

Impulse Control Disorders: Intermittent Explosive Disorder, Kleptomania, and Pyromania

Jonathan L. Kirsch, MD¹

Daphne Simeon, MD²

Heather Berlin, PhD, MPH³

Eric Hollander, MD¹

¹Department of Psychiatry, Albert Einstein College of Medicine, New York, NY, USA

²Department of Psychiatry, Beth Israel Medical Center, New York, NY, USA

³Departments of Psychiatry & Neuroscience, Icahn School of Medicine at Mount Sinai, New York, NY, USA

Psychiatry, Fourth Edition. Edited by Allan Tasman, Jerald Kay, Jeffrey A. Lieberman, Michael B. First and Michelle Riba, 2014, John Wiley & Sons, Ltd.

This chapter is based in part on the chapter “Impulse Control Disorders” by Daphne Simeon and Heather Berlin that appeared in *Psychiatry, Third Edition*, edited by Allan Tasman, Jerald Kay, Jeffrey A.

Lieberman, Michael B. First, and Mario Maj, 2008, John Wiley & Sons Ltd. ISBN: 978-0-470-06571-6.

Abstract The impulse control disorders (intermittent explosive disorder, kleptomania, and pyromania) share the feature of the irresistible urge to act in a given way. Effort to resist may be associated with increasing tension, giving rise to further need to act on the given impulse. This chapter reviews clinical features, epidemiology, neurobiology and clinical interventions.

Keywords

Impulse control disorders, impulsivity, intermittent explosive disorder, kleptomania, pyromania

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-V (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. 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'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 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 properly conceptualize ICDs, it is helpful to understand the role of impulsivity within them. The trait of impulsivity has been the subject of increasing interest in psychiatry. Impulsivity is a defining characteristic of many psychiatric illnesses, even those not classified as ICDs, including Cluster B personality disorders like borderline personality disorder (BPD) and antisocial personality disorder (ASPD), neurological disorders characterized by disinhibited behavior, attention-deficit/hyperactivity disorder (ADHD), substance and alcohol abuse, conduct disorder, binge-eating, bulimia, and paraphilias. Impulsivity research has been conducted both in disorders characterized by impulsivity, like BPD, ASPD, and conduct disorder, and in traditional ICDs like IED. Clinicians should recognize that individuals who are prone to impulsivity and ICDs are often afflicted with a cluster of related conditions including sexual compulsions, substance use disorders, and posttraumatic stress disorder, making screening for comorbid conditions that contribute to impulsivity, including these, as well as bipolar spectrum disorders and ADHD, especially important. A decision tree for the differential diagnosis of impulsive behaviors may be seen in Figure 80–1.

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 as well as a core feature of human behavior. An impulse is rash and lacks deliberation. It may be sudden and ephemeral, or a steady rise in tension that may reach a climax in an explosive expression of the impulse, 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, 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.

New 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 80–1). First is the increased sense of tension or arousal, followed by the failure to resist the urge to act. Third, there is a heightened sense of arousal. Once the act has been completed, there is a sense of relief from the urge. Finally, the patient experiences guilt and remorse at having committed the act. In conditions like trichotillomania, pyromania, and pathological gambling, the individual may spend considerable amounts of time fighting off the urge, trying not to carry out the impulse. 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.

Although impulse control disorders historically had been considered uncommon, later investigations suggest that some of them may occur more frequently than once assumed. Trichotillomania, (though 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 under-represented 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 20th 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. While 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).

While 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 (see box for diagnostic criteria). Impulsive aggression however, is not specific to IED. It is a key feature of several psychiatric disorders and nonpsychiatric conditions and may emerge during the course of yet other psychiatric disorders. The definition of IED as formulated in the DSM-5 is therefore essentially a diagnosis of exclusion. As described in criterion F, a diagnosis of IED is made only after other mental disorders and medical conditions that might account for recurrent aggressive outbursts have been ruled out. The individual may describe the aggressive episodes as “spells” or “attacks.” The symptoms appear within minutes to hours and, regardless of the duration of the episode, may remit almost as quickly. As in other ICDs, the explosive behavior may be preceded by a sense of tension or arousal and is followed immediately by a sense of relief or release of tension.

DSM-5 Diagnostic Criteria
Intermittent Explosive Disorder - 312.34 (F63.81)
A. Recurrent behavioral outbursts representing a failure to control aggressive impulses as manifested by either of the following:
1. Verbal aggression (e.g., temper tantrums, tirades, verbal arguments or fights) or physical aggression toward property, animals, or other individuals, occurring twice

weekly, on average for a period of 3 months. The physical aggression does not result in damage or destruction of property and does not result in physical injury to animals or other individuals.

2. Three behavioral outbursts involving damage or destruction of property and/or physical assault involving physical injury against animals or other individuals occurring within a 12-month period.

B. The magnitude of aggressiveness expressed during the recurrent outbursts is grossly out of proportion to the provocation or to any precipitation psychosocial stressors.

C. The recurrent aggressive outbursts are not premeditated (i.e., they are impulsive and/or anger-based) and are not committed to achieve some tangible objective (e.g., money, power, intimidation).

D. The recurrent aggressive outbursts cause either marked distress in the individual or impairment in occupational or interpersonal functioning, or are associated with financial or legal consequences.

E. Chronological age is at least 6 years (or equivalent developmental level).

F. The recurrent aggressive outbursts are not better explained by another mental disorder (e.g., major depressive disorder, bipolar disorder, disruptive mood dysregulation disorder, a psychotic disorder, antisocial personality disorder, borderline personality disorder) and are not attributable to another medical condition (e.g., head trauma, Alzheimer's disease) or to the physiologic effects of a substance (e.g., a drug of abuse, a medication). For children ages 6-18 years, aggressive behavior that occurs as part of an adjustment disorder should not be considered for this diagnosis.

Note: This diagnosis can be made in addition to the diagnosis of attention-deficit/hyperactivity

disorder, conduct disorder, oppositional defiant disorder, or autistic spectrum disorder when recurrent impulsive aggressive outbursts are in excess of those usually seen in these disorders and warrant independent clinical attention.

Although not explicitly stated in the DSM-5 definition of IED, impulsive aggressive behavior may have many motivations that are not meant to be included within this diagnosis. IED should not be diagnosed when the purpose of the aggression is monetary gain, vengeance, self-defense, social dominance, expressing a political statement, or when it occurs as a part of gang behavior. Typically, the aggressive behavior is ego-dystonic to individuals with IED, who feel genuinely upset, remorseful, regretful, bewildered, or embarrassed about their impulsive aggressive acts.

The primary change seen in DSM-5 with regard to IED is the type of aggressive outbursts that should be considered. While 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 in DSM-5 (Criterion A1). DMS-5 also includes those more destructive acts, involving damage or destruction of property or physical injury (Criterion A2) and provides more specific criteria defining the frequency needed (occurring twice weekly, on average, for a period of three 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). While 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, as well as 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., attention-deficit/hyperactivity disorder, 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 and 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 problematical (McElroy et al. 1992). To a large extent, this difficulty also reflects history: Freud himself never fully developed a theory of aggression and came to consider the existence of a “primary” destructive drive only late in his

life, after witnessing the death and devastation of World War I (*Beyond the Pleasure Principle*, published in 1920) (Freud 1955).

A study in which 15 men with rigorously diagnosed DSM-III-R IED were identified from among a group of 443 men who complained of violence, some systematic observations about the “typical violent episode” as reported by patients with IED was permitted (Felthous et al. 1991). In the vast majority of 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 lose track of where they were. None lost control of urine or bowel function during the episode. Various degrees 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 afterward, eight had partial recollection, and one lost memory of the event afterward. Most IED subjects tried to help or comfort the victim afterward.

Assessment

Psychiatric Examination and History

The DSM-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 dementias, focal frontal lesions, partial complex seizures, and post-concussion 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 a general medical condition, aggressive type, and it may be made with a careful history, as well as 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 or abuse. Blood and

urine toxicology screens may reveal the use of other substances, and track marks on the forearms may suggest intravenous drug use. Partial complex seizures and focal brain lesions may be evaluated by EEG and brain imaging. In cases without a grossly abnormal neurological examination, magnetic resonance imaging may be more useful than computed tomography of the head. Magnetic resonance imaging can reveal mesiotemporal scarring, which 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

IED has been subjected to little systematic study. As formulated in DSM-5, IED is probably a rare disorder. 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 the 1980 *Diagnostic and Statistical Manual of Mental Disorders*, Third Edition (DSM-III). The DSM-III and the revised third edition (DSM-III-R) definitions of IED required the absence of signs of generalized impulsivity or aggressiveness between episodes. Episodic behavioral disorders are quite common and exist across a continuum between 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 1-7%, 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 to study current or lifetime IED in 411 psychiatric outpatients. They reported a rate of 3.8% for current IED and 6.2% for lifetime IED by DSM-IV criteria. A 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 like schizophrenia or bipolar illness. In fact, the National Comorbidity Survey Replication (NCS-R) study found that IED is much more common than previously thought. Lifetime and 12-month prevalence estimates of DSM-IV IED were 7.3% and 3.9%, with a mean of 43 lifetime attacks resulting in 1,359 dollars in property damage. IED-related injuries occurred 180 times per 100 lifetime cases (Kessler et al. 2006).

In one study of the prevalence of DSM-III-R IED among violent men, Felthous et al. (1991) found that of 443 subjects who complained of violence, only 15 (3.4%) met criteria for IED. The DSM-III-R definition of IED was more restrictive than the 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 while 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 Patterns

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 et al. (1998) and McElroy (1999) studied 27 individuals who had symptoms that met criteria for IED and reported: “Twenty-five (93%) subjects had lifetime DSM-IV-TR diagnoses of mood disorders; 13 (48%), substance use disorders; 13 (48%), anxiety disorders; 6 (22%), eating disorders; and 12 (44%), an ICD other than IED. Subjects also displayed high rates of comorbid migraine headaches. First-degree relatives displayed high rates of mood, substance use, and impulse-control disorders.” McElroy et al. (1998) reported that the aggressive episodes observed in their subjects resembled “microdysphoric” manic episodes. Symptoms in common with both manic and IED episodes 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) suggests a much lower rate of comorbid bipolar illness, with a rate of 11% (bipolar I, 5%; bipolar II, 5%; and bipolar not otherwise specified, 1%). Due to symptomatic overlap between the two conditions, the co-occurrence of IED and bipolar disorder has been obscured in many epidemiologic studies. Regardless, clinicians should fully evaluate for bipolar disorder prior to determining treatment for IED, because mood stabilizers, rather than serotonin reuptake inhibitors (SSRIs), would be the first-line treatment for IED comorbid with bipolar disorder.

McElroy et al. (1998) reported that up to 44% of their IED subjects had another impulse-control-type disorder such as compulsive buying (37%) or kleptomania (19%). However, Coccaro et al. (1998b) found that 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 syndrome may be prone to rage attacks (Budman et al. 1998, 2000). The clinical manifestation and presentation of these attacks is similar to IED and may be more common among those children with Tourette syndrome 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 80–2). Coccaro et al. (1998b) reported the rate of BPD and/or ASPD in IED subjects to be 38%. However, rates of IED in subjects with BPD have been noted at 78% and in subjects with ASPD at 58% (Coccaro et al. 1998b). A review of unpublished data from the author’s lab (Hollander E 2007, personal communication) suggests that these rates are lower among subjects not seeking treatment and are lowest in the community (23% for BPD and/or ASPD); see also Coccaro et al. 2004. Regardless, BPD and ASPD subjects 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 abuse. A concurrent diagnosis of substance intoxication excludes the diagnosis of IED. However, many patients with IED report past or family histories of substance abuse, and in particular alcohol abuse. In light of evidence linking personal and family history of alcohol abuse with impulsive

aggression (Linnoila et al. 1989) and linking both with low central serotonergic function (reviewed later), this connection may be clinically relevant. Thus, when there is evidence suggesting that alcohol abuse may be present, a systematic evaluation of IED is warranted, and vice versa.

3. Neurological disorders, especially severe head trauma, partial complex seizures, dementias, and inborn errors of metabolism. IED is not diagnosed if the aggressive episodes are a direct physiological consequence of a general medical condition. Such cases would be diagnosed as personality change due to a general medical condition, delirium, or dementia. However, individuals with IED often have nonspecific findings on neurological examination, such as reflex asymmetries 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 (American Psychiatric Association 2000), and anecdotal case reports, the onset appears to be from childhood to the early 20s, and may be abrupt and without a prodromal period. The age of onset and course of IED 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 has been described (McElroy et al.

1998). Preliminary data (Coccaro et al. 2005) confirm these findings and indicate that onset of DSM-IV-TR IED occurs by the end of the first decade in 31%, by the end of the second decade in 44%, by the end of the third decade in 19%, and by the end of the fourth decade in only 6%. The mean age at onset in the NCS-R study was 14 years (Kessler et al. 2006). 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. If provoked, it is usually from a known person and is seemingly minor in nature (McElroy et al. 1998). Many individuals frequently have minor aggressive episodes in the interim between severely aggressive/destructive episodes. Considerable distress, social, financial, occupational, or legal or impairments typically result from these episodes.

Differential Diagnosis

The differential diagnosis of IED covers 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 toward the self. The psychiatric nosology of aggression is still preliminary. Impulsivity is defined as the tendency to act in a sudden, unpremeditated, and excessively spontaneous fashion. The IED diagnosis should be considered only after all other disorders associated with impulsivity and aggression have been ruled out. Chronic impulsivity

and aggression may occur as part of a cluster B personality disorder (e.g. BPD and ASPD); during the course of substance use disorders and substance intoxication; in the setting of a general medical (usually neurological) condition; and as part of disorders first diagnosed during childhood and adolescence such as conduct disorder, oppositional defiant disorder, ADHD, and mental retardation. In addition, impulsive aggression may appear during the course of a mood disorder, especially during a manic episode, which precludes the diagnosis of IED, and during the course of an agitated depressive episode. Impulsive aggression may also be an associated feature of schizophrenia, in which it may occur in response to hallucinations or delusions. Impulsive aggression may also appear in variants of obsessive-compulsive disorder (OCD), which may present with concurrent impulsive and compulsive symptoms.

A special problem in the differential diagnosis of impulsive aggression, which may arise in forensic settings, is that it may represent purposeful behavior, which is distinguished from IED by the presence of motivation and gain in the aggressive act, such as monetary gain, vengeance, or social dominance. Another diagnostic problem in forensic settings is malingering, in which individuals may claim to have IED to avoid legal responsibility for their acts. Figure 80–2 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 80–1.

Differences in Gender and Cultural Presentations

Amok is an extremely rare culture-specific syndrome of episodic aggression first described in the Malay Peninsula but later found in Africa and Papua New Guinea. Amok is an episode of sudden, unprovoked rage in which the affected individual runs around with a weapon and attempts to kill a number of people or animals. Sometimes the perpetrator, typically a man, then

kills himself. If captured alive, the individual with amok claims no memory of the acts. The etiology of amok and its relation to IED are unclear. Episodic violent behavior is more common in males than in females (DSM-IV-R).

Etiology and Pathophysiology

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

A second line of explanation, which has yielded numerous positive findings over the past 15 years, views impulsive aggression as the result of variations, 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

and Mendlewicz 1998). Animal studies suggest that serotonergic neurons play a role in behavioral inhibition and thus provide an impetus to explore the role of serotonin in human impulsivity. Although the majority of the human studies involved patients who suffered from impulsive aggression in the context of disorders other than IED, their findings may be relevant to the behavioral dimension of impulsive aggression, of which IED is a “pure” form.

Measures examining central (and peripheral) serotonin function correlate inversely with laboratory measures of aggression, as well as with data obtained from questionnaires and that which is 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, Dolan et al. 2001, Manuck et al. 1998), and platelet measures of serotonin activity (Birmaher et al. 1990, Coccaro et al. 1996). The type of aggression associated with reduced central serotonin function appears to be impulsive, as opposed to nonimpulsive, aggression (Linnoila et al. 1983, Virkkunen et al. 1994). Linnoila et al. (1989) divided aggressive behaviors into impulsive and nonimpulsive forms and found that reduced CSF serotonin was correlated with impulsive aggression only. These findings suggest that impulsive aggressive behavior can be distinguished biologically from nonimpulsive aggression. Interestingly, the inverse relationship between aggression and serotonin is not observed when catecholamine system function is impaired (Coccaro et al. 1989, Wetzler et al. 1991). Siever et al. (1991) and Stein et al. (1993) have confirmed a relationship between levels of 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 alcoholics have further defined a probable

relationship between such behaviors and diminished serotonergic function (Virkkunen et al. 1995, Virkkunen and Linnoila 1993).

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

Another line of neurobiological evidence links impulsive aggression with dysfunction of the prefrontal cortex (PFC). Studies of neuropsychiatric patients with localized brain lesions have demonstrated that some bilateral lesions in the PFC may be specifically associated with a chronic pattern of impulsive aggressive behaviors. Neurological studies suggest that the PFC regions associated with impulsive aggression syndromes are involved in the processing of affective information and 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 non-patient 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, whose number is increased in suicide victims and correlated with aggressive social behavior in primates. Lower levels of CSF 5-HIAA were found in neurological patients with

frontal brain injuries than in patients with injuries in other brain regions. The fenfluramine challenge test, a neuroendocrine challenge to the serotonergic system, 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 suggests that such behavior may be associated with altered innate characteristics of central nervous system function (Bars et al. 2001).

Thus, biological studies implicate the serotonergic system and the 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 which have been committed.

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 and Fink 1987). McElroy et al. (1998) reported that nearly a third of first-degree relatives of IED probands also carried the diagnosis. A blinded, controlled, family history study using IED-integrated research criteria (Coccaro 1999) found a morbid risk of IED of 26% in relatives of IED-IR probands compared with 8% among the relatives of control probands, a

significant difference. Although twin studies have confirmed the hypothesis that both impulsivity (Seroczynski et al. 1999) and aggression (Coccaro et al. 1997a) are under substantial genetic influence, there are no twin studies of IED itself. Genetic influence for these two traits ranges from 28% to 47%, with nonshared environmental influences making up the lion's share of the remaining variance.

Molecular Genetic Studies

Studies of particular genes in aggressive populations have used the candidate gene approach. Candidate genes are the genes for proteins with a suspected, or proven, biological association to a disorder [e.g., serotonin (5-HT) receptors in aggression]. The polymorphism HTR1B/G861C and short tandem repeat locus D6S284 are part of the gene for the 5-HT_{1B} receptor for serotonin. These genetic sites were examined in 350 Finnish sibling pairs and 305 Southwestern American Indian sibling pairs, both with a high rate of alcoholism. The diagnoses of ASPD and IED were used to examine the traits of impulsivity and aggression. The rate of IED in relatives of ASPD probands was 15%, and the relatives of healthy control subjects had neither IED nor ASPD. Lappalainen et al. (1998) were able to discover that the gene predisposing to ASPD alcoholism resides close to the HTR1B version of the coding sequence. They concluded that impulsivity and aggression might be influenced, in part, by 5-HT_{1B} receptors. Other candidate genes include the genes for tryptophan hydroxylase and MAO-A. Manuck et al. (1999, 2000) found an association of the traits of aggression, impulsivity, and serotonin activity (tested by *D,L-fenfluramine* challenge) with variations in both the tryptophan hydroxylase and the MAO-A genes in community samples.

Imaging and Brain Localization

Few localization and functional studies have looked at impulsive aggression or IED. 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 (2007) 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. Compared with controls, patients with IED exhibited increased activation of the amygdala and reduced activation of the orbitofrontal cortex to faces expressing anger. Extent of the activation in both amygdala and orbitofrontal cortex in response to faces expressing anger were 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, Best et al.'s (2002) data supported a possible dysfunctional frontal circuit.

More work is needed to reveal the specific functional brain abnormalities in impulsive aggressive individuals.

Treatment

Given the rarity of pure IED, it is not surprising that few systematic data are available on its response to treatment and that some of the recommended treatment approaches to IED are based on treatment studies of impulsivity and aggression in the setting of other mental disorders and general medical conditions. Thus, no standard regimen for the treatment of IED can currently be 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 symptom 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 and SSRIs), and antianxiety agents (lorazepam, alprazolam, and buspirone). Mattes (1990) compared the effectiveness of two commonly used agents, carbamazepine and propranolol, for the treatment of rage outbursts in a heterogeneous group of patients. He found that although carbamazepine and propranolol were overall equally effective, carbamazepine was more effective in patients with IED and propranolol was more effective in patients with ADHD. A substantial body of evidence supports the use of propranolol—often in high doses—for impulsive aggression in patients with chronic psychotic disorders and mental retardation. Lithium has been shown to have antiaggressive properties and may be used to control temper outbursts. In patients with comorbid major depressive disorder, OCD, or cluster B

and C personality disorders, SSRIs may be useful. Overall, in the absence of more controlled clinical trials, the best approach may be to tailor the psychopharmacological agent to coexisting psychiatric comorbidity. In the absence of comorbid disorders, carbamazepine, titrated to antiepileptic blood levels, may be used empirically.

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 to first recognize and then verbalize their anger appropriately. In addition, during the course of insight-oriented psychotherapy, they are encouraged to identify and express the fantasies surrounding their rage. Group psychotherapy for temper-prone patients has also been described. The cognitive-behavioral model of psychological treatment may be usefully applied to problems with anger and rage management.

Anger treatment studies focus on treatment of anger as a component of other psychiatric illnesses, like substance abuse, post-traumatic stress disorder, depression, and domestic violence; as well as in forensic and mentally impaired populations. In a few rare cases, anger is addressed as the primary or only problem, and a limited number of treatments have been described. “Imaginational exposure therapy,” used frequently in anxiety disorders, was studied in a noncontrolled pilot study of anger treatment (Grodnitzky and Tafrate 2000). Subjects habituated to anger-provoking scenarios, and the treatment was felt to be useful. In a controlled trial of high driving anger college students, Deffenbacher et al. (2000) compared pure relaxation training with relaxation training combined with cognitive therapy and an assessment-only control. Neither

treatment condition improved general trait anger, but both treatments improved driving anger. When repeated in a new population of drivers with higher anger levels, both treatments lowered trait anger (Deffenbacher et al. 2002). Since relaxation training with cognitive therapy provided little gain over pure relaxation training, relaxation training in itself may be adequate treatment for driving anger.

Another form of behavioral therapy, dialectical behavior therapy (DBT), is widely used, and has been studied in patients with BPD. One study showed improvement in anger, global functioning, and social adjustment when compared to treatment-as-usual (Linehan et al. 1994). Improvement in anger and impulsivity has been shown with DBT across many disorders. While there are no published double-blind, placebo-controlled studies on IED subjects in therapy, there are studies that are ongoing.

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 a year. They were brief and apparently unpredictable and resulted in his separation from his first wife. Except during those episodes, Mr. A was a pleasant, rather timid

man who deferred to his wife in most important decisions. There was no history suggestive of antisocial or borderline personality disorder. Mr. A, who described himself as a shy, withdrawn child, gave a history of head trauma at the age of 12 years, while he was ice skating, with loss of consciousness for 10 minutes. Other than this, his medical history was normal. There were no neurological or behavioral sequelae. Mr. A also described prolonged physical abuse by his alcoholic father. Mr. A himself denied a history of substance abuse, involvement with the criminal justice system, and prior psychiatric treatment. He denied a history of manic and depressive episodes. Mr. A had few friends and was not popular at his job. Although he had never lost his temper there, he believed that his boss and coworkers could sense his “stress” while dealing with clients.

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

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

Kleptomania

Diagnosis

Definition and Diagnostic Features

Kleptomania shares with 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 (see box below for criteria). Criterion A, which focuses on the senselessness of the items stolen, has often been considered the criterion that distinguishes kleptomania patients from ordinary shoplifters (Goldman 1991), but interpretation of this criterion is controversial. The archetype of the middle-aged female kleptomania patient who steals peculiar items may not adequately account for all people with kleptomania (Goldman 1991, McElroy et al. 1991a). Patients with kleptomania may in fact desire the items they steal and be able to use them, but do not need them. This may be particularly the case with kleptomania patients who hoard items (Goldman 1991), for which

multiple versions of the same item are usually not needed, but the item itself may be desired and may be of practical use to the patient.

People with kleptomania often report amnesia surrounding the shoplifting act (Goldman 1991, Grant 2004), and deny feelings of tension or arousal prior to shoplifting and feelings of pleasure or relief after the thefts. They often recall entering and leaving a store but have no memory of events in the store, including the theft (Grant 2004). Others, who are not amnesic for the thefts, describe shoplifting as “automatic” or “a habit,” and may also deny feelings of tension prior to a theft or pleasure after the act (DSM-5 criterion B or C), although they report an inability to control their shoplifting (criterion A). Some report that they felt tension and pleasure when they started stealing, but it became a “habit” over time. Some speculate that patients who are amnesic for shoplifting or who do so “out of habit” represent two different subtypes of kleptomania.

At presentation, the typical patient suffering from kleptomania is a 35-year-old woman who has been stealing for about 15 years and may not mention kleptomania as the presenting complaint or in the initial history (Goldman 1991, McElroy et al. 1991a). The patient may complain instead of anxiety, depression, lability, dysphoria, or manifestations of character pathology. There is often a history of a tumultuous childhood and poor parenting, and in addition acute stressors may be present, such as marital or sexual conflicts. The patient experiences the urge to steal as irresistible, and the thefts are commonly associated with a thrill, a high, a sense of relief, or gratification. Generally, the behavior has been hard to control and has often gone undetected by others. The kleptomania may be restricted to specific settings or types of objects, and the patient may or may not be able to describe rationales for these preferences. Quite often, the objects taken are of inherently little financial value, or have meaningless financial value

relative to the income of the person who has taken the object. Additionally, the object may never actually be used. These factors often help distinguish criminal theft from kleptomania. The theft is followed by feelings of guilt or shame and, sometimes, attempts at atonement. The frequency of stealing episodes may greatly fluctuate in concordance with the degree of depression, anxiety, or stress. There may be periods of complete abstinence. The patient may have a past history of psychiatric treatments including hospitalizations or of arrests and convictions, whose impact on future kleptomaniac behavior can be variable.

DSM-5 Diagnostic Criteria

Kleptomania - 312.32 (F63.3)

- | |
|---|
| <ul style="list-style-type: none">A. Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value.B. Increasing sense of tension immediately before committing the theft.C. Pleasure, gratification, or relief at the time of committing the theft.D. The stealing is not committed to express anger or vengeance and is not in response to a delusion or a hallucination.E. The stealing is not better explained by conduct disorder, a manic episode, or antisocial personality disorder. |
|---|

Assessment

Generally, the diagnosis of kleptomania is not a complicated one to make. However, kleptomania may frequently go undetected because the patient may not mention it spontaneously and the psychiatrist may fail to inquire about it as part of the routine history. The index of suspicion should rise in the presence of commonly associated symptoms such as chronic depression, other impulsive or compulsive behaviors, tumultuous backgrounds, or unexplained legal troubles. It

could convincingly be argued that a cursory review of compulsivity and impulsivity, citing multiple examples for the patient, should be a part of any thorough and complete psychiatric evaluation. In addition, it is important to do a careful differential diagnosis and pay attention to the various exclusion criteria before diagnosing theft as kleptomania. Possible diagnoses of sociopathy, mania, or psychosis should be carefully considered. In this regard, the psychiatrist must inquire about the affective state of the patient during the episodes, the presence of delusions or hallucinations associated with the occurrence of the behavior, the motivation behind the stealing, and the fate and subsequent use of the objects.

Although the typical patient may be a 35-year-old woman, it is important to remember that men, children, and elderly persons may present with or engage in kleptomania. Interestingly, Goldman (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, dementia, 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 and Martin 1977, Mendez 1988, Wood and Garralda 1990, Coid 1984, McIntyre and Emsley 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 criteria for the disorder (Odlaug and 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, like bulimia nervosa. Such speculations suggest at least a 0.6% prevalence of kleptomania in the general population (Goldman 1991). However, given that people who shoplift are often not caught, this is almost certainly an underestimate. Also, the shame and embarrassment associated with stealing prevents most people from voluntarily reporting kleptomania symptoms (Grant and 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 recent 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%

(Grant and Kim 2003, Specker et al. 1995). A study of bulimia patients found that 24% met DSM-III (American Psychiatric Association 1980) criteria for kleptomania (Hudson et al. 1983).

The literature clearly suggests that the majority of patients with kleptomania are women (e.g. Grant and Kim 2002b, McElroy et al. 1991b, Presta et al. 2002). In a retrospective review of 56 cases that appeared to fulfill DSM-III-R criteria for kleptomania, McElroy et al. (1991b) found that 77% were women. Similarly, in a prospective series of 20 patients with DSM-III-R kleptomania, 75% were women (McElroy et al. 1991a). 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 it is 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 while sending male shoplifters to prison (Grant 2006, Goldman 1991). 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 and Kim 2002b).

Comorbidity Patterns

High rates of other psychiatric disorders found in patients with kleptomania have sparked debate over the proper characterization of this disorder. Among those with kleptomania who present for treatment, there is a high incidence of comorbid mood, anxiety, and eating disorders, when compared with rates in the general population. Rates of lifetime comorbid affective disorders range from 59% (Grant and Kim 2002b) to 100% (McElroy et al. 1991b). The rate of lifetime comorbid bipolar disorder has been reported as ranging from 9% (Grant and Kim 2002b) to 27% (Bayle et al. 2003) to 60% (McElroy et al. 1991b). Studies have also found high lifetime rates of

comorbid anxiety disorders (60–80%; McElroy et al. 1991b, 1992), other ICDs (20–46%; Grant and Kim 2003), substance use disorders (23–50%; Grant and Kim 2002b, McElroy et al. 1991b), and eating disorders (60%; McElroy et al. 1991b). Personality disorders have been found in 43–55% of 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 8 patients (31%), and bulimia nervosa in 3 patients (12%). Similar percentages are noted by McElroy et al. (1991b) in a review of 56 patients with probable kleptomania: 57% with mood disorder symptoms, 34% with anxiety disorder symptoms, and 11% with bulimic symptoms. Comorbidity patterns among those who present for treatment may be greater than among random samples. More reliable comorbidity rates can be found in a prospective study of 20 kleptomaniacs (McElroy et al. 1991a). Lifetime DSM-III-R comorbidity rates were 40% major depressive disorder, 50% substance abuse, 40% panic disorder, 40% social phobia, 45% OCD, 30% anorexia nervosa, 60% bulimia nervosa, and 40% other ICDs. Dissociative symptoms, significant character pathology, and trauma histories are commonly encountered among this group (Goldman 1991, McElroy et al. 1991b).

Course

While 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, Grant and Kim 2002b, McElroy et al. 1991a, 1991b; Presta et al. 2002). In two separate studies, the mean age at onset was 20 years (Goldman 1991, McElroy et al. 1991a), and included people who had begun stealing as early as 5 to 7 years old. Onset beyond the age of 50 is unusual, and in some of these cases remote histories of past kleptomania

can be elicited (Goldman 1991). Most clinical samples of kleptomaniacs report shoplifting for more than 10 years prior to entering treatment (Goldman 1991, Grant and Kim 2002c, McElroy et al. 1991b) and in some cases 15 or 16 years may elapse before treatment is sought (Goldman 1991, McElroy et al. 1991a).

The disorder appears to be chronic, but with varying intensity. At peak frequency, McElroy et al. (1991a) found a mean of 27 episodes a month, essentially daily stealing, with one patient reporting four acts daily. The majority of patients may eventually be apprehended for stealing once or more, and a minority may even be imprisoned. Usually these repercussions do not result in more than a temporary remission of the behavior. People with kleptomania may also have extensive histories of psychiatric treatments, including hospitalization for other conditions, most commonly depression or eating disorders. Because of the unavailability of longitudinal studies, the prognosis is unknown. However, it appears that without treatment the behavior may be likely to persist for decades, sometimes with significant associated morbidity, despite multiple 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, American Psychiatric Association 2013).

Differential Diagnosis

The vast majority of 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 (American Psychiatric Association, 2013). 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 which should be distinguished from kleptomania include antisocial personality disorder and conduct disorder, as well as 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 like depression, panic disorder, OCD, and bulimia nervosa (McElroy et al. 1992, 1991b, Hudson and Pope 1990). The apparent high comorbidity of kleptomania with depression and bulimia nervosa has already been noted. As early as 1911, Janet (1911) recognized the alleviation of depressive symptoms on the commission of kleptomaniac acts. Some individuals with kleptomania respond to treatment with thymoleptic agents or electroconvulsive therapy. These observations are cited as support for an affective spectrum model.

Although the affective spectrum has been claimed to encompass obsessive-compulsive pathology (Hudson and Pope 1990) there exists a more specific model conceptualizing kleptomania and other impulse disorders as obsessive-compulsive spectrum disorders (McElroy et al. 1993). Several lines of evidence support this model. First, there are phenomenological similarities between the classical obsessions and compulsions of OCD and the irresistible impulses and repetitive actions characteristic of kleptomania. Further, there appears to be a greater than chance occurrence of OCD in probands with kleptomania and in their relatives. 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. While 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 and Kim 2002b). Yet another parallel supporting this model 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 and Kim 2002c).

Biological Theories

Serotonin and Inhibition

Compared with controls, kleptomania patients report significant elevations of impulsivity and risk taking (Bailey et al. 2003, Grant and Kim 2002d), and diminished inhibitory mechanisms may underlie the risk-taking behavior of kleptomania. The most well-studied inhibitory pathways involve serotonin and the 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 are few biological studies of kleptomania, early evidence supports a theory of serotonergic involvement in the disorder. One study found a lower number of the platelet serotonin transporter in kleptomania patients versus healthy controls (Marazziti et al. 2000). Pharmacological case studies suggest that SSRIs, and TCAs such as clomipramine (Lepkifker et al. 1999, McElroy et al. 1991b) may reduce the impulsive behavior associated with kleptomania.

Dopamine and Reward Deficiency

Dopaminergic systems influencing rewarding and reinforcing behaviors have also been implicated in ICDs and may play a role in the pathogenesis of kleptomania. One proposed mechanism is “Reward Deficiency Syndrome,” a hypothesized hypodopaminergic state involving multiple genes and environmental stimuli that puts an individual at high risk for multiple addictive impulsive and compulsive behaviors (Blum et al. 2000). Alterations in dopaminergic pathways have been proposed as underlying the seeking of rewards (e.g., shoplifting) that trigger the release of dopamine and produce feelings of pleasure (Blum et al. 2000). Further, dopamine release into the nucleus accumbens has been implicated in the translation of motivated drive into action, serving as a “go” signal (Chambers et al. 2003). Dopamine release into the nucleus accumbens seems maximal when reward probability is most uncertain, suggesting that it plays a central role in guiding behavior during risk-taking situations (Fiorillo et al. 2003). The structure and function of dopamine neurons within the nucleus accumbens, in conjunction with glutamatergic afferent and intrinsic GABAergic activities, appear to change in response to experiences that influence the function of the nucleus accumbens. 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-methyl-transferase (COMT) isoenzymes with differential enzymatic activity have shown that the higher cortical dopamine levels associated with reduced COMT activity results in improved frontal cortical cognitive performance (Malhorta 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, that result in theft twice weekly on average (Grant and Kim 2002b). Thus, urges linked to the experience of reward and pleasure may represent an important clinical target for treatment. Many indicate that the act of stealing reduces the urges or the tension these urges produce (McElroy et al. 1991b). While many report the urges as intrusive, the act of stealing is often a “thrill” for some, producing a pleasurable feeling (Goldman 1991, Grant and Kim 2002b). The μ -opioid system is thought to underlie urge regulation by processing

reward, pleasure, and pain, in part through modulation of dopamine neurons in the mesolimbic pathway via γ -aminobutyric acid interneurons (Potenza and Hollander 2002). Studies of naltrexone, a μ -opioid antagonist, have shown its efficacy in reducing urges in those with kleptomania and other ICDs (Dannon et al. 1999, Grant and Kim 2002c, Kim et al. 2001).

In sum, repeated kleptomaniac 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 kleptomaniac episodes were closely related to depressive bouts and who experienced an apparent antidepressant effect from the thrill and excitement of her risk-taking behavior. So kleptomania may result from an attempt to relieve feelings of depression through stimulation (Goldman 1991, McElroy et al. 1991a) and risk-taking behavior may produce an antidepressant effect for some patients (Fishbain 1987, Goldman 1991). Shoplifting may distract depressed patients from stressors and unpleasant cognitions. Ironically, problems resulting directly from shoplifting (e.g., embarrassment and shame from getting caught) may in turn lead to even more shoplifting as a misguided attempt of symptom management (Goldman 1991). Supporting the self-medication hypothesis of shoplifting, patients with kleptomania report high lifetime rates of depression (45-100%; Bayle

et al. 2003, McElroy et al. 1991b) that usually (60% of cases) precedes the kleptomaniac behavior (McElroy et al. 1991b). Further, several case studies report patients who described shoplifting as relief for their depressed moods (Fishbain 1987) and suggest that kleptomania symptoms improve with antidepressants (Lepkifker et al. 1999, McElroy et al. 1991b).

From a psychodynamic point of view, kleptomania has been viewed over the decades as a manifestation of a variety of unconscious conflicts, with sexual conflicts figuring prominently in the literature. Case reports have described conscious sexual gratification, sometimes accompanied by frank masturbation or orgasm during kleptomaniac acts (Fishbain 1987, Fenichel 1945). 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 (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 explore 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 (i.e., the removal of a punishing stimulus) hypothesizes that shoplifting is performed to experience relief from the aversive arousal of urges. The self-medication theory of kleptomania may represent a negative reinforcement. This could explain why kleptomaniac behavior continues despite the offender being frequently apprehended.

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

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

Psychiatrist-Patient Relationship

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

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

Somatic Treatments

No medication is currently approved by the US Food and Drug Administration for treating kleptomania. So, it is important to inform patients of “off-label” uses of medications for this disorder and the empirical basis for considering medication treatment.

Various medications, including tricyclic antidepressants, SSRIs (Lepkifker et al. 1999), mood stabilizers, and opioid antagonists have been examined for the treatment of kleptomania (Grant and Kim 2002c, McElroy et al. 1989) with mixed results. In a literature review of 56 kleptomania cases, McElroy et al. (1991a) noted that somatic treatments were described for 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 had partial or complete remission of both kleptomaniac urges and behavior. It has been suspected that kleptomania may respond selectively to SSRIs because of the anticomulsive 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 reinstated (McElroy et al. 1991a, Fishbain 1987). But 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. These include antipsychotics (McElroy et al. 1991b, Fishbain 1987), stimulants (McElroy et al. 1991b), valproic acid (McElroy et al. 1991a), carbamazepine (McElroy et al. 1991a), clonazepam (McElroy et al. 1991a) and lithium (McElroy et al. 1991a, Monopolis and Lion 1983). Lithium augmentation may be of benefit when kleptomania does not respond to an antidepressant alone (Burstein 1992). Other agents used successfully as monotherapy for kleptomania include fluvoxamine (Chong and Low 1996) and paroxetine (Kraus 1999). Combinations of medications have also been effective in case reports: lithium plus fluoxetine (Burstein 1992), fluvoxamine plus buspirone (Durst et al. 1997), fluoxetine plus lithium, fluoxetine plus imipramine (McElroy et al. 1991b), and fluvoxamine plus valproate (Kmetz et al. 1997). Finally, there have been some

reports of successful treatment of kleptomania with electroconvulsive therapy, which may have been administered for a concurrent mood disorder (McElroy et al. 1991b).

The findings from case reports have not been consistent. Seven cases of fluoxetine, three of imipramine, two of lithium as monotherapy, two of lithium augmentation, four of tranylcypromine, and one of carbamazepine combined with clomipramine all failed to reduce kleptomania symptoms (McElroy et al. 1991b). Some evidence suggests that SSRIs may even induce kleptomania symptoms (Kindler et al. 1997). One case series found that kleptomania symptoms responded to topiramate (Dannon 2003), while 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 evaluating the safety and efficacy of oral naltrexone, Grant, et al. (2009) randomized 25 subjects with DSM-IV kleptomania to naltrexone (dosing ranging from 50 mg/day 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 and Kim 2002c). A lower dosage, possibly 50 mg/day, may be effective in younger people with kleptomania (Grant and Kim 2002a). Opioid antagonists like naltrexone may be effective in reducing both the urges to shoplift and shoplifting behavior 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 and 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 fourteen 15-minute sessions over five days, two patients reported complete

remission of symptoms for a two-year period (McConaghy and Blaszczynski 1988). A woman who was treated weekly for five months, and reported two 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. Insight-oriented psychotherapy used for the treatment of Kleptomania however has been unsuccessful in 11 published cases (McElroy et al. 1991b). Psychoanalysis has resulted in some limited success for kleptomania symptoms, but usually with the addition of medication (Fishbain 1988, Schwartz 1992). The psychodynamic treatment of kleptomania centers on the exploration and working through of the underlying conflict or conflicts. In a review of 26 case reports, McElroy et al. (1991a) reported that 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, like depression, bulimia nervosa, OCD, or substance abuse, must be addressed along with the kleptomania. In addition to the inherent suffering and morbidity of these other disorders, their course and severity could compound the kleptomaniac behavior. In the rare cases of a precipitating or exacerbating organic etiology, the underlying organic cause must be treated. In addition, the treatment of particular groups such as children or the elderly should take into account special contributing life stage or situational factors. The involvement of family or others on whom the patient is dependent may be indicated.

Refractoriness to Initial Treatment

There has not been sufficient study of the treatment of kleptomania to systematically delineate approaches to the refractory patient. However, general clinical principles can be applied. Medication trials should be maximized, predominantly employing antidepressants and mood stabilizers, alone or in combination. In addition, it is important that comorbid conditions such as depression or OCD be monitored and treated, because they complicate the course of kleptomania. For patients who have no response or a partial response to pharmacotherapy alone, or 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.

Pyromania and Fire Setting Behavior

Diagnosis

Definition and Diagnostic Features

Pyromania is characterized by deliberate and purposeful fire setting on more than one occasion (American Psychiatric Association, 2013). Other defining features of pyromania include the experience of tension or affective arousal before the fire setting, an attraction or fascination with fire and its contexts, and a feeling of gratification or relief associated with the fire setting or when witnessing or participating in its aftermath. True pyromania is present in only a small subset of fire setters. Prins et al. (1985) have suggested the following motivations for intentional arson: financial reward, to conceal another crime, for political purposes, as a means of revenge, as 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 and 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 of 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 and Tierney 1999), pyromania is still considered an uncontrolled and often impulsive behavior.

DSM-5 Diagnostic Criteria

Pyromania - 312.33 (F63.1)

- | |
|--|
| <ul style="list-style-type: none">A. Deliberate and purposeful fire setting on more than one occasion.B. Tension or affective arousal before the act.C. Fascination with, interest in, curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences).D. Pleasure, gratification, or relief when setting fires, or when witnessing or participating in their aftermath.E. The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one’s living circumstances, in response to a delusion or hallucination, or as a result of impaired judgment (e.g., in major neurocognitive disorder, intellectual disability [intellectual developmental disorder], substance intoxication).F. The fire setting is not better 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, as well as the feeling of tension relief or pleasure in witnessing the outcome. This is useful in distinguishing between pyromania and fire setting elicited by other motives (i.e., financial gain, concealment of other crimes, political, arson related to other mental illness, revenge, attention seeking, erotic pleasure, a component of conduct disorder). In children and adolescents, the most common elements are excitation caused by fires, enjoyment produced by fires, relief of frustration by fire setting, and expression of anger through fire setting (Brandford and Dimock 1986). The most frequent motives for arson by juveniles are revenge on parents or other authorities, the search for heroism or excitement, self-destructiveness, the craving for sensation, and an expression of outrage (RŠsŠnen 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 and Nelson 2001). Thus, pyromania could be a displacement of aggression in people with a history of sexual trauma. The channeling of aggression by their fire setting may be an attempt to influence their environment and improve their self-esteem where other means have failed. Fire setting may also be an attempt at communication by individuals with few social skills (Geller and Bertsch 1985).

Pyromania onset has been reported to occur as early as the age of three, though it may present initially present in adulthood. Because of the legal implications of fire setting, individuals may not admit previous events, which may result in biased perceptions of the common age at onset. Men greatly outnumber women with the disorder. Further, nongeriatric

state hospital in-patients who had engaged in fire setting behavior were significantly more likely to have a history of nonlethal self-injurious behavior and had a significantly greater number of admissions to the state hospital (Geller and Bertsch 1985).

Assessment

The Psychiatric Interview

The interviewer must bear in mind that the circumstances of arson, whatever the motive, may pose legal and criminal problems for the individual, 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. Thus, 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.

While 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 and 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 Lewis and Yarnell (1951) data that only 14.8% of those with pyromania are female.

Fire setting for profit or revenge, or secondary to delusions or hallucinations, is more frequent than “authentic” pyromania. Although pyromania, by definition, 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%), as well as 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 and 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. Thirty-nine percent of the fire setters from the study received the diagnosis of pyromania. Of those, 22% had borderline to dull normal intelligence, and 13% had between dull and low average intelligence.

Fire setters were also described as driven by an irresistible impulse to set fires. The findings suggested that the peak incidence of fire setting was between the ages of 16 and 18 years, though this observation has not been confirmed by more recent studies. Pyromania is found in adolescents and is also present at any age. Among females, the diversity of ages is particularly apparent (Barker 1994). Also, neither confirmed, nor reproduced in more 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 Patterns

Limited data are available regarding individuals with pyromania. Reported data of comorbid diagnoses are generally derived from forensic samples and do not distinguish between criminally motivated fire setters and impulsive fire setters. Fire setting in subjects who do not have pyromania appears frequently and is often under-recognized. While 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. Thirty-six percent had schizophrenia or bipolar disorder, and 64% were misusing alcohol or drugs at the time of their fire setting.

Repo et al. (1997) examined the medical and criminal records of 282 arsonists in order to compare first time and repeat offenders. They found that alcohol dependence and ASPD were

common among recidivist offenders, especially among those who committed violent crimes. Recidivist offenders commonly had a history of enuresis during their childhood, were younger than first time offenders at the time of their first offence, and were more often intoxicated with alcohol during the arson attempt. Psychosis was common among those with no record of recidivist criminal offences. Puri et al. (1995) examined a group of 36 forensically referred fire setters. They found that about one-third had no other evidence of mental illness, about a quarter were female, psychoactive substance abuse was common, and 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 met criteria for ICDs: 18 had IED, 3 had pathological gambling, 4 had kleptomania, 3 had pyromania, and 3 had trichotillomania. Patients with pyromania had a higher number of previous depressions (3.3 versus 1.3, $p = 0.01$). Bipolar disorders were more frequent in the ICD group than in the group without ICDs (19% versus 1.3%, $p = 0.002$).

Laubichler et al. (1996) compared the files of 103 criminal fire setters and subjects with pyromania. Subjects with pyromania were younger (average age 20 years) than criminal fire setters (average age 30 years). Seventy (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. Rššnen et al. (1995) found that young arsonists frequently have problems with alcohol: 82% had alcoholism and 82% were intoxicated at the time of committing the crime. The excessive consumption of alcohol had a close connection with the arson committed. Lejoyeux et al. (1999) searched for ICDs among consecutive admissions for detoxification of alcohol-

dependent patients in a French department of psychiatry. Among the 79 patients included in the study, they found that 30 (38%) met criteria for one ICD (19 with IED, 7 with pathological gambling, 3 with kleptomania, and 1 case of 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. While 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 and Lion 1977) to 28% (Lewis and Yarnell 1951). In a cross-sectional and 10-year follow-up study, Barnett et al. (1997, 1999) compared mentally ill and mentally “healthy” fire setters from trial records in Germany where a defendant with a psychiatric disorder can be found to be not responsible, to have diminished responsibility, or to be fully responsible. 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 like theft, traffic violations, and alcohol-related offenses, had a higher rate of recurrence, and committed fewer common offenses other than fire setting. Among all arsonists who committed crimes in addition to arson, those who were found to be partly responsible for

their arson committed the highest number of offenses followed by those who were deemed not responsible for their actions and those who were fully responsible.

Differential Diagnosis

Other causes of fire setting must be ruled out. Fire setting behavior may be motivated by circumstances unrelated to mental disorders. Such motivations include profit, crime concealment, revenge, vandalism, and political statement or action (Geller 1987, Lowenstein 1989). Furthermore, fire setting may be 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 these individuals, 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-Hydroxyindoleacetic acid (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 (like 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 (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), as well as 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

alcohol dependence was increased in those who were violent offenders, as well as in the fathers of these subjects.

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 two and three years and was almost universal in a study of normal schoolboys at the ages of six, eight, and ten 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 of pyromania were more often involved in fire setting, threatening to set a fire, sounding a false fire alarm, or calling the fire department with a false report of fire than were control subjects. Thus, there may be a continuum between excessive interest in fire and “pure” pyromania.

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

Treatment

Treatment Goals

Because of the danger inherent in fire setting behavior, the primary goal is elimination of the behavior 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 be frequently embedded in other psychiatric illness, therapeutic attention may be directed primarily to the underlying disorder. However, the dangerous nature of fire setting requires that the behavior be controlled. Much in the same fashion that one would seek to educate impaired patients about the functional risks associated with their symptoms—and to establish boundaries of acceptable behavior—the fire setting behavior must be directly addressed, even if it is not a core symptom of the associated disorder.

Psychosocial Treatments

Treatment for fire setters is problematic because they frequently refuse to take responsibility for their acts, are in denial, have alcoholism, and lack insight (Mavromatis and Lion 1977). It has been estimated that up to 60% of childhood fire setting is motivated by curiosity. Such behavior often responds to direct educational efforts. In children and adolescents, focus on interpersonal problems in the family and clarification of events preceding the behavior may help to control the behavior (Lowenstein 1989). Principles of 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 like aversion therapy have helped fire setters (McGrath and Marshall 1979, Koles and 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. (2002b) confirmed the positive effect of a prevention program for pyromania. In 1999, they developed the Trauma Burn Outreach Prevention Program. All subjects

arrested and convicted after setting a fire attended a one day informational program. The program's interactive content focused on the medical, financial, legal, and societal impact of fire setting behavior. The rate of recidivism was less than 1% in the group who attended the program, versus 36% in the control group.

Clinical Vignette 2

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

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

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

In the past three 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/ICD-10-CM Diagnostic Criteria

Unlike its predecessors, ICD-10-CM (2014) contains a specific diagnosis code (F63.81) for IED, and describes the disorder in a manner reminiscent to that of criteria used in DSM-IV (i.e., prior to the changes made in DSM-5). A disorder characterized by recurrent episodes of serious assaultive acts or destruction of property due to a failure to resist aggressive impulses; the degree of aggression during these episodes is grossly out of proportion to any psychosocial provocation. The aggressive episodes are not etiologically linked to another mental disorder, a general medical condition, or substance use. Additional descriptors are available, including explosive disorder, intermittent; explosive disorder, isolated; and isolated explosive disorder.

The ICD-10-CM (2014) diagnostic criteria for pyromania (63.1) and kleptomania (F63.2) are essentially equivalent to their DSM-5 counterparts. Both including exclusion criteria, the ICD-10-CM diagnosis for pyromania lists fire-setting in/by ASPD, alcohol or psychoactive substance intoxication, conduct disorder, mental disorders due to known physiological conditions (i.e., medical conditions), and schizophrenia all as Type 2 excludes, denoting that the condition excluded is not part of the condition represented by the code (pyromania), but a patient may have both conditions at the same time. The ICD-10-CM diagnostic code for kleptomania also contains Type 2 excludes, including depressive disorder with stealing, stealing due to underlying mental condition-code to mental condition, and stealing in mental disorders due to known physiological condition. In addition, the code for kleptomania contains a Type 1, or “pure” exclude for shoplifting as the reason for observation for suspected mental disorder. This refers to, and indicates that the code excluded (shoplifting) should never be used at the same time as the code being defined (kleptomania).

References

- American Psychiatric Association (1980) Diagnostic and Statistical Manual of Mental Disorders, 3rd edition. APA, Washington, DC, USA.
- American Psychiatric Association (1987) Diagnostic and Statistical Manual of Mental Disorders, 3rd edition Revised. APA, Washington, DC, USA.
- American Psychiatric Association (2000) Diagnostic and Statistical Manual of Mental Disorders, 4th edition. Text Rev. APA, Washington, DC, USA.
- American Psychiatric Association (2013) Diagnostic and Statistical Manual of Mental Disorders, 5th edition. APA. Washington, DC, USA
- Barker AF (1994) Arson: A Review of the Psychiatric Literature. Institute of Psychiatry, Maudsley Monographs No 35. Oxford University Press, Oxford, UK.
- Barnett W, Richter P, and Renneberg B (1999) Repeated arson: Data from criminal records. *Forensic Science International* 101, 49–54.
- Barnett W, Richter P, Sigmund D, et al. (1997) Recidivism and concomitant criminality in pathological firesetters. *Journal of Forensic Science* 42, 879–883.
- Bars DR, Heyrend FL, Simpson CD, et al. (2001) Use of visual evoked potential studies and EEG data to classify aggressive, explosive behavior of youths. *Psychiatric Services* 52, 81–86.
- Bayle FJ, Caci H, Millet B, et al. (2003) Psychopathology and comorbidity of psychiatric disorders in patients with kleptomania. *American Journal of Psychiatry* 160, 1509–1513.
- Bechara A (2003) Risky business: Emotion, decision-making, and addiction. *Journal of Gambling Studies* 19, 23–51.
- Beldoch M (1991) Stolen objects as transitional objects. *American Journal of Psychiatry* 148, 1754.

- Best M, Williams JM, and Coccaro EF (2002) Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. *Proceedings of the National Academy of Sciences USA* 99, 8448–8453.
- Birmaher B, Stanley M, Greenhill L, et al. (1990) Platelet imipramine binding in children and adolescents with impulsive behavior. *Journal of the American Academy of Child & Adolescent Psychiatry* 29, 914–918.
- Blum K, Braverman ER, Holder JM, et al. (2000) Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors. *Journal of Psychoactive Drugs* 32 (suppl), 1–68.
- Brandford J and Dimock J (1986) A comparative study of adolescents and adults who willfully set fires. *Psychiatric Journal of University of Ottawa* 11, 228–234.
- Budman CL, Bruun RD, Park KS, et al. (2000) Explosive outbursts in children with Tourette’s disorder. *Journal of the American Academy of Child & Adolescent Psychiatry* 39, 1270–1276.
- Budman CL, Bruun RD, Park KS, et al. (1998) Rage attacks in children and adolescents with Tourette’s disorder: A pilot study. *Journal of Clinical Psychiatry* 59, 576–580.
- Bumpass ER, Fagelman FD, and Brix RJ (1983) Intervention with children who set fires. *American Journal of Psychotherapy* 37, 328–345.
- Burstein A (1992) Fluoxetine-lithium treatment for kleptomania. *Journal of Clinical Psychiatry* 53, 28–129.
- Chambers RA, Taylor JR, and Potenza MN (2003) Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *American Journal of Psychiatry* 160, 1041–1052.
- Chiswick D (1976) Shoplifting, depression and an unusual intracranial lesion (a case report). *Medicine, Science and the Law* 16, 266–268.
- Chong SA and Low BL (1996) Treatment of kleptomania with fluvoxamine. *Acta Psychiatrica Scandinavica* 93, 314–315.

- Coccaro EF (1999) Family history study of intermittent explosive disorder. Paper presented at the American Psychiatric Association 152nd Annual Meeting, Washington DC.
- Coccaro EF, Bergeman CS, Kavoussi RJ, et al. (1997a) Heritability of aggression and irritability: a twin study of the Buss-Durkee Aggression Scales in adult male subjects. *Biol Psychiatry* 41, 273–284.
- Coccaro EF, Kavoussi RJ, Berman ME, et al. (1998b) Intermittent explosive disorder-revised: development, reliability and validity of research criteria. *Compr Psychiatry* 39, 368–376.
- Coccaro EF, Kavoussi RJ, and Hauger RL (1997b) Serotonin function and anti-aggressive responses to fluoxetine: a pilot study. *Biol Psychiatry* 42, 546–552.
- Coccaro EF, Kavoussi RJ, Hauger RL, et al. (1998a) Cerebrospinal fluid vasopressin: Correlates with aggression and serotonin function in personality disordered subjects. *Archives of General Psychiatry* 55, 708–714.
- Coccaro EF, Kavoussi RJ, Sheline YI, et al. (1996) Impulsive aggression in personality disorder correlates with tritiated paroxetine binding in the platelet. *Archives of General Psychiatry* 53, 531–536.
- Coccaro EF, McCloskey MS, Fitzgerald DA, Phan KL (2007) Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry* 62, 168-178.
- Coccaro EF, Lawrence T, Trestman R, et al. (1991) Growth hormone responses to intravenous clonidine challenge correlate with behavioral irritability in psychiatric patients and healthy volunteers. *Psychiatry Res* 39, 129–139.
- Coccaro EF, Posternak MA, and Zimmerman M (2005) Prevalence and features of intermittent explosive disorder in a clinical setting. *Journal of Clinical Psychiatry* 66, 1221–1227.
- Coccaro EF, Schmidt CA, Samuels JF, et al. (2004) Lifetime and 1-month prevalence rates of intermittent explosive disorder in a community sample. *Journal of Clinical Psychiatry* 65, 820–824.
- Coccaro EF, Siever LJ, Klar HM, et al. (1989) Serotonergic studies in patients with affective and personality disorders: Correlates with suicidal and impulsive-aggressive behavior. *Archives of General Psychiatry* 46, 587–599.

- Coid J (1984) Relief of diazepam-withdrawal syndrome by shoplifting. *British Journal of Psychiatry* 145, 552–554.
- Dannon PN (2003) Topiramate for the treatment of kleptomania: A case series and review of the literature. *Clinical Neuropharmacology* 26, 1–4.
- Dannon PN, Iancu I, and Grunhaus L (1999) Naltrexone treatment in kleptomaniac patients. *Human Psychopharmacology* 14, 583–585.
- Deffenbacher JL, Filetti LB, Lynch RS, et al. (2002) Cognitive-behavioral treatment of high anger drivers. *Behavior Research and Therapy* 40, 895–910.
- Deffenbacher JL, Lynch RS, Oetting ER, et al. (2000) Characteristics and treatment of high-anger drivers. *Journal of Counseling Psychology* 47, 5–17.
- DeJong J, Virkkunen M, and Linnoila M (1992) Factors associated with recidivism in a criminal population. *Journal of Nervous and Mental Disease* 180, 543–550.
- DePue RA, Luciana M, Arbisi P, et al. (1994) Dopamine and the structure of personality: Relation of agonist-induced dopamine activity to positive emotionality. *Journal of Personality and Social Psychology* 67, 485–498.
- Dolan M, Anderson IM, and Deakin JF (2001) Relationship between 5-HT function and impulsivity and aggression in males offenders with personality disorders. *British Journal of Psychiatry* 178, 352–359.
- Durst R, Katz G, and Knobler H (1997) Buspirone augmentation of fluvoxamine in the treatment of kleptomania. *Journal of Nervous and Mental Disease* 185, 586–588.
- Felthous AR, Bryant SG, Wingerter CB, et al. (1991) The diagnosis of intermittent explosive disorder in violent men. *Bulletin of the American Academy of Psychiatry and the Law* 19, 71–79.
- Fenichel O (1945) *The Psychoanalytic Theory of Neurosis*. WW Norton, New York, USA.
- Fiorillo CD, Tobler PN, and Schultz W (2003) Discrete coding of reward probability and uncertainty by dopamine neurons. *Science* 299, 1898–1902.

- First M and Frances A (eds) (1995) *DSM-IV Handbook of Differential Diagnosis*. American Psychiatric Press, Washington, DC, USA, pp 196–197.
- Fishbain DA (1987) Kleptomania as risk-taking behavior in response to depression. *American Journal of Psychotherapy* 41, 598–603.
- Fishbain DA (1988) Kleptomaniac behavior response to perphenazine-amitriptyline HCl combination. *Canadian Journal of Psychiatry* 33, 241–242.
- Franklin GA, Pucci PS, Arbabi S, et al. (2002b) Decreased juvenile arson and firesetting recidivism after implementation of a multidisciplinary prevention program. *Journal of Trauma* 53, 260–266.
- Freud S (1955) Beyond the pleasure principle. In *The Standard Edition of the Complete Psychological Works of Sigmund Freud*, Vol. 18, Strachey J (ed). Hogarth Press, London, UK, pp 7–64; (Originally published in 1920).
- Gauthier J and Pellerin D (1982) Management of compulsive shoplifting through covert sensitization. *Journal of Behavior Therapy and Experimental Psychiatry* 13, 73–75.
- Geller JL (1987) Firesetting in the adult psychiatric population. *Hospital & Community Psychiatry* 38, 501–506.
- Geller JL and Bertsch G (1985) Fire-setting behavior in the histories of a state hospital population. *American Journal of Psychiatry* 142, 464–468.
- Giammanco M, Tabacchi G, Giammanco S, et al. (2005) Testosterone and aggressiveness. *Medical Science Monitor* 11, RA136-RA145.
- Glover JH (1985) A case of kleptomania treated by covert sensitization. *British Journal of Clinical Psychology* 24, 213–214.
- Goldman MJ (1991) Kleptomania: Making sense of the nonsensical. *American Journal of Psychiatry* 148, 986–996.
- Grant JE (2004) Co-occurrence of personality disorders in persons with kleptomania: A preliminary investigation. *Journal of the American Academy of Law and Psychiatry* 34, 395–398.

- Grant JE (2006) Understanding and treating kleptomania: New models and new treatments. *Israeli Journal of Psychiatry and Related Sciences*, 43, 81-87.
- Grant JE (2011) Kleptomania treated with tolcapone, a catechol-O-methyl-transferase (COMT) inhibitor. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 35, 295-296
- Grant JE, Correia S, Brennan-Krohn T (2006) White matter integrity in kleptomania: a pilot study. *Psychiatry Research – Neuroimaging*, 147, 233-237.
- Grant JE and Kim SW (2003) Comorbidity of impulse control disorders among pathological gamblers. *Acta Psychiatrica Scandinavica* 108, 207–213.
- Grant JE and Kim SW (2002a) Adolescent kleptomania treated with naltrexone: A case report. *European Child and Adolescent Psychiatry* 11, 92–95.
- Grant JE and Kim SW (2002b) Clinical characteristics and associated psychopathology of 22 patients with kleptomania. *Comprehensive Psychiatry* 43, 378–384.
- Grant JE and Kim SW (2002c) An open label study of naltrexone in the treatment of kleptomania. *Journal of Clinical Psychiatry* 63, 349–356.
- Grant JE and Kim SW (2002d) Temperament and early environmental influences in kleptomania. *Comprehensive Psychiatry* 43, 223–229.
- Grant JE, Kim SW, Odlaug BL (2009) A double-blind, placebo-controlled study of the opiate antagonist naltrexone, in the treatment of kleptomania. *Journal of Biological Psychiatry*, 65, 600-606.
- Grant JE, Levine L, Kim D, Potenza MN (2005) Prevalence of impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry* 162, 2184–2188.
- Grant JE, Williams KA, and Potenza MN (2007) Impulse-control disorders in adolescent psychiatric inpatients: co-occurring disorders and sex differences. *Journal of Clinical Psychiatry* 68, 1584-1592.
- Groditzky GR and Tafrate RC (2000) Imaginal exposure for anger reduction in adult outpatients: a pilot study. *Journal of Behavior Therapy and Experimental Psychiatry* 31, 259–279.

- Gudjonsson GH (1987) The significance of depression in the mechanism of “compulsive” shoplifting. *Medicine, Science and the Law* 27, 171–176.
- Herzog DB, Keller MB, Sacks NR, et al. (1992) Psychiatric comorbidity in treatment-seeking anorexics and bulimics. *Journal of Child and Adolescent Psychopharmacology* 31, 810–818.
- Hollander E, Posner N, and Cherkasky S (2002) Neuropsychiatric aspects of impulse-control disorders. In *American Psychiatric Press Textbook of Neuropsychiatry*, Yudofsky SC and Hales RE (eds). American Psychiatric Publishing, Washington, DC, USA, pp 579–596.
- Hudson JI and Pope HG (1990) Affective spectrum disorder: Does antidepressant response identify a family of disorders with a common pathophysiology? *American Journal of Psychiatry* 147, 552–564.
- Hudson JI, Pope HG, Jonas JM, et al. (1983) Phenomenological relationship of eating disorders to major affective disorder. *Psychiatry Research* 9, 345–354.
- Huesmann LR, Leonard E, Lefkowitz M, et al. (1984) Stability of aggression over time and generations. *Development and Psychopathology* 20, 1120–1134.
- Janet P (1911) La Kleptomanie et al. depression mentale. *Journal de Psychologie et Normale Pathologique* 8, 97–103.
- Kafry D (1980) Playing with matches: Children and fire. In *Fires and Human Behavior*, (ed). Canter D (ed). Wiley, New York, USA, pp 47–62.
- Kavoussi R, Armstead P, and Coccaro E (1997) The neurobiology of impulsive aggression. *Psychiatric Clinics of North America* 20, 395–403.
- Kessler RC, Coccaro EF, Fava M, et al. (2006) The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry* 63(6), 669–678.
- Keutzer CS (1972) Kleptomania: A direct approach to treatment. *British Journal of Medical Psychology* 45, 159–163.

- Khan K and Martin IC (1977) Kleptomania as a presenting feature of cortical atrophy. *Acta Psychiatrica Scandinavica* 56, 168–172.
- Khantzian EJ and Mack JE (1983) Self-preservation and the care of the self. Ego instincts reconsidered. *Psychoanalytic Study of the Child* 38, 209–232.
- Kim SW, Grant JE, Adson DE, et al. (2001) Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. *Biological Psychiatry* 49, 914–921.
- Kindler S, Dannon PN, Iancu I, et al. (1997) Emergence of kleptomania during treatment for depression with serotonin selective reuptake inhibitors. *Clinical Neuropharmacology* 20, 126–129.
- Kmetz GF, McElroy SL, and Collins DJ (1997) Response of kleptomania and mixed mania to valproate. *American Journal of Psychiatry* 154, 580–581.
- Koles MR and Jenson WR (1985) Comprehensive treatment of chronic fire setting in a severely disordered boy. *Journal of Behavior Therapy and Experimental Psychiatry* 16, 81–85.
- Kolko DJ (2001) Efficacy of cognitive-behavioral treatment and fire safety education for children who set fires: Initial and follow-up outcomes. *Journal of Child Psychology and Psychiatry* 42, 359–369.
- Kolko DJ and Kazdin AE (1988) Parent-child correspondence in identification of firesetting among child psychiatric patients. *Journal of Child Psychology and Psychiatry* 29, 175–184.
- Kolko D and Kazdin AE (1989) Assessment of dimensions of childhood fire-setting among patients and nonpatients: the fire-setting risk interview. *Journal of Abnormal Child Psychology* 17, 157–176.
- Koson DF and Dvoskin J (1982) Arson: a diagnostic study. *Bulletin of the American Academy of Psychiatry and the Law* 10, 39–49.
- Kraus JE (1999) Treatment of kleptomania with paroxetine. *Journal of Clinical Psychiatry* 60, 793.
- Kriegsfeld LJ, Dawson TM, Dawson VL, et al. (1997) Aggressive behavior in male mice lacking the gene for neuronal nitric oxide synthase requires testosterone. *Brain Research* 769, 66–70.
- Kuperman S, Kramer J, and Loney J (1988) Enzyme activity and behavior in hyperactive children grown up. *Biological Psychiatry* 24, 375–383.

- Lappalainen J, Long JC, Eggert M, et al. (1998) Linkage of antisocial alcoholism to the serotonin 5-HT_{1B} receptor gene in 2 populations. *Archives of General Psychiatry* 55, 989–994.
- Laubichler W, Kuhberger A, and Sedlmeier P (1996) “Pyromania” and arson. A psychiatric and criminological data analysis [in German]. *Nervenarzt* 67, 774–780.
- Lee S (1994) The heterogeneity of stealing behaviors in Chinese patients with anorexia nervosa in Hong Kong. *The Journal of Nervous and Mental Disease* 182, 304–307.
- Lejoyeux M, Arbaretaz M, McLoughlin M, et al. (2002) Impulse control disorders and depression. *The Journal of Nervous and Mental Disease* 190, 310–314.
- Lejoyeux M, Feuché N, Loi S, et al. (1999) Study of impulse control disorders among alcohol-dependent patients. *Journal of Clinical Psychiatry* 40, 302–305.
- Lejoyeux M, McLoughlin M, Ades, J (2006) Pyromania. In: Hollander E, Stein DJ, eds. *Clinical Manual of Impulse Control Disorders*, Washington DC, APPI; 229-250
- Lepkifker E, Dannon PN, Ziv R, et al. (1999) The treatment of kleptomania with serotonin reuptake inhibitors. *Clinical Neuropharmacology* 22, 40–43.
- Lewis NDC and Yarnell H (1951) *Pathological Firesetting (Pyromania): Nervous and Mental Disease Monograph No 82*. Coolidge Foundation, New York, USA.
- Lindberg N, Holi MM, Tani P, et al. (2005) Looking for pyromania: characteristics of a consecutive sample of Finnish male criminals with histories of recidivist fire-setting behavior between 1973 and 1993. *BMC Psychiatry* 5, 47-52
- Linehan MM, Tutek DA, Heard HL, et al. (1994) Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. *American Journal of Psychiatry* 151, 1771–1776.
- Linnoila M, De Jong J, and Virkkunen M (1989) Family history of alcoholism in violent offenders and impulsive fire setters. *Archives of General Psychiatry* 46, 613–616.
- Linnoila M, Virkkunen M, Scheinin M, et al. (1983) Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. *Life Science* 33, 2609–2614.

- Lion JR (1992) The intermittent explosive disorder. *Psychiatric Annals* 2, 64–66.
- Lowenstein LF (1989) The etiology, diagnosis and treatment of the fire-setting behaviour of children. *Child Psychiatry and Human Development* 19, 186–194.
- Lyons WE, Mamounas LA, and Ricaurte GA (1991) Brain-derived neurotrophic factor-deficient mice develop aggressiveness and hyperphagia in conjunction with brain serotonergic abnormalities. *Proceedings of the National Academy of Sciences USA*. 96, 15239–15244.
- Malhotra AK, Kestler LJ, Mazzanti, C, et al. (2002) A functional polymorphism in the COMT gene and performance on a test of prefrontal cognition. *American Journal of Psychiatry*, 159, 652-652.
- Manuck SB, Flory JD, Ferrell RE, et al. (2000) A regulatory polymorphism of the monoamine oxidase-A gene may be associated with variability in aggression, impulsivity, and central nervous system serotonergic responsivity. *Psychiatry Research* 95, 9–23.
- Manuck SB, Flory JD, Ferrell RE, et al. (1999) Aggression and anger-related traits associated with a polymorphism of the tryptophan hydroxylase gene. *Biological Psychiatry* 45, 603–614.
- Manuck SB, Flory JD, McCaffery JM, et al. (1998) Aggression, impulsivity, and central nervous system serotonergic responsivity in a nonpatient sample. *Neuropsychopharmacology* 19, 287–299.
- Marazziti D, Presta S, Pfanner C, et al. (2000) The biological basis of kleptomania and compulsive buying. Paper presented at the American College of Neuropsychopharmacology 39th Annual Meeting, San Juan, Puerto Rico.
- Marks I (1990) Behavioural (non-chemical) addictions. *British Journal of Addiction* 85, 1389–1394.
- Marzagao LR (1972) Systemic desensitization treatment of kleptomania. *Journal of Behavior Therapy and Experimental Psychiatry* 3, 327–328.
- Mattes JA (1990) Comparative effectiveness of carbamazepine and propranolol for rage outbursts. *Journal of Neuropsychiatry and Clinical Neuroscience* 2, 159–164.
- Mattes JA and Fink M (1987) A family study of patients with temper outbursts. *Journal of Psychiatric Research* 21, 249–255.
- Mavromatis M and Lion JR (1977) A primer on pyromania. *Disease of Nervous System* 38, 954–955.

- McConaghy N and Blaszczynski A (1988) Imaginal desensitization: A cost-effective treatment in two shoplifters and a binge-eater resistant to previous therapy. *Australian and New Zealand Journal of Psychiatry* 22, 78–82.
- McElroy SL (1999) Recognition and treatment of DSM-IV intermittent explosive disorder. *Journal of Clinical Psychiatry* 60, 12–16.
- McElroy SL, Keck PE Jr, Pope HG Jr, et al. (1989) Pharmacological treatment of kleptomania and bulimia nervosa. *Journal of Clinical Psychopharmacology* 9, 358–360.
- McElroy SL, Hudson JI, Phillips KA, et al. (1993) Clinical and theoretical implications of a possible link between obsessive-compulsive and impulse control disorders. *Depression* 1, 121–132.
- McElroy SL, Hudson JI, Pope HG Jr, et al. (1992) The DSM-III-R impulse control disorders not elsewhere classified: Clinical characteristics and relationship to other psychiatric disorders. *American Journal of Psychiatry* 149, 318–327.
- McElroy SL, Hudson JI, Pope HG, et al. (1991a) Kleptomania: Clinical characteristics and associated psychopathology. *Psychology Medicine* 21, 93–108.
- McElroy SL, Soutullo CA, Beckman DA, et al. (1998) DSM-IV intermittent explosive disorder: A report of 27 cases. *Journal of Clinical Psychiatry* 59, 203–210; quiz 211.
- McElroy SL, Pope HG Jr, Hudson JI, et al. (1991b) Kleptomania: A report of 20 cases. *American Journal of Psychiatry* 148, 652–657.
- McGrath P and Marshall PG (1979) A comprehensive treatment program for a fire-setting child. *Journal of Behavior Therapy and Experimental Psychiatry* 10, 69–72.
- McGuire RJ, Carlisle JM, and Young BG (1965) Sexual deviations as conditioned behaviour: A hypothesis. *Behavior Research and Therapy* 2, 185–190.
- McIntyre AW and Emsley RA (1990) Shoplifting associated with normal-pressure hydrocephalus: Report of a case. *Journal of Geriatric Psychiatry and Neurology* 3, 229–230.
- Mendez MF (1988) Pathological stealing in dementia. *Journal of Geriatric Psychiatry and Neurology* 36, 825–826.

- Monopolis S and Lion JR (1983) Problems in the diagnosis of intermittent explosive disorder. *American Journal of Psychiatry* 140, 1200–1202.
- Moreno I, Saiz-Ruiz J, and Lopez-Ibor JJ (1991) Serotonin and gambling dependence. *Human Psychopharmacology: Clinical and Experimental* 6, 9–12.
- New AS, Hazlett EA, and Buchsbaum MS (2002) Blunted prefrontal cortical ¹⁸fluorodeoxyglucose positron emission tomography response to metachlorophenylpiperazine in impulsive aggression. *Archives of General Psychiatry* 59, 621–629.
- Ninan PT, Rothbaum BO, Stipetic M, et al. (1992) CSF 5-HIAA as a predictor of treatment response in trichotillomania. *Psychopharmacology Bulletin* 28, 451–455.
- Noblett S and Nelson B (2001) A psychosocial approach to arson: A case controlled study of female offenders. *Medicine, Science and the Law* 41, 325–330.
- Odlaug, BL and Grant JE (2010) Impulse-control disorders in a college sample: results from the self-administered Minnesota Impulse Disorders Interview (MIDI). *Primary Care Companion to Journal of Clinical Psychiatry* 12, e1-e5.
- O’Sullivan GH and Kelleher MJ (1987) A study of fire-setters in the southwest of Ireland. *British Journal of Psychiatry* 151, 818–823.
- Parsey RV, Oquendo MA, Simpson NR, et al. (2002) Effects of sex, age, and aggressive traits in man on brain serotonin 5-HT_{1A} receptor binding potential measured by PET using [¹¹C]WAY-100635. *Brain Research* 954, 173–182.
- Post RM, Pickar D, Ballenger JC, et al. (1984) Endogenous opiates in cerebrospinal fluid: relationship to mood and anxiety, in *Neurobiology of Mood Disorders*. (eds) Post RM and Ballenger JC. Williams & Wilkins Baltimore, MD, USA, pp 356–368.
- Potenza MN and Hollander E (2002) Pathological gambling and impulse control disorders. In *Neuropsychopharmacology: The 5th Generation of Progress*, Coyle JT, Nemeroff C, Charney D, et al. (eds). Lippincott Williams & Wilkins, Baltimore, MD, USA, pp 1725–1741.

- Presta S, Marazziti D, Dell'Osso L, et al. (2002) Kleptomania: clinical features and comorbidity in an Italian sample. *Comprehensive Psychiatry* 43, 7–12.
- Prins H, Tennent G, and Trick K (1985) Motives for arson (fire raising). *Medicine, Science and the Law* 25, 275–278.
- Puri BK, Baxter R, and Cordess CC (1995) Characteristics of fire-setters. A study and proposed multiaxial psychiatric classification. *British Journal of Psychiatry* 166, 393–396.
- Raine A, Buchsbaum MS, Stanley J, et al. (1994) Selective reductions in prefrontal glucose metabolism in murderers. *Biological Psychiatry* 36, 365–373.
- Repo E, Virkkunen M, Rawlings R, et al. (1997) Criminal and psychiatric histories of Finnish arsonists. *Acta Psychiatrica Scandinavica* 95, 318–323.
- Ritchie EC and Huff TG (1999) Psychiatric aspects of arsonists. *Journal of Forensic Science* 44, 733–740.
- Robins LN and Regier DA (1992) *Psychiatric Disorders in America*. Free Press, New York.
- RŠsŠnen P, Hirvenoja R, Hakko H, et al. (1995) A portrait of a juvenile arsonist. *Forensic Science International* 73, 41–47.
- Schwartz HJ (1992) Psychoanalytic psychotherapy for a woman with diagnoses of kleptomania and bulimia. *Hospital & Comm Psychiatr* 43, 109–110.
- Seroczynski AD, Bergeman CS, and Coccaro EF (1999) Etiology of the impulsivity/aggression relationship: Genes or environment? *Psychiatry Research* 86, 41–57.
- Siever LJ (2008) Neurobiology of aggression and violence. *American Journal of Psychiatry* 165, 429–442
- Siever LJ, Buchsbaum MS, New AS, et al. (1999) D,L-Fenfluramine response in impulsive personality disorder assessed with [18F]fluorodeoxyglucose positron emission tomography. *Neuropsychopharmacology* 20, 413–423.
- Siever LJ, Kahn RS, Lawlor BA, et al. (1991) Critical issues in defining the role of serotonin in psychiatric disorders. *Pharmacology Review* 43, 509–525.
- Specker SM, Carlson GA, Christenson GA, et al. (1995) Impulse control disorders and attention deficit disorder in pathological gamblers. *Annals of Clinical Psychiatry* 7, 175–179.

- Staner L and Mendlewicz J (1998) Heredity and role of serotonin in aggressive impulsive behavior. *Encephale* 24, 355–364.
- Stein DJ, Hollander E, and Liebowitz MR (1993) Neurobiology of impulsivity and the impulse control disorders. *Journal of Neuropsychiatry and Clinical Neuroscience* 5, 9–17.
- Turnbull JM (1987) Sexual relationships of patients with kleptomania. *Southern Medical Journal* 80, 995–998.
- Van Ameringen M, Mancini C, Oakman JM, et al. (1999) The potential role of haloperidol in the treatment of trichotillomania. *Journal of Affective Disorders* 56, 219–226.
- Virkkunen M and Linnoila M (1993) Brain serotonin, type II alcoholism and impulsive violence. *Journal of Studies on Alcohol (Suppl 11)*, 163–169.
- Virkkunen M, Eggert M, Rawlings R, et al. (1996) A prospective follow-up study of alcoholic violent offenders and fire setters. *Archives of General Psychiatry* 53, 523–529.
- Virkkunen M, Goldman D, Nielsen DA, et al. (1995) Low brain serotonin turnover rate (low CSF 5-HIAA) and impulsive violence. *Journal of Psychiatry and Neuroscience* 20, 271–275.
- Virkkunen M, Nuutila A, Goodwin FK (1987) Cerebrospinal fluid monoamin metabolite levels in male arsonists. *Archives of General Psychiatry* 44, 241–247.
- Virkkunen M, Rawlings R, Tokola R, et al. (1994) CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholic, violent offenders, fire setters, and healthy volunteers. *Archives of General Psychiatry* 51, 20–27.
- Wetzel R (1966) Use of behavior techniques in a case of compulsive stealing. *Journal of Consulting Psychology* 5, 367–374.
- Wetzler S, Kahn RS, Asnis GM, et al. (1991) Serotonin receptor sensitivity and aggression. *Psychiatry Research* 37, 271–279.
- Wise MG and Tierney JG (1999) Impulse control disorders not elsewhere classified. In *The American Psychiatric Press Textbook of Psychiatry*, 3rd edition, Hales RE, Yudofsky SC, Talbott JA, et al. (eds). American Psychiatric Press, Washington, DC, USA, pp 773–794.

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

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

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

Figure 80–1 *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.)*

Figure 80–2 *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.*

Table 80–1 Differential Diagnosis of Intermittent Explosive Disorder

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

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