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CHAPTER 21

Impulse Control Disorders Not Elsewhere Classified

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The DSM-IV (American Psychiatric Association 1994a) diagnostic category called "Impulse Control Disorders Not Elsewhere Classified" is a "residual" diagnostic category, even though there is no other distinct group of disorders in DSM-IV classified as impulse disorders. The diagnoses found in this category are intermittent explosive disorder, kleptomania, pyromania, pathological gambling, trichotillomania, and impulse control disorder not otherwise specified (NOS). The features common to all of these impulse disorders are listed in Table 21–1.

In the 19th century, Pinel and Esquirol introduced the concept of an "instinctive impulse" and the term "instinctive monomania." The original monomanias included alcoholism, fire setting, and homicide. Kleptomania, a disorder first described by Marc in 1838, was later added to the monomanias by Mathey (Gibbens and Prince 1962). Many changes in the monomanias have occurred since the 19th century. Kleptomania, pyromania, pathological gambling, and trichotillomania were not listed as mental disorders in either DSM-I (American Psychiatric Association 1952) or DSM-II (American Psychiatric Association 1968). In 1980, kleptomania, pyromania, and pathological gambling were all added to the official DSM nomenclature in DSM-III (American Psychiatric Association 1980), along with two new disorders, intermittent explosive disorder and isolated explosive disorder. Seven years later, in DSM-III-R (American Psychiatric Association 1987), *isolated* explosive disorder was deleted "because of the high potential for misdiagnosis based on a single episode of aggressive behavior" (p. 427). *Intermittent* explosive disorder was retained, even though it was noted that "serious questions have been raised about its validity" (p. 427), and trichotillomania was added.

As more research on impulse disorders is conducted, other changes in this category will occur. Recent research indicates that there is a relationship between low cerebrospinal fluid (CSF) 5-hydroxyindoleacetic acid (5-HIAA; a metabolite of serotonin) and impulsivity (Virkkunen et al. 1987, 1989). In addition, antidepressants, especially those antidepressants with the ability to block the reuptake of serotonin in a selective fashion, are often effective in the treatment of these disorders (McElroy et al. 1991c). This new research has stimulated discussion as to whether the impulse control disorders are "affective spectrum disorders" (McElroy et al. 1992), are related to obsessive-compulsive disorder (OCD) (Swedo et al. 1989), or are a convergence of mood, impulse, and compulsive disorders (Kafka and Coleman 1991).

■ INTERMITTENT EXPLOSIVE DISORDER

□ Definition and Diagnostic Criteria

The classification of individuals who exhibit episodic violent behavior has undergone considerable change in the literature (Table 21–2). DSM-I described an aggressive type of person who mani-

fested "a persistent reaction to frustration with irritability, temper tantrums and destructive behavior" (American Psychiatric Association 1952, p. 37), as a "Passive Aggressive Personality." In 1956, Menninger and Mayman introduced the term "episodic dyscontrol," and Menninger, in his 1963 book, *The Vital Balance,* subdivided dyscontrol into three distinct types: 1) chronic, repetitive aggressive behavior (antisocial personality); 2) episodic, impulsive violence (homicidal assaultiveness, "shell-shock," hypomania, delirious syndromes); and 3) disorganized episodic violence (seizure disorders and brain-damage syndromes).

In 1968, DSM-II introduced a new diagnostic category, "Explosive Personality (Epileptoid Personality Disorder)." The diagnostic criteria for this category seem contradictory in that the intermittent violent behavior needed to occur in an aggressive person who has "gross outbursts of rage or of verbal or physical aggressiveness" that are "strikingly different from the patient's usual behavior." Nonetheless, as DSM-II notes, "these patients are generally considered excitable, aggressive and over-responsive to environmental pressures" (p. 42).

In 1970, Mark and Ervin described a "dyscontrol syndrome," characterized by 1) a history of physical assault, especially wife and child beating; 2) the symptom of pathological intoxication; 3) a history of impulsive sexual behavior, at times including sexual assaults; and 4) a history of many traffic violations and serious automobile accidents. This syndrome was felt to represent behavioral manifestations of disordered brain physiology, particularly in the limbic system. That same year, Monroe (1970) reinforced the idea that subtle brain dysfunction could cause episodic violent behavior and also reiterated the use of the term "episodic dyscontrol." Despite a lack of diagnostic specificity, the label episodic dyscontrol has persisted, primarily in the neurological literature (Elliott 1990).

Table 21–1. Essential features of impulse control disorders not elsewhere classified

1. Failure to resist an impulse, drive, or temptation to perform some act that is harmful to the person or others.
2. An increasing sense of tension or arousal before committing the act.
3. A sense of pleasure, gratification, or release at the time of committing the act, or shortly thereafter.

Table 21–2. Diagnosis of episodic violent behavior: historical perspective

1952	DSM-I	Passive-aggressive personality (aggressive type)
1955	ICD-7	Immature personality (aggressiveness subtype)
1956	Menninger and Mayman	"Episodic dyscontrol"
1963	Menninger	Dyscontrol: chronic, repetitive; episodic, impulsive; disorganized
1968	DSM-II	Explosive personality
1970	Monroe	Episodic behavioral disorders
1970	Mark and Ervin	"Dyscontrol syndrome"
1977	ICD-9	Explosive personality (exclude: dyssocial, hysterical)
1979	ICD-9-CM	Intermittent explosive disorder Recurrent, significant outbursts Not due to other mental disorder Aggression disproportionate to stressors Regret, self-reproach (remorse) present
1980	DSM-III	Intermittent explosive disorder Several discrete, serious episodes Aggression disproportionate to stressors No other impulsivity, aggression Exclude: schizophrenia, antisocial personality disorder, conduct disorder
1987	DSM-III-R	Intermittent explosive disorder Several discrete, serious episodes Aggression disproportionate to stressors No other impulsivity, aggression Exclude: psychosis, organic personality syndrome, antisocial personality disorder, borderline personality disorder, conduct disorder, intoxication
1994	DSM-IV	Intermittent explosive disorder Several discrete, serious episodes Aggression disproportionate to stressor Exclude: antisocial personality disorder, borderline personality disorder, a psychotic disorder, a manic episode, conduct disorder, attention-deficit/hyperactivity disorder, substance intoxication, and a general medical condition that caused the aggression

The diagnostic term "intermittent explosive disorder" first appeared in ICD-9-CM (World Health Organization 1978). This was the first time that an official diagnostic nomenclature had categorized episodic violence as a disorder separate from personality. Intermittent explosive disorder with different diagnostic criteria appeared in DSM-III, and then in DSM-III-R, but with this disclaimer:

> This category has been retained in DSM-III-R despite the fact that many doubt the existence of a clinical syndrome characterized by episodic loss of control that is not symptomatic of one of the disorders that must be ruled out before the diagnosis of intermittent explosive disorder can be made. (American Psychiatric Association 1987, p. 321).

Despite some reservations, intermittent explosive disorder was retained in DSM-IV. The DSM-IV diagnostic criteria for intermittent explosive disorder (Table 21–3) eliminate the requirement that impulsivity be absent between episodes, and add additional exclusionary diagnoses.

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

Table 21–3. DSM-IV diagnostic criteria for intermittent explosive disorder

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

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

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

□ Epidemiology

Monopolis and Lion (1983) call attention to the tendency of clinicians to diagnose intermittent explosive disorder without using any diagnostic criteria. A literature review prepared for the sourcebook for DSM-IV (American Psychiatric Association 1994b) reaffirms this impression and notes that authors typically use the occurrence of one or more explosive outbursts as sufficient for the diagnosis of intermittent explosive disorder. This means that the information published about intermittent explosive disorder, a relatively rare disorder, is actually information about individuals who are violent, an all too common phenomenon. Males account for 80% of persons who display episodic violence (American Psychiatric Association 1994b).

□ Etiology

The characteristics of 842 individuals who were reported to display episodic violent behavior are summarized in Table 21–4 (American Psychiatric Association 1994b). When all 842 cases were carefully reviewed and compared to DSM-III-R criteria for intermittent explosive disorder, only 17 patients who might have intermittent explosive disorder were found. Mattes (1990) reported 4 out of 51 (8%) patients diagnosed with intermittent explosive disorder who were free of any evidence of organicity. No systematic personality analysis was done, although the authors stated that the patients did not have borderline or antisocial personality disorders. In the only study that used DSM-III-R criteria for the diagnosis of intermittent explosive disorder, Felthous et al. (1991), after an extensive evaluation process, reported 13 patients with intermittent explosive disorder, although no neuropsychological testing or systematic personality assessment was performed.

Despite the knowledge that individuals who exhibit episodic violence often have personality disorders, no investigator of intermittent explosive disorder has evaluated personality in a systematic fashion. This is a critical factor, because antisocial

personality disorder and borderline personality disorder are part of the exclusion criteria for the intermittent explosive disorder diagnosis.

Monroe (1970) originally noted that episodic violent behavior occurs in patients because of excessive neuronal discharges or purely motivational causes. He described a continuum between "faulty learning" and "faulty equipment." Patients with episodic violent behavior frequently have neurological abnormalities. When examined, a significant percentage have abnormal neurological exams (65%), abnormal neuropsychological tests (58%), abnormal electroencephalograms (EEGs) (55%), a history of attention-deficit hyperactivity disorder (45%), or a history of learning disability (38%) (Table 21–4). Despite evidence of CNS dysfunction, it is often impossible to establish a clear cause-and-effect relationship between the central nervous system (CNS) dysfunction and episodic violent behavior. Occasionally, special diagnostic techniques, such as EEG activation with alpha-chloralose (Monroe 1970), may prove useful in evaluation of these patients.

Table 21–4. Characteristics of 842 patients with episodic violent behavior

	Percentage of patients examined out of total sample (N = 842)	Number positive/total examined (%)
History of seizures	87%	215/733 (29%)
Legal problems	74%	216/621 (35%)
Head trauma	73%	182/617 (30%)
History of attention deficit	69%	262/582 (45%)
Drugs involved	65%	82/547 (15%)
Neurological abnormality	64%	350/539 (65%)
History of psychosis	62%	33/527 (6%)
Antisocial personality	53%	15/445 (3%)
Alcohol abuse/ pathological intoxication	50%	238/417 (57%)
Electroencephalogram (EEG)	44%	202/368 (55%)
Family history of violence	31%	109/264 (41%)
Prodromal symptoms	24%	77/202 (38%)
Other personality disorder	20%	39/168 (23%)
Neuropsychological tests	20%	97/167 (58%)
Presence of remorse	18%	96/153 (63%)
Genetic abnormality	18%	4/151 (3%)
Learning disability	12%	38/99 (38%)
Computed tomography scan	12%	16/98 (16%)

Source. Reprinted from literature review on intermittent explosive disorder authored by Michael Wise et al. in American Psychiatric Association 1994b. Used with permission.

Research has implicated abnormalities in noradrenergic and serotonergic function in individuals who display episodic violence (Eichelman 1992; Kruesi et al. 1992; Tardiff 1992). This research is promising, and further investigation into the relationship between biological factors and behavioral disorders is warranted.

□ Treatment/Course and Prognosis

According to a review of the literature published between 1937 and 1991, episodic violent behavior is quite common in the general population, but strictly diagnosed intermittent explosive disorder is quite rare (American Psychiatric Association 1994b). Consequently, although information on the management and treatment of aggressive behavior is available, no information exists on the treatment, course, or prognosis of rigorously diagnosed intermittent explosive disorder. In addition, studies of aggressive behavior consist primarily of anecdotal case reports or open drug trials; few studies are placebo controlled. Research in this area is also complicated by the ethical dilemma of randomizing potentially violent patients to placebo treatment.

The development of a treatment plan for a patient who has long-standing, episodic aggressive behavior is complicated and involves the assessment and amelioration (when possible) of multiple factors, such as temperament, sensory cues, neuroanatomy, neurochemistry, neuroendocrine function, stress, and social conditions (Eichelman 1992). Presently, there is no drug specifically approved by the Food and Drug Administration (FDA) for the treatment of aggression. However, numerous pharmacological agents, including neuroleptics, benzodiazepines, lithium, beta-blockers (especially propranolol), anticonvulsants (espe-

cially carbamazepine), serotonin-modulating drugs (tryptophan, trazodone, buspirone, clomipramine, fluoxetine), polycyclic antidepressants, monoamine oxidase inhibitors, and psychostimulants, and long-term psychotherapy are effective in diminishing violent behavior in some individuals (Eichelman 1992; Tardiff 1992). The task for the clinician is to select the most effective and safest intervention for an individual patient who either is acutely violent or has chronic difficulty controlling violent impulses.

The management of a patient who becomes acutely violent, regardless of the underlying etiology, commonly involves physical restraint, seclusion, and sedation. Neuroleptics and benzodiazepines, such as haloperidol and lorazepam (or a combination of the two), are often appropriate and effective interventions to control an acutely violent individual. A more difficult decision is how to treat a patient who has long-standing bouts of aggressive behavior.

Because there are no universally effective "anti-aggression" medications, selection of a pharmacological agent is based on the clinical diagnosis of the patient. For example, when aggressive behavior is the result of psychotic ideation or mania, treatment with a neuroleptic or lithium will likely decrease aggressivity. In the absence of a treatable psychiatric condition, lithium, carbamazepine, propranolol, and more recently serotonin-selective agents are increasingly being used in the management of chronic aggressive behavior.

A small body of literature exists that evaluates the efficacy of lithium in the treatment of aggression not associated with a manic episode. Campbell et al. (1984) treated children who had conduct disorder with lithium and reported decreases in aggressive behavior, especially when the behavior contained strong affective components. Sheard et al. (1976) conducted a double-blind, placebo-controlled study which demonstrated that lithium reduced aggression in prisoners who did not have affective disorders.

Based on the hypothetical relationship between seizures and aggressive behavior, carbamazepine was used in the early 1970s to treat patients with rage outbursts, especially patients who had seizure foci located in the temporal lobe or limbic structures (Mattes 1986). Several additional studies reported that carbamazepine reduces aggressive behavior in patients without overt epilepsy (Mattes 1990; Mattes et al. 1984; Stone et al. 1986).

Elliott (1977) was among the first to treat the aggressive behavior seen in brain-injured patients with propranolol. Numerous other studies demonstrate the relative benefit of beta-blockers in patients with and without overt brain injury (Mattes 1990; Mattes et al. 1984; Sheard 1988; Williams et al. 1982; Yudofsky et al. 1981). More recent research has suggested that compounds that modulate serotonin transmission, such as buspirone, fluoxetine, trazodone, and clomipramine, may benefit some patients.

■ KLEPTOMANIA

□ Definition and Diagnostic Criteria

There is no systematic research on kleptomania to establish or refute the validity of the existing DSM criteria (American Psychiatric Association 1994b). The DSM-IV diagnostic criteria for kleptomania are given in Table 21–5. The only modification in these criteria from those in DSM-III-R is the addition of mania as an exclusionary diagnosis.

The reader must exercise caution when reading literature about "kleptomania." Much of the literature presents information about shoplifters and thiefs, and does not discuss the rare subgroup of those individuals who meet the diagnostic criteria for kleptomania. Shoplifters and thieves are different from kleptomanic persons in that thieves steal for financial gain or to use the stolen object for personal use (and are excluded by criterion A of the DSM-IV diagnostic criteria for kleptomania).

Table 21–5. DSM-IV diagnostic criteria for kleptomania

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

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

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

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

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

□ Epidemiology

Little is known about the epidemiology of kleptomania, because it is a relatively rare disorder and is seldom the subject of research. According to McElroy et al. (1991a, 1991b), most of the information on kleptomania is derived from three sources: studies of "legally referred" shoplifters, case reports or small series of psychiatric patients, and cases of kleptomania patients with eating disorders. Consequently, estimates on the incidence and sex ratios of kleptomania vary widely. Among shoplifters (Table 21–6), the incidence of kleptomania has been estimated as "no clear entity exists" (Gibbens and Prince 1962), 3.8 % (Arieff and Bowie 1947), 8% (Medlicott 1968), and less than 5% (American Psychiatric Association 1994b). In a recent review, Goldman (1991), noting that "kleptomania may account for a substantial portion of the staggering $40 billion in business losses attributed to shoplifting each year" (p. 986), estimates that the rate of kleptomania is at least 6 per 1,000 persons. Cupchik (1992), however, has questioned the validity of Goldman's statements.

Goldman (1991), based on his review, concluded that the "typical" individual with kleptomania is a 35-year-old woman who began to steal when she was 20 years old. In a study by Bradford and Balmaceda (1983), a 1:1 male-to-female ratio among shoplifters was found; however, more females (62%) than males (38%) were sent for pretrial psychiatric evaluation, which would skew the data. In a study by McElroy et al. (1991a), 15 of 20 (75%) individuals who met DSM-III-R criteria for kleptomania were female. The peak frequency of stealing was 27 episodes per month (range = 0.3 to 120); the mean age was 36 years (range = 21–48 years); and the mean duration of illness was 16 years (range = 3–38 years).

□ Etiology

Hypotheses about the cause of kleptomania are legion and little agreement exists.[†] In a detailed discussion of the analyses of three males with kleptomania, Wilhelm Stekel (1924) noted that "all three are dominated by the Oedipus complex—which is certainly far from an accident. All presented a tremendous sexual energy which may be released, as kinetic energy, temporarily, in cleptomaniac deeds" (p. 122). Fenichel (1945) pointed out that the unconscious formula for kleptomania is, "If you don't give it to me, I'll take it" (p. 370).

Bradford and Balmaceda (1983) in their study found an association between shoplifting and psychosocial stress: 78% of shoplifters had a mild to moderate psychosocial stressor on DSM-III Axis IV, and an additional 14% had a severe level of stress. McElroy et al. (1991a) found that none of the 20 persons with kleptomania whom they studied developed stealing as a result of stressful or traumatic events.

Gibbens (1981) has commented on the motivation behind kleptomanic behavior:

> The motive is often obscure, and the objects stolen useless or very trivial, but most often it seems to be a sudden impulse to give themselves a treat, like a child stealing for lack of love; to punish others by punishing themselves; hysterical secondary gain; or, in the newly poor, to keep up appearances. (p. 347)

Stealing is occasionally a presenting feature of brain disease (Wood and Garralda 1990) or a response to medications. McIntyre and Emsley (1990) described the case of an individual who "impulsively" stole inexpensive cosmetics who was found to have normal-pressure hydrocephalus. Khan and Martin (1977) described a man who stole as a presenting feature of presenile dementia, and Mendez (1988) discussed the case of a 66-year-old man who compulsively stole as a presenting feature of multi-infarct dementia. Coid (1984) reported on the case of a 54-year-old woman who apparently sought relief of her withdrawal symptoms from diazepam through stealing.

McElroy et al. (1991a) found that all 20 of her patients who met the DSM-III-R diagnostic criteria for kleptomania had either a current diagnosis (65%) or a lifetime history (100%) of depression. Also, a particularly high association with bipolar disorder was noted (35%). In addition, 17 (85%) met criteria during their lifetime for at least four or more other psychiatric disorders, including psychoactive substance use disorders (50%), anxiety disorders (80%), eating disorders (60%), and other impulse control disorders (60%).

[†]Hypotheses to explain the stealing behavior in kleptomania are that such behavior is an antidepressant, compensation for an actual or anticipated loss, an act for intrapsychic profit, a fetishistic behavior, a sexual act, a symptom of an underlying conflict, a behavior related to depression, a defense, a neurotic conflict, a form of psychopathy, or an OCD-related disorder (Goldman 1991). Kleptomanic behavior can also result from brain disease or medications.

□ Treatment/Course and Prognosis

Recent literature reviews of kleptomania report no systematic studies of rigorously diagnosed kleptomanic individuals (American Psychiatric Association 1994b; Goldman 1991; McElroy et al. 1991a). The secretive nature of the disorder also complicates systematic study. In addition, as noted in the foregoing discussion, many case reports and studies fail to adequately distinguish between shoplifting and kleptomania. Without systematic studies and careful differentiation of kleptomania from shoplifting, little useful information exists about treatment, course, or prognosis of kleptomanic individuals. Available information on treatment is limited to a number of case reports that use a broad range of therapeutic interventions.

Table 21–6. Frequency of kleptomania among shoplifters

Study	*N*	Clinical features
Arieff and Bowie (1947)	338	93% female; 70% some psychiatric disorder; sample 1.8% of total group of shoplifters arrested during the time period of study; kleptomania only in 3.8% of total sample.
Gibbens and Prince (1962)	776	69% female; depression common; higher than average psychiatric hospital admission; 0% kleptomanic.
Ordway (1964)	85	43% depressed (DSM-I); unknown % kleptomanic.
Cameron (1964)	873	Only females in the study; 1.4% depressed; < 1% kleptomanic.
Medlicott (1968)	50	52% female; 28% depressed (all female); all had some psychiatric disorder; 8% kleptomanic.
Gillen (1976)	48	100% females; 100% psychiatric disorder; < 5% kleptomanic.
Bradford and Balmaceda (1983)	50	62% female; 42% depressed; 4% kleptomanic.
Cupchik and Atcheson (1983)	24	71% female; unknown % kleptomanic.
Silverman and Brener (1988)	34	100% female; unknown % kleptomanic; shoplifters were compared with agoraphobic, depressive subjects; shoplifters had high levels of psychosocial stress (e.g., marital discord).

Source. Reprinted from literature review on kleptomania authored by John Bradford in American Psychiatric Association 1994b. Used with permission.

The psychoanalytic view suggests that kleptomania is a symptom of an underlying conflict (Goldman 1991). Unfortunately, because systematic studies are lacking, the success of psychoanalytic treatment of kleptomania is unknown. In one example, Fishbain (1987) reports the cure of a kleptomanic patient using a combination of insight-oriented and supportive psychotherapy, as well as antidepressant medication.

There are a number of reports of the use of behavior therapy to treat kleptomania. Glover (1985) has described the successful use of covert sensitization in the treatment of this disorder. Guidry (1975) and Wetzel (1966) have described single case reports using covert punishing contingency and behavioral modification, respectively. Also, Marzagao (1972) reported success using systematic desensitization to reduce the anxiety that had prompted the stealing behavior of a kleptomanic patient.

Somatic therapies are credited with partial or full remission in kleptomanic symptoms. For example, McElroy et al. (1991a) cite several reports of electroconvulsive therapy (ECT) alone, or ECT in combination with antidepressants, decreasing kleptomanic behavior. Burstein (1992) reports remission of kleptomanic behavior in one patient treated with a combination of fluoxetine and lithium. McElroy's group is responsible for much of the recent work on pharmacotherapeutic interventions in kleptomania. McElroy suggests that kleptomania is part of a group of disorders called affective spectrum disorders. Included in this category are OCD, eating disorders, and major mood disorders. These disorders are hypothesized to represent a spectrum of behaviors that occur secondary to abnormalities in the serotonergic system. Consequently, much of their research focuses on treatment using antidepressants that modulate serotonergic activity. For example, McElroy et al. (1989) reported a complete or partial decrease in kleptomania in three bulimic patients treated with a serotonergic antidepressant (either trazodone or fluoxetine). In another study, McElroy et al. (1991c), reported that 10 of 18 (56%) kleptomanic patients

had a partial or complete remission of stealing as a result of treatment with antidepressants.

Future research on kleptomania must differentiate individuals who shoplift from patients who have kleptomania. It must also focus on associated clinical characteristics, such as OCD, eating disorders, and major mood disorders. This will allow for the scientific evaluation of various treatments and help delineate the course and prognosis of kleptomania (American Psychiatric Association 1994b).

■ PYROMANIA

□ Definition and Diagnostic Criteria

Pyromania has been described as "motiveless arson" (Koson and Dvoskin 1982). This description would imply that if no motivation can be determined, pyromania exists. The problem with this diagnostic approach is that arsonists often do not admit motivation, or even the crime for that matter. To do so would admit guilt. This has led to misclassification of arsonists as pyromaniacs and contaminates much of the data on pyromania. Geller (1987) cautions that "pathologic fire setting needs to be viewed not as pathognomonic of pyromania but as a symptom, present in a range of psychiatric disorders, that must be addressed clinically" (p. 501).

In a literature review on pyromania written by Geller for the DSM-IV Sourcebook (American Psychiatric Association 1994b), there was found to be "a small frequency of pyromania in reported cases of fire setting since 1970 and *no* cases reported in the literature from the United States since 1970" (Table 21–7). Because there is little new literature on pyromania, DSM-IV made few changes to the DSM-III-R diagnostic criteria for pyromania. The additions to DSM-IV state that pyromania is not diagnosed if fire setting occurs only during a manic episode or if fire setting is better accounted for by a conduct disorder or an antisocial personality disorder (Table 21–8).

Geller notes that the pyromanic individual may make considerable advanced preparation before setting the fire, be an avid fire watcher, set off false fire alarms, be interested in fire-fighting paraphernalia, and even seek work as a firefighter.

□ Epidemiology

The diagnosis of pyromania is rarely made when DSM-III or DSM-III-R criteria are applied; the diagnosis is much more readily given in studies of arsonists in which no clear diagnostic criteria are used. Therefore, one cannot be sure whether pyromanic individuals are adequately differentiated in the literature from persons who exhibit other types of fire setting or arson. This flaw brings into question the characteristics often associated with pyromania.

The classic monograph *Pathological Fire Setting (Pyromania)*, by Lewis and Yarnell (1951), is the largest study of this topic. Lewis and Yarnell collected cases from a wide variety of sources, including about 2,000 records from the National Board of Fire Underwriters. Additional cases were provided through fire departments, psychiatric clinics and institutions, and police departments in the vicinity of New York City. A detailed survey of 1,145 adult male cases was made. (It is interesting that even with intense efforts, these authors were able to find only 201 records of adult female fire setters.) The peak incidence of fire setting occurred at age 17. Table 21–9 contains a summary of motivations found in the 1,145 cases of fire setting. Concerning the pyromanic individuals (39% of the selected sample), Lewis and Yarnell noted that "many of them offered the excuse of finding themselves controlled by the 'irresistible impulse' and, though their stories implied a mixture of all the above motives, they more often denied such motives, and for this reason we have allowed them to remain loosely classified as pyromaniacs" (p. 32). Of the 1,145

Table 21–7. Frequency of pyromania in adult fire setters

Dates	Cases of fire setting	Cases of pyromania	Percentage of fire setters diagnosed with pyromania
1840–1919	22	3–4	14–18%
1920–1959	1,496	781	52
1960–1969	169	42	25
1970–1979	161	0	0
1980–1989	932	27[a]	3

[a]Contains 2 cases from Canada and 25 cases from Finland. The number of cases from Finland (Virkkunen) may be less if individuals were reported in more than one article.

Source. Reprinted literature review on pyromania authored by Jeffrey Geller in American Psychiatric Association 1994b. Used with permission.

Table 21–8. DSM-IV diagnostic criteria for pyromania

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

B. Tension or affective arousal before the act.

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

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

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

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

Note. Italics indicate changes to DSM-III-R criteria.

cases, 48% were classified as "morons" or "imbeciles"; 22% as having borderline to dull normal intelligence; and 13% as having between dull to low average intelligence. Only 17% of the entire group were rated as having average to superior intelligence.

Other studies indicate that the clinical phenomenon of pyromania is more rare than was found in Lewis and Yarnell's samples. Robbins and Robbins (1967) reported that 23% of 239 convicted arsonists were identified as pyromaniacs; Koson and Dvoskin (1982) reported that no cases (0%) of pyromania were found during the pretrial evaluation of 26 arsonists. Bradford (1982) found only one individual (3% of sample) who "had some features of this phenomenon" in 34 pretrial arson evaluations. In two studies of fire setting among psychiatric patients at Northampton State Hospital (Geller 1984; Geller and Bertsch 1985), only one pyromaniac (2%) was found among a total of 45 fire setters.

Whereas pyromania is a rare disorder, fire-setting behavior among adults and children with other psychiatric disorders is not (Kolko and Kazdin 1992). In a study of psychiatric patients in a state hospital, 26% had a history of fire-setting behaviors and 16% had actually set fires (Geller and Bertsch 1985). Preliminary data on another chronic mentally ill population showed a lifetime prevalence rate of 30% in fire-setting behaviors.

In DSM-III (American Psychiatric Association 1980) a number of features are associated with pyromania. These include alcohol intoxication, psychosexual dysfunction, lower than average IQ, chronic personal frustrations, resentment of authority figures, and the occurrence of sexual arousal secondary to fires. Whether these features are associated with pyromania per se or represent a general characteristic of arsonists/fire setters remains a question for further study. Neither DSM-III-R (American Psychiatric Association 1987) nor DSM-IV (American Psychiatric Association 1994a) lists any features associated with pyromania.

□ Etiology

Fire symbolizes many things, from the "fires of Hell" to "fiery passion." The diverse symbolism of fire is represented in the psychoanalytic interpretations of pyromania. Wilhelm Stekel (1924), in his discussion of 95 cases and a case report of an analysis of a patient with pyromania, emphasized that "awakening and ungratified sexuality impels the individual to seek a symbolic solution to his conflict between instinct and reality" (p. 126). Sigmund Freud (1932[1931]/1964) considered fire setting a masturbatory equivalent with homosexual features. He noted that "in order to gain control over fire, men had to renounce the homosexually-tinged desire to put it out with a stream of urine" (p. 187). He further noted that "the warmth that is radiated by fire calls up the same sensation that accompanies a state of sexual excitation, and the shape and movement of a flame suggest a phallus in activity" (p. 190). Fenichel (1945) discussed pyromania as a specific form of urethral-erotic fixation and emphasized the sadistic and destructive symbolism of fire. Later writers, such as Lewis and Yarnell (1951), stressed that revenge is an important underlying motive for pyromania. Geller (1987) suggests that

Table 21–9. Survey of motivation in 1,145 fire setters

Motivation	Percentage of sample
Pyromania	39%
Revenge/jealous resentment	23
Psychosis	13
Volunteer firefighters or fire "buffs"	9
Tramps/migrant workers	7
"Would-be heroes"	6
Associated with burglary	3

fire setting as a symptom can best be understood as a communication from an individual with few social skills.

Research (Virkkunen 1984; Virkkunen et al. 1987) raises questions about whether the behavior of arson is associated with reactive hypoglycemia and/or lower concentration of 3-methoxy-4-hydroxyphenylglycol (MHPG) and 5-HIAA in CSF. Virkkunen et al. (1987) note that their results "support the hypothesis that poor impulse control in criminal offenders is associated with low levels of certain CSF monoamine metabolites and with a hypoglycemic tendency" (p. 241). A small subgroup of pyromanic subjects in this study (3 of 20 arsonists) had the lowest blood glucose nadirs among the arsonists. In addition, impulse fire setters who are violent offenders are often alcoholics and have a father who is an alcoholic (Linnoila et al. 1989).

□ Treatment

Most writers on the treatment of pyromania approach the patients from a psychoanalytic perspective (Macht and Mack 1968; Stekel 1924). Mavromatis and Lion (1977) point out that "treatment for fire setters has been traditionally problematic due to the frequent refusal to take responsibility for the act, the use of denial, the existence of alcoholism, and the lack of insight" (p. 955).

Most behavioral researchers have used aversive therapy to treat fire setters (McGrath and Marshall 1979), although others have used positive reinforcement with threats of punishment, stimulus satiation, and operant structured fantasies with positive reinforcement (Bumpass et al. 1983). Bumpass et al. (1983) treated 29 child fire setters using a graphing technique that sequentially correlated external stress, behavior, and feeling on graph paper. These authors reported that following treatment (average follow-up of 2.5 years), only 2 of the 29 children subsequently set fires.

□ Course and Prognosis

The pyromanic impulse to set fires is episodic and often self-limited, and frequently appears during a developmental or situational crisis. Fire setting associated with mental retardation, alcoholism, or a ritualistic pattern indicates a poor prognosis. A better prognosis exists if the patient can verbalize and work through frustrations in therapy. Studies indicate that the recidivism rate for fire setters ranges from 4.5% (Mavromatis and Lion 1977) to 28% (Lewis and Yarnell 1951).

■ PATHOLOGICAL GAMBLING

□ Definition and Diagnostic Criteria

Gambling is now legal in some form in 48 out of 50 states and in over 90 countries. The amount of money spent gambling legally has grown from $17 billion in 1974 to $210 billion in 1988, and consequently increases the likelihood that clinicians will encounter individuals who have this disorder (Lesieur and Rosenthal 1990).

The diagnostic criteria for pathological gambling in DSM-IV incorporate features from the criteria found in DSM-III and DSM-III-R (Table 21–10). The reader will note that the criteria

Table 21–10. DSM-IV diagnostic criteria for pathological gambling

A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:
   1. Is preoccupied with gambling (e.g., preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble).
   2. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.
   3. Has repeated unsuccessful efforts to control, cut back, or stop gambling.
   4. Is restless or irritable when attempting to cut down or stop gambling.
   5. Gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression).
   6. After losing money gambling, often returns another day to get even ("chasing" one's losses).
   7. Lies to family members, therapist, or others to conceal the extent of involvement with gambling.
   8. Has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling.
   9. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling.
   10. Relies on others to provide money to relieve a desperate financial situation caused by gambling.

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

for pathological gambling are similar to the criteria for psychoactive substance abuse disorders. Freud was one of the first to recognize this similarity, which prompted him to categorize pathological gambling as an addiction along with alcoholism and drug dependence (Lesieur and Rosenthal 1990).

□ Clinical Features

Edmond Bergler (1957), a psychoanalyst who has treated more than 60 compulsive gamblers, characterizes the compulsive gambler as a risk taker who fails to profit from his gambling misadventures. The compulsive gambler is often described as fiercely competitive, highly independent, individualistic, overconfident, and profoundly optimistic. He resents the intrusion of authority figures into his life, just as he resented his parents' intrusions during his childhood. The compulsive gambler is likely to marry and provide reasonably well for his family before his gambling losses precipitate a financial crisis. Contrary to what one might suspect, the compulsive gambler is extremely knowledgeable about the technical aspects of gambling and his skills are impressive, particularly when winning. It is when he is "chasing" his losses by larger and larger wagers that he disregards his technical knowledge. The compulsive gambler rarely seeks psychiatric help on his own, but is generally forced into consultation.

Speculation about the personality structure of a pathological gambler deserves comment. Comparison of the pathological gambler's premorbid personality with the "premorbid" personality of an individual with alcoholism seems appropriate, especially because the two disorders are so often linked. George Vaillant's (1980) prospective research on the personality of the alcoholic individual shows that this person does not have a premorbid oral, passive, dependent personality. Rather, when the person with alcoholism drinks excessively and continuously, these traits emerge as a secondary rather than a primary phenomenon. The same may be true of the pathological gambler.

The clinical features of a pathological gambler are listed in Table 21–11. The individual is progressively preoccupied with gambling; spends more time gambling and needs higher bets to experience excitement; experiences withdrawal symptoms if gambling is abruptly discontinued; may use gambling to forget or avoid dysphoric mood states; wagers larger and larger amounts to win back losses (called "chasing"); creates family and job disruption by telling lies to sustain gambling and performing illegal acts to pay debts; will request and often receive financial help (a "bailout") from family and/or friends to pay off debts and to sustain gambling; and attempts unsuccessfully to cut back or stop gambling.

□ Epidemiology

Dickerson (1984) estimates that 1% of adult males are pathological gamblers, an estimate that is consistent with Volberg and Steadman's (1988) data; in DSM-IV (American Psychiatric Association 1994a) the prevalence is estimated as being 2% to 3% of the adult population. The rate among psychiatric inpatients is higher and goes largely unrecognized. Lesieur and Blume (1990) found that 6.7% of patients admitted to an adult general psychiatry ward were pathological gamblers. Prevalence rates among alcohol- and substance-abusing individuals are estimated from 8% to 25% (Lesieur and Rosenthal 1990). It is likely that equal numbers of men and women gamble, but the vast majority of compulsive gamblers are men. The membership of Gamblers Anonymous ranges from 1% to 2% women in the United Kingdom to 5% to 10% in Australia. Pathological gambling and alcoholism are more common in the fathers of males with the disorder and in the mothers of females with the disorder.

□ Etiology

Numerous theories have been invoked to explain the origin of pathological gambling, including unconscious motivations, behavioral anomalies, the presence of an affective disorder, addiction, and biological abnormalities. Hollander et al. (1992) recently reiterated the idea that pathological gam-

Table 21–11. Clinical features of pathological gambling

Progressive gambling
Development of *tolerance*
Symptoms upon discontinuation (*withdrawal*)
Gambling as *escape* from dysphoria
Chasing of losses
Lies/deception
Illegal acts
Family/job disruption
Financial *bailout*
Inability to stop (*loss of control*)

bling might be an OCD-related disorder. Any one explanation seems a gross oversimplification given the heterogeneous nature of the patient population.

Bergler (1957) feels that the compulsive gambler's illogical, senseless certainty that he will win stems from a childhood sense of omnipotence. His unconscious aggression against the reality principle leads to an unconscious need for punishment. The punishment, achieved through losing, becomes essential for psychic equilibrium. H. R. Greenberg (1980) has reviewed the psychodynamic formulations of other analysts, including the analogies between gambling, childhood play, and masturbation; the classification of compulsive gamblers as compulsive neurotics with latent homosexual tendencies; and a variant of Shicksal neurosis in which the person surrenders responsibility for his actions to an omnipotent force, such as lady luck.

Gambling is also an activity that is reinforced by both the cash that one may win and the many exciting stimuli associated with the process. The validity of this hypothesis to explain pathological gambling is challenged by the observation that individuals have losing streaks that last for months. The compulsive gambler will continue gambling regardless of the losses. He appears desperate and oblivious to the rest of the world as he "chases" his losses.

Many studies (Lesieur and Rosenthal 1990; Linden et al. 1986; McCormick et al. 1984) have reported an extremely high incidence of affective disorders among pathological gamblers. McCormick et al. (1984) examined 25 compulsive gamblers and found a 72% incidence of major depression around the time they stopped gambling. Linden et al. (1986) reported that 76% of 50 compulsive gamblers had a major depressive disorder. These authors also found that 32% of the subjects had first-degree relatives with major affective disorders, and 36% had at least one first-degree relative with alcohol abuse or dependence. Gambling may also be an antidepressant, protecting the gambler from dysphoria and depression. The analogy can be drawn between the manic and depressive cycles of a bipolar patient, and the frenetic, high-energy mood of a winning gambler versus the desperate low of the losing gambler.

There are many interesting similarities between substance abuse, particularly alcoholism, and compulsive gambling. In both disorders dependencies are developed that exclude basic human needs such as sleep, food, and sex. The insidious downward trajectory of both disorders leads to loss of family, friends, and position. Custer (1982) noted that compulsive gamblers who abruptly stop gambling during a hospital admission are frequently tremulous and experience headaches, abdominal pain, diarrhea, nightmares, and cold sweats. Blaszczynski et al. (1986) found that a subgroup of gamblers had lower baseline β-endorphin levels. Roy and co-workers (1988) found evidence of elevated noradrenergic function in pathological gamblers and, in a later study (Roy et al. 1989), noted that elevation in noradrenergic function was correlated with personality extraversion. The pathological gambler's EEG activation patterns to right and left brain tasks are similar to patterns found in alcoholic adults and unmedicated children diagnosed as having attention-deficit disorder (ADD). Goldstein et al. (1985) noted that "both pathological gambling and alcoholism may be related to dysfunctional attention mechanisms and, more to the point, to the deficits in impulse control that characterize ADD" (p. 1233). The course of a recovering substance abuser and that of a compulsive gambler are very much alike in that relapses are common and occur at times of increased stress. Abstinence is felt by many to be an essential part in the recovery from both disorders. The mainstay of treatment, Gamblers Anonymous, is patterned after Alcoholics Anonymous.

□ Treatment

A number of diverse treatments for compulsive gambling have been reported (Legg England and Götestam 1991). These include psychoanalysis, behavior therapy, cognitive therapy, medications, and ECT. There are no controlled studies that compare these treatment modalities. Regardless of the choice of therapy, the pathway to recovery is likely to be fraught with difficulties. Relapses are common, as are missed sessions. During treatment, financial crises may occur (some clinicians experienced in treating compulsive gamblers recommend collecting the fee prior to each session!), and legal sequelae of gambling may arise.

The high incidence of major affective disorders among pathological gamblers leads one to question the relationship between these disorders. The dearth of articles on somatic treatments of compulsive gamblers leaves this question unanswered. Moskowitz (1980) did report improvement in three compulsive gamblers using lithium carbonate. Hollander et al. (1992), in a double-blind, placebo-controlled trial with clomipramine, reported a successful response in one treatment-resistant case. In some gamblers the affective disorder may promote the gambling, whereas in other gamblers it seems

likely that the depletion of resources (e.g., emotional, family, friends, financial) is responsible for the affective state of the gambler when he enters treatment. There may be a subgroup of compulsive gamblers who remain depressed in spite of abstinence. In a follow-up study of gamblers who continued abstinence 6 months after inpatient treatment, 18% reported "significant improvement in work and family life . . . but still were significantly depressed" (Taber et al. 1987, p. 761).

Behavioral treatments, particularly aversive therapy, have been used to treat compulsive gamblers. However, review of the literature on aversive treatments reveals disappointing results. McConaghy et al. (1983) compared aversive therapy with imaginal desensitization and found the latter more effective. Dickerson (1984) noted that there is "a trend away from the use of single limited procedures such as aversion therapy toward a multi-modal approach" (p. 113). Greenberg and Rankin (1982) reported on the behavioral treatment of 25 compulsive gamblers. Following treatment, 5 (20%) had their gambling "well under control," 7 (28%) alternated between periods of control and periods of gambling, and 14 (56%) were gambling when last followed up.

Bolen and Boyd (1968) stated that psychoanalysis was the treatment of choice for the compulsive gambler. In Bergler's (1957) collection of 60 individual case reports, 15 patients (25%) discontinued treatment in the first 6 weeks; of those who underwent treatment, 33 (55%) received an analysis of their neurosis, and 30 (50%) were judged to be cured.

Custer (1982) recommends that the compulsive gambler be admitted to an inpatient psychiatric treatment center, particularly when there is a risk of suicide, emotional decompensation, or exhaustion. The initial assessment must include the compulsive gambler's areas of "high risk": marital problems, large debts, demands or threats from creditors, loss of employment, legal problems, and isolation from friends and relatives. The treatment plan is then designed to treat problems identified during the intake process. In addition, Custer recommends group therapy with other compulsive gamblers and involvement of the compulsive gambler with Gamblers Anonymous.

Gamblers Anonymous and its sister group Gam-Anon (for family and spouses of compulsive gamblers) and Gam-a-Teen (for adolescent children of compulsive gamblers) are important resources for treatment. The only requirement for membership in Gamblers Anonymous is an expressed desire to stop gambling.

□ Course and Prognosis

The clinical course of compulsive gambling is outlined in Figure 21–1. The compulsive gambler's early history often involves winning and periods of considerable profits. Once the gambler falls behind, he is unable to cut his losses. Instead, he increases his wagers and begins to "chase" his losses on long shots. This leads to a tightening spiral of involvements and fewer options (Lesieur 1979). In a study of 50 compulsive gamblers, Lesieur (1979) noted that all 50 participated in some activity such as pool, golf, and bowling hustling; bookmaking; obtaining loans from friends, loan sharks, or loan companies; "borrowing" from personal checking accounts and from work; and committing petty larceny. Seventeen (34%) participated in check forgery, burglary, fencing stolen goods, stealing company checks, or swindling.

The prognosis of the untreated compulsive gambler is unknown. Few creditable data exist about the prognosis of the treated pathological gambler. Taber et al.'s (1987) follow-up study of 66 male veterans 6 months after the completion of a 28-day inpatient program indicates that 56% were totally abstinent. Outcome in this study was found to correlate with attendance at Gamblers Anonymous meetings. It does seem clear that keeping the compulsive gambler engaged in any form of therapy is very difficult. In the present authors' opinion, data currently available on the long-term prognosis of alcoholism might come close to predicting the outcome of pathological gambling.

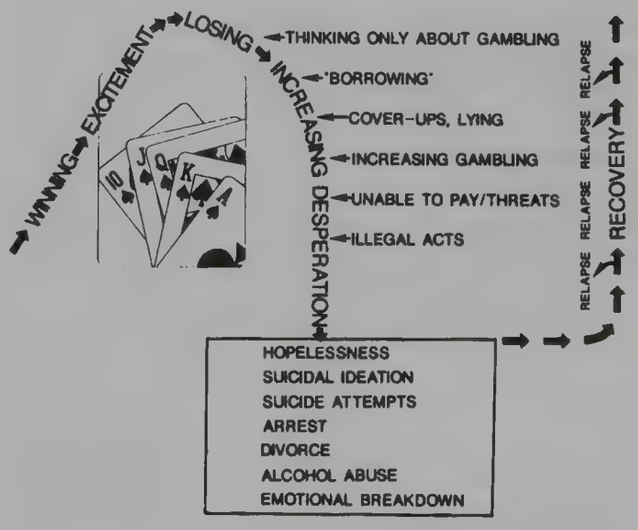

Figure 21–1. Clinical course of pathological gambling.

■ TRICHOTILLOMANIA

□ Definition and Diagnostic Criteria

Trichotillomania is a term created by Hallopeau in 1889 (Krishnan et al. 1985) to describe a compulsion to pull out one's own hair. Trichotillomania was not listed in DSM-III, but was added to DSM-III-R because "this well-recognized disorder involving the pulling out of hair fulfills the general criteria for the Impulse Control Disorders" (American Psychiatric Association 1987, p. 427). Although there has been more recent research on trichotillomania than on most of the other disorders in this chapter, insufficient information was available at the time DSM-IV was developed to warrant significant changes to the DSM-III-R diagnostic criteria (Table 21–12).

Trichotillomania produces irregular, nonscarring focal patches of hair loss that are linear, rectangular, or oval (Figure 21–2). Hair loss usually occurs in the scalp region but can involve eyebrows, eyelashes, or pubic hair. These areas of hair loss are more likely found on the opposite side of the body from the dominant hand. Within the area of hair loss, broken hairs of varying lengths are found, and the scalp may have a slight brownish discoloration secondary to rubbing the area. Two clinical findings can help with the diagnosis. In trichotillomania, the patient should not have changes in fingernails or toenails (except possibly signs of nail biting) usually associated with dermatologic conditions. Second, the application of collodion to the area of hair loss for 1 week permits regrowth of the hair.

Table 21–12. DSM-IV diagnostic criteria for trichotillomania

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

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

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

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

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

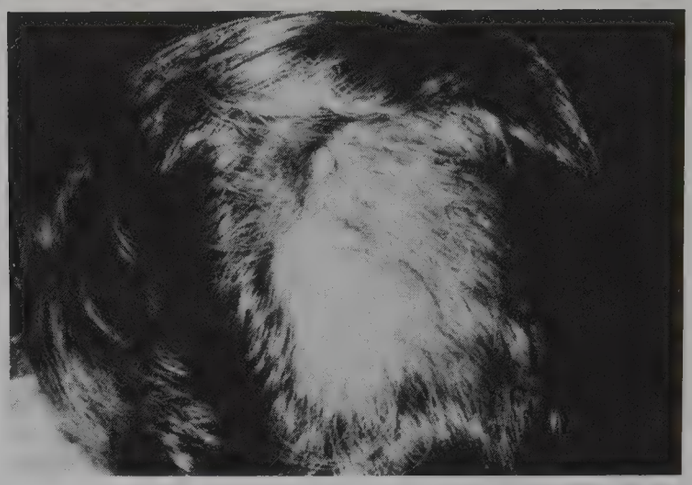

Figure 21–2. Trichotillomania. Note the irregular pattern of hair loss, the hair of varying lengths within the patch of hair loss, and the scalp, which has no evidence of scarring.

Parents may resist the suggestion that a child is pulling out his or her own hair. In these cases, a biopsy is diagnostic and provides evidence for the doubting parents. Careful parental observation of the child, which includes looking for hair among the child's playthings, is often helpful in making the diagnosis. The child may also practice onychophagy (i.e., nail biting). If the child complains of gastrointestinal symptoms such as abdominal pain, diarrhea and/or constipation, or decreased appetite, examination of the oral cavity for evidence of trichophagy (i.e., hair biting) and X-ray examination of the stomach for a trichobezoar (i.e., hairball) are warranted.

□ Epidemiology

The frequency of trichotillomania in the adult population is unknown, but most literature suggests that it is rare. The literature also suggests that females have the disorder much more frequently than do males. However, Christenson et al. (1991d), in a more recent questionnaire survey of 2,579 freshman college students, concluded that trichotillomania may not be as rare as previously suspected and that males may be affected as frequently as females. Prevalence for males ranged between 0.6% and 1.5%, and for females between 0.6% and 3.4%.

Christenson et al. (1991a) recently reported on the characteristics of 60 adult hair pullers. The mean age at onset was 13 years, and 93% of the subjects were female. Hair was pulled primarily from the scalp (67%); however, other locations were

involved, such as eyelashes (22%), eyebrows (8%), facial hair (2%), and pubic hair (2%). Subjects in this study were just as likely to use the nondominant as the dominant hand for hair pulling. All subjects felt that their hair pulling was an excessive or unusual behavior, and 95% reported a diurnal variation, with the worst hair pulling in the evening. The comorbidity with other psychiatric disorders was striking (Table 21–13).

The dermatological literature most commonly reports pathological hair pulling in preadolescent children. For example, Stroud (1983), a dermatologist, reviewed diagnoses of all patients who presented to his office with hair loss. During 1982, 59 children under the age of 13 presented to his office with hair loss. The children were found to have the following diagnoses: 31 children (53%) had tinea capitis (i.e., a fungal infection), 12 (20%) had alopecia areata (i.e., a suspected autoimmune phenomenon), 7 (11%) had traction alopecia (i.e., a disorder associated with hair styles that apply excessive prolonged tension to the hair), and 6 (10%) had trichotillomania. In another report of trichotillomania in children, Oranje et al. (1986) found that the female-to-male ratio was 2.5 to 1 and also that 25% of the children with trichotillomania had associated onychophagy, trichophagy, or automutilation. A small sample of 10 children with trichotillomania was systematically evaluated by Reeve et al. (1992), who found that hair pulling was not always a "benign" habit and was frequently associated with anxiety and affective disorders.

Table 21–13. Lifetime prevalence of psychiatric disorders in a population of 60 chronic hair pullers

Diagnosis	Percentage of sample
Trichotillomania	83%[a]
Mood disorders	65
Psychotic disorders	2
Anxiety disorders	57
Eating	20
Substance abuse	22
No disorder (except trichotillomania)	18

[a]Not all individuals had both an increasing sense of tension before pulling the hair and gratification, or a relief after pulling out the hair (both are required in DSM-IV).

Source. Modified and reprinted from Christenson GA, Mackenzie TB, Mitchell JE: "Characteristics of 60 Adult Chronic Hair Pullers. *American Journal of Psychiatry* 158:365–370, 1991a. Copyright 1991, American Psychiatric Association. Used with permission.

□ Etiology

From a psychoanalytic perspective, hair may have many possible symbolic meanings. According to Krishnan et al. (1985), hair can represent beauty, virility, sexual conflicts, physical prowess, and sexuality; hair cutting or plucking can signify castration. Oguchi and Miura (1977) believe that when trichotillomania occurs in a child, the hair pulling is a manifestation of mild frustration and is analogous to nail biting. In children, the syndrome usually develops at a time of psychosocial stress (Oranje et al. 1986), such as when there is a disturbed mother-child relationship, hospitalization, or when there is family stress associated with raising a mentally retarded child. These authors believe that the hair pulling can develop into a habit even though the stressor(s) is no longer present.

Stroud (1983) points out that "trichotillomania in adolescents and adults may indicate a more serious psychological problem and require psychiatric help" (p. 648). Krishnan et al. (1985) noted that trichotillomania can be present as a major symptom in OCD, mental retardation, schizophrenia, borderline personality disorder, and depression. Some authors have even questioned the validity of trichotillomania as a unique diagnostic entity (Dean et al. 1992; Werry 1990).

Recent literature has suggested that trichotillomania might be a type of OCD (Jenike 1989; Swedo et al. 1989). Stanley et al. (1992), however, report a number of differences between OCD and trichotillomania, including the fact that hair pulling is usually associated with pleasure, patients with trichotillomania have few associated obsessive-compulsive symptoms, and the two groups differ in terms of anxiety, depression, and personality characteristics. In addition, findings from regional cerebral blood flow studies of women with trichotillomania appear different from those of women with OCD (Swedo et al. 1991). In a single case report (Graae et al. 1992), a 13-year-old girl's OCD responded to fluoxetine and to clomipramine, but her trichotillomania responded minimally to each medication.

□ Treatment

According to Dean et al. (1992), "treatment modalities reported to be successful in ameliorating pathologic hair-pulling include a large part of the armamentarium of psychiatry and clinical psychol-

ogy" (p. 89). Consequently, there is no specific treatment for trichotillomania; rather psychoanalytic, behavioral, or pharmacological treatment may each potentially decrease hair pulling.

Recently, Swedo et al. (1989) found that clomipramine was significantly more effective than desipramine in a double-blind crossover treatment of trichotillomania. This study has been criticized for selection bias toward OCD patients, because 9 of 20 subjects were self-referred to the study after a television advertisement on OCD (Dean et al. 1992). Christenson et al. (1991b), in another placebo-controlled, double-blind crossover study, reported the response of 21 patients with chronic hair pulling to fluoxetine. In this 6-week trial, fluoxetine was no better than placebo. In a 16-week open-label trial of fluoxetine, Winchel et al. (1992) reported that 8 of 12 patients treated had a meaningful decrease in hair pulling. Christenson et al. (1991c) reported success in an open trial using lithium carbonate; 8 of 10 patients on lithium had mild to moderate improvement. Stein and Hollander (1992) found that augmentation of serotonergic agents with pimozide (a dopamine blocker) led to improvement in 6 of 7 patients.

Isolated case reports of successful treatment of trichotillomania have been published discussing the use of medications (chlorpromazine [in a schizophrenic individual]; amitriptyline; a monoamine oxidase inhibitor), hypnosis (with and without other treatments), and numerous behavior modification techniques (Krishnan et al. 1985).

□ Course and Prognosis

According to Stroud (1983), most cases of trichotillomania in young children resolve spontaneously. In younger children, trichotillomania usually represents a transient behavior in response to a psychosocial stressor, or it may represent a habit, without the presence of an obvious precipitant. However, if hair loss persists, psychiatric consultation is indicated, and inquiry into areas of parent-child relationships or other areas of potential conflict may illuminate the problem. Oranje et al. (1986), in their study of 21 children under the age of 15, found that psychiatric consultation was necessary in 11 cases (52%), with 4 of these consultees (19%) requiring psychiatric intervention. Sullivan (1989) has raised two issues in patients with trichotillomania: the possibility of trichophagia (i.e., hair eating) leading to the complication of a trichobezoar, and the rare possibility that iron deficiency may cause the hair pulling/eating behavior.

Psychiatric evaluation is indicated when trichotillomania occurs in adolescents and adults. Trichotillomania in adults, as Christenson et al. (1991a) have noted, "follows a chronic course, frequently involves multiple hair sites, and is associated with high rates of psychiatric comorbidity" (p. 370).

■ IMPULSE CONTROL DISORDER NOT OTHERWISE SPECIFIED

The DSM-IV criteria for impulse control disorder not otherwise specified (ICDNOS) are essentially unchanged; therefore, the diagnosis of ICDNOS remains a residual category for impulse control disorders that do not meet the criteria for other impulse control disorders discussed in this chapter.

The Impulse Control Disorders Committee of the DSM-IV Task Force reviewed diagnoses such as *amok*, pathological spending, pathological shopping, and self-mutilation disorder for listings as examples of Impulse Control Disorders in DSM-IV. The Task Force determined that insufficient scientific data existed to support including any of these conditions as examples of an ICDNOS diagnosis.

■ CONCLUSIONS

The disorders that are considered impulse control disorders not elsewhere classified represent a diverse array, including intermittent explosive disorder, kleptomania, pathological gambling, pyromania, trichotillomania, and impulse control disorder not otherwise specified. The disorders in this diagnostic category, when diagnosed using DSM criteria, are rare, except for pathological gambling. For example, in the United States literature during the last 20 years no new cases of pyromania were reported. The limited number of available cases hampers research and gathering of information on the epidemiology, treatment, course, and prognosis of these disorders, as well as refinement of diagnostic criteria.

Promising research is underway, especially on the relationship between serotonin, selective serotonin reuptake inhibitors, and impulsivity. This research may help clarify this diagnostic category, as well as answer questions as to whether the impulse control disorders not elsewhere classified are part of an affective spectrum that includes disorders such as obsessive-compulsive disorder, eating disorders, and major mood disorders.

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