

American Medical Care. New York, Josiah Macy Jr Foundation, 1995.

7. Greene J: Report examines docs. Modern Health Care 1994, pp 20-21.

Agitated Delirium Versus Positional Asphyxia

To the Editor:

The recent article by Stratton et al [May 1995;25:710-712] describes two violent and combative patients who were placed, prone, in hobble restraints and died while being transported and monitored by paramedical personnel. The authors are to be commended for bringing this problem to the attention of emergency physicians.

Unfortunately, they appear to have confused agitated delirium and positional asphyxia, without making a good case for the occurrence of either condition. The deaths remain unexplained, and any causal relationship between these deaths and the hobble restraints remains unproved.

Because both individuals were being monitored during transport, it is difficult to see how they could have died as a result of positional asphyxia: Had respiratory difficulty become apparent, the paramedics would certainly have changed the patients' positions! Most reported cases of positional asphyxia have been associated with the use of alcohol and depressant drugs, not stimulants¹, and then only in individuals who had been hog tied.²

The authors repeatedly used the term "nonlethal" with regard to levels of cocaine and methamphetamine. No scientific basis exists for the designation of lethal and nonlethal blood cocaine levels; this practice was abandoned some years ago.³ In the last 4 years, the San Francisco medical examiner has certified cocaine toxicity as the cause of death in more than 75 cases in which no other drug was

involved. Blood cocaine levels in these individuals ranged from near zero to more than 15,000 ng/mL. Drug reaction may certainly have been the sole cause of the deaths of the restrained patients described by Stratton et al, particularly in the victim with myocardial fibrosis and measurable levels of cocaethylene. More information is, however, needed.

Long-term cocaine users are hyperadrenergic, with high circulating levels of catecholamines.^{4,5} As a consequence of chronic catecholamine stimulation, and probably also of immediate early gene activation, alterations occur in the hearts⁶ and brains⁷ of cocaine abusers.

Myocardial hypertrophy and fibrosis are common^{8,9}, and both favor the occurrence of arrhythmic sudden death.¹⁰ Such anatomic alterations are sufficient to cause death even in the face of negligible blood cocaine levels. Another problem with trying to assign "lethal" and "nonlethal" levels is that tolerance rapidly emerges. Extraordinarily high levels (more than 10,000 ng) are sometimes seen as incidental findings in cases in which trauma, not cocaine, is the cause of death.¹¹

Blood cocaine levels in most cases of agitated delirium are on the order of 1,000 ng^{12,13}, similar to the levels found in crack smokers.¹⁴ However, just knowing the blood cocaine level is not enough to rule in, or rule out, cocaine as the cause of death.¹⁵ Similar considerations apply to amphetamine-related deaths as well. The two victims described by Stratton et al might have been experiencing agitated delirium, but if this had been the case, certain other findings would also be expected. Benzoylcegonine levels are generally much higher and hyperthermia is inevitably present, as are the other behavior changes typical of that condition.

Positional asphyxia and agitated delirium are not two names for the same disease. Not all deaths from

positional asphyxia involve drugs, and many individuals with agitated delirium die without ever being restrained. Deaths from agitated delirium tend to occur in summer, especially when the weather is warm and humid. Two thirds of the victims die at the scene or during transport by paramedics to the hospital. The few victims who live long enough to be hospitalized succumb to disseminated intravascular coagulation, rhabdomyolysis, and kidney failure. In the Miami patients, the average body temperature at the time of first medical encounter of 48 patients with agitated delirium was 40.2°C.

In Miami, most drug-related deaths are caused by cocaine, and 1 of every 10 cocaine-related deaths is due to agitated delirium.¹³ In San Francisco, agitated delirium is much less frequent, but then, so is the number of cocaine-related deaths. When victims of agitated delirium die, litigation is inevitable, with allegations of police brutality or medical mismanagement. However, some steps can be taken that will improve the patient's chances of survival and decrease the chance of litigation.

Sometimes an individual with agitated delirium must simply be restrained. But if the patient is restrained, he or she should never be taken directly to jail for booking. Body temperature should be documented at the scene and aggressive cooling measures undertaken when the patient arrives at the hospital. If the individual must be transported in a police van, every effort should be made to ensure that someone can see the patient. If the patient does die, accusations of police brutality can be ruled out only if the post-mortem examination includes a meticulous, well-documented neck dissection. Some medical examiners have gone so far as to ask the family of the decedent to retain their own pathologist to witness the autopsy. This may sound extreme, but in our

experience, settlements in cases of this type have not been insubstantial.

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1. Bell M, Rao V, Wetli C, et al: Positional asphyxia in adults. Am J Forensic Med Pathol 1992;13:101-107.
2. Reay D, Fligner C, Stilwell A, et al: Positional asphyxia during law enforcement transport. Am J Forensic Med Pathol 1992;13:90-97.
3. Smart R, Anglin R: Do we know the lethal dose of cocaine? J Forensic Sci 1986;32:303-312.
4. Karch S: Serum catecholamines in cocaine-intoxicated patients with cardiac symptoms. Am J Emerg Med 1987;16:481.
5. Nahas G, Trouvé R, Manger W: Cocaine, catecholamines and cardiac toxicity. Acta Anaesthesiol Scand 1990;34(suppl 94):77-81.
6. Tazelaar H, Karch S, Billingham M, et al: Cocaine and the heart. Hum Pathol 1987;18:195-199.
7. Staley J, Hearn L, Ruttenber a, et al: High affinity cocaine recognition sites on the dopamine transporter are elevated in fatal cocaine overdose victims. J Pharmacol Exp Ther 1994;271:1678-1685.
8. Karch S, Billingham M: The pathology and etiology of cocaine-induced heart disease. Arch Pathol Lab Med 1988;112:225-230.
9. Karch S, Green G, Young S: Myocardial hypertrophy and coronary artery disease in male cocaine users. J Forensic Sci 1995;40:589-593.
10. Dunn F, Pringle S: Sudden cardiac death, ventricular arrhythmias and hypertensive left ventricular hypertrophy. J Hypertension 1993;11:1003-1010.
11. Howell S, Ezell A: An example of cocaine tolerance in a gunshot wound fatality. J Anal Toxicol 1990;14:60-61.

12. Wetli C, Fishbain D: Cocaine-induced psychosis and sudden death in recreational cocaine users. *J Forensic Sci* 1985;30:873-880.

13. Wetli C, Raval C: Deaths from agitated delirium among cocaine users in Dade County: A review of 48 cases. *American Academy of Forensic Science Annual Meeting*, 1995.

14. Isenschmid D, Fischman M, Foltin R, et al: Concentration of cocaine and metabolites in plasma of humans following intravenous administration and smoking of cocaine. *J Anal Toxicol* 1992;16:311-314.

15. Karch S, Stephens B: When is cocaine the cause of death? *Am J Forensic Med Pathol* 1991;12:1-2.

In reply:

The interest of Dr Karch and Dr Wetli in our article is appreciated. They present several important issues in their letter.

These physicians state that respiratory difficulty would have become apparent as it developed during transport of the individuals described. After reviewing these cases it was our impression that, during transport, respiratory depression did manifest as unresponsiveness. Alerting emergency medical personnel to the potential danger indicated by unresponsiveness or calmness in this setting was one of the main reasons we submitted these case reports.

Karch and Wetli noted in their letter that most reported cases of positional asphyxia have been associated with "hogtie" restraints. Both our patients, as we reported, were hogtied. We used the term "hobble" because "hogtie" is offensive to many ethnic and socioeconomic groups.

The statement by Karch and Wetli that lethal levels of cocaine and amphetamine cannot be established because of the wide range of levels found in individuals with minimal and severe toxicity is correct. Toxicity from cocaine or

amphetamine could have been the sole cause of death in the individuals we described, but if this was the case it is doubtful that their terminal cardiac rhythms would have been bradycardia progressing to asystole. As stated in the letter, long-term cocaine users are hyperadrenergic, with high levels of circulating catecholamines. Tachycardia, hypertension, agitation, and seizures are expected effects of cocaine and amphetamine toxicity.¹⁻⁴ Far from hyperadrenergic tachycardias and agitation—and in fact consistent with asphyxia—the patients we described became calm and unresponsive, with bradycardia progressing to asystole.

Our article does not state that positional asphyxia and excited delirium are the same disease, as Karch and Wetli inferred. As we reported, further study is needed for the determination of whether hobble restraints, alone or in combination with causes of delirium, have the potential to cause sudden death. In truth, multiple factors probably caused the deaths of the individuals we described. For example, respiratory complications of long-term cocaine abuse and cocaine-induced respiratory depression, described in animal studies, could have predisposed the individuals to positional asphyxia.^{5,6} It is also important to note that complications can occur as a result of restraint of people with delirium unrelated to drug use. This was demonstrated by a Los Angeles—area case in which a young man with a chronic seizure disorder, who was in postictal delirium, was placed in hobble restraints and suffered cardiopulmonary arrest in a manner similar to that of the cases we reported.

Our intent in submitting our article was to alert emergency medical personnel to the danger of unexpected cardiopulmonary arrest in agitated, tightly restrained individuals. Our review of the cases reported in that paper indicates that positional

asphyxia was a factor in these unexpected deaths. Until clarification by further study becomes available, the emergency medicine clinician should be aware that tight restraint of agitated patients with the hobble technique is a high-risk procedure that requires measures to avoid positional asphyxia.

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1. Foltin RW, Fischman MW, Levin FR: Cardiovascular effects of cocaine in humans: Laboratory studies. *Drug Alcohol Depend* 1995;37:193-210.
2. Newlin DB: Effect of cocaine on vagal tone: A common factors approach. *Drug Alcohol Depend* 1995;37:211-216.
3. Gillis RA, Hernandez YM, Erzouki HK, et al: Sympathetic nervous system mediated cardiovascular effects of cocaine are primarily due to a peripheral site of action of the drug. *Drug Alcohol Depend* 1995;37:217-230.
4. Merigan KS, Park LJ, Leeper KV, et al: Adrenergic crisis from crack cocaine ingestion: Report of five cases. *J Emerg Med* 1994;12:485-490.
5. Perper JA, Van Thiel DH: Respiratory complications of cocaine abuse. *Recent Dev Alcohol* 1992;10:363-377.
6. Tseng CC, Derlet RW, Albertson TE: Cocaine-induced respiratory depression and seizures are synergistic mechanisms of cocaine-induced death in rats. *Ann Emerg Med* 1992;21:486-493.

Cruise Ship Medicine

To the Editor:

I believe readers and prospective cruise ship physicians would be interested in my experience as a cruise ship physician, which began after I responded to an advertisement that runs frequently in *Annals*.

In February and March 1995, I worked on two cruise ships in the

Caribbean and along the western coast of Mexico. Each ship carried about 700 passengers (average age, 65 years) and 400 crew members. The medical staff on each ship was one physician and one nurse.

Although I understand that shipboard medicine has its limitations, it was certainly a surprise, to say the least, to learn the following facts.

(1) Both ships lacked effective means of protecting medical staff from blood and airborne diseases. Only paper masks and rubber gloves were available.

(2) Equipment was old, unreliable, or missing. On one ship the heart monitor/defibrillator batteries lasted only 20 minutes, even with constant recharging. The other ship had no long cervical board until one was made. Bag-valve masks and water-testing materials were of poor quality.

(3) Drugs and supplies were outdated or missing. Aspirin and acetaminophen were lacking on one ship. Many "emergency" drugs were outdated or absent on both ships. One ship had no chest tubes or pleurovacs. Each ship, when "fully" stocked, carried about 12 L of normal saline solution. My last voyage ended with 1 L of solution remaining; the ship was scheduled to depart again later that day without a resupply.

Most disturbing and astonishing is that, despite reports made by fax and other means to those responsible for the medical operations of the ships, no action was taken.

Remember, "caveat doctor" if you are considering working on one of these ships. If you do undertake such work, bon voyage!

Wayne C Draper, MD

In reply:

Dr Draper's experience during his tour of duty aboard two cruise ships earlier this year illustrates some of the major issues that prompted the