At 5:45 a.m., fire department personnel responded to a call concerning an unresponsive person outside a fast-food restaurant. Upon their arrival, they found a 41-year-old, 350-lb. male acting strangely and “becoming a nuisance.” In accordance with standard operating procedures, the fire department requested the assistance of police department personnel. The first patrol car arrived at 5:58 a.m., whereupon the responding engine left the scene. A video-recording device installed in the responding police car recorded one police officer approaching the individual and saying, “You have to tell me what is going on.”

At this point, the individual cursed repeatedly at the officers and attempted to place one in a headlock. After instructing the individual to stay back, the two officers attempted to restrain him, striking him repeatedly with their batons. Although knocked to the ground by the police, the individual continued to struggle, and resisted verbal orders and physical attempts to place his hands behind his back. With the assistance of four additional officers, the individual was successfully handcuffed. At this point, outside the view of the police camera, he ceased struggling. He was rolled onto his back, and an officer said, “He’s still got a pulse. I don’t see him breathing.”

Paramedics responded to the scene, and the patient was pronounced dead soon after arrival at the regional Level 1 trauma center. While the death was officially ruled a homicide by the county coroner, he noted that the patient had an enlarged heart, and that both phencyclidine (PCP) and cocaine were detected on toxicological tests. No evidence of internal injury was noted.1,2

Cocaine, Excited Delirium and Sudden Unexpected Death

By Matthew D. Sztajnkrycer, MD, PhD, & Amado A. Baez, MD, MSc
Effects of Cocaine

According to data from the U.S. Drug Enforcement Administration (DEA), cocaine remains the second most common illicit drug used in the United States. Ten percent of people aged 12 years or over have used cocaine. In 2002, the U.S. DEA seized 61,594 kg of cocaine.

Cocaine exerts its effects through several distinct pathways. Centrally, it modulates the effects of brain neurotransmitters (especially dopamine), accounting for the euphoria associated with cocaine use, but also for the rapid development of tolerance and addiction.1 Cocaine causes epinephrine (also known as adrenaline) release from the adrenal glands, and blocks the reuptake of norepinephrine, thereby resulting in a state of increased physical stimulation.6 Finally, cocaine has effects on the electrical conduction systems of the heart similar to those of tricyclic antidepressants. The concurrent use of alcohol and cocaine produces a new compound, cocaethylene, which lasts longer in the body and has even more potent toxic effects.5,7

Excited Delirium

The term excited delirium (also known as Bell’s mania, lethal catatonia, acute exhaustive mania, agitated delirium) was first used in 1849 to describe psychiatric patients who developed continuous agitation and mania in the presence of fever, then suddenly collapsed and died.9 Excited delirium is currently characterized by the acute onset of bizarre or violent behaviors, including aggression, combativeness, hyperactivity, extreme paranoia, hallucinations, superhuman strength or incoherent shouting.6 Hyperthermia is frequently present.5,6

Fatal excited delirium was first described in seven cocaine users between April 1983 and May 1984.9 Since that time, more than 130 cases of fatal, cocaine-associated excited delirium have been reported in the medical and forensic literature.6,13 In Miami, excited delirium accounted for 10% of male cocaine deaths; 39% of excited delirium deaths occurred when the victim was in police custody.10 In a review of excited delirium deaths during custody, victims were predominantly male (97%), had an average weight of 220 lbs. and a mean body temperature of 104°F.10 No apparent racial differences have been noted in recent studies.11

Although the majority of reported drug-associated fatal excited delirium cases have involved the use of cocaine, other stimulant agents, including LSD, phencyclidine (PCP) and methamphetamine, have been implicated in excited delirium deaths.12,13 In the case presented above, both cocaine and phencyclidine were detected during postmortem toxicological testing.12 Lethal excited delirium appears clinically to consist of four distinct phases, which occur sequentially: elevated temperature, agitation, terrorized delirium, respiratory arrest and death.10

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Patients initially appear agitated to grossly psychotic, and exhibit feats of superhuman strength, especially during attempts to restrain them. In one study, an average of four officers was required to subdue suspects, with a range of 3–6 officers. Shortly after the patient’s being restrained, the violent struggling appears to cease, and a labored or shallow breathing pattern is noted. The patients are usually found dead or near death moments later. Death typically occurs within one hour of first contact with police. More than 75% of patients die either at the scene or during initial transport. In another study, 18 cases of excited delirium death were witnessed by EMS personnel; initial cardiac rhythms were described in 13 cases. In contrast with acute cocaine toxicity, ventricular dysrhythmias occurred in only one patient. Asystole was the most common presenting rhythm. Interestingly, toxicology investigations of these patients demonstrated cocaine levels similar to those found in recreational cocaine users and lower than those found in individuals who died secondary to acute cocaine intoxication.

Considerable attention has been paid to restraint use for controlling patients with excited delirium and the potential for death secondary to restraint. Studies in healthy volunteers suggest that the prone, hog-tied position impairs cardiovascular recovery from moderate exercise. Placing restrained individuals in a prone position has the potential to physically interfere with diaphragm movement. In one study, 18 of 21 deaths occurred in victims in a prone position. Moreover, eight of the 18 deaths occurred under the additional body weight of 1–5 restraining officers. While in one study all excited delirium sudden death patients were found by EMS in the prone position and in hobble restraints, the majority of surviving excited delirium patients were also found in this position. Not all deaths occurred in police custody. Current data neither support nor refute positional asphyxia secondary to prone positioning and use of hobble restraints in the initial arrest of these patients.

The actual cause of cocaine-associated excited delirium and sudden death remains to be determined. Studies have suggested that the elevated temperatures seen in these patients is due to abnormal changes in brain dopamine receptors, while violent behaviors may be due to changes in other neurotransmitter receptors. Levels of brain dopamine transporter proteins are also abnormal in these patients. The vast majority of patients died after a struggle. Struggling increases the levels of circulating epinephrine (adrenaline), the so-called fight-or-flight response, which may place further stress upon the heart. Animal studies have shown that rats given cocaine and then restrained are three times more likely to die than rats given the same dose of cocaine and allowed to wander freely about. Similarly, rats given cocaine and an infusion of epinephrine, norepinephrine and dopamine stopped breathing sooner than rats simply given cocaine. These effects were dose-related. The heart stress caused by elevated levels of these hormones, such as would be expected during a prolonged struggle, would only be worsened by any breathing impairment.

Any death in police custody can be expected to cause significant repercussions. Seven of 11 cases of excited delirium-associated death led to wrongful-death lawsuits against the agencies involved. The case presented at the beginning of this article received national attention; the video played repeatedly on CNN. The FBI, the Justice Department’s Civil Rights Division and the NAACP all began independent investigations. In circumstances where resuscitation is unsuccessful and excited delirium-associated death is suspected, it has been suggested that postmortem examination of the brain is capable of confirming the diagnosis.

While unexpected death is by definition unexpected, the stunning similarities observed in all these excited delirium cases provide law enforcement and EMS person-
nel with potential warning signs. Since the actual etiology of fatal excited delirium is not currently known, specific management guidelines do not exist. However, there are certain principles of general management when caring for these patients. Prompt recognition of excited delirium by first responders and strong cooperation between emergency medical services and law enforcement are essential in managing these patients. All individuals who demonstrate evidence of excited delirium should immediately be taken to a medical facility for evaluation, rather than to a law-enforcement facility. Individuals should be placed in a non-prone position as soon as possible. Continuous oximetry should be instituted to both detect hypoxia and document the absence of positional asphyxia, and cardiac monitoring should be initiated, given the potential for rapid deterioration. Rapid determination of blood glucose should be performed in all patients with altered mental status.

Benzodiazepines are the first-line treatment of psychomotor agitation in cocaine and other stimulant toxicity. Doses should be titrated to clinical effect, specifically the resolution of agitation. Beta-adrenergic receptor antagonists are contraindicated in the setting of sympathomimetic drug toxicity, due to the potential for unopposed alpha-adrenergic receptor stimulation, with subsequent vasoconstriction and end-organ toxicity. Similarly, neuroleptic medications such as haloperidol are contraindicated, due to adverse effects on temperature regulation, lowered seizure threshold and potential for dysrhythmias. Finally, although rarely documented, elevated temperature has been noted in many of these patients. Patient temperature should be recorded as soon as feasible and aggressive temperature control measures, analogous to those used in caring for heat-stroke patients, should be instituted. Aggressive fluid support, correction of acid-base status, and detection and treatment of rhabdomyolysis in a hyperthermic, agitated patient may be beneficial.

The cessation of struggling by an agitated excited delirium patient should be regarded as an ominous, near-terminal event, as should the development of shallow or labored breathing, and should immediately prompt aggressive evaluation and reassessment. The initial decompensation in these patients appears to be respiratory arrest, rather than cardiac arrest. In the presented case, a police officer was heard saying that the individual had a pulse but was no longer breathing. It is as yet undetermined whether aggressive airway management and advanced cardiac life support protocols at the onset of these symptoms might be lifesaving, or whether these near-terminal events represent an already intractable process. Ventricular dysrhythmias were noted in only one patient in the study by Stratton, et al. As such, defibrillation will likely not be beneficial in the management of these patients. Based upon available animal studies, the use of epinephrine for resuscitation of excited delirium patients in extremis may be contraindicated.

**Conclusion**

Cocaine-associated excitation delirium is responsible for approximately 10% of all cocaine deaths. While the death is often referred to as “unexpected” by responding personnel, the purpose of this article has been to make law enforcement and EMS personnel aware of the well-characterized progression of symptoms leading to death. It is hoped that increased awareness of warning signs might prevent future deaths.

**References**


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