



## Letter to the Editor

### Positional asphyxia: inadequate oxygen, or inadequate theory?

The sudden, unexplained death of a prisoner in custody presents a difficult challenge for pathologists and police alike, especially if the decedent was a drug user. It is the position of the *National Association of Medical Examiners* (NAME) that when there is “a clinical or investigative history of acute psychosis (paranoid behavior, undressing, violent behavior, and often with hyperthermia), and a complete investigation and forensic autopsy does not reveal a pathologic process as the proximate or underlying cause of death, then a central nervous system active drug should be suspected [1]”. In modern America (now, unfortunately, in modern Europe as well), pathologists have become increasingly aware that the underlying drug is likely to be a stimulant: cocaine or methamphetamine, and that it causes a syndrome known as “agitated delirium”.

Problems certifying these deaths arise because blood cocaine and methamphetamine concentrations may be modest [2], or the drugs may be absent from the blood altogether, with only small amounts of drug or metabolite detected in the urine. There exists a widely held misconception that stimulant drug toxicity is always the result of a single drug overdose. In fact, it is NAME’s position, that “chronic drug use is necessary to induce the changes in the neurochemistry that lead to agitated delirium” in the first place [1]. For many, the notion that negligible blood levels can be a manifestation of a lethal disorder is difficult to accept. The result is that other, more “believable”, but ultimately less plausible, explanations are accepted. The concept of “positional asphyxia” is one of these. Most of these deaths occur after an individual has been “hog-tied”, restrained in a prone position with their wrists and ankles bound behind the back. Alternatively, they may have been forced to the ground, with several officers exerting counter pressure on their limbs and back. When cardiac arrest occurs the event is often attributed to some unspecified combination of oxygen-consuming motor hyperactivity, excessive catecholamine release, and impaired breathing.

There is ample evidence that high concentrations of catecholamines are proarrhythmic [3], and there is even solid scientific evidence that “hog-tying” does limit pulmonary function, albeit negligibly. In the control clinical trial published by Chan et al. [4], the decrease in pulmonary function would not have disqualified any of the participants from active military duty. While there is ample evidence that intense exercise increases the risk for sudden death [5], there

is no evidence whatsoever that physical activity causes life-threatening episodes of hypoxia, no matter how strenuous the activity.

We submit the following calculations in support of our position. The analysis is based on the key assumption that the individual has no underlying disease. This assumption is critical because under American law (and, we believe, under the laws of Britain and most European countries), police cannot be held negligent, or liable, for the in-custody death of a prisoner who is afflicted with an unrecognized disease (such as cocaine-related cardiomyopathy). The police cannot adjust their behavior to account for a condition they do not know exists. The same argument does not apply to felonious assault where, if death results, it would be classified as felony homicide.

Our conclusions about “positional asphyxia” are not based upon any new research or insight. We have simply applied the fundamental tenets of basic exercise physiology; specifically the concept of  $VO_{2max}$ , the body’s ability to transport and utilize oxygen. The assumptions listed in Table 1 can be verified in any basic physiology textbook.

These numbers are a rough approximation, and they are based on the most conservative of assumptions. A 40-year-old man who was able to consume 40 ml/kg of  $O_2$ /min would, in fact, be classified as highly fit, almost certainly able to run in a marathon. Most victims of excited delirium are overweight and not fit. Their predicted maximal oxygen consumption might be less than half that of our hypothetical 40-year-old man.

The mere act of restraining an agitated individual cannot possibly lead to significant hypoxia (and thus death) unless, of course, there is some preexisting problem with central cardiac output, peripheral oxygen extraction, or oxygen utilization. “Positional asphyxia” in and of itself cannot cause this outcome. It can make any undiagnosed, preexisting disease, worse. But if such a pre-existing medical condition exists and was not known when the subject was detained, then by definition, the individual is not normal, and the restraining officers would not be liable for the death.

If the calculations are so clear, why is there still so much confusion and contention? We believe that most of the confusion is semantic, not scientific. The term “positional” asphyxia was originally used to describe the mechanism of death when an inebriate, usually obese, became wedged into a confined space, such as the area between the end of a bed and the wall, and suffocated. Such cases were easily diagnosed, since the decedent would inevitably be covered with a shower of petichae [6].

Table 1  
Assumptions

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1 g of hemoglobin carries 1.31 ml oxygen  
 1 l blood containing 15 mg/dl contains 200 ml oxygen  
 (150 g  $\times$  1.31 = 196.5 ml of oxygen)  
 Amount of oxygen available = cardiac output  $\times$  blood oxygen content  
 If cardiac output is 5 l, then available oxygen is 5 l  $\times$  200 ml O<sub>2</sub>/l = 1000 ml oxygen  
 Normal oxygen consumption at rest = 250 ml, leaving 750 ml oxygen unused  
 During exercise, a healthy 40-year-old man weighing 100 kg should be able to utilize 40 ml oxygen/kg m/min), or 4000 ml  
 Average cardiac stroke volume is 70 ml of blood per contraction  
 During maximal exertion, a healthy 40-year-old man should have a pulse of at least 160 beats/min  
 Thus, 160 beats/min  $\times$  70 ml O<sub>2</sub>/beat = 11,000 ml oxygen available/min. But the most that a 40-year-old man with a 40 ml VO<sub>2max</sub> and a weight of 100 kg could metabolize would be 40 ml/min  $\times$  100 kg = 4000 ml. That leaves 7200 ml oxygen/min unused

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However, in the early 1990s, based on the results of just one set of experiments showing that “hog-tying” lowered blood oxygen saturation in volunteers [7], this sentiment changed. Pathologists, especially those with little experience in the investigation of cocaine-related deaths, began to apply the term to any death of a restrained person where no other cause of death was immediately evident. The results of more recent studies, using modern technology have disproved their early studies about restraint and clarified the neurochemical causes responsible for death [4,8].

Since the body has such massive oxygen reserves, and since it has been amply demonstrated that “hog-tying” has only negligible effects on ventilation, we therefore conclude that the diagnosis of “positional asphyxia”, by itself, is not a sufficient cause of death, and that other causes for the death should be considered in those cases where an otherwise healthy individual dies suddenly while in police custody. The most obvious cause to consider is genetic abnormality.

Within the last several years it has become increasingly obvious that there could be a genetic basis for many of these deaths, particularly in cocaine users, where interactions with the HERG channel have already been documented [9].

## References

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