The Intermittent Explosive Disorder

by JOHN R. LION, MD

The intermittent explosive disorder (IED), currently under evaluation for continued inclusion in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV), is an entity of complex heritage. In large measure, the concept of ictal aggression reflects Monroe's work[1] on the episodic disorders in which limbic dysfunction has been correlated with a variety of neurotic, psychotic, and behavioral disturbances. Much earlier, Menninger[2] had conceptualized a "third order of dyscontrol" characterized by explosive outbursts of psychotic rage. And in DSM-II, the term "Explosive Personality" had described character pathology with outbursts of aggression. Thus, explosive violence has historically been associated with a wide array of dysfunctions ranging from functional to neurophysiologic disturbances.

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The IED was first formally conceptualized in the 1980 edition of DSM-III. It was one of two aggressive disorders subsumed under the rubric of the Disorders of Impulse Control; among them was also the isolated explosive disorder representing a unitary outburst of catastrophic rage. The isolated explosive disorder was deleted from the revised version of DSM-III[3] because it was seen as a one-time condition of uncertain *etiology*, most likely the result of psychosis.

The IED itself also underwent changes in DSM-III-R[3] as a result of disagreement among members of the Task Force charged with the revision of the Disorders of Impulse Control. Some clinicians questioned the phenomenology of an entity characterized by a normal baseline state upon which rage outbursts were superimposed. Others felt that outbursts of anger could be seen in many other disease entities—a view that continues to plague the existence of the IED. And most important of all, there began to be doubt about "ictal" aggression, sufficient doubt to remove the neurologic flavoring of the IED in DSM-III-R. Eliminated, therefore, were the paragraphs *from* the Essential Features section describing prodromal affective or autonomic symptoms, changes in sensorium, amnesia, and Associated Features that described such parameters as nonspecific EEG changes or signs and symptoms reflective of subcortical dysfunction.[3] Both scientific and political developments had contributed to this doubt.

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Monroe's work on ictal aggression had been complemented by the efforts of other clinicians to understand seizure states accompanied by violence. Mark and Ervin,[4] working with implanted limbic electrodes in animals and man, showed that amygdala stimulation could lead to rages, and lesions to pacification. Emboldened by their findings, these authors had gone on to suggest in their monograph that all forms of human aggression reflected limbic dysfunction. Such a generalization created a great outcry on the part of both the scientific community and the National Institutes of Health, who challenged the notion of epileptoid violence.[5,6] The net effect was to induce great caution toward the ictal view of violence. Simply put, it appears that not all temper-proneness reflects limbic dysfunction. Patients may have temper outbursts, but not have limbic system dysfunction—at least not diagnosed as such.

Other research cast a shadow on the IED. In looking at how accurately the actual diagnosis of the IED was made, Monopolis and Lion[7] determined that subjective parameters were used more prominently than the requisite Essential Features defined in the DSM-III. And in preparation for the forthcoming DSM-IV, Wise and coworkers (Wise MG, Gray K, Tierney J, et al. Intermittent explosive disorder: a review of the literature for DSM-IV, unpublished data) reviewed literature pertaining to the IED and found "pure" cases of the disorder to be rare; that is, the IED was invariably linked to organic dysfunction.

The conclusions of both studies were both obvious and troubling. There already existed an entity in the DSM called the Organic Personality Disorder, Explosive Subtype. It made sense to relegate temper-proneness here. But how should nonorganic temper-proneness be identified? And, once again, was there such a thing as an IED?

It seemed to the DSM-IV Work Group in the Disorders of Impulsive Control that there was "pure" temper-proneness, but such a condition needed to be clearly delineated from other diagnostic entities. For example, rages were often seen in the borderline personality disorder. Manic conditions, likewise, might include temper, as could certain of the psychotic conditions. Consequently, the IED is presently conceptualized in the DSM-III Options Book as an entity of exclusion. It is paroxysmal violence without a clear organic etiology, unrelated to other DSM entities containing violence.

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AGGRESSION IN NOMENCLATURE

Although violence is commonly encountered in clinical practice, its diagnostic acknowledgement within psychiatry has always been problematic. To a large extent, this also reflects history; thus Freud himself never fully developed a theory of aggression. Violence, unlike anxiety or depression, has long been a curious stepchild symptom, often relegated to the sphere of criminality and deviance. This is at variance with its clinical occurrence.

Few practitioners have been spared encounters with agitated and rageful patients; indeed, violence spans the entire gamut of neurotic, psychotic, and characterologic disorders. Yet there are very few places in which to put the symptom, and aggression rarely justifies the continuation of hospital stay in this day of managed care scrutiny. Temper-proneness is often debilitating, yet insurance companies would tend to view it with skepticism; the affective disorders such as panic or depression are generally viewed as much more traditional and more worthy of therapeutic intervention. Aggression continues to be viewed as indicative of "badness" and not a reflection of "madness."

DIAGNOSIS OF THE IED

As stated, the IED is currently a diagnosis of exclusion. Consequently, the clinician's principle efforts should be to rule out an organic etiology. Neurologic examination, neuropsychiatric assessment, and an EEG (sleep study) may be indicated. If a definitive seizure condition is found, the diagnosis shifts from the IED to the former Organic Personality section, currently referred to in DSM-IV Options Book drafts as "Personality Change Disorder, Disinhibited Type." If there is equivocal finding of brain dysfunction, the diagnosis of IED can be made. Other evidence of organicity includes neurologic "soft" signs, or neuropsychiatric testing. The more specific the organicity, the more the diagnosis shifts to the organic realm; the vaguer, the more the IED diagnosis is utilized.

The IED should not be diagnosed if another condition can explain the aggressiveness of the patient; the case of the borderline personality disorder has already been mentioned. But other existing pathology may not include descriptions of violence. For example, the obsessive-compulsive personality is occasionally prone to temper outbursts and it is common in practice to see overcontrolled patients whose affect overwhelms them. But descriptive parameters for this disorder do not include a tendency to ragefulness. Thus, the clinician confronted with such a patient would make two diagnoses if applicable: the primary personality disorder and the IED.

The IED diagnosis can be made concurrent with other disorders. Depressive states, for example, may include temper-proneness in association with agitation. The clinician must decide if the irritability of the patient can be accounted for

by the primary affective disorder, or if an additional diagnosis of IED is warranted. The same is true for psychotic conditions.

The description of the IED disorder roughly follows that of DSM-III-R. Further refinements in the actual text of the Diagnostic and Associated Feature sections are currently being formulated by the Task Force.

TREATMENT

The elements of therapy for the IED have been mentioned elsewhere.[8,9] In brief, treatment is pharmacologic, psychotherapeutic, and social/behavioral. With the exception of a new class of serenic compounds currently being tested in Europe for antiaggressive efficacy,[10] there are not specific pharmacologic agents for use in the treatment of the IED. Anticonvulsants such as carbamazepine have been utilized in the absence of definitive seizure activity. Lithium and beta-blockers have been used nonspecifically. Antianxiety agents such as the benzodiazepines can be employed to treat certain obsessive patients given to tension states and explosive outbursts; the same drugs may be useful for paranoid patients who find their anger building up. For example, a PRN regimen of alprazolam or lorazepam may be useful in quenching unbearable affect that spills over into explosive anger.

Rarely is medication alone sufficient for treatment. Patients experiencing the IED often have little affective awareness and need education to recognize when they are becoming angry. Identification of psychosocial stressors is crucial to avoid tempers. Knowledge about psychodynamics helps patients grapple with stimuli that would otherwise trigger rages. Deconditioning is helpful. Thus patients need to be taught to verbally (and appropriately) defuse their rages.

CLINICAL EXAMPLES

Case 1

A 30-year-old postal worker is referred by his employer because of temper outbursts on the job. He has become enraged at his supervisor who has criticized his productivity. During these outbursts, he has been verbally threatening and has, on several occasions, thrown articles in the workroom. Once, he struck the supervisor and was suspended from work.

On mental status examination, the patient exhibits mildly paranoid characterologic traits. He is suspicious, defensive, and a brittle man with limited education and insight. He has no history of head injury, nor any evidence of organicity.

He agrees to a PRN trial of alprazolam. A small amount of Xanax, 0.25 mg, is furnished him and he is instructed to take one when he feels himself become enraged. At the same time, he is to leave the premises for a 15-minute "time out" until the medication takes effect. This strategy works, and he is able to be calmer and more productively confrontative with his supervisor. In continued therapy, he talks about some issues of self-esteem and displays introspectiveness.

Case 2

A 36-year-old surgeon is referred by his colleagues because of temper outbursts in the operating room. He is described as an extremely compulsive man who insists that everyone conform to his perfectionist standards of work. Recently, while operating, he threw an instrument at a nurse who did not react to his demand quickly enough. With patients, he is very brusque and intolerant.

He has struggled with both temper and depression all his life. He is an anhedonic man with few pleasures in life. He incurred head injury in an automobile accident in childhood and was unconscious for several hours without neurologic sequelae. A sleep EEG reveals some left-sided slow waves. No seizure activity is noted.

In weekly psychotherapy, the nature of his moodiness and temper becomes apparent. Both are significant. Eventually, he is placed on Tegretol, 800 mg QD. The drug has a stabilizing effect on the rages and evidently helps him to regulate his mood as well.

COMMENT

The above cases show the complexities of empirical treatment. The latter case reveals the further complications of diagnosis, for both mood instability and temper-proneness existed. Base rates are important parameters of both disease and treatment. To have temper-proneness, a patient must have "several" episodes of rage. How many constitute pathology is a subjective matter. But treatment must, in part, be measured by a reduction in both the quality and quantity of the outbursts.

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