# 000 Impulse-Control Disorders

Daphne Simeon[1]
Heather Berlin[2]

[1,2]Department of Psychiatry, Mount Sinai School of Medicine, New York, NY, USA

## Introduction

Although dissimilar in behavioral expressions, the disorders in this chapter share the feature of impulse dyscontrol. Individuals who experience such dyscontrol are overwhelmed by the urge to commit certain acts that are often apparently illogical or harmful (McElroy et al. 1992). Whereas impulse-control disorders (ICDs) were once conceptualized as either addictive or compulsive behaviors, they are now classified within the DSM-IV-TR (American Psychiatric Association 2000) ICD category. These include intermittent explosive disorder (IED) (failure to resist aggressive impulses), kleptomania (failure to resist urges to steal items), pyromania (failure to resist urges to set fires), pathological gambling (failure to resist urges to gamble), and trichotillomania (failure to resist urges to pull one's hair). It should be noted that behaviors characteristic of these disorders may be notable in individuals as symptoms of another mental disorder. If the symptoms progress to such a point that they occur in distinct, frequent episodes and begin to interfere with the person's normal functioning, they may then be classified as a distinct ICD.

There are also a number of other disorders that are not included as a distinct category but are categorized as ICDs not otherwise specified in DSM-IV-TR. These include sexual compulsions (impulsive-compulsive sexual behavior), compulsive shopping (impulsive-compulsive buying disorder), skin picking (impulsive-compulsive psychogenic excoriation), and internet addiction (impulsive-compulsive computer usage disorder). These disorders are unique in that they share features of both impulsivity and compulsivity and might be labeled as ICDs. Patients with these disorders engage in the behavior to increase arousal. However, there is a compulsive component in which the patient continues to engage in the behavior to decrease dysphoria.

To properly conceptualize ICDs, it is helpful to understand the role of impulsivity within them. The trait of impulsivity has been the subject of increasing interest in psychiatry. Impulsivity is a defining characteristic of many psychiatric illnesses, even those not classified as ICDs, including Cluster B personality disorders like borderline personality disorder (BPD) and antisocial personality disorder (ASPD), neurological disorders characterized by disinhibited behavior, attention-deficit/hyperactivity disorder (ADHD), substance and alcohol abuse, conduct disorder, binge eating, bulimia, and paraphilias. Impulsivity research has been conducted both in disorders characterized by impulsivity, like BPD, ASPD, and conduct disorder, and in traditional ICDs like IED. Clinicians should recognize that individuals who are prone to impulsivity and ICDs are often afflicted with a cluster of related conditions including sexual compulsions, substance use disorders, and post-traumatic stress disorder and to screen for comorbid conditions such as bipolar spectrum disorders and ADHD that contribute to impulsivity (Figure 000–1).

Impulsivity is the failure to resist an impulse, drive, or temptation that is potentially harmful to oneself (e.g. trichotillomania, pathological gambling) or others (e.g. IED, pyromania, kleptomania) is a common clinical problem and a core feature of human behavior. An impulse is rash and lacks deliberation. It may be sudden and ephemeral, or a steady rise in tension that may reach a climax in an explosive expression of the impulse, which may result in careless actions without regard to the consequences to self or others. Impulsivity is evidenced behaviorally as an underestimated sense of harm, carelessness, extroversion, impatience, including the inability to delay gratification, and a tendency toward risk taking, and sensation seeking (Hollander et al. 2002). What makes an impulse pathological is the person's inability to resist it and its expression.

New research findings associate various forms of impulsive behavior with biological markers of altered serotonergic function. These include impulsive suicidal behavior, impulsive aggression, and impulsive fire setting (Stein et al. 1993). In all these circumstances, impulsivity is conceived of as the rapid expression of unplanned behavior, occurring in response to a sudden thought. (This is seen by some as the polar opposite of obsessional behavior, in which deliberation over an act may seem never ending.) Although the sudden and unplanned aspect of the behavior may be present in the impulse disorders (such as in IED and kleptomania),
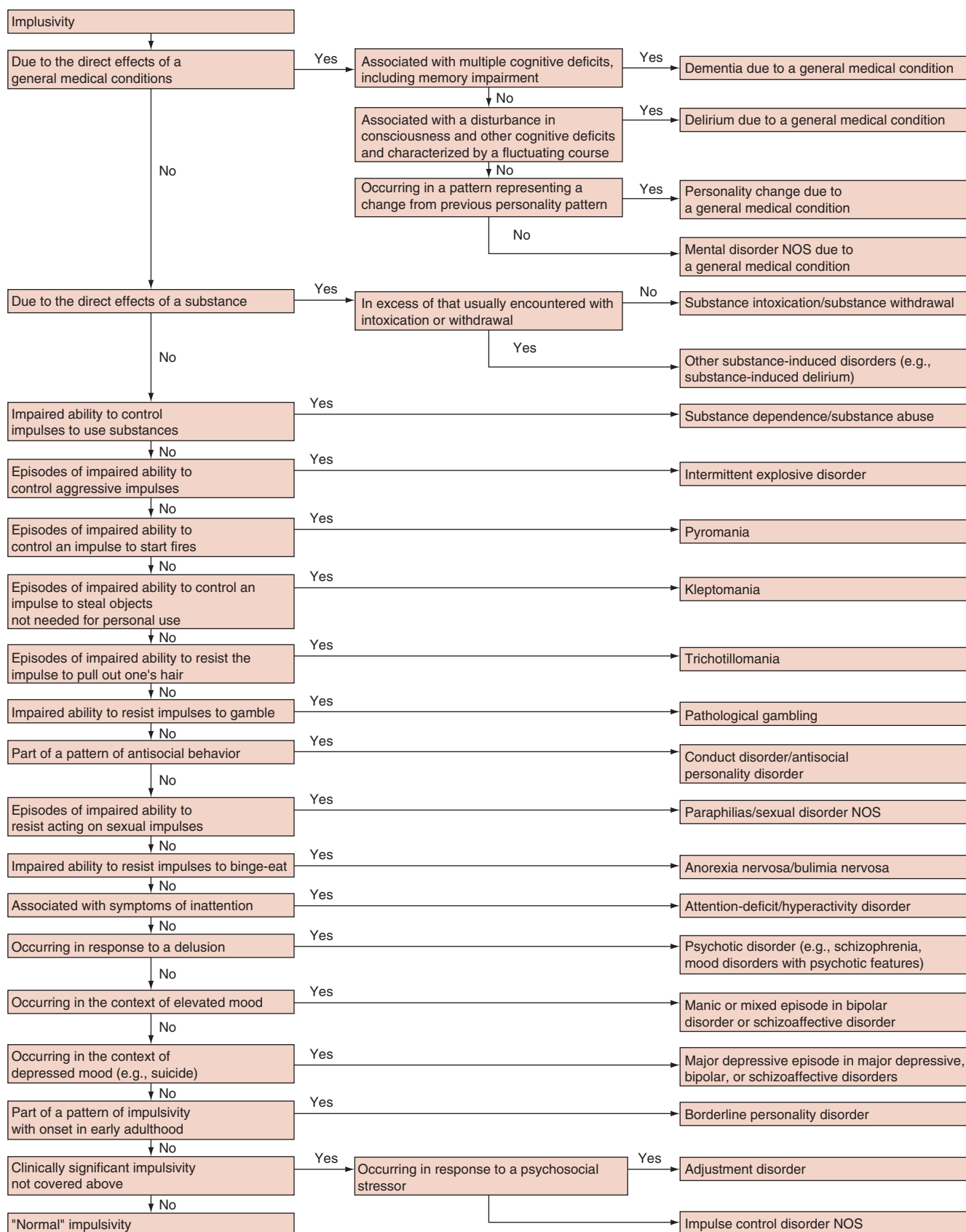
| | | |
|---|---|---|
| Implusivity | | |

Due to the direct effects of a general medical conditions — **Yes** → Associated with multiple cognitive deficits, including memory impairment — **Yes** → Dementia due to a general medical condition

— **No** → Associated with a disturbance in consciousness and other cognitive deficits and characterized by a fluctuating course — **Yes** → Delirium due to a general medical condition

— **No** → Occurring in a pattern representing a change from previous personality pattern — **Yes** → Personality change due to a general medical condition

— **No** → Mental disorder NOS due to a general medical condition

**No** ↓

Due to the direct effects of a substance — **Yes** → In excess of that usually encountered with intoxication or withdrawal — **No** → Substance intoxication/substance withdrawal

— **Yes** → Other substance-induced disorders (e.g., substance-induced delirium)

**No** ↓

Impaired ability to control impulses to use substances — **Yes** → Substance dependence/substance abuse

**No** ↓

Episodes of impaired ability to control aggressive impulses — **Yes** → Intermittent explosive disorder

**No** ↓

Episodes of impaired ability to control an impulse to start fires — **Yes** → Pyromania

**No** ↓

Episodes of impaired ability to control an impulse to steal objects not needed for personal use — **Yes** → Kleptomania

**No** ↓

Episodes of impaired ability to resist the impulse to pull out one's hair — **Yes** → Trichotillomania

**No** ↓

Impaired ability to resist impulses to gamble — **Yes** → Pathological gambling

**No** ↓

Part of a pattern of antisocial behavior — **Yes** → Conduct disorder/antisocial personality disorder

**No** ↓

Episodes of impaired ability to resist acting on sexual impulses — **Yes** → Paraphilias/sexual disorder NOS

**No** ↓

Impaired ability to resist impulses to binge-eat — **Yes** → Anorexia nervosa/bulimia nervosa

**No** ↓

Associated with symptoms of inattention — **Yes** → Attention-deficit/hyperactivity disorder

**No** ↓

Occurring in response to a delusion — **Yes** → Psychotic disorder (e.g., schizophrenia, mood disorders with psychotic features)

**No** ↓

Occurring in the context of elevated mood — **Yes** → Manic or mixed episode in bipolar disorder or schizoaffective disorder

**No** ↓

Occurring in the context of depressed mood (e.g., suicide) — **Yes** → Major depressive episode in major depressive, bipolar, or schizoaffective disorders

**No** ↓

Part of a pattern of impulsivity with onset in early adulthood — **Yes** → Borderline personality disorder

**No** ↓

Clinically significant impulsivity not covered above — **Yes** → Occurring in response to a psychosocial stressor — **Yes** → Adjustment disorder

— **No** → Impulse control disorder NOS

**No** ↓

"Normal" impulsivity

**Figure 000–1** *Differential diagnosis of impulsivity. Impulsivity is a tendency to act in a sudden, unpremeditated, and excessively spontaneous fashion. Other decision trees that should be considered are those for aggressive behavior, catatonia, delusions, depressed mood, euphoric or irritable mood, disorganized or unusual behavior, distractibility, eating behavior, self-mutilation, and suicide ideation or attempt. (NOS, not otherwise specified.)*

| Table 000–1 | Differential Diagnosis of Intermittent Explosive Disorder |
|---|---|
| IED Must Be Differentiated from Aggressive Behavior in | In Contrast to IED, the Other Condition |
| Substance intoxication or withdrawal | Is due to the direct physiological effects of a substance |
| Delirium or dementia (substance induced or due to a general medical condition) | Includes characteristic symptoms (e.g., memory impairment, impaired attention) |
|  | Requires the presence of an etiological general medical condition or substance use |
| Personality change due to a general medical condition, aggressive type | Requires presence of an etiological general medical condition |
| Conduct disorder or ASPD | Is characterized by more general pattern of antisocial behavior |
| Other mental disorders (schizophrenia, manic episode, oppositional defiant disorder, BPD) | Includes the characteristic symptoms of the other mental disorder |

*Source*: [Reproduced from First and Frances A (1995) with permission of American Psychiatric Press.]

the primary connotation of the word impulsivity, as used to describe these conditions, is the irresistibility of the urge to act. In DSM-IV-TR, ICDs are characterized by five stages of symptomatic behavior (Table 000–1). First is the increased sense of tension or arousal, followed by the failure to resist the urge to act. Third, there is a heightened sense of arousal. Once the act has been completed, there is a sense of relief from the urge. Finally, the patient experiences guilt and remorse at having committed the act. In conditions like trichotillomania, pyromania, and pathological gambling, the individual may spend considerable amounts of time fighting off the urge, trying not to carry out the impulse. The inability to resist the impulse is the common core of these disorders, rather than the rapid transduction of thought to action. A decision tree for the differential diagnosis of impulsive behaviors may be seen in Figure 000–1.

Other than sharing the essential feature of impulse dyscontrol, it is unclear whether the conditions in this chapter bear any relationship to each other. Emerging perspectives on the neurobiology of impulsivity suggest that impulsive behaviors, across diagnostic boundaries, may share an underlying pathophysiological diathesis. As noted earlier, markers of altered serotonergic neurotransmission have been associated with a variety of impulsive behaviors: suicidality, aggressive violence, pyromania, and conduct disorder. These observations have led to speculation that decreased serotonergic neurotransmission may result in decreased ability to control urges to act. In accord with this model, these disorders may be varying expressions of a single disturbance—or closely related disturbances—of serotonergic function. Although such markers of altered serotonergic function have been demonstrated among impulsive fire setters and impulsive violent offenders, there is, as yet, insufficient research on these conditions to accept or dismiss this theory.

It has been noted that these conditions are embedded in similar patterns of comorbidity with other psychiatric disorders. High rates of comorbid mood disorder and anxiety disorder appear typical of these disorders. This contextual similarity, combined with the common feature of impulsivity, may further support the notion that these conditions are—at the level of core diathesis—related to each other.

Although these conditions have historically been considered uncommon, later investigations suggest that some of them may be fairly common. Trichotillomania, for example,

was once considered rare. However, surveys indicate that the lifetime prevalence of the condition may exceed 1% of the population. Pathological gambling may be present in up to 3% of the population. Extrapolation from the known incidence of comorbid conditions suggests that kleptomania may have a 0.6% incidence. It would seem reasonable to suspect that individuals with pyromania and kleptomania may seek to avoid detection and may therefore be underrepresented in research and clinical samples.

Few treatment studies of these specific conditions have been conducted. Attempts to treat these conditions are usually formulated by extrapolation from treatments that have been developed for other conditions. The treatment literature for most of these conditions reflects the general development of psychiatric theory. Papers from the early part of the 20th century are largely restricted to reports of the psychoanalytic treatment of individual cases or of small series. The aggressive quality of kleptomania, pyromania, and IED and the self-damaging nature of trichotillomania and pathological gambling have presented tempting substrates for the application of traditional analytical concepts. From this perspective, these behaviors have been seen as symptomatic expressions of unconscious conflict, often sexual in nature. Other formulations include desires for oral gratification and masochistic wishes to be caught and punished, motivated by a harsh, guilt-inducing superego. The increasing influence of object relations theory was reflected in increasing emphasis on narcissistic psychopathology and histories of disturbed early parenting. As successful behavioral interventions were developed for other conditions, case reports of behavioral treatments for these conditions emerged. Reports of hypnotic treatments are also prominent in the literature.

As pharmacological treatments are applied to an increasing range of symptoms, the ICDs in this chapter present new opportunities to widen the application of thymoleptic and anxiolytic and, more recently, atypical neuroleptic medication. As part ongoing evolving theory, the very concept of impulsivity is still in ferment. Attempts to further refine the idea of impulsivity are reflected in a perspective offered by Van Ameringen et al. (1999).

Trichotillomania, IED, and pathological gambling have become the focus of increasing interest of late. However, kleptomania and pyromania remain stepchildren of research. Perhaps the legal implications of these behaviors and their entanglement with similar—but not impulsively

motivated—behaviors complicate the availability of sufficient cases to facilitate research. Because of the limited body of systematically collected data, the following sections largely reflect accumulated clinical experience. Therefore, the practicing psychiatrist should be particularly careful to consider the exigencies of individual patients in applying treatment recommendations.

## Intermittent Explosive Disorder

### Diagnosis

### Definition and Diagnostic Features

IED is a DSM diagnosis used to describe people with pathological impulsive aggression (see box for diagnostic criteria). Impulsive aggression, however, is not specific to IED. It is a key feature of several psychiatric disorders and nonpsychiatric conditions and may emerge during the course of yet other psychiatric disorders. Therefore, the definition of IED as formulated in the DSM-IV-TR is essentially a diagnosis of exclusion. As described in criterion C, a diagnosis of IED is made only after other mental disorders that might account for episodes of aggressive behavior have been ruled out. The individual may describe the aggressive episodes as "spells" or "attacks." The symptoms appear within minutes to hours and, regardless of the duration of the episode, may remit almost as quickly. As in other ICDs, the explosive behavior may be preceded by a sense of tension or arousal and is followed immediately by a sense of relief or release of tension.

---

**DSM-IV-TR Criteria**

**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).

---

Reproduced with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Copyright 2000 American Psychiatric Association.

Although not explicitly stated in the DSM-IV-TR definition of IED, impulsive aggressive behavior may have many motivations that are not meant to be included within this diagnosis. IED should not be diagnosed when the purpose of the aggression is monetary gain, vengeance, self-defense, social dominance, or expressing a political statement or when it occurs as a part of gang behavior. Typically, the aggressive behavior is ego-dystonic to individuals with IED, who feel genuinely upset, remorseful, regretful, bewildered, or embarrassed about their impulsive aggressive acts. Because the essential feature of IED is the occurrence of serious assaultive acts or destruction of property, it is a diagnosis relevant to the interface between psychiatry and the law.

Many clinicians and researchers rarely consider the diagnosis of IED, although impulsive aggressive behavior is relatively common. 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 1992). Impulsive aggressive behavior is usually pathological and causes substantial psychosocial distress/dysfunction (McElroy et al. 1998). Being the recipient of impulsive aggressive behavior can lead to similar behavior in children who grow up in this environment (Huesmann et al. 1984).

Violence is underreported in Western societies. As discussed by Lion (1992) although violence is commonly encountered in clinical psychiatric practice, its diagnostic acknowledgment within psychiatry has always been problematical (McElroy et al. 1992). To a large extent, this difficulty also reflects history: Freud himself never fully developed a theory of aggression and came to consider the existence of a "primary" destructive drive only late in his life, after the death and devastation of World War I (*Beyond the Pleasure Principle*, published in 1920) (Freud 1955).

Episodes of violent behavior appear in several common psychiatric disorders such as ASPD, BPD, and substance use disorders and need to be distinguished from the violent episodes of patients with IED, which are apparently rare. The study of Felthous et al. (1991) in which 15 men with rigorously diagnosed DSM-III-R IED were identified from among a group of 443 men who complained of violence, permitted some systematic observations about the "typical violent episode" as reported by patients with IED.

In the vast majority of instances, the subjects with IED identified their spouse, lover, or girl/boy friend as a provocateur of their violent episodes. Only one was provoked by a stranger. For most, the reactions occurred immediately and without a noticeable prodromal period. Only one subject stated that the outburst occurred between 1 and 24 hours after the perceived provocation. All subjects with IED denied that they intended the outburst to occur in advance. Most subjects remained well oriented during the outbursts, although two claimed to lose track of where they were. None lost control of urine or bowel function during the episode. Subjects reported various degrees of subjective feelings of behavioral dyscontrol. Only four felt that they completely lost control. Six had good recollection of the event afterward, eight had partial recollection, and one lost memory of the event afterward. Most IED subjects tried to help or comfort the victim afterward.

### Assessment

### Psychiatric Examination and History

The DSM-IV-TR diagnosis of IED is essentially a diagnosis of exclusion, and the psychiatrist should evaluate and carefully rule out more common diagnoses that are associated with impulsive violence. The lifelong nonremitting

history of impulsive aggression associated with ASPD and BPD, together with other features of antisocial behavior (in ASPD) or impulsive behaviors in other spheres (in BPD) may distinguish them from IED, in which baseline behavior and functioning are in marked contrast to the violent outbursts. Other features of BPD such as unstable and intense interpersonal relationships, frantic efforts to avoid abandonment, and identity disturbance may also be elicited by a careful history. More than in most psychiatric diagnoses, collateral information from an independent historian may be extremely helpful, especially in forensic settings. Of note, patients with IED are usually genuinely distressed by their impulsive aggressive outbursts and may voluntarily seek psychiatric help to control them. In contrast, patients with ASPD do not feel true remorse for their actions and view them as a problem only insofar as they suffer their consequences, such as incarceration and fines. Although patients with BPD, like patients with IED, are often distressed by their impulsive actions, the rapid development of intense and unstable transference toward the psychiatrist during the evaluation period of patients with BPD may be helpful in distinguishing it from IED.

Other causes of episodic impulsive aggression are substance use disorders, in particular alcohol abuse and intoxication. When the episodic impulsive aggression is associated only with intoxication, IED is ruled out. However, IED and alcohol abuse may be related, and the diagnosis of one should lead the psychiatrist to search for the other.

Neurological conditions such as dementias, focal frontal lesions, partial complex seizures, and postconcussion syndrome after recent head trauma may all present as episodic impulsive aggression and need to be differentiated from IED. Other neurological causes of impulsive aggression include encephalitis, brain abscess, normal-pressure hydrocephalus, subarachnoid hemorrhage, and stroke. In these instances, the diagnosis would be personality change due to a general medical condition, aggressive type, and it may be made with a careful history and the characteristic physical and laboratory findings.

Individuals with IED may have comorbid mood disorders. Although the diagnosis of a manic episode excludes IED, the evidence for serotonergic abnormalities in both major depressive disorder and ICDs supports the clinical observation that impulsive aggression may be increased in depressed patients, leading ultimately to completed suicide.

### Physical Examination and Laboratory Findings

The physical and laboratory findings relevant to the diagnosis of IED and the differential diagnosis of impulsive aggression may be divided into two main groups: those associated with episodic impulsive aggression but not diagnostic of a particular disorder and those, which suggest the diagnosis of a psychiatric or medical disorder other than IED. No laboratory or physical findings are specific for IED.

The first group of findings that are associated with impulsive aggression across a spectrum of disorders includes soft neurological signs such as subtle impairments in hand-eye coordination and minor reflex asymmetries. These signs may be elicited by a comprehensive neurological examination and simple pencil-and-paper tests such as parts A and B of the Trail Making Test. Measures of central serotonergic function such as cerebrospinal fluid

(CSF) 5-hydroxyindoleacetic acid (5-HIAA) levels, the fenfluramine challenge test, and positron emission tomography (PET) of prefrontal metabolism also belong to this group. Although these measures advanced our neurobiological understanding of impulsive aggression, their utility in the diagnosis of individual cases of IED and other disorders with impulsive aggression is yet to be demonstrated.

The second group of physical and laboratory findings is useful in the diagnosis of causes of impulsive aggression other than IED. The smell of alcohol on a patient's breath or a positive alcohol reading with a Breathalyzer may help reveal alcohol intoxication. Blood and urine toxicology screens may reveal the use of other substances, and track marks on the forearms may suggest intravenous drug use. Partial complex seizures and focal brain lesions may be evaluated by EEG and brain imaging. In cases without a grossly abnormal neurological examination, magnetic resonance imaging may be more useful than computed tomography of the head. Magnetic resonance imaging can reveal mesiotemporal scarring, which may be the only evidence for a latent seizure disorder, sometimes in the presence of a normal or inconclusive EEG. Diffuse slowing on the EEG is a nonspecific finding that is probably more common in, but not diagnostic of, patients with impulsive aggression. Hypoglycemia, a rare cause of impulsive aggression, may be detected by blood chemistry screens.

### Epidemiology

IED has been subjected to little systematic study. As formulated in DSM-IV-TR, IED is probably a rare disorder. The exclusionary criterion in the DSM-IV-TR definition (criterion C) reflects an ongoing debate over the boundaries of this disorder. The current definition of IED is the result of a succession of attempts by researchers to classify syndromes associated with impulsive aggression. The diagnostic term "IED" first appeared in the 1980 *Diagnostic and Statistical Manual of Mental Disorders*, Third Edition (DSM-III). The DSM-III and the revised third edition (DSM-III-R) definitions of IED required the absence of signs of generalized impulsivity or aggressiveness between episodes. Episodic behavioral disorders are quite common and exist across a continuum between ictal causes (excessive neuronal discharges) and purely motivational causes (psychogenic). Temper proneness is a relatively common clinical syndrome that is associated with a wide variety of psychiatric disorders and is usually found in patients with central nervous system dysfunction, character disorders, and psychoactive substance abuse. "Pure" IED, on the other hand, was found to be a rare clinical entity.

A number of 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%. The evaluation of studies is complicated by the variety of defining criteria used, from DSM-III to current research criteria. Zimmerman et al. (1998) used the Structured Clinical Interview for DSM-IV to study current or lifetime IED in 411 psychiatric outpatients. They reported a rate of 3.8% for current IED and 6.2% for lifetime IED by DSM-IV criteria. A recent reanalysis of a much larger sample from the same population revealed similar rates of IED (Coccaro et al. 2005). Further, data from a pilot community sample study revealed a community rate of lifetime

IED by DSM-IV-TR criteria at 4% and by IED-integrated research criteria at 5.1% (Coccaro et al. 2004). Considering the rates found in these more recent studies, IED could be as common as other major psychiatric disorders like schizophrenia or bipolar illness. In fact, the National Comorbidity Survey Replication (NCS-R) study found that IED is much more common than previously thought. Lifetime and 12-month prevalence estimates of DSM-IV IED were 7.3% and 3.9%, with a mean 43 lifetime attacks resulting in 1359 dollars in property damage. IED-related injuries occurred 180 times per 100 lifetime cases (Kessler et al. 2006).

In one study of the prevalence of DSM-III-R IED among violent men, Felthous et al. (1991) found that of 443 subjects who complained of violence, only 15 (3.4%) met criteria for IED. The DSM-III-R definition of IED was more restrictive than the current DSM-IV-TR diagnosis because it required the absence of signs of generalized impulsivity or aggressiveness between episodes. The EEGs of 13 of the men with IED were normal; two showed excessive slowing.

Most of the limited published data on gender differences suggest that males outnumber females with IED, and men with the disorder are more likely to be encountered in forensic settings, whereas women with the disorder are more likely to be found in psychiatric settings. This difference in presentation may reflect the reduced severity of the aggressive acts committed by women with IED. But, more recent data suggest that the male:female ratio is closer to 1:1 (Coccaro et al. 2005).

## Comorbidity Patterns

In contrast to the more restrictive DSM-III and DSM-III-R criteria, the DSM-IV-TR definition of IED allows signs of generalized impulsivity or aggressiveness to be present between episodes. It also allows the psychiatrist to give an additional diagnosis of IED in the presence of another disorder if the episodes are not better accounted for by the other disorder. These changes were deemed necessary because the clinical reality is that most individuals who have intermittent episodes of aggressive behavior also have some impulsivity between episodes and often present with other past or current psychiatric disorders.

Subjects with IED most frequently have other Axis I and II disorders. The most frequent Axis I diagnoses comorbid with IED lifetime include mood, anxiety, substance, eating, and other ICDs ranging in frequency from 7% to 89% (Coccaro et al. 1998b, McElroy et al. 1998). Such Axis I comorbidity rates raise the question of whether IED constitutes a separate disorder. However, recent data finding earlier onset of IED compared with all disorders, except for phobic-type anxiety disorders, suggest that IED is not secondary to these other disorders (Coccaro et al. 2005).

McElroy et al. (1998) and McElroy (1999) studied 27 individuals who had symptoms that met criteria for IED and reported: "Twenty-five (93%) subjects had lifetime DSM-IV-TR diagnoses of mood disorders; 13 (48%), substance use disorders; 13 (48%), anxiety disorders; 6 (22%), eating disorders; and 12 (44%), an ICD other than IED. Subjects also displayed high rates of comorbid migraine headaches. First-degree relatives displayed high rates of mood, substance use, and impulse-control disorders." McElroy et al. (1998) reported that the aggressive episodes observed in their subjects resembled "microdysphoric" manic episodes.

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%). However, this finding may not be surprising, because 56% of the subjects in question had a comorbid bipolar diagnosis of some type (bipolar I, 33%; bipolar II, 11%; and bipolar not otherwise specified or cyclothymia, 11%). The Rhode Island Hospital Study (Coccaro et al. 2005) suggests a much lower rate of comorbid bipolar illness, with a rate of 11% (bipolar I, 5%; bipolar II, 5%; and bipolar not otherwise specified, 1%). Regardless, clinicians should fully evaluate for bipolar disorder prior to determining treatment for IED, because mood stabilizers, rather than serotonin reuptake inhibitors (SSRIs), would be the first-line treatment for IED comorbid with bipolar disorder.

McElroy et al. (1998) reported that up to 44% of their IED subjects had another impulse-control–type disorder such as compulsive buying (37%) or kleptomania (19%). However, Coccaro et al. (1998b) found that IED subjects had a comorbid ICD, and only 5% of IED subjects had another ICD in the Rhode Island Hospital Study (Coccaro et al. 2005).

Some children with Tourette's disorder may be prone to rage attacks (Budman et al. 1998, 2000). The clinical manifestation of these attacks is similar to IED and may be more common among children with Tourette's who have comorbid mood disorders. On the basis of these observations, the rage attacks of these children may flow from an underlying dysregulation of brain function (Budman et al. 1998, 2000).

The limited literature on the comorbidity of impulsive aggressive episodes suggests that it often occurs with three classes of disorders:

1. Personality disorders, especially ASPD and BPD. By definition, ASPD and BPD are chronic and include impulsive aggression as an essential feature. Therefore, their diagnosis effectively excludes the diagnosis of IED (Figure 000–2). Coccaro et al. (1998b) reported the rate of BPD and/or ASPD in IED subjects to be 38%. However, rates of IED in subjects with BPD have been noted at 78% and in subjects with ASPD at 58% (Coccaro et al. 1998b). A review of unpublished data from the author's lab (Hollander 2007) (Hollander E [2007], personal communication) suggests that these rates are lower among subjects not seeking treatment and are lowest in the community (23% for BPD and/or ASPD; see also Coccaro et al. 2004). Regardless, BPD and ASPD 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/ASPD subjects without IED (Coccaro et al. 2005).

2. A history of substance use disorders, especially alcohol abuse. A concurrent diagnosis of substance intoxication excludes the diagnosis of IED. However, many patients with IED report past or family histories of substance abuse, and in particular alcohol abuse. In light of evidence linking personal and family history of alcohol abuse with impulsive aggression (Linnoila et al. 1989) and linking both with low central serotonergic function (reviewed later), this connection may be clinically
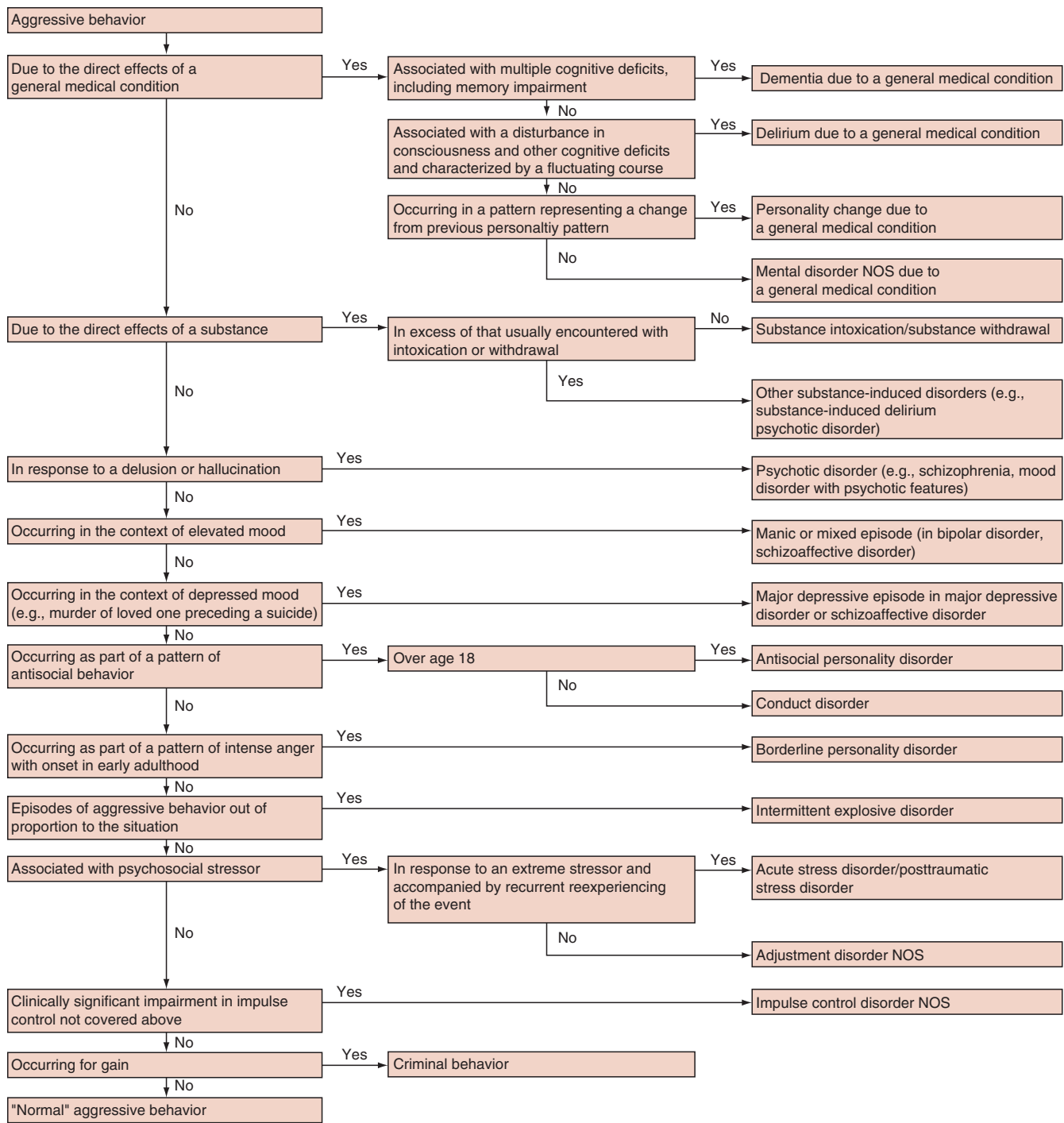
**Figure 000–2** *Differential diagnosis of aggression. The psychiatric nosology of aggression has not been well worked out and requires much additional study. This is a particularly unfortunate state of affairs because the attribution (or misattribution) of aggression to a mental disorder is a frequent focus of forensic attention and can mean the difference between a life term in prison or a promotional tour for a bestseller. Because of the inherent difficulties in making these determinations, psychiatric testimony in this regard should be interpreted with caution. Other decision trees that may be of interest include those for catatonia; delusions; euphoria or irritability; disorganized, agitated, or unusual behavior; impulsivity; hallucinations; substance use; and general medical condition.*

relevant. Thus, when there is evidence suggesting that alcohol abuse may be present, a systematic evaluation of IED is warranted, and vice versa.

3. Neurological disorders, especially severe head trauma, partial complex seizures, dementias, and inborn errors of metabolism. IED is not diagnosed if the aggressive episodes are a direct physiological consequence of a

general medical condition. Such cases would be diagnosed as personality change due to a general medical condition, delirium, or dementia. However, individuals with IED often have nonspecific findings on neurological examination, such as reflex asymmetries, mild hand-eye coordination deficits, and childhood histories of head trauma with or without loss of consciousness.

Their EEGs may show nonspecific changes. Such isolated findings are compatible with the diagnosis of IED and preempt the diagnosis only when they are indicative of a definitely diagnosable general medical or neurological condition. Such "soft" neurological signs may be diagnosed by a full neurological examination and neuropsychological testing.

## Course

Given the rarity of pure IED, limited research is available concerning the age at onset and natural course of IED. But, according to the DSM IV-TR (American Psychiatric Association 2000), and anecdotal case reports, the onset appears to be from childhood to the early 20s, and may be abrupt and without a prodromal period. The age of onset and course of IED distinguish it as separate from its comorbid diagnoses. The course of IED is variable, with an episodic course in some and a more chronic course in others. IED may persist well into middle life unless treated successfully. In some cases, it may decrease in severity or remit completely with old age. However, cognitive impairment caused by Alzheimer's disease and other age-related causes of dementia may result in the reappearance of impulsive aggressive behavior. A mean age at onset of 16 years and an average duration of about 20 years has been described (McElroy et al. 1998). Preliminary data (Coccaro et al. 2005) confirm these findings and indicate that onset of DSM-IV-TR IED occurs by the end of the first decade in 31%, by the end of the second decade in 44%, by the end of the third decade in 19%, and by the end of the fourth decade in only 6%. The mean age at onset in the NCS-R study was 14 years (Kessler et al. 2006).

Episodes typically last less than 30 minutes and involve one of or a combination of physical assault, verbal assault, or destruction of property. If provoked, it is usually from a known person and is seemingly minor in nature (McElroy et al. 1998). Many individuals frequently have minor aggressive episodes in the interim between severely aggressive/destructive episodes. Considerable distress, social, financial, occupational, or legal or impairments typically result from these episodes.

## Differential Diagnosis

The differential diagnosis of IED covers the differential diagnosis of impulsivity and aggressive behavior in general. Aggression is defined as forceful physical or verbal action, which may be appropriate and self-protective or inappropriate as in hostile or destructive behavior. It may be directed against another person or the environment, or toward the self. The psychiatric nosology of aggression is still preliminary. Impulsivity is defined as the tendency to act in a sudden, unpremeditated, and excessively spontaneous fashion. The IED diagnosis should be considered only after all other disorders associated with impulsivity and aggression have been ruled out. Chronic impulsivity and aggression may occur as part of a cluster B personality disorder (e.g. BPD and ASPD); during the course of substance use disorders and substance intoxication; in the setting of a general medical (usually neurological) condition; and as part of disorders first diagnosed during childhood and adolescence such as conduct disorder, oppositional defiant disorder, ADHD, and mental retardation. In addition, impulsive aggression may appear during the course of a mood disorder, especially during a manic episode, which precludes the diagnosis of IED, and during the course of an agitated depressive episode. Impulsive aggression may also be an associated feature of schizophrenia, in which it may occur in response to hallucinations or delusions. Impulsive aggression may also appear in variants of obsessive-compulsive disorder (OCD), which may present with concurrent impulsive and compulsive symptoms.

A special problem in the differential diagnosis of impulsive aggression, which may arise in forensic settings, is that it may represent purposeful behavior, which is distinguished from IED by the presence of motivation and gain in the aggressive act, such as monetary gain, vengeance, or social dominance. Another diagnostic problem in forensic settings is malingering, in which individuals may claim to have IED to avoid legal responsibility for their acts. Figure 000–2 presents the differential diagnosis of aggression.

Common disorders that should be excluded before IED is diagnosed and features that may be helpful in the differential diagnosis are summarized in Table 000–1.

## Differences in Gender and Cultural Presentations

Amok is an extremely rare culture-specific syndrome of episodic aggression first described in the Malay Peninsula but later found in Africa and Papua New Guinea. Amok is an episode of sudden, unprovoked rage in which the affected individual runs around with a weapon and attempts to kill a number of people or animals. Sometimes the perpetrator, typically a man, then kills himself. If captured alive, the individual with amok claims no memory of the acts. The etiology of amok and its relation to IED are unclear. Episodic violent behavior is more common in males than in females (DSM-IV-R).

## Etiology and Pathophysiology

Theories about the etiology of impulsive aggressive outbursts and IED have been part of psychiatry from its origins. Possession by spirits, humoral imbalances, and "moral weakness" were all suggested to play a role. Since the second-half of the 19th century, two main lines of explanation, which are to a large extent complementary, have been developed to account for the existence of individuals with episodic impulsive aggression. One line of explanation viewed the etiology of impulsive aggression as stemming from the effects of early childhood experiences and possibly childhood trauma on the development of self-control, frustration tolerance, planning ability, and gratification delay, which are all important for self-prevention of impulsive aggressive outbursts. Early experiences with "good-enough" mothering that fosters phase-appropriate delay of gratification and the development of the potential for imitation and identification with the mother are considered important for normal development. Too much or too little frustration, as well as overgratification or undergratification, may impair the normal development of the ability to anticipate frustration and delay gratification (Khantzian and Mack 1983).

A second line of explanation, which has yielded numerous positive findings over the past 15 years, views impulsive aggression as the result of variations in brain mechanisms that mediate behavioral arousal and behavioral inhibition. A rapidly growing body of evidence has shown that impulsive

aggression may be related to defects in the brain serotonergic system, which acts as an inhibitor of motor activity (Kavoussi et al. 1997, Staner and Mendlewicz 1998). Animal studies suggest that serotonergic neurons play a role in behavioral inhibition and thus provide an impetus to explore the role of serotonin in human impulsivity. Although the majority of the human studies involved patients who suffered from impulsive aggression in the context of disorders other than IED, their findings may be relevant to the behavioral dimension of impulsive aggression, of which IED is a "pure" form.

Measures examining central (and peripheral) serotonin function correlate inversely with life history, questionnaire, and laboratory measures of aggression. This relationship has been demonstrated by CSF 5-HIAA (Linnoila et al. 1983, Virkkunen et al. 1994), physiological responses to serotonin agonist probes (Coccaro et al. 1989, 1997b, Dolan et al. 2001, Manuck et al. 1998), and platelet measures of serotonin activity (Birmaher et al. 1990, Coccaro et al. 1996). 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). Linnoila et al. (1989) divided aggressive behaviors into impulsive and nonimpulsive forms and found that reduced CSF 5-HIAA was correlated with impulsive aggression only. These findings suggest that impulsive aggressive behavior can be distinguished biologically from nonimpulsive aggression. Interestingly, the inverse relationship between aggression and serotonin is not observed when catecholamine system function is impaired (Coccaro et al. 1989, Wetzler et al. 1991). Siever et al. (1991) and Stein et al. (1993) have confirmed a relationship between levels of 5-HIAA in the CSF and impulsive or aggressive behaviors. Pharmacological challenge studies have also demonstrated that low serotonergic responsiveness (measured by the neuroendocrine response to serotonergic agonists) correlates with scores of impulsive aggression. Studies of impulsive aggression among alcoholics have further defined a probable relationship between such behaviors and diminished serotonergic function (Virkkunen et al. 1995, Virkkunen and Linnoila 1993).

There is also evidence to support the role of nonserotonergic brain systems and modulators in impulsive aggression. These findings suggest a role for dopamine (Depue et al. 1994), norepinephrine (Coccaro et al. 1991), vasopressin (Coccaro et al. 1998a), 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).

Another line of neurobiological evidence links impulsive aggression with dysfunction of the prefrontal cortex (PFC). Studies of neuropsychiatric patients with localized brain lesions have demonstrated that some bilateral lesions in the PFC may be specifically associated with a chronic pattern of impulsive aggressive behaviors. Neurological studies suggest that the PFC regions associated with impulsive aggression syndromes are involved in the processing of affective information and the inhibition of motor responsiveness, both of which are impaired in impulsive aggressive patients. Interictal episodes of aggression may occur among some people with epilepsy. In a quantitative MRI study of such episodes among people with temporal lobe epilepsy (TLE) (Woermann et al. 2000) three groups (24 TLE patients with aggressive behavior, 24 TLE patients without such behavior, and 35 nonpatient controls) were compared. The aggressive behavior was associated with a reduction of frontal neocortical gray matter.

Further evidence linking the PFC with the serotonergic system and impulsive aggression comes from postmortem and animal studies suggesting that the PFC is rich in excitatory 5-HT$_2$ receptors, whose number is increased in suicide victims and correlated with aggressive social behavior in primates. Lower levels of CSF 5-HIAA were found in neurological patients with frontal brain injuries than in patients with injuries in other brain regions. The fenfluramine challenge test, a neuroendocrine challenge to the serotonergic system, was found to increase cerebral prefrontal glucose metabolism in normal control subjects. PET studies have found selective reductions in glucose metabolism in the prefrontal and frontal cortex of patients with impulsive aggression. The regional reductions in glucose metabolism in impulsive aggressive patients were more significant during a continuous performance task, whose performance was impaired in neurological patients with frontal lesions and was found to increase frontal glucose metabolism in normal subjects (Raine et al. 1994). A visual-evoked potential and EEG study in a large group of aggressive children and adolescents also suggest that such behavior may be associated with altered innate characteristics of central nervous system function (Bars et al. 2001).

Thus, biological studies implicate the serotonergic system and the PFC in the pathogenesis of impulsive aggression. The diagnosis of IED is sometimes considered in forensic settings; the biological correlates of impulsive aggression focus attention on, but do not solve, the complicated problem of personal responsibility for impulsive violent acts that are correlated with objective biological findings.

## Family and Twin Studies

Clinical observation and family history data suggest that IED is familial. Familial aggregation of temper outbursts and IED has been reported in psychiatric patients with "temper problems" (Mattes and Fink 1987). McElroy et al. (1998) reported that nearly a third of first-degree relatives of IED probands had IED. A blinded, controlled, family history study using IED-integrated research criteria (Coccaro 1999) found a morbid risk of IED of 26% in relatives of IED-IR probands compared with 8% among the relatives of control probands, a significant difference. Although twin studies have confirmed the hypothesis that both impulsivity (Seroczynski et al. 1999) and aggression (Coccaro et al. 1997a) are under substantial genetic influence, there are no twin studies of IED itself. Genetic influence for these two traits ranges from 28% to 47%, with nonshared environmental influences making up the lion's share of the remaining variance.

## Molecular Genetic Studies

Studies of particular genes in aggressive populations have used the candidate gene approach. Candidate genes are the genes for proteins with a suspected, or proven, biological association to a disorder [e.g., serotonin (5-HT) receptors in aggression]. The polymorphism HTR1B/G861C and

short tandem repeat locus D6S284 are part of the gene for the 5-HT$_{1B}$ receptor for serotonin. These genetic sites were examined in 350 Finnish sibling pairs and 305 Southwestern American Indian sibling pairs, both with a high rate of alcoholism. The diagnoses of ASPD and IED were used to examine the traits of impulsivity and aggression. The rate of IED in relatives of ASPD probands was 15%, and the relatives of healthy control subjects had neither IED nor ASPD. Lappalainen et al. (1998) were able to discover that the gene predisposing to ASPD alcoholism resides close to the HTR1B version of the coding sequence. They concluded that impulsivity and aggression might be influenced, in part, by 5-HT$_{1B}$ receptors. Other candidate genes include the genes for tryptophan hydroxylase and MAO-A. Manuck et al. (1999, 2000) found an association of the traits of aggression, impulsivity, and serotonin activity (tested by *D,L-fenfluramine* challenge) with variations in both the tryptophan hydroxylase and the MAO-A genes in community samples.

## Imaging and Brain Localization

Few localization and functional studies have looked at impulsive aggression or IED. Using fluorodeoxyglucose positron emission tomography (FDG-PET), Siever et al. (1999) found blunted glucose utilization responses to serotonin stimulation in the orbitofrontal cortex (an area associated with impulsive aggression) of IED subjects with BPD. A similar finding was reported in the anterior cingulate and anteromedial orbital cortex of impulsive aggressive subjects after stimulation with the direct serotonin agonist *m*-chlorophenylpiperazine (New et al. 2002). Using PET with a 5-HT$_{1A}$ antagonist in healthy volunteers, Parsey et al. (2002) found a significant inverse correlation between lifetime aggression and serotonin receptor binding in the dorsal raphe, anterior cingulate cortex, amygdala, medial PFC, and orbital PFC. Using neuropsychological testing in impulsive aggressive subjects, Best et al.'s (2002) data supported a possible dysfunctional frontal circuit. More work is needed to reveal the specific functional brain abnormalities in impulsive aggressive individuals.

## Treatment

Given the rarity of pure IED, it is not surprising that few systematic data are available on its response to treatment and that some of the recommended treatment approaches to IED are based on treatment studies of impulsivity and aggression in the setting of other mental disorders and general medical conditions. Thus, no standard regimen for the treatment of IED can currently be recommended. Both psychological and somatic therapies have been utilized in the treatment of IED. A prerequisite for both modalities is the willingness of the individual to acknowledge some responsibility for the behavior and participate in attempts to control it.

### Psychological Treatment

Lion (1992) has described the major psychotherapeutic task of teaching individuals with IED how to recognize their own feeling states and especially the affective state of rage. Lack of awareness of their own mounting anger is presumed to lead to the buildup of intolerable rage that is then discharged suddenly and inappropriately in a temper outburst. Patients with IED are therefore taught how to first recognize and then verbalize their anger appropriately. In addition, during the course of insight-oriented psychotherapy, they are encouraged to identify and express the fantasies surrounding their rage. Group psychotherapy for temper-prone patients has also been described. The cognitive-behavioral model of psychological treatment may be usefully applied to problems with anger and rage management.

Anger treatment studies focus on treatment of anger as a component of other psychiatric illnesses, like substance abuse, post-traumatic stress disorder, depression, and domestic violence; and in forensic and mentally impaired populations. In a few rare cases, anger is addressed as the primary or only problem, and a limited number of treatments have been described. "Imaginational 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 felt to be useful. In a controlled trial of high driving anger college students, Deffenbacher et al. (2000) compared pure relaxation training with relaxation training combined with cognitive therapy and an assessment-only control. Neither treatment condition improved general trait anger, but both treatments improved driving anger. When repeated in a new population of drivers with higher anger levels, both treatments lowered trait anger (Deffenbacher et al. 2002). Since relaxation training with cognitive therapy provided little gain over pure relaxation training, relaxation training in itself may be adequate treatment for driving anger.

Other versions of cognitive-behavioral therapy (CBT), like dialectical behavior therapy (DBT), have been studied in BPD patients. One study showed improvement in anger, global functioning, and social adjustment compared with a treatment-as-usual condition (Linehan et al. 1994). Improvement in anger and impulsivity has been shown with DBT across many disorders. There are no published double-blind, placebo-controlled studies on IED subjects in therapy, but studies of therapy in IED subjects are ongoing.

### Somatic Treatments

Several classes of medications have been used to treat IED and impulsive aggression in the context of other disorders. These included beta-blockers (propranolol and metoprolol), anticonvulsants (carbamazepine and valproic acid), lithium, antidepressants (tricyclic antidepressants and SSRIs), and antianxiety agents (lorazepam, alprazolam, and buspirone). Mattes (1990) compared the effectiveness of two commonly used agents, carbamazepine and propranolol, for the treatment of rage outbursts in a heterogeneous group of patients. He found that although carbamazepine and propranolol were overall equally effective, carbamazepine was more effective in patients with IED and propranolol was more effective in patients with ADHD. A substantial body of evidence supports the use of propranolol—often in high doses—for impulsive aggression in patients with chronic psychotic disorders and mental retardation. Lithium has been shown to have antiaggressive properties and may be used to control temper outbursts. In patients with comorbid major depressive disorder, OCD, or cluster B and C personality disorders, SSRIs may be useful. Overall, in the absence of more controlled clinical trials, the best approach may be to tailor the psychopharmacological agent to coexisting psychiatric comorbidity. In the absence of comorbid disorders, carbamazepine, titrated to antiepileptic blood levels, may be used empirically.

Mr. A is a 42-year-old separated man who works as a bank clerk. He came to seek outpatient psychiatric treatment after an angry outburst that led to the breakdown of his second marriage: his wife issued an order of protection against him after a rage attack in which he slapped her across the face and destroyed most of the kitchen and living room furniture. His rage was triggered by his wife's decision to buy a new microwave oven without consulting him. Mr. A, who remembered the episode clearly and with remorse, said that he realized how angry he was only after he actually struck at his wife.

During the course of his evaluation, Mr. A became tearful and admitted to several similar episodes during the course of his current and previous marriages. These episodes were rare, occurring once or twice a year. They were brief and apparently unpredictable and resulted in his separation from his first wife. Except during those episodes, Mr. A was a pleasant, rather timid man who deferred to his wife in most important decisions. There was no history suggestive of antisocial or borderline personality disorder. Mr. A, who described himself as a shy, withdrawn child, gave a history of head trauma at the age of 12 years, while he was ice skating, with loss of consciousness for 10 minutes. Other than this, his medical history was normal. There were no neurological or behavioral sequelae. Mr. A also described prolonged physical abuse by his alcoholic father. Mr. A himself denied a history of substance abuse, involvement with the criminal justice system, and prior psychiatric treatment. He denied a history of manic and depressive episodes. Mr. A had few friends and was not popular at his job. Although he had never lost his temper there, he believed that his boss and coworkers could sense his "stress" while dealing with clients.

Mr. A's physical and neurological examination was notable only for mild bilateral difficulty with rapid alternating hand movements. Except for his tearfulness while describing the episode, Mr. A's Mental Status Examination was unremarkable. Results of routine laboratory blood work and computed tomography of the head were within normal limits. An EEG was notable for diffuse slowing without an epileptic focus.

Mr. A's treatment was started with carbamazepine. at standard dosage. He also received a short course of psychotherapy that focused on recognizing his anger and venting it appropriately, on his memories of childhood physical abuse, and on his current sense of himself as a helpless person who was being controlled by his wife and boss. In addition, it was recommended that he transfer to a position that would not involve contact with clients. During a 2-year follow-up, Mr. A had no further rage episodes. He continued to have few friends but was able to maintain a long-term relationship with a woman he was planning to marry.

## Kleptomania

### Diagnosis

### Definition and Diagnostic Features

Kleptomania shares with all other ICDs the recurrent failure to resist impulses. Unfortunately, in the absence of epidemiological studies, little is known about kleptomania. Clinical case series and case reports are limited. Family, neurobiological, and genetic investigations are not available.

There are no established treatments of choice. Therefore, in reading this section the reader must keep in mind that much of what is described is based on limited data or on anecdotal information.

Kleptomania was designated a psychiatric disorder in 1980 in DSM-III (American Psychiatric Association 1980), and in DSM-III-R (American Psychiatric Association 1987) it was grouped under "disorders of impulse control not elsewhere classified." Kleptomania is currently classified in DSM-IV-TR (American Psychiatric Association 2000) as an ICD (see box below for criteria), but it is still poorly understood. Criterion A, which focuses on the senselessness of the items stolen, has often been considered the criterion that distinguishes kleptomania patients from ordinary shoplifters (Goldman 1991), but interpretation of this criterion is controversial. The archetype of the middle-aged female kleptomania patient who steals peculiar items may not adequately account for all people with kleptomania (Goldman 1991, McElroy et al. 1991a). Patients with kleptomania may in fact desire the items they steal and be able to use them, but do not need them. This may be particularly the case with kleptomania patients who hoard items (Goldman 1991), for which multiple versions of the same item are usually not needed, but the item itself may be desired and may be of practical use to the patient.

People with kleptomania often report amnesia surrounding the shoplifting act (Goldman 1991, Grant 2004), and deny feelings of tension or arousal prior to shoplifting and feelings of pleasure or relief after the thefts. They often recall entering and leaving a store but have no memory of events in the store, including the theft (Grant 2004). Others, who are not amnestic for the thefts, describe shoplifting as "automatic" or "a habit", and may also deny feelings of tension prior to a theft or pleasure after the act (DSM-IV-TR criterion B or C), although they report an inability to control their shoplifting (criterion A). Some report that they felt tension and pleasure when they started stealing, but it became a "habit" over time. Some speculate that patients who are amnestic for shoplifting or who do so "out of habit" represent two subtypes of kleptomania.

At presentation, the typical patient suffering from kleptomania is a 35-year-old woman who has been stealing for about 15 years and may not mention kleptomania as the presenting complaint or in the initial history (Goldman 1991, McElroy et al. 1991a). The patient may complain instead of anxiety, depression, lability, dysphoria, or manifestations of character pathology. There is often a history of a tumultuous childhood and poor parenting, and in addition acute stressors may be present, such as marital or sexual conflicts. The patient experiences the urge to steal as irresistible, and the thefts are commonly associated with a thrill, a high, a sense of relief, or gratification. Generally, the behavior has been hard to control and has often gone undetected by others. The kleptomania may be restricted to specific settings or types of objects, and the patient may or may not be able to describe rationales for these preferences. Quite often, the objects taken are of inherently little financial value, or have meaningless financial value relative to the income of the person who has taken the object. Additionally, the object may never actually be used. These factors often help distinguish criminal theft from kleptomania. The theft is followed by feelings of guilt or shame

and, sometimes, attempts at atonement. The frequency of stealing episodes may greatly fluctuate in concordance with the degree of depression, anxiety, or stress. There may be periods of complete abstinence. The patient may have a past history of psychiatric treatments including hospitalizations or of arrests and convictions, whose impact on future kleptomanic behavior can be variable.

---

**DSM-IV-TR Criteria**

**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.

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

---

Reproduced with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Copyright 2000 American Psychiatric Association.

## Assessment

Generally, the diagnosis of kleptomania is not a complicated one to make. However, kleptomania may frequently go undetected because the patient may not mention it spontaneously and the psychiatrist may fail to inquire about it as part of the routine history. The index of suspicion should rise in the presence of commonly associated symptoms such as chronic depression, other impulsive or compulsive behaviors, tumultuous backgrounds, or unexplained legal troubles. It could convincingly be argued that a cursory review of compulsivity and impulsivity, citing multiple examples for the patient, should be a part of any thorough and complete psychiatric evaluation. In addition, it is important to do a careful differential diagnosis and pay attention to the various exclusion criteria before diagnosing theft as kleptomania. Possible diagnoses of sociopathy, mania, or psychosis should be carefully considered. In this regard, the psychiatrist must inquire about the affective state of the patient during the episodes, the presence of delusions or hallucinations associated with the occurrence of the behavior, the motivation behind the stealing, and the fate and subsequent use of the objects.

Although the typical patient may be a 35-year-old woman, it is important to remember that men, children, and elderly persons may present with or engage in kleptomania. Interestingly, Goldman's suggested that men may first present for evaluation 15 years later than women. Kleptomania occurs transculturally and has been described in various Western and Eastern cultures. Asian observers have also noted an overlap with eating disorders (Lee 1994). Atypical presentations should raise a greater suspicion of an organic etiology for whom a medical evaluation would then be indicated. Medical conditions that have been associated with kleptomania include cortical atrophy, dementia, intracranial mass lesions, encephalitis, normal-pressure hydrocephalus, benzodiazepine withdrawal, and TLE. A complete evaluation when such suspicions are present includes a physical and neurological examination, general serum chemistry and hematological panels, and an EEG with temporal leads or computed tomography of the brain (Chiswick 1976, Khan and Martin 1977, Mendez 1988, Wood and Garralda 1990, Coid 1984, McIntyre and Emsley 1990).

## Epidemiology

No epidemiological studies of kleptomania have been conducted, and thus its prevalence can be estimated only grossly and indirectly. In a thorough review of the existing literature, Goldman (1991) found that in a series of shoplifters, the estimate of kleptomania ranged from 0% to 24%. The frequency of kleptomania may be indirectly extrapolated from incidence rates of kleptomania in comorbid disorders with known prevalence, like bulimia nervosa. Such speculations suggest at least a 0.6% prevalence of kleptomania in the general population (Goldman 1991). However, given that people who shoplift are often not caught, this is almost certainly an underestimate. Also, the shame and embarrassment associated with stealing prevents most people from voluntarily reporting kleptomania symptoms (Grant and Kimm 2002c). In addition, studies examining comorbidity of other disorders may neglect to inquire about kleptomania. Studies of kleptomania in various clinical samples suggest a higher prevalence. A recent study of 204 adult psychiatric inpatients in the US, with multiple disorders, revealed that kleptomania may in fact be fairly common. The study found that 7.8% ($n=16$) endorsed current symptoms consistent with a diagnosis of kleptomania and 9.3% ($n = 19$) had a lifetime diagnosis of kleptomania (Grant et al. 2005). Kleptomania appeared equally common in patients with mood, anxiety, substance use, or psychotic disorders. These findings are further supported by two French studies. One study of 107 depressed inpatients found that 4 (3.7%) had kleptomania (Lejoyeux et al. 2002); in another study of 79 alcohol-dependent inpatients, 3 (3.8%) reported symptoms consistent with kleptomania (Lejoyeux et al. 1999). In two studies examining comorbidity in pathological gamblers, rates of comorbid kleptomania ranged from 2.1% to 5% (Grant and Kim 2003, Specker et al. 1995). A study of bulimia patients found that 24% met DSM-III (American Psychiatric Association 1980) criteria for kleptomania (Hudson et al. 1983).

The literature clearly suggests that the majority of patients with kleptomania are women (e.g. Grant and Kim 2002b, McElroy et al. 1991b, Presta et al. 2002). In a retrospective review of 56 cases that appeared to fulfill DSM-III-R criteria for kleptomania, McElroy et al. (1991b) found that 77% were women. Similarly, in a prospective series of 20 patients with DSM-III-R kleptomania, 75% were women

(McElroy et al. 1991a). However, women generally seek psychiatric help more frequently than men, whereas men are more likely to become involved with the penal system (Goldman 1991). Consequently, this may not reflect true gender distribution. The severity of kleptomania symptoms and the clinical presentation of symptoms do not appear to differ based on gender (Grant and Kim 2002b).

## Comorbidity Patterns

High rates of other psychiatric disorders found in patients with kleptomania have sparked debate over the proper characterization of this disorder. Among those with kleptomania who present for treatment, there is a high incidence of comorbid mood, anxiety, and eating disorders, when compared with rates in the general population. 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 have also found high lifetime rates of comorbid anxiety disorders (60-80%; McElroy et al.1991b, 1992), 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 kleptomania patients, the most common being paranoid and histrionic personality disorder (Bayle et al. 2003, Grant 2004).

In reviewing 26 case reports of kleptomania, Goldman (1991) reported mention of histories of depression in 13 patients (50%), anxiety in 8 patients (31%), and bulimia nervosa in 3 patients (12%). Similar percentages are noted by McElroy et al. (1991b) in a review of 56 patients with probable kleptomania: 57% with mood disorder symptoms, 34% with anxiety disorder symptoms, and 11% with bulimic symptoms. Comorbidity patterns among those who present for treatment may be greater than among random samples. More reliable comorbidity rates can be found in a prospective study of 20 kleptomaniacs (McElroy et al. 1991a). Lifetime DSM-III-R comorbidity rates were 40% major depressive disorder, 50% substance abuse, 40% panic disorder, 40% social phobia, 45% OCD, 30% anorexia nervosa, 60% bulimia nervosa, and 40% other ICDs. Dissociative symptoms, significant character pathology, and trauma histories are commonly encountered among this group (Goldman 1991, McElroy et al. 1991b). Unfortunately, Axis I dissociative pathology and Axis II pathology have not yet been systematically investigated in these patients.

## Course

Kleptomania may begin in childhood, adolescence, or adulthood, and sometimes in late adulthood. However, most patients have an onset of symptoms before the age of 21 years, that is, by late adolescence (Goldman 1991, Grant and Kim 2002b, McElroy et al. 1991a, 1991b; Presta et al. 2002). In two separate studies, the mean age at onset was 20 years (Goldman 1991, McElroy et al. 1991a), and included people who had begun stealing as early as 5 to 7 years old. Onset beyond 50 years is unusual, and in some of these cases remote histories of past kleptomania can be elicited (Goldman 1991). Most clinical samples of kleptomaniacs report shoplifting for more than 10 years prior to

entering treatment (Goldman 1991, Grant and Kim 2002c, McElroy et al. 1991b) and in some cases 15 or 16 years may elapse before treatment is sought (Goldman 1991, McElroy et al. 1991a).

The disorder appears to be chronic, but with varying intensity. At peak frequency, McElroy et al. (1991a) found a mean of 27 episodes a month, essentially daily stealing, with one patient reporting four acts daily. The majority of patients may eventually be apprehended for stealing once or more, and a minority may even be imprisoned; usually these repercussions do not result in more than a temporary remission of the behavior. People with kleptomania may also have extensive histories of psychiatric treatments, including hospitalization for other conditions, most commonly depression or eating disorders. Because of the unavailability of longitudinal studies, the prognosis is unknown. However, it appears that without treatment the behavior may be likely to persist for decades, sometimes with significant associated morbidity, despite multiple convictions for shoplifting (arrest or imprisonment), with transient periods of remission. Three typical courses have been described: sporadic with brief episodes and long periods of remission; episodic with protracted periods of stealing and periods of remission; and chronic with varying intensity (DSM-IV-TR, American Psychiatric Association 2000).

## Etiology and Pathophysiology

The etiology of kleptomania is essentially unknown, although various models have been proposed in an effort to conceptualize the disorder. At present, the available empirical data are insufficient to substantiate any of these models. With the exception of scant information on family history, data regarding possible familial or genetic transmission of a kleptomania diathesis are unavailable. One study found the risk for major mood disorders in first-degree relatives of probands with kleptomania to be 0.31; similar to the familial risk for probands with major depressive disorder (McElroy et al. 1991a). In the same study, 7% of first-degree relatives of kleptomania patients had histories of OCD. These findings, along with other lines of evidence, suggest that kleptomania shares a common biological diathesis with mood disorders or OCD. The affective spectrum model suggests that kleptomania and other ICDs may share a common underlying biological diathesis with other disorders like depression, panic disorder, OCD, and bulimia nervosa (McElroy et al. 1992, 1991b, Hudson and Pope 1990). The apparent high comorbidity of kleptomania with depression and bulimia nervosa has already been noted. As early as 1911, Janet (1911) recognized the alleviation of depressive symptoms on the commission of kleptomanic acts. Some individuals with kleptomania respond to treatment with thymoleptic agents or electroconvulsive therapy. These observations are cited as support for an affective spectrum model.

Although the affective spectrum has been claimed to encompass obsessive-compulsive pathology (Hudson and Pope 1990) there exists a more specific model conceptualizing kleptomania and other impulse disorders as obsessive-compulsive spectrum disorders (McElroy et al. 1993). Several lines of evidence support this model. First, there are phenomenological similarities between the classical obsessions and compulsions of OCD and the irresistible impulses and repetitive actions characteristic of kleptomania. Further, there appears

to be a greater than chance occurrence of OCD in probands with kleptomania and in their relatives. In addition, both conditions have significant comorbidity with mood, anxiety, substance use, and eating disorders. However, OCD rituals are more clearly associated with relief of anxiety and harm avoidance, whereas kleptomania acts seem to be associated with gratification or pleasure. In addition, OCD is associated with a clear preferential response to SSRIs as opposed to general thymoleptics. The limited treatment literature (see later) does not support a similar response pattern in kleptomania. Unfortunately, the role of the serotonergic or of any other neurotransmitter system has not been sufficiently investigated in kleptomania. Interestingly, a large study found subjects with mixed anorexia and bulimia nervosa to have a higher lifetime prevalence of kleptomania than those with either anorexia or bulimia nervosa alone (Herzog et al. 1992). This could suggest a relationship between kleptomania and both the obsessive-compulsive (anorexic) and the affective (bulimic) spectrum.

Alternatively, kleptomania may be conceptualized as an addictive disorder. The irresistible impulse to steal is reminiscent of the urge and highly associated with drinking or drug use (McElroy et al. 1992). Marks (1990) proposed a constellation of behavioral (i.e., nonchemical) addictions encompassing OCD, compulsive spending, gambling, binging, hypersexuality, and kleptomania. This model postulates certain concepts thought to be common in all these disorders, like craving, mounting tension, "quick fixing," withdrawal, external cuing, and habituation. These components have not yet been well investigated in kleptomania.

## Biological Theories

### Serotonin and Inhibition

Compared with controls, kleptomania patients report significant elevations of impulsivity and risk taking (Bayle et al. 2003, Grant and Kim 2002d), and diminished inhibitory mechanisms may underlie the risk-taking behavior of kleptomania. The most well-studied inhibitory pathways involve serotonin and the PFC (Chambers et al. 2003). Decreased measures of serotonin have long been associated with a variety of adult risk-taking behaviors including alcoholism, fire setting, and pathological gambling (Moreno et al. 1991, Virkkunen et al. 1994). Blunted serotonergic responses in the ventromedial PFC have been seen in people with impulsive aggression (New et al. 2002), and this region has also been implicated in poor decision making (Bechara 2003), as seen in those with kleptomania. Although there are few biological studies of kleptomania, early evidence supports a theory of serotonergic involvement in the disorder. One study found a lower number of the platelet serotonin transporter in kleptomania patients versus healthy controls (Marazziti et al. 2000). Pharmacological case studies suggest that SSRIs like clomipramine and SSRIs (Lepkifker et al. 1999, McElroy et al. 1991b) may reduce the impulsive behavior associated with kleptomania.

### Dopamine and Reward Deficiency

Dopaminergic systems influencing rewarding and reinforcing behaviors have also been implicated in ICDs and may play a role in the pathogenesis of kleptomania. One proposed mechanism is "Reward deficiency syndrome," a hypothesized hypodopaminergic state involving multiple genes and environmental stimuli that puts an individual at high risk for multiple addictive impulsive and compulsive behaviors (Blum et al. 2000). Alterations in dopaminergic pathways have been proposed as underlying the seeking of rewards (e.g., shoplifting) that trigger the release of dopamine and produce feelings of pleasure (Blum et al. 2000). Further, 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 behavior 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 GABAergic activities, appear to change in response to experiences that influence the function of the nucleus accumbens. So, future behavior may be determined in part by prior rewarding experiences via neuroplastic changes in the nucleus accumbens. This may explain why, over time, many kleptomania patients report shoplifting "out of habit" even without a pronounced urge or craving.

### Opioid System, Cravings, and Pleasure

Kleptomaniacs report frequent urges to steal, that result in theft twice weekly on average (Grant and Kim 2002b). Thus, urges linked to the experience of reward and pleasure may represent an important clinical target for treatment. Many indicate that the act of stealing reduces the urges or the tension these urges produce (McElroy et al. 1991b). While many report the urges as intrusive, the act of stealing is often a "thrill" for some, producing a pleasurable feeling (Goldman 1991, Grant and Kim 2002b). The μ-opioid system is thought to underlie urge regulation by processing reward, pleasure, and pain, in part through modulation of dopamine neurons in the mesolimbic pathway via γ-aminobutyric acid 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).

In sum, repeated kleptomanic behavior may be a result of an imbalance between a pathologically increased urge and a pathologically decreased inhibition. The repeated shoplifting may therefore be due to increased activity of the mesocorticolimbic dopamine circuitry, indirectly enhanced through the opioid system, and decreased activity in the cortical inhibitor processes, largely influenced via serotonin.

Numerous psychological formulations of kleptomania have also been postulated over the years. A frequent theme reported by many authors and reviewed by Goldman (1991) and McElroy et al. (1991b) is that of kleptomania as an acting-out aimed at alleviating depressive symptoms. Fishbain (1987) carefully described the case of a woman whose kleptomanic episodes were closely related to depressive bouts and who experienced an apparent antidepressant effect from the thrill and excitement of her risk-taking behavior. So kleptomania may result from an attempt to relieve feelings of depression through stimulation (Goldman 1991, McElroy et al. 1991a) and risk-taking behavior 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 in turn lead to even more shoplifting as a misguided attempt of symptom management (Goldman 1991). Supporting the self-medication hypothesis of shoplifting, patients with kleptomania report high lifetime rates of depression (45-100%; Bayle et al. 2003, McElroy et al. 1991b) that usually (60% of cases) precedes the kleptomanic behavior (McElroy et al. 1991b). Further, several case studies report patients who described shoplifting as relief for their depressed moods (Fishbain 1987) and suggest that kleptomania symptoms improve with antidepressants (Lepkifker et al. 1999, McElroy et al. 1991b).

From a psychodynamic point of view, kleptomania has been viewed over the decades as a manifestation of a variety of unconscious conflicts, with sexual conflicts figuring prominently in the literature. Case reports have described conscious sexual gratification, sometimes accompanied by frank masturbation or orgasm during kleptomanic acts (Fishbain 1987, Fenichel 1945). Thus, it has been suggested that kleptomanic behavior serves to discharge a sexual drive that may have forbidden connotations similar to those of masturbation, and the stolen object itself may have unconscious symbolic or overt fetishistic significance. Although no systematic studies exist, there has long been an implication in the literature on kleptomania that those afflicted with kleptomania suffer disproportionately from a variety of sexual dysfunctions. Turnbull (1987) described six patients with a primary diagnosis of kleptomania, all of whom had dysfunctional sexual relationships with their partners, compulsive promiscuity, or anorgasmia.

Other cases of kleptomania have been understood as reflecting conflictual infantile needs and attempts at oral gratification, masochistic wishes to be caught and punished related to a harsh guilt-inducing superego or primitive aggressive strivings, penis envy or castration anxiety with the stolen object representing a penis, a defense against unwelcome passive homosexual longings, restitution of the self in the presence of narcissistic injuries, or the acquisition of transitional objects (Beldoch 1991). These various formulations are presented in detail in Goldman's review (1991). Psychodynamic interpretations associated with kleptomania should be carefully tailored to the individual. The literature on kleptomania has frequently implicated disturbed childhoods, inadequate parenting, and significant character disturbances in kleptomanic patients. From this perspective kleptomania can be more effectively understood in the context of an individual's overall character. Unfortunately, no clinical studies exist that systematically explore Axis II psychopathology in these patients.

Behavioral models also provide clues as to the pathogenesis of kleptomania. From an operant viewpoint, the positive reinforcer in kleptomania is the acquisition of items for nothing, and the intermittent reinforcement (e.g., not always being able to shoplift because of store security) of kleptomanic behavior may therefore be particularly resistant to extinction. Physiological arousal related to shoplifting (Goldman 1991) may be another reinforcer that initiates and perpetuates the behavior. Negative reinforcement (i.e., the removal of a punishing stimulus) hypothesizes that shoplifting is performed to experience relief from the aversive arousal of urges. The self-medication theory of kleptomania may represent a negative reinforcement. This could explain why kleptomaniac behavior continues despite the offender being frequently apprehended.

There may also be specific cognitive errors that are directly linked to kleptomanic behavior: (1) believing that only shoplifting will reduce the urge or the depressive state, (2) selective memory (e.g., remembering the thrill of shoplifting and ignoring the shame and embarrassment from being apprehended), and (3) erroneous self-assessment (e.g., that one deserves to be caught stealing because one is not intrinsically worth anything). A biopsychological perspective will most likely provide the most useful understanding for the treatment and prevention of kleptomania.

## Treatment

### Treatment Goals

The general goal of treatment is the eradication of kleptomanic behavior. Treatment typically occurs in the outpatient setting, unless comorbid conditions like severe depression, eating disturbances, or more dangerous impulsive behaviors dictate hospitalization. In the initial contact with the psychiatrist, as described earlier, it is important that the appropriate differential diagnoses be considered. The interview must be conducted in a respectful climate that ensures confidentiality. Patients may not only experience considerable guilt or shame for stealing, but also may be unrevealing because of the fear of legal repercussions. In the acute treatment phase, the aim is to decrease significantly or, ideally, eradicate episodes of stealing during a period of weeks to months. Concurrent conditions may compound the problem and require independently targeted treatment.

The acute treatment of kleptomania has not been, to date, systematically investigated. Recommendations are based on retrospective reviews, case reports, and small case series. Maintenance treatment for kleptomania has not been investigated either, and only anecdotal data exist for patients who have been followed up for significant periods after initial remission.

### Psychiatrist-Patient Relationship

As with any condition that may be associated with intense guilt or shame, kleptomania must be approached respectfully by the psychiatrist. Patients can be reassured and their negative feelings alleviated to some degree with proper initial psychoeducation. The treatment alliance can be strengthened by consistently maintaining a nonjudgmental and supportive stance. In addition, patients' fears regarding breaks of confidentiality and criminal repercussions must be addressed.

No treatments have been systematically shown to be effective for kleptomania. These treatment recommendations are supported by case reports and retrospective reviews only. In general, it appears that thymoleptic medications and behavioral therapy may be the most efficacious treatments for the short term, while long-term psychodynamic psychotherapy may be indicated and have good results for selected patients.

### Somatic Treatments

No medication is currently approved by the US Food and Drug Administration for treating kleptomania. So,

it is important to inform patients of "off-label" uses of medications for this disorder and the empirical basis for considering medication treatment.

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) with mixed results. In a literature review of 56 kleptomania cases, McElroy et al. (1991a) noted that somatic treatments were described for 8 patients. Significant improvement was reported for seven of these. Treatment included antidepressants alone, antidepressants with antipsychotics or stimulants, electroconvulsive therapy alone, or electroconvulsive therapy with antidepressants. The medications most commonly used to treat kleptomania are the antidepressants. In a series of 20 patients fulfilling DSM-III-R criteria for kleptomania, McElroy et al. (1991b) found that 18 had received antidepressants and of those patients 10 had partial or complete remission of both kleptomanic urges and behavior. It has been suspected that kleptomania may respond selectively to SSRIs because of the anticompulsive and anti-impulsive properties of these compounds. Of these 18 patients, 10 were administered fluoxetine alone and only 2 had a full response and 1 had a partial response. These data are not suggestive of a high response rate to SSRIs, but dose and duration of treatment were not explicitly stated. In a report on three patients with concurrent DSM-III-R kleptomania and bulimia nervosa treated with serotonergic antidepressants, two received high-dose fluoxetine and one trazodone; all three showed significant improvement in kleptomania, independent of the course of bulimia nervosa and depression (McElroy et al. 1989). It is still unclear whether kleptomania responds preferentially to serotonergic antidepressants, and this question awaits further study. Other agents reported to have treated kleptomania successfully include nortriptyline (McElroy et al.1991b) and amitriptyline (Fishbain 1987). So, although little is known about maintenance pharmacological treatment for kleptomania, the literature suggests that symptoms tend to recur with cessation of thymoleptic treatment and again remit when treatment is reinstituted (McElroy et al.1991a, Fishbain 1987). But it remains unclear if the antikleptomanic effect of thymoleptics is dependent on or independent of their antidepressant effect.

A number of other medications have been employed to treat kleptomania. These include antipsychotics (McElroy et al.1991b, Fishbain 1987), stimulants (McElroy et al. 1991b), valproic acid (McElroy et al. 1991a), carbamazepine (McElroy et al. 1991a), clonazepam (McElroy et al. 1991a) and lithium (McElroy et al. 1991a, Monopolis and Lion 1983). Lithium augmentation may be of benefit when kleptomania does not respond to an antidepressant alone (Burstein 1992). Other agents used successfully as monotherapy for kleptomania include fluvoxamine (Chong and Low 1996) and paroxetine (Kraus 1999). Combinations of medications have also 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). Finally, there have been some reports of successful treatment of kleptomania with electroconvulsive therapy, which may have been administered for a concurrent mood disorder (McElroy et al. 1991b).

The findings from case reports have not been consistent. Seven cases of fluoxetine, three of imipramine, two of lithium as monotherapy, two of lithium augmentation, four of tranylcypromine, and one of carbamazepine combined with clomipramine all failed to reduce kleptomania symptoms (McElroy et al. 1991b). Some evidence suggests that SSRIs may even induce kleptomania symptoms (Kindler et al. 1997). A case series found that kleptomania symptoms respond to topiramate (Dannon 2003). In another case series, the two subjects treated with naltrexone responded (Dannon et al. 1999).

In the only open-label trial for kleptomania, naltrexone resulted in a significant decline in the intensity of urges to steal, stealing thoughts, and stealing behavior (Grant and Kim 2002c) (mean effective dosage 145 mg/day). A lower dosage, possibly 50 mg/day, may be effective in younger people with kleptomania (Grant and Kim 2002a). Opioid antagonists like naltrexone may be effective in reducing both the urges to shoplift and shoplifting behavior, by reducing the "thrill" associated with shoplifting and thus preventing the positive reinforcement of the behavior. Antidepressants, particularly those that influence serotonergic systems (e.g., SSRIs), may also be effective in reducing the symptoms of kleptomania, by targeting serotonergic systems implicated in impaired impulse regulation. If kleptomania represents both impaired urge regulation and inhibition of behavior, both opioid antagonists and antidepressants may play a role in controlling this behavior.

## Psychosocial Treatments

Formal studies of psychosocial interventions for kleptomania have not been performed. However, a number of clinical reports have supported behavioral therapy for kleptomania. Different behavioral techniques have been employed with some success, including aversive conditioning, systematic desensitization, covert sensitization, and behavior modification. In their review of 56 cases of kleptomania, McElroy et al. (1991a) noted that the 8 patients who were treated with behavioral therapy—mostly aversive conditioning—showed significant improvement. We give here some specific examples of behavioral techniques that have been successfully employed and described. One patient was taught to hold her breath as a negative reinforcer whenever she experienced an impulse to steal (Keutzer 1972). Another patient was taught to use systematic desensitization techniques to control the mounting anxiety associated with the impulse to steal (Marzagao 1972). A patient treated by covert sensitization learned to associate images of nausea and vomiting with the desire to steal (Glover 1985). A woman who experienced sexual excitement associated with shoplifting and would masturbate at the site of the act was instructed to practice masturbation at home, while fantasizing kleptomanic acts (Fishbain 1987). The literature suggests that these techniques remain effective over the long term (Gauthier and Pellerin 1982, Glover 1985). In imaginal desensitization the patient imagines the steps of stealing and her ability to not steal in that context, while maintaining a relaxed state. Undergoing fourteen 15-minute sessions over 5 days, two patients reported complete remission of symptoms for a 2-year period (McConaghy and Blaszczynski 1988). Learning to substitute alternative sources of satisfaction and excitement when urges to steal occur has been successful in a

woman treated weekly for 5 months, who reported 2 years of remitted symptoms (Gudjonsson 1987).

It appears that the most effective behavioral treatment of all may be complete abstinence, that is, the patient should no longer visit any of the stores or settings where kleptomanic acts occur. A number of patients who never come to psychiatric attention apparently employ this technique successfully, and it may be an appropriate treatment goal if it does not result in excessive restrictions of activity and lifestyle.

The clinical literature suggests that for most patients, behavioral therapy may be a more efficacious approach than insight-oriented psychotherapy. Insight-oriented psychotherapy, however, has been unsuccessful in treating this disorder in 11 published cases (McElroy et al. 1991b). Psychoanalysis has resulted in some limited success for kleptomania symptoms, but usually with the addition of medication (Fishbain 1988, Schwartz 1992). The psychodynamic treatment of kleptomania centers on the exploration and working through of the underlying conflict or conflicts. In a review of 26 case reports, McElroy et al. (1991a) reported that 4 of 5 patients had a good response to psychoanalysis or related therapy. However, in another review of 20 cases (meeting DSM-III-R criteria) McElroy et al. (1991b) reported that of 11 patients treated with psychotherapy, none showed improvement. There are case reports in the literature of successful psychodynamic treatment of kleptomania (Schwartz 1992). Such treatment, possibly in combination with other approaches, may be indicated for patients for whom a clear conflictual basis for the behavior can be formulated, who also have the needed insight and motivation to undertake this type of treatment. In proposing such treatments, which may be long term, the psychiatrist should consider whether there are immediate risks that must be addressed, such as a high risk of legal consequences.

As few empirical studies are available, research is needed to guide the selection of which psychotherapy to use, and to investigate of the combination of medication and psychotherapy in treating kleptomania.

### Special Treatment Considerations
Little is known about treating kleptomania and therefore special treatment considerations have not been elucidated. However, it is clear that comorbid conditions, like depression, bulimia nervosa, OCD, or substance abuse, must be addressed along with the kleptomania. In addition to the inherent suffering and morbidity of these other disorders, their course and severity could compound the kleptomanic behavior. In the rare cases of a precipitating or exacerbating organic etiology, the underlying organic cause must be treated. In addition, the treatment of particular groups such as children or the elderly should take into account special contributing life stage or situational factors. The involvement of family or others on whom the patient is dependent may be indicated.

### Refractoriness to Initial Treatment
There has not been sufficient study of the treatment of kleptomania to systematically delineate approaches to the refractory patient. However, general clinical principles can be applied. Medication trials should be maximized, predominantly employing antidepressants and mood stabilizers, alone or in combination. In addition, it is important that comorbid conditions such as depression or OCD be monitored and treated, because they complicate the course of kleptomania. For patients who have no response or a partial response to pharmacotherapy alone or who do not want medication treatment, behavioral therapy is indicated. Behavioral therapy can be used alone or in combination with medication. There are no systematic comparisons of medication, behavioral therapy, or combined treatments. Therefore, the initial treatment choice is based in the assessment of the particular circumstances of each presentation. The patient's past treatment history, comorbid diagnoses, and personal resources should be weighed in choosing a course of treatment. Finally, there may be refractory patients for whom a multiple combination approach is helpful. Fishbain (1987) described the treatment of a middle-aged woman with a long history of kleptomania, depression, and suicidality and extensive past psychiatric treatments who responded to a combination of supportive and insight-oriented therapy, medication, and behavior modification.

## Pyromania and Fire Setting Behavior

### Diagnosis

#### Definition and Diagnostic Features
The primary characteristics of pyromania are recurrent, deliberate fire setting, the experience of tension or affective arousal before the fire setting, an attraction or fascination with fire and its contexts, and a feeling of gratification or relief associated with the fire setting or its aftermath. True pyromania is present in only a small subset of fire setters. Prins et al. (1985) have suggested the following motivations for intentional arson: financial reward, to conceal another crime, for political purposes, as a means of revenge, as a

---

**DSM-IV-TR Criteria**

**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 sociopathical 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

Reproduced with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Copyright 2000 American Psychiatric Association.

symptom of other (nonpyromania) psychiatric conditions (e.g., in response to a delusional belief), as attention-seeking behavior, as a means of deriving sexual satisfaction, and as an act of curiosity when committed by children. Revenge and anger appear to be the most common motivations for fire setting (O'Sullivan and Kelleher 1987). Fire setting may also be associated with other psychiatric diagnoses (see differential diagnosis section). Fire-setting behavior may be a focus of clinical attention, even when criteria for pyromania are not present. Because the large majority of fire-setting events are not associated with true pyromania (which is rare), this section also addresses fire-setting behavior in general. Recent diagnostic classifications include pyromania among the ICDs and fire setting is thought to result from a failure to resist an impulse. Although pyromaniacs may methodically prepare the fire and leave obvious clues of this preparation behind (Wise and Tierney 1999), pyromania is still considered an uncontrolled and often impulsive behavior.

The diagnosis of pyromania emphasizes the affective arousal, thrill, or tension preceding the act, as well as the feeling of tension relief or pleasure in witnessing the outcome. This is useful in distinguishing between pyromania and fire-setting elicited by other motives (i.e., financial gain, concealment of other crimes, political, arson related to other mental illness, revenge, attention seeking, erotic pleasure, a component of conduct disorder). In children and adolescents, the most common elements are excitation caused by fires, enjoyment produced by fires, relief of frustration by fire setting, and expression of anger through fire setting (Brandford and Dimock 1986). The most frequent motives for arson by juveniles (RŠsŠnen et al. 1995) are revenge on parents or other authorities, the search for heroism or excitement, self-destructiveness, the craving for sensation, and an expression of outrage. There is also a lot of self-destructive behavior by juveniles before committing arson; 74% have suicidal thoughts and 44% have tried to commit suicide before committing their crimes. Females with pyromania frequently have a history of self-harm, sexual abuse, and psychosocial traumas (Noblett and Nelson 2001). Thus, pyromania could be a displacement of aggression in people with a history of sexual trauma. The channeling of aggression by their fire setting may be an attempt to influence their environment and improve their self-esteem where other means have failed. Fire setting may also be an attempt at communication by individuals with few social skills (Geller and Bertsch 1985).

Pyromania onset has been reported to occur as early as age of 3 years, but it may initially present in adulthood. Because of the legal implications of fire setting, individuals may not admit previous events, which may result in biased perceptions of the common age at onset. Men greatly outnumber women with the disorder. Further, nongeriatric state hospital inpatients who had engaged in fire-setting behavior were significantly more likely to have a history of nonlethal self-injurious behavior and had a significantly greater number of admissions to the state hospital (Geller and Bertsch 1985).

## Assessment

### The Psychiatric Interview

The interviewer must bear in mind that the circumstances of arson, whatever the motive, may pose legal and criminal problems for the individual. This may provide motivation to skew the reporting of events. Individuals who may be at risk for the legal consequences of fire setting may be motivated to represent themselves as victims of psychiatric illness, hoping that a presumed psychiatric basis of the behavior may attenuate legal penalties. Thus, the interviewer must maintain a guarded view of the information presented.

## Epidemiology

Most epidemiological studies have not directly focused on pyromania but instead on various populations of arsonists or fire setters. Most studies suggest that true pyromania is rare and reveal a preponderance of males with a history of fire fascination (Barker 1994). According to DSM-IV-TR, pyromania occurs more often in males, especially those with poorer social skills and learning difficulties. This notation confirms the Lewis and Yarnell (1951) data that only 14.8% of those with pyromania are female. Fire setting for profit or revenge or secondary to delusions or hallucinations is more frequent than "authentic" ICD. Although pyromania is a rare event, fire-setting behavior is common in the histories of psychiatric patients. Geller and Bertsch (1985) found that 50 (26%) of 191 nongeriatric state hospital patients had histories of some form of fire-setting behavior. Unlike pyromania, which is rare among women, fire-setting behavior was common in the histories of female patients (22%), as well as male patients (28.8%).

"True" pyromania in childhood is rare, but fire setting is frequent in children and in adolescents. Juvenile fire setting is most often associated with conduct disorder, ADHD, or adjustment disorder. Among children with psychiatric conditions, fire-setting behavior is quite common. Kolko and Kazdin (1988) found that among a sample of children attending an outpatient psychiatry clinic, approximately 20% had histories of fire setting. For a sample of inpatient children, the rate was approximately 35% (Kolko and Kazdin 1988).

The classic study *Pathological Fire-Setting (Pyromania)* by Lewis and Yarnell (1951) is one of the largest epidemiological studies of this topic and includes approximately 2000 records from the National Board of Fire Underwriters and cases provided from fire departments, psychiatric clinics and institutions, and police departments near New York City. Thirty-nine percent of the fire setters from the study received the diagnosis of pyromania. Twenty-two percent had borderline to dull normal intelligence, and 13% had between dull and low average intelligence. Fire setters were also described as driven by an irresistible impulse to set fires. The peak incidence of fire setting was between the ages of 16 and 18 years although. This observation has not been confirmed by more recent studies. The reported high prevalence rates of pyromania have not been confirmed by more recent studies. Koson and Dvoskin (1982) found no cases of pyromania in a population of 26 arsonists. Ritchie and Huff (1999) identified only 3 cases of pyromania in 283 cases of arson.

## Comorbidity Patterns

Limited data are available regarding individuals with pyromania. Reported data of comorbid diagnoses are generally derived from forensic samples and do not distinguish between criminally motivated fire setters and compulsive fire setters. Fire-setting behavior may be associated with other

mental conditions like mental retardation, conduct disorder, alcohol and other substance use disorders, personality disorders, and schizophrenia. In most cases, fire-setting behavior is not directly related to pyromania. On the other hand, fire setting in subjects who do not have pyromania appears frequent and often underrecognized. Among psychiatric patients, Geller and Bertsch (1985) found that 26% of the patients had a history of fire-setting behavior, and 16% of these patients had actually set fires. Ritchie and Huff (1999) reviewed mental health records and prison files from 283 arsonists, 90% of whom had a recorded history of mental health problems. Thirty-six percent had schizophrenia or bipolar disorder, and 64% were misusing alcohol or drugs at the time of their fire setting.

Repo et al. (1997) examined the medical and criminal records of 282 arsonists in order to compare first time and repeat offenders. They found that alcohol dependence and ASPD were common among recidivist offenders, especially among those who committed violent crimes. Recidivist offenders commonly had a history of enuresis during their childhood, were younger than first time offenders at the time of their first offence, and were more often intoxicated with alcohol during the arson attempt. Psychosis was common among those with no record of recidivist criminal offences. Puri et al. (1995) examined a group of 36 forensically referred fire setters. They found that about one-third had no other evidence of mental illness, about a quarter were female, psychoactive substance abuse was common and interpersonal relationships were often disturbed. Lejoyeux et al. (2002) assessed ICDs, using the Minnesota Impulsive Disorders Interview, in 107 depressed inpatients who met DSM-IV-TR criteria for major depressive episodes. Thirty-one depressed patients met criteria for ICDs: 18 had IED, 3 had pathological gambling, 4 had kleptomania, 3 had pyromania, and 3 had trichotillomania. Patients with pyromania had a higher number of previous depressions (3.3 versus 1.3, $P = 0.01$). Bipolar disorders were more frequent in the ICD group than in the group without ICDs (19% versus 1.3%, $P = 0.002$).

Laubichler et al. (1996) compared the files of 103 criminal fire setters and subjects with pyromania. Subjects with pyromania were younger (average age 20 years) than criminal fire setters (average age 30 years). Seventy of the 103 subjects had consumed alcohol before setting a fire. Fifty-four presented with alcohol dependence. The authors suggested a correlation between the amount of alcohol consumed and the frequency of fire setting. RŠsŠnen et al. (1995) found that young arsonists have frequent alcohol problems: 82% had alcoholism and 82% were intoxicated at the time of committing the crime. The excessive consumption of alcohol had a close connection with the arson committed. Lejoyeux et al. (1999) searched for ICDs among consecutive admissions for detoxification of alcohol-dependent patients in a French department of psychiatry. They found 30 alcohol-dependent persons presenting with at least one ICD (19 with IED, 7 with pathological gambling, 3 with kleptomania, and 1 case of trichotillomania), but none of the patients presented with two or more ICDs, and no patient presented with pyromania. However, it cannot be concluded from such a limited population that pyromania is not associated with alcohol dependence. Further studies are needed.

## Course

According to the DSM-TR, there are insufficient data to establish a typical age at onset of pyromania and to predict the longitudinal course. However, the impulsive nature of the disorder suggests a repetitive pattern. Again, because legal consequences may occur, the individual may be motivated to represent the index episode as a unique event. Fire setting for nonpsychiatric reasons may be more likely to be a single event. 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). In a cross-sectional and 10-year follow-up study Barnett et al. (1997, 1999) compared mentally ill and mentally "healthy" fire setters from trial records in Germany where a defendant with a psychiatric disorder can be found to be not responsible, to have diminished responsibility, or to be fully responsible. Mentally disordered arsonists were more likely than those with no disorder to have a history of arson before their trial, were more often convicted of arson again (11% relapse compared with 4%), had fewer registrations of common offenses like theft, traffic violations, and alcohol-related offenses, had a higher rate of recurrence, and committed fewer common offenses other than fire setting. Among all arsonists who committed crimes in addition to arson, those who were found to be partly responsible for their arson committed the highest number of offenses followed by those who were deemed not responsible for their actions and those who were fully responsible.

## Differential Diagnosis

Other causes of fire setting must be ruled out. Fire-setting behavior may be motivated by circumstances unrelated to mental disorders. Such motivations include profit, crime concealment, revenge, vandalism, and political statement or action (Geller 1987, Lowenstein 1989). Furthermore, fire setting may be a part of ritual, cultural, or religious practices in some cultures.

Fire setting may occur in the presence of other mental disorders. A diagnosis of fire setting is not made when the behavior occurs as a part of conduct disorder, ASPD, or a manic episode or if it occurs in response to a delusion or hallucination. The diagnosis is also not given if the individual suffers from impaired judgment associated with mental retardation, dementia, or substance intoxication.

## Etiology and Pathophysiology

Because pyromania is rare, there is little reliable scientific literature available regarding individuals who fit diagnostic criteria. But because of the morbid impact that arson has on society, fire-setting behavior (which often does not fulfill criteria for pyromania) has been the focus of scientific investigation and literature.

Arson has been the subject of several investigations of altered neuroamine function. These findings include the observation that platelet monoamine oxidase is negatively correlated with fire-setting behavior of adults who had been diagnosed with attention-deficit disorder in childhood (Kuperman et al. 1988). Investigation of the function of serotonergic neurotransmission in individuals with aggressive and violent behaviors has included studies of CSF

concentrations of 5-HIAA in individuals with a history of fire setting. 5-HIAA is the primary metabolite of serotonin, and its concentration in the CSF is a valid marker of serotonin function in the brain. Virkkunen et al. (1987, 1994) demonstrated that impulsive fire setting was associated with low CSF concentrations of 5-HIAA. This finding was consistent with other observations associating impulsive behaviors with low CSF 5-HIAA levels (like impulsive violence and impulsive suicidal behavior). A history of suicide attempt strongly predicts recidivism of arson (DeJong et al. 1992).

Impulse fire setters who are violent offenders are often dependent on alcohol and have an alcohol-dependent father (Linnoila et al. 1989). Virkkunen et al. (1996) investigated biochemical and family variables and predictors of recidivism among forensic psychiatric patients who had set fires. Male alcoholic patients and fire setters ($N = 114$) were followed for an average of 4.5 years after their release from prison. Low CSF 5-HIAA and homovanillic acid concentrations were associated with a family history of paternal alcoholism with violence. A low plasma cholesterol concentration was associated with a family history positive for paternal alcoholism without violence. Compared with nonrecidivists, the recidivists, who set fires during the follow-up period, had low CSF 5-HIAA and MHPG (a metabolite of norepinephrine) concentrations and early family environments characterized by paternal absence and the presence of brothers at home.

Psychodynamic models refer to the symbolism of fire which is complemented by "normal" human interest in fire. Fire interest starts between the ages of 2 and 3 years and was almost universal in a study of normal schoolboys at the ages of 6, 8, and 10 years (Kafry, 1980). The distinction between normal interest in fire and excessive interest leading to pyromania is not always clear among children. Playing with matches is not a symptom of pyromania. Kolko and Kazdin (1989) showed that "future" pyromaniacs had more curiosity about fire and liked to be exposed to people (parents/peers) who are involved with fire. According to Geller and Bertsch (1985), children at risk of pyromania were more often involved in fire setting, threatening to set a fire, sounding a false fire alarm, or calling the fire department with a false report of fire than were control subjects. Thus, there may be a continuum between excessive interest in fire and "pure" pyromania.

Since the first description of pyromania in 1833 by the French psychiatrist Marc, the symbolic sexual dimension of pyromania has been noted. Many pyromaniacs were later described as having fire fetishes. A "fire experience" may become a "fire fetish" via conditioning with positive feedback by for example imagining/recalling a fire fantasy just before orgasm (McGuire et al. 1965). Lewis and Yarnell (1951) suggest three main groups of fire setters: the accidental, the occasional, and the habitual.

## Treatment

### Treatment Goals

Because of the danger inherent in fire-setting behavior, the primary goal is elimination of the behavior. The treatment literature does not distinguish between pyromania and fire-setting behavior of other causes. Much of the literature is focused on controlling fire-setting behavior in children and adolescents.

### Psychiatrist-Patient Relationship

Because of the potential legal risks for people who acknowledge fire-setting behavior, the psychiatrist must take particular pains to ensure an environment of empathy and confidentiality. A corollary concern involves obligations that may be incumbent on the psychiatrist. Because of the legal implications of these behaviors and the potential for harm to another individual should fire setting recur, psychiatrists should consider both the ethical and the legal constraints that may follow from information learned in the course of treatment.

### Somatic Treatments

There are no reports of pharmacological treatment of pyromania. Because fire setting may be frequently embedded in other psychiatric illness, therapeutic attention may be directed primarily to the underlying disorder. However, the dangerous nature of fire setting requires that the behavior be controlled. Much in the same fashion that one would seek to educate impaired patients about the functional risks associated with their symptoms—and to establish boundaries of acceptable behavior—the fire-setting behavior must be directly addressed, even if it is not a core symptom of the associated disorder.

### Psychosocial Treatments

Treatment for fire setters is problematic because they frequently refuse to take responsibility for their acts, are in denial, have alcoholism, and lack insight (Mavromatis and Lion 1977). It has been estimated that up to 60% of childhood fire setting is motivated by curiosity. Such behavior often responds to direct educational efforts. In children and adolescents, focus on interpersonal problems in the family and clarification of events preceding the behavior may help to control the behavior (Lowenstein 1989). Principles of CBT have been also applied to childhood fire setting (Kolko 2001).

Treatments for fire setting are largely behavioral or focused on intervening in family or intrapersonal stresses that may precipitate episodes of fire setting. Behavioral treatments like aversive therapy have helped fire setters (McGrath and Marshall 1979, Koles and Jenson, 1985). Other treatment methods rely on positive reinforcement with threats of punishment an dstimulus satiation (Bumpass et al. 1983). Bumpass et al. (1983) treated 29 child fire setters and used a graphing technique that correlated external stress, behavior, and feelings on graph paper. After treatment (average follow-up, 2.5 years), only 2 of the 29 children continued to set fires. Relaxation training may also be used (or added to graphing techniques) to assist in the development of alternative modes of dealing with the stress that may precede fire setting. Another technique combines overcorrection, satiation, and negative practice with corrective consequences. The child is supervised in constructing a controlled, small fire in a safe location, which is then extinguished by the child. Throughout the process, the parent verbally instructs the child in safety techniques. Franklin et al. (2002b) confirmed the positive effect of a prevention program for pyromania. In 1999, they developed the Trauma Burn Outreach Prevention Program. All subjects arrested and convicted after setting a fire received 1 day of information. The program's interactive content focused on the medical, financial,

A 34-year-old man came to a medical emergency department for the treatment of third-degree burns on his hands and face. He claimed to have been accidentally caught in a fire at a warehouse. Because of the patient's severe agitation and inability to explain the circumstances of the injury coherently, the treating surgeon asked that the patient be seen by a psychiatrist.

On meeting the psychiatrist the patient became even more severely agitated. He began to complain of the pain caused by his burns and was reluctant to speak with the psychiatrist. The patient insisted that he was in substantial pain and that he had no need to speak with "some shrink." Because the patient was going to be admitted for medical monitoring, the psychiatrist withdrew, planning to visit the patient again the next day in his hospital room. The next day the young man was more amenable to an interview. At this time he seemed sad and, although anxious, less visibly agitated than he was on the preceding day. He no longer questioned the psychiatrist's purpose in visiting him and participated in a brief discussion about his burns, the pain they caused, and the misfortune he suffered, having been caught in a fire. The psychiatrist again decided to withdraw after this brief conversation. Despite the passive cooperation the patient offered, the psychiatrist was still impressed with how guarded he seemed about the question of the events that led up to the fire. The psychiatrist concluded that the patient seemed to want to avoid discussing the details and decided that several visits might be necessary to engage the patient sufficiently to obtain an adequate history.

On the following day the patient seemed relieved when the psychiatrist entered the room. He said that he had something to tell the psychiatrist. He then proceeded to describe a history of fascination with fire since the age of 16 years. He had set a couple of small fires in wastebaskets at that age and found himself drawn to trade magazines that specialized in fire control equipment. He would often walk by the local firehouse and tried to follow the fire crews when they responded to a fire alarm. For a number of years he was aware of a growing urge to set fires. He worried about this compulsion and managed to avoid acting on it.

In the past 3 years his forbearance began to erode. In that period he had set several fires in isolated parts of the city. He was careful to do so in areas where he knew few people might be caught in the fire. He tried to arrange circumstances in which the fire would be quickly discovered. Indeed, he reported one of the fires himself—both because he was fearful of the harm that might occur and because he had a great urge to see the firefighters arrive and battle the flames. In a recent fire a firefighter had been mildly injured. At that point he realized the dangers of his compulsion. Several days ago he went out to set another fire. He did not realize how quickly the fire would progress and he was injured. After telling the psychiatrist this story he expressed great relief that he finally had shared his shame with someone. He also expressed the hope that it would be understood that he suffered from a compulsion and asked the psychiatrist if there might be some way to reduce or erase the need to set fires. He realized he faced criminal prosecution but felt relieved that his behavior had been interrupted before another person was seriously hurt. Eventually this patient committed to treatment and his pyromanic behavior remitted with a combination of cognitive−behavior therapy and medication treatment.

legal, and societal impact of fire-setting behavior. The rate of recidivism was less than 1% in the group who attended the program, versus 36% in the control group.

## Pathological Gambling

## Diagnosis

### Definition and Diagnostic Features
Pathological gambling has been considered a distinct diagnostic entity since 1980, when it was first included in DSM-III (American Psychiatric Association 1980). DSM-IV-TR (American Psychiatric Association 2000) currently classifies pathological gambling as an ICD not

**DSM-IV-TR Criteria**

**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.

Reproduced with permission from the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision. Copyright 2000 American Psychiatric Association.

elsewhere classified. Gambling as a behavior is common. Current estimates suggest that approximately 80% of the adult population in the US gamble. The amount of money wagered legally in the US grew from $17 to $210 billion in from 1974 to 1988, an increase of more than 1200%, making gambling the fastest growing industry in America at the time (Lesieur and Rosenthal 1991). DSM-IV-TR, like DSM-III-R before it, covertly recognized the ubiquity of gambling behavior and the desire to gamble by the careful wording of criterion A for pathological gambling: "Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following." This definition of pathological gambling differs from some other definitions of ICDs not elsewhere classified, which are worded as "Failure to resist an impulse to." This difference implies that neither gambling behavior nor failure to resist an impulse to engage in it is viewed as pathological in and of itself. Rather, the maladaptive nature of the gambling behavior is the essential feature of pathological gambling and defines it as a disorder.

## Assessment

The most established measure for pathological gambling is the South Oaks Gambling Screen (SOGS) (Lesieur and Blume 1987, 1993). It is a 20-item questionnaire, which assesses recurrent and maladaptive gambling behavior that disrupts personal, family, and vocational pursuits. However, the SOGS has some limitations in that it does not correspond exactly with the DSM-IV diagnosis of pathological gambling or take into account frequency of gambling behaviors. While the SOGS is a self-report screening measure for PG, the Pathological Gambling Modification of the Yale-Brown Obsessive-Compulsive Scale (PG-YBOCS) (DeCaria et al. 1998b) is a clinician-rated outcome measure of PG. The PG-YBOCS consists of 10 questions that measure the severity and change in severity of pathological gambling symptoms over a recent time interval (usually within the past 1/2 week(s)). The first five questions assess urges and thoughts associated with pathological gambling, while the last five questions assess the behavioral component of the disorder. Although the PG-YBOCS is a relatively new measure of PG, it is one of the most widely used clinician-rated measures of PG. In addition to the SOGS and PG-YBOCS, the Gambling Symptom Assessment Scale (Kim et al. 2001) is a 12-item self-rated outcome measure designed to assess gambling symptom severity and change of gambling symptoms during treatment. It is an alternative pathological gambling measure that can be used in clinical studies.

It is not difficult to diagnose pathological gambling once one has the facts. It is much more of a challenge to elicit the facts, because the vast majority of patients with pathological gambling view their gambling behavior and gambling impulses as ego-syntonic and may often lie about the extent of their gambling (criterion A7). Patients with pathological gambling may first seek medical or psychological attention because of comorbid disorders. Given the high prevalence of addictive disorders in pathological gambling and the increased prevalence of pathological gambling in those with alcoholism and other substance abuse, an investigation of gambling patterns and their consequences is warranted for any patient who presents with a substance abuse problem. Likewise, the high rates of comorbidity with mood disorders suggest the utility of investigating gambling patterns of patients presenting with an affective episode.

The spouses and significant others of patients with pathological gambling deserve special attention. Individuals with pathological gambling usually feel entitled to their behavior and often rely on their families to bail them out (criterion A10). As a consequence, it is often the spouse of the patient with pathological gambling who first realizes the need for treatment and who bears the consequences of the disorder. Lorenz (1981) conducted a survey of 103 wives of pathological gamblers who attended Gam-Anon meetings (for family members of patients with pathological gambling). She found that most spouses had to borrow money and were harassed or threatened by bill collectors. Most spouses physically assaulted the gambler, verbally abused their children, and experienced murderous or destructive impulses toward the gambler. Although the gamblers themselves appeared less violent than the general population norms, their spouses were more violent, possibly because of desperation and anger. Eleven percent of the spouses of patients with pathological gambling admitted to having attempted suicide, and this result was replicated in a later study. These findings have two main implications for the assessment of pathological gambling: first, the spouse may be a valuable and motivated informant who should be questioned about the patient's behavior, and second, spouses should be specifically asked about the effects of the patient's illness on their own well-being and functioning and about suicidal ideation and attempts and the control of their own impulsivity.

## Epidemiology

Pathological gambling is considered to be the most common of the ICDs not elsewhere classified. The number of people whose gambling behavior meets criteria for pathological gambling in the US is estimated to be between 2 and 6 million (Volberg and Steadman 1988) . Surveys conducted between 1986 and 1990 in Maryland, Massachusetts, New York, New Jersey, and California estimated the prevalence of "probable pathological gamblers" among the adult population to be between 1.2% and 2.3%. These states have a broad range of legal wagering opportunities and a heterogeneous population. Similar surveys in Minnesota and Iowa, states with limited legal wagering opportunities and more homogeneous populations, yielded prevalence rates of 0.9% and 0.1%, respectively (Rosenthal 1992). Thus, availability of gambling opportunities and demographic makeup may influence the prevalence of pathological gambling. A 1998 study of national prevalence, using DSM-IV-TR criteria, determined that the prevalence of pathological gambling was 1.2% (1.7% for men and 0.8% for women). In addition to those who fulfilled DSM-IV-TR criteria, the researchers classified an additional 1.5% as "problem gamblers." The combined total of "pathological gamblers" and "problem gamblers" is 5.5 million adult Americans (Gerstein et al. 1999). During the past 20 years, many states have turned to lotteries as a way of increasing their revenues without increasing taxes. Currently, some form of gambling is legal in 47 of the 50 states, as well as in more than 90 countries worldwide. From 1975 to 1999 revenues from legal gambling in the US has risen from $3 to 58 billion (Volberg 2002) thus the prevalence and incidence of pathological gambling are expected to increase. A meta-analysis of 120 published stud-

ies indicated that the lifetime prevalence of serious gambling (meeting DSM criteria for pathological gambling) among adults is 1.6% (Shaffer et al. 1999). Among those younger than 18 years, the prevalence is 3.9%, with past-year rates for adults and adolescents being 1.1% and 5.8%, respectively (Shaffer and Hall 1996). Although it is illegal for adolescents to gamble, this high rate may be due to illegal gambling of adolescents (e.g. via the internet or gambling with peers). Women make up about one-third of all Americans with pathological gambling. However, they are underrepresented in GA, in which only 2% to 4% of the members are women. This pattern is echoed in England and Australia, where women make up 7% and 10% of Gamblers Anonymous (GA) members, respectively. The reason for this discrepancy may be the greater social stigma attached to pathological gambling in women and their characteristic pattern of solitary gambling. Nonwhites and those with less than a high-school education are more highly represented among pathological gamblers than in the general population. The demographic makeup of patients in treatment for pathological gambling differs substantially from the demographics of all patients with pathological gambling. Jewish persons are overrepresented in treatment settings and in GA, while women, minorities, and those younger than the age of 30 years are underrepresented in GA and in treatment (Lesieur and Rosenthal 1991).

## Comorbidity Patterns

Overall, patients with pathological gambling have high rates of comorbidity with several other psychiatric disorders and conditions. Individuals presenting for clinical treatment of pathological gambling apparently have impressive rates of comorbidity. Ibanez et al. (2001) reported 62.3% of one group seeking treatment had a comorbid psychiatric disorder. The most frequent current comorbid disorders were personality disorders (42%), alcohol abuse or dependence (33.3%), adjustment disorders (17.4%), and mood disorders (8.7%) Lifetime comorbid diagnoses included alcohol abuse or dependence (34.8%), mood disorders (15.9%), and anxiety disorders (7.2%). The relatively low rate of comorbid mood disorders compared to the rates reported below may be due to the outpatient setting of this study.

### Mood Disorders

Pathological gambling is highly comorbid with affective disorders, among inpatient (McCormick et al. 1984) and outpatient samples (Linden et al. 1986). In several surveys, between 70% and 80% of all patients with pathological gambling also had mood symptoms that met criteria for a major depressive episode, a manic episode, or a hypomanic episode at some point in their life. More than 50% had recurrent major depressive episodes (Lesieur and Rosenthal 1991). A complicating factor is that recovering pathological gamblers may experience depressive episodes after cessation of gambling. In addition, some pathological gamblers may gamble to relieve feelings of depression (criterion A5). Despite criterion B for pathological gambling, which essentially precludes the diagnosis of pathological gambling if the behavior occurs exclusively during the course of a manic episode, many patients have a disturbance that meets criteria for both disorders because they gamble both during and between manic and hypomanic episodes. Between 32% and

46% of patients with pathological gambling were reported also to have mood symptoms that meet criteria for bipolar disorder, bipolar II disorder, or cyclothymic disorder (McElroy et al. 1992).

### Suicide

Although data are not yet conclusive, a meaningful association between problem gambling and suicidal behavior and/or ideation appears to exist. Phillips et al. (1997) conclude that "Las Vegas, the premier US gambling setting, displays the highest levels of suicide in the nation, both for residents of Las Vegas and for visitors to that setting. In general, visitors to and residents of major gaming communities experience significantly elevated suicide levels. In Atlantic City, abnormally high suicide levels for visitors and residents appeared only after gambling casinos were opened. The findings do not seem to result merely because gaming settings attract suicidal individuals." Others report that between 12% and 24% of pathological gamblers in various settings have had a history of at least one suicide attempt. In one study, 80% of pathological gamblers had a history of either suicide attempts or ideation (Lesieur and Rosenthal 1991).

### Substance Abuse and Dependence

There appears to be a strong relationship between pathological gambling and substance abuse as evidenced by the high rates of comorbid substance abuse and dependency with pathological gambling (Lesieur et al. 1986, McCormick et al. 1984, Linden et al. 1986, Lesieur 1988). Studies of prevalence of comorbid substance use disorders yield widely varying results; from 9.9% for alcohol and other substance dependence (Gerstein et al. 1999) to 44% for alcohol dependence and 40% for illicit drug dependence (Bland et al. 1993, Cunningham-Williams et al. 1998). Using a structured instrument, between 5% and 25% of substance-abusing patients in several settings were found to meet criteria for pathological gambling and an additional 10% to 15% were considered to have "gambling problems" (Lesieur and Rosenthal 1991). Among individuals with pathological gambling, individuals with higher socioeconomic status (SES) are more likely to have concurrent problems with alcohol abuse then are gamblers with lower SES (Welte et al. 2001). Failure to treat comorbid substance use disorders in gamblers may lead to higher relapse rates (Maccallum and Blaszczynski 2002).

### Other Disorders

Again, current data are inconclusive, but OCD, panic disorder, generalized anxiety disorder, and eating disorders have all been reported to be present in higher rates in patients with pathological gambling than in the general population. Pathological gambling has been described as being part of the obsessive-compulsive spectrum and sharing features with both OCD and impulsive cluster of obsessive-compulsive spectrum disorders (Dell'Osso et al. 2005, Bienvenu et al. 2000). The reported prevalence of OCD among pathological gamblers ranges from 0.9% (Cunningham-Williams et al. 1998) to 16% (Bland et al. 1993). Pathological gambling has also been associated with ADHD (Carlton and Goldstein 1987). Retrospective studies suggest that many pathological gamblers have had symptoms that met criteria for ADHD as children (McElroy et al. 1992). Compulsive sexual behavior,

compulsive buying disorder, and IED are relatively frequent in pathological gamblers, as are personality disorders. Murray (1993) found that pathological gamblers fit no particular personality profile, but several investigators have reported abnormal personality traits in pathological gamblers based on dimensional assessments (e.g., Roy et al. 1989). Taber et al. (1987) reported that 20% of 66 pathological gambling inpatients had personality disorders. Narcissistic and ASPD are believed to be overrepresented in these patients, and pathological narcissism is assumed by some psychoanalysts to underlie the entitlement displayed by many patients with pathological gambling. In addition to psychiatric disorders, patients with pathological gambling may manifest greater prevalence of stress-related medical conditions, like peptic ulcer disease, hypertension, and migraine.

## Course

The onset of pathological gambling is usually insidious, although some may be "hooked" by their first bet. There may be years of social gambling with minimal or no impairment followed by an abrupt onset of pathological gambling that may be precipitated by greater exposure to gambling or by a psychosocial stressor. The gambling pattern may be regular or episodic, and the course tends to be chronic. Over time, there is usually a progression in the frequency of gambling, the amounts wagered, and the preoccupation with gambling and with obtaining money with which to gamble. The urge to gamble and gambling activity generally increase during periods of stress or depression, as an attempted escape or relief (criterion A5).

Pathological gambling usually begins in adolescence in men (Hollander et al. 2000a) with gradual development of dependence, and may remain undiagnosed for years, they often present with a 20- to 30-year-gambling history. In contrast, onset in females is usually later in life. Prior to seeking treatment, the duration of pathological gambling is about 3 years. Thus, as a result of the differences in onset and duration, female pathological gamblers generally have a better prognosis than male pathological gamblers (Rosenthal 1992). Female pathological gamblers also tend to be depressed and may use gambling as an anesthetic, accompanied by excitement, to escape from life's problems (i.e., as in a dissociative state; Jacobs 1988).

Psychiatric disorders like major depression and alcohol or substance abuse and dependence, may develop from or be exacerbated by pathological gambling. There is also a mortality risk associated with the disorder. Estimates of suicide attempts in pathological gamblers range from 17% to 24% (Ciarrochi and Richardson 1989, 2000a). One study found that the suicide rate in cities where gambling is legalized is four times higher than in cities where it is not (Phillips et al. 1997). Younger patients are more likely to have suicidal tendencies and major depressive disorders (McCormick et al. 1984), and most pathological gambling begins during adolescence (Hollander et al. 2000a), thus early identification and intervention are crucial.

Rosenthal (1992) described four typical phases in the course of a typical male patient with pathological gambling: winning, losing, desperation, and hopelessness (Linnoila et al. 1989).

### Winning

Many male gamblers become involved with gambling because they are good at it and receive recognition for their early successes. Women with pathological gambling are less likely to have a winning phase. Traits that foster a winning phase and are typical of male patients with pathological gambling are competitiveness, high energy, ability with numbers, and interest in the strategy of games. The early winnings lead to a state in which a large proportion of the gambler's self-esteem derives from gambling, with accompanying fantasies of winning and spectacular success.

### Losing

A string of bad luck or a feeling that losing is intolerable may be the precipitant of chasing behavior; previous gambling strategies are abandoned as the gambler attempts to win back everything at once. The gambler experiences a state of urgency, and bets become more frequent and heavy. Debts accumulate, and only the most essential are paid. Covering up and lying about gambling become more frequent. As this is discovered, relationships with family members deteriorate. Losing gamblers use their own and their family's money, go through savings, take out loans, and finally exhaust all legitimate sources. Eventually, they cannot borrow any more, and faced with threats from creditors or loss of a job or marriage, they go to their family and finally confess. This results in the "bailout": debts are paid in return for a promise to stop or cut down gambling. Any remission, if achieved, is short lived. After the bailout there is an upsurge of omnipotence; the gambler believes it is possible to get away with anything, bets more heavily, and loses control altogether.

### Desperation

This stage is reached when the gambler begins to do things that would previously be inconceivable: writing bad checks, stealing from an employer, or other illegal activities. Done once, these behaviors are much more likely to be repeated. The behavior is rationalized as a short-term loan with an intention to pay it back as soon as the winning streak arrives. The gambler feels just one step away from winning and solving all the problems. Attention is increasingly taken up with illegal loans and various scams to make money. The gambler becomes irritable and quick tempered. When reminded of responsibilities or put in touch with guilt feelings, the gambler responds with anger and projective blame. Appetite and sleep deteriorate and life holds little pleasure. A common fantasy at this stage is of starting life over with a new name and identity, the ultimate "clean slate."

### Hopelessness

For some gamblers, there is a fourth stage in which they suddenly realize that they can never get even, but they no longer care. This is often a revelation, and the precise moment when it occurred is often remembered. From this point on, just playing is all that matters. Gamblers often acknowledge knowing in advance that they will lose and play sloppily so that they lose even if they have the right horse or a winning hand. They seek action or excitement for its own sake and gamble to the point of exhaustion.

Few gamblers seek help in the winning phase. Most do so only during the later phases and only after a friend, family member, or employer has intervened. Two-thirds of the gamblers have committed illegal activities by then, and the risk of suicide increases as they progress through the phases of the illness.

Without treatment, the prognosis of pathological gambling is poor. It tends to run a chronic course with increasing morbidity and comorbidity, gradual disruption of family and work roles and relationships, depletion of financial reserves, entanglement with criminals and the criminal justice system, and, often, suicide attempts. In the hands of an experienced psychiatrist, it is an "extremely treatable disorder" with a favorable prognosis (Rosenthal 1992). The difference between a poor and a good prognosis depends on treatment, and treatment depends on a diagnosis. As noted earlier, the diagnosis of pathological gambling is often missed in clinical settings because mental health professionals do not think to ask about it. Because most pathological gamblers do not see themselves as having a disorder and many of them do not even consider themselves as having a problem, collateral information from a family member may be very helpful.

## Differential Diagnosis

The differential diagnosis of pathological gambling is relatively straightforward (Table 000–2). Pathological gambling should be differentiated from professional gambling, social gambling, and a manic episode. Social gambling, engaged in by a majority of adult Americans, typically occurs with friends or colleagues, lasts for a specified time, and is limited by predetermined acceptable losses. Professional gambling is practiced by highly skilled and disciplined people and involves carefully limited risks. Many individuals with pathological gambling may feel that they are actually professional gamblers. Chasing behavior and unplanned losses distinguish pathological gamblers. Patients in a manic episode may exhibit a loss of judgment and excessive gambling resulting in financial disasters. A diagnosis of pathological gambling should be given only if a history of maladaptive gambling behavior exists at times other than during a manic episode. Problems with gambling may also occur in people with ASPD. If criteria are met for both disorders, both can be diagnosed.

## Differences in Gender and Cultural Presentations

An important and understudied area is the clinical presentation of pathological gambling in women. Women constitute a third of patients with pathological gambling in epidemiological studies. But, they are extremely underrepresented in treatment populations, and most psychoanalytic theories of pathological gambling ignore them completely. Part of this bias may be due to the fact that gambling carries a greater social stigma for women, women gamblers are more likely to live and to gamble alone, and treatment programs for pathological gambling in the US were first pioneered in Veterans Hospitals. Compared with men with pathological gambling, women with pathological gambling are more likely to be depressed and to gamble as an escape rather than because of a craving for action and excitement. Pathological gambling begins at a later age in female than in male gamblers, often after adult roles have been established. Big winning is usually less important than the need to impress. Women typically play less competitive forms of gambling in which luck is more important than skill, and they play alone. Their progression into the disorder is often more rapid, and the time between the onset of the disorder and the time they present for treatment is usually much shorter than for men (3 versus 20 years). The shorter duration makes for a better prognosis in treatment, but, unfortunately, few of the women with pathological gambling come to treatment.

The choice of gambling activities is dictated by local availability and cultural norms. Horseracing, cockfights, roulette, slot machines, casino card games, state-sponsored lotteries, and the stock market may all be used by the gambler. Likewise, the extent of gambling considered normal varies across cultures. DSM-IV-TR approaches this by concentrating on the consequences of gambling rather than on its frequency and type.

## Etiology and Pathophysiology

Pathological gambling has been included in DSM-III, DSM-III-R, and DSM-IV as a disorder of impulse control. Pathological gambling can also be viewed as an addictive disorder (Murray 1993), an affective spectrum disorder (McElroy et al. 1992) and an obsessive-compulsive spectrum disorder (Hollander et al. 1992b). DSM-IV-TR maintains a close relationship between pathological gambling and addictive disorders in that several of the diagnostic criteria for pathological gambling were intentionally made to resemble criteria for substance dependence (Table 000–3).

The parallels between pathological gambling and addictive disorders are manifold. Pathological gambling has been viewed as the "pure" addiction, because it involves several aspects of addictive behavior without the use of a chemical

| Table 000–2 | Differential Diagnosis of Pathological Gambling |
| --- | --- |
| Pathological Gambling Must Be Differentiated From | In Contrast to Pathological Gambling, the Other Condition |
| Professional gambling | Is characterized by discipline and limited risk taking |
| | Is intended to be a source of income |
| Social gambling | Usually occurs among friends |
| Is characterized by limited time spent on gambling and limited risk taking | |
| Manic episode | Involves episodes of characteristic symptoms (e.g., flight of ideas) |
| Is characterized by symptoms that persist at times when individual is not gambling | |

*Source:* [Reproduced from First and Frances A (1995) with permission of American Psychiatric Press.]

| Table 000–3 | Comparison of DSM-IV-TR Criteria for Pathological Gambling and Substance Dependence |
|---|---|

| Pathological Gambling | Substance Dependence |
|---|---|
| A. Persistent and recurrent maladaptive gambling behavior as indicated by at least five (or more) of the following: | A maladaptive pattern of substance use, leading to clinically significant impairment or distress, as manifested by three (or more) of the following: |
| A1. 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) | 1. A great deal of time is spent in activities necessary to obtain the substance or recover from its effects |
| A2. Needs to gamble with increasing amounts of money to achieve the desired excitement | 2. (Tolerance) (a). A need for markedly increased amounts of the substance to achieve intoxication or desired effect or (b). markedly diminished effect with continued use of the same amount of substance |
| A3. Has repeated unsuccessful efforts to control, cut back, or stop gambling | 3. There is a persistent desire or unsuccessful attempts to cut down or control substance use |
| A4. Is restless or irritable when attempting to cut down or stop gambling | 4. (Withdrawal) (a). The characteristic withdrawal syndrome for the substance |
| A5. Gambles as a way of escaping from problems or relieving a dysphoric mood | 5. (Withdrawal) (b). The same substance is taken to relieve or avoid relieving withdrawal symptoms |
| A6. After losing money gambling, often returns another day to get even ("chasing" one's losses) | 6. The substance is often taken in larger amounts or over a longer period than was intended |
| A7. Lies to family members, therapist, or others to conceal the extent of involvement with gambling | |
| A8. Has committed illegal activities such as forgery, fraud, theft, or embezzlement to finance gambling | |
| A9. Has jeopardized or lost a significant relationship, job, or educational opportunity because of gambling | 7. Important social, occupational, or recreational activities are given up or reduced because of substance use |
| A10. 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 | 8. The substance use is 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 |

Source: [Reproduced from American Psychiatric Association (2000) with permission of American Psychiatric Press.]

substance. The parallels between substance dependence, in particular alcohol dependence, and pathological gambling have led to the successful adoption of the self-help group model of Alcoholics Anonymous (AA) to GA. Patterns of comorbidity also suggest a possible link between pathological gambling and addictions, in particular alcoholism. In addition to the comorbidity of pathological gambling and substance use disorders, family studies have demonstrated a familial clustering of alcoholism and pathological gambling. Ramirez et al. (1983) found that 50% of their patients with pathological gambling had a parent with alcoholism; other studies have also found high rates of a family history of substance dependence in patients with pathological gambling. There is also a greater prevalence of pathological gambling in parents of patients with pathological gambling.

The links between pathological gambling and affective disorders are also supported by family studies that demonstrate high rates of affective disorders in first-degree relatives of patients with pathological gambling (McElroy et al. 1992), as well as by high rates of comorbidity of pathological gambling and affective disorders. In addition, as noted by many authors and incorporated in the DSM-IV-TR criteria for pathological gambling, many patients with pathological gambling gamble as a way of relieving dysphoric moods (criterion A5), and cessation of gambling may be associated with depressive episodes in the majority of recovering gamblers (Linden et al. 1986).

The links between pathological gambling and obsessive spectrum disorders are less clear. Although a popular name for pathological gambling is compulsive gambling, the majority of people with pathological gambling do not experience the urge to gamble as ego-dystonic until late in the course of their illness, after they have suffered some of its consequences. The rates of comorbidity of pathological gambling and OCD and obsessive-compulsive personality disorder are not nearly as high as the rates of comorbidity of pathological gambling and affective and addictive disorders. Nevertheless, pathological gambling shares several characteristics with compulsions: it is repetitive, often has ritualized aspects, and is meant to relieve or reduce distress. Moreover, sporadic reports on the effectiveness of SSRIs in the treatment of pathological gambling suggest a possible link to obsessive spectrum disorders (Hollander et al. 1992b).

## Neurotransmitter Function

The association between altered function of the serotonin neurotransmitter system and impulsive behaviors has focused attention on a potential role for serotonin function in the neurophysiology of pathological gambling. Evidence of serotonergic dysfunction in pathological gamblers comes from neurobiological studies (Moreno et al. 1991, Carrasco et al. 1994, DeCaria et al. 1998a). These findings include: blunted prolactin response after intravenous administration

of the SSRI, clomipramine (Moreno et al. 1991), increased prolactin response after the administration of a serotonin agonist, m-CPP (DeCaria et al. 1996), and low platelet MAO-B activity (a correlate central nervous system concentrations of the serotonin metabolite 5-HIAA) (Blanco et al. 1996, Carrasco et al. 1994). However, direct measure of CSF 5-HIAA in pathological gamblers has yielded mixed results (Roy et al. 1988b, Bergh et al. 1997, Ibanez et al. 2002). Preliminary data support potential utility of SSRI medications in the treatment of pathological gambling (Hollander et al. 1998, 2000b, Ibanez et al. 2002). There is evidence of serotonergic dysfunction in depression (Coccaro et al. 1989), impulsivity (Linnoila et al. 1983), suicidality (Mann et al. 1992), and alcoholism (Tollefson 1991). This is of interest because pathological gambling is strongly associated with depression (Roy et al. 1988a, 1988b), impulsivity (Moreno et al. 1991), suicidality (Ciarrochi and Richardson 1989), and alcohol or drug abuse (Linden et al. 1986, McCormick et al. 1984). Thus, pathological gambling may also be associated with serotonergic dysfunction as it relates to these comorbid features.

Because of the "addictive" aspects of pathological gambling—and the role that dopaminergic function plays in chemical addictions—attention has been directed at dopamine function among pathological gamblers. Two available studies have yielded contradictory data (Roy et al. 1988b, Bergh et al. 1997). The role of noradrenergic function has also been explored. In support for such a role, pathological gamblers have been shown to have higher urinary and CSF concentrations of noradrenaline and metabolites (Roy et al. 1988b, Bergh et al. 1997). Measures of extroversion in pathological gamblers significantly correlate with indices of noradrenergic function (Roy et al. 1989). Further, increased noradrenergic function has been associated with arousal, irritability, and risk-taking behavior (Coccaro et al. 1991) and pathological gambling has been associated with increased arousal and tonic activity of the central noradrenergic system (Brown 1986, Dickerson et al. 1987, Roy et al. 1988b). In addition, increased growth hormone secretion, a measure of noradrenergic reactivity, was found in pathological gamblers in response to oral administration of clonidine, an alpha-2-adrenergic agonist (Ibanez et al. 2002).

### Genetic Contribution

The incidence of pathological gambling among first-degree family members of pathological gamblers appears to be approximately 20% (Ibanez et al. 2002). Inherited factors may explain 62% of variance in the diagnosis (Eisen et al. 1998) and some of these genetic factors may also contribute to the risk for conduct disorder, ASPD, and alcohol abuse (Eisen et al. 2001). Gambino et al. (1993) found that patients who perceived that their parents had gambling problems were three times more likely to score as probable pathological gamblers on the SOGS. Those who also perceived that their grandparents had gambling problems had a 12-fold increased risk compared with patients who did not perceive gambling problems in their parents and grandparents.

Serotonergic, noradrenergic, and dopaminergic genes have been investigated because of the putative role of these neurotransmitters in pathological gambling, and a number of molecular genetic studies performed to date have reported findings consistent with the involvement of these neurotransmitter systems in pathological gambling (e.g., Ibanez et al. 2001, Perez de Castro et al. 2002, Comings et al. 1996, 1997). However, some of the studies performed to date have not been adequately controlled for potential differences in racial and ethnic compositions, factors that could account for differences in allelic variant distributions. Thus, these studies should be regarded as preliminary.

At present, the main source of evidence for the genetic influence in the etiology of pathological gambling comes from a study of 3359 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), a genetic susceptibility model in the pathogenesis of pathological gambling (Eisen et al. 1998), and indicates a common genetic vulnerability for pathological gambling and alcohol dependence in men (Slutske et al. 2000). In a smaller twin study, Winters and Rich (1999) found a significant heritability explaining "high action" gambling, like casinos and gambling slot machines, in 92 monozygotic and dizygotic male twin pairs. But, no significant differences in heritability were found among males for "low action" games and among 63 female monozygotic and dizygotic twin pairs for either "high action" or "low action" gambling.

### Neuropsychology

Clinical comorbidities and observations that pathological gambling involves strong motivations to engage in gambling and subjective feelings of reward, withdrawal, and craving for gambling, support the categorization of pathological gambling as "a nonpharmacological addiction" (Blanco et al. 2001, Holden 2001). This view is corroborated by neuroimaging findings that gambling-associated cognitive and motivational events, or responses of pathological gamblers to gambling-related stimuli, are associated with metabolic changes in brain regions implicated in studies of substance use disorders (Breiter et al. 2001, Holden 2001, Potenza et al. 2003). Using FDG-PET in unmedicated pathological gamblers without comorbid substance use disorders ($N = 7$), Hollander et al. (2001) found heightened limbic and sensory activation in a gambling-for-money condition, with increased emotional valence and greater risk and reward, which confirms the salience of monetary reward in the development of pathological gambling.

Some data support the notion that people with impaired impulse control exhibit abnormalities in risk-benefit decision making in both gambling and nongambling activities and that their cognitive or emotional sense of what distinguishes gambling from other decisions of daily living may be compromised (Crean et al. 2000, Petry 2001a, 2001b; Petry and Casarella 1999, Bechara 2001, Bechara et al. 2000, 2001, Potenza 2001). These deficits may produce an inability to inhibit motivated drives to gamble, leading to persistent gambling. Myopia for the future and insensitivity to punishment has also been shown in orbitofrontal and ventromedial PFC lesion patients (Bechara et al. 1994, Berlin et al. 2004) using gambling tasks. Cavedini et al. (2002) data suggest a link between pathological gambling and other disorders (i.e., OCD and drug addiction) all having diminished ability to evaluate future consequences, which may be explained in part by an abnormal functioning of the orbitofrontal cortex.

Attention problems and impulsivity in pathological gamblers could reflect deficits in executive functioning that are often a consequence of minimal brain damage with orbitofrontal cortex impairment (Rugle and Melamed 1993, Specker et al. 1995, Berlin et al. 2004).

## Psychodynamic Considerations

Psychoanalytic theories of gambling were the first systematic attempts to account for pathological gambling. Erotization of the fear, tension, and aggression involved in gambling behavior, as well as themes of grandiosity and exhibitionism, were explored by several authors during the first quarter of the 20th century. Freud (1961) in his influential essay on Dostoyevsky, suggested that the pathological gambler actually gambled to lose, not to win, and traced the roots of the disorder to the ambivalence felt by the young man toward his father. The father, the object of his love, is not only loved but also hated, and this results in unconscious guilt. The gambler then loses to punish himself, in what Freud labeled "moral masochism." Freud also spoke of "feminine masochism" in which losing is a way of gaining love from the father, who will somehow reward the loser for loyalty. To lose is to suffer, and for the feminine masochist, suffering equals love. Interestingly, in the later spirit of DSM-IV-TR, Freud also conceptualized pathological gambling as an addiction and included it in a triad with alcoholism and drug dependence. He saw all three as manifestations of that primary addiction, masturbation, or at least masturbatory fantasies. Like most researchers after him, Freud focused only on male gamblers.

Bergler, a psychoanalyst who treated many pathological gambling patients, expanded on Freud's idea that pathological gamblers gamble to lose (Lesieur and Rosenthal 1991). He traced the roots of this desire to lose to the rebellion of gamblers against the authority of their parents and the parents' intrusive introduction of the reality principle into their lives. The rebellion causes guilt, and the guilt creates the need for self-punishment. Bergler thought that the gambler's characteristic aggression is actually pseudoaggression, a craving for defeat and rejection. He saw the gambler as one who perpetuates an adversarial relationship with the world. The dealer in the casino, the gambler's opponents at the card table, the stock exchange, and the roulette wheel are all unconsciously identified with the refusing mother or the rejecting father. Overall, psychoanalytic approaches to pathological gambling (Lesieur and Rosenthal 1991) generally conceptualized it as either a compulsive neurosis (Freud, Bergler, and Rosenthal) or an impulse disorder (Fenichel). Fenichel (1945) focused on the gambler's entitlement and intense need to "get the stuff," an oral fixation. Several published case reports documented the successful treatment of pathological gambling by psychoanalysis.

Learning theories of pathological gambling focus on the learned and conditioned aspects of gambling and use the quantifiable nature of the behavior to test specific hypotheses. One hypothesis was that patients with pathological gambling crave the excitement and tension associated with their gambling, as evidenced by the fact that they are much more likely to place last-second wagers than are low-frequency gamblers, to prolong their excitement. Higher wagers placed by patients with pathological gambling also produce greater excitement, and greater amounts of money are required to achieve the same "buzz" over time, an observation incorporated in the diagnostic criteria for pathological gambling (criterion A2).

## Treatment

## Treatment Goals

The goals of treatment of an individual with pathological gambling are the achievement of abstinence from gambling, rehabilitation of the damaged family and work roles and relationships, treatment of comorbid disorders, and relapse prevention. This approach echoes the goals of treatment of an individual with substance dependence. There are many similarities and several important differences between the treatment of pathological gambling and the treatment of substance dependence. For most patients without severe acute psychiatric comorbidity, such as major depressive disorder with suicidal ideation or alcohol dependence with a history of delirium tremens, treatment may be given on an outpatient basis. Inpatient treatment in specialized programs may be considered if the gambler is unable to stop gambling, lacks significant family or peer support, or is suicidal, acutely depressed, multiply addicted, or contemplating some dangerous activity.

No standard treatment of pathological gambling has emerged. Despite many reports of behavioral and cognitive interventions for pathological gambling, there are minimal data available from well-designed or clearly detailed treatment studies (Petry 2002). Pharmacological treatments (see below) offer promise, but research-guided approaches are still insufficient to offer a standardized approach. Thus, general approaches, based in clinical experience and available resources (like GA or other support groups) should be considered. The treatment of pathological gambling may consist of participation in GA, individual therapy, family therapy, treatment of comorbid disorders, and medication treatment. Many treatment modalities of pathological gambling are similar to that of substance abuse disorders and were created based on the addiction model, like self-help groups, inpatient treatment programs, and rehabilitation programs. As is the case for substance dependence, the gambler needs to be abstinent to be accessible to any or all of these treatment modalities. Essential features of any therapeutic intervention for pathological gambling include the need to establish both a therapeutic alliance and network, address the underlying pathology, interrupt the behavior and maintain abstinence, problem solve, and improve quality of life.

The most popular intervention for problem gambling is GA, a 12-step group built on the same principles as AA, which utilizes empathic confrontation by peers who struggle with the same impulses. For many gamblers, participation in GA is sufficient, and it is an essential part of most treatment plans. Extensive data are lacking, but overall GA appears somewhat less effective than AA in achieving and maintaining abstinence. Evidence suggests that GA 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 GA members report total abstinence at 1-year follow-up and 7% at 2-year

follow-up (Brown 1985). Although participation in GA's spousal component, Gam-Anon, may be helpful for some family members, little evidence suggests that it reduces disordered gambling (Petry and Armentano 1999).

Individual therapy is often useful as an adjunct to GA. Rosenthal (1992) stressed that to maintain abstinence and use GA successfully, many gamblers need to understand why they gamble. Therapy involves confronting and teasing out the vicissitudes of the patient's sense of omnipotence and dealing with the various self-deceptions and the defensive aspects of the patient's lying, and problems involving magical thinking and reality. Relapse prevention involves knowledge and avoidance of specific triggers. In addition to psychodynamic therapy, behavioral treatment of pathological gambling has been proposed, with imagined desensitization achieving better rates of remission than aversive conditioning.

The greatest differences between the treatment of pathological gambling and other addictions are in the area of family therapy. Because relapse may be difficult to detect (no substance smelled on their breath, dilated or constricted pupils, or slurred speech or staggered gait) and because of a long history of exploitative behavior by the patient, the spouse and other family members tend to be more suspicious of, and angry at, the pathological gambling patient compared with families of alcoholic patients. Family sessions are often essential to offer the gambler an opportunity to make amends, learn communication skills, and deal with preexisting intimacy problems. The spouse and other family members often acquire their own psychiatric illnesses during the course of the patient's illness and may need individualized treatment to recover.

## Somatic Treatments

Although research reports of the pharmacological treatment of pathological gambling have reported some efficacy, there are still, as yet, insufficient data to come to any conclusions about the utility of medication. Pharmacological treatment studies of pathological gambling have demonstrated some promising results with the use of SSRIs (Hollander et al. 1992b, Hollander et al. 1998, 2000b, de la Gandara 1999, Zimmerman et al. 2002, Kim et al. 2002), serotonin reuptake inhibitors (Pallanti et al. 2002b), mood stabilizers (Haller and Hinterhuber 1994, Pallanti et al. 2002a, Hollander et al. 2002), opiate antagonists (Kim et al. 2001), and atypical antipsychotics (Potenza and Chambers 2001). Doses at the higher end of the usual treatment range should be considered with both SSRIs and opiate antagonists. Some studies have not reported significant findings, possibly due to high placebo response rates, high rates of discontinuation, as well as the possibility that different patients have differential responses to the various medication options based on endophenotypes that have yet to be elucidated (Blanco et al. 2002, Grant et al. 2003). At this time, pharmacological agent algorithms are still not definitive for the treatment of PG (Grant and Kim 2002e, Haller and Hinterhuber 1994, Hollander et al. 2000b, Kim and Grant 2001, Kim et al. 2001, Grant and Potenza 2004)). Treatment should ultimately target all symptom domains within the individual patient that contribute to compulsive gambling, including common comorbid conditions like bipolar spectrum disorder, ADHD, and substance abuse/dependence disorders.

## Psychotherapy

Inpatient programs for pathological gambling have included various combinations of individual and group psychotherapy and substance use treatment (Taber 1981), and most strongly encouraged or required attendance at GA 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). Although methodologically flawed, these reports suggest that professionally delivered multimodal therapy programs, given alone or in combination with GA, may be more effective than GA alone. Self-help manuals may also be useful for some (Dickerson et al. 1990), and studies comparing their effectiveness with professionally delivered CBT are ongoing (Petry and Armentano 1999).

Early psychoanalytic reports suggest that problem gambling is regressive and representative of various pregenital and genital instincts, unconscious conflicts, or painful affects. Most studies that report good outcome are based on single-case studies, and some authors believe that purely psychodynamic treatment of pathological gambling is difficult. Rosenthal and Rugle (1994) published a psychodynamic approach to pathological gambling treatment, which integrates traditional psychodynamic psychotherapy with an addiction model.

Behavioral, cognitive, and combined cognitive-behavioral methods have been used in treating pathological gambling. Aversive therapy has been employed to reach the goal of total abstinence of gambling, as have behavior monitoring, contingency management, contingency contracting, covert sensitization, systematic desensitization, imaginal desensitization, in vivo exposure, imaginal relaxation, psychoeducation, cognitive restructuring, problem-solving skills

---

### Clinical Vignette 3

Mr. Z is a 44-year-old automobile mechanic who is married and has two adolescent children. He was referred for inpatient treatment from the emergency department of his local hospital after an overdose of a full bottle of over-the-counter sleeping pills taken as a suicide attempt. He took the pills in the setting of a major depressive episode without psychotic features. This was his first psychiatric contact. During his initial evaluation, Mr. Z admitted to several past depressive episodes for which he did not seek treatment and which resolved spontaneously. He also admitted to a history of alcohol abuse and stated that his drinking may have escalated recently. His family history was significant for a father with alcohol dependence and a mother with bipolar disorder. Mr. Z was treated as an inpatient with fluoxetine and improved rapidly, with full resolution of his suicidal ideation and other depressive symptoms.

Two days before his planned discharge from the hospital, a family meeting was held. During the meeting, his treatment team was surprised when his wife announced that she refused to let Mr. Z return home before "the other problem" was taken care of. At this point, a parallel history was taken and revealed that Mr. Z has had a problem with "excessive gambling" for the last 25 years. His wife meant that Mr. Z gambled with money that he did not have and went on gambling sprees that ended only when he lost all the money available to him and exhausted his current abilities to borrow money. During such episodes he had lost

*continues*

the family home, was caught stealing spare parts from his job and was fired, and borrowed several thousand dollars from his in-laws and his sister, which he was unable to repay. More recently he became involved with loan sharks, who were now threatening him physically. His suicide attempt followed a threat to hurt him and his family if his debts were not paid.

Mr. Z began to gamble when he was 16 years old and stated that "there is nothing else like it." He frequented off-track-betting establishments and considered himself an expert in horseracing. His only social acquaintances were other gamblers, and he found other recreational activities dull and unsatisfying. When he was confronted with this history, Mr. Z felt that his wife was "being hysterical." He admitted that he loved to gamble but described himself as a professional gambler and cited several occasions on which he won significant amounts of money. He was not interested in any kind of treatment but agreed to attend GA if his extended family would help him out of his current financial problems.

During the course of a 2-year follow-up, Mr. Z continued to gamble and lose money. He stopped going to meetings. He was fired from his job again, discontinued his medication, became depressed, and started drinking. At that time, he came to treatment with his wife, who was attending Gam-Anon and began seeing a psychiatrist for the treatment of her own mild depression. Mr. Z agreed to an arrangement in which his wife would have complete power of attorney over his financial affairs. His participation in GA and AA became a prerequisite for his continued living at home. He was again given fluoxetine and began a course of individual psychotherapy with biweekly family sessions. For the first time in his adult life, Mr. Z was able to abstain from gambling for a period of 3 months.

training, social skills training, and relapse prevention. Use of cognitive restructuring facilitates a decrease in the frequency of gambling and irrational verbalizations associated with gambling (Ladouceur 1990).

## Trichotillomania

### Diagnosis

#### Definition and Diagnostic Features
The essential feature of trichotillomania is the recurrent failure to resist impulses to pull out one's own hair. Resulting hair loss may range in severity from mild (hair loss may be negligible) to severe (complete baldness and involving multiple sites on the scalp or body). Individuals with this condition do not want to engage in the behavior, but attempts to resist the urge result in great tension. Thus, hair pulling is motivated by a desire to reduce this dysphoric state. In some cases, the hair pulling results in a pleasurable sensation, in addition to the relief of tension. Tension may precede the act or may occur when attempting to stop. Distress over the symptom and the resultant hair loss may be severe.

Typically, the person complaining of unwanted hair pulling is a young adult or parent of a child who has been seen pulling out hair (Winchel 1992). Hair pulling tends to occur in small bursts that may last minutes to hours. Episodes may occur once or many times each day. Hairs are

---

### DSM-IV-TR Criteria

**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.

pulled out individually and may be pulled out rapidly and indiscriminately. Often, however, the hand of the individual may roam the afflicted area of scalp or body, searching for a shaft of hair that may feel particularly coarse or thick. Satisfaction with having pulled out a complete hair (shaft and root) is often expressed. Occasionally the experience of hair pulling is described as quite pleasurable. Some experience an itch-like sensation in the scalp that is eased by the act of pulling. The person may then toss away the hair shaft or inspect it. A substantial number of people then chew or consume the hair (trichophagia). Hair-pulling is most commonly limited to the eyebrows and eyelashes. The scalp is the next most frequently afflicted site. However, hairs in any location of the body may be the focus of hair-pulling urges, including facial, axillary, chest, pubic, and even perineal hairs.

Anxiety is almost always associated with the act of hair pulling. Such anxiety may occur in advance of the hair-pulling behavior. A state of tension may occur spontaneously, driving the person to pull out hair in an attempt to reduce dysphoric feelings. Varying lengths of time must pass before the tension abates. Consequently, the amount of hair that may be extracted in an episode varies from episode to episode and from person to person. Frequently, hair pulling begins automatically and without conscious awareness. In such circumstances, individuals discover themselves pulling out hairs after some have already been pulled out. In these situations, dysphoric tension is associated with the attempt to stop the behavior.

Circumstances that seem to predispose to episodes of hair pulling include both states of stress and, paradoxically, moments of relaxation. Frequently, hair pulling occurs when at-risk individuals are engaged in a relaxing activity that promotes distraction and ease (e.g., watching television, reading, talking on the phone). It is common for hair pullers to report that the behavior does not occur in the presence of other people. A frequent exception may be that many pull hair in the presence of members of the nuclear family.

Some individuals have urges to pull hairs from other people and may sometimes try to find opportunities to do so surreptitiously (like initiating bouts of play fighting). There have been reports of individuals pulling hairs from pets, dolls, and other fibrous materials, like sweaters or carpets (Tabatabai and Salari-Lak 1981).

The distress that usually accompanies trichotillomania varies in severity. Concerns tend to focus on the social and vocational consequences of the behavior. Themes of worry include fear of exposure, feeling that "something is wrong with me," anxiety about intimate relationships, and sometimes inability to pursue a vocation. Because certain kinds of work, like reading and writing at a desk, seem to precipitate episodes of hair pulling, some individuals make career choices based on the avoidance of desk work. Leisure activities that may involve a risk of exposure (ranging from gymnastics class to sexual intimacy) may be avoided.

Patterns of hair-pulling behavior among children are less well described. Usually, the parent observes a child pulling out hair and may note patches of hair loss. Children may sometimes be unaware of the behavior or may, at times, deny it. Childhood trichotillomania has been reported to be frequently associated with thumb sucking or nail biting (Friman and Hove 1987). It has been suggested that trichotillomania with onset in early childhood may occur frequently with spontaneous remissions. Thus, some have recommended that trichotillomania in early childhood be considered a benign habit with a self-limited course. But, many people who present with chronic trichotillomania in adulthood report onset in early childhood (Reeve et al. 1992).

## Assessment

In general, the diagnosis of trichotillomania is not complicated. The essential symptom—recurrently pulling out hair in response to unwanted urges—is easily described by the patient. When the patient acknowledges the hair-pulling behavior and areas of patchy hair loss are evident, the diagnosis is not usually in doubt. Problems in diagnosis may arise when the diagnosis is suspected but the patient denies it. Such denial may occur in younger individuals and some adults. When the problem is suspected but denied by the patient, a skin biopsy from the affected area (see later) may aid in making the diagnosis.

### The Psychiatric Interview

The psychiatrist should carefully inquire into the nature of the distress and the patient's concerns. Although the cosmetic impact may appear slight, distress may be severe. Concerns about disclosure, anticipation of social rejection, and concerns about limitations in career choices are frequent and may result in chronic dysphoria. The psychiatrist should be aware of the embarrassment that may accompany inspection of the hair loss, particularly when located in regions of the body that are not usually accessible in the course of a standard psychiatric examination. Because of the apparent frequency of comorbid mood disorders (past or current), the interviewer should pay special attention to the presence of these features.

### Physical Examination and Laboratory Findings

Areas of hair loss can be marked by complete alopecia or can appear diffusely thinned or "ratty." Altered scalp appearance can range from small areas of thinned hair to complete baldness. For unclear reasons, several patterns of scalp loss are typical. Frequently, coin-sized areas of alopecia are noted at the vertex or at temporal or occipital regions. Among more severely afflicted people a peculiar pattern, the so-called tonsure trichotillomania, may appear: a completely bald head except for a narrow, circular fringe circumscribing the outer boundary of the scalp, producing a look reminiscent of medieval friars.

Despite the hair loss, most individuals with this condition have no overtly unusual appearance on cursory inspection. If the hair loss is not covered by clothing or accessories, artful combing of hair or use of eyeliner and false eyelashes may easily hide it. The ease with which the condition may often be hidden may explain the general underappreciation of its apparent frequency and potential associated distress.

### Associated Laboratory Findings

Histological findings are considered characteristic and may aid diagnosis when it is suspected despite denial by the individual. Biopsy samples from involved areas may have the following features. Short and broken hairs are present. The surface of the scalp usually shows no evidence of excoriation. On histological examination, normal and damaged follicles are found in the same area, as well as an increased number of catagen (i.e., nongrowing) hairs. Inflammation is usually minimal or absent. Some hair follicles may show signs of trauma (wrinkling of the outer root sheath). Involved follicles may be empty or contain a deeply pigmented keratinous material. The absence of inflammation distinguishes trichotillomania-induced alopecia from alopecia areata, the principal condition in the differential diagnosis (Mehregan 1970, Muller 1990).

## Epidemiology

Trichotillomania was long thought to be uncommon, often accompanied by other psychiatric conditions. Although definitive studies of frequency rates in the general population are still lacking, three surveys of nonclinical college-age samples support the emerging view that trichotillomania is more common than originally suggested. In two of these samples, totaling approximately 3000 undergraduate students, 10–13% of students reported hair pulling, with the prevalence of clinically significant pulling ranging between 1% and 3.5% (Christenson et al. 1991b, Rothbaum et al. 1993). A epidemiological study of trichotillomania and skin picking using self-report instruments (the Massachusetts General Hospital Hairpulling Scale and the Skin Picking Scale) in a sample of 1324 college freshmen found that 5.4% (72) endorsed relatively frequent hair pulling or skin picking (Hajcak et al. 2006). One epidemiological survey of 17-year-old adolescents in Israel suggests a prevalence rate of 1% for current or past hair pulling, with fewer reporting noticeable hair loss or distress from these symptoms (1995a).

These may be underestimates of the lifetime incidence of the disorder. Had these studies applied DSM-IV-TR criteria, which have become slightly less restrictive than DSM-III-R criteria, the rates might be higher. In addition, because onset may occur later in life than the mean ages of individuals in these groups, the true lifetime incidence would probably be higher. Moreover, these samples consist of a

selected population—largely first-year college students—and may not reflect the general population. Nonetheless, these studies indicate that the condition is likely to be far more common than previously assumed. But definitive, controlled studies of the prevalence of the condition have not yet been performed.

## Comorbidity Patterns

Many experts (e.g., Christenson and Mansueto 1999) have noted a common co-occurrence and formal similarities between trichotillomania and other body-focused ICDs like skin picking and severe nail biting. Nail biting and skin picking (excoriation) are often associated with trichotillomania and these three pathological behaviors often co-occur with each other and are thought to be related (Wilhelm and Margraf 1993, Bhatia et al. 1991, Simeon et al. 1997, O'Sullivan et al. 2000, Cohen et al. 1995, Wilhelm et al. 1999). If the skin picking and nail biting appear to be largely negatively reinforcing—that is, reducing anxiety associated with specific obsessional thoughts and/or reducing the likelihood of feared outcomes—they may be better conceptualized as OCD behaviors and addressed accordingly. However, Franklin et al. (2006) suggest, based on clinical experience, that these conditions are much more likely to formally resemble trichotillomania. In accordance, many trichotillomania patients have comorbid skin picking and nail biting (O'Sullivan et al. 2000, Lochner et al. 2002, Christenson et al. 1991a, Simeon et al. 1997).

Individuals with trichotillomania have increased risk for mood disorders (major depressive disorder, dysthymic disorder) and anxiety symptoms (Table 000–4). The frequency of specific anxiety disorders (like generalized anxiety and panic disorders and OCD) may be increased as well. Compared to controls, college students who reported frequent hair pulling or skin picking also reported significantly more symptoms of anxiety and stress reactivity, and had higher scores on a measure of obsessive-compulsive symptoms (Hajcak et al. 2006). Although it has been suggested that trichotillomania in childhood or adolescence is associated with schizophrenia or severe disruptions of the family system, no systematically collected data support such conclusions.

Christenson et al. (1991a) 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 affective, 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). In a larger sample of adults seeking treatment for trichotillomania, Christenson (1995) found comorbidity rates of 57% for major depression, 27% for generalized anxiety disorder, 20% for eating disorders, 19% for alcohol abuse, and 16% for other substance abuse. In a mixed sample of children, adolescents, and adults with trichotillomania, Swedo and Leonard (1992) found comorbidity rates of 39% for unipolar depression, 32% for generalized anxiety disorder, 16% for OCD, and 15% for substance abuse. Reeve et al. (1992) and King et al. (1995b) found that 7 of 10 and 9 of 15 children with trichotillomania had at least one comorbid Axis I disorder, respectively. Franklin et al. (2002a) and Tolin et al. (2002) reported little comorbidity in their pediatric treatment-seeking samples, suggesting that comorbidity may develop secondarily in the wake of trichotillomania. Sampling issues most likely underlie these observed differences. Nevertheless, if it is indeed the case that children and adolescents with trichotillomania are less comorbid than adults, early intervention in children and adolescents with trichotillomania may help reduce the rates and severity of later adult psychiatric comorbidity and functional impairment (Keuthen et al. 2002).

## Course

The age at onset typically ranges from early childhood to young adulthood. Peak ages at presentation may be bimodal, with an earlier peak about age 5 to 8 years among children in whom it has a self-limited course, whereas among patients who present to clinicians in adulthood the mean age at onset is approximately 13 years (Rothbaum et al. 1993, Winchel 1992, Swedo et al. 1989). Initial onset after young adulthood is apparently uncommon. There have been reports of onset as early as 14 months of age and as late as 61 years.

Trichotillomania may be one of the earliest occurring conditions in psychiatry. Some parents insist that their child began pulling hair before 1 year of age. When trichotillomania begins before age 6 years it tends to be a milder condition. It often responds to simple interventions and may be self-limited, with a duration of several weeks to several months, even if not treated. It often occurs in association with thumb sucking. In some cases it remits spontaneously when therapeutic attention is directed at concurrent, severe thumb -sucking (Watson and Allen 1993). It has been suggested that trichotillomania in childhood may be associated with severe intrapsychic or familial psychiatric conditions. But there is no reliable evidence that supports such a conclusion. Indeed, some have suggested that because it may be common and frequently self-limiting, it should be considered a normal behavior among young children.

Tricotillomania in adolescents and adults typically follows a chronic course, involves multiple hair sites, and

| Table 000–4 | Lifetime Comorbidity and Trichotillomania | | | | | |
|---|---|---|---|---|---|---|
| Reference | No Axis I | Anxiety Disorder | Mood Disorder | PSUD[*] | OCD | Eating Disorder |
| Christenson et al. (1991a) (*n* = 60) | 18% | 57% | 65% | 22% | 15% | 20% |
| Winchel et al. (1992) (*n* = 20) | 45% | 5% | 45% | 15% | 5% | 0% |
| Swedo et al. (1989) (*n* = 14) | ? | 43% | 57% | 29% | † | 0% |

[*]PSUD, Psychoactive substance use disorder.

[†]Patients with OCD were excluded from this sample, as were patients with psychosis.

is associated with high rates of psychiatric comobidity (Christenson et al. 1991a). The chronic course may take one of two patterns: in one the frequency and severity of hair pulling waxes and wanes over months, without any true remissions; in the other, episodes are characterized by frequent hair pulling separated by long periods of remission (Moore and Jefferson, 2004). Some individuals have continuous symptoms for decades. For others, the disorder may come and go for weeks, months, or years at a time. Sites of hair pulling may vary over time. Circumscribed periods of hair pulling (weeks to months) followed by complete remission are reported among children. Progression of the condition appears to be unpredictable. It is not known which factors may predict a protracted and unremitting course.

Because of the unavailability of longitudinal studies of trichotillomania, generalizations about prognosis cannot be made. Patients who present in research clinics typically have histories of many years (up to decades) of hair pulling. Presentation after age 40 years appears to be far less common than in the previous three decades of life, suggesting that the condition may eventually remit spontaneously, even when untreated. It is likely that the persistent cases seen in research environments reflect the more severe end of the spectrum. As noted earlier, trichotillomania in children may often be a time-limited phenomenon.

### Differential Diagnosis

Among individuals presenting with alopecia who complain of hair-pulling urges, the diagnosis is not usually in doubt. When patients deny hair pulling, other (dermatological) causes of alopecia should be considered. These include alopecia areata, male pattern hair loss, chronic discoid lupus erythematosus, lichen planopilaris, folliculitis decalvans, pseudopelade, and alopecia mucinosa.

Trichotillomania is not diagnosed when hair pulling occurs in response to a delusion or hallucination. Many people twist and play with their hair. This may be exacerbated in states of heightened anxiety but does not qualify for a diagnosis of trichotillomania. Some may present with features of trichotillomania but hair damage may be so slight as to be virtually undetectable, even under close examination. In such conditions the disorder should be diagnosed only if it results in significant distress to the individual.

Trichotillomania may have a short, self-limited course among children and may be considered a temporary habit. Therefore, among children the diagnosis should be reserved for situations in which the behavior has persisted during several months.

### Differences in Gender and Cultural Presentations

Secondary avoidance of intimate relationships, which occurs among some individuals with trichotillomania, may be exacerbated for women in cultures in which physical appearance is weighted differently for men and women. Avoidance of sports activities, in which disguised hair loss can be revealed, may also have gender-related effects in cultures in which athletic participation has different social meanings for men and women. Although culture-based expectations regarding appearance may make hair loss a greater burden for women, women may have a greater opportunity to hide hair loss through the use of wigs, hats, and scarves.

Reliable data regarding sex ratio in the general population are not yet available. It has long been suggested that women greatly outnumber men. However, surveys of college students suggest that the true ratio may be near parity. The apparent preponderance of women presenting for treatment may alternatively reflect self-selection for presentation for treatment. Self-selection may reflect gender related, culturally based attitudes regarding appearance, as well as an acceptance of normative hair loss among men. Because such gender-related distinctions may not be made by parents who are concerned about hair-pulling habits in their children, the apparent equal presentation of male and female children may more accurately reflect the true sex ratio. For many women, hair pulling may worsen during the premenstrual phase (Keuthen et al. 1997).

### Etiology and Pathophysiology

The etiology of trichotillomania is unknown. The phenomenological similarities between trichotillomania and OCD have prompted speculations that the pathophysiology of the two conditions may be related. The apparent association between altered serotonergic function and OCD has guided attention toward the possible role of serotonergic function in the underlying cause of trichotillomania. Thus, interest has been spurred in examining serotonergic function in patients with trichotillomania.

Ninan et al. (1992) obtained CSF from eight individuals with trichotillomania and measured concentrations of the primary serotonin metabolite 5-HIAA. Baseline concentrations of 5-HIAA did not differ from those of control subjects, nor was there a relationship between the baseline 5-HIAA concentration and the severity of trichotillomania symptoms. But, seven of these patients were then treated with SSRIs (fluoxetine and clomipramine). The researchers found a negative correlation between baseline CSF 5-HIAA concentration and the degree of improvement after treatment. This observation does not, however, directly support a conclusion that altered serotonin function is etiologically related to trichotillomania.

Swedo et al. (1991) used PET to measure regional brain glucose in three groups: trichotillomania patients, OCD patients, and normal controls. Like OCD patients, those with trichotillomania had altered patterns of glucose utilization compared with normal controls. However, the regional patterns of altered glucose utilization differed between trichotillomania and OCD groups. In a morphometric MRI study, left putamen volume was found to be significantly smaller in 10 female trichotillomania subjects as compared with 10 normal matched controls (O'Sullivan et al. 1997).

Performance on neuropsychological tests may offer an additional basis for defining the underlying neuropathological process in people with trichotillomania. Because impaired performance on such tests may indicate altered function in particular brain regions, they may also help localize brain regions in which altered function may be associated with trichotillomania. On the basis of such tests, Rettew et al. (1991) suggested that trichotillomania patients may have deficits in spatial processing. Patterns of deficits on such tests may provide further support for a relationship between trichotillomania and other psychiatric conditions. Rettew et al. (1991) found similarities between trichotillomania and OCD subjects.

Keuthen et al. (1996) also speculated that people with trichotillomania would demonstrate alterations in neuropsychological function similar to individuals with OCD, who have been shown to have impairments in executive, visual-spatial and nonverbal memory function. In a study of 20 trichotillomania subjects and 20 matched healthy controls, they demonstrated the presence of impaired performance on two of these three parameters (nonverbal memory and executive function). These results were interpreted as supporting the presumed relationship between trichotillomania and OCD. However, Stanley et al. (1997a), in their study of 21 trichotillomania subjects versus 17 healthy controls, did not find evidence of deficits in visual-spatial ability, motor function, or executive function. But, differences were found on measures of divided attention, leading to the suggestion that trichotillomania might be more properly conceptualized as an affective or anxiety-based disorder, and that any demonstrated similarities with OCD may be related to their shared overlap with anxiety/affective disorders.

Additional support for a possible relationship between trichotillomania and OCD may come from family studies. In a preliminary investigation of psychiatric diagnoses among first-degree relatives of probands with trichotillomania, Lenane et al. (1992) found increased frequencies (compared with normal control subjects) of OCD, as well as mood and anxiety disorders. Bienvenu et al. (2000) examined 300 first-degree relatives of 343 OCD patients, and found increased rates of "grooming" conditions (e.g., nail-biting, skin-picking, trichotillomania), and other ICDs (e.g., kleptomania, pathological gambling, pyromania).

In sum, few data are available to support any particular model of the etiological pathophysiology of trichotillomania. Early studies point to some alteration of brain activity. There is inconsistent support in these studies for a relationship with OCD. Figure 000–2 shows a schematic diagram of a preliminary biopsychosocial model of trichotillomania (Franklin et al. 2006). This model is a heuristic, not explanatory, but is hoped to stimulate new studies on the mechanisms of trichotillomania and to be modified as new data emerges.

## Treatment

### Treatment Goals

Treatment of trichotillomania typically occurs in an outpatient setting. Eradication of hair-pulling behavior is the general focus of treatment. Distress, avoidant behaviors, and cosmetic impairment are secondary to the hair-pulling behavior and would be likely to remit if the hair-pulling behavior is controlled. However, if sufficient control of hair pulling cannot be attained, treatment goals should emphasize these associated problems as well. Even if hair pulling persists, therapeutic interventions may be targeted at reducing secondary avoidance and diminishing distress. Treatment may be considered in three phases:

*Initial Contact*: The diagnosis is made and the patient and psychiatrist agree on a strategy that may incorporate both pharmacological and psychological interventions. If distress is severe, supportive interventions should be immediately considered in anticipation of incomplete treatment response or of a delay of weeks to months before interventions may be beneficial.

*Acute Treatment*: Even when treatment of hair-pulling behavior is optimally successful, there may be a delay of several weeks to months before adequate control is attained. So, the acute treatment phase may be prolonged.

*Maintenance*: It is not known how long patients must maintain active treatment interventions to prevent relapse. It should be anticipated that a substantial number of patients require ongoing treatment for an extended time. Pharmacological treatments may need to be maintained for open-ended periods. Behavioral or hypnotic intervention may require periodic "booster shots" to support continuation of benefits.

### Psychiatrist-Patient Relationship

It is important to bear in mind the particular nature of embarrassment that often accompanies this condition. Several factors contribute to feelings of shame for many with trichotillomania. When hair pulling has had its onset in childhood or adolescence, there is often a history of the hair pulling being treated as a family secret. Patients have been frequently castigated by parents or spouses for lack of self-control. There may also be a feeling that the problem is largely cosmetic, causing some individuals to fear they do not have the "right" to utilize health resources for its treatment. This may be manifest as fears of having their problem minimized or of being derided for seeking help. It is helpful for the clinician to share with patients an understanding that the problem pervades their daily life and may result in meaningful distress and functional inhibition.

A variety of treatment approaches have been advocated for trichotillomania. However, there have, as yet, been few controlled studies of the efficacy of any treatment approach. A review of the literature reveals that only eight randomized trials have been conducted thus far, seven of which included a control condition (see below). A number of investigations of the use of antidepressants with specific inhibition of serotonin reuptake (i.e., fluoxetine and clomipramine) have yielded mixed results (Rothbaum et al. 1993, Winchel et al. 1992, Swedo et al. 1989, Stein et al. 1997, Jaspers 1996). A multimodal approach, simultaneously utilizing several complementary treatment options, may turn out to be the most effective approach for most patients.

While a number of treatment options can be currently offered to individuals with trichotillomania, the durability of long-term outcomes is unclear. Keuthen et al. (1998, 2001) followed a group of hair pullers who had "naturalistic" treatment in the community. Treatments were pharmacological, behavioral or both. Among those who had benefits, improvements were often lost over time, and persistent treatment and ongoing treatment was common over the course of several years.

### Stress Management

Before embarking on a course of treatment, the psychiatrist and the patient should first consider the course and severity of the individual's condition. Because early remission may occur in cases of recent onset, mild trichotillomania of short duration does not necessarily require immediate intervention. In particular, if the hair pulling first occurred during a

period of stress, the behavior may spontaneously diminish as the stressful circumstances abate. In such circumstances, therapeutic attention may best be directed toward examining and seeking to diminish the basis for stress. Teaching alternative stress reduction methods may be useful in reducing recent-onset trichotillomania. However, when individuals with trichotillomania present to the psychiatrist, it is often likely to have been a persistent condition and may have been present for many years or decades. Among such patients, stress reduction may also be useful in reducing trichotillomania but complete remission is less likely.

## Somatic Treatments

The literature is generally made up of case studies, with progressively more controlled investigation in recent years. In general, knowledge about trichotillomania treatments is limited by small sample sizes, lack of specificity regarding sample characteristics, nonrandom assignment to treatment, dearth of long-term follow-up data, exclusive reliance on patient self-report measures, and lack of information regarding rates of treatment refusal and dropout.

A variety of medications have been used in the treatment of trichotillomania. In 1989, initial reports demonstrated the apparent benefits of fluoxetine and clomipramine. Clomipramine was found to be superior to desipramine (Swedo et al. 1989). Fluoxetine was reported beneficial in open treatment (Winchel et al. 1992). Although reports for more than 60 patients have subsequently added support for the use of these medications, the two double-blind studies in which fluoxetine has been compared with placebo did not demonstrate any improvement compared to placebo (Christenson et al. 1991b, Streichenwein and Thornby 1995). Fluvoxamine (Stanley et al. 1997b), citalopram (Stein et al. 1997), escitalopram (Gadde et al. 2007), and venlafaxine (Ninan et al. 1998) have been reported to be efficacious in open trials. Although more controlled trials of SSRIs are needed, the use of such medications would be a prudent first step if a pharmacological approach has been agreed upon.

Of the six randomized, controlled trials evaluating the efficacy of pharmacotherapy conducted to date, five involved SSRIs. This may reflect the previously prevailing view that trichotillomania is a variant of OCD and thus ought to be responsive to the same pharmacological agents proven successful in for OCD. In sum, results from these controlled studies of SSRIs are equivocal at best, although in view of the small sample sizes more controlled research should be conducted to determine their efficacy (Swedo et al. 1989, 1993, Christenson et al. 1991c, Streichenwein and Thornby 1995, Ninan et al. 2000, van Minnen et al. 2003). Perhaps important differences between OCD and trichotillomania underlie this apparent difference in treatment response. However, several case studies indicated that augmentation of SSRIs with atypical neuroleptics may be beneficial (Epperson et al. 1999, Stein and Hollander 1992), and an open trial suggested that olanzapine may be efficacious as a monotherapy for trichotillomania (Stewart and Nejtek 2003). Interestingly, naltrexone, an opioid antagonist, thought to decrease positive reinforcement, has also been found superior to placebo in reducing trichotillomania symptoms (Christenson et al. 1994).

Although no double-blind discontinuation studies have been conducted in trichotillomania, evidence from open studies suggests that treatment response gained from pharmacotherapy may not be maintained in the long run (Pollard et al. 1991, Iancu et al. 1996). The absence of a single randomized, controlled trial in pediatric trichotillomania limits treatment recommendations for this population.

Initial evidence of improvement is usually first reported by the patient as greater awareness of the inclination to pull hair. This is usually followed by an ability to abort hair-pulling episodes more quickly than in the past. The ability to resist the urge follows. In cases with a good outcome, the inclination to pull diminishes and may eventually disappear. Patients who pull from several sites may find that the rate of improvement varies from site to site.

There have been conflicting reports of early relapse of symptoms in some patients treated with clomipramine or fluoxetine. Although good maintenance of benefit has been reported for some patients 6 months and longer after the initiation of treatment, early relapse after several weeks to months has also been reported. Keuthen et al. (2001) provided long-term data on maintenance of response over time. Following a group of people who had varying forms of treatment (pharmacological and psychological) for several years after an index evaluation, the authors concluded that initial improvement was common, but over time there was an increase in symptom scores and self-esteem scores worsened. This problem needs further exploration in long-term treatment studies. If early relapse turns out to be common, it would distinguish trichotillomania from depression and OCD, in which medication benefits are often well maintained as long as medication is continued. Optimal duration of treatment for well-treated individuals is still unknown. In accord with standards developed for the treatment of other conditions, it would be reasonable to continue medication for at least 6 months before tapering. Reinitiation of treatment may be needed.

In a 16-week open trial, the anticonvusant drug topiramate, which has shown promising results in the treatment of ICDs, significantly decreased the severity of hair pulling in adults with trichotillomania (Lochner et al. 2006). Christenson et al. (1991c) have reported successful treatment with lithium. This observation awaits replication. Because trichotillomania is often accompanied by other manifestations of anxiety—and for many individuals is exacerbated by stressful conditions—attempts at treatment with anxiolytic agents may be useful as well. There are no published reports of such treatments.

Adjunctive treatment with pimozide, a neuroleptic agent, has been advocated for some patients who are refractory to other medications (Stein and Hollander 1992). The potential benefits of neuroleptics have been reported now by several authors (Potenza et al. 1998, Gabriel 2001, Gupta and Gupta 2000, Epperson et al. 1999). Most of these reports describe individuals for whom SSRIs provided insufficient benefits. The addition of atypical neuroleptics much improved their outcomes. The greater margin of safety and tolerability associated with atypical neuroleptics may make this a more viable treatment option, but the potential side effects of atypicals should still be taken into consideration. Van Ameringen et al. (1999) found that eight of nine trichotillomania patients responded to haloperidol. Six previously failed treatment with SSRIs. The possible superiority of neuroleptics prompted these authors to speculate

that trichotillomania may be similar to Tourette's syndrome, which responds preferentially to neuroleptics.

## Psychosocial Treatments

### Behavioral Treatment

Various behavioral techniques have been tried (Diefenbach et al. 2000). The most successful technique, habit reversal, is based on designing competitive behaviors that should inhibit the hair-pulling behavior (Azrin and Nunn 1977, Azrin et al. 1980, Rosenbaum and Ayllon 1981). For example, if hair pulling requires raising the arm to the scalp and contracting the muscles of the hand to grasp a hair, the behaviorist may design a behavioral program in which the patient is taught to lower the arm and extend the muscles of the hand. As with most behavioral techniques, these interventions are most successful when the patient is strongly motivated and compliant. Also, the treating psychiatrist should be experienced in the use of such techniques. If necessary, a referral should be made to an experienced individual. Modified behavioral approaches have been described for children and adolescents (Vitulano et al. 1992, Rapp et al. 1998).

In a randomized, controlled trial (Diefenbach et al. 2006), patients completing group behavior therapy ($n=12$) experienced significantly greater decreases in self-reported hair-pulling symptoms and clinician-rated hair loss severity than those in group supportive therapy ($n=12$). In addition, a significantly higher percentage of those in the behavior therapy condition were rated as much improved or very much improved on the Clinical Global Impression scale at post-treatment. But, despite substantial symptom improvement, trichotillomania severity remained problematic at post-treatment, few patients in either treatment met criteria for clinically significant change at post-treatment, and relapse of symptoms occurred over the 6-month follow-up period. So these results provided support for the short-term efficacy of group behavior therapy.

### Cognitive-Behavioral Therapy

CBT has been developed for, and applied to, trichotillomania patients. A variety of techniques have been used, and although the current CBT literature justifies only cautious recommendations, habit reversal, awareness training, and stimulus control are generally purported as the core efficacious interventions. Successful outcome has been reported on several of these interventions. But, since most of the literature consists of uncontrolled case reports or small case series, confident conclusions cannot be drawn. This is evidenced by the three randomized trials with adults exploring CBT efficacy. Ninan et al. (2000) found CBT superior to clomipramine and placebo at post-treatment; the same pattern was reported by van Minnen et al. (2003) in their randomized, controlled trial of CBT, fluoxetine, and a wait-list condition. Azrin et al. (1980) found that habit reversal was more effective than negative practice, where subjects were told to act out the motions of hair pulling in front of a mirror, without doing any damage, for 30 seconds every hour, and then to maintain the exercises for 4 days after entirely breaking their habit and finally to gradually decrease the exercises over a 2-week time period. The treatment rationale involves the principles of satiation and heightened awareness. The problem of relapse following CBT has been highlighted in several studies (Lerner et al.

1998, Keuthen et al. 2001, Mouton and Stanley 1996). The limited and equivocal treatment literature suggests that there is neither a universal nor a complete response to any treatment for trichotillomania. Controlled studies of the efficacy of CBT treatments involving habit reversal, pharmacotherapy, and their combination are needed.

### Hypnotherapy

There are no formal studies of the use of hypnosis for trichotillomania, but there are many published reports of beneficial treatment (Barabasz 1987, Cohen et al. 1999, Fabbri and Dy 1974, Kohen 1996, Rowen 1981, Zalsman et al. 2001). Benefits may be variable. Some patients may have dramatic improvement. For some who improve, the benefits may be short lived. As with behavioral interventions, the benefits of this approach are sometimes dependent on a highly motivated patient who can regularly carry out self-hypnotic measures as instructed by the psychiatrist. Some patients who have obtained partial benefits from either hypnosis or medication do well when both treatments are combined. Successful use of hypnotherapy for children with trichotillomania has also been reported (Cohen et al. 1999).

### Dynamic Psychotherapy

Many psychoanalytically oriented descriptions of people with trichotillomania have been published. These reports generally describe the psychodynamic formulations of individual cases and should not be the basis for generalizations about most individuals with trichotillomania. Although patients with trichotillomania may benefit from exploration and attempts to reduce intrapsychic conflict, the literature does not provide persuasive evidence of the efficacy of this approach in reducing hair pulling.

### Self-help and Other Groups

Self-help groups for patients with trichotillomania have appeared. Some are based in the structure of other 12-step programs. Some patients appear to experience meaningful reduction in hair-pulling symptoms after beginning participation in such a group. Although the efficacy of such groups in reducing symptoms remains to be established, most patients with trichotillomania can benefit from meeting other individuals with similar symptoms. Because of the lack of general awareness of trichotillomania, these individuals frequently believe that they are "oddball" individuals with a behavior that is unique. Many have experienced parental condemnation for the behavior and have been frequently castigated for a "habit" that may be viewed by others as under their voluntary control. The experience of meeting others with the condition is extremely supportive for such individuals and may help to reduce the attendant stress while supporting self-esteem. Where programs specifically oriented toward trichotillomania may not be generally available, these individuals may benefit from groups oriented toward OCD.

## Treatment of Comorbid Conditions

Depression, dysthymic disorder, and anxiety symptoms occur frequently in patients with trichotillomania. Successful treatment of depression may not be associated with reduction in trichotillomania. If depression or dysthymic disorder is present and independently provides an indication for medication, one of the antidepressants discussed earlier should be

chosen. If fluoxetine is used, the psychiatrist should be aware that a dose that is sufficient for reduction of the depressive symptoms may not be sufficient for reduction of trichotillomania. If panic disorder is present, either medication may still be used, but fluoxetine may initially exacerbate panic attacks in such patients and initiation of treatment at low doses (2.5–5 mg/day) should be considered. With slow titration upward, the patient should generally be able to tolerate usual doses with concomitant amelioration of the panic disorder. Combined treatment with anxiolytics may be useful for some and may contribute to the reduction in symptoms of trichotillomania. Other conditions that may be present, like OCD or eating disorders, may require special attention. Although fluoxetine may be useful for patients with eating disorders, medication treatment alone is unlikely to be adequate and the usual multimodal approaches for the treatment of bulimia nervosa or anorexia are appropriate. OCD may respond to treatment directed at trichotillomania, but adjunctive behavioral treatment of symptoms of OCD may be desirable.

## Age- and Cultural-Related Features

When trichotillomania presents in early childhood, as discussed earlier, the condition may be likely to be inherently self-limited. Often, all that may be necessary is to draw the child's attention to the behavior in some systematic way and to clarify for the child that the behavior is undesirable. Such methods include daily application of a nonmedicinal ointment to the affected region and reminding the child that the purpose is elimination of the hair-pulling habit. Some suggest that the child be given the responsibility of applying the ointment with parental supervision. Others suggest that parents should monitor the child as much as possible and respond with reminders that the hair should not be pulled and rewards with verbal encouragement for ceasing to pull hair. There have been no systematic studies of the benefits of such interventions, but dermatologists who specialize in the treatment of children have noted that hair-pulling behavior may frequently disappear within a few weeks of initiating such an approach. In circumstances in which childhood trichotillomania is more persistent, the parent and psychiatrist are faced with a dilemma. More elaborate behavioral interventions, such as habit reversal, should be tried. This, however, may be difficult with a child. Rosenbaum and Ayllon (1981) have described a modified version of habit reversal that may be employed with children. Hypnosis has been also used in the treatment of habit disorders in children. Medication should be cautiously considered when treating childhood trichotillomania. Although medication may be useful, the absence of data supporting the benefits of such treatments in children indicates a conservative approach. If medication is considered, its use in the treatment of childhood OCD should serve as a guideline.

Should the psychiatrist be presented with trichotillomania in a person of advanced age, special attention should be paid to usual concerns regarding the use of these medications in the elderly. Lower doses of medication should be considered because of potential altered pharmacokinetics in older persons. Medications with anticholinergic side effects (such as clomipramine) may present greater hazards for the older person. Sedative-hypnotic anxiolytics should be used sparingly because of greater vulnerability to cognitive side effects and the increased risk of falling. Women of

childbearing potential (perhaps the majority of people who may present for treatment) should be advised regarding the potential risks of these medications to a developing fetus. If the patient is pregnant or considering pregnancy, behavioral treatments may be favored.

Clinicians should be sensitive to the interaction between cultural values and trichotillomania. Women of some cultures may be more prone to distress if trichotillomania is perceived as a hindrance to achieving valued goals, like marriage. Also, in some communities, wigs and other hair accessories are generally acceptable and may present a comfortable means of diminishing the cosmetic impact of hair loss. In other communities, such accoutrements may themselves draw undesired attention.

## Refractory Response or Nonresponse to Initial Treatment

Because research in the treatment of trichotillomania is still limited, it is not possible to recommend an initial best treatment for all patients. However, the decision is often determined by available resources. Support groups may not be easily found in many areas. Hypnotherapy and behavioral therapy may be more easily available, but psychiatrists with these skills may not be experienced in the specific techniques used in this condition. Pharmacological interventions may be more readily available. Wherever possible, simultaneous multimodal interventions should be considered. Pharmacological, behavioral, and hypnotic interventions, which may each be only partially useful, may be synergistic when used in combination.

If therapy with a single medication is not successful, the psychiatrist may consider augmenting one agent with another. Augmentation strategies in the treatment of trichotillomania have not been studied. General principles of augmentation used in the treatment of depression or OCD may be considered. There may be particular benefit in combining anxiolytic agents (such as buspirone or clonazepam) with an SSRI antidepressant. As noted above, the advent of atypical neuroleptics may offer a new and possibly efficacious treatment option. Despite the increasing safety of these medications, caution should be used in the introduction of a neuroleptic for the treatment of a persistent condition.

### Clinical Vignette 4

Mr. G, a 32-year-old podiatrist, began pulling out hairs in his second year of college. He had always been a generally anxious person and thought of it as a nervous habit. Never particularly concerned about his appearance and noting the familial disposition to male pattern hair loss, he felt resigned to eventual baldness and thought little about it. He noted that his hair pulling tended to be worse in a variety of circumstances: before examinations, after a breakup with a girlfriend, while studying, and while watching television. He thought the last circumstance surprising. The others seemed to be situations of understandable stress, but television relaxed him. Indeed, at those times he was hardly aware of it until he would find his hands roaming searchingly through his scalp and would then find a small pile of hairs beside him on the sofa. Occasionally, an acquaintance would comment—with varying degrees of tact—on the ratty appearance of his hair, particularly above the left temple. Nevertheless, he was still not too

concerned. He did note a progression of the habit until he was pulling from virtually every spot on his body where hair grew, including his perineum.

Despite Mr. G's relative lack of concern, his new wife was not so resigned to the habit. She could not deny the mild revulsion she felt when she saw the range of locations from which he pulled hair. (In the beginning of their courtship, she was aware of only the thinned scalp.) She insisted that he go to a psychiatrist.

Mr. G's new psychiatrist tried a variety of interventions. Noting Mr. G's general anxiety, the psychiatrist instructed him in relaxation techniques, while exploring with him the sources of stress in his life. This was not too helpful. They then embarked on a course of medication trials, including three different SSRIs. There was encouraging improvement at first, but a partial relapse occurred and the benefits were limited. The psychiatrist then heard about a local therapist who led a group therapy for people with trichotillomania and also practiced hypnotic techniques for the problem. With some hesitancy, Mr. G joined the group. He was initially uncomfortable, being the only man in the group, and he did not identify with many of the concerns expressed by the women. But he settled in, became an active participant, and was surprised to find that his hair-pulling diminished further. He found that he was pulling from fewer sites and was able to abort hair-pulling episodes more quickly. Fortified by the partial successes and encouraged by the other group members, he went to a behavioral psychologist, who used a technique called habit reversal to supply him with a repertoire of skills he could employ to control urges to pull his hair. Finally, with this multimodal approach (medication, hypnosis, and group and behavioral therapy) his hair pulling virtually disappeared. He had occasional relapses when under significant stress, but by and large he was relieved of his symptom. He and his wife thought the substantial efforts he had made to rid himself of the problem were worthwhile.

## Comparison of DSM-IV/ICD-10 Diagnostic Criteria

The ICD-10 Diagnostic Criteria for Research do not include diagnostic criteria for IED. It is included in ICD-10 as an "other habit and Impulse-Control Disorder." The ICD-10 Diagnostic Criteria for Research and the DSM-IV-TR criteria for kleptomania, pyromania, and trichotillomania are essentially equivalent. Finally, the ICD-10 Diagnostic Criteria for Research for pathological gambling are monothetic (i.e., A plus B plus C plus D are required) whereas the DSM-IV-TR criteria set is polythetic (i.e., 5 out of 10 required) with different items. Furthermore, the ICD-10 criteria specify "two or more episodes of gambling over a period of at least 1 year," whereas DSM-IV-TR does not specify a duration.

## References

American Psychiatric Association (1980) Diagnostic and Statistical Manual of Mental Disorders, 3rd edition. APA, Washington, DC, USA.

American Psychiatric Association (1987) Diagnostic and Statistical Manual of Mental Disorders, 3rd edition Revised. APA, Washington, DC, USA.

American Psychiatric Association (2000) Diagnostic and Statistical Manual of Mental Disorders, 4th edition. Text Rev. APA, Washington, DC, USA.

Azrin NA and Nunn R (1977) Habit Control in a Day. Simon & Schuster, New York, USA.

Azrin NS, Nunn RG, and Frantz SE (1980) Treatment of hair-pulling (trichotillomania): A comparitive study of habit reversal and negative practice training. Journal of Behavior Therapy and Experimental Psychiatry 11, 13–20.

Barabasz M (1987) Trichotillomania: A new treatment. International Journal of Clinical and Experimental Hypnosis 35, 146–154.

Barker AF (1994) Arson: A Review of the Psychiatric Literature. Institute of Psychiatry, Maudsley Monographs No 35. Oxford University Press, Oxford, UK.

Barnett W, Richter P, and Renneberg B (1999) Repeated arson: Data from criminal records. Forensic Science International 101, 49–54.

Barnett W, Richter P, Sigmund D, et al. (1997) Recidivism and concomitant criminality in pathological firesetters. Journal of Forensic Science 42, 879–883.

Bars DR, Heyrend FL, Simpson CD, et al. (2001) Use of visual evoked-potential studies and EEG data to classify aggressive, explosive behavior of youths. Psychiatric Services 52, 81–86.

Bayle FJ, Caci H, Millet B, et al. (2003) Psychopathology and comorbidity of psychiatric disorders in patients with kleptomania. American Journal of Psychiatry 160, 1509–1513.

Bechara A (2001) Neurobiology of decision-making: Risk and reward. Seminars in Clinical Neuropsychiatry 6, 205–216.

Bechara A (2003) Risky business: Emotion, decision-making, and addiction. Journal of Gambling Studies 19, 23–51.

Bechara A, Damasio AR, Damasio H, et al. (1994) Insensitivity to future consequences following damage to human prefrontal cortex. Cognition 50, 7–15.

Bechara A, Damasio H, and Damasio AR (2000) Emotion, decision making and the orbitofrontal cortex. Cerebral Cortex 10, 295–307.

Bechara A, Dolan S, Denburg N, et al. (2001) Decision-making deficits, linked to a dysfunctional ventromedial prefrontal cortex, revealed in alcohol and stimulant abusers. Neuropsychologia 39, 376–389.

Beldoch M (1991) Stolen objects as transitional objects. American Journal of Psychiatry 148, 1754.

Bergh C, Eklund T, Sodersten P, et al. (1997) Altered dopamine function in pathological gambling. Psychological Medicine 27, 473–475.

Berlin HA, Rolls ET, and Kischka U (2004) Impulsivity, time perception, emotion and reinforcement sensitivity in patients with orbitofrontal cortex lesions. Brain 127, 1108–1126.

Best M, Williams JM, and Coccaro EF (2002) Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. Proceedings of the National Academy of Sciences USA 99, 8448–8453.

Bhatia MS, Singhal PK, Rastogi V, et al. (1991) Clinical profile of trichotillomania. The Journal of the Indian Medical Association 89, 137–139.

Bienvenu OJ, Samuels JF, Riddle MA, et al. (2000) The relationship of obsessive-compulsive disorder to possible spectrum disorders: Results from a family study. Biological Psychiatry 48, 287–293.

Birmaher B, Stanley M, Greenhill L, et al. (1990) Platelet imipramine binding in children and adolescents with impulsive behavior. Journal of the American Academy of Child & Adolescent Psychiatry 29, 914–918.

Blanco C, Petkova E, Ibanez A, et al. (2002) A pilot placebo-controlled study of fluvoxamine for pathological gambling. Annals of Clinical Psychiatry 14, 9–15.

Blanco C, Moreyra P, Nunes EV, et al. (2001) Pathological gambling: Addiction or compulsion? Seminars in Clinical Neuropsychiatry 6, 167–176.

Blanco C, Orensanz-Munoz L, Blanco-Jerez C, et al. (1996) Pathological gambling and platelet MAO activity: A psychobiological study. American Journal of Psychiatry 153, 119–121.

Bland RC, Newman SC, Orn H, et al. (1993) Epidemiology of pathological gambling in Edmonton. Canadian Journal of Psychiatry 38, 108–112.

Blum K, Braverman ER, Holder JM, et al. (2000) Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors. Journal of Psychoactive Drugs 32 (suppl), 1–68.

Brandford J and Dimock J (1986) A comparative study of adolescents and adults who wilfully set fires. Psychiatric Journal of University of Ottawa 11, 228–234.

Breiter HC, Aharon I, Kahneman D, et al. (2001) Functional imaging of neural responses to expectancy and experience of monetary gains and losses. Neuron 30, 619–639.

Brown RI (1986) Arousal and sensation-seeking components in the general explanation of gambling and gambling addictions. International Journal of the Addictions 21, 1001–1016.

Brown RI (1985) The effectiveness of Gamblers Anonymous. In The Gambling Studies: Proceedings of the 6th National Conference on Gambling and Risk-Taking, Eadington WR and Reno NV (eds). University of Nevada.

Budman CL, Bruun RD, Park KS, et al. (2000) Explosive outbursts in children with Tourette's disorder. Journal of the American Academy of Child & Adolescent Psychiatry 39, 1270–1276.

Budman CL, Bruun RD, Park KS, et al. (1998) Rage attacks in children and adolescents with Tourette's disorder: A pilot study. Journal of Clinical Psychiatry 59, 576–580.

Bumpass ER, Fagelman FD, and Brix RJ (1983) Intervention with children who set fires. American Journal of Psychotherapy 37, 328–345.

Burstein A (1992) Fluoxetine-lithium treatment for kleptomania. Journal of Clinical Psychiatry 53, 28–129.

Carlton PL and Goldstein L (1987) Physiological determinants of pathological gambling. In A Handbook of Pathological Gambling, Glaski T (ed), Charles C Thomas, Springfield IL, pp 657–663.

Carrasco JL, Saiz-Ruiz J, Hollander E, et al. (1994) Low platelet monoamine oxidase activity in pathological gambling. Acta Psychiatrica Scandinavica 90, 427–431.

Cavedini P, Riboldi G, Keller R, et al. (2002) Frontal lobe dysfunction in pathological gambling patients. Biological Psychiatry 51, 334–341.

Chambers RA, Taylor JR, and Potenza MN (2003) Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. American Journal of Psychiatry 160, 1041–1052.

Chiswick D (1976) Shoplifting, depression and an unusual intracranial lesion (a case report). Medicine, Science and the Law 16, 266–268.

Chong SA and Low BL (1996) Treatment of kleptomania with fluvoxamine. Acta Psychiatrica Scandinavica 93, 314–315.

Christenson GA (1995) Trichotillomania: from prevalence to comorbidity. Psychiatric Times 12, 44–48.

Christenson GA and Mansueto CS (1999) Trichotillomania: Descriptive characteristics and phenomenology. In Trichotillomania, Stein DJ, Christenson GA, and Hollander E (eds). American Psychiatric Press, Washington, DC, USA, pp 1–41.

Christenson GA, Crow SJ, and Mackenzie TB (1994) A placebo controlled double blind study of naltrexone for trichotillomania. Paper presented at the 147th annual meeting of the American Psychiatric Association, Philadelphia, PA, USA.

Christenson GA, Chernoff-Clementz E, and Clementz BA (1992) Personality and clinical characteristics in patients with trichotillomania. Journal of Clinical Psychiatry 53, 407–413.

Christenson GA, Mackenzie TB, and Mitchell JE (1991a) Characteristics of 60 adult chronic hair pullers. American Journal of Psychiatry 148, 365–370.

Christenson GA, Mackenzie TB, Mitchell JE, et al. (1991b) A placebo-controlled, double-blind crossover study of fluoxetine in trichotillomania. American Journal of Psychiatry 148, 1566–1571.

Christenson GA, Popkin MK, Mackenzie TB, et al. (1991c) Lithium treatment of chronic hair-pulling. Journal of Clinical Psychiatry 52, 116–120.

Ciarrochi J and Richardson R (1989) Profile of compulsive gamblers in treatment: update and comparisons. Journal of Gambling Behavior 5, 53–65.

Coccaro EF, Posternak MA, and Zimmerman M (2005) Prevalence and features of intermittent explosive disorder in a clinical setting. Journal of Clinical Psychiatry 66, 1221–1227.

Coccaro EF, Schmidt CA, Samuels JF, et al. (2004) Lifetime and 1-month prevalence rates of intermittent explosive disorder in a community sample. Journal of Clinical Psychiatry 65, 820–824.

Coccaro EF, Kavoussi RJ, Hauger RL, et al. (1998a) Cerebrospinal fluid vasopressin: Correlates with aggression and serotonin function in personality disordered subjects. Archives of General Psychiatry 55, 708–714.

Coccaro EF, Kavoussi RJ, Berman ME, et al. (1998b) Intermittent explosive disorder-revised: development, reliability and validity of research criteria. Compr Psychiatry 39, 368–376.

Coccaro EF, Bergeman CS, Kavoussi RJ, et al. (1997a) Heritability of aggression and irritability: a twin study of the Buss-Durkee Aggression Scales in adult male subjects. Biol Psychiatry 41, 273–284.

Coccaro EF, Kavoussi RJ, and Hauger RL (1997b) Serotonin function and anti-aggressive responses to fluoxetine: a pilot study. Biol Psychiatry 42, 546–552.

Coccaro EF, Kavoussi RJ, Sheline YI, et al. (1996) Impulsive aggression in personality disorder correlates with tritiated paroxetine binding in the platelet. Archives of General Psychiatry 53, 531–536.

Coccaro EF, Lawrence T, Trestman R, et al. (1991) Growth hormone responses to intravenous clonidine challenge correlate with behavioral irritability in psychiatric patients and healthy volunteers. Psychiatry Res 39, 129–139.

Coccaro EF, Siever LJ, Klar HM, et al. (1989) Serotonergic studies in patients with affective and personality disorders: Correlates with suicidal and impulsive-aggressive behavior. Archives of General Psychiatry 46, 587–599.

Cohen HA, Barzilai A, and Lahat E (1999) Hypnotherapy: An effective treatment modality for trichotillomania. Acta Paediatrica 88, 407–410.

Cohen LJ, Stein DJ, Simeon D, et al. (1995) Clinical profile, comorbidity, and treatment history in 123 hair pullers: A survey study. Journal of Clinical Psychiatry 56, 319–326.

Coid J (1984) Relief of diazepam-withdrawal syndrome by shoplifting. British Journal of Psychiatry 145, 552–554.

Comings DE, Rosenthal RJ, Lesieur HR, et al. (1996) A study of the dopamine D2 receptor gene in pathological gambling. Pharmacogenetics 6, 223–234.

Comings DE, Gade R, Wu S, et al. (1997) Studies of the potential role of the dopamine D1 receptor gene in addictive behaviors. Molecular Psychiatry 2, 44–56.

Crean JP, de Wit H, and Richards JB (2000) Reward discounting as a measure of impulsive behavior in a psychiatric outpatient population. Experimental and Clinical Psychopharmacology 8, 155–162.

Cunningham-Williams RM, Cottler LB, Compton WM, et al. (1998) Taking chances: Problem gamblers and mental health disorders—results from the St. Louis Epidemiologic Catchment Area Study. American Journal of Public Health 88, 1093–1096.

Dannon PN (2003) Topiramate for the treatment of kleptomania: A case series and review of the literature. Clinical Neuropharmacology 26, 1–4.

Dannon PN, Iancu I, and Grunhaus L (1999) Naltrexone treatment in kleptomanic patients. Human Psychopharmacology 14, 583–585.

DeCaria CM, Begaz T, and Hollander E (1998a) Serotonergic and noradrenergic function in pathological gambling. CNS Spectrums 3, 38–47.

DeCaria CM, Hollander E, Begaz T, et al. (1998b) Reliability and validity of a pathological gambling modification of the Yale-Brown Obsessive-Compulsive Scale (PG-YBOCS): Preliminary findings. 12th National Conference of Problem Gambling, Las Vegas, NV, USA, July 17–20.

DeCaria CM, Hollander E, Grossman R, et al. (1996) Diagnosis, neurobiology, and treatment of pathological gambling. Journal of Clinical Psychiatry 57, 80–83; discussion 83–84.

Deffenbacher JL, Filetti LB, Lynch RS, et al. (2002) Cognitive-behavioral treatment of high anger drivers. Behavior Research and Therapy 40, 895–910.

Deffenbacher JL, Lynch RS, Oetting ER, et al. (2000) Characteristics and treatment of high-anger drivers. Journal of Counseling Psychology 47, 5–17.

DeJong J, Virkkunen M, and Linnoila M (1992) Factors associated with recidivism in a criminal population. Journal of Nervous and Mental Disease 180, 543–550.

de la Gandara JJ (1999) Fluoxetine: Open-trial in pathological gambling. Paper presented at the 152nd annual meeting of the American Psychiatric Association, Washington, DC, USA.

Dell'Osso B, Allen A, and Hollander E (2005) Comorbidity issues in the pharmacological treatment of Pathological Gambling: a critical review. Clinical Practice and Epidemiology in Mental Health 10, 1–21.

DePue RA, Luciana M, Arbisi P, et al. (1994) Dopamine and the structure of personality: Relation of agonist-induced dopamine activity to positive emotionality. Journal of Personality and Social Psychology 67, 485–498.

Dickerson M, Hinchy J, and England SL (1990) Minimal treatments and problem gamblers: A preliminary investigation. Journal of Gambling Studies 6, 87–102.

Dickerson M, Hinchy J, and Falve J (1987) Chasing, arousal and sensation-seeking in off-course gamblers. British Journal of Addictions 82, 673–680.

Diefenbach GJ, Reitman D, and Williamson DA (2000) Trichotillomania: A challenge to research and practice. Clinical Psychology Review 20, 289–309.

Diefenbach GJ, Tolin DF, Hannan S, et al. (2006) Group treatment for trichotillomania: Behavior therapy versus supportive therapy. Behavior Therapy 37(4), 353–363.

Dolan M, Anderson IM, and Deakin JF (2001) Relationship between 5-HT function and impulsivity and aggression in males offenders with personality disorders. British Journal of Psychiatry 178, 352–359.

Durst R, Katz G, and Knobler H (1997) Buspirone augmentation of fluvoxamine in the treatment of kleptomania. Journal of Nervous and Mental Disease 185, 586–588.

Eisen SA, Lin N, Lyons MJ, et al. (1998) Familial influences on gambling behaviour: An analysis of 3359 twin pairs. Addiction 93, 1375–1384.

Eisen SA, Slutske WS, Lyons MJ, et al. (2001) The genetics of pathological gambling. Seminars in Clinical Neuropsychiatry 6, 195–204.

Epperson CN, Fasula D, Wasylink S, et al. (1999) Risperidone addition in serotonin reuptake inhibitor-resistant trichotillomania: Three cases. Journal of Child and Adolescent Psychopharmacology 9, 43–49.

Fabbri R Jr and Dy AJ (1974) Hypnotic treatment of trichotillomania: Two cases. International Journal of Clinical and Experimental Hypnosis 22, 210–215.

Felthous AR, Bryant SG, Wingerter CB, et al. (1991) The diagnosis of intermittent explosive disorder in violent men. Bulletin of the American Academy of Psychiatry and the Law 19, 71–79.

Fenichel O (1945) The Psychoanalytic Theory of Neurosis. WW Norton, New York, USA.

Fiorillo CD, Tobler PN, and Schultz W (2003) Discrete coding of reward probability and uncertainty by dopamine neurons. Science 299, 1898–1902.

First M and Frances A (eds) (1995) DSM-IV Handbook of Differential Diagnosis. American Psychiatric Press, Washington, DC, USA, pp 196–197.

Fishbain DA (1987) Kleptomania as risk-taking behavior in response to depression. American Journal of Psychotherapy 41, 598–603.

Fishbain DA (1988) Kleptomanic behavior response to perphenazine-amitriptyline HCl combination. Canadian Journal of Psychiatry 33, 241–242.

Franklin ME, Tolin DF, and Diefenbach GJ (2006). Trichotillomania. In Clinical Manual of Impulse Control Disorders, Hollander E and Stein D (eds). American Psychiatric Publishing Inc., Arlington, VA, USA.

Franklin ME, Keuthen NJ, Spokas ME, et al. (2002a) Pediatric trichotillomania: Descriptive psychopathology and comorbid symptomatology. Paper presented at the Trichotillomania: Psychopathology and Treatment Development Symposium, conducted at the 36th annual meeting of the Association for the Advancement of Behavior Therapy, Reno, NV, USA.

Franklin GA, Pucci PS, Arbabi S, et al. (2002b) Decreased juvenile arson and firesetting recidivism after implementation of a multidisciplinary prevention program. Journal of Trauma 53, 260–266.

Freud S (1955) Beyond the pleasure principle. In The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. 18, Strachey J (ed). Hogarth Press, London, UK, pp 7–64; (Originally published in 1920).

Freud S (1961) Dostoevsky and parricide. In The Standard Edition of the Complete Psychological Works of Sigmund Freud, Vol. 21, Strachey J (ed). Hogarth Press, London, UK.

Friman PC and Hove G (1987) Apparent covariation between child habit disorders: Effects of successful treatment for thumb-sucking on untargeted chronic hair-pulling. Journal of Applied Behavior Analysis 20, 421–425.

Gabriel A (2001) A case of resistant trichotillomania treated with risperidone-augmented fluvoxamine. Canadian Journal of Psychiatry 46, 285–286.

Gadde KM, Ryan Wagner H 2nd, Connor KM, et al. (2007) Escitalopram treatment of trichotillomania. International Clinical Psychopharmacology 22(1), 39–42.

Gambino B, Fitzgerald R, Shaffer HJ, et al. (1993) Perceived family history of problem gambling and scores on SOGS. Journal of Gambling Studies 9, 169–184.

Gauthier J and Pellerin D (1982) Management of compulsive shoplifting through covert sensitization. Journal of Behavior Therapy and Experimental Psychiatry 13, 73–75.

Geller JL (1987) Firesetting in the adult psychiatric population. Hospital & Community Psychiatry 38, 501–506.

Geller JL and Bertsch G (1985) Fire-setting behavior in the histories of a state hospital population. Americn Journal of Psychiatry 142, 464–468.

Gerstein DR, Volberg RA, Harwood R, et al. (1999) Gambling Impact and Behavior Study: Report to the National Gambling Impact Study Commission. National Opinion Research Center at the University of Chicago, Chicago, IL, USA.

Giammanco M, Tabacchi G, Giammanco S, et al. (2005) Testosterone and aggressiveness. Medical Science Monitor 11, RA136–RA145.

Glover JH (1985) A case of kleptomania treated by covert sensitization. British Journal of Clinical Psychology 24, 213–214.

Goldman MJ (1991) Kleptomania: Making sense of the nonsensical. American Journal of Psychiatry 148, 986–996.

Grant JE (2004) Co-occurrence of personality disorders in persons with kleptomania: A preliminary investigation. Journal of the American Academy of Law and Psychiatry 34, 395–398.

Grant JE and Kim SW (2003) Comorbidity of impulse control disorders among pathological gamblers. Acta Psychiatrica Scandinavica 108, 207–213.

Grant JE and Kim SW (2002a) Adolescent kleptomania treated with naltrexone: A case report. European Child and Adolescent Psychiatry 11, 92–95.

Grant JE and Kim SW (2002b) Clinical characteristics and associated psychopathology of 22 patients with kleptomania. Comprehensive Psychiatry 43, 378–384.

Grant JE and Kim SW (2002c) An open label study of naltrexone in the treatment of kleptomania. Journal of Clinical Psychiatry 63, 349–356.

Grant JE and Kim SW (2002d) Temperament and early environmental influences in kleptomania. Comprehensive Psychiatry 43, 223–229.

Grant JE and Kim SW (2002e) Pharmacotherapy of pathological gambling. Psychiatry Annals 32, 186–191.

Grant JE, Kim SW, Potenza MN, et al. (2003) Paroxetine treatment of pathological gambling: a multi-centre randomized controlled trial International Clinical Psychopharmacology 18, 243–249.

Grant JE and Potenza MN (eds) (2004) Pathological Gambling: A Clinical Guide to Treatment. American Psychiatric Publishing, Washington, DC, USA.

Grant JE, Potenza MN, Levine L, et al. (2005) Prevalence of impulse control disorders in adult psychiatric inpatients. American Journal of Psychiatryy 162, 2184–2188.

Grodnitzky GR and Tafrate RC (2000) Imaginal exposure for anger reduction in adult outpatients: a pilot study. Journal of Behavior Therapy and Experimental Psychiatryy 31, 259–279.

Gudjonsson GH (1987) The significance of depression in the mechanism of "compulsive" shoplifting. Medicine, Science and the Law 27, 171–176.

Gupta MA and Gupta AK (2000) Olanzapine is effective in the management of some self-induced dermatoses: Three case reports. Cutis 66, 143–146.

Hajcak G, Franklin ME, Simons RF, et al. (2006) Hairpulling and skin picking in relation to affective distress and obsessive-compulsive symptoms. Journal of Psychopathology and Behavioral Assessment 28, 177–185.

Haller R and Hinterhuber H (1994) Treatment of pathological gambling with carbamazepine. Pharmacopsychiatry 27, 129.

Herzog DB, Keller MB, Sacks NR, et al. (1992) Psychiatric comorbidity in treatment-seeking anorexics and bulimics. Journal of Child and Adolescent Psychopharmacology 31, 810–818.

Holden C (2001) "Behavioral" addictions: do they exist? Science 294, 980–982.

Hollander E, Buchalter AJ, and DeCaria CM (2000a) Pathological gambling. Psychiatric Clinics of North America 23, 629–642.

Hollander E, DeCaria CM, Finkell JN, et al. (2000b) A randomized double-blind fluvoxamine/placebo crossover trial in pathologic gambling. Biological Psychiatr 47, 813–817.

Hollander E, DeCaria CM, Mari E, et al. (1998) Short-term single-blind fluvoxamine treatment of pathological gambling. American Journal of Psychiatry 155, 1781–1783.

Hollander E, Frenkel M, Decaria C, et al. (1992b) Treatment of pathological gambling with clomipramine. American Journal of Psychiatry 149, 710–711.

Hollander E, Posner N, and Cherkasky S (2002) Neuropsychiatric aspects of impulse-control disorders. In American Psychiatric Press Textbook of Neuropsychiatry, Yudofsky SC and Hales RE (eds). American Psychiatric Publishing, Washington, DC, USA, pp 579–596.

Hollander E, Pallanti S, Baldini Rossi N, et al. (2001) Sustained release lithium/placebo treatment response and FDG-PET imaging of wagering in bipolar spectrum pathological gamblers. Paper presented at the American College of Neuropsychopharmacology Annual Conference, Waikoloa Village, HI, USA.

Hudson JI and Pope HG (1990) Affective spectrum disorder: Does antidepressant response identify a family of disorders with a common pathophysiology? American Journal of Psychiatry 147, 552–564.

Hudson JI, Pope HG, Jonas JM, et al. (1983) Phenomenological relationship of eating disorders to major affective disorder. Psychiatry Research 9, 345–354.

Huesmann LR, Leonard E, Lefkowitz M, et al. (1984) Stability of aggression over time and generations. Development and Psychopathology 20, 1120–1134.

Iancu I, Weizman A, Kindler S, et al. (1996) Serotonergic drugs in trichotillomania: Treatment results in 12 patients. The Journal of Nervous and Mental Disease 184, 641–644.

Ibanez A, Blanco C, and Saiz-Ruiz J (2002) Neurobiology and genetics of pathological gambling. Psychiatric Annals 32, 181–185.

Ibanez A, Blanco C, Donahue E, et al. (2001) Psychiatric comorbidity in pathological gamblers seeking treatment. American Journal of Psychiatry 158, 1733–1735.

Jacobs DF (1988) Evidence for a common dissociative-like reaction among addicts. Journal of Gambling Behavior 4, 27–37.

Janet P (1911) La Kleptomanie et al. depression mentale. Journal de Psychologie et Normale Pathologique 8, 97–103.

Jaspers JP (1996) The diagnosis and psychopharmacological treatment of trichotillomania: A review. Pharmacopsychiatry 29, 115–120.

Kafry D (1980) Playing with matches: Children and fire. In Fires and Human Behavior, (ed). Canter D (ed). Wiley, New York, USA, pp 47–62.

Kavoussi R, Armstead P, and Coccaro E (1997) The neurobiology of impulsive aggression. Psychiatric Clinics of North America 20, 395–403.

Kessler RC, Coccaro EF, Fava M, et al. (2006) The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. Archives of General Psychiatry 63(6), 669–678.

Keuthen NJ, Fraim C, Deckersbach T, et al. (2001) Longitudinal follow-up of naturalistic treatment outcome in patients with trichotillomania. Journal of Clinical Psychiatry 62, 101–107.

Keuthen NJ, Franklin ME, Bohne A, et al. (2002) Functional impairment associated with trichotillomania and implications for treatment development. Paper presented at the Trichotillomania: Psychopathology and Treatment Development Symposium, conducted at the 36th annual meeting of the Association for the Advancement of Behavior Therapy, Reno, NV, USA.

Keuthen NJ, Savage CR, O'Sullivan RL, et al. (1996) Neuropsychological functioning in trichotillomania. Biological Psychiatry 39, 747–749.

Keuthen NJ, O'Sullivan RL, Goodchild P, et al. (1998) Retrospective review of treatment outcome for 63 patients with trichotillomania. American Journal of Psychiatry 155, 560–561.

Keuthen NJ, O'Sullivan RL, Hayday CF, et al. (1997) The relationship of menstrual cycle and pregnancy to compulsive hair-pulling. Psychotherapy and Psychosomatics 66, 33–37.

Keutzer CS (1972) Kleptomania: A direct approach to treatment. British Journal of Medical Psychology 45, 159–163.

Khan K and Martin IC (1977) Kleptomania as a presenting feature of cortical atrophy. Acta Psychiatrica Scandinavica 56, 168–172.

Khantzian EJ and Mack JE (1983) Self-preservation and the care of the self. Ego instincts reconsidered. Psychoanalytic Study of the Child 38, 209–232.

Kim SW and Grant JE (2001) The psychopharmacology of pathological gambling. Seminars in Clinical Neuropsychiatry 6, 184–194.

Kim SW, Grant JE, Adson DE, et al. (2002) A double-blind placebo-controlled study of the efficacy and safety of paroxetine in the treatment of pathological gambling. Journal of Clinical Psychiatry 63, 501–507.

Kim SW, Grant JE, Adson DE, et al. (2001) Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. Biological Psychiatry 49, 914–921.

Kindler S, Dannon PN, Iancu I, et al. (1997) Emergence of kleptomania during treatment for depression with serotonin selective reuptake inhibitors. Clinical Neuropharmacology 20, 126–129.

King RA, Zohar AH, Ratzoni G, et al. (1995a) An epidemiological study of trichotillomania in Israeli adolescents. Journal of Child and Adolescent Psychopharmacologyy 34, 1212–1215.

King RA, Scahill L, Vitulano LA, et al. (1995b) Childhood trichotillomania: Clinical phenomenology, comorbidity, and family genetics. Journal of Child and Adolescent Psychopharmacology 34, 1451–1459.

Kmetz GF, McElroy SL, and Collins DJ (1997) Response of kleptomania and mixed mania to valproate. American Journal of Psychiatry 154, 580–581.

Kohen DP (1996) Hypnotherapeutic management of pediatric and adolescent trichotillomania. Journal of Developmental and Behavioral Pediatrics 17, 328–334.

Koles MR and Jenson WR (1985) Comprehensive treatment of chronic fire setting in a severely disordered boy. Journal of Behavior Therapy and Experimental Psychiatry 16, 81–85.

Kolko DJ (2001) Efficacy of cognitive-behavioral treatment and fire safety education for children who set fires: Initial and follow-up outcomes. Journal of Child Psychology and Psychiatry 42, 359–369.

Kolko DJ and Kazdin AE (1988) Parent–child correspondence in identification of firesetting among child psychiatric patients. Journal of Child Psychology and Psychiatry 29, 175–184.

Kolko D and Kazdin AE (1989) Assessment of dimensions of childhood fire-setting among patients and nonpatients: the fire-setting risk interview. Journal of Abnormal Child Psychology 17, 157–176.

Koson DF and Dvoskin J (1982) Arson: a diagnostic study. Bulletin of the American Academy of Psychiatry and the Law 10, 39–49.

Kraus JE (1999) Treatment of kleptomania with paroxetine. Journal of Clinical Psychiatry 60, 793.

Kriegsfeld LJ, Dawson TM, Dawson VL, et al. (1997) Aggressive behavior in male mice lacking the gene for neuronal nitric oxide synthase requires testosterone. Brain Research 769, 66–70.

Kuperman S, Kramer J, and Loney J (1988) Enzyme activity and behavior in hyperactive children grown up. Biological Psychiatry 24, 375–383.

Ladouceur R (1990) Cognitive activities among gamblers. Paper presented at the Association for Advancement of Behavior Therapy (AABT) Convention, San Francisco, CA, USA.

Lappalainen J, Long JC, Eggert M, et al. (1998) Linkage of antisocial alcoholism to the serotonin 5-HT1B receptor gene in 2 populations. Archives of General Psychiatry 55, 989–994.

Laubichler W, Kuhberger A, and Sedlmeier P (1996) "Pyromania" and arson. A psychiatric and criminological data analysis [in German]. Nervenarzt 67, 774–780.

Lee S (1994) The heterogeneity of stealing behaviors in Chinese patients with anorexia nervosa in Hong Kong. The Journal of Nervous and Mental Disease 182, 304–307.

Lejoyeux M, Arbaretaz M, McLoughlin M, et al. (2002) Impulse control disorders and depression. The Journal of Nervous and Mental Disease 190, 310–314.

Lejoyeux M, Feuché N, Loi S, et al. (1999) Study of impulse control disorders among alcohol-dependent patients. Journal of Clinical Psychiatry 40, 302–305.

Lenane MC, Swedo SE, Rapoport JL, et al. (1992) Rates of obsessive-compulsive disorder in first degree relatives of patients with trichotillomania: A research note. Journal of Child Psychology and Psychiatry 33, 925–933.

Lepkifker E, Dannon PN, Ziv R, et al. (1999) The treatment of kleptomania with serotonin reuptake inhibitors. Clinical Neuropharmacology 22, 40–43.

Lerner J, Franklin ME, Meadows EA, et al. (1998) Effectiveness of a cognitive-behavioral treatment program for trichotillomania: An uncontrolled evaluation. Behavior Therapy 29, 157–171.

Lesieur HR (1988) The female pathological gambler, in Gambling Studies: Proceedings of the 7th International Conference on Gambling and Risk-Taking. Edited by Eadington WR. Reno, NV, University of Nevada, pp 230–258.

Lesieur HR and Blume SB (1987) The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers. American Journal of Psychiatryy 144, 1184–1188.

Lesieur HR and Blume S (1993) Revising the South Oaks Gambling Screen in different settings. Journal of Gambling Studies 9, 213–223.

Lesieur HR and Rosenthal RJ (1991) Pathological gambling: A review of the literature. Journal of Gambling Studies 7, 5–39.

Lesieur H, Blume S, and Zoppa R (1986) Alcoholism, drug abuse, and gambling. Alcoholism: Clinical and Experimental Research 10, 33–38.

Lewis NDC and Yarnell H (1951) Pathological Firesetting (Pyromania): Nervous and Mental Disease Monograph No 82. Coolidge Foundation, New York, USA.

Linden RD, Pope HG Jr, and Jonas JM (1986) Pathological gambling and major affective disorder: Preliminary findings. Journal of Clinical Psychiatry 47, 201–203.

Linehan MM, Tutek DA, Heard HL, et al. (1994) Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. American Journal of Psychiatry 151, 1771–1776.

Linnoila M, De Jong J, and Virkkunen M (1989) Family history of alcoholism in violent offenders and impulsive fire setters. Archives of General Psychiatry 46, 613–616.

Linnoila M, Virkkunen M, Scheinin M, et al. (1983) Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. Life Science 33, 2609–2614.

Lion JR (1992) The intermittent explosive disorder. Psychiatric Annals 2, 64–66.

Lochner C, Simeon D, Niehaus DJ, et al. (2002) Trichotillomania and skin-picking: a phenomenological comparison. Depress Anxiety 15(2), 83–86.

Lochner C, Seedat S, Niehaus DJ, et al. (2006) Topiramate in the treatment of trichotillomania: an open-label pilot study. International Clinical Psychopharmacology 21(5), 255–259.

Lorenz V (1981) Differences found among Catholic, Protestant, and Jewish families of pathological gamblers. In Fifth National Conference on Gambling and Risk Taking, Lake Tahoe, CA, USA.

Lowenstein LF (1989) The etiology, diagnosis and treatment of the fire-setting behaviour of children. Child Psychiatry and Human Development 19, 186–194.

Lyons WE, Mamounas LA, and Ricaurte GA (1991) Brain-derived neurotrophic factor-deficient mice develop aggressiveness and hyperphagia in conjunction with brain serotonergic abnormalities. Proceedings of the National Academy of Sciences USA. 96, 15239–15244.

Maccallum F and Blaszczynski A (2002) Pathological gambling and comorbid substance use. Australian and New Zealand Journal of Psychiatry 36, 411–415.

Mann JJ, McBride PA, Brown RP, et al. (1992) Relationship between central and peripheral serotonin indexes in depressed and suicidal psychiatric inpatients. Archives of General Psychiatry 49, 442–446.

Manuck SB, Flory JD, Ferrell RE, et al. (2000) A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. Psychiatry Research 95, 9–23.

Manuck SB, Flory JD, Ferrell RE, et al. (1999) Aggression and anger-related traits associated with a polymorphism of the tryptophan hydroxylase gene. Biological Psychiatry 45, 603–614.

Manuck SB, Flory JD, McCaffery JM, et al. (1998) Aggression, impulsivity, and central nervous system serotonergic responsivity in a nonpatient sample. Neuropsychopharmacology 19, 287–299.

Marazziti D, Presta S, Pfanner C, et al. (2000) The biological basis of kleptomania and compulsive buying. Paper presented at the American College of Neuropsychopharmacology 39th Annual Meeting, San Juan, Puerto Rico.

Marks I (1990) Behavioural (non-chemical) addictions. British Journal of Addiction 85, 1389–1394.

Marzagao LR (1972) Systemic desensitization treatment of kleptomania. Journal of Behavior Therapy and Experimental Psychiatry 3, 327–328.

Mattes JA (1990) Comparative effectiveness of carbamazepine and propranolol for rage outbursts. Journal of Neuropsychiatry and Clinical Neuroscience 2, 159–164.

Mattes JA and Fink M (1987) A family study of patients with temper outbursts. Journal of Psychiatric Research 21, 249–255.

Mavromatis M and Lion JR (1977) A primer on pyromania. Disease of Nervous System 38, 954–955.

McConaghy N and Blaszczynski A (1988) Imaginal desensitization: A cost-effective treatment in two shoplifters and a binge-eater resistant to previous therapy. Australian and New Zealand Journal of Psychiatry 22, 78–82.

McCormick RA, Russo AM, Ramirez LF, et al. (1984) Affective disorders among pathological gamblers seeking treatment. American Journal of Psychiatryy 141, 215–218.

McElroy SL (1999) Recognition and treatment of DSM-IV intermittent explosive disorder. Journal of Clinical Psychiatry 60, 12–16.

McElroy SL, Keck PE Jr, Pope HG Jr, et al. (1989) Pharmacological treatment of kleptomania and bulimia nervosa. Journal of Clinical Psychopharmacology 9, 358–360.

McElroy SL, Hudson JI, Phillips KA, et al. (1993) Clinical and theoretical implications of a possible link between obsessive-compuslive and impulse control disorders. Depression 1, 121–132.

McElroy SL, Hudson JI, Pope HG Jr, et al. (1992) The DSM-III-R impulse control disorders not elsewhere classified: Clinical characteristics and relationship to other psychiatric disorders. American Journal of Psychiatry 149, 318–327.

McElroy SL, Hudson JI, Pope HG, et al. (1991a) Kleptomania: Clinical characteristics and associated psychopathology. Psychology Medicine 21, 93–108.

McElroy SL, Soutullo CA, Beckman DA, et al. (1998) DSM-IV intermittent explosive disorder: A report of 27 cases. Journal of Clinical Psychiatry 59, 203–210; quiz 211.

McElroy SL, Pope HG Jr, Hudson JI, et al. (1991b) Kleptomania: A report of 20 cases. American Journal of Psychiatry 148, 652–657.

McGrath P and Marshall PG (1979) A comprehensive treatment program for a fire-setting child. Journal of Behavior Therapy and Experimental Psychiatry 10, 69–72.

McGuire RJ, Carlisle JM, and Young BG (1965) Sexual deviations as conditioned behaviour: A hypothesis. Behavior Research and Therapy 2, 185–190.

McIntyre AW and Emsley RA (1990) Shoplifting associated with normal-pressure hydrocephalus: Report of a case. Journal of Geriatric Psychiatry and Neurology 3, 229–230.

Mehregan AH (1970) Trichotillomania. A clinicopathologic study. Archives of Dermatology 102, 129–133.

Mendez MF (1988) Pathological stealing in dementia. Journal of Geriatric Psychiatry and Neurology 36, 825–826.

Monopolis S and Lion JR (1983) Problems in the diagnosis of intermittent explosive disorder. American Journal of Psychiatry 140, 1200–1202.

Moody G (1990) Quit Compulsive Gambling. Thorsons, London.

Moore DP and Jefferson JW (2004) Handbook of Medical Psychiatry, 2nd edition. Mosby, Inc, St. Louis, MO, USA.

Moreno I, Saiz-Ruiz J, and Lopez-Ibor JJ (1991) Serotonin and gambling dependence. Human Psychopharmacology: Clinical and Experimental 6, 9–12.

Mouton SG and Stanley MA (1996) Habit reversal training for trichotillomania: A group approach. Cognitive and Behavioral Practice 3, 159–182.

Muller SA (1990) Trichotillomania: A histopathologic study in sixty-six patients. Journal of American Academy of Dermatology 23, 56–62.

Murray JB (1993) Review of research on pathological gambling. Psychology Report 72, 791–810.

New AS, Hazlett EA, and Buchsbaum MS (2002) Blunted prefrontal cortical $^{18}$fluorodeoxyglucose positron emission tomography response to meta-chlorophenylpiperazine in impulsive aggression. Archives of General Psychiatry 59, 621–629.

Ninan PT, Knight B, Kirk L, et al. (1998) A controlled trial of venlafaxine in trichotillomania: Interim phase I results. Psychopharmacology Bulletin 34, 221–224.

Ninan PT, Rothbaum BO, Marsteller FA, et al. (2000) A placebo-controlled trial of cognitive-behavioral therapy and clomipramine in trichotillomania. Journal of Clinical Psychiatry 61, 47–50.

Ninan PT, Rothbaum BO, Stipetic M, et al. (1992) CSF 5-HIAA as a predictor of treatment response in trichotillomania. Psychopharmacology Bulletin 28, 451–455.

Noblett S and Nelson B (2001) A psychosocial approach to arson: A case controlled study of female offenders. Medicine, Science and the Law 41, 325–330.

O'Sullivan GH and Kelleher MJ (1987) A study of fire-setters in the southwest of Ireland. British Journal of Psychiatry 151, 818–823.

O'Sullivan RL, Mansueto CS, Lerner EA, et al. (2000) Characterization of trichotillomania. A phenomenological model with clinical relevance to obsessive-compulsive spectrum disorders. Psychiatric Clinics of North America 23(3), 587–604.

O'Sullivan RL, Rauch SL, Breiter HC, et al. (1997) Reduced basal ganglia volumes in trichotillomania measured via morphometric magnetic resonance imaging. Biological Psychiatry 42, 39–45.

Pallanti S, Quercioli L, Sood E, et al. (2002a) Lithium and valproate treatment of pathological gambling: a randomized single-blind study. Journal of Clinical Psychiatry 63, 559–564.

Pallanti S, Baldini-Rossi N, Sood E, et al. (2002b) Nefazodone treatment of pathological gambling: a prospective open-label controlled trial. Journal of Clinical Psychiatry 63, 1034–1039.

Parsey RV, Oquendo MA, Simpson NR, et al. (2002) Effects of sex, age, and aggressive traits in man on brain serotonin 5-HT$_{1A}$ receptor binding potential measured by PET using $_{[C-11]}$WAY-100635. Brain Research 954, 173–182.

Perez de Castro I, Ibanez A, Saiz-Ruiz J, et al. (2002) Concurrent positive association between pathological gambling and functional DNA polymorphisms at the MAO-A and the 5-HT transporter genes. Molecular Psychiatry 7, 927–928.

Petry NM (2001a) Pathological gamblers, with and without substance use disorders, discount delayed rewards at high rates. Journal of Abnormal Child Psychology 110, 482–487.

Petry NM (2001b) Substance abuse, pathological gambling, and impulsiveness. Drug and Alcohol Dependence 63, 29–38.

Petry NM (2002) Psychosocial treatments for pathologiocal gambling: Current status and future directions. Psychiatric Annals 32, 192–196.

Petry NM and Armentano C (1999) Prevalence, assessment, and treatment of pathological gambling: a review. Psychiatric Services 50, 1021–1027.

Petry NM and Casarella T (1999) Excessive discounting of delayed rewards in substance abusers with gambling problems. Drug and Alcohol Dependence 56, 25–32.

Phillips DP, Welty WR, and Smith MM (1997) Elevated suicide levels associated with legalized gambling. Suicide and Life-Threatening Behavior 27, 373–378.

Pollard CA, Ibe IO, Krojanker DN, et al. (1991) Clomipramine treatment of trichotillomania: A follow-up report on four cases. Journal of Clinical Psychiatry 52, 128–130.

Post RM, Pickar D, Ballenger JC, et al. (1984) Endogenous opiates in cerebrospinal fluid: relationship to mood and anxiety, in Neurobiology of Mood Disorders. (eds) Post RM and Ballenger JC. Williams & Wilkins Baltimore, MD, USA, , pp 356–368.

Potenza MN (2001) The neurobiology of pathological gambling. Seminars in Clinical Neuropsychiatry 6, 217–226.

Potenza MN and Chambers RA (2001) Schizophrenia and pathological gambling. American Journal of Psychiatry 158, 497–498.

Potenza MN and Hollander E (2002) Pathological gambling and impulse control disorders. In Neuropsychopharmacology: The $5^{th}$ Generation of Progress, Coyle JT, Nemeroff C, Charney D, et al. (eds). Lippincott Williams & Wilkins, Baltimore, MD, USA, pp 1725–1741.

Potenza MN, Steinberg MA, Skudlarski P, et al. (2003) Gambling urges in pathological gambling: a functional magnetic resonance imaging study. Archives of General Psychiatry 60, 828–836.

Potenza MN, Wasylink S, Epperson CN, et al. (1998) Olanzapine augmentation of fluoxetine in the treatment of trichotillomania. American Journal of Psychiatry 155, 1299–3000.

Presta S, Marazziti D, Dell'Osso L, et al. (2002) Kleptomania: clinical features and comorbidity in an Italian sample. Comprehensive Psychiatry 43, 7–12.

Prins H, Tennent G, and Trick K (1985) Motives for arson (fire raising). Medicine, Science and the Law 25, 275–278.

Puri BK, Baxter R, and Cordess CC (1995) Characteristics of fire-setters. A study and proposed multiaxial psychiatric classification. British Journal of Psychiatry 166, 393–396.

Raine A, Buchsbaum MS, Stanley J, et al. (1994) Selective reductions in prefrontal glucose metabolism in murderers. Biological Psychiatry 36, 365–373.

Ramirez LF, McCormick RA, Russo AM, et al. (1983) Patterns of substance abuse in pathological gamblers undergoing treatment. Addictive Behaviors 8, 425–428.

Rapp JT, Miltenberger RG, Long ES, et al. (1998) Simplified habit reversal treatment for chronic hair-pulling in three adolescents: A clinical replication with direct observation. Journal of Applied Behavior Analysis 31, 299–302.

Reeve EA, Bernstein GA, and Christenson GA (1992) Clinical characteristics and psychiatric comorbidity in children with trichotillomania. Journal of Child and Adolescent Psychopharmacology 31, 132–138.

Repo E, Virkkunen M, Rawlings R, et al. (1997) Criminal and psychiatric histories of Finnish arsonists. Acta Psychiatrica Scandinavica 95, 318–323.

Rettew DC, Cheslow DL, Rapoport JL, et al. (1991) Neuropsychological test performance in trichotillomania: A further link with obsessive-compulsive disorder. Journal of Anxiety Disorders 5, 225–235.

Ritchie EC and Huff TG (1999) Psychiatric aspects of arsonists. Journal of Forensic Science 44, 733–740.

Robins LN and Regier DA (1992) Psychiatric Disorders in America. Free Press, New York.

Rosenbaum MS and Ayllon T (1981) The habit-reversal technique in treating trichotillomania. Behavior Therapy 12, 473–481.

Rosenthal RJ (1992) Pathological gambling. Psychiatric Annals 22, 72–78.

Rosenthal R and Rugle L (1994) A psychodynamic approach to the treatment of pathological gambling, part I: Achieving abstinence. Journal of Gambling Studies 10, 21–42.

Rothbaum BO, Shaw L, Morris R, et al. (1993) Prevalence of trichotillomania in a college freshman population. Journal of Clinical Psychiatry 54, 72–73.

Rowen R (1981) Hypnotic age regression in the treatment of a self-destructive habit: Trichotillomania. American Journal of Clinical Hypnosis 23, 195–197.

Roy A, DeJong J, and Linnoila M (1989) Extroversion in pathological gamblers: Correlates with indexes of noradrenergic function. Archives of General Psychiatry 46, 679–681.

Roy A, Custer R, Lorenz V, et al. (1988a) Depressed pathological gamblers. Acta Psychiatrica Scandinavica 77, 163–165.

Roy A, Adinoff B, Roehrich L, et al. (1988b) Pathological gambling. A psychobiological study. Archives of General Psychiatry 45, 369–373.

RŠsŠnen P, Hirvenoja R, Hakko H, et al. (1995) A portrait of a juvenile arsonist. Forensic Science International 73, 41–47.

Rugle L and Melamed L (1993) Neuropsychological assessment of attention problems in pathological gamblers. The Journal of Nervous and Mental Disease 181, 107–112.

Russo AM, Taber JI, McCormick RA, et al. (1984) An outcome study of an inpatient treatment program for pathological gambling. Hospital & Community Psychiatry 35, 823–827.

Schlosser S, Black DW, Blum N, et al. (1994) The demography, phenomenology, and family history of 22 persons with compulsive hair pulling. Annals of Clinical Psychiatry 6, 147–152.

Schwartz HJ (1992) Psychoanalytic psychotherapy for a woman with diagnoses of kleptomania and bulimia. Hospital & Comm Psychiatr 43, 109–110.

Seroczynski AD, Bergeman CS, and Coccaro EF (1999) Etiology of the impulsivity/aggression relationship: Genes or environment? Psychiatry Research 86, 41–57.

Shaffer HJ and Hall MN (1996) Estimating the prevalence of adolescent gambling disorders: A quantitative synthesis and guide toward standard gambling nomenclature. Journal of Gambling Studies 12, 193–214.

Shaffer HJ, Hall MN, and Vanderbilt J (1999) Estimating the prevalence of disordered gambling behavior in the United States and Canada: a research synthesis. American Journal of Public Health 89, 1369–1376.

Siever LJ, Buchsbaum MS, New AS, et al. (1999) D,L-Fenfluramine response in impulsive personality disorder assessed with [18F]fluorodeoxyglucose positron emission tomography. Neuropsychopharmacology 20, 413–423.

Siever LJ, Kahn RS, Lawlor BA, et al. (1991) Critical issues in defining the role of serotonin in psychiatric disorders. Pharmacology Review 43, 509–525.

Simeon D, Cohen LJ, Stein DJ, et al. (1997) Comorbid self-injurious behaviors in 71 female hair-pullers: A survey study. The Journal of Nervous and Mental Disease 185, 117–119.

Slutske WS, Eisen S, True WR, et al. (2000) Common genetic vulnerability for pathological gambling and alcohol dependence in men. Archives of General Psychiatry 57, 666–673.

Specker SM, Carlson GA, Christenson GA, et al. (1995) Impulse control disorders and attention deficit disorder in pathological gamblers. Annals of Clinical Psychiatry 7, 175–179.

Staner L and Mendlewicz J (1998) Heredity and role of serotonin in aggressive impulsive behavior. Encephale 24, 355–364.

Stanley MA, Breckenridge JK, Swann AC, et al. (1997b) Fluvoxamine treatment of trichotillomania. Journal of Clinical Psychopharmacology 17, 278–283.

Stanley MA, Hannay HJ, and Breckenridge JK (1997a) The neuropsychology of trichotillomania. Journal of Anxiety Disorders 11, 473–488.

Stein DJ and Hollander E (1992) Low-dose pimozide augmentation of serotonin reuptake blockers in the treatment of trichotillomania. Journal of Clinical Psychiatry 53, 123–126.

Stein DJ, Bouwer C, and Maud CM (1997) Use of the selective serotonin reuptake inhibitor citalopram in treatment of trichotillomania. European Archives of Psychiatry and Clinical Neuroscience 247, 234–236.

Stein DJ, Hollander E, and Liebowitz MR (1993) Neurobiology of impulsivity and the impulse control disorders. Journal of Neuropsychiatry and Clinical Neuroscience 5, 9–17.

Stewart R and Brown An outcome study of Gamblers Anonymous. British Journal of Psychiatry 152, 284–288.

Stewart RS and Nejtek VA (2003) An open-label, flexible-dose study of olanzapine in the treatment of trichotillomania. Journal of Clinical Psychiatry 64, 49–52.

Streichenwein SM and Thornby JI (1995) A long-term, double-blind, placebo-controlled crossover trial of the efficacy of fluoxetine for trichotillomania. American Journal of Psychiatry 152, 1192–1196.

Swedo SE, Lenane MC, and Leonard HL (1993) Long-term treatment of trichotillomania (hair pulling) (letter to the editor). New England Journal of Medicine 329, 141–142.

Swedo SE and Leonard HL (1992) Trichotillomania: an obsessive-compulsive spectrum disorder? Psychiatric Clinics of North America 15, 777–790.

Swedo SE, Leonard HL, Rapoport JL, et al. (1989) A double-blind comparison of clomipramine and desipramine in the treatment of trichotillomania (hair-pulling). New England Journal of Medicine 321, 497–501.

Swedo SE, Rapoport JL, Leonard HL, et al. (1991) Regional cerebral glucose metabolism of women with trichotillomania. Archives of General Psychiatry 48, 828–833.

Tabatabai SE and Salari-Lak M (1981) Alopecia in dolls! Cutis 28, 206.

Taber JI, McCormick RA, Russo AM, et al. (1987) Follow-up of pathological gamblers after treatment. American Journal of Psychiatryy 144, 757–761.

Taber JI (1981) Group psychotherapy with pathological gamblers. Paper presented at the 5th National Conference on Gambling and Risk-Taking. South Lake Tahoe, NV, USA.

Tolin D, Franklin ME, Diefenbach G, et al. (2002) CBT for pediatric trichotillomania: An open trial. Paper presented at the Trichotillomania: Psychopathology and Treatment Development Symposium, conducted at the 36th annual meeting of the Association for the Advancement of Behavior Therapy, Reno, NV, USA.

Tollefson GD (1991) Anxiety and alcoholism: A serotonin link. British Journal of Psychiatry y 159, 34–39.

Turnbull JM (1987) Sexual relationships of patients with kleptomania. Southern Medical Journal 80, 995–998.

Van Ameringen M, Mancini C, Oakman JM, et al. (1999) The potential role of haloperidol in the treatment of trichotillomania. Journal of Affective Disorders 56, 219–226.

van Minnen A, Hoogduin KA, Keijsers GP, et al. (2003) Treatment of trichotillomania with behavioral therapy or fluoxetine. Archives of General Psychiatry 60, 517–522.

Virkkunen M and Linnoila M (1993) Brain serotonin, type II alcoholism and impulsive violence. Journal of Studies on Alcohol (Suppl 11), 163–169.

Virkkunen M, Eggert M, Rawlings R, et al. (1996) A prospective follow-up study of alcoholic violent offenders and fire setters. Archives of General Psychiatry 53, 523–529.

Virkkunen M, Goldman D, Nielsen DA, et al. (1995) Low brain serotonin turnover rate (low CSF 5-HIAA) and impulsive violence. Journal of Psychiatry and Neuroscience 20, 271–275.

Virkkunen M, Nuutila A, Goodwin FK (1987) Cerebrospinal fluid monoamin metabolite levels in male arsonists. Archives of General Psychiatry 44, 241–247.

Virkkunen M, Rawlings R, Tokola R, et al. (1994) CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholic, violent offenders, fire setters, and healthy volunteers. Archives of General Psychiatry 51, 20–27.

Vitulano LA, King RA, Scahill L, et al. (1992) Behavioral treatment of children and adolescents with trichotillomania. Journal of Child and Adolescent Psychopharmacology 31, 139–146.

Volberg RA (2002) The epidemiology of pathological gambling. Psychiatric Annals 32, 171–178.

Volberg RA and Steadman HJ (1988) Refining prevalence estimates of pathological gambling. American Journal of Psychiatry 145, 502–505.

Watson TS and Allen KD (1993) Elimination of thumb-sucking as a treatment for severe trichotillomania. Journal of Child and Adolescent Psychopharmacology 32, 830–834.

Welte J, Barnes G, Wieczorek W, et al. (2001) Alcohol and gambling pathology among US adults: Prevalence, demographic patterns and comorbidity. Journal of Studies on Alcohol 62, 706–712.

Wetzel R (1966) Use of behavior techniques in a case of compulsive stealing. Journal of Consulting Psychology 5, 367–374.

Wetzler S, Kahn RS, Asnis GM, et al. (1991) Serotonin receptor sensitivity and aggression. Psychiatry Research 37, 271–279.

Wilhelm F and Margraf J (1993) Nail-biting: Description, etiological models and treatment. Verhaltenstherapie 3, 176–196.

Wilhelm S, Keuthen NJ, Deckersbach T, et al. (1999) Self-injurious skin picking: Clinical characteristics and comorbidity. Journal of Clinical Psychiatry 60(7), 454–459.

Winchel RM (1992) Trichotillomania: Presentation and treatment. Psychiatric Annals 22, 8–89.

Winchel RM, Jones JS, Stanley B, et al. (1992) Clinical characteristics of trichotillomania and its response to fluoxetine. Journal of Clinical Psychiatry 53, 304–308.

Winters KC and Rich T (1999) A twin study of adult gambling behavior. Journal of Gambling Studies 14, 213–215.

Wise MG and Tierney JG (1999) Impulse control disorders not elsewhere classified. In The American Psychiatric Press Textbook of Psychiatry, 3rd edition, Hales RE, Yudofsky SC, Talbott JA, et al. (eds). American Psychiatric Press, Washington, DC, USA, pp 773–794.

Woermann FG, van Elst LT, Koepp MJ, et al. (2000) Reduction of frontal neocortical grey matter associated with affective aggression in patients with temporal lobe epilepsy: An objective voxel by voxel analysis of automatically segmented MRI. Journal of Neurology, Neurosurgy & Psychiatry 68, 162–169.

Wood A and Garralda ME (1990) Kleptomania in a 13-year-old boy. A sequel of a "lethargic" encephalitic/depressive process? British Journal of Psychiatry 157, 770–772.

Zalsman G, Hermesh H, and Sever J (2001) Hypnotherapy in adolescents with trichotillomania: Three cases. American Journal of Clinical Hypnosis 44, 63–68.

Zimmerman M, Mattia J, Younken S, et al. (1998) The prevalence of DSM-IV impulse control disorders in psychiatric outpatients (NR265), in 1998 New Research Program and Abstracts, American Psychiatric Association 151st Annual Meeting, Toronto, Ontario.

Zimmerman M, Breen RB, and Posternak MA (2002) An open-label study of citalopram in the treatment of pathological gambling. Journal of Clinical Psychiatry 63, 44–48.

**Abstract:** The impulse-control disorders (intermittent explosive disorder, kleptomania, pyromania, pathological gambling, and trichotillomania) share the feature of the irresistible urge to act in a given way. Effort to resist may be associated with rising tension, giving rise to further need to act on the given impulse. This chapter reviews clinical features, epidemiology, and clinical interventions. Data suggesting perturbations of central serotonin function and other neurotransmitter systems are reviewed.