

Management of patients with suspected but unidentified poisoning in the emergency department: a joint Royal College of Emergency Medicine and National Poisons Information Service best practice guideline

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ABSTRACT

The management of patients suspected, but not confirmed, as being poisoned is challenging. The Royal College of Emergency Medicine and National Poisons Information Service have produced this guidance to provide a generalised clinical approach to any poisoned patient in the emergency department. This guideline provides a clinical approach to support the initial assessment of a patient, identification of potential toxic agents and emergency management. A toxidromic approach is used, with emphasis on consideration of the toxicokinetics of potential poisons and how the patient's clinical condition may change. It does not replace poison-specific guidance available from TOXBASE and the NPIS or a locally appropriate poisons centre.

PREFACE

The Royal College of Emergency Medicine (RCEM) in collaboration with the National Poisons Information Service (NPIS) has produced this best practice guideline (BPG) to assist clinicians in the initial assessment of a patient presenting to a UK emergency department (ED) with a suspected but unidentified poisoning.

This guideline was developed in line with RCEM BPG policy by circulation through all members of the RCEM Medical Toxicology Special Interest Group, the Quality in Emergency Care Committee and the regional directors and consultants from NPIS for review and comment prior to endorsement by both organisations. This guideline was first published on the RCEM website in April 2025.¹

SUMMARY OF RECOMMENDATIONS

- For up-to-date poison-specific guidance use TOXBASE (poisons information database <https://www.toxbase.org/>) and the National Poisons Information Service (NPIS), if in the UK, or a locally appropriate Poisons Centre and database.² (Recommendation level C)
- Poisoning may be implicated whenever diagnostic uncertainty exists in the undifferentiated patient. (Recommendation level C) Traumatic

injury may need to be ruled out in potentially poisoned patients. (Recommendation level C)

- Identification of causative agents should not delay the administration of emergency treatments to stabilise respiratory, cardiovascular or central nervous system function. (Recommendation level C)
- The NPIS 'Poisoning with an unknown substance' document (annex A on the RCEM website¹ or at TOXBASE)² provides useful guidance to aid toxidrome and potential agent identification. (Recommendation level C)
- ECGs and blood gases may need to be repeated several times based on the clinical situation. (Recommendation level C)
- Management should not be based solely on the reported exposed substance as other agents may be involved. In particular, illicit street drugs and drugs bought over the internet may not contain the advertised or claimed active substance or may contain multiple substances and adulterants. (Recommendation level C)
- Patients having been subjected to an unknown poison can be considered safe for discharge if they are physiologically and biochemically stable, have capacity and no further deterioration is anticipated. The time period of observation for signs of further deterioration should be based on the presenting toxidromes and potential poisons guided by TOXBASE-specific information. (Recommendation level C)

An explanation of the Strength of Recommendation Taxonomy is given in [table 1](#).

SCOPE

This guideline deals with patients presenting to the ED suspected of having been poisoned with an unknown substance. The diagnosis of acute poisoning can be challenging when no clear agent has been identified. This guideline provides a generalised approach to all patients suspected of being poisoned. It does not replace specific guidance available from TOXBASE and the NPIS.

This guideline is focused on the individual ED patient managed in the UK. The general approach to a poisoned patient, recommended in this guideline, may aid in recognition of a deliberate or accidental

Table 1 Strength of Recommendation Taxonomy²⁵

Strength of recommendation	Definition
A	Recommendation based on consistent and good-quality patient-oriented evidence.
B	Recommendation based on inconsistent or limited-quality patient-oriented evidence.
C	Recommendation based on consensus, usual practice, opinion, disease-oriented evidence or case series for studies of diagnosis, treatment, prevention or screening.

chemical release. However, the specific clinical or operational issues associated with Chemical, Biological, Radiological and Nuclear (CBRN) or Hazardous Materials (HAZMAT) incidents are not addressed in this work.

REASON FOR DEVELOPMENT

The assessment and treatment of poisoned patients is common in Emergency Medicine. Emergency physicians should be able to recognise acute poisoning, anticipate deterioration and initiate management.

The majority of clinically significant UK poisonings, for which the TOXBASE poisons database is accessed, result from exposure to pharmaceutical agents.³ However, the causative agent is not always clear, and patients may require emergency management regardless of whether likely agents have been identified.

The toxidromic approach taken within this document seeks to avoid diagnostic bias when assessing potentially poisoned patients so that toxicological and non-toxicological causes are considered, ruled out or managed as appropriate.

GENERAL APPROACH TO THE POISONED PATIENT

Figure 1 illustrates a general paradigm for the approach to a potentially poisoned patient. An initial assessment should be conducted and immediate interventions made as required.

Poisoning is a consideration whenever diagnostic uncertainty exists in an undifferentiated patient. *Other pathologies, including trauma, may also be present and require investigation and radiological imaging.*

The following initial investigations may guide diagnosis: ECG, venous blood gas (including lactate, chloride, sodium, potassium, glucose) and laboratory tests for full blood count, renal function, liver function tests, magnesium, creatine kinase and international normalised ratio.

Paracetamol concentrations should be measured in all cases that may result from self-harm and be considered in all cases of unknown poisoning.⁴ Additional specific blood tests, for example, salicylate, toxic alcohols and ethanol concentrations, may be useful on a case-by-case basis or when recommended by NPIS.

The history, clinical examination and initial bedside test results may suggest one or more toxidromes or particular class of poison. *This should guide the initial administration of emergency treatments to stabilise the patient.* Potential agents should be considered from both their clinical effects and toxicokinetics. This will allow prediction of how the poisoning may progress, identify additional interventions and pre-empt what management may be required in the future. Toxicokinetic considerations include: *time since exposure, potential ongoing absorption, anticipated time to peak plasma concentration and the elimination half-life.* Continual re-evaluation must take place and clinicians should be prepared to reconsider initial diagnoses as the situation changes.

TOXIDROMES

The term toxidrome refers to a collection of signs, symptoms and investigation findings that suggest a particular class of chemical or individual poison. Not all poisonings result in a clearly identifiable toxidrome, but when present, may assist in clinical management. *It may be necessary to initiate treatment based on the toxidrome alone before a specific agent has been identified.*

The NPIS 'Poisoning with an unknown substance' document provides detailed toxidrome tables and can be found on TOXBASE. TOXBASE also includes a 'symptom search' option, found below the search box, which will produce a list of substances associated with that clinical feature.²

TOXICOLOGICAL HISTORY

The elements of a focused toxicological history are given in box 1.

TOXICOLOGICAL EXAMINATION AND IMMEDIATE INTERVENTIONS

Stabilisation is the clinical priority and interventions may be required before a toxidrome is identified or differential diagnosis developed. See table 2 for potential investigations and interventions.

The CRESS assessment (online supplemental material)—consciousness, respirations, eyes, secretions, skin—has particular usage when a CBRN incident is suspected and will identify the toxidromes associated with the major classes of chemical weapons. It may also be useful in other poisonings, rapidly identifying clinical signs that may aid in diagnosis. It can be conducted at the same time as the A—airway assessment.

Airway

Poisoning via any route of exposure but particularly ingestion may result in aspiration of both normal gastric contents and poisons directly damaging to the respiratory system. Signs of thermal or caustic damage to the airway should be sought. Consider airway protection with a cuffed tube if protective reflexes are lost or airway swelling is anticipated. Testing the gag reflex is not recommended as it may induce vomiting and aspiration. A Glasgow Coma Scale (GCS) Score <9 should not be used as the sole determinant of the need for invasive airway protection.⁵

Breathing

Pulse oximetry should not be relied upon in cases of potential carbon monoxide poisoning, methaemoglobinaemia, sulphaemoglobinaemia or exposure to pigmented dyes such as methylthioninium chloride.

Inhalation may cause direct damage to alveoli and impair gas exchange.

Poisoning may also affect neuromuscular function, resulting in respiratory muscle failure. If respiratory failure is suspected, serial arterial blood gases and end-tidal carbon dioxide monitoring should be used to guide intervention.

Circulation

Hypotension may result from reduced cardiac output, vasodilation or hypovolaemia. If not resolving with initial intravenous fluids, vasopressor and inotropes may be required. The use of high-dose insulin euglycaemia therapy may be required in severe β -adrenergic or calcium channel blockade.^{6,7} Early discussion with NPIS can assist in the selection of supportive agents.

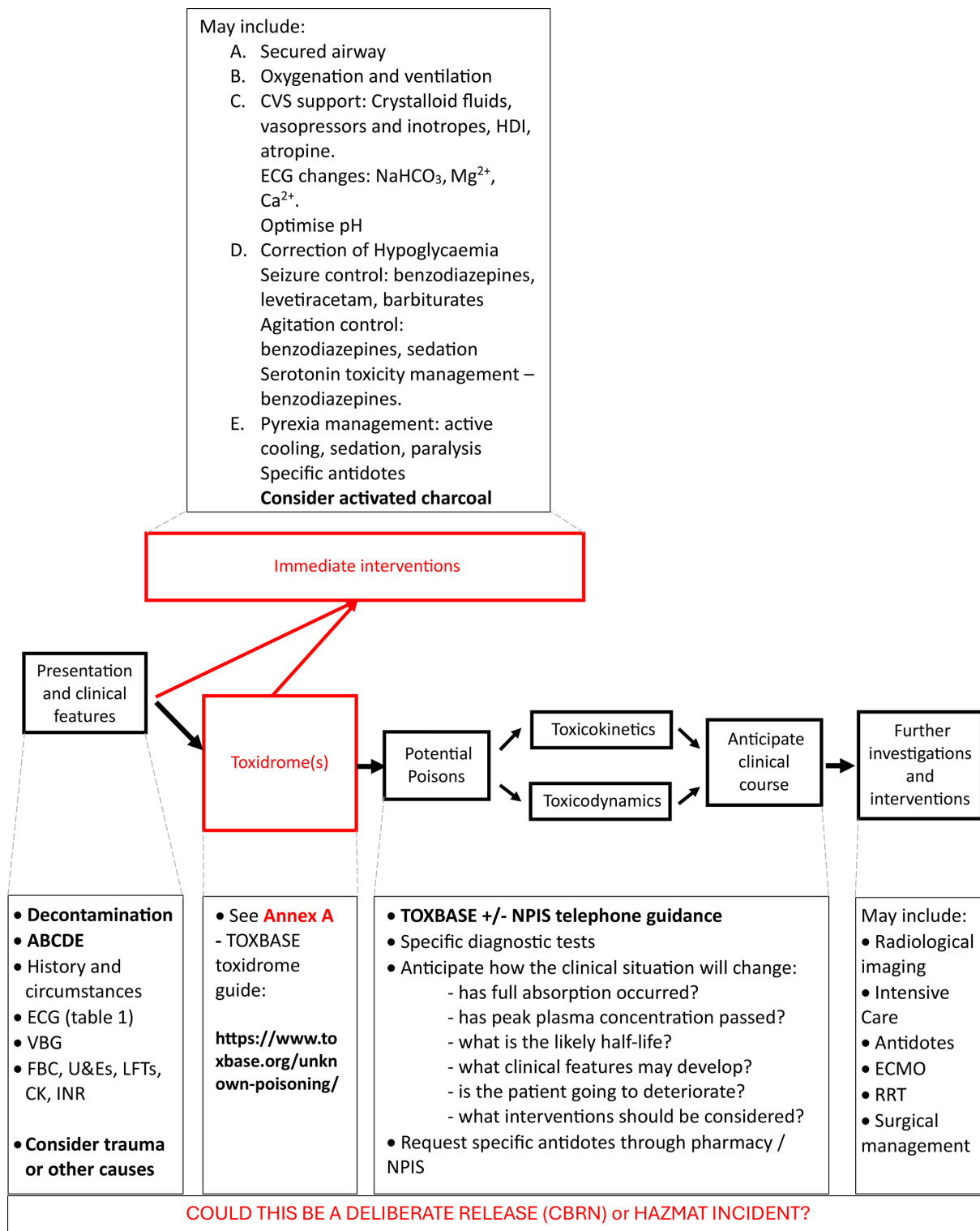


Figure 1 The general approach to a poisoned patient. CBRN, Chemical, Biological, Radiological and Nuclear Materials; CK, creatine kinase; CVS, cardiovascular system; ECMO, extracorporeal membrane oxygenation; FBC, full blood count; HAZMAT, Hazardous Materials; HDI, high dose insulin; INR, international normalised ratio; LFTs, liver function tests; NaHCO_3 , sodium bicarbonate; NPIS, National Poisons Information Service; RRT, renal replacement therapy; U&Es, urea and electrolytes; VBG, venous blood gas.

Seek ECG abnormalities early and treat as soon as identified to prevent deterioration. These include electrolyte-associated changes, QRS abnormalities resulting from sodium channel blockade and QT prolongation.

ECG QT calculation table 3: Prolongation of the QT interval is associated with torsades de pointes (TdP) and

ventricular arrhythmias. The QT interval is best calculated manually from the start of the Q-wave to where the T-wave returns to the isoelectric baseline. This is ideally done in three chest leads and three limb leads, with the median of the measured QT intervals being plotted against the heart rate on the nomogram in figure 2.^{8,9}

Box 1 Focused toxicological history

- ⇒ Circumstances of current presentation: time of exposure/time last seen well, potential agent(s) involved and dose
- ⇒ Prior medical history
- ⇒ Prescribed medications
- ⇒ Over-the-counter medications, herbal, ayurvedic, Chinese or other traditional medicines
- ⇒ Access to other medications
- ⇒ Use of illicit drugs, new psychoactive substances or alcohol
- ⇒ Plants, mushrooms, venomous animals
- ⇒ Occupational history and exposures
- ⇒ Other affected people
- ⇒ Personal history of political or criminal activity

Arrhythmia management [table 3](#): In poisoned patients, arrhythmias may result from disruption of cardiac conduction by the agent(s) or from secondary electrolyte disturbance. In the context of an unknown poisoning, TOXBASE/NPIS recommend urgent treatment with intravenous sodium bicarbonate in patients with drug-induced QRS prolongation, to manage potential sodium channel blockade from commonly exposed drugs, for example, tricyclic antidepressants, cocaine and class 1A and 1C antiarrhythmics.¹⁰

If the QT-HR lies above the nomogram line ([figure 2](#)), check serum potassium, calcium and magnesium and, if the concentrations are low, replace as required. Continuous cardiac monitoring and repeated 12-lead ECGs will identify evolving abnormalities.¹¹ If ECG instability develops in the context of QT prolongation, for example, TdP, replace potassium, magnesium and calcium as required and manage as per advanced life support protocols. NPIS can be contacted for further advice.

Toxic-induced arrhythmias may be refractory to electrical cardioversion without appropriate antidote administration.¹⁰ NPIS and intensive care medicine advice should be sought early in these cases.

A venous or arterial *blood gas sample*, including point-of-care electrolytes and the anion gap, may point towards specific poisons. If toxic alcohols are suspected, the anion gap should be repeated after a few hours to look for a rise and compared with the osmolar gap taken on admission and with the repeat blood gas. Advice on interpretation can be sought from NPIS.

Disability

Consciousness may fluctuate in poisoned patients and should be frequently reassessed. The GCS, although useful as a shorthand for the degree of altered mental status, must never be used for prognostic purposes in the acutely poisoned patient. Most patients in a toxic-induced coma can be expected to make a full recovery with appropriate medical management.^{5 12 13} Where the diagnosis remains unclear and consciousness does not improve, patients with reduced consciousness will require brain imaging, initially with CT head, to investigate for intracranial pathology.

Recurrent or prolonged seizures:

Benzodiazepines are recommended for initial management. TOXBASE provides further guidance for second-line treatments. *Phenytoin should be avoided due to its sodium channel blockade properties.*¹⁴

Poisoning-focused neurological signs:

- ▶ Pupillary size may aid diagnosis but is a poor marker of treatment responsiveness in opioid and cholinergic toxidromes.
- ▶ Impaired visual acuity may suggest quinine or methanol poisoning.

Table 2 Examination and interventions—poisoning-related considerations

	Considerations	Potential investigations	Potential interventions
Airway (and CRESS assessment)	Vomiting Aspiration Swelling Burns (chemical or thermal)	Flexible nasal endoscopy	Early consideration of intubation Left lateral position Airway adjuncts
Breathing	<i>Is pulse oximetry reliable?</i> Pneumonitis Neuromuscular failure Hypoventilation	ABG ETCO ₂ MetHb CO	Supplemental O ₂ , HFNO Positive pressure ventilation ECMO
Circulation	Arrhythmias are common Hypotension may be multifactorial	Repeated ECGs Continual cardiac monitoring Frequent blood pressures Invasive BP monitoring VBG/ABG including point-of-care electrolytes	Treat ECG changes immediately (table 3) Intravenous fluids Vasopressors and inotropes Atropine High-dose insulin therapy
Disability	Consciousness may fluctuate Neuroimaging should be considered for non-toxicological causes or if focal neurology is present Seizure control. Hyperthermia Serotonin toxicity, neuroleptic malignant syndrome	ABG/VBG Glucose Consider CT head	Seizure control with benzodiazepines Naloxone when appropriate
Exposure and environmental	Hyperthermia and hypothermia management Signs of residual agent	Invasive temperature monitoring Residual agents should be collected and stored for testing	Decontamination should be done as early as possible alongside treatment

ABG, arterial blood gas; BP, blood pressure; CO, carbon monoxide; CRESS assessment, Consciousness, Respirations, Eyes, Secretions, Skin assessment; ECMO, extracorporeal membrane oxygenation; ETCO₂, end tidal carbon dioxide monitoring; HFNO, high flow nasal oxygen; MetHb, methaemoglobinaemia; VBG, venous blood gas.

Table 3 Poisoning-related ECG abnormalities^{26 27}

Initial ECG changes	Arrhythmia after deterioration	Potential poisons	Initial management
QRS prolongation (>120 ms) May indicate sodium channel blockade. Compare with previous ECGs if available. May be preceded by: Dominant R wave aVR Dominant S wave aVL	Ventricular arrhythmias	Antiepileptics, Tricyclic antidepressants, Cocaine, Diphenhydramine, Class 1A and 1C antiarrhythmics	Sodium bicarbonate (NaHCO₃): QRS > 160 ms, cardiac arrest or VT: 100 mL 8.4% NaHCO ₃ intravenous bolus QRS 120–160 ms: 50 mL 8.4% NaHCO ₃ intravenous bolus Children with QRS prolongation: 1–2 mL/kg 8.4% (centrally) or 2–4 mL/kg 4.2% (peripherally). If cardiac arrest or VT as a bolus. If prolonged QRS alone over 20 mins.
QT interval prolongation ► The uncorrected QT interval should be compared with the nomogram in figure 2 ²⁸ ► Automated QTc should not be used	Ventricular arrhythmias TdP	Antipsychotics, for example, haloperidol, risperidone, quetiapine, amisulpride Selective serotonin reuptake inhibitors (SSRI) e.g. citalopram, escitalopram Tricyclic antidepressants, for example, amitriptyline Opioids, for example, methadone Cardiac drugs, for example, amiodarone, sotalol. Antihistamines, for example, diphenhydramine Antimicrobials, for example, fluoroquinolones, macrolides Arsenics	For QT: HR above the nomogram line: Check serum magnesium, potassium and calcium. Replace if concentrations are low. For TdP: Magnesium sulphate: Adults: 2 grams magnesium sulphate intravenous over 10 mins Children: 25–50 mg/kg magnesium sulphate intravenous (max 2 grams) over 10–15 mins Pharmacological or electrical pacing may be considered
P wave flattening PR interval prolongation	Junctional or ventricular escape rhythms	Cardiac glycosides (digoxin, foxglove, oleander) β-blockers, Calcium channel blockers, Cholinergics (organophosphates, carbamates, pilocarpine, muscarine containing mushrooms)	Agent specific Digoxin/cardiac glycosides: consider DigiFab as per TOXBASE Correct electrolyte abnormalities
ST segment elevation		Cocaine Hypotension from multiple agents.	Manage as acute coronary syndrome Benzodiazepines and nitrates for cocaine
ST ‘sagging’ with QRS prolongation, T wave inversion, shortened QT with PR prolongation	Bradycardia Ventricular arrhythmias	Digoxin and other cardiac glycosides	Atropine
Brugada type pattern: Coved ST elevation V1–3	Ventricular arrhythmias	Tricyclic antidepressants Cocaine Lithium Class 1A and 1C antiarrhythmics	Sodium bicarbonate Agent-specific management

TdP, torsades de pointes.

- Nystagmus and optical divergence are common with agents affecting the central nervous system (CNS) including ethanol, antiepileptics, benzodiazepines and tricyclic antidepressants.
- Ankle/ocular clonus and hyperreflexia should be assessed as signs of serotonin toxicity.

Serotonin toxicity is suggested in the presence of the following:

- Altered mental state: confusion, agitation, progressing to reduced consciousness and coma.
- Neuromuscular hyperactivity: hyperreflexia, clonus, tremors.
- Autonomic instability: tachycardia, fever, hypertension or hypotension. Diarrhoea and vomiting may also occur.

Neuroleptic malignant syndrome is associated with antipsychotic use and can occur at any time during use or cessation. It most commonly occurs following initiation and may be triggered by rapid dose increases or switching between medications. It may present similarly to serotonin toxicity but differs in producing ‘lead pipe’ rigidity and usually with a slower onset.

Exposure and environmental factors

Temperature management. Temperature monitoring may need to be repeated at regular intervals or invasively monitored.

- Hyperthermia (>38.5°C), if felt to be the result of intoxication, should be managed by active cooling, the administration of benzodiazepines and antidotes specific to the suspected causative agent. Further detail is available on TOXBASE.
- Hypothermia may require active rewarming.

Diarrhoea and vomiting. Antiemetics and antidiarrhoeals may cause QT prolongation and have CNS effects in interaction with other exposed substances.

Cutaneous injury, rashes and skin changes may aid diagnosis.

Residual agent or any unidentified substance must be removed immediately by both dry and wet decontamination, *with appropriate personal protective equipment if considered necessary.*

Other considerations

Rhabdomyolysis may result from many poisons as well as pressure necrosis, seizures and extreme muscular activity. Adequate hydration and urinary output may prevent acute renal failure.

Urinary retention may complicate anticholinergic toxicity or result from treatment with atropine, particularly for organophosphorus insecticide or nerve agent poisoning. Early urinary catheterisation may be helpful.

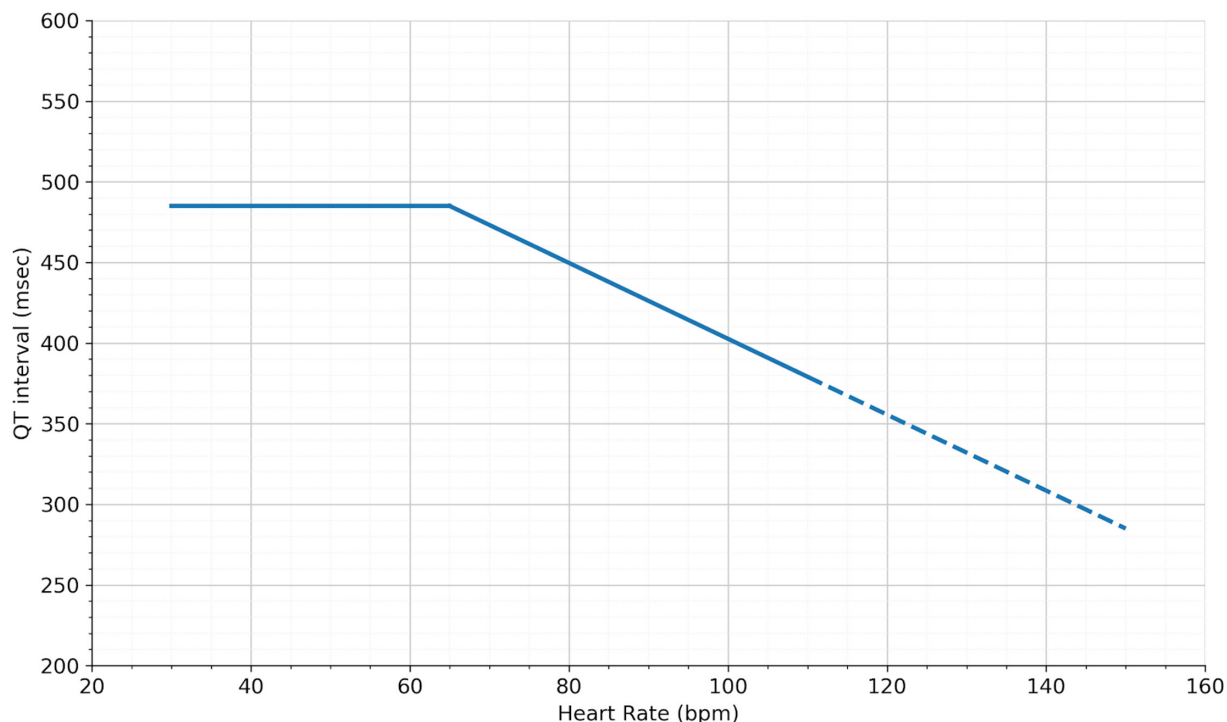


Figure 2 QT interval nomogram for the interpretation of QT and heart rate (HR). QT-HR above the line is 'at-risk' of torsades de pointes (adapted from Chan *et al* [8]).

Sustained release preparations of pharmaceuticals will prolong absorption time and increase the elimination half-life of a substance.

Bezoar formation. Ingestion of large quantities of pharmaceutical tablets may result in the formation of a gastric pharmacobezoar—a packed mass of tablets which may slow absorption of the total dose and then break down to suddenly release high concentrations of drug. This may result in a sudden unexpected clinical deterioration.

Adulteration. Illicit drugs may be adulterated with other active substances, in addition to the intended drug, and unknown to the purchaser. This is done to enhance or alter the effect of the drug, or as a bulking agent to improve marketability. *The name the drug is sold under, or the patient's belief of what has been taken, may not relate to the active chemical.*

Additional investigations

Serum osmolality should be requested and repeated in cases of potential toxic alcohol poisoning. The osmolar gap can then be compared with the anion gap calculated at the same times. TOXBASE and NPIS can provide assistance with interpretation of results.

Drug assays. Specific toxicological assays may be required to aid diagnosis and guide treatment, for example, paracetamol, salicylate, iron, lithium, digoxin, phenytoin, carbamazepine, valproate, theophylline, methotrexate, ethylene glycol or methanol. Carboxyhaemoglobin and methaemoglobin concentration measurements are routinely available through point-of-care blood gas analysers in most EDs. Specific drug assays may also guide the continuation or re-initiation of long-term therapies, have a role in diagnosis of brain death and organ donation, and have medicolegal implications. Local clinical biochemistry staff should be consulted regarding the availability of specific tests and further advice is available from NPIS.¹⁵

Toxicological screening. Drug screening is rarely of clinical value in the acute phase of an intoxication due to the time taken to get laboratory results. *Point-of-care rapid testing should not be used to guide clinical management* due to the low sensitivity and specificity of the tests used and the potential for false positives. In cases of severe unknown poisoning, laboratory assays or time-of-flight mass spectrometry may guide subsequent management, but results must be interpreted with care as part of the wider clinical situation.

Blood ethanol concentrations are not routinely useful in the management of intoxicated patients and patients with raised blood ethanol may have ingested other substances that are contributing to their current illness. Habitual alcohol drinkers may tolerate high blood ethanol concentrations while alcohol-naïve patients may be symptomatic at relatively low concentrations. Ethanol concentrations should be measured urgently in cases of:

- ▶ Coma of unclear cause
- ▶ Widened osmolar gap
- ▶ Suspected severe ethanol poisoning
- ▶ Suspected toxic alcohol poisoning (including methanol and ethylene glycol)
- ▶ Children with unexplained acidosis.¹⁵

Failure to clinically improve, after a period of observation, in patients with high blood alcohol, should prompt investigation for other causes.

CT

- ▶ **CT head.** A CT head should be considered in patients with a reduced consciousness or focal neurological signs.
- ▶ **CT thorax, abdomen and pelvis.** CT scans may be required to confirm or rule out other non-toxic diagnoses. If internal drug packets are suspected, a low-dose CT abdomen and pelvis scan should be considered in line with RCEM guidance.¹⁶

Box 2 Poisons poorly adsorbed by activated charcoal or for which it is not recommended

Boric acid
Cyanide
Ethanol
Ethylene glycol
Ferrous salts (iron)
Lithium
Lead
Malathion
Methanol
Mercury
Hydrocarbons (aspiration risk)
Strong acids
Strong alkalis

Other emergency treatments

Decontamination refers to the removal of a poisonous substance from the patient, *in order to prevent further absorption*. The route of exposure and the likely chemical contaminant determine the type of decontamination required. *Decontamination is a treatment priority to reduce the total exposed dose; however, it must not delay the administration of time-critical treatments which may be life-saving*. Detailed guidance can be found on TOXBASE.²

- ▶ **Gaseous exposures.** Exposure to gases does not generally require decontamination beyond the removal of exposed clothes.
- ▶ **Skin.** Clothing should be removed and the patient dry decontaminated with absorbent material such as blue roll. Most cases will then require cleaning with water and soap. Care should be taken to avoid the exposure of staff to the agent and their vapours.¹⁷
- ▶ **Eyes.** Irrigate eyes thoroughly with crystalloid solution for a minimum of 10–15 min. Test the corneal pH following irrigation aiming for pH 7–7.2.¹⁸
- ▶ **Gut decontamination** is discussed below.

Single dose activated charcoal (SDAC) should be considered for administration either orally or by nasogastric tube if a potentially toxic amount of a substance, known to be bound by activated charcoal, has been ingested. TOXBASE recommends this within an hour of ingestion. *SDAC is contraindicated if the patient is unable to protect their own airway and is not intubated with a cuffed endotracheal tube*. It is also contraindicated when aspiration may be high-risk, such as with hydrocarbon ingestion. Although effective for many poisons, activated charcoal is ineffective at adsorbing the chemicals listed in box 2.¹⁹

Gastric lavage should not be routinely performed for acute poisoning and may cause additional harm.²⁰ In rare cases of life-threatening overdose, not amenable to activated charcoal, it may be considered following discussion with NPIS.

Other treatments including renal replacement therapy, whole bowel irrigation, multidose activated charcoal and urine alkalisation are agent-specific and should be initiated following TOXBASE/NPIS advice.

ANTIDOTES

Antidotes should be given when indicated based on the toxidrome or ideally specific agent. Some antidotes may cause side effects or toxicity if given without the presence of their target agent. The RCEM and NPIS guideline on antidote availability specifies

which antidotes should be immediately available in the ED (category A), which should be held in the hospital and available within 1 hour (category B) and which are held supraregionally (category C) requiring release through contact with NPIS. Exotic animal antivenoms are also held in hubs around the UK and requests for their supply should be discussed with NPIS.²¹

PAEDIATRIC CONSIDERATIONS

Poisoning is a consideration in children whenever a diagnosis is unclear. Children under 5 years of age may accidentally ingest poisons while exploring their environment, deliberate poisoning can be a form of non-accidental injury, and rates of deliberate self-poisoning increase during adolescence and young adulthood. Although the physiology of young children may affect susceptibility to particular agents, the general principles of management, recommended in this guideline, remain the same for all ages. TOXBASE/NPIS should be referred to for specific paediatric guidelines once a likely poison is identified.

PREGNANCY CONSIDERATIONS

Pregnant patients should be managed with the same general principles recommended in this guideline. TOXBASE should be referred to as with all poisonings. The United Kingdom Teratology Information Service (UKTIS) provides systematic evidence reviews for the use of medicines in pregnancy and advises on management after in utero exposure.²² TOXBASE links to UKTIS reviews and provides contact details.²

DISCHARGE PLANNING

The decision of when to discharge is made on a case-by-case basis and an assessment of the risk of clinical deterioration. If patients wish to self-discharge from the ED, a capacity assessment should be conducted with the result recorded in the patient's notes. Many intoxicated patients will not have the capacity to make the decision to leave the ED; the rationale for this decision should be clearly stated in the note and they should be managed in line with the RCEM guideline 'The Mental Capacity Act in Emergency Medicine Practice'.²³

Patients who have been subjected to an unknown poison or poisons can be considered safe for discharge once they are physiologically and biochemically stable, have returned to their baseline level of cognitive function and no further deterioration is anticipated. The time period of observation for signs of further deterioration should be based on the presenting toxidromes and potential poisons guided by TOXBASE specific information.

Patients who have self-harmed either through deliberate overdose or by physical means should have a mental health assessment in line with the RCEM Mental Health in ED guideline.²⁴

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