



Excited Delirium and Sudden Unexpected Death

Matthew D. Sztajnkrycer, MD, PhD

Amado A. Baez, MD, MSc

Department of Emergency Medicine

Mayo Clinic

Rochester, MN

During a three-day period in June 2004, 3 individuals died after being restrained by police in Florida, Minnesota, and California. All had exhibited bizarre behavior necessitating police intervention. All arrests involved struggle and subsequent restraint, including one situation where the individual was struggling with neighbors prior to police and EMS arrival. All arrests eventually required the deployment of OC spray, a TASER device, or both. In the California case, the suspect was evaluated and cleared by EMS prior to being transported to jail. Within two miles from the scene, the patient began to complain of difficulty breathing and subsequently died. In the Minnesota case, the suspect became unresponsive shortly after being cuffed, and was pronounced dead at a local emergency department. In the Florida case, the patient was taken to an emergency department where he died. All final autopsy reports are pending at this time.

These cases show a striking similarity to a recent Cincinnati, Ohio case, which received international attention. During a videotaped arrest, an agitated male suspect attacked the responding officers. The two officers subsequently attempted to subdue the suspect, striking him repeatedly with their batons. Although knocked to the ground, he continued to struggle, requiring a total of 6 officers to place him in handcuffs. At this point, outside the view of the police camera, the suspect ceased struggling. An officer can be heard on the videotape stating "He's still got a pulse. I don't see him breathing." The suspect was pronounced dead soon after arrival at the emergency department. The Hamilton County coroner noted that the suspect had an "enlarged heart", and that both PCP and cocaine were detected on toxicological tests. No evidence of internal injury was noted.

The term excited delirium (ED) was first used in 1849 to describe psychiatric patients who developed onset of continuous agitation and mania, in the presence of fever, and then suddenly collapsed and died. Fatal ED was first described in 7 cocaine users between April 1983 and May 1984 [1]. Since that time, more than 130 cases of fatal, cocaine-associated ED have been reported in the medical and forensic literature [1-5].

Fatal ED appears clinically to consist of 4 distinct phases, which occur sequentially: elevated temperature, agitated delirium, respiratory arrest, and death [6]. Patients initially appear agitated to grossly psychotic, and exhibit feats of superhuman strength, especially during attempts to restrain them. Shortly after being restrained, the violent struggling appears to cease, and a labored or shallow breathing pattern is noted [2-4]. The patients are typically found dead or near dead moments later. Death typically occurs within 1 hour of first contact with police [2]. More than 75% of patients died either at the scene or during initial transportation [2]. In one study, initial cardiac rhythms were described in 13 cases [3]. In contrast with acute cocaine toxicity, ventricular dysrhythmias occurred in only 1 patient. Asystole was the most common presenting rhythm.

The actual cause of cocaine-associated ED and sudden death is unknown. Studies have suggested that the elevated temperatures seen in these patients is due to abnormal changes in brain dopamine receptors [7]. The vast majority of these patients died after a struggle. Such struggles increase the levels of circulating epinephrine [5,6], and may also result in a metabolic acidosis.

While unexpected death is by definition unexpected, the stunning similarities observed in all these fatal ED cases provide law enforcement and EMS personnel with potential warning signs. All individuals who demonstrate evidence of ED should 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, and continuous oximetry instituted to document the absence of asphyxia.

The cessation of struggling by an agitated ED patient should be regarded as an ominous, near-terminal event, as should the development of shallow or labored breathing. The initial decompensation appears to be respiratory arrest, rather than cardiac arrest. Aggressive airway management and advanced cardiac life support protocols might be life-saving in these circumstances, although there is insufficient data to make any firm conclusions. Aggressive temperature control measures, analogous to those used in caring for heat-stroke patients, should be instituted.

In conclusion, excitatory delirium has been attributed to approximately 10% of all cocaine deaths [6]. While the death is often referred to as "unexpected" by responding personnel, there is a well-characterized progression of symptoms leading to death. It is hoped that increased awareness of warning signs might prevent future deaths.

References

1. Rutenber AJ, Lawler-Heavner J, Yin M, et al. Fatal Excited Delirium Following Cocaine Use: Epidemiologic Findings Provide New Evidence for Mechanisms of Cocaine Toxicity. *J Forensic Sci* 1997; 42: 25 - 31.
2. Ross DL. Factors Associated with Excited Delirium Deaths in Police Custody. *Mod Pathol* 1998; 11: 1127 - 1137.
3. Stratton SJ, Rogers C, Brickett K, et al. Factors Associated with Sudden Death of Individuals Requiring Restraint for Excited Delirium. *Am J Emerg Med* 2001; 19: 187 - 191.

4. Pollanen MS, Chiasson DA, Cairns JT, et al. Unexpected Death Related to Restraint for Excited Delirium: A Retrospective Study of Deaths in Police Custody and in the Community. *CMAJ* 1998; 158: 1603 - 1607.
5. O'Halloran RL and Lewman LV. Restraint Asphyxiation in Excited Delirium. *Am J Forensic Med Pathol* 1993; 14: 289 - 295.
6. Wetli CV, Mash D, and Karch SB. Cocaine-Associated Agitated Delirium and the Neuroleptic Malignant Syndrome. *Am J Emerg Med* 1996; 14: 425 - 428.
7. Karch SB and Stephens BG. Acute Excited States are Not Caused by High Blood Concentrations of Cocaine. *BMJ* 1998; 316: 1171.