Royal College of Emergency Medicine

Best Practice Guideline

Management of Patients with Suspected but Unidentified Poisoning in the Emergency Department

NPIS

April 2025

Summary of Recommendations

1. For up-to-date poison specific guidance use TOXBASE® and the National Poisons Information Service (NPIS).

2. Poisoning may be implicated whenever diagnostic uncertainty exists in the undifferentiated patient.

3. Traumatic injury may need to be ruled out in potentially poisoned patients.

4. Identification of causative agents should not delay the administration of emergency treatments to stabilise respiratory, cardiovascular or central nervous system function.

5. The NPIS '*Poisoning with an unknown substance'* **(Annex A)** document provides useful guidance to aid toxidrome and potential agent identification.

6. ECGs and blood gases may need to be repeated several times based on the clinical situation.

7. Illicit street drugs, and drugs bought over the internet, may not contain the advertised or claimed active substance, and may contain multiple substances and adulterants.

8. Patients presenting 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.

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Scope

This guideline addresses the management of patients presenting to the Emergency Department with suspected poisoning from an unknown substance. Diagnosing acute poisoning can be challenging in the absence of a clearly identified agent. This document outlines a generalised approach for assessing and treating all patients suspected of poisoning.

It does not replace, specific guidance provided by TOXBASE® and the National Poisons Information Service (NPIS).

This guideline focuses on individual patients in the Emergency Department setting. It is not intended for use in Chemical, Biological, Radiological, and Nuclear (CBRN) or Hazardous Materials (HAZMAT) incidents, and does not cover the clinical or operational aspects of accidental or deliberate chemical releases, or scenarios involving multiple casualties.

Reason for Development

The assessment and treatment of poisoned patients is a common aspect of Emergency Medicine. Emergency physicians should be able to recognise acute poisoning, anticipate potential deterioration, and initiate appropriate management.
The majority of clinically significant poisonings in the UK—those for which the TOXBASE® poisons database is accessed—result from exposure to pharmaceutical agents [1]. However, the causative agent is not always apparent, and patients may require emergency management regardless of whether likely agents have been identified.

The toxidromic approach adopted in this guideline aims to minimise diagnostic bias when assessing potentially poisoned patients. It encourages consideration of both toxicological and non-toxicological causes, ensuring that each is appropriately ruled out or managed.

Glossary

CBRN - Chemical, Biological, Radiological and Nuclear threats from deliberate, weaponised release.

CRESS Assessment - Consciousness, Respirations, Eyes, Secretions, Skin. A rapid clinical assessment used in suspected CBRN or HAZMAT situations.

CVS - Cardiovascular System.

ECMO - Extracorporeal Membrane Oxygenation.

GCS - Glasgow Coma Scale.

HAZMAT - Hazardous Materials.

HDI - High Dose Insulin Euglycaemic Therapy.

HFNO - High Flow Nasal Oxygen.

RRT - Renal Replacement Therapy.

TdP - Torsade De Pointes.

TOXBASE® - Poisons Information Database https://www.toxbase.org/.

NPIS - National Poisons Information Service.

Quick Reference Guides

Figure 1: The general approach to a poisoned patient

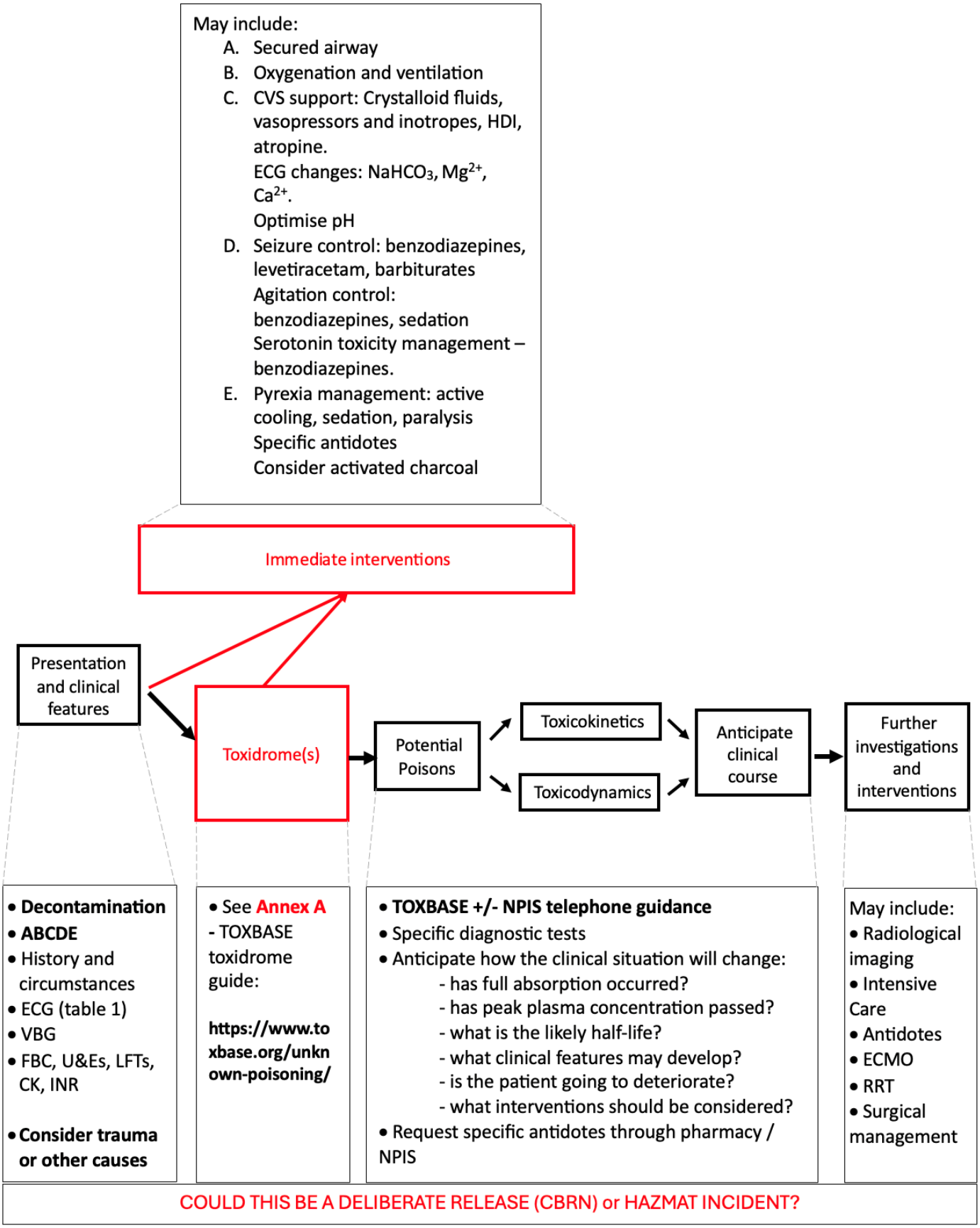


Table 1: Focused Toxicological History

- Circumstances of current presentation: time of exposure, 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.

Table 2: Examination and interventions

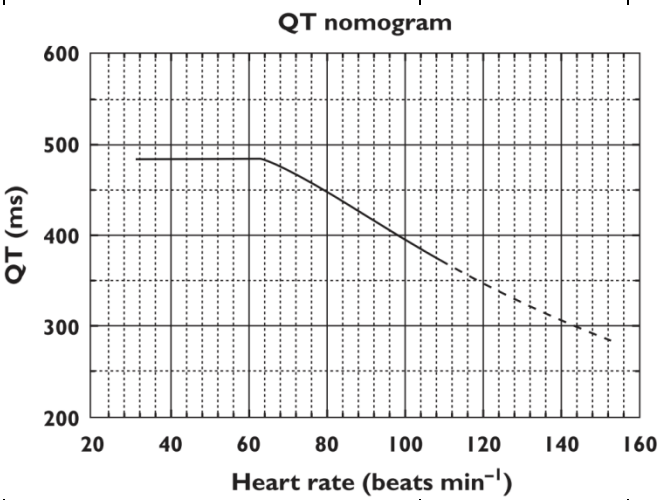
	Considerations	**Potential Investigations**	**Potential Interventions**
Airway (and CRESS assessment *table 3*)	Vomiting Aspiration Swelling Burns (chemical or thermal)	Flexible nasal endoscopy	Early consideration of intubation Left lateral position Airway adjuncts
Breathing	*Is pulse oximetry reliable?* 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 4)* 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 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	Hyper 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

Table 3: CRESS – Rapid toxidrome assessment for CBRN / HAZMAT incidents.(2)

	Opioids	**Cholinergics** (organophosphates / nerve agents)	**Anticholinergics**	**Vesicants (Blister agents)**	**Cyanides / Hydrogen sulphide**	**MetHb**	**Pulmonary agents** – chlorine, phosgene	**Botulinum toxin**	**Riot control agents**	**Sepsis**	**Heat stroke**
	NOT ALL FEATURES MAY BE PRESENT AT THE TIME OF PRESENTATION – THIS LIST IS NOT EXHAUSTIVE - Refer to AnnexA for full toxidrome list										
Consciousness	Reduced / Unconscious	Convulsions / Unconscious	Agitated / confused Reduced/ Unconscious Convulsions	Normal	Unconscious / Convulsions	Agitated/ Unconscious	Normal / Agitated	Normal	Normal	Normal / reduced / altered	Altered
Respiration	Reduced / stopped	Increased → reduced / stopped	Increased	Normal / increased	Increased or stopped	Normal / Increased / Stopped	Increased	Reduced	Normal / increased	Increased	Increased
Eyes	Pinpoint pupils*	Pinpoint pupils	Dilated pupils / Blurred vision	Normal / inflamed	Normal / Dilated	Normal	Normal / Inflamed	Dilated pupils / Blurred vision	Normal / inflamed	Normal	Normal / Dilated pupils
Secretions	Normal	Increased, vomiting	Dry mouth / Thirst	Normal or mildly increased	Normal	Normal	Increased ± Blood	Dry mouth / Thirst	Increased	Normal / sputum	Normal
Skin	Normal / cyanosed	Sweating	Flushed / Dry	Red / blistered / painful	Pink → cyanosed	Cyanosed	Cyanosed	Dry	Normal	Warm → pale / mottled Non-blanching rash	Varied
Other features	Naloxone responsive	Bradycardia, bronchorrhea, bronchospasm	Tachycardia	Rapid: caustic agent, Lewisite Delayed (6-24 hours): Sulphur mustard Airway and systemic features may be present	Sudden onset Raised lactate Arterialised venous blood	Pulse oximetry not accurate No improvement with O2	Pulmonary oedema / ARDS may develop rapidly	Rapidly descending paralysis – ptosis, diplopia, dysarthria. Rapid respiratory failure.	Bronchospasm may be present.	Pyrexia Tachycardia Hypotension Bio-syndromes♦	Pyrexia

* Pinpoint pupils (and/or increased secretions) may be delayed if skin absorption or eye protection worn.
♦ Bio-syndromes include respiratory, cutaneous, lymphadenopathy, haemorrhagic, gastrointestinal and neurological (central & peripheral)

Table 4: ECG abnormalities(3)

Initial ECG Changes	Arrythmia after deterioration	Potential xenobiotics	Initial Management
QRS prolongation (>120ms) 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 anti-arrhythmics	**Sodium bicarbonate:** ***QRS >160 msec, cardiac arrest or VT:*** 100 ml 8.4% $NaHCO_3$ IV bolus **QRS 120-160 msec:** 50 ml 8.4% $NaHCO_3$ IV 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 to the nomogram below.(4) • Automated QTc should not be used.  	Ventricular arrhythmias Torsade de Pointe (TdP)	Antipsychotics, SSRIs, Tricyclic antidepressants, Opioids, Antiemetics, Antihistamines.	**Magnesium Sulphate:** Adults: 2 grams magnesium sulphate IV over 10-15 mins Children: 25-50 mg/kg magnesium sulphate IV (max 2 grams) over 10-15 mins
P wave flattening **PR interval prolongation**	Junctional or ventricular escape rhythms	β-blockers, Calcium channel blockers, Cholinergics (organophosphates, carbamates, pilocarpine, muscarine containing mushrooms), Cardiac glycosides (digoxin, foxglove, oleander)	Agent specific 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 anti-arrhythmics	Sodium bicarbonate Agent specific management

General approach to the poisoned patient

Figure 1 illustrates a general paradigm for the approach to a potentially poisoned patient.

Poisoning is a consideration whenever diagnostic uncertainty exists in an undifferentiated patient. Poisoning and injury may both be present and **major traumatic injury should be considered, and if required, ruled out with appropriate 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 INR.

Paracetamol concentrations should be measured in all cases that may result from deliberate self-harm and be considered in all cases of unknown poisoning. Additional specific bloods tests e.g. salicylates, toxic alcohols, 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 particularly clinical signs of poisoning. **This should guide the initial administration of emergency treatments to stabilise the patient.** Concurrently an identified toxidrome will guide diagnosis towards likely poisons. These potential agents should be considered from both their potential 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: **when was the exposure and is the agent still being absorbed, has anticipated peak plasma concentration been reached and what is the likely half-life**. Continual re-evaluation must take place and clinicians should be prepared to reconsider initial diagnoses as the situation changes.

Toxidromes

(See Annex A for a list of toxidromes)

The term toxidrome refers to a collection of signs, symptoms and investigation findings that suggest a particular class of xenobiotic or individual poison. **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 **Annex A** provides detailed toxidrome tables and can be found on TOXBASE® (https://www.toxbase.org/unknown-poisoning). TOXBASE® (https://www.toxbase.org) also includes a “symptom search” option, found below the search box, which will produce a list of substances associated with that clinical feature.

Toxicological History:

The elements of a focused toxicological history are in *Table 1.*

Toxicological Examination and Immediate interventions

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

The **CRESS assessment** *[Table 3]* – consciousness, respirations, eyes, secretions, skin will rapidly identify a limited number of toxidromes and should be conducted at the same time as the A – airway assessment. It has particular usage when a CBRN incident is suspected and will identify the major classes of chemical weapons.

Airway:

Poisoning via any route of exposure but particularly ingestion, may result in aspiration of both normal gastric contents and xenobiotic substances 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.

Breathing:

Pulse oximetry should not be relied upon in cases of potential carbon monoxide poisoning, methaemoglobinaemia, sulfhaemoglobinaemia, 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 ETCO2 monitoring should be used to guide intervention.

Circulation:

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. The ECG may need repeating at regular intervals to identify evolving abnormalities.

Arrhythmia management [Table 4]: Urgent treatment with sodium bicarbonate (in patients with QRS prolongation) or magnesium (in patients with QT prolongation) and correction of electrolyte abnormalities is a priority before administration of additional antiarrhythmic drugs e.g. amiodarone. Toxic induced arrhythmias may be refractory to electrical cardioversion without appropriate antidote administration.

Hypotension may result from reduced cardiac output, vasodilation or hypovolaemia requiring supportive measures with vasopressors, inotropes and intravenous fluids. The use of high dose insulin euglycaemia therapy may be required in severe beta-adrenergic or calcium channel blockade.

A venous or arterial blood gas sample, including point of care electrolytes and the anion gap, may point towards specific poisons [Annex A, Table 3]. If toxic alcohols are suspected the anion gap should be repeated after a few hours to look for a rise and compared to the osmolar gap taken on admission and with the repeat blood gas.

Disability

Consciousness may fluctuate in poisoned patients and should be frequently reassessed. The Glasgow coma score (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] Where the diagnosis remains unclear and consciousness does not improve, patients with a 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 not be used as it causes sodium channel blockade.**

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.
- Nystagmus and optical divergence are common with agents affecting the 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, myoclonus, tremors.
- Autonomic instability: tachycardia, pyrexia, hyper 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 after starting treatment and may by 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.

Hypoxia, **hypercapnia**, **hypoglycaemia** and **acidaemia** may need correcting.

Exposure and Environmental

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

- Hyperthermia (>38.5^{o}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 should be noted. Antiemetics may cause QT prolongation and have CNS effects. Patients not able to protect their own airway will require intubation to prevent aspiration.

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.

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.

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 xenobiotic and then breakdown to release large concentrations of the forming substances. This may result in a sudden unexpected clinical deterioration.

Adulteration. Illicit drugs may be adulterated with other xenobiotics, 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 [Annex A, Table 3] for calculation is then be compared to the anion gap calculated at the same times.

Drug assays - Specific toxicological assays may be required to aid diagnosis and guide treatment e.g. 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 availability of specific tests and further advice is available from NPIS.[6]

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. Patients may have been exposed to xenobiotics in recent days or weeks which will still produce positive test results unrelated to the current clinical pathology. 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 - 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 whilst alcohol naive patients may be symptomatic at relatively low concentrations. Ethanol concentrations should be measured urgently in cases of:

- Undiagnosed coma
- Widened osmolar gap
- Suspected severe ethanol poisoning
- Suspected toxic alcohol poisoning (including methanol and ethylene glycol)
- Children with unexplained acidosis [6]

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

Computed Tomography:

- **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.[7]

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 determines 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 lifesaving**.

Detailed guidance can be found on TOXBASE®:
https://www.toxbase.org/resources/incidents/decontamination/

- **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.[8]
- **Eyes -** Irrigate eyes thoroughly with crystalloid solution for a minimum of 10-15 minutes. Test the corneal pH following irrigation.
- **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 SDAC, has been ingested within the last hour. ***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 *Table 5.* [9]

Table 5. 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

Gastric lavage should not be routinely performed for acute poisoning and may cause additional harm.(10) In rare cases of life-threatening overdose, not amenable to activated charcoal, it may be considered following discussion with NPIS.

Other specific treatments, including renal replacement therapy, whole bowel irrigation, multidose activated charcoal and urine alkalinisation 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 one hour (Category B) and which are held supra-regionally (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.[11]

Paediatric considerations

Poisoning is a consideration in children whenever a diagnosis is unclear. Children under five years of age may accidentally ingest poisons whilst exploring their environment, deliberate poisoning can be a form of non-accidental injury (NAI) 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. The TOXBASE® 'Exposure in

Pregnancy' page (https://www.toxbase.org/exposure-in-pregnancy/) links to UKTIS reviews and provides contact details.

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 Emergency Department a capacity assessment may be appropriate with the result recorded in the patients notes. Many intoxicated patients will not have 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*'.[12]

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 Emergency Department guideline.[13]

References

1. National Poisons Information Service. National Poisons Information Service Report 2022 to 2023. 2023.

2. Todd S, Bland S, Ritson J. Environmental Trauma: CBRN Incidents. In: Textbook of Acute Trauma Care [Internet]. Cham: Springer International Publishing; 2022. p. 783–99. Available from: https://link.springer.com/10.1007/978-3-030-83628-3_41

3. Clancy C. Cardiologic Principles I: Electrophysiologic and Electrocardiographic Principles. In: Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS, editors. Goldfrank's Toxicologic Emergencies, 11e [Internet]. New York, NY: McGraw-Hill Education; 2019. Available from: https://accessemergencymedicine.mhmedical.com/content.aspx?bookid=2569&sectionid=210259136

4. Isbister GK, Page CB. Drug induced QT prolongation: The measurement and assessment of the QT interval in clinical practice. Br J Clin Pharmacol. 2013 Jul;76(1):48–57. Available from: https://pubmed.ncbi.nlm.nih.gov/23167578/

5. Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS. Principles of Managing the Acutely Poisoned or Overdosed Patient. In: Nelson LS, Howland MA, Lewin NA, Smith SW, Goldfrank LR, Hoffman RS, editors. Goldfrank's Toxicologic Emergencies, 11e [Internet]. New York, NY: McGraw-Hill Education; 2019. Available from: https://accessemergencymedicine.mhmedical.com/content.aspx?bookid=2569&sectionid=210267250

6. Thompson JP, Watson ID, Thanacoody HKR, Morley S, Thomas SHL, Eddleston M, et al. Guidelines for laboratory analyses for poisoned patients in the United Kingdom. Vol. 51, Annals of Clinical Biochemistry. Royal Society of Medicine Press Ltd; 2014. p. 312–25. Available from: https://pubmed.ncbi.nlm.nih.gov/24477115/

7. Aw-Yong M, Grundlingh J, Andi A. Management of Suspected Internal Drug Trafficker Management of Suspected Internal Drug Trafficker (SIDT). 2020. Available from: https://rcem.ac.uk/wp-content/uploads/2021/10/Management_of_Suspected_Internal_Drug_Trafficker_December_2020.pdf

8. Brent J. Water-based solutions are the best decontaminating fluids for dermal corrosive exposures: A mini review. Clin Toxicol. 2013 Sep;51(8):731–6. Available from: https://pubmed.ncbi.nlm.nih.gov/24003912/

9. Chyka PA, Seger D. Position paper: Single-dose activated charcoal. Vol. 43, Journal of Toxicology - Clinical Toxicology. 2005. p. 61–87. Available from: https://pubmed.ncbi.nlm.nih.gov/15822758/

10. Benson BE, Hoppu K, Troutman WG, Bedry R, Erdman A, Höjer J, et al. Position paper update: Gastric lavage for gastrointestinal decontamination. Vol. 51, Clinical Toxicology. 2013. p. 140–6. Available from: https://pubmed.ncbi.nlm.nih.gov/23418938/

11. Royal College of Emergency Medicine, National Poisons Information Service. Royal College of Emergency Medicine and National Poisons Information Service Guideline on Antidote

Availability for Emergency Departments ([Internet]. [cited 2024 Jul 4]. Available from: https://rcem.ac.uk/wp-content/uploads/2023/08/RCEM_NPIS_Antidote_Guideline_List_2021_FINAL_V7.pdf

12. Royal College of Emergency Medicine. The Mental Capacity Act in Emergency Medicine Practice [Internet]. 2017 [cited 2024 Jul 5]. Available from: https://rcem.ac.uk/wp-content/uploads/2024/12/RCEM_Mental_Capacity_Act_in_EM_Practice-Feb_2017_V2-Copy.pdf

13. Royal College of Emergency Medicine. Mental Health in Emergency Departments A toolkit for improving care [Internet]. [cited 2024 Jul 5]. Available from: https://rcem.ac.uk/wp-content/uploads/2021/10/Mental_Health_Toolkit_June21.pdf

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RCEM Best Practice Sub-Committee
RCEM Toxicology Special Interest Group

Endoresments

National Poisons Information Service.

Review

Usually within three years or sooner if important information becomes available.

Declaration of Interests

None.

Disclaimers

The College recognises that patients, their situations, Emergency Departments and staff all vary. This guideline cannot cover all possible scenarios. The ultimate responsibility for the interpretation and application of this guideline, the use of current information and a patient's overall care and wellbeing resides with the treating clinician.

Research Recommendations

Further research is needed into the epidemiology of poisonings presenting to UK emergency departments.

Audit Standards

None.

Key Words for Search

Poisoning, toxicology, poison, toxidrome, drugs.

Annex A – Downloaded from TOXBASE® October 2024 and subject to revision. Always check online or via the App for clinical advice.

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POISONING WITH AN UNKNOWN SUBSTANCE

Poisoned patients are sometimes unaware, unable or, occasionally, unwilling to give a history of substances that they may have been exposed to. In such cases the potential poison(s) can sometimes be inferred by comparing patient features to those commonly associated with known poisons (toxidromes). The information below is intended to assist in this process. This may be helpful in differential diagnosis, in determining the need for specific toxicological interventions and in anticipating and avoiding complications. Poisoning from animal toxins (including envenoming) is presented separately for convenience. Microbial pathogens have not been presented here; they are outside the scope of this document.

- It is important to be aware that some non-toxicological conditions can produce features similar to poisons.
- The list of poisons presented here is not exhaustive, and features described here may sometimes be presented by other poisons.
- Poisons may not produce all the features listed here in any one patient, and may sometimes also produce other unlisted features.

Discuss severe cases with the National Poisons Information Service 0844 892 0111 (in Ireland NPIC (01) 809 2566).

The index table lists:

1) Symptoms/signs often seen in intoxicated patients

2) Symptoms/signs that are sometimes caused by poisons

3) Biochemical abnormalities sometimes associated with poisons

4) ECG changes sometimes associated with poisons

5) Poisoning from toxins

Tables 1 to 5 expand on these in more detail (left hand column) and provide a list of suggested intoxicants (right hand column)

INDEX TABLE

1. Symptoms/signs often seen in intoxicated patients:
    - Acute liver failure
    - Acute kidney injury
    - Anticholinergic syndrome
    - Antimitotic syndrome
    - Cholinergic syndrome
    - Corrosive syndrome
    - Fume fever
    - Hypotension with bradycardia
    - Metabolic acidosis
    - Methaemoglobinaemia
    - Opioid syndrome
    - Sedative-hypnotic syndrome
    - Serotonin-agonist syndrome
    - Sympathomimetic syndrome
    - Vesicant poisoning

2. Symptoms/signs that are sometimes caused by poisons:
    - Blindness
    - Bone marrow suppression
    - Acneiform eruptions
    - Deafness
    - Intermittent ‘flu-like’ illness
    - Hyperventilation
    - Lung fibrosis
    - Neuropathy

3. Biochemical abnormalities sometimes associated with poisons:
    - Hypernatraemia
    - Hyponatraemia
    - Hyperkalaemia
    - Hypokalaemia
    - Hyperglycaemia
    - Hypoglycaemia
    - Hypocalcaemia
    - Increased anion gap
    - Increased osmolar gap
    - Metabolic acidosis
    - Respiratory acidosis
    - Metabolic alkalosis
    - Respiratory alkalosis

4. ECG changes associated with poisons:
    - QRS prolongation
    - QT prolongation
    - Bradycardia (+/- AV block)

5. Poisoning from animal toxins
    Jelly fish stings
    - Less serious insect stings
    - Scorpion stings
    - Scombrotoxic fish poisoning
    - Shell fish poisoning
    - Snake envenoming
    - Spider bites
    - Venomous fish stings

TABLE 1: SYMPTOMS/SIGNS SOMETIMES SEEN IN INTOXICATED PATIENTS

Clinical features	Example agent(s)
Acute liver failure	Paracetamol Ethanol Iron Carbon tetrachloride Kava Kava Amanita and other hepatotoxic mushrooms
Acute kidney injury	Ethylene glycol Methanol ACE-I Cortinarius mushrooms Any toxicological cause of severe hypotension NSAIDs Paracetamol (rarely in the absence of liver failure)
Anticholinergic syndrome (muscarinic antagonist) Confusion Agitation Dry skin Hyperthermia Thirst Dry mouth Mydriasis Tachycardia Urinary retention Paralytic ileus and decreased bowel sounds	Antimuscarinic drugs (e.g. hyoscine) Tricyclic antidepressants (TCA) Antipsychotics (e.g. chlorpromazine, pericyazine) Antihistamines (sedating e.g. diphenhydramine) Atropa *belladona* Inocybe mushrooms Datura *stramonium* (Jimson weed)
Antimitotic syndrome (cytotoxic to dividing cells) Bone marrow suppression (aplastic anaemia, leucopaenia, thrombocytopaenia) Alopecia Vomiting, diarrhoea, mucositis	Antineoplastic drugs Colchicine Immunosuppressants Podophylline Radiation exposure Arsenic Thallium
Cholinergic syndrome (nicotinic and muscarinic agonist) Increased sweating and lacrimation Wheezing and breathing difficulty Pupillary constriction and visual disturbance Vomiting Involuntary defecation or urination Bradycardia (nicotinic agonists may initially cause tachycardia) Muscle paralysis and respiratory failure	Organophosphorus insecticides Carbamate insecticides Nicotine Laburnum species Hemlock species Inocybe mushrooms

Corrosive poisoning GI tract pain Vomiting Hematemesis Dyspnoea Drooling Stridor/pneumonitis (if aspirated) Pain, ulceration or necrosis from skin contact Inflammation of all layers from eye contact	Hydrofluoric acid Strong acids (sulphuric, nitric, hydrochloric) Strong alkalis (e.g. sodium hydroxide) Paraquat Copper salts
Fume fever History of unpleasant smells Chills Cough Dyspnoea Headache Myalgia Malaise	Metal oxides (especially zinc oxide) Polymer fumes (e.g. fumes released during heat-decomposition of fluorine containing polymers such as Teflon) Other toxic industrial chemicals
Gastrointestinal irritation (severe) Severe vomiting Abdominal pain Diarrhoea Haematemesis Melaena Hypovolaemia may lead to shock and/or acute kidney injury.	Ingestion of corrosives NSAIDs Salts of iron, arsenic, thallium and other metals Cardiac glycosides (mainly upper GI symptoms associated with ECG abnormalities) Aconitine (associated with ECG changes and neuropathy) Ricin (mainly lower GI symptoms, even with injection, may be associated with seizures and eye or lung signs) Aluminium phosphide (may be associated with local swelling, metabolic abnormalities and eye or lung signs)
Hypotension with bradycardia	Beta blockers Calcium channel blockers Digoxin and other cardiac glycosides
Metabolic acidosis (More details under Biochemical abnormalities sometimes associated with toxins) Deep and rapid (Kussmaul's) breathing Obtunded consciousness Tachycardia Hypotension	Ethylene glycol Methanol Aspirin Paracetamol (uncommon unless AKI) Iron Cyanide Carbon monoxide Acids Sodium fluoroacetate

Methaemoglobinaemia Blue-grey 'apparent' central cyanosis (blue to grey lips, tongue and mucus membranes, and slate grey skin) Persistent cyanosis despite oxygenation Fatigue, dizziness, headaches Depressed consciousness Seizures Urine may be discoloured black or brown	Benzene derivatives (Phenols, Cresols, Aniline) Sodium nitrite Organic nitrites Chlorates Copper salts Prilocaine Benzocaine
Opioid syndrome Depressed consciousness Decreased respiratory rate (although this may not occur in patients with airway obstruction) Decreased tidal volume Miosis Naloxone response Hypotension Pulmonary oedema	Opioids (morphine, heroin, methadone, codeine, oxycodone, etc) Olanzapine can also cause coma and miosis
Sedative – hypnotic poisoning Depressed consciousness Ataxia Dysarthria Nystagmus	Ethanol Benzodiazepines and related drugs Gamma hydroxybutyrate (GHB) Gamma butyrolactone (GBL) Barbiturates
Serotonin agonist syndrome Neuromuscular features (hyperreflexia and clonus, tremor, shivering, hypertonia) Altered sensorium (restlessness, agitation, confusion) Autonomic instability (fever/hyperthermia, unstable BP or pulse, bladder/bowel problems) Flushing Seizures	Serotonin-Specific Re-uptake Inhibitors (SSRI) Monoamine Oxidase Inhibitors (MAOI) Tricyclic antidepressants Venlafaxine Methylenedioxymethamphetamine (MDMA) Amphetamines Cocaine Tramadol Triptans Linezolid St John's Wort 'Legal highs' Psilocybe mushrooms Any combination of the above, even in therapeutic doses
Sympathomimetic syndrome Hyper/hypotension Tachycardia Neurological excitation Tremor Hyperreflexia Seizures	Cocaine Amphetamines 'Legal highs'

Vesicant poisoning Conjunctivitis Keratitis Dermatitis Severe blistering	Nitrogen and sulphur mustards Methyl bromide Hexylresorcinol Croton oil

TABLE 2: SYMPTOMS/SIGNS THAT ARE SOMETIMES CAUSED BY POISONS

Clinical features	**Example agent(s)**
Blindness	Methanol Quinine
Bone marrow suppression	Chemotherapy agents Colchicine Radiation poisoning
Acneiform eruptions	Dioxins Steroids (chronic use)
Deafness	Salicylates Quinine Loop diuretics
'Flu-like' symptoms only occurring in a certain place or in several people present in the same place	Noxious gas or vapour exposure (eg Carbon Monoxide)
Hyperventilation	Salicylates Metabolic acidosis
Lung fibrosis	Paraquat poisoning Adverse reaction to antineoplastic drugs (cyclophosphamide, busulfan, bleomycin, chlorambucil) or amiodarone
Neuropathy	Lead Chronic arsenic poisoning Thallium Ethylene glycol Organophosphates (intermediate syndrome or delayed effects)

TABLE 3: BIOCHEMICAL ABNORMALITIES SOMETIMES ASSOCIATED WITH POISONS[1]

Clinical features	Example agent(s)
Hypernatraemia	Ecstasy (rarely)
Hyponatraemia	Ecstasy (commonly) SSRIs Levamisole (adulterant in cocaine) Diuretics (chronic)
Hyperkalaemia	Digoxin Potassium sparing diuretics ACE inhibitors
Hypokalaemia	Theophylline Salbutamol Digoxin Diuretics (chronic) Insulin Sulphonylureas Paracetamol
Hyperglycaemia	Theophylline Salicylates Calcium channel antagonists Beta blockers
Hypoglycaemia	Insulin Sulphonylureas Ethanol Salicylates Sodium valproate
Hypocalcaemia	Ethylene glycol Hydrofluoric acid Sodium monofluoroacetate
Increased anion gap = (Na + K) - (HCO_3 + Cl) Normal (12 – 16 mmol/L)	Ethanol Ethylene glycol Iron salts Isoniazid Methanol Metformin Paraldehyde Salicylates Toluene
Increased osmolar gap = measured – calculated osmolarity Calculated osmolarity = (glucose + urea + 1.86xNa) ÷ 0.93 Normal (less than10 mmol/L)}	Ethanol Ethylene glycol Acetone Isopropanol Propylene glycol

	Hyperosmolar IV solutions (e.g. mannitol)
Metabolic acidosis {pH less than 7.35, pCO_2 less than 4.5 kPa (34 mmHg) base deficit present}	Carbon monoxide Cyanide Ecstasy Ethylene glycol Gamma hydroxybutyrate Iron Isoniazid Metformin Methanol Paracetamol Paraldehyde Salicylates Sodium valproate Theophylline Tricyclic antidepressants
Respiratory acidosis {pH less than 7.35, pCO_2 greater than 6.0 kPa (45 mmHg), base deficit absent}	Sedative agents e.g. Barbiturates Benzodiazepines Gamma hydroxybutyrate Ethanol Opiates Tricyclic antidepressants
Metabolic alkalosis {pH greater than 7.45, pCO_2 normal base excess present}	Bicarbonate
Respiratory alkalosis {pH greater than 7.45, pCO_2 less than 4.5 kPa (34 mmHg) base excess absent}	Salicylates Theophylline Ecstasy

TABLE 4: ECG CHANGES ASSOCIATED WITH POISONS

Clinical features	**Example agent(s)**
QRS prolongation	Tricyclic antidepressants Local anaesthetics Quinine
QT prolongation	Antipsychotics Serotonin-Specific Re-uptake Inhibitors (SSRI) Tricyclic antidepressants

Bradycardia (often associated with delayed atrio-ventricular conduction)	Beta-blockers Rate-limiting calcium channel blockers Cardiac glycosides (e.g. digoxin)

TABLE 5: ENVENOMING SYNDROMES

Clinical features	Example agent(s)
Jellyfish stings Immediate pain and local urticaria. Immediate or delayed systemic features may occur including headache, dizziness, muscle cramps and sweating. In severe cases severe chest and abdominal pain, abdominal rigidity, dysphagia and anaphylaxis.	Most jellyfish found around the UK are harmless. Those which may sting include: • *Chrysaora hyoscella* (compass jellyfish) • *Cyanea capillata* (lion's mane) • *Cyanea lamarckii* (sea nettle) • *Physalia physalis* (Portuguese man-o-war).
Less serious insect stings These may cause local pain and swelling but very rarely cause severe toxicity. Severe anaphylactic reactions have occurred in individuals sensitive to the insect venoms, occasionally resulting in fatalities. Deaths have occurred as a result of upper airway blockage due to oedema caused by stings in the mouth or on the neck or head regions.	Bees, wasps, etc.
Scorpion stings Usually cause only severe pain at sting site. Occasional features include local paraesthesia or paralysis or skin changes including necrosis or allergy. Rare effects include autonomic dysfunction including autonomic storm, severe gastrointestinal, haematological, and systemic neurological	Various scorpions
Scombrotoxic fish poisoning Closely resembles features of histamine reaction, including flushing, dizziness, headache, palpitations, nausea, vomiting, abdominal pain, diarrhoea. Bronchospasm and urticaria are less common.	Spoiled **dark meated marine fish** e.g. mackerel, tuna, bonito and skipjack.

Shell fish poisoning **Paralytic** poisoning usually starts within 3 hours with a feeling of floating, dizziness, incoordination, weakness, numbness and paraesthesia around the mouth and in the extremities. Respiratory failure (due to muscle weakness) may develop. **Diarrhetic** poisoning is characterised by gastro-intestinal disorders including nausea, vomiting, diarrhoea, abdominal pain, headache and fever. Symptoms can develop in between 30 minutes and 3 hours after consumption	Naturally occurring algal blooms on which the shellfish feed can sometimes contain toxins that accumulate in the shellfish. The toxins responsible for **paralytic** poisoning are derivatives of saxitoxin, while it is thought that dinophysis toxins, pectenotoxins and yessotoxins, cause **diarrhetic** poisoning. **Neurotoxic** poisoning is caused by brevetoxins and **amnesic** poisoning is caused by domoic acid, a contaminant in shellfish.
Neurotoxic poisoning is very rare in the UK, and causes rapid onset of tingling, numbness of legs, tongue and throat, muscular aches, dizziness, diarrhoea and vomiting. Paralysis does not occur **Amnesic** poisoning starts within 24 hours of consumption with gastro-intestinal disorders including vomiting, diarrhoea, and abdominal pain; and later onset of neurological effects including confusion, memory loss, disorientation, seizure and coma.	
Snake envenoming Bites usually occur in summer and envenoming usually results in local features (swelling, bleeding, bruising, lymphangitis, blistering, necrosis, secondary infection, and painful regional lymph node enlargement), but can very rarely cause anaphylaxis, bleeding diatheses, acute kidney injury (especially in children), dysrhythmias and shock or GI effects.	***Vipera berus*** (adder or viper) is the only native British species to cause envenoming. For more details see [link to adder entry] Exotic snakes may cause features similar to adder envenoming, or cause other syndromes including descending paralysis, eye effects from venom spat into eyes. For more details see [link to non-British snake entry]
Spider bites A bite feels like a painful bee-sting. Redness, local swelling and irritation at the site of the bite may occur. Enzymes injected by the spider cause necrosis of the skin. Systemic features are very unlikely, except for allergic reactions in susceptible individuals.	Various spiders

Venomous fish stings In the UK, the most common effects are local severe pain with a burning sensation and swelling. Rare features include vomiting and headache with tachycardia and respiratory distress.	**Weever fish** envenoming occurs in shallow UK waters, such as that favoured by bathers and paddlers, with most injuries occurring to the feet. **Lion fish and stone fish** may be kept by tropical fish enthusiasts and cause stings to hands.

[1] Adapted from: National Poisons Information Service; Association of Clinical Biochemists. Laboratory analyses for poisoned patients: joint position paper. Ann Clin Biochem. 2002;39(Pt 4):337.

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