

Disruptive, Impulse-Control, and Conduct Disorders: Intermittent Explosive Disorder, Kleptomania, and Pyromania

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DSM-5 Diagnoses

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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, in the DSM-5 (American Psychiatric Association, 2013) impulsive behavior is recognized as an important feature in numerous psychiatric diagnoses. Those not classified and described elsewhere are grouped together in the chapter disruptive, impulse control, and conduct disorders. Included in this chapter are intermittent explosive disorder (IED) (failure to resist aggressive impulses), kleptomania (failure to resist urges to steal items), and pyromania (failure to resist urges to set fires). It was noted, as is still the case in DSM-5, that behaviors characteristic of these disorders may be present 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.

DSM-IV-TR classified a number of other, related, disorders, not appearing in distinct categories, as ICDs not otherwise specified (American Psychiatric Association, 2000). These included 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.

In DSM-5, published in 2013, three disorders (IED, pyromania, and kleptomania) of those previously classified as ICDs remained as such, and were incorporated into a new chapter of Disruptive, Impulse Control, and Conduct Disorders. This chapter brought together disorders previously included in DSM-IV-TR's disorders usually first diagnosed in infancy, childhood, and adolescence (oppositional defiant disorder, conduct disorder, and disruptive behavior disorder not otherwise specified, now categorized as other specified and unspecified disruptive impulse control, and conduct disorders) and IED, pyromania, and kleptomania from the ICD chapter. All of these disorders, now classified together, are characterized by problems in emotional and behavioral control and regulation. Trichotillomania (hair-pulling disorder has been added parenthetically), previously classified as an ICD in DSM-IV-TR, and excoriation (skin-picking disorder), which is newly added though it had been had been mentioned in DSM-IV-TR as an ICD not otherwise specified, have both been incorporated into the DSM-5 chapter on Obsessive–Compulsive and Related Disorders. Pathological gambling (now gambling disorder), also having been classified as an ICD in DSM-IV-TR, is now categorized in the Non-Substance-Related Disorders section of DSM-5's chapter on Substance-Related and Addictive Disorders.

To conceptualize ICDs properly, 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 such as borderline personality disorder (BPD) and antisocial personality disorder (ASPD), neurological disorders characterized by disinhibited behavior, attention-deficit/hyperactivity disorder (ADHD), substance and alcohol use disorders, conduct disorder, binge-eating, bulimia, and paraphilias. Impulsivity research has been conducted both in disorders characterized by impulsivity, such as BPD, ASPD, and conduct disorder, and in traditional ICDs such as 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 posttraumatic stress disorder, making screening for comorbid conditions that contribute to impulsivity, including these, in addition to bipolar spectrum disorders and ADHD, especially important. A decision tree for the differential diagnosis of impulsive behaviors is presented in Figure 70–1.

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) and is a common clinical problem and also a core feature of human behavior. An impulse is rash and lacks deliberation. It may be sudden and ephemeral, or be characterized by a steady rise in tension that may reach a climax in an explosive expression of the impulse, resulting in careless actions without regard to the consequences to self or others. Impulsivity is evidenced behaviorally as an underestimated sense of harm, carelessness, extroversion, and impatience, and includes 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.

Research findings associate various forms of impulsive behavior, including impulsive suicidal behavior, impulsive aggression, and impulsive fire setting, with biological markers of altered serotonergic function (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 impulsive behaviors may be present in the impulse disorders (such as in IED and kleptomania), the primary connotation of the word impulsivity, as used to describe these conditions, is the irresistibility of the urge to act.

In DSM-5, ICDs are characterized by five stages of symptomatic behavior (Table 70–1). 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 such as trichotillomania, pyromania, and pathological gambling, the individual may spend considerable amounts of time fighting off the urge, trying not to carry out the impulse. It is the inability to resist the impulse that is the common core of these disorders, rather than the rapid transduction of thought to action.

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, including 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.

Table 70–1 Five Stages of Symptomatic Behavior in ICDs

1. Increased sense of tension or arousal
2. A failure to resist the urge to act
3. Heightened sense of arousal
4. Sense of relief from the urge
5. Guilt and remorse at having committed the act

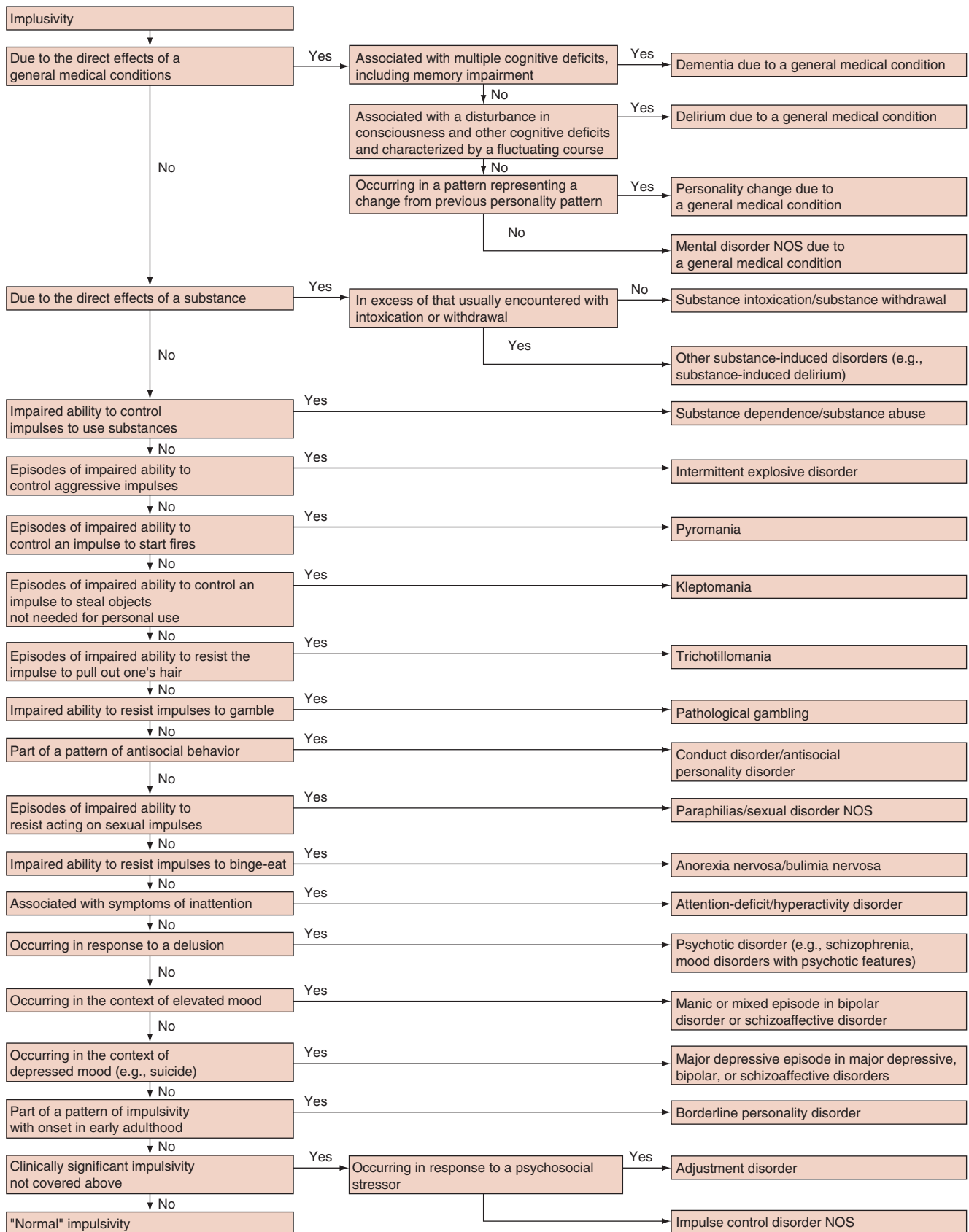


Figure 70–1 Differential diagnosis of impulsivity. Impulsivity is a tendency to act in a sudden, unpremeditated, and excessively spontaneous fashion. Other decision trees that should be considered are those for aggressive behavior, catatonia, delusions, depressed mood, euphoric or irritable mood, disorganized or unusual behavior, distractibility, eating behavior, self-mutilation, and suicide ideation or attempt. NOS, not otherwise specified.

Although impulse control disorders historically had been considered uncommon, later investigations suggest that some of them may occur more frequently than once assumed. Trichotillomania (although not categorized as an ICD in DSM-5), for example, was once considered rare. However, surveys indicate that the lifetime prevalence of the condition may exceed 1% 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 aimed at these specific conditions have been conducted. Attempts at treatment are usually formulated by extrapolation from protocols 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 twentieth century are largely limited to reports of the psychoanalytic treatment of individual cases or of small series. The aggressive quality of kleptomania, pyromania, and IED presents a tempting substrate 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 thymoleptics, anxiolytics, and, more recently, atypical neuroleptic medications. Although the concept of impulsivity is still in ferment, attempts at further refining it are reflected in a perspective offered by Van Ameringen et al. (1999).

Whereas IED has been the focus of increasing interest of late, 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

Intermittent Explosive Disorder (IED) is a DSM diagnosis used to describe patients with pathological and recurrent

impulsive aggression. Impulsive aggression, however, is a key feature of several psychiatric disorders and nonpsychiatric conditions, and is not specific to IED. The diagnosis of IED as formulated in the DSM-5 is therefore essentially a diagnosis of exclusion, and can be made only after other mental disorders and medical conditions that might account for recurrent aggressive outbursts have been ruled out. This is described by Criterion F of the DSM-5, which, in addition to containing a brief list of other conditions to be considered, also specifies that aggressive behavior occurring as part of an adjustment disorder in children (aged 6–18 years) should not be considered for the diagnosis of IED. With regard to the aggressive episodes, the individual may describe them 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 with 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 (Table 70–1). 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. Although not explicitly stated in the DSM-5 definition of IED, impulsive aggressive behavior may be driven by 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.

The primary change seen in DSM-5 with regard to IED is the type of aggressive outbursts that should be considered. Whereas only aggressive outbursts resulting in serious assaultive or destruction of property were considered in DSM-IV-TR, verbal aggression (e.g., temper tantrums, tirades, verbal arguments, or fights) and nondestructive/noninjurious physical aggression also meet criteria

Table 70–2 Differential Diagnosis of Intermittent Explosive Disorder

- Unipolar major depressive disorder
- Bipolar disorder
- Schizoaffective disorder
- Psychotic disorder
- Schizophrenia
- Substance intoxication and/or withdrawal
- Childhood-onset disorders
 - Attention-deficit/hyperactivity disorder
 - Conduct disorder
 - Oppositional defiant disorder
 - Autistic spectrum disorder
 - Disruptive mood dysregulation disorder
- Personality disorders
 - Antisocial personality disorder
 - Borderline personality disorder
- General medical disorders
 - Dementia
 - Delirium
- Major cognitive disorder
- Personality change due to another medical condition, aggressive type
- Temporal lobe epilepsy

in DSM-5 (Criterion A1). DSM-5 also includes those more destructive acts involving damage or destruction of property and/or physical injury against animals or individuals (Criterion A2), and provides more specific criteria defining the frequency needed (occurring twice weekly, on average, for a period of 3 months for Criterion A1 or three outbursts meeting Criterion A2 in a 12-month period). The aggressive outbursts are now specified as impulsive and/or anger-based in nature (Criterion C), and must cause marked distress in the individual or impairment in occupational or interpersonal functioning, or be associated with negative financial or legal consequences (Criterion D). Although the salient feature of IED is failure to control impulsive aggressive behavior in response to subjectively experienced provocation that would not typically result in an aggressive outburst (Criterion B), it is a diagnosis relevant to the interface between psychiatry and the law.

Because of the paucity of research on this disorder in young children, and also the potential difficulty of distinguishing IED-associated outbursts from those temper tantrums considered normal in childhood, a minimum age of 6 years (or equivalent developmental level) is now required (Criterion E). Finally, the relationship of IED to other disorders especially relevant in the child and adolescent population (e.g., ADHD, disruptive mood dysregulation disorder) has been further clarified.

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 & Regier, 1992). 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 relatively rare. Impulsive aggressive behavior is usually pathological and causes substantial psychosocial distress/dysfunction (McElroy et al., 1998) regardless of its diagnostic origin. 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 problematic (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 witnessing the death and devastation of World War I (*Beyond the Pleasure Principle*, published in 1920) (Freud, 1955).

Felthous et al. (1991) diagnosed 15 men with IED [via DSM-III-R (American Psychiatric Association, 1987)] from amongst a group of 443 men who presented with complaints of violence. Using this cohort, some systematic observations about the “typical violent episode” as reported by patients with IED were made (Felthous et al., 1991). In most instances, the subjects with IED identified their spouse, lover, or girl-/boyfriend 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 have lost track of where they were. None lost control of urine or bowel function during the episode. Various degrees of feelings of behavioral dyscontrol were reported by subjects, with only four having felt that they completely lost control. Six had good recollection of the event afterwards, eight had partial recollection, and one lost memory of the event afterwards. Most IED subjects tried to help or comfort the victim afterwards.

Assessment Issues

Psychiatric Examination and History

The DSM-5 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 coexistence of the other.

Neurological conditions such as degenerative brain diseases, focal frontal lesions, partial complex seizures, and postconcussion syndrome after recent head trauma may all present with episodic impulsive aggression and also 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 another medical condition, aggressive type, and it may be made with a careful history, in addition to noting the characteristic physical, laboratory, and radiographic 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 do suggest an alternative psychiatric or medical diagnosis. There are no specific physical, laboratory, or imaging findings that are pathognomonic 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 levels of 5-hydroxyindoleacetic acid (5-HIAA) in the cerebrospinal fluid (CSF), 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 (MRI) may be more useful than computed tomography of the head. MRI can reveal mesiotemporal scarring, which sometimes in the presence of a normal or inconclusive EEG may be the only evidence for a latent seizure disorder. 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

While the DSM-IV-TR states that IED is “apparently rare,” more recent research, along with a broadening of the diagnostic criteria to include verbal aggression, has identified and acknowledged it to be at least as common as other major psychiatric disorders. The exclusionary criterion in the DSM-5 definition (Criterion F) 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 DSM-III (American Psychiatric Association, 1980). The DSM-III and the revised edition DSM-III-R (American Psychiatric Association, 1987) definitions of IED required the absence of signs of

generalized impulsivity or aggressiveness between episodes. Episodic behavioral disorders are fairly common and exist across a continuum between causes that are purely ictal (excessive neuronal discharges) and those that are purely motivational (psychogenic) in nature. 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 various clinical populations, and one community survey has been done to determine the prevalence of IED. Estimates of the lifetime prevalence of IED range from 1 to 7%, depending on the study population (Grant et al., 2005). 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-TR 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-TR criteria. A subsequent 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 such as schizophrenia and 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-TR IED were 7.3 and 3.9%, respectively, with a mean of 43 lifetime attacks resulting in \$1359 of 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 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 whereas those of the other 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. More recent data suggests that the male:female ratio is closer to 1:1 (Coccaro et al., 2005).

Comorbidity

In contrast to the more restrictive, earlier criteria in DSM-III and DSM-III-R, the definition of IED in DSM-IV-TR and DSM-5 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 generally have a lifetime history of other psychiatric disorders (Coccaro, 2005). The most frequent 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 comorbidity rates raise the question of whether IED constitutes a separate disorder. Recent data finding the onset of IED usually preceding that of comorbid psychopathology, except for phobic-type anxiety disorders, suggest that IED is indeed a separate entity, and not secondary to these other disorders (Coccaro et al., 2005).

McElroy's group 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; McElroy, 1999). 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 include irritability (79–92%), increased energy (83–96%), racing thoughts (62–67%), anxiety (21–42%), and depressed or 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) suggested 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%). Owing to symptomatic overlap between the two conditions, the co-occurrence of IED and bipolar disorder has been obscured in many epidemiological studies. Regardless, clinicians should fully evaluate for bipolar disorder prior to determining treatment for IED, because mood stabilizers, rather than selective 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 few 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 disorder may be prone to rage attacks (Budman et al., 1998, 2000). The clinical manifestation and presentation of these attacks is similar to those of IED and may be more common among those children with Tourette disorder and who also 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 in nature and both include impulsive aggression as an essential feature. Therefore, their diagnosis effectively excludes a diagnosis of IED (Figure 70–2). 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 our laboratories (Hollander E, unpublished work, 2007) 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 who carry a comorbid diagnosis of IED do appear to have higher scores for aggression and lower scores for general psychosocial function than do BPD and ASPD subjects without IED (Coccaro et al., 2005).
2. A history of substance use disorders, especially alcohol use disorder. If explosive outbursts occur only during episodes of substance intoxication, then the diagnosis of IED is not made. However, many patients with IED report past or family histories of a substance use disorder, and in particular alcohol use disorder. In light of evidence linking personal and family history of an alcohol use disorder with impulsive aggression (Linnoila et al., 1989) and linking both with low central serotonergic function (reviewed later), this connection may be clinically relevant. Thus, when there is evidence suggesting that an alcohol use disorder 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 another medical condition, delirium, or major neurocognitive disorder. However, individuals with IED often have nonspecific findings on neurological examination, such as reflex asymmetries or mild hand–eye coordination deficits accompanied by EEGs which may show nonspecific changes. In addition, a childhood history of head trauma, with or without loss of consciousness, may be elicited in a thorough history. 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 typical age at onset and the natural course of IED. That notwithstanding, according to the DSM IV-TR, and anecdotal case reports, the onset appears to be from

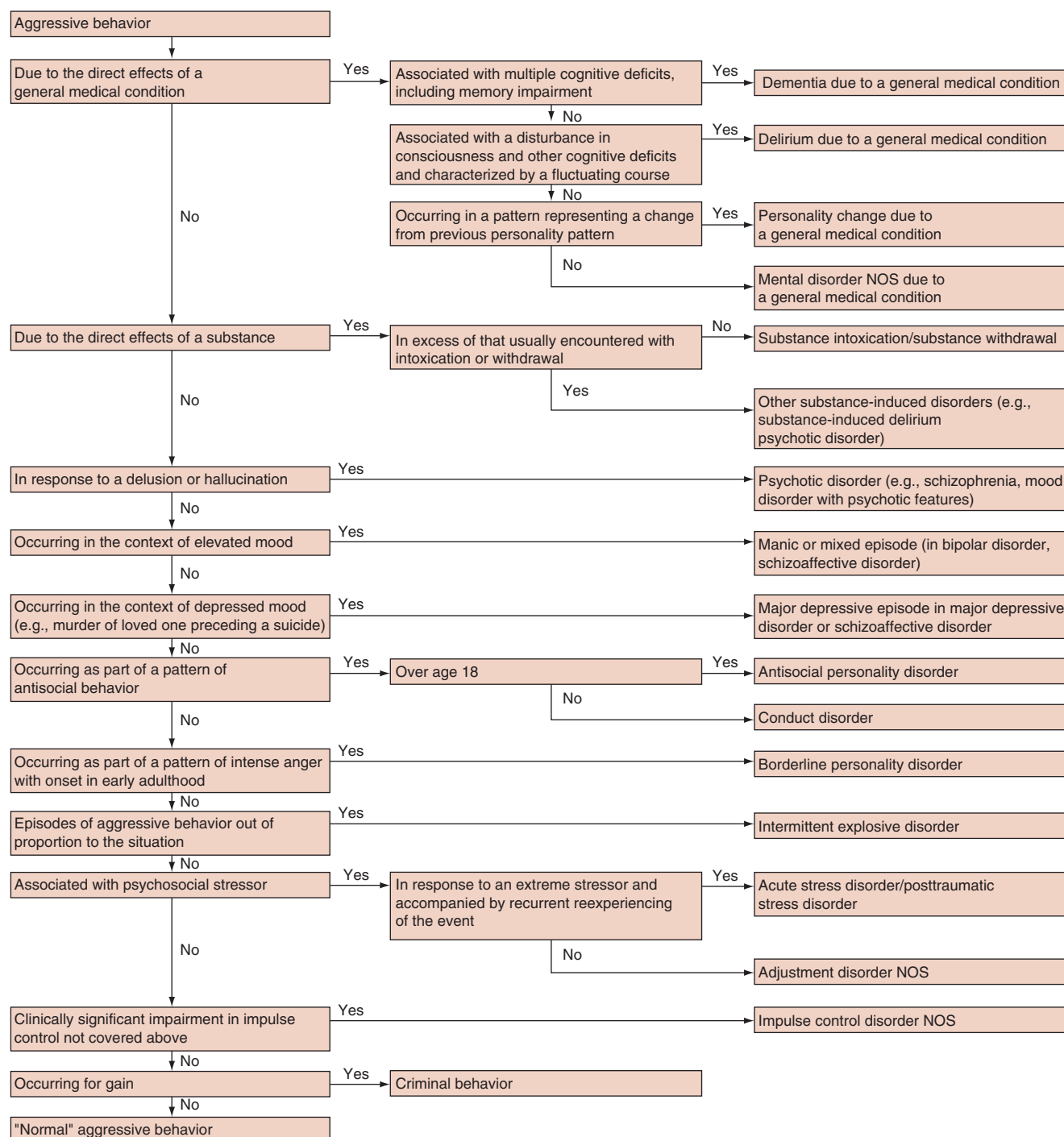


Figure 70–2 Differential diagnosis of aggression. The psychiatric nosology of aggression has not been well worked out and requires much additional study. This is a particularly unfortunate state of affairs because the attribution (or misattribution) of aggression to a mental disorder is a frequent focus 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. NOS, not otherwise specified.

childhood to the early 20s, and may be abrupt and without a prodromal period. The age of onset and course of IED help to distinguish it as separate from its comorbid diagnoses. A mean age at onset of 16 years and an average duration of about 20 years have been described (McElroy et al., 1998). Preliminary data (Coccaro et al., 2005) confirmed these findings and indicated 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). The course of IED is variable, as it tends to be episodic in some cases and more chronic 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.

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

Differential Diagnosis

The differential diagnosis of IED covers that 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 towards 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 disruptive behavior or impulse control disorders such as conduct disorder and oppositional defiant disorder, or a neurodevelopmental disorder such as ADHD and intellectual disability. The DSM-5 helps to clarify and distinguish the impulsive aggression characteristic of IED to that seen in conduct disorder and oppositional defiant disorder. In conduct disorder, the form of aggression characterized by the diagnostic criteria is proactive and predatory whereas the aggression typically seen in oppositional defiant disorder is characterized by temper tantrums and verbal arguments with authority figures. In contrast, the impulsive aggressive outbursts in intermittent explosive disorder are in response to a broader array of provocation, can include physical assault, and have been reported as more severe than that seen in the former two.

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. In children, it is especially important to rule out disruptive mood dysregulation disorder, which can also present with instances of severe temper outbursts. In contrast to intermittent explosive disorder, it is characterized by a persistently negative mood state (i.e., irritability, anger) most of the day, nearly every day, between impulsive aggressive outbursts. In addition to not requiring a persistent disruption in mood between outbursts, intermittent explosive disorder requires only 3 months of active symptoms, in contrast to the 12-month requirement for disruptive mood dysregulation disorder. Impulsive aggression may also be an associated feature of schizophrenia, in which it may occur in response to hallucinations or delusions and it may also appear in variants of obsessive-compulsive

disorder (OCD), presenting 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 70–2 presents the differential diagnosis of aggression.

Common disorders that should be excluded before IED is diagnosed and features that may be helpful in formulating the diagnosis are summarized in Table 70–2.

Differences in 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.

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” have all been suggested to play a role. Since the second half of the nineteenth 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, and also over- or undergratification, may impair the normal development of the ability to anticipate frustration and delay gratification (Khantzian & Mack, 1983).

A second line of explanation, which has yielded numerous positive findings over the past 20 years, views impulsive aggression as the result of variations, or imbalances 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 & 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 laboratory measures of aggression, and also with data obtained from questionnaires and those solicited during the life history review. This relationship has been demonstrated by levels of serotonin in the CSF (Linnoila et al., 1983; Virkkunen et al., 1994), physiological responses to serotonin agonist probes (Coccaro et al., 1989, 1997b; Manuck et al., 1998; Dolan et al., 2001), 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 serotonin was correlated with impulsive aggression only. These findings suggest that impulsive aggressive behavior can be distinguished biologically from nonimpulsive aggression. Interestingly, when impairments to the catecholamine system are experimentally introduced, this inverse relationship between aggression and serotonin is not observed (Coccaro et al., 1989; Wetzler et al., 1991). Siever et al. (1991) and Stein et al. (1993) have confirmed a relationship between levels of serotonin 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 measuring impulsive aggression. Studies of impulsive aggression among individuals with alcohol dependence have further defined a probable relationship between such behaviors and diminished serotonergic function (Virkkunen & Linnoila, 1993; Virkkunen et al., 1995).

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 (Virkkunen et al., 1994; Giammanco et al., 2005), 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 in 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-HT₂ receptors, the number of which 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, is 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. Performance was impaired in neurological patients with frontal lesions, whereas it remained intact in normal subjects, resulting in an increase in their frontal glucose metabolism (Raine et al., 1994). A visual-evoked potential and EEG study in a large group of aggressive children and adolescents also suggested that such behavior may be associated with altered innate characteristics of central nervous system function (Bars et al., 2001).

Hence biological studies implicate the serotonergic system and the prefrontal cortex 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 have been committed. Despite the recent advances in the understanding of biological underpinnings of violence, the implementation of cognitive neuroscience and molecular genetics within the criminal responsibility assessment is at the center of a harsh debate. The legal doctrine of responsibility is not challenged by identifying biological factors as partially determining crime any more than it is by findings of social causation. Only in cases in which abnormal biological factors are exceptionally powerful influences might responsibility be challenged. Such cases will be rare (Mednick & Finello, 1983).

Family and Twin Studies

Clinical observation and family history data suggest that IED is a familial disorder. Familial aggregation of temper outbursts and IED has been reported in psychiatric patients with “temper problems” (Mattes & Fink, 1987). McElroy et al. (1998) reported that nearly one-third of first-degree relatives of IED probands also carried the diagnosis. A blinded, controlled, family history study (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 have been no twin studies of IED itself. Genetic influence for these two traits ranges from 28 to 47%, with nonshared environmental influences making up the majority of the remaining variance. In this study, Coccaro et al. used an alternative criteria set that integrated their research findings with the DSM’s definition and conceptualizations of IED.

This “integrated research” criterion (IED-IR) differs from that defined in the DSM-IV-TR on several major points. IED-IR allows for inclusion of low-intensity, including verbal, aggression. In addition, it requires that the aggressive behavior be impulsive in nature and be accompanied by subjective distress and/or social or occupational dysfunction. Furthermore, the criteria allow patients with borderline and/or antisocial personality disorder to receive a comorbid diagnosis of IED.

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 with 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 alcohol dependence. 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. Impulsive aggressive behavior has been conceptualized as an imbalance between excessive, aggressive drives originating in limbic brain structures such as the amygdala, and insufficient control of these impulses by cortical structures such as the orbital frontal cortex and anterior cingulate cortex (Siever, 2008). Coccaro et al.'s study in which 20 unmedicated subjects (10 patients with IED and 10 healthy, matched controls) underwent functional magnetic resonance imaging while viewing blocks of emotionally salient faces supports this hypothesis (Coccaro et al., 2007). Compared with controls, patients with IED exhibited increased activation of the amygdala and reduced activation of the orbitofrontal cortex to faces expressing anger. The extent of the activation in both amygdala and orbitofrontal cortex in response to faces expressing anger was differentially related to prior aggressive behavior across subjects.

In an earlier study, 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, the data of Best et al. (2002) 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 prescribed. Both psychological and somatic therapies have been utilized in the treatment of IED, and a prerequisite for both modalities is the willingness of the individual to acknowledge some responsibility for the behavior and to participate in attempts to control it.

Somatic Treatments

Several classes of medications have been used to treat IED and the symptoms of impulsive aggression in the context of other disorders. These include beta-blockers (propranolol and metoprolol), anticonvulsants (carbamazepine and valproic acid), lithium, antidepressants [tricyclic antidepressants (TCAs) and SSRIs], and anti-anxiety 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 intellectual disability. Lithium has been shown to have anti-aggressive 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. In the absence of comorbid disorders, carbamazepine, titrated to anti-epileptic blood levels, may be used empirically.

Although both first- and second-generation antipsychotics have certainly been used to treat impulsive aggression, there are no data supporting their use specifically in the treatment of IED. Given the side-effect profile of these medications, they should be used with caution and with monitoring of potential side effects. Overall, in the absence of more controlled clinical trials, the best approach may be to tailor the psychopharmacological agent to coexisting psychiatric comorbidity.

Psychosocial Treatments

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 first to 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 the treatment of anger as a component of other psychiatric illnesses, such as substance use disorders, posttraumatic stress disorder, depression, and domestic violence, and also 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 (Groditzky & Tafrate, 2000). Subjects habituated to anger-provoking scenarios, and the treatment was felt to be useful. In a controlled trial of volunteer college students who self-identified as angry drivers, 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 the subjects' anger while driving. 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.

Another form of behavioral therapy, dialectical behavior therapy (DBT), is widely used, and has been studied extensively in patients with BPD. One study showed improvement in anger, global functioning, and social adjustment when compared with treatment as usual (Linehan et al., 1994). Improvement in anger and impulsivity has been shown with DBT across many disorders. Components of programs such as DBT for individuals with borderline personality disorder may prove efficacious for individuals with IED. Individuals with IED have deficits in the ability to recognize and be mindful of behavioral consequences, necessitating training in self-control in order to inhibit stress responses to benign environmental cues. One additional component of treating anger and the lack of premeditation characteristic of individuals with IED is mindfulness training. Further research is needed in both the use of entire DBT programs and in mindfulness training alone for the treatment of IED.

Clinical Vignette 1

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 per 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 tests 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 the 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, as are familial, neurobiological, and genetic investigations. 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 first designated as a psychiatric disorder in 1980 in DSM-III (American Psychiatric Association, 1980) and was later grouped under “disorders of impulse control not elsewhere classified” in DSM-III-R (American Psychiatric Association, 1987). In DSM-IV-TR (American Psychiatric Association, 2000), it was classified as an ICD, as it continues to be in DSM-5 (American Psychiatric Association, 2013), categorized in the chapter on Disruptive, Impulse Control, and Conduct Disorders. Remaining a poorly understood entity, changes were not made to the diagnostic criteria between those of DSM-IV-TR and DSM-5. Criterion A focuses not only the impulsive nature of the stealing, but also on the senselessness of the items stolen, specifying that the objects are not needed either for personal use or for their monetary value. This 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.

Diagnostic Criteria B and C speak to sense of tension which rises prior to committing the theft, followed by pleasure, gratification, or relief at the time the act is completed. While this mounting pressure is typically experienced and relieved by the impulsive act, 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 may recall entering and leaving a store but have no memory of events in the store, including the theft (Grant, 2004). Others, who are not amnesic for the thefts, describe shoplifting as “automatic” or “a habit,” and may also deny feelings of tension prior to a theft (Criterion B) or pleasure after the act (Criterion 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 amnesic for shoplifting or who do so “out of habit” represent two different subtypes of kleptomania. Criteria D and E are both exclusionary, ruling out other causes of theft, including that committed to express anger or vengeance (Criterion D), in response to a delusion or hallucination (Criterion D), or stealing that is better explained by conduct disorder, a manic episode, or antisocial personality disorder (Criterion E).

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. Fairly 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.

Assessment Issues

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 (1991) 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). A medical evaluation is indicated for patients presenting atypically, as such presentations should raise a greater suspicion of an organic etiology. Medical conditions that have been associated with kleptomania include cortical atrophy, degenerative brain diseases, intracranial mass lesions, encephalitis, normal-pressure hydrocephalus, benzodiazepine withdrawal, and temporal lobe epilepsy. 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 & Martin, 1977; Coid, 1984; Mendez, 1988; McIntyre & Emsley, 1990; Wood & Garraalda, 1990).

Epidemiology

Although no larger scale epidemiological studies have been conducted to assess the prevalence of kleptomania in the general population, a survey of college students ($n = 791$) found that three (0.38%) met DSM-IV-TR criteria for the disorder (Odlaug & Grant, 2010). 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, such as 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 & Kim, 2002c). In addition, studies examining comorbidity of other disorders may neglect to inquire about all of the criteria required for making a diagnosis of kleptomania. Studies of kleptomania in various clinical samples suggest a higher prevalence. A study of 204 adult psychiatric inpatients in the United States, 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 four (3.7%) had kleptomania (Lejoyeux et al., 2002); in another study of 79 alcohol-dependent inpatients, three (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% (Specker et al., 1995; Grant & Kim, 2003). A study of bulimia patients found that 24% had symptoms that met DSM-III criteria for kleptomania (Hudson et al., 1983).

The literature clearly suggests that the majority of patients with kleptomania are women (e.g., McElroy et al., 1991b; Grant & Kim, 2002b; 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). Grant (2006) extrapolated the results of four studies which assembled large numbers of patients with kleptomania ($n = 108$) and found 68 (63%) of them to be female. Although a majority of reported cases of kleptomania indicate a female predominance, these findings may be biased as women may be more likely than men to present for psychiatric evaluation and treatment. Furthermore, the legal system may be more likely to send female shoplifters for psychiatric evaluation and send male shoplifters to prison (Goldman, 1991; Grant, 2006). Consequently, these figures may not reflect true gender distribution. The severity of kleptomania symptoms and the clinical presentation do not appear to differ based on gender (Grant & Kim, 2002b).

Comorbidity

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 compared with rates in the general population. Rates of lifetime comorbid affective disorders range from 59% (Grant & Kim, 2002b) to 100% (McElroy et al., 1991b). The rate of lifetime comorbid bipolar disorder has been reported as ranging from 9% (Grant & 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), other ICDs (20–46%; Grant & Kim, 2003), substance use disorders (23–50%; McElroy et al., 1991b; Grant & Kim, 2002b), 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 disorders (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 eight patients (31%), and bulimia nervosa in three patients (12%). Similar percentages were 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).

Course

Although the age at onset of kleptomania is variable, the disorder most often begins in adolescence. The disorder may begin in childhood, adolescence, or adulthood, and in rare cases in late adulthood (Goldman, 1991; McElroy et al., 1991a,b; Grant & Kim, 2002b; 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 5–7 years old. Onset beyond the age of 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; McElroy et al., 1991b; Grant & Kim, 2002c) 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 per 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 arrests, convictions, or imprisonment for shoplifting, with transient periods of remission.

There is little systematic information on the course of kleptomania, but 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-5).

Differential Diagnosis

Most individuals with kleptomania steal from stores. However, kleptomania is not synonymous with shoplifting, and those with the disorder differ from “ordinary” shoplifters in that they do not steal for personal gain, but rather for symptomatic relief (McElroy et al., 1991b). Ordinary theft, whether planned or impulsive, is deliberate and is motivated by the usefulness of the object or its monetary worth. Some individuals, especially adolescents, may also steal on a dare, as an act of rebellion, or as a rite of passage. The diagnosis is not made unless other characteristic features are also present. Kleptomania is exceedingly rare, whereas shoplifting is relatively common. According to the DSM-5, kleptomania occurs in 4–24% of individuals arrested for shoplifting. These rates may be falsely low due to incomplete psychiatric evaluations, lack of strict diagnostic criteria for kleptomania, and selection bias in these samples (McElroy et al., 1991b). Malingering, in which individuals may simulate the symptoms of kleptomania to avoid criminal prosecution, must also be ruled out when considering a diagnosis of kleptomania. Other diagnoses that should be distinguished from kleptomania include antisocial personality disorder and conduct disorder, and also the intentional or inadvertent stealing that may occur during a manic episode, in response to delusions or hallucinations, or as a result of a major neurocognitive disorder.

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 such as depression, panic

disorder, OCD, and bulimia nervosa (Hudson & Pope, 1990; McElroy et al., 1991b, 1992). The apparent high comorbidity of kleptomania with depression and bulimia nervosa has already been noted. As early as 1911, Janet recognized the alleviation of depressive symptoms on the commission of kleptomaniac acts (Janet, 1911). 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 & 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. Both conditions also 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 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, bingeing, hypersexuality, and kleptomania. This model postulates certain concepts thought to be common in all these disorders, including craving, mounting tension, “quick fixing,” withdrawal, external cuing, and habituation. Although these components have not yet been well investigated in kleptomania, in one study, 68.2% of individuals reported that the value of stolen items had increased over the duration of the disorder, suggesting tolerance (Grant & Kim, 2002b). Further supporting this model of addictive-type behavior is that most individuals with kleptomania try unsuccessfully to stop stealing, and their inability to do so often leads to feelings of shame and guilt (Grant & Kim, 2002c).

Biological Theories

Serotonin and Inhibition

Compared with controls, kleptomania patients report significant elevations of impulsivity and risk taking (Grant & Kim, 2002d; Bayle et al., 2003), and diminished inhibitory

mechanisms may underlie the risk-taking behavior of kleptomania. The most well-studied inhibitory pathways involve serotonin and the prefrontal cortex (Chambers et al., 2003). Decreased measures of serotonin have long been associated with a variety of adult risk-taking 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 have been 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 and TCAs such as clomipramine (McElroy et al., 1991b; Lepkifker et al., 1999) 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. Thus, future behavior may be determined in part by neuroplastic changes in the nucleus accumbens, as a consequence of previously rewarding experiences. This may explain why, over time, many kleptomania patients report shoplifting “out of habit” even without a pronounced urge or craving.

The poor decision-making and problems with inhibitory control seen in kleptomania may be due to impairments in prefrontally mediated cognitive functions. Some support for this hypothesis comes from a diffusion tensor imaging study which found that kleptomania was associated with decreased white matter microstructural integrity in inferior frontal brain regions (Grant et al., 2006). Studies of naturally occurring catechol-*O*-methyltransferase (COMT) isoenzymes with differential enzymatic activity have shown that the higher cortical dopamine levels associated with reduced COMT activity result in improved frontal cortical cognitive performance (Malhotra et al.,

2002). Impairments in cortical information processing may increase the risk for making impulsive decisions that are focused on short-term gains and lack inhibitory control, thereby placing the individual at high risk for continued impulsive behavior. Shoplifting, as one example of impulsive behavior, may be related to less efficient prefrontal neural signaling and possible deficits in executive functioning (Grant et al., 2006). A COMT inhibitor therefore may offer a unique mechanism to address cognitive deficits associated with kleptomania and possibly other impulse control disorders (Grant, 2011).

Opioid System, Cravings, and Pleasure

Kleptomaniacs report frequent urges to steal, which result in theft twice weekly on average (Grant & Kim, 2002b). Hence 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 that 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 & 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 & 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; Kim et al., 2001; Grant & Kim, 2002c).

In sum, repeated kleptomanic behavior may be a result of an imbalance between a pathologically increased urge and a pathologically decreased inhibition. The repeated stealing 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.

Psychodynamic Models

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 at symptom management (Goldman, 1991). Supporting the self-medication hypothesis of shoplifting, patients with kleptomania report high lifetime rates of depression (45–100%; McElroy et al., 1991b; Bayle et al., 2003) 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 (McElroy et al., 1991b; Lepkifker et al., 1999).

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 kleptomaniac acts (Fenichel, 1945; Fishbain, 1987). Thus, it has been suggested that kleptomaniac 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 (Goldman, 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 kleptomaniac 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 explored the presence of personality disorders 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 kleptomaniac 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 also serves to increase the behavior, as the stealing is performed to experience relief from the aversive tension which mounts prior to the act (i.e., the removal of a punishing stimulus). This "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 kleptomaniac 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 biopsychosocial 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 kleptomaniac behavior. Treatment typically occurs in the outpatient setting, unless comorbid conditions such as 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 about 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 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 U.S. Food and Drug Administration (FDA) for treating kleptomania. Therefore, 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, including TCAs, SSRIs (Lepkifker et al., 1999), mood stabilizers, and opioid antagonists have been examined for the treatment of klepto-

mania (McElroy et al., 1989; Grant & Kim, 2002c), with mixed results. In a literature review of 56 kleptomania cases, McElroy et al. (1991a) noted that somatic treatments were described for eight patients, with significant improvement reported in seven of them. 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 showed partial or complete remission of both kleptomaniac 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, with only two showing a full response and one responding partially. 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 of 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). 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 (Fishbain, 1987; McElroy et al., 1991a). However, it remains unclear if the antikleptomaniac effect of thymoleptics is dependent on or independent of their antidepressant effect.

A number of other medications have been employed to treat kleptomania, including antipsychotics (Fishbain, 1987; McElroy et al., 1991b), stimulants (McElroy et al., 1991b), valproic acid (McElroy et al., 1991a), carbamazepine (McElroy et al., 1991a), clonazepam (McElroy et al., 1991a), and lithium (Monopolis & Lion, 1983; McElroy et al., 1991a). 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 & 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 and 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). One case series found that kleptomania symptoms responded to topiramate (Dannon, 2003), and another reported a response in two subjects treated with naltrexone (Dannon et al., 1999).

Only one randomized, double-blind, placebo-controlled trial has been conducted to date for the treatment of kleptomania. In their evaluation of the safety and efficacy of oral naltrexone, Grant et al. (2009) randomized 25 subjects with DSM-IV-TR kleptomania to naltrexone (dosing ranging from 50 to 150 mg/day) or placebo. Of the 23 individuals who completed the study, those assigned to naltrexone (mean effective dosage 117 mg/day) were found to have statistically significant reductions in stealing urges and stealing behavior compared with subjects on placebo (Grant et al., 2009). This clinical trial was an extension of their previous open-label trial for kleptomania, in which naltrexone resulted in a significant decline in the intensity of urges to steal, stealing thoughts, and stealing behavior (mean effective dosage 145 mg/day) (Grant & Kim, 2002c). A lower dosage, possibly 50 mg/day, may be effective in younger people with kleptomania (Grant & Kim, 2002a). Opioid antagonists such as naltrexone may be effective in reducing both the urges to shoplift and shoplifting behavior itself, by reducing the associated “thrill,” 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 impairment in both urge regulation and behavior inhibition, 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 eight patients who were treated with behavioral therapy – mostly aversive conditioning – showed significant improvement. The literature contains specific examples of different behavioral techniques that have been successfully employed and described, and it is suggested that these techniques remain effective over the long term (Gauthier & Pellerin, 1982; Glover, 1985). 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 kleptomaniac acts (Fishbain, 1987). 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 14 15-minute sessions over 5 days, two patients reported complete remission of symptoms for a 2-year period (McConaghy & Blaszczyński, 1988). A woman who was treated weekly for 5 months, and reported 2 years of continued remittance, used substitution of alternative sources of satisfaction and excitement in place of her urges to steal (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. 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 four of five 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 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, such as 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 delineate systematically 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 for those 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 on 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, suicidality, and extensive past psychiatric treatments who responded to a combination of supportive and insight-oriented therapy, medication, and behavior modification.

Clinical Vignette 2

Ms. B is a 34-year-old divorced Caucasian female with four children who was referred for a court-mandated mental health evaluation after she was arrested for shoplifting cosmetic products from a store. She reports that she first began stealing at the age of 15 years, when she would occasionally steal candy with her friends. This progressed over a period of several years and developed into an almost daily ritual, which she would do by herself. She currently reports that she shoplifts one or two times each week. Although always employed, and in fact financially comfortable, she describes often stealing unnecessary items from retail stores and sometimes also from friends. The objects she steals from stores are typically personal hygiene and cosmetic products of little value, which she keeps hidden in a box in her closet at home. Her friends and family do not know about her problem.

She has been caught by store security staff several times but this is her first encounter with the law. She reports feeling extremely embarrassed as a result of her arrest, similar to how she has felt in the past when stopped and questioned by store employees. She is now afraid that she may lose her job and is fearful that she will not be able to stop stealing. She feels that her shoplifting has become uncontrollable and reports thinking about it often, on a daily basis.

Ms. B reports that upon entering a store, an overwhelming urge to steal comes over her, and that a tension or anxiety builds which does not subside until she completes the theft. After leaving the store, Ms. B reports feeling conflicted, as she feels a rush and a sense of relief, yet also feel guilty and remorseful about having taken the item. Detailed diagnostic assessment shows that Ms. B has no other psychiatric problems. Her family history is positive for both alcohol and substance use disorders.

PYROMANIA AND FIRE-SETTING BEHAVIOR

Diagnosis

Definition and Diagnostic Features

Pyromania is characterized in the DSM-5 by deliberate and purposeful fire setting on more than one occasion (Criterion A). Other defining features of pyromania include the experience of tension or affective arousal before the fire setting (Criterion B), an attraction to, curiosity about, interest in, or fascination with, fire and its contexts, including paraphernalia, uses, and consequences (Criterion C), and a feeling of pleasure, gratification, or relief associated with the act of setting a fire or when witnessing or participating in its aftermath (Criterion D). As with the last two diagnostic criteria of kleptomania, Criteria E and F are exclusionary, with Criterion E specifying that the fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, or to improve one's living circumstances, nor is it in response to a delusion or hallucination, or as a result of impaired judgment related to a major neurocognitive disorder, intellectual disability, or substance intoxication. Criterion F excludes fire-setting behavior that can be explained by conduct disorder, a manic episode, or antisocial personality disorder.

The diagnosis of pyromania, the criteria of which did not change from DSM-IV-TR to DSM-5, emphasizes the affective arousal, thrill, or tension preceding the act, in addition to 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 & Dimock, 1986). The most frequent motives for arson by juveniles are revenge on parents or other authorities, a search for excitement and motive to help the firefighters as would-be heroes, self-destructiveness, a craving for sensation, and an expression of outrage (Räsänen et al., 1995). There is also a considerable rate of self-destructive behavior amongst juveniles prior to committing arson; 74% have suicidal thoughts and 44% have tried to commit suicide before setting a fire. Females with pyromania frequently have a history of self-harm, sexual abuse, and psychosocial traumas (Noblett & 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 & Bertsch, 1985).

True pyromania is present in only a small subset of fire setters. Prins et al. (1985) suggested the following motivations for intentional arson: financial reward, to conceal another crime, for political purposes, as a means of revenge, as attention-seeking behavior, as a means of deriving sexual satisfaction, and as an act of curiosity when committed by children. They also recognized that fire setting can exist as

a symptom of other psychiatric conditions (e.g., in response to a delusional belief), which is discussed along with other differential diagnoses in that section. Revenge and anger appear to be the most common motivations for fire setting (O'Sullivan & Kelleher, 1987). 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. Fire-setting behavior may be a focus of clinical attention, even when criteria for pyromania are not present. Diagnostic classifications including in DSM-5, where it appears in the chapter on Disruptive, Impulse Control, and Conduct Disorders, list pyromania among the ICDs as the associated fire-setting act is thought to result from the failure to resist an impulse. Although pyromaniacs may methodically prepare the fire and leave obvious clues of this preparation behind (Wise & Tierney, 1999), pyromania is still considered an uncontrolled and often impulsive behavior.

Pyromania onset has been reported to occur as early as the age of 3 years, although it may present initially 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 & Bertsch, 1985).

Assessment Issues

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, providing 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. Hence the interviewer must maintain a guarded view of the information presented.

Epidemiology

Although fire setting has been examined in the medical literature for the last 200 years, there has been only limited research concerning the unique characteristics of pyromania (Lejoyeux et al., 2006). Most epidemiological studies have not focused directly on pyromania, but instead on various populations of arsonists or fire setters.

Although the prevalence of pyromania is not well established, one study looked at a sample of 791 college students and reported a rate of 1% (Odlaug & Grant, 2010). In another study, also suggesting the rare nature of pyromania, the authors found that only three (3.3%) of 90 arson recidivists had pure pyromania and that an additional nine subjects (10%) met DSM-IV-TR criteria for pyromania only when intoxicated at the time of the arson (Lindberg et al., 2005). 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-5, pyromania occurs much more often in males, especially those with poorer social skills and learning difficulties. This notation

confirms the data of Lewis and Yarnell (1951) 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” pyromania. Although true pyromania, meeting DSM criteria, is a rare entity, 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%), and also in male patients (28.8%).

As pyromania typically emerges during late childhood and adolescence, prevalence in this age group has been found to be more common than in adults. A study of 102 adolescent psychiatric inpatients reported that 6.9% met criteria for current pyromania (Grant et al., 2007). Fire-setting behavior is frequently seen in children and in adolescents, especially those with psychiatric conditions. 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 & Kazdin, 1988). Those conditions most often associated with juvenile fire setting include conduct disorder, attention deficit/hyperactivity disorder, and adjustment disorder.

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. It was found that 39% of the fire setters from the study received the diagnosis of pyromania. Of those, 22% had borderline intellectual functioning, and 13% low average intelligence. Fire setters were also described as driven by an irresistible impulse to set fires. The findings suggested that 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. Although the highest incidence is typically seen in adolescents, pyromania can present and be seen in subjects of any age, especially among females, where the diversity of ages is particularly apparent (Barker, 1994). Also, neither confirmed nor reproduced in more recent studies are the high prevalence rates of pyromania noted by Lewis and Yarnell (1951). Koson and Dvoskin (1982) found no cases of pyromania in a population of 26 arsonists. Ritchie and Huff (1999) identified only three cases of pyromania in 283 cases of arson.

Comorbidity

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 impulsive fire setters. Fire setting in subjects who do not have pyromania appears frequently and is often underrecognized. Although in most cases fire-setting behavior is not directly related to pyromania, it may be associated with other mental conditions, including substance use disorders, gambling disorder, depressive and bipolar disorders, and other disruptive, impulse control, and conduct disorders. 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; 36% 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 one-quarter were female, psychoactive substance abuse was common, and that 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 had symptoms that met criteria for ICDs: 18 had IED, three had pathological gambling, four had kleptomania, three had pyromania, and three 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 (68%) of the 103 subjects had consumed alcohol before setting a fire and 54 (52%) of the subjects presented with alcohol dependence. The authors suggested a correlation between the amount of alcohol consumed and the frequency of fire setting. Räsänen et al. (1995) found that young arsonists frequently have problems with alcohol: 82% had alcohol dependence 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. Among the 79 patients included in the study, they found that 30 (38%) had symptoms that met criteria for one ICD (19 with IED, seven with pathological gambling, three with kleptomania, and one with trichotillomania), but that none of the patients presented with two or more ICDs, and no patients 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-5, there are insufficient data to establish a typical age at onset of pyromania. Although

the longitudinal course is also unknown, 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 & Lion, 1977) to 28% (Lewis & 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. Those arsonists considered to be mentally disordered 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 such as theft, traffic violations, and alcohol-related offenses, had a higher rate of recurrence, and committed fewer common offenses other than fire setters. 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 part of a ritual, cultural, or religious practice in some cultures. Fire setting may also occur as part of developmental experimentation in childhood (e.g., playing with matches, lighters, or fire).

As was also the case in the differential diagnosis of kleptomania, malingering should be considered and ruled out, as symptoms may be simulated to avoid criminal prosecution. Fire setting may occur in the presence of other mental disorders. A separate diagnosis of pyromania is not made when the fire setting occurs in response to a delusion or hallucination, or as a part of conduct disorder, a manic episode, or ASPD. Likewise, behavior that is attributable to the physiological effects of another medical condition or to the impaired judgment associated with major neurocognitive disorder, intellectual disability, or substance intoxication precludes a diagnosis of pyromania.

Etiology and Pathophysiology

Because pyromania is such a rare diagnosis, there is little reliable scientific literature available regarding individuals who fit diagnostic criteria. 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 and is presented here.

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). 5-HIAA is the primary metabolite of serotonin, and its concentration in the CSF is a valid marker of serotonin function in the brain. Investigation of the function of serotonergic neurotransmission in individuals with aggressive and violent behaviors has included studies of CSF 5-HIAA concentrations in individuals with a history of fire setting. Virkkunen et al. (1987, 1994) demonstrated that impulsive fire setting was associated with low 5-HIAA levels found in the CSF. This finding was consistent with other observations associating impulsive behaviors with low CSF 5-HIAA levels (such as impulsive violence and impulsive suicidal behavior). DeJong et al.’s determination that a history of suicide attempt strongly predicts the recidivism of arson further supports this correlation (DeJong et al., 1992).

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 concentrations of both 5-HIAA and 3-methoxy-4-hydroxyphenylglycol (MHPG), and also early family environments characterized by paternal absence and the presence of brothers at home. Linnoila et al. (1989) also studied the correlation between impulse fire setters and alcohol dependence, and found that in those with a history of violent fire-related offenses, there was an increase in alcohol dependence not only in the offenders themselves, but also in their fathers.

Psychodynamic Models

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) involved with fire. According to Geller and Bertsch (1985), children at risk for 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. Hence 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 imagining/recalling a fire fantasy just before orgasm (McGuire et al., 1965). Lewis and Yarnell (1951) suggested 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 altogether. 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 frequently be 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 as 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 alcohol dependence, and lack insight (Mavromatis & 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 cognitive behavioral therapy 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 such as aversion therapy have helped fire setters (McGrath & Marshall, 1979; Koles & Jenson, 1985). Other treatment methods rely on positive reinforcement with threats of punishment and stimulus 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 two 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. (2002) 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 attended a 1-day informational program. The program's interactive content focused on the medical, financial, legal, and societal impacts 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.

Clinical Vignette 3

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.

During 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.

Comparison of DSM-5 and ICD-10 Diagnostic Criteria

The ICD-10 diagnostic criteria for research do not include diagnostic criteria for IED. It is included in ICD-10 only as an “Other Habit and Impulse Control Disorder.” The ICD-10 diagnostic criteria for research and the DSM-5 criteria for kleptomania and pyromania are essentially equivalent.

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