CHAPTER

19

Impulse-Control Disorders Not Elsewhere Classified

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Whereas impulse-control disorders (ICDs) were once conceptualized as either addictive or compulsive behaviours, they are now classified within the DSM-IV-TR (American Psychiatric Association 2000) ICD category (Table 19–1).

In DSM-IV-TR, ICDs are characterized by five stages of symptomatic behaviour (Table 19–2).

TABLE 19–1. DSM-IV-TR impulse-control disorders

Impulse-control disorders not elsewhere classified
Intermittent explosive disorder
Kleptomania
Pyromania
Pathological gambling
Trichotillomania
Impulse-control disorders not otherwise specified
Impulsive-compulsive sexual disorder
Impulsive-compulsive self-injurious disorder
Impulsive-compulsive Internet usage disorder
Impulsive-compulsive buying disorder

Source. American Psychiatric Association 2000.

Intermittent Explosive Disorder

Definition and Diagnostic Criteria

Intermittent explosive disorder (IED) is a DSM diagnosis used to describe people with pathological impulsive aggression. In community surveys, 12%–25% of men and women in the United States reported engaging in physical fights as adults, a frequent manifestation of impulsive aggression (Robins and Regier 1991). Impulsive aggressive behaviour usually is pathological and causes substantial psychosocial distress or dysfunction (McElroy et al. 1998) (see Table 19–3).

Epidemiology

Several studies have looked at clinical populations, and one community survey has been done to determine the prevalence of IED. Numbers range between 1.1% and 6.3%. Some data suggest that the male-to-female ratio is approximately 1:1 (Coccaro et al. 2005).

Comorbidity

The most frequent Axis I diagnoses comorbid with IED are mood, anxiety, substance, eating, and other ICDs, with lifetime rates ranging from 7% to 89% (Coccaro et al. 1998a; McElroy et al. 1998).

TABLE 19–2. Core features of impulse-control disorders

Essential features	The individual fails to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others
Before the act	The individual feels an increasing sense of tension or arousal
At the time of committing the act	The individual experiences pleasure, gratification, or relief
After the act	The individual experiences a sense of relief from the urge
	The individual may or may not feel regret, self-reproach, or guilt

Source. American Psychiatric Association 2000.

Bipolar Disorder

Symptoms in common with both manic and IED episodes included irritability (79%–92%), increased energy (83%–96%), racing thoughts (62%–67%), anxiety (21%–42%), and depressed (dysphoric) mood (17%–33%); 56% of the subjects in question had a comorbid bipolar diagnosis of some type (McElroy et al. 1998). Mood stabilizers, rather than selective serotonin reuptake inhibitors (SSRIs), would be the first-line treatment for IED comorbid with bipolar disorder.

Other Impulse-Control Disorders

McElroy et al. (1998) reported that up to 44% of their IED subjects had another ICD, such as compulsive buying (37%) or kleptomania (19%).

Borderline and Antisocial Personality Disorders

Coccaro et al. (1998a) reported the rate of borderline personality disorder (BPD) and/or antisocial personality disorder in IED subjects to be 38%. However, higher rates of IED have been noted in BPD subjects (78%) and in subjects with antisocial personality disorder (58%) (Coccaro et al. 1998a). Regardless, BPD and antisocial personality disorder subjects with a comorbid diagnosis of IED do appear to have higher scores for aggression and lower scores for general psychosocial function than do BPD and antisocial personality disorder subjects without IED (Coccaro et al. 2005).

TABLE 19–3. DSM-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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Pathogenesis

Family and Twin Studies

Familial aggregation of temper outbursts and IED has been reported in psychiatric patients with "temper problems" (Mattes and Fink 1987), and McElroy et al. (1998) reported that nearly a third of first-degree relatives of IED probands had IED.

Biological Correlates

Measures examining central (as well as peripheral) serotonin function correlate inversely with life history, questionnaire, and laboratory measures of aggression. The type of aggression associated with reduced central serotonin function appears to be *impulsive*, as opposed to *nonimpulsive*, aggression (Linnoila et al. 1983; Virkkunen et al. 1994).

Evidence also supports the role of other nonserotonergic brain systems and modulators in impulsive aggression. These findings suggest a facilitating role for dopamine (DePue et al. 1994), norepinephrine (Coccaro et al. 1991), vasopressin (Coccaro et al. 1998b), brain-derived neurotrophic factor (Lyons et al. 1991), opiates (Post et al. 1984), and testosterone (Giammanco et al. 2005; Virkkunen et al. 1994) and an inhibitory interaction between neuronal nitric oxide synthase and testosterone in rodents (Kriegsfeld et al. 1997).

Course

According to DSM-IV-TR, the onset appears to be from childhood to the early 20s. The course of IED is variable, with an episodic course in some and a more chronic course in others.

Treatment

Pharmacotherapy

Several medications have been used to treat impulsive aggression, such as tricyclic antidepressants, benzodiazepines, mood stabilizers, and neuroleptics. Recently, pharmacotherapy studies of aggression have turned to SSRIs and mood stabilizers as first-line treatments. In a treatment trial of subjects meeting Integrated Research Criteria for IED, impulsive aggressive behaviour did respond to fluoxetine (Coccaro and Kavoussi 1997), but non-serotonin-specific antidepressants had little benefit for impulsive aggression and many side effects in treatment studies. Soloff et al. (1986a) found that affective symptoms improved with amitriptyline in some BPD and schizotypal personality disorder inpatients, but impulsivity and aggression worsened in a set of patients, perhaps because of the noradrenergic effects of tricyclic antidepressants (Links et al. 1990).

Soloff et al. (1993) found that compared with placebo and haloperidol, phenelzine produced a moderate reduction in anger and hostility in BPD patients. Yet in a 16-week continuation phase, the subjects had experienced only minor benefits in depression and irritability and remained substantially impaired after the treatment phase (Cornelius et al. 1993; Soloff et al. 1993). In a double-blind crossover trial (Cowdry and Gardner 1988), treatment-resistant BPD patients with a history of impulsive aggression showed improvement with tranylcypromine, carbamazepine (decreased severity of behavioural dyscontrol), and trifluoperazine but had an increase in the severity and frequency of the episodes of serious dyscontrol with alprazolam.

Links et al. (1990) found that objective ratings of anger and suicidality in BPD outpatients improved the most on lithium compared with desipramine and placebo, but subjects and their clinicians did not report any improvement in mood. Sheard et al. (1976) found an improvement with lithium compared with placebo in chronically aggressive prisoners. Barratt et al. (1997) also reported a reduction in aggression with phenytoin in impulsive aggressive prisoners.

In the Cowdry and Gardner (1988) study, carbamazepine lessened episodes of impulsive aggression in BPD subjects, but 18% of the subjects had a worsening of mood that improved once carbamazepine was stopped. Kavoussi and Coccaro (1998) and Hollander et al. (2003) reported an antiaggressive effect of divalproex sodium in IED subjects with a Cluster B personality disorder. Given the relative adverse event profiles for SSRIs versus mood stabilizers, it is likely that clinical treatment of IED should start with SSRIs unless the subject is extremely aggressive or has a history of a bipolar disorder, in which case treatment with a mood stabilizer would be more appropriate.

Cowdry and Gardner's (1988) subjects showed significant improvement in depression and anxiety objective ratings with trifluoperazine. Trifluoperazine was seen as less useful than tranylcypromine (a monoamine oxidase inhibitor) and carbamazepine in improving behaviour and affect among subjects. Soloff et al. (1986b, 1989) found that BPD inpatients improved on hostility and global function measurements with haloperidol, but considerable depression remained. Montgomery and Montgomery (1982) found that suicidal and parasuicidal behaviour, in subjects with a history of such behaviours, decreased in 4a depot flupenthixol treatment group. Zanarini and Frankenburg (2001) compared the atypical antipsychotic olanzapine with placebo in outpatients with BPD. The treatment improved anger, hostility, and other symptoms but did not improve depression.

Psychotherapy

Psychotherapy for anger and aggression focuses on cognitive-behavioural group therapy. In a few rare cases, anger is addressed as the primary or only problem, and a limited number of treatments have been described. Imaginal exposure therapy, used frequently in anxiety disorders, was studied in a noncontrolled pilot study of anger treatment (Grodnitzky and Tafrate 2000). Subjects habituated to anger-provoking scenarios, and the treatment was believed to be useful.

Other versions of cognitive-behavioural therapy (CBT), such as dialectical behaviour therapy, have been studied in BPD patients. One study showed improvement in anger, social adjustment, and global functioning compared with a treatment-as-usual condition (Linehan et al. 1994). Improvement in anger and impulsivity has been shown with dialectical behaviour therapy across many disorders.

Kleptomania

Definition and Diagnostic Criteria

The DSM-IV-TR criteria for kleptomania are listed in Table 19–4.

TABLE 19–4. DSM-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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Patients with kleptomania often report amnesia surrounding the act of shoplifting (Goldman 1991; Grant 2004) and deny feelings of tension or arousal prior to shoplifting and feelings of pleasure or relief after the thefts. Other patients, who are not amnestic for the thefts, describe shoplifting as "automatic" or "a habit" and also may deny feelings of tension prior to a theft or pleasure after the act, although they report an inability to control their shoplifting.

Epidemiology

A study in the United States of 204 adult psychiatric inpatients with multiple disorders found that 7.8% endorsed current symptoms consistent with a diagnosis of kleptomania and 9.3% had a lifetime diagnosis of kleptomania (Grant et al. 2005). Kleptomania appeared equally common in patients with mood, anxiety, substance use, or psychotic disorders. Most patients with kleptomania are women (Grant and Kim 2002b; McElroy et al. 1991b; Presta et al. 2002). The severity of kleptomania symptoms and the clinical presentation of symptoms do not appear to differ based on gender (Grant and Kim 2002b).

Comorbidity

Rates of lifetime comorbid affective disorders range from 59% (Grant and Kim 2002b) to 100% (McElroy et al. 1991b). The rate of lifetime comorbid bipolar disorder has been reported as ranging from 9% (Grant and Kim 2002b) to 27% (Bayle et al. 2003) to 60% (McElroy et al. 1991b). Studies also have found high lifetime rates of comorbid anxiety disorders (60%–80%; McElroy et al. 1991b, 1992), other ICDs (20%–46%; Grant and Kim 2003), substance use disorders (23%–50%; Grant and Kim 2002b; McElroy et al. 1991b), and eating disorders (60%; McElroy et al. 1991b). Personality disorders have been found in 43%–55% of patients with kleptomania, the most common being paranoid personality disorder and histrionic personality disorder (Bayle et al. 2003; Grant 2004).

Pathogenesis

Biological Theories

Serotonin and inhibition. The most well-studied inhibitory pathways involve serotonin and the prefrontal cortex (Chambers et al. 2003). Decreased measures of serotonin have long been associated with a variety of adult risk-taking behaviours, including alcoholism, fire setting, and pathological gambling (Moreno et al. 1991; Virkkunen et al. 1994). Blunted serotonergic responses in the ventromedial prefrontal cortex have been seen in people with impulsive aggression (New et al. 2002), and this region also has been implicated in poor decision making (Bechara 2003), as seen in those with kleptomania.

Dopamine and reward deficiency. Alterations in dopaminergic pathways have been proposed as underlying the seeking of rewards (e.g., shoplifting) that triggers the release of dopamine and produces feelings of pleasure (Blum et al. 2000). Furthermore, dopamine release into the nucleus accumbens has been implicated in the translation of motivated drive into action, serving as a "go" signal (Chambers et al. 2003). Dopamine release into the nucleus accumbens seems maximal when reward probability is most uncertain, suggesting that it plays a central role in guiding behaviour during risk-taking situations (Fiorillo et al. 2003). The structure and function of dopamine neurons within the nucleus accumbens, in conjunction with glutamatergic afferent and intrinsic γ-aminobutyric acid (GABA)–ergic activities, appear to change in response to experiences that influence the function of the nucleus accumbens.

Opioid system, cravings, and pleasure. The μ-opioid system is thought to underlie urge regulation by processing reward, pleasure, and pain at least in part via modulation of dopamine neurons in the mesolimbic pathway through GABA interneurons (Potenza and Hollander

2002). Studies of naltrexone, a μ-opioid antagonist, have shown its efficacy in reducing urges in those with kleptomania and other ICDs (Dannon et al. 1999; Grant and Kim 2002c; Kim et al. 2001).

Psychological Theories

Kleptomania may result from an attempt to relieve feelings of depression through stimulation (Goldman 1991; McElroy et al. 1991a). Risk-taking behaviour may produce an antidepressant effect for some patients (Fishbain 1987; Goldman 1991). Shoplifting may distract depressed patients from stressors and unpleasant cognitions. Ironically, problems resulting directly from shoplifting (e.g., embarrassment and shame from getting caught) may lead to even more shoplifting as a misguided means of symptom management (Goldman 1991).

From an operant viewpoint, the positive reinforcer in kleptomania is the acquisition of items for nothing, and the intermittent reinforcement of kleptomanic behaviour may therefore be particularly resistant to extinction. Physiological arousal related to shoplifting (Goldman 1991) may be another reinforcer that initiates and perpetuates the behaviour.

Course

Most patients have an onset of symptoms before age 21 (Goldman 1991; Grant and Kim 2002b; McElroy et al. 1991a, 1991b; Presta et al. 2002).

Treatment

Pharmacotherapy

Various medications—tricyclic antidepressants, SSRIs (Lepkifker et al. 1999), mood stabilizers, and opioid antagonists—have been examined for the treatment of kleptomania (Grant and Kim 2002c; McElroy et al. 1989). McElroy et al. (1991b) reported treatment response in 10 of 20 patients with the following single agents: fluoxetine, nortriptyline, trazodone, clonazepam, valproate, and lithium. Other agents used successfully as monotherapy for kleptomania include fluvoxamine (Chong and Low 1996) and paroxetine (Kraus 1999). Combinations of medications also have been effective in case reports: lithium plus fluoxetine (Burstein 1992), fluvoxamine plus buspirone (Durst et al. 1997), fluoxetine plus lithium, fluoxetine plus imipramine (McElroy et al. 1991b), and fluvoxamine plus valproate (Kmetz et al. 1997).

In an open-label medication trial for kleptomania, naltrexone (mean effective dosage, 145 mg/day) resulted in a significant decline in the intensity of urges to steal, stealing thoughts, and stealing behaviour (Grant and Kim 2002c). A lower dosage, possibly 50 mg/day, may be effective in younger people with kleptomania (Grant and Kim 2002a). A double-blind, placebo-controlled study of naltrexone (mean effective dosage, 116.7±44.4 mg/day) also demonstrated statistically significant reductions in stealing urges and behaviour in kleptomania (Grant et al. 2009).

Psychotherapy

Imaginal desensitization uses the idea of imagining the steps of stealing while maintaining a relaxed state. The patient then imagines the potential scene of stealing but also imagines his or her ability to not steal in that context.

Pyromania

Definition and Diagnostic Criteria

The essential feature of pyromania is multiple deliberate and purposeful (rather than accidental) fire setting (Table 19–5).

TABLE 19–5. DSM-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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Another important clinical feature of pyromania is the fascination with fire. People with pyromania are prone to being "watchers" at fires in their neighbourhoods, setting off false fire alarms, or working or volunteering as firefighters.

Epidemiology

Lewis and Yarnell (1951), in their classic study *Pathological Firesetting (Pyromania)*, found that of those arrested for fire setting, 39% did not have a profit motive and were diagnosed with pyromania.

Comorbidity

Comorbid conditions with pyromania often include substance use disorders, mental retardation, conduct disorder, mania, schizophrenia, and antisocial personality disorder.

Pathogenesis

Biological Markers

Virkkunen et al. (1987, 1994) suggested that pyromania may be associated with reactive hypoglycemia and/or lower concentrations of 3-methoxy-4-hydroxyphenylglycol (MHPG) and cerebrospinal fluid 5-hydroxyindoleacetic acid (5-HIAA).

Course

In individuals with pyromania, fire-setting incidents are episodic and may wax and wane in frequency. Studies indicate that the recidivism rate for fire setters ranges from 4.5% (Mavromatis and Lion 1977) to 28% (Lewis and Yarnell 1951).

Treatment

The treatment of pyromania is summarized in Table 19–6.

Pathological Gambling

Definition and Diagnostic Criteria

The essential feature of pathological gambling is recurrent gambling behaviour that is maladaptive (e.g., loss of judgment, excessive gambling) and in which personal, family, or vocational endeavours are disrupted (Table 19–7).

Epidemiology

A meta-analysis of 120 published studies indicated that the lifetime prevalence of serious gambling (meeting DSM criteria for pathological gambling) among adults is 1.6% (Shaffer et al. 1999).

Comorbidity

Three Axis I disorders frequently co-occur with pathological gambling: substance abuse or dependence, bipolar spectrum disorders, and attention-deficit/hyperactivity disorder. Dopamine agonists, in particular pramipexole, have been implicated in the development of compulsive gambling and other excessive behaviour patterns (Dodd et al. 2005). Results of a large cross-sectional study of 3,090 patients with treated idiopathic Parkinson's disease at 46 movement disorder centres in Canada and the United States showed that ICDs, including behaviours such as compulsive gambling, occur in about 14% of all patients with Parkinson's disease (Weintraub et al. 2010). That number increases to about 17% of those patients taking a dopamine agonist.

Pathogenesis

Neurobiology

There is evidence of serotonergic, noradrenergic, and dopaminergic dysfunction in the pathogenesis of pathological gambling (Table 19–8).

Genetics

At present, the main source of evidence for the genetic influence in the etiology of pathological gambling derives from a study of 3,359 male twin pairs from the Vietnam Era Twin Registry cohort (Eisen et al. 1998, 2001; Slutske et al. 2000). These data suggest that gambling problems of increasing severity represent a single continuum of vulnerability rather than distinct entities (Eisen et al. 1998, 2001), indicate genetic susceptibility in pathological gambling (Eisen et al. 1998), and suggest a common genetic vulnerability for pathological gambling and alcohol dependence in men (Slutske et al. 2000).

Course

The course of pathological gambling tends to be chronic, but the pattern of gambling may be regular or episodic. Chronicity is usually associated with increases in the fre-

TABLE 19–6. Pyromania: treatment summary

Authors	Treatment	Description
McGrath and Marshall 1979	Behavioural therapy	Child fire setter ($N=1$); successful
Koles and Jenson 1985	Behavioural therapy	Child fire setter ($N=1$); successful
Bumpass et al. 1983	Technique that sequentially correlates external stress, behaviour, and feelings on graph paper to help patients become aware of the cause-effect relation between feelings and behaviour so as to substitute an acceptable behaviour	Child fire setters ($N=29$); after treatment (average follow-up, 2.5 years), only 2 of the 29 children continued to set fires
Franklin et al. 2002	Trauma Burn Outreach Prevention Program (TBOPP), 1-day interactive program focusing on the medical, financial, legal, and societal impact of fire setting, emphasizing individual accountability and responsibility	132 juveniles (66 arsonists, 66 fire setters) in the TBOPP group; 102 juveniles (33 arsonists and 66 fire setters) in the no-TBOPP group; TBOPP participants had essentially no recidivism compared with the no-TBOPP group

quency of gambling and the amount gambled. Gambling may increase during periods of increased stress.

Psychiatric disorders such as major depression and alcohol or substance abuse and dependence may develop from or be exacerbated by pathological gambling. Estimates of suicide attempts in pathological gamblers range from 17% to 24% (Ciarrochi and Richardson 1989; Hollander et al. 2000a).

In males, the disorder usually begins in adolescence (Hollander et al. 2000a) and may remain undiagnosed for years; male pathological gamblers often present with a 20- to 30-year gambling history, with gradual development of dependence. In contrast, onset of pathological gambling in females is more likely to occur later in life. Prior to their seeking treatment, the duration of pathological gambling in women is approximately 3 years.

Treatment

Pharmacotherapy

Pharmacological treatment studies of pathological gambling have had some promising results with the use of serotonin reuptake inhibitors (de la Gandara 1999; Hollander et al. 1992, 1998, 2000b; Kim et al. 2002; Zimmerman et al. 2002), serotonin antagonists (Pallanti et al. 2002a), mood stabilizers (Haller and Hinterhuber 1994; Hollander et al. 2002; Pallanti et al. 2002b), opiate antagonists (Kim et al. 2001), and atypical antipsychotics (Potenza and Chambers 2001).

Psychotherapy

The most popular intervention for problem gambling is Gamblers Anonymous, which is similar to Alcoholics Anonymous and Narcotics Anonymous. However, evidence suggests that Gamblers Anonymous may not be very effective when used without other treatment modalities (Petry and Armentano 1999). Retrospective studies show a dropout rate of up to 70% within the first year (Stewart and Brown 1988), and overall dropout rates range from 75% to 90% (Moody 1990). Only 8% of members report total abstinence at 1-year follow-up and 7% at 2-year follow-up (Brown 1985).

Inpatient programs for pathological gambling have included various combinations of individual and group psychotherapy and substance use treatment (Taber 1981), and most of these strongly encouraged or required attendance at Gamblers Anonymous meetings. Many patients improved in all programs, and outcome studies have shown 55% of patients reporting abstinence at 1-year follow-up (Russo et al. 1984; Taber et al. 1987).

Behavioural, cognitive, and combined cognitive-behavioural methods have been used in treating pathological gambling. Aversive therapy has been used to reach the goal of total abstinence of gambling, as have behaviour monitoring, contingency management, contingency contracting, covert sensitization, systematic desensitization, imaginal desensitization, in vivo exposure, imaginal relaxation, psychoeducation, cognitive restructuring, problem-solving skills training, social skills training, and relapse prevention (Ladouceur 1990).

TABLE 19–7. DSM-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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TABLE 19–8. Developmental and neurobiological model of pathological gambling

Vulnerable state

Primed genetically/neurobiologically

Repeated environmental exposure

Gambling cycle: behavioural mechanisms

Stimulation readiness → norepinephrine

Behavioural initiation → serotonin

Reward/reinforcement → dopamine

Behavioural disinhibition → serotonin

Source. Reprinted from Pallanti S, Baldini Rossi N, Hollander E: "Pathological Gambling," in *Clinical Manual of Impulse-Control Disorders*. Edited by Hollander E, Stein DJ. Washington, DC, American Psychiatric Publishing, 2006, p. 262. Used with permission.

TABLE 19–9. DSM-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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Trichotillomania

Definition and Diagnostic Criteria

Trichotillomania is a chronic ICD characterized by repetitive pulling out of one's own hair, resulting in noticeable hair loss. The DSM-IV-TR criteria for trichotillomania are listed in Table 19–9.

Epidemiology

In studies involving college samples, 10%–13% of students reported hair pulling, with the prevalence of clinically significant pulling ranging between 1% and 3.5% (Christenson et al. 1991a; Rothbaum et al. 1993).

Comorbidity

Christenson et al. (1991b) found that approximately 82% of an adult sample with trichotillomania met criteria for a past or current comorbid Axis I disorder, the most common

being mood, anxiety, and addictive disorders. Of the patients with comorbid disorders, there was a lifetime prevalence rate of 65% for mood disorders, 57% for anxiety disorders, 22% for substance abuse disorders, 20% for eating disorders, and 42% for personality disorders. The most frequently cited comorbid personality disorders are histrionic, borderline, and obsessive-compulsive (Christenson et al. 1992; Schlosser et al. 1994; Swedo and Leonard 1992).

A key debate in the field is whether trichotillomania should be conceptualized as an ICD or a variant of obsessive-compulsive disorder (OCD). In support of the classification as an obsessive-compulsive spectrum disorder is the apparent similarity between compulsions and the repetitive and perceived uncontrollable nature of hair pulling and accompanying anxiety relief (Swedo 1993; Swedo and Leonard 1992), the possible selective responsiveness of trichotillomania to serotonin reuptake inhibitors, and the elevated rates of OCD in patients with trichotillomania (Christenson et al. 1991a).

Pathogenesis

Biological Vulnerability

Familial research suggests that trichotillomania may be associated with increased rates of OCD or other excessive habits among first-degree relatives (Bienvenu et al. 2000; King et al. 1995).

Hair-Pulling Cues

The behavioural model of hair pulling suggests that pulling begins as a normal response to stress but eventually becomes associated with a variety of internal and external cues through conditioning (Mansueto et al. 1997).

Reinforcement

Hair pulling is often preceded by negative internal states such as unpleasant emotions, aversive physiological sensations, or dysregulated arousal. Hair pulling appears to result in a decrease of these states. Over time, hair-pulling urges that are reinforced by pulling lead to stronger urges to pull, which perpetuates the behavioural cycle. Trichotillomania patients report retrospectively that pulling leads to reduced feelings of tension, boredom, and anxiety, and nonclinical hair pullers also report reductions in sadness and anger (Stanley et al. 1995).

Course

Age at onset usually ranges from early childhood to young adulthood. Initial onset after young adulthood is uncommon.

Trichotillomania in adolescents and adults typically follows a chronic course, involves multiple hair sites, and is associated with high rates of psychiatric comorbidity (Christenson et al. 1991a).

Treatment

Pharmacotherapy

Results from controlled studies of serotonin reuptake inhibitors are equivocal at best, although in view of the small sample sizes, more controlled research should be conducted to determine the efficacy of these medications more definitively (Christenson et al. 1991b; Ninan et al. 2000; Streichenwein and Thornby 1995; Swedo et al. 1989, 1993; van Minnen et al. 2003). However, several case studies indicated that augmentation of SSRIs with atypical antipsychotics may be beneficial (Epperson et al. 1999).

Psychotherapy

With respect to behavioural approaches and CBT, a variety of specific techniques have been applied, including awareness training, self-monitoring, aversion, covert sensitization, negative practice, relaxation training, habit reversal, competing response training, stimulus control, and overcorrection.

Key Points: Impulse-Control Disorders

- Pathological impulsivity is a useful construct in understanding a broad range of psychiatric symptoms and disorders, including the ICDs not otherwise specified.
- ICDs are highly prevalent and associated with significant disability and costs but receive disproportionately little attention from clinicians and researchers.
- There are now structured diagnostic instruments and standardized rating scales that allow reliable diagnosis and assessment of the ICDs.

- There have been significant advances in our understanding of the neuronal circuitry that mediates impulsivity, as well as in the delineating of the contributing genes and proteins in this circuitry.
- Ultimately, a better understanding of the psychobiological underpinnings of impulsivity, behaviour addiction, and other related constructs may lead to changes in our classification of these disorders.
- Although no medication is registered for the treatment of ICDs, a number of randomized controlled trials have demonstrated the potential value of pharmacotherapy.
- Current clinical practice also emphasizes the need for a comprehensive approach to management that includes psychotherapy and family intervention. Additional work is needed to improve efficacy.

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