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Pathological Intoxication and Alcohol Idiosyncratic Intoxication—Part I: Diagnostic and Clinical Aspects

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ABSTRACT: The concept of pathological intoxication and its successor term, alcohol idiosyncratic reaction, has been one of ambiguity and professional disagreement. The history of such an entity reveals contradictions and varying usage—particularly in regard to the amount of alcohol required. Some feel that the current classification system is broad enough to include such reactions without the use of such terms. Laboratory and electroencephalographic findings are not diagnostic. The author suggests that if the concept is to be retained, psychiatrists utilize where possible the term "alcohol idiosyncractic reaction" in accord with *Diagnostic and Statistical Manual of Mental Disorders* (DSM) III guidelines, particularly in view of the medicolegal implications discussed in Part II, which follows as a separate paper.

KEYWORDS: psychiatry, intoxication, alcohol, pathological intoxication, responsibility, alcoholism

The practice of psychiatry requires meticulous attention to the use of words in an exact, uniform, and unambiguous fashion. Scientific studies would be chaotic if each clinician or researcher used language in an individualized way or if several languages were in use, each using identical words but with different meanings. The goal of a standardized diagnostic system can be met only if the users of medical terminology all use the same words. As the meaning of words changes, the use of the scientific literature becomes immensely complicated unless the reader is attuned to the evolution of the terminology. Similarly, unnecessary confusion arises when medicine interacts with other social systems such as the law. Lack of clarity may also open the door to misuse or abuse of psychiatric concepts.

The purpose of this and its companion paper is the clarification of the concept of pathological intoxication (PI) and its successor, alcohol idiosyncratic intoxication (AII), both in its clinical and legal dimensions. For simplicity, the abbreviation PI will generally be used to reflect both terms.

The DSMs and the Diagnosis

Diagnostic and Statistical Manual of Mental Disorders (DSM) I used a coverall diagnosis of acute brain syndrome, (ABS), alcohol intoxication to refer to recoverable ABSs, such as delirium tremens (DTs) and alcoholic hallucinosis as well as simple intoxication with an

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acute brain syndrome. Pathological intoxication was also included in this category, referring to a marked behavioral or psychotic reaction with ABS after minimal alcohol intake and where there was no preexisting mental disorder. Where a preexisting disorder was made more manifest after minimal alcohol intake, the case would be classified under the diagnosis of the underlying condition. Thus no specific requirement of psychosis was needed for a diagnosis.

DSM II defined pathological intoxication as "an acute brain syndrome manifested by psychosis after minimal alcohol intake." This short description was quite explicit in each of the required elements. That which would distinguish such intoxication from other alcohol reactions is the requirement of minimal alcohol intake.

DSM III altered the criteria by focusing on behavioral change with aggressivity and does not specify that an acute brain syndrome or psychosis must be present.

The essential behavioral change is related to the recent ingestion of an amount of alcohol insufficient to induce intoxication in most people with usually subsequent amnesia for the period of intoxication. The behavior is atypical of the person when not drinking—for example, "a shy, retiring, mild-mannered person may, after one weak drink, become belligerent and assaultive." During the episode, the individual seems out of contact with others. The duration is quite brief and ceases after a few hours. The individual returns to a normal state as the blood alcohol falls (although if the individual had but one drink, the blood alcohol levels themselves would be insignificant). Such a patient may do harm to himself, herself, or others. A small percentage of individuals reportedly show temporal spikes on the electroencephalogram (EEG). Loss of tolerance has been postulated in some. Some may show evidences of brain injury. Fatigue and debilitating illnesses may lower tolerance. Differential diagnosis includes a similar reaction to other substances such as barbiturates and temporal lobe epilepsy which may be associated with "fits" of destructive rage. Malingering is a particular consideration where criminal charges are present and the person may attribute the adverse behaviors to a small amount of alcohol.

The essential point of all the definitions is the marked change with minimal alcohol intake.

Despite the adoption of the terms as a diagnostic entity, considerable disagreement exists as to the frequency of the reaction and its merit as a clinical entity.

Historical Review

Banay [*1*], in his review of the literature in 1944, attributes the expression "pathological reaction to alcohol" to Krafft-Ebing, who described the condition in 1869. Other terms used were pathological intoxication, complicated intoxication, and atypical intoxication. At that time, pathological intoxication referred both to short psychotic reactions to minimal intake of alcohol as well as to an atypical reaction in a chronic alcoholic. In New York and nationwide, the diagnosis was used in 2 to 9% of admissions for alcoholic psychosis. Among his criteria, Krafft-Ebing noted (1) the effect was unrelated to alcoholic intake, (2) the timing occurred both early in drinking and after drinking had stopped, (3) the quality differed from simple intoxication, (4) movements were uncoordinated, and (5) amnesia was present for the entire period.

Banay also summarized the observations of Heilbronner, Bonhoeffer, Voss, Schroeder, Silbert, Meggendorfer, Seelert, Binswanger, and Bleuler.

Heilbronner described anxiety, loss of orientation, misrepresentation of the environment, visual and auditory hallucinations, sudden onset, short duration, and sleep followed by amnesia. This definition had similarities both to delirium tremens and alcoholic hallucinosis.

Bonhoeffer described both psychotic symptoms and epileptoid characteristics with motor agitation and rage. A person might react with violence to the slightest provocation from the environment.

Voss stressed the differentiation from alcoholic twilight states related to hysteria and trauma. Because of the overlapping and ambiguity of terminology, various authors have described the panoply of symptoms associated with alcoholic psychoses. Schroeder suggested the term "transitory alcoholic psychosis," with numerous predisposing factors such as personality, exhaustion, overexertion, skull injuries, and carbon monoxide poisoning. Silbert felt that the criteria should include previous occurrence of a pathological reaction to alcohol and that amnesia was not a sufficient criterion.

Others included overactivity of speech and behavior and abortive delirium. Some focused on reduced or absent pupillary and deep tendon reflexes. Meggendorfer described epileptoid and delirious forms as well as the frequency of sexual offenses, usually involving exhibitionism and homosexuality. Seelert felt that there was no effective external provocation and stated that hypoglycemia was involved. Binswanger felt an epileptoid reaction was the essence of PI and was present almost exclusively in psychopaths. It was also felt to be common in the mentally retarded. Binswanger differentiated quantitatively abnormal and qualitatively abnormal forms. Bleuler's view was that the delirious form of PI was related to abortive DTs and that the reaction was also related to schizoid character and schizophrenic episodes.

Bowman and Jellinek, in their review [*2*], stated that PI was a direct effect of alcohol, frequently as a result of small amounts of alcohol, with fits of rage that were not constant but required special conditions such as temporary dysphorias or hypoglycemia. Lewis [*3*] refers to pathological intoxication as a "drunken furor," produced by a very large amount of alcohol or by a small amount in one who is intolerant to the substance.

May and Ebaugh [*4*] stated that PI bears a greater relation to legendary figures such as the minotaur and the centaur than to clinical reality and that claims of PI have been made in cases of alcohol ingestion followed by antisocial behavior with a subsequent claim of amnesia. Many do not show psychoses, and "senseless" violence is often emphasized as an important diagnostic point—another ambiguous concept. No characteristic physical findings are valid indications of the condition. Of 732 cases of alcoholic psychosis, 41 were classified as PI (there were also 1289 cases of chronic alcoholism without psychosis). In none of the cases reported by May and Ebaugh was there evidence of minimal intake. They felt that there was no justification for the use of PI as a special diagnostic category, that it was a diagnostic catch-all, and that in all their cases the symptoms were related to alcohol intoxication and that other diagnostic categories and consideration of dynamic factors were adequate for logical classification and diagnosis.

Tinklenberg et al. [*5*] discussed alcohol as a disinhibitor in their review of criminal assault by adolescents and noted that alcohol and barbiturates are central nervous system (CNS) depressants that are linked with the phenomenon of increased aggression without necessary resort to the concept to PI.

The EEG and Blood Alcohol Levels

Greenblatt et al. [*6*] reported that three of five cases of PI had abnormal EEGs, with slow and rapid potentials but with none showing paroxysmal dysrhythmia.

Bach-y-Rita et al. [*7*] studied the relationship of the EEG to PI in ten patients. These patients had sporadic drinking, depression, insomnia, and overt psychosis followed by sleep. They were given 150 to 300 cm^3 of a 25% solution of alcohol. All showed clinical signs of intoxication with mystagmus, slurred speech, tremors, incoordination, and somnolence. None showed seizure activity on the EEG. All had problems in sexual identity. Bach-y-Rita felt that perhaps the low stress environment of the project accounted for the lack of seizure activity.

Maletzky [*8*] discussed the confusing literature on PI and the lack of support in the literature for the concept, noting many authors question the merit of this type of disinhibition as a separate clinical entity and the notion that minimal alcohol intake is required. Further, the

concept of pathological intoxication implies that there is such an entity as "normal intoxication." Various authors have noted that alcohol can activate the EEG in those with a history of violence, that alcohol does not produce violence or psychosis in those without a prior history, and that alcohol can produce aggression and psychosis in those with such a history. He administered alcohol in a 25% intravenous drip at a rate of 200 cm^3 per hour. EEG changes occurred in 15 of 22 patients, usually with generalized slowing with occasional sharp waves. Two with no behavioral change showed no EEG change, while two with psychotic behavior showed no EEG change. Nine became violent, four psychotic (hallucinations and delusions), two mixed, and seven showed a normal reaction with signs only of simple alcohol intoxication. Patients had as much as 1200 cm^3 of the 25% solution with blood alcohols varying from 0.095 to 0.275%; the mean infusion was 980 cm^3, equivalent to 15 cans of beer. All slept heavily after the procedure. This study did not support the concept of minimal alcohol intake; obviously all had significant alcohol intake. Maletzky [*9,10*] has also reviewed the subject of episodic dyscontrol, which overlaps the concept of PI.

Kosbab and Kuhnley [*11*] and Skelton [*12*] each present single cases that they felt represented PI. Skelton's case showed some EEG slowing when alcohol was administered in a case where the patient had three episodes of destructive behavior in association with alcohol and a homicidal act "inconsistent with the subject's reputation in the community."

Criticisms and Other Comments

Coid [*13*] in his thorough review dates the reaction to Krafft-Ebing's work in 1869 and traces its roots from "pathologische alkoholreaction" to mania a potu, temporal lobe epilepsy, abortive delirium tremens, and the recent pathological intoxication. He noted the varying clinical standards and differing thoughts about etiology of various authors. He concludes:

> The literature on PI is confusing, so that a psychiatrist confronted with so motley an array of opinions is unlikely to make use of such a 'label'. . . . The origin of a 'small amount' of alcohol as a crucial factor is unclear from the literature; there is no study based on eye-witness accounts or experimental evidence that 'small' quantities of alcohol have ever induced the condition. It is also impossible to define a 'relatively small amount,' as the dose-related effects vary so much between different individuals and in the same individual at different times . . . (T)he majority of experimental studies have unsuccessfully attempted to find an underlying aetiological explanation for PI. The subjects involved are likely to have been a highly heterogenous group, due to lack of adequate diagnostic criteria, which may explain to some extent why few workers agree and why no aetiological factors have been established. . . . The possibility that PI is an epileptic phenomenon induced by alcohol has been extensively examined without being confirmed. . . .
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> However, the need for a diagnostic category to span the gap between murder committed while drunk and the range of the McNaughten Rules cannot justify the manufacture of a fictitious label which, furthermore, risks shifting the responsibility away from the individual to an assumed cerebral disease or to the unwarranted power of the drug itself. Although the concept of PI has survived from the last century, it may well be that with further research there is now sufficient diagnostic understanding to allocate many states of supposed PI to more appropriate categories. Meanwhile, the present inflexible and unsupported criteria would appear to have rendered such a nosological entity obsolete.

Hollender [*14*] expressed the view that so-called PIs represented dissociative reactions and that PI as an entity remains in doubt. He concluded:

> It is high time that we ask ourselves if a notion without substance is being perpetuated. Are we attributing a disturbed state to pathological intoxication when alcohol is incidental rather than causal—merely a culturally-fostered rationalization? We should accept the diagnosis of pathological intoxication only if in suspected cases changes in the EEG and in behavior can be produced by the administration of alcohol under double-blind conditions.

Cohen [*15*] restricts his use of PI or AII to the standards of DSM III, pointing out that PI is a rare but definable condition, related to minimal intake, behavior differing from the person's usual comportment, explosive fury, and so forth. He, too, reviews the arguments over its existence, comparing it to amok, negi negi, phencyclidine toxicity, amphetamine and cocaine reactions, and so forth, but these represent reactions to chemicals and not the hypersensitivity of a few that is the current hallmark. He notes, as did others, that persons may lose tolerance to alcohol after other organic disorders. He comments that "in order to make a secure diagnosis of (PI), a blood alcohol concentration of less than 0.1% would seem necessary." Such a criterion would eliminate Maletzky's cases with their significant blood alcohol: "Otherwise (PI) could not be differentiated from alcohol intoxication with an associated rage reaction."

The issue of reproducible noxious behaviors as confirmatory is another area of dispute. Cohen concluded:

> A wide variety of bellicose disturbances that happen to occur in connection with drinking are randomly grouped together by some clinicians as (PI). These include psychomotor epilepsy, (DTs), violence resulting from drunkenness, and certain dissociation states. Unfortunately, no reliable objective test is available to support the diagnosis.

Conclusions

This cursory review of relevant literature reflects the chaos in the use of the concept PI or AII. Previous studies indicate clearly that the expression PI expands and contracts according to its use by a given author and that differentiation from other alcohol states, other psychiatric conditions, and a variety of related factors is most difficult. Because of this, the literature itself is only of limited use.

Many authors have questioned whether there is a distinct entity meriting a classification of PI. In particular, the use of the term implies that there is such a thing as normal intoxication, although the person is suffering from a toxic poisoning in either case. No criteria exist physically or by electroencephalography that provide significant support for such a diagnosis. If the concept is to be retained, then it probably should be restricted as it is in DSM III to conditions arising with minimal intake—one or two drinks—at a level insufficient to cause drunkenness in a person who characteristically does not drink and who behaves in a way out of keeping with prior modes of functioning.

This would eliminate applicability of much that has been reported—particularly those studies that indicate explosive, poorly explained violent behaviors with significant alcohol intake. Obviously, 15 bottles of beer are not minimal intake.

The literature thus indicates a difference between "*a* pathological intoxication" and "*the* pathological intoxication," the former being broader and including similar reactions but with significant alcohol intake.

Even the use of the former is problematic because it implies a certain type of reaction in association with the use of alcohol which is pathological as contrasted broadly with other severe alcohol reactions such as withdrawal syndromes, DTs, and a plethora of other alcohol-related conditions that overlap considerably.

Psychiatrists therefore should attempt to avoid use of the term PI because of its semantic ambiguity and instead utilize AII (while indicating the interchangeability with PI in communications to attorneys and courts). *The* pathologic intoxication implies a unique legal status in many states. Since it is being used in statutes, psychiatrists may appropriately use the term in medicolegal situations. But they should do so narrowly and in strict conformance with the requirements of DSM III. No therapeutic purpose is gained by using PI otherwise, and the practice of adopting alcohol idiosyncratic intoxication in all future reports would help to create a more uniform usage within the profession. Most important is the recommendation that acute rage reactions, no matter how unclearly motivated, in associations with

significant alcohol intake should no longer be classified as pathological intoxication, an expression that should be allowed to die through deliberate neglect. As review of the legal aspects demonstrates, this is not a picayune matter, but one which has significant social import.

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