

FIFTH EDITION



CONCISE
GUIDE TO

Child & Adolescent PSYCHIATRY

DSM-5
EDITION

Mina K. Dulcan, M.D.
Rachel R. Ballard, M.D.
Poonam Jha, M.D.
Julie M. Sadhu, M.D.

Concise Guide to Child and Adolescent Psychiatry

Fifth Edition

Concise Guide to Child and Adolescent Psychiatry

Fifth Edition

Mina K. Dulcan, M.D.

Rachel R. Ballard, M.D.

Poonam Jha, M.D.

Julie M. Sadhu, M.D.

AMERICAN
PSYCHIATRIC
ASSOCIATION

PUBLISHING



Note: The authors have worked to ensure that all information in this book is accurate at the time of publication and consistent with general psychiatric and medical standards, and that information concerning drug dosages, schedules, and routes of administration is accurate at the time of publication and consistent with standards set by the U.S. Food and Drug Administration and the general medical community. As medical research and practice continue to advance, however, therapeutic standards may change. Moreover, specific situations may require a specific therapeutic response not included in this book. For these reasons and because human and mechanical errors sometimes occur, we recommend that readers follow the advice of physicians directly involved in their care or the care of a member of their family.

Books published by American Psychiatric Association Publishing represent the findings, conclusions, and views of the individual authors and do not necessarily represent the policies and opinions of American Psychiatric Association Publishing or the American Psychiatric Association.

If you wish to buy 50 or more copies of the same title, please go to www.appi.org/specialdiscounts for more information.

Diagnostic criteria included in this book are reprinted, with permission, from *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition. Copyright © 2013, American Psychiatric Association.

Copyright © 2018 American Psychiatric Association Publishing

ALL RIGHTS RESERVED

Manufactured in the United States of America on acid-free paper

21 20 19 18 17 5 4 3 2 1

Fifth Edition

American Psychiatric Association Publishing

1000 Wilson Boulevard

Arlington, VA 22209-3901

www.appi.org

Typeset in Adobe's Times and Helvetica

Library of Congress Cataloging-in-Publication Data

Names: Dulcan, Mina K., author. | Ballard, Rachel, author. | Jha, Poonam, author. | Sadhu, Julie (Julie M.), author. | American Psychiatric Association Publishing, issuing body.

Title: Concise guide to child and adolescent psychiatry / by Mina K. Dulcan, Rachel R. Ballard, Poonam Jha, Julie Sadhu.

Description: Fifth edition. | Arlington, VA : American Psychiatric Association Publishing, [2018] | Includes bibliographical references.

Identifiers: LCCN 2017016412 (print) | LCCN 2017017260 (ebook) | ISBN 9781615371457 (ebook) | ISBN 9781615370788 (pbk. : alk. paper)

Subjects: | MESH: Mental Disorders | Adolescent | Child | Handbooks

Classification: LCC RJ499.3 (ebook) | LCC RJ499.3 (print) | NLM WS 39 | DDC 618.92/89—dc23

LC record available at <https://lcn.loc.gov/2017016412>

British Library Cataloguing in Publication Data

A CIP record is available from the British Library.

CONTENTS

List of Tables	xi
About the Authors	xv
Preface to the Fifth Edition	xvii

1 Introduction **1**

Change in Children and Adolescents	2
Overview of Diagnosis	3
Use of DSM-5 for Children and Adolescents	3
Comorbidity	4
Overview of Treatment	5

2 Evaluation and Treatment Planning **7**

Evaluation	7
Use of Multiple Informants	9
History From Parents	10
Patient Interview	10
Family Evaluation	12
Standardized Evaluation Instruments	14
Medical Evaluation	14
School Assessment	15
Psychological Testing	15
Treatment Planning	20
Feedback	22

3	Neurodevelopmental Disorders	25
	Intellectual Disability	25
	Communication Disorders	36
	Autism Spectrum Disorder	41
	Attention-Deficit/Hyperactivity Disorder	50
	Specific Learning Disorder	71
	Developmental Coordination Disorder	75
	Tic Disorders	76

4	Schizophrenia	87
----------	----------------------	-----------

5	Bipolar Disorder	93
----------	-------------------------	-----------

6	Depressive Disorders	101
	Disruptive Mood Dysregulation Disorder	101
	Major Depressive Disorder and Persistent Depressive Disorder	104

7	Anxiety Disorders	115
	Separation Anxiety Disorder	115
	Selective Mutism	125
	Social Anxiety Disorder (Social Phobia) and Specific Phobias	128
	Panic Disorder	133
	Generalized Anxiety Disorder	135

8	Obsessive-Compulsive and Related Disorders	145
	Obsessive-Compulsive Disorder	145
	Trichotillomania (Hair-Pulling Disorder)	150
	Excoriation (Skin-Picking) Disorder	151

9	Trauma- and Stressor-Related Disorders . .	155
	Reactive Attachment Disorder and Disinhibited Social Engagement Disorder	155
	Posttraumatic Stress Disorder and Acute Stress Disorder	158
	Adjustment Disorders	164

10	Feeding and Eating Disorders	171
	Pica	171
	Rumination Disorder	173
	Avoidant/Restrictive Food Intake Disorder	175
	Anorexia Nervosa and Bulimia Nervosa	177
	Anorexia Nervosa	177
	Bulimia Nervosa	185

11	Elimination Disorders	193
	Enuresis	193
	Encopresis	197

12	Sleep-Wake Disorders	203
	Evaluation of Sleep-Related Complaints	203
	Insomnia	205
	Narcolepsy	207
	Obstructive Sleep Apnea	208
	Circadian Rhythm Sleep-Wake Disorders	210
	Parasomnias	210
	NREM Sleep Arousal Disorders	210
	Nightmare Disorder	212
	Restless Legs Syndrome	213
	Psychiatric Disorders and Sleep Problems	214

13 Gender Dysphoria217

14 Disruptive, Impulse-Control, and Conduct Disorders225

Oppositional Defiant Disorder 225

Conduct Disorder 230

15 Substance-Related and Addictive Disorders243

16 Special Clinical Circumstances 255

Emergencies 255

Assessment and Triage 255

Suicide 260

Child Maltreatment 264

Out-of-Control Behavior 271

Family Transitions 274

Divorce 275

Physical Illness in Parents or Siblings 279

Death of a Family Member 280

Adoption 282

Cultural Factors for Immigrant Families 283

Adolescent Pregnancy 284

Obesity 286

Physically Ill Children And Adolescents 289

Developmental Factors in Reaction to Acute

Illness, Hospitalization, and Surgery 289

Chronic Illness 291

Adherence to Treatment 294

Treatment 295

Children of Psychiatrically Ill Parents 297

Risks and Resilience 297

Interventions 299

17 Psychopharmacology305

Special Issues for Children and Adolescents	305
Stimulant Medications	310
Atomoxetine	322
Guanfacine and Clonidine	324
Antidepressants	328
Lithium Carbonate	338
Antipsychotics	342
Anticonvulsants	356
Melatonin	361
Omega-3 Fatty Acids	362

18 Psychosocial Treatments373

Communication With Children and Adolescents	374
The Resistant Child or Adolescent	375
Types of Psychotherapy	375
Supportive Therapy	375
Psychodynamically Oriented Therapy	376
Time-Limited Therapy	377
Other Models of Therapy	378
Parent Counseling and Psychoeducation	380
Behavior Therapy	380
Parent Management Training	381
Classroom Behavior Modification	383
Family-Based Assessment and Treatment	383
Group Therapy	386
Systems of Care	389
Milieu Treatment: Inpatient Hospitalization, Residential Treatment, and Partial Hospitalization	390
Inpatient Hospitalization and Residential Programs	390
Partial Hospitalization	393

Adjunctive Treatments	393
Special Education Placements	394
Recreation	395
Foster Care	395
Parent Support Groups	396

Appendix: Resources for Parents	399
--	------------

Information on the Internet	399
Books	402

Index	407
--------------------	------------

LIST OF TABLES

1 Introduction

- 1-1 DSM-5 conditions relevant to children and adolescents that may be a focus of clinical attention . . . 4

2 Evaluation and Treatment Planning

- 2-1 Outline of biopsychosocial history 8
- 2-2 Dimensions of temperament 11
- 2-3 Temperament clusters 11
- 2-4 Mental status examination. 12
- 2-5 Family developmental tasks 13
- 2-6 Examples of standardized diagnostic assessment interviews 15
- 2-7 Individually administered tests of intellectual capacity, learning, and adaptive functioning 16

3 Neurodevelopmental Disorders

- 3-1 Clinical features of intellectual disability 27
- 3-2 Psychosocial causes of intellectual disability 30
- 3-3 Biological causes of intellectual disability. 31
- 3-4 Suggested medical contributions to etiology of attention-deficit/hyperactivity disorder 57
- 3-5 Child Attention Problems (CAP) Rating Scale 65
- 3-6 Child Attention Problems (CAP) Rating Scale scoring 66
- 3-7 DSM-5 specific learning disorders 72

6	Depressive Disorders	
6-1	Developmental differences in DSM-5 criteria for depressive disorders	105

7	Anxiety Disorders	
7-1	Common normal fears	116
7-2	Causes of school absenteeism	119

10	Feeding and Eating Disorders	
10-1	Physical signs and symptoms and complications associated with anorexia nervosa and bulimia nervosa	180
10-2	Differential diagnosis of anorexia nervosa	183

11	Elimination Disorders	
11-1	Medical causes of urinary incontinence	195

14	Disruptive, Impulse-Control, and Conduct Disorders	
14-1	DSM-5 symptom criteria for oppositional defiant disorder and suggested epidemiological cutoff frequencies	227
14-2	Common psychological characteristics of children and adolescents with conduct disorder . . .	232

15	Substance-Related and Addictive Disorders	
15-1	Risk factors associated with serious substance abuse in adolescence	247

16 Special Clinical Circumstances

- 16-1 Common psychiatric emergencies in
children and adolescents 256
- 16-2 Outline of emergency history. 257
- 16-3 Psychiatric emergency medical evaluation 259
- 16-4 Options for emergency dispositions. 259
- 16-5 Risk factors for repeat suicide attempt. 263
- 16-6 Causes of out-of-control behavior in
children and adolescents 272

17 Psychopharmacology

- 17-1 Stimulant preparations 311
- 17-2 Clinical effects of stimulant medications. 315
- 17-3 Side effects of stimulant medications 320
- 17-4 Antidepressant medications most often used
in children and adolescents 329
- 17-5 Antipsychotic medications 344

18 Psychosocial Treatments

- 18-1 Common themes of individual therapies 376

ABOUT THE AUTHORS

Mina K. Dulcan, M.D.

Margaret C. Osterman Professor of Child Psychiatry; Head, Department of Child and Adolescent Psychiatry, Ann & Robert H. Lurie Children's Hospital of Chicago; Professor of Psychiatry and Behavioral Sciences and Pediatrics and Chief, Child and Adolescent Psychiatry, Northwestern University Feinberg School of Medicine, Chicago, Illinois

Rachel R. Ballard, M.D.

Assistant Professor of Psychiatry and Behavioral Sciences and Pediatrics, Northwestern University Feinberg School of Medicine, Chicago, Illinois; Attending Psychiatrist, Department of Child and Adolescent Psychiatry, Ann & Robert H. Lurie Children's Hospital of Chicago

Poonam Jha, M.D.

Assistant Professor of Psychiatry and Behavioral Sciences, Northwestern University Feinberg School of Medicine, Chicago, Illinois; Attending Psychiatrist, Department of Child and Adolescent Psychiatry, Ann & Robert H. Lurie Children's Hospital of Chicago

Julie M. Sadhu, M.D.

Director of Education in Child and Adolescent Psychiatry and Assistant Professor of Psychiatry and Behavioral Sciences, Northwestern University Feinberg School of Medicine, Chicago, Illinois; Attending Psychiatrist, Department of Child and Adolescent Psychiatry, Ann & Robert H. Lurie Children's Hospital of Chicago

Disclosure of Competing Interests

The following contributors to this book have indicated a financial interest in or other affiliation with a commercial supporter, a manufacturer of a commercial product, a provider of a commercial service, a nongovernmental organization, and /or a government agency, as listed below:

Mina K. Dulcan, M.D.—*Advisor:* Ironshore Pharmaceuticals; *Editorial Board:* Care Management Technologies; *Royalties:* American Psychiatric Association Publishing.

Julie M. Sadhu, M.D.—*Member:* Child and Adolescent Psychiatry Maintenance of Certification Committee, American Board of Psychiatry and Neurology; *Member:* Assessments Committee, American Association of Directors of Psychiatric Residency Training.

The following authors of this book have reported no competing interests during the year preceding manuscript submission:

Rachel R. Ballard, M.D.

Poonam Jha, M.D.

PREFACE TO THE FIFTH EDITION

The fourth edition of *Concise Guide to Child and Adolescent Psychiatry* was published in 2012. In 2013, DSM-5 made significant changes to psychiatric nomenclature and criteria, stimulating the creation of this new edition. The field of pediatric mental health continues to grow and develop. The body of clinical research and empirically supported treatments has grown, evaluation practices have been refined, awareness of emotional and behavioral problems in youth has increased, progress has been made in decreasing the stigma of mental illness and obtaining parity for insurance coverage, and the lay media continue to be flooded with controversies regarding psychiatric disorders and their treatments.

Findings that experts agree are relevant to clinical practice have been distilled and incorporated into this fifth edition. Older content has been pruned. Each section of the book has been revised and updated, and the diagnostic chapters have been reorganized to match DSM-5. The American Psychiatric Association has graciously permitted the reprinting of diagnostic criteria from DSM-5.

■ ACKNOWLEDGMENTS

MaryBeth Lake, who made important contributions to the third and fourth editions, graciously gave an opportunity for three other child and adolescent psychiatrists to coauthor this book. We are deeply grateful to the child and adolescent psychiatry fellows and the fac-

ulty and staff in the Department of Child and Adolescent Psychiatry at Ann & Robert H. Lurie Children's Hospital of Chicago, who inspire us to learn more and who teach us every day.

INTRODUCTION

The *Concise Guide to Child and Adolescent Psychiatry*, 5th Edition, offers an introduction to mental health care in children and adolescents to be used in conjunction with clinical supervision and consultation. This book was written to be used as a primer on child and adolescent psychiatry for medical and other health or mental health students or clinical trainees and as a brief update for practicing physicians, nurses, and advanced practice nurses in general psychiatry, child and adolescent psychiatry, pediatrics, neurology, and family medicine. It may also be useful for professionals in special education, child welfare, and juvenile justice, as well as parents. In the interest of brevity, complex theoretical notions, new research, and areas of controversy have been simplified. Each section on a disorder or clinical situation includes a listing of relevant treatment methods. Treatment techniques are described in Chapters 17 (“Psychopharmacology”) and 18 (“Psychosocial Treatments”). Each chapter has suggested additional reading for those who wish more detail. For more in-depth, comprehensive coverage of child mental health, see *Dulcan’s Textbook of Child and Adolescent Psychiatry*, 2nd Edition (Dulcan 2016). The American Academy of Child and Adolescent Psychiatry practice parameters—clinical guidelines that are based on the scientific literature and the wisdom of experienced clinicians—are published in the *Journal of the American Academy of Child and Adolescent Psychiatry*. They are cited in the corresponding chapters.

Throughout this book, *children* refers to both prepubertal children and adolescents, unless otherwise specified. *Parent* is used for the child’s primary adult caregiver(s), whoever that may be.

■ CHANGE IN CHILDREN AND ADOLESCENTS

The primary “work” of children is to grow and change in multiple dimensions. Rigid descriptions of mental disorders and psychiatric symptoms do not capture the liveliness and energy of children as they develop and cope with internal and external difficulties. A child’s tendency to change is a therapeutic ally. As clinicians, we work with the natural dynamics of the interaction among our interventions, environmental influences, and developmental processes.

Disorders in childhood can exert lasting effects beyond the primary psychiatric disorder. Developmental complications are often cumulative and may disrupt a wide range of functions. Social, cognitive, and psychological development, and even physical growth (Pine et al. 1996), may be impaired. Progressive learning delays, school failure, low self-esteem, demoralization, impaired relationships with family members, and rejection or neglect by peers are common complications of childhood-onset disorders. Prompt intervention can reduce these developmental consequences.

Regardless of the etiology of the primary disorder, biological, cognitive, psychological, familial, social, economic, and cultural factors are critical in determining the course of illness. Genes can interact with environment to increase vulnerability or to reduce risk. The effects of early developmental deficits may be compensated for or exacerbated by later opportunities or barriers. The family or social environment can amplify strengths or aggravate weaknesses. The adult outcome of a childhood disorder in a specific patient is a result of the interaction among therapeutic forces and risk and protective factors. The ultimate prognosis may depend more on the ability of the child and family to learn to cope with the illness than on the severity of the disorder. Resilient individuals may even turn childhood symptoms such as excessive sensitivity (separation anxiety disorder), unrelenting stubbornness (oppositional defiant disorder), or uncontrolled activity and enthusiasm (attention-deficit/hyperactivity disorder) into strengths in adulthood. Compensatory abilities and an enhancing environment can result in achievement and adaptation far above that predicted from early deficits.

■ OVERVIEW OF DIAGNOSIS

Use of DSM-5 for Children and Adolescents

DSM-5 (American Psychiatric Association 2013) introduced significant changes in the organization of disorders. No longer are some disorders of youth relegated to a separate chapter. The new neurodevelopmental disorders chapter includes many disorders that typically are first diagnosed in childhood. The former DSM multi-axial structure has been eliminated, and all psychiatric and “other medical diagnoses” are placed in a single list. Any diagnosis can be used for a child if the criteria are met. A few disorders have slightly adapted criteria for children. Some disorders include behavior or emotions that may be normal at certain developmental stages but become pathological if they persist (becoming, e.g., separation anxiety disorder, enuresis, encopresis, or oppositional defiant disorder). Most disorders, however, are not “normal” at any age. In this book, key DSM-5 criteria for disorders are highlighted. For full details, refer to DSM-5 (American Psychiatric Association 2013).

DSM-5 contains an expanded list of clinical circumstances, classified as “V codes” or “Other Conditions That May Be a Focus of Clinical Attention,” that are not psychiatric diagnoses but may prompt assessment and treatment. They include psychosocial and environmental problems relevant to diagnosis, treatment, and prognosis. Many of these codes are especially relevant to children, and the major categories are listed in Table 1–1 (see DSM-5 for detailed list).

All DSM-5 diagnoses (except for tic disorders) require evidence that the symptoms are causing significant distress or impairment in social, academic, and/or occupational functioning.

“Other” medical conditions (new terminology to indicate that psychiatric disorders are also medical conditions) are now included in the single list of the patient’s diagnoses. Many medical problems, from a fever or earache to a brain tumor, may be signaled first by behavioral or emotional symptoms or declining school performance. Children with chronic medical disorders or physical disabilities are at increased risk for psychiatric disorders (see Chapter 16).

TABLE 1-1. **DSM-5 conditions relevant to children and adolescents that may be a focus of clinical attention**

Relational problems

Problems related to family upbringing

Other problems related to primary support group

Abuse and neglect

Child maltreatment and neglect problems

Child physical abuse

Child sexual abuse

Child neglect

Child psychological abuse

Educational and occupational problems

Housing and economic problems

Other problems related to the social environment

Problems related to crime or interaction with the legal system

Problems related to other psychosocial, personal, and environmental circumstances

Other circumstances of personal history (includes child or adolescent antisocial behavior)

Nonadherence to medical treatment

Borderline intellectual functioning

Source. American Psychiatric Association 2013.

Replacing the former Axis V, DSM-5 includes separate measures of symptom severity and disability for individual disorders.

Comorbidity

Psychiatric disorders typically occur in combinations. This is true not only in young people treated in the most intensive settings, such as inpatient units and residential treatment facilities, but also in outpatients. Comorbidity is common even in community epidemiological surveys, although less so than in children who have been referred for clinical services. Combinations of disorders may be unexpected, such as a teenager with impulsive hyperactivity or aggressive conduct problems who also suffers from depression or anxiety. Broad-

spectrum rating scales are a useful supplement to the diagnostic process, to avoid overly narrow focus on the presenting problem.

■ OVERVIEW OF TREATMENT

Contemporary treatment planning includes use of evidence-based interventions selected with consideration of biological, psychological, and social factors; multimodal treatment approaches; and multidisciplinary teams. As diagnostic criteria have been refined, evaluation techniques have become more precise, both therapy models and medications have been more rigorously designed and tested for specific disorders or symptoms, and treatment selection has a stronger empirical foundation.

In contrast to treatment in adults, a child is typically brought to the clinical setting by someone else. Although the child is identified as “the patient,” each case has at least two clients: a parent or guardian and the child, whose needs and goals may conflict. In addition, treatment often involves other family members; teachers and school counselors; government agencies, such as child protective services or the juvenile court; community organizations; and financial providers, such as Medicaid or insurance companies, and state-funded services. Because children depend on adults for their basic needs and have little autonomy in choosing caregivers, residence, schools, or activities, the clinician must work in partnership with the parents and other support systems to reestablish developmental progress and maximize adaptive outcome.

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Dulcan MK (ed): Dulcan’s Textbook of Child and Adolescent Psychiatry, 2nd Edition. Arlington, VA, American Psychiatric Association Publishing, 2016

Pine DS, Cohen P, Brook J: Emotional problems during youth as predictors of stature during early adulthood: results from a prospective epidemiologic study. *Pediatrics* 97(6, Pt 1):856–863, 1996 8657527

■ ADDITIONAL READING

Hilt RJ, Nussbaum AM: *DSM-5 Pocket Guide for Child and Adolescent Mental Health*. Arlington, VA, American Psychiatric Association Publishing, 2016

EVALUATION AND TREATMENT PLANNING

■ EVALUATION

A comprehensive evaluation includes obtaining a biopsychosocial history (Table 2–1); performing a mental status examination; ordering any additional tests; and obtaining records (with parental permission) from the child's school, pediatrician, and agencies such as child protective services or the juvenile court. The clinician should request reports of all prior psychiatric, psychological, developmental, and medical evaluations and treatment. Assessment should continue throughout the course of treatment as the child, parents, and situation change. When the presenting problems are urgent or narrowly circumscribed, treatment may be initiated after a focused evaluation with a more detailed assessment completed as time permits.

Before the evaluation, the clinician should tell the parents how long the evaluation will take, what it will cost, and what they can expect at the end. The clinician should advise parents on how to prepare the child for the first visit. Some parents invite the child out for an ice-cream cone but bring him or her to the child psychiatry clinic instead. Needless to say, this does not enhance the child's cooperation, although it does provide the clinician with useful data about the parents.

In conducting an evaluation, the clinician must constantly be mindful of the patient's developmental level, which may determine whether a behavior (e.g., temper tantrums, separation anxiety) is pathological. Developmental stage influences the nature of symptoms, expectable reactions to stressors, ability to communicate and

TABLE 2-1. Outline of biopsychosocial history

Chief symptom and reasons for referral**History of present illness**

- Development of the symptoms
- Attitudes of child and parents toward the symptoms
- Effects of the symptoms on the child and family
- Stressors
- Prior psychological or psychiatric evaluations
- Prior treatment
 - Psychotherapy: type, frequency, duration, effects
 - Medication: exact doses, schedule, beneficial and adverse effects
 - Intensive treatments such as hospitalization or residential placement
 - Environmental changes and effects

Current developmental status

- Motor abilities and activity level
- Attention
- Speech and language
- Academic performance
- Relationships with peers
- Risk-taking behaviors
- Sexual development and behavior
- Hobbies, activities, athletic interests and skills
- Relationships with family members and other significant adults

Review of behavioral and psychological symptoms**Medical review of systems****Past history**

- Psychiatric
- Medical
- Neurological

Developmental history

- Pregnancy and delivery
- Neonatal period, infancy, early childhood
- Pubertal status
- Sexual interests and activities (for adolescents)
- Temperament

 TABLE 2-1. **Outline of biopsychosocial history (*continued*)**

Developmental history (*continued*)

Milestones

Motor

Cognitive

Speech and language

Social

Education history

Traumatic events

Psychosocial and psychiatric history of each parent**Developmental history of the couple/family life cycle stage****Family medical history****Current family circumstances, concerns, liabilities, resources**

to understand concepts, and capacity to participate in different types of treatment.

The structure of an evaluation is determined by the child's age, the nature of the presenting problems, and practical factors. It is often useful to begin by meeting briefly with the child and parents together, to clarify and understand each person's views of the presenting problems and the goals of the evaluation and to develop an initial impression of family interaction. For children younger than 6 years, it may be convenient to obtain the entire history from the parents before seeing the child. Older children, especially adolescents, should be involved early in the evaluation process. Adolescents are often concerned about confidentiality and should be told that what they say will be shared only with their permission, unless they are at risk for physical harm, such as from suicide, homicide, substance abuse, high-risk sexual behavior, or running away. Clinicians are legally mandated to report suspected physical or sexual abuse to child protection authorities.

Use of Multiple Informants

No single informant or technique can give a full description of a child. Children themselves, teachers, parents, other relatives, com-

munity members, and clinicians each have their own point of view and opportunities for observation. Lack of agreement between parents and teachers or between two parents often results from genuine variations in the behavior of children in different settings and with different people. Ideally, both parents should be interviewed, even if they are not living together, because their cooperation will enhance the likelihood of successful treatment, and each parent has a unique perspective on the child's development and environment. A telephone interview may be substituted if a parent is not available in person.

History From Parents

The elements of a complete history from the parents or caregivers are outlined in Table 2-1. The construction of a time line, including symptoms, life events, and changes in the family and environment, can help organize a complex history.

An important element in the developmental history is the child's *temperament*, or "style" of behavior. Children can be rated on each of the nine dimensions listed in Table 2-2. The *goodness of fit* between the child's temperament and the parents' temperament, expectations, and child-rearing skills significantly affects developmental course and outcome. In addition, certain trait clusters (Table 2-3) have predictive value. Both *difficult* and *slow-to-warm-up* children are at risk for emotional and behavior problems, whereas *easy* children are relatively protected.

Patient Interview

During the patient interview, the child provides his or her view of the history and his or her current symptoms, strengths, and concerns while the clinician makes observations. Children and adolescents often report their anxiety and depression, clandestine conduct problems, and drug use more accurately than their parents do. Parents typically report history, observable behavior problems, and family background more accurately than the child does. The details of the interview vary with developmental stage. The content of the mental status examination is outlined in Table 2-4.

TABLE 2-2. Dimensions of temperament

1. Activity level
 2. Rhythmicity (regularity and predictability of biological functions)
 3. Approach to or withdrawal from novel stimuli
 4. Adaptability to environmental change
 5. Intensity of reaction
 6. Threshold of responsiveness (intensity of stimulation required to evoke a response)
 7. Quality of mood (positive, neutral, or negative)
 8. Distractibility
 9. Attention span and persistence
-

Source. Adapted from Thomas A, Chess S: *Temperament in Clinical Practice*. New York, Guilford, 1986.

TABLE 2-3. Temperament clusters

Easy

- Positive mood
- Regular biological rhythms
- Adaptable
- Low intensity of reactions
- Positive approach to novelty

Difficult

- Negative mood
- Irregular biological rhythms
- Slow to adapt
- Intense reactions
- Negative response to novelty

Slow to warm up

- Gradual adaptation after repeated contact
 - Mild intensity of reactions
 - Negative response to novelty
-

Source. Adapted from Thomas A, Chess S: *Temperament in Clinical Practice*. New York, Guilford, 1986.

TABLE 2–4. Mental status examination

Physical appearance and grooming
Interactions with clinician
Understanding of the purpose of the interview
Motor activity level and coordination
Tics, stereotypies, mannerisms
Mood and affect
Anxiety
Obsessions or compulsions
Attention, persistence, frustration tolerance
Impulsivity
Oppositionality
Verbal or physical aggression
Speech and language
Hallucinations, delusions, thought disorder
Clinical estimate of intelligence
Judgment and insight

The therapist should begin the patient interview by informally discussing nonthreatening topics before focusing on the presenting symptoms. For patients who are not very verbal, an opportunity to draw (with pencils, crayons, or washable markers) can help them feel comfortable enough to engage in conversation. Young children initially may want the parent to be present and may be more comfortable with play materials (e.g., dollhouse, stuffed animals, blocks, clay) than with a formal interview. Asking children direct questions about their symptoms is not harmful and will not create new symptoms that the child does not already have. Clinicians need to ask children direct questions (using wording that is adapted to the child's developmental level) to understand their emotional states.

Family Evaluation

Each person living with the patient, as well as noncustodial parents, grandparents, and siblings who are no longer living at home, may be crucial to understanding family dynamics, including both unex-

TABLE 2–5. Family developmental tasks

Forming a “marital coalition” to meet the needs of the adults for intimacy, sexuality, and emotional support

Establishing a “parental coalition” to form flexible relationships with children and present a consistent disciplinary front

Providing for nurturance, enculturation, and emancipation of children

Coping with crises

Source. Adapted from Fleck S: “A General Systems Approach to Severe Family Pathology.” *American Journal of Psychiatry* 133:669–673, 1973.

pected sources of emotional support and areas of conflict. Meeting simultaneously with all significant family members to collect information and to observe interactions is often useful. Families with young children may benefit from the use of role playing, family drawings, or puppet play. The clinician may ask the family to complete a task during the session in order to assess family interactions. A family tree or genogram helps the clinician to organize data on family members and their relationships.

Regardless of a family’s structure or members, the well-being of the child requires that certain tasks be accomplished (Table 2–5). Well-functioning families respond resiliently to stress, communicate effectively, assign roles that suit the needs and abilities of each family member, respond appropriately to emotions, solve problems both within and outside the boundaries of the family, and find effective and humane ways to control the behavior of family members.

Tasks of the initial session of a family assessment include the following:

- Ascertain each family member’s view of the problem.
- Begin to establish a relationship with each family member to facilitate the treatment alliance.
- Gather data by observation and with questions.
- Make test interventions and assess their effects.
- Propose and negotiate a provisional plan for the next steps.

Mental health care for young people is unlikely to succeed without considering their parents' needs. Parents struggling with their own untreated psychiatric disorders may be unable to meet the child's emotional and physical needs, and there may be a "contagious" effect on the child. The clinician's most important contribution may be to arrange for the parent to receive psychiatric assessment and pharmacological and/or psychotherapeutic treatment. The clinician may need to use both empathy and judicious persuasion to induce the parent to assume a "patient" role.

Standardized Evaluation Instruments

Selected parent, teacher, and self-report behavior checklists, questionnaires, and rating scales supplement the clinical evaluation by providing a systematic review of behaviors and psychiatric symptoms. Scores may be compared with norms obtained from large community-based samples or groups of clinically referred children. Ratings may also be done at intervals to measure progress. The most commonly used broad-spectrum package consists of the Child Behavior Checklist (CBCL) parent rating form, the Teacher Report Form (TRF), and the Youth Self-Report (YSR) Form (Achenbach System of Empirically Based Assessment; <http://www.aseba.org>). More specific instruments are available for one or more diagnoses. Structured and semistructured diagnostic interview protocols (see Table 2–6) are more commonly used in research but often have clinical usefulness.

Medical Evaluation

A standard evaluation includes a medical history and physical examination (within the past year or more recently if the onset of problems is acute) to identify any medical causes of symptoms or co-existing medical disorders. Neurological consultation or testing (e.g., electroencephalogram, brain scan) is indicated only if focal neurological signs or symptoms are present or if the history suggests seizures, loss of cognitive or physical skills, or sequelae of brain injury. Laboratory tests may be obtained, especially if phar-

TABLE 2–6. **Examples of standardized diagnostic assessment interviews**

Structured diagnostic interview

Diagnostic Interview Schedule for Children (DISC)

Semistructured diagnostic interviews

Child and Adolescent Psychiatric Assessment (CAPA)

Diagnostic Interview for Children and Adolescents (DICA)

Schedule for Affective Disorders and Schizophrenia for School-Aged Children and Adolescents (Kiddie-SADS)

Note. For references and more detail, see “Diagnostic Interviews” (Chapter 7) in *Dulcan’s Textbook of Child and Adolescent Psychiatry* (Carlisle and McClellan 2010).

macological treatment is anticipated. Urine testing for drugs and, in female adolescents, a pregnancy test are often indicated. Scientific evidence does not support the use of brain imaging in clinical diagnosis or treatment of psychiatric disorders in youth.

School Assessment

School reports are almost always useful. Attention, learning, quality and quantity of homework and classwork, behavior in class and on the playground, and social relationships are sensitive indicators of the presence of psychiatric symptoms and of developmental status. After obtaining parental consent, the clinician may talk with the teacher or school counselor, obtain school records (educational testing, behavior, grades, and attendance), arrange for teachers to complete standardized checklists, and perhaps even visit the school to observe the youngster.

Psychological Testing

Standardized tests administered by a clinical psychologist are used to assess intellectual potential, cognitive skills, fund of knowledge, and adaptive functioning (Table 2–7). Individually administered tests provide a more accurate evaluation than those given to chil-

TABLE 2-7. **Individually administered tests of intellectual capacity, learning, and adaptive functioning**

Intelligence

Differential Ability Scales, Second Edition (DAS-II)	2.6–17 years Subtests estimate General Cognitive Ability (GCA) and the subdomains of Verbal, Nonverbal, and Spatial Ability
Kaufman Adolescent and Adult Intelligence Test (KAIT)	11 years through adulthood Distinguishes between learned information and capacity to solve novel problems
Kaufman Assessment Battery for Children, Second Edition (K-ABC-II)	3–18 years Subtests estimate general Mental Processing and the subdomains of Sequential and Simultaneous Processing, Learning, Planning, and Knowledge
Kaufman Brief Intelligence Test, Second Edition (KBIT-2)	4 years through adulthood Screening test to estimate IQ
Leiter International Performance Scale—Third Edition	3 years through adulthood Nonverbal test for use with persons who are hearing impaired or autistic or who do not speak English
Peabody Picture Vocabulary Test, Fourth Edition (PPVT-4)	2.6 years through adulthood Brief test of receptive vocabulary used to estimate verbal intelligence
Shipley-2	7 years through adulthood Brief pencil-and-paper test that does not require a psychologist to administer Subtests estimate general cognitive functioning and the subdomains of Crystallized/Verbal and Fluid/Nonverbal cognitive ability

TABLE 2-7. **Individually administered tests of intellectual capacity, learning, and adaptive functioning (*continued*)**

Intelligence (*continued*)

Stanford-Binet Intelligence Scale, Fifth Edition	2 years through adulthood Subtests estimate Verbal IQ, Nonverbal IQ, and Full Scale IQ, as well as the subdomains of Fluid Reasoning, Knowledge, Quantitative Reasoning, Visual Spatial Processing, and Working Memory
Universal Nonverbal Intelligence Test, Second Edition (UNIT-2)	5-21 years Subtests estimate Full Scale IQ and the subdomains of Memory, Reasoning, Symbolic, and Quantitative Ability
Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II)	6 years through adulthood Both 4-subtest and 2-subtest versions estimate Full Scale IQ; 4-subtest version also estimates the Verbal Comprehension Index and Perceptual Reasoning Index
Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV)	16 years through adulthood Subtests estimate Full Scale IQ and the subdomains of Verbal Comprehension, Perceptual Reasoning, Working Memory, and Processing Speed
Wechsler Intelligence Scale for Children, Fifth Edition (WISC-V)	6-16 years Subtests estimate Full Scale IQ and the subdomains of Verbal Comprehension, Visual Spatial, Fluid Reasoning, Working Memory, and Processing Speed
Wechsler Preschool and Primary Scale of Intelligence, Fourth Edition (WPPSI-IV)	2.6-7.7 years Subtests estimate Full Scale IQ and the subdomains of Verbal Comprehension, Visual Spatial, Working Memory, Fluid Reasoning, and Processing Speed

TABLE 2–7. **Individually administered tests of intellectual capacity, learning, and adaptive functioning (*continued*)**

Intelligence (*continued*)

Woodcock-Johnson Tests of Cognitive Ability, Fourth Edition (WJ IV)	2 years through adulthood Frequently used by schools Subtests estimate General Intellectual Ability and the subdomains of Comprehension Knowledge, Fluid Reasoning, Short-Term Working Memory, and Cognitive Efficiency
---	---

Academic achievement

Kaufman Test of Educational Achievement, Third Edition (KTEA-3)	4 years through adulthood Subtests estimate academic mastery of reading, writing, math, and oral language skills
Wechsler Individual Achievement Tests, Third Edition (WIAT-III)	4–50 years Subtests estimate academic mastery of reading, writing, math, and oral language skills
Wide Range Achievement Test, Fourth Edition (WRAT-4)	5 years through adulthood Subtests screen for academic mastery of reading, writing, math, and oral comprehension skills
Woodcock-Johnson Tests of Achievement, Fourth Edition (WJ IV)	2 years through adulthood Subtests estimate academic mastery of reading, writing, math, academic knowledge, and oral language skills

TABLE 2–7. **Individually administered tests of intellectual capacity, learning, and adaptive functioning** (*continued*)

Adaptive behavior (required to diagnose intellectual disability)

Adaptive Behavior Assessment System, Third Edition (ABAS-III)	Birth through adulthood Estimates overall adaptive functioning (General Adaptive Composite) and functioning in the subdomains of Conceptual, Social, and Practical skills Parent and teacher report forms
Vineland Adaptive Behavior Scales, Second Edition	Birth through adulthood Semistructured interview with parent (or brief written form for teacher) Estimates overall adaptive functioning (Adaptive Behavior Composite) and functioning in the subdomains of Communication, Daily Living Skills, Socialization, and Motor Skills
Woodcock-Johnson Scales of Independent Behavior—Revised (SIB-R)	Birth through adulthood Semistructured interview with caregiver Estimates overall adaptive functioning (Broad Independence Index) and functioning in the subdomains of Motor, Social Interaction and Communication, Personal Living, and Community Living skills

dren in the classroom. Tests have been criticized because of cultural influences on performance, unresponsiveness to “creative” responses, the dangers of using a rigid construct of intelligence that masks individual strengths, the use of test results to exclude children from mainstream education, and potential insults to developing self-esteem. Despite these concerns, IQ tests provide a global assessment that has clinical predictive value, particularly when combined with an evaluation of adaptive behavior. Projective tests such as the Children’s Apperception Test (CAT) or the Rorschach Inkblot Technique are not useful in making diagnostic or treatment decisions. The most commonly used test for assessing infants (ages birth to 3 years) is the Bayley Scales of Infant Development, Third Edition. It is used to evaluate motor (fine and gross), language (receptive and expressive), and cognitive development.

In neuropsychological testing, a neurodiagnostic battery of tests is used to provide a detailed assessment of a patient’s cognitive strengths and weaknesses and compare them with patterns seen in individuals with developmental, neurological, or other medical conditions. It can assist in the diagnosis, localization, and monitoring of neurodevelopmental, neurodegenerative, or acquired disorders of brain functioning or adverse effects of treatments such as cranial radiation and chemotherapy. Neuropsychological testing is not indicated as a diagnostic procedure for attention-deficit/hyperactivity disorder, although it can assist in the assessment of specific learning disabilities.

■ TREATMENT PLANNING

Treatment plans are based on both psychiatric diagnosis and identified target symptoms. The strengths and vulnerabilities of the patient and the resources and liabilities of the family are critical factors in treatment planning. The social environment, including school, neighborhood, and social support networks, strongly influences choice of treatment strategy. The practical realities of the quality and availability of community resources and the family’s ability to pay for or

to attend treatment sessions often compel the clinician to modify an “ideal” or comprehensive plan. Realistic and efficient selection and sequencing of treatment modalities are central to effective decision making. Clinical judgments regarding anticipated treatment effectiveness, efficiency, and risk–benefit ratio may lead to selection of a single form of treatment or multimodal therapies. Interventions may be administered simultaneously or sequentially, as the child or family requires or is able to make use of additional treatment.

Parental motivation or ability to carry out the treatment plan may strongly influence treatment decisions. For example, unusual strengths of a family may avert hospitalization of a psychotic or suicidal child or family limitations may prevent implementation of family therapy or maintenance of the child living at home. For children from disrupted homes or abusive or neglectful environments, the first priority may not be psychiatric care, but social services, such as a safe and stable home, food, supervision, and medical care. In complex cases, a case manager who coordinates the involvement of various agencies and services (sometimes called “wraparound”) may be able to maintain a child in the community, which may result in a better outcome at a lower overall cost.

Each of a child’s symptoms may appear to call for a different intervention. In setting priorities, factors to consider include the following:

- Risk of physical harm to the child or to others
- Symptoms that will likely increase in severity and chronicity if not treated rapidly (e.g., school avoidance)
- Patient and family motivation and resistance
- Symptoms or family members that are most accessible to treatment
- Problems that are most urgent to the patient or family

Treatment planning is an ongoing process. Continual reassessment is necessary as the effects of interventions are seen and as additional information about the child and family comes to light.

■ FEEDBACK

The clinician's findings and recommendations are typically presented to both the parents and the child. The clinician decides whether to meet with them together or separately, and in what order. Depending on the ability of each to listen or understand, parents and child are educated about the nature of the child's and the family's strengths, psychiatric liabilities or disorders (including DSM-5 diagnoses), and the expected course and possible complications of the disorder. The clinician should answer questions about etiology at the level that scientific knowledge allows, while assiduously avoiding blaming or being judgmental. Parents and many children already feel guilty about real or perceived failures, and the empathic clinician is cautious to ameliorate these feelings. It is important to address possible opinions and information (which may or may not be correct) that parents have learned from relatives, friends, and the Internet.

Parents, and usually the child, should help determine which treatment strategy to follow. The clinician describes recommended and alternative interventions in terms of the process (duration, costs) and the anticipated benefits and risks. A successful feedback conference helps the family to understand their strengths and weaknesses, respect their child's abilities and the difficulties he or she faces, sense the interplay of multiple etiological factors, realize the implications of the child's diagnosis and prognosis, ponder the practicalities involved, acknowledge hopes and fears, and integrate the recommendations with the rest of their lives. Even the best treatment has little chance of success without the cooperation of the family and the child (to the extent possible for developmental stage and psychopathology). The treatment plan should be consistent with the family's resources. Finding areas in which improvement may be quickly attained builds the family's confidence in themselves and in the therapeutic process.

■ REFERENCE

Carlisle L, McClellan JM: Diagnostic interviews, in Dulcan's Textbook of Child and Adolescent Psychiatry. Edited by Dulcan MK. Washington, DC, American Psychiatric Publishing, 2010, pp 79–88

■ ADDITIONAL READING

Caplan R, Bursch B: "How Many More Questions?": Techniques for Clinical Interviews of Young Medically Ill Children. New York, Oxford University Press, 2013

Cepeda C, Gotanco L: Psychiatric Interview of Children and Adolescents. Arlington, VA, American Psychiatric Association Publishing, 2017

Dulcan MK (ed): Part I: Assessment and Diagnosis, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 3–104

Gerson R, McGoldrick M, Petry S: Genograms: Assessment and Intervention, 3rd Edition. New York, WW Norton, 2008

NEURODEVELOPMENTAL DISORDERS

Neurodevelopmental disorders form a new section in DSM-5 (American Psychiatric Association 2013), grouping conditions with very early onset. Consistent with the overall organization of DSM-5, they are all placed in the single list of all diagnoses. The neurodevelopmental disorders are characterized by a variety of developmental deficits that lead to impairment in functioning in multiple domains. The disorders included are *intellectual disabilities*, *communication disorders*, *autism spectrum disorder*, *attention-deficit/hyperactivity disorder*, *specific learning disorder*, and *motor disorders*.

■ INTELLECTUAL DISABILITY

Clinical Description

DSM-5 replaced the diagnosis of mental retardation (coded on Axis II in DSM-IV [American Psychiatric Association 2000]) with the term *intellectual disability* (also referred to as *intellectual developmental disorder*, the ICD-11 term). Intellectual disability was the preferred nomenclature used by many experts and advocates since the mid 2000s, as well as in a federal statute. The DSM-5 diagnosis of intellectual disability (ID) requires deficits in general mental abilities; deficits in adaptive functioning compared with peers of similar age, gender, and culture; and onset in the developmental period. Clinical assessment and standardized testing are both necessary in making the diagnosis of ID. Individuals with ID have IQ scores of two

or more standard deviations below the mean (IQ of 65–75 or lower). Adaptive functioning is subdivided into three domains: conceptual (language, math, reading, writing, reasoning, knowledge, memory); social (empathy, social judgment, interpersonal communication, awareness of social and legal rules, friendships); and practical (abilities in self-care, employment, money management). Severity levels of ID are now classified by adaptive functioning rather than IQ, as was done in DSM-IV. Clinical features of the four levels of severity are listed in Table 3–1.

Epidemiology

Prevalence of ID in the United States ranges from 1% to 3%, depending on age, how ID is defined, methods used for diagnosis, and the population being evaluated. All levels of severity of ID are more common in males (approximately 30% more common in males), but this difference decreases as severity of ID increases.

Comorbidity

Of children and adolescents with ID, 30%–50% have a comorbid psychiatric disorder (Munir 2016). This rate is significantly higher than in youngsters of normal intelligence, in part because neurobiological and psychosocial causes of ID place the child at greater risk of psychiatric disorder. Almost every diagnostic category in psychiatry is represented. Prevalence estimates of comorbid conditions vary because of the challenge of distinguishing symptoms due to ID from those of a separate condition. Factors that increase the risk of psychiatric comorbidity include severity of disability, lower adaptive functioning, language impairment, poor socialization, lower socioeconomic status, and being cared for by a single parent.

The most common presentation is a constellation of symptoms that includes impulsivity, irritability, hyperactivity, short attention span, and language delay. The prevalence of attention-deficit hyperactivity disorder (ADHD) in individuals with ID is between 4% and 11%, similar to that in the general population. Symptoms of ADHD can be observed even in nonverbal children. However, in children

TABLE 3-1. **Clinical features of intellectual disability**

	Mild	Moderate	Severe	Profound
Percentage of intellectual disability population	85	10	5	<1
Predominant socioeconomic class	Low	Less low	Even distribution	Even distribution
Academic level achieved by adulthood	4th to 6th grade	2nd grade	Below 1st grade	Below 1st grade
Language	Concrete and limited	More limited	Single words and phrases	Nonverbal
Vocation	Can acquire skills for minimum wage work	Rote work under protected conditions	Simple skills	—
Residence	Community	Sheltered	Mostly living in highly structured and closely supervised settings	Mostly living in highly structured and closely supervised settings
Economic	Able to make change	Able to make small change	Able to use coin machines	Dependent on others for money exchange
	Budget planning with effort or assistance	Usually able to manage pocket money	Dependent on others for money management	Dependent on others for money management

with ID, inattention may be a sign of developmentally inappropriate expectations rather than ADHD. Frustration and limited expressive and receptive language may lead to aggressive temper outbursts.

Autism spectrum disorder (ASD) is the second most common co-occurring disorder for individuals with ID (Matson and Shoemaker 2009). There is much overlap in ID and ASD. Approximately 30%–40% of persons with ID have ASD, and 70% of persons with ASD have ID. Both ID and ASD can be caused by congenital rubella syndrome, tuberous sclerosis complex, and phenylketonuria. Some symptoms seen in ASD, including self-injurious behaviors, social isolation, and communication deficits, also may be found in persons with severe ID. ASD is diagnosed when there is evidence of severe social impairment relative to the patient's developmental level. Stereotypic movements, including hand shaking or waving, body rocking, head banging, mouthing of objects, self-biting, picking at skin, and self-hitting, are among the more common behavioral presentations in individuals with ID, especially those with more severe impairment. Pica, rumination, cluttering, stuttering, and other language and speech disorders occur at increased rates in association with ID.

Depression can be a complication of ID (e.g., in response to extra burdens, poor self-image, and social stigmatization) or simply a coincidence, and occurs at a rate similar to or higher than that in the general population (Hurley 2006). Depression is probably underdiagnosed in persons with ID, given the reliance on the individual to express internal thoughts and feelings. Behaviors, such as aggression, may be attributed incorrectly to the ID, instead of to depressed mood. Depression in verbal, mildly intellectually disabled individuals presents more similarly to that in typically developing peers, although the patient's reports may be more concrete. The diagnosis in moderately or severely intellectually disabled youth, however, requires observation in several settings. Occasionally, pharmacological or behavioral treatment is undertaken without a confirmed diagnosis of depression, with the hope that symptoms will improve. Rarely, individuals with ID may have rapid-cycling bipolar mood disorders. Suicidal ideation and behavior may occur among intellectually dis-

abled individuals, but completed suicides are less prevalent than in typically developing youth.

Twenty-five percent of youth with ID have significant symptoms of anxiety. Diagnoses include generalized anxiety disorder, phobias, panic disorder, posttraumatic stress disorder, and obsessive-compulsive disorder (OCD). Fears of failing and loss of caregivers are among the environmental factors that increase the vulnerability of persons with ID to anxiety disorders. As in individuals affected by depression, patients with ID may not have the cognitive or verbal skills to describe specific cognitive or emotional symptoms, increasing the clinician's reliance on behavioral observation and report of caregivers.

Etiology

Intellectual disability results from psychosocial, biological, and environmental factors. In approximately 30% of cases, the etiology is unknown. Both biological and social factors influence risk for ID of unknown cause. Risk factors for ID of unknown etiology include low birth weight; male gender; mothers of African American, Latina, or Asian ethnicity; older mothers; lower level of maternal education; lower socioeconomic status; multiple births; or second or later birth order.

Most ID is idiopathic, associated with sociocultural or psychosocial disadvantage. Intellectual and adaptive deficits are presumed to be determined by the interaction of a polygenic mechanism and social factors (Table 3–2).

Intellectual disability of moderate, severe, or profound severity is less likely to be idiopathic. ID is associated with more than 200 recognized syndromes. Known biomedical etiologies are identified in 60%–90% of cases of severe or profound ID (Table 3–3). Virtually all types of genetic inheritance paradigms are represented when genetic causes of ID are considered (recessive, dominant, X-linked, single gene, polygenic).

Down syndrome, the most common genetic cause of ID, occurs in about 1 of 1,000 births. Down syndrome is caused by a triplicate

TABLE 3-2. **Psychosocial causes of intellectual disability**

Poverty	Malnutrition
	Disease and infection
	Inadequate preventive care and medical treatment
	Sociocultural deprivation
Parental factors	Limited intelligence and education
	Psychiatric disorders
	Inadequate help-seeking behavior
	Lack of psychosocial stimulation
	Poor parenting skills
Lack of community programs	Child abuse and neglect
	Early identification and diagnosis
	Early intervention and infant stimulation
	Specialized education
	Vocational training and independent living

state of all or part of the critical region on chromosome 21, most often caused by errors in cell division of maternal origin. Risk increases with increase in maternal age. The phenotype is characterized by more than 80 features, including cognitive impairments (average IQ of 50), hypotonia, short stature, cardiac malformation, gastrointestinal malformation, distinctive facial appearance, and obesity. Individuals with Down syndrome can develop early-onset Alzheimer's disease. Relative cognitive strengths include visual processing and relative weaknesses include language. A generalized deficit in executive function can lead to problems with attention, impulsivity, hyperactivity, and aggression. Social skills tend to be relatively stronger than other domains of adaptive functioning.

Fragile X syndrome (FXS), caused by trinucleotide expansion of the FMR1 gene, is the most common inherited cause of ID (1 in 4,000 males and 1 in 8,000 females). Characteristic physical stigmata—long face, prominent ears, prominent jaw, and macroorchid-

 TABLE 3–3. **Biological causes of intellectual disability**

Genetic	Single gene defects (e.g., 22q11.2 deletion syndrome [velocardiofacial syndrome]) Inborn errors of metabolism (e.g., phenylketonuria) Chromosomal abnormalities (e.g., fragile X syndrome, trisomy 21 [Down syndrome]) Polygenic inheritance
Prenatal	Maternal illness (e.g., diabetes, toxemia) Maternal infection passed to fetus (e.g., rubella, cytomegalovirus, toxoplasmosis, syphilis, herpes, HIV) Toxins (e.g., alcohol, tobacco, narcotics, lead, anticonvulsants) Brain malformations Extreme malnutrition Intrauterine growth retardation Gestational complications (e.g., placenta previa, umbilical cord prolapse, vaginal hemorrhages)
Perinatal	Extreme prematurity Blood group incompatibility Brain trauma Cerebrovascular accident Neonatal anoxia Asphyxia Encephalopathy from hyperbilirubinemia
Infancy or childhood	Brain infection (e.g., meningitis, encephalitis) Head trauma Neurological disease Brain tumor Hypothyroidism Radiation Lead intoxication Severe malnutrition

dism (large testes)—are seen most clearly after puberty. Patients may have a high arched palate; hyperextensible finger joints; soft, velvetlike skin; and flat feet. The medical history may include seizures, recurrent infections, hernias, strabismus, or scoliosis. In some individuals with FXS, cognitive impairment may be absent. However, most affected males have moderate impairment, and affected females have mild impairment. Behavioral characteristics of individuals with FXS include hyperarousal, hyperactivity, social anxiety, shyness, and gaze aversion. Up to 25% of individuals with FXS meet criteria for autism (conversely, however, only 2% of individuals with autism have FXS). Other comorbid psychopathology includes schizotypal personality disorder and ADHD.

Course and Prognosis

The course of ID varies according to the etiology, severity, underlying genetic syndromes, and associated medical conditions. Medical care should address underlying conditions that can cause brain injury (e.g., shunting for hydrocephalus, diet for phenylketonuria). Associated medical conditions that compromise the child's functioning can be prevented or adequately managed. Such conditions include seizures, otitis media, injury secondary to self-abusive behavior, and deafness and congenital cataracts (associated with Down syndrome). Treatment of comorbid psychiatric conditions can also influence the course (e.g., treatment of comorbid ADHD can enhance the ability to learn skills). Adaptive functioning outcome is also influenced by environmental variables, including parental intelligence, psychological resilience, and material resources, as well as community resources and barriers.

More severe levels of ID are diagnosed before the child is of school age, particularly when the ID is associated with a known phenotype or syndrome. In ID of moderate to mild severity, diagnosis is uncommon before age 5 years, rises sharply in the early school years, and peaks in the later school years. Higher observed prevalence during the school years is usually attributed to the adaptive and intellectual demands of school (especially for social interaction and

abstract thinking). At all levels of severity, the normal sequence of cognitive developmental stages occurs, but the rate of developmental progress 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. ID is not necessarily a lifelong affliction. Proper training that addresses clinical needs and uses the patient's strengths can improve the level of functioning.

Evaluation and Differential Diagnosis

Intelligence tests are a necessary component of the diagnostic process (although there are potential challenges in using standardized tests in individuals with ID). The scores provide a general index of the individual's developmental level. Test selection is based on age of the child and the child's verbal ability. Tests measuring adaptive functioning vary in the domains assessed, child's age, the identified informant, and setting of assessment. Table 2–7 (Chapter 2, “Evaluation and Treatment Planning”) lists individually administered tests of intellectual capacity, learning, and adaptive functioning. For very young children, the Bayley Scales of Infant and Toddler Development, Third Edition (Bayley-III) or the Denver Developmental Screening Test (DDST) may be used to estimate level of cognitive development. In 2015, the American Association on Intellectual Disability and Developmental Disabilities (AAIDD) published the Diagnostic Adaptive Behavior Scale (DABS), a test of adaptive functioning focused on diagnostic clarity.

Medical evaluation begins with a careful history that includes inquiry regarding potential familial disorders, neurophysiological insults, diseases with progressive deterioration, and seizure disorder. The physical examination should include a neurodevelopmental assessment of functional abilities and impairments and a search for abnormal neurological findings, dysmorphic features, or dermato-

logical signs. Laboratory investigations are particularly important when there is no clear etiology for the ID. Genetic testing and recommendations are rapidly evolving. The American College of Medical Genetics and Genomics (ACMG) provides current guidelines for cytogenetic analysis in the diagnostic process (www.acmg.net). Brain imaging may identify lesions that are surgically correctable or at least explanatory. If seizures are suspected, an electroencephalogram (EEG) is indicated. Individuals with ID require modifications in the standard psychiatric examination, according to their level of language and cognitive development. These patients often suffer from multiple disabilities and medical disorders that affect mood and behavior. Clinical assessment must be comprehensive and based on the patient's presentation in multiple settings. Hyperactivity, aggression, sadness, lack of enthusiasm, excessive anxiety, and formal thought disorder are not primary features of ID and should lead to a full psychiatric evaluation, as should a significant change in mood, behavior, sleep, appetite, or level of adaptive functioning.

The differential diagnosis of ID includes *environmental deprivation, learning disorders, autism spectrum disorder, communication disorders, borderline intellectual functioning, and impaired vision and/or hearing*.

Treatment

To address the multiple disabilities and complications associated with ID, developmentally sensitive multimodal treatment is optimal. The clinician should coordinate medical and psychiatric evaluations, parental guidance (support, education, behavior management, educational and environmental planning, long-term monitoring, and advocacy), and the standard therapies for concomitant psychiatric disorders. It is important to ensure the safety of the patient and those around him or her.

Behavior modification techniques, such as removing inappropriate attention; changing dysfunctional communication patterns (e.g., rewarding use of words or sign language); consistently applying social, self-care, academic, and vocational expectations; and structur-

ing environmental contingencies, can reduce the frequency and severity of self-injury, stereotypes, pica, and asocial behavior. The behavioral program should be consistent and applied across settings, including home and school. Specialists may provide educational and developmental training to enhance speech and language; motor, cognitive, social, and occupational functioning; and adaptive skills such as toileting, dressing, grooming, and eating.

Developmentally oriented psychotherapeutic interventions may be effective in managing crises or in addressing long-term psychosocial goals. Therapy can focus on tangible objectives for the patient, including achieving greater independence, recognizing and expressing emotion, dealing with the reactions of others, developing social skills, and handling personal and practical challenges. Group therapy for adolescents and young adults can be useful for social skills training. Family therapy can focus on learning about the disability, emphasizing the patient's strengths, reducing guilt and overprotection, and fostering greater independence.

Although no pharmacological treatments are available for ID per se, the frequency of comorbid psychiatric disorders may suggest the use of psychotropic medication. Treatment effects are often difficult to assess in developmentally disabled patients. Their impaired verbal skills may complicate the diagnostic process, measurement of efficacy, and detection of adverse effects. Heterogeneity among children with ID leads to variability in treatment effect. Youngsters with structural brain abnormalities often react differently to medications than do typically developing children, even when target symptoms are similar.

Before psychotropic medications are prescribed to individuals with ID, special considerations apply. Individuals with ID may have limited ability to communicate adverse effects, thus caregiver input is often necessary. Medications used for medical or neurological disorders associated with ID may have problematic side effects and/or ceilings on effectiveness and should be started at lower doses with more gradual increases. A significant change in the individual's typical behavior warrants an investigation for a medical cause of discomfort. Medications typically used for ADHD are effective in the

treatment of symptoms of impulsivity, hyperactivity, and inattention in ADHD in children with ID, although effect size is less and side effects are more prominent. Alpha-adrenergic agents also have shown to be effective in treating ADHD symptoms. Few studies exist on the psychopharmacological treatment of depression in these patients, but selective serotonin reuptake inhibitors (SSRIs) may improve mood, energy level, interest, and motivation. Individuals with ID may be more susceptible to behavioral activation with SSRI use. Mood lability and aggression can be treated with mood stabilizers and antipsychotics. Of the antipsychotics, the second-generation class of medications is generally preferred. Risperidone has been studied the most, showing positive effects on behaviors. However, weight gain and metabolic changes are also seen. Sleep disturbance is commonly seen in this population. Treating the sleep disturbance may indirectly yield improvement in behaviors. Melatonin has shown promising effects in small, open trials.

■ COMMUNICATION DISORDERS

Clinical Description

DSM-5 communication disorders include language disorder, speech sound disorder, childhood-onset fluency disorder, and social (pragmatic) communication disorder. Communication disorders are deficits in speech, language, or communication abilities that interfere with academic or adaptive functioning. *Speech* is the expressive production of sounds and includes articulation, fluency, voice, and resonance. *Language* involves the effective, rule-governed use of a conventional system of symbols for communication. *Communication* includes verbal and nonverbal behavior that influences the behavior or ideas of other individuals. A child may have a disorder in more than one area of communication, and multiple cortical deficits are usually observed, particularly in sensory information processing and temporal auditory processing.

Epidemiology

Speech and language disorders are evident in about 5% of the school-age population. Language disorders are more common in males and in children with psychiatric disorders, ID, or hearing impairment.

Etiology

Many aspects of motor, sensory, and cognitive development must be intact for speech and language to develop normally. By age 5 years, children are expected to speak fluently and to comprehend speech. Adult articulation skills should be present by age 8 years. The range of normal functioning is broad, and development of language and speech skills continues for many years. Development of articulation and vocabulary is highly influenced by the child's environment. Verbal and nonverbal communication skills include word finding (access and retrieval of verbal information), word relationships (semantics), sentence formation (syntax), along with giving and receiving feedback, following conversational structure and flow, responding to the context, adapting to meanings and external events (pragmatics), responding to one's internal sense of events, and monitoring one's own communication (metalinguistic skills). The development of these skills is a formidable task to achieve in 5 years.

The etiology of deficits is often unknown, but there is strong evidence for the heritability of expressive language disorders, both with and without articulation problems. Environmental factors also contribute and may include faulty speech models within the family and lack of stimulation of language. Hearing loss, even if mild, plays a significant role in the etiology of language and speech disorders. During the period of language development, fluctuating hearing capacity or degrees of hearing loss that are considered medically insignificant can diminish measured verbal IQ and academic performance. Even mild hearing loss (25–40 decibels) may delay development of articulation, expressive and receptive language, reading, and spelling.

Course and Prognosis

Relative deficits in articulation (speech sound production), expression (oral language production and use), and reception (comprehension) may be observable by age 2–3 years. Delays in speech and language frequently improve during development, so that early delays are not strongly predictive of subsequent psychiatric and learning disorders. Speech and language skills are eventually acquired in most cases, but other characteristics (concomitant psychiatric disorders, neuromotor problems, low IQ) may predict worse outcome.

Complications of speech and language disorders include progressive academic impairment, psychological distress, low self-esteem, anxiety regarding learning, and school dropout. Children with these disorders may have difficulty maintaining a conversation or expanding on a topic. Problems in social interactions may lead to peer problems and overdependence on family members. When frustrated, the young child may have tantrums or the older child may refuse to speak.

Evaluation and Differential Diagnosis

Multidisciplinary assessment is required to evaluate communication disorders. Specialized speech and language evaluation includes articulation, receptive skills (understanding single words, word combinations, and sentences), and expressive language skills (syntactic structures, vocabulary, and social appropriateness). The clinician may observe family characteristics and free speech between parents and child to assess social skills and nonverbal communication (vocalizations, gestures, and gazes). The clinician should obtain hearing acuity evaluation using audiometry or auditory evoked response (which does not require the child's cooperation) and evaluate auditory attention (e.g., losing flow of conversation, inability to hear in a crowd, distractibility), discrimination, and memory. Nonverbal measures of IQ (see Table 2–7) are used in cases of suspected language delay.

Comorbid psychiatric disorders, including other developmental disorders, are common and should be carefully sought in the evaluation.

Differential diagnosis includes *autism spectrum disorder*, *selective mutism*, *deafness*, *intellectual disability*, *medical and neurological disorders*, and *acquired aphasia*. Children with a communication disorder may exhibit social awkwardness, resistance to change, and low frustration tolerance that approach the severity of ASD, but they have better social communication, empathic awareness, and abstraction than do children with ASD.

Treatment

Hearing deficits should be addressed. Social involvement, imitation, and imaginative play are encouraged to increase verbal, communicative, and symbolic skills. Referral to a speech-language pathologist (SLP) for evaluation and ongoing therapy is essential. Psychiatric treatment of concurrent attentional, emotional, or behavior problems and educational management of academic skills disorders may be needed.

Specific Disorders

Language Disorder

Language disorder is characterized by persistent difficulties in the acquisition and use of language (spoken, written, signed, or other) due to deficits in language comprehension or production including reduced vocabulary, limited sentence structure, and impairments in discourse. This disorder includes both expressive and receptive language skills, which were classified as separate disorders in DSM-IV. Deficits result in limitations in communication, social participation, academic performance, and/or occupational function. Language disorder becomes evident in toddlerhood. There is early variability in prognosis, but function at age 4 years may be predictive of long-term prognosis. Children with receptive language difficulties have a poor prognosis compared with children who have only expressive deficits.

Speech Sound Disorder

Speech production requires both the knowledge of phonological speech sounds and the ability to move the jaw, tongue, and lips in co-

ordination with breath to vocalize. In speech sound disorder (phonological disorder in DSM-IV), there are persistent difficulties with the production of speech sounds that interfere with speech intelligibility and cause limitations in communication, social participation, or academic achievement. This disorder may occur together with language disorder. It may be associated with other oral-motor skills deficits, including feeding problems. Speech sound disorder usually responds to speech therapy.

Childhood-Onset Fluency Disorder (Stuttering)

Stuttering is a disturbance of the normal fluency and time patterning of speech that is inappropriate for the age and language level of the individual. It may include sound or syllable repetitions, broken words, blocking, or tension in the production of words. Stuttering severity varies with the perceived stress of a situation, and fear and avoidance of speaking may develop. It typically begins by age 6 years, and 65%–85% of children with this disorder have full recovery. The severity of the disorder at age 8 years is predictive of persistence into adolescence or adulthood.

Speech therapy for stuttering, conducted by an SLP, involves intensive training of fluent speech skills; the fostering of self-esteem and social assertiveness; and the use of behavior therapy methods, such as modification of environmental and conversational factors that trigger stuttering, relaxation, role-playing, feedback, practice in speaking in different settings (e.g., reading aloud in a group, alone, in front of a classroom, on a telephone), and talking with different people (parents, relatives, friends, strangers). Education and counseling of family members regarding the disorder and ways to support the child's speech efforts is advised.

Social (Pragmatic) Communication Disorder

Social (pragmatic) communication disorder (SCD), introduced in DSM-5, is characterized by persistent deficits in the social use of verbal and nonverbal communication as manifested by impairment in greeting or sharing information, in adjusting communication to

match the social context and audience, in following the rules of conversation and storytelling (including reciprocity and using nonverbal cues), and in understanding nonliteral meanings of language or making inferences. Although syntax, word structure, and grammar of language are intact, pragmatics (i.e., the social use of language and communication) is impaired. These deficits in social communication result in impaired communication, social participation, relationships, and academic or occupational achievement.

A diagnosis of SCD should not be made in the presence of ID unless the social communication deficits are clearly in excess of the cognitive limitations. The diagnosis also cannot be made in very young children, because language development and acquisition of vocabulary typically are not sufficiently developed to assess social pragmatic deficits. Milder cases may not be recognized until adolescence.

A family history of ASD, communication disorders, or specific learning disorder increases the risk for SCD. It has been proposed that some of the children who were diagnosed under DSM-IV with pervasive developmental disorder not otherwise specified (PDD NOS) may now qualify for a diagnosis of SCD. SCD can be distinguished from *autism spectrum disorder* by the absence of current or previous history of repetitive or restricted patterns of behavior, interests, or activities. Other considerations in differential diagnosis are *attention-deficit/hyperactivity disorder* and *social anxiety disorder*. Because SCD is a new diagnosis, research (and even clinical experience) is lacking with regard to epidemiology, treatment, and prognosis.

■ AUTISM SPECTRUM DISORDER

Clinical Description

Autism spectrum disorder is a diagnostic category introduced in DSM-5 to replace the pervasive developmental disorders in DSM-IV: autistic disorder, Asperger's disorder, PDD NOS, and childhood disintegrative disorder. This change was made because the separate diagnoses were not reliably made or consistent over time. The diag-

nostic criteria of ASD (Box 3–1) focus on impairment, relative to chronological and mental age, in two key areas: social interaction/communication and behavior, interests, and activities. Because ASD encompasses a broad range of clinical presentations, diagnostic specifiers are included to indicate level of severity; intellectual or language impairment; known associated medical, genetic, or environmental factors; comorbid neurodevelopmental, mental, or behavioral disorder; or catatonia. Although there is no longer a specific age-at-onset criterion, symptoms must be present in early childhood. Symptoms of ASD manifest most often in the second year of life. Individuals with milder ASD symptoms and normal IQ may first present for clinical attention in middle school or even in adolescence as their long-standing deficits become more salient with expectations for more advanced social functioning. A childhood history of completely normal social development, intact communication, and age-appropriate peer relationships precludes a later diagnosis of ASD.

Box 3–1 DSM-5 Diagnostic Criteria for Autism Spectrum Disorder

- A. Persistent deficits in social communication and social interaction across multiple contexts, as manifested by all of the following, currently or by history (examples are illustrative, not exhaustive; see text):
1. Deficits in social-emotional reciprocity, ranging, for example, from abnormal social approach and failure of normal back-and-forth conversation; to reduced sharing of interests, emotions, or affect; to failure to initiate or respond to social interactions.
 2. Deficits in nonverbal communicative behaviors used for social interaction, ranging, for example, from poorly integrated verbal and nonverbal communication; to abnormalities in eye contact and body language or deficits in understanding and use of gestures;

to a total lack of facial expressions and nonverbal communication.

3. Deficits in developing, maintaining, and understanding relationships, ranging, for example, from difficulties adjusting behavior to suit various social contexts; to difficulties in sharing imaginative play or in making friends; to absence of interest in peers.
- B. Restricted, repetitive patterns of behavior, interests, or activities, as manifested by at least two of the following, currently or by history (examples are illustrative, not exhaustive):
1. Stereotyped or repetitive motor movements, use of objects, or speech (e.g., simple motor stereotypies, lining up toys or flipping objects, echolalia, idiosyncratic phrases).
 2. Insistence on sameness, inflexible adherence to routines, or ritualized patterns of verbal or nonverbal behavior (e.g., extreme distress at small changes, difficulties with transitions, rigid thinking patterns, greeting rituals, need to take same route or eat same food every day).
 3. Highly restricted, fixated interests that are abnormal in intensity or focus (e.g., strong attachment to or preoccupation with unusual objects, excessively circumscribed or perseverative interests).
 4. Hyper- or hyporeactivity to sensory input or unusual interest in sensory aspects of the environment (e.g., apparent indifference to pain/temperature, adverse response to specific sounds or textures, excessive smelling or touching of objects, visual fascination with lights or movement).
- C. Symptoms must be present in the early developmental period (but may not become fully manifest until social demands exceed limited capacities, or may be masked by learned strategies in later life).

- D. Symptoms cause clinically significant impairment in social, occupational, or other important areas of current functioning.
- E. These disturbances are not better explained by intellectual disability (intellectual developmental disorder) or global developmental delay. Intellectual disability and autism spectrum disorder frequently co-occur; to make comorbid diagnoses of autism spectrum disorder and intellectual disability, social communication should be below that expected for general developmental level.

Note: Individuals with a well-established DSM-IV diagnosis of autistic disorder, Asperger's disorder, or pervasive developmental disorder not otherwise specified should be given the diagnosis of autism spectrum disorder. Individuals who have marked deficits in social communication, but whose symptoms do not otherwise meet criteria for autism spectrum disorder, should be evaluated for social (pragmatic) communication disorder.

Social deficits are the most consistent and reliable indication of ASD. Younger children fail to seek out peer interactions, whereas older individuals may express an interest in friendships and seek interpersonal experiences but lack the skills to initiate and maintain relationships. Individuals with ASD demonstrate persistent deficits in appreciating the feelings and thoughts of other people and in understanding the process and nuances of social communication. They prefer solitary games and relate to others primarily as "objects" in their play.

In many children with ASD, spoken language is either delayed or totally absent. Even in patients with normal language and IQ, deficits in pragmatic language are present, as seen in these patients' inability to initiate or sustain reciprocal conversation. Communication may be characterized by echolalia, pronoun reversals, scripted or stereotyped phrases, and idiosyncratic meanings. Phonological (sound production) and syntactic (grammar) functions may be rela-

tively spared, with more significant impairment of verbal semantics (sociocultural meanings) and pragmatics (rules of interpersonal exchange) and nonverbal aspects of communication (eye contact, facial expression, body language). Imaginative and symbolic functions (e.g., use of toys in play) are significantly impaired. Play is characterized by rituals, stereotypies, motor mannerisms, and pre-occupations with parts of objects. Individuals with ASD are often cognitively rigid, finding it difficult to adjust to transitions between activities and unexpected changes in routine or in the environment and to compromise in social interactions. Some individuals with ASD are hypersensitive and may avoid sensory stimuli (certain tastes, loud sounds, textures of food or clothing).

Many individuals with ASD have subnormal intelligence, but a few show significant improvements in measured IQ with time or treatment. According to 2012 data from the Centers for Disease Control and Prevention (CDC), 31.6% of ASD patients had ID (IQ<70), 24.5% had borderline intelligence (IQ 71–85), and only 43.9% had average to above average intelligence. Even in individuals with normal IQ, neuropsychological test results are often greatly scattered (often with discrepancies between verbal and nonverbal IQ) and inconsistent over time. Some individuals (known as *savants*) have unusual or special capacities in music, drawing, arithmetic, or calendar calculation.

Epidemiology

Prevalence estimates of ASD vary widely. The most recent report from the CDC estimates national prevalence at 14.6 per 1,000 (1 in 68) 8-year-olds (Christensen et al. 2016). The reported overall prevalence rate has increased 123% from 2002 to 2010 but seems to have stabilized between 2010 and 2012. Increased prevalence estimates are likely due primarily to broader diagnostic criteria and increased awareness. There is a male predominance of 4 to 1. White children are more likely to be diagnosed with ASD than Hispanic or black children, though this is likely due to cultural or socioeconomic barriers to assessment and treatment.

Etiology

ASD is a highly heritable condition with multiple implicated genes. The prevalence of autism in siblings is 4.5%. Monozygotic twins have a higher rate of concordance (60%–90%) than dizygotic twins, but it is not 100%, thus implicating the role of early environmental biological insults as well. There is no evidence that psychosocial factors or parenting patterns cause ASD.

A specific genetic cause of autism can be identified in about 15% of individuals, most often in those with profound or severe ID, developmental delays, or physical anomalies. Known medical causes include fragile X syndrome (5% of ASD individuals), tuberous sclerosis, and certain chromosomal abnormalities (15q11-13 maternal duplications; 16p11.2 duplications and deletions) (Volkmar et al. 2014). Seizure disorders appear in 20%–25% of patients with ASD (Volkmar et al. 2014). Neuroimaging studies demonstrate a wide range of structural and functional abnormalities but no consistent, diagnostic pattern.

Identified risk factors include advanced maternal or paternal age, a sibling with ASD, extremely premature birth, and closer spacing of pregnancies (Volkmar et al. 2014).

In multiple studies, the mumps, measles, and rubella (MMR) vaccine or the vaccine preservative thimerosal have not been found to increase the risk of ASD.

Course and Prognosis

ASD is often apparent in early infancy, but in some patients, the full disorder does not appear until after age 3 years. An initial period of seemingly normal development may be followed by developmental arrest or by regression with loss of previously developed abilities.

Children with ASD usually make gradual but erratic improvement, particularly during the school-age years. Occasionally patients make rapid unexplained developmental progress. Medical illness and environmental changes may precipitate behavioral dysregulation that may include compulsive, aggressive, or self-injurious behaviors, occasionally requiring pharmacological intervention.

Adolescents may experience either continued developmental progress or behavioral deterioration. Conditions frequently comorbid with ASD include ADHD, anxiety, depression, motor incoordination, constipation, epilepsy, avoidant/restrictive food intake disorder, and sleep problems. DSM-5 allows a diagnosis of ADHD to be made in the presence of ASD, unlike DSM-IV.

Educational and supportive services have a marked beneficial effect. For the less severely impaired, treatment may lead to social skills and adaptations that permit employment and independent or group home living. Predictors of good adaptive outcome include higher IQ, better language skills (especially the ability to communicate verbally by age 5 years), greater social skills, and later appearance of symptoms.

Depending on severity, perhaps one-third of individuals with ASD are able to function independently as adults. However, even in these higher-functioning individuals, social deficits and cognitive rigidity may persist, making navigation of social interactions and life demands difficult, thereby increasing the likelihood of anxiety and depression.

Evaluation

The evaluation of the child with suspected ASD should be a thorough, multidisciplinary, collaborative process. The American Academy of Pediatrics recommends screening of all children for ASD at age 18 and 24 months. Examples of brief screening instruments that can be used by primary care physicians or nurses with very young children are the Checklist for Autism in Toddlers (CHAT), which includes parent interview and observations of the child (Scambler et al. 2001), and the Modified Checklist for Autism in Toddlers (M-CHAT-R; available at www.firstsigns.org/downloads/m-chat.PDF), which includes only parent interview. Early developmental, family, and medical histories are important. If ASD is suspected, the assessment should include a clinical evaluation by a child and adolescent psychiatrist, psychologist, or developmental pediatrician experienced in assessing children with ASD. In addition, it is im-

portant to assess language, cognition, social skills, and adaptive functioning. Testing by a speech and language pathologist can document abnormalities in abstract and pragmatic language, encoding of complex information, and attentional skills. The medical evaluation should screen for possible inborn metabolic, structural, or degenerative diseases or seizures. An EEG (indicated in the presence of seizure disorder or developmental regression), Wood's lamp exam for tuberous sclerosis, and genetic testing are recommended to rule out medical etiologies (Volkmar et al. 2014). Audiological examination for possible deafness and examinations for other sensory deficits may be done. Patients who have a tendency to mouth objects should have serum lead levels drawn to check for lead intoxication. Psychological testing to assess cognitive ability and adaptive functioning and to establish the diagnosis of ASD may be indicated. Standardized diagnostic checklists (e.g., the Childhood Autism Rating Scale, Second Edition [CARS-2]) are based on clinical observation and parental recall of early behavior. The “gold standard” instruments for autism diagnosis in research are the Autism Diagnostic Interview, Revised (ADI-R), and the Autism Diagnostic Observation Schedule (ADOS), developed by Lord and colleagues. (CARS-2, ADI-R, and ADOS are available from Western Psychological Services; www.wpspublish.com). These instruments are not required for clinical diagnosis.

Differential Diagnosis

Rett syndrome occurs predominantly in females and includes deceleration of head growth, marked ID, hand-washing stereotypies, and loss of purposeful motor skills. Differential diagnosis also includes congenital *deafness* (but deaf children typically learn an alternative oral or sign language, lose their isolative behaviors, and develop expressive communication), congenital blindness (but blind children relate socially), *developmental expressive and receptive language disorders* (but children with these disorders are typically more sociable, communicate well in gestures, and do not have stereotypic and repetitive behavioral patterns), and juvenile-onset *schizophre-*

nia (distinguished by hallucinations, delusions, and thought disorder). Children with pure *intellectual disability* have a more even or global distribution of delays and fewer abnormalities in behavior or social relatedness. The differential diagnosis is particularly difficult in children with severe to profound ID. In very young children, *reactive attachment disorder* may be a consideration, but these cases are typically characterized by a history of severe neglect. In *selective mutism* the patient is able to speak in certain situations, such as at home or with familiar adults. *Degenerative neurological diseases* or *Landau-Kleffner syndrome* (acquired epileptic aphasia) may resemble ASD. *Social (pragmatic) communication disorder* overlaps with ASD in the domain of social interaction/communication but lacks past or current restricted and repetitive behavior or interests.

Treatment

Patients fortunate enough to have early access to rigorous multimodal treatment show significant improvement. The milieu should be highly structured and should include special education, speech and language instruction, vocational training (for adolescents), and teaching of adaptive skills. Behavior therapy reduces unwanted symptoms; promotes speech, social interaction, and assertiveness; increases self-reliance and self-care skills; and facilitates exploration. Parent guidance is critical, both to provide education and to deal with emotional reactions such as guilt or denial. Parents can contribute to the child's learning of language and self-care and adaptive skills, arrange for special education and for adjunctive services, and make long-term plans for the child. Training in behavior management skills is essential for a tolerable home environment and for maximizing the child's potential.

Acute hospitalization or longer-term residential treatment may be needed in the context of severe aggression. Long-term outpatient follow-up is required, including periodic reassessment for seizures or mood or anxiety disorders.

Psychotropic medications may be used to ameliorate disruptive behavior or to treat coexisting psychiatric disorders. However,

in the context of new or worsening aggression or self-injurious behavior, medication treatment should only occur after environmental stressors (e.g., changes in teacher, caregiver, routine, or environment) and medical etiologies (e.g., pain, infection) have been ruled out. Especially in nonverbal patients, physical discomfort can frequently be a cause for new or increased aggression or self-injurious behavior. Low doses of nonsedating antipsychotics, in conjunction with a highly structured treatment program, may help control behavioral symptoms, reduce excessive activity levels, and enhance the effect of behavior therapy. Controlled studies of atypical antipsychotics, such as risperidone, demonstrate clinical effectiveness with a relatively tolerable side-effect profile, although weight gain and metabolic syndrome are concerns in chronic use. The strongest evidence is for risperidone (McCracken et al. 2002) and aripiprazole (Owen et al. 2009) in reducing disruptive behavior and irritability (FDA-approved indications). Common side effects are sedation and weight gain. The risk of tardive dyskinesia may be higher in these patients because of the length of treatment and perhaps biological vulnerability. The α_2 -adrenergic receptor agonist clonidine has also demonstrated efficacy in treating irritability and aggression (Fung et al. 2016).

In patients with comorbid ADHD, one may consider stimulant medications, atomoxetine, or an α_2 agonist such as guanfacine (Scahill et al. 2015) or clonidine. SSRIs are sometimes effective in reducing comorbid anxiety or depressive symptoms in patients with ASD, though one should titrate cautiously and monitor for agitation or behavioral activation.

■ ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

Clinical Description

The syndrome of hyperactivity, impulsivity, and inattention was historically labeled “minimal brain damage,” “minimal brain dysfunction,” “hyperkinetic syndrome,” “hyperactivity,” or “ADD.”

The prior diagnostic term, *attention-deficit disorder* (ADD), is no longer correct but is often used in the lay media, sometimes to refer to inattentive ADHD, and sometimes for the entire syndrome.

DSM-5 made few changes to ADHD, which was moved to the new neurodevelopmental disorders section (see Box 3–2 for DSM-5 criteria). Because of low reliability and longitudinal instability, the DSM-IV subtypes have been replaced by similar “presentations”: *combined* (meeting criteria for both inattention and hyperactivity–impulsivity), *predominantly inattentive*, and *predominantly hyperactive/impulsive*. Children with the predominantly inattentive presentation tend to be described as “daydreamers” or “spacey,” more often have comorbid anxiety and depression, are more likely to be neglected by peers, have fewer conduct and behavior problems, and present less frequently to psychiatric settings than do those with the combined presentation. The predominantly hyperactive/impulsive group consists largely of very young children for whom the inattention criteria are not yet developmentally appropriate. Longitudinal follow-up studies have found that for the majority of these children, the criteria for combined type/presentation are eventually met.

Box 3–2 DSM-5 Diagnostic Criteria for Attention-Deficit/Hyperactivity Disorder

- A. A persistent pattern of inattention and/or hyperactivity–impulsivity that interferes with functioning or development, as characterized by (1) and/or (2):
 1. **Inattention:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

- a. Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or during other activities (e.g., overlooks or misses details, work is inaccurate).
- b. Often has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lectures, conversations, or lengthy reading).
- c. Often does not seem to listen when spoken to directly (e.g., mind seems elsewhere, even in the absence of any obvious distraction).
- d. Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., starts tasks but quickly loses focus and is easily sidetracked).
- e. Often has difficulty organizing tasks and activities (e.g., difficulty managing sequential tasks; difficulty keeping materials and belongings in order; messy, disorganized work; has poor time management; fails to meet deadlines).
- f. Often avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g., schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
- g. Often loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).
- h. Is often easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).
- i. Is often forgetful in daily activities (e.g., doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

2. **Hyperactivity and impulsivity:** Six (or more) of the following symptoms have persisted for at least 6 months to a degree that is inconsistent with developmental level and that negatively impacts directly on social and academic/occupational activities:

Note: The symptoms are not solely a manifestation of oppositional behavior, defiance, hostility, or a failure to understand tasks or instructions. For older adolescents and adults (age 17 and older), at least five symptoms are required.

- a. Often fidgets with or taps hands or feet or squirms in seat.
- b. Often leaves seat in situations when remaining seated is expected (e.g., leaves his or her place in the classroom, in the office or other workplace, or in other situations that require remaining in place).
- c. Often runs about or climbs in situations where it is inappropriate. (**Note:** In adolescents or adults, may be limited to feeling restless.)
- d. Often unable to play or engage in leisure activities quietly.
- e. Is often “on the go,” acting as if “driven by a motor” (e.g., is unable to be or uncomfortable being still for extended time, as in restaurants, meetings; may be experienced by others as being restless or difficult to keep up with).
- f. Often talks excessively.
- g. Often blurts out an answer before a question has been completed (e.g., completes people’s sentences; cannot wait for turn in conversation).
- h. Often has difficulty waiting his or her turn (e.g., while waiting in line).
- i. Often interrupts or intrudes on others (e.g., butts into conversations, games, or activities; may start using other people’s things without asking or re-

ceiving permission; for adolescents and adults, may intrude into or take over what others are doing).

- B. Several inattentive or hyperactive-impulsive symptoms were present prior to age 12 years.
- C. Several inattentive or hyperactive-impulsive symptoms are present in two or more settings (e.g., at home, school, or work; with friends or relatives; in other activities).
- D. There is clear evidence that the symptoms interfere with, or reduce the quality of, social, academic, or occupational functioning.
- E. The symptoms do not occur exclusively during the course of schizophrenia or another psychotic disorder and are not better explained by another mental disorder (e.g., mood disorder, anxiety disorder, dissociative disorder, personality disorder, substance intoxication or withdrawal).

Specify whether:

Combined presentation: If both Criterion A1 (inattention) and Criterion A2 (hyperactivity-impulsivity) are met for the past 6 months.

Predominantly inattentive presentation: If Criterion A1 (inattention) is met but Criterion A2 (hyperactivity-impulsivity) is not met for the past 6 months.

Predominantly hyperactive/impulsive presentation: If Criterion A2 (hyperactivity-impulsivity) is met and Criterion A1 (inattention) is not met for the past 6 months.

Core deficits in ADHD include impairment relative to expected developmental level in learning and following rules (e.g., how to solve academic problems or how to behave in school and with friends), difficulty in inhibiting impulsive responses to internal wishes or needs or to external attractions or provocations, and less than expected ability to exert mental effort without immediate reward.

Many children with ADHD have high levels of motor activity in a variety of settings. When children are expected to be highly active (e.g., on the playground), children with and without ADHD

typically have similar activity levels. In the classroom, however, restlessness, fidgeting, and walking or running around without permission cause problems. The child with ADHD often energetically explores new places and things. On entering a room, the child may immediately begin to touch and climb. He or she may inadvertently break things or hurt him- or herself or others.

Children with ADHD have difficulty with motivation, sustained attention, organization, and completion when tasks are difficult, complex, long, or boring. These youngsters are unusually prone to seek immediate gratification. Resulting school problems include delayed learning, poor study skills, incomplete homework, tests with careless mistakes, and disruptive behavior. Deficits have been identified in OTMP (organization, time management, and planning) (Gallagher et al. 2014) in many youth with ADHD. These problems may lead to erratic or failing grades, special class placement, suspension, expulsion, or dropping out of school altogether.

Peers perceive some children with ADHD as immature and irritating and often avoid or neglect them because of their low frustration tolerance; difficulty following rules; and intrusive, bossy, and 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 estimates of ADHD vary, because of differences in diagnostic criteria, samples, and assessment methods. DSM-5 cites a rate of 5% in school-age children. Community surveys have found ADHD in as many as 17% of boys and 8% of girls of elementary school age and 11% of boys and 6% of girls in adolescence. ADHD is present in 30%–50% of child psychiatric outpatients and 40%–70% of child psychiatric inpatients, often in combination with other psychiatric disorders. In elementary school-age children, the boy-to-girl ratio is typically 9 to 1 in clinical settings but 2–3 to 1 in community surveys. Girls are more likely to have the predominantly inattentive presentation than the combined presentation and, compared with

boys, are less likely to have comorbid disruptive behavior disorders and more likely to have intellectual impairment and comorbid anxiety and depression. Although in most ways, boys and girls with ADHD are similar in symptoms, impairment, and response to treatment, girls with ADHD are less likely to be identified or receive treatment than are boys with the disorder.

ADHD has been found to exist in all countries where studies have been done. The actual prevalence of ADHD is at least as high in other countries as in the United States, although diagnostic and treatment practices vary widely (Faraone et al. 2003). The rate of identification and treatment of ADHD is influenced by cultural expectations. Because all children in the United States are expected to attend school until age 18 years, academic and behavior problems are detected in youth who in other countries would no longer be in school.

Comorbidity

ADHD commonly occurs in association with other psychiatric disorders, particularly oppositional defiant disorder (ODD) and conduct disorder (CD). As many as 50% of clinically referred children with ADHD also have CD, and many others have ODD; therefore, much of the ADHD literature is actually about the combination of ADHD plus ODD or CD. In clinical settings, approximately one-third of patients with ADHD also have a mood and/or anxiety disorder. Although ADHD is very common in youth with Tourette's disorder, relatively few children with ADHD also have Tourette's. The presence of ADHD often complicates the clinical course of youth with ID or specific learning disorders. In adolescents, comorbid substance abuse may appear.

Etiology

ADHD is a heterogeneous syndrome, and many different factors contribute to etiology. Genetics (and epigenetics) interact with multiple biological environmental factors (Table 3–4) to increase risk.

TABLE 3—4. **Suggested medical contributions to etiology of attention-deficit/hyperactivity disorder**

Prenatal	Young mother Poor maternal health Maternal use of cigarettes or alcohol
Perinatal	Prematurity Intrauterine growth retardation
Infancy	Malnutrition
Toxicity	Lead exposure
Genetic disorders	Fragile X syndrome Glucose-6-phosphate dehydrogenase deficiency Generalized resistance to thyroid hormone Phenylketonuria
Brain injury	Trauma Infection

Although the school and home environments can influence the severity of ADHD symptoms, child-rearing practices do not cause ADHD.

A common neurobiological feature is relative immaturity of the prefrontal cortex, which controls many “executive functions,” such as planning, organization, working memory, sense of time, and impulse control. Many children with ADHD have less tolerance for delay and different response to rewards than typically developing children. However, not all children with ADHD show deficits on neuropsychological testing and deficits that are found vary widely. Neurochemical studies of persons with ADHD suggest that multiple neurotransmitter systems are involved, including norepinephrine, dopamine, and serotonin. Anatomic imaging studies have found that, as a group, children with ADHD, compared with control subjects, have reduced volume in multiple brain areas. A longitudinal imaging study of children with ADHD and control children (Shaw et al. 2013) found a relative global maturational delay in de-

velopment of the cerebral cortex. Imaging findings are not, however, specific enough to be useful in the diagnosis of ADHD. Imaging may be helpful in evaluation only if a neurological lesion or disorder is suspected. Functional magnetic resonance imaging offers promise in research on understanding the underlying brain mechanisms of attention and impulse control.

Genetics

The genetic contribution to the etiology of ADHD is substantial, with an average heritability of 75%–80%. In addition, children with ADHD without conduct problems are likely to have relatives with learning problems. Children with both ADHD and CD are more likely to have relatives with CD, adult antisocial behavior or personality, and substance abuse. Mood disorders are common in families with ADHD. The hereditary component of ADHD is polygenic, rather than a single-gene defect. The specific mechanism of genetic transmission in ADHD has not been identified, although multiple possibly contributing genes have been found. Many of the identified genetic risk factors have a small effect size or are relatively rare. In one prospective study, ADHD subjects with certain genes were more likely to develop aggression and other symptoms of CD (Caspi et al. 2008).

Medical Factors

Children with identifiable medical or neurological causes (see Table 3–4) represent a small proportion of the ADHD population, and many children with medical risk factors do not have ADHD.

Nonlocalizing neurological “soft signs” (e.g., clumsiness, left–right confusion, perceptual–motor dyscoordination, dysgraphia) are common in children with ADHD, but 15% of children without ADHD have as many as five soft signs, and children with anxiety disorders also have these signs. The presence or absence of soft signs, therefore, is not helpful in diagnosis but may identify target symptoms for treatment.

Course and Prognosis

Mothers of some youngsters with ADHD (especially those meeting hyperactive–impulsive criteria) recall excessive intrauterine kicking or report: “When he began to walk, he ran.” Although ADHD can be diagnosed by age 3 years, considerable overlap with normal high activity level, impulsivity, limited frustration tolerance, and short attention span is found before age 5 years. Preschoolers with severe hyperactivity and impulsivity, often paired with oppositional behavior, come to diagnostic attention early, often after being expelled from daycare or preschool. In children with less severe behavioral problems, diagnosis is often delayed until elementary school, where expectations for physical stillness, prolonged attention, and conformity to social norms are greater and children are compared to peers. Verbally skilled, nonoppositional children who do not have learning disabilities and who are not severely hyperactive may not be given a diagnosis until they experience the greater academic demands for organization and concentrated effort in middle school or high school, or even college or graduate school.

ADHD is not a benign or self-limited childhood disorder; it has a chronic or even lifelong course. With age, physical hyperactivity typically decreases. However, in adolescence, most continue to be symptomatic, although specifics differ among studies, related to characteristics of the sample and assessment methods. As many as 30%–50% of clinically diagnosed hyperactive children continue to have the diagnosis of ADHD in adulthood, and other young adults continue to have subthreshold symptoms of ADHD with impaired functioning. Secondary effects include low self-esteem and significantly compromised social skills. Youth with ADHD are at increased risk of dropping out before high school graduation and have more car accidents, court appearances, suicide attempts, early sexual activity, and problems with relationships than do young adults without ADHD. In comparison to controls, both academic and employment accomplishment may be impaired. Children with ADHD are at increased risk for substance abuse or delinquency as adolescents only if they also had CD in childhood. “Pure” ADHD may increase the

risk for substance abuse disorders in adulthood, however (Charach et al. 2011). ADHD is associated with increased risk of cigarette smoking, including earlier onset and more difficulty quitting. Comorbid ADHD and CD in childhood also predict antisocial behavior and antisocial personality disorder in adolescence and adulthood. The absence of significant conduct problems or defiant, aggressive behavior toward adults in childhood and the presence of high IQ may predict a better prognosis for ADHD. More severe and prolonged symptoms of ADHD, comorbidity, and parental mental health problems have been linked to worse prognosis. The risk of early death may be increased, due to suicide, homicide, and accidents.

Parental responses to children with ADHD can aggravate or improve the course of the child's illness. Observation studies indicate that mothers tend to have more controlling, directive, structure-setting, and negative responses to their children with ADHD than control mothers have to their children (Tallmadge and Barkley 1983). Interestingly, when the ADHD child's behavior is improved by stimulant medication, the parents become more positive and less controlling (Tallmadge and Barkley 1983). Much of the parent-child conflict appears to be caused by comorbid ODD rather than by the ADHD per se. Children with ADHD are often more compliant with their fathers' requests than with their mothers'. Living with a child who has ADHD, especially one who also has CD or ODD, can be very difficult for parents and siblings. Given the strong genetic contribution to ADHD, it is not uncommon for one or both parents to suffer from their own inattention and impulsivity. ADHD provides an example of how an early-onset disorder, often with a genetic or neurological etiology, can be modified by life experiences and developmental processes, including educational programming; neighborhood supports; individual compensatory strengths, such as easy temperament, engaging personality, athletic abilities, and intelligence; and family emotional, social, and financial resources.

Evaluation

Clinical skill, familiarity with normal development, multiple informants, and rigorous application of the diagnostic criteria are needed

to diagnose ADHD, because some degree of inattention, impulsivity, and overactivity is common in children, especially boys. The behavior of youth with ADHD may appear quite different in different environments. Teachers expect more extended concentration and physical stillness and organizational abilities than do parents. When the child is in a highly structured or novel setting, is engaged in a stimulating activity (e.g., a computer game), or is alone with an interested adult, symptoms may not be apparent at all, except in the most severe cases of ADHD. Symptoms typically worsen in situations that are unstructured, boring, and minimally supervised or that require sustained attention or mental effort. Problems, therefore, are often far more apparent in a busy classroom than in the clinician's office. The clinician's waiting area may provide a more realistic sample of behavior.

Children and adolescents with ADHD commonly do not report their symptoms of hyperactivity, inattention, and impulsivity. The patient interview is useful, nonetheless, for the clinician to observe the child's behavior and to identify any comorbid or alternative psychiatric disorders. The parent interview is crucial for both ADHD diagnosis and more comprehensive assessment. Key parts of the history include obstetrical history (e.g., maternal substance use, prenatal or perinatal injury), medical history (e.g., seizures, thyroid disorder, head trauma, tics, medication use), screening questions for sleep disorders (e.g., loud snoring), traumatic events (including neglect or abuse), family residence (e.g., lead exposure from paint or soil), family psychiatric history, family medical history (e.g., thyroid disorder), and any factors that may increase the risk of medication abuse by the child, siblings, or parents. Special emphasis is placed on school reports of grades, learning, and behavior both in the classroom and in less structured settings such as the playground, cafeteria, and school bus. Normed parent and teacher rating scales are key—as part of the initial evaluation, broad-band dimensional scales like the Child Behavior Checklist (CBCL) and the Teacher Report Form (TRF) or DSM-based symptom ratings such as the Child Symptom Inventory are used. More narrowly focused ADHD scales include the SNAP-IV and the ADHD Rating Scale–5. The

Vanderbilt ADHD Diagnostic Teacher Rating Scale is commonly used by pediatricians and schools.

The physical examination should include possible signs of physical anomalies, thyroid disorder, or chronic illness, as well as baseline findings relevant to subsequent medication treatment such as height and weight, tics, and cardiac status. Neurological examination or tests (e.g., EEG or brain scan) are indicated only in the presence of focal neurological signs or clinical suggestions of a seizure disorder or a decline in neurological or cognitive functioning. Acuity of vision and hearing should be tested. Laboratory measurement of thyroid function or blood lead level is indicated only if suggested by history or physical examination. A baseline electrocardiogram prior to medication treatment is not indicated unless there are cardiac risk factors in the patient history or examination or family history.

Psychological testing is useful to assess intellectual ability, academic achievement, and possible specific learning disorders. Normal performance on individual testing does not preclude the diagnosis of ADHD, although observations during testing may provide data on attention, distractibility, impulsivity, ability to stay seated, and frustration tolerance. Neuropsychological evaluation may be requested to evaluate specific deficits suggested by medical history, physical examination, or basic psychological testing. Speech and language evaluation may be needed.

ADHD is a clinical diagnosis made on the basis of interviews and standardized parent and teacher behavior rating scales. EEG, brain imaging, laboratory tests, psychological tests, activity monitoring, and computerized tests of vigilance are not diagnostic for ADHD.

Differential Diagnosis

Because the usual long-term course of ADHD is gradual improvement, sustained worsening behavior (apart from periods of environmental stress) suggests the emergence of a different psychiatric disorder. A positive treatment response to stimulant medication does not confirm a diagnosis of ADHD or eliminate another diagnosis. Clinical expertise is necessary to differentiate ADHD from

normal high activity level. Problems of recent onset and brief duration may represent an *adjustment disorder*. For a diagnosis of ADHD, DSM-5 requires that some symptoms causing impairment must have been present in more than one setting before age 12 years (a change from 7 years in DSM-IV), and sufficient symptom criteria must have been met for at least the past 6 months.

Various *physical problems* (medical disorders, sleep disorders, or lack of adequate sleep or nutrition) can interfere with attention. Hyper- or hypothyroidism, as well as the effects of prescribed medications or the abuse of drugs, can mimic ADHD. In addition, *situational anxiety*, *child abuse or neglect*, *posttraumatic stress disorder*, or *boredom in school* can manifest 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 seen in ADHD, although these disorders often occur together with ADHD. Youth with *depression* or *anxiety* may present with deficits in attention. Anxious children may appear fidgety and restless, but they can verbalize worries or fears. Symptoms of anxiety are most apparent in new situations, whereas ADHD symptom expression typically worsens with increased familiarity. *Mania* or *bipolar mixed state* may take uncharacteristic forms in prepubertal children and should be considered, especially in patients with a family history of bipolar disorder. *Depressive disorders* are generally more episodic than ADHD and have prominent sadness or irritability. The new DSM-5 diagnosis *disruptive mood dysregulation disorder* (DMDD), characterized by chronic irritability, reactive aggression, and severe mood lability, may be confused with ADHD, although the two disorders are often comorbid. Some degree of emotional dysregulation is found in ADHD itself, often in the context of low frustration tolerance, but the mood symptoms are more severe in DMDD. DMDD alone does not have the core features of ADHD, outside of the context of the mood symptoms.

Accurate differential diagnosis is particularly crucial to rule out *psychosis*, because stimulant treatment can exacerbate psychotic symptoms and disorganization. Children with ADHD do not have

hallucinations, delusions, or formal thought disorder. However, distractibility and excessive talking may resemble thought disorder, and impulsivity may lead to potentially dangerous behavior and a lack of awareness of the environment that may be confused with poor reality testing. Children with *autism spectrum disorder* (ASD) often show hyperactivity, inattention, and impulsivity. DSM-5 now permits ADHD to be diagnosed in the presence of ASD, if criteria for both are met. Children with undiagnosed *intellectual disability* or *learning disorders* are often mistakenly referred for stimulant treatment because of inattention, distractibility, and inability to follow directions in school that stem from inability to do the work rather than from ADHD, although both may be present.

Monitoring Treatment

Treatment outcome is best evaluated by parent observations and by reports from teachers on behavior, attention, and academic progress. Brief rating scales completed by teachers weekly during dose titration and at intervals thereafter are an efficient means of gathering school-related information. The items on the Child Attention Problems (CAP) Rating Scale (Table 3–5) (Barkley 1990) are similar to many of the DSM-5 symptoms of ADHD (see Box 3–2). Scoring of inattention and overactivity is based on a large normative sample (Table 3–6).

The IOWA Conners Teacher Rating Scale is a short report form for treatment monitoring that covers both ADHD symptoms and defiance or oppositional behavior (Loney and Milich 1982; Pelham et al. 1989). Some investigators and clinicians use computerized laboratory tests of attention, vigilance, and impulsivity, such as the Continuous Performance Test (CPT), to assess medication effect. Because computerized tests measure a limited domain of performance in an artificial environment, they should not be substituted for clinical judgment based on the reports of observers in different settings. Behavioral observations and measurement of productivity and accuracy on timed samples of reading or math problems are more ecologically valid than CPTs.

TABLE 3-5. **Child Attention Problems (CAP) Rating Scale**

Child's name: _____ Child's age: _____
 Today's date: _____ Child's sex: Male ☐
 Female ☐
 Filled out by: _____

Below is a list of items that describes pupils. For each item that describes the pupil now or within the past week, check whether the item is Not true, Somewhat or sometimes true, or Very or often true. Please check all items as well as you can, even if some do not seem to apply to this pupil.

	Not true	Somewhat or sometimes true	Very or often true
1. Fails to finish things he/she starts	☐	☐	☐
2. Can't concentrate, can't pay attention for long	☐	☐	☐
3. Can't sit still, restless, or hyperactive	☐	☐	☐
4. Fidgets	☐	☐	☐
5. Daydreams or gets lost in his/her thoughts	☐	☐	☐
6. Impulsive or acts without thinking	☐	☐	☐
7. Difficulty following directions	☐	☐	☐
8. Talks out of turn	☐	☐	☐
9. Messy work	☐	☐	☐
10. Inattentive, easily distracted	☐	☐	☐
11. Talks too much	☐	☐	☐
12. Fails to carry out assigned tasks	☐	☐	☐

Please feel free to write any comments about the pupil's work or behavior in the last week.

Source. Reprinted with permission from Craig Edelbrock, Ph.D., 1967. (In the public domain; may be reproduced but not changed or sold.)

TABLE 3-6. **Child Attention Problems (CAP) Rating Scale scoring**

Each of the 12 items is scored 0, 1, or 2.

Total score: Sum of the scores on all items

Subscores:

Inattention: Sum of scores on items 1, 2, 5, 7, 9, 10, and 12

Overactivity: Sum of scores on items 3, 4, 6, 8, and 11

Scores recommended as the upper limit of the normal range (93rd percentile):

	Boys		Girls	
Age (years)	6-11	12-16	6-11	12-16
Inattention	9	9	8	7
Overactivity	6	7	5	4
Total score	15	16	13	11

Source. Personal communication, Craig Edelbrock, Ph.D., 1986.

Treatment

The cornerstones of treatment for ADHD are education about the disorder, appropriate school and class placement and academic remediation (if necessary), and medication treatment. Behavior modification is effective if consistently applied, but improvement is seen only when and where the behavioral program is in effect. Mild cases of ADHD may respond to parent education, behavior modification, and school consultation alone, especially in young children. For preschool-age children, behavior modification is recommended prior to considering treatment with medication. For all youth with ADHD, if medication and environmental modification do not lead to sufficiently improved behavior, academic performance, and social adjustment, or if comorbid psychiatric conditions are present, then additional treatments should be considered, based on the specific problems of the child and family.

Parent Education

Education of parents regarding the nature of the disorder and its management is key to the treatment of ADHD. Efficient parent ed-

ucation methods include parent groups in the clinical setting, books (see “Books for Parents” in Appendix, “Resources”), and referral to a support group such as CHADD (Children and Adults with Attention-Deficit/Hyperactivity Disorder) (see “Information on the Internet” in Appendix). Parents are taught to manage the child’s environment by providing consistent routines and structure, reducing excessive stimulation, averting predictable opportunity for misbehavior, and designing and implementing behavior modification strategies. At home, parents are advised to establish specific spaces for homework, closely monitor peer activities, and tailor activities to the child’s strengths and weaknesses. Parents can be coached on how best to work with the school to meet their child’s needs.

School-Related Interventions

Special education is not always required but is useful for many patients with ADHD. The clinician may assess the need for special services after the child is receiving the maximum benefit from medication. The parent (and the clinician, with parental permission) should inform school officials about the child’s vulnerabilities and strengths. Regular reports from the teacher on behavior and academic performance are essential for best outcome. An Individualized Education Program (IEP) or a 504 plan (mandated by Section 504 of the federal Rehabilitation Act of 1973) can be developed with the school to ensure that the child will receive needed services.

Useful and practical adaptations by teachers include providing consistent classroom structure and routines, seating the child near the teacher and away from disruptive peers or distractions, dividing assignments into small segments, reducing the quantity of repetitive classwork or homework, and communicating consistently with parents. A notebook for documenting assignment and completion of homework that is carried daily by the child between teacher and parent can be effective. Posting on Web sites by teachers of assignments is also useful. The Daily Report Card (see Chapter 18, “Psychosocial Treatments”) targets specific behaviors and can be completed by the teacher with consequences provided by the parent. Environmental modification with closely supervised recess, physi-

cal education, bus, and cafeteria arrangements is sometimes needed. For some children, a small and perhaps self-contained classroom or resource room with a high teacher-to-student ratio, one-to-one tutoring, or remediation of specific learning disorders is needed. Collaborative Life Skills (Pfiffner et al. 2016) is an empirically supported treatment model that combines many behavioral interventions, implemented by school-based staff with parents, teachers, and primary school students with ADHD, that can improve behavioral and academic functioning. An empirically supported model of organizational skills training has been developed to teach and assist elementary school students in using organization, time management, and planning skills essential for school success (Gallagher et al. 2014), although it is difficult to find the resources to implement fully.

Academic transitions require careful planning. The move from elementary school, with smaller classes and fewer teachers, to middle school and then to high school is often very difficult for youngsters with ADHD. The decrease in structure and supervision, along with the increased expectations for autonomous functioning and organization, may result in dramatic exacerbation of academic and behavior problems.

Pharmacotherapy

Medication is the primary treatment for the core symptoms of ADHD. The vast majority of children with ADHD have a positive response to one or more drugs. Unfortunately, despite more than 50 years of research, specific predictors of response to one or all psychostimulants have not been found. Dose titration requires individual tailoring to optimize behavior and learning in different settings throughout the day. Drug treatment may continue for years, with periodic dose adjustments needed. A few patients no longer need treatment by adolescence, but many individuals require medication through adolescence and into adulthood.

Prevalence of medication prescription for ADHD varies widely among communities. Medications appear to be underused in some

areas, especially when schools and parents have difficulty accessing diagnostic and treatment resources. In some medical practices, however, medications may be prescribed too freely, with insufficient evaluation and follow-up. Specific medications and their use are covered in Chapter 17 (“Psychopharmacology”).

The two classes of psychostimulants, methylphenidate and amphetamine (and related compounds; see Table 17–1 in Chapter 17), are the most established pharmacological agents in child psychiatry. Their short-term clinical effectiveness and safety have been confirmed in more than 100 double-blind studies. A stimulant medication is the first-line drug for ADHD, with one class being tried if the other is insufficiently effective or has poorly tolerated side effects. The second-line choice is atomoxetine (Strattera), a norepinephrine reuptake inhibitor that has an FDA indication for ADHD. Long-acting forms of the α_2 -adrenergic agonists guanfacine (Intuniv) and clonidine (Kapvay) have received FDA approval for treating pediatric ADHD, either as a single agent or added to a stimulant. The immediate-release forms of both drugs continue to be used at times, especially when formularies do not include the extended-release forms. At the end of the algorithm would be the antidepressant bupropion, which has some research supporting use in ADHD but does not have an FDA indication for ADHD. When comorbid aggression is not manageable with optimal use of the above medications plus behavioral therapy, cautious adjunctive use of an atypical antipsychotic may be needed. If there is comorbid depression or anxiety, an SSRI may be added.

Psychotherapeutic Interventions

Behavior modification can improve academic achievement, reduce specifically targeted behavior problems, and decrease symptoms that remain in spite of medication treatment. Both punishment (time-out and response cost) and contingent rewards are required, and consistent, intensive, and prolonged (months to years) treatment may be needed. *Response cost* is the removal of points, rewards, or privileges following misbehavior as specified in a behavior modifi-

cation plan. Parents and teachers must learn the specific techniques and be willing to invest the time and energy to implement them. Stimulants and behavior modification at home and at school may have separate and additive positive effects. Generalization to other settings and maintenance of improvement due to behavior modification is unfortunately limited. Children may need to be taught social skills before behavior modification or medication can improve peer relationships. In a highly structured behavioral program (often not feasible to implement or maintain, however), the functioning of unmedicated children with ADHD can be nearly normalized, although 30%–60% will improve further with the addition of a low dose of stimulant medication.

Cognitive-behavioral therapy, in which children learn social and academic problem-solving strategies, monitor their own performance, remind themselves of rules, and praise themselves for accomplishments, generally is not effective in reducing ADHD symptoms, most likely because the child rarely uses the learned skills unless he or she is specifically cued to do so by an adult. Individual psychotherapy may be useful in addressing comorbid disorders or sequelae of trauma, not core symptoms of ADHD. Family psychotherapy may reduce the conflict that results from raising a child with ADHD or may address primary marital dysfunction. Group therapy provides a setting where youth social skills deficits can be observed and remediated.

Dietary Treatments

There is evidence to support modest positive effects on ADHD symptoms from supplementation with polyunsaturated fatty acids (omega-3, PUFA, fish oil). Research on micronutrient vitamin/mineral supplements has yielded inconsistent results. Melatonin may be helpful in reducing initial insomnia in children with ADHD, but it does not appear to be effective for core symptoms of ADHD. Food elimination diets remain popular with some families despite the minimal evidence of efficacy and the difficulty implementing special diets. Research on the Feingold diet (which omits both natural and artificial salicylates and food dyes) is inconclusive, but elimina-

tion of additives such as food dyes and preservatives (both found in processed foods) might be helpful for a very small subgroup of children with ADHD, particularly those younger than 6 years. However, even for those few children whose condition improves with a restricted diet, stimulants are a more potent treatment than diet.

Unestablished Treatments

Despite anecdotal reports, there is no scientific evidence that sugar, food allergies, gluten, herbal remedies, yeast, trace minerals, or megavitamins influence the etiology or treatment of ADHD. Systemic reactions (irritability, fatigue) due to a variety of allergies or deficiencies or excesses in food intake may affect behavior and attention in some children. Some nonclinicians have recommended caffeine as a more “natural” treatment of hyperactivity, but studies have found significant side effects and no evidence of efficacy.

Other unproven treatments that have enthusiastic advocates (often with financial interests) include computer-based cognitive training, neurofeedback (biofeedback training of brain waves), sensory integrative training, optometric vision training, chiropractic manipulation, metronome therapy, herbal regimens, homeopathy, massage, and acupuncture.

■ SPECIFIC LEARNING DISORDER

The specific learning disorders are all characterized by difficulties in learning and using an academic skill, with symptoms persisting for at least 6 months, despite targeted intervention. DSM-5 places them in the neurodevelopmental disorders section. The disorders are listed in Table 3–7.

Clinical Description

Learning disorders are defined by delay in expected cognitive development in specific abilities. The child functions below age or grade level. Academic or adaptive impairment is required. The de-

TABLE 3–7. **DSM-5 specific learning disorders**

With impairment in reading, specify if deficits in:

- Word reading accuracy
- Reading rate or fluency
- Reading comprehension

With impairment in mathematics, specify if deficits in:

- Number sense
- Memorization of arithmetic facts
- Accurate or fluent calculation
- Accurate math reasoning

With impairment in written expression, specify if deficits in:

- Spelling accuracy
 - Grammar and punctuation accuracy
 - Clarity or organization of written expression
-

lay is not secondary to a sensory defect or known neurological disorder. *Dyslexia* is an alternative term used for a learning disorder consisting of difficulties in word recognition, decoding, and spelling. *Dyscalculia* is an alternative term that refers to difficulties in processing numbers, learning arithmetic, and performing accurate or fluent calculations. About 5% of all schoolchildren in the United States are classified as having a learning disorder. Comorbidity among developmental disorders and with psychiatric disorders is common.

Etiology

Genetic influence is substantial. Family histories in all subtypes of specific developmental disorder show an overrepresentation of reading, speech, and language disorders in the siblings and parents of affected children, although pedigrees are not consistent with a single mode of transmission. Monozygotic (MZ) and dizygotic (DZ) twin concordance approximates 85% and 50% for reading, 75% and 45% for language, and 70% and 50% for math (Plomin and Kovas 2005). There is a male predominance of at least 2:1.

Maternal smoking during pregnancy, low birth weight, and prenatal and perinatal mishaps, in addition to strong genetic influence, appear to be associated with the appearance of developmental reading disorder.

Course and Prognosis

Learning disorders are frequently unrecognized and manifest instead as school refusal, oppositional defiant disorder, depression, or somatoform disorder. Children with learning disorders are embarrassed by their academic difficulties, dislike school, and avoid schoolwork. Diagnoses are typically made in grade school. Over time, mild cases may resolve through persistent remediation, practice, and compensation for deficits. In later school years, problems involving organizational skills may remain, even if basic skills were well remediated. In college and graduate school, individuals may have difficulty learning foreign languages, writing efficiently, or reading fluently. Often, secondary emotional and behavior problems persist beyond the duration or direct relevance of the developmental deficits. Adults may need to make accommodations in their lives and choice of career to cope with residual deficits.

Psychological and behavioral complications of learning disorder include low self-esteem, anxiety, demoralization, poor frustration tolerance, lack of enjoyment in learning, passivity or rigidity in new learning situations, truancy, delinquency, and school dropout. A strong relationship exists between specific learning disorder and ADHD; some of this is attributable to heredity. The interaction between conduct disorder and learning disorder is likely to be complex and multifactorial. Comorbid depression or anxiety disorders are relatively common.

Evaluation and Differential Diagnosis

Specific learning disorder is characterized by persistent difficulties in reading, writing, or mathematics during formal years of schooling. Diagnosis requires a clinical review of the child's developmen-

tal, medical, and educational history, reports of test scores and teacher observations, and response to academic interventions. Symptoms may include slow or inaccurate reading, poor written expression, difficulties remembering number facts, or inaccurate mathematical reasoning. Academic skills must be well below the average range of scores in culturally and linguistically appropriate tests of reading, writing, or mathematics. The child's difficulties must not be better explained by developmental, neurological, sensory (vision or hearing), or motor disorders and must significantly interfere with academic achievement. After the diagnosis of specific learning disorder is made, specifiers indicate the area or areas in which the difficulties occur and degree of severity.

Environmental causes of academic delays include suboptimal teaching or classroom, cultural background, lack of fluency in English, and poor school attendance. A child with suspected learning disability should also be assessed for possible *intellectual disability*, *ADHD*, *mood disorder* (causing low motivation), *anxiety disorder* (causing reduced attention), and other psychiatric and neurological disorders. Children with *communication disorders* may need tests that use nonverbal measures of intelligence to evaluate learning disorders.

Treatment

Because a variety of specialists and teachers may be involved in the education of a child with multiple deficits, multidisciplinary communication is vital, particularly during school transitions. *Response to intervention* (RTI) is a multilevel support system including screening and progress monitoring with early and focused interventions for struggling students, before a learning disorder diagnosis is made. The intervention and the child's response are both diagnostic and therapeutic, with failure to respond contributing to a learning disorder diagnosis. The Individuals with Disabilities Education Act (IDEA) mandates for all children with disabilities, including specific learning disorder, the right to a "free appropriate public education," including special education and related services designed to address their needs in the least restrictive context possible (see

<http://idea.ed.gov/>). In practice, remediation of only basic skills is funded, and deficits must be severe to qualify (e.g., 2 years behind expected level). An IEP is designed for each qualifying child. Part-time resource rooms, full-time self-contained classrooms, and mainstream classrooms (with special education consultants) provide the major part of special education.

The IEP includes specific accommodations, including (depending on the type of disorder) use of a calculator or word processor; time-extended or oral tests; individual or small-group tutoring; or self-paced programmed texts or computerized self-instruction. Behavioral techniques are often used to improve motivation, emphasize success, foster enjoyment of new skills, reduce rigidity in learning, and promote application in new situations.

Parents of children with learning 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 ID) with no clear ceiling on educational achievement.

Individual psychotherapy may help reduce secondary psychological complications such as low self-esteem, temper tantrums, lack of assertiveness, and inflexibility. Medication treatment is not helpful for specific learning disorders but may be for concurrent disorders, especially ADHD.

■ DEVELOPMENTAL COORDINATION DISORDER

In developmental coordination disorder, there is delayed acquisition of motor skills that is sufficient to cause functional impairment and is not caused by a known physical disorder. About 6% of children ages 5–11 years have significant impairments of gross or fine motor abilities. ADHD is commonly also present. Treatment, including occupational and physical therapy, is warranted to minimize sequelae such as academic difficulties (due to slow and illegible handwriting), sports avoidance, and vocational impairment.

■ TIC DISORDERS

Clinical Description

A *tic* is a sudden, rapid, recurrent, relatively involuntary, nonrhythmic motor movement or vocalization. Tics are described as relatively involuntary because they may be temporarily suppressed by conscious effort. They can derive from muscle contractions in any part of the body. Tics can be categorized as *simple tics*, which are very brief tics confined to one or a few muscle groups, or *complex tics*, which are tics that involve multiple muscle groups and can involve serial movements. Tics are often preceded by a specific premonitory urge. DSM-5 subdivides tic disorders into *Tourette's disorder*; *persistent (chronic) motor or vocal tic disorder*; and *provisional tic disorder* (previously referred to as *transient tic disorder*). Tourette's disorder is characterized by its chronicity and the presence at some time of both motor and vocal tics. Unlike with other DSM-5 disorders, functional impairment is not required to make the diagnosis (American Psychiatric Association 2013).

Epidemiology

Provisional tic disorder is the most common tic disorder, estimated to affect nearly 3% of children. Prevalence of Tourette's disorder is 0.77%. The prevalence of Tourette's disorder is four times higher in boys than in girls. In contrast, the prevalence rates are equivalent among boys and girls for persistent motor tic disorder and provisional tic disorder. Tic disorders are more common when the population of children in special education is surveyed (Knight et al. 2012).

Comorbidity

Comorbid disorders are common and are often more disabling than the tics. About one half of children with Tourette's disorder also have ADHD, with a range of 20%–90% in various samples (Robertson 2006). Comorbidity of Tourette's disorder and OCD or symptoms

of obsessions and compulsions is also high, with rates ranging from 11% to 80% (Cavanna et al. 2009). Anxiety disorders may correlate with tic severity in Tourette's disorder (Coffey et al. 2000). Depression is found in 10%–75% of patients with Tourette's disorder (Robertson 2006). Subdiagnostic threshold symptoms that are often seen in patients with tic disorders include aggression, temper outbursts, oppositional behaviors, behavioral rigidity, sleep problems, and interpersonal problems.

Etiology

Tourette's disorder is a neuropsychiatric illness with a variety of etiologies. Evidence suggests cortical-striatal-thalamic circuit involvement. Numerous neurotransmitters have been implicated, including γ -aminobutyric acid (GABA), dopamine, and serotonin (Leckman et al. 2010).

Extensive genetic research supports the high heritability of this disorder. The concordance rate of Tourette's disorder in MZ twins is 53%–56%, and when criteria are broadened to include chronic tic disorders, the concordance rate is 77%–94% (State 2011). The interactions of genetic and (biological) environmental influences are complex. Genetic linkage studies have not been conclusive, and the disorder appears to be genetically heterogeneous.

A subset of patients with tics have been proposed to be suffering from PANDAS (pediatric autoimmune neurological disorders associated with streptococcal infection), in which there was sudden onset or worsening of tics in the setting of an infection. This connection has been highly controversial, with conflict over boundaries of the syndrome, characteristics, treatment, and even its existence (Martino et al. 2015). Tics have been removed from the required constellation of symptoms associated with this postinfectious state (now referred to as *pediatric acute-onset neuropsychiatric syndrome*, or PANS). Approximately 10% of children with ADHD have tics, regardless of treatment with stimulant medication. Stimulants and some antidepressants may temporarily aggravate tics, but they do not appear to cause tic disorders or hasten the appearance of tics in a

vulnerable child. Medication use is often simply coincidental with the onset or worsening of tics.

Course and Prognosis

Symptoms of tic disorders typically wax and wane, and the location of tics changes over time. Transitions or stressors, such as the start of the school year, vacations, and family disruption or geographic move, typically precipitate increased frequency and severity of tics. Anxiety, pleasurable excitement, boredom, or fatigue can exacerbate tics.

Onset of Tourette's disorder is typically during childhood and early adolescence. Symptoms of ADHD often appear before any tics. Seven years is the mean age at onset of motor symptoms, which usually begin as a single motor tic and have a rostrocaudal (i.e., head before trunk and limbs) progression over time. Motor tics can include eye blinking, eyebrow raising, facial grimacing, head jerks, abdominal tics, and leg/foot movements. Complex gestures may appear later. Complex motor tics may appear purposeless or may be camouflaged by being blended into purposeful movements. They may mirror someone else's movements (*echokinesis* or *echopraxia*). By age 9–10 years, many patients with Tourette's disorder can describe premonitory sensory urges (uncomfortable feelings in a specific part of the body that are relieved with the performance of a tic). At an average age of 11 years, phonic and vocal tics may appear, along with obsessions and compulsive behaviors. Vocal tics may start as a single syllable or sounds such as throat clearing, grunting, sniffing, snorting, or barking, and progress to longer exclamations. Classic *coprolalia* (i.e., swearing or obscene language) is present in fewer than 10% of patients with Tourette's disorder. Other complex vocal tics include *palilalia* (i.e., repeating one's own sounds) and *echolalia* (i.e., repeating others' words).

In most cases, the severity of symptoms diminishes after puberty; about one-third of those affected recover by late adolescence, and another one-third significantly improve by adulthood. In the remaining one-third, symptoms range from mild to (rarely) severe.

Complications of Tourette's disorder include self-consciousness, shame, impaired self-esteem and social adaptation, and avoidance of social situations that results from being teased and rejected by peers and even parents and teachers. Severe symptoms may interfere with forming satisfying friendships and romantic relationships. The unemployment rate in adults with Tourette's disorder is reportedly as high as 50%. Comorbid disorders and symptoms can also contribute to impairment.

Evaluation and Differential Diagnosis

Patients with Tourette's disorder often attempt to hide symptoms or are unaware of them. They typically suppress tics while in the clinician's office. As a result, the clinician may underestimate the severity or overlook the diagnosis altogether. There are often many years between the first symptoms and the diagnosis.

Neurological history and examination are needed to rule out other movement disorders. Baseline dyskinesias should be assessed before medication is started. If myoclonic seizures are suspected, an EEG may be helpful.

Psychiatric evaluation focuses on identifying concomitant anxiety, mood, behavior, and developmental disorders and stressors that may be exacerbating symptoms. Checklists and standard self-report measures may be used to assess and monitor tics. Specific questions regarding obsessions and compulsions are indicated. School reports of academic performance, standardized test results, tic severity, social skills, and behavioral observations are useful. If school performance is impaired, psychological testing that includes assessment of intellectual functioning and academic achievement can clarify the diagnosis of a comorbid learning disorder or lower intellectual capacity. Other possible interference with school success includes direct effects of tics or premonitory urges, efforts to suppress tics or compulsions, intrusive obsessions, ADHD, anxiety symptoms, social stigmatization, cognitive dulling or anxiety secondary to medication, and excessive absenteeism. The clinician evaluates the child's self-consciousness, ability to manage peer teasing,

social ostracism, and assertiveness. Family history of tics and associated disorders is important, and evaluation of relatives for tic disorders may be considered.

The differential diagnosis includes a wide variety of *neurological disorders* that present with involuntary movements. Patients with *autism spectrum disorder* or *stereotypic movement disorder* may have stereotyped movements that resemble tics. Typical children may have isolated, fleeting tics, but such twitches (blinks, grimaces) and habits are not diagnosed even as a provisional tic disorder unless they persist nearly every day for at least 4 weeks. The clinical boundaries between Tourette's disorder, ADHD, and OCD are blurred in many patients who have symptoms of all three disorders. Complex tics may be difficult to distinguish from compulsions, and some patients have both.

Treatment

Provisional tic disorder does not require treatment. The clinician should advise the family to reduce attention to the symptom and avoid criticism. The clinician may also educate, reassure the patient and family, and encourage them to return if symptoms persist. Chronic tic disorder alone rarely requires specific treatment, unless the tics are disabling or impairing, in which case the same treatments may be used as for Tourette's disorder. There is evidence that tics are highly associated with school problems; therefore, special education programming may be needed (Kurlan et al. 2001).

For Tourette's disorder, a period of initial monitoring helps to establish a baseline of symptom severity. A log or diary can be useful. Children are often brought to clinical attention at a time of crisis, and the evaluation itself, along with reassurance about the nature of the illness, may result in a decrease in symptoms and distress.

Psychosocial Treatments

Sometimes, simple identification and monitoring of tics can lead to sufficient improvement. Taking this conservative approach is espe-

cially warranted in those who are treatment-naïve, have historically been sensitive to medication adverse effects, or who have minimal impairment from tics. Education of the patient and parents, teachers, and other important figures in the child's life is vital. Referral to the Tourette Association of America (formerly known as the Tourette Syndrome Association) local chapter and/or Web site (www.tourette.org) may be invaluable to provide information and support. Psychosocial interventions often precede pharmacological treatment. In many cases, the control of tics is less urgent than the treatment of the accompanying attentional, behavioral, or obsessive-compulsive symptoms. Comprehensive behavioral intervention including habit reversal (a competing response procedure) can yield positive results in managing tics (Piacentini et al. 2010). Supportive psychotherapy may help an individual cope with the stigma of illness, promote self-esteem, improve interpersonal comfort and social skills, and reduce anxiety. Family therapy may assist in reducing the child's stress and helping the family manage associated symptoms. Behavior modification at home and school is recommended for comorbid symptoms of ADHD and ODD. Cognitive-behavioral therapy may be used for obsessive-compulsive symptoms. A school IEP or 504 plan with tutoring, resource room, or special education classroom may be indicated for children with comorbid learning disabilities or behavior problems.

Pharmacotherapy

Medication treatment is complicated by possible adverse effects and the difficulty in determining effectiveness because of the disorder's natural waxing and waning course. The goal is not to eliminate tics but to reduce them to a level that does not interfere with the child's development and functioning. The first step in pharmacological treatment planning is to identify the most troubling target symptoms and choose a medication that is likely to address those symptoms while producing the fewest adverse effects.

For mild to moderate tics, α_2 -adrenergic agonists are generally considered first-line pharmacological treatment because of their rela-

tively more tolerable adverse-effect profile. Both guanfacine and clonidine have demonstrated superior efficacy over placebo in multiple randomized controlled trials (RCTs) (Tourette's Syndrome Study Group 2002; Weisman et al. 2013). Guanfacine is usually preferred because of its association with less sedation and the option of less frequent daily dosing (though both guanfacine and clonidine are now available in once- or twice-daily long-acting formulations). Although there is considerable evidence for the efficacy of antipsychotics, their side-effect profile limits their use as first-line treatment. For more severe tics, antipsychotics may be considered. FDA labeling for pediatric tics exists for the typical antipsychotics pimozide and haloperidol. Of the second-generation antipsychotics, risperidone and ziprasidone have demonstrated efficacy in RCTs (Weisman et al. 2013).

When comorbid symptoms are responsible for greater impairment than the tics, treatment of comorbid conditions should be considered first. Although there was past concern regarding the use of stimulants to treat symptoms of ADHD in the presence of tics, subsequent research has resolved this issue (Cohen et al. 2015). Best practice is to balance the possible risk of tic exacerbation with the impairment caused by the ADHD symptoms. Methylphenidate is effective in treatment of ADHD without exacerbation of tics in children with tic disorders (Tourette's Syndrome Study Group 2002). Although stimulants are safe and effective for many children with tics, careful monitoring is prudent to evaluate for possible stimulant-induced tic exacerbation. Atomoxetine has been shown to improve ADHD symptoms without tic exacerbation in children with ADHD and a tic disorder (Allen et al. 2005). Treatment of OCD with CBT and/or an SSRI may be indicated.

■ REFERENCES

- Allen AJ, Kurlan RM, Gilbert DL, et al: Atomoxetine treatment in children and adolescents with ADHD and comorbid tic disorders. *Neurology* 65(12):1941–1949, 2005 16380617

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 4th Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000
- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Barkley RA : Attention Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment. New York, Guilford, 1990
- Caspi A, Langley K, Milne B, et al: A replicated molecular genetic basis for subtyping antisocial behavior in children with ADHD. *Arch Gen Psychiatry* 65:203–210, 2008 18250258
- Cavanna AE, Servo S, Monaco F, Robertson MM: The behavioral spectrum of Gilles de la Tourette syndrome. *J Neuropsychiatry Clin Neurosci* 21(1):13–23, 2009 19359447
- Charach A, Yeung E, Climans T, Lillie E: Childhood attention-deficit/hyperactivity disorder and future substance use disorders: comparative meta-analyses. *J Am Acad Child Adolesc Psychiatry* 50(1):9–21, 2011 21156266
- Christensen DL, Baio J, Van Naarden Braun K, et al; Centers for Disease Control and Prevention (CDC): Prevalence and characteristics of autism spectrum disorder among children aged 8 years—autism and developmental disabilities monitoring network, 11 sites, United States, 2012. *MMWR Surveill Summ* 65(3):1–23, 2016 27031587
- Coffey BJ, Biederman J, Smoller JW, et al: Anxiety disorders and tic severity in juveniles with Tourette's disorder. *J Am Acad Child Adolesc Psychiatry* 39(5):562–568, 2000 10802973
- Cohen SC, Mulqueen JM, Ferracioli-Oda E, et al: Meta-analysis: risk of tics associated with psychostimulant use in randomized, placebo-controlled trials. *J Am Acad Child Adolesc Psychiatry* 54(9):728–736, 2015 26299294
- Faraone SV, Sergeant J, Gillberg C, Biederman J: The worldwide prevalence of ADHD: is it an American condition? *World Psychiatry* 2(2):104–113, 2003 16946911
- Fung LK, Mahajan R, Nozzolillo A, et al: Pharmacologic treatment of severe irritability and problem behaviors in autism: a systematic review and meta-analysis. *Pediatrics* 137(Suppl 2):S124–S135, 2016 26908468
- Gallagher R, Abikoff H, Spira E: Organizational Skills Training for Children With ADHD: An Empirically Supported Treatment. New York, Guilford Press, 2014

- Hurley AD: Mood disorders in intellectual disability. *Curr Opin Psychiatry* 19(5):465–469, 2006 16874117
- Knight T, Steeves T, Day L, et al: Prevalence of tic disorders: a systematic review and meta-analysis. *Pediatr Neurol* 47(2):77–90, 2012 22759682
- Kurlan R, McDermott MP, Deeley C, et al: Prevalence of tics in schoolchildren and association with placement in special education. *Neurology* 57(8):1383–1388, 2001 11673576
- Leckman JF, Bloch MH, Smith ME, et al: Neurobiological substrates of Tourette's disorder. *J Child Adolesc Psychopharmacol* 20(4):237–247, 2010 20807062
- Loney J, Milich R: Hyperactivity, inattention, and aggression in clinical practice. *Advances in Developmental and Behavioral Pediatrics* 3:113–147, 1982
- Martino D, Zis P, Buttiglione M: The role of immune mechanisms in Tourette syndrome. *Brain Res* 1617:126–143, 2015 24845720
- Matson JL, Shoemaker M: Intellectual disability and its relationship to autism spectrum disorders. *Res Dev Disabil* 30(6):1107–1114, 2009 19604668
- McCracken JT, McGough J, Shah B, et al: Research Units on Pediatric Psychopharmacology Autism Network: Risperidone in children with autism and serious behavioral problems. *N Engl J Med* 347(5):314–321, 2002 12151468
- Munir KM: The co-occurrence of mental disorders in children and adolescents with intellectual disability/intellectual developmental disorder. *Curr Opin Psychiatry* 29(2):95–102, 2016 26779862
- Owen R, Sikich L, Marcus RN, et al: Aripiprazole in the treatment of irritability in children and adolescents with autistic disorder. *Pediatrics* 124(6):1533–1540, 2009 19948625
- Pelham WE, Milich R, Murphy DA, et al: Normative data on the IOWA Conners Teacher Rating Scale. *J Clin Child Psychol* 18:259–262, 1989
- Pfiffner LJ, Rooney M, Haack L, et al: A randomized controlled trial of a school-implemented school-home intervention for attention-deficit/hyperactivity disorder symptoms and impairment. *J Am Acad Child Adolesc Psychiatry* 55(9):762–770, 2016 27566117
- Piacentini J, Woods DW, Scahill L, et al: Behavior therapy for children with Tourette disorder: a randomized controlled trial. *JAMA* 303(19):1929–1937, 2010 20483969
- Plomin R, Kovas Y: Generalist genes and learning disabilities. *Psychol Bull* 131(4):592–617, 2005 16060804

- Robertson MM: Attention deficit hyperactivity disorder, tics and Tourette's syndrome: the relationship and treatment implications. A commentary. *Eur Child Adolesc Psychiatry* 15(1):1–11, 2006 16514504
- Scahill L, McCracken JT, King BH, et al; Research Units on Pediatric Psychopharmacology Autism Network: Extended-release guanfacine for hyperactivity in children with autism spectrum disorder. *Am J Psychiatry* 172(12):1197–1206, 2015 26315981
- Scambler D, Rogers SJ, Wehner EA: Can the checklist for autism in toddlers differentiate young children with autism from those with developmental delays? *J Am Acad Child Adolesc Psychiatry* 40(12):1457–1463, 2001 11765292
- Shaw P, Malek M, Watson B, et al: Trajectories of cerebral cortical development in childhood and adolescence and adult attention-deficit/hyperactivity disorder. *Biol Psychiatry* 74(8):599–606, 2013 23726514
- State MW: The genetics of Tourette disorder. *Curr Opin Genet Dev* 21(3):302–309, 2011 21277193
- Tallmadge J, Barkley RA: The interactions of hyperactive and normal boys with their fathers and mothers. *J Abnorm Child Psychol* 11(4):565–579, 1983 6655156
- Tourette's Syndrome Study Group: Treatment of ADHD in children with tics: a randomized controlled trial. *Neurology* 58(4):527–536, 2002 11865128
- Volkmar F, Siegel M, Woodbury-Smith M, et al; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder. *J Am Acad Child Adolesc Psychiatry* 53(2):237–257, 2014 24472258
- Weisman H, Qureshi IA, Leckman JF, et al: Systematic review: pharmacological treatment of tic disorders—efficacy of antipsychotic and alpha-2 adrenergic agonist agents. *Neurosci Biobehav Rev* 37(6):1162–1171, 2013 23099282

■ ADDITIONAL READING

- Barkley RA: Attention-Deficit Hyperactivity Disorder: A Handbook for Diagnosis and Treatment, 4th Edition. New York, Guilford, 2014
- Faraone SV, Antshel KM: ADHD: non-pharmacologic interventions. *Child Adolesc Psychiatr Clin N Am* 23(4):xiii–xiv, 2014
- Murphy TK, Lewin AB, Storch EA, Stock S; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues

- (CQI): Practice parameter for the assessment and treatment of children and adolescents with tic disorders. *J Am Acad Child Adolesc Psychiatry* 52(12):1341–1359, 2013
- Norbury CF, Gooch D, Wray C, et al: The impact of nonverbal ability on prevalence and clinical presentation of language disorder: evidence from a population study. *J Child Psychol Psychiatry* 57(11):1247–1257, 2016
- Pierce K: Neurodevelopmental disorders: specific learning disorder, communication disorders, and motor disorders, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 157–171
- Pliszka SR: Attention-deficit/hyperactivity disorder, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 173–194
- Tanguay PE, Lohr WD: Autism spectrum disorders, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 135–155
- Toth K, de lacy N, King BH: Intellectual disability, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 105–133
- Towbin KE: Tic disorders, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 461–478

SCHIZOPHRENIA

■ CLINICAL DESCRIPTION

Schizophrenia is a neurodevelopmental disorder that most commonly emerges in early adulthood. DSM-5 (American Psychiatric Association 2013) criteria for schizophrenia are the same at all ages. At least one of three “positive symptoms”—delusions, hallucinations, and disorganized speech—is required for diagnosis. The DSM-IV subtypes have been eliminated because of poor reliability, lack of stability, and limited utility for treatment or prognosis. When the disorder develops before age 18 years, it is called *early-onset schizophrenia* (EOS) or, in the rare cases in which it develops before age 13 years, *childhood-onset schizophrenia* (COS).

Schizophrenia is characterized by positive and negative symptoms. *Positive symptoms* may include hallucinations, delusions, disorganized speech, and grossly disorganized or catatonic behavior. *Negative symptoms* include apathy, avolition, and poverty of speech. Cognitive decline, including deficits in attention, memory, processing speed, and executive function, often occurs at the time of illness onset.

■ EPIDEMIOLOGY

EOS is rare, and COS is even more so. Peak age at onset of schizophrenia is between 15 and 25 years in men and between 20 and 30 years in women. Lifetime prevalence is estimated at 0.3%–0.7%.

■ ETIOLOGY

There is a strong genetic contribution to risk for schizophrenia, with first-degree relatives of an affected person having 5%–20% higher lifetime risk of developing the disorder than the general population. No single gene or genetic locus is implicated; rather, multiple genes and genomic loci are involved, and factors such as epigenetics, somatic mutations, and gene-by-environment interactions likely contribute. Risk factors likely act through these mechanisms and include prenatal infection, obstetric complications, in utero exposure to famine, advanced paternal age, marijuana use, migration, and lack of social support (Kodish and McClellan 2016).

■ COURSE AND PROGNOSIS

As schizophrenia affects cognitive, emotional, and social functioning and development, EOS often has a severe course and poor prognosis. Schizophrenia is characterized by four phases: prodromal, acute, recovery, and residual (McClellan et al. 2013). The *prodromal phase* is marked by a decline in function, which in youth may present as social isolation, academic decline, odd preoccupations, and mood symptoms. This is followed, after a brief or extended course, by the *acute phase*, in which positive symptoms appear. Hallucinations, delusions, and disorganized or dangerous behavior generally bring the child to treatment. *Recovery* from the acute phase may take 6 months or longer and is often incomplete with persistent deficits. The *residual phase* is generally marked by chronic impairment in social and educational function. Adolescents with schizophrenia are at increased risk for death from suicide or accidents.

■ EVALUATION AND DIFFERENTIAL DIAGNOSIS

A comprehensive history is necessary to clarify premorbid functioning and current positive and negative symptoms. Information from multiple sources will increase the likelihood of getting a full assessment of family history, prenatal exposures, exposure to trauma or

neglect, and longitudinal academic and social function. A complete physical exam is indicated to rule out any nonpsychiatric medical condition that might cause psychotic symptoms. Use of neurological consultation, laboratory tests, and neuroimaging evaluations should be guided by the history and physical examination.

Urgent *medical conditions* that must be ruled out or treated in a child presenting with psychosis include delirium; substance intoxication; medication reaction; brain neoplasm; head injury; stroke; autoimmune encephalitis; meningitis; AIDS; fluid, electrolyte, or glucose abnormalities; endocrine abnormalities; Wilson's disease; and porphyria.

Children and adolescents with *mood disorders* may present with psychotic symptoms that overlap with those of schizophrenia. A child with *psychotic depression* may have affective flattening and mood congruent or incongruent hallucinations. Adolescents with *mania* may present with hallucinations, delusions, and disorganized behavior. Longitudinal observation while treating the mood symptoms should clarify the diagnosis over time.

Children and adolescents who have been victims of *trauma* may report psychotic-like symptoms, including reexperiencing in the form of hallucinations, as well as restricted and repetitive play. The intrusive thoughts and compulsive behaviors of *obsessive-compulsive disorder* (OCD) may suggest impaired reality testing without the presence of true psychosis. The symptoms of *autism spectrum disorder* (ASD), including deficits in communication and social aloofness, may mimic the negative symptoms of schizophrenia, but symptoms of ASD are generally earlier in onset, more pervasive, and more stable over time.

Normative experiences can be mistaken for psychotic symptoms. Children may have vivid fantasy play and magical thinking beyond the typical age. They may confuse hallucinations with dreams or the hallucinations that can occur with falling asleep (*hypnagogic*) and waking (*hypnopompic*). They may report apparent hallucinations or delusions that actually reflect shared familial, religious, or cultural beliefs. Young children with *fever* may be prone to acute hallucinations, unrelated to a psychiatric disorder.

■ TREATMENT

Treatment of schizophrenia in youth requires a comprehensive approach that involves the patient, family, and community and incorporates medication as well as psychosocial and educational interventions.

The cornerstone of psychopharmacological treatment for EOS is antipsychotic medication (see Chapter 17, “Psychopharmacology”). Controlled trials have confirmed the effectiveness of antipsychotics in improving psychotic symptoms and overall functioning. Trials have not established clear superiority of second-generation over first-generation antipsychotics in terms of efficacy or tolerability. The U.S. Food and Drug Administration has approved indications for risperidone, olanzapine, paliperidone, quetiapine, aripiprazole, and lurasidone for treatment of youth age 13 years and older with schizophrenia, but controlled trials have also supported haloperidol, loxapine, thioridazine, and thiothixene. Clozapine may be considered in refractory cases (Schneider et al. 2015).

Antipsychotic treatment in youth requires ongoing monitoring for side effects. Both first- and second-generation antipsychotics can produce extrapyramidal symptoms, including dystonia, akathisia, tardive dyskinesia, and neuroleptic malignant syndrome. The second-generation agents are more strongly associated with weight gain and adverse effects on glucose and lipid metabolism. Monitoring should be accompanied by education on diet, exercise, and sleep.

Psychosocial interventions include cognitive-behavioral therapy to address deficits in thought processes, skills training for improved role functioning and self-efficacy, and family-based therapies. There is growing evidence for cognitive remediation in improving verbal memory and executive function in youth with schizophrenia (Armando et al. 2015). Family education and support are crucial to help families with the complex emotional, social, and financial burden of a child with schizophrenia, and to help promote a stable environment for the child. The NIMH RAISE project for first-episode psychosis has demonstrated the superiority of an integrated approach, including supportive education and employment, in im-

proving length of time in treatment, quality of life, participation in work and school, and symptoms (Kane et al. 2016).

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Armando M, Pontillo M, Vicari S: Psychosocial interventions for very early and early-onset schizophrenia: a review of treatment efficacy. *Curr Opin Psychiatry* 28(4):312–323, 2015 26001923
- Kodish I, McClellan JM: Early-onset schizophrenia, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 389–408
- Kane JM, Robinson DG, Schooler NR, et al: Comprehensive versus usual community care for first-episode psychosis: 2-year outcomes from the NIMH RAISE early treatment program. *Am J Psychiatry* 173(4):362–372, 2016 26481174
- McClellan J, Stock S; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter for the assessment and treatment of children and adolescents with schizophrenia. *J Am Acad Child Adolesc Psychiatry* 52(9):976–990, 2013 23972700
- Schneider C, Papachristou E, Wimberley T, et al: Clozapine use in childhood and adolescent schizophrenia: a nationwide population-based study. *Eur Neuropsychopharmacol* 25(6):857–863, 2015 25769917

■ ADDITIONAL READING

- Harvey RC, James AC, Shields GE: A systematic review and network meta-analysis to assess the relative efficacy of antipsychotics for the treatment of positive and negative symptoms in early-onset schizophrenia. *CNS Drugs* 30(1):27–39, 2016 26801655

BIPOLAR DISORDER

■ CLINICAL DESCRIPTION

DSM-5 breaks out bipolar and related disorders into their own chapter (American Psychiatric Association 2013). The DSM-5 criteria for bipolar disorders are the same for children and adults with a few exceptions: cyclothymic disorder (the required duration is 1 year instead of 2 years) and major depressive episode (in children and adolescents, irritable mood can be substituted for depressed mood, and in children, lack of expected weight gain can qualify instead of weight loss).

In the past three decades, the diagnosis of bipolar disorder in children was a focus of significant scrutiny and controversy as some researchers postulated that bipolar disorder manifested differently in children than in adults. They asserted that children presented with a “broad phenotype” of bipolar disorder marked by ultra-rapid-cycling or sustained mixed episodes, chronic irritability, and explosive outbursts. Subsequent research at the National Institute of Mental Health has since suggested that this group of children represents a different clinical entity and that these children do not progress to develop classic or narrow-phenotype bipolar disorder in adulthood. *Disruptive mood dysregulation disorder* (DMDD), introduced in DSM-5, was designed to capture this population. The intent was that only children for whom the strict application of diagnostic symptom criteria (i.e., present with discrete manic or hypomanic episodes of sufficient duration that are a clear departure from their baseline functioning and mood) is met should be assigned a diagnosis of bipolar type I or bipolar type II disorder.

DSM-5 criteria for mania and hypomania have an increased emphasis on changes in activity and energy, in addition to mood. Of the mania diagnostic criteria, children have far less opportunity than adults for spending sprees or foolish investments but may present with hypersexualized behavior or attire. The classic pattern of alternating episodes of depressed and elated mood with corresponding vegetative (sleep, activity, appetite) symptoms does not typically emerge until late adolescence or adulthood. The initial mood episodes are more likely to be depressive episodes. The DSM-IV diagnosis of bipolar I disorder, mixed episode (American Psychiatric Association 2000), has been replaced in DSM-5 with a new specifier: “with mixed features.”

■ EPIDEMIOLOGY

Bipolar disorder in children is extremely rare. Mania is rare before middle adolescence but by late adolescence is nearly as common as in adults. A community survey of high school students found a lifetime prevalence of 1% for all bipolar disorders. An additional 5.7% of the sample had persistent subthreshold hypomanic and associated symptoms (Lewinsohn et al. 1995). In one study, 45% of children and adolescents diagnosed with DSM-IV-TR bipolar disorder not otherwise specified converted to bipolar type I and type II over 5 years (Axelson et al. 2011). Family history of mania or hypomania was the strongest predictor of conversion. Approximately 20% of all patients with bipolar disorder have their first episode during adolescence.

■ ETIOLOGY

Biological and genetic factors play a large role in the etiology of bipolar disorder. More than one third of first-degree relatives of individuals with bipolar spectrum disorder have symptoms that meet criteria for a bipolar spectrum disorder. Twin studies also demonstrate significant heritability for the disorder (Althoff et al. 2005). In addition, schizophrenia and bipolar disorder likely share genetic contributions (Lichtenstein et al. 2009), and relatives of individuals

with schizophrenia have a higher risk of developing either bipolar disorder or schizophrenia. However, environmental factors such as physical and sexual abuse, negative parenting styles, poor social supports, and prenatal alcohol exposure may influence development of the disorder.

■ COURSE AND PROGNOSIS

Mania and hypomania occurring before adulthood are often misdiagnosed or not recognized. Heedless risk taking, highly energized affect, and developmentally inappropriate sexual preoccupation and behavior are useful markers for mania. Precocious sexuality in a child with manic symptoms should not be presumed to be evidence of sexual abuse. Grandiosity is expressed in different ways than in adults, but the experienced clinician can distinguish these beliefs from normal bragging or childhood fantasy (Geller et al. 2002b). Manic or hypomanic decreased need for sleep must be differentiated from insomnia, resistance to bedtime, or substance use. Family history and longitudinal course may provide important clues. Childhood-onset bipolar disorder carries a worse prognosis than bipolar disorder of adult onset. There is frequent comorbidity with anxiety disorders (62.9%), behavior disorders (attention-deficit/hyperactivity disorder [ADHD], intermittent explosive disorder, oppositional defiant disorder [ODD], conduct disorder) (44.8%), and substance use disorders (36.6%) (Merikangas et al. 2011).

Prospective studies of adolescents and longitudinal studies of children of parents with bipolar disorder suggest that anxiety disorders may be an early manifestation in individuals who subsequently develop bipolar disorder (Duffy et al. 2007; Henin et al. 2005; Lewinsohn et al. 2002).

■ EVALUATION AND DIFFERENTIAL DIAGNOSIS

Both mania and agitated depression may be confused with *ADHD*, but mood disorders are typically episodic, whereas *ADHD* is a

chronic condition with onset in early childhood. Symptoms of bipolar disorder that best discriminate from ADHD are elation, grandiosity, flight of ideas/racing thoughts, decreased need for sleep, and hypersexuality (Geller et al. 2002a). Mania in childhood and adolescence is frequently misdiagnosed as *schizophrenia* because the initial manic episode can include psychotic symptoms. Manic or depressed youth may have some symptoms of *ODD* or *conduct disorder* that are secondary to their mood symptoms. Of course, *disruptive behavior disorder* may precede bipolar disorder or develop in parallel. *Secondary mania* may result from prescribed medication (e.g., steroids, carbamazepine, antidepressants, stimulants), illegal drugs (e.g., cocaine, amphetamines), metabolic abnormalities (especially hyperthyroidism), or central nervous system disturbances (e.g., tumor, trauma, multiple sclerosis, epilepsy, infections). Of note, DSM-5 allows for a manic episode or hypomanic episode to be diagnosed if the episode emerges in the context of antidepressant treatment as long as the full syndrome persists beyond the physiological effects of the treatment. This should be distinguished from a medication-related side effect of sleeplessness, agitation, or irritability that desists after cessation of the medication.

Bipolar disorder is frequently comorbid with ADHD, ODD, conduct disorder, substance and alcohol use disorders, and anxiety, but teasing apart the distinct diagnoses can be challenging during an acute manic episode.

■ TREATMENT

It is extremely difficult to implement controlled trials in youth with bipolar disorder. Attempts have been made to extrapolate from research on adults, but the findings are not consistent. All of the mood-stabilizing medications have significant side effects. Drugs with current U.S. Food and Drug Administration indications for pediatric bipolar disorder/mania are lithium (age 12 years and older); the atypical antipsychotics aripiprazole, risperidone, asenapine, and quetiapine (age 10 years and older); and olanzapine (age 13 years

and older; other drugs should be considered first because of weight gain). Aripiprazole and lithium have approved indications for prevention of recurrence of mania. Compared with adults, youth with mania have greater response to the atypical antipsychotics, but youth experience lower effect sizes with the mood stabilizers divalproex and lithium. In a direct comparison in youth with bipolar disorder, risperidone was faster and more effective than divalproex in acutely reducing symptoms of mania (Pavuluri et al. 2010). The Treatment of Early Age Mania (TEAM) study (Geller et al. 2012) found risperidone to be superior to both divalproex and lithium.

In contrast to the usual preference for monotherapy in pediatric psychopharmacology, combinations of medications are often needed for mania. Efficacy in bipolar depression remains elusive. Because of the risk of precipitating mania, antidepressants are generally not used in bipolar depression until treatment with a mood stabilizer is established. See Chapter 17 (“Psychopharmacology”) for information on the use of lithium, antipsychotic medications, and anticonvulsants.

There are multiple empirically supported psychosocial interventions to be added to medication treatment, especially for adolescents. Models include Family-Focused Treatment for Adolescents (FFT-A) (Miklowitz et al. 2008), family psychoeducation, child- and family-focused cognitive-behavioral therapy, and others (Weinstein et al. 2013).

■ REFERENCES

- Althoff RR, Faraone SV, Rettew DC, et al: Family, twin, adoption, and molecular genetic studies of juvenile bipolar disorder. *Bipolar Disord* 7(6):598–609, 2005 16403185
- American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 4th Edition, Text Revision. Washington, DC, American Psychiatric Association, 2000
- American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition. Arlington, VA, American Psychiatric Association, 2013

- Axelsson DA, Birmaher B, Strober MA, et al: Course of subthreshold bipolar disorder in youth: diagnostic progression from bipolar disorder not otherwise specified. *J Am Acad Child Adolesc Psychiatry* 50(10):1001–16.e3, 2011 21961775
- Duffy A, Alda M, Crawford L, et al: The early manifestations of bipolar disorder: a longitudinal prospective study of the offspring of bipolar parents. *Bipolar Disord* 9(8):828–838, 2007 18076532
- Geller B, Zimmerman B, Williams M, et al: DSM-IV mania symptoms in a prepubertal and early adolescent bipolar disorder phenotype compared to attention-deficit hyperactive and normal controls. *J Child Adolesc Psychopharmacol* 12(1):11–25, 2002a 12014591
- Geller B, Zimmerman B, Williams M, et al: Phenomenology of prepubertal and early adolescent bipolar disorder: examples of elated mood, grandiose behaviors, decreased need for sleep, racing thoughts and hypersexuality. *J Child Adolesc Psychopharmacol* 12(1):3–9, 2002b 12014593
- Geller B, Luby JL, Joshi P, et al: A randomized controlled trial of risperidone, lithium, or divalproex sodium for initial treatment of bipolar I disorder, manic or mixed phase, in children and adolescents. *Arch Gen Psychiatry* 69(5):515–528, 2012 22213771
- Henin A, Biederman J, Mick E, et al: Psychopathology in the offspring of parents with bipolar disorder: a controlled study. *Biol Psychiatry* 58(7):554–561, 2005 16112654
- Lewinsohn PM, Klein DN, Seeley JR: Bipolar disorders in a community sample of older adolescents: prevalence, phenomenology, comorbidity, and course. *J Am Acad Child Adolesc Psychiatry* 34(4):454–463, 1995 7751259
- Lewinsohn PM, Seeley JR, Buckley ME, Klein DN: Bipolar disorder in adolescence and young adulthood. *Child Adolesc Psychiatr Clin N Am* 11(3):461–475, vii, 2002 12222078
- Lichtenstein P, Yip BH, Björk C, et al: Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: a population-based study. *Lancet* 373(9659):234–239, 2009 19150704
- Merikangas KR, Jin R, He JP, et al: Prevalence and correlates of bipolar spectrum disorder in the world mental health survey initiative. *Arch Gen Psychiatry* 68(3):241–251, 2011 21383262
- Miklowitz DJ, Axelsson DA, Birmaher B, et al: Family-focused treatment for adolescents with bipolar disorder: results of a 2-year randomized trial. *Arch Gen Psychiatry* 65(9):1053–1061, 2008 18762591

- Pavuluri MN, Henry DB, Findling RL, et al: Double-blind randomized trial of risperidone versus divalproex in pediatric bipolar disorder. *Bipolar Disord* 12(6):593–605, 2010 20868458
- Weinstein SM, West AE, Pavuluri M: Psychosocial intervention for pediatric bipolar disorder: current and future directions. *Expert Rev Neurother* 13(7):843–850, 2013 23898854

■ ADDITIONAL READING

- Carlson GA, Pataki C, Meyer SE: Bipolar disorder, in Dulcan's *Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 277–304

DEPRESSIVE DISORDERS

■ DISRUPTIVE MOOD DYSREGULATION DISORDER

Clinical Description

Disruptive mood dysregulation disorder (DMDD) was introduced in DSM-5 (American Psychiatric Association 2013) in order to provide a diagnostic home for a group of children and adolescents who were formerly being diagnosed with pediatric or “broad phenotype” bipolar disorder. The diagnosis of DMDD arose out of research on a subgroup of children with “severe mood dysregulation” (SMD), a syndrome characterized by severe, nonepisodic, chronic irritability and many of the hyperarousal symptoms of mania without the discrete, episodic nature of mania or hypomania. Unlike youth with narrow-phenotype or classic bipolar disorder, youth with SMD had high rates of comorbid attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), and anxiety disorders; did not have mood episodes; had lower familial rates of bipolar disorder; and demonstrated a different neurocognitive and neuroanatomic profile. Longitudinal follow-up of individuals with SMD to adulthood demonstrated an increased risk of developing depressive and anxiety disorders but not bipolar disorder (Leibenluft 2011).

The diagnosis of DMDD requires the presence of at least a 12-month period of persistently irritable or angry mood punctuated by severe, recurrent temper outbursts of physical and/or verbal aggression that are excessive for the situation or provocation, in at

least two settings (not only at home). The outbursts must be developmentally inappropriate and occur at least three times per week (on average). There cannot be a period of 3 or more consecutive months during the 12 months in which the diagnostic symptom criteria were not met. The diagnosis cannot be initially made before age 6 years or after age 18 years, and the onset of symptoms must be before age 10 years. The angry mood and dysregulated behavior of children with DMDD impair home, school, and social life. If intermittent explosive disorder or bipolar disorder is present or if full symptom criteria for a hypomanic or manic episode (with the exception of duration) are met for more than a day, DMDD cannot be diagnosed. If a child's symptoms meet criteria for both ODD and DMDD, he or she should be diagnosed only with DMDD. DMDD and ADHD can be diagnosed simultaneously if criteria for both are met.

Given the significant clinical overlap and comorbidity with other psychiatric disorders (especially ODD, conduct disorder, and depressive disorders), more studies are required to establish the distinct phenomenology of DMDD.

Epidemiology and Comorbidity

Reported prevalence rates vary widely, with low rates in community samples and significantly higher rates in clinic settings and in younger children. An analysis of two large community samples of children ages 9–17 years (Great Smoky Mountains Study of Youth and Caring for Children in the Community) yielded a prevalence rate of DMDD of approximately 1% (Copeland et al. 2013), with a cumulative prevalence of 4.4%. Comorbidity with ODD, conduct disorder, and depressive disorders was substantial. When the age at onset criterion was disregarded, the prevalence rate in a preschool community cohort (ages 2–5 years) was higher (3.3%) (Copeland et al. 2013). In a different general population sample (ages 6–12 years, $N=665$), the prevalence of DMDD was 9%. Of the 9%, 92% had symptoms that met criteria for ODD, whereas only 66% of children with ODD had symptoms that met criteria for DMDD (Mayes et al. 2016).

DMDD rates in an outpatient clinical sample of 706 youth (ages 6–12) enrolled in the Longitudinal Assessment of Manic Symptoms (LAMS) study were 26% at intake and a cumulative 40% by the end of 2 years. DMDD had low diagnostic stability, with 52% of those who qualified for a diagnosis of DMDD meeting criteria at only one assessment during the 2-year study. DMDD overlapped significantly with ODD and conduct disorder, and was not associated with current, new onset, or parental history of mood or anxiety disorders (Axelson et al. 2012). In a community mental health clinic sample, the prevalence rate of DMDD was found to be 31%, with high rates of comorbidity with ODD (96%), conduct disorder (24%), and ADHD (81%) (Freeman et al. 2016).

The significant overlap between DMDD and ODD has raised concerns about the diagnostic validity and utility of the DMDD construct and has led some to argue that it should be an ODD specifier rather than a separate DSM diagnosis (Mayes et al. 2016).

Etiology

As DMDD is a new diagnosis, little is known about its etiology. A family history of bipolar disorder may increase the risk of DMDD (Sparks et al. 2014).

Course and Prognosis

Young adults who retrospectively met criteria for DMDD in childhood had higher rates of anxiety, depression, and other psychiatric illness; poorer health; lower socioeconomic status; more criminal problems; and lower overall functioning in adulthood than individuals who did not qualify for the diagnosis in childhood (Copeland et al. 2014).

Evaluation and Differential Diagnosis

It is important to obtain parent and teacher reports to establish the presence of impairment in more than one setting. It may be challenging for parents to recall whether temper outbursts meet duration

and frequency criteria for diagnosis. It is important to rule out other disorders that may manifest with irritability and outbursts, such as *major depressive disorder*, *persistent depressive disorder*, untreated *ADHD*, or undiagnosed *communication or learning disorders*. In addition, in adolescents, it is important to rule out *substance or alcohol use disorders*, which can present with irritability and angry outbursts. One should obtain a detailed medication history to determine if *medication side effects* (such as from corticosteroids or stimulant medications) may be causing irritability. The diagnosis of DMDD precludes making a diagnosis of ODD, even if the criteria are met for both diagnoses, since DMDD is considered to be a more severe disorder.

Treatment

Treatment guidelines have not been established for DMDD (Roy et al. 2014). Preliminary data from studies of children with SMD suggest a positive effect of stimulant medications (especially in children with comorbid ADHD and aggression) and of risperidone. Valproate has shown some benefit in treatment of aggression in children with ADHD, ODD, and conduct disorder (Roy et al. 2014). More recently, an open trial of stimulants in children with both ADHD and DMDD demonstrated good tolerance and clinically significant improvements in ADHD, depressive, and ODD symptoms, although many patients continued to have significant impairment (Baweja et al. 2016).

■ MAJOR DEPRESSIVE DISORDER AND PERSISTENT DEPRESSIVE DISORDER

Clinical Description

DSM-5 criteria for depressive disorders are the same for children and adults, with a few exceptions (Table 6–1). Persistent depressive disorder (PDD) (also called *dysthymia*) encompasses the DSM-IV di-

TABLE 6–1. **Developmental differences in DSM-5 criteria for depressive disorders**

Disorder	Adults	Children
Major depression	Depressed mood Change in weight or appetite	Can be irritable mood Can be failure to make expected weight gains
Persistent depressive disorder (dysthymia)	Depressed mood At least 2-year duration	Can be irritable mood At least 1-year duration

agnoses of both dysthymic disorder and chronic major depressive disorder (MDD). If the criteria for PDD are met, it can be diagnosed even when full criteria for MDD are also present. Children with PDD present with a history of irritable or sad mood for at least 1 year with at least two of the following: disturbance in appetite, disturbance in sleep, low energy or fatigue, low self-esteem, poor concentration or difficulty making decisions, and hopelessness. On the basis of research, DSM-5 has removed the previous MDD exclusion criterion for recent bereavement.

At all ages, depressed mood inferred from observation of the patient (appears sad or tearful) can be substituted for the patient's report. The anhedonia criterion of diminished interest or pleasure in activities can be met by observed apathy. Children and adolescents often report this as pervasive boredom. Mood-related symptoms may be manifested in different ways at different developmental levels. Key indicators of depression in young people are declining school performance, withdrawal from social activities, somatic symptoms (especially headaches and abdominal pain), sleep difficulties, and conduct problems. Consistent neurovegetative symptoms are rare in childhood depression or dysthymia.

Epidemiology

The prevalence of major depression has been estimated at 1%–3% in prepubertal children and 3%–9% in adolescents, although rates

vary with the population, the diagnostic criteria, and the methods of assessment. In the Oregon Adolescent Depression Project (Lewinsohn et al. 1994), the lifetime prevalence of at least one episode of major depression by late adolescence was 20%–25%, and the lifetime prevalence of dysthymic disorder was 3%. Many more youth have depressive symptoms than have full major depression or dysthymia. Before puberty, depression is equally common in boys and girls, with a change in adolescence to the female predominance found in adults.

Etiology

Etiological factors for mood disorders in children are similar to those in adults. Depression in a parent is a significant risk factor via both genetic transmission and interpersonal influence (i.e., parent's modeling, emotional unavailability, decreased capacity for parenting). Abuse and neglect may be significant precipitants, especially in very young children.

Course and Prognosis

Mood disorders in childhood are serious and potentially fatal problems. In a 15-year follow-up of children with major depression, 4% had died by suicide (Wolk and Weissman 1996). A 10- to 15-year follow-up of a clinical sample of subjects with adolescent onset of major depression found that nearly 8% had committed suicide (Weissman et al. 1999). Risk of suicide increases with a past history of suicide attempt, comorbid substance abuse, access to lethal means (such as a gun), impulsivity, conduct disorder, family history of suicide attempts, or physical or sexual abuse. Over the full age range, earlier age at onset implies a lengthier and more severe course and greater genetic loading. Over the past 25 years, the age at onset of major depression appears to have decreased. In a prospective natural history observation study of clinically referred prepubertal children, the median length of the major depressive episode was 9 months. The median length of time for recovery from dysthy-

mia was 4 years. Dysthymia often progressed to a major depressive episode before resolving. Risk for subsequent episodes of major depression or dysthymia is high (Kovacs 1996). In a sample of high school students, episodes of major depression lasted as long as 10 years (mean=26 weeks; median=8 weeks). Of those who recovered, one-third had another episode within 4 years. Suicidal ideation was associated with earlier onset, longer episodes, and earlier relapse of depression (Lewinsohn et al. 1994). Follow-up studies of depressed youth consistently find increased risk of repeated mood disorders and other psychiatric disorders, personality disorders, substance abuse, and impairment in subsequent educational and vocational achievement and peer and family relationships.

Childhood-onset depression is more likely than adult-onset depression to evolve into bipolar disorder. Among adolescents with major depression, subsequent bipolar disorder was predicted by precipitous onset of symptoms, psychomotor retardation, psychotic features, psychopharmacologically precipitated hypomania, and family history of bipolar disorder (Strober and Carlson 1982).

Evaluation and Differential Diagnosis

The clinician should ask children direct questions about depression. Young children have more difficulty recognizing and verbalizing their feelings and may use idiosyncratic words to describe dysphoria or anhedonia. Both parent and child reports are essential. Some children report their mood states more accurately than their parents can, although young children may have limited insight into other symptoms. A trained clinician should observe children for depressed affect. The Children's Depression Inventory (CDI; Kovacs 1985) may be a useful self-report screening instrument.

Assessment of the degree of potentially dangerous behavior is crucial. The clinician can question children directly regarding suicidal ideation, suicide plans, and suicide attempts. No evidence indicates that inquiries about wishes or attempts to die increase the risk of self-destructive behavior. Substance abuse increases the risk of suicide. The clinician should ask specifically about emotional or

physical abuse or neglect of the child, access to lethal weapons, family members or friends who have attempted suicide, nonsuicidal self-injury, prior suicide attempts, and any potential protective factors (relationships or spiritual beliefs).

Even in nonclinical epidemiological samples, anxiety disorders, ADHD, and disruptive behavior disorders frequently coexist with depression and dysthymia, with onset before or after the onset of the mood disorder. *Alcohol or drug abuse* may cause a secondary depression or may represent an attempt to “self-medicate” dysphoria. Diagnosis of a mood disorder may be impossible without observing the patient in a drug-free state. *Separation anxiety disorder* may resemble major depression or dysthymic disorder or may coexist with it. Children younger than 4 years may develop a clinical picture similar to major depression when separated from their parents. Children with *reactive attachment disorder* secondary to parental abuse or neglect who present with lethargy, apathy, and withdrawal may appear depressed.

The strong family clustering of mood disorders suggests that parents and siblings should be evaluated routinely for mood and anxiety disorders, and treatment should be provided or arranged as necessary.

Treatment

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

Up to 60% of patients with mild to moderate depression respond to supportive treatment. However, early-onset mood disorders often have devastating effects on development. Long-term treatment is often needed. Even after spontaneous remission or successful treatment, reduced coping skills, cognitive patterns associated with depression, and impaired interpersonal relationships with peers and family members may require individual, group, or family therapy to address developmental deficits or sequelae of the depression. Whatever

the treatment, involvement of the family is even more crucial than for adult patients. Both patients and families benefit from psychoeducation (provision of information about the disorder and its treatment) (see Appendix, “Resources for Parents”) and from instruction in relapse prevention (taking medication as prescribed, recognizing early symptoms of relapse, and avoiding precipitants of relapse such as sleep deprivation and drug abuse).

For nonpsychotic depression, psychotherapy is typically the first treatment. Medication is added if symptoms do not improve in 4–6 weeks (see Chapter 17, “Psychopharmacology”). For more severe depression, medication is indicated as initial treatment. Fluoxetine (age 8 years and older) and escitalopram (age 12 years and older) are currently approved by the U.S. Food and Drug Administration (FDA) for the treatment of pediatric major depressive disorder.

Cognitive-behavioral therapy (CBT) and interpersonal therapy (IPT) techniques developed for the treatment of depression in adults have been adapted for use in children and adolescents (Mufson et al. 2004) (see also “Additional Reading”). Social withdrawal and limited peer relationships may respond to behavioral activation and social skills training. Remedial education or tutoring may be needed when the illness has interfered with learning in school.

For adolescent depression, a National Institute of Mental Health (NIMH)–funded multisite study (Treatment for Adolescents With Depression Study Team 2003) compared randomized assignment to fluoxetine, CBT, the combination, or placebo in 439 adolescents with MDD. Summary findings were that on the Child Depression Rating Scale, fluoxetine (with or without CBT) was superior to placebo in efficacy with an acceptable risk-benefit profile, and that at week 12 of treatment, combined treatment and fluoxetine alone were equal in benefit and had greater benefit than CBT, which was not significantly different from placebo (March et al. 2004). Response was faster with combined treatment than with CBT alone, but the results of all active treatments converged by 18 weeks (Kennard et al. 2009). Further analysis showed (somewhat counterintuitively) that combined treatment had an advantage over fluoxetine alone for

mild to moderate depression but no significant advantage for moderate to severe depression.

The subsequent NIMH-funded six-site Treatment of Resistant Depression in Adolescents (TORDIA) study included 334 adolescents with MDD or dysthymia who had not responded to an adequate trial of a selective serotonin reuptake inhibitor (SSRI) for at least 8 weeks. Subjects were randomly assigned to one of four conditions: switch to a different SSRI (predominantly citalopram) or to venlafaxine or add CBT to either of those medication switches. Adding CBT to either medication switch was more effective than a medication switch alone. There was no difference in efficacy between switch to another SSRI or venlafaxine, but venlafaxine produced more side effects (Brent et al. 2008). The initial trajectory by 6 weeks of treatment predicted whether the patient would remit, suggesting that clinicians should not wait longer than 6–8 weeks to switch or add treatments if the patient is not responding.

Even with evidence-based treatments, many youth remain impaired and vulnerable to relapse. Research is ongoing to find strategies to improve remission rate and reduce or delay relapses. For severe (especially if psychotic) adolescent depression unresponsive to adequate trials of pharmacotherapy, electroconvulsive therapy may be considered, although there are no research studies in adolescents. Transcranial magnetic stimulation appears to be useful in depressed adults, but there is no completed controlled study in adolescents.

Children and adolescents who have seasonal affective disorder may respond to morning treatment with bright white light (Swedo et al. 1997).

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Axelson D, Findling RL, Fristad MA, et al: Examining the proposed disruptive mood dysregulation disorder diagnosis in children in the Longitudinal Assessment of Manic Symptoms study. *J Clin Psychiatry* 73(10):1342–1350, 2012 23140653

- Baweja R, Belin PJ, Humphrey HH, et al: The effectiveness and tolerability of central nervous stimulants in school-age children with attention-deficit/hyperactivity disorder and disruptive mood dysregulation disorder across home and school. *J Child Adolesc Psychopharmacol* 26(2):154–163, 2016 26771437
- Brent D, Emslie G, Clarke G, et al: Switching to another SSRI or to venlafaxine with or without cognitive behavioral therapy for adolescents with SSRI-resistant depression: the TORDIA randomized controlled trial. *JAMA* 299(8):901–913, 2008 18314433
- Copeland WE, Angold A, Costello EJ, Egger H: Prevalence, comorbidity, and correlates of DSM-5 proposed disruptive mood dysregulation disorder. *Am J Psychiatry* 170(2):173–179, 2013 23377638
- Copeland WE, Shanahan L, Egger H, et al: Adult diagnostic and functional outcomes of DSM-5 disruptive mood dysregulation disorder. *Am J Psychiatry* 171(6):668–674, 2014 24781389
- Freeman AJ, Youngstrom EA, Youngstrom JK, Findling RL: Disruptive mood dysregulation disorder in a community mental health clinic: prevalence, comorbidity and correlates. *J Child Adolesc Psychopharmacol* 26(2):123–130, 2016 26745325
- Kennard BD, Silva SG, Tonev S, et al: Remission and recovery in the Treatment for Adolescents with Depression Study (TADS): acute and long-term outcomes. *J Am Acad Child Adolesc Psychiatry* 48(2):186–195, 2009 19127172
- Kovacs M: The Children's Depression Inventory (CDI). *Psychopharmacol Bull* 21(4):995–998, 1985 4089116
- Kovacs M: The course of childhood-onset depressive disorders. *Psychiatr Ann* 26:326–330, 1996
- Leibenluft E: Severe mood dysregulation, irritability, and the diagnostic boundaries of bipolar disorder in youths. *Am J Psychiatry* 168(2):129–142, 2011 21123313
- Lewinsohn PM, Clarke GN, Seeley JR, Rohde P: Major depression in community adolescents: age at onset, episode duration, and time to recurrence. *J Am Acad Child Adolesc Psychiatry* 33(6):809–818, 1994 7598758
- March J, Silva S, Petrycki S, et al; Treatment for Adolescents With Depression Study (TADS) Team: Fluoxetine, cognitive-behavioral therapy, and their combination for adolescents with depression: Treatment for Adolescents With Depression Study (TADS) randomized controlled trial. *JAMA* 292(7):807–820, 2004 15315995

- Mayes SD, Waxmonsky JD, Calhoun SL, Bixler EO: Disruptive mood dysregulation symptoms and association with oppositional defiant and other disorders in a general population child sample. *J Child Adolesc Psychopharmacol* 26(2):101–106, 2016 26745442
- Mufson L, Dorta KP, Wickramaratne P, et al: A randomized effectiveness trial of interpersonal psychotherapy for depressed adolescents. *Arch Gen Psychiatry* 61(6):577–584, 2004 15184237
- Roy AK, Lopes V, Klein RG: Disruptive mood dysregulation disorder: a new diagnostic approach to chronic irritability in youth. *Am J Psychiatry* 171(9):918–924, 2014 25178749
- Sparks GM, Axelson DA, Yu H, et al: Disruptive mood dysregulation disorder and chronic irritability in youth at familial risk for bipolar disorder. *J Am Acad Child Adolesc Psychiatry* 53(4):408–416, 2014 24655650
- Strober M, Carlson G: Bipolar illness in adolescents with major depression: clinical, genetic, and psychopharmacologic investigation. *Arch Gen Psychiatry* 39:549–555, 1982 7092488
- Swedo SE, Allen AJ, Glod CA, et al: A controlled trial of light therapy for the treatment of pediatric seasonal affective disorder. *J Am Acad Child Adolesc Psychiatry* 36(6):816–821, 1997 9183137
- Treatment for Adolescents With Depression Study Team: Treatment for Adolescents With Depression Study (TADS): rationale, design, and methods. *J Am Acad Child Adolesc Psychiatry* 42(5):531–542, 2003 12707557
- Weissman MM, Wolk S, Goldstein RB, et al: Depressed adolescents grown up. *JAMA* 281(18):1707–1713, 1999 10328070
- Wolk SI, Weissman MM: Suicidal behavior in depressed children grown up: preliminary results of a longitudinal study. *Psychiatr Ann* 26:331–335, 1996

■ ADDITIONAL READING

- Birmaher B, Brent DA: Depressive and disruptive mood dysregulation disorders, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 245–276
- Friedberg RD, McClure JM: *Clinical Practice of Cognitive Therapy With Children and Adolescents: The Nuts and Bolts*, 2nd Edition. New York, Guilford, 2015

- Kennard BD, Hughes JL, Foxwell AA: CBT for Depression in Children and Adolescents: A Guide to Relapse Prevention. New York, Guilford, 2016
- Mufson L, Dorta KP, Moreau D, Weissman MM: Interpersonal Psychotherapy for Depressed Adolescents, 2nd Edition. New York, Guilford, 2004

ANXIETY DISORDERS

Although fears and anxiety occur normally during development (Table 7-1), anxiety disorders in children remain underrecognized and undertreated and can lead to lack of social competence, rejection or neglect by peers, academic underachievement, and eventual reduced occupational and social relationship functioning. In contrast to the disruptive behavior disorders, anxiety disorders often cause more distress in the child than in the parents and are thus considered “internalizing” disorders. The Anxiety Disorders Interview Schedule for Children and Parents (ADIS; Silverman et al. 2001) is a semistructured interview that clinicians may use to evaluate many anxiety disorders.

■ SEPARATION ANXIETY DISORDER

Clinical Description

Separation anxiety disorder (SAD) was moved to the anxiety disorders section in DSM-5 (American Psychiatric Association 2013). The core symptoms remain essentially the same, but the description was expanded to include adult separation anxiety disorder (e.g., the adult’s attachment figure may be the child). Onset no longer is required to be before 18 years of age, and the disorder may occur in adulthood. The required duration remains at least 4 weeks for children and adolescents but was increased to at least 6 months in adults. In SAD, cognitive, affective, somatic, and behavioral symptoms appear in response to genuine or fantasized separation from the individual(s) to whom the child is most attached. The key feature of SAD is excessive and age-inappropriate anxiety, fear, or

TABLE 7-1. Common normal fears

Developmental stage	Feared object or situation
Birth to 6 months	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/sickness Burglars Being sent to the principal Punishment Natural disasters Failure/rejection
12-18 years	Tests in school Low social competence Social evaluation Social embarrassment Psychological abnormality

worry concerning separation from home or primary attachment figures (Box 7-1). The anxiety can be experienced and expressed as an unrealistic fear of or worry about the child and parent being permanently separated due to injury, kidnapping, harm, or death. Symptoms can include nightmares with separation themes and inability to sleep alone, attend school, visit friends, go on errands, or stay at camp. To avoid leaving home, children with SAD will often complain of somatic symptoms such as stomachaches or headaches (which they may actually be experiencing) or report distressing events such as bullying by peers or being picked on by teachers.

Box 7–1 DSM-5 Diagnostic Criteria for Separation Anxiety Disorder

- A. Developmentally inappropriate and excessive fear or anxiety concerning separation from those to whom the individual is attached, as evidenced by at least three of the following:
 - 1. Recurrent excessive distress when anticipating or experiencing separation from home or from major attachment figures.
 - 2. Persistent and excessive worry about losing major attachment figures or about possible harm to them, such as illness, injury, disasters, or death.
 - 3. Persistent and excessive worry about experiencing an untoward event (e.g., getting lost, being kidnapped, having an accident, becoming ill) that causes separation from a major attachment figure.
 - 4. Persistent reluctance or refusal to go out, away from home, to school, to work, or elsewhere because of fear of separation.
 - 5. Persistent and excessive fear of or reluctance about being alone or without major attachment figures at home or in other settings.
 - 6. Persistent reluctance or refusal to sleep away from home or to go to sleep without being near a major attachment figure.
 - 7. Repeated nightmares involving the theme of separation.
 - 8. Repeated complaints of physical symptoms (e.g., headaches, stomachaches, nausea, vomiting) when separation from major attachment figures occurs or is anticipated.
- B. The fear, anxiety, or avoidance is persistent, lasting at least 4 weeks in children and adolescents.
- C. The disturbance causes clinically significant distress or impairment in social, academic, occupational, or other important areas of functioning.

- D. The disturbance is not better explained by another mental disorder, such as refusing to leave home because of excessive resistance to change in autism spectrum disorder; delusions or hallucinations concerning separation in psychotic disorders; refusal to go outside without a trusted companion in agoraphobia; worries about ill health or other harm befalling significant others in generalized anxiety disorder; or concerns about having an illness in illness anxiety disorder.
-

Not all children with SAD miss excessive amounts of school, and not all children with school absenteeism have SAD. However, school refusal often occurs within the context of psychiatric disorders, psychosocial vulnerabilities, impaired family functioning, parental psychopathology, and/or stressful life events. Approximately three-quarters of children with SAD exhibit school avoidance (Last et al. 1987). School absenteeism has various etiologies (Table 7–2). In clinical samples of children and adolescents with school refusal, anxiety and depressive disorders, alone or in combination, are very common.

Epidemiology

SAD has a reported prevalence of 3%–5% in youth (Shear et al. 2006), with higher prevalence in childhood compared with adolescence (Breton et al. 1999) and much higher rates of symptoms that do not meet full diagnostic criteria (Kashani and Orvaschel 1990). More recent research has shown that females have a higher prevalence of SAD than males, in contrast to older studies that found no gender differences. As with other anxiety disorders, SAD appears to aggregate in families.

Etiology

A variety of theoretical perspectives exist on the etiology of SAD. The high familial concordance of anxiety disorders makes it difficult to separate the contributions of genetic inheritance, tempera-

TABLE 7-2. Causes of school absenteeism

Separation anxiety disorder
Other psychiatric disorders
Mood disorder
Anxiety disorder
Generalized anxiety disorder
Social anxiety disorder
Panic disorder
Obsessive-compulsive disorder
Psychotic disorder
Truancy (often associated with conduct disorder)
Substance use
Sociocultural conformity
Permission granted by family (overt or covert)
Normative peer behavior
Realistic fear
Excessive teasing/humiliation or verbal harassment/bullying
Physical bullying—intimidation or bodily harm
Academic struggles or avoidance
Social stressors
Poverty
Homelessness

ment, family dynamics, modeling, and other environmental factors. An interactive mechanism is most likely.

Developmental theorists focus on the interplay between the exploring toddler's normal uncertainty about the location of the caregiver and the at-risk child's anxious or insecure attachment. Behaviorally oriented theories emphasize the maintenance of symptoms by conditioned fear, based on stimulus generalization and reinforcement. Research using an attachment theory model found that insecure mother-infant attachment and maternal sensitivity each separately predicted separation anxiety in children at age 6 years (Dallaire and Weinraub 2005).

Biological theories focus on temperamental, genetic, and physiological factors. More specifically, research reveals age-specific ef-

fects of both genes and shared environment on anxiety, depression, and withdrawn behavior in childhood and adolescence (Lamb et al. 2010). SAD and other anxiety disorders may be linked to dysregulation of the fear and stress response system in the brain (Bagnell 2011). Mood and/or anxiety disorders are commonly found in the parents and other relatives of children with SAD. A possible subtype of SAD has been identified in children who may be at particularly high risk for developing panic disorder as adults (Roberson-Nay et al. 2010). *Behavioral inhibition* as described by Kagan (1998) refers to a genetically based temperament trait characterized by response to a new or unfamiliar situation with avoidance, distress, caution, and/or reticence. Both family studies of subjects with anxiety disorders and prospective studies of toddlers have found that behavioral inhibition is correlated with increased risk of developing anxiety disorders. Specific parenting styles characterized by rejection, control, and intrusiveness are also likely related to increased anxiety in youth (Rapee 1997; Wood 2006).

Course and Prognosis

SAD typically begins around the age of school entry and is usually recognized in early or middle childhood. The disorder is often recurrent, with acute exacerbations at the beginning of the school year, following holiday breaks from school, or when starting a new school. Early predictors of SAD include parental depression, parental panic disorder, and strong stranger anxiety during infancy (Lavalley et al. 2011). Precipitants include actual separations, parental divorce, deaths, family moves, family changes, and family crises. Symptoms may worsen during or after medical illnesses or procedures. Comorbidity with other anxiety disorders and non-anxiety psychiatric disorders is common, although rates vary among studies.

Most children who suffer from SAD do not receive mental health treatment. A substantial proportion of children with SAD eventually recover, with or without treatment. Others experience repeated remissions and relapses and may develop a chronic course with impairment extending into adulthood. Adults with SAD typi-

cally have a history of childhood SAD (Silove et al. 2010). In addition to suggested links between childhood SAD and adult agoraphobia or panic disorder, SAD may be considered a risk factor for subsequent depression and other anxiety disorders (Lewinsohn et al. 2008).

Follow-up studies of childhood school absenteeism have reported a high prevalence of adult maladaptation and psychiatric symptoms. Although many of these children return to school eventually, poor overall emotional and social functioning is common. School absentees often have academic underachievement and social avoidance and can be at risk for excessive absenteeism from work or chronic unemployment in adulthood.

Evaluation and Differential Diagnosis

A parent may need to be present with the child for the entire initial interview, if separation is too difficult. Evaluation should include probing for symptoms of anxiety or mood disorders in both the child and the parents, assessing for syndromes that may mimic anxiety. Rating scales such as the Multidimensional Anxiety Scale for Children–2 (MASC-2), by John March (available from Multi-Health Systems), or the Screen for Child Anxiety Related Emotional Disorders (SCARED; Birmaher et al. 1997) are useful in quantifying symptoms. Physical symptoms such as nausea, vomiting, abdominal pain, or headaches require judicious medical evaluation. In SAD, somatic symptoms are typically worse on evenings and mornings before school and absent on weekends and holidays, except as separations approach. A careful focus is needed on identifying any factors contributing to school refusal, using multiple informants (child, parent, teacher, and other school staff).

Separation anxiety is a *normal developmental phenomenon* in children ages 6–30 months, with intensification between 13 and 18 months of age (Kearney et al. 2003). In children younger than 6 years, normal separation anxiety can reappear in stressful situations. In an older child, this may represent an *adjustment disorder with anxiety*. Other anxiety disorders in the differential diagnosis include *gener-*

alized anxiety disorder (GAD), panic disorder, and social anxiety disorder. A *depressive disorder* may also cause clinginess and refusal to separate from the parent. Less commonly, children with *schizophrenia* or an *autism spectrum disorder* may present with symptoms of separation anxiety.

When evaluating the patient with chronic school absenteeism, the clinician should investigate possible causes other than separation anxiety disorder (see Table 7–2). The most common cause of school absenteeism in adolescents is truancy, a feature of conduct disorder. Youngsters with truancy typically show delinquent behavior. They may leave home in the morning and spend the day with friends, while their parents are unaware of or not concerned about their activities, in contrast to children with SAD, who remain at home with their parents' knowledge.

Treatment

SAD is treated using a multimodal approach, including psychoeducation (see Appendix, "Resources for Parents"), collaboration with the primary care provider, school consultation, cognitive-behavioral therapy (CBT), family therapy, and selective serotonin reuptake inhibitors (SSRIs).

School absenteeism requires prompt intervention because, regardless of the cause, the longer the child is out of school, the higher the likelihood of treatment resistance, chronicity, and school failure. The goals are age-appropriate separation of parent and child and a rapid return to school. Cooperation of the family and the school is essential.

For psychotherapy of SAD in youth, CBT has strongest empirical support (Kearney and Albano 2000; Velting et al. 2004). Key elements are psychoeducation of parents and youth, management of somatic symptoms, relaxation training, cognitive restructuring, behavioral exposures, contracting and contingency management, social skills training, and relapse prevention. Specific techniques include using limit setting, eliminating secondary gain for staying home, escorting the child to school, incorporating gradual exposure

to increasing lengths of time at school, and rewarding the child for increasing successful attendance. Parent-child interaction therapy (PCIT) is another effective treatment for younger children (Pincus et al. 2005).

Multimodal treatment planning for youth with SAD must be done sensitively, with understanding of the child's symptoms and concerns. Home schooling is contraindicated, but proactive problem-solving around any actual school issues, such as bullying, is essential. If the child also has panic disorder or a major depressive episode, aggressive return to school prior to addressing these comorbid issues can exacerbate symptoms. A thoughtful treatment plan for school reentry includes planning calm and predictable morning routines, encouraging children and parents to use new cognitive-behavioral skills, addressing positive and/or negative reinforcement of school refusal behavior, and ensuring safety in the environment (such as a school social worker providing support during the school day). Classroom-based accommodations and modifications may also be appropriate under an Individualized Education Plan or Response to Intervention planning.

When moderate to severe anxiety symptoms persist despite appropriate psychosocial and family-based therapeutic interventions, an antidepressant may be added to the treatment of separation anxiety or comorbid mood disorder. SSRIs (see Chapter 17, "Psychopharmacology") have replaced imipramine and other tricyclic antidepressants as a result of their more favorable side-effect profile and improved tolerability. The age of the child should always be taken into consideration. Older children are more commonly treated with medication than younger children. Benzodiazepines have been used rarely in youth as adjuncts to psychological interventions, although behavioral disinhibition, sedation, and dependence/withdrawal are serious potential side effects. These agents should be used sparingly, temporarily, and in combination with an SSRI, if at all.

Given the enhanced outcomes of combination treatment (sertraline plus CBT) compared with either treatment alone in a large randomized study (Walkup et al. 2008), an SSRI plus CBT is recom-

mended for youth with SAD, GAD, and/or social anxiety disorder. However, depending on availability, cost, time factors, and family preference, an SSRI alone, CBT alone, or the combination may be selected in an individual case.

SAD-related sleep disturbance or resistance to sleeping alone is generally treated behaviorally. Techniques include developing a predictable and calming bedtime routine, monitoring bedtime behavior, teaching the child relaxation and other coping skills, and supporting parents while coaching them to positively reinforce the child for sleeping in his or her own bed (McMenamy and Katz 1989).

If a parent's own anxiety or mood disorder is making it harder to separate from the child, the parent should also receive direct psychiatric treatment (possibly including appropriate medication) in addition to guidance in child management and parenting in the context of individual or family therapy.

Common issues in any modality of treatment of SAD are planned and unplanned absences (e.g., vacations by the patient or therapist, school transitions, parental absence, losses of people important to the child) and the termination of treatment. Anticipatory guidance for the parents and the child regarding expected separations is useful. Once treatment is completed, the therapist should be specific about when and how to return to treatment, for example at times of increased vulnerability or if symptoms return.

Referral to a partial hospitalization program (PHP) (day treatment) (see Chapter 18, "Psychosocial Treatments") may be warranted as a transition to school reentry, particularly if school refusal persists despite adequate outpatient treatment. Severe SAD that results in total avoidance of school and does not respond to environmental, psychotherapeutic, and pharmacological interventions may require psychiatric hospitalization. However, prompt referral to a PHP can often avoid the need for inpatient hospitalization. When available, intensive outpatient programs (IOP) that offer treatment several days a week (typically after school) can also be useful as a "step up" from traditional outpatient care if more intensive treatment is needed, or as a "step down" to ease the transition to school reentry following hospitalization or PHP.

In the treatment of adolescents with truancy, firm limit setting with supervision and contingency contracting is necessary. When school absenteeism includes family permission, socializing with peers, risk of physical danger, or substance abuse, involvement of juvenile authorities may be needed.

■ SELECTIVE MUTISM

Clinical Description

In DSM-5 (American Psychiatric Association 2013), selective mutism (SM) was placed in the anxiety disorders section. Children with SM do not speak in one or several important settings despite having the ability to comprehend spoken language and to speak in other situations. Symptoms persist for at least 1 month (outside of the first month of school when transient mutism can occur) and are severe enough to affect educational and interpersonal functioning. These children have an adequate knowledge of the language yet may experience specific developmental communication disorders. 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 these children are separated from a familiar or comfortable setting, they might use gestures, nods, monosyllabic responses, written notes, or whispers but avoid full vocalization. Many of these children are shy, anxious, withdrawn, socially inhibited, fearful of new experiences, and resistant to separation from parents. Some have oppositional behavior, as well.

Epidemiology

SM is estimated to occur in a very small percentage of school-age children and typically begins between 3 and 8 years of age. Point prevalence ranges from 0.03% to 1%, depending on the age of the population studied, setting of study, and the length of their exposure to school. Prevalence is higher in younger children than in adoles-

cents. There is no difference in the prevalence due to sex, race, or ethnicity, but a higher prevalence rate of 2% was reported in immigrant families (Elizur and Perednik 2003).

Etiology

Genetic, temperamental, environmental, and developmental factors all contribute to the development of SM. Family aggregation of SM and related symptoms (social anxiety, shyness, or speech and language disorders) suggests that genetic factors may contribute to the development of SM (Keeton and Crosby Budinger 2012). There is an association between a temperament marked by behavioral inhibition and SM (Muris et al. 2016). SM is seen at higher rates in bilingual children of immigrant families. It can be challenging to distinguish SM from a typical “silent period” of newly immigrated children, but SM should be suspected if the mutism is prolonged despite adequate exposure to the host language, if SM exists in both languages, or if there is associated anxiety or inhibited behavior (Toppelberg et al. 2005).

There may be a link between SM in children and social anxiety disorder in adults. These adults recall feeling intensely anxious as children when they were asked to speak, with accompanying symptoms that approximate panic. SM may have been an early observable form of developing social anxiety disorder. Parents of selectively mute children tend to have a variety of anxiety disorders, including panic, separation anxiety, and social anxiety.

Selectively mute children have higher rates of many developmental conditions, including elimination disorders, motor delays, and lower intelligence quotient (Kristensen 2000). Approximately 25% of children with SM also had delayed onset of speech, and 50% have a speech disorder or speech immaturities. These associations suggest that there is a neurodevelopmental etiology or that developmental disorders may worsen communication impairments. Global mutism can result from cerebellar lesions and is known to accompany cerebellar hemorrhages, subarachnoid hemorrhages, vertebral artery injuries, basilar artery occlusion, and head trauma.

Course and Prognosis

SM is usually discovered when the child attends kindergarten or first grade and is expected to speak. Excessive shyness may be identified retrospectively. The diagnosis is typically made between 3 and 8 years of age. Symptoms may last for weeks, months, or years and usually resolve by age 10. When SM persists beyond age 12, patients are less likely to completely recover. Complications of SM include academic underachievement and impaired peer relationships. The child's persistent silence may lead to unhelpful special class and school placements. Many of these children have comorbid psychiatric problems, including social anxiety disorder, obsessive-compulsive disorder, or school avoidance. In clinically referred samples, some patients have comorbid oppositional defiant disorder. Social anxiety disorder may emerge in adulthood.

Evaluation and Differential Diagnosis

Standard psychiatric evaluation is indicated, including family history of language or anxiety disorders. Speech and language evaluation is warranted. The clinician should review the child's medical history for evidence of neurological injury or delay or hearing deficit, with neurological evaluation or audiological testing if indicated. Although the child may not speak directly to the clinician, observation of the quality of interaction and ability to communicate nonverbally can yield valuable information. The clinician should evaluate the possible presence of physical or sexual abuse, depression, and anxiety disorders.

The differential diagnosis of failure to speak includes *hearing impairment, intellectual disability, communication disorder, aphasia, autism spectrum disorder, schizophrenia, and conversion disorder*. Global impairment of speech is characteristic of all but the latter three disorders.

Treatment

First-line treatment is behavioral, combining office-based desensitization with active involvement of parents at home and teachers at

school (Cohan et al. 2006). Speech and language therapy is often needed, as well. Therapy is based on the assumption that the child will speak again. Any form of communication is encouraged through behavioral plans that shape behavior by reinforcing attempts to speak. Short-term therapy may be effective, although in more resistant cases longer-term treatment may be required. Family therapy may help to identify and change dysfunctional patterns that maintain symptoms. Although teachers and parents often make accommodations to the child's muteness, 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 should be explicit with the child about these expectations: to talk at school and in therapy.

In cases resistant to psychosocial approaches and with more pronounced anxiety symptoms, there is some evidence to support pharmacologic treatment with fluoxetine or sertraline.

■ SOCIAL ANXIETY DISORDER (SOCIAL PHOBIA) AND SPECIFIC PHOBIAS

Clinical Description

A *specific phobia* is defined as marked fear or anxiety about a specific object or situation. In DSM-5 (American Psychiatric Association 2013), there is no longer a distinction between pediatric and adult criteria for specific phobia and social anxiety disorder. At all ages, the anxiety must be out of proportion to the actual danger or threat in the situation, after cultural contextual factors are taken into account. A 6-month duration is required. Transient developmentally appropriate fears (see Table 7-1) do not usually require treatment. Phobias are distinguished from normal fears by their severity, irrationality, persistence, and functional impairment, usually secondary to avoidance of the feared object or situation. Persons with social anxiety disorder suffer from marked fear or anxiety about one or more social situations in which they are exposed to possible scrutiny and negative evaluation by others. DSM-5 examples include interacting socially, being observed (e.g., eating), and performing in front of others (e.g., reading aloud in class, giving a speech).

Epidemiology

Many children with phobias are never seen in a clinical setting because parents and teachers rarely refer children with anxiety or excessive shyness for treatment. The prevalence of specific phobias is 5% in children and 16% in 13- to 17-year-olds (American Psychiatric Association 2013). Most phobias are more common among girls and younger children, though gender rates are equal for blood-injection-injury phobia. Social anxiety disorder is estimated to occur in 2%–5% of youth (March et al. 2007), though an epidemiological study of adolescents and young adults found the prevalence to be about 9% (Burststein et al. 2011). Lifetime prevalence in the general population may be as high as 16% (Lipsitz and Schneier 2000). While nonreferred youth with specific phobia generally have less comorbidity than do those with other anxiety disorders, the children who do present for treatment tend to be more symptomatic with regard to both anxiety and comorbidities.

Etiology

Contributions to the development of childhood anxiety include biological influences (e.g., heredity, behavioral inhibition/temperament, autonomic reactivity, anxiety sensitivity) and environmental influences (e.g., insecure attachment style, overcontrolling/critical/anxious parenting style, peer and social problems, negative/stressful life events). Interactions among these factors are complex.

Course and Prognosis

Specific phobias may begin at any time during development. Phobic symptoms may follow association of a stimulus with an unexpected panic attack or a traumatic event. Most simple phobias remit spontaneously, but some persist. A recent longitudinal study showed that specific phobia may predict the development of many other psychiatric disorders, including other anxiety disorders, mood disorders, eating disorders, and pain disorders (Lieb et al. 2016). Youth with natural environment-type phobias (e.g., storms) fare more poorly

and are more difficult to treat than youth with animal-type phobias (Ollendick et al. 2010). Social anxiety disorder, which tends to start in adolescence, can result in impaired social and academic/occupational functioning due to school avoidance, social withdrawal, substance abuse, and difficulty with dating and intimacy. In one large sample of youth, stressful life events were a significant factor in the development of social anxiety (Aune and Stiles 2009). The disorder tends to persist into adulthood and is associated with professional underachievement, depression, generalized anxiety symptoms, constrained social functioning, and significant functional impairment.

Evaluation and Differential Diagnosis

Parents are often unaware of phobic symptoms or social anxiety in their children, so the clinical interview with the child is especially important. However, younger children may not be able to describe avoidant behaviors, thus parent input regarding the child's behaviors may be necessary. Younger children may express their phobic anxiety as crying, irritability, argumentativeness, tantrums, freezing, or clinging, rather than verbalizing it or explicitly avoiding the feared object or situation. Older youth may quietly avoid the feared stimulus or present with general irritability. A careful patient history aims to elucidate the feared stimulus, circumstances surrounding the development of the phobia, behavior in response to the phobic object or situation, anticipatory or avoidant behaviors, and any secondary gain. Observations of behavior may also be useful. A self-report questionnaire or clinician-administered measure designed for the specific presenting anxiety symptoms can be helpful to measure symptom severity or track treatment response, especially when symptoms are difficult to elicit by interview. Family assessment is also critical.

Differential diagnosis includes *panic disorder*, *agoraphobia*, *social anxiety disorder*, *generalized anxiety disorder*, *posttraumatic stress disorder*, *obsessive-compulsive disorder*, *learning disorders*, *mood and psychotic disorders*, *autism spectrum disorder*, and *eating disorders* (if food or eating is avoided).

Treatment

Clinicians often integrate several approaches to treat phobic disorders. Cognitive-behavioral treatments are the best studied and most efficient interventions for children with phobias. The positive effects of cognitive-behavioral therapy (CBT) appear to generalize, as seen in a study in which treatment of the targeted specific phobia led to improvement of comorbid specific phobias and other anxiety disorders (Ollendick et al. 2010). Effective behavioral treatment requires that the child have skills and the opportunities to manage the problem situation in alternative ways. The therapist evaluates the child's skill set, teaches new skills when necessary, and works with the family (and school if appropriate) to eliminate inadvertent reinforcement of phobic behavior.

For specific phobias, CBT approaches developed for adults, such as systematic desensitization and exposure and response prevention (ERP) techniques, can be used in children, with strategies modified for developmental and cognitive levels. Systematic desensitization techniques (depending on specific phobia type) can include real life ("in vivo") exposure, narrative stories ("emotive imagery"), modeling of coping behavior, and contingency management using shaping, positive reinforcement, and extinction techniques (King et al. 2005).

For treatment of social anxiety disorder, CBT programs incorporating psychoeducation, exposure techniques (practicing feared situations with gradual exposures using fear hierarchies), and social skills training are used with youth and caregivers. Published resources are available to provide instruction and worksheets for role plays of naturalistic exposures, social skill development such as meeting new people, and nonverbal communication skills for youth with social anxiety (Chorpita 2007). Supplemental CBT strategies to facilitate exposures include cognitive restructuring, active ignoring, time-out procedures, and use of positive reinforcement (reward contingency plans). Cognitive restructuring techniques aim to reduce cognitive, emotional, and behavioral symptoms by specifically addressing maladaptive, distorted, self-defeating, or unrealistic thought

patterns. By challenging these negative cognitions (thought patterns) associated with anxiety-producing situations (e.g., school exams or evaluative social settings), youth learn to generate more positive and realistic thoughts, thus improving related feelings and reinforcing mastery, competence, assertiveness, and healthy problem-solving abilities.

Pharmacological studies often include subjects with mixed anxiety disorders. Randomized placebo-controlled trials (RCTs) support the use of the SSRIs fluoxetine, fluvoxamine, sertraline, or paroxetine (very rarely used in children) in the treatment of social anxiety disorder (generally with comorbidities of selective mutism, SAD, and/or GAD). Venlafaxine ER has also shown superior efficacy over placebo in the treatment of social anxiety disorder (March et al. 2007). Medication generally is reserved for cases in which severe symptoms interfere with psychotherapy or in which there is only a partial response to psychotherapy alone. A study comparing fluoxetine alone, psychotherapy (specifically Social Effectiveness Therapy for Children, or SET-C) alone, and placebo in the treatment of social anxiety disorder found that SET-C yielded superior results in reducing social distress, reducing avoidance, improving general functioning, improving social skills, and enhancing social competence (Beidel et al. 2007), underscoring the value of psychotherapy in treating social anxiety disorder. Benzodiazepines have potentially problematic adverse effects, are contraindicated in youth with a history of substance abuse, and have not shown efficacy in controlled trials; they are therefore not recommended other than possibly as an adjunctive short-term treatment with SSRIs in severe cases.

The U.S. Food and Drug Administration (FDA) has issued a “black box warning” for the use of any antidepressant medication, including SSRIs, in the pediatric population because of a small but statistically significant increased risk of suicidal thoughts (compared with subjects on placebo). Pharmacological studies of pediatric anxiety disorder did not find this increased risk (as opposed to pediatric depression, where this effect was seen), but careful monitoring is nonetheless essential when SSRIs are being used, with par-

ticular attention paid to mood, agitation, and suicidal thoughts or behaviors (see Chapter 17, “Psychopharmacology”).

■ PANIC DISORDER

Clinical Description

DSM-5 (American Psychiatric Association 2013) unlinks the diagnoses of panic disorder and agoraphobia. Panic disorder and agoraphobia are relatively rare in children and somewhat more common in adolescents. Diagnostic criteria and physical symptom profile are the same as in adults. Family studies and retrospective reports note continuity between pediatric and adult presentations. *Panic disorder* requires recurrent unexpected abrupt surges of intense fear or discomfort that peaks rapidly (panic attack). Four or more of the 13 panic symptoms are required. They include both physiological and psychological features. In youth, cognitive immaturity may preclude some characteristic cognitions during an attack (fear of dying, going crazy, doing something uncontrolled). Panic attacks generally include shortness of breath, palpitations, chest pain, paresthesias, trembling, dizziness, tachycardia, sweating, and hyperventilation. *Agoraphobia* is described as intense fear or anxiety triggered by real or anticipated exposure to at least two different situations (e.g., using public transportation, being in open spaces, being in enclosed spaces, standing in line or in crowds, being outside of the home alone). For both diagnoses, the anxiety must be out of proportion to the actual danger or threat and impairment, and a 6-month duration of symptoms is required.

Epidemiology

Prevalence of panic disorder is 1%–2% in children and 2%–4% in adolescents (Beesdo et al. 2009), with age at onset typically in late adolescence and early adulthood (American Psychiatric Association 2013). However, panic symptoms are much more common, with reported prevalence in adolescents of 13%–16% (Mattis and Ollendick 2002; Reed and Wittchen 1998). Panic disorder is more

common in females than in males. Agoraphobia is even rarer in children, with a typical age at onset of 17 years. Less than 2% of adolescents and adults have a diagnosis of agoraphobia in any given year (American Psychiatric Association 2013).

Etiology

Twin studies suggest genetic contributions. Modeling may also contribute. Offspring of parents with panic disorder are at high risk for anxiety disorders. A review of 24 studies concluded that cigarette smoking tends to both precede the onset of panic disorder and promote panic (Cosci et al. 2010). Individuals with panic disorder have an increased sensitivity to somatic sensations, referred to as interoceptive sensitivity (Domschke et al. 2010).

Course and Prognosis

The evolution of panic symptoms and the natural history of the disorder in children are not clear, but evidence supports chronicity (Biederman et al. 1997). Children with panic disorder and agoraphobia have high rates of other anxiety and mood disorders as well as attention-deficit/hyperactivity disorder (ADHD) (Biederman et al. 1997). Subclinical panic attacks, referred to as fear *spells*, may also predict later development of various psychiatric disorders (Asselmann et al. 2014). Prepubertal onset may signal greater severity (Vitiello et al. 1990). Youth with the somatic symptoms of panic disorder are likely to first seek the help of a pediatrician or an emergency room. Panic attacks can begin at the onset of or during an episode of major depression or SAD. Agoraphobia is typically chronic, though the type of triggering situation may vary (younger children are more likely to fear leaving the home alone).

Evaluation and Differential Diagnosis

Children can report current panic attacks, but parent report is helpful to verify duration and history. Self-monitoring techniques may be useful in children, as in adults. Panic disorder in children is likely

underdiagnosed, with the symptoms often attributed to *separation anxiety disorder*, *hyperventilation syndrome*, or *situational or “normal” anxiety*. Panic attacks can be the anxiety manifestation in other anxiety disorders, so a careful history asking about triggers can help to differentiate panic disorder from other anxiety disorders. For unexplained somatic concerns, a careful (but not excessive) medical evaluation should be done to rule out medical conditions.

Treatment

Education is essential for patients, families, and school staff (if symptoms interfere with school functioning). An RCT found a CBT-based treatment (Panic Control Treatment for Adolescents [PCT-A]) to be feasible and efficacious in a small sample of adolescents followed for up to 6 months posttreatment (Pincus et al. 2010). CBT strategies used with panic disorder are aimed at the reduction of the fear of sensations that trigger the anxiety response. They include relaxation techniques, cognitive strategies, and ERP, which can involve interoceptive exposure (gradual exposure to physical sensations by using exercises such as breath holding, running in place, or spinning to precipitate dizziness, shortness of breath, or sweating). SSRIs should be considered for panic symptoms that are persistent and/or impair functioning despite psychotherapeutic interventions. Although SSRIs have shown efficacy in adult panic disorder, there are no pediatric controlled trials. A pilot study suggests that fluoxetine and sertraline may be useful in treating panic symptoms (Renaud et al. 1999). In more severely affected youth with panic disorder, benzodiazepines may be considered for very cautious use, only short-term, given their many risks.

■ GENERALIZED ANXIETY DISORDER

Clinical Description

Children with GAD have pervasive worries for at least 6 months about a variety of areas (Box 7–2). The DSM-5 criteria require only one accompanying symptom for children, whereas adults must have three.

Box 7–2 DSM-5 Diagnostic Criteria for Generalized Anxiety Disorder

- A. Excessive anxiety and worry (apprehensive expectation), occurring more days than not for at least 6 months, about a number of events or activities (such as work or school performance).
- B. The individual finds it difficult to control the worry.
- C. The anxiety and worry are associated with three (or more) of the following six symptoms (with at least some symptoms having been present for more days than not for the past 6 months):

Note: Only one item is required in children.

- 1. Restlessness or feeling keyed up or on edge.
 - 2. Being easily fatigued.
 - 3. Difficulty concentrating or mind going blank.
 - 4. Irritability.
 - 5. Muscle tension.
 - 6. Sleep disturbance (difficulty falling or staying asleep, or restless, unsatisfying sleep).
- D. The anxiety, worry, or physical symptoms cause clinically significant distress or impairment in social, occupational, or other important areas of functioning.
 - E. The disturbance is not attributable to the physiological effects of a substance (e.g., a drug of abuse, a medication) or another medical condition (e.g., hyperthyroidism).
 - F. The disturbance is not better explained by another mental disorder (e.g., anxiety or worry about having panic attacks in panic disorder, negative evaluation in social anxiety disorder [social phobia], contamination or other obsessions in obsessive-compulsive disorder, separation from attachment figures in separation anxiety disorder, reminders of traumatic events in posttraumatic stress disorder, gaining weight in anorexia nervosa, physical complaints in somatic symptom disorder, perceived appearance flaws in body dysmorphic disorder, having a serious illness in illness anx-

xiety disorder, or the content of delusional beliefs in schizophrenia or delusional disorder).

The key feature of GAD is uncontrollable worry. It is important to clarify in an individual child that the excessive anxiety is actually *out of proportion* to the situation rather than an expectable reaction to a significant life stressor. Habit disturbances such as nail biting, hair pulling, or thumb sucking are common in youth with GAD. Somatic symptoms such as headache, upset stomach, or fatigue may result in parental requests for extensive medical evaluations.

Epidemiology

Transient symptoms of anxiety are common in typically developing children. Although typical age at onset for GAD is adulthood, estimates of the prevalence of adolescent GAD are 1% in younger adolescents to 3% in older adolescents (Merikangas et al. 2010). GAD is diagnosed twice as often in females as in males. Comorbidity among the anxiety and depressive disorders is common (Wehry et al. 2015).

Etiology

Family genetic studies reveal important contributing factors (e.g., behaviorally inhibited temperament or information processing biases), and research on environmental factors has implicated adverse life events, exposure to negative information, and modeling as important. Parent and child contributions to symptoms are presumed to be bidirectional, including an association between child anxiety and parental control (van der Bruggen et al. 2008). Children at risk of developing more severe anxiety symptoms are characterized by psychosocial adversity, inattention, low prosociality in school, and high levels of maternal discipline (Duchesne et al. 2010).

Course and Prognosis

GAD is a chronic disorder with low rates of full remission (American Psychiatric Association 2013). Although many anxious chil-

dren improve with or without treatment, most experience a chronic course with waxing and waning symptoms. A childhood diagnosis of anxiety disorder is associated with increased heritability, symptom severity, impairment, and development of a range of disorders across the life span (Egger and Angold 2006).

Evaluation and Differential Diagnosis

In general, children are more accurate than their parents are in reporting their anxiety symptoms. Parents may either underreport because they are not aware of the child's symptoms or report an exaggerated degree of child anxiety, influenced by the parent's own heightened anxiety. Parents and children may actually be reporting different symptoms as "anxiety." Self-report instruments such as the SCARED (Birmaher et al. 1997), with both child and parent scales, and the Multidimensional Anxiety Scale for Children (MASC) (March et al. 1997), and its subsequent version (MASC-2), can aid in assessment of anxiety symptoms.

Other anxiety disorders, such as *SAD*, *panic disorder*, *specific phobia*, *social anxiety disorder*, and *adjustment disorder with anxiety*, and the *depressive disorders* should be considered in the differential diagnosis of GAD. Unlike children with ADHD, who are also restless and fidgety, children with GAD worry excessively (although the two disorders can be comorbid). Because children with GAD often have somatic symptoms, a wide variety of physical illnesses is in the differential diagnosis. Determining the extent of an appropriate medical workup for symptoms such as recurrent headache or abdominal pain can be difficult, because children with documented physical etiologies also report anxiety and depression. Medical disorders that can mimic an anxiety disorder include *substance withdrawal or intoxication*, *hyperthyroidism*, *pheochromocytoma*, *asthma*, and *medication side effects*.

Treatment

CBT is the first-line psychotherapy approach for the treatment of anxiety disorders. The therapeutic alliance, parental ability to partic-

ipate in treatment, and likelihood of completing therapeutic homework are important to consider when assessing for CBT suitability. CBT techniques used in the treatment of youth with GAD include relaxation exercises, cognitive restructuring, ERP, and problem-solving techniques in order to target the physical signs of anxiety and excessive worry. CBT can be provided in individual, family, group, or school-based settings.

SSRIs, particularly fluvoxamine, sertraline, and fluoxetine, have empirical support for treatment of GAD, although there is no evidence that a particular SSRI is more effective than another. The combination of CBT with sertraline offers additional benefit compared with either treatment alone, based on a large RCT (Walkup et al. 2008). The use of medication requires close monitoring for possible side effects, and long-term use has not been studied. While the association of SSRI use and suicidal thinking is seen more in the treatment of depressive disorders, such monitoring is necessary when using an SSRI in the treatment of anxiety disorders. Clinical guidelines suggest maintenance medication treatment for approximately 1 year from symptom remission, with tapering and discontinuation during a low-stress time of year and attention to resuming medication if symptoms recur. Venlafaxine, a serotonin-norepinephrine reuptake inhibitor, also has empirical support for use in GAD (Rynn et al. 2007). Most recently, duloxetine, another serotonin-norepinephrine reuptake inhibitor, demonstrated superior efficacy to placebo in pediatric GAD (Strawn et al. 2015). Duloxetine received an FDA indication for pediatric GAD, making it the only FDA-labeled medication for pediatric GAD. Although tricyclic antidepressants, buspirone, and benzodiazepines have been suggested as alternative drugs, either as monotherapy or in combination with SSRIs, their safety and efficacy have not been established, because of limited or inconclusive research.

Referral of parents for their own treatment is often important, particularly if a parent also has an anxiety, mood, or other psychiatric disorder. The therapist should work with the parents to anticipate future developmental events that may lead to parental overprotectiveness or unrealistically high expectations for the child's performance.

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Asselmann E, Wittchen HU, Lieb R, et al: Associations of fearful spells and panic attacks with incident anxiety, depressive, and substance use disorders: a 10-year prospective-longitudinal community study of adolescents and young adults. *J Psychiatr Res* 55:8–14, 2014 24774646
- Aune T, Stiles TC: The effects of depression and stressful life events on the development and maintenance of syndromal social anxiety: sex and age differences. *J Clin Child Adolesc Psychol* 38(4):501–512, 2009 20183637
- Bagnell AL: Anxiety and separation disorders. *Pediatr Rev* 32(10):440–445, quiz 446, 2011 21965711
- Beesdo K, Knappe S, Pine DS: Anxiety and anxiety disorders in children and adolescents: developmental issues and implications for DSM-V. *Psychiatr Clin North Am* 32(3):483–524, 2009 19716988
- Beidel DC, Turner SM, Sallee FR, et al: SET-C versus fluoxetine in the treatment of childhood social phobia. *J Am Acad Child Adolesc Psychiatry* 46(12):1622–1632, 2007 18030084
- Biederman J, Faraone SV, Marris A, et al: Panic disorder and agoraphobia in consecutively referred children and adolescents. *J Am Acad Child Adolesc Psychiatry* 36(2):214–223, 1997 9031574
- Birmaher B, Khetarpal S, Brent D, et al: The Screen for Child Anxiety Related Emotional Disorders (SCARED): scale construction and psychometric characteristics. *J Am Acad Child Adolesc Psychiatry* 36(4):545–553, 1997 9100430
- Breton JJ, Bergeron L, Valla JP, et al: Quebec child mental health survey: prevalence of DSM-III-R mental health disorders. *J Child Psychol Psychiatry* 40(3):375–384, 1999 10190339
- Burstein M, He JP, Kattan G, et al: Social phobia and subtypes in the national comorbidity survey-adolescent supplement: prevalence, correlates, and comorbidity. *J Am Acad Child Adolesc Psychiatry* 50(9):870–880, 2011 21871369
- Chorpita BF: Modular Cognitive-Behavioral Therapy for Childhood Anxiety Disorders. New York, Guilford, 2007
- Cohan SL, Chavira DA, Stein MB: Practitioner review: Psychosocial interventions for children with selective mutism: a critical evaluation of the literature from 1990-2005. *J Child Psychol Psychiatry* 47(11):1085–1097, 2006 17076747

- Cosci F, Knuts IJ, Abrams K, et al: Cigarette smoking and panic: a critical review of the literature. *J Clin Psychiatry* 71(5):606–615, 2010 19961810
- Dallaire DH, Weinraub M: Predicting children's separation anxiety at age 6: the contributions of infant-mother attachment security, maternal sensitivity, and maternal separation anxiety. *Attach Hum Dev* 7(4):393–408, 2005 16332583
- Domschke K, Stevens S, Pfleiderer B, Gerlach AL: Interoceptive sensitivity in anxiety and anxiety disorders: an overview and integration of neurobiological findings. *Clin Psychol Rev* 30(1):1–11, 2010 19751958
- Duchesne S, Larose S, Vitaro F, Tremblay RE: Trajectories of anxiety in a population sample of children: clarifying the role of children's behavioral characteristics and maternal parenting. *Dev Psychopathol* 22(2):361–373, 2010 20423547
- Egger HL, Angold A: Common emotional and behavioral disorders in pre-school children: presentation, nosology, and epidemiology. *J Child Psychol Psychiatry* 47(3-4):313–337, 2006 16492262
- Elizur Y, Perednik R: Prevalence and description of selective mutism in immigrant and native families: a controlled study. *J Am Acad Child Adolesc Psychiatry* 42(12):1451–1459, 2003 14627880
- Kagan J: *Galen's Prophecy: Temperament in Human Nature*. Boulder, CO, Westview Press, 1998
- Kashani JH, Orvaschel H: A community study of anxiety in children and adolescents. *Am J Psychiatry* 147(3):313–318, 1990 2309948
- Kearney CA, Albano AM: *When Children Refuse School: A Cognitive-Behavioral Therapy Approach (Therapist Guide)*. San Antonio, TX, Psychological Corporation, 2000
- Kearney CA, Sims KE, Pursell CR, Tillotson CA: Separation anxiety disorder in young children: a longitudinal and family analysis. *J Clin Child Adolesc Psychol* 32(4):593–598, 2003 14710468
- Keeton CP, Crosby Budinger M: Social phobia and selective mutism. *Child Adolesc Psychiatr Clin N Am* 21(3):621–641, 2012 22800998
- King NJ, Muris P, Ollendick TH: Childhood fears and phobias: assessment and treatment. *Child Adolesc Ment Health* 10:50–56, 2005
- Kristensen H: Selective mutism and comorbidity with developmental disorder/delay, anxiety disorder, and elimination disorder. *J Am Acad Child Adolesc Psychiatry* 39(2):249–256, 2000 10673837
- Lamb DJ, Middeldorp CM, van Beijsterveldt CE, et al: Heritability of anxious-depressive and withdrawn behavior: age-related changes during adolescence. *J Am Acad Child Adolesc Psychiatry* 49(3):248–255, 2010 20410714

- Last CG, Francis G, Hersen M, et al: Separation anxiety and school phobia: a comparison using DSM-III criteria. *Am J Psychiatry* 144(5):653–657, 1987 3578577
- Lavallee K, Herren C, Blatter-Meunier J, et al: Early predictors of separation anxiety disorder: early stranger anxiety, parental pathology and prenatal factors. *Psychopathology* 44(6):354–361, 2011 21847002
- Lewinsohn PM, Holm-Denoma JM, Small JW, et al: Separation anxiety disorder in childhood as a risk factor for future mental illness. *J Am Acad Child Adolesc Psychiatry* 47(5):548–555, 2008 18356763
- Lieb R, Miché M, Gloster AT, et al: Impact of specific phobia on the risk of onset of mental disorders: a 10-year prospective-longitudinal community study of adolescents and young adults. *Depress Anxiety* 33(7):667–675, 2016 26990012
- Lipsitz JD, Schneier FR: Social phobia. *Epidemiology and cost of illness. Pharmacoeconomics* 18(1):23–32, 2000 11010601
- March JS, Parker JD, Sullivan K, et al: The Multidimensional Anxiety Scale for Children (MASC): factor structure, reliability, and validity. *J Am Acad Child Adolesc Psychiatry* 36(4):554–565, 1997 9100431
- March JS, Entusah AR, Rynn M, et al: A randomized controlled trial of venlafaxine ER versus placebo in pediatric social anxiety disorder. *Biol Psychiatry* 62(10):1149–1154, 2007 17553467
- Mattis SG, Ollendick TH: Nonclinical panic attacks in late adolescence prevalence and associated psychopathology. *J Anxiety Disord* 16(4):351–367, 2002
- Merikangas KR, He JP, Burstein M, et al: Lifetime prevalence of mental disorders in U.S. adolescents: results from the National Comorbidity Survey Replication—Adolescent Supplement (NCS-A). *J Am Acad Child Adolesc Psychiatry* 49(10):980–989, 2010 20855043
- McMenamy C, Katz RC: Brief parent-assisted treatment for children's nighttime fears. *J Dev Behav Pediatr* 10(3):145–148, 1989 2663926
- Muris P, Hendriks E, Bot S: Children of few words: relations among selective mutism, behavioral inhibition, and (social) anxiety symptoms in 3- to 6-year olds. *Child Psychiatry Hum Dev* 47(1):94–101, 2016 25842046
- Ollendick TH, Ost LG, Reuterskiöld L, Costa N: Comorbidity in youth with specific phobias: impact of comorbidity on treatment outcome and the impact of treatment on comorbid disorders. *Behav Res Ther* 48(9):827–831, 2010 20573338
- Pincus D, Eyberg S, Choate M: Adapting parent-child interaction therapy for young children with separation anxiety disorder. *Educ Treat Child* 28(2):163–181, 2005

- Pincus DB, May JE, Whitton SW, et al: Cognitive-behavioral treatment of panic disorder in adolescence. *J Clin Child Adolesc Psychol* 39(5):638–649, 2010 20706917
- Rapee RM: Potential role of childrearing practices in the development of anxiety and depression. *Clin Psychol Rev* 17(1):47–67, 1997 9125367
- Reed V, Wittchen HU: DSM-IV panic attacks and panic disorder in a community sample of adolescents and young adults: how specific are panic attacks? *J Psychiatr Res* 32(6):335–345, 1998 9844949
- Renaud J, Birmaher B, Wassick SC, Bridge J: Use of selective serotonin reuptake inhibitors for the treatment of childhood panic disorder: a pilot study. *J Child Adolesc Psychopharmacol* 9(2):73–83, 1999 10461817
- Roberson-Nay R, Klein DF, Klein RG, et al: Carbon dioxide hypersensitivity in separation-anxious offspring of parents with panic disorder. *Biol Psychiatry* 67(12):1171–1177, 2010 20172505
- Rynn MA, Riddle MA, Yeung PP, Kunz NR: Efficacy and safety of extended-release venlafaxine in the treatment of generalized anxiety disorder in children and adolescents: two placebo-controlled trials. *Am J Psychiatry* 164(2):290–300, 2007 17267793
- Shear K, Jin R, Ruscio AM, et al: Prevalence and correlates of estimated DSM-IV child and adult separation anxiety disorder in the National Comorbidity Survey Replication. *Am J Psychiatry* 163(6):1074–1083, 2006 16741209
- Silove DM, Marnane CL, Wagner R, et al: The prevalence and correlates of adult separation anxiety disorder in an anxiety clinic. *BMC Psychiatry* 10:21–27, 2010 20219138
- Silverman WK, Saavedra LM, Pina AA: Test-retest reliability of anxiety symptoms and diagnoses with the Anxiety Disorders Interview Schedule for DSM-IV: child and parent versions. *J Am Acad Child Adolesc Psychiatry* 40(8):937–944, 2001 11501694
- Strawn JR, Prakash A, Zhang Q, et al: A randomized, placebo-controlled study of duloxetine for the treatment of children and adolescents with generalized anxiety disorder. *J Am Acad Child Adolesc Psychiatry* 54(4):283–293, 2015 25791145
- Toppelberg CO, Tabors P, Coggins A, et al: Differential diagnosis of selective mutism in bilingual children. *J Am Acad Child Adolesc Psychiatry* 44(6):592–595, 2005 15908842
- van der Bruggen CO, Stams GJ, Bögels SM: Research review: the relation between child and parent anxiety and parental control: a meta-analytic review. *J Child Psychol Psychiatry* 49(12):1257–1269, 2008 18355216

- Velting ON, Setzer NJ, Albano AM: Update on and advances in assessment and cognitive-behavioral treatment of anxiety disorders in children and adolescents. *Prof Psychol Res Pr* 35:42–54, 2004
- Vitiello B, Behar D, Wolfson S, McLeer SV: Diagnosis of panic disorder in prepubertal children. *J Am Acad Child Adolesc Psychiatry* 29(5):782–784, 1990 2228933
- Walkup JT, Albano AM, Piacentini J, et al: Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *N Engl J Med* 359(26):2753–2766, 2008 18974308
- Wehry AM, Beesdo-Baum K, Hennelly MM, et al: Assessment and treatment of anxiety disorders in children and adolescents. *Curr Psychiatry Rep* 17(7):52, 2015 25980507
- Wood JJ: Parental intrusiveness and children's separation anxiety in a clinical sample. *Child Psychiatry Hum Dev* 37(1):73–87, 2006 16932853

■ ADDITIONAL READING

- Connolly SD, Suarez LM, Victor AM, et al: Anxiety disorders, in Dulcan's *Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 305–343
- Rynn MA, Yanes-Lukin PK, Braun DA, Goldberg PH: Anxiety disorders, in *Clinical Manual of Child and Adolescent Psychopharmacology*, 3rd Edition. Edited by McVoy M, Findling RL. Arlington, VA, American Psychiatric Association Publishing, 2017, pp 169–219

OBSESSIVE-COMPULSIVE AND RELATED DISORDERS

In DSM-5 (American Psychiatric Association 2013), obsessive-compulsive disorder (OCD) was removed from the anxiety disorders chapter and placed in a newly created section entitled “Obsessive-Compulsive and Related Disorders,” which also includes body dysmorphic disorder, hoarding disorder, trichotillomania (hair-pulling disorder), and excoriation (skin-picking) disorder.

■ OBSESSIVE-COMPULSIVE DISORDER

Clinical Description

The child may have either obsessions (worries) or compulsions (rituals), although most patients have both. Recognition by the affected individual that the obsession or compulsion is excessive or unreasonable is no longer required. In DSM-5 (American Psychiatric Association 2013), there are three revised specifiers for insight: good or fair, poor, and absent insight/delusional beliefs.

Epidemiology

OCD is underrecognized and underdiagnosed and is more common than clinical populations would suggest. Symptoms are poorly understood by and embarrassing to children and therefore often concealed, which delays assessment and diagnosis. The prevalence of OCD in children and adolescents is thought to be 1%–2%, although

rates as high as 4% have been reported. Mild or transient rituals, obsessions, or compulsions are common in the general population. At various stages of development, ritualistic behaviors and compulsions are normal. Examples are rigid bedtime routines; collecting, arranging, and storing of objects; and concerns about dirt and germs in preschool and early-school-age children (Leckman and Bloch 2008). Onset of OCD peaks in preadolescence and again in early adulthood (Geller et al. 1998). Boys tend to have earlier onset than girls; however, by adolescence gender distribution is likely equal (Swedo et al. 1989). Neither gender nor age at onset influences the number, specific type, or severity of OCD symptoms. Childhood OCD generally has a more favorable outcome than adult-onset OCD.

Etiology

OCD is a model neuropsychiatric disorder. The “serotonin hypothesis” for the etiology of the disorder was initially developed following the success of selective serotonin reuptake inhibitors (SSRIs) in the treatment of OCD. Family studies indicate a genetic diathesis, with greater genetic loading in pediatric OCD compared with adult-onset OCD. Neuroimaging suggests abnormalities in connections between the basal ganglia and the cortex. A cortico-striatal-thalamic circuitry mechanism has been implicated, involving the neurotransmitters glutamate, dopamine, and serotonin (Rosenberg and Keshavan 1998). The scarcity of a known family history of OCD in many cases raises speculation about environmental influences. Perinatal events among children with OCD are associated with earlier age at onset; increased symptom severity; and comorbidity with attention-deficit/hyperactivity disorder (ADHD), persistent tic disorder, anxiety disorder, and major depressive disorder (Geller et al. 2008). Both comorbid anxiety and externalizing psychopathology are associated with increased symptom severity and impairment (Langley et al. 2010).

Although the existence of pediatric autoimmune neuropsychiatric disorders associated with streptococcus infections (PANDAS) is controversial, there is some evidence supporting the hypothesis that group A beta-hemolytic streptococcus (GABHS) infection is

linked to disease onset and clinical exacerbations in a subset of youth with OCD and/or tic disorders (Kurlan et al. 2008). On the other hand, a prospective study found that documented streptococcal infections were not associated with exacerbations of tic or obsessive-compulsive symptoms in children whose symptoms met strict criteria for PANDAS, compared with children with Tourette's disorder and/or OCD who did not have a PANDAS history (Leckman et al. 2011). The current consensus is that a subset of children with OCD may have onset or exacerbation linked to GABHS. However, in a blinded randomized controlled trial (RCT) with patients strictly diagnosed with PANDAS and OCD, intravenous immunoglobulin was not superior to intravenous saline (Williams et al. 2016). More recently, there has been a decoupling of the specific pathogen, GABHS, from the dramatic onset of OCD symptoms, now described as pediatric acute-onset neuropsychiatric syndrome (PANS) (Murphy et al. 2015). These cases may have other, not yet identified, causes. Traumatic experiences, such as being a victim of assault or witnessing harm/violence, are occasionally associated with onset of OCD (Lafleur et al. 2011).

Course and Prognosis

OCD in children (as in adults) appears to be a chronic condition, following a waxing and waning course. A follow-up study found that 41% of youth with OCD continued to have OCD 9 years later and that 40% of the participants had an additional psychiatric diagnosis at follow-up (Micali et al. 2010). However, many youth will experience partial or full remission. The most commonly reported obsessions include contamination, sexual or somatic thoughts, and overly moralistic worries, and the most commonly reported compulsions are washing, repeating, checking, and ordering (Geller et al. 1998).

Factors associated with a more severe course include very early age at onset, psychiatric comorbidities, poor initial treatment response, longer illness duration, and having a first-degree relative with OCD. Complications of OCD can include impairment in peer relations, adult intimacy, and employment, as well as physical sequelae

such as dermatitis secondary to washing rituals. OCD does not appear to negatively affect educational achievement (Geller et al. 1998).

OCD in children is usually accompanied by other psychiatric disorders, which may include anxiety and mood disorders; tics or Tourette's disorder; speech and developmental disorders; ADHD; and disruptive, impulse-control, and conduct disorders.

Evaluation and Differential Diagnosis

Children and adolescents are often secretive about obsessions and compulsions. Temper outbursts, academic struggles, or changes in eating may be the presenting concerns until obsessions and compulsions are carefully elicited with specific questions to the child, parents, and teachers. Pathological rituals need to be distinguished from normal developmental childhood routines and careful assessment of impairment is critical. The clinician-rated Children's Yale-Brown Obsessive Compulsive Scale (C-YBOCS; Scahill et al. 1997) is the gold standard assessment tool for use at initial diagnosis and in symptom monitoring during treatment. It provides a standardized inventory of symptoms and aids in assessing severity, subjective distress, impairment, internal resistance, and degree of control.

Alternative diagnoses include *phobic disorder*, *Tourette's disorder* (which may be comorbid), *anorexia* and *bulimia nervosa*, and *schizophrenia*. *Neurological conditions* can precipitate OCD symptoms. The usefulness of throat culture and streptococcal antibody titers in the absence of sore throat is highly controversial. Complex tics may be hard to distinguish from compulsions. Patients with *autism spectrum disorder* (ASD) can exhibit obsessive-compulsive symptoms. *Stimulant medication* can induce over-focused or perseverative behaviors, especially at higher doses.

Treatment

Cognitive-behavioral therapy (CBT) is the first-line treatment for mild to moderate OCD in youth. Medication should be considered

when OCD symptoms are severe, in the presence of comorbid disorder or family dysfunction, when the patient or family resists engaging in CBT, or when a skilled CBT clinician is not available. In the Pediatric OCD Treatment Study (POTS; March et al. 2004), CBT and medication treatment with sertraline were equally effective, and each was better than placebo treatment, although subsequent data analysis examining OCD with or without tics revealed somewhat different outcomes. Combining CBT and medication yielded the greatest treatment response. Specific CBT techniques used in the POTS included psychoeducation, cognitive training (cognitive restructuring), mapping OCD, exposure and response prevention (ERP), and relapse prevention/generalization training. CBT has been extended to group therapy and family-based approaches. Family-based CBT was shown to be superior to family-based relaxation treatment in OCD treatment for younger children ages 5–8 years (Freeman et al. 2014). Acceptance and commitment therapy (ACT) has shown some effect in reducing anxiety. The goal of ACT is for patients to accept their thoughts as just thoughts (instead of reality) while avoiding specific experiences that do not support acceptance (Park and Geller 2014). However, empirical evidence for ACT in pediatric OCD is lacking.

In addition to sertraline, the SSRIs paroxetine, fluoxetine, and fluvoxamine, and the tricyclic antidepressant clomipramine, have been shown to be effective in childhood OCD (Geller et al. 2003), although clomipramine is typically reserved for treatment-resistant cases because of its side-effect profile and interactions with other medications, and paroxetine is very rarely used in children. Because there are no comparative efficacy data, clinical factors determine which SSRI to select (including citalopram and escitalopram, which have not been studied in OCD trials). Clomipramine, fluoxetine, fluvoxamine, and sertraline have U.S. Food and Drug Administration (FDA) labeling for pediatric OCD. If medication augmentation is required, the most common addition (extrapolating from research with adults) is an atypical antipsychotic, particularly if there are concurrent tic disorders, ASD symptoms, or mood dysregulation.

■ TRICHOTILLOMANIA (HAIR-PULLING DISORDER)

Trichotillomania is the pulling out of one's own hair, resulting in hair loss, despite repeated attempts to stop, and causing significant distress or impairment. Trichotillomania often emerges in childhood and is comorbid with anxiety, OCD, depression, or ADHD.

Children who pull out their hair may do so as a result of anxiety or boredom or to relieve a specific sense of tension or may experience gratification or relief with pulling. Others may pull automatically, with little awareness. Many have a combination of these patterns. They may have accompanying rituals such as examining, rolling, biting, or ingesting the hair. Pulling may involve hair from the scalp, eyebrows, eyelashes, axillae, body, or pubic area, and the pattern of hair loss may be either localized or diffuse. Children may cover the hair loss with hats, scarves, wigs, or makeup. Some may pull the hair of others, or of pets, wigs, or dolls.

Consequences of hair pulling may include social embarrassment, occupational impairment, or permanent damage to hair growth or quality. Swallowing of hair may result in trichobezoars with resultant anemia, abdominal pain, and risk for obstruction or perforation.

The differential diagnosis of trichotillomania includes *normative hair pulling, rolling, or twisting; hair pulling for cosmetic reasons; compulsive rituals in OCD; body dysmorphic disorder; and medication-induced stereotypies*.

The most effective treatment for trichotillomania is behavior therapy, including habit reversal therapy (HRT). Pharmacotherapy for trichotillomania in youth has been less successful than in adults. Trials of SSRIs have had mixed results in adults and adolescents, although a more recent meta-analysis suggests a moderate effect across both age groups (McGuire et al. 2014). *N*-Acetyl cysteine has shown promise in adults with trichotillomania, but a randomized controlled trial of this supplement in children showed no benefit over placebo (Bloch et al. 2013).

■ EXCORIATION (SKIN-PICKING) DISORDER

Excoriation disorder is newly added to DSM-5 (American Psychiatric Association 2013). Skin picking can begin at any age, often occurs in childhood or adolescence, and is frequently comorbid with anxiety disorders. It is characterized by repeated picking at the skin, causing lesions, with unsuccessful attempts to stop, leading to distress or impairment.

Children with skin picking will often present with multiple irregular, angular lesions on accessible areas of the body in various stages of healing. They may have sought medical treatment for a “rash” without success. They will often find a skin irregularity and pick at it until it opens and bleeds.

Differential diagnosis includes *body dysmorphic disorder* and *OCD*, which may co-occur. It also commonly co-occurs with *other grooming disorders*, including trichotillomania and nail-biting. It is more common in females. Skin picking is a characteristic of *Prader-Willi syndrome* and is not diagnosed separately in those individuals.

Treatment includes behavior therapy, with best evidence for HRT. SSRIs are often used, especially if there is a comorbid anxiety disorder, but there is limited evidence for their efficacy in skin picking in children or adults (Schumer et al. 2016). *N*-Acetylcysteine has shown promise for this condition in adults and in patients with Prader-Willi syndrome, but there are insufficient data to recommend its use in children with excoriation (skin-picking) disorder (Miller and Angulo 2014).

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Bloch MH, Panza KE, Grant JE, et al: N-Acetylcysteine in the treatment of pediatric trichotillomania: a randomized, double-blind, placebo-controlled add-on trial. *J Am Acad Child Adolesc Psychiatry* 52(3):231–240, 2013 23452680

- Freeman J, Sapyta J, Garcia A, et al: Family-based treatment of early childhood obsessive-compulsive disorder: the Pediatric Obsessive-Compulsive Disorder Treatment Study for Young Children (POTS Jr)—a randomized clinical trial. *JAMA Psychiatry* 71(6):689–698, 2014 24759852
- Geller D, Biederman J, Jones J, et al: Is juvenile obsessive-compulsive disorder a developmental subtype of the disorder? A review of the pediatric literature. *J Am Acad Child Adolesc Psychiatry* 37(4):420–427, 1998 9549963
- Geller DA, Biederman J, Stewart SE, et al: Which SSRI? A meta-analysis of pharmacotherapy trials in pediatric obsessive-compulsive disorder. *Am J Psychiatry* 160(11):1919–1928, 2003 14594734
- Geller DA, Wieland N, Carey K, et al: Perinatal factors affecting expression of obsessive compulsive disorder in children and adolescents. *J Child Adolesc Psychopharmacol* 18(4):373–379, 2008 18759647
- Kurlan R, Johnson D, Kaplan EL; Tourette Syndrome Study Group: Streptococcal infection and exacerbations of childhood tics and obsessive-compulsive symptoms: a prospective blinded cohort study. *Pediatrics* 121(6):1188–1197, 2008 18519489
- Lafleur DL, Petty C, Mancuso E, et al: Traumatic events and obsessive compulsive disorder in children and adolescents: is there a link? *J Anxiety Disord* 25(4):513–519, 2011 21295942
- Langley AK, Lewin AB, Bergman RL, et al: Correlates of comorbid anxiety and externalizing disorders in childhood obsessive compulsive disorder. *Eur Child Adolesc Psychiatry* 19(8):637–645, 2010 20349255
- Leckman JF, Bloch MH: A developmental and evolutionary perspective on obsessive-compulsive disorder: whence and whither compulsive hoarding? *Am J Psychiatry* 165(10):1229–1233, 2008 18829875
- Leckman JF, King RA, Gilbert DL, et al: Streptococcal upper respiratory tract infections and exacerbations of tic and obsessive-compulsive symptoms: a prospective longitudinal study. *J Am Acad Child Adolesc Psychiatry* 50(2):108–118.e3, 2011 21241948
- March JS, Foa EB, Gammon E, et al; Pediatric OCD Treatment Study (POTS) Team: Cognitive-behavior therapy, sertraline, and their combination for children and adolescents with obsessive-compulsive disorder: the Pediatric OCD Treatment Study (POTS) randomized controlled trial. *JAMA* 292(16):1969–1976, 2004 15507582
- McGuire JF, Ung D, Selles RR, et al: Treating trichotillomania: a meta-analysis of treatment effects and moderators for behavior therapy and serotonin reuptake inhibitors. *J Psychiatr Res* 58:76–83, 2014 25108618

- Micali N, Heyman I, Perez M, et al: Long-term outcomes of obsessive-compulsive disorder: follow-up of 142 children and adolescents. *Br J Psychiatry* 197(2):128–134, 2010 20679265
- Miller JL, Angulo M: An open-label pilot study of N-acetylcysteine for skin-picking in Prader-Willi syndrome. *Am J Med Genet A* 164A(2):421–424, 2014 24311388
- Murphy TK, Patel PD, McGuire JF, et al: Characterization of the pediatric acute-onset neuropsychiatric syndrome phenotype. *J Child Adolesc Psychopharmacol* 25(1):14–25, 2015 25314221
- Park JM, Geller DA: Novel approaches in treatment of pediatric anxiety (Epub only). *F1000Prime Rep* 6:30, 2014 24860652
- Rosenberg DR, Keshavan MS: A.E. Bennett Research Award. Toward a neurodevelopmental model of obsessive-compulsive disorder. *Biol Psychiatry* 43(9):623–640, 1998 9582996
- Scahill L, Riddle MA, McSwiggin-Hardin M, et al: Children's Yale-Brown Obsessive Compulsive Scale: reliability and validity. *J Am Acad Child Adolesc Psychiatry* 36(6):844–852, 1997 9183141
- Schumer MC, Bartley CA, Bloch MH: Systematic review of pharmacological and behavioral treatments for skin picking disorder. *J Clin Psychopharmacol* 36(2):147–152, 2016 26872117
- Swedo SE, Rapoport JL, Leonard H, et al: Obsessive-compulsive disorder in children and adolescents. Clinical phenomenology of 70 consecutive cases. *Arch Gen Psychiatry* 46(4):335–341, 1989 2930330
- Williams KA, Swedo SE, Farmer CA, et al: Randomized, controlled trial of intravenous immunoglobulin for pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections. *J Am Acad Child Adolesc Psychiatry* 55(10):860–867.e2, 2016 27663941

■ ADDITIONAL READING

- Geller DA, March J; AACAP Committee on Quality Issues: Practice parameter for the assessment and treatment of children and adolescents with obsessive-compulsive disorder. *J Am Acad Child Adolesc Psychiatry* 51(5):541–557, 2012
- Geller DA, Williams K: Obsessive-compulsive disorder, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 365–388

TRAUMA- AND STRESSOR-RELATED DISORDERS

Trauma- and stressor-related disorders are a new category in DSM-5 (American Psychiatric Association 2013) that includes reactive attachment disorder, disinhibited social engagement disorder, post-traumatic stress disorder, acute stress disorder, and adjustment disorders.

■ REACTIVE ATTACHMENT DISORDER AND DISINHIBITED SOCIAL ENGAGEMENT DISORDER

Clinical Description

Attachment disorders develop in early childhood as a result of severely deficient caregiving environments. They comprise two patterns, which DSM-5 divides into two clinical disorders. In *reactive attachment disorder* (RAD), young children fail to focus attachment behaviors toward a preferred caregiver. They do not seek or respond to comfort, and they have reduced social and emotional reciprocity, reduced positive affect, increased irritability or fearfulness, and disturbances of emotional regulation. Symptoms must be apparent before age 5 years. In *disinhibited social engagement disorder* (DSED), children show lack of wariness of strangers, will indiscriminately approach unfamiliar adults, and may interact with adults in an overly proximate or intrusive way. Attachment disorder-

ders cannot be diagnosed before the child has a developmental age of 9 months, when focused attachment is expected to occur.

Epidemiology

In an economically deprived urban population sample in the United Kingdom, the prevalence of attachment disorders was 1.4% (Minnis et al. 2013). Prevalence of attachment disorders is highly dependent on the environment of the population studied.

Etiology

RAD and DSED both arise from a background of extreme insufficient care. Abuse may have also been present. DSM-5 requires at least one of the following in the patient's history: social neglect or deprivation with persistent unmet needs for comfort, stimulation, and affection; repeated changes of primary caregivers (as in frequent changes of foster placements or extremely disorganized families); or rearing in unusual settings that severely limit opportunities to form selective attachments (e.g., institutional settings with a high child-to-caregiver ratio). Children with *Williams syndrome* may exhibit a behavioral phenotype very similar to DSED, but they are not given the DSED diagnosis because the symptoms do not stem from a history of deprivation or abuse. Not all children who experience neglect or institutionalization will develop an attachment disorder. Factors that may contribute include length of time in an institution, the age at which a corrective attachment opportunity occurs, the presence of growth retardation, and genetic predisposition.

Course and Prognosis

Symptoms of RAD generally improve and may eventually resolve if affected children are placed in supportive, sensitive, and secure foster or adoptive homes. With placement in an environment where the child can develop secure attachments, some children with DSED will have marked reduction in indiscriminate behavior, but others will have continued difficulty with peer relationships. Long-term

outcomes for adult interpersonal or occupational function are not known for either RAD or DSED.

Evaluation

Attachment disorders are diagnosed through assessment instruments completed by caregivers and by clinical observation. The history of the caretaking environment is key. The Strange Situation Procedure, in which the child is alternately exposed to his or her mother and then a stranger, is one formal way to assess the quality of attachment, although it was not designed for routine clinical use. Clinically based structured observations of attachment have been developed (see Zeanah et al. 2016 in Additional Reading).

RAD and DSED are most often diagnosed in children who have already been institutionalized or identified as victims of neglect. If the child is residing with his or her parent(s) and exhibiting symptoms of an attachment disorder, evaluation of the parent's (parents') capacity and willingness to provide adequate care is indicated.

Differential Diagnosis

RAD can be differentiated from *autism spectrum disorder* by improvement in cognitive and social deficits when children with RAD are placed in a caring environment and the absence in RAD of abnormalities in social communication and restricted interests and preoccupations that are characteristic of autism. *Global developmental delay*, *depression*, and *posttraumatic stress disorder* are in the differential diagnosis for RAD. There is overlap between symptoms of DSED and *attention-deficit/hyperactivity disorder* (ADHD). If criteria are met, both may be diagnosed.

Treatment

For both RAD and DSED, the core feature of treatment is placing the child in a nurturing environment with the opportunity to form secure attachment. For children who will remain in their family of origin, specific parent training fosters improved attachment

(Bernard et al. 2012). The therapeutic approach generally involves working with the caregiver and with the caregiver-child dyad to strengthen attachment. There is no role for psychopharmacological treatment for the core symptoms of RAD or DSED (Zeanah and Gleason 2015).

■ POSTTRAUMATIC STRESS DISORDER AND ACUTE STRESS DISORDER

Clinical Description

Posttraumatic stress disorder (PTSD) encompasses the emotional, cognitive, behavioral, and physiological reactions that may occur after directly experiencing, witnessing, or learning of a loved one's experience of a traumatic event, such as actual or threatened death, serious injury, or sexual violence. Examples of traumatic events that may affect children and adolescents include child abuse; domestic, community, or school violence; natural disasters; vehicular or other accidents; medical traumas; war, terrorism, or refugee trauma; or the traumatic death of significant others. DSM-5 no longer classifies PTSD as an anxiety disorder. The diagnostic symptom clusters have been expanded from three to four: intrusion symptoms (memories, dreams, dissociative reactions, distress or physiological reactions in response to reminders), avoidance of internal or external reminders of the event(s), negative mood or cognition symptoms, and symptoms of altered arousal (anger or irritability, reckless or self-destructive behavior, hypervigilance, exaggerated startle response, difficulty concentrating, and sleep disturbance).

Based on research that identified developmental factors affecting symptom presentation, alternative DSM-5 diagnostic criteria have been added for children 6 years and younger. Even older children may express symptoms differently than older adolescents or adults. Intrusive symptoms in children may be expressed through repetitive, trauma-themed play or trauma-themed reenactment. Children may report "scary" dreams but not be able to fully verbalize nightmares. Reminders of the trauma may trigger somatic symp-

toms. Avoidance of trauma cues may manifest as increased clinginess or separation anxiety fears. Negative alterations in mood or cognition may present as confusion, forgetfulness, alterations in concentration, inhibition, sense of foreshortened future, preoccupation with death of self or loved ones, self-blame (that may lead to lack of disclosure in situations of abuse), and behavioral regression. Although children may accurately remember many details of the experience, sequencing or duration of events is often distorted. Alterations in arousal may manifest as temper outbursts, irritability, physical or verbal aggression, emotional numbing, hypoactivity, sleep disturbance, and increased awareness of the environment. Perceptual distortions can occur, including tactile, olfactory, visual, and auditory misperceptions.

The diagnosis of PTSD requires symptom duration of at least 1 month. *Acute stress disorder* refers to trauma-reactive symptoms that last for at least 3 days but no longer than 30 days. Other than duration, acute stress disorder has diagnostic criteria similar to those for PTSD, and both disorders cause significant distress or impairment in social, occupational, or other important areas of functioning.

Epidemiology

The rate of experiencing a traumatic event in childhood or adolescence is as high as 25% (Costello et al. 2002). Fortunately, the majority of individuals who experienced such an event do not develop PTSD. Prevalence rates of acute stress disorder range from 14% to 51% (Kassam-Adams et al. 2012). In a recent meta-analysis, the rate of childhood PTSD was estimated at 16% (Alisic et al. 2014). Rates varied with gender and type of trauma. Girls with a history of interpersonal trauma developed PTSD at rates of nearly 33%, compared with 8.4% in boys with a history of non-interpersonal trauma. Rates of both trauma exposure and PTSD are higher in girls than in boys. Risk of development of PTSD is increased with more intense or longer trauma exposure, proximity to the traumatic event, presence of potential serious injury or violence, and interpersonal violence (particularly if a parent or caregiver is the perpetrator). Risk

factors identified for developing PTSD after a disaster include media exposure, peritraumatic panic symptoms, delayed evacuation, sense of endangerment, and premorbid anxiety disorder (Cohen and Mannarino 2016).

Etiology

For diagnosis of PTSD and acute stress disorder, DSM-5 requires an identifiable etiological agent (i.e., the extreme traumatic event). Type of traumatic event may predict development of acute stress disorder, with higher rates in children who have experienced bodily injury, pain, or violence. Children who have experienced multiple stressors, prior loss, disturbances in family functioning, or psychiatric comorbidity are vulnerable to more severe and prolonged symptoms, but a sufficiently severe stressor can produce the disorder in a person without any predisposition. The onset of PTSD likely involves a complicated interaction of neuroendocrine dysregulation with genetic, psychological, and social factors (Caspi et al. 2002). Abuse or neglect of children may cause cognitive and developmental delays as well as increased arousal or withdrawal secondary to changes in brain physiology. Given the high degree of comorbidity with both internalizing and externalizing disorders, it is possible that trauma affects functioning in a more generalized manner.

Course and Prognosis

Whether a “natural recovery” occurs from PTSD is controversial. One meta-analysis found that without intervention, 50% of children and adolescents no longer met PTSD symptom criteria 6 months posttrauma (Hiller et al. 2016). However, other studies have found maintenance of PTSD symptoms with time (Scheeringa et al. 2005). Predictors of acute stress disorder symptom persistence in youth with severe injuries were female gender, presence of peritraumatic dissociation, and lower socioeconomic status (Brown et al. 2016). Another study found no difference in rates of acute stress disorder and

PTSD rates in boys who had been traumatized in motor vehicle accidents versus assault. Presence of dissociation and hyperarousal symptoms predicted development of PTSD (Meiser-Stedman et al. 2005). In addition to the initial trauma, disasters often result in loss of home, isolation from usual social supports, and loss of parents or other family members, all of which can exacerbate the PTSD. Symptoms may be partially ameliorated by a stable, cohesive, and supportive family and safe environment. In cases of child abuse or domestic violence, potential legal and child welfare involvement, which may lead to disruption of the family unit, numerous interviews, or change in homes, may also trigger or prolong trauma-reactive symptoms.

Symptoms of 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. For the subset of youth with persistent PTSD, prognosis is poor if untreated. PTSD in childhood is a significant risk factor for adult suicide attempts, major depression, dissociation, and impaired overall functioning (Warshaw et al. 1993). Maltreatment in childhood significantly increases the risk of adult depression and anxiety (Li et al. 2016). A history of sexual or physical abuse during childhood is associated with increased risk of lifetime psychopathology, particularly for women (MacMillan et al. 2001).

Evaluation and Differential Diagnosis

At the time of clinical presentation, a traumatic event may or may not be known. Acute stress disorder or PTSD should be suspected in any child or adolescent who has had a significant change in behavior or emotional state. Multiple informants should be interviewed, as avoidance can lead to underreporting, the child may have difficulty describing trauma and symptoms, and caregivers may not know about or wish to disclose the trauma and may not be aware of the child's emotional reaction. The developmental history may sug-

gest increased vulnerability. The Child PTSD Symptom Scale (Foa et al. 2001) and the Child Stress Disorders Checklist (Saxe et al. 2003) are potentially useful rating scales. Teacher observations regarding changes in school behavior or achievement can contribute to the evaluation.

Comorbidity is common in PTSD and frequently includes mood, anxiety, and/or behavioral disorder diagnoses. Acute stress disorder and PTSD are distinguished from *adjustment disorder* by the severity and type of the stressor and the distinctive trauma-related symptoms, such as repetitive re-experiencing. PTSD can mimic many other disorders (any of which could be comorbid), such as *ADHD*, *oppositional defiant disorder*, *panic disorder*, *social anxiety disorder*, *depression*, *substance use*, *bipolar disorder*, *psychotic disorders*, or a variety of *physical conditions*.

Treatment

Established by Congress in 2000, the National Child Traumatic Stress Network (www.nctsn.org) serves as a resource for developing and disseminating evidence-based treatment and education.

Early identification of at-risk youth following a traumatic event can greatly lessen psychological morbidity. A preventative approach that includes screening and rapid referral to appropriate services is particularly applicable to traumas that affect many children (i.e., natural disasters). The Child and Family Traumatic Stress Intervention (CFTSI; Berkowitz et al. 2011), a four-session intervention that incorporates psychoeducation, relaxation training, and coping skills training, was more effective than care-as-usual in preventing progression to PTSD.

Evidence-based psychotherapy treatment models for youth with PTSD symptoms (described in Cohen and Mannarino 2016) share the following components: developmental and cultural sensitivity; informed by the neurobiological impact of trauma on children; inclusion of parents/caretakers/families; and aims of re-establishing safety and trust and addressing trauma reminders and significant areas of dysfunction. Selection among the evidence-

informed treatments for an individual child is influenced by developmental level, treatment setting, acceptance by the family, type of trauma, and other individual factors. Trauma-focused cognitive-behavioral therapy (TF-CBT) has the strongest empirical support. TF-CBT treatment components are summarized by PRACTICE: Psychoeducation and Parenting skills, Relaxation, Affective modulation, Cognitive processing, Trauma narrative, In vivo mastery of trauma memories, Conjoint child-parent sessions, and Enhancing safety. Cognitive-Behavioral Intervention for Trauma in Schools (CBITS) is a model that adapts TF-CBT to group therapy during the school day with limited parental involvement. Child-Parent Psychotherapy (CPP), a relationship-based model with some empirical support, is designed to improve interactions between young children and parents. Individual trauma-focused CBT for single-episode traumas, called Cognitive-Based CBT, has been developed and studied. More specialized modalities include the Surviving Cancer Competently Intervention Program (SCCIP), Trauma Grief Component Therapy for Adolescents (TGCT-A), Trauma Systems Therapy (TST), and Narrative Exposure Therapy for Children (KidNET; for youth exposed to war and refugee experiences). Other models, designed to address complex trauma or trauma with comorbidity, have shown some promising results. Trauma Affect Regulation: Guidelines for Education and Therapy (TARGET; Ford et al. 2012) was shown to be effective for girls in the juvenile justice system with complex trauma. The adolescent adaptation of Seeking Safety, a treatment curriculum for youth with PTSD and substance use disorders, is promising (Najavits et al. 2006). Web-based curricula have been developed to enhance dissemination of evidence-based treatments. It is important to screen for trauma-reactive symptoms in the parent or caregiver, who may have had shared or prior trauma, and refer for treatment if needed. Parental trauma-related symptoms predict greater symptoms in children.

Treatment of PTSD in youth should start with psychotherapy (rather than pharmacotherapy) unless there is a compelling reason to add medication, such as a comorbid diagnosis that requires medication, clear dangerousness requiring immediate medication, or

severe symptoms that persist despite psychotherapy. No randomized control trials have demonstrated efficacy of medication in pediatric PTSD. Limited evidence suggests that selective serotonin reuptake inhibitors, propranolol, clonidine, prazosin (for nightmares), morphine (for pain in burn victims), or risperidone can benefit traumatized youth (Cohen and Mannarino 2016). However, because of weak empirical support and the high placebo response in youth, medications should be used only if necessary.

■ ADJUSTMENT DISORDERS

Clinical Description

Adjustment disorder is characterized by the development of emotional and/or behavioral symptoms within 3 months of the onset of a stressor. Diagnosis requires marked distress that is out of proportion to the severity or intensity of the stressor and/or significant functional impairment. In DSM-5 (American Psychiatric Association 2013), adjustment disorders are classified as stressor-related disorders, instead of the prior residual category for subdiagnostic threshold clusters of symptoms. An adjustment disorder may be diagnosed in a person with another mental disorder only if an identifiable stressor leads to the development of symptoms that are not characteristic of the original disorder. Adjustment disorders have six subtypes, classified by patterns of symptoms of depression, anxiety, and behavior (conduct).

Common stressors in childhood and adolescence include parental divorce, change in schools, physical illness, the birth of a sibling, parental unemployment, and abuse or neglect. Adolescents may be particularly vulnerable to disruptions in a relationship with a boyfriend or girlfriend. Normal bereavement is exclusionary.

Epidemiology

The prevalence of adjustment disorders in community samples of youth is between 2% and 8%. Populations with particularly severe

stressors (i.e., surgical patients) have high rates of adjustment disorder. Children and adolescents usually have mixed presentations, with symptoms that are not exclusively emotional (mood, anxiety) or behavioral.

Etiology

The adjustment disorder is presumed to be precipitated by the identified stressor. The child's reaction to the stressor rather than the characteristics of the stressor determine whether the diagnosis is present. Even if the patient's emotional response to the stressor is developmentally expected, if the symptom presentation is causing significant impairment, a diagnosis of adjustment disorder is made.

Course and Prognosis

Symptoms of adjustment disorder will typically 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 after the stressor or its consequences have stopped, then a different diagnosis is warranted. The prognosis depends on the severity and duration of the stressor and its meaning to the child; the vulnerability of the individual; and the response of the family, school, and peers to both the stressor and the young person's reaction. In general, the prognosis is assumed to be benign, although patients can present with suicidal ideation and/or behavior, substance abuse, or recurrent somatic complaints. Patients with adjustment disorder are more frequently seen in medical emergency rooms and admitted to inpatient units than are control groups without psychiatric symptoms.

Follow-up of adolescents 5 years after receiving a clinical diagnosis of adjustment disorder found that 57% were well, although 23% of these patients had qualified for another psychiatric diagnosis during the intervening period. Research diagnoses at follow-up included schizophrenia, affective disorders, antisocial personality disorder, and substance abuse disorders. One patient had committed suicide (Andreasen and Hoenk 1982). Another study found a 25%

rate of suicidal ideation, suicide threats, or suicide attempts in clinic-referred adolescent outpatients diagnosed with adjustment disorder (Pelkonen et al. 2005).

Evaluation and Differential Diagnosis

The clinician should obtain reports from the child, parent, and teacher to identify all stressors, rather than assuming that the first reported or obvious stressor is the crucial one. Other psychiatric diagnoses should be sought, and if diagnostic criteria are met, the diagnosis of adjustment disorder is precluded. In the past, overuse of the diagnosis of adjustment disorder (in an effort to avoid “labeling” children) has often obscured another psychiatric diagnosis. If the reaction to the death of a loved one exceeds what might be expected in intensity, quality, or persistence, then *persistent complex bereavement disorder* (included in DSM-5 under Conditions for Further Study) should be considered. If specific psychological aspects (emotional symptoms and behaviors) exacerbate a medical condition (e.g., asthma or diabetes), then *psychological factors affecting other medical conditions* should be considered. In contrast, adjustment disorder is descriptive of the reaction to the illness. If the stressor is sudden and catastrophic or potentially so, and the other diagnostic criteria are met, then *acute stress disorder* or *PTSD* should be considered.

Treatment

Crisis intervention and time-limited psychotherapy techniques may be useful for treatment of adjustment disorder. Cognitive therapy 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 and to mobilize family and community support systems. Explanations to parents and teachers of the child’s or adolescent’s reactions may reduce impairment and shorten the course of the disorder. Treatments established for similar diagnoses

may be useful. For example, if adjustment disorder with depressed mood is debilitating, the same treatments as for major depression may be indicated.

■ REFERENCES

- Alisic E, Zalta AK, van Wesel F, et al: Rates of post-traumatic stress disorder in trauma-exposed children and adolescents: meta-analysis. *Br J Psychiatry* 204:335–340, 2014 24785767
- American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Andreasen NC, Hoenk PR: The predictive value of adjustment disorders: a follow-up study. *Am J Psychiatry* 139(5):584–590, 1982 7072842
- Berkowitz SJ, Stover CS, Marans SR: The Child and Family Traumatic Stress Intervention: secondary prevention for youth at risk of developing PTSD. *J Child Psychol Psychiatry* 52(6):676–685, 2011 20868370
- Bernard K, Dozier M, Bick J, et al: Enhancing attachment organization among maltreated children: results of a randomized clinical trial. *Child Dev* 83(2):623–636, 2012 22239483
- Brown RC, Nugent NR, Hawn SE, et al: Predicting the transition from acute stress disorder to posttraumatic stress disorder in children with severe injuries. *J Pediatr Health Care* 30(6):558–568, 2016 26776839
- Caspi A, McClay J, Moffitt TE, et al: Role of genotype in the cycle of violence in maltreated children. *Science* 297(5582):851–854, 2002 12161658
- Cohen JA, Mannarino AP: Posttraumatic stress disorder and persistent complex bereavement disorder, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 345–364
- Costello EJ, Erkanli A, Fairbank JA, Angold A: The prevalence of potentially traumatic events in childhood and adolescence. *J Trauma Stress* 15(2):99–112, 2002 12013070
- Foa EB, Johnson KM, Feeny NC, Treadwell KR: The child PTSD Symptom Scale: a preliminary examination of its psychometric properties. *J Clin Child Psychol* 30(3):376–384, 2001 11501254
- Ford JD, Steinberg KL, Hawke J, et al: Randomized trial comparison of emotion regulation and relational psychotherapies for PTSD with girls involved in delinquency. *J Clin Child Adolesc Psychol* 41(1):27–37, 2012 22233243

- Hiller RM, Meiser-Stedman R, Fearon P, et al: Research Review: Changes in the prevalence and symptom severity of child post-traumatic stress disorder in the year following trauma—a meta-analytic study. *J Child Psychol Psychiatry* 57(8):884–898, 2016 27169987
- Kassam-Adams N, Palmieri PA, Rork K, et al: Acute stress symptoms in children: results from an international data archive. *J Am Acad Child Adolesc Psychiatry* 51(8):812–820, 2012 22840552
- Li M, D’Arcy C, Meng X: Maltreatment in childhood substantially increases the risk of adult depression and anxiety in prospective cohort studies: systematic review, meta-analysis, and proportional attributable fractions. *Psychol Med* 46(4):717–730, 2016 26708271
- MacMillan HL, Fleming JE, Streiner DL, et al: Childhood abuse and lifetime psychopathology in a community sample. *Am J Psychiatry* 158(11):1878–1883, 2001 11691695
- Meiser-Stedman R, Yule W, Smith P, et al: Acute stress disorder and post-traumatic stress disorder in children and adolescents involved in assaults or motor vehicle accidents. *Am J Psychiatry* 162(7):1381–1383, 2005 15994725
- Minnis H, Macmillan S, Pritchett R, et al: Prevalence of reactive attachment disorder in a deprived population. *Br J Psychiatry* 202(5):342–346, 2013 23580380
- Najavits LM, Gallop RJ, Weiss RD: Seeking safety therapy for adolescent girls with PTSD and substance use disorder: a randomized controlled trial. *J Behav Health Serv Res* 33(4):453–463, 2006 16858633
- Pelkonen M, Marttunen M, Henriksson M, Lönqvist J: Suicidality in adjustment disorder—clinical characteristics of adolescent outpatients. *Eur Child Adolesc Psychiatry* 14(3):174–180, 2005 15959663
- Saxe G, Chawla N, Stoddard F, et al: Child stress disorders checklist: a measure of ASD and PTSD in children. *J Am Acad Child Adolesc Psychiatry* 42(8):972–978, 2003 12874500
- Scheeringa MS, Zeanah CH, Myers L, Putnam FW: Predictive validity in a prospective follow-up of PTSD in preschool children. *J Am Acad Child Adolesc Psychiatry* 44(9):899–906, 2005 16113618
- Warshaw MG, Fierman E, Pratt L, et al: Quality of life and dissociation in anxiety disorder patients with histories of trauma or PTSD. *Am J Psychiatry* 150(10):1512–1516, 1993 8379556
- Zeanah CH, Gleason MM: Annual research review: Attachment disorders in early childhood—clinical presentation, causes, correlates, and treatment. *J Child Psychol Psychiatry* 56(3):207–222, 2015 25359236

■ ADDITIONAL READING

Nelson CA, Fox NA, Zeanah CH: Romania's Abandoned Children: Deprivation, Brain Development and the Struggle for Recovery. Cambridge, MA, Harvard University Press, 2014

Zeanah CH, Chesher T, Boris NW; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter for the assessment and treatment of children and adolescents with Reactive Attachment Disorder and Disinhibited Social Engagement Disorder. *J Am Acad Child Adolesc Psychiatry* 55(11):990–1003, 2016

FEEDING AND EATING DISORDERS

DSM-5 (American Psychiatric Association 2013) placed all of the eating and feeding disorders in a single section, whether the typical onset is in early childhood (pica, rumination disorder), at any time in the lifespan (avoidant/restrictive food intake), or in adolescence and young adulthood (anorexia nervosa [AN], bulimia nervosa [BN]). None of these diagnoses has onset restricted to a single developmental period.

■ PICA

Clinical Description

Pica is the persistent consumption of nonnutritive, nonfood materials that is not developmentally appropriate and not culturally sanctioned or socially normative. Pica in children most often comes to clinical attention in association with other behavior and medical problems.

Epidemiology

Pica can occur at any age, most commonly with childhood onset. A minimum age of 2 years for the diagnosis distinguishes pica from developmentally normative mouthing and ingestion of objects in infants and toddlers. Pica may manifest in pregnancy with cravings for nonfood items such as chalk or clay, but the diagnosis is considered only if the ingestion poses medical risk. Pica may be more prevalent in individuals with intellectual disability, but overall prevalence is unknown.

Etiology

Possible causes include nutritional deficiencies, insufficient stimulation, parent–child relationship difficulties, or cultural tradition, although there is no definitive support for any of these.

Course and Prognosis

Pica usually starts in young children and resolves by school age except in persons with intellectual disability, who may have pica into adulthood. Common nonfood items ingested include ice, chalk, paper, clay, and soil. Delayed motor and mental development, neurological deficits, and behavioral abnormalities may predate (and perhaps contribute to) or result from pica. Pica has numerous potentially severe medical complications, including heavy metal poisoning, parasitic infections, or intestinal obstruction. Lead toxicity (from ingestion of peeling paint, dust from home renovation, plaster, or soil) can lead to learning disorders, hyperactivity, fatigue, weight loss, and constipation, and even to toxic encephalopathy.

Evaluation

Pica should be considered in all cases of developmental delays, learning difficulties, intellectual disability, unusual behavioral symptoms, and chronic constipation. In order to diagnose pica, the clinician must complete a full psychiatric evaluation (see Chapter 2, “Evaluation and Treatment Planning”) as well as determine the child’s nutritional status and feeding history. Inadequate supervision of children or parental neglect should be considered. Questions should be asked about peeling paint or renovation in the home that might increase lead exposure. Periodic blood tests for lead in children with pica are recommended. Consultation with pediatric colleagues is indicated if blood lead levels are greater than 4 micrograms per deciliter.

Treatment

Behavior therapy has been successful in the treatment of pica in individuals with intellectual disability and may be applicable to other

children. Components include education of caregivers, rewards for appropriate eating, and teaching how to differentiate edible foods. Appropriate responses to instances of pica include overcorrection (enforced immediate oral hygiene) and negative reinforcement (time-outs, restriction of privileges), combined with a positive reinforcement contingency behavior plan (reward system). Additional interventions include increased supervision, promotion of appropriate stimulation, improvement of play opportunities, or placement in day care. Concomitant treatment of medical complications may be required.

■ RUMINATION DISORDER

Clinical Description

Rumination is the regurgitation and rechewing or spitting out of recently ingested food, which, when it recurs repeatedly, is called *rumination disorder*. Once thought to occur only in infants or in individuals with intellectual disability, rumination disorder is now recognized to occur in developmentally typical children and adults. In older children and adults, rumination begins with a rise in intra-gastric pressure produced by voluntary, but not always intentional, contracture of the abdominal muscles.

Etiology

The etiology is unknown. Rumination is highly associated with gastroesophageal reflux. Clinicians generally presume that the infant, lacking external sources of gratification, uses rumination for self-stimulation or social reinforcement. However, although an understimulating or overstimulating environment can contribute to the appearance of rumination disorder, some infants with the disorder appear happy, are developing normally otherwise, and have emotionally supportive and interactive parents.

Course and Prognosis

Rumination typically occurs during the first year of life, resolving by the second year, or in adolescence, often accompanied by anxiety, depression or other eating disorders. Tooth decay, failure to thrive, dehydration, electrolyte imbalance, and malnutrition can be complications.

Evaluation and Differential Diagnosis

Behavioral and psychiatric evaluation of the child and parents emphasizes developmental history and psychosocial assessment, feeding and eating history, nutritional status, attachment, and observation of parent–child interactions during feeding. Medical conditions, including gastroesophageal reflux and cyclic vomiting, must be considered.

Treatment

Behavioral consultation is highly recommended in treating rumination disorder. In infants, parent training focusing on behavioral techniques, including positive attention and interaction (cuddling and playing with the child before, during, and after mealtimes), will reduce social deprivation and behavioral withdrawal. Negative reinforcement (ignoring) combined with a reward for time not ruminating (parental attention and social interaction, such as playing) can be used in outpatient treatment. In adolescents, behavioral interventions include habit reversal training (HRT), diaphragmatic breathing, and biofeedback (Mousa et al. 2014).

Pediatric hospitalization may be necessary if rumination results in significant malnutrition. Hospitalization can be beneficial by providing a partial separation of the child from the primary caregiver, an alternative feeding environment for the child (to “decondition” the symptoms), and a period of respite for the parent. Continuing treatment facilitates attachment between child and parent, monitors the psychosocial environment at home, and provides support in the event of emerging developmental problems.

■ AVOIDANT/RESTRICTIVE FOOD INTAKE DISORDER

Clinical Description

DSM-5 (American Psychiatric Association 2013) renamed this disorder and expanded the criteria. *Avoidant/restrictive food intake disorder* (ARFID) is characterized by restrictive or food avoidant behaviors that result in failure to meet energy or nutritional requirements. These behaviors may result from lack of interest in eating, sensory sensitivities, fear of choking, or history of traumatic experiences such as forceful feeding. They do not, by definition, have fear of gaining weight as a motivation. Limited information is available on this heterogeneous disorder.

Epidemiology

Prevalence data are currently limited to clinical samples. In an eating disorders clinic (patients age 8–18 years), 14% of patients presented with a diagnosis of ARFID (Fisher et al. 2014). Compared with youth with diagnoses of anorexia nervosa or bulimia nervosa, patients with ARFID were younger, more likely to be male, and more likely to have comorbid medical or anxiety disorders, and had a median body weight percentile between that of patients with AN and BN. Eddy et al. (2015) identified 1.5% of patients presenting to a gastroenterology clinic network as having symptoms that met full criteria for ARFID, also with more males than females.

Etiology

Many children who develop ARFID have a history of picky eating from early childhood. The most common underlying pattern in the Eddy et al. (2015) series was lack of interest in food, followed by limited diet due to sensory issues. The aversive/traumatic experience type is least common.

Course and Prognosis

Because the diagnosis of ARFID is relatively new, little is known about the long-term course and prognosis. In a study of children hospitalized with ARFID, a year after admission about half the patients met criteria for readmission and 20% were readmitted. These were similar numbers to patients with AN (Strandjord et al. 2015).

Evaluation and Differential Diagnosis

ARFID must be distinguished from failure to take in adequate nutrients due to *medical conditions*, including malabsorption, neoplasm, gastroesophageal reflux, or other gastrointestinal disorders. It differs from *anorexia nervosa* because patients with ARFID are not motivated to lose weight and are not dissatisfied with their weight or body image. ARFID is distinct from *normative picky eating* in that the child fails to meet nutritional requirements, has substantial weight loss (or, in children, absence of expected weight gain), becomes dependent on enteral feeding or dietary supplements, and/or has significant psychosocial interference.

Evaluation includes medical, dietary, and psychosocial histories and an examination to measure height and weight (and calculate body mass index). Review of a longitudinal growth record is useful. Laboratory evaluation should be done to assess for nutritional deficiencies (e.g., electrolyte abnormalities, anemia).

Treatment

Pediatric hospitalization may be required for individuals with severe nutritional deficiencies. There are very limited data on treatment, but for those individuals who can be managed as outpatients, a multidisciplinary, family-based approach that addresses the restrictive or food-averse behaviors is indicated. Behavioral or cognitive-behavioral treatments should be tailored to the underlying reasons for the disordered eating (Norris et al. 2016).

■ ANOREXIA NERVOSA AND BULIMIA NERVOSA

Anorexia Nervosa

Clinical Description

Anorexia nervosa is characterized by a refusal to maintain normal weight or a failure to reach expected weight gain as a result of intentional diet restriction or other extreme measures. Patients have either a distorted perception of their body shape and size or a denial of the seriousness of the weight loss. The preoccupation with food and body shape is obsessive. In severe cases, these patients may appear delusional. The appetite is not lost; rather there is an intense fear of gaining weight. DSM-5 (American Psychiatric Association 2013) removed the prior diagnostic requirement for amenorrhea. The *restricting type* of AN is characterized by strict dieting, fasting, or excessive exercise. In the *binge-eating/purging type*, huge quantities of food are eaten and then purged.

The physiological process of starvation leads to the development of additional psychological symptoms. Patients may become socially withdrawn, irritable, and anhedonic, with decreased interest in sex, low self-esteem, and recurrent feelings of helplessness and inadequacy. This picture may be consistent with major depression. Depression impairs the individual's ability to function in the classroom, in social situations, and within the family. Patients tend to be controlling, not only of their own habits but also of those around them. Patients are often preoccupied with personal academic achievement and exercise. An accompanying diagnosis of obsessive-compulsive disorder (OCD) should be made if criteria are met.

Epidemiology

The majority of patients with AN are female, with an estimated prevalence of 0.3%–0.7% of adolescent girls in the United States. Onset of AN prior to puberty is rare. The incidence peaks in the 14- to 24-year age group. Milder forms of eating disorders are even more

common. AN is found predominantly in Western industrialized nations, and the prevalence is probably significantly affected by social and societal factors. The prevalence in boys, minority populations, and non-Western countries is lower but increasing.

Etiology

Various biological, psychological, and environmental factors have been associated with AN, although whether these are correlative or etiological is not clear (Le Grange 2016). Genetic and neurophysiological mechanisms contribute to the development of both AN and BN. First-degree relatives of anorexic and bulimic patients are 6–10 times more likely to have an eating disorder than are control populations. However, it remains difficult to separate environmental from heritable causative factors. Patients experience a variety of psychological, physical, academic, and social problems, although distinguishing cause and effect is often difficult. These patients tend to be dissatisfied with their bodies at puberty. Dieting is chosen as a socially acceptable method to improve the patient's sense of well-being and control. Patients with AN tend to be perfectionistic, cognitively rigid, and detail-oriented, with an obsessive and conflict-avoidant personality style. Some studies have found that societal ideals of beauty (particularly in Western cultures), peer group influences, and engagement in certain activities (e.g., gymnastics, wrestling, dance, modeling) increase the risk of AN.

Family dynamics, including parental over-involvement, lack of appropriate boundaries within the family, and insufficient autonomy, have received much attention in the literature for the past four decades (Minuchin et al. 1978). However, few studies have tested these theories, and these characteristics are also present in families in which eating disorders do not develop. Thus, the etiological role, if any, of family dynamics remains unclear.

Course and Prognosis

AN usually manifests during adolescence, between ages 14 and 18 years, a time of rapid growth with accompanying weight gain and

changes in body shape. Although onset before puberty is less common, prepubertal children may develop AN or problem eating behaviors, including food avoidance, body image disturbance, inappropriate dieting, overeating, ritualistic behavior during meals, and selective eating. The first evidence of dissatisfaction with body shape may be found in a preoccupation with dieting. In one community survey, more than half of the girls considered themselves to be overweight, although only 15% were actually overweight (Mellin et al. 1992).

Physiological sequelae are common. Serious medical complications may require hospitalization (Palla and Litt 1988; Table 10–1). The course of AN is typically prolonged, and the disorder does not remit without treatment. Continuing symptoms include low weight for height and age, peak bone mass reduction, excessive concern with weight or appearance, pubertal delay or interruption, amenorrhea, morbidity and mortality associated with serious medical complications, and troubled social and sexual relationships. Poor outcome is associated with longer duration of illness, extreme weight loss, and poor interpersonal relationships. Adolescent patients have a better prognosis, lower mortality, and better response to treatment than do adults.

Mortality in part depends on chronicity. Risk of death from medical complications of AN is estimated at between 5% and 7%, with as many as 50% of these deaths due to suicide (Arcelus et al. 2011). Comorbid psychiatric diagnoses contribute to a poor prognosis, and partial remission is common, especially in patients with psychiatric comorbidity.

Evaluation and Differential Diagnosis

A complete history, physical examination, and routine laboratory studies are necessary to rule out other psychiatric or medical causes for loss of weight or appetite. In addition, starvation can produce physiological disturbances that should be identified through laboratory studies (see Table 10–1). An electrocardiogram and DEXA scan (to evaluate for osteoporosis) should be ordered in addition to

TABLE 10–1. **Physical signs and symptoms and complications associated with anorexia nervosa and bulimia nervosa**

Cardiovascular	Hypotension (especially postural)
	Bradycardia (rates between 40 and 50 beats per minute)
	Arrhythmias (prolonged QT interval may be a marker for risk of sudden death)
	Mitral valve prolapse
	Cardiac arrest
	Edema and congestive heart failure during refeeding
Neuroendocrine	Cardiac failure secondary to cardiomyopathy from ipecac (emetine) poisoning
	Amenorrhea or irregular menses (low levels of FSH and LH despite low estrogen levels)
	Low basal metabolism rate
	Abnormal glucose tolerance test with insulin resistance
	Hypothermia
	Elevated levels of growth hormone and cortisol
Bone	Sleep disturbances
	Osteopenia
Fluid disturbance	Dehydration
	Electrolyte imbalance
	Abnormal urinalysis
Gastrointestinal	Constipation
	Diarrhea

TABLE 10-1. **Physical signs and symptoms and complications associated with anorexia nervosa and bulimia nervosa (*continued*)**

Hematological	Leukopenia
	Anemia
	Thrombocytopenia
	Low sedimentation rate
Dermatological	Dry skin
	Lanugo (baby-fine body hair)
Oral, esophageal, and gastric damage from vomiting and/or binge eating	Loss of dental enamel
	Gastritis
	Enlarged salivary glands
	Esophagitis
	Esophageal tear
	Pancreatitis

Note. FSH=follicle-stimulating hormone; LH=luteinizing hormone.

Source. Adapted from Palla and Litt 1988.

blood tests. Hypoglycemia is a particularly poor prognostic sign. Dehydration can lead to elevations in blood urea nitrogen, liver function test values, and serum cholesterol levels. Signs of infection may be masked by leukopenia and hypothermia. Abnormal thyroid function studies are classified as “sick-euthyroid syndrome,” with normal to low levels of thyroxine (T_4) and free T_4 and variable levels of thyroid-stimulating hormone.

The psychiatric interview of a patient with suspected AN includes details about the onset and course of the eating disorder, the highest and lowest weight, and the weight identified by the patient as most comfortable. The clinician should also explore daily eating patterns, including not only amounts of food but also times for meals. Questions about the use of laxatives, emetics, or diuretics and exercise can be incorporated into this eating history. Although anorexic patients learn to disguise their continuing wish to lose weight, hide excessive exercise and purging, and claim to eat more than they do, they may inadvertently reveal important diagnostic information during an eating history. Family members may provide supplementary information, although patients with AN often hide their symptoms.

In addition to individual assessments, a family evaluation is essential in treating young patients. The therapist may ask family members about their impressions of the illness and its origins. A review of at-home treatment attempts uncovers family dynamics and individual perceptions about the problem's origins and possible solutions. A family eating or weight history may show patterns of behavior imitated by the child or adolescent. Family history of psychiatric illness may suggest similar diagnoses in the patient.

The differential diagnosis of AN is shown in Table 10–2.

Treatment

The treatment of AN should be comprehensive and emphasize a return to normal eating patterns. In most cases, the initial focus of treatment is on acceptance of the disorder and weight gain. In severe cases, this initial phase may take place on an inpatient unit. Typical indications for inpatient hospitalization include weight

TABLE 10-2. **Differential diagnosis of anorexia nervosa**

Normal thinness

Physical disorders causing weight loss

Hyperthyroidism
Cystic fibrosis
Other endocrine disorders, such as Addison's disease and diabetes mellitus
Gastrointestinal disorders resulting in vomiting, loss of appetite, and/or malabsorption
Malignancy
Chronic infection

Psychiatric disorders causing loss of appetite and weight loss

Depression
Avoidant/restrictive food intake disorder
Rumination disorder
Peculiar eating behavior secondary to obsessive-compulsive disorder or to delusions in schizophrenia or psychotic depression
Avoidance of eating caused by phobia of choking, with or without psychosis
Vomiting secondary to conversion disorder

Hypothyroidism producing hypothermia and amenorrhea

more than 25% below ideal body weight, rapid and severe weight loss refractory to outpatient treatment, hypothermia, symptomatic hypotension or syncope, heart rate below 50 beats per minute, severe electrolyte abnormalities, severe dehydration or evidence of arrhythmia, or a prolonged QTc interval. The denial of illness and fears of loss of control and of becoming fat generate severe resistance to treatment, even when the patient and family acknowledge the diagnosis. A firm focus on a target weight within 90% of the ideal body weight is helpful. The patient should reach this goal through gradual weight gain of 1 pound per week as an outpatient or 2–3 pounds per week while hospitalized (Mehler 2001). Too-rapid increase in food intake can lead to medically dangerous “refeeding syndrome.” Appetite stimulants are not recommended and may be contraindicated. Involvement of a primary care physician and a pediatric dietitian with experience in eating disorders is essential, whether treatment is inpatient or outpatient. Psychotherapeutic approaches can act as an adjunct to the refeeding regimen. Forced weight gain alone is futile, and too-rapid weight gain exacerbates fears of loss of control and may be medically hazardous. Tube or intravenous feeding is reserved for medical emergencies, because these methods are viewed by patients as punitive. The patient’s sabotage of these methods can be dangerous (e.g., bleeding from the site of a pulled intravenous line, aspiration of tube feedings). Regardless of the success of refeeding, a comprehensive treatment plan that includes an exploration of underlying psychological factors is essential.

Family therapy is particularly effective in adolescents with AN. The Maudsley model of *family-based treatment* (FBT) has the strongest empirical support (Lock et al. 2005). Families are affected secondarily by the presence of the eating disorder, and the clinician should not assume that every family has premorbid problems. Psychoeducational programs for family members greatly facilitate treatment and may be done in an individual or group setting (see Appendix, “Resources for Parents”). Family involvement in treatment, whatever the primary modality, clearly benefits young patients. Current FBT emphasizes parents as part of the solution, not the cause of

the problem. *Parent-focused treatment* (PFT) is a modification of FBT in which the therapist meets with the parents only, while a nurse monitors the patient. In a randomized controlled trial, PFT was more efficient at weight restoration in adolescent patients with AN (Le Grange et al. 2016).

Individual psychotherapy is generally a second choice for adolescent patients but may be indicated for patients whose families are unwilling or unable to participate. Adolescent focused therapy (AFT) and cognitive-behavioral therapy (CBT) have demonstrated efficacy in treatment of AN (Lock et al. 2015). CBT initially emphasizes changing incorrect beliefs and dysfunctional cognitions about food and eating. The focus of treatment eventually extends to include issues of body weight, appearance, peer relationships, and individual control. Cognitive-behavioral interventions (e.g., the use of food diaries) have been effective, particularly in preventing relapse in patients once weight has returned to normal.

Once the patient's eating patterns have normalized, interpersonal psychotherapies may provide insight for the patient that will facilitate long-term recovery. Older patients tend to do well with individual therapies, while younger patients respond best to family interventions.

There is little evidence supporting the use of medication in the treatment of AN. Adjunctive atypical antipsychotic medication prescribed to increase appetite and reduce rumination has not demonstrated significant efficacy in studies. Although use of selective serotonin reuptake inhibitors (SSRIs) has not been systematically studied in adolescents with AN, studies of SSRIs in adults with AN have not been encouraging. However, SSRIs may assist with comorbid anxiety and depression, once weight has stabilized. Current treatment guidelines recommend reserving medications for comorbid conditions and refractory cases (Lock et al. 2015).

Bulimia Nervosa

Clinical Description

Bulimia nervosa is characterized by repeated episodes of uncontrollable binge eating of huge amounts of food in a short time (2-hour

period) accompanied by excessive attempts to compensate for this caloric intake. Bingeing and compensatory behaviors occur (on average) at least once a week for at least 3 months. Binge eating is typically done secretly and may initially be pleasurable. The patient may engage in the behavior during a dysphoric episode or in response to a recent stressor. The binge usually brings the patient relief, but self-deprecatory thoughts return. Patients feel that their eating is out of control.

Exercise and strict fasting are the most common compensatory behaviors, followed by induced vomiting. The use of laxatives, enemas, diuretics, or thyroid medication is less common, particularly in the pediatric population.

The frequency of binges increases as the disorder develops. Patients with BN are generally of normal weight but may be slightly over or under their ideal weight, and may have been overweight before the onset of the disorder. The patient's self-image is unduly influenced by body shape and weight.

Epidemiology

Studies of the prevalence of BN are affected by changing diagnostic criteria, the relative recent definition of the diagnosis, and the secrecy of the behavior. BN is more common than AN. In almost 50% of cases, onset occurs before age 18 years. Binge eating is common among adolescents, but relatively few have signs and symptoms that meet diagnostic criteria for BN. Approximately 1%–2% of adolescent females and 0.5% of adolescent males in the United States had signs and symptoms that met DSM-IV criteria for BN. Males account for 10%–15% of all clinically referred patients with BN. Patients generally become ill in the latter half of adolescence. Risk is increased in ballet dancers and elite athletes whose sport emphasizes thinness. Bulimic patients have increased rates of drug and alcohol use, anxiety, depression, and PTSD. They tend to be of higher socioeconomic status and are typically white or Hispanic. Recent studies have noted increasing rates of BN and purging with laxatives in the African American population, although the prevalence is still lower than in whites or Latinas. Dysfunctional behaviors re-

lated to eating and weight are relatively common among adolescent and young adult females. Patients who binge but lack the compensatory behaviors (e.g., purging, excessive exercise) may meet criteria for binge-eating disorder. Studies of adolescent females note that between 40% and 60% are “dieting” to lose weight. This is particularly true among white adolescent girls from high-income families. In more than 10% of these, dieting may include induced vomiting or the use of diet pills and diuretics. Dieting behavior clearly increases the risk for eating disorders.

Etiology

AN and BN frequently present on a continuum, and many patients demonstrate symptoms of each. Approximately 50% of patients with AN will develop bulimic symptoms, and up to a quarter of patients with BN become anorectic. Identical twins of patients with BN have higher rates of the disorder. Monozygotic twins have higher concordance rates than do dizygotic twins. A family history of obesity, depression, or alcoholism is common. Sexual and physical abuse predispose children to various psychiatric disorders but not preferentially to the development of eating disorders.

Course and Prognosis

Dieting usually precedes the development of BN. The patient first begins to binge-eat as a direct result of food restriction; the behavior then becomes a compulsion or an addiction. The patient regards bingeing as abhorrent behavior, which contributes to feelings of depression and self-criticism. The patient once again begins dieting, often augmented with vigorous exercise or purging, in an attempt to undo the damage of the binge. Although these behaviors give the patient temporary relief from the emotional pain, they become part of a vicious cycle that maintains symptoms of BN and depression. A constant state of semi-starvation results, which places the patient at additional risk for mood disorders. The rates of suicidal ideation (53%) and suicide attempts (35%) are elevated in adolescents with BN (Lock et al. 2015).

Few studies have examined either the short- or the long-term prognosis for treated bulimic patients. Relapse rates are 30%–50% when patients are followed for 6 months to 6 years. Some speculate that improvement continues for 10–15 years. Risk factors for relapse include induced vomiting and the use of alcohol or drugs. Patients with milder symptoms at the onset of treatment appear to have a better prognosis. Among bulimic adolescents, comorbid anxiety or mood disorders predict continued eating-disordered behaviors. No factors have been identified as predictive of treatment success. Studies note a BN mortality rate of approximately 5%.

Social complications can be severe, with time and finances depleted by obtaining food, binge eating, and purging. School functioning and peer relationships typically deteriorate. Associated disorders include anxiety, depression, PTSD, substance abuse, and (in adults) borderline personality disorder. The presence of a comorbid medical diagnosis (i.e., diabetes, cystic fibrosis) may cause otherwise benign levels of symptoms to become dangerous. The risk of suicide or death from medical complications is significant. Risk factors for death from bulimia include a greater than 2-year duration; daily vomiting or bingeing; and use of laxatives, diuretics, ipecac, and stimulants (diet pills).

Evaluation and Differential Diagnosis

The clinician should evaluate the patient by 1) obtaining a detailed lifetime history of weight changes, noting periods of greatest fluctuation; 2) inquiring about current eating patterns in a typical day, including the number of calories consumed and any use of diet pills; 3) determining the onset and current status of bingeing and purging behaviors; and 4) asking about possible use of thyroid hormone, excessive exercise, laxatives, and diuretics. The medical history may suggest a neurological, endocrinological, or genetic (e.g., Prader-Willi syndrome) etiology of binge eating. Patients may have medical complications (see Table 10–1). The abuse of laxatives can lead to abdominal cramping, diarrhea, or rectal bleeding. Vomiting or abuse of diuretics or laxatives can produce metabolic alkalosis, elevations in serum amylase, hypomagnesemia and hypophosphate-

mia, and hypokalemia. A review of psychiatric history focuses on comorbidity and on previous treatment attempts. The individual's social and personal history may reveal contributing factors. Family assessment considers dynamics; previous attempts to help the patient; eating habits; and a review of significant social, medical, and psychiatric family history.

Treatment

Goals of treatment include eliminating the binge-purge cycle, establishing healthy eating habits, and promoting new strategies and skills to deal with emotions and problematic situations. The treatment plan progresses in a stepwise fashion, beginning with nutritional rehabilitation. Nutritional consultation can facilitate eating of regular, well-balanced meals to avoid the hunger that triggers the urge to binge. Patients with BN who maintain a normal weight generally do not require hospitalization. Hospitalization is indicated when the patient is suicidal, has out-of-control eating and vomiting, is metabolically unstable, or does not respond to 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.

A manualized family-based treatment for BN in adolescents has empirical support (Le Grange et al. 2015). CBT is the most effective treatment for BN in adults and may be preferable to family-based treatment for adolescents from high-conflict families. Case series and one controlled trial support the use of CBT in adolescents (Schmidt et al. 2007). It can help patients overcome feelings of helplessness and the habit of using food to deal with their uncomfortable feelings. The patient learns skills and strategies for problem solving, coping with stress, identifying feelings, and avoiding relapse. Patients are more successful when they have lower weights and do not abuse laxatives or diuretics.

Pharmacological treatment of BN in adolescents does not have research support, although in an open-label medication trial of adolescents with BN, fluoxetine at treatment dosages of 60 mg/day was

well tolerated and associated with improvement (Kotler et al. 2003). SSRIs may be useful for comorbid psychiatric disorders. Bupropion increases the risk of seizures and should not be used in these patients.

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Arlington, VA, American Psychiatric Association, 2013
- Arcelus J, Mitchell AJ, Wales J, Nielsen S: Mortality rates in patients with anorexia nervosa and other eating disorders: a meta-analysis of 36 studies. *Arch Gen Psychiatry* 68(7):724–731, 2011 21727255
- Eddy KT, Thomas JJ, Hastings E, et al: Prevalence of DSM-5 avoidant/restrictive food intake disorder in a pediatric gastroenterology healthcare network. *Int J Eat Disord* 48(5):464–470, 2015 25142784
- Fisher MM, Rosen DS, Ornstein RM, et al: Characteristics of avoidant/restrictive food intake disorder in children and adolescents: a “new disorder” in DSM-5. *J Adolesc Health* 55(1):49–52, 2014 24506978
- Kotler LA, Devlin MJ, Davies M, Walsh BT: An open trial of fluoxetine for adolescents with bulimia nervosa. *J Child Adolesc Psychopharmacol* 13(3):329–335, 2003 14642021
- Le Grange D: Elusive etiology of anorexia nervosa: finding answers in an integrative biopsychosocial approach. *J Am Acad Child Adolesc Psychiatry* 55(1):12–13, 2016 26703904
- Le Grange D, Lock J, Agras WS, et al: Randomized clinical trial of family-based treatment and cognitive-behavioral therapy for adolescent bulimia nervosa. *J Am Acad Child Adolesc Psychiatry* 54(11):886–94.e2, 2015 26506579
- Le Grange D, Hughes EK, Court A, et al: Randomized clinical trial of parent-focused treatment and family-based treatment for adolescent anorexia nervosa. *J Am Acad Child Adolesc Psychiatry* 55(8):683–692, 2016 27453082
- Lock J, Agras WS, Bryson S, Kraemer HC: A comparison of short- and long-term family therapy for adolescent anorexia nervosa. *J Am Acad Child Adolesc Psychiatry* 44(7):632–639, 2005 15968231
- Lock J, La Via MC; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter for the assessment and treatment of children and adolescents with eating disorders. *J Am Acad Child Adolesc Psychiatry* 54(5):412–425, 2015 25901778

- Mehler PS: Diagnosis and care of patients with anorexia nervosa in primary care settings. *Ann Intern Med* 134(11):1048–1059, 2001 11388818
- Mellin LM, Irwin CE Jr, Scully S: Prevalence of disordered eating in girls: a survey of middle-class children. *J Am Diet Assoc* 92(7):851–853, 1992 1624655
- Minuchin S, Rosman BL, Baker L: *Psychosomatic Families: Anorexia Nervosa in Context*. Cambridge, MA, Harvard University Press, 1978
- Mousa HM, Montgomery M, Alioto A: Adolescent rumination syndrome. *Curr Gastroenterol Rep* 16(8):398, 2014 25064317
- Norris ML, Spettigue WJ, Katzman DK: Update on eating disorders: current perspectives on avoidant/restrictive food intake disorder in children and youth. *Neuropsychiatr Dis Treat* 12:213–218, 2016 26855577
- Palla B, Litt IF: Medical complications of eating disorders in adolescents. *Pediatrics* 81(5):613–623, 1988 3162764
- Schmidt U, Lee S, Beecham J, et al: A randomized controlled trial of family therapy and cognitive behavior therapy guided self-care for adolescents with bulimia nervosa and related conditions. *Am J Psychiatry* 164:591–598, 2007 17403972
- Strandjord SE, Sieke EH, Richmond M, Rome ES: Avoidant/restrictive food intake disorder: illness and hospital course in patients hospitalized for nutritional insufficiency. *J Adolesc Health* 57(6):673–678, 2015 26422290

■ ADDITIONAL READING

- Eddy KT, Murray HB, Le Grange D: Eating and feeding disorders, in Dulcan's *Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 435–460
- Le Grange D, Lock J: *Treating Bulimia in Adolescents: A Family Based Approach*. New York, Guilford, 2007
- Lock J, La Via MC; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter for the assessment and treatment of children and adolescents with eating disorders. *J Am Acad Child Adolesc Psychiatry* 54(5):412–425, 2015
- Lock J, Le Grange D, Agras WS, et al: *Treatment Manual for Anorexia Nervosa: A Family Based Approach*, 2nd Edition. New York, Guilford, 2013

- Rasquin A, Di Lorenzo C, Forbes D, et al: Childhood functional gastrointestinal disorders: child/adolescent. *Gastroenterology* 130(5):1527–1537, 2006
- Rosen DS; American Academy of Pediatrics Committee on Adolescence: Identification and management of eating disorders in children and adolescents. *Pediatrics* 126(6):1240–1253, 2010

ELIMINATION DISORDERS

DSM-5 (American Psychiatric Association 2013) placed elimination disorders in their own chapter (formerly grouped, in DSM-IV, under “Disorders Usually First Diagnosed in Infancy, Childhood, or Adolescence”).

■ ENURESIS

Clinical Description

Enuresis is defined as repeated voiding of urine into bed or clothes, after a chronological age (or equivalent developmental level) of at least 5 years and occurring at least twice a week for at least 3 consecutive months or causing clinically significant distress or impairment in functioning (American Psychiatric Association 2013). Urinary incontinence in young children, and occasionally in older children after toilet training has been completed, may be a normal developmental phenomenon. Monosymptomatic enuresis (MSE) is nocturnal, does not involve bladder dysfunction, and includes no daytime symptoms. Children with MSE produce more urine at night than children without MSE and may have decreased functional bladder capacity. Children with nonmonosymptomatic enuresis (NMSE) have daytime (and also often nighttime) wetting. They may have urinary urgency, leaking, or frequency and may have bladder dysfunction, including emptying and storage abnormalities.

Nocturnal enuresis typically occurs 30 minutes to 3 hours after sleep onset but may occur at any time during the night. The child may sleep through the episode or be awakened by the moisture. Daytime

bladder control usually precedes nocturnal control by 1–2 years. Some clinicians do not diagnose nocturnal enuresis until the child is age 6 or 7 years. If bladder control has not yet been achieved, the enuresis is *primary*. In *secondary* enuresis, wetting reappears after a period of established urinary continence.

Epidemiology

Enuresis is seen in 10%–15% of 7-year-olds. The male predominance of primary enuresis decreases with age. After age 5 years, the prevalence of enuresis in both boys and girls spontaneously decreases by 15% per year. Between 3% and 9% of school-age girls experience daytime wetting (Mattsson and Gladh 2003). The general prevalence in older adolescents and adults is 1%. Few of the children with nocturnal enuresis also have diurnal enuresis or encopresis. In contrast, 50%–60% of patients with diurnal enuresis are likely to experience nocturnal incontinence. Enuresis is of the primary type in more than 85% of cases, a percentage that gradually decreases with age. Secondary enuresis is equally prevalent in boys and girls.

Etiology

Approximately 70% of children with enuresis (particularly boys) have a first-degree relative with enuresis. Studies of monozygotic and dizygotic twins show a strong genetic factor, although the mode of transmission is unclear. A “maturational” etiology is suggested in patients with primary enuresis who have small-volume voidings, short stature, low mean bone age, and delayed sexual maturation. Some patients have a relative inability to concentrate urine. Excessive fluid intake may contribute to the problem. Anatomical abnormalities of the urinary tract are not typical causes of enuresis. Constipation with or without encopresis is common in children with enuresis, and should be treated first as this often improves enuresis. Vaginal reflux of urine (incontinence within 10 minutes of voiding in girls) and “giggle incontinence” (solely associated with laughter between ages 10 and 20) are not considered enuresis. Enuresis is more common in patients with obstructive sleep apnea and with attention-deficit/

TABLE 11-1. Medical causes of urinary incontinence

Urinary tract infection
Urethritis—bubble bath, sexual abuse
Diabetes mellitus
Diabetes insipidus
Sickle cell trait
Seizure disorder
Sleep apnea
Neurogenic bladder—myelodysplasia, trauma, other neurological disorder
Congenital malformation of the genitourinary tract
Urinary obstruction—stone, pelvic mass
Constipation
Medication side effect

hyperactivity disorder (ADHD). See Table 11-1 for medical causes of enuresis.

Psychological factors are not generally thought to be causal of enuresis. 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. ADHD is often comorbid with enuresis, and both may represent central nervous system maturational delays (Shreeram et al. 2009). However, children with ADHD may wait until the last minute to urinate and then lose control on the way to the bathroom. Secondary enuresis may be related to stress, trauma, or psychosocial crisis. Enuresis that continues into adolescence is associated with higher rates of psychopathology.

Course and Prognosis

Primary enuresis has a high rate of spontaneous remission. Only about 1% of boys (and a smaller percentage of girls) still have this condition at age 18 years. Secondary enuresis usually begins between ages 5 and 8 years. Onset in adolescence may signify more psychiatric problems and less favorable outcome.

Complications include embarrassment, anger from and punishment by caregivers, teasing by peers, avoidance of overnight visits and camp, social withdrawal, and angry outbursts. The development of psychiatric disorders is higher in enuretic children than in the general population.

Evaluation and Differential Diagnosis

Initial medical evaluation is required to rule out medical causes (see Table 11-1). This includes a medical history (including questions about frequency, urgency, dysuria, and bowel habits), family history, and a physical examination. For MSE, the only laboratory study initially needed is a urinalysis, with appropriate follow-up if the results are abnormal. Evaluation of NMSE includes a urinalysis, urine culture, bladder ultrasound, and uroflow testing. Urodynamic studies are reserved for children who do not respond to treatment or who have physical exam findings suggestive of tethered spinal cord. A family history of enuresis is generally reassuring and implies that it will eventually be outgrown.

Psychiatric evaluation of the child and family includes assessment of associated psychiatric symptoms, recent psychosocial stressors, and family concern about and management of the symptoms. Developmental evaluation can identify the child who is not mature enough to achieve continence.

Treatment

Treatment of MSE begins with behavior modification. For younger children who wet only at night, the most useful strategy is to minimize symptoms by advising the parents not to punish or ridicule the child while awaiting maturation. Children can be taught to change their own beds to reduce negative parental reactions. Restricting fluids before bedtime, eliminating caffeinated beverages, and voiding at bedtime may be helpful.

For older children who are motivated to stop bed wetting, a monitoring and reward procedure (a chart with stars to be exchanged for rewards) may be effective. Daytime urinary continence may be

achieved rapidly with a behavioral program. A program of “bladder training” exercises may be helpful. Practice in delaying bladder emptying may increase bladder capacity. Interruption of the stream while urinating may strengthen sphincter muscles and improve awareness of bladder sensations. Instruction on adapted voiding posture and hygiene may be effective for urethrovaginal reflux (Mattsson and Gladh 2003).

If simple interventions are unsuccessful, a urine alarm is recommended. This device, an improvement on the “bell and pad,” has a high success rate of 75% at 6 months and 56% at 12 months. This rate compares favorably to rates using observation alone of 6% at 6 months and 16% at 12 months. If the alarm is set up to awaken the parents so they awaken the child, the relapse rate is low. A combination of the urine alarm, cleanliness training, retention control, and overlearning can stop bed wetting in two-thirds of children with primary enuresis (see Bennett 2005 in “Additional Reading”).

Although behavioral treatments consistently demonstrate higher success rates than medication, DDAVP (desmopressin) at bedtime can be helpful in the short term in patients who are resistant to behavioral interventions or who need a rapid result (e.g., for camp). The majority of patients relapse when medication is withdrawn (unless maturation has intervened). The oral form of DDAVP is preferred over the intranasal spray because of the risk of hyponatremia associated with the spray. Neither form should be given to a child who has fever, vomiting, diarrhea, or other condition that may result in sodium imbalance.

Psychotherapy is rarely useful in the treatment of enuresis. However, it may be indicated for the treatment of psychosocial sequelae of enuresis. Children who experience enuresis as a consequence of trauma or stress may also benefit from psychotherapeutic interventions. Associated disorders may require psychiatric treatment.

■ ENCOPRESIS

Clinical Description

Encopresis is defined as fecal soiling of clothes or excretion into inappropriate places that occurs at least once a month for at least

3 months. The child's chronological age (or equivalent developmental level) must be at least 4 years, when full bowel control is developmentally expected. Medical evaluation is necessary before labeling the disorder as "functional."

Encopresis is associated with constipation about 80% of the time. Children may have constipation or voluntary stool withholding with continuous leaking overflow incontinence, a problem that resolves with treatment of the constipation. This problem is classified as "retentive encopresis." Children with incontinence without constipation, or "functional nonretentive fecal incontinence," tend to produce formed stools in inappropriate contexts.

Epidemiology

Prevalence of encopresis gradually decreases with age and is reported in approximately 3% of 4-year-olds, 2% of 6-year-olds, and 1.6% of 10- to 11-year-olds. The problem is rare in adolescents. Among school-age children, males predominate in ratios from 2.5:1 to 6:1.

Etiology

Retentive encopresis may initially be triggered by painful defecation, inadequate or punitive toilet training, fear of using the school bathroom, or toilet-related fears. Once retention and constipation are initiated by emotional or medical factors, bowel physiology may maintain them independently. Parents may not recognize that the soiling is related to chronic constipation rather than reluctance to use the toilet. Pathophysiological mechanisms include altered colon motility and contraction patterns, obstruction, stretched and thinned colon walls (megacolon), or decreased sensation or perception secondary to a neurological disorder. Liquid stool leaks around the impaction and the child is unaware and unable to exert control.

The etiology of nonretentive encopresis is less well understood. It is often associated with younger age, positive family history, male gender, and life events such as birth of a sibling, parental discord, or other stressors. It may be a deliberate attempt by the child to effect change or communicate anger. Thirty to 50 percent of these children

will have a comorbid emotional or behavioral disorder (Koppen et al. 2016).

Course and Prognosis

The course and prognosis for encopresis are variable. Although the majority of children with encopresis will recover within a year of treatment, some studies indicate persistence of symptoms into adulthood. One study of constipation with encopresis found that among 16-year-olds, one-third were still symptomatic. A 10-year follow-up of children with nonretentive encopresis found that 49% of 12-year-olds were symptomatic, as were 15% of 18-year-olds (Rajindrajith et al. 2013). Psychiatric or medical comorbidity may be the primary determinant of prognosis.

Behavior problems are more common in the psychiatrically referred population than in those seen by pediatricians. In the psychiatric population, 25% of children with encopresis also have enuresis. Encopresis is also more common in children with ADHD.

Evaluation and Differential Diagnosis

A detailed history of bowel function, nature and pattern of soiling, attempts to train or treat, and bathroom habits and environment is needed. Physical examination should include an abdominal examination for evidence of a fecal mass, an anal examination for evidence of fecal material, a rectal digital examination for stool consistency, and a neurological examination with perianal sensation testing. The need for additional laboratory tests is based on the history and physical examination. A barium enema is not necessary in uncomplicated cases of encopresis but may be helpful in diagnosing *Hirschsprung's disease* (congenital megacolon). Urinalysis will detect a secondary urinary tract infection, which is common in girls with encopresis. *Medical causes of fecal incontinence* include thyroid disease, hypercalcemia, lactase deficiency, pseudo-obstruction, myelomeningocele (spina bifida), cerebral palsy with hypotonia, rectal stenosis, anal fissure, Hirschsprung's disease (which is usually associated, however, with large feces rather than incontinence), and anorectal trauma.

Psychiatric evaluation includes assessment for associated psychiatric disorders. Oppositional children may soil willfully. Children with ADHD do not plan ahead, so they may be caught by an urge to defecate when a bathroom is not available. They also may be prone to constipation (because of spending insufficient time toileting) or fecal soiling of underwear (due to careless hygiene). Anxious children may be 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 encopresis can be treated by a pediatrician, but more complex cases need psychological intervention. The pediatrician educates the child and parents about bowel function. For encopresis without constipation, a behavior-shaping program gives rewards first for just sitting on the toilet and later for moving the bowels appropriately. Manipulative soiling requires family behavioral treatment and attention to any exacerbating dynamic factors. For children with severe stool retention, impaction, and loss of bowel tone, initial bowel cleaning (most often with oral polyethylene glycol [PEG], or with enemas) followed by “retraining” the bowel (e.g., with stool softener or mineral oil, a high-fiber diet, development of a toileting routine, and use of a mild suppository if necessary) must be used in conjunction with the behavioral program (Rowan-Legg and Canadian Paediatric Society 2011). Repeated administration of enemas by parents is harmful to the parent–child relationship. Individual and family psychiatric interventions are indicated in resistant cases, in which the focus of treatment shifts to the associated psychiatric disorders.

■ REFERENCES

American Psychiatric Association: *Diagnostic and Statistical Manual of Mental Disorders*, 5th Edition. Arlington, VA, American Psychiatric Association, 2013

- Koppen IJ, von Gontard A, Chase J, et al: Management of functional nonretentive fecal incontinence in children: Recommendations from the International Children's Continence Society. *J Pediatr Urol* 12(1):56–64, 2016 26654481
- Mattsson S, Gladh G: Urethrovaginal reflux—a common cause of daytime incontinence in girls. *Pediatrics* 111(1):136–139, 2003 12509566
- Rajindrajith S, Devanarayana NM, Benninga MA: Review article: faecal incontinence in children: epidemiology, pathophysiology, clinical evaluation and management. *Aliment Pharmacol Ther* 37(1):37–48, 2013 23106105
- Rowan-Legg A; Canadian Paediatric Society, Community Paediatrics Committee: Managing functional constipation in children. *Paediatr Child Health* 16(10):661–670, 2011 23204909
- Shreeram S, He JP, Kalaydjian A, et al: Prevalence of enuresis and its association with attention-deficit/hyperactivity disorder among U.S. children: results from a nationally representative study. *J Am Acad Child Adolesc Psychiatry* 48(1):35–41, 2009 19096296

■ ADDITIONAL READING

- Bennett HJ: *Waking Up Dry: A Guide to Help Children Overcome Bedwetting*. Elk Grove Village, IL, American Academy of Pediatrics, 2005 (full text available on-line at <http://reader.aappublications.org/waking-up-dry/9?ajax>)
- Mikkelsen EJ: Elimination disorders, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 479–493
- Traisman ES: Enuresis: evaluation and treatment. *Pediatr Ann* 44(4):133–137, 2015
- Vande Walle J, Rittig S, Bauer S, et al; American Academy of Pediatrics; European Society for Paediatric Urology; European Society for Paediatric Nephrology; International Children's Continence Society: Practical consensus guidelines for the management of enuresis. *Eur J Pediatr* 171(6):971–983, 2012

SLEEP-WAKE DISORDERS

Nearly 25% of all children experience a sleep disorder at some time. Sleep complaints are related to the quality, timing, and amount of sleep. The common feature in all sleep disorders is the resultant daytime distress and impairment that results from disturbed sleep. The most common problems in children and adolescents are sleep talking, nightmares, nighttime waking, difficulty falling asleep, enuresis, bruxism, sleep rocking, restless legs syndrome, and night terrors. A self-report survey of adolescents found a 10% lifetime prevalence of difficulties with sleep initiation, sleep maintenance, and excessive daytime somnolence (Johnson et al. 2006). Children with chronic medical, neurodevelopmental, and psychiatric disorders are at greater risk of sleep disorders. An estimated 30%–80% of children with intellectual disability and 50%–70% of children with autism spectrum disorder (ASD) have associated sleep problems (Cortesi et al. 2010; Johnson 1996). Abnormalities in sleep are also symptoms of mood and anxiety disorders; thus, sleep assessment is critical for a thorough psychiatric evaluation. DSM-5 (American Psychiatric Association 2013) reclassified the sleep disorders into 10 major disorders or disorder groups.

■ EVALUATION OF SLEEP-RELATED COMPLAINTS

Sleep requirements vary by age. Preschool children typically require 11–12 hours of sleep, school-age children require an average of 10 hours of sleep, and adolescents require an average of 8.5–9.0

hours of sleep. Assessment of sleep disorders includes pediatric history and physical examination, particularly seeking evidence of obesity, enlarged tonsils, middle ear problems, a seizure disorder, allergies, asthma, and medication use. Although abnormal physical findings in a child with a sleep continuity disorder are relatively infrequent, the evaluation of the airway should include tonsillar size, nasal airflow, and facial abnormalities. A sleep history includes details of the patient's environment for sleeping and inquiries about whether the patient awakens screaming and confused, walks or talks while asleep, grinds his or her teeth, snores, experiences restless legs or nocturnal leg jerks, rocks or bangs his or her head at night, or has nighttime fears, frequent nightmares, and/or enuresis. A helpful mnemonic to assist in screening for sleep-related problems is BEARS, which stands for Bedtime, Excessive daytime sleepiness, Awakenings, Regularity, and Snoring (Mindell and Owens 2003). Sleep habits are reviewed and include the physical sleep environment, sleep schedules, intake of caffeinated beverages, level of activity, and evening exposure to screens (e.g., computer, phone, television, tablet, game devices). Children may resist bedtime or develop diurnal patterns that include difficulty awakening, daytime sleepiness, fatigue, and naps. A sleep log or diary is useful.

Developmental and psychiatric histories are necessary to assess possible psychiatric etiology or comorbidity. In addition, details of recent stressors, parental reactions to sleep-related problems, prescribed and over-the-counter medications, illicit substance use (in older children or adolescents), and the effects of any prior behavioral and pharmacological interventions are needed. When there is persistent nighttime waking or sleep loss or sleep apnea or nocturnal epilepsy is suspected, a sleep laboratory evaluation with polysomnography (PSG) may be indicated. PSG may include sleep electroencephalogram (EEG) and electrocardiogram as well as video monitoring and measurement of eye movements, electromyogram, airflow, and respiratory effort. A sleep-deprived EEG is useful in the evaluation of possible seizures. The Multiple Sleep Latency Test (MSLT) may be used to evaluate excessive daytime sleepiness (if a cause is not apparent). A drug screen may be needed in adolescents.

Insufficient sleep is particularly prevalent during adolescence, when late-night activities and early-morning academic schedules limit the available hours for sleep. Many adolescents tend toward delayed sleep-onset and awakening (phase shift), making it difficult to obtain adequate sleep.

■ INSOMNIA

Clinical Description

Patients with insomnia have persistent difficulty (compared with norms for age) initiating or maintaining sleep that results in impaired functioning. Bedtime resistance, disruptive nocturnal awakenings, and other behavioral sleep problems are most commonly seen in younger children, with a prevalence of 25%–50% (Owens et al. 2000). By age 6–9 months, most infants sleep through the night (or at least do not fuss when they awaken). Up to one-half of all infants, however, have irregular sleep patterns and occasional or persistent night waking throughout the first year of life. A study of children without sleep disorders found that 21% of 18- to 23-month-olds awakened during the night. Of the 24- to 29-month-olds, 31% took more than 30 minutes to fall asleep on more than three nights per week. Children ages 30–36 months are most likely to have difficulty settling for the night (16%) and express fears of the dark (24%) (Crowell et al. 1987). At age 5 years, more than 20% of children surveyed at least occasionally awakened during the night and called out to their parents. Parental reinforcement may have inadvertently contributed to this behavior. By school age, children are normally asleep for 95%–97% of the time they are in bed (Carskadon and Dement 1987).

Etiology

Sleep patterns in infants and toddlers may be affected by perinatal complications, colic, separation anxiety, the absence of a favorite transitional object, and parent–child interactions. The schedule of daytime naps for preschoolers can contribute to problems sleeping at

night. Young children with sleep continuity disorders have a higher rate of family stressors, including parental absence because of employment, family illness, and maternal depression.

Chronic insomnia is much more frequent in children with psychiatric disorders. It is often related to behavior or habit problems in settling for the night, especially in the child with attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder, or separation anxiety disorder. Or, insomnia may be a symptom of a mood disorder, ASD, or schizophrenia.

Insomnia may be secondary to the use of caffeine or prescribed (e.g., stimulants) or over-the-counter medication (e.g., decongestants) or substance use. Patients suffering from physical discomfort may have difficulty staying asleep.

Treatment

After any psychiatric or medical cause is addressed, in young children the first steps are to remove any factors that interfere with sleep and to enhance structure that encourages sleep (e.g., a bedtime routine, the use of a transitional object, or a night light for the child who is afraid of the dark). Behavior modification removes the secondary gain of parental attention at night and provides positive reinforcement for the child who stays quietly in his or her own room. Older children and adolescents may benefit from hypnosis or relaxation techniques. Consistency among all family members is critical to success.

In adolescents with insomnia, behavior therapy can disrupt the conditioned association between bedtime habits and anxiety regarding inability to sleep. Sleep hygiene is improved by using the bed only for sleeping, establishing a regular sleep schedule, avoiding naps, and removing all electronic media and communication devices from the bedroom. Cognitive-behavioral therapy in older children and adolescents has proven to be an effective intervention (Kuhn and Roane 2011).

Hypnotic medications are not recommended for chronic use. To date, there are no pharmacological agents approved by the U. S. Food and Drug Administration (FDA) for use in pediatric insomnia. Moreover, many children respond to sedatives with paradoxical ag-

itation. Melatonin has shown to be helpful in short-term use with relatively few adverse effects (see Chapter 17, “Psychopharmacology”). It may assist with circadian regulation and assist in falling asleep at bedtime.

■ NARCOLEPSY

Clinical Description and Evaluation

The classic tetrad of symptoms associated with narcolepsy includes daytime sleepiness, cataplexy, hypnagogic hallucinations, and sleep paralysis. Children and adolescents have variable presentations, with few experiencing all four symptoms concurrently. DSM-5 (American Psychiatric Association 2013) refined the definition of narcolepsy to include more precise parameters and specific test results. Narcolepsy is characterized by recurrent periods of an irrepressible need to sleep, lapsing into sleep, or napping occurring within the same day. This symptom must also be accompanied by either episodes of cataplexy (i.e., sudden bilateral loss of muscle tone with maintained consciousness precipitated by heightened emotional state; in children, it can present as spontaneous grimaces, jaw-opening episodes with tongue thrusting, or global hypotonia, without an emotional trigger), cerebrospinal fluid (CSF) hypocretin deficiency, or nocturnal PSG showing short rapid eye movement (REM) sleep latency (≤ 15 minutes) or MSLT showing shortened mean sleep latency with at least two sleep-onset REM periods. Narcolepsy is relatively rare, though the exact childhood prevalence is unknown. The prevalence of narcolepsy in adults is approximately 2–5 in 10,000.

In evaluation, because narcolepsy is relatively rare, other causes of daytime sleepiness should be considered. The most likely alternative diagnosis to narcolepsy is the normal increase in daytime sleep and reports of sleepiness in adolescence. Truly excessive daytime sleep may be an avoidance mechanism, even in the classroom, or secondary to insomnia 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 dis-

order or conversion disorder. Substance use or withdrawal should also be suspected when sleep continuity is disrupted. Typically, referral to a sleep medicine program is required to make the diagnosis by clinical examination, PSG, MSLT, and/or lumbar puncture.

Course and Prognosis

Onset of narcolepsy is typically in late adolescence or early adulthood. Sleep attacks and cataplexy may interfere significantly with schoolwork and peer relationships. Behavioral and emotional changes can develop early in the clinical presentation.

Treatment

Treatment of narcolepsy begins with educating the patient and family members about the nature and course of the disorder. A useful Web site is that of the Narcolepsy Network (www.narcolepsynetwork.org). A regular sleep schedule with scheduled naps is helpful. Stimulant drugs (methylphenidate, dextroamphetamine) are used to reduce sleep attacks, particularly while the child is at school. Modafinil (Provigil) is a nonstimulant wakefulness-promoting agent that is FDA approved for use in narcolepsy. Sodium oxybate (Xyrem) was shown to be effective and well tolerated in children and adolescents with excessive daytime sleepiness associated with narcolepsy (Aran et al. 2010). Other medications that can be useful in the treatment of cataplexy, sleep paralysis, and hypnagogic hallucinations include the tricyclic antidepressants (clomipramine, imipramine, protriptyline), the mixed-action antidepressants, and the selective serotonin reuptake inhibitors.

■ OBSTRUCTIVE SLEEP APNEA

Clinical Description

Obstructive sleep apnea (OSA) is a common and treatable sleep disorder. It is defined by episodes of complete or partial cessation of

airflow associated with oxygen desaturation. Symptoms of OSA include persistent snoring, witnessed apneas or gasping for air, restless sleep, and nocturnal diaphoresis. The prevalence of pediatric OSA is estimated to be 1%–4% (Brockmann et al. 2012). The prevalence is higher in children with neuromuscular and craniofacial abnormalities and genetic syndromes.

Etiology

OSA results from the loss of patency of the upper airway, caused by structural or neurological factors. With obstruction, respiratory efforts increase and greater muscle activity and arousal lead to relief of the obstruction. Causes of OSA include obesity, adenotonsillar hypertrophy, nocturnal asthma, lax upper airway structures, maxillofacial abnormalities, neuromuscular disease, Down syndrome, and hypothyroidism.

Course and Prognosis

The degree of adenotonsillar hypertrophy does not correlate with the severity of symptoms. PSG is required to make the diagnosis. Children with OSA may have medical complications, including pulmonary hypertension, systemic hypertension, right heart failure, failure to thrive, short stature, and enuresis. Greater awareness of the disorder has decreased the incidence of medical complications. Psychiatric and academic difficulties may include developmental delay, irritability, aggressiveness, distractibility, inattention, and hyperactivity.

Treatment

The treatment of OSA is most often surgical, typically an adenotonsillectomy, which is curative in the majority of cases. Continuous positive airway pressure (CPAP) is an option for children who have either failed the surgical intervention or who are not considered an appropriate surgical candidate.

■ CIRCADIAN RHYTHM SLEEP-WAKE DISORDERS

Circadian rhythm sleep-wake disorders result from disruption of the internal body rhythms that regulate sleep and wakefulness. There are six subtypes in DSM-5. Delayed sleep-phase syndrome (DSPS) (i.e., delay of sleep phase in relation to the light cycle) occurs following puberty in 5%–10% of adolescents. Adolescents with DSPS have excessive daytime sleepiness, which can impact academic performance, mood, attention, and relationships. Treatment for DSPS includes sleep hygiene, psychoeducation, and gradual advancement of sleep phase. Blue light therapy has shown efficacy in shifting sleep (Revell et al. 2012), as has bright light therapy with morning exposure. Melatonin can also be used to help advance the sleep phase. Chronotherapy, a behavioral technique in which bedtime is incrementally and systematically delayed, can be used for severe DSPS.

■ PARASOMNIAS

Parasomnias are more commonly seen in children, especially those with psychiatric or neurological disorders, than in adults. They can appear as early as age 2 years and typically resolve by adolescence. These disorders disrupt sleep with abnormalities of arousal, partial arousal, or sleep-stage transitions. The patient does not complain of insomnia or sleepiness. Parasomnias include non-rapid eye movement (NREM) sleep arousal disorders, nightmare disorder, and rapid eye movement (REM) sleep behavior disorder.

NREM Sleep Arousal Disorders

NREM sleep disorders are subdivided into sleepwalking type and sleep terror type.

Sleepwalking Type

Sleepwalking disorder is characterized by repeated episodes of arising from bed and engaging in motor activities while still asleep. Episodes,

which last a few minutes to a half hour, typically occur 1–3 hours after the child falls asleep, during Stage 3 and 4 delta (NREM) sleep. The prevalence of occasional sleepwalking is 15%. Frequent episodes occur in 1%–6% of individuals. The child or adolescent arises quietly and engages in perseverative, 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. Occasionally, the patient engages in inappropriate behavior, such as urinating in the closet. Young children tend to walk toward a light or sound. Older children may wake in an agitated state with garbled speech and a tendency to recoil when touched. Sleepwalking tends to be familial. Of patients who sleepwalk, 10%–20% have first-degree relatives with the disorder. Sleepwalking is usually seen between ages 4 and 12 years, with most cases remitting spontaneously by age 15 years. Parents should be advised to remove hazards in the environment. Children should maintain regular sleep schedules. Scheduled awakenings prior to the expected time of sleepwalking episode may be helpful. If sleepwalking is frequent or dangerous, benzodiazepines may be considered.

Sleep Terror Type

Episodes of sleep terror disorder typically occur during the first third of the night, in Stage 3 and 4 delta (NREM) sleep, and last 1–10 minutes. The child looks 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 may have brief recall of a feeling of terror or of dream fragments. The child rapidly returns to sleep when the episode is over and in the morning has no memory of the event. The parents are far more distressed than the child. The estimated prevalence of the full disorder

der in children is 1%–6%, and it is more common in boys than in girls. Sleep terror disorder is considered to be developmental and is not caused by a psychiatric disorder. Sleep terrors can be increased by sleep deprivation or anything that fragments sleep, including fever, illness, a full bladder, and certain medications. Patients (or parents) may experience symptoms due to cumulative sleep loss, if night terrors are frequent. Family history of sleep terror is common. Age at onset is typically between 3 and 12 years, with spontaneous resolution by adolescence. The number and frequency of episodes are highly variable. Consecutive episodes may be separated by days or weeks, but in rare instances they may occur on consecutive nights.

Parents should be educated about the disorder and reassured that the episodes do not indicate a psychiatric or neurological problem. The child's sleep schedule is monitored to provide a sufficient amount of time spent in bed. The patient's safety should be ensured by parents' erecting gates across stairs and locking doors and windows to prevent leaving the house. Waking the child before the usual time of the night terror may abort attacks. However, waking the child during the event should be avoided because it may exacerbate or prolong the episode. Medication such as a benzodiazepine is used only if the episodes are frequent, put the child in physical danger, severely disrupt the family, or interfere with daytime functioning.

Nightmare Disorder

Occasional normal frightening dreams 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. Although the child may rapidly fall asleep again, sometimes he or she cannot return to sleep and will ask to sleep with the parents. Frequency of nightmares typically waxes and wanes as the child develops. Among children ages 3–5 years, 10%–50% have recurrent nightmares that disturb their parents. Symptoms typically begin during preschool years and decrease in frequency with age. Occasionally, the disorder persists into adulthood. No psychiatric conditions are associated consistently with nightmare disorder in

children. Nightmares tend to increase with stress, sleep deprivation, fatigue, and change in sleep environment. Medications may increase nightmares. Reassurance is generally the best approach. The child should not be pressured to describe the nightmare but should be given an opportunity to talk about his or her fears. Sleep schedules should be normalized and sleep time increased if patients suffer from sleep deprivation. Children and adolescents with more persistent problems may require anxiety reduction techniques, such as relaxation, imagery combined with systematic desensitization, and dream reorganization. If nightmares are a symptom of posttraumatic stress disorder or another syndrome, the primary etiology should be addressed.

■ RESTLESS LEGS SYNDROME

Clinical Description

Restless legs syndrome (RLS) is defined as the urge to move the legs, usually accompanied by uncomfortable sensations in the legs. The urge begins or worsens during periods of rest, is worse in the evening or night, and is partially or totally relieved with movement. Developmental issues should be considered as children may not express the symptoms as “urges.” Prevalence rates range from 2% to 7%, depending on the frequency criterion. Females are more likely to have RLS, and the prevalence of RLS increases with age. The typical age at onset is young adulthood, but many adults with RLS reported having symptoms in late childhood or adolescence (American Psychiatric Association 2013).

Etiology

RLS can be seen in individuals with iron deficiency. RLS also has a strong genetic component, more common in individuals of European descent as compared with African or Asian descent. Disturbances in the central dopaminergic system have also been associated with RLS.

Treatment

Behavioral interventions include maintaining a regular sleep-wake schedule, avoiding sleep deprivation, reducing caffeine intake, avoiding tobacco and alcohol, and avoiding stimulating activities near bedtime. Pharmacological interventions include iron supplementation for children with iron deficiency. Dopaminergic agents have been shown to be safe and well tolerated in children (England et al. 2011). Gabapentin also has shown efficacy in pediatric RLS (Frenette 2011). Other medications that can be used in pediatric RLS include benzodiazepines, α -agonists, and carbamazepine.

■ PSYCHIATRIC DISORDERS AND SLEEP PROBLEMS

Children with ADHD often have sleep-related problems including difficulty settling down to sleep, delayed sleep onset, frequent nocturnal awakenings, restless sleep, and reduced total amount of sleep. OSA should be considered in children with inattention, hyperactivity, and snoring. Studies have conflicting results regarding whether adenotonsillectomy reduces symptoms of ADHD in patients with both OSA and ADHD. Many children with ADHD also have RLS and periodic limb movement disorder. Sleep complaints are included in symptom criteria for children with mood disorders. Sleep complaints can include impaired sleep initiation or sleep maintenance, hypersomnia, or insomnia. Sleep difficulties can be seen in many children with ASD, with the most frequent concerns being difficulty falling asleep, frequent nocturnal awakenings, early morning awakenings, irregular sleep-wake cycle, restless sleep, and symptoms consistent with parasomnias. Adolescents with substance use disorders often have disturbed sleep, including excessive daytime sleepiness and insomnia. Children with anxiety disorders, notably generalized anxiety disorder, separation anxiety disorder, and specific phobia, also commonly present with sleep-related concerns.

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Arlington, VA, American Psychiatric Association, 2013
- Aran A, Einen M, Lin L, et al: Clinical and therapeutic aspects of childhood narcolepsy-cataplexy: a retrospective study of 51 children. *Sleep* 33(11):1457–1464, 2010 21102987
- Brockmann PE, Urschitz MS, Schlaud M, Poets CF: Primary snoring in school-children: prevalence and neurocognitive impairments. *Sleep Breath* 16(1): 23–29, 2012 21240656
- Carskadon MA, Dement WC: Daytime sleepiness: quantification of a behavioral state. *Neurosci Biobehav Rev* 11(3):307–317, 1987 3684058
- Cortesi F, Giannotti F, Ivanenko A, Johnson K: Sleep in children with autistic spectrum disorder. *Sleep Med* 11(7):659–664, 2010 20605110
- Crowell J, Keener M, Ginsburg N, Anders T: Sleep habits in toddlers 18 to 36 months old. *J Am Acad Child Adolesc Psychiatry* 26(4):510–515, 1987 3654502
- England SJ, Picchietti DL, Couvadelli BV, et al: L-Dopa improves Restless Legs Syndrome and periodic limb movements in sleep but not Attention-Deficit-Hyperactivity Disorder in a double-blind trial in children. *Sleep Med* 12(5):471–477, 2011 21463967
- Frenette E: Restless legs syndrome in children: a review and update on pharmacological options. *Curr Pharm Des* 17(15):1436–1442, 2011 21476956
- Johnson CR: Sleep problems in children with mental retardation and autism. *Child Adolesc Psychiatr Clin N Am* 5(3):673–681, 1996
- Johnson EO, Roth T, Schultz L, Breslau N: Epidemiology of DSM-IV insomnia in adolescence: lifetime prevalence, chronicity, and an emergent gender difference. *Pediatrics* 117(2):e247–256, 2006
- Kuhn BR, Roane BM: Pediatric insomnia and behavioral interventions, in *Therapy in Sleep Medicine*. Edited by Barkoukis T, Matheson J, Ferber R, et al. Philadelphia, PA, Elsevier, 2011, pp 448–456
- Mindell JA, Owens JA: Sleep problems in pediatric practice: clinical issues for the pediatric nurse practitioner. *J Pediatr Health Care* 17(6):324–331, 2003 14610449
- Owens JA, Spirito A, McGuinn M, Nobile C: Sleep habits and sleep disturbance in elementary school-aged children. *J Dev Behav Pediatr* 21(1):27–36, 2000 10706346

Revell VL, Molina TA, Eastman CI: Human phase response curve to intermittent blue light using a commercially available device. *J Physiol* 590(19):4859–4868, 2012 22753544

■ ADDITIONAL READING

Ivanenko A, Johnson KP: Sleep disorders, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 495–519

GENDER DYSPHORIA

■ KEY CONCEPTS AND TERMINOLOGY

Sex refers to anatomical and genetic characteristics associated with being male or female. For most babies, a *natal sex* is assigned at birth based on appearance of the genitalia. *Gender identity* refers to the child or adolescent's own sense of self as a boy or a girl, which is typically established by age 3 years and consolidated by age 5 or 6 years. Social norms regarding *gender expression* or *gender role behavior* are substantially determined by the prevailing culture. Characteristics include mannerisms, gait, clothing, toys, play activities, and preferred playmates. *Gender nonconformity* (or *gender variance*) (i.e., gender role behavior that does not conform to culturally defined norms) and *gender discordance* (i.e., incongruence between anatomical sex and gender identity) may or may not go together in a particular individual. In general, girls are societally permitted more latitude in role behavior than boys are. In nonclinical populations of boys, cross-gender behavior is rare after age 6 years. *Gender dysphoria* refers to the distress experienced by some (but not all) persons with gender discordance. *Transsexual* describes individuals who identify as the "opposite" gender and who may seek social, medical, and/or surgical gender reassignment. As there has been increasing awareness of nonbinary and fluid identification and expression, *transgender* has become the preferred term for people with a gender identity that is discordant with their anatomical sex.

■ CLINICAL DESCRIPTION

Gender dysphoria was one of the most controversial sections in the development of DSM-5 (American Psychiatric Association 2013), as there have been rapid but divergent changes in societal attitudes toward conventional gender binary concepts, emotions, and behavior. Youth with gay, lesbian, or bisexual sexual orientation or gender nonconformity have a variety of developmental and social issues (see “Additional Reading”), but the only remaining related psychiatric diagnosis is gender dysphoria. DSM-5 renamed this syndrome (previously *gender identity disorder*) and placed it in its own chapter, rather than with identity disorders. Criteria for gender dysphoria differ for children and for adolescents and adults, but at all ages the lead criterion is “marked incongruence between one’s experienced/expressed gender and assigned gender, of at least 6 months’ duration” (American Psychiatric Association 2013, p. 452). In addition, at all ages DSM-5 requires that there be clinically significant distress or impairment in order to make the diagnosis. For child diagnosis, at least six criteria must be met (see Box 13–1), one of which must be Criterion A1 (i.e., a strong desire to be of the other gender or an insistence that one is the other gender). For adolescents, at least two of the eight criteria listed in Box 13–1 must be present.

Box 13–1 DSM-5 Diagnostic Criteria for Gender Dysphoria (Criterion A Only)

Gender Dysphoria in Children

- A. A marked incongruence between one’s experienced/expressed gender and assigned gender, of at least 6 months’ duration, as manifested by at least six of the following (one of which must be Criterion A1):
 - 1. A strong desire to be of the other gender or an insistence that one is the other gender (or some alternative gender different from one’s assigned gender).
 - 2. In boys (assigned gender), a strong preference for cross-dressing or simulating female attire; or in girls

(assigned gender), a strong preference for wearing only typical masculine clothing and a strong resistance to the wearing of typical feminine clothing.

3. A strong preference for cross-gender roles in make-believe play or fantasy play.
4. A strong preference for the toys, games, or activities stereotypically used or engaged in by the other gender.
5. A strong preference for playmates of the other gender.
6. In boys (assigned gender), a strong rejection of typically masculine toys, games, and activities and a strong avoidance of rough-and-tumble play; or in girls (assigned gender), a strong rejection of typically feminine toys, games, and activities.
7. A strong dislike of one's sexual anatomy.
8. A strong desire for the primary and/or secondary sex characteristics that match one's experienced gender.

Gender Dysphoria in Adolescents and Adults

- A. A marked incongruence between one's experienced/expressed gender and assigned gender, of at least 6 months' duration, as manifested by at least two of the following:
 1. A marked incongruence between one's experienced/expressed gender and primary and/or secondary sex characteristics (or in young adolescents, the anticipated secondary sex characteristics).
 2. A strong desire to be rid of one's primary and/or secondary sex characteristics because of a marked incongruence with one's experienced/expressed gender (or in young adolescents, a desire to prevent the development of the anticipated secondary sex characteristics).
 3. A strong desire for the primary and/or secondary sex characteristics of the other gender.
 4. A strong desire to be of the other gender (or some alternative gender different from one's assigned gender).

5. A strong desire to be treated as the other gender (or some alternative gender different from one's assigned gender).
 6. A strong conviction that one has the typical feelings and reactions of the other gender (or some alternative gender different from one's assigned gender).
-

■ EPIDEMIOLOGY

There are no epidemiological data on the prevalence of gender dysphoria in children or adolescents. As children, more boys than girls come to clinical attention, perhaps because of greater cultural permissiveness regarding gender roles and behavior for females than for males. The clinically referred adolescent population has a more even distribution of natal sex. As attention in lay media has increased, youth with gender dysphoria are presenting at younger ages than in the past.

■ ETIOLOGY

Various biological and psychological causative factors for gender dysphoria have been proposed, but no specific psychological, genetic, or hormonal factor has yet been identified. Some evidence suggests a contribution by prenatal hormones acting on the developing brain. Family characteristics—for example, lack of appropriate gender role modeling; parental wish for opposite-sex child; parent who treats child as opposite sex; same-sex parent who is absent, distant, or depressed; disturbance in mother–child relationship; mother who is dissatisfied with her own gender or gender role; and violence in the family—have been proposed as etiological factors in individual patients. However, many patients with gender dysphoria do not have these factors, and many children exposed to these dynamics do not develop gender dysphoria, so causal validity is highly unlikely. Children raised by transgender, lesbian, or gay

parents show no evidence of confusion about gender identity or role behavior.

■ COURSE AND PROGNOSIS

Peer relationships may be impaired. In general, children have rigid gender role expectations and do not welcome a child who does not conform to these. Boys with discordant gender role behavior are particularly likely to be teased and ostracized. Adolescents can be particularly cruel to peers who do not conform to conventional notions of gender role and physical, emotional, and/or social media bullying may result. The pervasive discomfort of the young patient with gender dysphoria leads to low self-esteem and often anxiety and/or depression, and interferes with school performance.

As a group, patients with gender dysphoria have levels of comorbid psychiatric disorders equal to those of other clinically referred patients, although some youngsters with gender dysphoria have few, if any, symptoms of other disorders.

Prospective studies find that of children with gender dysphoria, the majority “desist” in adolescence. As adults, they are more likely to identify as gay, lesbian, or bisexual than as heterosexual. Adolescents with gender dysphoria, particularly those with more pronounced and consistent identification as transgender, are likely to “persist” with this identification into adulthood.

■ EVALUATION AND DIFFERENTIAL DIAGNOSIS

Sensitive clinical evaluation is required. Older children or adolescents with gender dysphoria often learn from repeated negative feedback not to verbalize their wishes or beliefs about their gender and their anatomy. Children with gender dysphoria must be differentiated from those who simply do not meet the gender role expectations of their families or their culture. Children with gender dysphoria can be distinguished from “tomboys” or from boys who have more studious or musical interests (rather than rough-and-

tumble play and sports) by the rigidity of their avoidance of the expected gender role, their profound unhappiness with their physical gender, and their discomfort with or dislike of their sexual anatomy. It is important to establish the duration, consistency, and strength of cross-gender feelings and behavior, as well as peer relationships with and reactions to the patient. A complete mental health evaluation (see Chapter 2, “Evaluation and Treatment Planning”) is needed to establish the diagnosis and any comorbidity, as well as problematic family relationships resulting from or contributing to distress. In adolescents, specific inquiry is needed into suicidality, high-risk behaviors, and substance abuse. Pediatric evaluation is indicated to rule out a *physical or hormonal abnormality* resulting in a *disorder of sex development*.

■ TREATMENT

Approaches to treatment have changed dramatically and remain in a state of continuing evolution. International standards exist describing generally accepted approaches to treatment (Coleman et al. 2011). No randomized controlled trials exist. Interventions for prepubertal children may include psychoeducation (for both parents and child), supportive therapy, enhancement of coping strategies, encouragement of recreational and peer activities with which the child is comfortable, collaboration with school personnel, and treatment of any coexisting emotional, behavioral, or family problems. More recent approaches include social gender transition, including adoption of a preferred name and gender pronoun, and (at Tanner stage 2) hormonal suppression/delay of pubertal changes, for which consultation with a pediatric endocrinologist with special expertise is indicated. For adolescents, similar psychosocial approaches are clinically advised, and work with parents and schools is likely to be even more important than for younger patients. Support groups for youth and for parents may be helpful. Adolescents may be more insistent on social gender transition. Questions regarding decisions for and timing of cross-sex hormone therapy and/or surgical interventions require specialized consultation.

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Arlington, VA, American Psychiatric Association, 2013
- Coleman E, Bockting W, Botzer M, et al: Standards of care for the health of transsexual, transgender and gender nonconforming people, version 7. *Int J Transgenderism* 13:165–232, 2011, www.wpath.org

■ ADDITIONAL READING

- Adelson SL; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter on gay, lesbian, or bisexual sexual orientation, gender nonconformity, and gender discordance in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 51(9):957–974, 2012 22917211
- Leibowitz S, Chen D, Hidalgo MA: Gender dysphoria and nonconformity, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 585–602

DISRUPTIVE, IMPULSE-CONTROL, AND CONDUCT DISORDERS

DSM-5 (American Psychiatric Association 2013) placed oppositional defiant disorder and conduct disorder into a new chapter that also includes intermittent explosive disorder (very rarely used in youth) and antisocial personality disorder (limited to adults).

■ OPPOSITIONAL DEFIANT DISORDER

Clinical Description

Oppositional defiant disorder (ODD) is often considered the less severe, developmental precursor of conduct disorder (CD), although most children with ODD do not develop CD. Children with ODD demonstrate argumentative, disobedient, and defiant behavior, without serious violation of the rights of other people. These children are stubborn, negativistic, and provocative. Anger-related symptoms are typically directed at parents and teachers. A lesser degree of angry outbursts may be seen in peer relationships.

DSM-5 defines ODD as a pattern of angry/irritable mood, argumentative/defiant behavior, or vindictiveness lasting at least 6 months, during which at least four of the behavioral criteria are present at a frequency greater than that typical for the child's age and developmental level. The diagnostic symptom criteria are divided into three categories (see Table 14–1), whose prognostic implications differ.

The argumentative/defiant and vindictive symptoms more likely predict development of conduct disorder, whereas the angry-irritable mood symptoms more likely predict later mood and anxiety disorders (Stringaris and Goodman 2009).

Data from a community survey provide guidance in determining when a behavior occurs more frequently than expected for age (Angold and Costello 1996). DSM-5 symptom criteria and epidemiological suggested cutoff frequencies are listed in Table 14–1. DSM-5 has slightly different recommended frequencies: that symptoms occur “most days” for children under 5 years and at least once a week for children older than 5 years (American Psychiatric Association 2013, p. 462). For a diagnosis of ODD to be made, the symptoms must not occur exclusively during the course of a psychotic, substance use, or mood disorder and must cause distress (in patient or others) or impair functioning.

A crucial feature of ODD is the antagonistic stance 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. The child may experience “rational” interventions as continuing arguments. Disobedience can take the form of overt defiance and provocation or dawdling and procrastination, as well as “sneaky” behavior. Parents, and sometimes teachers, become exhausted, frustrated, and angry. As a result, discipline veers between being overly punitive and hopelessly lax. By the time of clinical referral, symptoms are the result of an interactive, negative spiral between parents and child.

Epidemiology

The changing criteria for ODD have prevented consistent prevalence estimates. DSM-5 reports prevalence ranging from 1% to 11%, with an average estimate of 3.3%, with equal or slightly higher rates in boys than girls. ODD is common in psychiatric clinics and in special education classrooms. It often occurs concurrently with attention-deficit/hyperactivity disorder (ADHD).

TABLE 14–1. **DSM-5 symptom criteria for oppositional defiant disorder and suggested epidemiological cutoff frequencies**

	Suggested cutoff frequency
Angry/irritable mood	
1. Often loses temper	Twice a week
2. Is often touchy or easily annoyed	Twice a week
3. Is often angry and resentful	Four times a week
Argumentative/defiant behavior	
4. Often argues with authority figures or, for children and adolescents, with adults	Twice a week
5. Often actively defies or refuses to comply with requests from authority figures or with rules	Twice a week
6. Often deliberately annoys others	Four times a week
7. Often blames others for his or her mistakes or misbehavior	Once in 3 months
Vindictiveness	
8. Has been spiteful or vindictive at least twice within the past 6 months	Once in 3 months

Source. American Psychiatric Association 2013; Angold and Costello 1996.

Etiology

Several psychosocial etiological models have been proposed with a common theme of reciprocal, negative interaction between parent and child resulting in inconsistent discipline, structure, and limit-setting that reinforces oppositional behavior in the child.

Marital problems are common in the parents of children with ODD, but it is difficult to distinguish a contributing factor from the effect of raising a difficult child. Genetic, neurobiological, and temperamental factors may also contribute. The features of ODD and difficult temperament (see Chapter 2, “Evaluation and Treatment Planning”) overlap significantly and may be hard to distinguish. Environmental factors such as poverty, family dysfunction, child

abuse, and parental mental illness have also been associated with an increased risk of ODD.

Course and Prognosis

Some children with ODD develop CD, but many do not. In one sample of 7- to 12-year-old clinic-referred boys with ODD, 44% developed CD over a 3-year period (Loeber et al. 1995). Risk factors for progression of ODD to CD include poverty and parental characteristics such as a mother who is young when her first child is born and parents who abuse substances, discipline children inconsistently, and supervise children inadequately. Risk factors in the child are low IQ, physical aggression, and resistance to parental discipline. Many children with ODD subsequently develop anxiety and mood disorders (Loeber et al. 2009).

Evaluation and Differential Diagnosis

Psychiatric evaluation of the child and family is needed to rule out alternative or comorbid disorders and to identify family and psychosocial contributing factors. The ODD symptoms are generally reported by parents and caregivers. The children may not view themselves as defiant or argumentative and often blame parents, authority figures, and peers. Symptoms are more prominent when the child is with familiar people.

Although these behaviors can be *normal* for children in phases during toddlerhood and adolescence, the 6-month duration criterion of ODD and specification that in children younger than 5 years the symptoms occur on “most days” ordinarily exclude these developmental phenomena. Some children are simply *temperamentally stubborn* but lack the pattern of more severe disturbance characteristic of ODD. Children with ODD are often annoying or spiteful but stop short of the pattern of serious behavior problems seen in *conduct disorder*. DSM-5 now allows for ODD and CD to be concurrently diagnosed if symptoms of both disorders are present. Severe *separation anxiety disorder*, *panic disorder*, or *obsessive-compulsive*

disorder may lead to temper tantrums and dramatic resistance, but in these disorders the problem behavior is restricted to the feared situations, and in most cases these children can verbalize specific triggers of anxiety.

A key to the diagnosis of ODD is its lifelong pattern. Discrete periods of irritability and resistance to adult direction may be secondary to *major affective episodes* (depression or hypomania), *psychosis*, or an *adjustment disorder*. The intentional and provocative noncompliance characteristic of ODD should be differentiated from the noncompliance resulting from impulsivity and inattention in *attention-deficit/hyperactivity disorder*, although both disorders are often present. If criteria for both ODD and ADHD are met, both are diagnosed.

Oppositional behavior that is restricted to the school context may be a result of *intellectual disability*, *borderline intelligence*, a *communication or other developmental disorder*, *academic difficulty secondary to a learning disorder*, or lack of training in cultural norms and expectations.

Treatment

Multimodal programs such as Fast Track can reduce progression to CD in young children identified as at-risk (Conduct Problems Prevention Research Group 2002). Parent management training in behavior modification techniques such as positive reinforcement, giving more effective commands, “time-out,” and token economies can reduce power struggles and modify oppositionality. Maximal improvement may result from combining the use of a social skills, problem-solving, and conflict management training group for the child with a behavior modification training group for parents (Webster-Stratton and Hammond 1997). Interventions have been developed and tested to use technology, including DVDs, online programs, and parent training using interactive software, to replace the therapist for treatment of disruptive behavior. Bibliotherapy (i.e., recommending books to parents) may be useful (see Appendix, “Resources for Parents”).

In children with coexisting ADHD, anxiety, or mood disorder, medication for the comorbid condition may reduce oppositional behavior and improve compliance.

■ CONDUCT DISORDER

Clinical Description

Children and adolescents with CD repeatedly violate important societal rules or the personal rights of others. Youth with CD who exhibit overt physical aggression may be differentiated from those who engage in covert, nonconfrontational behaviors (e.g., stealing and truancy). In addition, whether the aggressive behavior is “predatory” (goal oriented, planned) or “affective” (impulsive, reactive) may have implications for etiology and treatment.

DSM-5 specifies three types of CD: *childhood onset*, in which at least one criterion is met before age 10 years; *adolescent onset*; and *unspecified onset*. Longitudinal studies (Langbehn et al. 1998) have found that childhood onset is associated with male predominance, increased physical aggression, impaired peer relationships, and comorbid ADHD. A typical course for this subtype starts with ODD in early childhood, which develops into full-blown CD by puberty, followed by a risk of persistent CD and development of adult antisocial personality disorder. In contrast, individuals with adolescent onset typically have few symptoms before puberty, are less likely to be aggressive, and are more likely to be female than those in the early-onset group. Most of those with adolescent onset have friends in the context of a gang or other delinquent peer group. The prognosis for cessation of conduct problems is better if onset is in adolescence; however, course and outcome may be influenced by the nature of the delinquent group and the availability of alternative social supports.

Long-term consequences of conduct disorder include loss of interest in school, school failure and dropout, and eventual unemployment. Youth with CD are at increased medical risk for early pregnancy; sexually transmitted diseases; physical injury from fighting, accidents, rape, or murder; and the sequelae of smoking, drink-

ing, and drug abuse. Rates of suicidal ideation and behavior are increased, especially in the presence of substance abuse.

DSM-5 introduced to the conduct disorder diagnosis a specifier, “with limited prosocial emotions,” that is meant to capture the presence of callous-unemotional (CU) traits. The CU traits include a lack of empathy, lack of remorse, shallow affect, manipulativeness, deceitfulness, and grandiosity. These traits have demonstrated significant heritability but are also influenced by environmental factors. Negative, harsh, inconsistent parenting and poor parent-child communication have been associated with elevated levels of CU traits in children and adolescents. Children with high levels of CU traits often exhibit a fearless temperament and are less responsive to punishment for negative behavior. They seem to respond better to warm nurturing parenting styles with positive reinforcement (Kimonis and Frick 2010; Loeber et al. 2009). The presence of CU traits is associated with a more severe and persistent course of conduct disorder and increases the risk of progression to antisocial personality disorder and psychopathy. For children with CU traits, treatment with stimulant medication has also demonstrated some efficacy in reducing conduct problems.

Despite heterogeneity among youth with CD, certain psychological characteristics are common (Table 14–2).

Epidemiology

DSM-5 cites prevalence estimates of CD among children and adolescents in the United States ranging from 2% to more than 10%. Although the prevalence of CD in girls has increased, boys still outnumber girls by 3–4:1. Less is known about CD in girls than in boys. Boys commit far more violent crimes than girls do (8:1). When self-report data are used, the overall prevalence of misconduct and delinquent behaviors increases, and the male predominance for crime declines to about 2:1. Cultural attitudes toward gender, race, and class may affect the relative likelihood of a youth being identified as having CD. CD is frequent among youth referred to outpatient psychiatric services.

TABLE 14-2. **Common psychological characteristics of children and adolescents with conduct disorder**

Attention deficits, low frustration tolerance
Impulsivity, recklessness
Learning disorders, especially in reading
Negative mood
Sullenness
Irritability
Volatile anger
Low self-esteem
Impaired cognitions
Distortions of size and time awareness
Lack of or distorted connection between prior events and consequences
Limited ability to generate, evaluate, and implement alternative problem-solving strategies
Emotional deficits
Minimization of fear and sadness, exaggeration of anger
Lack of empathy
Lack of guilt
Impaired interpersonal relations
Suspiciousness, with cognitive distortions
Attributional bias: misperception of others' actions as hostile
Preference for nonverbal, action-oriented, aggressive solutions to problems
Callousness

Comorbidity

Comorbid psychiatric or neurological disorders are common in association with CD and contribute to its severity and chronicity. ADHD occurs in as many as half of children with CD in community surveys. In psychiatric clinical programs, CD without ADHD is rare. Posttraumatic stress disorder (PTSD) and dissociative disorders are often reported, especially by incarcerated delinquent youth. Hypervigilance, irritability, and flashbacks may contribute to aggression when youth feel threatened. Learning disorders (especially reading disorder and expressive language disorder) are common. Depres-

sion or bipolar disorder may be seen. Although it seems counter-intuitive, anxiety and mood disorders are found in many youth with CD, especially girls, with increased rates after puberty. Substance use is often present and can aggravate impulsivity, risk taking, aggression, suicidality, and school failure.

Etiology

Many factors have been identified as contributing to the development of CD, which is a heterogeneous disorder. Identifying the contributing factors for the individual patient is important in planning treatment. Current views emphasize an interaction among socioeconomic, cultural, family dynamic, temperamental, genetic, neurobiological, and psychiatric factors to explain the development and persistence of CD and its pattern of transmission from one generation to the next.

Data from a study of male twins suggest that, compared with adult antisocial behavior, juvenile conduct problems are more strongly related to environmental factors and less strongly related to genetic factors (Lyons et al. 1995). The interaction of inadequate and often abusive parenting with characteristics intrinsic to the child results in noncompliant and aggressive behaviors and deficient academic and social skills. Temperamental characteristics such as negative emotions, intense reactions, and inflexibility are associated with a higher risk of CD. Multiple studies have found that birth complications combined with early maternal rejection (i.e., unwanted pregnancy, attempt to abort fetus, and placement outside the home before age 1 year), poor parenting, and parental mental illness increase the likelihood of violent criminal behavior (Raine 2002). Patterson and colleagues (1989) emphasized the importance of a parent-child negative spiral of ever-increasing aversive and coercive behaviors. In effect, these children train their parents to use harsh but inconsistent discipline, and the parents train the children in noncompliant, defiant, and antisocial behaviors. Although less is known about girls with CD, some data suggest that their families are even more dysfunctional than those of boys with CD. Rejection

by peers and school failure encourage affiliation with similarly troubled peers. One naturalistic study found that approximately one-third of prepubertal children with depressive mood disorders developed a DSM-III diagnosis of CD by age 19 years (Kovacs et al. 1988).

Although multiple risk factors appear to have a cumulative effect, most children with risk factors do not develop CD. Divorce does not appear to be a major risk factor; family discord rather than separation appears to mediate the risk for CD. Protective factors are not well understood, but an easy or behaviorally inhibited temperament, areas of competence, adequate supervision at home, or a good relationship with one parent or another adult can reduce an at-risk child's chance of developing CD. Associating with nondelinquent peers and attending a school with a positive environment also offer some protection.

Course and Prognosis

The first signs of behavior problems—aggression, impulsivity, and noncompliance—may be seen as early as age 4 years. Several studies have found that the combination of aggression and shyness in first grade predicts adolescent delinquency and substance abuse. Symptoms tend to emerge in a predictable developmental sequence, with milder behaviors followed by more severe ones. Progression may stop at any stage. Early onset; greater frequency, number, and variety of conduct symptoms; and comorbid ADHD are associated with more severe and prolonged CD (Loeber et al. 2000).

CD remits in many youth, but some lead lives of delinquency or develop antisocial personality disorder. Low IQ score and parental antisocial personality disorder predict persistence of CD (Loeber et al. 1995). A relatively small number of chronic offenders account for most juvenile crime. Recidivists are more likely to have early onset, poor school grades, and low socioeconomic status. In a 7-year follow-up of clinic-referred boys ages 7–12 years with CD, the majority showed fluctuating or increasing CD behaviors. At baseline, the minority with a more positive outcome were more likely to have

lower severity of CD, fewer symptoms of ADHD, higher verbal IQ, greater family socioeconomic advantage, and biological parents who were not antisocial. Treatment or incarceration did not account for improvement (Lahey et al. 2002).

Despite the high risk for major psychiatric symptoms, substance abuse, functional impairment, and incarceration, many children with CD achieve a favorable adult adjustment. More adaptive social skills, more positive peer experiences, and adolescent onset predict a better long-term outcome. Innovative early childhood programs have been able to reduce delinquent outcomes and improve academic achievement in children at risk for CD, but successful model programs are rarely replicated or disseminated.

Evaluation

Youth with more severe and complex forms of CD benefit from a comprehensive biopsychosocial evaluation by a multidisciplinary team that might include a child and adolescent psychiatrist, a clinical psychologist, a social worker, a pediatrician or specialist in adolescent medicine, a neurologist, an educational diagnostician and school consultant, a speech and language pathologist, occupational and recreational therapists, a legal advisor, and a case manager or probation officer.

Conventional diagnostic interviews may be difficult, because many youth with CD are uncomfortable or hostile when talking with adults in roles of authority and some have a limited ability to express themselves verbally and to think abstractly. Efforts to establish rapport and careful questioning are essential to identify comorbid psychiatric disorders or neurological impairment. Sources of information in addition to the patient are essential, in part because these youth often underreport criminal behaviors. Compared with parents, teenagers generally report more of their covert behaviors, such as lying, stealing, vandalism, and fire setting, whereas parents are more likely to report their child's overt behaviors, such as aggression. However, parents who are trying to shield their child from legal consequences may not disclose criminal behaviors.

Medical evaluation (especially in adolescents) should consider pregnancy, sexually transmitted diseases, and hepatitis, as well as any untreated medical disorder and a urine screen for drugs. Evaluation of neurological history is needed because of the frequency of head trauma (both contributing to and resulting from the CD) and seizures. Cognitive and educational assessment are indicated because of the frequent association with specific learning disorders and borderline intellectual functioning or intellectual disability. In addition, truancy, comorbid ADHD, and lack of socialization to the value and culture of school may contribute to educational deficits.

Differential Diagnosis

CD is a heterogeneous descriptive diagnosis that is made if the symptoms meet the behavioral criteria. Because virtually any psychiatric disorder can present with disturbance of conduct, the clinician must assess the full range of psychiatric diagnoses, cognitive abilities, language and speech functioning, social competence and relationships, and family functioning. During an episode of depression or mania in *bipolar disorder*, irritability and impaired judgment can lead to behavior problems that can be distinguished from primary CD by their time course and associated mood symptoms. *Psychosis* in children and adolescents may result in behaviors consistent with CD. Differential diagnosis also includes *intermittent explosive disorder* and a spectrum of less severe disorders: *ODD*, *adjustment disorder with disturbance of conduct*, and child or adolescent *antisocial behavior* (a DSM-5 V code), as well as typical mischief.

Treatment

Despite the cost of CD to individuals, families, and society, few treatments with proven efficacy are available. The patient is rarely motivated to change, and the family and social environments may lack the necessary resources. Early-onset CD becomes increasingly resistant to treatment as the child enters adolescence; therefore, early intervention with young children is crucial. Treatment may take a variety of forms: family interventions, social support, behavior mod-

ification, psychopharmacology, or legal sanctions. The treatment setting may be the home; a school, clinic, hospital, or residential treatment program; or a specialized delinquency program. In complex cases of CD, youth often need (but rarely receive) lengthy multimodal therapeutic interventions. The environment in which the patient is living or to which he or she will return must be considered. Comorbid conditions also require attention.

A containment structure and effective limit setting must be established quickly to provide a safe and stable environment for treatment. Limit setting at home may be compromised by parental conflict, parental absence, inconsistent discipline, vague or low expectations for appropriate behavior, or parental depression, substance abuse, or other psychiatric illness. Creating or reinforcing limits for the child with CD may require counseling of parents, treatment of parents' psychiatric problems, increased supervision at home, surveillance at school, and/or use of legal mechanisms. Guardianship, hearings before judges, supervision by parole officers, or brief incarceration may be required for effective limit setting and communicating the significance of behavioral violations. Families may need concrete assistance with income, housing, legal matters, or medical care. If there are significant comorbid psychiatric disorders, hospitalizing the youth briefly may be useful for containment and intensive evaluation, perhaps including a trial of medication. More stringent criteria for "medical necessity" have essentially eliminated hospitalization for pure CD, in part because hospitalization is not effective as definitive treatment.

Psychotherapeutic Interventions

Several treatments based on behavioral and family systems principles have shown efficacy, when youth and families can be motivated to participate in and complete treatment programs. In many cases, however, even with treatment, the problem behavior remains outside the normal range.

Parent management training (PMT) and functional family therapy (FFT) can be helpful for relatively motivated and intact families.

Principles of behavior modification and family systems theory are used to improve communication and negotiation skills, encourage positive reinforcement, correct dysfunctional parent–child interaction patterns, and promote more effective and less damaging methods of discipline. Younger children are more likely than adolescents to benefit from parent training interventions. Multisystemic therapy (MST) (see the book by Henggeler et al. in “Additional Reading”), a comprehensive treatment combining home-based systemic family therapy with behavior modification and direct intervention in the youth’s social system, is an effective treatment of delinquency in youth, even those from chaotic, multiproblem families. It improves adjustment of patients and family members and reduces future criminal behavior. The therapist empowers parents; enlists social service agencies; and actively reaches out into the home, school, and neighborhood. Individual supportive therapy or pharmacotherapy for the identified patient or a family member is added when necessary. For MST to be implemented with fidelity to the model, program funding from government or foundation sources is typically required.

Group therapy, particularly in residential treatment or therapeutic group homes, uses peer pressure to promote positive change and to improve socialization skills. Caution is needed, because treating antisocial youth together in groups can lead to contagion and worsening of problem behaviors. Insight-oriented individual psychotherapy is not useful.

Pharmacotherapy

There is no medication treatment for CD, but treatment of comorbid disorder(s) can be useful (see Chapter 17, “Psychopharmacology”). In patients with coexisting ADHD, stimulants can reduce impulsive conduct symptoms, verbal and physical aggression, hyperactivity, and inattention.

CD that is secondary to major depression may remit when the depression is successfully treated. Lithium may be considered in the treatment of severe impulsive aggression, especially when it is accompanied by explosive, irritable, labile affect. In a well-designed con-

trolled study, lithium was shown to be equal or superior to haloperidol in reducing aggression, hostility, and tantrums and to have fewer side effects (Campbell et al. 1995). Lithium may be the first pharmacological choice if the patient with CD has a family history of bipolar disorder.

Patients who have severe impulsive aggression with emotional lability and irritability, an abnormal electroencephalogram, a strong clinical suggestion of epileptic phenomena, or nonresponse to lithium may benefit from a trial of carbamazepine or valproate. Antipsychotic medications may reduce aggression that is secondary to psychosis. Even in nonpsychotic, severely aggressive children, atypical antipsychotics such as risperidone may reduce aggression, hostility, negativism, and explosiveness, although the side-effect profile (cognitive dulling, weight gain, metabolic syndrome, and risk of tardive dyskinesia) may be problematic. Propranolol, a β -adrenergic blocker, may be useful in patients with otherwise uncontrollable rage reactions and impulsive aggression, especially in those with evidence of organicity.

School and Juvenile Justice Interventions

School interventions include special attention to behavior control; individualized educational programming; vocational training; and remediation of language, speech, and other specific learning disorders. Despite their mainstream popularity, “boot camps” are not effective in reducing future crimes committed by juvenile delinquents (Henggeler and Schoenwald 1994).

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Angold A, Costello EJ: Toward establishing an empirical basis for the diagnosis of oppositional defiant disorder. *J Am Acad Child Adolesc Psychiatry* 35(9):1205–1212, 1996 8824064

- Campbell M, Adams PB, Small AM, et al: Lithium in hospitalized aggressive children with conduct disorder: a double-blind and placebo-controlled study. *J Am Acad Child Adolesc Psychiatry* 34(4):445–453, 1995 7751258
- Conduct Problems Prevention Research Group: Evaluation of the first 3 years of the Fast Track prevention trial with children at high risk for adolescent conduct problems. *J Abnorm Child Psychol* 30(1):19–35, 2002 11930969
- Henggeler SW, Schoenwald SK: Boot camps for juvenile offenders: just say no. *J Child Fam Stud* 3:243–248, 1994
- Kimonis ER, Frick PJ: Oppositional defiant disorder and conduct disorder grown-up. *J Dev Behav Pediatr* 31(3):244–254, 2010 20410703
- Kovacs M, Paulauskas S, Gatsonis C, Richards C: Depressive disorders in childhood, III: a longitudinal study of comorbidity with and risk for conduct disorders. *J Affect Disord* 15(3):205–217, 1988 2975293
- Lahey BB, Loeber R, Burke J, Rathouz PJ: Adolescent outcomes of childhood conduct disorder among clinic-referred boys: predictors of improvement. *J Abnorm Child Psychol* 30(4):333–348, 2002 12108765
- Langbehn DR, Cadoret RJ, Yates WR, et al: Distinct contributions of conduct and oppositional defiant symptoms to adult antisocial behavior: evidence from an adoption study. *Arch Gen Psychiatry* 55(9):821–829, 1998 9736009
- Loeber R, Green SM, Keenan K, Lahey BB: Which boys will fare worse? Early predictors of the onset of conduct disorder in a six-year longitudinal study. *J Am Acad Child Adolesc Psychiatry* 34(4):499–509, 1995 7751264
- Loeber R, Burke JD, Lahey BB, et al: Oppositional defiant and conduct disorder: a review of the past 10 years, part I. *J Am Acad Child Adolesc Psychiatry* 39(12):1468–1484, 2000 11128323
- Loeber R, Burke J, Pardini DA: Perspectives on oppositional defiant disorder, conduct disorder, and psychopathic features. *J Child Psychol Psychiatry* 50(1-2):133–142, 2009 19220596
- Lyons MJ, True WR, Eisen SA, et al: Differential heritability of adult and juvenile antisocial traits. *Arch Gen Psychiatry* 52(11):906–915, 1995 7487339
- Patterson GR, DeBaryshe BD, Ramsey E: A developmental perspective on antisocial behavior. *Am Psychol* 44(2):329–335, 1989 2653143
- Raine A: Biosocial studies of antisocial and violent behavior in children and adults: a review. *J Abnorm Child Psychol* 30(4):311–326, 2002 12108763

- Stringaris A, Goodman R: Longitudinal outcome of youth oppositionality: irritable, headstrong, and hurtful behaviors have distinctive predictions. *J Am Acad Child Adolesc Psychiatry* 48(4):404–412, 2009 19318881
- Webster-Stratton C, Hammond M: Treating children with early-onset conduct problems: a comparison of child and parent training interventions. *J Consult Clin Psychol* 65(1):93–109, 1997 9103739

■ ADDITIONAL READING

- Henggeler SW, Schoenwald SK, Borduin CM, et al: *Multisystemic Therapy for Antisocial Behavior in Children and Adolescents*, 2nd Edition. New York, Guilford, 2009
- Newcorn JH, Ivanov I, Chacko A, Javdani S: Aggression and violence, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 603–620
- Thomas CR: Oppositional defiant disorder and conduct disorder, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 195–217

SUBSTANCE-RELATED AND ADDICTIVE DISORDERS

■ CLINICAL DESCRIPTION

DSM-5 (American Psychiatric Association 2013) no longer includes separate diagnoses for substance abuse and dependence. A single list of criteria is provided for substance use disorder (SUD) (Box 15–1), with the type of substance specified. DSM-5 deleted the criterion of recurrent legal problems and added craving or strong desire to use. The threshold for making the diagnosis of substance use disorder is two criteria met, along with impairment or distress. DSM-5 also includes for each substance, where applicable, criteria for the diagnoses of intoxication and withdrawal. The continuum of adolescent substance use ranges from nonuse (abstinence), through experimental and casual use, to abuse and dependence. The line between use and abuse is crossed more easily by young persons than by adults, and some experts recommend that any use of alcohol or illicit drugs by a person under the legal drinking age be called abuse. Physical dependence is rare in adolescents. Gambling disorder is a new diagnosis in this chapter of DSM-5.

Box 15–1 DSM-5 symptom criteria for substance use disorder

1. Often taken in larger amounts or over a longer period than was intended
2. Persistent desire or unsuccessful efforts to cut down or control use

3. A great deal of time spent in activities necessary to obtain the substance
 4. Craving, or a strong desire or urge to use the substance
 5. Recurrent use resulting in failure to fulfill major role obligations
 6. Continued use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance
 7. Important activities given up or reduced because of the substance use
 8. Recurrent use in situations in which it is physically hazardous
 9. Use continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance
 10. Tolerance, as defined by either:
 - a. Need for markedly increased amounts to achieve intoxication or desired effect
 - b. Markedly diminished effect with continued use of the same amount
 11. Withdrawal, as manifested by either:
 - a. Characteristic withdrawal symptoms
 - b. Taking of a substance to relieve or avoid withdrawal symptoms
-

■ EPIDEMIOLOGY

Three national surveys provide regularly updated data on substance use in youth. The National Institute on Drug Abuse (NIDA)–sponsored Monitoring the Future (MTF) project is an annual in-school survey of nationally representative samples of approximately 50,000 8th-, 10th-, and 12th-grade students (<http://monitoringthefuture.org>). The reported rates are likely underestimates, because many of the heaviest

drug users drop out of school or are likely to be absent when surveys are taken. The National Survey on Drug Use and Health (NSDUH), conducted by the Substance Abuse and Mental Health Services Administration (SAMHSA), uses an interactive, computer-based questionnaire to provide annual reports and detailed tables on the prevalence, patterns, and consequences of drug and alcohol use and abuse in the general U.S. civilian noninstitutionalized population ages 12 years and older (<https://www.samhsa.gov/samhsa-data-outcomes-quality/major-data-collections/reports-detailed-tables-2015-NSDUH>). Every 2 years the Centers for Disease Control and Prevention (CDC) conduct school-based surveys (<https://www.cdc.gov/healthyyouth/data/yrbs/index.htm>). This Youth Risk Behavior Surveillance System (YRBSS) monitors health-risk behaviors in 9th- through 12th-grade students that contribute to the leading causes of death and disability among youth and adults, including behaviors that contribute to unintentional injuries and violence; sexual behaviors related to unintended pregnancy and sexually transmitted diseases, including HIV infection; alcohol and other drug use; and tobacco use.

The 2015 MTF found that teen smoking (7% past-month use) and alcohol (22.1% past-month use) decreased from the prior year, but use of marijuana (3.4% past-month use) and of e-cigarettes remained constant. Of concern is that among 12th graders, daily marijuana use (6%) exceeded daily tobacco use. The perception by 12th graders that marijuana use is risky continues to decline. Newer concerns are hookah water pipes, small cigars, and e-cigarettes, which are raising concern that they may increase rates of nicotine dependence, introducing “vaping” to teens, who then progress to tobacco use. MTF found more than twice as many 8th and 10th graders using e-cigarettes as were using conventional cigarettes. However, more than half reported vaping with only “flavoring,” not nicotine (Miech et al. 2016).

YRBSS 2015 data show a continuing decrease (since 1991) in rates of “ever tried” cigarette smoking (32.3%), currently smoking cigarettes (10.8%), and daily smoking cigarettes (2.3%). Current use of smokeless tobacco remains relatively steady at 7.3%. An elec-

tronic vapor product was reported to have been used at least once by 45% of surveyed high school youth. Rates of alcohol use have decreased since 1991, but 2015 rates are essentially unchanged from 2013. At least one drink of alcohol on at least one day in the past 30 days was reported by 32.8% of respondents. Use of a nonprescribed prescription drug (including opiates, stimulants, and benzodiazepines) was reported by 17%. Rates of cocaine and hallucinogen use have decreased since 2001 but have been stable since 2013. Use of inhalants and ecstasy continues to slowly decline. The rate of heroin use (“ever used” 2.1%) has remained between 2% and 3% since 2005 but is lower than in 2003 (3.3%).

There are no studies of prevalence of SUDs as defined by DSM-5. New or unconventional compounds for drug use appear frequently, including various herbs as well as synthetic drugs (e.g., bath salts, cannabinoids, fentanyl). Often these compounds are not detected by standard drug screening tests. All substances obtained illegally may have possible dangerous contaminants. A particularly deadly example is fentanyl sold as heroin.

■ COMORBIDITY

Adolescents often present with multiple SUD diagnoses. Virtually all adolescents referred for treatment of substance use have additional disorders, including attention-deficit/hyperactivity disorder (ADHD), oppositional defiant disorder (ODD), conduct disorder (CD), depression, anxiety disorders, posttraumatic stress disorder (PTSD), psychosis, and specific learning disorders. Virtually any psychiatric disorder may occur in association with substance use as a cause, an effect, or a correlate. Psychiatric disorders may predate substance abuse or be secondary to pharmacological or situational effects of drug use. In a longitudinal school-based study of adolescents, major depression and heavy smoking each increased the prospective risk for the other. This association does not appear to be due to shared risk factors (Windle and Windle 2001). The presence of ADHD, especially when accompanied by ODD or CD, is associated with early onset of substance abuse.

■ ETIOLOGY

Children of persons with substance use disorders appear to be particularly vulnerable to adolescent drug use, likely resulting from a combination of genetic and family dynamic factors with learned attitudes toward substance use (Table 15–1). Genetic contributions to alcoholism are strongest in males. Peer influence mediates avoidance of drugs, as well as both initiation and maintenance of substance 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. Among youth, determinants of use are often specific to each drug, related to some extent to the perceived risks and benefits of the substance.

TABLE 15–1. Risk factors associated with serious substance abuse in adolescence

Early childhood trauma, including history of physical or sexual abuse
Rebelliousness
Aggression
Impulsivity
Low self-esteem
Elementary school underachievement
Failure to value education
Absence of strong religious convictions
Experimentation with drugs before age 15 years
Relationships with peers who have behavior problems and use drugs
Alienation from parents
Family lacking in clear discipline, praise, and positive relationships
Family history of substance abuse

■ COURSE AND PROGNOSIS

Adolescence is the critical period for initiation of drug abuse. Onset is rare in adulthood, except for the abuse of prescription drugs. Substance abuse typically progresses in predictable stages (Kandel 1975). Each stage serves as a “gateway” to the next—from abstinence to

cigarettes, then beer or wine, to hard liquor, to marijuana, and then to other illicit drugs. The increasing availability of marijuana with medical and even recreational legalization for adults has increased the likelihood that cannabis will function as a gateway substance and that some adolescents will begin with cannabis and bypass tobacco and alcohol. At each stage, many youth do not progress further, but when progression occurs, stages are rarely skipped. Almost all adolescents who have tried cocaine and heroin first used alcohol, tobacco, and cannabis. The earlier cannabis use is begun and the more often cannabis is used, the more likely is the progression to other illicit drugs. Of concern is that the marijuana available now is far more potent than the marijuana available in the past. In general, drugs from each stage are continued into the next, leading to a pattern of abuse of multiple drugs. Abuse of inhalants is an exception. Children may begin to use these easily available volatile substances but then desist as they gain access to other drugs. Although many young people experiment with alcohol and drugs, a smaller number proceed to regular use, and only a fraction of those become dependent. Unfortunately, it is difficult to predict which young people will only experiment with substances or continue social use of alcohol, in contrast to those who will progress to a more severe SUD. Early onset and rapid progression appear to increase the risk for subsequent serious problems.

In children and adolescents, substance use interferes with developing cognitive, social, and physical abilities. Critical developmental experiences that are missed may be difficult or impossible to replace, leading to high risk for impairment of future functioning in every sphere. Potential morbidity and mortality from substance use are substantial. Rates of suicidal ideation and behavior are increased. The risk of death from intentional or accidental overdose, dangerous behavior while intoxicated (especially automobile accidents), or homicide related to drug dealing is significant. Chronic use of marijuana may result in apathy and resulting arrest of academic and social development. There is increasing concern regarding potential risks of cognitive impairment, decreased motivation, or psychotic disorder subsequent to early, frequent, and heavy adolescent cannabis expo-

sure (Volkow et al. 2016). Injection drug use is the current major vehicle for the spread of HIV among adolescents. Hepatitis is also a risk. Indiscriminate sexual activity (related to direct drug effect on impulse control and judgment or induced by prostitution to buy drugs) places the adolescent at high risk for exposure to HIV and other sexually transmitted diseases. Adolescent female drug users may become pregnant and place the developing fetus at risk for drug-induced damage or HIV infection. Inhalant abuse may result in brain damage, cardiac arrest, liver and kidney damage, or lead poisoning.

■ EVALUATION AND DIFFERENTIAL DIAGNOSIS

A high index of suspicion for substance use is essential in all clinical settings. Virtually any change in emotional state, behavior, social activities, or academic performance can signal a problem with substance use. The clinician should question in a nonjudgmental manner all patients older than 9 years about substance use. If there is evidence of substance use, the patient should be asked about every category of substance, including details of amount, method of use, frequency, impairment, and social and emotional context. Verification by parents, teachers, other professionals, or peers may be crucial. Specific areas of inquiry should include intoxication at school, missing classes because of substance use, evidence of tolerance, and preferences among drugs that indicate experience. Sequelae of substance use, such as decline in school attendance or grades, increase in family conflict, cessation of previously important activities, association with a marginal peer group, and involvement in risky behavior (especially driving while intoxicated and reckless sexual behavior), should be specifically queried, as well as motivation and any attempts to decrease or stop use.

Self-report questionnaires are available for use with adolescents, including the revised Drug Use Screening Inventory (DUSIR; Tarter 1990), the Drug and Alcohol Problem (DAP) Quick Screen (Schwartz and Wirtz 1990), and the CAGE (Dias 2002).

Because most young substance users have “dual diagnoses” (i.e., psychiatric disorders in addition to substance use disorders),

the clinician should attempt to establish the chronology of substance use with respect to the emotional and behavioral symptoms. A detailed family history regarding psychiatric disorders and substance use is essential. The risk of substance abuse in parents and siblings is high.

Physical or neurological examination may disclose effects of substance use. Physical signs and symptoms of inhalant abuse may include spots or sores around the mouth, red or runny eyes or nose, dazed or dizzy appearance, and nausea or loss of appetite. Laboratory screening for drug use can provide valuable information, although false-positive and false-negative results occur, and verification and integration with the rest of the assessment are essential. Medical evaluation for HIV, hepatitis, and other sequelae is indicated.

■ TREATMENT

The primary goal is achieving and maintaining abstinence. Medical detoxification is rarely necessary in adolescents. Common features of treatment programs for substance abuse include developmentally appropriate approaches to abstinence, group therapy with other substance abusers, participation in self-help “12-Step” programs such as Alcoholics Anonymous (AA) and Narcotics Anonymous (NA), and the concept of “recovery” rather than cure. The effects of denial, lack of motivation, and the peer drug culture make conventional individual psychotherapy unlikely to succeed. Pharmacological treatments for SUDs are rarely used in adolescents. Better response to treatment may be associated with motivation and cooperation in the patient and family, the patient’s willingness to undergo urine testing, earlier stages of drug use, and remaining in treatment longer. Legal interventions and sanctions may be needed. Suggested therapeutic interventions include using motivational interviewing techniques; teaching social skills and strategies for problem solving, coping, and relapse prevention; and encouraging structured and supervised recreational activities with drug-free peers. Comorbid psychiatric disorders and specific learning disorders must be addressed and treated, although they are difficult to validly assess without a period

of abstinence. Academic deficits need to be remedied, and vocational testing and training may be useful for older adolescents for whom return to school is unlikely.

Family therapy approaches have been shown to be effective, particularly those that use structural and behavioral techniques to address parent–youth relationships and interaction patterns, as well as behavior management skills training for parents. Other effective models add interventions with peers, teachers, and other parts of the youth's social environment, as well as job and school skills training for youth (Henggeler et al. 2002).

Short-term hospitalization is now used only for acute medical indications, medical treatment of overdose or intoxication, or comorbidity with other psychiatric conditions that increase the acute risk of harm to self or others. Both the patient and the family should be actively involved in group treatment and education regarding drugs. Psychotropic medications may alleviate concomitant disorders, reduce withdrawal symptoms, or facilitate abstinence. Residential treatment for 1–12 months is used only for the most severe, complex, or recalcitrant cases or when a parent is an active drug user.

Long-term continuation of treatment is important as an outpatient or in a day treatment program or halfway house. Intensive participation in AA or NA is often required. Adolescents generally do best in groups with other adolescents rather than mixed with adults. Family therapy is an integral part of treatment. Goals include educating parents about substance abuse and its consequences, thus decreasing denial and facilitating their support of treatment and of abstinence; improving parental skills of firm and consistent, but supportive, limit setting and supervision; and enhancing communication between family members. Parents may require referral for treatment of their own substance use or other psychiatric disorders. Effective treatment results in decreased substance abuse as well as improved school performance and fewer behavioral and psychological symptoms.

Relapses are common and should be viewed as predictable complications rather than as catastrophes or reasons for terminating treatment. Relapse prevention (i.e., specific attention to situations

in which drug use is likely, with training in coping strategies) may reduce the number and severity of relapses. Periodic urine testing can facilitate abstinence.

■ REFERENCES

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, 5th Edition. Arlington, VA, American Psychiatric Association, 2013
- Dias PJ: Adolescent substance abuse. Assessment in the office. *Pediatr Clin North Am* 49(2):269–300, 2002 11993283
- Henggeler SW, Clingempeel WG, Brondino MJ, Pickrel SG: Four-year follow-up of multisystemic therapy with substance-abusing and substance-dependent juvenile offenders. *J Am Acad Child Adolesc Psychiatry* 41(7):868–874, 2002 12108813
- Kandel D: Stages in adolescent involvement in drug use. *Science* 190(4217):912–914, 1975 1188374
- Miech R, Patrick ME, O'Malley PM, Johnston LD: What are kids vaping? Results from a national survey of US adolescents. *Tob Control* August 25, 2016. Available at: doi:10.1136/tobaccocontrol-2016-053014, Accessed January 17, 2017.
- Schwartz RH, Wirtz PW: Potential substance abuse. Detection among adolescent patients. Using the Drug and Alcohol Problem (DAP) Quick Screen, a 30-item questionnaire. *Clin Pediatr (Phila)* 29(1):38–43, 1990 2403500
- Tarter RE: Evaluation and treatment of adolescent substance abuse: a decision tree method. *Am J Drug Alcohol Abuse* 16(1-2):1–46, 1990 2330931
- Volkow ND, Swanson JM, Evins E, et al: Effects of cannabis use on human behavior, including cognition, motivation, and psychosis: a review. *JAMA Psychiatry* 73(3):292–297, 2016 26842658
- Windle M, Windle RC: Depressive symptoms and cigarette smoking among middle adolescents: prospective associations and intrapersonal and interpersonal influences. *J Consult Clin Psychol* 69(2):215–226, 2001 11393599

■ ADDITIONAL READING

Bukstein OG: Substance use disorders and addictions, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 219–244

SPECIAL CLINICAL CIRCUMSTANCES

This chapter covers a wide variety of situations and conditions that are not psychiatric diagnoses but in which psychiatric clinical skills may be useful or even required. Emergencies require expert assessment and prompt intervention. The effects of family transitions or adolescent pregnancy may become apparent in pediatric or school settings. Childhood obesity and physical illness raise psychological issues. The children of parents with psychiatric disorders are often ignored by systems of care designed for adults.

■ EMERGENCIES

Assessment and Triage

Emergencies present most often in psychiatric, pediatric, or general hospital emergency rooms, but they also occur in other psychiatric and pediatric settings and in schools (Table 16–1). The clinician rapidly assesses the potential for physical danger or acute psychiatric deterioration, evaluates support systems, may initiate treatment, and makes a disposition for further evaluation and treatment. In situations that are potentially dangerous to the youth or to others, a delay in intervention may result in acute exacerbation or increased resistance to treatment or actual harm.

An emergency evaluation must be brief and focused (Table 16–2). The clinician should talk with the child or adolescent and the relevant adults both alone and together. Important collateral infor-

TABLE 16-1. **Common psychiatric emergencies in children and adolescents**

Suicidal behavior, threats, or intent
Victim of physical abuse or severe neglect
Victim of sexual abuse or rape
Violent behavior or threats of violence
Delirium or other behavior or mental status changes secondary to medical illness or to prescribed medication
Nonpsychotic hallucinations in young children
Night terrors
Acute phobic hallucinations
Psychosis
Acute anxiety reactions, hyperventilation, panic attack
Acute school refusal
Misconduct/legal violations (i.e., fire setting, property destruction, theft)
Running away
Substance use
Anorexia nervosa or bulimia nervosa
Conversion symptoms

mation can be gathered by telephone, from as many informants as possible (with appropriate consent). To ensure patient and staff safety and facilitate immediate intervention, the clinician must assess immediately for

- Medical illness or medication side effect.
- Head trauma.
- Intentional or accidental overdose.
- Drug or alcohol intoxication or withdrawal.
- Need for physical restraint or containment to prevent aggression or the child escaping.
- Need to prevent a parent from removing the child from the emergency room.
- Drugs or weapons carried by or available to the patient or accompanying adult(s).

TABLE 16–2. **Outline of emergency history**

Demographics	Age Residence School and current grade Health insurance status Primary caregiver Legal guardian
Chief complaint	Who initiated the emergency visit? What happened? What did the patient intend to do? How was the patient discovered? How did the patient and others react?
Any past suicide attempts and intent	Overdose of prescribed or over-the-counter medication Use of firearms Hanging Other self-injury Use of alcohol or illicit drugs
History of present illness	Development of symptoms Previous evaluations Treatment attempts and their results Involvement of social agencies or legal authorities
History of emotional or behavior problems	Depression Aggression Conduct disorder Drug or alcohol use Psychosis Autism spectrum disorder
Adaptive functioning with family, school, and peers	Problem-solving skills, social skills, ability to ask for and obtain help
Stressors in patient and family	Recent romantic breakup, peer conflict, bullying, cyberbullying, academic disappointment, disciplinary action, family discord, family social stressors

TABLE 16-2. **Outline of emergency history (*continued*)**

Abbreviated developmental history	Intellectual disability Developmental delay Regression from previous level of functioning
Family situation	Family living arrangements Other family or adults involved or who could be involved Adult stability, competence, relationship with the patient, attitude toward the patient and his or her problems Physical abuse, neglect, substance abuse
Family history of psychiatric illness or suicide	
History of medical illness and review of symptoms	

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. Medical evaluation should be pursued as indicated (Table 16-3). The cause of a sudden change in behavior should be considered organic until proven otherwise. “Medical clearance” in the emergency room does not guarantee the absence of physical disorder as a cause of psychiatric symptoms.

In a single evaluation in an emergency setting, especially if collateral information is limited, the clinician often has difficulty making a definitive DSM-5 (American Psychiatric Association 2013) diagnosis. It would be appropriate to use “unspecified” disorders until more definitive information is available. Only a triage decision regarding further evaluation and treatment may be possible. Abbreviated feedback, education, and explanation are appropriate in the emergency setting. When the disposition or treatment is uncertain, the primary issue is safety (Table 16-4). The decision

TABLE 16-3. Psychiatric emergency medical evaluation

Physical and neurological examination

- Potential neurological causes
- Evidence of drug ingestion
- Evidence of physical or sexual abuse
- Medical complications of an eating disorder

Laboratory tests—according to clinical indications

- Follow-up findings from medical history and physical examination
 - Blood level of prescribed drug
 - Toxicology if overdose is suspected
 - Pregnancy test
 - Blood and urine drug screen
 - Blood alcohol level
 - Tests for sexually transmitted diseases
-

TABLE 16-4. Options for emergency dispositions

- Begin crisis intervention in emergency setting
 - Send home with outpatient or crisis center referral or appointment
 - Provide family members with information on outpatient referral or crisis center
 - Send home with return to emergency room as needed
 - Observe in the emergency department
 - Contact child welfare or protective services for evaluation for placement outside the home in a shelter or foster home or for supervision in the home (hospital protective services team may consult or assist)
 - If the patient is medically unstable, hospitalize in pediatric unit (may need constant observation by “sitter”) with psychiatric consultation
 - Partial hospitalization program (day hospital) or intensive outpatient program
 - Psychiatric hospitalization
 - Involve juvenile court
-

whether to hospitalize a young person is often determined by the ability of supervising adults to tolerate the young person’s behavior or to ensure safety.

Suicide

Suicidal behavior is life threatening in all of its forms and should, therefore, be taken seriously regardless of age or circumstance. In pediatric patients, adults may assume that the intention of the youth's suicidal behavior is not serious and may not seek treatment. Very young children may lack the cognitive and physical skills to act on suicidal ideation, but a preoccupation with self-destructive behavior suggests significant psychiatric disturbance. Even the most blatant "gesture" can prove fatal, especially in a child whose assessment of physical danger may be immature and unrealistic. The need for accurate and prompt identification of suicidal behaviors is essential given the tendency for these symptoms to recur within days or weeks.

Epidemiology

The National Youth Risk Behavior Survey, conducted every 2 years by the Centers for Disease Control and Prevention (CDC), found in 2015 that 17.7% of high school students had in the past year "seriously considered attempting suicide," and 8.6% made a suicide attempt. In prepubertal children, the male-to-female ratio of completed suicide attempts is 3:1; a figure that increases to 5:1 in the 15- to 24-year age group. However, the prevalence of suicidal ideation and attempts is higher in females by a ratio of more than 2:1. Males choose the more lethal method of firearms to complete suicide, whereas the most used method in completed suicide in females is hanging. Suicide attempts using firearms are particularly lethal, especially when combined with substance use. In males, a history of suicidal behavior increases the risk of completed suicide. This prediction is not as strong in females, for whom a history of major depressive disorder is the strongest risk factor for suicide attempt. Gay, lesbian, bisexual, and transgender youth may be more likely to become suicidal because of multiple potential risk factors, including depression, substance abuse, sexual victimization, rejection by family, and bullying by peers.

Course and Prognosis

Psychiatric symptoms are a risk factor for serious and recurrent suicidal behavior. Nearly all suicide victims have a psychiatric disorder, particularly mood disorder, which in boys is often comorbid with substance abuse and/or conduct problems. Suicide in elementary school-age children is rare but is more often associated with attention-deficit/hyperactivity disorder (ADHD). Unfortunately, only one-third of suicide attempters are ever referred to a mental health service, and lack of treatment is common even in those referred. Additional risk factors include previous noncompliance with psychiatric treatment, social isolation, poor school performance, abuse and neglect, parental psychiatric illness, and family history of completed suicide. Medical illness increases suicide risk, especially epilepsy or central nervous system damage secondary to trauma, infection, or chemotherapy. Adolescent suicides are often preceded by stressful events, including the loss of a romantic relationship; reprimands in school or at home; or academic, family, or legal difficulties. Access to firearms greatly increases risk.

Evaluation

When evaluating a youth after a suicide attempt, the clinician should obtain a detailed history of the circumstances preceding and following the attempt; symptoms of mood disorder, 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 (to identify “contagion”); amount of preparation and planning invested in the attempt; and coping skills and supports in the patient and family. Patients may present with a decline in social functioning characterized by isolation, legal problems, school suspensions, and running away from home. The seriousness of the attempt is determined by a consideration of both lethality and intention. *Lethality* is a measure of the likelihood of death and is based on method, location, and the likelihood of being found. *Intention* is a description of what the patient wished to happen and is based on information provided by the patient and the interpretation by the clinician. Patients

with a previous history of suicide attempts or who use methods other than ingestion and superficial cutting should be considered at highest risk. Assessment of risk factors for subsequent suicide attempts is essential (Table 16–5). Information should be collected from multiple sources including the patient, caregiver, school, and any other individual close to the patient. The Columbia Suicide Severity Rating Scale (C-SSRS; Posner et al. 2011) is a useful tool for structuring the assessment of suicidality (www.cssrs.columbia.edu).

Treatment

In determining disposition, safety is the first concern. A brief hospitalization may be useful to complete a more detailed assessment and begin treatment, even in youths who deny continuing suicidality. Compliance with outpatient treatment recommendations may be improved if an appointment date and time are provided and the therapist contacts the patient and family before the initial visit. Family involvement in treatment planning draws them into the therapy and educates them about psychiatric diagnoses, the lethality of the behavior, and the purposes of treatment. Caretakers should be told directly to remove firearms from the home (or at least lock them securely, with ammunition stored and locked in a separate location) and to securely lock up medications (prescribed and over-the-counter), sharp objects (e.g., knives, blades), ropes or cords, and household cleaners/chemicals, and to prevent access to alcohol or illicit drugs. Although “no-suicide contracts” do not eliminate risk, the discussion can provide insight into the thinking of both patient and caregivers. When a safety plan is being created, warning signals, triggers, and potential youth coping strategies and external supports are identified.

Subsequent treatment includes treating primary psychiatric disorders, improving problem solving and stress reduction skills, and stabilizing the home and school environment. Several models of cognitive-behavioral therapy (CBT) for suicidal youth, with or without medication added, have been developed (see the chapter by Goldstein and Brent [2016] in “Additional Reading”). Dialectical

TABLE 16-5. Risk factors for repeat suicide attempt

Patient history

- Verbalization or threats regarding suicide
- Substance abuse
- Poor impulse control
- A recent loss or other severe stressor
- More than one previous suicide attempt
- A friend or family member who has committed suicide
- Exposure to recent news stories or movies about suicide
- Poor social supports
- Victim of physical or sexual abuse

Nature of the current attempt

- Accidental discovery (versus attempt in view of others or telling others immediately)
- Careful plans to avoid discovery
- Hanging or gunshot

Family

- Wishes to be rid of child or adolescent
- Does not take child's problems seriously
- Overly angry and punitive
- Depression or suicidality in family member
- Unwilling or unable to provide support and supervision

Mental status examination

- Depression
- Hopelessness
- Regret at being rescued
- Belief that things would be better for self or others if dead
- Wish to rejoin a dead loved one
- Belief that death is temporary and pleasant
- Unwillingness to promise to call before attempting suicide
- Psychosis
- Intoxication

behavior therapy (DBT) has been shown to reduce suicidal ideation and suicidal behaviors in adolescents (MacPherson et al. 2013).

Child Maltreatment

In the psychiatric evaluation, clinicians should maintain a high index of suspicion and collect information about the possibility of physical or sexual abuse. All states have laws requiring professionals who suspect child abuse to report it to the designated authorities. Mandated reporting is not confined to *confirmed* abuse. If evidence is sufficient to raise a serious question, the situation must be reported in order to prompt an official investigation. Clinicians acting in good faith are immune from liability for reporting, but legal penalties may be assessed for failing to report. When the clinician suspects abuse, he or she should tell the parents that an investigation will ensue and that helpful services may be available from the child protection agency. Abusive parents are often frightened by their loss of control and remorseful for the injury to the child. They are overwhelmed by stressors and may be relieved to have aid in protecting the child. If the child is in immediate danger, he or she should be admitted to a pediatric service (by means of a court order, if necessary) until the investigation and determination of disposition are complete. In cases of significant abuse in which the parent is the suspected perpetrator or is unable to protect the child, the protective service agency should place the child in an emergency shelter or foster home. At times, security guards or police may be necessary to complete the disposition and keep the parent from absconding with an abused child.

Epidemiology

The U.S. Department of Health and Human Services National Child Abuse and Neglect Data System (NCANDS) reported that some form of maltreatment will be confirmed in one in eight children by age 18 years (Wildeman et al. 2014). Of the reported cases, 75% were due to neglect, 17% due to physical abuse, and 8% due to sexual abuse. In 2014, almost 1,600 childhood fatalities were caused by abuse or ne-

glect. This figure is likely an underestimate, given the official tendency to report ambiguous deaths as accidental. Fatalities are more likely in younger children, with more than 75% in children younger than 3 years. More boys than girls died from abuse or neglect.

Physical and Emotional Abuse and Neglect

Clinical description and etiology. *Physical abuse* is defined as injury or risk of injury to a child younger than age 18 years as a consequence of being hit with a hand or object or being kicked, shaken, thrown, burned, stabbed, or choked by a parent or parent substitute. Children who are threatened, verbally abused, or given harsh but nonphysical forms of punishment are considered to be emotionally abused. *Emotional abuse* is defined as a "repeated pattern of caregiver behavior or extreme incident(s) that convey to children that they are worthless, flawed, unloved, unwanted, endangered, or only of value in meeting another's needs" (American Professional Society on the Abuse of Children 1995, p. 2). This abuse can include spurning behaviors or terrorizing, isolating, exploiting or corrupting, or denying appropriate emotional responsiveness to the child. The most common abuser is the child's parent or guardian. Risk factors for abuse include social isolation of parents, violence between parents, management of behavior problems with corporal punishment, inappropriate expectations for the child's developmental level, young parental age, parental history of abuse, caretaker use of alcohol and drugs, and stressors on the family such as unemployment and overcrowding. Children with a history of prematurity, intellectual disability, or physical disability may be at higher risk of victimization.

Neglect is more difficult to define than abuse, and clarification often requires a pediatric hospitalization with a comprehensive medical, psychiatric, and social evaluation. Neglect may be considered in five categories: 1) medical care neglect, 2) gross safety neglect (lack of supervision), 3) physical neglect (food and shelter), 4) emotional neglect, and 5) educational neglect (the latter two types are not recognized by all states). Emotional neglect applies when the patient is not given adequate attention and affection or is exposed to

traumatic and inappropriate behaviors. Poverty contributes even more powerfully to neglect than to abuse. Many neglectful parents have not experienced or even witnessed appropriate parenting. Contributing factors may include cognitive limitation, drug or alcohol use, or mental illness.

Evaluation. Clinicians should have a high index of suspicion for abuse when evaluating an injured child whose presentation is atypical. The history may yield clues such as delayed seeking of medical care, an explanation that does not fit the injury or the child's developmental level, and variable descriptions of how the injury occurred. Injuries characteristic of abuse include but are not limited to

- Multiple injuries in various stages of healing.
- Bruises in the pattern of fingers or belts.
- Burns, especially from cigarettes or scalding by water.
- Spiral bone fractures.
- Head and eye injuries.
- Ear injuries.
- Abdominal injuries.
- Chest injuries.

Evaluation of suspected abuse or neglect includes a complete physical examination (with imaging studies as indicated) and private interviews with the parents; the child (if old enough to talk) or adolescent; and other persons, such as siblings, babysitters, relatives, and neighbors. Youth may hide abuse because of fears of punishment or abandonment by peers or family members. Although corporal punishment of children by parents (and, in many states, by teachers or principals) is still accepted by many cultural groups in the United States, any injury inflicted by a hard object or by burning, shaking, or throwing should be considered abuse, as should prolonged or severe spanking.

Effects. Abused and neglected children are at risk for cognitive deficits, neurological impairment, blindness, physical disability, or even death. Psychological sequelae vary widely in severity and nature,

commonly including low self-esteem, difficulty trusting others, impaired social relationships, increased impulsivity, irritability, anhedonia, aggression, poor school performance, and self-destructive activities, including suicide and a variety of risk-taking behaviors. Posttraumatic stress disorder (PTSD), attachment disorders, or symptoms of dissociation may ensue. Virtually any psychiatric disorder can be present in victims of abuse, whether as risk factor, comorbidity, or sequela.

Interventions. The initial goal of treatment is to prevent the recurrence of abuse. Interventions include hotlines and self-help groups (organized by Parents Anonymous) that are useful for motivated but socially isolated parents who have difficulty controlling their angry responses toward their children.

Victims need interventions for abuse-related symptoms, as well as a psychiatric evaluation with multimodal treatment as indicated. Day hospital programs provide a safe and nurturing environment to begin abuse-specific interventions. Group play therapy can encourage interaction with peers and avoid the solitary play that frequently develops in maltreated children.

Parents at high risk include teenagers, new parents, impoverished single parents, and individuals with histories of substance use or cognitive limitations. Perpetrators have high rates of depression, substance use, and antisocial behavior requiring assessment and treatment. Education in normal child development and principles of behavior management may be useful. Some parents need concrete assistance with housing, food, and medical care. Therapeutic and supportive services are often best provided in the home, because many families are both poorly motivated and insufficiently organized to make use of traditional outpatient services. Short-term placement of the child in foster care may be necessary while the home is stabilized and parents obtain treatment. Child welfare agencies and family courts should be encouraged to determine rapidly whether parents can be rehabilitated and the children returned or whether proceedings should be initiated to terminate parental rights. Too often a child is placed in a succession of foster homes, punctuated by unsuccessful returns to the parents.

Sexual Abuse

Clinical description. *Sexual abuse* refers to touching offenses (ranging from fondling to sexual penetration) and nontouching offenses (exposure to pornographic material). Although the reported incidence of sexual abuse and rape is increasing with mandatory reporting laws and heightened public awareness, it is still likely to be an underestimate. Females are more likely to be victimized than males by 4–5:1. The most common age of initial sexual abuse is 8–11 years. Most perpetrators are male and usually known to the victim. Although the offenders in most reported abuse cases are fathers or stepfathers (or boyfriends), retrospective studies also identify uncles or brothers as frequent perpetrators. Extrafamilial and intrafamilial sexual abuse of boys is less frequently reported and prosecuted, even though the psychological consequences may be debilitating.

Evaluation. Sexual abuse varies widely in degree of sexual contact; amount of physical or psychological coercion; single vs. repeated incidents; and whether the abuser is a member of the household, another person known to the child, or a stranger. The child may tell a parent, a friend, or a teacher, or there may be physical evidence such as prepubertal vaginal bleeding; recurrent urinary tract infections; inflammation, bruises, or lacerations of the genitals or anus; sexually transmitted disease; or pregnancy. Sudden onset of compulsive masturbation, precocious sexual knowledge or behaviors, oppositional behavior, fears, running away, depression, sleep disturbance, somatic symptoms, or decreased academic performance may raise suspicion.

Rape or sexual abuse by a stranger is usually reported by the victim immediately, although boys are less likely to acknowledge abuse. The child is often seen acutely in the emergency room. Sexual abuse by a relative or family friend is more difficult for children to report, is often discredited by the mother, and may result in a family crisis. In cases of father–daughter incest, the mother may suspect or be aware of the abuse, but fear of the father or a wish to “protect the family” prevents her from taking action. The mother may even tacitly encourage the abuse. In these families, a daughter takes on many

aspects of the role of the mother and wife who is less available because of depression or physical illness.

The purposes of the initial evaluation are

- To determine whether abuse is likely to have occurred.
- To ensure that the child is protected.
- To establish the need for medical and psychological treatment.

It is important that the evaluation itself does not further traumatize victims of sexual abuse. A team approach, in which health and mental health care professionals work together with the child protection agency and the legal system, avoids subjecting the child to repeated inquisitions. A complete physical examination is indicated, preferably by a pediatrician or pediatric gynecologist who has experience in evaluating abused children and who can carry out the procedures required for legal evidence. The clinician should interview (together and separately) the child, parents, and siblings. Questions about the specifics of the abuse are best saved for private or forensic interviews. For children who have difficulty verbally describing events, the opportunity to draw or use puppets may be helpful. The use of anatomically correct dolls is controversial. More complete evaluation of the patient's psychiatric and developmental status should be deferred for a subsequent interview. If the alleged perpetrator is not a member of the immediate family, his or her interrogation is often best left to child welfare or legal officials.

Children rarely make false allegations of sexual abuse, except in the context of prompting by a parent embroiled in a custody dispute, adolescent vindictiveness (e.g., to remove a disliked stepfather), or a wish to disguise the teenager's voluntary sexual activity. When false allegations are suspected, a child and adolescent mental health professional with special expertise should be consulted.

Effects. Victims of sexual abuse are at increased risk of behavioral and emotional problems, and are overrepresented in clinical psychiatric settings. Rates of psychiatric disorders increase as the children approach school age, probably because they begin to recog-

nize the inappropriateness of the sexual contact. As adults, victims report higher rates of many psychiatric disorders. A history of sexual abuse increases the likelihood of attempted suicide 2- to 14-fold. Full or partial PTSD is common. Symptoms include fear, startle reactions, reenactment of the trauma, flashbacks, sleep disturbance, and depression. Children and adolescents who are victims of particularly severe or long-lasting abuse may experience dissociative reactions and conversion symptoms. The tendency to reenact the trauma may lead to sexual behavior problems. Among victims, risk factors for emotional and behavior problems include family disruption, economic hardship, preexisting psychiatric diagnosis, and scapegoating of the child for reporting the abuse. Psychological symptoms include suicidal ideation or behavior, fears, sleep disorder, low self-esteem, sexual precocity or preoccupation, impaired social adjustment, and subsequent sexual dysfunction as an adult. Promiscuity or, in contrast, a phobic reaction with sexual inhibition may develop.

Medical sequelae include damage to the genitals or rectum and/or acquisition of sexually transmitted diseases and their sequelae.

Interventions. The child must be protected from both further sexual abuse and the effects of reporting it, especially if the perpetrator is the father or the mother's partner. The child should return home only if the abuser has been removed from and does not have access to the home and if the mother can and will protect the child.

Research on treatment for sexually abused children and adolescents is limited. Sexual abuse is an experience rather than a disorder and the treatment targets symptoms and dynamics. Symptoms resulting from abuse vary based on developmental level, with children more likely to experience anxiety and sleep disorders and adolescents more likely to experiment with drugs and delinquent behavior. There is an empirically supported model of CBT for sexual abuse-related PTSD symptoms in preschool-age children (Cohen et al. 2004).

Treatment interventions are "abuse-specific" and encourage the expression of abuse-related feelings, correct cognitive distortions

regarding the abuse, teach prevention skills, and encourage support from other abuse victims. Specific symptoms may persist, however, and require targeted interventions that may include psychopharmacology. The therapist should select a treatment approach based on the characteristics of the child, the presence of psychiatric symptoms, and the family context. Intermittent treatment may be required for effects of abuse that may not appear for months or years. There is no evidence, however, that psychiatric treatment for asymptomatic victims of abuse will prevent future problems.

In incest cases, legal pressure may be required to initiate and maintain treatment of the perpetrator. Therapy models that aim at improving family functioning have not been particularly successful. Many parents (whether perpetrator or spouse) of sexually abused children have been abused themselves, making it difficult for them to deal with their child's situation. These parents may benefit from a support group for adult victims of childhood incest. If alcohol is a precipitating factor, successful treatment of the alcoholism markedly decreases the risk of recidivism.

Out-of-Control Behavior

When a child or an adolescent is brought to an emergency room because his or her behavior is out of control (Table 16-6), the clinician must first use whatever physical means are necessary to ensure the safety of the patient, family, and staff.

Evaluation

If a patient has been aggressive, it is important to determine whether a person or object actually has been harmed or only threatened verbally or with gestures. Detailed description of the aggressive behavior includes: precipitants, warning signs, evidence of an altered mental state, actual damage, need for physical restraint, repetitive behaviors, lethality (including access to firearms), solitary vs. group action, and response to any previous treatment. Cultural factors may affect both the precipitant and the response. Predictors of violent

TABLE 16-6. **Causes of out-of-control behavior in children and adolescents**

Temper outbursts

Attention-deficit/hyperactivity disorder

Oppositional defiant disorder

Conduct disorder

Intellectual disability

Autism spectrum disorder

Anxiety-provoked aggression

Separation anxiety disorder

Panic disorder

Obsessive-compulsive disorder

Acute phobic hallucinations (especially in children ages 2–6 years)

Organic delirium

Medical illness

Fever

Electrolyte imbalance

Central nervous system infection, tumor, trauma, or vascular accident

Seizures

Endocrine or autoimmune disorder

Hypoxia

Metabolic disorder

Adverse reaction to prescribed or over-the-counter medication

Toxic ingestion

Reaction to illicit substance

Acute intoxication or toxic reaction

Flashback

Withdrawal

Psychotic reaction to chronic drug use

Schizophrenia

Mania

Abuse or neglect

Posttraumatic stress disorder

behavior include male sex, emergency room arrival under police custody, and history of prior violence, violence victimization, substance use, and psychiatric illness (Marzullo 2014).

The mental status examination includes an assessment of current anger or violent intent, organic cognitive impairment, paranoia, delusions or hallucinations, and impulsivity.

Patients with intellectual disability, autism spectrum disorder, neurological disorders, or delayed expressive and receptive language are more likely to communicate distress through aggression. The older and larger the youngster, the more seriously aggression must be taken. If ADHD, conduct disorder, psychosis, intellectual disability, or drug use causes impulsivity and impaired judgment, a more restrictive environment may be needed.

Children and adolescents with a history of escalating violence and pervasive hostility are most problematic. Occasionally youth will show little guilt or remorse for their actions. The risk of aggressive behavior is increased by neuropsychological dysfunction and exposure to violence and/or physical abuse or witnessed violence between caregivers. In such instances, parents model the choice of aggression as a solution to problems. Family assessment is important because relationships among all family members are affected by violence.

Treatment

In the acute situation, medication is sometimes considered, although children have usually calmed and rarely require medication in the emergency department. Special caution in pharmacotherapy is needed if a medical disorder or drug ingestion is suspected. If necessary, acutely psychotic adolescents can be given an antipsychotic agent. The second-generation antipsychotics (see Chapter 17, “Psychopharmacology”), particularly risperidone and olanzapine, because of their comparable efficacy, rapid onset of action, sedative effects, rapid orally disintegrating form, and comparatively lower extrapyramidal effects, are becoming the preferred choice over haloperidol for the acutely agitated patient (Baeza et al. 2013).

Benzodiazepines may cause paradoxical disinhibition and worsening of the agitation. Particular attention should be paid to the safe and appropriate use of seclusion and restraint, if needed, because of risk of harm to self or others and the failure of less restrictive interventions to control the patient's behavior.

In considering disposition from the emergency room, the clinician must consider the safety of vulnerable persons in the home (e.g., a baby) and the ability of the adults to supervise. Even if the behavior resolves quickly in the emergency department, these youngsters often have multiple social, psychological, academic, and behavior problems that require psychiatric follow-up. The courts and child protective services are frequently involved in disposition planning.

After safety is ensured, the goal of treatment is to address the underlying cause of out-of-control behavior. Psychiatric hospitalization is not effective in the treatment of severe conduct disorders. Possibly more effective alternatives are extended partial hospitalization, residential treatment, therapeutic school placement, and comprehensive home-based treatment programs. The success of outpatient therapy depends on the patient's and family's cognitive abilities and motivation, the severity of antisocial behavior, and use of a systems approach. Cognitive-behavioral programs like anger management training encourage greater individual control. Social skills training increases alternatives to aggression, if the environmental contingencies can be managed to encourage their use. Family and, when necessary, the legal system should be involved to actively support the treatment process.

■ FAMILY TRANSITIONS

A substantial number of children spend some part of their life with just one parent, whether due to single motherhood, separation, divorce, or death of a parent. Children in single-parent households typically have more responsibility, not only for chores but also for management, decision making, babysitting for younger siblings, and emotional support of the parent. As a result, these children may

be more independent, competent, and responsible than other children but at the expense of having less freedom, more worries, and less closeness with peers. The parent–child relationship can suffer when children resent the additional responsibility.

Divorce

Effects

Divorce affects nearly all aspects of a child's life, but the long-term outcome varies depending on both risk and protective factors. Divorce is a process that often begins with marital strain and discord (that may last for years), is punctuated by the marital separation, and results in dramatic changes in family life. To the child, divorce represents the failure of a relationship that was to serve as a model of love and commitment. A conflict-ridden intact family is, however, more detrimental to children than a stable one-parent home. In fact, marital discord is a more important risk factor for child maladjustment than actual divorce or conflict following divorce (Buehler et al. 1998). High-intensity marital conflict during childhood is associated with attachment difficulties, externalizing and internalizing symptoms in childhood, and psychological disorders as young adults. Parental physical violence is more detrimental to children's adjustment than verbal conflict. Rates of child abuse and sibling violence are increased in violent compared with nonviolent high-conflict marriages. Protective factors that have been identified for children in high-conflict marriages include a positive relationship with a caregiver and supportive siblings. In addition, for teenagers, peer relationships and a positive self-concept appear to be helpful. Another factor mediating the impact of high-conflict parental relationships is the manner in which the parents settle their disputes. Unresolved or chronic discord is associated with more difficulties for children. Parental conflict in which each parent criticizes the other to the child and attempts to gain the child's alliance is particularly damaging, whether in an intact family or after divorce. Intense parental conflict may also reflect psychiatric symptoms in one or both adults. Children and adolescents commonly blame them-

selves for the divorce, view themselves as potential saviors, and harbor fantasies that their parents will reunite.

Reactions to parental divorce may vary with the age and developmental level of the child and the time since divorce. Infants may react with irritability and sleep and feeding difficulties. Children ages 2–6 years commonly experience separation anxiety and behavioral regression such as clinging or loss of toilet training. Children ages 5–9 years may be anxious, sad, and preoccupied with loss of their intact family and separation from their nonresidential parent, whatever the nature of the prior relationship. Slightly older children may feel shame and anger or complain of somatic symptoms, but they have somewhat better coping mechanisms to deal with their feelings. Disruptions in peer relationships and academic learning are common. Pre-teens may feel powerless, frightened, and intensely angry with one or both parents. They are more likely than younger children to take active sides in ongoing battles between their parents. Adolescents tend to react either with depression, acting out, and emotional and social withdrawal from friends and school or with a developmental spurt, showing unexpected maturity, empathy, and compassion and providing significant help to one or both parents. School social adjustment and academic performance may be negatively affected.

The relative effects of different custody options are difficult to study, although in one 12-year follow-up study of low-income families with contested custody in divorce who had been randomly assigned to mediation or litigation, mediation resulted in greater continuing involvement with the nonresidential parent without an associated increase in co-parenting conflict (Emery et al. 2001). Some research has found a positive correlation between joint legal custody and children's adjustment; however, parents' psychological well-being and the quality of the parent-child relationship are key variables. Divorcing parents who *request* joint custody are likely to be those who are better able to maintain a parental coalition and active involvement of both parents, despite the dissolution of the marriage. Fathers with joint custody are more likely to remain emotionally involved with and financially supportive of the children. If a judge awards joint custody against the wishes of one or both par-

ents, severe conflict is likely to continue, to the detriment of the child. This is particularly true for infants and toddlers who are often forced into overnight visits at a time when separations may be traumatic. Single-parent custody relationships have been effective, particularly when contact with the noncustodial parent remains an option for the child. Even when the noncustodial parent has been unreliable or has acted violently, the child may continue to request visits. Unfortunately, divorce almost always has a detrimental effect on the relationship between the nonresidential parent and the child. Contacts become primarily social, and these parents rarely are partners in the child's discipline or education. There is evidence, however, that for noncustodial fathers the quality of paternal parenting and the child-father relationship are more important than frequency of contact (Amato and Gilbreth 1999).

Marital separation and divorce create social upheaval for the child. The standard of living typically declines, due to spreading the family income over two households instead of one, inadequate child support payment, and mothers who may be poorly equipped to enter the job market. When a stay-at-home mother must return to work, children may be traumatized by yet another separation. Reduced income may lead to a move to a more modest neighborhood, with resulting separation from familiar peers, neighbors, and school.

Divorce has phases that require repeated adjustment. Some children do poorly during the first year after divorce, a time that forces change on the parent as well as the child. Many parents, coping with their own anger, grief, depression, anxiety, and loneliness, as well as struggling with the financial settlement and the practicalities of maintaining separate households, are emotionally unavailable to their children and unable to provide consistent routine and discipline. Typically, hostility between parents decreases over time. Long-term child adjustment is dependent on the ability of the custodial parent to maintain a functional household, continue the child's relationship with the other parent, avoid severe economic hardship, and provide emotional support to the child.

Children of divorced families have more social, emotional, and behavioral difficulties, including substance use and difficulties with

relationships, than those in intact families, although less so than previously believed. Age of the child, time since divorce, parenting styles, and degree of parental/family conflict likely influence these complex issues of postdivorce adjustment. Adolescents and young adults may experience delayed effects of divorce as they enter dating, falling in love, and marriage.

Most children of divorced parents eventually must readjust to one or more new families, as one or both of their parents find new partners or remarry. Remarriage by a custodial mother is associated with increased problems in the mother–daughter relationship but decreased behavioral symptoms in boys. Young adolescents who have functioned in parental roles often have special difficulty adjusting to a stepparent, who usurps some of their duties and privileges. Awareness of the parent's sexual activity is acutely embarrassing to teenagers.

Although some young adults who have faced divorce as children have significant consequences, the majority have resilient outcomes.

Interventions

Education regarding typical reactions to divorce is useful for parents and children. A support group may reduce feelings of isolation and guilt, especially in communities where divorce is uncommon. Postdivorce groups or workshops based in schools offer services with less stigma. Parents Without Partners, a self-help group with chapters nationwide, offers companionship, advice, and support from adult peers, as well as educational programs and role models for children. Brief focused therapy can help children and parents deal with their feelings and aid parents in resolving conflict and establishing new lives. In a controlled trial with white, middle-class families undergoing divorce, a 6-year follow-up of a group and individual prevention program with mothers, with or without their school-age children, found that the adolescents from the intervention groups (compared with controls, who received reading material) had fewer mental disorders and fewer sexual partners. Among those

initially at higher risk, the intervention subjects had fewer behavior problems and less substance use (Wolchik et al. 2002). Some states provide court-connected programs that offer or mandate evaluation, counseling, and crisis intervention and make recommendations to the judge regarding custody, visitation rights, and child support. Divorce and custody mediation is available as an effective alternative to the adversarial litigation process. However, in circumstances of abuse, violence, mental limitations or illness, or severe and unresolved parental conflict, the judicial system is appropriate for protection of children and parents. Children and parents with significant symptoms may require more extensive psychiatric evaluation including individual and/or family therapy. Parental psychiatric problems following divorce are associated with more difficulties for children; therefore, prompt treatment for these adults is critical.

Physical Illness in Parents or Siblings

When a family member is chronically or seriously ill, parental resources in time, money, and emotional energy available to other family members are reduced. In addition, nearly all children have at some time wished that something bad would happen to a parent or sibling. If that person then falls ill, the child may literally believe that he or she caused the illness. This “magical thinking” is normal in young children and appears under stress even at later developmental stages.

To the extent that an ill child receives special treatment, sibling rivalry is exacerbated. This may be overt or concealed by the child’s guilt or parental shaming. A child or an adolescent often must take on extra chores to substitute for a parent or sibling. Parents can help by equalizing chores, discipline, positive parental attention, privileges, and treats as much as developmental stages and physical condition permit.

Childhood disability and illness have an impact on the whole family system. The sibling of a disabled child may be teased by peers. Providing accurate information about the disability can facilitate coping. Some siblings of children with severe, chronic disabil-

ities are more likely to have symptoms of aggression, depression, social isolation, and oppositional behavior. Families who are already stressed (e.g., by financial strain or single parenthood) are at higher risk for emotional or behavioral symptoms.

Death of a Family Member

The death of a family member, particularly a parent or primary caregiver, is a traumatic event for a child that can elicit strong emotional or behavioral responses. One in 20 children experiences the death of a parent. Bereaved children often experience depressed mood, sadness, longing for the deceased, appetite changes, irritability, social withdrawal, declining school performance, and sleep disturbances, although children's expressions of grief tend to be more intermittent than those of adults. In the first 2 months after parental death, the effect can be profound and may lead to the development of major depression and suicidal ideation. However, the severity of these symptoms generally wanes over time. In a longitudinal study of the psychosocial functioning of bereaved children from stable families, the children appeared similar in function to healthy control subjects (Fristad et al. 1993). If the death occurred under traumatic circumstances, the expected anger and grief may escalate to *childhood traumatic grief* or *persistent complex bereavement disorder*. If the death involves a child, the siblings must face their own grief in addition to their grieving parents' emotional withdrawal. Suicide-bereaved children have to address grief as well as the stigma and anguish surrounding suicide. Suicide-bereaved children experienced more anxiety, anger, and shame than children bereaved from parental death not caused by suicide (Cerel et al. 1999).

The terminal phase of the parental illness may, in fact, be more stressful for the child than the death. During this time, the child is exposed to the physical changes and painful consequences of life-threatening disease. The child also becomes aware of the irreversible nature of the illness and the likelihood of death. Parental illness changes the structure and organization of the family. Family roles and routines change, and family cohesiveness may suffer as the ill

parent becomes less available physically and emotionally, and the other parent may be exhausted by caretaking and grief. Terminal phases of illness require more resources for care, which may jeopardize the family's financial stability. When a parent has been ill, the death may mark the end of a tragic and difficult period for the family and begin the process of healing and reorganization.

When a family member dies, the child should be allowed some choice in whether to attend the funeral (if developmentally appropriate). One study (Weller et al. 1988) showed that attendance at funeral activities did not result in increased psychiatric symptoms 2 months later. Extra support, such as an adult friend or relative, can be helpful through the funeral process, because the surviving parent may be too preoccupied with his or her own grief to be available to the child. Children can mourn, although they may express their grief in behavior rather than verbally. If the child is especially vulnerable because of a psychiatric disorder or additional stressors, clinical judgment should influence the decision about how to handle the funeral, and extra therapeutic support may be needed.

Parents should explain death in a matter-of-fact, concrete way. Religious explanations should be used only if these are consistent with family beliefs. The magical thinking and egocentricity that are characteristics of young children may lead them to believe that they are responsible for the death. They will often be too frightened or guilty to mention this and should be spontaneously reassured that they had nothing to do with the cause of the death.

The child's conceptualization of death plays a role in adjustment. As children mature, they gradually develop an understanding that death is universal and irreversible. Children younger than 2 or 3 years do not understand at all; they are simply made anxious by the separation. At ages 4–5 years, death seems reversible, like sleep or a long journey. For 5- to 10-year-olds, death can be personified. A more realistic concept of death develops in children ages 10–11 years. Although by then most understand that death is universal, more than half of fourth graders do not yet clearly understand the irreversibility of death. Elementary school-age children tend to view death as a punishment for bad behavior. Although most adolescents cogni-

tively understand the meaning of death, some behave as though they do not accept their own mortality.

The long-term effects of family bereavement depend on how well the surviving parents cope and are able to restore family life functioning. Open and honest expressiveness among mutually supportive family members facilitates this process. If the child's life is further disrupted by economic hardship or changes in school, home, and friends or the child's relationship with the deceased parent had been conflictual, the risk of significant adjustment problems may be increased.

Adoption

Adopted children are overrepresented in psychiatric treatment settings because of factors such as genetic or prenatal risks from the birth parents; disruptions, trauma, or neglect in the child's early life; adoptive home problems (such as unrealistic expectations by both the adoptive parents and the child, differential treatment by parents or grandparents); and the child's unresolved feelings about being given up for adoption or identity issues. The understanding of temperament (see Chapter 2, "Evaluation and Treatment Planning") emphasizes the importance of "goodness of fit" between parent and child. To the extent that there is a genetic contribution to temperament, adopted children may be more likely to be mismatched with one or both parents, increasing the risk of behavior and emotional problems.

Children adopted past infancy are at higher risk for psychiatric problems as a result of the experiences that led to placement for adoption. These children have endured not only the loss of their parent(s) or primary attachment figure, but perhaps also neglect, abuse, and frequent changes of caregiver. Some children adopted after spending their first years in a residential nursery or orphanage are capable of forming stable affectionate relationships with their adoptive parents, but many show significant social and attentional problems and attachment difficulties.

It is normal for children and adolescents, even those who have not been adopted, to go through stages of questioning whether they

are with their “real” parents and whether another set of parents would be better. It is important for adoptive parents not to take these stages too seriously or personally. Although there is not a clear consensus of when the best time is to disclose to a child his or her adoption status, the child’s cognitive functioning should be taken into account. The trend is increasingly toward “open” adoption, where children are told early and there may be contact with the birth mother.

■ CULTURAL FACTORS FOR IMMIGRANT FAMILIES

Immigrant families face unique challenges, both within the family unit and in relation to the external environment, that can have a profound impact on the psychological well-being and functioning of children. Factors such as lack of social supports, financial strain, prior or current trauma associated with the immigration process, perceived racism or prejudice, ongoing concerns about immigrant status or risk of deportation, language barriers, and neighborhood crime and poverty can affect the family’s functioning and that of the children. In addition, children of immigrants often acculturate at a faster rate than their parents, leading to a phenomenon known as an “acculturation gap.” This can increase parent–child conflict, especially for adolescents, as their adoption of “American” values may be perceived by their parents as a rejection of their native culture. The children may feel that their parents are being unreasonable or unfair. Research has found that adoption of a “bicultural” identity, in which an individual integrates values from both the native and host cultures, is the most adaptive stance and produces the most favorable outcomes. Helping parents understand their bicultural children and helping children appreciate their parents’ immigration narrative can be beneficial in facilitating communication. In work with families, it is important for the clinicians to understand the immigration experience of the family and the cultural identity of the parents and children. Identifying the key decision-makers and caregivers within the family and recognizing the cultural factors impacting treatment seeking, understanding of illness, and attitudes

toward mental health care can greatly improve rapport and treatment adherence.

■ ADOLESCENT PREGNANCY

Adolescent development includes exploration of new relationships, including attraction, dating, breaking up, and sexual activity. Mental health disorders in teenagers may significantly affect this aspect of social development. Teens with severe social anxiety or social skills deficits may be frustrated by their inhibition or difficulty in interacting with potential partners. Adolescents with depression, bipolar disorder, or impulsivity may be more likely to engage in unprotected sexual activity that puts them at risk for pregnancy or sexually transmitted diseases.

Mental health clinicians may be the health professionals with the most regular contact with youth. Psychiatric assessment and ongoing care should regularly address the emotional, developmental, and medical aspects of reproductive health in adolescents.

Demographics

By age 15, 15% of unmarried adolescents have had sexual intercourse; by age 19, 46% have. In 2015 data reported by the CDC, the birthrate among U.S. females ages 15–19 was 22.3 per 1,000, an all-time low, although remaining higher than in other industrialized nations. The recent sharp decline in American teenage pregnancy and birthrates is attributable to increased use of contraceptives, particularly use of highly effective methods (Lindberg et al. 2016). The proportion of teen pregnancies ending in abortion continued to decline, to 30% of pregnancies in 2010. There are large disparities by state of residence and by race, with adolescent girls of color at higher risk for unintended pregnancy.

The Mother

Adolescents who become pregnant are not a uniform group; they differ by race, socioeconomic status, and age. In studies controlled

for academic aptitude and financial status, teenage mothers are less likely to complete high school, less likely to achieve a stable and well-paying job, more likely to be dependent on public assistance, and less likely to enter a stable marriage than their peers. Interestingly, those who are able to finish high school, avoid another pregnancy in their teens, and marry do not show negative effects at long-term follow-up when compared with peers who delayed their first pregnancy.

The Child

Lack of adequate prenatal care is a risk factor for prematurity, low birth weight, and neonatal mortality, especially for mothers younger than age 15 years. Children of teenage mothers have higher postneonatal mortality rates and more subsequent illnesses and injuries.

Children, especially sons, of teenage parents appear to be at a developmental disadvantage and may be at higher risk for emotional problems, ADHD, aggression, school failure, and incarceration. The daughters of adolescent mothers are more likely to become pregnant before age 18 years than offspring of mothers in their 20s. Poor prenatal care, genetic factors, parental limited education, and suboptimal parenting because of immaturity, financial stresses, and living in disadvantaged neighborhoods with low-quality schools may contribute to these outcomes.

Interventions

Mental health providers should regularly assess adolescents for high-risk sexual activity and provide guidance on abstinence, consent, contraception, and prevention of sexually transmitted disease. Potential effects of psychotropic medications on a developing fetus or newborn must be reviewed with all adolescents of reproductive potential.

Once an adolescent becomes pregnant, nonjudgmental support for the young woman and her family, along with practical and educational assistance, may facilitate making an informed decision among

adoption, abortion, and raising the child. Young mothers are most satisfied with the outcome of their pregnancy when they receive parental support for their decision (Siegel and Brandon 2014). For teenage mothers raising a child, outreach programs offering prenatal care, child care, parenting training, assistance in completing high school, further academic or vocational training, and enrichment programs such as Head Start for the children can positively affect long-term outcome.

■ OBESITY

Clinical Description

Body mass index (BMI; weight in kg/height in m²), the most appropriate measure to screen for childhood obesity, has norms by age and gender. *Overweight* is defined as a BMI at or above the 85th percentile and below the 95th percentile of age- and sex-specific norms. *Obesity* is defined as a BMI greater than the 95th percentile.

Epidemiology

The prevalence of childhood obesity is increasing. Data from the 2011–2014 National Health and Nutrition Examination Survey (NHANES) found 17% of youth to be obese (Ogden et al. 2016). African American youth and Hispanic boys are at greatest risk for both obesity and overweight.

Etiology

Genetic, medical, family, and societal factors contribute to the development of obesity. Parental obesity greatly increases the risk. Feeding practices from infancy impact later weight gain. The ubiquitous presence of calorie-dense and nutrition-poor foods, the relative absence of fresh fruits and vegetables in urban “food deserts,” the challenges of exercise in many neighborhoods, and the increase in screen time for many children are all implicated in pediatric obesity.

Course and Prognosis

Obesity in middle childhood (especially if it persists through adolescence) predicts adult obesity. Childhood obesity can result in medical complications, including cardiovascular disease and diabetes. Severe liver disease with hepatitis and fibrosis may result. Obstructive sleep apnea as a consequence of weight gain can lead to nighttime hypoxemia, cardiac arrhythmias, and right heart failure. Obesity carries a social stigma as well. These children have more difficulty making and keeping friends, negatively affecting levels of self-esteem. However, there is increasing evidence that obesity is not always associated with poor health outcomes and that it is possible to be metabolically healthy at higher weights (Saul and Rodgers 2016).

Evaluation and Differential Diagnosis

The mental health provider may be part of a multidisciplinary team working with the obese child and his or her family. Comprehensive assessment should include medical, family, social, nutritional, physical activity, and sleep history, as well as a physical exam and laboratory tests as indicated to assess metabolic risk factors and possibly rare endocrine or genetic causes of obesity. The nutritional history includes not only what and how much the child consumes but behavioral patterns as well, including where the child eats and with whom; times of day; accompanying activities; emotional state before, during, and after eating; and family attitudes toward food, exercise, and weight. Clinicians must strive to avoid communicating weight bias or shame, respectfully taking into account cultural and socioeconomic factors that influence health and diet behaviors.

Treatment

The treatment team should emphasize family-based efforts rather than targeting the identified patient and make the primary goal healthy behaviors rather than weight change. For children, the medical recommendation is generally to stabilize weight and allow the child to grow into it, rather than weight loss. Prescribing dietary or

behavioral changes is less effective than collaborative treatment. Collaboration begins with motivational interviewing, in which the clinician helps the child and family develop their goals and assess their readiness for, and obstacles to, meeting them.

Specific treatment plans vary based on the family presentation, the child's needs, and resources available, but experts make the following general recommendations (Children's Hospital Association 2012):

For individuals:

1. Use a hunger scale to develop awareness of hunger and satiety.
2. Review and encourage sleep hygiene, as poor sleep is associated with overeating.
3. Recommend eating in a common area, such as the kitchen table, without television screens or other distractions.
4. Consume five small meals per day.
5. Recommend leaving the table after a normal meal and waiting 20 minutes before second helpings, giving time to experience satiety.
6. Encourage regular exercise.
7. Decrease sedentary activities.

For caregivers:

1. Recommend that the whole family participate in health and exercise behaviors, and explore barriers to these.
2. Show love and nurturing without using food as a reward.
3. Create a positive family culture around eating.
4. Foster mindfulness around eating, encouraging children to enjoy foods and to attend to the sensory experience of eating.
5. Avoid buying foods that family members overeat.
6. Avoid nagging or criticizing around food—this is likely to be counterproductive.

Certain behavioral patterns related to diet and nutrition may be best addressed in CBT, interpersonal psychotherapy, or DBT. These

include boredom eating, emotional eating, binge eating, nighttime eating syndrome, hoarding or hiding foods, and food aversions.

Children who are treated with certain psychotropic medications, particularly the atypical antipsychotics and the mood stabilizers, are at risk for medication-induced weight gain. Counseling regarding dietary habits must be part of their care, and the continued need for treatment must be carefully balanced against the risks associated with development of obesity. Addition of metformin may mitigate medication-induced weight gain (Zheng et al. 2015).

Bariatric surgery is increasingly used to treat severe obesity in adolescents who have not responded to more conservative weight loss strategies, particularly when there is comorbid diabetes or metabolic syndrome. Bariatric surgery can reverse these disease processes (Mirensky 2016).

■ PHYSICALLY ILL CHILDREN AND ADOLESCENTS

Developmental Factors in Reaction to Acute Illness, Hospitalization, and Surgery

The emotional response to illness is dependent on the child's stage of development, the amount of discomfort, the type of treatment and its side effects, the practical limitations that result from the illness, premorbid psychiatric problems in the patient, and the child's level of understanding. Parents influence the child's reaction through their own coping mechanisms and the support they are able to provide. Clinicians can often predict the most stressful times in the course of an illness and should take these opportunities to provide additional emotional support. Practices such as parental rooming-in, unlimited parental visiting, permitting parents to be present while the child is sedated preoperatively, and outpatient surgery to avoid hospital admissions have reduced emotional aftereffects. The use of primary nurses and child development specialists in the hospital has also facilitated children's adjustment. Preparation, individualized to coping style and developmental level, may be done via talks explaining procedures, visits, books, puppet shows, and films.

Infancy

Hospitalized infants younger than 6 months are most upset by the change in the usual routine. It is helpful to have the parents do as much of the caregiving as possible and to arrange for consistent nurses. For the older infant who has formed strong differential attachments, separation is traumatic, and stranger anxiety adds to the infant's distress. Infants respond to the emotional reactions of the primary caregivers. An anxious, tense parent will be less able to soothe and comfort her child than one who is calmer and in better control. Most pediatric hospital settings encourage parents to "live in" while their infant is hospitalized.

Early Childhood

Hospitalized children ages 1–3 years may react to separation from their parents by rejecting parents when they visit, being aggressive toward medical staff, regressing in bowel and bladder control, and/or refusing to eat. If parents are absent, children may develop depression, sleep disturbance, diarrhea, or vomiting. Maximizing parental presence and providing the child with familiar items from home, especially the child's favorite toy or blanket (transitional object), are helpful. Developmentally, this is a time of increasing exploration and autonomy. Unfortunately, toddlers frequently attempt to master their environment by becoming more oppositional. Offering the child as much predictability, consistency, and control (choices) as possible can reduce distress and resistance.

For children ages 3–5 years, separation from parents by hospitalization is difficult, even for a child who is comfortably able to separate in other circumstances. Anesthesia and surgery are especially frightening because of normal developmental fears of bodily injury. Children believe that illness and painful treatments are punishment for real or fantasized misbehavior. When possible, preparation by simple explanations and a visit to the hospital may help. Frequent presence of a parent is important. Following the acute phase of illness, if understandably anxious parents attempt to protect the child's health by limiting the child's activities, including delaying school enrollment, unintended increased passivity, dependence, and fearfulness in the child may result.

School Age

Children ages 6–12 years usually tolerate acute illness and hospitalization relatively well, especially when they are prepared, their parents are present, and the child's preceding development was on course. Even so, the child may have irrational explanations of illness (e.g., that she is being punished or that his parents were unable to protect him). Peer relationships become paramount during this period, and peers notice and may comment on physical appearance and capabilities. Medically ill children become aware of their differences and limitations; occasionally their friendships are affected. School attendance and participation are major developmental tasks of childhood; medically ill children have more academic problems than their non-medically ill peers. These problems may be caused by the effects of the illness and its treatment, decreased expectations in the classroom, school absences, or psychosocial stressors. Reactions often include regression or oppositional behavior.

Adolescence

Adolescents have more realistic fears regarding the outcome of illness, such as changes in appearance or inability to continue favorite activities. An injury may make impossible a planned career (e.g., sports, the military). Loss of autonomy and privacy is especially painful for adolescents. The adolescent should be given some control over treatment to avoid struggles between the patient and the caregiver and to convey that the adolescent's opinions are important. Occasionally, lack of adherence to medical requirements can be an expression of suicidal ideation, particularly when the consequences could be life threatening. Physical appearance and sexuality emerge as priorities during adolescence, and patients with sequelae of illness, injury, or treatment may need additional support.

Chronic Illness

The Child's Reaction

Chronic illness can lead to interference with academic, social, and recreational development. Autonomy and control of the child's own

body are jeopardized, and self-esteem may be lowered. Especially in adolescence, peers have little tolerance for differences in appearance or behavior. Children and adolescents with chronic illness without disability are twice as likely as control children to have a psychiatric disorder. Those with disability as well as chronic illness are even more likely to have emotional symptoms, ADHD, social isolation, and school performance problems. Central nervous system disorders are more likely to have psychiatric sequelae. Episodic illness is more stressful than persistent medical disorders because of the unpredictability of the problem and the need to respond to sudden changes in physical condition. Psychiatric distress may present as an increase in dependent, fearful behavior; an escalation in risk-taking or acting-out behaviors; or, in older children and adolescents, a tendency to become angry, hostile, and isolative. Somatic symptom disorder, manifested by headaches, recurrent abdominal pain, limb pain, chest pain, or fatigue that is affected by psychological factors, interacts with symptoms of medical disorders and may be difficult to distinguish. The combination can lead to increased physical disability. Catastrophic injury and devastating illness can lead to symptoms of PTSD in both the pediatric patient and the parent. Support should be provided to the child, parent, and medical staff. Psychiatric disorder is not inevitable in chronically ill children and adolescents. Knight et al. (2015) assessed mental health problems in matched pairs of youth with diabetes and systemic lupus erythematosus and found depression symptoms in 23%, suicidal ideation in 15%, and anxiety in 27% of participants.

The Parents' Reaction

The most critical factor in the child's ability to cope with chronic illness is the response of the family. Medical illness in a child tends to exaggerate all the strengths and weaknesses of the family. At the time of diagnosis, the parents must go through a period of mourning with stages similar to those following a death: anger, denial, grief, and resignation. Medical problems in a child may be viewed by the parent (and others) as a negative reflection on the parent. Parents feel

guilty, especially for genetic diseases or pregnancy complications. The psychological status of parents is strongly related to their perception of the child's illness. Clinicians must, therefore, attend to the parents' understanding of the disorder and all of its implications. Additional caregiving and financial burdens may stress parents beyond their ability to cope. Chronic illness increases the strain on a marriage. The parents' anger, resentment, guilt, and/or denial may interfere with their ability to communicate with each other and to work with the pediatric team. Parents may distance themselves from the child, become overly close or intrusive, or alternate between the two. Mothers of children with chronic illness are at increased risk for depression.

The Terminally Ill Child or Adolescent

When a young person is dying, parents or medical personnel may make misguided attempts to "protect" the child. These are perceived as in the best interests of the child but more often relate to the difficulties adults have with a child's death. Although young patients may not fully understand their clinical situation, children are astute observers of emotions in their parents, nurses, and doctors. If the child is not dealt with honestly, his or her imagination can be even worse than the reality. Children understand the permanence of death by age 10 or 11 years, although they may not understand the relationship between it and the biological aspects of their illness. Parents or physicians may need to tell children about their terminal illness in stages, as understanding progresses and the child is able to cope with the knowledge. Children are often more concerned about immediate details of treatment and its effect on them than about ultimate survival. The child or adolescent should be given the opportunity, but not be forced, to talk about disease, disability, or death. Drawing, painting, or imaginative play may be useful symbolic outlets and means of communication.

The terminal phase of an illness leads to a recapitulation in parents of the feelings of denial, anger, grief, depression, and guilt that followed the initial diagnosis. Discussions about death between

parent and child should be encouraged as a way to prepare the child, answer his or her questions and fears, and say good-bye. If a child is terminally ill for a long time, anticipatory mourning may be completed, and some parents gradually detach, leaving the child no emotional place in the family. Similarly, physicians may inadvertently avoid the child and family when aggressive treatment is no longer an option and the medical team is resigned to the patient's death. Alternatively, if a child who is expected to die recovers, parents often experience a "Damocles syndrome," living in constant fear of disease exacerbation and death. Other parents come into conflict with medical caregivers by demanding additional active treatments that are medically considered to be futile. A hospital ethics committee or palliative care consultation may be helpful.

Adherence to Treatment

Lack of adherence to or compliance with medical procedures and regimens can be a major problem in the care of children and adolescents. Factors in the child and parent, family dynamics, the nature of the treatment, and the relationship between the child and family and the medical team can all contribute to poor adherence. Severe nonadherence may warrant a full psychiatric evaluation and treatment of psychiatric disorder(s) and/or family dysfunction.

Adherence is facilitated by giving the child or adolescent as much responsibility for the treatment as appropriate, gradually increasing responsibility as interest, understanding, and behavior control improve. The child should participate in decision making and be offered as many choices as possible. Explanations can be targeted to the child's ability to understand. The normal compulsiveness of children ages 6–12 years can be harnessed to develop a habit of charting or record keeping related to the treatment. If children or adolescents are able to develop their own relationships with medical personnel, adherence may be facilitated by avoiding the struggles for independence inherent in the parent–child relationship. Adults should minimize power struggles and, whenever possible, negotiate and solve problems with the young person. At times, a child's non-

adherence may signal a parent's ambivalence about or even opposition to treatment.

Treatment

Psychotherapy

Supportive individual, family, and/or group education and psychotherapy are often valuable for the patient and parents. Instruction in social problem-solving and coping skills may be beneficial.

Support Groups

Support groups for children and adolescents and for parents, both separately and as families, may be helpful. These groups provide emotional support and education on challenges the disease is likely to present. Chronically ill children may benefit from disease-specific camps and recreation programs with medical supervision, which permit them more normal activities and provide a respite for parents.

Pharmacotherapy

Indications for the use of pharmacotherapy in medically ill children and adolescents do not differ from those in medically healthy children. Medication choice and management decisions are, however, affected by the illness and should be made in conjunction with the pediatric specialist. Risk of side effects is higher because of both medication interactions and illness. Disorders that are the direct consequence of medical illness, including delirium and organic affective states, should be addressed by treating the primary cause. Situational and anticipatory anxiety related to medical procedures may benefit from hydroxyzine, diazepam, or alprazolam if psychosocial intervention is insufficient. Because benzodiazepines may cause paradoxical agitation in children, a test dose may be indicated. Treatable depression should be actively sought, because depression is not an inevitable response to illness and may dramatically impede recovery.

Behavior Modification

Techniques such as behavior contracting with contingency management and self-monitoring with self-reinforcement are invaluable in improving medical and behavioral adherence. Children who refuse or are unable to swallow oral medication can be taught to take pills using instruction, modeling, contingent rewards, and a behavior-shaping protocol that involves successively larger candies or placebos (Patel et al. 2015).

Cognitive-behavioral techniques can be used to ameliorate a variety of chronic physical symptoms and symptom-related behaviors. For example, in treating headache, the antecedents and consequences of pain are determined (by keeping a pain diary, if the child is old enough), and attempts are made to modify events and situations that precipitate or positively reinforce pain. The clinician works with the patient, parents, teachers, pediatrician, and significant others to emphasize functioning normally despite pain as well as stress management techniques. Reductions in pain and pain-related behaviors give patients a sense of control and mastery and a return to age-appropriate activities. *Stress inoculation* helps to prevent anxiety in children before medical and dental procedures and reduces anxiety, pain, or discomfort connected to repeated procedures such as chemotherapy injections, bone marrow aspirations, and spinal taps in chronically ill children. Stress inoculation consists of multicomponent cognitive-behavioral approaches, including education, modeling procedures, systematic desensitization, hypnosis, contingency management, imagery, and breathing exercises.

Hypnosis is readily used in children, who are generally more hypnotizable than adults. Different techniques are appropriate for children of different ages. Hypnosis is used in treating physical symptoms with a psychological component or managing severe physical symptoms (pain, nausea) associated with a medical disorder or treatment.

Relaxation training for children and adolescents is used to manage pain in pediatric migraine, juvenile rheumatoid arthritis, and hemophilia. These techniques can result in decreased subjective experience of pain, reduced need for analgesics, and even improved

mood, self-esteem, and physical and social functioning. Relaxation techniques are also used in treating asthma or cystic fibrosis in patients who hyperventilate.

■ CHILDREN OF PSYCHIATRICAL- LY ILL PARENTS

Risks and Resilience

Children of psychiatrically ill parents are at increased risk for developing a psychiatric disorder themselves, due to both genetic and environmental risk factors. Psychiatric illness in a parent can impact his or her parenting abilities, create marital discord, and disrupt the family environment. Poor decision making or decreased functioning may result in inadequate supervision or neglect. Family disruption may lead to divorce or placement of the child outside of the home. A population-based study in Denmark found that children of parents with schizophrenia, bipolar disorder, or depression were less likely to live in a nuclear family and more likely to live in a single parent-headed household. There was also an increased risk of living with neither parent, especially if a parent has schizophrenia (Ranning et al. 2016). The risk of psychopathology in the child is related to the severity and chronicity of psychiatric illness in the parent.

Children may be particularly distressed when their parents are admitted to a psychiatric hospital. Younger children may show sleep disturbance, decreased appetite, attention-seeking behavior, separation anxiety, crying (especially at bedtime), and social withdrawal. Adolescents are better able to verbalize their fears, guilt, and concerns about themselves and their parents.

Children whose parents are depressed are at increased risk for the development of psychopathology, as well as behavioral, academic, and health problems. Parents who are depressed may be less affectionate and emotionally unavailable to their children, often resulting in elevated rates of depression in their children. These children are more likely to experience a variety of internalizing symptoms, including recurrent feelings of guilt, interpersonal problems, and dif-

difficulties with attachment. Remission in maternal depression with treatment is associated with a decrease in symptoms in the child (Weissman et al. 2006).

A 7-year longitudinal study found that 75% of children of a parent with bipolar disorder qualified for a psychiatric diagnosis, most commonly bipolar spectrum disorder, substance use disorder, anxiety disorder, disruptive behavior disorder, or ADHD (Axelson et al. 2015). Children who have a parent with schizophrenia are more likely to have abnormalities in attention and information processing, even before any overt symptoms emerge. Children of patients with anxiety disorders are at increased risk of an anxiety disorder themselves, as well as fears and worries, school difficulties, somatic complaints, and social isolation.

A significant minority of children of alcoholic parents have increased behavior problems, especially conduct problems, hyperactivity, impulsivity, hypersensitivity to stimuli, poor self-control, inattention, and emotional symptoms such as anxiety and depression. Prenatal alcohol exposure is a common nongenetic cause of intellectual disability and can contribute to the development of learning, language, and behavioral disabilities. Children of alcohol abusers are more likely to develop alcohol use problems themselves, and the disinhibition associated with substance use places them at greater risk for suicide, accidents, unplanned pregnancy, HIV exposure, and school failure.

Some “resilient” children not only resist and cope with unusual stress but also thrive and succeed despite adversity. Protective factors include a cohesive and emotionally supportive family environment and external support systems, such as caring extended family members, other adults, and/or institutional supports (e.g., school or community agencies). These children often have a strong commitment to extracurricular activities and interpersonal relationships and tend to be confident and self-reliant. They are realistic in their assessment of the family and its limitations, and understand their own coping style when dealing with parental psychiatric illness.

Adolescence is a particularly high-risk period for the development of major depression in the offspring of parents with mood dis-

order. Successful adaptation is characterized by close, confiding relationships; persistence and success in school and work; and involvement in activities. They are able to separate themselves, cognitively and emotionally, from their parent and their parent's illness, while often functioning as caregivers in the family. Coping skills include accurate cognitive appraisal of the stress to be dealt with, realistic assessment of their responsibilities and powers, and an understanding of the parent's illness (especially that they are not responsible for their parent's depression).

Interventions

Parents can promote resilient traits in their children and moderate the emotional consequences of their psychiatric illness. Communication between spouses and among family members can improve the level of understanding and reduces guilt and emotional distress. Children should be referred to appropriate support groups. Individual strengths in the child should be encouraged and nurtured. When parents are receiving psychiatric treatment, the treating clinician should be alert to possible effects of the illness on the children. Parental psychiatric hospitalization is particularly stressful. There should be at least one family session near the time of admission to explain the need for hospitalization and the anticipated treatment course. Optimally, a member of the treatment team interviews each child to assess developmental level, school performance, peer relationships, coping skills for dealing with and understanding parental illness, and the possible presence of psychiatric symptoms. Efforts can be made to enrich and strengthen the child's support system. Placement with a relative or in foster care may be necessary until the parent can resume child-care responsibilities.

■ REFERENCES

- Amato PR, Gilbreth JG: Nonresident fathers and children's well-being: a meta-analysis. *J Marriage Fam* 61:557-573, 1999
- American Professional Society on the Abuse of Children: Practice Guidelines: Descriptive Terminology in Child Sexual Abuse Medical Evaluations. Oklahoma City, OK, APSAC, 1995

- American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. Arlington, VA, American Psychiatric Association, 2013
- Axelson D, Goldstein B, Goldstein T, et al: Diagnostic precursors to bipolar disorder in offspring of parents with bipolar disorder: a longitudinal study. *Am J Psychiatry* 172(7):638–646, 2015 25734353
- Baeza I, Correll CU, Saito E, et al: Frequency, characteristics and management of adolescent inpatient aggression. *J Child Adolesc Psychopharmacol* 23(4):271–281, 2013 23647136
- Buehler C, Krishnakumar A, Stone G, et al: Interparental conflict styles and youth problem behaviors: a two-sample replication study. *J Marriage Fam* 60:119–132, 1998
- Cerel J, Fristad MA, Weller EB, Weller RA: Suicide-bereaved children and adolescents: a controlled longitudinal examination. *J Am Acad Child Adolesc Psychiatry* 38(6):672–679, 1999 10361784
- Children's Hospital Association: Expert Guidelines: Role of Psychologists in Assessment and Treatment of Obese Youth. Lenexa, KS, Children's Hospital Association, 2012
- Cohen JA, Deblinger E, Mannarino AP, Steer RA: A multisite, randomized controlled trial for children with sexual abuse-related PTSD symptoms. *J Am Acad Child Adolesc Psychiatry* 43(4):393–402, 2004 15187799
- Emery RE, Laumann-Billings L, Waldron MC, et al: Child custody mediation and litigation: custody, contact, and coparenting 12 years after initial dispute resolution. *J Consult Clin Psychol* 69(2):323–332, 2001 11393609
- Fristad MA, Jedel R, Weller RA, Weller EB: Psychosocial functioning in children after the death of a parent. *Am J Psychiatry* 150(3):511–513, 1993 8434672
- Knight A, Weiss P, Morales K, et al: Identifying differences in risk factors for depression and anxiety in pediatric chronic disease: a matched cross-sectional study of youth with lupus/mixed connective tissue disease and their peers with diabetes. *J Pediatr* 167(6):1397–403.e1, 2015 26316371
- Lindberg L, Santelli J, Desai S: Understanding the decline in adolescent fertility in the United States, 2007–2012. *J Adolesc Health* 59(5):577–583, 2016 27595471
- MacPherson HA, Cheavens JS, Fristad MA: Dialectical behavior therapy for adolescents: theory, treatment adaptations, and empirical outcomes. *Clin Child Fam Psychol Rev* 16(1):59–80, 2013 23224757
- Marzullo LR: Pharmacologic management of the agitated child. *Pediatr Emerg Care* 30(4):269–275, quiz 276–278, 2014 24694885

- Mirensky TL: Bariatric surgery in youth. *Endocrinol Metab Clin North Am* 45(2):419–431, 2016 27241972
- Ogden CL, Carroll MD, Lawman HG, et al: Trends in obesity prevalence among children and adolescents in the United States, 1988-1994 through 2013-2014. *JAMA* 315(21):2292–2299, 2016 27272581
- Patel A, Jacobsen L, Jhaveri R, Bradford KK: Effectiveness of pediatric pill swallowing interventions: a systematic review. *Pediatrics* 135(5):883–889, 2015 25896843
- Posner K, Brown GK, Stanley B, et al: The Columbia-Suicide Severity Rating Scale: initial validity and internal consistency findings from three multi-site studies with adolescents and adults. *Am J Psychiatry* 168(12):1266–1277, 2011 22193671
- Ranning A, Munk Laursen T, Thorup A, et al: Children of parents with serious mental illness: with whom do they grow up? A prospective, population-based study. *J Am Acad Child Adolesc Psychiatry* 55(11):953–961, 2016 27806863
- Saul J, Rodgers RF: Wellness, not weight: changing the focus in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 55(1):7–9.e1, 2016 26703902
- Siegel RS, Brandon AR: Adolescents, pregnancy, and mental health. *J Pediatr Adolesc Gynecol* 27(3):138–150, 2014 24559618
- Weissman MM, Pilowsky DJ, Wickramaratne PJ, et al; STAR*D-Child Team: Remissions in maternal depression and child psychopathology: a STAR*D-child report. *JAMA* 295(12):1389–1398, 2006 16551710
- Weller EB, Weller RA, Fristad MA, et al: Should children attend their parent's funeral? *J Am Acad Child Adolesc Psychiatry* 27(5):559–562, 1988 3182618
- Wildeman C, Emanuel N, Leventhal JM, et al: The prevalence of confirmed maltreatment among US children, 2004 to 2011. *JAMA Pediatr* 168(8):706–713, 2014 24887073
- Wolchik SA, Sandler IN, Millsap RE, et al: Six-year follow-up of preventive interventions for children of divorce: a randomized controlled trial. *JAMA* 288(15):1874–1881, 2002 12377086
- Zheng W, Li XB, Tang YL, et al: Metformin for weight gain and metabolic abnormalities associated with antipsychotic treatment: meta-analysis of randomized placebo-controlled trials. *J Clin Psychopharmacol* 35(5):499–509, 2015 26280837

■ ADDITIONAL READING

- Beardslee WR, Martin JL: Children of parents with psychiatric and substance abuse disorders, in Dulcan's Textbook of Child and Adolescent Psychiatry. Edited by Dulcan MK. Washington, DC, American Psychiatric Publishing, 2010, pp 623–635
- Cohen JA, Mannarino AP: Posttraumatic stress disorder and persistent complex bereavement disorder, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 345–364
- Cohen JA, Bukstein O, Walter H, et al; AACAP Work Group on Quality Issues: Practice parameter for the assessment and treatment of children and adolescents with posttraumatic stress disorder. *J Am Acad Child Adolesc Psychiatry* 49(4):414–430, 2010 20410735
- DeMaso DR, Martini DR, Cahen LA, et al; Work Group on Quality Issues: Practice parameter for the psychiatric assessment and management of physically ill children and adolescents. *J Am Acad Child Adolesc Psychiatry* 48(2):213–233, 2009 20040826
- Giles LL, Martini DR: Psychiatric emergencies, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 621–635
- Goldstein TR, Brent DA: Youth suicide, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 571–584
- Joshi PT, Cullins LM, Southammakosane CA: Child abuse and neglect, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 539–558
- Kelly JB: Children's adjustment in conflicted marriage and divorce: a decade review of research. *J Am Acad Child Adolesc Psychiatry* 39(8):963–973, 2000 10939225
- Martini DR, Lowry KW, Lavigne JV: Psychiatric aspects of chronic physical disorders, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 411–433
- Miller AL, Rathus JH, Linehan MM: Dialectical Behavior Therapy With Suicidal Adolescents. New York, Guilford, 2007

- Pumariega AJ, Rothe E, Mian A, et al; American Academy of Child and Adolescent Psychiatry (AACAP) Committee on Quality Issues (CQI): Practice parameter for cultural competence in child and adolescent psychiatric practice. *J Am Acad Child Adolesc Psychiatry* 52(10):1101–1115, 2013
- Walsh F: Family transitions: challenges and resilience, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 637–652

PSYCHOPHARMACOLOGY

■ SPECIAL ISSUES FOR CHILDREN AND ADOLESCENTS

General Principles

In the treatment of psychiatric disturbances in children and adolescents, psychopharmacology may be a key part of a multimodal treatment plan, the primary intervention, or an adjunct to other forms of treatment. The clinician must educate the family regarding the disorder, treatment options, and the child's needs at each developmental stage. The clinician also must consider the meaning of the prescription and administration of a drug to the child, the family, and the child's teachers and peer group.

Evidence-based clinical practice of pediatric psychopharmacology is impeded by the relative lack of double-blind randomized placebo-controlled trials (RCTs). Many medications that initially appeared to be effective in anecdotal reports, case series, and open trials were not shown to be more effective than placebo in double-blind studies. In evaluating the medical literature on drug effects, it is important to distinguish between *statistically* significant and *clinically* significant effects: to know whether the target symptoms are reduced to near-normal levels or merely changed and to determine the clinical meaning of the change. Within a heterogeneous sample, some patients' conditions may improve and others may worsen, resulting in nonsignificant change as a group. On the other hand, statistically significant group changes may translate into only modest

improvement in individual patient functioning and may not be worth the effort, expense, and potential risk of the treatment.

Simultaneous use of more than one medication (“polypharmacy”) should be cautious, judicious, and as rare as possible because of the increased risk of significant side effects, toxic drug interactions, and difficulty assessing effectiveness of each drug. It is important to give a medication an adequate trial of long enough duration for that drug and at a sufficient dose to determine effectiveness before switching to or adding another drug.

Developmental Toxicity

The interactions between drug treatment and physical, cognitive, social, and emotional development may produce unique or especially severe side effects. All psychotropic medications have the potential for cognitive toxicity. Because some drug treatments last for years, there is a risk of chronic and cumulative effects. Cognitive blunting can impair developing academic skills, social skills, and self-esteem even before physical side effects are observed, especially in young children. Young patients may have behavioral toxicity (i.e., the worsening of preexisting behaviors or affective states or the development of new symptoms). Behavioral toxicity typically abates after dose adjustments or change of drug.

Indications and Dosing

Once a drug is approved for any indication, the U.S. Food and Drug Administration (FDA) regulates only the company’s advertising of the drug, not the prescribing behavior of physicians. Because in the past, pharmaceutical companies had little incentive to test drugs in children, the majority of psychopharmacological agents and indications (and most drugs used in pediatrics as well) lack pediatric labeling and are “unapproved” or “off-label” for children. For preschool-age children, even fewer drugs have FDA psychiatric indications. As a result, the FDA guidelines as published in the *Physicians’ Desk Reference* (PDR) cannot be relied on for appropriate indications, age ranges, or doses for children. Although lack of approval for an age group or a

disorder does not imply improper or illegal use, it is prudent to inform the family of these labeling issues, as well as of evidence in the literature for safe and effective use. The physician should make clinical judgments based on the medical literature rather than the PDR.

Dose may be determined by titration using effectiveness and side effects; within guidelines based on age, weight, or blood levels; or by extrapolation from adult doses. Although it is ideal for pediatric drug doses to be derived from data on children, such dosing studies are rare. A useful general principle is “start low and go slow.”

The age-dependent processes of drug absorption, distribution, protein binding, metabolism, and elimination influence optimal dose size and schedule (Vitiello 2017). Young children absorb some drugs more rapidly than do adults, leading to higher peak levels. Age-related factors that may influence distribution include uptake by actively growing tissue and proportional size of organs and tissue masses. Prior to puberty, children have, compared with adults, relatively more body water and less fat (which serves as a reservoir for lipid-soluble compounds). In children, drugs such as lithium (which are primarily distributed in body water) have a proportionally larger volume of distribution and therefore a lower concentration. By age 1 year, glomerular filtration rate and renal tubular secretion mechanisms reach adult levels. Hepatic enzyme activity develops early, and the rate of drug metabolism is then related to liver size. Both kidney and liver parenchyma in children are larger than in adults, relative to body size. Therefore, compared with adults or older adolescents, children generally require a larger dose per kilogram of body weight of drugs that are primarily metabolized by the liver. They may also require divided doses to minimize fluctuations in blood level. Compared with adults, children also tend to have less protein binding of drugs, leaving a greater proportion of the drug biologically active.

Outcome

Children often differ from adults in both effectiveness and adverse effects of a medication. Assessing the risk–benefit ratio, especially for long-term treatment, is particularly complicated in children and adolescents. Many scientific unknowns persist regarding drug ef-

fects during development. Evaluation is complicated by a variety of nonpharmacological effects, including nonspecific therapeutic effects of evaluation and treatment; expectancy in the patient, parent, or teacher; changes in the environment; and the natural course of the disorder (e.g., the waxing and waning of symptoms in Tourette's disorder).

The clinician must specify target symptoms and obtain emotional, behavioral, cognitive, and physical data at baseline and during treatment. Treatment effects can be assessed by interviews of and standardized symptom rating scales by the patient, the parent, and other caregivers (e.g., teachers, inpatient nurses); direct observation in the office, waiting room, or classroom; physical examination; and, as appropriate, specific cognitive, learning, or laboratory tests. The clinician must actively question regarding both therapeutic and adverse effects because many young patients will not report them spontaneously, and parents may not notice.

The interactions between treatment and the environment raise complex issues for children and adolescents. To assess medication effects, the clinician must evaluate and monitor the adult and peer environments as well as the patient. It is important to know what the therapeutic contacts and any clinical change mean to the patient and to family members. The adults (parents, teachers, or staff in an inpatient unit or residential treatment setting) who care for children may misinterpret the youngster's response to the environment as indicating a need for medication or an improvement that is a result of medication. Some of these adults mistakenly seek to use psychotropic medication alone to control or eliminate a child's troublesome behavior, instead of investigating the family or institutional dynamics that may be provoking and maintaining such behavior or implementing more time-consuming, difficult, and expensive therapeutic or behavioral management strategies or changes in living situation.

Adherence to Treatment

Effective treatment requires a therapeutic alliance and the cooperation of the patient, parents, school personnel, and often other caregivers.

Adherence to a treatment regimen can be reduced by factors such as lack of perceived need for treatment, failure to understand the disorder, poor organization, lack of money, misunderstanding of instructions, or refusal to cooperate. Overly complex schedules of drug administration may make accurate administration nearly impossible. Media attention to alleged inappropriate use of medications has made some families and teachers highly resistant to pharmacotherapy.

Some children cannot or will not swallow pills. Some drugs are available in elixir or dissolving tablet form or can be dissolved in juice or sprinkled on pudding or applesauce. Problems with this method of administration may include unpleasant taste, chemical interaction resulting in precipitation of the medication, and inaccurate dosing. Information on shaping the ability to swallow pills is available on the Web at <http://www.massgeneral.org/children/assets/pdf/Lurie-Center-Teaching%20Your%20Child%20to%20Swallow%20a%20Pill.pdf>.

Ethical Issues

The careful clinician attempts to balance the risks of medication, the risks of the untreated disorder, and the expected benefits of medication relative to other treatments. With the exception of drugs used for attention-deficit/hyperactivity disorder (ADHD), it is generally preferable that there be substantial clinical experience with a new drug in adults before it is used with children and young adolescents.

Consent for pharmacotherapy in children is a complex issue that can be made even more difficult if the parents or guardians are in conflict (e.g., in angry divorces or with children in the custody of child welfare). Informed consent is best considered an ongoing process rather than a single event. "Assent" to medication use is considered possible to obtain from a patient older than 7 years, with more complex understanding at older ages. Formal consent forms are less useful than a documented discussion of therapeutic options with potential risks and benefits and an opportunity for questions and answers. Published information sheets for parents and teachers are available to supplement discussions with the prescribing clinician about specific medications (Dulcan and Ballard 2015).

■ STIMULANT MEDICATIONS

The number of available stimulant preparations (Table 17–1) has increased dramatically, which enhances the clinician's ability to tailor the medication regimen to the patient. They include a variety of forms of methylphenidate and amphetamine. Unfortunately, increasingly narrow payor formularies often drastically limit available choices.

Indications and Efficacy

Stimulant medications, including various formulations of methylphenidate, dexamethylphenidate, dextroamphetamine, amphetamine mixed salts, and lisdexamfetamine (see Table 17–1), are the first-line treatment for *ADHD*. Stimulants retain their effectiveness for ADHD treatment in adolescents (and adults). In preschool children, stimulant effect is more variable, and the rate of side effects is higher, especially sadness, irritability, clinging, insomnia, anorexia, and repetitive behaviors (Greenhill et al. 2006). Stimulants can improve attention and reduce excessive distractibility in patients with ADHD, whether or not hyperactivity is present. Many children whose symptoms respond positively to stimulants, however, continue to have deficits such as specific learning disabilities and gaps in knowledge and skills caused by inattention, poor social skills, and family problems. Intensive behavior modification may add to stimulant effect or permit the use of a lower dose of medication, but it is often difficult to implement and sustain behavioral treatment and to transfer improvements from one setting to another. The National Institute of Mental Health (NIMH) Multimodal Treatment of ADHD (MTA) study showed that optimally titrated methylphenidate was more effective for core ADHD symptoms than intensive behavioral therapy, optimal methylphenidate treatment plus behavioral treatment was more effective than behavioral treatment alone, and each of the MTA treatments was more effective than treatment as usual in the community (mostly stimulants, but at lower doses, fewer doses per day, shorter duration of treatment, and less close monitoring) (MTA Cooperative Group 1999).

TABLE 17-1. **Stimulant preparations**

Drug	Formulation	Doses
Methylphenidate Generic (IR) (3–4 hours)	Tablet	5, 10, 20
	Oral solution	5 mg/5 mL, 10 mg/5 mL
	Chewable tablet	2.5, 5, 10
Methylin (IR) (3–4 hours)	Oral solution (grape)	5 mg/5 mL, 10 mg/5 mL
Ritalin (IR) (3–4 hours)	Tablet	5, 10, 20
Aptensio XR (10–12 hours)	Capsule with beads	10, 15, 20, 30, 40, 50, 60
	Ratio of IR:LA=40:60	
Metadate ER (6–8 hours) and generic	Wax matrix tablet	10, 20
Ritalin LA (8–9 hours) and generic	Capsule with beads (sprinkle)	10, 20, 30, 40
	Ratio of IR:LA=50:50	
Metadate CD (6–8 hours)	Capsule Diffucap with beads (sprinkle)	10, 20, 30, 40, 50, 60
	Ratio of IR:LA=30:70	
Concerta (10–12 hours) and generics	Oros Osmotic Controlled Release	18, 27, 36, 54
	Ratio of IR:LA=4:14	18 mg Oros=5 mg IR bid or tid
Quillivant XR (8 hours)	Extended release oral suspension	25 mg/5 mL–60 mL, 120 mL,
	25 mg/mL	150 mL, 180 mL
	Ratio of IR:LA=20:80	Bottles: 300 mg (60 mL), 600 mg, 750 mg, 900 mg

TABLE 17-1. Stimulant preparations (*continued*)

Drug	Formulation	Doses
Methylphenidate (<i>continued</i>)		
QuilliChew ER (8 hours)	Chewable tablet	20, 30, 40
Daytrana	Transdermal system (patch)	10, 15, 20, 30
	Worn for 9 hours for 12-hour duration	
Dexmethylphenidate (only the <i>dextro</i> isomer of methylphenidate)		
Focalin (3–4 hours) and generic	Tablet	2.5, 5, 10
Focalin XR (10–12 hours)	Capsule with beads (sprinkle)	5, 10, 15, 20, 25, 30, 35, 40
	Ratio of IR:LA=50:50	
Generic XR (10–12 hours)	See Focalin XR	5, 10, 15, 30, 40
Dextroamphetamine		
Generic (IR) (3–5 hours)	Tablet	5, 10
Dextroamphetamine sulphate (3–5 hours)		
ProCentra	Oral solution (bubblegum)	5 mg/mL
Zenzedi	Tablet	2.5, 5, 7.5, 10, 15, 20, 30
Generic	Tablet	5, 10
Dexedrine Spansule (6–8 hours) and generic	Capsule with beads (sprinkle)	5, 10, 15
Dyanavel XR (8–10 hours)	Liquid (bubblegum)	2.5 mg/mL
Vyvanse (lisdexamfetamine dimesylate) (12 hours)	Prodrug (may open capsule—dissolve in water or orange juice or mix in yogurt and give immediately; do not divide capsules)	20, 30, 40, 50, 60, 70

TABLE 17–1. **Stimulant preparations (*continued*)**

Drug	Formulation	Doses
Mixed salts amphetamine		
Adderall (IR) (3–5 hours) and generic	Tablet Ratio of isomers <i>d:l</i> =3:1	5, 7.5, 10, 12.5, 20, 30
Adderall XR (10–12 hours) and generic	Capsule with beads (sprinkle) Ratio of IR:LA=50:50	5, 10, 15, 20, 25, 30
Adzenys XR-ODT and generic (10–12 hours)	Orally disintegrating tablet Dissolve in mouth (fruit flavor) Ratio of isomers <i>d:l</i> =3:1 Ratio of IR:LA=50:50	3.1, 6.3, 9.4, 12.5, 15.7, 18.8 3.1 mg Adzenys=5 mg Adderall XR
Evekeo (4–6 hours)	Tablet Ratio of isomers <i>d:l</i> =1:1	5, 10

Note. CD=extended-release; ER=extended-release; IR=immediate-release; LA=long-acting; ODT=orally disintegrating tablet; XR=extended-release.

Long-acting capsules may not be chewed, crushed, or divided. A chart with color pictures of medications that have FDA approval for ADHD is available online from www.adhdmedicationguide.com.

When *conduct disorder* or *oppositional defiant disorder* coexists with ADHD, stimulant medication can reduce defiance, negativism, and impulsive verbal and physical aggression. In children and adolescents with *intellectual disability*, stimulants are effective in treating ADHD target symptoms, although the amount of improvement is smaller and side effects are more common. Stimulants may also reduce symptoms of inattention, impulsivity, and overactivity when ADHD is present along with *autism spectrum disorder* (ASD) or *intellectual disability*.

Boys and adults, with or without ADHD, have similar cognitive and behavioral responses to comparable doses of stimulants, except that children report feeling “funny,” whereas adults report euphoria (Donnelly and Rapoport 1985). Stimulants act in the brain by inhibiting the dopamine transporter, thereby increasing the amount of dopamine available in the synapse. Norepinephrine reuptake from the synapse is also blocked. Amphetamine also releases dopamine into the synapse. Stimulants may preferentially increase neurotransmitter activity at inhibitory synapses and in inhibitory brain areas. Stimulants are absorbed rapidly from the gut and metabolized rapidly. Stimulant medication effects on ADHD occur primarily as the drug is being absorbed and as the plasma (and brain) concentration is rising.

There are no useful predictors of whether an individual patient with ADHD will respond to a specific medication. Neurological soft signs, electroencephalogram (EEG), brain scans, neurochemical measures, or genetic tests are not useful predictors of response to stimulants. Although prior studies were mixed, the NIMH MTA study showed that children who have anxiety comorbid with ADHD do not have a reduced response to stimulants. More than 90% of patients with ADHD have some positive response to at least one of the stimulant medications. A substantial number of children respond to one stimulant but not another (Ramtvedt et al. 2013). Stimulant effects on individual target symptoms (Table 17–2), however, vary greatly from child to child and even from one symptom to another in a single patient. A given dose may produce improvement in some areas but no change or worsening in others. Therefore, if one stim-

TABLE 17-2. **Clinical effects of stimulant medications**

Motor effects	Reduce activity to level that fits the context Decrease excessive talking, noise, and disruption Decrease fidgeting and finger-tapping Improve handwriting Improve fine motor control
Social effects	Reduce off-task behavior Improve ability to play and work independently Reduce impulsivity Decrease intensity of behavior Reduce bossiness Reduce verbal and physical impulsive aggression Improve (but not normalize) peer social status Reduce noncompliance and defiance with adults Improve parent-child interactions Parents and teachers respond with less controlling and more positive behavior
Cognitive effects	Improve effort and attention, especially to boring tasks Increase on-task behavior Reduce distractibility Reduce impulsivity Increase quantity and accuracy of academic work

ulant is ineffective, another should be tried before using another drug class. Methylphenidate is the most commonly used and best-studied stimulant. Amphetamine disadvantages include negative attitudes of pharmacists and higher potential for abuse. Compared with methylphenidate, amphetamine may have a slightly greater incidence of side effects such as growth retardation, appetite suppression, and compulsive behaviors.

Dexmethylphenidate (Focalin) contains only the *dextro* isomer of methylphenidate. Compared with conventional *d,l*-methylphenidate, half the dose of dexmethylphenidate is just as effective, with fewer side effects, and perhaps longer duration. Longer-acting stimulant preparations are now most commonly used. They are especially

appealing for children in whom the duration of action of the standard formulations is very short (2.5–3.0 hours); when severe rebound occurs; or when administering medication every 4 hours (or at school) is inconvenient, inconsistent, stigmatizing, impossible, or insufficiently supervised to prevent diversion of the drug. The many different formulations of methylphenidate and amphetamine (see Table 17–1) are useful in tailoring treatment to each patient.

Although short-term efficacy of stimulants has been clearly demonstrated, it is much more difficult to demonstrate long-term effects. Existing studies have had methodological problems (e.g., naturalistic treatment assignment; comorbidity; premature termination of drug; doses too high, too low, or poorly timed; inconsistent adherence to medication; individual variation in response; insensitive outcome instruments). A long-term prospective randomized controlled trial assigning children with ADHD to stimulant medication or placebo or other treatment is neither ethical nor feasible.

Initiation and Ongoing Treatment

The decision to medicate a child with ADHD is based on the child's inattention, impulsivity, and often hyperactivity that are not due to another treatable cause and that are persistent and severe enough to cause functional impairment at school and usually at home and with peers. For safety reasons, parents must be willing to monitor the medication and to attend appointments with the child. An important part of treatment is education of the child, family, and teacher, including explicitly debunking common myths about stimulant treatment. Stimulants do *not* have a paradoxical sedative action, do *not* lead to drug abuse, and *do* continue to be effective after puberty.

Multiple outcome measures that use more than one source and setting are essential. The clinician obtains baseline data from the school on behavior and academic performance before initiating stimulant medication. The physician should work closely with parents on adjusting the size and timing of doses and obtain frequent reports from teachers and annual academic testing. Brief symptom checklists, such as the Child Attention Problems (CAP) Rating Scale (see

Tables 3–5 and 3–6) or the IOWA Conners Scale (see Chapter 3, “Neurodevelopmental Disorders”), are useful in gathering weekly data from teachers.

Many experts recommend a systematic stimulant titration using the full range of doses—for example, 5, 10, 15, and 20 mg of methylphenidate (highest dose omitted for very young or small children). Body weight serves only as a rough guide. Doses of amphetamine or dexamethylphenidate (Focalin) are half the milligrams of methylphenidate. A strategy that is preferred by many clinicians and parents is to start stimulant medication at a low dose and increase by half (if necessary and if pill is scored) or whole pills (within the usual recommended range) every week or two, monitoring response (ideally including teacher ratings) and side effects. By age 3 years, children’s absorption, distribution, protein binding, and metabolism of stimulants are similar to those of an adult, although adults may experience more side effects than do children at equivalent therapeutic doses.

The onset of clinical effect for immediate-release methylphenidate and amphetamine is within 30 minutes after each dose. A single dose is usually effective for 3–4 hours. Of the immediate-release forms, amphetamine typically has about an hour longer therapeutic duration compared with methylphenidate. A typical regimen for the immediate-release preparations would be thrice-daily dosing, with medication given after breakfast, after lunch, and after school. Starting with only a morning dose may be useful in assessing drug effect, by comparing morning and afternoon school performance. The need for an after-school dose or for medication on weekends is individually determined by considering target symptoms. A third dose after school has been shown to improve behavior without increasing sleep problems (Kent et al. 1995). Most children with moderate or severe symptoms of ADHD need full coverage, all day and all week. The long-acting formulations are typically given once a day, in the morning, with supplementation with immediate release if necessary, timed according to target symptoms.

If the initial stimulant drug choice is not effective or not well tolerated, more than half of nonresponders to methylphenidate or amphetamine respond to the other stimulant.

Medical monitoring includes, at a minimum, pulse rate and blood pressure initially and at times of dose change; weight at baseline, during titration, and two to three times a year; and height at baseline and then several times a year. Prior to prescribing stimulant medication, the clinician should take a cardiac history, including any patient structural abnormalities, chest pain, palpitations, fainting, and reduced exercise tolerance and family history of arrhythmias, early cardiac death, or sudden unexplained death. Electrocardiogram (ECG) monitoring prior to and during stimulant treatment has been controversial. Following the FDA warnings in 2006 regarding sudden death in patients with structural cardiac abnormalities or other serious heart problems, the American Heart Association issued a statement that recommended obtaining an ECG prior to stimulant treatment (Vetter et al. 2008). This recommendation lacked specific supporting evidence, and soon after, the American Academy of Pediatrics (supported by the American Academy of Child and Adolescent Psychiatry) issued a statement (Perrin et al. 2008) concluding that routine pretreatment cardiac tests are not indicated unless there is known or suspected cardiac disorder or symptoms. If there are such findings on history or pediatric examination, an ECG and often a pediatric cardiology consultation are indicated. Several large naturalistic studies have not found increased cardiac risk when stimulants are used for ADHD in youth. The clinician should look for and inquire about tics at baseline and at every visit. Periodic re-evaluation of the need for a dose increase or decrease or a change in timing of administration will optimize improvement.

Although pharmacological tolerance has been reported occasionally, medication administration is often irregular, and lack of adherence should be considered when medication appears to become ineffective. The child should not be responsible for his or her own medication, because youngsters with ADHD are impulsive and forgetful at best, and most dislike the idea of taking medication, even when they can verbalize its positive effects and report few if any side effects. They will often avoid, “forget” to take, surreptitiously spit out, or simply refuse to take a dose of medication. Apparent decreased drug effect may be caused by a reaction to a stressful

change at home or school, lower efficacy of a generic preparation, or abatement of an initial positive placebo effect. If tolerance does occur, the other stimulant may be substituted.

The duration of medication treatment is individually determined by whether drug-responsive target symptoms are still present. Treatment may be required through adolescence and into adulthood. If behavioral symptoms are not severe outside of the school setting, the young person may have an annual drug-free trial of at least 2 weeks, or even the whole summer if symptoms are mild. If school behavior and academic performance are stable, a carefully monitored trial off medication during the school year (but *not* at the beginning) will provide data on whether medication is still needed.

Risks and Side Effects

Most side effects are similar for all stimulants (Table 17–3). Giving medication after meals reduces appetite suppression. Difficulty falling or staying asleep may be due to ADHD symptoms, oppositional refusal to go to bed, separation anxiety, stimulant effect or rebound, or excess environmental stimulation (especially electronic devices). Preexisting sleep problems are common in patients with ADHD. Stimulants may either worsen or improve irritable mood. Other than mildly elevated blood pressure, cardiovascular side effects are exceedingly rare. Adverse effects may result if a child chews one of the long-acting forms instead of swallowing it.

The use of stimulants in patients with a personal or family history of tics has been controversial because of concern that new, persistent tics might be precipitated, especially in those children who are at genetic risk. The physician must balance the impairment resulting from tics with that from ADHD symptoms, considering the efficacy and side-effect profile of alternative medications. With appropriate informed consent and careful clinical monitoring, a stimulant (methylphenidate) may remain the first choice (Friedland and Walkup 2015). Tics are extremely common in children with ADHD, with or without stimulant medication, and tend to wax and wane. A comprehensive review of controlled trials concluded that new onset

TABLE 17-3. Side effects of stimulant medications

Common side effects (try dose reduction)	Anorexia Weight loss or slowed weight gain (may be worse with amphetamine) Irritability (may be worse with amphetamine) Abdominal pain Headaches Easy crying
Less common side effects	Mildly elevated blood pressure Insomnia (may be worse with amphetamine) Dysphoria (may be worse with amphetamine) Social withdrawal Impaired cognitive test performance (especially at very high doses) Decrease in expected weight gain Rebound overactivity and irritability (try adding small afternoon or evening dose) Habits such as skin picking and nail biting Allergic rash, hives, or conjunctivitis Transient motor tics (may be worse with amphetamine)
Infrequent side effects	Dizziness Nausea Anxiety and fearfulness Stuttering
Rare but potentially serious side effects (usually reversible)	Exacerbation or precipitation of tics (may be worse with amphetamine) Depression Growth retardation Tachycardia Hypertension Psychosis with hallucinations Stereotyped activities or compulsions Raynaud's phenomenon Priapism

TABLE 17-3. **Side effects of stimulant medications (*continued*)**

Reported with Concerta only	Capsule lodged in throat
Reported with Daytrana only	Skin irritation under patch Allergy to methylphenidate Skin depigmentation

or worsening of tics is no greater while taking stimulant medication than with placebo (Cohen et al. 2015). There is some evidence that very high doses of amphetamine can increase tic severity, which may persist (Kurlan 2002).

Although stimulant-induced growth retardation has been a concern, and weight and height should be monitored, any decreases in expected weight gain and growth are small and rarely clinically significant (Faraone et al. 2008). The magnitude may be dose related and may be greater with dextroamphetamine than with methylphenidate. Attenuation of effect has been reported. Medication-free summers (if clinically appropriate) may facilitate height or weight normalization.

Rebound effects such as increased excitability, activity, talkativeness, irritability, and insomnia, beginning 3–15 hours after a dose, may be seen as each dose or the last dose of the day wears off or for up to several days after sudden withdrawal of high daily doses of stimulants. These effects may resemble a worsening of the original symptoms. Management strategies include increasing structure after school, giving a dose of medication in the afternoon that is smaller than the midday dose, or using a long-acting formulation. If the rebound effect is severe, an alternative agent (atomoxetine, guanfacine, or clonidine) can be added to or substituted for the stimulant.

Clinically relevant contraindications to the use of stimulant medication are schizophrenia or other acute psychosis, glaucoma, or recent stimulant drug abuse. When potential abuse of stimulant medication by the patient—or, more often, by peers or family members—is a concern, Concerta may be a good choice, as its once-a-day ad-

ministration is easier to supervise, and the physical characteristics of the capsule contents (methylphenidate mixed with an osmotic “sponge”) make it impossible to crush and snort or inject. Vyvanse is also not abusable, because of the need for enzyme cleavage of the amino acid before the amphetamine is active.

No evidence indicates that stimulants as clinically prescribed decrease the seizure threshold or precipitate bipolar disorder.

A selective serotonin reuptake inhibitor (SSRI) may be added to a stimulant for the treatment of comorbid ADHD and depression or anxiety (for symptoms remaining after stimulant treatment).

Stimulants are used in combination with atomoxetine, clonidine, or guanfacine to treat symptoms resistant to a stimulant alone. This is theoretically appealing because of complementary actions and nonoverlapping side-effect profiles.

■ ATOMOXETINE

Atomoxetine (Strattera) is a specific norepinephrine reuptake inhibitor with minimal affinity for other receptors or transporters.

Indications and Efficacy

Atomoxetine was the first nonstimulant approved by the FDA for the treatment of ADHD. RCTs have shown efficacy in inattentive and hyperactive-impulsive symptoms of ADHD in preschoolers through adolescents as reported by parents and teachers (Kelsey et al. 2004; Michelson et al. 2001). Atomoxetine is effective in some youth who have not responded to stimulant treatment. In RCTs, about half of subjects are atomoxetine responders (Newcorn et al. 2009). In responders, the effect size is lower than that typically seen with stimulants. Atomoxetine improves comorbid oppositional symptoms (improvement greater at higher doses) (Newcorn et al. 2005) and comorbid anxiety symptoms. In addition to having a different side-effect profile, which is useful for patients who do not tolerate stimulants, atomoxetine provides 24-hour coverage, which is especially helpful with the evening and early-morning symptoms not cov-

ered by stimulants. Atomoxetine might be the first choice for patients with ADHD if stimulants (which are controlled substances) are refused or not feasible or for patients with preexisting severe early-morning behavior problems, insomnia, or low weight.

Initiation and Ongoing Treatment

Atomoxetine may be given once a day (morning or evening) or divided into two doses (with breakfast and dinner) to reduce side effects. The starting dose is 0.3 mg/kg/day (usually in a single daily dose) for a week, with the dosage then increased over 1–3 weeks to an initial target dose of 1.2 mg/kg/day. The FDA maximum dose is 1.4 mg/kg/day or 100 mg, whichever is less, but the off-label maximum dose (demonstrated safe in clinical trials) is 1.8 mg/kg/day. The maximum response at a particular dose may be delayed several weeks or even months, requiring patience (especially when compared with the immediate effects of the stimulants). Tapering is not required when discontinuing this medicine. Atomoxetine is metabolized via the cytochrome P450 2D6 pathway. Some patients are poor metabolizers because of genetic makeup or drug interactions, but there are no adverse clinical consequences and genotyping is not recommended.

Atomoxetine may be combined with a stimulant in patients who are partially responsive to each.

Risks and Side Effects

Side effects are generally mild and include sedation, decreased appetite, nausea, abdominal pain, and dizziness. Taking the medication with food improves tolerability. There are slight increases in blood pressure and pulse (rarely clinically significant), but no QTc prolongation on the ECG. Atomoxetine does not increase tics (Allen et al. 2005) or lower the seizure threshold. The capsules must not be opened, as the contents are caustic to the eye. Rare side effects include syncope, vomiting, constipation, irritability, and, very rarely, priapism and (in patients with comorbid bipolar disorder) manic activation. Atomoxetine has two FDA black box warnings: one regard-

ing extremely rare severe liver injury and one (based on weak evidence) of increased hostility and aggression or suicidal ideation. Routine liver function monitoring is not recommended, but parents should be instructed that if jaundice, unexplained abdominal symptoms, pruritus, or dark urine appear, the medication should be stopped and the physician should be called so that liver function tests can be obtained. Atomoxetine does not have a physiological withdrawal syndrome if stopped suddenly, although symptoms of ADHD are likely to return.

■ GUANFACINE AND CLONIDINE

Guanfacine hydrochloride and clonidine are α -adrenergic agonists developed for the treatment of hypertension. They have been used as second- or third-line treatments for ADHD, despite relatively sparse supporting research. More recently, extended-release formulations of guanfacine (Intuniv) (once a day) and clonidine (Kapvay) (twice a day) have been studied in RCTs and given FDA indications for the treatment of ADHD, either alone or in addition to a stimulant medication. These longer-acting formulations have more research to support their use, and they avoid daily rebound of symptoms or gaps in effectiveness as well as the inconvenience of needing to be given three or four times a day. Also, like atomoxetine, they provide coverage for symptoms of ADHD in the early morning and the evening, when stimulants are not active. Comparing the two drugs, in general, guanfacine has more evidence for treatment effect in inattention, and clonidine is more sedating.

Indications and Efficacy

Attention-Deficit/Hyperactivity Disorder

Clonidine improves frustration tolerance and compliance and reduces emotional outbursts in ADHD. It may be used at bedtime to decrease ADHD overarousal or oppositional behavior or to ameliorate insomnia caused by stimulant effect or rebound.

In a study comparing immediate-release methylphenidate and clonidine, the combination, and placebo, methylphenidate was most effective by teacher report and class observation, with the fewest adverse effects of the active treatments. Clonidine was less effective for ADHD symptoms and caused sedation, but it demonstrated improvement on parent ratings. The combination showed little evidence of added benefit and no increase in side effects (Palumbo et al. 2008). A small RCT comparing immediate-release methylphenidate and clonidine and the combination in youth with ADHD plus aggressive oppositional or conduct symptoms found that all three treatment conditions were associated with significant improvement in attention, impulsivity, and oppositional and conduct symptoms on parent and teacher rating scales and laboratory measures, with few differences among groups. There were no safety findings related to the combination (Connor et al. 2000).

An RCT (Jain et al. 2011) of two doses of extended-release clonidine (0.2 mg/day and 0.4 mg/day) versus placebo in children with hyperactive/impulsive or combined type ADHD found efficacy by parent report of both doses of clonidine, starting at 2 weeks after the target dose was reached.

Two RCTs have demonstrated efficacy of extended-release guanfacine as monotherapy for both hyperactive/impulsive and inattentive symptoms of ADHD as well as for oppositional symptoms by parent, teacher, and clinician ratings (Biederman et al. 2008; Sallee et al. 2009). Extended-release guanfacine has also been shown to be effective for symptoms of ADHD when added to stimulant medication in stimulant partial responders (Wilens et al. 2012). Immediate-release guanfacine combined with dexamethylphenidate has been demonstrated to be more effective in treating ADHD than either guanfacine or the stimulant alone (McCracken et al. 2016).

Tourette's Disorder

A modest-sized RCT in children with tic disorders and ADHD showed that guanfacine (immediate release) significantly improved (compared with placebo) teacher and clinician ratings of ADHD

symptoms, led to decreased errors on a continuous performance test (while placebo subjects had increased errors), and decreased tic severity by 31% (compared with 0% in the placebo group) (Scahill et al. 2001).

The efficacy of clonidine for Tourette's disorder per se has been controversial. However, an RCT in children with both chronic tics and ADHD showed efficacy of clonidine in reducing both tics and impulsivity/hyperactivity (Tourette's Syndrome Study Group 2002).

Other Symptoms

Guanfacine or clonidine may be useful in the management of aggressive behavior, impulsivity, oppositional behavior, self-injurious behavior, and agitation in a variety of conditions, including posttraumatic stress disorder and intellectual disability. Extended-release guanfacine has been shown to decrease symptoms of inattention and hyperactivity/impulsivity in children with ASD (Scahill et al. 2015).

Initiation and Ongoing Treatment

Before guanfacine or clonidine is initiated, a cardiovascular history and a physical examination (including blood pressure and pulse rate) are needed. An ECG may be advisable for preschool children, although the American Heart Association guidelines do not mandate ECG monitoring with these drugs (Vetter et al. 2008). If there is a family history of diabetes or clinical symptoms in the patient, a fasting blood glucose may be indicated. These drugs are contraindicated in patients with a history of syncope, bradycardia, or heart block.

One mg of guanfacine is equivalent to 0.1 mg of clonidine. One of these α -adrenergic agonists can be switched to the other by a gradual cross-taper.

Immediate-release guanfacine is started at 0.5 mg/day, with the dosage gradually increased by 0.5 mg every 3–4 days to a maximum daily dose of 4 mg in three divided doses. Extended-release guanfacine is given once a day, titrated from 1 mg/day, with the dos-

age increased by 1 mg/day each week to a maximum of 4 mg/day. The target dose is 0.05–0.08 mg/kg/day. The FDA approved maximum dose is 4 mg/day or 0.12 mg/kg/day, whichever is less, although adolescents may require up to 6 mg/day (Wilens et al. 2015). The extended-release form is not mg-to-mg equivalent with immediate-release guanfacine. Discontinuation should be by gradual taper (1 mg every 3–7 days).

Immediate-release clonidine is started at a low dose of 0.05 mg/day at bedtime and titrated gradually over 2–4 weeks to 0.15–0.40 mg/day (0.003–0.01 mg/kg/day) in four divided doses (for immediate release), in order to minimize sedation. The medication should be continued for 2–8 weeks at the maximal dose before determining effectiveness (Pliszka et al. 2000). Clonidine is available in a transdermal skin patch that may improve ability to administer medication as prescribed and may reduce effects on blood pressure by smoothing blood levels. The transdermal form is effective for only 4–5 days in children, compared with 7 days in adults (Hunt 1987). Clonidine extended-release (Kapvay) is started at 0.1 mg/day in two divided doses and the dosage increased by 0.1 mg/day each week to a target of 0.4 mg/day, divided into every-12-hour doses. Doses of immediate- and extended-release clonidine are not mg-for-mg equivalent. Any form of clonidine should be discontinued by gradual tapering to avoid withdrawal tachycardia, hypertension, headache, and nausea.

Risks and Side Effects

The extended-release forms must not be crushed, chewed, or broken.

Guanfacine has fewer and milder side effects (primarily irritability, sedation, headache, and abdominal pain) and less rebound than clonidine. Surprisingly, guanfacine used for the treatment of ADHD in children has little effect on pulse or blood pressure, although syncope has been reported rarely in patients with ADHD or Tourette's disorder.

The most troublesome side effect of clonidine is somnolence, which is most prominent early in treatment and generally decreases

after 4–8 weeks. Patients may experience daily rebound hyperactivity and irritability or insomnia when the immediate-release form is used. The extended-release formulation of clonidine may produce less somnolence and rebound. The most serious potential adverse effects of clonidine are cardiovascular and include hypotension, bradycardia, rebound tachycardia and hypertension, and asymptomatic ECG conduction changes. Clonidine may worsen symptoms of dysphoria. Less common side effects include headache, abdominal pain, irritability, nightmares, rash, and decreased glucose tolerance. The skin patch often causes a local hypersensitivity reaction and may produce allergic sensitization.

In the past, anecdotal reports of sudden death in children who at one time had been taking both methylphenidate and clonidine generated some concern, but evidence linking the drugs to the deaths is tenuous (Wilens et al. 1999). Pending further clarification, extra caution has been advised when treating preschoolers or children with cardiac disease, when combining clonidine with additional medications, or when adherence to consistent medication administration is uncertain.

■ ANTIDEPRESSANTS

Medications called *antidepressants* that are commonly used for the treatment of a variety of disorders in children and adolescents are listed in Table 17–4. Evidence related to their pediatric use is growing, although more research is needed, particularly studies comparing drugs of the same class. Interestingly, despite the name of the drug class, in children and adolescents, evidence is stronger for benefit in anxiety disorders than in depression. The safest and most commonly used class of antidepressants is the *selective serotonin reuptake inhibitors* (SSRIs). Antidepressants with other mechanisms of action are collectively referred to as *atypical antidepressants*. Because of problematic adverse effects and potential lethality in overdose, *tricyclic antidepressants* (TCAs) are very rarely used in children, with the exception of clomipramine for treatment-resistant obsessive-compulsive disorder (OCD).

TABLE 17-4. **Antidepressant medications most often used in children and adolescents**

Generic name	Brand name	Typical ^a daily dose <18 years	Indications supported by RCT <18 years
Selective serotonin reuptake inhibitors			
Citalopram	Celexa	20–40 mg	Depression
Escitalopram	Lexapro	10–20 mg	Depression
Fluoxetine	Prozac	10–80 mg	Depression, GAD, OCD, SM, SoAD
Fluvoxamine	Luvox	50–200 mg	GAD, OCD, SAD, SoAD
Paroxetine	Paxil	10–60 mg	Depression, OCD, SoAD
Sertraline	Zoloft	25–250 mg	Depression, GAD, OCD, SAD, SoAD
Atypical antidepressants			
Bupropion	Wellbutrin (IR, SR)	150–300 mg	ADHD
	Wellbutrin XL	150–300 mg children 150–450 mg adolescents	
Desvenlafaxine	Pristiq	50–100 mg	None
Duloxetine	Cymbalta	40–60 mg ^b	GAD
Mirtazapine	Remeron	7.5–45 mg	None
Trazodone	Oleptro	25–150 mg	None
Venlafaxine	Effexor (IR, XR)	37.5–300 mg	Depression, GAD, SoAD

TABLE 17-4. Antidepressant medications most often used in children and adolescents (*continued*)

Generic name	Brand name	Typical ^a daily dose <18 years	Indications supported by RCT <18 years
Tricyclic antidepressant^b			
Clomipramine	Anafranil	50–200 mg 1–3 mg/kg	OCD

Note. ADHD=attention-deficit/hyperactivity disorder; GAD=generalized anxiety disorder; IR=immediate release; OCD = obsessive-compulsive disorder; RCT=randomized controlled trial; SAD=separation anxiety disorder; SM=selective mutism; SoAD=social anxiety disorder; SR=sustained release; XL=extended release; XR=extended release.

^aStarting doses are lower. Within this range, children require lower doses than do adolescents.

^bDivided doses.

Mechanisms of Action

Selective serotonin reuptake inhibitors, as the name implies, block the presynaptic reuptake of serotonin. Chronic use of SSRIs also results in the down-regulation of serotonin receptors, which then modulates serotonergic transmission. The SSRIs differ in how they affect other neurotransmitter receptors and therefore the strength of serotonin reuptake selectivity.

The atypical antidepressants vary in mechanism of action. Bupropion (Wellbutrin) has a novel chemical structure and inhibits norepinephrine and dopamine reuptake. Venlafaxine (Effexor), and its derivative, desvenlafaxine (Pristiq), inhibit serotonin and norepinephrine reuptake and weakly inhibit dopamine uptake. Duloxetine (Cymbalta) is a selective serotonin-norepinephrine reuptake inhibitor with weak dopaminergic reuptake inhibition. Mirtazapine (Remeron) is unique in its noradrenergic and specific serotonergic mechanisms. Trazodone (Oleptro) has a distinctive mechanism of action as both a serotonin reuptake inhibitor and a mixed serotonergic agonist and antagonist.

Indications and Efficacy

Depression

SSRIs are the first-line medication treatment for youth with depression. In RCTs, the response rate for SSRIs is 50%–60%, versus 30%–50% for placebo. The high “placebo response” (more accurately described as nonspecific positive response to being in a clinical trial) has made it difficult to demonstrate superiority of antidepressants to placebo in pediatric depression.

The FDA has approved pediatric indications in major depressive disorder (MDD) for fluoxetine (age 8 years and older) and escitalopram (age 12 years and older). Fluoxetine has the best-documented clinical trial efficacy. Two RCTs of escitalopram yielded positive responses in adolescents only (thus resulting in the FDA indication only for older children) (Emslie et al. 2009; Wagner et al. 2006). Escitalopram was shown to be superior to placebo in the continua-

tion treatment of depression (Findling et al. 2013). Paroxetine is rarely used, because of mixed evidence on efficacy and concern about side effects in youth. The results of two studies of sertraline (reported as one merged study) (Wagner et al. 2003) support efficacy in depressed youth. Data from citalopram trials are inconsistent. Fluvoxamine has not been studied in pediatric depression. Case reports suggest that duloxetine may be beneficial in adolescents with pain and depression (Meighen 2007). TCAs have not been shown to be efficacious in pediatric depression.

Research evidence is lacking with regard to comparative effectiveness among the SSRIs, so choice is based on target symptoms, comorbidity, side effects, half-life, interactions with other medications, positive familial experience with or response to the agent, patient/family preference, cost, insurance formulary, or a trial of the drug in the particular patient. For example, fluoxetine's long half-life may be an advantage if missed doses are likely, but side effects or drug-drug interactions can persist for weeks after fluoxetine is discontinued, and fine-tuning of the dose may be difficult. If a patient does not respond well to the first one chosen, a trial with another SSRI is typically indicated. Treatment of Resistant Depression in Adolescents (TORDIA) found that in teens with MDD or dysthymia who had failed to respond to an adequate trial of an SSRI, a medication change with added cognitive-behavioral therapy (CBT) was more effective than switching medications alone. Switching to fluoxetine or citalopram was as effective as switching to venlafaxine, and venlafaxine produced more side effects (Brent et al. 2008).

Obsessive-Compulsive Disorder

Multiple SSRIs, including fluoxetine, fluvoxamine, paroxetine, and sertraline, have been shown in RCTs to be efficacious in the treatment of OCD in youth. Drugs with FDA indications for OCD in children and adolescents are sertraline (age 6 years and older), fluoxetine (7 years and older), fluvoxamine (8 years and older), and the TCA clomipramine (10 years and older), which is a third-line choice due to safety and tolerability issues. Clomipramine is generally reserved for use

as an augmentation strategy to an SSRI, and is less commonly used for this purpose than are atypical antipsychotics.

Anxiety Disorders

The first-choice medication for treating pediatric anxiety disorders is an SSRI, although medication is considered to be only one component of a multimodal treatment plan and is recommended only when symptoms are moderate to severe with significant functional impairment. In a large RCT of 7- to 17-year-olds with separation anxiety disorder (SAD), generalized anxiety disorder (GAD), or social anxiety disorder, combination treatment (CBT plus sertraline) was more effective than either monotherapy or placebo, and each active treatment (CBT or sertraline) as monotherapy was significantly superior to placebo (Walkup et al. 2008). Consensus among experts is that there is no evidence that one SSRI is more effective than another for these disorders. Fluoxetine was shown to be effective for youth with GAD and social anxiety disorder, but not for youth with SAD (Birmaher et al. 2003). The efficacy of fluoxetine in selective mutism is suggested by one small RCT (although most subjects had remaining symptoms) (Black and Uhde 1994). A multisite RCT demonstrated efficacy of fluvoxamine in children and adolescents with social anxiety disorder, SAD, or GAD (Walkup et al. 2001). In a large study of youth with SAD, GAD, or social anxiety disorder, sertraline was superior to placebo (Walkup et al. 2008). Sertraline was also efficacious in a small RCT for GAD in children and adolescents (Rynn et al. 2001). Wagner et al. (2004) found youth with social anxiety disorder to respond favorably to paroxetine. Despite the lack of research, SSRIs are sometimes used to treat panic disorder in youth, based on extrapolation from use in adults.

There is also a growing body of evidence to support the use of the atypical antidepressants in pediatric anxiety disorders. A large RCT in youth with social anxiety disorder found venlafaxine extended release (ER) to be superior in efficacy to placebo (March et al. 2007). Venlafaxine ER also has shown efficacy in GAD (Rynn et al. 2007). Duloxetine demonstrated efficacy in pediatric GAD

(Strawn et al. 2015); this resulted in FDA labeling for pediatric GAD. Duloxetine is the only medication that has FDA labeling for any pediatric anxiety disorder (excluding OCD, formerly categorized as an anxiety disorder). A very small case series of youth with chronic and cyclical vomiting showed a reduction in vomiting with use of mirtazapine (Coskun and Alyanak 2011).

Attention-Deficit/Hyperactivity Disorder

Bupropion has been shown to be effective in both children and adults with ADHD, with an effect size equivalent to that of methylphenidate (Barrickman et al. 1995).

Several TCAs have demonstrated efficacy in the treatment of ADHD. Given that a variety of safer alternative drugs (stimulants in many formulations, atomoxetine, guanfacine, clonidine) have demonstrated efficacy in ADHD, a TCA (most often nortriptyline) would be a fourth- or fifth-line choice for the treatment of ADHD, used only if other drugs are ineffective or not tolerated.

Autism Spectrum Disorder

There is some evidence that the SSRIs such as fluoxetine (Hollander et al. 2005) may be useful in reducing aggression, temper problems, self-injurious behavior, and stereotyped behavior seen in patients with ASD, although other studies have negative results. An RCT of citalopram failed to show reduction in repetitive behaviors in children with ASD (King et al. 2009). The atypical antipsychotics are more commonly used to treat severe irritability or aggression in youth with ASD.

Enuresis

Enuresis can be addressed by the child's primary care physician or in a multimodal treatment program that addresses any medical contributing factors and implements behavioral strategies. Imipramine has been replaced by desmopressin (DDAVP) as the drug of first choice in the treatment of enuresis.

Initiation and Ongoing Treatment

Typical therapeutic daily dose ranges are listed in Table 17–4. The general principle “start low and go slow” applies. In medication trials for anxiety disorders, often relatively high doses were used to achieve symptom reduction. Thus, for anxiety disorders, the principle can be expanded to “start low, go slow, and aim high.” Many of the medications are metabolized in the liver; thus, patients with hepatic insufficiency may require additional monitoring when antidepressants are prescribed. An especially careful assessment of all prescribed and over-the-counter (OTC) medications needs to be completed prior to treatment initiation, because many of the antidepressants have drug–drug interactions.

SSRIs do not require medical assessment prior to or during treatment other than pregnancy testing, when indicated. Particularly in patients with anxiety disorders, it is advisable to use very low initial doses and slow titration in order to minimize side effects that could lead to refusal to take medication. A useful starting strategy is to prescribe the lowest dose pill available, or half a pill, if scored. The liquid formulation of fluoxetine may be used for very gradual titration. Fluoxetine is sometimes prescribed on an every-other-day basis because it has a long half-life and active metabolites. A very long-acting form (Prozac Weekly) is available for patients who may be nonadherent with daily medication, once a therapeutic dose is found and tolerability in that patient is established.

Bupropion is available in immediate-, sustained-, and extended-release forms. The immediate-release formulation may be used to start treatment; conversion to a longer-acting formulation may be done once an adequate dose has been attained. Because of the side effect of increased seizure risk (likely dose-related), bupropion is contraindicated in youth with epilepsy, eating disorders, or other risk factors for seizures. Use of venlafaxine, desvenlafaxine, or duloxetine may result in blood pressure elevation; thus, blood pressure monitoring is necessary. Mirtazapine may cause elevation in liver enzymes and leukopenia; if a child develops flulike symptoms, checking the hepatic enzymes and complete blood count may be indicated.

The goal of treatment should be full remission of symptoms. After a child or adolescent has been asymptomatic for 6–12 months, the clinician may consider slowly tapering the medication. Timing of a discontinuation trial should avoid stressful life events or predictably difficult times such as a new school year. Many factors should be considered in determining the need for maintenance therapy in depression. These factors include the severity of the initial depressive episode (i.e., presence of suicidal thinking, psychosis, or severe functional impairment), number and severity of prior depressive episodes, chronicity, comorbidity, family psychopathology, and presence of support. It is recommended, based on extrapolation from adult data, that youth with multiple depressive episodes continue maintenance medication indefinitely.

Risks and Side Effects

With SSRI use, side effects are mild, typically emerge early in treatment, and resolve over time. Low starting doses and slow, gradual titration can limit the development of adverse effects. Common somatic side effects include headaches, anorexia, weight loss, weight gain, bruxism, nausea, tremors, drowsiness, and vivid or strange dreams. Increased activity levels can occur, perhaps related in part to akathisia. Symptoms of behavioral activation include restlessness, insomnia, social disinhibition, and agitation or aggression. Bipolar switching or manic reaction is a less common but potentially serious adverse effect of any antidepressant and can include changes in mood, sleep, behavior, and impulse control. All antidepressant medications, including SSRIs, have an FDA “black box warning” regarding risk of increased suicidal thoughts and behaviors. Although the increased risk is small, careful monitoring is necessary, especially as medication is started and following dose adjustments. In reported studies, children who reported new or increased suicidal thoughts or behaviors typically had preexisting risk factors. Some patients develop apathy or an amotivational syndrome after weeks or months of SSRI treatment, consisting of emotional blunting, decreased motivation, passivity, and loss of interest and energy. This can

superficially resemble sedation or worsening of depression, and parents and patients may not identify the cause. A small decrease in dose may be helpful. Pediatric clinicians are not used to considering sexual side effects, but the sexual dysfunction commonly associated with SSRIs (i.e., decreased libido, anorgasmia, and erectile dysfunction) may be distressing for adolescents. Bleeding or bruising is very rare but possible (Lake et al. 2000).

Because SSRIs inhibit the cytochrome P450 isoenzymes, there is considerable potential for adverse drug interactions. *Serotonin syndrome* is a very rare, potentially fatal reaction to the addition or increase in dose of a serotonergic agent (most often in combination with another prescribed or over-the-counter medication or an herbal remedy) characterized by extreme restlessness, agitation, fever, myoclonic jerking movements, severe hyperreflexia, clonus, fasciculation, nausea, vomiting, and diarrhea. Seizures, severe hypotension, ventricular tachycardia, and disseminated intravascular coagulation can occur in severe cases.

Although the margin of safety of SSRIs is greater than for other antidepressants, deaths have been reported following large ingestions of SSRIs.

Withdrawal symptoms, including dizziness, headache, chills, tiredness, nausea, vomiting, and diarrhea, may occur after sudden discontinuation of an SSRI. This is less of a problem with fluoxetine, because of its very long half-life, and is more likely with paroxetine and fluvoxamine. Mild withdrawal symptoms can occur even with missed doses in some youth. Citalopram and escitalopram have few significant drug interactions because of their weak hepatic cytochrome P450 enzyme inhibition, and they are good options in patients who are on multiple medications. Fluoxetine's long half-life reduces the risk of discontinuation withdrawal symptoms, but the extensive washout period required complicates a subsequent medication trial, if switching drugs is necessary. Among SSRIs, fluvoxamine likely has the lowest incidence of sexual side effects, but sleep disturbances can be a problem. Paroxetine requires exact adherence and slow taper if discontinued, because of its shorter half-life and increased risk for withdrawal symptoms.

The atypical antidepressants are also fairly well tolerated. They have the same general side-effect profile as do SSRIs, though they do not cause sexual side effects (except trazodone). Each atypical antidepressant may have distinctive side effects. Bupropion is associated with an increased seizure risk. Blood pressure elevations can be seen with venlafaxine, desvenlafaxine, and duloxetine. Trazodone is associated with the potentially serious side effect of priapism (i.e., prolonged painful erection of the penis). The atypical antidepressants, notably venlafaxine, should also be tapered slowly to avoid discontinuation symptoms. The FDA warning regarding risk of increased suicidal thinking and behavior also applies to the atypical antidepressants.

Common adverse effects of bupropion include irritability, insomnia, anorexia, and tic exacerbations. Less commonly, edema, rashes, and nocturia have been reported. Increased seizure risk was discussed above.

■ LITHIUM CARBONATE

Lithium carbonate, a naturally occurring salt, is the oldest mood stabilizer.

Indications and Efficacy

Mood Disorder

Lithium has an FDA indication for the treatment of mania in patients 12 years and older. The Collaborative Lithium Trials (CoLT) provide the most recent data on lithium's efficacy and safety in pediatric mania (Findling et al. 2011, 2015). Lithium may be considered in the treatment of bipolar affective disorder, mixed or manic, and for prophylaxis of bipolar disorder in children and adolescents who have a documented history of recurrent episodes. It is effective for acute stabilization in many adolescents with mania, although adjunctive antipsychotic medication is often required. Children and adolescents with mania tend to have a less dramatic response to lith-

ium than do adults, and mania with preadolescent onset tends to have a poorer response to lithium than does adolescent-onset mania (Strober et al. 1988). The Treatment of Early Age Mania (TEAM) study found risperidone to be more efficacious than lithium (Geller et al. 2012), although the side-effect profile in long-term use of risperidone in youth is concerning.

Aggression

Several small studies of youth with severe aggression, especially with impulsivity and explosive affect, showed lithium to be effective in reducing aggression, hostility, and tantrums. Lithium may be useful in intellectually disabled youth with severe aggression directed toward themselves or others.

Initiation and Ongoing Treatment

Lithium should not be prescribed unless the family is willing and able to consistently administer multiple daily doses and obtain lithium blood levels. In addition to the usual medical history and physical examination, complete blood count (CBC) with differential, electrolytes, thyroid function studies, calcium, blood urea nitrogen (BUN), and creatinine should be determined before lithium is started. A urinalysis and ECG also should be obtained. Girls of reproductive age should have a pregnancy test.

Lithium carbonate is the most commonly used formulation because of its reliable serum levels and reasonable cost. Immediate-release (lithium carbonate, 300 mg; Eskalith, 300 mg), controlled-release (Lithobid, 300 mg; Eskalith CR, 450 mg), and elixir (lithium citrate, 300 mg/5 mL) formulations are available. Because the slow-release formulations are not cleared as rapidly as regular lithium, twice-daily administration is sufficient, and more steady blood levels are achieved.

No systematic studies have been done in children to compare the efficacy and side effects of different dosing schedules. In prepubertal children, traditional practice is to start lithium at 300 mg/day for

several weeks and slowly increase the dosage to 900 mg/day in divided doses. Usual child and adolescent therapeutic doses range from 900 to 1,200 mg/day, although total daily doses of up to 2,000 mg may be required. Therapeutic levels can be safely attained in a much shorter time by using a weight-based dosing guide (Weller et al. 1986). Also, published nomograms can be used to calculate doses based on blood levels after single test doses (Alessi et al. 1994; Geller and Fetner 1989). The higher glomerular filtration rate in children compared with adults usually requires a higher milligram-per-kilogram dose before puberty. A weight-based pediatric titration strategy starts with 15–20 mg/kg/day in two or three divided doses. The total daily dose is increased by 300 mg every 4–5 days, based on clinical response, side effects, and serum levels. The titration strategy recommended based on the CoLT trial is for patients weighing at least 30 kg to begin treatment with lithium at a daily dose of 300 mg tid, followed by 300-mg weekly increases (an additional 300-mg increase during the first week), with monitoring of lithium levels and positive and adverse effects. The maximum allowable lithium level is 1.4 mEq/L (Findling et al. 2011).

Lithium's half-life (approximately 18 hours) could permit once-a-day dosing in adolescents. However, because children have more rapid lithium clearance than do adults, multiple divided doses may be necessary to maintain therapeutic levels. In addition, some patients have gastrointestinal distress when they take the entire daily dose at bedtime. Lithium is therefore usually given two or three times a day with meals, even though divided doses have the disadvantage of potentially decreasing adherence to the prescribed regimen.

Therapeutic levels in children are generally similar to those in adults: 0.6–1.2 mEq/L. Under usual circumstances, levels should not exceed 1.4 mEq/L. Peak serum levels will occur within 1–2 hours after ingestion. Steady-state serum levels are achieved after 5 days. To measure the serum level, blood should be drawn 8–12 hours after the last evening dose and before the first morning dose. Levels are obtained once or twice weekly during dose adjustment; levels are checked every 3–6 months after a stable dose has been reached.

Clinical status may suggest more frequent checks of the lithium level.

The clinician should periodically check BUN and creatinine or creatinine clearance because lithium may alter kidney function. A thyroid-stimulating hormone (TSH) test should be obtained every 4–6 months. The clinician must be alert to possible clinical signs of hypothyroidism that could be mistaken for fatigue or a retarded depression.

No studies have addressed the issue of how long to continue lithium. A naturalistic study (Strober et al. 1990) found that adolescents who discontinued lithium were three times more likely to relapse compared with those who continued taking the medication. Most relapses occurred within the first year after cessation of treatment. Once lithium is started, it seems advisable to continue administration for at least 6 months, and preferably a year. If studies of lithium termination in adults are applicable to children, an even longer duration of treatment might be considered. Experience in adult patients with worsening of cycling and decreased response to treatment following intermittent lithium use suggests special caution regarding discontinuation of mood stabilizers. Lithium should be discontinued by gradual tapering. The clinician should closely follow up with the patient after lithium is discontinued and should monitor the patient for signs of relapse so that episodes can be treated early.

Risks and Side Effects

Lithium is well tolerated by many children and adolescents, but younger children are more prone to side effects, especially at higher lithium doses and serum levels. The most common side effects in children (i.e., tremor, weight gain, headache, nausea, diarrhea) rarely require discontinuation of lithium. Polydipsia and polyuria may cause enuresis and prevent attainment of a therapeutic level. Lithium may produce goiter and/or hypothyroidism, which may have more significant consequences in developing children than in adults. Lithium effects on blood glucose level are controversial, but reactive hypoglycemia is possible. Acne may be induced or aggra-

vated. Hypokalemia is a very rare side effect that can be managed by dietary supplementation (e.g., two bananas, two large carrots, two cups of skim milk, half of a honeydew melon, or an avocado daily), which is preferable to taking potassium tablets that taste bad and can further irritate the gastrointestinal tract. Because of its teratogenic potential (highest risk in first trimester), lithium is relatively contraindicated in sexually active girls.

Toxicity is closely related to serum levels, and the therapeutic margin is narrow. The patient and the family should be told to call the doctor immediately if the patient develops a febrile or gastrointestinal illness, uses rigorous dieting to lose weight, or takes diuretics or nonsteroidal anti-inflammatory agents (often taken by adolescent girls to relieve menstrual distress). Lithium should be stopped while a patient has fever, vomiting, or diarrhea. Vigorous exercise in hot weather can lead to lithium toxicity, and parents should be cautioned to make sure the patient drinks enough water. Erratic consumption of large amounts of salty snack foods may cause wide fluctuations in lithium blood levels. Caffeine may decrease the lithium level.

■ ANTIPSYCHOTICS

“Antipsychotics” are used in children and adolescents to treat both psychotic and severe nonpsychotic mental and behavioral disorders. These medications are divided into two groups: the first-generation antipsychotics (FGAs), or “typicals” (also called “neuroleptics”), and the second-generation antipsychotics (SGAs), or “atypicals.” While all antipsychotics block dopamine D_2 receptors, the two groups differ in the extent to which they affect dopamine pathways implicated in psychosis, mania, tics, and aggression, as well as dopamine-related adverse effects on motor symptoms, prolactin secretion, and cognition. Antipsychotics also bind to varying degrees to serotonergic, histaminic, muscarinic, and α -adrenergic receptors, determining the therapeutic and side-effect profile of each particular drug.

Indications, Efficacy, and Use

Atypical antipsychotics are generally preferred for most pediatric indications because of their lower risk of potentially irreversible side effects such as tardive dyskinesia. Table 17–5 outlines the current FDA indications for the SGAs and selected FGAs that have grandfathered indications extrapolated from adult data.

Schizophrenia

Several typical and atypical antipsychotics have modest efficacy in children and adolescents with schizophrenia. Difference in efficacy among these drugs when used for schizophrenia has not been demonstrated, with the exception of clozapine, which has been shown to benefit patients who have been unresponsive to the other agents. Concerns about the development of tardive dyskinesia with long-term use of the typical antipsychotics and the prominence of negative symptoms in young patients with schizophrenia suggest preference for the atypicals, although these medications also have problematic side effects (see below). RCTs have demonstrated efficacy superior to placebo for agents in both groups of antipsychotics. Improvement in positive symptoms can be seen by the second week of treatment, but improvement in positive symptoms may not plateau for a month or longer, and negative symptoms may continue to improve for 6 months to a year.

Treatment is generally started with an SGA that has an FDA indication for schizophrenia in children and adolescents. (See Table 17–5 for indicated drugs, starting dose, and dose range.) If there is no response to a drug after a 3- to 4-week trial at adequate dose (and assured adherence), another antipsychotic may be tried. Because of its side-effect profile, clozapine is considered only after failed treatment with two or three of the other SGAs, as well as one of the typical neuroleptics.

Full efficacy may not appear for up to 6 months. Positive symptoms (delusions and hallucinations) tend to abate first, with improvements seen by the second week of treatment, plateauing after a month or longer. Improvement in cognitive symptoms (thought

TABLE 17-5. Antipsychotic medications

Drug (generic name)	Typical pediatric starting dose, mg	Typical pediatric dose range, mg	Pediatric indications in the United States	Side effects					
				Weight gain	EPS	↑Lipids, risk of diabetes	↑ Pro- lactin	↑QTc ^a	Sedation
Second-generation ^b									
ARI	2–5	10–30	Irritability associated with ASD , ages 6–17 years Bipolar I disorder , manic or mixed, ages 10–17 years (acute monotherapy, adjunctive with lithium or valproate, maintenance) Schizophrenia , ages 13–17 years (acute, maintenance) Tourette’s disorder , ages 6–17 years	0/+	+	0/+	0 ^c	0/+	0/+

TABLE 17–5. Antipsychotic medications (*continued*)

Drug (generic name)	Typical pediatric starting dose, mg	Typical pediatric dose range, mg	Pediatric indications in the United States	Side effects					
				Weight gain	EPS	↑Lipids, risk of diabetes	↑ Pro- lactin	↑QTc ^a	Sedation
Second-generation ^b (<i>continued</i>)									
ASE	2.5 bid	2.5–10	Bipolar I disorder , manic or mixed, ages 10–17 years (acute)	+	++	0/+	+	+	+
CLO	12.5	50–600	—	+++	0	+++	0	+	+++
ILO	NA (adults 1 mg bid)	12–24	—	+ / ++	0/+	+	0/+	++	0/+
LUR	SCZ: 40–80 BD depres- sion, autism: 20	40–120	Schizophrenia , ages 13–17 years	0/+	+ / ++	0/+	+	0/+	+ / ++

TABLE 17-5. Antipsychotic medications (*continued*)

Drug (generic name)	Typical pediatric starting dose, mg	Typical pediatric dose range, mg	Pediatric indications in the United States	Side effects					
				Weight gain	EPS	↑Lipids, risk of diabetes	↑ Pro- lactin	↑QTc ^a	Sedation
Second-generation ^b (continued)									
OLA	2.5–5	10–20	Bipolar I disorder , manic or mixed, ages 13–17 years (acute) Schizophrenia , ages 13–17 years (acute)	+++	0/+	+++	+	0/+	+ / ++
PAL	6	3–12	Schizophrenia , ages 12–17 years (acute)	++	++	+	+++	+	0/+
QUE	25–50	SCZ: 400–800 BD: 400–600	Bipolar I disorder , manic, ages 10–17 years (acute monotherapy and adjunctive with lithium or valproate) Schizophrenia , ages 13–17 years (acute)	++	0	++	0	+	++ ^d

TABLE 17–5. Antipsychotic medications (*continued*)

Drug (generic name)	Typical pediatric starting dose, mg	Typical pediatric dose range, mg	Pediatric indications in the United States	Side effects					
				Weight gain	EPS	↑Lipids, risk of diabetes	↑ Pro- lactin	↑QTc ^a	Sedation
Second-generation ^b (<i>continued</i>)									
RIS	0.25–0.5	1–6	Irritability associated with ASD , ages 5–17 years Bipolar I disorder , manic or mixed, ages 10–17 years (acute) Schizophrenia , ages 13–17 years (acute)	++	++	+	+++	+	+
ZIP	20	SCZ: 60–160 BD: 40–160	—	0/+	+	0/+	+	++	+

TABLE 17-5. Antipsychotic medications (*continued*)

Drug (generic name)	Typical pediatric starting dose, mg	Typical pediatric dose range, mg	Pediatric indications in the United States	Side effects					
				Weight gain	EPS	↑Lipids, risk of diabetes	↑ Pro- lactin	↑QTc ^a	Sedation
First-generation									
CHLOR	25–100	50–300	Severe behavioral problems and short-term treatment of hyperactive children with accompanying conduct disorders, ages 1–12 years ^e	+++	+	+++	+	0/+	++
HAL	0.5–2	2–10	Psychotic disorders , ages 3–17 years ^e Nonpsychotic behavior disorders and Tourette’s disorder , ages 3–17 years ^e	+	+++	0/+	++/ +++	0/+	+
PER	2–4	8–32	Schizophrenia , ages 12–17 years ^e	++	+	+	++	+	+

TABLE 17-5. **Antipsychotic medications (continued)**

Drug (generic name)	Typical pediatric starting dose, mg	Typical pediatric dose range, mg	Pediatric indications in the United States	Side effects				
				Weight gain	EPS	↑Lipids, risk of diabetes	↑ Pro- lactin	↑QTc ^a Sedation

Note. Much of these data are extrapolated from adult populations, and the information may change as more pediatric data become available.

0=none; 0/+ =minimal; +=mild; ++=moderate; +++=severe.

ARI=aripiprazole; ASD=autism spectrum disorder; ASE=asenapine; BD=bipolar disorder; CHLOR=chlorpromazine; CLO=clozapine; EPS=extrapyramidal symptoms; HAL=haloperidol; ILO=iloperidone; LUR=lurasidone; OLA=olanzapine; PAL=paliperidone; PER=perphenazine; QUE=quetiapine; RIS=risperidone; SCZ=schizophrenia; ZIP=ziprasidone.

^aRelevance for development of torsades de pointes not established.

^bSecond-generation antipsychotics have low risk of causing tardive dyskinesia, except for CLO, which has none. The first-generation antipsychotics in this table all have moderate risk of causing tardive dyskinesia.

^cAripiprazole may reduce prolactin levels.

^dLess at higher doses than at lower doses (potential threshold ≥ 300 mg/day).

^eExtrapolated from adults.

Source. Adapted from Correll 2016.

disorder) follows, and, very slowly, negative symptoms (apathy, anergy, withdrawal) may improve. To monitor outcome, parent and teacher reports are essential, in addition to self-reports from adolescents. Standardized clinician rating scales, such as the Positive and Negative Syndrome Scale for Schizophrenia, derived from the Children's Psychiatric Rating Scale, are sensitive to improvement in children (Spencer et al. 1994).

Current practice for adults with schizophrenia is to continue antipsychotic treatment indefinitely; however, firm recommendations regarding children are lacking because of the difficulty in making a definitive diagnosis and the possibility of developmental toxicity. Antipsychotics should be discontinued by gradual tapering to prevent rebound symptoms or relapse.

Bipolar Disorder

RCTs have demonstrated efficacy of SGAs (aripiprazole, olanzapine, quetiapine, risperidone, asenapine, and ziprasidone) as monotherapy for mania or mixed episodes in pediatric patients with bipolar disorder. For bipolar depression, combined olanzapine-fluoxetine was superior to placebo, but two studies of quetiapine yielded negative findings (Correll 2016). Monotherapy is infrequently sufficient for full remission, however, and atypical antipsychotics are often used adjunctively with lithium or valproate. The effect size in youth with bipolar mania, compared with that in adults, is larger for the atypicals and smaller for lithium and anticonvulsant mood stabilizers.

Developmental Disorders

Risperidone, olanzapine, and aripiprazole have been shown in RCTs to reduce severe tantrums, irritability, aggression, stereotypies, and self-injurious behavior (but not the core symptoms of autism) in children with ASD. The majority of these studies have assessed risperidone and aripiprazole in trials of 6 weeks to 6 months (Correll 2016). A single RCT of lurasidone for irritability in children ages 6–17 years with ASD did not show superiority over pla-

cebo (Loebel et al. 2016). Studies of longer-term maintenance treatment with antipsychotics in ASD suggest that risperidone is superior to placebo for relapse prevention (McCracken et al. 2002; Troost et al. 2005). Aripiprazole use has support for longer-term maintenance in ASD (Findling et al. 2014).

By 2011, as many as 1 in 6 children and adolescents with ASD and/or intellectual disability had been prescribed an SGA (Park et al. 2016). The young age at initiation of treatment in these children, the chronicity of their conditions, and the lack of long-term studies of efficacy and neurodevelopmental and metabolic effects of antipsychotics require that their use be continually reevaluated in each child, with assurance that nonpharmacological interventions to address problematic behaviors are implemented. Antipsychotic medications often increase appetite. If food-seeking behaviors become disruptive or aggressive, especially in nonverbal children, it is critical to evaluate the extent to which the medication may be actually increasing symptoms that it is meant to target.

Tourette's Disorder

Efficacy is often difficult to evaluate in Tourette's disorder because of the natural waxing and waning of symptoms. If tics cause impairment, habit reversal training is effective and is a more benign alternative to medication. Alpha-adrenergic receptor agonists (guanfacine, clonidine) have a more favorable benefit–risk ratio than other classes of medications used for tics. If disabling tics do not respond to these interventions, antipsychotics may be considered. Aripiprazole, haloperidol, pimozide, risperidone, and ziprasidone have been shown to be superior to placebo in RCTs for pediatric Tourette's disorder (Whittington et al. 2016), although all have significant side effects.

Aggressive Behavior Unresponsive to Other Interventions

Studies of hospitalized severely aggressive children ages 6–12 years have found that several typical neuroleptics are effective compared with placebo in reducing aggression, hostility, negativism, and explo-

siveness (Correll 2016). The risks of cognitive dulling and tardive dyskinesia place these drugs low on the list of medication options for aggression, however. Chlorpromazine leads to unacceptable sedation at relatively low doses.

Off-label use of atypical antipsychotics in youth with disruptive behavior disorders is increasingly common. Best practice dictates that antipsychotics be used for aggression or impulsivity only when nonpharmacological treatments have been employed, other appropriate medications with more benign profiles (e.g., stimulants, α agonists) have been optimally tried, and any comorbid or underlying disorders are adequately treated. Most RCTs of atypical antipsychotics for disruptive behavior disorders in children and adolescents have evaluated risperidone and found it to be superior to placebo in reducing aggression.

Initiation and Ongoing Treatment

Before an antipsychotic medication is started, a medical history (with special focus on personal and family history of obesity, hypertension, hyperlipidemia, diabetes mellitus, and cardiovascular symptoms or disease) and a physical examination (including blood pressure, pulse, height, weight, and body mass index [BMI]) should be obtained. Initial laboratory studies include CBC with differential, liver functions, fasting glucose, and lipid profile. Counseling regarding nutrition and exercise should be started immediately and refreshed at every visit, if the patient's clinical condition permits. In addition to verbal discussion and opportunity for questions, published medication information sheets can be used (e.g., Dulcan and Ballard 2015).

Detailed informed consent is especially crucial for clozapine, and monitoring is more extensive (FDA Drug Safety Communication 2015). An ECG should be obtained before starting ziprasidone, clozapine, or pimozide. An EEG is indicated before clozapine treatment because of the potential for EEG abnormalities and seizures.

The clinician should carefully examine each patient for abnormal movements with a scale such as the Abnormal Involuntary

Movement Scale (AIMS) at baseline and every 3–6 months thereafter. Especially in children with ASD or Tourette's disorder, it may be difficult to distinguish medication-induced movements of tardive dyskinesia or withdrawal dyskinesias from those movements characteristic of the disorder itself. The clinician should explain the risk of movement disorders to parents and patients (as appropriate) before starting treatment and regularly as treatment continues.

Dose must be titrated with careful attention to reduction in target symptoms and to side effects. Age, weight, and severity of symptoms do not provide clear dose guidelines. Children generally require higher mg/kg doses than do adults, and divided doses may be preferable in younger children. The half-lives of antipsychotics vary, and the titration schedules differ between those that reach a steady state rapidly (quetiapine, ziprasidone) and those with longer half-lives. Antipsychotics should be maintained at the lowest effective dose. Regular specific queries regarding side effects are required.

As treatment continues, pulse, blood pressure, height, weight, and BMI are followed at 3-month intervals. Blood triglycerides and high-density lipoprotein cholesterol (HDL) and fasting glucose are measured every 6 months. Prolactin is measured only if the patient is symptomatic (true gynecomastia, galactorrhea, breast tenderness, or, in females, amenorrhea). Patients who have significant weight gain in the first 3 months of treatment should have liver function enzymes measured, as fatty liver infiltration may develop.

Risperidone, aripiprazole, and olanzapine are available as a disk that dissolves rapidly in the mouth, a useful formulation when quick drug action is needed or when pill swallowing is difficult or resisted. Risperidone, aripiprazole, and several of the typical neuroleptics are available as a liquid. Risperidone, olanzapine, and paliperidone (and several typical neuroleptics) are available as a long-acting injection. Quetiapine is available in an extended-release form.

If a typical neuroleptic is to be used, one of the higher-potency drugs, such as perphenazine or thiorixene, may be best. The lower-potency compounds (e.g., chlorpromazine) are best avoided for chronic use because of sedation, cognitive dulling, and memory deficits that can interfere with learning.

Risks and Side Effects

Pediatric patients are more prone to side effects from antipsychotic drugs than are adults, and they are at risk for prolonged exposure, including during key developmental stages. Common side effects of antipsychotic medications are summarized in Table 17–5. The most common problematic side effect of the SGAs is immediate and chronic weight gain, often associated, after as short a treatment period as 12 weeks, with clinically significant hyperlipidemia and insulin resistance (Correll et al. 2009). *Metabolic syndrome*, a common chronic complication of antipsychotic medication, is characterized by abdominal obesity, elevated fasting triglycerides, reduced HDL, elevated blood pressure, and elevated fasting glucose due to insulin resistance leading to type 2 diabetes. Weight gain and associated morbidity are highest with olanzapine, quetiapine, and clozapine. Of the SGAs, lurasidone has the least effect on weight.

Sedation and cognitive dulling may exacerbate the effect of negative symptoms and interfere with school functioning. Priapism has been reported with use of SGAs. Clozapine is associated with blood dyscrasias, seizures, and hypersalivation, as well as very rare myocarditis.

Acute EPS, including dystonic reactions, parkinsonian tremor and rigidity, drooling, and akathisia, are commonly seen with typical neuroleptic treatment and are more prevalent than initially hoped with atypical antipsychotic use. Laryngeal dystonia is potentially fatal. Acute dystonia may be treated with oral or intramuscular diphenhydramine (25 or 50 mg) or benztropine (0.5–2.0 mg). When medication is started in an outpatient, the clinician should instruct a responsible adult to watch for a dystonic reaction and may prescribe diphenhydramine or benztropine to use acutely if needed (or recommend an immediate visit to the local emergency room). Adolescent boys seem to be especially vulnerable to acute dystonic reactions, so prophylactic antiparkinsonian medication may be indicated (benztropine 1–2 mg/day in divided doses). Clinical experience suggests that prepubertal children do not respond well to anticholinergics; therefore, reduction of antipsychotic dose is preferable to adding

another drug if EPS appear. Chronic parkinsonian symptoms are often underrecognized by clinicians and may impair performance of age-appropriate activities and lead to resistance to taking medication.

Akathisia may be especially difficult to identify in very young patients or those with limited verbal ability. It may be misinterpreted as anxiety or agitation and mistakenly exacerbated with an increase in antipsychotic dose. Clonazepam, beta-blockers, or mirtazapine may reduce neuroleptic-induced akathisia in adolescents.

Tardive dyskinesia or withdrawal dyskinesias are frequent in children treated with typical neuroleptics. Most withdrawal dyskinesias are transient. Very rarely, potentially irreversible tardive dyskinesia has been documented in children and adolescents after treatment as brief as 5 months. Other withdrawal-emergent symptoms include nausea, vomiting, loss of appetite, diaphoresis, and hyperactivity. Various behavioral withdrawal symptoms may appear up to several weeks after antipsychotic discontinuation and persist for as long as 8 weeks. These must be distinguished from a return of symptoms of the original disorder. Withdrawal dyskinesia has also been reported after risperidone discontinuation.

Neuroleptic malignant syndrome (NMS), a potentially fatal side effect, is manifested by hyperthermia, muscle rigidity, autonomic hyperactivity, and changes in consciousness. It has been reported to be associated with the atypicals, as well. Adolescents may present with serious medical complications or may have NMS without fever. NMS is treated by discontinuation of the antipsychotic and use of aggressive supportive measures. The use of specific medications to treat NMS has not been studied in adolescents, although case reports suggest the use of bromocriptine or dantrolene.

Anticholinergic side effects, such as hypotension, dry mouth, constipation, nasal congestion, blurred vision, and urinary retention, are uncommon but may occur. Children may develop enuresis on antipsychotics. Chlorpromazine increases the risk of sunburn.

■ ANTICONVULSANTS

Indications and Efficacy

Anticonvulsants are medications that were initially developed for the treatment of epilepsy. Although some anticonvulsants have FDA approval for psychiatric use in adults, none of them has an FDA indication for treatment of psychiatric conditions in children. Despite this, they are often used in clinical practice for treatment of bipolar disorder and severe aggression associated with emotional lability and irritability, with or without evidence of primary neurological findings. The pediatric research literature supporting the safety and efficacy of anticonvulsants is limited, and clinical use of these medications is largely based on extrapolation from adult studies, which may not apply in younger patients. When used for these conditions, the drugs are often called *mood stabilizers*, a grouping that also includes lithium. Among the anticonvulsants, divalproex sodium has the most robust data supporting its use in children. There are some limited data regarding carbamazepine and lamotrigine, but virtually no empirical data on the pediatric psychiatric use of other anticonvulsants.

Divalproex Sodium

Sodium valproate (valproic acid [Depakene]; divalproex sodium [Depakote]) has an FDA indication in adults for the treatment of mania in bipolar disorder. The data supporting the use of divalproex (DVP) for treatment of pediatric bipolar disorder are mixed, with both positive and negative study findings. Efficacy was demonstrated in an NIMH-sponsored RCT that found DVP to be superior to both lithium and placebo in the treatment of youth with bipolar I disorder (Kowatch et al. 2007). However, a subsequent industry-sponsored RCT failed to find divalproex extended-release to be superior to placebo in the treatment of manic or mixed episodes in youth ages 10–17 years (Wagner et al. 2009). Findling and colleagues (2005) found that after stabilization with combination lithium and divalproex treatment, neither monotherapy with divalproex nor lithium monotherapy was sufficiently effective for maintenance therapy.

Generally, studies comparing divalproex with atypical antipsychotics have demonstrated equal or greater efficacy for atypical antipsychotics. A head-to-head randomized study of quetiapine versus divalproex for adolescent mania found quetiapine to be as effective as divalproex with a more rapid onset of action (DelBello et al. 2006), and another RCT found that combination divalproex and quetiapine was more effective than divalproex alone (DelBello et al. 2002). The TEAM multisite RCT found that subjects treated with risperidone had a higher response rate (68.5%) than those treated with lithium (35.6%) or divalproex (24%), but the magnitude of this effect was influenced by site-related characteristics and the presence of ADHD (Geller et al. 2012; Vitiello et al. 2012). These findings are supported by a randomized trial comparing treatment of pediatric mania with risperidone versus divalproex that found risperidone was associated with more rapid improvement, greater efficacy, and better tolerability (Pavuluri et al. 2010). Another RCT of youth with pediatric bipolar disorder demonstrated greater improvement in manic symptoms in response to risperidone than to divalproex in the youth who had comorbid disruptive behavior disorder (DBD), whereas those without comorbid DBD responded similarly to both medications (West et al. 2011).

Carbamazepine

Carbamazepine (Carbatrol, Tegretol) is an older anticonvulsant that has demonstrated efficacy in adult mania, but lack of controlled trials and problematic side effects and drug interactions limit its use in pediatric bipolar disorder. Although some evidence supports use of this drug for aggression in conduct disorder, a controlled study did not find carbamazepine to be more effective than placebo in reducing aggression (Cueva et al. 1996). Carbamazepine has largely been replaced by the atypical antipsychotics for this use.

Lamotrigine

Lamotrigine (Lamictal) has an FDA indication for use in maintenance treatment in adults with bipolar disorder. Some open-label tri-

als support its use in pediatric bipolar depression, both as monotherapy and as adjunctive therapy (Chang et al. 2006); in acute mania (Biederman et al. 2010); and as maintenance treatment after acute stabilization of a manic or hypomanic episode (Pavuluri et al. 2009). Other anticonvulsants have insufficient pediatric data to recommend their use for psychiatric indications.

Initiation and Ongoing Treatment

Baseline assessments prior to using anticonvulsants include a recent medical history and physical examination, menstrual and sexual history, CBC with differential, liver function tests, and a pregnancy test for postpubertal females, because both valproate and carbamazepine carry risks of teratogenicity. Prior to carbamazepine being used in patients of Asian ancestry, genetic testing is recommended for HLA-B*1502, an allele that increases risk of dangerous skin reactions. CBC with differential, serum levels of the drug, and liver function tests should be checked every 6 months.

Valproate

For children and adolescents, valproate is typically started at 10–15 mg/kg/day in two or three divided doses and then titrated up gradually according to tolerance and response. Serum valproate levels are optimally measured 12 hours after the last dose. Target serum levels for treating mania are thought to be 50–125 micrograms/mL. Depakote is often the preferred formulation (over Depakene) because it is enteric-coated to reduce gastrointestinal side effects. An extended-release formulation (Depakote ER) permits fewer daily doses. A soft-gel capsule of valproic acid delayed release (Stavzor) is easier to swallow.

Coadministration of guanfacine has been shown to increase plasma levels of valproate (Ambrosini and Sheikh 1998).

Carbamazepine

Typical starting dose of carbamazepine is 15 mg/kg/day, or for children 100 mg twice a day and for adolescents 100 mg three times a

day. Dose may be increased by 100–200 mg/day at weekly intervals. The maximum total daily dose of carbamazepine is 1,000 mg in children and 1,200 mg in adolescents. Usual maintenance total doses are 10–20 mg/kg/day divided into two or three doses per day. There is an extended-release form (Equetro) that permits once-a-day dosing. There are no systematic data on optimal therapeutic levels for treatment of pediatric aggression or bipolar disorder. The commonly used maintenance level is 7–10 micrograms/mL.

Lamotrigine

The risk of potentially fatal Stevens-Johnson syndrome or toxic epidermal necrolysis is increased when lamotrigine is started at a high dose or titrated too rapidly, especially in children. Educating the patient and family to closely monitor for and to report any rash is essential. If a rash occurs, stopping the medication is advised. Because coadministration with divalproex doubles the concentration of lamotrigine, lamotrigine should be started at an even lower dose and titrated more slowly when the two drugs are used together. Lamotrigine is typically started at 25 mg/day and titrated by 25 mg every 2 weeks to minimize side effects. There are starter packs available for patients who are taking divalproex and lamotrigine concurrently. Carbamazepine and some other anticonvulsants can induce the metabolism of lamotrigine so higher end-doses may be required.

Risks and Side Effects

Data on pediatric safety and tolerability of the anticonvulsants are mostly derived from experience with the use of these drugs in the treatment of epilepsy. Behavioral toxicity is possible with any of these drugs (more likely with carbamazepine and gabapentin). Topiramate may cause cognitive dulling and memory problems. Drug interactions between anticonvulsants and other medications are often problematic.

The FDA has placed a warning on all anticonvulsants of increased risk of suicidal thoughts and behavior.

Valproate

Common side effects include gastrointestinal symptoms, weight gain, sedation, transient hair loss, and tremor. Rare and serious adverse effects include liver toxicity (primarily in children younger than 3 years who are taking multiple anticonvulsants and usually reversible when the drug is stopped), hyperammonemia, blood dyscrasias, thrombocytopenia, and, very rarely, pancreatitis. Patients should be monitored for nausea, vomiting, easy bruising, lethargy, disorientation, malaise, or persistent abdominal pain (possible pancreatitis). Significant concern has been raised by the connection between valproate use and polycystic ovary syndrome, manifested by obesity, insulin resistance, acne, hirsutism, and irregular or absent menstrual periods (associated with decreased fertility). Female patients should be monitored closely for these symptoms.

Valproate is contraindicated in pregnancy because of increased rates of adverse neurodevelopmental outcomes and neural tube defects in exposed babies. The drug should not be used in females of childbearing potential in the absence of highly reliable use of birth control. Some authorities have suggested that valproate is contraindicated in all females of childbearing potential.

Carbamazepine

Common side effects include nausea, vomiting, vertigo, decreased coordination, drowsiness, blurred vision, and nystagmus (reversible by lowering dose). Rare adverse events include tics, ataxia, blood dyscrasias such as aplastic anemia and agranulocytosis, decreased thyroid function, hyponatremia, hepatitis, exacerbation of seizures, and life-threatening skin reaction. Adverse behavioral reactions may occur, with mania, extreme irritability, agitation, insomnia, obsessive thinking, hallucinations, delirium, psychosis, paranoia, hyperactivity, and aggression, especially in the first month of treatment. Carbamazepine is contraindicated in females of childbearing potential because of teratogenicity. Because of its metabolism by cytochrome P450 enzyme 3A4, carbamazepine has the potential to interact with many other medications, thereby increasing or decreasing the levels of these medications.

Lamotrigine

Lamotrigine is associated with Stevens-Johnson syndrome (see “Initiation and Ongoing Treatment” above). Youth under age 16 are at a threefold greater risk of developing Stevens-Johnson syndrome compared with adults. In 2010, the FDA mandated a warning regarding association of lamotrigine with aseptic meningitis.

■ MELATONIN

Melatonin is a naturally occurring neurotransmitter related to serotonin and tryptophan. It influences circadian rhythms in the anterior hypothalamus and is capable of shifting sleep phase. Melatonin is frequently recommended by clinicians or used independently by parents for initial insomnia (delayed sleep onset) in typically developing children or in youth with ADHD, ASD, and neurodevelopmental disabilities, as well as in adolescents with delayed sleep phase syndrome. In a study of currently stimulant-treated children (6–14 years old, 91% boys) with ADHD and initial insomnia (Weiss et al. 2006), the first intervention was sleep hygiene modifications for all. Those with insufficient response (the majority of subjects) entered a double-blind crossover trial of placebo versus 5 mg melatonin given 20 minutes before bedtime. Relative to placebo, melatonin decreased initial insomnia by 16 minutes. Combined sleep hygiene and melatonin resulted in a mean decrease in initial insomnia of 60 minutes. Total sleep time did not change, and improved sleep was not associated with improvement in symptoms of ADHD. Adverse events did not differ from those seen with placebo.

In the treatment of initial insomnia, melatonin is given approximately 30 minutes before desired sleep-onset time. One may start with a dose of 1.5 mg and increase in 1.5-mg increments every 4–5 days as needed, to a dose of 10–15 mg. Lower doses are used to normalize the circadian sleep-wake cycle in delayed sleep phase syndrome. Approximately 0.3 mg is given about 4–5 hours before the current habitual time of falling asleep. The same dose is gradually moved earlier as the falling asleep time is moved earlier (advanced).

Melatonin is not regulated as a drug and is sold as a food or nutritional supplement over the counter, raising concerns about variable potency of preparations and possible impurities. Side effects of melatonin per se are minimal, and melatonin may be used long-term with few, if any, adverse effects (Hoebert et al. 2009).

■ OMEGA-3 FATTY ACIDS

Omega-3 fatty acids are a subgroup of the long-chain polyunsaturated fatty acids (LC-PUFAs), which reduce inflammation and are implicated in cardiac health, cancer prevention, and neurodevelopment as well as regulation of mood and behavior. Omega-3 fatty acids are essential, meaning that humans cannot synthesize them but can metabolize naturally occurring plant-based α -linolenic acid into eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), the two major omega-3 fatty acids. Other sources are seafood, vegetable oils, and nuts.

Omega-3 supplementation has been shown to have a moderate effect size on symptoms in pediatric bipolar depression and ADHD (see Barragán et al. 2017; Bloch and Qawasmi 2011; Fristad et al. 2015). Children with ADHD may have low baseline levels of omega-3 fatty acids and may benefit from supplementation either as monotherapy or adjunctive therapy to stimulants. Studies of omega-3 fatty acids in autism spectrum disorder have not shown benefit.

Current recommended dose is an EPA/DHA combination at about 1,000 mg/day, generally in the form of fish oil. Side effects may include stomach upset, diarrhea, and “fish burps.” Relative contraindications include hepatic impairment and bleeding disorders. As with all supplements, it is best to purchase a product with the U.S. Pharmacopeial Convention (USP)–verified mark that indicates that the product has been voluntarily submitted for verification of federal standards of quality, purity, and potency. This includes exclusion of heavy metals such as mercury, which may accumulate in fish oil.

■ REFERENCES

- Alessi N, Naylor MW, Ghaziuddin M, Zubieta JK: Update on lithium carbonate therapy in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 33(3):291–304, 1994 8169173
- Allen AJ, Kurlan RM, Gilbert DL, et al: Atomoxetine treatment in children and adolescents with ADHD and comorbid tic disorders. *Neurology* 65(12):1941–1949, 2005 16380617
- Ambrosini PJ, Sheikh RM: Increased plasma valproate concentrations when coadministered with guanfacine. *J Child Adolesc Psychopharmacol* 8(2):143–147, 1998 9730080
- Barragán E, Breuer D, Döpfner M: Efficacy and safety of omega-3/6 fatty acids, methylphenidate, and a combined treatment in children with ADHD. *J Atten Disord* 21(5):433–441, 2017 24464327
- Barrickman LL, Perry PJ, Allen AJ, et al: Bupropion versus methylphenidate in the treatment of attention-deficit hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 34(5):649–657, 1995 7775360
- Biederman J, Melmed RD, Patel A, et al; SPD503 Study Group: A randomized, double-blind, placebo-controlled study of guanfacine extended release in children and adolescents with attention-deficit/hyperactivity disorder. *Pediatrics* 121(1):e73–e84, 2008 18166547
- Biederman J, Joshi G, Mick E, et al: A prospective open-label trial of lamotrigine monotherapy in children and adolescents with bipolar disorder. *CNS Neurosci Ther* 16(2):91–102, 2010 20415838
- Birmaher B, Axelson DA, Monk K, et al: Fluoxetine for the treatment of childhood anxiety disorders. *J Am Acad Child Adolesc Psychiatry* 42(4):415–423, 2003 12649628
- Black B, Uhde TW: Treatment of elective mutism with fluoxetine: a double-blind, placebo-controlled study. *J Am Acad Child Adolesc Psychiatry* 33(7):1000–1006, 1994 7961338
- Bloch MH, Qawasmi A: Omega-3 fatty acid supplementation for the treatment of children with attention-deficit/hyperactivity disorder symptomatology: systematic review and meta-analysis. *J Am Acad Child Adolesc Psychiatry* 50(10):991–1000, 2011 21961774
- Brent D, Emslie G, Clarke G, et al: Switching to another SSRI or to venlafaxine with or without cognitive behavioral therapy for adolescents with SSRI-resistant depression: the TORDIA randomized controlled trial. *JAMA* 299(8):901–913, 2008 18314433

- Chang K, Saxena K, Howe M: An open-label study of lamotrigine adjunct or monotherapy for the treatment of adolescents with bipolar depression. *J Am Acad Child Adolesc Psychiatry* 45(3):298–304, 2006 16540814
- Cohen SC, Mulqueen JM, Ferracioli-Oda E, et al: Meta-analysis: risk of tics associated with psychostimulant use in randomized, placebo-controlled trials. *J Am Acad Child Adolesc Psychiatry* 54(9):728–736, 2015 26299294
- Connor DF, Barkley RA, Davis HT: A pilot study of methylphenidate, clonidine, or the combination in ADHD comorbid with aggressive oppositional defiant or conduct disorder. *Clin Pediatr (Phila)* 39(1):15–25, 2000 10660814
- Correll CU: Antipsychotic medications, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 795–846
- Correll CU, Manu P, Olshansky V, et al: Cardiometabolic risk of second-generation antipsychotic medications during first-time use in children and adolescents. *JAMA* 302(16):1765–1773, 2009 19861668
- Coskun M, Alyanak B: Psychiatric co-morbidity and efficacy of mirtazapine treatment in young subjects with chronic or cyclic vomiting syndromes: a case series. *J Neurogastroenterol Motil* 17(3):305–311, 2011 21860824
- Cueva JE, Overall JE, Small AM, et al: Carbamazepine in aggressive children with conduct disorder: a double-blind and placebo-controlled study. *J Am Acad Child Adolesc Psychiatry* 35(4):480–490, 1996 8919710
- DelBello MP, Schwiers ML, Rosenberg HL, Strakowski SM: A double-blind, randomized, placebo-controlled study of quetiapine as adjunctive treatment for adolescent mania. *J Am Acad Child Adolesc Psychiatry* 41(10):1216–1223, 2002 12364843
- DelBello MP, Kowatch RA, Adler CM, et al: A double-blind randomized pilot study comparing quetiapine and divalproex for adolescent mania. *J Am Acad Child Adolesc Psychiatry* 45(3):305–313, 2006 16540815
- Donnelly M, Rapoport JL: Attention deficit disorders, in *Diagnosis and Psychopharmacology of Childhood and Adolescent Disorders*. Edited by Weiner JM. New York, Wiley, 1985, pp 178–197
- Dulcan MK, Ballard R (eds): *Helping Parents and Teachers Understand Medications for Behavioral and Emotional Problems: A Resource Book of Medication Information Handouts*, 4th Edition. Arlington, VA, American Psychiatric Publishing, 2015

- Emslie GJ, Ventura D, Korotzer A, et al: Escitalopram in the treatment of adolescent depression: a randomized placebo-controlled multisite trial. *J Am Acad Child Adolesc Psychiatry* 48(7):721–729, 2009 19465881
- Faraone SV, Biederman J, Morley CP, Spencer TJ: Effect of stimulants on height and weight: a review of the literature. *J Am Acad Child Adolesc Psychiatry* 47(9):994–1009, 2008 18580502
- FDA Drug Safety Communication: FDA modifies monitoring for neutropenia associated with schizophrenia medicine clozapine; approves new shared REMS program for all clozapine medicines. <http://www.fda.gov/downloads/Drugs/DrugSafety/UCM462193.pdf>, 2015, Accessed July 5, 2016.
- Findling RL, McNamara NK, Youngstrom EA, et al: Double-blind 18-month trial of lithium versus divalproex maintenance treatment in pediatric bipolar disorder. *J Am Acad Child Adolesc Psychiatry* 44(5):409–417, 2005 15843762
- Findling RL, Kafantaris V, Pavuluri M, et al: Dosing strategies for lithium monotherapy in children and adolescents with bipolar I disorder. *J Child Adolesc Psychopharmacol* 21(3):195–205, 2011 21663422
- Findling RL, Robb A, Bose A: Escitalopram in the treatment of adolescent depression: a randomized, double-blind, placebo-controlled extension trial. *J Child Adolesc Psychopharmacol* 23(7):468–480, 2013 24041408
- Findling RL, Mankoski R, Timko K, et al: A randomized controlled trial investigating the safety and efficacy of aripiprazole in the long-term maintenance treatment of pediatric patients with irritability associated with autistic disorder. *J Clin Psychiatry* 75(1):22–30, 2014 24502859
- Findling RL, Robb A, McNamara NK, et al: Lithium in the acute treatment of bipolar I disorder: a double-blind, placebo-controlled study. *Pediatrics* 136(5):885–894, 2015 26459650
- Friedland S, Walkup JT: Meta-assurance: no tic exacerbation caused by stimulants. *J Am Acad Child Adolesc Psychiatry* 54(9):706–708, 2015 26299291
- Fristad MA, Young AS, Vesco AT, et al: A randomized controlled trial of individual family psychoeducational psychotherapy and omega-3 fatty acids in youth with subsyndromal bipolar disorder. *J Child Adolesc Psychopharmacol* 25(10):764–774, 2015 26682997
- Geller B, Fetner HH: Children's 24-hour serum lithium level after a single dose predicts initial dose and steady-state plasma level. *J Clin Psychopharmacol* 9(2):155, 1989 2723138
- Geller B, Luby JL, Joshi P, et al: A randomized controlled trial of risperidone, lithium, or divalproex sodium for initial treatment of bipolar I disorder, manic or mixed phase, in children and adolescents. *Arch Gen Psychiatry* 69(5):515–528, 2012 22213771

- Greenhill L, Kollins S, Abikoff H, et al: Efficacy and safety of immediate-release methylphenidate treatment for preschoolers with ADHD. *J Am Acad Child Adolesc Psychiatry* 45(11):1284–1293, 2006 17023867
- Hoebert M, van der Heijden KB, van Geijlswijk IM, Smits MG: Long-term follow-up of melatonin treatment in children with ADHD and chronic sleep onset insomnia. *J Pineal Res* 47(1):1–7, 2009 19486273
- Hollander E, Phillips A, Chaplin W, et al: A placebo controlled crossover trial of liquid fluoxetine on repetitive behaviors in childhood and adolescent autism. *Neuropsychopharmacology* 30(3):582–589, 2005 15602505
- Hunt RD: Treatment effects of oral and transdermal clonidine in relation to methylphenidate: an open pilot study in ADD-H. *Psychopharmacol Bull* 23(1):111–114, 1987 3602304
- Jain R, Segal S, Kollins SH, Khayrallah M: Clonidine extended-release tablets for pediatric patients with attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 50(2):171–179, 2011 21241954
- Kelsey DK, Sumner CR, Casat CD, et al: Once-daily atomoxetine treatment for children with attention-deficit/hyperactivity disorder, including an assessment of evening and morning behavior: a double-blind, placebo-controlled trial. *Pediatrics* 114(1):e1–e8, 2004 15231966
- Kent JD, Blader JC, Koplewicz HS, et al: Effects of late-afternoon methylphenidate administration on behavior and sleep in attention-deficit hyperactivity disorder. *Pediatrics* 96(2 Pt 1):320–325, 1995 7630692
- King BH, Hollander E, Sikich L, et al; STAART Psychopharmacology Network: Lack of efficacy of citalopram in children with autism spectrum disorders and high levels of repetitive behavior: citalopram ineffective in children with autism. *Arch Gen Psychiatry* 66(6):583–590, 2009 19487623
- Kowatch R, Findling R, Scheffer R, et al: Placebo controlled trial of divalproex versus lithium for bipolar disorder. Presented at the annual meeting of the American Academy of Child and Adolescent Psychiatry, Boston, MA, October 2007
- Kurlan R: Methylphenidate to treat ADHD is not contraindicated in children with tics. *Mov Disord* 17(1):5–6, 2002 11835432
- Lake MB, Birmaher B, Wassick S, et al: Bleeding and selective serotonin reuptake inhibitors in childhood and adolescence. *J Child Adolesc Psychopharmacol* 10(1):35–38, 2000 10755580
- Loebel A, Brams M, Goldman RS, et al: Lurasidone for the treatment of irritability associated with autistic disorder. *J Autism Dev Disord* 46(4):1153–1163, 2016 26659550

- March JS, Entusah AR, Rynn M, et al: A randomized controlled trial of venlafaxine ER versus placebo in pediatric social anxiety disorder. *Biol Psychiatry* 62(10):1149–1154, 2007 17553467
- McCracken JT, McGough J, Shah B, et al; Research Units on Pediatric Psychopharmacology Autism Network: Risperidone in children with autism and serious behavioral problems. *N Engl J Med* 347(5):314–321, 2002 12151468
- McCracken JT, McGough JJ, Loo SK, et al: Combined stimulant and guanfacine administration in attention-deficit/hyperactivity disorder: a controlled, comparative study. *J Am Acad Child Adolesc Psychiatry* 55(8):657–666.e1, 2016 27453079
- Meighen KG: Duloxetine treatment of pediatric chronic pain and co-morbid major depressive disorder. *J Child Adolesc Psychopharmacol* 17(1):121–127, 2007 17343560
- Michelson D, Faries D, Wernicke J, et al; Atomoxetine ADHD Study Group: Atomoxetine in the treatment of children and adolescents with attention-deficit/hyperactivity disorder: a randomized, placebo-controlled, dose-response study. *Pediatrics* 108(5):E83, 2001 11694667
- MTA Cooperative Group. Multimodal Treatment Study of Children with ADHD: A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Arch Gen Psychiatry* 56(12):1073–1086, 1999 10591283
- Newcorn JH, Spencer TJ, Biederman J, et al: Atomoxetine treatment in children and adolescents with attention-deficit/hyperactivity disorder and comorbid oppositional defiant disorder. *J Am Acad Child Adolesc Psychiatry* 44(3):240–248, 2005 15725968
- Newcorn JH, Sutton VK, Weiss MD, Sumner CR: Clinical responses to atomoxetine in attention-deficit/hyperactivity disorder: the Integrated Data Exploratory Analysis (IDEA) study. *J Am Acad Child Adolesc Psychiatry* 48(5):511–518, 2009 19318988
- Palumbo DR, Sallee FR, Pelham WE Jr, et al: Clonidine for attention-deficit/hyperactivity disorder: I. Efficacy and tolerability outcomes. *J Am Acad Child Adolesc Psychiatry* 47(2):180–188, 2008 18182963
- Park SY, Cervesi C, Galling B, et al: Antipsychotic use trends in youth with autism spectrum disorder and/or intellectual disability: a meta-analysis. *J Am Acad Child Adolesc Psychiatry* 55(6):456–468.e4, 2016 27238064
- Pavuluri MN, Henry DB, Moss M, et al: Effectiveness of lamotrigine in maintaining symptom control in pediatric bipolar disorder. *J Child Adolesc Psychopharmacol* 19(1):75–82, 2009 19232025

- Pavuluri MN, Henry DB, Findling RL, et al: Double-blind randomized trial of risperidone versus divalproex in pediatric bipolar disorder. *Bipolar Disord* 12(6):593–605, 2010 20868458
- Perrin JM, Friedman RA, Knilans TK; Black Box Working Group; Section on Cardiology and Cardiac Surgery: Cardiovascular monitoring and stimulant drugs for attention-deficit/hyperactivity disorder. *Pediatrics* 122(2):451–453, 2008 18676566
- Pliszka SR, Greenhill LL, Crismon ML, et al: The Texas Children's Medication Algorithm Project: Report of the Texas Consensus Conference Panel on Medication Treatment of Childhood Attention-Deficit/Hyperactivity Disorder. Part II: Tactics. *Attention-Deficit/Hyperactivity Disorder. J Am Acad Child Adolesc Psychiatry* 39(7):920–927, 2000 10892235
- Ramtevedt BE, Røinås E, Aabech HS, Sundet KS: Clinical gains from including both dextroamphetamine and methylphenidate in stimulant trials. *J Child Adolesc Psychopharmacol* 23(9):597–604, 2013 23659360
- Rynn MA, Siqueland L, Rickels K: Placebo-controlled trial of sertraline in the treatment of children with generalized anxiety disorder. *Am J Psychiatry* 158(12):2008–2014, 2001 11729017
- Rynn MA, Riddle MA, Yeung PP, Kunz NR: Efficacy and safety of extended-release venlafaxine in the treatment of generalized anxiety disorder in children and adolescents: two placebo-controlled trials. *Am J Psychiatry* 164(2):290–300, 2007 17267793
- Sallee FR, McGough J, Wigal T, et al; SPD503 Study Group: Guanfacine extended release in children and adolescents with attention-deficit/hyperactivity disorder: a placebo-controlled trial. *J Am Acad Child Adolesc Psychiatry* 48(2):155–165, 2009 19106767
- Scahill L, Chappell PB, Kim YS, et al: A placebo-controlled study of guanfacine in the treatment of children with tic disorders and attention deficit hyperactivity disorder. *Am J Psychiatry* 158(7):1067–1074, 2001 11431228
- Scahill L, McCracken JT, King BH, et al; Research Units on Pediatric Psychopharmacology Autism Network: Extended-release guanfacine for hyperactivity in children with autism spectrum disorder. *Am J Psychiatry* 172(12):1197–1206, 2015 26315981
- Spencer EK, Alpert M, Pouget ER: Scales for the assessment of neuroleptic response in schizophrenic children: specific measures derived from the CPRS. *Psychopharmacol Bull* 30(2):199–202, 1994 7831455

- Strawn JR, Prakash A, Zhang Q, et al: A randomized, placebo-controlled study of duloxetine for the treatment of children and adolescents with generalized anxiety disorder. *J Am Acad Child Adolesc Psychiatry* 54(4):283–293, 2015 25791145
- Strober M, Morrell W, Burroughs J, et al: A family study of bipolar I disorder in adolescence. Early onset of symptoms linked to increased familial loading and lithium resistance. *J Affect Disord* 15(3):255–268, 1988 2975298
- Strober M, Morrell W, Lampert C, Burroughs J: Relapse following discontinuation of lithium maintenance therapy in adolescents with bipolar I illness: a naturalistic study. *Am J Psychiatry* 147(4):457–461, 1990 2107763
- Tourette's Syndrome Study Group: Treatment of ADHD in children with tics: a randomized controlled trial. *Neurology* 58(4):527–536, 2002 11865128
- Troost PW, Lahuis BE, Steenhuis MP, et al: Long-term effects of risperidone in children with autism spectrum disorders: a placebo discontinuation study. *J Am Acad Child Adolesc Psychiatry* 44(11):1137–1144, 2005 16239862
- Vetter VL, Elia J, Erickson C, et al: Cardiovascular monitoring of children and adolescents with heart disease receiving medications for attention-deficit/hyperactivity disorder: a scientific statement from the American Heart Association Council on Cardiovascular Disease in the Young Congenital Cardiac Defects Committee and the Council on Cardiovascular Nursing. *Circulation* 117(18):2407–2423, 2008 18427125
- Vitiello B: Developmental aspects of pediatric psychopharmacology, in *Clinical Manual of Child and Adolescent Psychopharmacology*, 3rd Edition. Edited by McVoy M, Findling RL. Arlington, VA, American Psychiatric Association Publishing, 2017, pp 1–34
- Vitiello B, Riddle MA, Yenokyan G, et al: Treatment moderators and predictors of outcome in the Treatment of Early Age Mania (TEAM) study. *J Am Acad Child Adolesc Psychiatry* 51(9):867–878, 2012 22917200
- Wagner KD, Ambrosini P, Rynn M, et al: Efficacy of sertraline in the treatment of children and adolescents with major depressive disorder: two randomized controlled trials. *JAMA* 290(8):1033–1041, 2003 12941675
- Wagner KD, Berard R, Stein MB, et al: A multicenter, randomized, double-blind, placebo-controlled trial of paroxetine in children and adolescents with social anxiety disorder. *Arch Gen Psychiatry* 61(11):1153–1162, 2004 15520363
- Wagner KD, Jonas J, Findling RL, et al: A double-blind, randomized, placebo-controlled trial of escitalopram in the treatment of pediatric depression. *J Am Acad Child Adolesc Psychiatry* 45(3):280–288, 2006 16540812

- Wagner KD, Redden L, Kowatch RA, et al: A double-blind, randomized, placebo-controlled trial of divalproex extended-release in the treatment of bipolar disorder in children and adolescents. *J Am Acad Child Adolesc Psychiatry* 48(5):519–532, 2009 19325497
- Walkup JT, Labellarte MJ, Riddle MA, et al; The Research Unit on Pediatric Psychopharmacology Anxiety Study Group: Fluvoxamine for the treatment of anxiety disorders in children and adolescents. *N Engl J Med* 344(17):1279–1285, 2001 11323729
- Walkup JT, Albano AM, Piacentini J, et al: Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *N Engl J Med* 359(26):2753–2766, 2008 18974308
- Weiss MD, Wasdell MB, Bomben MM, et al: Sleep hygiene and melatonin treatment for children and adolescents with ADHD and initial insomnia. *J Am Acad Child Adolesc Psychiatry* 45(5):512–519, 2006 16670647
- Weller EB, Weller RA, Fristad MA: Lithium dosage guide for prepubertal children: a preliminary report. *J Am Acad Child Psychiatry* 25(1):92–95, 1986 3081617
- West AE, Weinstein SM, Celio CI, et al: Co-morbid disruptive behavior disorder and aggression predict functional outcomes and differential response to risperidone versus divalproex in pharmacotherapy for pediatric bipolar disorder. *J Child Adolesc Psychopharmacol* 21(6):545–553, 2011 22136096
- Whittington C, Pennant M, Kendall T, et al: Practitioner review: Treatments for Tourette syndrome in children and young people—a systematic review. *J Child Psychol Psychiatry* 57(9):988–1004; (Epub ahead of print), 2016 27132945
- Wilens TE, Spencer TJ, Swanson JM, et al: Combining methylphenidate and clonidine: a clinically sound medication option. *J Am Acad Child Adolesc Psychiatry* 38(5):614–619, discussion 619–622, 1999 10230195
- Wilens TE, Bukstein O, Brams M, et al: A controlled trial of extended-release guanfacine and psychostimulants for attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 51(1):74–85.e2, 2012 22176941
- Wilens TE, Robertson B, Sikirica V, et al: A randomized, placebo-controlled trial of guanfacine extended release in adolescents with attention-deficit/hyperactivity disorder. *J Am Acad Child Adolesc Psychiatry* 54(11):916–25.e2, 2015 26506582

■ ADDITIONAL READING

- Emslie GJ, Croarkin P, Chapman MR, Mayes TL: Antidepressants, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 737–768
- Gracious BL, Danielyan A, Kowatch RA: Mood stabilizers, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 769–794
- Lee ES, Findling RL: Principles of psychopharmacology, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 691–708
- McVoy M, Findling RL (eds): Clinical Manual of Child and Adolescent Psychopharmacology, 3rd Edition. Arlington, VA, American Psychiatric Association Publishing, 2017
- Spencer TJ, Biederman J, Wilens TE: Medications used for attention-deficit/hyperactivity disorder, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 709–736

PSYCHOSOCIAL TREATMENTS

The ideal mental health service delivery system provides a continuum of care (i.e., integrated programs at all levels of intensity) in which the child and family can move easily from one service to another as the clinical situation warrants.

Child and adolescent psychiatric disorders cannot be successfully treated unless the family dynamics and the school environment are considered. The parents are always involved, at a minimum to ensure coordinated treatment and to remove any secondary gain that inadvertently maintains the child's symptoms. For many disorders, therapeutic work with the parents or the family is just as important, or even more important, than direct therapy with the child. Often, the therapist coordinates with the school, the pediatrician, a social welfare agency, juvenile court personnel, and/or a community recreation leader. Whatever the modalities of therapy used, the therapist must be aware of the patient's level of physical, cognitive, and emotional development in order to understand the symptoms, set appropriate goals, and tailor effective interventions. A focus on the skills necessary for successful development and adaptation, with attention to improving those at which the child or parent is not sufficiently competent, may facilitate successful therapy.

In the outpatient setting, treatment by a single therapist is generally most efficient and effective. Indications for collaborative treatment (two or more therapists working as a team) include an adolescent who is unusually concerned about confidentiality, the need for different types of skills that one therapist does not have, or a clear need of the patient and parent(s) for different qualities in a therapist (e.g., the child would benefit from a male role model, but

the mother has great difficulty relating to men). In collaborative treatment, the therapists must maintain free and open communication, discuss and agree on treatment plans, and avoid aligning into competitive “teams.” Unresolved conflicts over relative power and authority of the therapists will lead to difficulties in treatment.

Therapists must maintain clear guidelines for confidentiality and for relaying information between the parent and child. Adolescents are usually more sensitive to this issue than children. In general, the therapist should tell each party when and what information from his or her session will be relayed to the other. Parents and children may participate in the decision or in the communication itself. When children or adolescents are engaging in potentially dangerous activities or have serious thoughts of harming themselves or others, parents must be informed. Carefully planned joint parent–child sessions, in which the therapist coaches and supports the parent or child in sharing information, may be more useful than reports from the therapist.

Recommended books and chapters in “Additional Reading” offer guidance in implementing psychosocial treatments.

■ COMMUNICATION WITH CHILDREN AND ADOLESCENTS

Children’s ability to use language is limited by their cognitive immaturity. Young children often use play to express feelings, to narrate past events, and to work through trauma. In play therapy, the therapist uses the metaphor of the child’s symbolic play and bases questions and comments on characters in the play rather than focusing directly on the child’s own feelings and experiences (even if the connection is clear to the therapist). The skilled therapist tailors communication to the child’s stage of language and cognitive development and must be aware that the vocabulary of some bright and precocious children exceeds their emotional understanding of events and concepts. Dramatic play with dolls or puppets; drawing and other art techniques; and questions about dreams, wishes, or favorite stories or television shows can provide access to children’s fantasies, emotions, and concerns.

■ THE RESISTANT CHILD OR ADOLESCENT

It is not surprising that many children and adolescents do not wish to engage in therapy. Treatment is typically sought for them by parents or teachers. Many children and adolescents do not perceive a reason for them to change; they view that as “giving in” to parents or teachers. A wide variety of motivations may lead a child or an adolescent to refuse to participate in or attempt to sabotage therapy. It is important to understand the patient’s viewpoint and agenda and tailor resistance-reducing strategies to the cause. A child who is anxious or having difficulty separating from a parent may be helped by having the parent initially present in therapy. The therapist may address, either directly or through play, the patient’s reluctance to participate and may suggest possible causes that the child is unwilling or unable to verbalize. Long silences in therapy are generally not helpful and tend to lead to increased anxiety or struggle for control. Attractive play materials help to make the therapy situation less threatening and encourage participation while the therapist builds an alliance. Even adolescents often appreciate the availability of paper and markers. Play can be combined with therapy in techniques such as storytelling, drama, and specially designed games. The therapist must guard, however, against the sessions becoming only play or recreation instead of therapy. Use of a token economy (i.e., rewards for desired behavior and loss of rewards for disruptive behavior; see below) in the therapy situation may improve motivation, especially for materially deprived or oppositional children.

■ TYPES OF PSYCHOTHERAPY

The common themes of individual therapies are listed in Table 18–1.

Supportive Therapy

Supportive therapy may be especially useful for children and adolescents who do not have satisfying relationships with adults because their symptoms make it very difficult to establish a positive

TABLE 18–1. Common themes of individual therapies

A relationship with a therapist who is identified as a helping person and who has some degree of control and influence over the patient
Instillation of hope, pride, and improved morale
Use of attention, encouragement, and suggestion
Goals of helping the patient to achieve greater control, competence, mastery, autonomy, and coping skills
Goals to abandon or modify unrealistic expectations of self, others, and the environment

Source. Adapted from Strupp 1973.

relationship or their parents are emotionally or physically unavailable, or even hostile. For the patient in crisis, the therapist provides support until a stressor resolves, a developmental crisis has passed, or the patient or environment changes sufficiently that other adults can take on the supportive role. The patient has a real relationship with the therapist, who facilitates catharsis and provides understanding and judicious advice. Psychoeducation is often included in supportive therapy.

Psychodynamically Oriented Therapy

Psychodynamically oriented therapy is grounded in psychoanalytic theory but is more flexible and emphasizes the real relationship with the therapist, the provision of a corrective emotional experience, and the experience of transference. Goals include resolution of symptoms, change in behavior, and resumption of the normal developmental process. Mechanisms of change include understanding and working with transference feelings, catharsis, development of insight, strengthening of ego skills and adaptive defenses, and improvement in reality testing. The therapist forms an alliance with the child or adolescent, identifies feelings, clarifies thoughts and events, makes interpretations, judiciously gives information and advice, and acts as an advocate for the patient. Sessions are held once or twice a week.

Possible candidates for psychodynamically oriented individual therapy include verbal youngsters (or those who can use symbolic play) who are in significant emotional distress or who are struggling to deal with a stressor or traumatic event (e.g., parental death, divorce, or abandonment; physical illness). Patients with attention-deficit/hyperactivity disorder (ADHD) or disruptive behavior disorders are unlikely to benefit. Youngsters with ADHD have little insight into their behavior and its effect on others and may be genuinely unable to report their problems or to reflect on them. Patients with oppositional defiant and conduct disorders refuse to acknowledge problem behavior and are better treated in family or group therapy or in a structured milieu.

Time-Limited Therapy

All of the models of time-limited therapy have in common a planned relatively brief duration (several sessions to 6 months), a predominant focus on the present, and a high degree of structure and attention to specific, limited goals. Theoretical foundations of various models include psychodynamic, crisis, family systems, cognitive, behavioral or social learning, and guidance or educational. Both the therapist and the patient must take active roles. The short duration is used to increase patient motivation and participation and limit nonadaptive dependency and regression. A great deal of attention is paid to the process of termination and to how the patient will continue to make progress after therapy stops. Psychodynamic models emphasize a firm termination of therapy, while cognitive, behavioral, and supportive models often include periodic “booster” sessions.

Time-limited treatment appears to be at least as effective as longer-term therapy for some patients. Time-limited methods have been recommended for multiproblem, crisis-oriented families who are unlikely to persist in longer-term treatment and for well-functioning children and families with circumscribed problems of recent onset. Brief treatment is relatively contraindicated for long-standing severe problems and for children and adolescents who have endured serious losses and/or deprivation.

Other Models of Therapy

Specific structured therapy programs and techniques have been developed and tested for a variety of pediatric psychiatric disorders and symptoms (see “Additional Reading”). These techniques may be used individually or in diagnosis-specific groups. Parents may be included in psychoeducation, support, implementation of strategies at home, and/or interventions for their own symptoms, to improve outcome for the youth.

Cognitive-Behavioral Therapy

Manualized cognitive-behavioral therapy (CBT) techniques have been developed and adapted for children and adolescents with many disorders, including depression, obsessive-compulsive disorder, anxiety disorders, and bulimia nervosa. These empirically supported techniques may be used individually, in diagnosis-specific group therapy settings, or in family based treatment (see Beidel and Reinecke 2016; Szegedy et al. 2012 in “Additional Reading”). There is also an evidence-based model of trauma-focused CBT for youth (see Cohen et al. 2017 in “Additional Reading” and www.musc.edu/tfcbt).

CBT encompasses a range of techniques used to target specific symptoms. The key components include exposure and response prevention (a hierarchy of feared situations is gradually and successfully approached with graduated exposures), cognitive restructuring (recognition of negative thought processes that are then challenged and replaced with more realistic and positive self-statements to improve emotional/behavioral responses to situations), relaxation training (reducing arousal by progressive use of relaxation exercises or cognitive meditation techniques), pleasant activity scheduling or behavioral activation (particularly for patients with anergy, anhedonia, lack of motivation, or social isolation), and problem-solving skills (exercises to sequentially examine problems, goals, and possible solutions, with selection of an agreed-upon strategy to employ and subsequent self-evaluation of the results). Parents and caregivers can play a critical “coaching” role for CBT assignments between sessions and their collaboration is essential in psychoeducation and treatment planning.

Interpersonal Psychotherapy

Interpersonal psychotherapy for depressed adolescents (IPT-A) (see Gunlicks-Stoessel and Mufson 2016; Mufson et al. 2004 in “Additional Reading”) was developed as a time-limited psychotherapy based on the work of interpersonal theorists and developmental adaptation from the interpersonal psychotherapy (IPT) as used with adults. The IPT model focuses on the premise that depression occurs within an interpersonal framework. IPT serves to relieve symptoms of depression by improving communication patterns, which then positively influence relationships, thus improving depression. IPT also incorporates attachment theory by addressing interpersonal conflicts, transitions, and grief in the context of relationships. IPT-A was designed as a 12-week treatment for nonpsychotic depressed adolescents. It has been used when comorbidities exist, although it is most effective when depression is the primary diagnosis and comorbidities are minimal. Parental participation is recommended.

Motivational Interviewing

Motivational interviewing has its roots in the work of Carl Rogers on client-centered therapy. It is particularly useful with patients who do not come to treatment of their own accord, such as adolescents who abuse substances. The basic principle is that if a person experiences a safe, comfortable, and collaborative therapeutic environment, he or she will be more likely to participate with the therapist to acknowledge and examine his or her problems and make progress toward their resolution. Techniques deal explicitly with patient resistance and ambivalence to enhance motivation and willingness to consider change in behavior (see Nagy and Armstrong 2016 in “Additional Reading”).

Dialectical Behavior Therapy

Dialectical behavior therapy (DBT), based on the theoretical and empirical work of Linehan with adults suffering from borderline personality disorder, has been developmentally extended to work with suicidal adolescents and those who engage in nonsuicidal self-injury (see Miller et al. 2007 in “Additional Reading”). This approach

(DBT-A) combines creative individual, family, and group therapy and skills training seminars from a variety of conceptual frameworks with telephone-based coaching and crisis intervention and group consultation for therapists in the treatment of this very difficult and crisis-prone population.

■ PARENT COUNSELING AND PSYCHOEDUCATION

Parent counseling or guidance is primarily an educational intervention, conducted with individual parents or couples in groups (see Mendenhall et al. 2016 in “Additional Reading”). Parents learn about normal child development. The therapist helps parents to understand their child and his or her problems and to modify parental attitudes and behaviors that seem to be contributing to the difficulties. The therapist must try to understand the parents’ point of view and to be sympathetic to the hardships of living with a troubled child or adolescent. For parents who have serious difficulties of their own, parent counseling may merge into or pave the way for marital therapy or individual treatment of the adult.

Virtually all parents of children with psychiatric or learning problems need and deserve education on the nature of their child’s disorder and how to select among treatments and manage difficult behavior. Parents spend far more time with their children than the therapist does and can powerfully assist or impede treatment. Parents of children with chronic problems must become skilled advocates, to ensure that their children receive the treatment and schooling that they need. Carefully selected reading material (“bibliotherapy”) or Web sites may be extremely useful to parents (see Appendix, “Resources for Parents”).

■ BEHAVIOR THERAPY

Behavioral therapists view symptoms as resulting from habits, faulty learning, dysfunctional interaction patterns, or neurodevelopmental deficits, rather than from unconscious or intrapsychic motivation.

In an operant approach, positive and negative environmental contingencies (responses to the child's behavior) are identified and then modified in an attempt to decrease problem behaviors and increase adaptive ones. A *token economy*, one type of operant approach (see below), can be used successfully by parents, teachers, therapists (with groups or individuals), and staff of inpatient or day treatment programs.

Social learning theory integrates operant conditioning theory with an understanding of cognitive processes and emphasizes the importance of learning new behaviors by observing or imitating others. For example, modeling is used in the treatment of children's anxiety and fears to decrease social withdrawal and teach adaptive skills.

Behavior therapy is commonly used to reduce symptoms of ADHD, oppositional defiant disorder, and conduct disorder. Central to assessment and treatment planning is a functional behavior analysis that evaluates the "ABCs" of problem behaviors—that is, the antecedents, the behaviors themselves, and the consequences, intended or not, of the behaviors. This often illuminates ways in which children and adults may both have a role in triggering and maintaining the problem behaviors and suggests strategies for modifying the interactions and the behaviors.

Parent Management Training

Parent management training may be done with individual families or in groups. Programs have been developed for families of young children, older children, and adolescents (see "Additional Reading"). Families begin by charting targeted behaviors and their antecedents and consequences. Initial treatment goals should focus on a limited number of target behaviors with which families are likely to achieve early success. As family members gain skills and reach goals, more complex or difficult behaviors are approached. Teaching techniques include written and verbal instruction in principles of social learning and behavior modification, modeling by the therapist, behavioral rehearsal of skills to be used, and homework assignments with subsequent review, feedback, and repetition.

Children with disruptive and noncompliant behavior often elicit high levels of negative attention from the adults in their lives, while their appropriate behavior often goes unnoticed. The parent-child relationship may have become strained or antagonistic. A first step in parent management training is to emphasize and teach how to reinforce positive behaviors with attention, praise, or other positive reinforcers. Parents are encouraged to devote at least brief periods of time to child-directed activities without questioning or redirection. Parents are also advised to ignore mild forms of misbehavior. Once these habits are established, the problematic behaviors are addressed using a reward system or token economy in which a child can earn tokens or points for positive behavior which they can later cash in for positive reinforcers (rewards). As the child masters the behaviors, the rewards can be gradually faded (reduced in frequency) or transferred to another behavioral target. Punishment generally should be brief, proximate to the misbehavior, and consistently enforced. Time out, which puts the child in a quiet, boring area where he or she experiences a "time-out" from accidental or naturally occurring positive reinforcement, is an effective punishment if properly applied, especially for younger children. Some parent management programs also address parental stress and parental cognitions about their children and their behaviors. These interventions help parents stay calm and rational rather than escalating negative situations.

Treatment is most effective for young children and those with less severe and persistent behavior problems. Characteristics that have been associated with less positive outcome in parent training include low socioeconomic status, parental psychiatric problems, marital conflict, lack of a social support network, harsh punishment practices, and parent history of antisocial behavior (Kazdin 1997). Families with these characteristics should receive maximally potent interventions, with attention to the parents' individual or marital problems as necessary. Additional topics may need to be addressed, such as skills for resolving marital conflict or managing parental anger. More highly functioning families may be able to succeed with written materials only or by using manuals or videotapes supplemented by group discussion. The therapist must be aware of ethnic

and cultural beliefs and customs regarding child development and parenting.

Behavioral intervention can be done in the context of family therapy, including techniques such as parent-child contingency contracting. A social contract is written that specifies the behaviors that the parent and child will change, with contingencies. The family is trained to negotiate and solve problems. These techniques may be particularly useful for adolescents.

Classroom Behavior Modification

Techniques for use in schools include class rules, attention to positive behavior, token economies, and response-cost programs (reinforcers are withdrawn in response to undesirable behavior). The teacher may dispense simple reinforcers such as praise, stars on a chart, or classroom privileges, or parents may provide positive reinforcement and/or response cost based on a "daily report card" that the teacher sends home, rating the child's performance that day on selected target behaviors (Kelley 1990).

■ FAMILY-BASED ASSESSMENT AND TREATMENT

Role of the Family in Treatment

Attempts to treat disorders in children or adolescents without considering the persons with whom they live or have significant relationships are doomed to failure. Change in one family member, whether as a result of a psychiatric disorder, psychiatric treatment, normal developmental process, or life events, will affect other family members and their relationships. Family constellations vary immensely, from the traditional (but increasingly rare) nuclear family to grandparents functioning as parents, a single-parent family (with or without contact with a second parent), a stepfamily, or an adoptive or foster family. The term *parents* refers here to adults filling the parenting role regardless of their biological or legal relationship to the patient.

Supportive therapy with families includes counseling in methods of changing behavior; encouraging more positive and realistic parental feelings and attitudes toward children; helping family members to manage their emotional reactions to the child's psychiatric disorder; detecting and obtaining treatment for psychiatric disorders in parents and siblings; and advising parents about schools, treatment modalities, community or leisure activities, and sometimes complex custody and placement decisions.

Family Therapy

Indications

There is empirical support for the effectiveness of family-based interventions (see Wendel and Gouze 2016 in "Additional Reading") as the primary or adjunctive treatment for behavior problems (including adolescent substance abuse), depression, bipolar disorder, anxiety, and eating disorders.

Family therapy may be particularly useful when dysfunctional interactions or impaired communication within the family appear to be related to the presenting problem or when symptoms begin or worsen with a new developmental stage or a change in the family such as divorce, remarriage, adoption, or foster placement. If more than one family member has symptoms, family therapy may be more efficient and effective than multiple individual treatments. It should be considered when one family member improves with treatment but another, not in treatment, worsens. Cases in which the identified patient is relatively unmotivated to participate or to change are likely to be more successful in family therapy than in individual therapy. Attention to family systems issues may be useful when progress is stalled in individual therapy or in behavior therapy. Often family therapy is part of a multimodal treatment plan.

If patients have clearly organic physical or mental illness or if the family equilibrium is precarious and one or more family members are at serious risk for decompensation, family therapy may be useful as an adjunct to other treatments, such as medication or hos-

pitalization. A patient who is acutely psychotic, violent, or delusional regarding the family should not be included in family therapy sessions. Family sessions may not be helpful when a parent has severe but unworkable psychiatric disturbance or when the child or adolescent strongly prefers individual treatment. Children should not be included in family sessions when parents continue (despite redirection) to criticize them or to share inappropriate information, when the most critical issues are marital, or when parents primarily need specific concrete help with practical affairs or parent training. Cultural sensitivity and competence are even more important when working with families than with individuals.

Types of Family Therapy

Structural family therapy (developed by Minuchin) has been the model most used and studied in families in which a child or an adolescent is the identified patient. Its focus is on the present; the identified patient's symptoms are seen as serving a function for the family. The assessment process includes mapping the structure of the family, including the location and permeability of boundaries between family members and around the family and its subsystems. Other important variables are the character and flexibility of alignments of family members, including alliances (joining two or more members in a common interest or task) and coalitions (joint actions directed against one or more family members). Data are gathered on communication patterns and the distribution of power within the family and on the family's sources of stress and support in the environment.

The therapist uses assigned tasks and his or her own interactions with family members to influence the family to change its structure and thereby its functioning, resulting in resolution of the presenting symptoms. Relabeling (i.e., redefining a behavior or symptom to give it a different, usually less negative, meaning for the family) opens alternative pathways for family interactions.

Other types of family-based interventions are used for specific circumstances, such as *family grief therapy*, *in-home family preserva-*

tion services, infant or toddler–parent interactive psychotherapy, and family approaches for chronic physical illness. Models that have been used to treat conduct disorders include Patterson’s *behavioral family therapy*; Alexander’s *functional family therapy*, which combines behavioral and family systems theories and techniques with attention to cognitive processes; and *multisystemic therapy*, developed by Henggeler and Borduin (see “Additional Reading”), which uses various home-based therapeutic techniques (family therapy, parent training, and cognitive-behavior therapy) along with direct practical assistance to the family in the context of the adolescent’s natural environment of home, school, and neighborhood. For eating disorders, *Maudsley family therapy* is used (see Chapter 10, “Feeding and Eating Disorders”).

Psychoeducational family therapy, initially developed for families of adults with schizophrenia, has been extended to families of patients with childhood disorders, such as eating disorders, ADHD, and anxiety and mood disorders. Detailed didactic presentations about the disorder are designed to enhance the family’s support networks and to improve the family’s coping skills through increased understanding of the illness, its treatment, and home behavior management techniques. Ongoing treatment uses family systems interventions when educational and behavioral techniques are blocked by maladaptive family structures or processes. Multiple family group interventions are also used in outpatient treatment programs for various disorders and for families of patients in inpatient or partial hospitalization programs.

■ GROUP THERAPY

Indications

Group therapy may be particularly useful for children and adolescents, who are more willing to reveal their thoughts and feelings to peers than to adults. It also offers the clinician an opportunity to observe in vivo patients’ interpersonal skills and interactions with peers and allows patients to observe and practice important social skills and to benefit from peer companionship and support. Feedback and

input from peers may be given more credence than observations and suggestions from an adult therapist. Often target symptoms such as aggression, withdrawal, shyness, and/or deficient social skills with peers are not apparent or accessible to intervention in individual therapy. Group therapy can provide a powerful context in which to teach social skills and language, especially for children with autism spectrum disorder or developmental delays. CBT and IPT-A for depressed adolescents, CBT for anxiety disorders, and DBT can all be delivered in a group setting. Motivational therapy and twelve-step groups are used for adolescents who have problems with drug or alcohol abuse. Multifamily psychoeducational groups are often used for outpatients and on inpatient units or partial hospitalization programs. Support groups include members who share a common stressor (e.g., sexual abuse, parental divorce, a chronic physical illness, loss of a loved one). Other groups are specifically targeted to a single psychiatric disorder. Groups that focus on social skills work best with a mixture of patients.

Group psychotherapy is contraindicated for extremely fragile youth and those who are psychotic or paranoid. Adolescents with sociopathic traits or behaviors should not be included in groups with others who might be victimized or intimidated. Severely aggressive or hyperactive children should probably not be included in outpatient groups because of the difficulty in controlling their behavior, the risk of their modeling of problem behaviors for other children, and their intimidation of less assertive children.

Technical Considerations

All of the theoretical models used in individual therapy may be used in group therapy. Therapy may be exclusively verbal or may include expressive arts techniques (such as psychodrama, dance, or arts and crafts), sports activities, or behavioral techniques (such as anger management skills, modeling and practicing social skills, cooperation, and negotiation). Whatever the type of group, the therapist must understand the dynamics of group process. Psychoeducation and supportive treatment can be provided efficiently in groups.

Some groups have a defined, limited duration, from 6 weeks to an academic year; others are longer term. Therapy groups conducted in therapeutic schools, inpatient units, or day hospitals are typically open ended and often include all children enrolled in the program, with youth typically grouped by age. Special topic groups focusing on anger management and problem-solving skills, cognitive behavior techniques, substance use, social skills, abuse-related issues, or preparation for discharge from the program may also be offered.

Group members should be in the same or adjacent developmental stages. Children and adolescents change so dramatically as they develop that an age span broader than 2 or 3 years is likely to impede the therapeutic group process. When groups for pre- and early adolescents are being formed, developmental stage is often more important than chronological age. Early adolescent girls mature physically, emotionally, and socially more rapidly than same-age boys. If not taken into account when forming a group, this differential maturation, as well as individual differences in developmental trajectories, may negatively affect group dynamics.

Opinions differ on whether to include boys and girls in the same group. Some issues might be better handled in single-sex groups, although children and adolescents benefit from interactions with opposite-sex peers. Combining boys and girls, although initially more difficult, may ultimately be more productive, depending on the setting. In mixed-sex groups, efforts should be made to have approximately equal numbers of boys and girls. In adolescent groups, the leader(s) must be alert to possible sexual undercurrents and acting out, while facilitating the discussion of sexual concerns and the practicing of social skills.

Children with ADHD are often referred to group therapy because of their difficulty with peer relationships and their lack of insight into their difficulties. Children who are taking stimulant medication may need to receive a dose before the group meets to help them benefit from group therapy and not disrupt it for others.

Of all modalities of therapy, the need for co-therapists is most evident in group treatment. Groups are complex, with many events occurring simultaneously, and a second observer is valuable. In

groups of younger children, an extra pair of hands is often needed. Co-leaders who differ from each other in age, sex, race, or culture may expand the opportunities for different types of patient–therapist relationships. The group structure should fit the nature of the group and the patients. The leaders are responsible for maintaining structure and control of behavior within the group. The leaders must be explicit about the rules of confidentiality for the group because the group setting increases the risk of breach of confidentiality.

For younger children, games and craft activities can provide a useful structure, but the leader(s) must ensure that recreation does not become the only function of the group. Behavior modification, cognitive problem-solving techniques, and anger management skills may be employed. Many child and adolescent patients will not spontaneously attempt to relate to other children. Others have been rejected, bullied, or scapegoated by peers. If the group therapy is successful, the patients will be able to generalize the skills learned in the group to form relationships with peers at school and in their neighborhoods.

Parental involvement is especially crucial for preschool- and school-age children because parents can provide valuable information regarding important events in the child's environment and treatment progress outside of the group setting. Adolescents are better able than younger children to report on external events and progress. They are also more sensitive to confidentiality issues; thus it may be advisable to keep parents more distant from the content of sessions. For children of all ages, the therapist should inform parents of the general goals of the group and their child's progress toward specific goals. Parent education in development and behavior management is often effectively provided in a coordinated parent group session. Parents may also appreciate the opportunity to meet with other parents whose children have similar problems.

■ SYSTEMS OF CARE

Innovative community services, often called “wraparound” programs, are used to avoid hospitalization or placement in residential treat-

ment. Such services attempt to address complex needs (psychological, school, family, peer, spiritual) in a strength-based community model. Wraparound services involve a variety of interventions with individualized programming and therapy, active involvement of family and community members, integration with social services (such as child welfare, financial support, and housing), as well as the educational and justice systems; and use of interventions in the home, neighborhood, community, and school rather than in the traditional office or hospital setting. Crisis intervention teams, brief respite placement, and in-home therapy/supervision are typically included. Funding sources vary depending on the specific community or state.

■ MILIEU TREATMENT: INPATIENT HOSPITALIZATION, RESIDENTIAL TREATMENT, AND PARTIAL HOSPITALIZATION

Inpatient Hospitalization and Residential Programs

Indications

Because children and adolescents should be treated in the setting that is least restrictive and disruptive to their lives, hospital or residential treatment is reserved for youngsters who have not responded to outpatient treatment due to severity of symptoms, lack of motivation, or severe disorganization of the patient or family. If there is concomitant physical illness requiring skilled medical/nursing care, pediatric hospitalization may be necessary. In cases where the family's presentation for psychiatric services has been delayed, symptom severity may require more intensive treatment from the outset, even before outpatient services are attempted.

Inpatient hospitalization is usually an acute event that is precipitated by immediate physical danger to self or others, acute psychosis, a crisis in the environment that reduces the ability of the caregiving adults to cope with the child or adolescent, or failure of

less intensive forms of treatment. Hospitalization may be needed for a more intensive, systematic, and detailed evaluation and observation of the patient and family than is possible in an outpatient or a day treatment program or if the patient is resistant to outpatient or day treatment. Most hospital lengths of stay are very short (i.e., 5–10 days). With exceptions only for the most severely ill children and adolescents (or those unfortunately awaiting residential placement), hospitalization is now typically used only to stabilize the acute clinical situation and to arrange for treatment in a less restrictive setting. Placement in a residential treatment center may be indicated for children and adolescents with chronic behavior problems such as aggression, running away, truancy, substance abuse, school refusal, or self-destructive acts that the family, foster home, and/or community cannot manage.

Components of Treatment

Pharmacotherapy. Hospitalization offers an opportunity for trials of medications in children or adolescents whose conditions have not responded to previous treatment, who have complicated or unclear diagnoses, who have medical problems complicating pharmacotherapy, or whose parents are not able to reliably administer medication and report on efficacy and side effects. Resumption of previously prescribed, but not reliably taken, medication is often needed.

Individual psychotherapy. As newer treatment methods have evolved and hospital stays have become shorter, individual psychotherapy is less often used as an inpatient primary treatment modality. Regularly scheduled individual sessions with a therapist with whom the child or adolescent can develop a trusting relationship continue to be important in developing a more thorough understanding of the patient's developmental status and intrapsychic, familial, and social dynamics and assisting him or her to develop more adaptive methods of coping with strong emotions. The therapist may be able to help the patient better understand his or her own difficulties and benefit from the other treatments offered. In a short

hospital stay, the patient's trauma history may become apparent, but addressing traumas and losses is deferred to longer-term therapy.

Milieu therapy. Milieu therapy encompasses the entire treatment environment, including interactions with staff outside of scheduled therapy sessions. The 24/7 setting offers a valuable opportunity to observe the patient during meals, sleep, self-care, and play. Milieu structure provides clear rules and a regular routine to promote a sense of security and predictability as well as teaching of specific skills to increase self-esteem and competence. Many settings include a behavioral program that uses a token economy or privilege level system to manage behavior and to modify specific symptoms.

Group therapy. In addition to general or special topic groups, many programs include therapist-led community meetings in which patients practice coping and social skills, and learn to observe their own and others' behavior and to recognize the effect of their behavior on others.

Education. The majority of children and adolescents who require out-of-home placement have had problems in school. The small classes and highly trained teachers of a hospital unit or residential center can provide a detailed evaluation of a patient's academic strengths and weaknesses. One of the most important components of discharge planning is arranging for an appropriate educational placement and working with the teacher to set appropriate target goals. Youth in residential treatment centers are gradually integrated into a special education or mainstream program in the local public or private schools, although some larger residential centers have their own school on campus.

Family treatment. Work with families is an essential part of hospital treatment. Interventions may include family therapy, parent counseling in behavior management, and education about the nature of the child's disorder and treatment plan. Parents may require marital therapy; referral for individual assessment and treatment; or help with housing, finances, day care, or medical care.

Partial Hospitalization

A partial hospitalization program (PHP), also known as a day treatment program, may be best for those children or adolescents who require more intensive intervention than can be provided in outpatient visits but who are able to safely stay at home, in foster care, or in a group home. Patients who are at immediate risk of serious self-harm or harm to others would not be appropriate for a PHP and require an inpatient level of care. Compared with hospitalization or residential placement, day treatment is less disruptive to the patient and the family and can offer an opportunity for intensive work with parents, who typically attend the program on a regular basis. Daily planning and review of home management strategies enhance generalization and maintenance of gains made in treatment. A day program may be used to avoid the necessity of inpatient hospitalization; to aggressively address school refusal, psychiatric, and behavioral problems; or as a transition (“step-down”) for a patient who has been hospitalized.

Day treatment programs vary in design, treatment techniques, and patient populations, although all treat moderately to severely ill children and adolescents. Some programs provide a full 8-hour day, 5 days a week, and include a school program. Acute PHPs have typical lengths of stay of 1–3 weeks. Other programs, often called intensive outpatient programs (IOPs), meet in the late afternoon and evening hours (typically for 3 hours), after patients attend community schools, and on weekends, to facilitate parental attendance. Some agencies offer intensive summer day treatment programs or a therapeutic day camp. The modalities of treatment are variable but tend to be similar to those described for inpatient units; however, the staff-to-patient ratio is typically lower.

■ ADJUNCTIVE TREATMENTS

At times, an intervention that is not, strictly speaking, a psychiatric treatment may be recommended as part of a treatment plan. These could include spiritual, recreational, or extracurricular activities. These programs may be crucial to the child’s or adolescent’s well-

being and the treatment of the psychiatric disorder, or they may encourage progress or improve level of functioning.

Special Education Placements

Modified school programs are indicated for children and adolescents who cannot perform satisfactorily in regular classrooms or who need special structure or teaching techniques to reach their academic potential. These programs range in intensity from tutoring or resource classrooms several hours a week, to special classrooms in mainstream schools, to public or private schools that serve only children and adolescents with special educational needs. Resources differ from community to community, but most have programs for youth with intellectual disability, for those with specific learning disorders (learning disabilities), and for those whose emotional and/or behavior problems require a special setting for learning. Classes are small, with a high teacher-to-student ratio and specially trained teachers. Vocational evaluation and education may be crucial, especially for adolescents.

Students who do not need a special education placement but who need accommodations to meet standard academic goals may benefit from a 504 plan. The 504 plan (which refers to the section of the Rehabilitation Act of 1973 that prohibits discrimination based on disability) can specify accommodations such as extra time for testing, testing in a low-distraction setting, preferential seating, or check-ins with teachers or counselors for students with psychiatric diagnoses such as ADHD or anxiety.

Child and adolescent psychiatrists may be part of the interdisciplinary team as consultants or in school-based health and mental health clinics. These programs efficiently provide coordinated medical and mental health care, although funding can be difficult to maintain.

A boarding school may be useful when a parent-child problem is unresponsive to treatment or an appropriate placement is not available in the home community. Some boarding schools have special programs for children with specific learning disorders or psychiatric disorders.

Recreation

Learning to perform a sport or skill competently may be an important adjunct to treatment for children and adolescents who lack positive relationships with peers or adults because of social isolation, withdrawal, or being ignored or actively rejected. Trained recreation therapists work in various psychiatric and community settings and focus on teaching adaptive leisure skills and improving interactions with peers. A relationship with an adult such as a Big Brother or Sister or a YMCA counselor or sports coach offers an opportunity to interact with a peer group under supervision and may provide support and build self-esteem until the child or adolescent improves enough to establish relationships independently. Some families employ a high school or college student one or more afternoons a week to teach the child social and play skills, develop a relationship with the child, and provide structured time. This method also gives parents a respite and an opportunity to spend time with their other children.

Day or overnight summer camps are potentially a very helpful experience. Some youngsters can attend regular camp, whereas others need a therapeutic camp program geared toward children and adolescents with psychiatric or medical problems.

Foster Care

Placement in a foster home may be needed when parents are unwilling or unable to care for their child. Indications are clearest in cases of physical or medical neglect or physical or sexual abuse. Some families may not be able to provide the appropriate emotional nurturance and supervision. Court intervention is required for placement. Foster care should be short term, until parents are rehabilitated or the courts decide that more permanent placement is needed. Unfortunately, child welfare agencies in many communities are overwhelmed, and children and adolescents may require advocacy with the appointment of a *guardian ad litem* attorney to facilitate return to parents, placement in a foster home or group home, or termination of parental rights to free the child for adoption, according to the conditions of the child and family. Children in foster care have higher rates of

both psychiatric and physical disorders than do children in the general population (Turney and Wildeman 2016).

Children with severe behavior or physical problems or older adolescents who are difficult to place or maintain in foster or adoptive homes may be placed in group homes with trained staff. These programs vary in staffing and intensity of the treatment offered. Some resemble residential treatment, whereas others simply provide a supervised residence.

Parent Support Groups

Various groups that provide education and support for parents, as well as conduct fund-raising and advocacy for services, have been organized by parents with professional support. Examples are listed in the Appendix. Numerous local groups (many of which are affiliated with national organizations) focus on specific medical or psychiatric disorders or on more generic psychological problems of childhood. They can provide a powerful adjunct to more traditional professional services.

■ REFERENCES

- Kazdin AE: Parent management training: evidence, outcomes, and issues. *J Am Acad Child Adolesc Psychiatry* 36:1349–1356, 1997
- Kelley ML: *School-Home Notes: Promoting Children's Classroom Success*. New York, Guilford, 1990
- Strupp HH: *Psychotherapy: Clinical Research, and Theoretical Issues*. New York, Jason Aronson, 1973
- Turney K, Wildeman C: Mental and physical health of children in foster care. *Pediatrics* 138(5), pii e20161118 Epub 2016 Oct 17 27940775

■ ADDITIONAL READING

- Barkley RA: *Defiant Children: A Clinician's Manual for Assessment and Parent Training*, 3rd Edition. New York, Guilford, 2013
- Barkley RA, Robin AL: *Defiant Teens: A Clinician's Manual for Assessment and Family Intervention*, 2nd Edition. New York, Guilford, 2014

- Beidel DC, Reinecke MA: Cognitive-behavioral treatment for anxiety and depression, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 973–991
- Chorpita BF, Weisz JR: MATCH-ADTC: Modular Approach to Therapy for Children With Anxiety, Depression, Trauma, or Conduct Problems. Satellite Beach, FL, PracticeWise LLC, 2009
- Cohen JA, Mannarino AP, Deblinger E: Treating Trauma and Traumatic Grief in Children and Adolescents, 2nd Edition. New York, Guilford, 2017
- Greene RW, Ablon JS: Treating Explosive Kids: The Collaborative Problem-Solving Approach. New York, Guilford, 2006
- Gunlicks-Stoessel ML, Mufson L: Interpersonal psychotherapy for depressed adolescents, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 959–971
- Henggeler SW, Schoenwald SK, Borduin CM, et al: Multisystemic Therapy for Antisocial Behavior in Children and Adolescents, 2nd Edition. New York, Guilford, 2009
- McMahon RJ, Forehand RL: Helping the Non-Compliant Child: Family Based Treatment for Oppositional Behavior, 2nd Edition. New York, Guilford, 2003
- Mendenhall AN, Arnold LE, Fristad MA: Parent counseling, psychoeducation, and parent support groups, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 875–899
- Miller AL, Rathus HK, Linehan MM: Dialectical Behavior Therapy With Suicidal Adolescents. New York, Guilford, 2007
- Mufson L, Dorta KP, Moreau D, et al: Interpersonal Psychotherapy for Depressed Adolescents, 2nd Edition. New York, Guilford, 2004
- Nagy P, Armstrong S: Motivational interviewing, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 993–1006
- Petti TA: Milieu treatment: inpatient, partial hospitalization, and residential programs, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 1027–1046
- Pfiffner LJ, Kaiser NM: Behavioral parent training, in Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd Edition. Edited by Dulcan MK. Ar-

- lington, VA, American Psychiatric Association Publishing, 2016, pp 901–935
- Poncin Y, Woolston J: Systems of care, wraparound services, and home-based services, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 1007–1026
- Szigethy E, Weisz JR, Findling RL: *Cognitive-Behavior Therapy for Children and Adolescents*. Arlington, VA, American Psychiatric Publishing, 2012
- Terr L: Individual psychotherapy, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 849–874
- Webster-Stratton C, Herbert M: *Troubled Families-Problem Children: Working With Parents: A Collaborative Process*. New York, Wiley, 1994
- Wendel R, Gouze KR: Family-based assessment and treatment, in *Dulcan's Textbook of Child and Adolescent Psychiatry*, 2nd Edition. Edited by Dulcan MK. Arlington, VA, American Psychiatric Association Publishing, 2016, pp 937–958

APPENDIX

RESOURCES FOR PARENTS

■ INFORMATION ON THE INTERNET

American Academy of Child and Adolescent Psychiatry

<http://www.aacap.org/>

3615 Wisconsin Ave., N.W.

Washington, D.C. 20016

Facts for Families

A large collection of information sheets written for the general public on development, problem behaviors, stressors, and psychiatric disorders. Available in English, Spanish, and Chinese.

Resource Centers

Contain consumer-friendly definitions, answers to frequently asked questions, clinical resources, expert videos, and abstracts from JAACAP and AACAP Annual Meeting Scientific Proceedings, and Facts for Families relevant to each topic. Topics include anxiety disorders, ADHD, autism, bipolar disorder, bullying, child abuse, conduct disorder, depression, disaster, military families, moving into adulthood, oppositional defiant disorder, and substance use.

Note. As scientific research advances, information and advice change. Many topics are controversial, even among experts. Parents should be encouraged to discuss questions with their child's pediatrician, child and adolescent psychiatrist, or other health or mental health professional.

Autism Spectrum Disorder: Parents' Medication Guide

Guides for Parents (<http://www.parentsmedguide.org/>)

A joint project of the American Psychiatric Association and the American Academy of Child and Adolescent Psychiatry—Extensive resources with information about diagnosis and medication.

- *ADHD Parents Medication Guide* (2013) (also in Spanish)
- *Parents' Medication Guide for Bipolar Disorder in Children and Adolescents* (2010)
- *The Use of Medication in Treating Childhood and Adolescent Depression: Information for Patients and Families* (2010)

Anxiety and Depression Association of America

<https://www.adaa.org/>

Autism Society of America

<http://www.autism-society.org/>

4340 East-West Highway, Suite 350

Bethesda, MD 20814

1-800-328-8476

Autism Spectrum Connection

<http://www.aspergersyndrome.org/>

Bright Futures

<http://www.brightfutures.org/>

Publications to promote and improve the health and well-being of children and adolescents.

CHADD (Children and Adults with Attention-Deficit/Hyperactivity Disorder), The National Resource Center on AD/HD

<http://www.chadd.org/>

800-233-4050

Child Mind Institute

<http://www.childmind.org/>

National Alliance on Mental Illness (NAMI)

<http://www.nami.org/>

3803 N. Fairfax Drive, Suite 100

Arlington, VA 22203

800-950-6264

National Child Traumatic Stress Network

<http://www.nctsn.org/>

**National Federation of Families for
Children's Mental Health**

<http://www.ffcmh.org/>

12320 Parklawn Drive

Rockville, Maryland 20852

240-403-1901

National Institute of Mental Health

<http://www.nimh.nih.gov/>

Tourette Association of America

<https://www.tourette.org/>

42-40 Bell Boulevard

Bayside, NY 11361

888-4-TOURET

Zero to Three

<http://www.zerotothree.org/>

1255 23rd Street, N.W.

Suite 350

Washington, DC 20037

800-899-4301

Developmental information for parents of children 4 years and younger, including how to enhance children's learning and social-emotional growth.

■ BOOKS

Child Development

American Academy of Child and Adolescent Psychiatry: *Your Child: What Every Parent Needs to Know*. New York, HarperCollins, 1998

American Academy of Child and Adolescent Psychiatry: *Your Adolescent: Emotional, Behavioral and Cognitive Development From Early Adolescence Through the Teen Years*. New York, HarperCollins, 1999

Managing Child Behavior

Barkley RA, Benton CM: *Your Defiant Child: 8 Steps to Better Behavior*, 2nd Edition. New York, Guilford, 2013

Barkley RA, Robin AL: *Your Defiant Teen: 10 Steps to Resolve Conflict and Rebuild Your Relationship*, 2nd Edition. New York, Guilford, 2014

Clark L: *SOS! Help for Parents: A Practical Guide for Handling Common Everyday Behavior Problems*, 2nd Edition. Bowling Green, KY, Parents Press, 1985

Faber A, Mazlish E: *How to Talk So Kids Will Listen and Listen So Kids Will Talk*. New York, HarperCollins, 1980

Forehand R, Long N: *Parenting the Strong-Willed Child: The Clinically Proven Five-Week Program for Parents of Two- to Six-Year-Olds*, 3rd Edition. New York, McGraw-Hill, 2010

Green RW: *The Explosive Child: A New Approach for Understanding and Parenting Easily Frustrated, Chronically Inflexible Children*, 3rd Edition. New York, HarperCollins, 2014

Kazdin AE: *The Kazdin Method for Parenting the Defiant Child*. New York, Mariner Books, 2009 (includes a DVD)

Nichols MP: *Stop Arguing with Your Kids: How to Win the Battle of Wills by Making Your Children Feel Heard*. New York, Guilford, 2004

Phelan TW: *1–2–3 Magic: Effective Discipline for Children 2–12*, 6th Edition. Naperville, IL, Sourcebooks Inc, 2016

Anger Management for Parents

Clark L: *SOS! Help for Emotions: Managing Anxiety, Anger, and Depression*, 2nd Edition. Bowling Green, KY, Parents Press, 2002

Psychiatric Disorders

Anxiety

Foa EB, Andrews LW: *If Your Adolescent Has an Anxiety Disorder: An Essential Resource for Parents*. New York, Oxford University Press, 2006

Last CG: *Help for Worried Kids: How Your Child Can Conquer Anxiety and Fear*. New York, Guilford, 2006

Manassis K: *Keys to Parenting Your Anxious Child*. Hauppauge, NY, Barron's Educational Series, 1996

March JS: *Talking Back to OCD*. New York, Guilford, 2007

McHolm AE, Cunningham CE, Vanier MK: *Helping Your Child With Selective Mutism: Practical Steps to Overcome a Fear of Speaking*. Oakland, CA, New Harbinger Publications, 2005

Rapee RM, Spence SH, Cobham V, et al: *Helping Your Anxious Child: A Step-by-Step Guide for Parents*. Oakland, CA, New Harbinger Publications, 2000

Wagner AP: *Worried No More: Help and Hope for Anxious Children*, 2nd Edition. Mobile, AL, Lighthouse Press, 2005

Attention-Deficit/Hyperactivity Disorder

Barkley R: *Taking Charge of ADHD: The Complete, Authoritative Guide for Parents*, 3rd Edition. New York, Guilford, 2013

Hinshaw SP, Ellison K: *ADHD: What Everyone Needs to Know*. New York, Oxford University Press, 2016

Jensen PS: *Making the System Work for Your Child With ADHD*. New York, Guilford, 2004

Zeigler Dendy CA: *Teenagers With ADD and ADHD: A Guide for Parents and Professionals*, 2nd Edition. Bethesda, MD, Woodbine House, 2006

Autism Spectrum Disorder (Including Asperger's Disorder)

Rosenblatt AI, Carbone PS: *Autism Spectrum Disorders: What Every Parent Needs to Know*. Elk Grove, IL, American Academy of Pediatrics, 2013

Siegel B: *Getting the Best for Your Child With Autism: An Expert's Guide to Treatment*. New York, Guilford, 2008

Depression and Bipolar Disorder

Birmaher B: *New Hope for Children and Teens With Bipolar Disorder*. New York, Three Rivers Press, 2004

Evans DL, Andrews LW: *If Your Adolescent Has Depression or Bipolar Disorder: An Essential Resource for Parents*. New York, Oxford University Press, 2005

Fristad MA, Arnold JSG: *Raising a Moody Child: How to Cope With Depression and Bipolar Disorder*. New York, Guilford, 2004

Miklowitz DJ, George EL: *The Bipolar Teen: What You Can Do to Help Your Child and Your Family*. New York, Guilford, 2008

Eating Disorders

Lock J, Le Grange, D: *Help Your Teenager Beat an Eating Disorder*. New York, Guilford, 2005

Walsh BT, Cameron VL: *If Your Adolescent Has an Eating Disorder: An Essential Resource for Parents*. New York, Oxford University Press, 2005

Tourette's Disorder

Walkup JT, Mink JW, McNaught KStP: *A Family's Guide to Tourette Syndrome*. Bayside NY, Tourette Syndrome Association, 2012

Psychiatric Medication

Dulcan MK, Ballard R (eds): *Helping Parents and Teachers Understand Medications for Behavioral and Emotional Problems: A Resource Book of Medication Information Handouts*, 4th edition. Washington, DC, American Psychiatric Publishing, 2015

Wilens TE, Hammerness PG: *Straight Talk About Psychiatric Medications for Kids*, 4th Edition. New York, Guilford, 2016

INDEX

Page numbers printed in boldface type refer to tables or figures.

- Abnormal Involuntary Movement Scale (AIMS), 352–353
- Abortion, and adolescent pregnancy, 284
- Abstinence, and treatment of substance use disorder, 250
- Academic achievement, standardized tests of, **18**
- Acceptance and commitment therapy (ACT), for obsessive-compulsive disorder, 149
- Acculturation gap, and immigration, 283
- Activity level, normal versus attention-deficit/hyperactivity disorder, 63
- Acute phase, of schizophrenia, 88
- Acute stress disorder
 - adjustment disorder and, 166
 - clinical description of, 158–159
 - course and prognosis of, 160–161
 - differential diagnosis of, 162, 166
 - epidemiology of, 159–160
 - etiology of, 160
 - evaluation of, 161–162
 - treatment of, 162–164
- Adaptive behavior
 - clinical description of intellectual disability and, 26
 - standardized tests of, **19**
- Adaptive Behavior Assessment System, Third Edition (ABAS-III), **19**
- Adenotonsillar hypertrophy, and obstructive sleep apnea, 209
- ADHD. *See* Attention-deficit/hyperactivity disorder
- ADHD Rating Scale–5, 61
- Adherence to treatment
 - chronic illness in children and, 294–295
 - pediatric psychopharmacology and, 308–309
 - stimulant medications for attention-deficit/hyperactivity disorder and, 318–319
- Adjunctive treatments, and psychosocial interventions, 393–396

Adjustment disorder

- clinical description of, 164
- course and prognosis of, 165–166
- differential diagnosis of, 162, 166
- epidemiology of, 164–165
- etiology of, 165
- evaluation of, 166
- high activity levels and, 63
- posttraumatic stress disorder and, 162
- separation anxiety disorder and, 121
- treatment of, 166–167

Administration methods, for pediatric psychopharmacology, 309

Adolescent(s). *See also* Age;

Children

- adjustment disorder in, 164, 165
- attention-deficit/hyperactivity disorder in, 59, 60
- autism spectrum disorder and, 47
- behavioral interventions for rumination disorder in, 174
- bipolar disorder in, 94, 107
- communication with during psychosocial interventions, 374
- discontinuation of lithium and relapse of mood disorders in, 341

- divorce of parents and, 276
- enuresis in, 195
- gender dysphoria in, **219–220**
- group therapy and, 389
- insomnia in, 206
- interpersonal psychotherapy for, 379, 387
- involvement of in evaluation process, 9
- major depression in, 106, 107, 298–299
- narcolepsy in, 208
- physical illness in, 289–297
- pregnancy in, 284–286
- process of change in, 2
- psychosocial interventions and resistant, 375
- risk factors for substance abuse in, **247**
- schizophrenia in, 88, 90
- sexual side effects of selective serotonin reuptake inhibitors and, 337
- side effects of antipsychotics and, 354
- sleep issues for, 203–204, 214
- special issues in psychopharmacology for, 305–309
- treatment of separation anxiety disorder in, 125
- understanding of death, 281–282
- use of DSM-5 for diagnosis in, 3–4

- Adolescent focused therapy
(AFT), for anorexia nervosa,
185
- Adolescent-onset conduct
disorder, 230
- Adoption, of children, 282–283
- Adults
antipsychotics for
schizophrenia in, 350
attention-deficit/hyperactivity
disorder in, 59–60
gender dysphoria and,
219–220, 221
rumination disorder and, 173
selective mutism and social
anxiety disorder in, 126
separation anxiety disorder in,
120–121
social anxiety disorder in, 130
Tourette's disorder and
unemployment in, 79
- Age. *See also* Adolescents;
Adults; Age at onset;
Children; Development;
Infancy
attention-deficit/hyperactivity
disorder and, 59
common fears by, **116**
conceptualization of death and,
281–282
development of language skills
and, 37
diagnosis of elimination
disorders and, 194, 198
dosages of drugs for children
and, 307
group therapy and span of, 388
parental divorce and, 276
reaction to physical illness and,
290–291
sexual abuse and, 268
sleep requirements by, 203
structure of evaluation and, 9
- Age at onset
of anorexia nervosa, 178
of autism spectrum disorder, 42
of bulimia nervosa, 186
of conduct disorder, 230, 234
of generalized anxiety disorder,
137
of major depression, 106
of mania, 94
of panic disorder, 134
of pica, 171
of restless legs syndrome, 213
of schizophrenia, 87
of selective mutism, 125
of separation anxiety disorder,
120
of sleep terrors, 212
of tic disorders, 78
- Aggression
anticonvulsants and, 356
antipsychotics and, 351–352
evaluation of, 271, 273
group therapy and, 387
lithium and, 339
treatment of, 274
- Agoraphobia, and panic disorder,
133, 134
- Akathisia, and antipsychotics,
355

- Alcohol abuse, and alcoholism.
See also Substance use disorder
 children of parents with, 298
 rates of in adolescents, 246
 secondary depression and, 108
 sexual abuse and, 271
- Alcoholics Anonymous (AA),
 250, 251
- Allergies, and attention-deficit/
 hyperactivity disorder, 71
- Alpha-adrenergic agonists, and tic
 disorders, 81–82, 351
- Alprazolam, 295
- American Academy of Child and
 Adolescent Psychiatry,
 399–400, 400
- American Academy of Pediatrics,
 47, 318
- American Association on
 Intellectual Disability and
 Developmental Disabilities
 (AAIDD), 33
- American College of Medical
 Genetics and Genomics
 (ACMG), 34
- American Heart Association, 318,
 326
- American Psychiatric Association,
 400
- Amotivational syndrome, as side
 effect of antidepressants,
 336–337
- Amphetamine
 attention-deficit/hyperactivity
 disorder and, 69, 314
 formulations and dosages of,
313
 side effects of, 315, 321
- Anger. *See also* Temper
 outbursts
 information resources for
 parents on management
 of, 403
 management training for,
 274
 oppositional defiant disorder
 and, 225, 226
- Anhedonia, and depressive
 disorders, 105, 107
- Anorexia nervosa (AN)
 avoidant/restrictive food intake
 disorder and, 176
 bulimic symptoms in, 187
 clinical description of, 177
 course and prognosis of,
 178–179
 differential diagnosis of, **183**
 epidemiology of, 177–178
 etiology of, 178
 evaluation of, 179, 182
 physical signs and medical
 complications associated
 with, **180–181**
 treatment of, 182, 184–185
- Anticholinergic side effects, of
 antipsychotics, 355
- Anticipatory guidance, for
 separation anxiety disorder,
 124
- Anticipatory mourning, and
 terminal illness, 294

- Anticonvulsants. *See also* Lithium
 indications for and efficacy of,
 356–358
 initiation of and ongoing
 treatment with, 358–359
 risks and side effects of,
 359–361
- Antidepressants. *See also* Atypical
 antidepressants; Selective
 serotonin reuptake
 inhibitors; Tricyclic
 antidepressants
 black box warning for use of by
 pediatric population, 132,
 336, 338
 dosages of, **329–330**, 335
 indications for, **329–330**,
 331–334
 initiation of and ongoing
 treatment with, 335–336
 manic or hypomanic episodes
 and, 96
 mechanisms of action, 331
 narcolepsy and, 208
 risks and side effects of,
 336–338
 separation anxiety disorder and,
 123
- Antipsychotics. *See also* Atypical
 antipsychotics
 autism spectrum disorder and,
 50
 conduct disorder and, 239
 dosages of, indications for,
 and side effects of,
344–349
 initiation of and ongoing
 treatment with, 352–353
 intellectual disability and, 36
 mechanisms of action, 342
 out-of-control behavior and,
 273
 risks and side effects of, 90,
 354–355
 schizophrenia and, 90
 tic disorders and, 82
- Antisocial behavior, and conduct
 disorder, 236
- Antisocial personality disorder,
 60, 230, 234
- Anxiety and Depression
 Association of America, 400
- Anxiety disorders. *See also*
 Generalized anxiety
 disorder; Panic disorder;
 Selective mutism;
 Separation anxiety disorder;
 Social anxiety disorder;
 Specific phobias
 aggressive behavior and, **272**
 attention-deficit/hyperactivity
 disorder and, 322
 atypical antidepressants for,
 333–334
 bipolar disorder and, 95
 children of parents with, 298
 comorbidity of, 95, 233, 322
 conduct disorder and, 233
 deficits in attention and, 63
 differential diagnosis of, 138
 information resources for
 parents on, 403

- Anxiety disorders (*continued*)
- intellectual disability and
 - symptoms of, 29
 - posttraumatic stress disorder
 - and symptoms of, 161
 - selective serotonin reuptake
 - inhibitors and, 322, 333
 - sleep-related concerns in, 214
 - Tourette's disorder and, 77
- Apathy
- depressive disorders and, 105
 - side effects of antidepressants
 - and, 336–337
- ARFID (Avoidant/restrictive food intake disorder), 175–176, **183**
- Argumentative/defiant symptoms,
 - of oppositional defiant disorder, 226
- Aripiprazole, 50, 96, 351
- Arousal, and posttraumatic stress disorder, 158, 159
- Asenapine, 96
- Asian Americans, and use of
 - carbamazepine, 358
- Asperger's disorder, 41. *See also*
 - Autism spectrum disorder
- Assent, for pharmacotherapy in children, 309
- Assessment, of emergencies,
 - 255–256, 258–259.
 - See also* Evaluation
- Atomoxetine
 - attention-deficit/hyperactivity disorder and, 69, 82, 322–324
 - indications for and efficacy of, 322–323
 - initiation of and treatment with, 323
 - risks and side effects of, 323–324
 - tic disorders and, 82
- Attachment disorders, 267.
 - See also* Disinhibited social engagement disorder; Reactive attachment disorder
- Attachment theory
 - interpersonal psychotherapy and, 379
 - separation anxiety disorder and, 119
- Attention-deficit disorder (ADD),
 - use of term, 51
- Attention-deficit hyperactivity disorder (ADHD)
 - antidepressants for, 334
 - atomoxetine and, 322–324
 - autism spectrum disorder and, 47, 50
 - behavior therapy for, 381
 - bipolar disorder and, 95
 - clinical description of, 50–51, 54–55
 - comorbidity in, 26, 28, 56, 75, 76, 77, 80, 82, 195, 199, 226, 232, 238, 246
 - conduct disorder and, 232, 238
 - course and prognosis of, 59–60
 - developmental coordination disorder and, 75

- differential diagnosis of, 62–64, 74, 95, 157, 229
- disinhibited social engagement disorder and, 157
- disruptive mood dysregulation disorder and, 102, 103
- DSM-5 diagnostic criteria for, **51–54**
- encopresis and, 199, 200
- enuresis and, 195
- epidemiology of, 55–56
- etiology of, 56–58
- evaluation of, 60–62
- genetics of, 56, **57**, 58
- group therapy for, 388
- guanfacine and clonidine for, 324–325
- information resources for parents on, 403–404
- intellectual disability and, 26, 28, 35–36
- medical factors in, **57**, 58
- melatonin for sleep problems in, 361
- obstructive sleep apnea and, 214
- omega-3 fatty acids and, 362
- oppositional defiant disorder and, 226, 229
- specific learning disorder and, 73, 74
- stimulant medications for, 310, 314, 316–322
- substance use disorder and, 246
- suicide and, 261
- tic disorders and, 76, 77, 80, 82
- treatment of, 35–36, 64, 66–71, 82
- Atypical antidepressants
 - anxiety disorders and, 333–334
 - autism spectrum disorder and, 334
 - definition of, 328
 - indications for and dosages of, **329**
 - mechanisms of action, 331
 - side effects of, 338
- Atypical antipsychotics
 - conduct disorder and, 239
 - indications for and efficacy of, 343, 350–352
 - mechanisms of action, 342
 - obesity and, 289
 - obsessive-compulsive disorder and, 149
- Autism Observation Schedule (ADOS), 48
- Autism Diagnostic Interview, Revised (ADI-R), 48
- Autism Society of America, 400
- Autism spectrum disorder (ASD)
 - antidepressants for, 334
 - antipsychotics for, 350–351
 - attention-deficit/hyperactivity disorder and, 64, 314
 - clinical description of, 41–45
 - communication disorders and differential diagnosis of, 39
 - comorbidity of, 28, 64, 314
 - course and prognosis of, 46–47
 - differential diagnosis of, 48–49, 122, 148, 157

Autism spectrum disorder (ASD)

(continued)

epidemiology of, 45

etiology of, 46

evaluation of, 47–48

fragile X syndrome and, 32

guanfacine or clonidine for, 326

information resources for

parents on, 404

intellectual disability and, 28

obsessive-compulsive disorder
and, 148reactive attachment disorder
and, 157

schizophrenia and, 89

separation anxiety disorder and,
122

sleep problems and, 203, 214

speech sound disorder and, 41

stimulant medications and,
314

treatment of, 49–50

Autonomy, medically ill

adolescents and loss of, 291.

See also Confidentiality

Avoidance, and posttraumatic

stress disorder, 158, 159

Avoidant/restrictive food intake

disorder (ARFID), 175–176,

183

Axis V, replacement of in DSM-5,

4

Bariatric surgery, and obesity, 289

Barium enema, and encopresis,

199

Bayley Scales of Infant and

Toddler Development, Third

Edition (Bayley-III), 20, 33

BEARS (Bedtime, Excessive day-
time sleepiness, Awakenings,
Regularity, and Snoring), 204Behavior. *See also* Adaptive

behavior; Aggression;

Anger; Antisocial behavior;

Apathy; Behavior

modification; Behavior

therapy; Impulsivity;

Inattention; Irritability;

Repetitive behaviors;

Ritualistic behaviors;

Self-injurious behaviors;

Stereotypic behaviors

antipsychotics and problems in,

348

information resources for

parents on, 402–403

out-of-control as emergency,

257, 271–274

selective mutism and inhibition

of, 126

separation anxiety disorder and

inhibition of, 120

side effects of carbamazepine

and, 360

Behavioral family therapy, 386

Behavioral toxicity, of

psychoactive drugs, 306, 359

Behavior modification. *See also*

Behavior therapy

attention-deficit/hyperactivity

disorder and, 69

- conduct disorder and, 238
- enuresis and, 196–197
- insomnia and, 206
- intellectual disability and, 34–35
- interventions for medically ill children and, 296–297
- oppositional defiant disorder and, 229
- techniques for use in schools, 383
- Behavior therapy. *See also*
 - Behavioral family therapy;
 - Behavior modification;
 - Cognitive-behavioral therapy
- autism spectrum disorder and, 49
- encopresis and, 200
- pica and, 172–173
- psychosocial interventions and, 380–383
- restless legs syndrome and, 214
- tic disorders and, 81
- Benzodiazepines
 - medically ill children and, 295
 - out-of-control behavior and, 274
 - panic disorder and, 135
 - separation anxiety disorder and, 123
 - sleep terrors and, 212
 - social anxiety disorder or specific phobias and, 132
- Benztrapine, 354
- Bereavement, and death of family member, 280. *See also* Persistent depressive disorder
- Bibliotherapy, and oppositional defiant disorder, 229
- Bicultural identity, and immigration, 283
- Binge eating, and bulimia nervosa, 186
- Binge-eating/purging type, of anorexia nervosa, 177
- Biomedical etiologies, for intellectual disability, 29, **31**
- Biopsychosocial evaluation, **8–9**, 235
- Bipolar depression, 350, 358, 362
- Bipolar disorder
 - anticonvulsants for, 356–357
 - antipsychotics for, **344**, **345**, **346**, **347**, 350
 - attention-deficit/hyperactivity disorder and, 63
 - childhood-onset depression and, 107
 - children of parents with, 298
 - clinical description of, 93–94
 - conduct disorder and, 236
 - course and prognosis of, 95
 - differential diagnosis of, 95–96, 236
 - epidemiology of, 94
 - etiology of, 94–95
 - information resources for parents on, 404
 - lamotrigine for, 357–358
 - lithium for, 338
 - treatment of, 96–97

- Black box warnings, on
 - medications, 132, 323–324, 336
- Bladder training exercises, and enuresis, 197
- Blood urea nitrogen (BUN), and lithium, 339, 341
- Blue light therapy, and circadian rhythm sleep-wake disorders, 210
- Boarding schools, and special education, 394
- Body dysmorphic disorder, 151
- Body image, and anorexia nervosa, 177, 179, 185
- Body mass index (BMI), 286
- Bone, and medical complications of anorexia nervosa and bulimia nervosa, **180**
- Brain imaging, and attention-deficit/hyperactivity disorder, 57–58. *See also* Neuroimaging
- Brain injury, and attention-deficit/hyperactivity disorder, **57**.
See also Head trauma
- Brief focused therapy, and divorce, 278
- Bright Futures, 400
- Bulimia nervosa
 - clinical description of, 185–186
 - course and prognosis of, 187–188
 - epidemiology of, 186–187
 - etiology of, 187
 - evaluation of, 188–189
 - physical signs and medical complications associated with, **180–181**
 - treatment of, 189–190
- Bupropion
 - attention-deficit/hyperactivity disorder and, 334
 - formulations of, 335
 - mechanism of action, 331
 - side effects of, 338
- Caffeine, 71, 206
- CAGE, 249
- Callous-unemotional (CU) traits, and conduct disorder, 231
- Carbamazepine
 - conduct disorder and, 239
 - initiation and ongoing treatment with, 358–359
 - mania and, 357
 - side effects of, 360
- Cardiac tests, and stimulant medications, 318
- Cardiovascular complications, of anorexia nervosa and bulimia nervosa, **180**
- Cardiovascular history, and initiation of guanfacine or clonidine, 326
- Case managers, and treatment planning, 21
- Cataplexy, 207
- CD. *See* Conduct disorder
- Centers for Disease Control and Prevention (CDC), 45, 245, 260, 284

- Central nervous system disorders, psychiatric sequelae of, 292
- CHADD (Children and Adults with Attention-Deficit/Hyperactivity Disorder), 67, 400
- Change, process of in children and adolescents, 2
- Checklist for Autism in Toddlers (CHAT), 47
- Child abuse. *See also* Sexual abuse
clinical description of, 265–266
domestic violence and, 275
epidemiology of, 264–265
etiology of, 265, 266
evaluation of, 266
interventions for, 267
mandated reporting of, 264
out-of-control behavior and, **272**
posttraumatic stress disorder and, 160, 161
psychological sequelae of, 266–267
- Child and Adolescent Psychiatric Assessment (CAPA), **15**
- Child Attention Problems (CAP) Rating Scale, 64, **65–66**, 316–317
- Child Behavior Checklist (CBCL), 14, 61
- Child Depression Rating Scale, 109
- Child and Family Traumatic Stress Intervention (CFTSI), 162
- Childhood disintegrative disorder, 41. *See also* Autism spectrum disorder
- Childhood-onset conduct disorder, 230
- Childhood-onset schizophrenia (COS), 87
- Childhood traumatic grief disorder, 280
- Child Mind Institute, 401
- Child-Parent Psychotherapy (CPP), 163
- Child PTSD Symptom Scale, 162
- Children. *See also* Age; Anxiety disorders; Bipolar disorder; Child abuse; Depressive disorders; Disruptive, impulse-control, and conduct disorders; Elimination disorders; Evaluation; Feeding and eating disorders; Gender dysphoria; Neurodevelopmental disorders; Obsessive-compulsive and related disorders; Schizophrenia; Sleep-wake disorders; Substance use disorder; Trauma- and stressor-related disorders; Treatment
adoption of, 282–283
communication with during psychosocial interventions, 374
comprehensive evaluation of, 7–20

Children (*continued*)

- controversy on diagnosis of
 - bipolar disorder in, 93
 - cultural factors for immigrant families and, 283–284
 - DSM-5 diagnostic criteria for gender dysphoria in, **218–219**
 - emergencies involving, 255–274
 - family transitions and, 274–283
 - frequency and common features of sleep problems in, 203
 - obesity in, 286–289
 - overview of treatment for, 5
 - physical illness in, 289–297
 - process of change in, 2
 - of psychiatrically ill parents, 297–299
 - psychosocial interventions and resistant, 375
 - special issues in
 - psychopharmacology for, 305–309
 - use of DSM-5 for diagnosis in, 3–4
 - use of term in context, 1
- Children's Apperception Test (CAT), 20
- Children's Depression Inventory (CDI), 107
- Children's Yale-Brown Obsessive Compulsive Scale (C-YBOCS), 148
- Child Stress Disorders Checklist, 162

- Child Symptom Inventory, 61
- Chlorpromazine, 352, 355
- Chronotherapy, for delayed sleep-phase syndrome, 210
- Cigarette smoking, and tobacco use, 245–246
- Circadian rhythm sleep-wake disorders, 210
- Clinical description
 - of acute stress disorder, 158–159
 - of adjustment disorders, 164
 - of anorexia nervosa, 177
 - of attention-deficit/hyperactivity disorder, 50–51, 54–55
 - of autism spectrum disorder, 41–45
 - of bipolar disorder, 93–94
 - of bulimia nervosa, 185–186
 - of child maltreatment, 265–266
 - of communication disorders, 36
 - of conduct disorder, 230–231
 - of disinhibited social engagement disorder
 - of disruptive mood dysregulation disorder, 101–102
 - of encopresis, 197–198
 - of enuresis, 193–194
 - of gender dysphoria, 218, **218–220**
 - of generalized anxiety disorder, 135, 137
 - of insomnia, 205
 - of intellectual disability, 25–26, **27**

- of major depressive disorder, 104–105
- of narcolepsy, 207
- of obstructive sleep apnea, 208–209
- of panic disorder, 133
- of persistent depressive disorder, 104–105
- of pica, 171
- of posttraumatic stress disorder, 158–159
- of reactive attachment disorder, 155–156
- of restless legs syndrome, 213
- of schizophrenia, 87
- of selective mutism, 125
- of separation anxiety disorder, 115–116, 118
- of social anxiety disorder, 128
- of specific learning disorder, 71–72
- of specific phobias, 128
- of substance use disorder, 243
- of tic disorders, 76
- Clinically significant effects, of psychopharmacology, 305–306
- Clomipramine, and obsessive-compulsive disorder, 149, 328, 332–333
- Clonidine
 - attention-deficit/hyperactivity disorder and, 69
 - autism spectrum disorder and, 50
 - indications for and efficacy of, 324–326
 - initiation and ongoing treatment with, 326–327
 - posttraumatic stress disorder and, 164
 - risks and side effects of, 327–328
 - tic disorders and, 82
- Clozapine, 90, 343, 354
- Cognition. *See also* Cognitive-behavioral therapy; Cognitive remediation; Cognitive restructuring techniques; Cognitive therapy
 - side effects of stimulants and, **315**
 - symptoms of PTSD and, 158, 159
 - toxicity of psychoactive drugs, 306
- Cognitive-Based CBT, 163
- Cognitive-Behavioral Intervention for Trauma in Schools (CBITS), 163
- Cognitive-behavioral therapy (CBT)
 - for anorexia nervosa, 185
 - for attention-deficit/hyperactivity disorder, 70
 - for bulimia nervosa, 189
 - for generalized anxiety disorder, 138–139
 - group therapy and, 387
 - for insomnia, 206
 - for major depressive disorder and persistent depressive disorder, 109, 110

Cognitive-behavioral therapy (CBT)

(continued)

for medically ill children, 296

for obsessive-compulsive disorder, 148–149

for out-of-control behavior, 274

for panic disorder, 135

range of techniques in, 378

for schizophrenia, 90

for separation anxiety disorder, 122–123, 123–124

for sexual abuse victims, 270

for social anxiety disorder and specific phobias, 131–132

for suicidal behavior, 262

for tic disorders, 81

Cognitive remediation, and schizophrenia, 90

Cognitive restructuring techniques, for social anxiety disorder and specific phobias, 131–132

Cognitive therapy, for adjustment disorder, 166

Collaborative Life Skills, 68

Collaborative Lithium Trials (CoLT), 338, 340

Columbia Suicide Severity Rating Scale (C-SSRS), 262

Combined presentation, of attention-deficit/hyperactivity disorder, 51,

54

Combined treatment

for generalized anxiety disorder, 139

for major depressive disorder and persistent depressive disorder, 109–110

for obsessive-compulsive disorder, 149

selective serotonin reuptake inhibitors for anxiety disorders and, 333

for separation anxiety disorder, 123–124

Communication, with children and adolescents during psychosocial interventions, 374

Communication disorders

clinical description of, 36

course and prognosis of, 38

differential diagnosis of, 39, 74

epidemiology of, 37

etiology of, 37

evaluation of, 38

specific learning disorder and, 74

treatment of, 39

types of, 39–41

Community services, and

psychosocial interventions, 389–390. *See also* Social services

Comorbidity

anxiety disorders and, 95, 233, 322

attention-deficit/hyperactivity disorder and, 26, 28, 56, 75, 76, 77, 80, 82, 195, 199, 226, 232, 238, 246

- autism spectrum disorder and, 28, 64, 314
- bipolar disorder and, 95, 96
- conduct disorder and, 232–233, 246, 314
- depression and, 28–29, 322
- disruptive mood dysregulation disorder and, 102
- intellectual disability and, 26, 28–29, 56, 64, 314
- obsessive-compulsive disorder and, 76, 80, 89
- oppositional defiant disorder and, 246, 314
- overview of issues in diagnosis and, 4–5
- posttraumatic stress disorder and, 162, 232
- selective mutism and, 127
- substance use disorder and, 246
- tic disorders and, 76–77
- Complete blood count (CBC), and lithium, 339
- Complex tics, 76
- Compulsions, and obsessive-compulsive disorder, 146
- Conduct disorder (CD)
 - antipsychotics and, **348**
 - attention-deficit/hyperactivity disorder and, 56, 58, 60, 63, 314
 - behavior therapy for, 381
 - bipolar disorder and, 95, 96
 - clinical description of, 230–231
 - comorbidity in, 232–233, 246, 314
 - course and prognosis of, 234–235
 - differential diagnosis of, 236
 - disruptive mood dysregulation disorder and, 103
 - epidemiology of, 231
 - etiology of, 233–234
 - evaluation of, 235–236
 - genetics of, 58
 - oppositional defiant disorder and, 225, 228
 - psychological characteristics of children and adolescents with, **232**
 - stimulant medications and, 314
 - substance use disorder and, 246
 - treatment of, 236–239
- Confidentiality, and group therapy for adolescents, 389. *See also* Autonomy; Privacy
- Consent, for pharmacotherapy in children, 309. *See also* Informed consent
- Constipation, and encopresis, 198, 199, 200
- Continuous Performance Test (CPT), 64
- Continuous positive airway pressure (CPAP), 209
- Contraceptives, and adolescents, 284
- Conversion disorder, **183**
- Coping skills, for children of psychiatrically ill parents, 299
- Coprolalia, 78

- Core deficits, in attention-deficit/hyperactivity disorder, 54
- Co-therapists, for group therapy, 388–389
- Course and prognosis
 - of acute stress disorder, 160–161
 - of adjustment disorder, 165–166
 - of anorexia nervosa, 178–179
 - of attention-deficit/hyperactivity disorder, 59–60
 - of autism spectrum disorder, 46–47
 - of bipolar disorder, 95
 - of bulimia nervosa, 187–188
 - of communication disorders, 38
 - of conduct disorder, 234–235
 - of disinhibited social engagement disorder, 156–157
 - of disruptive mood dysregulation disorder, 103
 - of enuresis, 195–196
 - of generalized anxiety disorder, 137–138
 - of intellectual disability, 32–33
 - of major depressive disorder and persistent depressive disorder, 106–107
 - of narcolepsy, 208
 - of obesity, 287
 - of obsessive-compulsive disorder, 147–148
 - of obstructive sleep apnea, 209
 - of oppositional defiant disorder, 228
 - of panic disorder, 134
 - of pica, 172
 - of posttraumatic stress disorder, 160–161
 - of reactive attachment disorder, 156–157
 - of schizophrenia, 88
 - of selective mutism, 127
 - of separation anxiety disorder, 120–121
 - of social anxiety disorder and specific phobias, 129–130
 - of specific learning disorder, 73
 - of substance use disorder, 247–249
 - of suicidal behavior, 261
 - of tic disorders, 78–79
- Crime, and conduct disorder, 234, 235
- Culture, and sociocultural issues
 - anorexia nervosa and, 178
 - attention-deficit/hyperactivity disorder and, 56
 - children of immigrant families and, 283–284
 - conduct disorder and, 231
 - standardized tests and, 20
- Custody options, and divorce, 276–277
- Cyclothymic disorder, 93

- Daily Report Card, 67
- “Damocles syndrome,” and terminal illness, 294
- Day treatment programs, 393
- DDAVP (desmopressin), 197, 334
- Deafness, and autism spectrum disorder, 48. *See also* Hearing loss
- Death. *See also* Mortality
 methylphenidate/clonidine combination and reports of sudden, 328
 reaction to family member’s, 280–282
 of terminally ill child or adolescent, 293–294
- Decision making, and chronic illness in children, 294
- Degenerative neurological diseases, 49
- Dehydration, and anorexia nervosa, 182
- Delayed sleep-phase syndrome (DSPS), 210, 361
- Delirium, **272**, 295
- Delusions, and schizophrenia, 89
- Denmark, and study of children of psychiatrically ill parents, 297
- Denver Developmental Screening Test (DDST), 33
- Department of Health and Human Services, National Child Abuse and Neglect Data System (NCANDS), 264
- Depression. *See also* Bipolar depression; Depressive disorders; Major depressive disorder; Persistent depressive disorder; Psychotic depression
 anorexia nervosa and, 177, **183**
 antidepressants for, 331–332
 attention-deficit/hyperactivity disorder and, 322
 comorbidity of, 28–29, 322
 conduct disorder and, 238–239
 deficits in attention and, 63
 disruptive mood dysregulation disorder and, 101–104
 information resources for parents on, 404
 intellectual disability and, 28–29
 posttraumatic stress disorder and, 161
 psychiatric illness in parents and, 297–298
 selective serotonin reuptake inhibitors and, 322
 Tourette’s disorder and, 77
 treatment of in medically ill children, 295
- Dermatological complications, of anorexia nervosa and bulimia nervosa, **180**.
See also Rash
- Desmopressin (DDAVP), 197, 334
- Desvenlafaxine, 335, 338

- Development. *See also* Age;
 Developmental disorders
 assessment of emergency and
 history of, **258**
 of children of adolescent
 parents, 285
 common fears and, **116**
 DSM-5 criteria for depressive
 disorders and, 104, **105**
 evaluation of acute stress
 disorder or posttraumatic
 stress disorder and, 158,
 161–162
 evaluation and level of, 7, 9
 evaluation of sleep-related
 problems and, 204
 family tasks in, **13**
 group therapy and, 388
 information resources for
 parents on, 402
 outline of biopsychosocial
 history and, **8–9**
 parental divorce and, 276
 psychopharmacology and
 toxicity for, 306
 reaction to acute illness,
 hospitalization, or surgery
 and, 289–291
 separation anxiety as normal
 phenomenon in, 121
 substance use disorder and,
 248
- Developmental coordination
 disorder, 75
- Developmental disorders, and
 antipsychotics, 350–351
- Developmental expressive and
 receptive language disorders,
 48
- Dexmethylphenidate, **312**,
 315–316
- Dextroamphetamine, **312**
- Diabetes, psychiatric sequelae of,
 292
- Diagnosis. *See also* Clinical
 description; Comorbidity;
 Differential diagnosis;
 DSM-5; Evaluation;
 Misdiagnosis;
 Underdiagnosis
 comorbidity as issue in, 4–5
 of disruptive mood
 dysregulation disorder,
 101–102
 use of DSM-5 for, 3–4
 use of “unspecified” in
 emergency settings, 258
- Diagnostic Adaptive Behavior
 Scale (DABS), 33
- Diagnostic Interview for Children
 and Adolescents (DICA), **15**
- Diagnostic Interview Schedule for
 Children (DISC), **15**
- Dialectical behavior therapy (DBT)
 in group settings, 387
 for suicidal behavior, 262, 264,
 379–380
- Diazepam, 295
- Diet, and dieting. *See also* Food;
 Nutritional history
 anorexia nervosa and, 178, 179
 bulimia nervosa and, 187

- treatment of attention-deficit/hyperactivity disorder and, 70–71
- Dietary supplements, and lithium, 342. *See also* Melatonin; Omega-3 fatty acids
- Differential Ability Scales, Second Edition (DAS-II), **16**
- Differential diagnosis
 - of acute stress disorder, 162, 166
 - of adjustment disorder, 162, 166
 - of anorexia nervosa, **183**
 - of anxiety disorders, 138
 - of attention-deficit/hyperactivity disorder, 62–64, 74, 95, 157, 229
 - of autism spectrum disorder, 48–49, 122, 148, 157
 - of bipolar disorder, 95–96, 236
 - of communication disorders, 39, 74
 - of conduct disorder, 236
 - of disinhibited social engagement disorder, 157
 - of disruptive mood dysregulation disorder, 104
 - of excoriation disorder, 150
 - of generalized anxiety disorder, 122, 138
 - of intellectual disability, 34, 74
 - of major depressive disorder and persistent depressive disorder, 108
 - of obesity, 287
 - of obsessive-compulsive disorder, 148, **183**
 - of oppositional defiant disorder, 228–229
 - of panic disorder, 122, 135
 - of persistent depressive disorder, 108
 - of posttraumatic stress disorder, 162, 166
 - of reactive attachment disorder, 49, 108, 157
 - of schizophrenia, 89
 - of selective mutism, 127
 - of separation anxiety disorder, 121–122
 - of social anxiety disorder and specific phobias, 122, 130
 - of specific learning disorder, 74
 - of speech sound disorder, 41
 - of substance use disorder, 249–250
 - of tic disorders, 80
- Difficult (temperament cluster), 10, **11**
- Diphenhydramine, 354
- Disability, family system and childhood, 279–280
- Discontinuation. *See also* Drug-free trials; Tapering; Withdrawal
 - of antidepressants, 336
 - of guanfacine or clonidine, 327
 - of lithium, 341

- Disinhibited social engagement disorder (DSED)
 clinical description and, 155–156
 course and prognosis of, 156–157
 differential diagnosis of, 157
 epidemiology of, 156
 etiology of, 156
 evaluation of, 157
 treatment of, 157–158
- Disruptive behavior disorder (DBD), 96, 357
- Disruptive, impulse-control, and conduct disorders. *See* Conduct disorder; Oppositional defiant disorder
- Disruptive mood dysregulation disorder (DMDD)
 attention-deficit/hyperactivity disorder and, 63
 bipolar disorder and, 93
 clinical description of, 101–102
 course and prognosis of, 103
 differential diagnosis of, 104, 63
 epidemiology of, 102–103
 evaluation of, 103–104
 treatment of, 104
- Dissociative disorders, and conduct disorder, 232
- Divalproex sodium, 256–257
- Divorce, and family transitions, 275–279
- DMDD. *See* Disruptive mood dysregulation disorder
- Docosahexaenoic acid (DHA), 362
- Domestic violence, 161, 275
- Dosages, and pediatric psychopharmacology
 of antidepressants, **329–330**, 335
 of antipsychotics, 353
 of carbamazepine, 358–359
 general principles for, 307
 of guanfacine or clonidine, 326–327
 of lithium, 339–340
 of melatonin, 361
 of stimulant medications for ADHD, 317
 of valproate, 358
- Down syndrome, 29–30
- Dreams, and PTSD, 158
- Drug and Alcohol Problem (DAP) Quick Screen, 249
- Drug-drug interactions, and antidepressants, 335
- Drug-free trials, and stimulant medications for ADHD, 319
- Drug Use Screening Inventory (DUSI-R), 249
- DSED. *See* Disinhibited social engagement disorder
- DSM-5
 adjustment disorders in, 164
 attention-deficit/hyperactivity disorder in, **51–54**

- autism spectrum disorder in,
 - 41, **42–44**, 47
- bipolar and related disorders in,
 - 93, 94, 96
- conduct disorder in, 230, 231
- developmental differences in
 - criteria for depressive disorders in, 104, **105**
- disruptive mood dysregulation disorder in, 101
- eating and feeding disorders in,
 - 171, 175
- gender dysphoria in, **218–220**
- generalized anxiety disorder in,
 - 135, **136–137**
- narcolepsy in, 207
- neurodevelopmental disorders in, 25
- obsessive-compulsive and related disorders in, 145
- oppositional defiant disorder in,
 - 225, 226, **227**
- overview of use for diagnosis in children and adolescents,
 - 3–4
- panic disorder and agoraphobia in, 133
- posttraumatic stress disorder in,
 - 158
- separation anxiety disorder in,
 - 115, **117–118**
- specific learning disorder in, **72**
- specific phobias and social anxiety disorder in, 128
- substance use disorder in,
 - 243–244**
- subtypes of sleep-wake disorders in, 210
- trauma- and stressor-related disorders in, 155
- V codes in, 3, **4**
- Duloxetine
 - depression and, 332
 - generalized anxiety disorder and, 139, 333–334
 - mechanism of action, 331
 - side effects of, 335, 338
- Dyslexia, and dyscalculia, 72
- Dysthymia. *See* Persistent depressive disorder
- Dystonia, and antipsychotics, 354
- Early-onset schizophrenia (EOS), 87
- Easy (temperament cluster), 10, **11**
- Eating disorders. *See* Feeding and eating disorders
- Echokinesis, echopraxia, and echolalia, 78
- E-cigarettes, 245–246
- Education. *See also* Psycho-education; Schools; Special education; Teachers
 - attention-deficit/hyperactivity disorder and parental, 66–67
 - child maltreatment and, 267
 - divorce and, 278
 - hospitalization and, 392
 - narcolepsy and, 208
 - pica and, 173
 - sleep terrors and parental, 212
 - tic disorders and, 81

- Eicosapentaenoic acid (EPA), 362
- Electrocardiograms (ECGs), and
pharmacotherapy, 318, 326,
352
- Electroconvulsive therapy, 110
- Elimination disorders, in DSM-5,
193. *See also* Encopresis;
Enuresis
- Emergencies, in psychiatric and
pediatric settings and in
schools
adjustment disorder and, 165
assessment of, 255–256,
258–259
child maltreatment as, 264–271
common forms of, **256**
options for dispositions in,
259
out-of-control behavior and,
271–274
outline of patient history in,
257–258
suicidal behavior and, **257**,
260–264
- Emotional abuse, of children,
265
- Emotions, and assessment of
emergency, **257**. *See also*
Anger; Irritability
- Encopresis
clinical description of, 197–198
course and prognosis of, 199
epidemiology of, 198
etiology of, 198–199
evaluation of, 199–200
treatment of, 200
- Enuresis
antidepressants and, 334
clinical description of, 193–194
course and prognosis of,
195–196
encopresis and, 199
epidemiology of, 194
etiology of, 194–195
evaluation of, 196
treatment of, 196–197
- Environmental factors
communication disorders and,
37
conduct disorder and, 233
oppositional defiant disorder
and, 227–228
in pediatric
psychopharmacology, 308
specific learning disorder and,
74
- Environmental interventions, for
adjustment disorder, 166
- Epidemiology
of acute stress disorder,
159–160
of adjustment disorders,
164–165
of anorexia nervosa, 177–178
of attention-deficit/
hyperactivity disorder,
55–56
of autism spectrum disorder, 45
of bipolar disorder, 94
of bulimia nervosa, 186–187
of child maltreatment, 264–265
of communication disorders, 37

- of conduct disorder, 231
- of disinhibited social engagement disorder, 156
- of disruptive mood dysregulation disorder, 102–103
- of encopresis, 198
- of enuresis, 194
- of gender dysphoria, 220
- of generalized anxiety disorder, 137
- of intellectual disability, 26
- of major depressive disorder and persistent depressive disorder, 105–106
- of obesity, 286
- of obsessive-compulsive disorder, 145–146
- of oppositional defiant disorder, 226
- of panic disorder, 133–134
- of pica, 171
- of posttraumatic stress disorder, 159–160
- of reactive attachment disorder, 156
- of schizophrenia, 87
- of selective mutism, 125–126
- of separation anxiety disorder, 118
- of social anxiety disorder and specific phobias, 129
- of substance use disorder, 244–246
- of suicidal behavior, 260
- of tic disorders, 76
- EPS, and side effects of antipsychotics, 354–355
- Escitalopram, 109, 331–332
- Esophageal complications, of anorexia nervosa and bulimia nervosa, **180**
- Ethical issues, in pediatric psychopharmacology, 309
- Etiology
 - of acute stress disorder, 160
 - of adjustment disorder, 165
 - of anorexia nervosa, 178
 - of attention-deficit/hyperactivity disorder, 56–58
 - of autism spectrum disorder, 46
 - of bipolar disorder, 94–95
 - of bulimia nervosa, 187
 - of child maltreatment, 265, 266
 - of communication disorders, 37
 - of conduct disorder, 233–234
 - of disinhibited social engagement disorder, 156
 - of encopresis, 198–199
 - of enuresis, 194–195
 - of gender dysphoria, 220–221
 - of generalized anxiety disorder, 137
 - of insomnia, 205–206
 - of intellectual disability, 29–30, 32
 - of major depressive disorder and persistent depressive disorder, 106
 - of obesity, 286
 - of obsessive-compulsive disorder, 146–147

Etiology (*continued*)

- of obstructive sleep apnea, 209
- of oppositional defiant disorder, 227–228
- of panic disorder, 134
- of pica, 172
- of posttraumatic stress disorder, 160
- of reactive attachment disorder, 156
- of restless legs syndrome, 213
- of schizophrenia, 88
- of selective mutism, 126
- of separation anxiety disorder, 118–120
- of social anxiety disorder and specific phobias, 129
- of specific learning disorder, 72–73
- of substance use disorder, 247
- of tic disorders, 77–78

Evaluation. *See also* Assessment;

- Biopsychosocial evaluation;
- Diagnosis
- of acute stress disorder, 161–162
- of adjustment disorder, 166
- of anorexia nervosa, 179, 182
- of attention-deficit/hyperactivity disorder, 60–62
- of autism spectrum disorder, 47–48
- of bulimia nervosa, 188–189
- of child abuse, 266
- of communication disorders, 38

- comprehensive process of for children and adolescents, 7–20
- of conduct disorder, 235–236
- of disinhibited social engagement disorder, 157
- of disruptive mood dysregulation disorder, 103–104
- of encopresis, 199–200
- of enuresis, 196
- of gender dysphoria, 221–222
- of generalized anxiety disorder, 138
- of intellectual disability, 33–34
- of major depressive disorder and persistent depressive disorder, 107–108
- of narcolepsy, 207–208
- of obesity, 287
- of obsessive-compulsive disorder, 148
- of oppositional defiant disorder, 228
- of panic disorder, 134–135
- of pica, 172
- of posttraumatic stress disorder, 161–162
- of reactive attachment disorder, 157
- of schizophrenia, 88–89
- of selective mutism, 127
- of separation anxiety disorder, 121–122
- of sleep-related complaints, 203–205

- of social anxiety disorder and specific phobias, 130
- of specific learning disorder, 73–74
- of substance use disorder, 249
- of suicidal behavior, 261–262
- of tic disorders, 79–80
- Evidence-based clinical practice, of pediatric psychopharmacology, 305
- Exercise
 - antipsychotics and, 352
 - bulimia nervosa and, 186
 - lithium toxicity and, 342
- Excoriation (skin-picking) disorder, 151
- Exposure and response prevention (ERP) techniques, 131, 135
- False allegations, of sexual abuse, 269
- Family. *See also* Family therapy; Genetics; Marriage; Parent(s)
 - anorexia nervosa and dynamics of, 178, 182
 - assessment of emergency and, **258**
 - comprehensive evaluation and, 12–14
 - feedback conferences and treatment planning for, 22
 - impact of transitions in on children, 274–283
 - involvement of in psychosocial interventions for children, 373, 383–384
 - risk factors for suicide attempts and, **263**
 - sexual abuse and, 268–269
 - speech sound disorder and history of, 41
 - treatment of major depression and, 109
 - treatment of suicidal behavior and, 262
- Family approaches for chronic physical illness, 386
- Family-based assessment and treatment, 383–386
- Family-based cognitive-behavioral therapy, for obsessive-compulsive disorder, 149
- Family-based treatment (FBT), for anorexia nervosa, 184–185
- Family grief therapy, 385
- Family systems theory, and conduct disorder, 238
- Family therapy. *See also* Functional family therapy
 - for attention-deficit/hyperactivity disorder, | 70
 - hospitalization and, 392
 - indications for, 384–385
 - for intellectual disability, 35
 - for selective mutism, 128
 - for substance use disorder, 251
 - for tic disorders, 81
 - types of, 385–386
- Fast Track program, 229

Fears

- development stages and normal, **116**
- school absenteeism and, **119**
- separation anxiety disorder and, **117**
- social anxiety disorder or specific phobias and, 128
- subclinical panic attacks and, 134

Feedback, and treatment planning, 22

Feeding and eating disorders.

- See also* Anorexia nervosa; Avoidant/restrictive food intake disorder; Bulimia nervosa; Pica; Rumination disorder
- in DSM-5, 171, 175
- information resources for parents on, 404

Feingold diet, 70

Fever, and hallucinations, 89

Firearms, and suicide risk, 260, 261, 262

Fish oil, and omega-3 fatty acids, 362

Fluid disturbance, and medical complications of anorexia nervosa and bulimia nervosa, **180**

Fluoxetine

- anxiety disorders and, 333
- bulimia nervosa and, 189–190
- initiation of, 335
- major depression and, 109, 331, 332

obsessive-compulsive disorder and, 149, 332

panic disorder and, 135

social anxiety disorder and specific phobias, 132

withdrawal symptoms and, 337

Fluvoxamine

obsessive-compulsive disorder and, 149, 332

pediatric depression and, 332

social anxiety disorder or specific phobias and, 132, 333

withdrawal symptoms and, 337

Food. *See also* Diet; Dietary

supplements; Feeding and eating disorders; Nutritional history

anorexia nervosa and

preoccupation with, 177

attention-deficit/hyperactivity disorder and elimination diets, 70–71

feeding practices in infancy and subsequent obesity, 286

Food and Drug Administration

(FDA), 306, 318, 322, 323, 334, 336, 338, 356, 359. *See also* Black box warnings

Foster care

adjunctive treatments and, 395–396

for children of psychiatrically ill parents, 299

interventions for child maltreatment and, 267

- Fragile X syndrome (FXS), 30, 32
- Functional family therapy (FFT), 237–238, 386
- Funerals, and death of family member, 281
- Gabapentin, and restless legs syndrome, 214
- GAD. *See* Generalized anxiety disorder
- Gastroesophageal reflux, and rumination disorder, 173, 174
- Gastrointestinal complications of anorexia nervosa and bulimia nervosa, **180, 181**
- side effects of lithium and, 342
- Gay, lesbian, bisexual, and transgender youth, and suicidal behavior, 260
- Gender. *See also* Gender dysphoria; Gender identity
- anorexia nervosa and, 177, 178
 - attention-deficit/hyperactivity disorder and, 55–56
 - autism spectrum disorder and, 45
 - bulimia nervosa and, 186
 - conduct disorder and, 231
 - enuresis and, 194
 - generalized anxiety disorder and, 137
 - group therapy and, 388
 - posttraumatic stress disorder and, 159
 - sexual abuse and, 268
 - suicide attempts and, 260
- Gender discordance, 217
- Gender dysphoria
- clinical description of, 218
 - course and prognosis of, 221
 - DSM-5 diagnostic criteria for, **218–220**
 - epidemiology of, 220
 - etiology of, 220–221
 - evaluation of, 221–222
 - key concepts and terminology in, 217
 - treatment of, 222
- Gender expression (gender role behavior), 217
- Gender identity, use of term, 217
- Gender identity disorder. *See* Gender dysphoria
- Gender nonconformity (gender variance), 217
- Generalized anxiety disorder (GAD)
- clinical description of, 135, 137
 - course and prognosis of, 137–138
 - differential diagnosis of, 122, 138
 - epidemiology of, 137
 - etiology of, 137
 - evaluation of, 138
 - selective serotonin reuptake inhibitors for, 333
 - separation anxiety disorder and, 122
 - treatment of, 138–139

Genetics, and etiology. *See also*

Genetic testing

of anorexia nervosa, 178

of attention-deficit/

hyperactivity disorder, 56,
57, 58

of autism spectrum disorder,
46

of bipolar disorder, 94–95

of expressive language
disorders, 37

of generalized anxiety disorder,
137

of intellectual disability, 29,
31

of obsessive-compulsive
disorder, 146

of panic disorder, 134

of restless legs syndrome, 213

of schizophrenia, 88

of selective mutism, 126

of specific learning disorder,
72

of substance use disorder, 247

of tic disorders, 77

Genetic testing

carbamazepine and, 358

intellectual disability and, 34

Genogram, 13

Global mutism, 126

Goodness of fit, between child and
parent, 10, 282

Grandiosity, and mania, 95

Group A beta-hemolytic
streptococcus (GABHS)
infection, 146–147

Group homes, 396

Group therapy

for attention-deficit/

hyperactivity disorder, 70

for conduct disorder, 238

hospitalization and, 392

indications for, 386–387

intellectual disability and,
35

play therapy for child

maltreatment and, 267

technical considerations for,
387–389

Growth record, and avoidant/
restrictive food intake
disorder, 176

Growth retardation, stimulant-
induced, 321

Guanfacine

attention-deficit/hyperactivity
disorder and, 69

indications for and efficacy of,
324–326

initiation of and ongoing
treatment with, 326–327

risks and side effects of,
327–328

tic disorders and, 82

valproate and, 358

Guardian ad litem attorney, 395

Guidelines. *See also*

Recommendations

principles of psychopharma-
cology for children and
adolescents as, 305–306

for treatment planning, 21

- Habit reversal therapy (HRT)
 rumination disorder and, 174
 Tourette's disorder and, 351
 trichotillomania and, 150
- Hallucinations, and schizophrenia, 89
- Haloperidol, 82
- Headache, treatment of, 296
- Head trauma, and conduct disorder, 236. *See also*
 Brain injury
- Hearing loss, and communication disorders, 37. *See also*
 Deafness
- Hematological complications,
 of anorexia nervosa and bulimia nervosa, **180**
- Hirschsprung's disease, 199
- HIV, and substance use disorder, 249
- Hormone therapy, and gender dysphoria, 222
- Hospitalization. *See also*
 Emergencies; Partial hospitalization
 anorexia nervosa and, 179, 182, 184
 autism spectrum disorder and, 49
 avoidant/restrictive food intake disorder and, 176
 bulimia nervosa and, 189
 components of treatment in, 391–392
 indications for, 390–391
 for major depressive disorder or persistent depressive disorder and, 108
 of psychiatrically ill parents, 297, 299
 rumination disorder and, 174
 substance use disorder and, 251
- Hotlines, and child abuse, 267
- Hunger scale, 288
- Hydroxyzine, 295
- Hyperactivity, and attention-deficit/hyperactivity disorder, **53–54**
- Hypersensitivity, and autism spectrum disorder, 45
- Hypnosis, for medically ill children, 296
- Hypoglycemia, and anorexia nervosa, 182
- Hypokalemia, and lithium, 342
- Hypothyroidism, and anorexia nervosa, **183**
- ID. *See* Intellectual disability
- Idiopathic, intellectual disability as, 29
- Immediate-release preparations, of stimulants, 317
- Immigrants
 cultural factors for families and children, 283–284
 selective mutism and, 126
- Impulsivity, and attention-deficit/hyperactivity disorder, **53–54**

- Inattention, and attention-deficit/hyperactivity disorder, **51–52**
- Incest, and sexual abuse, 268, 269
- Individualized Education Plan (IEP), 67, 123
- Individual psychotherapy, 185, 391
- Individuals with Disabilities Education Act (IDEA), 74–75
- Infancy. *See also* Pregnancy
causes of attention-deficit/hyperactivity disorder and, **57**
causes of intellectual disability and, **31**
- Infant or toddler-parent interactive psychotherapy, 386
- Information sheets, on
medications, 309, 352
- Informed consent, for
pharmacotherapy, 309, 352
- Inhalant abuse, 248, 249
- In-home family preservation services, 386
- Insomnia
clinical description of, 205
etiology of, 205–206
melatonin for, 361
treatment of, 206–207
- Institutionalization, and
attachment disorders, 156, 157
- Intellectual developmental disorder, 25
- Intellectual disability (ID)
antipsychotics for, 351
attention-deficit/hyperactivity disorder and, 56, 64, 314
autism spectrum disorder and, **44, 49**
clinical description of, 25–26, **27**
comorbidity in, 26, 28–29, 56, 64, 314
course and prognosis of, 32–33
differential diagnosis of, 34, 74
epidemiology of, 26
etiology of, 29–30, 32
evaluation of, 33–34
lithium and, 339
pica and, 171, 172
sleep problems and, 203
specific learning disorder and, 74
speech sound disorder and, 41
standardized tests of adaptive behavior and, **19**
stimulant medications and, 314
treatment of, 34–36
- Intelligence (IQ)
autism spectrum disorder and, 45
conduct disorder and, 234
intellectual disability and, 25–26
standardized tests of, **16–18, 20**
- Intensive outpatient programs (IOPs), 124, 393
- Intention, and suicidal behavior, 261
- Intermittent explosive disorder, and conduct disorder, 236

- Internet, and information for parents, 399–401
- Interpersonal psychotherapy (IPT), 109, 379
- Interpersonal psychotherapy for depressed adolescents (IPT-A), 379, 387
- Intrusion symptoms, and posttraumatic stress disorder, 158
- Iowa Conners Teacher Rating Scale, 64
- Iron deficiency, and restless legs syndrome, 213, 214
- Irritability
 - antipsychotics for, **344, 347**
 - oppositional defiant disorder and, 226
- Joint custody, and divorce, 276–277
- Journal of the American Academy of Child and Adolescent Psychiatry*, 1
- Judicial system, and divorce, 279.
 - See also* Custody options; Legal issues
- Juvenile justice interventions, for conduct disorder, 239.
 - See also* Crime
- Kaufman Adolescent and Adult Intelligence Test (KAIT), **16**
- Kaufman Assessment Battery for Children, Second Edition (K-ABC-II), **16**
- Kaufman Brief Intelligence Test, Second Edition (KBIT-2), **16**
- Kaufman Test of Educational Achievement, Third Edition (KTEA-3), **18**
- Labeling, and adjustment disorder, 166
- Laboratory tests
 - antipsychotics and, 352
 - comprehensive evaluation and, 14–15
 - intellectual disability and, 34
 - lithium and, 339
 - psychiatric emergencies and, **259**
 - substance use disorder and, 250
- Lamotrigine, 357–358, 359, 361
- Landau-Kleffner syndrome, 49
- Language. *See also* Speech
 - autism spectrum disorder and, 44–45
 - cognitive immaturity and children's ability to use, 374
 - communication disorders and, 36
- Language disorder, 39. *See also* Developmental expressive and receptive language disorders
- Laryngeal dystonia, and antipsychotics, 354
- Lead exposure
 - attention-deficit/hyperactivity disorder and, **57**

- Lead exposure (*continued*)
autism spectrum disorder and, 48
pica and, 172
- Learning disorders, 64, 232, 236.
See also Specific learning disorder
- Legal issues, and foster care, 395–396. *See also* Crime; Judicial system; Juvenile justice interventions
- Leiter International Performance Scale—Third Edition, **16**
- Lethality, of suicide attempts, 261
- Lifelong pattern, of oppositional defiant disorder, 229
- Limit setting, and conduct disorder, 237
- Lithium
age and dose of, 307
bipolar disorder and, 96
conduct disorder and, 238–239
indications for and efficacy of, 338–339
initiation of and ongoing treatment with, 339–341
risks and side effects of, 341–342
- Liver disease, and obesity, 287
- Liver injury, as side effect of atomoxetine, 324
- Longitudinal Assessment of Manic Symptoms (LAMS), 103
- Magical thinking, 279, 281
- Major depressive disorder (MDD).
See also Depression
in children of parents with mood disorders, 298–299
clinical description of, 104–105
course and prognosis of, 106–107
differential diagnosis of, 108
epidemiology of, 105–106
etiology of, 106
evaluation of, 107–108
fluoxetine and, 331
treatment of, 108–110
- Major depressive episode, 93
- Mandated reporting, of child maltreatment, 264
- Mania
age at onset of, 94
attention-deficit/hyperactivity disorder and, 63
divalproex sodium for, 356
DSM-5 criteria for, 94
lithium for, 338–339
out-of-control behavior and, **272**
schizophrenia and, 89
- Marijuana, 248–249
- Marriage
impact of conflict in on children, 275
marital problems in parents of children with oppositional defiant disorder, 227
remarriage by custodial mother and, 278

- Maturation etiology, of enuresis, 194
- Maudsley family therapy, 386
- Medical conditions. *See also*
 Laboratory tests; Medical history; Physical examination; Somatic symptoms
- anorexia nervosa and, 179, **180–181, 183**
- attention-deficit/hyperactivity disorder and, **57, 58, 63**
- avoidant/restrictive food intake disorder and, 176
- bulimia nervosa and, **180–181, 188**
- changes in DSM-5 and “other,” 3
- in children and adolescents, 289–297
- enuresis and, **195**
- intellectual disability and, 32
- obesity and, 287
- in parents or siblings, 279–280
- pica and, 172
- rumination disorder and, 174
- schizophrenia and, 89
- suicide risk and, 261
- terminal phase of parental illness and, 280–281
- Medical history, and initiation of antipsychotics, 352
- Medications. *See* Over-the-counter medications; Pharmacotherapy; Prescription drug abuse
- Melatonin
- attention-deficit/hyperactivity disorder and, 70
- circadian rhythm sleep-wake disorders and, 210
- indications for and efficacy of, 361–362
- insomnia and, 207
- intellectual disability and, 36
- Mental retardation. *See* Intellectual disability
- Mental status examination, **12, 258, 263, 273**
- Metabolic syndrome, and antipsychotics, 354
- Metformin, and obesity, 289
- Methylphenidate
- amphetamine compared with, 315
- attention-deficit/hyperactivity disorder and, 69, 82, 310, 325
- clonidine and, 325
- formulations and dosages of, **311–312**
- tic disorders and, 82
- Milieu treatment, and psychosocial interventions, 390–393
- Mirtazapine, 331, 334, 335
- Misdiagnosis, of bipolar disorder, 95, 96
- Modafinil, and narcolepsy, 208
- Modified Checklist for Autism in Toddlers—Revised (M-CHAT-R), 47

- Monitoring, of stimulant medications for attention-deficit/hyperactivity disorder, 318
- Monitoring the Future (MTF), 244, 245
- Monosymptomatic enuresis (MSE), 193, 196
- Mood disorders
 attention-deficit/hyperactivity disorder and family history of, 58
 bulimia nervosa and, 187
 conduct disorder and, 233
 lithium for, 338–339
 major depression in adolescent children of parents with, 298–299
 schizophrenia and, 89
 sleep complaints and, 214
 specific learning disorder and, 74
- Mood stabilizers. *See* Anticonvulsants; Lithium
- Mortality. *See also* Death
 anorexia nervosa and, 179
 bulimia nervosa and, 188
 child maltreatment and, 265
 substance use disorder and, 248
- Motivational interviewing, 288, 379
- Motor activity, and attention-deficit/hyperactivity disorder, 54–55
- Motor effects, of stimulant medications, **315**
- Movement disorders, and antipsychotics, 353
- Multidimensional Anxiety Scale for Children (MASC), 121, 138
- Multimodal treatment, of separation anxiety disorder, 122–123
- Multimodal Treatment of ADHD (MTA) study, 310, 314
- Multiple informants, use of in comprehensive evaluation, 9–10
- Multiple Sleep Latency Test (MSLT), 204
- Multisystemic therapy (MST), 238, 386
- N*-Acetyl cysteine, and trichotillomania or excoriation disorder, 150, 151
- Narcolepsy, 207–208
- Narcolepsy Network, 208
- Narcotics Anonymous (NA), 250, 251
- Narrative Exposure Therapy for Children (Kid-NET), 163
- Natal sex, 217
- National Alliance on Mental Illness (NAMI), 401
- National Child Abuse and Neglect Data System (NCANDS), 264
- National Child Traumatic Stress Network, 162, 401
- National Federation of Families for Children's Mental Health, 401
- National Health and Nutrition Examination Survey (NHANES), 286

- National Institute on Drug Abuse (NIDA), 244
- National Institute of Mental Health (NIMH), 90–91, 93, 109, 310, 314, 401
- National Survey on Drug Use and Health (NSDUH), 245
- National Youth Risk Behavior Survey, 260
- Negative reinforcement
 - pica and, 173
 - rumination disorder and, 174
- Negative symptoms, of schizophrenia, 87
- Neglect
 - definition and categories of, 265–266
 - cognitive and developmental delays and, 160
 - etiology of reactive attachment disorder or disinhibited social engagement disorder and, 156, 157
 - out-of-control behavior and, **272**
- Neurodevelopmental disorders, in DSM-5, 25. *See also* Attention-deficit/hyperactivity disorder; Autism spectrum disorder; Communication disorders; Developmental coordination disorder; Intellectual disability; Specific learning disorder; Tic disorders
- Neuroendocrine complications, of
 - anorexia nervosa and bulimia nervosa, **180**
- Neuroimaging, and obsessive-compulsive disorder, 146. *See also* Brain imaging
- Neuroleptic malignant syndrome (NMS), and antipsychotics, 355
- Neurological conditions, and
 - obsessive-compulsive disorder, 148
- Neurological examination
 - comprehensive evaluation and, 14
 - psychiatric emergencies and, **259**
- Neurological “soft signs,” of
 - attention-deficit/hyperactivity disorder, 58
- Neuropsychological testing, and
 - standardized tests, 20
- Neurotransmitters
 - attention-deficit/hyperactivity disorder and, 57
 - stimulant medications and, 314
 - tic disorders and, 77
- Nightmare disorder, 212–213
- Nonmonosymptomatic enuresis (NMSE), 193, 196
- Non-rapid eye movement (NREM) sleep arousal disorders, 210–213
- Normative experiences, and
 - psychotic symptoms, 89

Normative picky eating, and
avoidant/restrictive food
intake disorder, 176

“No-suicide contracts,” 262.

See also Social contract

Nutritional history, and obesity,
287. *See also* Diet; Food

Obesity, in children, 286–289

Obsessive-compulsive disorder
(OCD)

anorexia nervosa and, 177, **183**

clinical description of, 145

clomipramine for treatment-
resistant, 328

comorbidity in, 76, 80, 89

course and prognosis of,
147–148

differential diagnosis of, 148,

183

epidemiology of, 145–146

etiology of, 146–147

evaluation of, 148

excoriation disorder and, 151

schizophrenia and, 89

selective serotonin reuptake
inhibitors for, 332–333

tic disorders and, 76, 80

treatment of, 148–149

Obsessive-compulsive and related
disorders, in DSM-5, 145.

See also Excoriation
disorder; Obsessive-
compulsive disorder;
Trichotillomania

Obstructive sleep apnea (OSA),
208–209, 214, 287

ODD. *See* Oppositional defiant
disorder

“Off-label” uses, of

psychopharmacological
agents for children, 306, 352

Olanzapine, 96, 273

Omega-3 fatty acids, 362

Open adoption, 283

Oppositional defiant disorder
(ODD)

attention-deficit/hyperactivity
disorder and, 56, 60, 63,
314

behavior therapy for, 381

clinical description of, 225–
226, **227**

comorbidity and, 246, 314

course and prognosis of, 228

differential diagnosis of,
228–229

disruptive mood dysregulation
disorder and, 102, 103

enuresis and, 195

epidemiology of, 226

etiology of, 227–228

evaluation of, 228

stimulant medications for, 314

substance use disorder and, 246
treatment of, 229–230

Oregon Adolescent Depression
Project, 106

OSA. *See* Obstructive sleep
apnea

- OTMP (organization, time management, and planning), 55
- Over-the-counter medications
 drug-drug interactions and, 335
 insomnia and, 206
- Overweight, definition of, 286
- Palilalia, 78
- PANDAS (pediatric autoimmune neurological disorder associated with streptococcal infection), 77, 146, 147
- Panic attacks, 133, 134, 135
- Panic Control Treatment for Adolescents (PCT-A), 135
- Panic disorder
 clinical description of, 133
 differential diagnosis of, 122, 135
 epidemiology of, 133–134
 etiology of, 134
 evaluation of, 134–135
 selective serotonin reuptake inhibitors for, 333
 separation anxiety disorder and, 122
 treatment of, 135
- Paranoia, and group psychotherapy, 387
- Parasomnias, 210–213
- Parent(s). *See also* Education; Family; Marriage; Parent management training
 adoption and, 282, 283
 children of psychiatrically ill, 297–299
 conduct disorder and, 233
 counseling and psychoeducation for, 380
 course of attention-deficit/hyperactivity disorder and, 60
 death of terminally ill child and, 293–294
 education on attention-deficit/hyperactivity disorder for, 66–67
 evaluation of disruptive mood dysregulation disorder and, 103–104
 group therapy and, 389
 history from during evaluation of child, 9, 10
 hospitalization and separation of child from, 290–291
 information resources for, 399–405
 interventions for child maltreatment and, 267
 involvement of in psychosocial treatments for children, 373
 marital problems and oppositional defiant disorder in children, 227
 obesity in, 286
 physical illness in and impact on child, 279–280, 280–281
 preparation of child for evaluation, 7

Parent(s) (*continued*)

- psychosocial causes of intellectual disability and, **30**
- reactions to chronic illness in child, 292–293
- sexual abuse and, 271
- support groups for, 396
- treatment planning and, 21, 22
- treatment of specific learning disorder and, 75
- use of term in context, 1
- Parent-child interaction therapy (PCIT), 123
- Parent-focused treatment (PFT), for anorexia nervosa, 185
- Parent management training (PMT)
 - behavior therapy and, 381–383
 - conduct disorder and, 237–238
 - oppositional defiant disorder and, 229
 - rumination disorder and, 174
- Parents Anonymous, 267
- Parents Without Partners, 278
- Parkinsonian symptoms, and antipsychotics, 355
- Paroxetine
 - depression and, 332
 - obsessive-compulsive disorder and, 149
 - social anxiety disorder or specific phobias and, 132, 333
 - withdrawal symptoms and, 337

Partial hospitalization

- programs for day treatment and, 393
- for separation anxiety disorder, 124

Patient interviews

- comprehensive evaluations of children and, 10, 12
- conduct disorder and, 235
- evaluation of attention-deficit/hyperactivity disorder and, 61

Peabody Picture Vocabulary Test, Fourth Edition (PPVT-4), **16**

Pediatric acute-onset neuropsychiatric syndrome (PANS), 147

Pediatric OCD Treatment Study (POTS), 149

Peer relationships. *See also* Social impairment

- attention-deficit/hyperactivity disorder and, 55
- gender dysphoria and, 221
- physical illness in children and adolescents and, 291

Perinatal causes

- of attention-deficit/hyperactivity disorder, **57**
- of intellectual disability, **31**

Perphenazine, 353

Persistent complex bereavement disorder, 166, 280

Persistent depressive disorder (PDD)

- clinical description of, 104–105

- course and prognosis of, 106–107
- differential diagnosis of, 108
- epidemiology of, 105–106
- etiology of, 106
- evaluation of, 107–108
- treatment of, 108–110
- Persistent (chronic) motor or vocal tic disorder, 76
- Pervasive developmental disorder not otherwise specified (PDD NOS), 41. *See also* Autism spectrum disorder
- Pharmacotherapy. *See also*
 - Adherence to treatment;
 - Administration methods;
 - Anticonvulsants; Antidepressants; Antipsychotics;
 - Black box warnings; Combined treatment; Discontinuation; Dosages; “Off-label” uses; Rebound effects;
 - Selective serotonin reuptake inhibitors; Side effects;
 - Tapering; Withdrawal
- anorexia nervosa and, 185
- attention-deficit/hyperactivity disorder and, 68–69
- autism spectrum disorder and, 49–50
- bulimia nervosa and, 189–190
- conduct disorder and, 238–239
- enuresis and, 197
- hospitalization and, 391
- information resources for parents on, 405
- insomnia and, 206–207
- intellectual disability and, 35–36
- lithium and, 338–342
- major depressive disorder or persistent depressive disorder and, 109
- medically ill children and, 295
- narcolepsy and, 208
- obesity and, 289
- obsessive-compulsive disorder and, 148–149
- out-of-control behavior and, 273
- posttraumatic stress disorder and, 164
- restless legs syndrome and, 214
- schizophrenia and, 90
- social anxiety disorder or specific phobias and, 132
- special issues for children and adolescents, 305–309
- tic disorders and, 81–82
- trichotillomania and, 150
- Phobia, and differential diagnosis of anorexia nervosa, **183**.
See also Specific phobias
- Physical abuse, definition of, 265.
See also Child abuse
- Physical examination. *See also*
 - Laboratory tests;
 - Neurological examination
- attention-deficit/hyperactivity disorder and, 62
- autism spectrum disorder and, 48

Physical examination (*continued*)

- child abuse and, 266
- comprehensive evaluations of
 - children and, 14–15
- conduct disorder and, 236
- emergencies and, 258, **259**
- encopresis and, 199
- generalized anxiety disorder
 - and, 138
- intellectual disability and, 33–34
- pharmacotherapy and, 326
- sexual abuse and, 269
- substance use disorder and, 250

Physicians' Desk Reference

(PDR), 306

Pica

- clinical description of, 171
- course and prognosis of, 172
- epidemiology of, 171
- etiology of, 172
- evaluation of, 172
- treatment of, 172–173

Pimozide, 82

Placebo response, and antidepressants for pediatric depression, 331

Play, and autism spectrum disorder, 45

Play therapy

- child maltreatment and, 267
- communication with children
 - and, 374

Polycystic ovary syndrome, and valproate, 360

Polypharmacy, 306

Polysomnography (PSG), 204, 209

Polyunsaturated fatty acids, and attention-deficit/hyperactivity disorder, 70

Positive and Negative Syndrome Scale for Schizophrenia, 350

Positive reinforcement, and behavior therapy, 173, 382

Positive symptoms, of schizophrenia, 87

Posttraumatic stress disorder (PTSD)

- adjustment disorder and, 166
- catastrophic injury and chronic illness, 292
- child abuse and, 267
- clinical description of, 158–159
- comorbidity and, 162, 232
- conduct disorder and, 232
- course and prognosis of, 160–161
- differential diagnosis of, 162, 166
- epidemiology of, 159–160
- etiology of, 160
- nightmares as symptom of, 213
- sexual abuse and, 270
- treatment of, 162–164

PRACTICE: Psychoeducation and

- Parenting skills, Relaxation, Affective modulation, Cognitive processing, Trauma narrative, In vivo mastery of trauma memories, Conjoint child-parent sessions, and Enhancing safety, 163

- Prader-Willi syndrome, 151
- Prazosin, and posttraumatic stress disorder, 164
- Predominantly hyperactive/
impulsive and predominantly
inattentive presentations, of
attention-deficit/hyperactiv-
ity disorder, 51, **54**
- Pregnancy
in adolescents, 284–286
alcohol use and, 298
pica and, 171
prenatal causes of attention-
deficit/hyperactivity
disorder and, **57**
prenatal causes of intellectual
disability and, **31**
valproate and, 360
- Prescription drug abuse, 246
- Prevalence. *See* Epidemiology
- Primary enuresis, 194
- Privacy, medically ill adolescents
and loss of, 291. *See also*
Confidentiality
- Prodromal phase, of
schizophrenia, 88
- Prognosis. *See* Course and
prognosis
- Propranolol, 164, 239
- Protective factors, for children of
psychiatrically ill parents,
298
- Provisional tic disorder, 76
- PSG (polysomnography), 204,
209
- Psychodynamically oriented
therapy, 376–377
- Psychoeducation. *See also*
Education
family therapy and, 386
group therapy and, 387
parent counseling and, 380
for social anxiety disorder and
specific phobias, 131
- Psychological characteristics, of
children and adolescents
with conduct disorder, **232**
- Psychological factors affecting
other medical conditions,
166
- Psychological sequelae, of child
abuse, 266–267
- Psychological testing
attention-deficit/hyperactivity
disorder and, 62
comprehensive evaluation and,
15, 20
- Psychopharmacology. *See*
Pharmacotherapy
- Psychosis. *See also* Psychotic
disorders
conduct disorder and, 236
differential diagnosis of atten-
tion-deficit/hyperactivity
disorder and, 63–64
group therapy contraindicated
for, 387
- Psychosocial causes, of
intellectual disability, 29,
30

Psychosocial interventions. *See*

also Psychotherapy

adjunctive treatments and,
393–396

behavior therapy and, 380–383

communication with children
or adolescents and, 374

community services and,
389–390

family-based assessment and
treatment as, 383–386

for gender dysphoria, 222

group therapy and, 386–389

milieu treatment and, 390–393

parent counseling and
psychoeducation as, 380

resistant child or adolescent
and, 375

for schizophrenia, 90–91

for tic disorders, 80–81

Psychotherapy. *See also*

Cognitive-behavioral
therapy; Family therapy;

Group therapy; Individual

psychotherapy; Psychosocial
interventions

for anorexia nervosa, 184

for attention-deficit/
hyperactivity disorder,
69–70

common themes in, **376**

for conduct disorder, 237–238

developmentally oriented inter-
ventions for intellectual
disability and, 35

enuresis and, 197

hospitalization and, 391–392

for major depressive disorder
and persistent depressive
disorder, 109

medical illnesses in children
and, 295

for posttraumatic stress
disorder, 162–164

for separation anxiety disorder,
122–123

for specific learning disorder,
75

types of, 375–380

Psychotic depression, 89, **183**

Psychotic disorders, and anti-
psychotics, **348**. *See also*
Psychosis; Schizophrenia

PTSD. *See* Posttraumatic stress
disorder

Quetiapine, 96, 357

Race. *See also* Asian Americans
autism spectrum disorder and,
45

bulimia nervosa and, 186
restless legs syndrome and,
213

RAD. *See* Reactive attachment
disorder

RAISE project (NIMH), 90–91

Rash, as side effect of lamotrigine,
359

- Reactive attachment disorder (RAD)
- autism spectrum disorder and, 49
 - clinical description of, 155–156
 - course and prognosis of, 156–157
 - differential diagnosis of, 49, 108, 157
 - epidemiology of, 156
 - etiology of, 156
 - evaluation of, 157
 - major depressive disorder and, 108
 - treatment of, 157–158
- Rebound effects, and pharmacotherapy, 321, 328
- Recommendations, on treatment of obesity, 288. *See also* Guidelines
- Recovery, from acute phase of schizophrenia, 88
- Recreation, programs as adjunctive treatments, 395
- Refeeding syndrome, and anorexia nervosa, 184
- Relapse, and relapse prevention
- of bulimia nervosa, 188
 - lithium for mood disorders in adolescents and, 341
 - of major depression, 110
 - of substance use disorder, 251–252
- Relaxation training, for medically ill children and adolescents, 296–297
- Religious explanations, about death, 281
- Repetitive behaviors, and autism spectrum disorder, 43
- Residual phase, of schizophrenia, 88
- Response cost, and psychotherapy for attention-deficit/hyperactivity disorder, 69–70
- Response to intervention (RTI), and specific learning disorder, 74
- Restless legs syndrome (RLS), 213
- Restricting type, of anorexia nervosa, 177
- Retentive encopresis, 198
- Rett syndrome, 48
- Reward system, and behavior therapy, 382. *See also* Positive reinforcement
- Risk-benefit ratio, and pediatric psychopharmacology, 307–308
- Risk factors
- for autism spectrum disorder, 46
 - for bulimia nervosa, 188
 - for child abuse, 265
 - for conduct disorder, 234
 - for intellectual disability, 29
 - for progression of oppositional defiant disorder to conduct disorder, 228
 - for schizophrenia, 88

Risk factors (*continued*)

- for substance abuse in adolescents, **247**
- for suicide, 106, 261, 262, **263**

Risperidone

- autism spectrum disorder and, 50, 351
- bipolar disorder and, 96
- conduct disorder and, 239
- disruptive mood dysregulation disorder and, 104
- intellectual disability and, 36
- mania and, 339, 357
- out-of-control behavior and, 273
- posttraumatic stress disorder and, 164
- tic disorders and, 82

Ritualistic behaviors, and obsessive-compulsive disorder, 146

RLS (restless legs syndrome), 213

Rorschach Inkblot Technique, 20

Rumination disorder, 173–174, **183**SAD. *See* Separation anxiety disorder

Safety

- emergencies and, 258–259
- out-of-control behavior and, 274
- sleep terrors and, 212
- treatment of suicidal behavior and, 262

Salt consumption, and lithium blood levels, 342

Savants, and autism spectrum disorder, 45

Schedule for Affective Disorders and Schizophrenia for School-Aged Children and Adolescents (Kiddie-SADS), **15**

Schizophrenia

- anorexia nervosa and, **183**
 - antipsychotics for, 343, **344**, **345**, **346**, **347**, **348**, 350
 - autism spectrum disorder and, 48–49
 - children of parents with, 297, 298
 - clinical description of, 87
 - course and prognosis of, 88, 122
 - differential diagnosis of, 89, **183**
 - epidemiology of, 87
 - etiology of, 88
 - evaluation of, 88–89
 - misdiagnosis of bipolar disorder as, 96
 - out-of-control behavior and, **272**
 - separation anxiety disorder and, 122
 - treatment of, 90–91
- Schools. *See also* Education; Special education; Teachers attention-deficit/hyperactivity disorder and problems with, 55
- classroom behavior modification and, 383

- comprehensive evaluation and, 15
- interventions for conduct disorder in, 239
- medically ill children and, 291
- oppositional defiant disorder in context of, 229
- separation anxiety disorder and refusal or absenteeism, 118, **119**, 121, 122, 123, 125
- specific learning disorder and, 73
- surveys on drug use and, 245
- treatment of attention-deficit/hyperactivity disorder and, 67–68
- Screen for Child Anxiety Related Emotional Disorders (SCARED), 121, 138
- Seasonal affective disorder, 110
- Secondary enuresis, 194, 195
- Secondary mania, 96
- Second-generation antipsychotics (SGAs). *See* Atypical antipsychotics
- Sedation, and antipsychotics, 354
- Seeking Safety, 163
- Seizure disorders, and autism spectrum disorder, 46
- Selective mutism
 - autism spectrum disorder and, 49
 - clinical description of, 125
 - course and prognosis of, 127
 - differential diagnosis of, 49, 127
 - epidemiology of, 125–126
 - etiology of, 126
 - evaluation of, 127
 - treatment of, 127–128
- Selective serotonin reuptake inhibitors (SSRIs)
 - anorexia nervosa and, 185
 - anxiety disorders and, 333
 - attention-deficit/hyperactivity disorder and, 322
 - autism spectrum disorder and, 50, 334
 - bulimia nervosa and, 190
 - depression and, 331–332
 - excoriation disorders and, 151
 - generalized anxiety disorder and, 139
 - indications for and dosages of, **329**
 - initiation of and ongoing treatment with, 335–336
 - intellectual disability and, 36
 - major depression and, 110
 - mechanisms of action, 331
 - panic disorder and, 135
 - posttraumatic stress disorder and, 164
 - risks and side effects of, 336–338
 - separation anxiety disorder and, 123–124
 - social anxiety disorder or specific phobias and, 132
 - trichotillomania and, 150

- Self-help programs
 - for child abuse, 267
 - for substance abuse, 250
- Self-injurious behaviors
 - autism spectrum disorder and, 50
 - intellectual disability and, 28
- Self-report questionnaires, and
 - substance use disorder, 249
- Semistructured diagnostic
 - interviews, **15**
- Separation anxiety disorder (SAD)
 - clinical description of,
 - 115–116, 118
 - course and prognosis of,
 - 120–121
 - differential diagnosis of,
 - 121–122
 - DSM-5 diagnostic criteria for,
 - 117–118**
 - epidemiology of, 118
 - etiology of, 118–120
 - evaluation of, 121–122
 - major depression and, 108
 - selective serotonin reuptake
 - inhibitors for, 333
 - treatment of, 122–125
- Serotonin hypothesis, for
 - obsessive-compulsive
 - disorder, 146
- Serotonin syndrome, and selective
 - serotonin reuptake
 - inhibitors, 337
- Sertraline
 - depression and, 332
 - generalized anxiety disorder
 - and, 139, 333
 - obsessive-compulsive disorder
 - and, 332–333
 - panic disorder and, 135
 - social anxiety disorder or
 - specific phobias and, 132
- Severe mood dysregulation
 - (SMD), 101
- Severity levels, of intellectual
 - disability, 26
- Sex, use of term, 217
- Sexual abuse, 268–271. *See also*
 - Child abuse
- Sexuality, and manic symptoms,
 - 95
- Sexually transmitted diseases, and
 - substance use disorder, 249
- Sexual side effects, of selective
 - serotonin reuptake
 - inhibitors, 337
- Shipleigh-2, **16**
- Shyness, and selective mutism,
 - 127
- Sick-euthyroid syndrome, and
 - anorexia nervosa, 182
- Side effects, of medications
 - of anticonvulsants, 359–361
 - of antidepressants, 336–338
 - of antipsychotics, 90, 354–355
 - of atomoxetine, 323–324
 - differential diagnosis of
 - disruptive mood
 - dysregulation disorder
 - and, 104
 - of guanfacine and clonidine,
 - 327–328
 - of lithium, 341–342

- medically ill children and, 295
- of stimulants, **315**, 319, **320–321**, 321–322
- Simple tics, 76
- Sleep, and sleep disturbance. *See also* Insomnia; Sleep hygiene; Sleep-wake disorders
 - age and evaluation of, 203–205
 - excessive daytime sleepiness and, 207
 - intellectual disability and, 36
 - mania or hypomania and, 95
 - psychiatric disorders and problems with, 214
 - separation anxiety disorder and, 124
 - side effects of stimulants and, 319
- Sleep-deprived EEG, 204
- Sleep history, 204
- Sleep hygiene, 206, 288, 361
- Sleep terror type, of NREM sleep arousal disorder, 211–212
- Sleep-wake disorders,
 - reclassification of in DSM-5, 203. *See also* Circadian rhythm sleep-wake disorders; Insomnia; Narcolepsy; Obstructive sleep apnea; Parasomnias; Restless legs syndrome; Sleep
- Sleepwalking type, of NREM sleep arousal disorder, 210–211
- Slow to warm up (temperament cluster), 10, **11**
- SNAP-IV, 61
- Social anxiety disorder
 - clinical description of, 128
 - course and prognosis of, 129–130
 - differential diagnosis of, 122, 130
 - epidemiology of, 129
 - etiology of, 129
 - evaluation of, 130
 - selective mutism and, 126
 - selective serotonin reuptake inhibitors for, 333
 - separation anxiety disorder and, 122
 - treatment of, 131–133
- Social contract, and behavior therapy, 383. *See also* “No-suicide contracts”
- Social Effectiveness Therapy for Children (SET-C), 132
- Social factors. *See also* Culture; Social impairment; Social skills training
 - obesity and, 287
 - school absenteeism and, **119**
 - side effects of stimulant medications and, **315**
- Social gender transition, and gender dysphoria, 222
- Social impairment. *See also* Peer relationships; Social factors
 - autism spectrum disorder and, **42–43**, 44
 - intellectual disability and, 28

- Social (pragmatic) communication disorder, 49
- Social learning theory, and behavior therapy, 381
- Social services, and treatment planning, 21. *See also* Community services
- Social skills training, and aggression, 274
- Sodium oxybate, and narcolepsy, 208
- Somatic symptom(s)
 - of generalized anxiety disorder, 137, 138
 - of separation anxiety disorder, 121
- Somatic symptom disorder, and chronic illness, 292
- Somnolence, as side effect of clonidine, 327–328
- Special education
 - adjunctive treatments and programs for, 394
 - attention-deficit/hyperactivity disorder and, 67
 - tic disorders and, 81
- Specific learning disorder
 - clinical description of, 71–72
 - course and prognosis of, 73
 - differential diagnosis of, 74
 - etiology of, 72–73
 - evaluation of, 73–74
 - treatment of, 74–75
- Specific phobias
 - clinical description of, 128
 - course and prognosis of, 129–130
 - differential diagnosis of, 130
 - epidemiology of, 129
 - etiology of, 129
 - evaluation of, 130
 - treatment of, 131–133
- Speech. *See also* Language; Speech and language therapy; Speech sound disorder
 - communication disorders and, 36
 - selective mutism and, 126, 128
- Speech-language pathologist (SLP), 39
- Speech and language therapy, for selective mutism, 128
- Speech sound disorder, 39–40
- Standardized instruments, for evaluation, 14, **15**, **16–19** 20
- Standard of living, after divorce, 277
- Stanford-Binet Intelligence Scale, Fifth Edition, **17**
- Statistically significant effects, of psychopharmacology, 305–306
- Stereotypic behaviors, and autism spectrum disorder, **43**
- Stevens-Johnson syndrome, and lamotrigine, 359, 361
- Stimulant medications. *See also* Amphetamine; Methylphenidate
 - attention-deficit/hyperactivity disorder and, 68–69, 82
 - conduct disorder and, 238

- disruptive mood dysregulation disorder and, 104
- formulations and dosages of, **311–313**
- indications for and efficacy of, 310, 314–316
- initiation of and ongoing treatment with, 316–319
- obsessive-compulsive disorder and, 148
- risks and side effects of, 319, **320–321**, 321–322
- tic disorders and, 82
- Strange Situation Procedure, 157
- Stress inoculation, 296
- Stressors. *See also* Trauma- and stressor-related disorders
- adjustment disorder and reactions to, 165
- assessment of emergency and, **257**
- Structural family therapy, 385
- Structured diagnostic interview, **15**
- Subdiagnostic threshold symptoms, of tic disorders, 77
- Substance Abuse and Mental Health Services Administration (SAMHSA), 245
- Substance use disorder (SUD). *See also* Alcohol abuse
- bipolar disorder and, 95
- clinical description of, 243
- comorbidity of, 246
- conduct disorder and, 233, 246
- course and prognosis of, 247–249
- differential diagnosis of, 249–250
- DSM-5 diagnostic criteria for, **243–244**
- epidemiology of, 244–246
- etiology of, 247
- evaluation of, 249
- school absenteeism and, **119**
- sleep disturbances and, 214
- treatment of, 250–252
- Suicide, and suicidal ideation
- adjustment disorder and, 165–166
- anorexia nervosa and, 179
- anticonvulsants and risk of, 359
- antidepressants and risk of, 336, 338
- bereavement and, 280
- bulimia nervosa and, 187
- conduct disorder and, 231
- emergencies and, **257**, 260–264
- intellectual disability and, 28–29
- major depression and, 106, 107
- posttraumatic stress disorder and, 161
- risk factors for, 106, 261, 262, **263**
- schizophrenia and, 88
- substance use disorder and, 248
- Summer camps, 395

- Support groups. *See also* Group therapy
 for children of psychiatrically ill parents, 299
 divorce and, 278
 for gender dysphoria, 222
 medical illness in children and, 295
 for parents, 396
- Supportive psychotherapy, 81, 375–376, 384
- Surgery, for obstructive sleep apnea, 209
- Surviving Cancer Competently Intervention Program (SCCIP), 163
- Symptom duration, and diagnosis
 of posttraumatic stress disorder or acute stress disorder, 159
- Synthetic drugs, 246
- Systematic desensitization, for
 social anxiety disorder and specific phobias, 131
- Systemic lupus erythematosus, 292
- Tapering. *See also* Discontinuation
 of antidepressants, 336
 of lithium, 341
- Tardive dyskinesia, and
 antipsychotics, 355
- Teacher(s). *See also* Schools
 classroom behavior
 modification and, 383
 comprehensive evaluations of
 children and, 15
 attention-deficit/hyperactivity disorder,
 evaluation of 61
 treatment of, 67–68
- Teacher Report Form (TR), 14, 61
- Temperament, and comprehensive
 evaluation, 10, **11**
- Temper outbursts, and out-of-control behavior, **272**.
See also Anger
- Textbook of Child and Adolescent Psychiatry*, 2nd Edition
 (Dulcan 2016), 1
- Therapeutic levels, of lithium,
 340–341
- Thiothixene, 353
- Thyroid function, and anorexia
 nervosa, 182, **183**
- Thyroid-stimulating hormone
 (TSH), and lithium, 341
- Tic disorders. *See also* Tourette's
 disorder
 clinical description of, 76
 comorbidity of, 76–77
 course and prognosis of, 78–79
 differential diagnosis of, 80
 epidemiology of, 76
 etiology of, 77–78
 evaluation of, 79–80
 side effects of stimulants and,
 319, 321
 treatment of, 80–82
- Time-limited therapy, 377
- Time line, and developmental
 history, 10

- Token economy, and behavior therapy, 381, 382. *See also* Positive reinforcement
- Tolerance. *See also* Withdrawal stimulant medications for ADHD and, 318, 319 substance use disorder and, **244**
- Tourette Association of America, 81, 401
- Tourette's disorder
 antipsychotics for, **344, 348**, 351
 attention-deficit/hyperactivity disorder and, 56
 clinical description of, 76
 comorbidity and, 76–77
 course and prognosis of, 78–79
 differential diagnosis of, 80
 etiology of, 77–78
 evaluation of, 79–80
 guanfacine and clonidine for, 325–326
 information resources for parents on, 405
 treatment of, 80–82
- Toxicity, and side effects of medications, 306, 342, 359
- Transcranial magnetic stimulation, 110
- Transdermal skin patch, and clonidine, 327, 328
- Transgender and transsexual, use of terms, 217
- Transient tic disorder, 76
- Trauma. *See also* Trauma- and stressor-related disorders
 obsessive-compulsive disorder and, 147
 preventative approach following, 162
 schizophrenia and, 89
 type of and development of acute stress disorder, 160
- Trauma Affect Regulation: Guidelines for Education and Therapy (TARGET), 163
- Trauma-focused cognitive-behavioral therapy (TF-CBT), 163
- Trauma Grief Component Therapy for Adolescents (TGCT-A), 163
- Trauma- and stressor-related disorders, as new category in DSM-5, 155. *See also* Acute stress disorder; Adjustment disorders; Disinhibited social engagement disorder; Posttraumatic stress disorder; Reactive attachment disorder
- Trauma Systems Therapy (TST), 163
- Trazodone, 331, 338
- Treatment. *See also* Adjunctive treatments; Adherence to treatment; Combined treatment; Hospitalization; Pharmacotherapy; Play therapy; Psychosocial interventions; Psychotherapy of acute stress disorder, 162–164

Treatment (*continued*)

- of adjustment disorder, 166–167
- of adolescent pregnancy, 285–286
- of anorexia nervosa, 182, 184–185
- of attention-deficit/hyperactivity disorder, 64, 66–71
- of autism spectrum disorder, 49–50
- of bipolar disorder, 96–97
- of bulimia nervosa, 189–190
- of child abuse and neglect, 267
- for children of psychiatrically ill parents, 299
- of communication disorders, 39
- of conduct disorder, 236–239
- of disinhibited social engagement disorder, 157–158
- of disruptive mood dysregulation disorder, 104
- of encopresis, 200
- of enuresis, 196–197
- of excoriation disorder, 151
- of gender dysphoria, 222
- of generalized anxiety disorder, 138–139
- of insomnia, 206–207
- of intellectual disability, 34–36
- involvement of parents and family as requirement for successful, 373, 383–384
- of major depressive disorder and persistent depressive disorder, 108–110
- of narcolepsy, 208
- of obesity, 287–289
- of obsessive-compulsive disorder, 148–149
- of obstructive sleep apnea, 209
- of oppositional defiant disorder, 229–230
- of out-of-control behavior, 273–274
- overview of issues in, 5
- of panic disorder, 135
- of pica, 172–173
- planning of, 20–22
- of posttraumatic stress disorder, 162–164
- of reactive attachment disorder, 157–158
- of restless legs syndrome, 214
- of rumination disorder, 174
- of schizophrenia, 90–91
- of selective mutism, 127–128
- of separation anxiety disorder, 122–125
- for sexual abuse victims, 270–271
- of social anxiety disorder and specific phobias, 131–133
- of specific learning disorder, 74–75
- of substance use disorder, 250–252
- of suicidal behavior, 262, 264
- of tic disorders, 80–82
- of trichotillomania, 150

- Treatment for Adolescents With Depression Study, 109
- Treatment of Early Age Mania (TEAM) study, 339, 357
- Treatment of Resistant Depression in Adolescents (TORDIA), 110, 332
- Trichotillomania, 150
- Tricyclic antidepressants (TCAs), 328, **330**, 334
- Underdiagnosis
 - of depression in patients with intellectual disability, 28
 - of panic disorder, 135
- Unemployment, in adults with Tourette's disorder, 79
- U.S. Pharmacopeial Convention (USP), 362
- Universal Nonverbal Intelligence Test, Second Edition (UNIT-2), **17**
- Unspecified-onset conduct disorder, 230
- Urinalysis, and encopresis, 199
- Urinary incontinence, and enuresis, 193, **195**
- Urine alarms, and enuresis, 197
- Valproate
 - conduct disorder and, 239
 - disruptive mood dysregulation disorder and, 104
 - initiation of and ongoing treatment with, 358
 - side effects of, 360
- Vanderbilt ADHD Diagnostic Teacher Rating Scale, 62
- V codes, in DSM-5, 3, **4**
- Venlafaxine
 - generalized anxiety disorder and, 139, 333
 - major depression and, 110
 - mechanism of action, 331
 - side effects of, 335, 338
 - social anxiety disorder and specific phobias, 132
- Vindictive symptoms, of oppositional defiant disorder, 226
- Vineland Adaptive Behavior Scales, Second Edition, **19**
- Violence, and out-of-control behavior, 271, 273. *See also* Domestic violence
- Web-based curricula, for treatment of PTSD, 163
- Wechsler Abbreviated Scale of Intelligence, Second Edition (WASI-II), **17**
- Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV), **17**
- Wechsler Individual Achievement Tests, Third Edition (WIAT-III), **18**
- Wechsler Intelligence Scale for Children, Fifth Edition (WISC-V), **17**

- Wechsler Preschool and Primary Scale of Intelligence, Fourth Edition (WPPSI-IV), **17**
- Weight-based dosing guide, for lithium, 340
- Weight gain, and antipsychotics, 353, 354
- Western Psychological Services, 48
- Wide Range Achievement Test, Fourth Edition (WRAT-4), **18**
- Williams syndrome, 156
- Withdrawal
 - antipsychotics and, 355
 - selective serotonin reuptake inhibitors and, 337
 - substance use disorder and, **244**
- Woodcock-Johnson Scales of Independent Behavior—Revised (SIB-R), **19**
- Woodcock-Johnson Tests of Achievement, Fourth Edition (WJIV), **18**
- Woodcock-Johnson Tests of Cognitive Ability, Fourth Edition (WJIV), **18**
- Worry
 - generalized anxiety disorder and, 137
 - separation anxiety disorder and, **117**
- Wraparound services, 390
- Youth Risk Behavior Surveillance System (YRBSS), 245–246
- Youth Self-Report (YSR), 14
- Zero to Three, 401
- Ziprasidone, and tic disorders, 82

“The fifth edition of *Concise Guide to Child and Adolescent Psychiatry* includes important DSM-5 updates and clearly defined reviews of new and changed diagnoses. The handy size, conciseness of chapters, and readability remain as hallmarks of this classic book. This edition will continue to be the favorite reference in our division for all medical students and residents because it is replete with highlights of all important areas of child and adolescent psychiatry.”

Jeffrey Hunt, M.D., Professor and Program Director, Child and Adolescent Psychiatry Fellowship and Combined Program in Pediatrics, Psychiatry, and Child Psychiatry, Alpert Medical School of Brown University

“Dr. Dulcan and colleagues have done an outstanding job in summarizing the salient elements in a concise, digestible format. This guide will be helpful not only to trainees but to those preparing for MOC and to more seasoned professionals looking for summaries on a variety of child mental health topics. This volume is a must for any mental health professional’s bookshelf.”

Oscar G. Bukstein, M.D., M.P.H., Associate Psychiatrist-in-Chief and Vice Chairman of Psychiatry, Training Director, Child & Adolescent Psychiatry Fellowship, Boston Children’s Hospital; Professor of Psychiatry, Harvard Medical School

ISBN 978-1-61537-078-8



AMERICAN
PSYCHIATRIC
ASSOCIATION
PUBLISHING



WWW.APPI.ORG