

PCP INTOXICATION

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November 1, 2025

RECOMMENDED CITATION

mohammad looti (2025). *PCP INTOXICATION*. PSYCHOLOGICAL SCALES. Retrieved from <https://scales.arabpsychology.com/?p=63251>

PCP Intoxication

Primary Disciplinary Field(s): Psychiatry, Toxicology, Pharmacology

1. Core Definition

PCP Intoxication refers to a clinical syndrome characterized by a transient and **reversible cluster of maladaptive behavioral and psychological changes** that develop shortly after the recent ingestion, inhalation, or parenteral administration of Phencyclidine (PCP) or a related arylcyclohexylamine analog. The essential diagnostic criteria necessitate the presence of clinically significant modifications in behavior or psychology, coupled with at least one verifiable sign of physiological participation. This definition establishes PCP intoxication as a distinct, acute clinical condition arising from the psychoactive and dissociative effects of the drug on the central nervous system.

The core feature distinguishing PCP intoxication from general substance use is the unique combination of physical signs--particularly nystagmus and heightened vital signs--with profound psychological distress, often manifesting as belligerence, impulsivity, or acute psychosis. While the syndrome is described as reversible, the acute phase carries significant risks of morbidity and mortality, stemming both from the drug's direct physiological effects and the resultant unpredictable, often violent behavior exhibited by the intoxicated individual. Furthermore, the severity of the syndrome is often dose-dependent, though individual responses can vary significantly based on tolerance, environment, and co-ingestion of other substances.

Clinically, PCP intoxication falls under the category of Substance-Related and Addictive Disorders within the Diagnostic and Statistical Manual of Mental Disorders (DSM-5). The diagnosis requires that the signs and symptoms are not attributable to another medical condition, including non-substance-related mental disorders, and that they cause clinically significant distress or impairment in social, occupational, or other important areas of functioning. The rapid onset and resolution typically differentiate it from primary psychotic disorders, although the acute effects can closely mimic conditions like schizophrenia, necessitating careful differential diagnosis in the emergency setting.

2. Etymology and Historical Development

Phencyclidine, or PCP, was first synthesized in the late 1950s by Parke, Davis and Company, initially intended for use as an intravenous general anesthetic under the brand name Sernyl. Its chemical structure is that of an arylcyclohexylamine, and it was notable for its ability to induce a state of profound analgesia and amnesia without respiratory depression, which was a common side effect of traditional anesthetic agents. The term **dissociative anesthetic** was coined to

describe the unique state it induced, where patients appeared conscious and responsive but felt detached from their bodies and environment.

Despite its initial promise, clinical use in human medicine was short-lived. By the mid-1960s, PCP was discontinued due to the high incidence of severe postoperative psychological side effects, including agitation, delirium, vivid hallucinations, and distressing psychotic reactions persisting days or even weeks after administration. Although it found some continued use in veterinary medicine, particularly for large animals, its availability led to its diversion into the illicit drug market.

PCP emerged as a significant drug of abuse in the United States, particularly in the late 1960s and 1970s, where it became known by street names such as "Angel Dust," "Rocket Fuel," or "Hog." The recognition of PCP Intoxication as a specific clinical syndrome grew directly out of emergency room presentations involving individuals exhibiting extreme agitation, unexpected strength, and the unique combination of behavioral disturbance and specific physical signs like nystagmus. This period solidified the clinical understanding that PCP toxicity represents a severe medical and psychiatric emergency distinct from other common drug intoxications like those caused by opiates or stimulants.

3. Pharmacological Mechanism of Action

The fundamental mechanism underlying PCP intoxication is its action as a potent, non-competitive antagonist of the **N-methyl-D-aspartate (NMDA) receptor**, a key ionotropic receptor for the excitatory neurotransmitter glutamate. PCP binds specifically to a site within the ion channel pore of the NMDA receptor complex, effectively blocking the flow of ions, primarily calcium, into the neuron. This blockage disrupts normal glutamatergic neurotransmission, particularly in cortical and limbic structures, which is believed to be the primary cause of the drug's distinctive dissociative and hallucinogenic properties.

The NMDA receptor blockade leads to an overall hypofunction of the glutamatergic system. Since NMDA receptors are crucial for synaptic plasticity, learning, and memory, their inhibition produces the cognitive disruption, detachment, and altered perception characteristic of the intoxicated state. Furthermore, this blockade indirectly affects other neurotransmitter systems. PCP also acts as an agonist or reuptake inhibitor at several other receptors, adding complexity to its profile. It increases dopamine levels in the mesolimbic pathway, contributing to its addictive potential and the stimulant-like effects (e.g., paranoia, agitation, and psychosis).

Additional pharmacological targets include the sigma (σ) receptors, where PCP acts as an agonist, and possibly interactions with opioid receptors (specifically kappa and mu), which may contribute to its analgesic properties. The combined effect of NMDA antagonism, enhanced dopaminergic activity, and other receptor interactions culminates in the severe neurophysiological dysregulation observed during acute intoxication. This multifaceted pharmacological profile explains why PCP

intoxication presents as a complex toxic syndrome blending features of anesthesia, stimulant abuse, and severe mental illness.

4. Key Characteristics and Diagnostic Criteria

The diagnosis of PCP intoxication requires both behavioral and physiological evidence following recent use. Behaviorally, the individual typically displays a rapid onset of maladaptive changes that often center around aggression and unpredictability. These changes might include **belligerence, physical assault, impulsivity, marked psychomotor agitation, and severely impaired judgment**. The individual often displays a startling lack of reaction to pain due to the anesthetic properties of the drug, which compounds the risk of self-harm or violent actions toward others.

Physiological manifestations are crucial for confirming PCP intoxication and separating it from purely psychiatric emergencies. The classic physical signs include horizontal or vertical nystagmus (involuntary eye movement), hypertension (elevated blood pressure), and tachycardia (rapid heart rate). Depending on the dose, severe intoxication can present with more alarming symptoms such as generalized muscle rigidity, ataxia (impaired coordination), slurred speech (dysarthria), and diminished responsiveness, sometimes progressing to coma.

A particularly concerning diagnostic feature is the emergence of psychotic symptoms. Individuals may experience delusions, often of a paranoid nature, or visual and auditory hallucinations. This acute PCP-induced psychosis can be virtually indistinguishable from an acute schizophrenic episode, sometimes lasting for several days or weeks even after the drug has been metabolized. The combination of intense paranoia, dissociation, and analgesia contributes to the extreme danger associated with managing an intoxicated patient, who may perceive medical personnel as threats and respond with explosive violence.

5. Clinical Presentation and Psychological Effects

The psychological experience of PCP intoxication is rooted in the powerful dissociative effects. The user feels disconnected, or "dissociated," from their own body, thoughts, and external reality, leading to a profound sense of unreality. This detachment can initially be sought recreationally but often spirals into severe anxiety, depersonalization, and panic attacks, especially as the dose increases or the environment becomes stressful.

The emotional lability seen in intoxication is extreme. Rapid shifts between euphoria, calmness, and profound anxiety or rage are common. As the intoxication deepens, thinking becomes disorganized, and cognitive processes are severely impaired. Users exhibit poor attention, memory deficits, and a loss of temporal and spatial awareness. The impairment of judgment is a critical factor leading to risky behaviors, such as walking into traffic or jumping from high places, fueled by an exaggerated sense of invulnerability coupled with the analgesic effects.

In high-dose intoxications, or "runs," the psychological state transitions into acute delirium, often accompanied by violent behavior. Patients may require significant physical and chemical restraint due to their combative nature and unexpected strength, which is often cited in anecdotal reports but is primarily a function of their profound lack of pain sensation combined with extreme agitation. Following the resolution of the acute intoxication, some individuals report lingering affective and cognitive impairment, often characterized by depression or residual memory difficulties.

6. Physiological Manifestations and Risks

The physiological risks associated with PCP intoxication are extensive and potentially life-threatening. Cardiovascular effects are prominent, including significant **hypertension and tachycardia**, resulting from the drug's sympathomimetic activity, possibly through indirect catecholamine release. While moderate intoxication involves these signs, severe overdose can paradoxically lead to hypotension and bradycardia as the central nervous system depression deepens.

One of the most critical complications is **severe hyperthermia** (malignant hyperthermia). Due to intense muscular activity, agitation, rigidity, and possibly a central hypothalamic effect, the body temperature can rise rapidly to dangerous levels (e.g., above 105°F or 40.5°C). Untreated hyperthermia can lead quickly to irreversible brain damage, disseminated intravascular coagulation (DIC), and organ failure.

Furthermore, the combination of agitation, muscle rigidity (dystonia), and hyperthermia significantly increases the risk of **rhabdomyolysis**--the breakdown of damaged skeletal muscle tissue. Rhabdomyolysis releases muscle proteins (myoglobin) into the bloodstream, which are toxic to the kidneys and can precipitate acute kidney injury or renal failure. Neurologically, seizures are another serious risk, particularly in cases of massive overdose. The immediate management of PCP intoxication must therefore prioritize the control of agitation, the reduction of hyperthermia, and the monitoring of renal function to mitigate these severe physiological sequelae.

7. Management and Treatment

The management of acute PCP intoxication is primarily supportive and focused on stabilization, symptom control, and prevention of complications. Initial emergency treatment follows standard protocols: securing the airway, breathing, and circulation (A-B-Cs). Since the patient is often agitated or violent, the immediate goal is to ensure the safety of the patient and staff, often requiring adequate physical restraints followed by chemical sedation.

The primary pharmacological treatment for agitation and seizures involves **benzodiazepines** (e.g., lorazepam or diazepam). These agents help calm the patient, control muscle spasms, and reduce the risk of hyperthermia and rhabdomyolysis stemming from uncontrolled agitation. For severe,

refractory psychosis or agitation, antipsychotics such as haloperidol may be considered, although caution is advised due to their potential to lower the seizure threshold or exacerbate anticholinergic effects, especially if other substances are co-ingested.

Controlling physiological crises is paramount. Severe hyperthermia requires rapid aggressive cooling, which may involve ice packs, cooling blankets, or internal cooling methods. Hypertension is typically managed using sedatives, but persistent, severe elevations may necessitate specific antihypertensive agents. Historically, techniques such as urinary acidification or forced diuresis were sometimes employed to enhance PCP excretion, as the drug is a weak base, but these methods are generally discouraged today due to the risk of exacerbating rhabdomyolysis-induced renal failure. The patient must be maintained in a quiet, low-stimulus environment to minimize external triggers that could provoke agitation or psychosis.

8. Significance and Impact

PCP intoxication holds significant importance in toxicology and forensic psychiatry due to its unique clinical presentation and the high danger level associated with the acute syndrome. Unlike many other common intoxicants that primarily cause sedation or euphoria, PCP consistently produces a dangerous blend of anesthesia, dissociation, paranoia, and physical strength, making it one of the most challenging intoxications to manage in the acute care setting.

From a diagnostic perspective, PCP intoxication serves as a prime example of an acute substance-induced psychotic disorder. Its ability to produce symptoms virtually identical to chronic schizophrenia has historically informed research into the NMDA receptor hypothesis of psychosis, suggesting that dysregulation of glutamatergic pathways plays a crucial role in the etiology of primary psychotic illnesses. This linkage has driven pharmacological research attempting to modulate NMDA function to treat schizophrenia.

Furthermore, the legal and public health ramifications of PCP use remain critical. Because the intoxication leads to unpredictable, violent, and often amnesic behavior, cases involving PCP often present complex legal and ethical challenges. The ongoing threat of designer analogs of PCP (N-methoxy-PCP, 3-MeO-PCE, etc.) ensures that clinical understanding of the core intoxication syndrome remains essential for emergency medical and mental health professionals globally.

Further Reading

Diagnostic and Statistical Manual of Mental Disorders (DSM-5) - Official criteria for Substance-Related and Addictive Disorders.

Phencyclidine (PCP) - Wikipedia entry detailing chemistry and history.

N-methyl-D-aspartate Receptor - Information on the primary mechanism of action.