ARTICLE

Acute behavioural disturbance: a physical emergency psychiatrists need to understand

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SUMMARY

The phenomenon of acute behavioural disturbance (ABD) (also known as acute behavioural disorder or excited delirium) is an underrecognised and potentially life-threatening syndrome, and an emergency in psychiatric settings. Causes are discussed and the pathophysiology explained. The challenges faced by practitioners are highlighted, including how 'standard' control and restraint can exacerbate symptoms. Initial treatment strategies are suggested.

LEARNING OBJECTIVES

After reading this article you will be able to:

- understand the causes and presentation of ABD and be able to differentiate it from other forms of agitation
- understand the pathophysiology of ABD and why it is potentially so dangerous
- appreciate initial management of ABD by psychiatric services and the referral pathway to medical care.

KEYWORDS

Acute behavioural disorder; novel psychoactive substances; legal highs.

Acute behavioural disturbance (or acute behavioural disorder) (ABD) is a potentially life-threatening syndrome of delirium, dysregulated physiological responses and aggressive behaviour. It is most commonly caused by illicit drug use. Uncontrolled catecholamine release, metabolic acidosis and hyperthermia can lead to rapid physical deterioration and death. A lack of awareness about ABD means that it can be misrecognised as a more 'generalised disturbance'. This risks failure to provide timely appropriate care, and indeed 'standardised' interventions such as control and restraint can exacerbate the symptoms and worsen prognosis.

Readers might be most familiar with ABD from infamous police video footage of officers restraining a highly aroused and aggressive suspect, only for that person to quickly deteriorate, with increasingly laboured breathing and cardiac arrest.

ABD is challenged as a 'diagnostic entity' and is best considered as a common final pathway with a range of potential causes. This, added to the common failure to recognise the condition and use of other terms, such as 'excited delirium', means that it is inherently difficult to get reliable epidemiological information on ABD. However, there are concerning anecdotal reports of its rise in frequency, particularly in line with consumption of new/novel psychoactive substances.

This primer will describe the causes and known epidemiology of ABD, and how to recognise it. It will explain its pathophysiology and give initial management recommendations.

Causes and epidemiology

Historical to current understanding

Acute behavioural disturbance has likely existed throughout human history, with early occurrences attributed to various supernatural phenomena, such as spiritual and demonic possession. American psychiatrist Luther Bell wrote up the first scientific case reports on a group of patients admitted to hospital between 1836 and 1849, who were highly agitated and had a 75% mortality rate (Bell 1849). Autopsies found no anatomical abnormalities to explain the deaths, and the label 'Bell's mania' was subsequently adopted. Bell stated that a history of stimulant use would likely be a key cause of the phenomenon: of note, opium, heroin, morphine, cannabis, mescaline, codeine and cocaine were all available – to varying degrees – in Victorian times. Henry Maudsley also wrote on two cases of 'acute maniacal delirium' that, with hindsight, appear to be ABD (Maudsley 1897). The first presented with cycles of extreme agitation over a period of weeks, necessitating restraint; the individual's mental functioning deteriorated, and he was left in a state described as resembling dementia.

After the 1900s the diagnosis of Bell's mania was gradually replaced in the medical literature, with a wave of definitions and classifications as psychiatry became increasingly standardised.

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BOX 1 A representative case of acute behavioural disturbance, containing elements from several individuals treated by the authors, with some details changed and any individually identifying information removed to preserve anonymity

Police were called by the individual's mother as she had not heard from her son for days. She was concerned for his welfare and reported that he used cocaine and alcohol frequently, having been on a recent binge lasting a week, which ended up with him being assaulted and brought to the emergency department. The police attended the property and gained access under common law owing to the concern for his welfare. The man was shouting incoherently, reporting that persons were attempting to get through his windows (he lived at the top of a fourteen-storey building); examination of the scene revealed that he had piled furniture and bedding in the centre of the living room in an attempt to build a hiding place.

Several police officers and paramedics also in attendance were required to restrain the man. He was unable to follow simple commands and constantly battled the officers, who succeeded in gaining control using handcuffs and leg restraints. He was then transported to the emergency department by ambulance; the paramedics radioed ahead to alert staff. On arrival he was directed to the resuscitation room, where he was rapidly assessed to be suffering from acute behavioural disturbance. His agitation was such that he required half a dozen police officers, in addition to restraints, to avoid injury to himself and personnel present.

The emergency department consultant directed for intramuscular ketamine to be administered (dose 4 mg/kg); it was initially impossible to obtain investigations or establish intravenous access. After a couple of minutes, the patient showed signs of dissociation of consciousness but was still exhibiting severe aggression and combativeness; by this point intravenous access had been established and emergency anaesthesia was induced. Initial tests revealed a sinus tachycardia on ECG, a metabolic acidosis with compensatory hyperventilation and a serum creatine kinase (CK) level of almost 30 000 U/L (units per litre. The normal range is approximately 20 - 200 U/L). He was admitted to the intensive care unit and required sedation for around almost two days. His CK level fell to normal with intravenous hydration and he was discharged from hospital after a few days more, with no sequelae of mental or physical illness.

Interestingly, the incidence of such dramatic presentations (particularly with fatal outcomes) remarkably diminished. This is likely accounted for by the increased availability of sedative medications, notably a popularisation of barbiturates in the early 20th century and the introduction of antipsychotics in the 1950s.

In 1985 Charles Wetli published an article on a number of deaths occurring after police attendance and associated with recreational cocaine use (Wetli 1985). Wetli, a forensic pathologist, coined the term 'excited delirium', attributing this cause of death to such cases in which no anatomical or toxicological explanation could be found. It was noted that, although in most cases there was a detectable level of cocaine, this was well below that found in fatal overdoses and often below that seen in recreational users. Following on from his publication, further examples of fatal cases associated with a variety of other drugs were published.

Controversy surrounds the use of the term excited delirium (Lipsedge 2015), not least due to the link with death in custody, greater such rates in individuals from minority backgrounds and the association with complex issues such as institutional racism in police forces. Indeed, some civil rights campaigners have stated that it is a 'fake diagnosis', aimed at exonerating officers from charges of excessive use of force (Paquette 2003). Further, legal proceedings have contested ABD as a recognised medical condition (Anaïs 2014), and most hospital records do not have ABD as a diagnosis, instead ascribing patients' symptoms to conditions such as 'agitated delirium', 'cocaine-induced mental illness' or 'malignant catatonia' (Fink 1999; Detweiler 2009).

In 2009 the American College of Emergency Physicians published a white paper in an attempt to increase awareness and suggest possible treatment strategies (ACEP Excited Delirium Taskforce 2009). A largely unknown phenomenon in the UK, experience of the condition by medical staff and emergency services was, until quite recent times, vanishingly rare. However, the recent growth in the use of new/novel psychoactive substances (often referred to as 'legal highs') and increasing media coverage of deaths in custody have led to growth in awareness of what we now label acute behavioural disturbance. Box 1 gives a representative example of a case of ABD, containing elements from several individuals treated by the authors, with some details changed and any individually identifying information removed to preserve anonymity.

Rather than being seen as a distinct entity, ABD should be conceptualised as a 'final common pathway' with many underlying causes. It should be noted that ABD is not currently certifiable as a cause of death.

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BOX 2 Drugs associated with acute behavioural disturbance

- Cannabis
- Methamphetamine or amphetamine
- Lysergic acid diethylamide (LSD)
- Novel psychoactive substances, including synthetic cannabinoids ('spice'), mephedrone, MDPV, 5-MeO-DALT, 2C, and α-PVP

Causes

Illicit drug use

Investigations into fatal cases have determined illicit drugs, particularly stimulants, to be key precipitants, with cocaine the most commonly isolated agent (Box 2). Case reports have linked ABD with acute cannabis intoxication and a range of novel psychoactive substances, including the synthetic cannabinoids (sometimes generically referred to as 'spice'). Data on novel psychoactive substances are inevitably hindered by their large number and the general lack of information on many of them. Given the broad range of actions on neuronal receptors, the hypothesis of ABD as a final common pathway is strengthened. There is no evidence that alcohol in isolation precipitates ABD, but it is possible that, in combination with other factors, the risk is increased.

There is currently insufficient evidence to determine any importance of duration and quantity of drug use and, as noted earlier, when detected, cocaine is often found at concentrations lower than those typically seen in poisoning deaths. However, interpreting drug concentrations is complicated by the fact that some time may pass between drug consumption, onset of ABD and toxicological testing. Screening for chronic drug use by hair analysis has been shown to be of some utility in the forensic toxicology literature (Paterson 2009).

Substance withdrawal

The syndrome associated with alcohol withdrawal and its counterpart, delirium tremens, will be familiar to most clinicians. Drug withdrawal states from baclofen (Downes 2019) and gamma-hydroxybutyrate (GHB) (Lal Kashyap 2011; Corstens 2018) have been shown to precipitate problematic withdrawal states that prove a great challenge for carers trying to manage the agitation and autonomic instability.

Mental and physical illness

Recalling that ABD is a final pathway, it has been reported in instances of schizophrenia, bipolar affective disorder and malignant catatonia where individuals have been untreated (Bond 1980; O'Halloran 1993; Fink 1999; Morrison 2001; Park 2001; Detweiler 2009; Kennedy 2018). Such episodes, without other factors such as illicit drug use, are rare in contemporary mental health services. However, they were considerably more common in the era before modern pharmacological treatments, and the history of psychiatry records many deaths from forms of agitation, exhaustion and collapse that are likely to have been instances of ABD.

Any cause of delirium may induce ABD, and thus there are many medical illnesses that are potential risks, although the actual incidence in such cases seems low. The limited literature on the topic has implicated hypoglycaemia, sleep deprivation, head injury, brain tumours and brain infection (encephalitis or meningitis) and cerebrovascular accidents (Vatsavayi 2003).

Epidemiology

It is clear that obtaining good epidemiological data on ABD is difficult. It is sufficiently uncommon that most clinicians are unaware of it, with debate on it as a diagnostic term and on its included criteria, and most of the scientific literature is based on case reports.

Notably, it can be difficult to clearly separate it from what have sometimes been labelled 'angry man' episodes and, although death in custody is inevitably controversial, it is accepted that there are occasions when considerable force is required to subdue individuals, and such cases might get missed. This latter problem is amplified by the fact that the time from a death in custody to the completion of inquest and inquiry reports can be 5 years or more.

The number of fatal cases of ABD related to police contact has increased gradually since 1998, which has been linked with changes in patterns of illicit drug use and better training and awareness of ABD as an entity. Inferential statistical analyses have suggested fatality rates of under 10% (Baldwin 2018), although such a figure must remain somewhat circumspect at this time.

Diagnosing ABD

Not all individuals with ABD have the same symptom profile, and Box 3 describes the frequency of features described by Canadian police officers in the only prospective study to date on the topic (Hall 2013). Responding officers particularly commented on a pronounced skin temperature hot to the touch, what is known as 'tactile hyperthermia', which appears to be a distinguishing feature of ABD (Baldwin 2016).

BOX 3 Frequency of behaviours described in those with acute behavioural disturbance

In a prospective study on 1269 individuals who underwent use of force for acute behavioural disturbance, Canadian police officers reported the following symptoms:

- violent behaviour (66%)
- pain tolerance (20.8%)
- constant/near constant activity (24.7%)
- not responsive to police presence (21.7%)
- superhuman strength (10.8%)
- rapid breathing (9.7%)
- does not fatigue (8.8%)
- naked/inappropriately clothed (7.4%)
- sweating profusely (4.9%)
- hot to touch (3.5%)
- attraction to/destruction of glass/reflective surfaces (2.8%)

(Hall 2013)

For a working diagnosis of ABD, three core symptom clusters are classically described: delirium, dysregulation of physiology and aggressive or combative behaviour.

Box 4 gives an overview of assessment and initial management.

Delirium

Delirium will be well understood by psychiatrists – an acute disturbance of cognitive functioning with an organic aetiology. Individuals are liable to be disoriented in time, person and place. Hallucinations – gustatory, auditory, visual and olfactory – are commonly reported (Ross 1998).

Dysregulation of physiology

The pathophysiology of ABD is detailed below, but essentially it results from a sustained release of the sympathetic catecholamines adrenaline, noradrenaline and dopamine. These raise heart rate and blood pressure, and effect various metabolic changes. A notable occurrence in ABD is impairment of the normal homeostatic physiological processes that should ordinarily limit such release.

Aggressive or combative behaviour

The somewhat limited data suggest that in such instances most individuals, in a delirious state, believe that they are 'fighting for their lives' rather than being motivated by wishing to challenge or assault another. Their altered mental state also means that they may not recognise attempts to assist them, and indeed there are reports of individuals believing that professional services are imposters.

Pathophysiology

The onset of delirium typically has a marked impact on individuals' mental states, commonly defined by disorientation, fear, paranoia and a sense of persecution and terror, manifesting with attempts to escape from anyone attempting to help. The well-recognised 'fight or flight' changes associated with the fear response – namely enormous physical effort

BOX 4 Main factors to consider when assessing and instigating treatment of acute behavioural disturbance (ABD)

Assessment of the agitated patient

- REMEMBER: no symptom, or lack of symptom, is pathognomonic for ABD
- Delirium is common: is the individual disoriented in time, person and place?
- Agitation and aggression: is there hyperactivity, an apparent increase in strength and tolerance to pain?
- Is there a history or suspicion of illicit drug use?

Examination – ensuring safety of all

- Physiological disturbance: is the body hot to touch; is there abnormal sweating (too much/none)?
- Physiological distress: is there laboured breathing?
- Measure basic parameters – pulse, blood pressure, temperature – although this may be quite difficult to do safely and accurately
- Urinary drug screens for novel psychoactive substances can be of limited value, and there may not be the time or ability to collect a sample

Initial management

- Where possible, verbally de-escalate and calm the patient: standard delirium orientation cues may be helpful
- There are risks of harm to staff and bystanders/other patients: the wider environment will need to be contained
- Avoid or minimise physical contact and restraint where possible
- Call an ambulance and prepare an adequate handover for emergency department staff, who may be less familiar or comfortable with managing any mental illness component
- Intramuscular benzodiazepines or antipsychotic medications can be administered: a record of any medications given must accompany the patient to minimise the risk of unintentional overdose through repeat administration
- Ideally, mental health staff known to the patient should accompany them throughout, to assist emergency department colleagues and provide some continuity for a patient who is likely to be distressed and confused

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and catecholamine release – lead to three interplaying factors that serve to create a 'perfect storm' of physiological distress: hyperthermia, catecholamine excess and metabolic acidosis (Bunai 2008).

Hyperthermia

The hypothalamus acts as the body's thermostat, with normal temperature ranges between 36.5 and 37.5°C. Hyperthermia in ABD is a consequence of excessive muscular activity and generation of heat. Even when this excess movement ceases, the muscles act as a 'thermal reservoir', sustaining elevation in body temperature; obesity is an understandable recognised additional risk factor for this. Rhabdomyolysis – the breakdown of muscle tissue – is indicative of the duration and/or intensity of the episode; thus, measuring creatine kinase levels is important in the diagnostic workup of patients (Borek 2012). It is noteworthy that rhabdomyolysis may affect localised muscle groups, such as the neck, as opposed to producing more generalised damage (Debelmas 2018).

Compensatory mechanisms to counter the rise in temperature include increased respiration and sweating. However, owing to large amounts of fluid loss in advanced stages of ABD, sweating may actually cease and the individual may present with dry skin. Problematically for mental health services, body temperature from peripheral measurements (for example, the ear) are often inaccurate, with greater reliability attained by bladder (not rectal) measurement. As mentioned above, tactile hyperthermia is commonly encountered.

Hyperthermia will contribute to and worsen any delirium and metabolic acidosis (Otahbachi 2010) (see below). The risk of death or serious illness is generally related to the duration of hyperthermia and the peak temperature reached. Temperatures over 42°C have very poor outcomes, including life-changing injuries secondary to multi-organ failure, regardless of treatment administered, and the risk of death secondary to multi-organ failure is well recognised.

Catecholamine excess

The sympathetic and parasympathetic nervous systems control autonomic functions such as heart rate, gut motility and pupil size. Short-lived stressors lead to an adaptive surge in adrenally secreted catecholamines (adrenaline, noradrenaline and dopamine) and subsequent accelerated heart rate, raised blood pressure and diversion of blood flow away from the gastrointestinal circulation.

The levels typically encountered in ABD have yet to be adequately quantified. However, cardiac catecholamine receptors increase the rate and force of contraction, at the cost of reduction in the electrical stability of the heart. Excess sympathetic outflow is associated with a prolonged QT interval (Yadav 2004). Release of, and sensitivity to, adrenaline and noradrenaline is increased in metabolic acidotic states (Goldsmith 1990), and may result in 'stunning' of the myocardium (Yadav 2004). Sudden bursts of adrenaline may lead to an often-fatal form of heart injury – Takotsubo cardiomyopathy – whereby part of the left ventricle suddenly forms a 'pocket' (Otahbachi 2010). Simulated law enforcement use-of-force encounters suggest that physical resistance and fleeing on foot generate greater catecholamine release than dog unit, Taser® or irritant spray (Ho 2010). Catecholamines enhance the risk of convulsion and the respiratory and circulatory toxicity of cocaine (Mets 1996).

Metabolic acidosis

Acid production is a normal outcome of metabolic activity, with homeostatic mechanisms maintaining the stable blood pH of about 7.4 that is essential for optimal cellular functioning. This is achieved by a complex interplay of buffers within the blood, carbon dioxide levels and regulation of the excretion or conservation of acid by the kidneys. In pathological states the production of excess acidic compounds exceeds the buffering and excretion capability of the body, and a state of metabolic acidosis ensues. In ABD, contributors to this metabolic acidotic state include excess muscle activity, raised catecholamine levels, dehydration, hyperthermia, acute kidney failure (Plush 2015) and, if consumed, cocaine (Allam 2001).

The initial mechanism to attenuate the excess acid generation is its combination with circulating bicarbonate in the blood, producing carbon dioxide. This rise in carbon dioxide results in the stimulation of respiration, which may lead to tachypnoea and panting. This in turn may herald cardiorespiratory collapse as the physiological compensatory mechanisms are overwhelmed.

Metabolic acidosis further sensitises the heart tissue to circulating catecholamines. Crucially, this may result in sudden death when a surge occurs during restraint – in other words, the often standard response to manage agitation exacerbates it. The electrical conduction throughout the heart muscle is grossly disturbed and ultimately may lead to electromechanical dissociation, wherein the electrical current fails to trigger muscle contraction. Changes on an electrocardiogram (ECG) can be detected secondary to metabolic acidosis (Dreyfuss 1989; Bozeman 2013), although clearly, obtaining an ECG during an episode of ABD is almost impossible until the associated agitation has been treated.

Treatment

Early recognition and initiation of treatment is paramount to reduce fatalities attributed to ABD (Strote 2014). There is also an increase in risk to police officers and members of the public when faced with a person exhibiting ABD (Baldwin 2018). The location of the disturbed patient may make it challenging to safely contain the individual and arrange transfer to definitive care. Specific units that might face additional pressures include police custody suites, segregation units in prisons, and seclusion rooms in psychiatric intensive care units (PICUs) and forensic acute admissions units. Transfer from legal establishments, secure mental health settings and forensic mental health units can be impeded by the policies and legislation to detain a person. It may be seen as counterintuitive for staff to be removing a patient/detainee from a secure setting, but such facilities do not possess the necessary medical testing, care and potential treatment indicated by the condition.

Most cases of ABD appear to be brief, and once treatment has been initiated, resolution of the delirium typically occurs within 48 h. Full assessment and treatment needs to occur in the emergency department (Wetli 1996), and psychiatric units will not have the facilities or ability to effect appropriate care beyond first steps. The primary roles of mental health services are: recognition of potential ABD, contacting emergency services, providing a relevant medical handover and instigating appropriate sedation (Box 3). Mental health staff do have particular expertise in verbally managing agitated individuals and accompanying them to the emergency department will be appropriate where possible, including trying to assist a disoriented and delirious patient. For patient and staff safety, it is recommended that transfer to definitive care is via ambulance, to allow direct visual observation of the casualty (Karch 1999).

The importance of collaborative development of guidance for mental health and emergency department services cannot be overemphasised; general education on the topic, including for police services, is vital to reduce risk to staff and patient.

Restraint and transport

The use of restraint in cases of ABD is often unavoidable. The purpose of restraint is to facilitate transport, reduce the risk of harm to the individual and rescuers and facilitate the administration of medication. Sudden death may ensue after the application of restraint, characteristically laboured breathing followed by cardiorespiratory arrest (Stratton 2001); metabolic acidosis has been recorded in such cases (Hick 1999). It is recommended that the individual be supine or rolled onto the side (Stratton 1995; Pollanen 1998; Ross 1998; Michaud 2014). The degree and duration of restraint should be for the minimum time necessary to aide an intervention (Royal College of Emergency Medicine 2016). As mentioned above, transport to definitive care ideally should be via ambulance, to allow observation of the patient. Conflict between first responders regarding the appropriateness of emergency department attendance can lead to poor outcome.

Sedation

Sedation calms aggressive behaviour, but this is not its sole purpose. The cyclical agitation perpetuates biochemical and physiological abnormalities that may prove fatal. The greater the duration and intensity of agitation, the greater the risk of an adverse outcome; calming the individual reduces muscle activity and excess heat generation. Tranquillisation also reduces catecholamine production, further diminishing the agitation.

Over the years several options have been trialled, with varying success in terms of rapidity of onset, effectiveness, safety and administration into muscle. Intravenous access – something less likely to be attempted in a mental health setting – is not only challenging but may result in needle-stick injury to those involved. Drugs used to rapidly sedate patients presenting with ABD can be grouped into Salem 2009:

- benzodiazepines – such as diazepam, lorazepam and midazolam (Isbister 2010)
- antipsychotics – haloperidol, droperidol, olanzapine, chlorpromazine (Isbister 2010; Page 2018)
- ketamine – a dissociative anaesthetic agent (Cole 2018; Li 2020).

Some studies have recommended combination therapy of an antipsychotic and benzodiazepine (Chan 2013; O'Connor 2017), and practitioners will be familiar with the traditional pairing of intramuscular lorazepam and haloperidol used in mental health settings for many years to treat agitation and threatening behaviour. However, their effectiveness has not always been optimal. In emergency department settings, intramuscular ketamine has proved superior, having a predictable dose–response relationship with good effect (Hayes 2015; Riddell 2017), and may be used as a 'rescue therapy' after failure of traditional sedation options (Isbister 2016). Ketamine used at appropriate dosages (ranging from 2 to 4 mg/kg intramuscularly) will reliably terminate the agitated state of a person within 3–5 min, enabling the patient to be disrobed, intravenous access to be established and vital signs

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to be monitored (Takeuchi 2011; O'Brien 2020). In the prehospital arena, intramuscular ketamine is building an evidence base, with a call for more standardised regimens to be developed (Melamed 2007; Linder 2018). Airway complications are the most common reported adverse event (Burnett 2012; Hayes 2015; Riddell 2017; Yap 2019). Once more-extreme agitation has settled, benzodiazepines may be titrated intravenously to maintain an adequately sedated state.

Cooling

The risk of serious health complications and death from ABD primarily correlates with the peak temperature and duration of hyperthermia. Once control of the agitation has been established, rapid reduction of body temperature is imperative. Antipyretics such as paracetamol are ineffective, and physical methods are indicated: cooled intravenous fluids assist as a temporising measure but are limited by the volume that can be administered. The application of ice to the whole body (not just packs) and water sprays can rapidly reduce the body temperature; the deployment of medical equipment specifically to control body temperature offers a further option for cooling.

Intravenous fluids

Fluid loss secondary to hyperthermia and agitation increases the predisposition to sudden cardiac arrest. During the initial phase of resuscitation, a couple of litres of cooled fluids are typically administered to support the circulation and improve organ perfusion. Some practitioners administer bicarbonate in the prehospital environment to address the suspected acidosis (Kleinman 2009; Maher 2014). Although the total fluid deficit may be much more than this, further fluids are infused at a slower rate to avoid precipitating acute heart failure and the accumulation of fluid in the lungs.

Specialised inputs

Further interventions are dictated by the clinical picture; the use of renal replacement therapy in kidney failure, mechanical ventilation and the replacement of various electrolytes may be necessary.

Outcomes

Recovery

Current evidence suggests that the best determinants for the outcomes of ABD relate primarily to the duration and peak temperature of hyperthermia, the premorbid physical condition of the individual and the overall duration of the episode. Body mass indices (BMI) greater than 25 kg/m^2 – classed as overweight – are associated with a greater risk of death, regardless of whether the excess mass is due to muscle or fat.

Morbidity

Hyperthermia causes rhabdomyolysis – muscle protein leakage into the circulation – which can lead to acute kidney injury. Impairment of renal functioning further contributes to acidosis, as the failure to make urine leads to an accumulation of waste products.

The liver is also susceptible to varying levels of damage secondary to sustained hyperthermia. Metabolic processes vital for life are impaired, and a poor prognostic sign is the requirement for a glucose infusion – indicative that the liver is unable to maintain glucose homeostasis.

Cardiac contractility is weakened in acidotic states, and low blood pressure may require pharmacological vasopressor support. This can lead to pulmonary oedema as the strength of heart muscle contraction precipitously falls.

At any point, particularly if the temperature is above 41.5°C, disseminated intravascular coagulation (DIC) may occur – with an almost uniformly terminal prognosis in this context. DIC is an uncontrolled activation of the clotting mechanisms within the body. Here, it occurs as the raised temperature accelerates the activity of blood clot enzymes, leading to the formation and consumption of 'micro clots' in the blood. Consequently, spontaneous haemorrhages occur throughout the body, with bleeding into the brain, lungs, bowel and other organs. Death may occur at any stage secondary to haemorrhage, cardiac failure, sepsis or multi-organ failure.

Mortality

Mortality rates have been estimated at just under 10%, and death has been reported within minutes of first responders arriving. Professionals, especially police, will have to balance the risks the agitation poses to the affected individual and to others around them with the risks of restraint.

Postulated mechanisms of sudden death include hypoxia, respiratory compromise (suffocation), direct lethality of electronic control weapons, so-called 'hog-tying' restraint methods and direct drug toxicity; the area is heavily contested and there is a lack of scientific consensus.

ECG changes seen to occur during an episode of ABD are associated with sudden death in individuals who do not have ABD. From medical experience, it is well recognised that cardiac arrest from any cause in someone with metabolic acidosis has a very low probability of successful resuscitation. The gross physiological disturbance leading to the acidosis consumes the body's reserves to restore

MCQ answers
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normal functioning, and it is therefore usually impossible to restore the conditions necessary for the body to function. Sudden death after application of restraint is rarely due to the restraint procedure itself (Mirchandani 1994), rather the sudden release of catecholamines triggered by the arrival of first responders can, against a background of severe acidosis, stun the heart, dissociating the electrical activity from initiating muscle contraction, leading to cardiac arrest. This cardiac rhythm – termed pulseless electrical activity – is not amenable to defibrillation and even with immediate cardiopulmonary resuscitation the outcome is bleak (Stratton 2001).

Results from post-mortem studies usually do not identify any physical changes or injuries (other than those sustained from resisting restraint, falls or cardiopulmonary resuscitation). Testing for acidosis is only a 'point in time' measurement and can rapidly change within minutes; after cardiac arrest the measurements do not reflect those found prior to circulatory cessation. Catecholamine measurements are not considered reliable at post-mortem, owing to the rapid degradation and release from neurons secondary to death.

Conclusions

Acute behavioural disturbance (ABD) is an emergency condition with many causes and one final common pathway that may prove fatal. It is one that psychiatrists need to understand, especially those working in acute in-patient settings. Failure to recognise the condition and lack of 'standard' protocols for managing agitated patients risk deterioration and death. No symptom is pathognomonic, ABD is not a diagnosis and it is best considered as a common-end pathway with multiple causes. However, illicit drug use, especially cannabis and some new/novel psychoactive substances, appear particularly important in terms of causation, although good data remain difficult to obtain.

Prompt assessment and sedation are paramount in the treatment of the condition. However, the choice of sedation agent and depth of sedation are dependent on the treatment facilities available. It is most likely that transfer to the local emergency department is indicated and a review of transfer policies in conjunction with emergency medicine is recommended.

Nevertheless, psychiatric services can instigate several helpful initial steps, including sedation, verbal de-escalation, avoidance or minimisation of any control and restraint, and assisting the emergency department through clear handover and support of a patient whom they will understand better.

As well as staff awareness and training on ABD, individuals working in some specific environments might wish to consider any need for protocols on monitoring and moving patients in more secured or segregated units, where policies and Mental Health Act legislation might make rapid enactment of care more problematic. This includes, but is not limited to: police custody suites, segregation units in prisons, seclusion rooms in psychiatric intensive care units (PICUs) and forensic acute admissions units.

Finally, we are reminded of legal issues and litigation surrounding ABD that have, to date, largely been aimed at police services. Although we are unaware of any medicolegal action against National Health Service mental health trusts or professionals, this is a possibility. In particular, mental health control and restraint practice could be exposed to scrutiny and challenge in a case of ABD where it was argued that it led to, or exacerbated, symptoms. Equally, internationally, courts have shown recognition that restraint can at times be essential and unavoidable, for the safety of the patient and for others. It also seems possible that a charge of medical negligence could be levelled against a doctor who failed to recognise and treat ABD. In such instances more broadly, practice is typically measured against reasonable expectations for a doctor, given their training, experience and specialty, and what their peers might be anticipated to do. As ABD grows in incidence and visibility, this might become an increasing problem. Although we are unaware of any evidenced educational programmes on ABD aimed at staff or drug consumers, individual units might consider it appropriate to trial such engagement where local risks or drug consumption are considered particularly high, for example in some prison settings.

Author contributions

Both authors contributed to the writing of this article.

Declaration of interest

None.

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MCQs

Select the single best option for each question stem.

1 Which of the following is not a recognised cause of ABD?
- a schizophrenia
- b cocaine
- c antipsychotic medication
- d cerebrovascular accidents
- e bipolar affective disorder.

2 Which of the following is not a factor in the controversy regarding ABD?
- a it is linked with death in police custody
- b it is not an ICD-10 diagnosis
- c terminology and naming have varied widely
- d disproportionate rates have been seen in some minority communities
- e it was unknown before the 1990s.

3 Which of the following is not a recognised sign of ABD?
- a narrowed pinpoint pupils
- b skin hot to the touch
- c violent behaviour
- d rapid breathing
- e raised pain threshold.

4 Which of the following is not part of the pathophysiology of ABD?
- a hyperthermia
- b hypotension
- c metabolic acidosis
- d raised serum CO_2
- e electromechanical dissociation of the heart.

5 Treatment of ABD in psychiatric services:
- a should avoid the use of sedatives
- b must involve appropriate physical restraint
- c should involve administration of intravenous fluids
- d can involve antipsychotic medication and benzodiazepines
- e includes an up-to-date ECG.

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