Acute Behavioural Disturbance

A condensed version of the RCEM 2016 guide

Summary of recommendations

1. Excited Delirium / Acute Behavioural Disturbance (ABD)[1] is a medical emergency – affected individuals may suffer sudden cardiovascular collapse and/or cardiac arrest with little or no warning.
2. Patient restraint time in ABD should be kept to an absolute minimum - the degree of restraint used must be justifiable, reasonable, for the minimum time necessary and proportional to the situation.
3. Sedation should be with intravenous benzodiazepines, ketamine or antipsychotics. If the intravenous route is not immediately available then intramuscular administration should be used.
4. Early and aggressive management of hyperthermia and acidosis should be instituted and a high index of suspicion for the development of rhabdomyolysis and Disseminated Intravascular Coagulation (DIC) should be maintained.

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Introduction

Acute Behavioural Disturbance (ABD) is the accepted terminology adopted by the UK Police Forces, the Ambulance Services and the Faculty of Forensic and Legal Medicine[2]. It describes the sudden onset of aggressive and violent behaviour and autonomic dysfunction, typically in the setting of acute on chronic drug abuse or serious mental illness.

ABD is associated with sudden death in approximately 10% of cases[3].
High profile deaths of individuals displaying features of ABD have occurred whilst they have been in police custody.

Box 1: Physical symptoms and signs typical of ABD

- Extremely aggressive/violent behaviour
- Excessive strength/continued struggle despite restraint
- Insensitive to pain
- Acute psychosis with fear of impending doom
- Constant physical activity without fatigue
- Hot to touch/profusely sweating/inappropriate state of undress
- Hyperthermia
- Tachypnoea
- Tachycardia

Differential Diagnoses of ABD

- Heat Stroke
- Neuroleptic Malignant Syndrome
- Serotonin Syndrome
- Thyroid Storm
- Sepsis

- Substance intoxication / withdrawal
- Hypoxia
- Hypoglycaemia
- Head Injury / Seizures
- Akathisia

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Management

The initial aim of management of an individual with ABD should be the rapid tranquilisation and minimisation of their hyper-exertional state.

1. Restraint

- Verbal calming and de-escalation techniques maybe be used as the first line intervention.
- However, these patients are often highly agitated and aggressive with an altered mental status making their response to de-escalation techniques unpredictable.
- Physical restraint to facilitate their initial management may be inevitable. This should be kept to a minimum using a level of force that is justifiable, reasonable and proportional to the individual case and rapidly followed by sedation with close monitoring of vital signs[1]
- Physical restraint has been associated with injuries to patients as well been a contributing factor in patient deaths and particular care must be exercised to ensure that at no time the patient's airway is compromised, this is particularly likely if the patient is kept in a face down position (e.g. due to spitting or biting) with pressure applied on the patient's neck or shoulder region.

Keeping the patient in a prone position MUST be avoided.

- Remember that significant physiological derangements (acidosis, electrolyte abnormalities, cardiac arrhythmias etc.) can occur due to the underlying condition (eg. excited delirium) or as a result of resisting restraint and may be exacerbated by comorbidities (eg. cardiac disease) or medication / illicit substances.

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2. Sedation

- Ideally sedation should be administered via the intravenous route however this route is unlikely to be immediately available.
- An Intramuscular (IM) agent of sufficient strength to allow rapid control of the patient followed by cannulation and monitoring in a high dependency area, May be used
- IM medication can work far more rapidly when an individual is agitated or physically overactive.
- ABD may well need much higher doses of sedative agents than are typically required or recommended[12].

Whichever sedative agent is chosen, it must be one that the treating ED physician is familiar with.

Full patient monitoring (RCEM guidance on safe procedural sedation) including $EtCO_2$ monitoring, must be used in all cases in which sedation is administered if possible.

Early involvement of other specialties such as anaesthetics should be considered.

a.Benzodiazepines

- There is variability in the dose response and this often necessitates active titration – this may be undesirable in ABD where rapid and predictable sedation is essential.
- There is a relatively slow, and often unpredictable, onset time when benzodiazepines are given IM and this route is more commonly associated with adverse events.
- However, IM lorazepam is recommended by NICE as the first line agent for use in rapid tranquilisation for the short- term management of violence and aggression[1].
- The most common serious adverse effect with benzodiazepine use is respiratory depression which may exacerbate the acidosis. These may be compounded by alcohol or other hypnotics already consumed by the individual.

b.Ketamine

- Has a very rapid onset of action when administered IV or IM and has a wide therapeutic window producing consistent effects at predictable doses.
- Ketamine protects airway reflexes and increasing doses lead to more prolonged duration of sedation whilst rarely affecting respiratory drive[9].
- However, ketamine does inhibit the reuptake of catecholamines leading to the potential for sympathomimetic side effects such
    - Tachycardia
    - Hypertension
    - Increased cardiac output
    - Increased myocardial oxygen consumption.

  Thus a theoretical risk of worsening cardiovascular instability.
- Ketamine may be associated with unpleasant emergence phenomenon, although this is readily managed by the administration of benzodiazepines.

c.Antipsychotics

- Traditional neuroleptics such as haloperidol have well recognised side effects such as prolongation of the QTc interval and cardiac arrhythmias.
- NICE does recommend the use of haloperidol with promethazine for rapid tranquilisation in the management of violence and aggression but **ONLY if the patient has taken antipsychotic medication previously or they have previously had an ECG**[1].
- All antipsychotics can lower the seizure threshold, have anti cholinergic effects, precipitate acute dystonic reactions and may rarely lead to neuroleptic malignant syndrome.
- Antipsychotics such as droperidol, or the newer atypical agents such as olanzapine, have been shown to be more effective sedating agents than midazolam with less adverse events[11, 12]

3. Supportive Management in hospital

Procedural sedation should be followed by a rapid assessment of the patient.

- Thorough physical examination
- Temperature
- Bloods (including FBC, U&E, Bone profile, CK and coagulation profile)
- ECG
- Arterial blood gas - is essential
- Consider other investigations such as further imaging (e.g. CT).
- A collateral history should also be obtained to search for the possible causes.

a. **Hypovolaemia** is common in ABD, attributable to the excess physical activity and hyperthermia, so all patients should receive IV crystalloids[9]. These supplementary IV fluids will help to correct metabolic acidosis and prevent end organ damage.

b. **Acidosis**, the use of sodium bicarbonate to specifically treat metabolic acidosis in the absence of hyperkalaemia in ABD is not recommended - it may exacerbate intracellular acidosis and have a negative inotropic effect on an ischaemic myocardium[13].

c. **Hyperthermia** is common in individuals presenting with ABD. Hyperthermic patients need to be cooled to standard body temperature (and not below). This should be achieved with the institution of basic cooling methods such as the removal of clothing and placing the patient in a cool environment. Active cooling with cooled intravenous fluid, ice packs in the axilla and groins should be used as required[9].

d. **Others**, Patients should be closely monitored for signs of the development of rhabdomyolysis, hyperkalaemia and DIC, all having been reported in cases of ABD[10]. The provision of standard care such as urinary alkalinisation with sodium bicarbonate for rhabdomyolysis is advocated.

Each individual with ABD must have their medical needs met fully before usual police proceedings for any criminal activity begin.

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Appendix 1

Rapid Tranquilisation in ABD: please consult formulary

Medication	Route	Typical dose (mg)	Onset (min)	Duration (min)
Midazolam	I/N	5	3-5	30-60
	IM	5	10-15	120-360
	IV	2-5	1-5	30-60
Lorazepam	IM	4	15-30	60-120
	IV	2-4	2-5	60-120
Diazepam	IM	10	15-30	15-60
	IV	5-10	2-5	15-60
Haloperidol	IM	10-20	15-30	180-360
	IV	5-10	10	180-360
Droperidol	IM	5	10-30	120-240
	IV	2.5	10	120-240
Olanzapine	IM	10	15-45	
	IV (unlicensed)	5		
Ketamine	IM	2-4mg/kg	3-5	60-90
	IV	1-2mg/kg	1	20-30

References:

1. http://www.nice.org.uk/guidance/ng10
2. Faculty of Forensic and Legal Medicine. Acute behavioural disturbance: guidelines on management in police custody. January 2016
3. American College of Emergency Physicians White Paper Report on Excited Delirium Syndrome September 2009
4. Gill JR. The syndrome of excited delirium. *Forensic Sci Med Pathol* 2014; 10: 223-228.
5. Vilke GM, Bozeman WP, Dawes DM et al. Excited Delirium Syndrome (EXDS); Treatment Options and Considerations. *Journal of Forensic and Legal Medicine* 2012; 19:117-121.
6. Vilke GM, Payne-James J, Karch SB. "Excited delirium (ExDS): Redefining an old diagnosis. *Journal of Forensic and Legal Medicine* 2012; 19:7-11
7. Hick JL, Smith S, Lynch MT. Metabolic acidosis in restraint-associated cardiac arrest: a case series. *Academic Emergency Medicine* 1999; 6(3): 239-243.
8. Dimsdale JE, Hartley LH, Guiney T et al. Post exercise peril - plasma catecholamines and exercise. *JAMA* 1984; 251(5): 630-632.
9. Green SM, Roback MG, Kennedy RM, Krauss B. Clinical practice guideline for emergency department ketamine dissociative sedation: 2011 update. *Annals of Emergency Medicine* 2011; 57(5): 449-461.
10. Vilke GM, DEBard ML, Chan TC et al. Excited Delirium Syndrome (EXDS): Defining based on a review of the literature. *Journal of Emergency Medicine* 2012; 43(5): 897-905.
11. Chan EW, Taylor DM, Knott JC et al. Intravenous droperidol or olanzapine as an adjunct to midazolam for the acutely agitated patient: a multicenter, randomised, double blind, placebo controlled clinical trial. *Ann Emerg Med* 2013; 61:72-81.
12. Ibister GK, Calver LA, Page CB et al. Randomised controlled trial of intramuscular droperidol versus midazolam for violence and acute behavioral disturbance: The DORM study. *Ann Emerg Med* 2010; 56:392 - 401.
13. European Resuscitation Council Guidelines for Resuscitation 6th Edition January 2011.

Other useful publications

- Di Maio TG, Di Maio VJM. Excited Delirium Syndrome – Cause Of Death and Prevention. USA: CRC Press; 2006.
- Wetli CV, Fishbain DA. Cocaine induced psychosis and sudden death in recreational cocaine users. J Forensic Sci 1985; 30(3): 873-80.
- Hughes EL (ed). Special Panel Review of Excited Delirium. Seattle, Washington. April 2011.
- Takeuchi A, Ahern TL, Henderson SO. Excited Delirium. Western Journal of Emergency Medicine 2011; 12(1): 77-83.
- Stratton S, Rogers C, Brickett K, Gruzinski G. Factors associated with sudden cardiac death of individuals requiring restraint for excited delirium. American Journal of Emergency Medicine 2001; 19(3): 187-191.
- Ruttenber J, McAnally HB, Wetli CV. Cocaine associated Rhabdomyolysis and Excited delirium: different stages of the same syndrome. The American Journal of Forensic Medicine and Pathology 1999; 20(2): 120-127.
- Bozeman WP, Ali K, Winslow JE. Long QT syndrome unmasked in an adult subject presenting with excited delirium. Journal of Emergency Medicine 2013; 44(2): 207-210.
- Mash DC, Duque L, Pablo J et al. Brain biomarkers for identifying excited delirium as a cause of sudden death. Forensic Sci Int (2009), doi:10.1016/j.forsciint.2009.05.01.
- Otahbachi M, Cevik C, Bagdure S, Nugent K. Excited delirium, Restraints and Unexpected Death: A review of pathogenesis. Am J Forensic Med Pathol 2010; 31: 107-112.
- Chan TC, Vilke GM, Neuman T, Clausen J. Restraint position and positional asphyxia. Ann Emerg Med 1997; 30(5): 578-86.
- Chan TC, Neuman T, Clausen J et al. Weight force during prone restraint and respiratory function. Am J Forensic Med Pathol 2004; 25(3): 185-9.
- Hirota K, Lambert DG. Ketamine: its mechanism(s) of action and unusual clinical uses. BJ Anaesthesia 1996; 77(4): 441-444.
- Svenson JE, Abernathy MK. Ketamine for pre hospital use: new look at an old drug. American Journal of Emergency Medicine 2007; 25:977-98.
- Burnett AM, Watters BJ, Barringer KW et al. Laryngospasm after intramuscular administration of ketamine to a patient in excited delirium. PreHospital Emergency Care 2012; 16:412-414.
- Ho JD, Smith SW, Nystrom PC et al. Successful management of Excited Delirium Syndrome with pre hospital ketamine: two case example. Pre Hospital Emergency Care 2013; 17: 274-2.
- Maher PJ, Walsh M, Burns T, Strote J. Prehospital resuscitation of a man with excited delirium and cardiopulmonary arrest. CJEM 2014; 16(1): 80-83.
- Kodikara S, Cunningham K, Pollanen MS. Excited delirium syndrome: Is it a cause of death? Legal Medicine 2012; 14: 252-254.
- Penders TM, Gestring RE, Vilensky DA. Excited delirium following use of synthetic cathinones (bath salts). General Hospital Psychiatry 2012; 34: 647- 650.
- Hall C A, Kader AD, McHale AMD et al. Frequency of signs of excited delirium syndrome in subjects undergoing police use of force: Descriptive evaluation of a prospective, consecutive cohort. Journal of Forensic and Legal Medicine (2012), http://dx.doi.org/10.1016/j.jflm.2012.05.008.