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CHAPTER

27

Impulse-Control Disorders Not Elsewhere Classified

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Intermittent Explosive Disorder

The DSM-IV-TR diagnostic criteria for intermittent explosive disorder are shown in Table 27–1.

Two DSM-IV-TR diagnoses are currently available to the clinician who wishes to diagnose primarily episodic violent behavior: *intermittent explosive disorder* and *personality change due to a general medical condition, aggressive type*. Intermittent explosive disorder (see Table 27–1) has numerous exclusion criteria, whereas personality change due to a general medical condition requires the presence of a specific organic factor that is judged to be causally related to the violence (Shaw and Fletcher 2000). Most individuals with episodic violent behavior do not meet the diagnostic criteria for either disorder but have another psychiatric disorder such as schizophrenia, paranoid disorder, mania, substance abuse, drug withdrawal, delirium, a personality disorder (especially borderline or antisocial), mental retardation, a conduct disorder, or organic brain disease (Tardiff 1992).

TABLE 27–1. DMS-IV-TR diagnostic criteria for intermittent explosive disorder

A. Several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property.

B. The degree of aggressiveness expressed during the episodes is grossly out of proportion to any precipitating psychosocial stressors.

C. The aggressive episodes are not better accounted for by another mental disorder (e.g., antisocial personality disorder, borderline personality disorder, a psychotic disorder, a manic episode, conduct disorder, or attention-deficit/hyperactivity disorder) and are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., head trauma, Alzheimer's disease).

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Etiology

Episodic violent behavior occurs in patients for biological (e.g., neuronal discharges), psychological (e.g., motivational), and social reasons (Lesch and Merschdorf 2000). A continuum exists between "faulty learning" and "faulty equipment" (Monroe 1970). Individuals with episodic violence have abnormalities in serotonergic and noradrenergic function (Virkkunen et al. 1996), as well as increased testosterone, dopamine, and arginine vasopressin levels (Kavoussi et al. 1997). Reduced function of γ-aminobutyric acid (GABA) and acetylcholine also has been associated with agitation (Lindenmayer 2000). A review by Chernasky and Hollander (1997) of the neurobiology and neurophysiology of aggression and impulsivity indicated that it was unclear whether all of these findings pertain to those with intermittent explosive disorder.

Evaluation

The evaluation of the patient with potential intermittent explosive disorder includes a review of the chief complaint, history of the present illness, psychiatric and substance use history, family history, personal and developmental history, medical history (e.g., history of seizures), mental status, and results of physical examination. Information from the patient, relatives, therapist, primary care physician, prior evaluations of violence, and police and criminal records may be of utility. The onset, course, and severity of the violent behavior, as well as other types of reckless behavior (e.g., suicidal behavior, reckless driving, destruction of property, reckless spending, and reckless sexual behavior), are assessed. It may be helpful to quantify the behavior with the Overt Aggression Scale (Yudofsky et al. 1986) or Overt Agitation Severity Scale (Yudofsky et al. 1997). Visual evoked potential studies and special diagnostic techniques (e.g., electroencephalographic activation with chloralose) may prove useful in evaluation of these patients (Bars et al. 2001).

Treatment

No drug is currently specifically approved by the U.S. Food and Drug Administration (FDA) for the treatment of intermittent explosive disorder or other forms of aggression. However, numerous pharmacological agents have been reported to be effective in diminishing violent behavior in some individuals, largely in anecdotal reports: neuroleptics, benzodiazepines, lithium, β-blockers (especially propranolol), anticonvulsants (especially carbamazepine), serotonin-modulating drugs (tryptophan, trazodone, buspirone, clomipramine, selective serotonin reuptake inhibitors [SSRIs]), polycyclic antidepressants, monoamine oxidase inhibitors (MAOIs), and psychostimulants (Tardiff 1992). Medications with few long-term side effects are advantageous because the duration of the disorder is currently unknown but may be persistent, as with other aggressive disorders.

Lithium and other mood stabilizers may be useful in the treatment of aggression not associated with a manic episode. Campbell and colleagues (1984) treated children who had conduct disorder with lithium and reported decreases in aggressive behavior, especially when the behavior contained strong affective components. In a double-blind, placebo-controlled study, Sheard and coworkers (1976) found that lithium reduced aggression in prisoners who did not have mood disorders. Fava (1997) noted that lithium appears to be effective in the treatment of aggression in prison inmates without epilepsy, mentally retarded and disabled patients, children with conduct disorder, and patients with bipolar disorder. On the basis of the hypothetical relationship between seizures and aggressive behavior, carbamazepine was used in the early 1970s to treat patients with rage outbursts, especially patients who had seizure foci located in the temporal lobe or limbic structures (Mattes 1986). Carbamazepine reduces aggressive behavior in patients with intermittent explosive disorder and without overt epilepsy (Mattes 1990); the average dose was 860 mg, and the mean serum level was 8.6 μg/mL. Other studies have suggested the benefit of carbamazepine for developmentally disabled individuals with agitation (Folks et al. 1982) and for persons with dementia (Gleason and Schneider 1990), as well as valproate for those with borderline personality disorder (Stein et al. 1995) and dementia (Narayan and Nelson 1997).

Antipsychotic medication has been used for acute and long-term violence of many etiologies, although the side effect risk has prompted reevaluation of that practice. Atypical antipsychotics have a better profile than typical antipsychotics. In a 12-week double-blind, placebo-controlled trial, risperidone was better than haloperidol and placebo for behavioral disturbance associated with dementia (De Deyn et al. 1999).

Elliott (1977) was among the first to use propranolol to treat the aggressive behavior seen in brain-injured patients. Numerous other studies showed the relative benefit of β-blockers in patients with and without overt brain injury (Mattes 1990). More recent research has suggested that compounds that modulate serotonin transmission, such as buspirone, serotonin reuptake inhibitors, trazodone, and clomipramine, may benefit some patients. This also appears true in disorders of pathological aggression (Fava 1997).

Kleptomania

The DSM-IV-TR diagnostic criteria for kleptomania are shown in Table 27–2.

Etiology

Numerous theories have been proposed in an effort to understand the stealing behavior of individuals with kleptomania. Explanations have included a need for stimulation to help treat underlying depression (Fishbain 1987) and a desire to emotionally compensate for an actual or anticipated loss (Cupchik and Atcheson 1983).

Evaluation

The practitioner must consider whether the stealing behavior represents a choice to commit an antisocial act with potential secondary gain rather than the taking of items not needed for personal use. Because many patients with kleptomania do not report their stealing behavior to mental health evaluators, specific questions must be asked. Such questions involve the onset, frequency, duration, and magnitude of stealing behavior as well as any previous treatment. In addition, the clinician should evaluate for a comorbid psychiatric disorder, including depression, mania, alcohol or substance abuse, anxiety disorder, eating disorder, personality disorder, or other impulse-control disorder, as well as a family history of mental illness. The evaluator also should screen for any possible head injury or organic disorder, with particular attention in elderly individuals referred for new-onset stealing behavior (Moak 1988).

Treatment

Various treatment approaches have been described for kleptomania. Nonpharmacological treatments that have been used include systematic desensitization (Marzagao 1972), "assertive training" (Wolpe 1958), aversive conditioning (Keutzer 1972), covert sensitization (Glover 1985), self-imposed banning by shoppers (Goldman 1991), adjunctive sexual counseling for those with disturbed sexual relationships (Turnbull 1987), and Shoplifters Anonymous. Pharmacotherapy also has been used to help decrease impulsive urges to steal.

A few published case reports suggest that SSRIs, such as paroxetine (Kraus 1999), fluvoxamine (Chong and Low 1996), and fluoxetine (McElroy et al. 1989), may be useful in decreasing the frequency of stealing behavior in individuals with kleptomania. The use of antidepressants may be particularly helpful in individuals with kleptomania with a comorbid depressive disorder. Therapeutic efficacy has also been reported with lithium, valproate, amitriptyline, imipramine, nortriptyline, trazodone, and electroconvulsive therapy (ECT) in adults with kleptomania (McElroy et al. 1995).

TABLE 27–2. DMS-IV-TR diagnostic criteria for kleptomania

A. Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value.

B. Increasing sense of tension immediately before committing the theft.

C. Pleasure, gratification, or relief at the time of committing the theft.

D. The stealing is not committed to express anger or vengeance and is not in response to a delusion or a hallucination.

E. The stealing is not better accounted for by conduct disorder, a manic episode, or antisocial personality disorder.

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Pyromania

The DSM-IV-TR diagnostic criteria for pyromania are shown in Table 27–3.

Etiology

The vast majority of the literature related to fire setting addresses factors associated with arsonists, individuals who usually do not qualify for a diagnosis of pyromania. Family backgrounds of fire setters have indicated higher-than-expected rates of mental illness, antisocial personality disorder, and alcoholism.

Evaluation

When evaluating a fire setter for the diagnosis of pyromania, the clinician must carefully determine whether the individual meets the established diagnostic criteria for this disorder. Additional important areas to review include the following:

TABLE 27–3. DMS-IV-TR diagnostic criteria for pyromania

A. Deliberate and purposeful fire setting on more than one occasion.

B. Tension or affective arousal before the act.

C. Fascination with, interest in, curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences).

D. Pleasure, gratification, or relief when setting fires, or when witnessing or participating in their aftermath.

E. The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's living circumstances, in response to a delusion or hallucination, or as a result of impaired judgment (e.g., in dementia, mental retardation, substance intoxication).

F. The fire setting is not better accounted for by conduct disorder, a manic episode, or antisocial personality disorder.

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- Motive for starting the fire
- Location of the fire
- Means of starting the fire
- History and progression of fire-setting behavior
- Age at first offense
- Aggression toward other inanimate objects
- Presence or absence of a co-conspirator
- Intent to harm persons, property, or both
- History of violence
- History of "watching" other fires or spending time in fire stations
- History of setting off fire alarms
- Actions during the fire
- Behaviors after setting the fire
- Self-injurious behavior using fire
- History of smoking
- Access to flammables
- Family history of arson

An electroencephalogram (EEG) is appropriate for those individuals with symptoms consistent with a seizure disorder.

In some situations, fire setting represents a suicide attempt, and further inquiry regarding suicidal intent is recommended. Careful attention also must be given to the presence of intoxication, personality disorders, mental retardation, mood disorders, and psychosis. In particular, psychotic arsonists almost always act alone (Molnar et al. 1984), and their targets are frequently outsiders of the community rather than individuals they know (Virkkunen 1974). Virkkunen (1974) also found that hallucinations and delusions were the principal motivation for the fire-setting behavior in one-third of schizophrenic arsonists.

Treatment

Limited studies have been conducted on effective treatment strategies for pyromania. Traditional psychoanalytic approaches have not proven effective, and no research supports the theory that pyromania is caused by unresolved psychosexual conflicts. Other treatment modalities that have been implemented include behavior therapy with aversive conditioning (McGrath and Marshall 1979), positive reinforcement (Bumpass et al. 1983), social skills training (Jackson et al. 1987), and implementation of a relapse prevention plan (Stewart 1993). Because no single approach has been proven effective for all fire setters, treatment should be individualized with a combination of interventions as appropriate.

Pathological Gambling

The DSM-IV-TR diagnostic criteria for pathological gambling are shown in Table 27–4.

Studies of pathological gamblers classified two broad subtypes: *antisocial-impulsive* (also known as action subtype) and *obsessive-dependent* (escape subtype). Although both subtypes have characteristics common to many pathological gamblers (such as neuroticism, low self-esteem, and manipulativeness), important differences exist. *Action gamblers* are predominantly male and achieve a euphoric state through gambling. Such individuals tend to be domineering, controlling, and manipulative; have an above-average IQ; and view themselves as friendly, sociable, gregarious, and generous. They are often energetic, assertive, persuasive, and confident in their interpersonal interactions. Action gamblers are reluctant to recognize that they have a gambling problem and generally are resistant to treatment. In contrast, *escape gamblers* are represented nearly equally by men and women. These individuals tend to be nurturing, responsible, and active in their family life prior to the onset of pathological gambling. In their interpersonal relationships, they are passive-avoidant, unassertive,

TABLE 27–4. DMS-IV-TR diagnostic criteria for pathological gambling

A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:

(1) is preoccupied with gambling (e.g., preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble)

(2) needs to gamble with increasing amounts of money in order to achieve the desired excitement

(3) has repeated unsuccessful efforts to control, cut back, or stop gambling

(4) is restless or irritable when attempting to cut down or stop gambling

(5) gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)

(6) after losing money gambling, often returns another day to get even ("chasing" one's losses)

(7) lies to family members, therapist, or others to conceal the extent of involvement with gambling

(8) has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling

(9) has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling

(10) relies on others to provide money to relieve a desperate financial situation caused by gambling

B. The gambling behavior is not better accounted for by a manic episode.

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and in need of empowerment. Escape gamblers often have a history of physical, sexual, or emotional abuse and use gambling as a means to escape their problems. These individuals describe feelings of temporary elation and release from physical and emotional pain while gambling. They may seek professional help for assistance with their gambling, relationship issues, or both. Escape gamblers are more amenable to treatment, with a better prognosis when compared with action gamblers (Committee on the Social and Economic Impact of Pathological Gambling 1999).

Etiology

Anderson and Brown (1987) proposed that gamblers have low autonomic arousal that they seek to augment by gambling. Consequently, pathological gamblers become addicted to their own arousal because of its self-reinforcing physical and psychological effects. Follow-up studies suggest that increased arousal and risk taking described in pathological gamblers may be linked to noradrenergic system dysfunction (Roy et al. 1988). Although abnormalities in the endorphin system also have been suggested as a possible contributor to increased arousal in pathological gamblers, studies examining this link have not noted significant differences in plasma β-endorphin levels between pathological gamblers and nongamblers (Blaszczynski et al. 1986).

Associated Diagnoses

A significant relationship exists between pathological gambling and substance abuse, with more than 60% of pathological gamblers meeting criteria for a substance abuse disorder at least once in their lifetime. The incidence of pathological gambling is 8–10 times greater among those with alcohol dependence than in the general population (Lejoyeux et al. 2000). One study examining the overlap of alcoholism and pathological gambling suggested a common genetic vulnerability for these two disorders (Slutske et al. 2000). Depressive disorders co-occur in approximately 18% of pathological gamblers, a rate similar to that found among substance-dependent populations (Crockford and el-Guebaly 1998).

Personality disorders have been reported to have moderate comorbidity with pathological gambling. In a study of 82 inpatient pathological gamblers, more than 90% met criteria for a personality disorder, primarily narcissistic, histrionic, or borderline (Blaszczynski and Steel 1998). Although possible comorbidity with antisocial personality disorder has been alluded to in the literature, research shows that the antisocial behavior conducted by individuals with pathological gambling typically occurs after their gambling becomes problematic rather than as a result of a separate antisocial personality disorder (Blaszczynski and Silove 1996).

Evaluation

The evaluator's primary task is to carefully determine whether the individual meets the diagnostic criteria for pathological gambling and to assess the extent of comorbid conditions. In addition to the DSM-IV-TR criteria, the South Oaks Gambling Screen (Lesieur and Blume 1987) may be useful in assessing the severity of the gam-

bling behavior. Potential comorbid diagnoses to consider include alcohol and substance abuse, personality disorders, depression, and bipolar mania. Pathological gambling is not diagnosed if the gambling behavior is the result of a manic episode. The examiner's challenge is to distinguish between the elation sometimes induced by gambling behavior and the euphoria secondary to mania.

Treatment

Several pharmacological agents have been prescribed with varied rates of success, particularly in the presence of a comorbid psychiatric disorder. Lithium carbonate has helped decrease gambling behavior in pathological gamblers with bipolar disorder (Moskowitz 1980). SSRIs have been shown to assist particularly in alleviating impulsive and compulsive aspects of gambling behavior.

Hollander and colleagues (2000) reported complete cessation of gambling for 8 weeks in 7 of 10 pathological gamblers in response to 220 mg/day of fluvoxamine therapy. Studies note that comorbid diagnosis of depression may further warrant treatment with an antidepressant that affects serotonergic activity. Concerns have been raised about the possibility that SSRIs may induce underlying mania in some patients with mood disorders. Screening for comorbid mood disorders should diminish the possibility of a manic episode following SSRI treatment of pathological gambling.

Opioid antagonists have been shown to block the excitement or pleasure of addictive behavior and have been suggested as a possible treatment for pathological gambling. In particular, naltrexone has shown promising results for curbing intense cravings that often accompany pathological gambling behavior (Kim 1998).

Trichotillomania

The DSM-IV-TR diagnostic criteria for trichotillomania are shown in Table 27–5.

Etiology

A number of biological etiologies have been proposed for trichotillomania. Christenson and colleagues (1992) reported that 8% of 161 patients knew a first-degree relative who had pulled hair, and in other family studies, between 4% and 5% of relatives reported current or past hair-pulling behavior (Diefenbach et al. 2000). A neuroethological theory views trichotillomania and obsessive-compulsive disorder (OCD) as pathology of the neurobiological mechanisms responsible for grooming behavior. Neurotransmitter dysregulation is supported by phenomenological similarities of trichotillomania symptoms to those of other disorders, studies of neurotransmitter and other neuroendocrine functions, and treatment studies with SSRIs.

TABLE 27–5. DMS-IV-TR diagnostic criteria for trichotillomania

A. Recurrent pulling out of one's hair resulting in noticeable hair loss.

B. An increasing sense of tension immediately before pulling out the hair or when attempting to resist the behavior.

C. Pleasure, gratification, or relief when pulling out the hair.

D. The disturbance is not better accounted for by another mental disorder and is not due to a general medical condition (e.g., a dermatological condition).

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

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Some reports in the literature suggest that trichotillomania may be a type of OCD (Jenike 1989). Stanley and colleagues (1993), however, reported several differences between OCD and trichotillomania, including the facts that hair pulling usually is associated with pleasure, patients with trichotillomania have few associated obsessive-compulsive symptoms, and patients with OCD differ from patients with trichotillomania in terms of anxiety, depression, and personality characteristics.

Associated Diagnoses

Psychiatric disorders are commonly comorbid with trichotillomania (e.g., mood [65%], anxiety [57%], and personality [25%–55%] disorders) (Soriano et al. 1996).

Evaluation

The clinical interview requires a substantial collection of historical information, including quantitative and qualitative pulling of one's hair from the scalp, eyelashes, eyebrows, facial hair, and other regions; tension before pulling; and pleasure or gratification when pulling. A relation, if any, to psychosocial stressors, menstruation, and other triggers should be explored. Family history of hair pulling should be noted. Screening for symptoms of obsessive-compulsive, depressive, other anxiety, and tic disorders may identify

comorbid psychiatric conditions. Medical disorders (e.g., dermatological) are prudent to evaluate in collaboration with primary and other specialty physicians as indicated.

Physical examination and procedures may substantiate the diagnosis of trichotillomania. Adult patients generally do not have changes in fingernails or toenails (except possibly signs of nail biting) usually associated with dermatological conditions. Hair regrowth follows the application of collodion to the area of hair loss for 1 week. In children, careful parental observation of the child includes looking for hair among the child's playthings. The child also may practice onychophagy (i.e., nail biting). If the child complains of gastrointestinal symptoms such as abdominal pain, diarrhea and/or constipation, or decreased appetite, examination of the oral cavity for evidence of trichophagy (i.e., eating hair) and X-ray examination of the stomach for a trichobezoar (i.e., hairball) are warranted. In addition, a skin biopsy may provide evidence for the diagnosis by ruling out other conditions. Finally, longitudinal photographs may facilitate changes in the course or responses to treatment.

Treatment

Open trials indicate favorable response to fluoxetine (Winchel et al. 1992), fluvoxamine (Stanley et al. 1997), venlafaxine (Ninan et al. 1998), lithium carbonate (Christenson et al. 1991), and haloperidol (Van Ameringen et al. 1999). Augmentation of SSRIs also has been successful with haloperidol (Van Ameringen et al. 1999), pimozide (Stein and Hollander 1992), and risperidone (Epperson et al. 1999). Isolated case reports of successful treatment of trichotillomania in adults have been published regarding the following drugs: buspirone, citalopram, amitriptyline, a monoamine oxidase inhibitor, chlorpromazine in a patient with comorbid schizophrenia, and olanzapine augmentation of an SSRI. For children and adolescents, paroxetine and fluoxetine use has been reported. A steroid topical cream also has been used in combination with clomipramine when hair pulling is cued by itch (Black and Blum 1992). Another topical cream has been used to increase physical sensitivity (Ristvedt and Christenson 1996).

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Study Questions

Select the single best response for each question.

1. Intermittent explosive disorder may be associated with
   A. Decreased dopamine levels.
   B. Decreased serotonin levels.
   C. Low testosterone levels.
   D. Increased γ-aminobutyric acid levels.
   E. Increased acetylcholine levels.

2. A nonpharmacological treatment indicated for kleptomania is
   A. Group therapy.
   B. Cognitive-behavioral psychotherapy.
   C. Interpersonal psychotherapy.
   D. Psychodynamic psychotherapy.
   E. Systematic desensitization.

3. A nonpharmacological treatment indicated for pyromania is
   A. Aversive conditioning.
   B. Cognitive-behavioral psychotherapy.
   C. Interpersonal psychotherapy.
   D. Psychodynamic psychotherapy.
   E. Psychoanalytic psychotherapy.

4. A patient who fits the antisocial–impulsive (action) subtype of pathological gambling
   A. Is most likely a female.
   B. Experiences a euphoric state while gambling.
   C. Is generally passive in day-to-day life.
   D. Is less manipulative than the obsessive–dependent (escape) subtype.
   E. Is stingy about spending money.

5. Which of the following comorbid disorders is most likely to be present in a patient with trichotillomania?
   A. Body dysmorphic disorder.
   B. Eating disorder.
   C. Mood disorder.
   D. Psychotic disorder.
   E. Substance abuse.