# Drug abusers who die during arrest or in custody

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*J R Soc Med* 1999;**92**:110–113 SECTION OF CLINICAL FORENSIC & LEGAL MEDICINE, 20 JUNE 1998

The Police Complaints Authority of Great Britain recently announced that 56 prisoners had died in custody in 1997[1]. The rate, roughly 0.1 per 100 000 citizens per year, is surprisingly similar to the rate in California, where 35 to 40 such deaths are recorded annually[2]. The vast majority of the these deaths, both in California and in the UK, are suicides, but in the USA many of the remaining deaths are drug-related. Suicides are often preventable. Given appropriate resources, custodial officers can be trained to recognize potential suicides and see to their wellbeing. Drug-related deaths are more difficult to prevent.

Depending upon when they occur, drug-related deaths in prisoners can be divided into four different categories: (1) death during arrest and transport; (2) death within 12 hours of arrest; (3) death after 12 hours but while still on remand; and (4) death after trial. Excited delirium in chronic stimulant abusers is the principal cause of death during arrest and transport. Death from massive overdose in drug smugglers and dealers is the principal cause during the first 12 hours. Deaths that occur after 12 hours are almost always related to withdrawal, or other natural causes.

Even with optimal care, many drug-related deaths cannot be prevented. Drug dealers who swallow the evidence at the time of arrest stand a good chance of dying, as do chronic stimulant abusers suffering from excited delirium. Survival in these cases is theoretically possible but, given the ineffectiveness of current remedies, it is not likely. Thus drug-related deaths fall into a different category from readily preventable deaths such as those related to drug withdrawal, suicide, and other natural causes, and must be considered separately.

## BODYPACKERS AND BODYSTUFFERS

Most preventable drug deaths are due to massive ingestion. The concealment of drugs within body cavities, known as 'bodypacking', is used by both heroin and cocaine smugglers. Quantities in excess of half a kilogram may be smuggled in this fashion[3,4]. There is some controversy over how smugglers, with or without symptoms of toxicity, should be treated[5], but there is no question that their management includes direct medical supervision.

'Crack' cocaine dealers facing imminent arrest may resort to swallowing their inventory. This practice is referred to as 'bodystuffing'[5]. Because of the way crack is produced (by addition of bicarbonate to a heated solution of cocaine hydrochloride), results of ingestion are unpredictable[6]. If the ingested pieces of crack contain a great deal of bicarbonate, they are likely to pass harmlessly through the stomach and be slowly, if at all, absorbed from the intestines. If, however, the final product does not contain much bicarbonate, or if cocaine hydrochloride has been swallowed, the ingested material will dissolve rapidly, very high blood levels will be quickly attained, and the picture will resemble that seen in the bodypackers.

If the bodypacker or bodystuffer is a cocaine user, he or she may be able to tolerate very high blood concentrations with few, if any, symptoms. Otherwise, they will exhibit a stereotyped pattern of symptoms with agitation, followed by confusion, a rise in temperature, continuous seizures, and cardiac arrest[7,8]. If the dose is massive, seizures may be the first symptom. Necropsy findings include pulmonary and cerebral oedema[9]. Because cocaine rapidly crosses the blood–brain barrier and benzoylecgonine does not, cocaine concentrations in both blood and brain will be impressively high (>5 mg/kg), although brain concentrations of benzoylecgonine may be quite low (<1.0 mg/kg)[10]. If death occurs rapidly, little of the cocaine in the brain will have been converted to benzoylecgonine[11]. If heroin was the drug being smuggled, the picture will not be very different, though seizures are much less likely[4].

Prevention in such cases, no matter whether opioids or stimulants are involved, consists first and foremost in not taking the arrestee directly to jail. Common sense dictates that anyone suspected of ingesting substantial amounts of illicit drugs needs to be evaluated at a hospital. The need is not diminished even if such claims are clearly made as an attempt to avoid incarceration. The liability associated with not seeking medical clearance is simply too great.

## EXCITED DELIRIUM

Excited delirium was first observed in psychiatric patients more than one hundred years ago[12]. Today, most victims

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are chronic, long-term, high-dose stimulant abusers. Initially hyperthermic, they soon become psychotic, experiencing several hours of violent agitation. Cardiac arrest occurs a few hours later[13,14].

In some cases, death occurs at home, unattended. More often, police come into contact with the deceased during the phase of psychotic agitation, just as death is about to occur. A struggle will ensue, and chemical incapacitating agents, capsicum spray in the US or CS spray in the UK, may be employed with no effect. In the average case, the concerted efforts of five or six officers will be required to finally force the victim to the ground and apply restraints. Respiratory arrest tends to occur shortly after the restraints are applied, while the victim is on the ground or being transported. If attempts at resuscitation are successful, almost all victims die of rhabdomyolysis and multisystem failure a few days later[14,15].

Hyperthermia in this syndrome is a consequence of brainstem $D_1$ dopamine receptor downregulation[16]. The psychotic behaviour seems to be the result of upregulation of kappa-2 receptors in the amygdala[17]. The mechanism for respiratory arrest in these individuals is not understood, but whatever the cause, death occurs within hours of symptom onset. At necropsy, there will be pulmonary oedema and heart disease—mild to moderate cardiac enlargement, myocardial fibrosis, and coronary artery disease.

The changes seen in victims' hearts can be explained by the prevailing concentrations of adrenaline and noradrenaline, high concentrations of which result in myocyte calcium overload and cell destruction. The earliest visible lesions, called 'contraction bands', are illustrated in Figure 1 (thin arrows)[18]. These are a non-specific finding due to abnormally high intracytosolic calcium, either from loss of external membrane integrity (as in ischaemia) or from catecholamine excess (as in cocaine toxicity). As the cells die off they are replaced with collagen, producing a pattern of myocardial fibrosis best described as 'microfocal' (thick arrows); normal cells, with no evidence of contraction bands, can be seen surrounding one or two damaged cells that have been replaced by collagen. The identical pattern is seen with phaeochromocytoma[19]. Markers for intravenous drug abuse, such as inflammation of the portal triads and birefringent crystals in the lungs, may also be present, but their presence only provides confirmatory evidence of chronic drug abuse[20,21].

In cases of excited delirium, cocaine concentrations in blood and brain will be modest, but concentrations of the cocaine metabolite benzoylecgonine may be quite high[22]. The findings are explained by benzoylecgonine's longer half-life[23], and by the fact that it accumulates in the tissues of chronic users.

If death occurs while the officers are trying to restrain the victim, the police will be assumed to be responsible. The tendency to confuse proximity with causality becomes very great when the necropsy is cursory or incomplete and underlying heart disease and hyperthermia go undiagnosed or unappreciated[24,25]. When these cases come to legal review, as they inevitably do, the same set of issues is raised on every occasion. Because these issues are so predictable, measures should be taken to ensure that events and findings are clearly documented.

When an individual with excited delirium dies just after being restrained, the cause of death may be attributed to 'positional asphyxia.' This term was originally used to describe the mechanism of death when an intoxicated, usually obese, individual becomes wedged into a confined

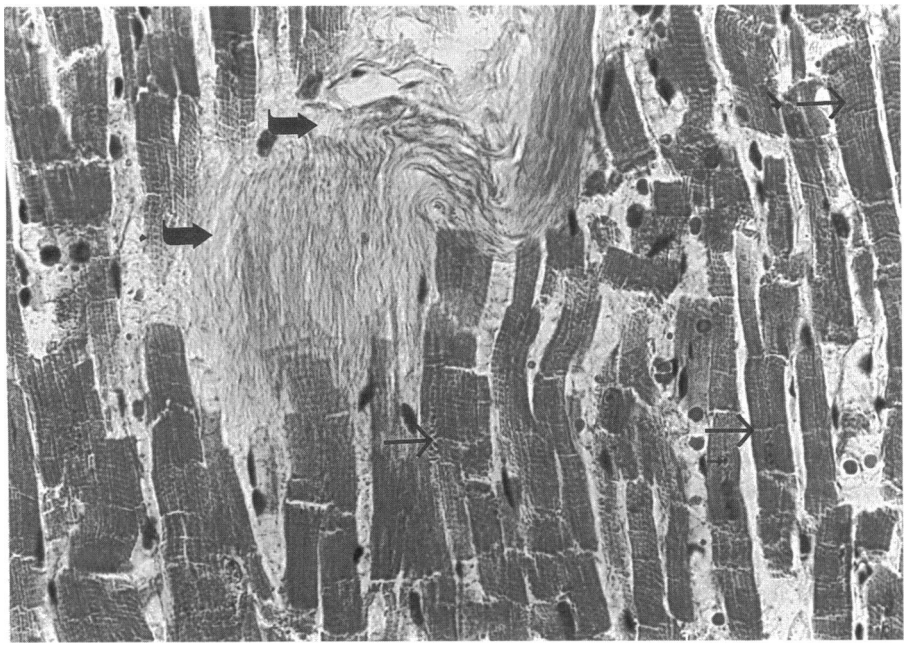

*Figure 1* **Microfocal myocardial fibrosis: from a 41-year-old female chronic cocaine abuser who died suddenly.** H&E stain. Eosinophilic transverse bands (contraction bands) are visible in several of the cells (thin arrows). Cells with severe contraction banding are replaced by collagen scars (thick arrows).

space, such as between the edge of a bed and the wall. In such cases, post-mortem examination will disclose marked congestion, along with cyanosis and, frequently, showers of petechiae[26]. But in the early 1990s, largely on the basis of a single set of experiments showing that a certain type of restraint referred to as hogtying (suspects are placed prone with their wrists tied together behind their backs and their ankles, also tied together, secured to the wrists) produced hypoxia, some pathologists began applying the term positional asphyxia to any death of any restrained detainee with a few conjunctival petechiae[27,28], where an alternative cause of death was not immediately obvious. The decedents were invariably chronic drug abusers or schizophrenics, their hearts were often not even weighed, let alone examined in detail, and temperatures were rarely recorded. More recent studies have shown that in the non-obese, restraint, even hogtying, has only modest effects on ventilatory function[29], and that the presence of occasional conjunctival petechiae is proof of nothing more than heart failure[30,31]. If the heart is examined in sufficient detail, important abnormalities will be evident.

Additional documentation is needed if chemical incapacitating agents, particularly capsicum spray, are employed. Our experience suggests that these agents have no effect on patients with excited delirium, but that if the victim dies, death will be blamed on their use. There are no tissue assays for these chemical agents, but capsicum can be recovered from the skin and clothing with methanolic swabs (saline swabs in the living). Failure to recover the sprayed capsicum from the facial area, or from the airway in cases of accidental death, is reasonable evidence that capsicum did not enter the lungs and did not directly cause toxicity or death.

Violently agitated prisoners should always be taken to a hospital, never to a jail, and they should not be transported in a police car. In some jurisdictions, that caveat may be impossible to heed, since ambulance attendants may refuse to transport acutely agitated patients. Whatever the means of transport, victims need close supervision. Monitoring with pulse oximetry will preclude later accusations of positional asphyxia. At a minimum, someone, a paramedic or an officer, should be at the victim's side during transport. The victim's temperature should be recorded on arrival at the hospital. Temperature is, in fact, rarely recorded when patients are agitated. Without any documentation of hyperthermia, proof that a decedent was suffering from excited delirium may become more problematic.

If the victim cannot be resuscitated, certain measures must be taken immediately. Core temperature should be recorded as soon after death as possible, and again at necropsy. If petechiae or bruises are present, they should be photographed. Documenting the absence of these lesions at the initial necropsy is just as important as documenting their presence. Petechiae can form after death[32]. If the absence of petechiae is not documented initially and petechiae are found during a second necropsy, charges of incompetence or cover-up may result.

The changes in brain dopamine and kappa receptors can be documented, but only if the brain is removed and frozen within 12 hours. Frozen samples can then be sent to a reference neurochemistry laboratory[33]. Obviously, a scrupulous well-photographed neck dissection will be required to determine whether a choke hold or neck compression has been applied[34]. The thoracic organs and the brain should be removed before the neck dissection to decompress venous return and prevent artifactual bleeding into the soft tissues of the neck, the presence of which may falsely suggest traumatic injury.

The heart must be examined in detail. That does not necessarily mean taking dozens of sections from the conduction system, but it does entail careful weighing, measurement of wall thickness, and the taking of multiple sections for histological examination[35]. Microfocal fibrosis favours the occurrence of lethal arrhythmias and so does increased heart size[36,37]. But increased size only becomes apparent if the heart is weighed and the result compared with weight predicted by standard nomograms. When such comparisons are made, the heart will be found to be at least 10% above predicted norms[38,39]. Since heart size is an independent risk factor for sudden death[40], the measurement may prove to be a very significant factor in determining the cause of death.

Even if all of these measures are taken, false allegations of brutality may still be made. Proving that the decedent had a generally fatal disease does not prove that he was not maltreated, but very careful documentation at the scene of the death and of necropsy findings does make it more likely that any mistreatment will be discovered. Of course, if vital signs and oxygen saturation are continuously recorded during transport, then it matters little what position the decedent was in or how he was restrained, provided it can be proven that his respiratory status was not impaired. Similarly, if capsicum and CS are not employed, death cannot be attributed to their use.

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