Approach to the acutely poisoned patient

Poisoning, accidental or deliberate, is a common presentation in emergency departments.

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Many patients present to emergency care professionals with suspected or actual poisoning. Exposure to poisons can be accidental or deliberate. Accidental exposure may result from environmental hazards, accidental ingestion, inappropriate use of pharmacological agents, etc. while deliberate self-harm and assault account for a distressing number of cases. Any substance may in theory be toxic – whether via its pharmacological actions, inappropriate dose, physiological or even mechanical impact – and hence the maxim of 'treat the patient, not the poison' applies. Many patients can be successfully resuscitated and treated with supportive management alone, while precious few poisons actually have or even need antidotes.

This article will advise that managing the airway, breathing and circulation (ABCs) remains the most important aspect of management, and that the ABCs come before the 'D' for 'drugs, disability or differential diagnosis'. Aspects of basic resuscitation that may need to be altered or at least considered in the presence of a specific toxin will be discussed, and the concept of 'toxidromes' will also be examined. The immense amount of information that is required for an exhaustive look at the identification and management of the many varied individual toxins that may be encountered in South Africa limits what can be included in a single article, and readers are encouraged to have access to a number of useful poison references, such as:

- a poison information centre – e.g. telephone 0800 333 444
- local and national emergency medical services (EMS) – state and private
- local accident and emergency (A&E) unit
- medical text references[1]
- the Internet – it's amazing what the intelligent use of Google can reveal.

Safety

Safety of the health care professional is paramount, and this proviso is deliberately addressed prior to any resuscitative intervention. Take universal safety precautions, and pay special attention to the following situations:

- Possible hazardous materials (hazmat) – require specialised protective garments, specialised facilities, training, etc. Local EMS and disaster management services can assist. This includes industrial or occupational toxins such as pesticides or cyanide.
- Possible agents used in terrorist or biological warfare situations. Local EMS and disaster management services can assist.
- Patients where illicit drugs have been involved – be aware of volatile behaviour, weapons, needles, criminal elements, etc.
- On a softer note, pay careful attention to the history of the event as provided by various parties; and also ensure that no future medico-legal investigation is compromised by thoughtless interventions.

Airway

A common factor contributing to morbidity or mortality in cases of poisoning or drug overdose is the loss of protective airway reflexes and subsequent airway obstruction, aspiration or respiratory compromise and even arrest.

In awake patients, ensure that their level of consciousness is monitored, and that they remain sufficiently awake to manage their airway. For all other patients definitive airway protection is recommended (e.g. tracheal intubation).

Remember

- Appropriate antidotes, e.g. naloxone for opioids or flumazenil for benzodiazepines can obviate the need for a definitive airway, but the half-life of many sedatives exceeds those of the antidotes.
- Anticipate vomiting, especially with ingested poisons.
- Have effective suction handy.
- Position the patient for optimal airway control.
- Consider interactions between induction agents/sedatives and the toxin(s).

Breathing

Patients may present with either or a combination of ventilatory failure, hypoxia or bronchospasm.

Ventilatory failure can have many causes, such as failure of respiratory muscles, central depression of ventilatory drive, pulmonary infection and/or pulmonary oedema.

In the context of poisoning, respiratory muscles can be paralysed by botulin toxin, neuromuscular blockers, organophosphates, neurotoxic snake venom, strychnine, tetanus, etc.

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Central respiratory drive can be depressed by barbiturates, opioids, sympatholytic agents, ethanol/alcohol, sedative-hypnotics and even tricyclic antidepressants.

Remember

- Use arterial blood gas measurements frequently.
- Don't wait for apnoea or pCO_2 > 60 mmHg before assisting ventilation.

Hypoxia could be the result of various situations such as:

- insufficient oxygen in the ambient air (think of carbon dioxide, methane, nitrogen)
- disrupted oxygen absorption from cardiogenic pulmonary oedema (think of beta-blockers, quinidine, tricyclics, verapamil)
- non-cardiogenic pulmonary oedema or pneumonia (think of aspiration, chlorine or irritant gases, cocaine, ethylene glycol, mercury, metal fumes, paraquat, phosgene, salicylates, sedative-hypnotics, smoke inhalation)
- cellular hypoxia (think of cyanide, carbon monoxide).

Remember

- Blood gases may be normal in cellular hypoxia.
- Watch out for pulmonary contusion due to trauma.

Bronchospasm can result from a direct irritant, the pharmacological effects of the toxin or drug, or a hypersensitivity or allergic reaction (think of irritant gases, beta-blockers, hydrocarbon aspiration, isocyanates, smoke inhalation, food allergy, anticholinesterases).

Circulation[2]

- Certain advanced cardiovascular life support (ACLS) drugs are not appropriate in certain poisoning circumstances, e.g. atropine is ineffective in beta-blocker overdose.
- Arrhythmias may be caused by or complicate many drug overdoses or toxic exposures. Always treat the patient, and focus on the actual or potential cardiovascular instability rather than the potential cardiac effects of the offending agent.
- Watch out for bradycardia or AV block due to calcium antagonists, drugs that depress sympathetic tone or drugs that increase parasympathetic tone.
- Hypothermia, acute myocardial infarction (AMI), electrolyte abnormalities and metabolic disturbances may occur.
- Young fit patients have huge physiological reserves.
- With tachycardias, consider causes such as occult blood loss, fluid loss, hypoxia, fever, AMI and anxiety.
- Be aware of ventricular irritability with illicit stimulants (e.g. cocaine and amphetamines) and solvents.
- Hypotension with a relative bradycardia could be due to sympatholytics (think of beta-blockers, opiates), membrane depressants (think of quinidine, tricyclics) and others (think of fluoride, organophosphates, sedative-hypnotics).
- Hypotension with a relative tachycardia could be due to fluid loss or third spacing (think of magic mushrooms, arsenic, colchicines, hyperthermia) or peripheral vasodilation (think of β_2-stimulants, caffeine, nitrites, phenothiazines).
- Hypertension is frequently overlooked in intoxicated patients; so beware of relative hypertension in young fit adults. Think of sympathomimetics and environmental hyperthermia.

Disability

Coma or stupor

A decreased level of consciousness is probably the most common significant complication of drug overdose or poisoning.[3] It is often due to global depression of the brain's reticular activating system, caused by agents such as anticholinergics or CNS depressants. Coma may be a post-ictal phenomenon after a toxin-induced seizure. Also, consider coma due to a cerebrovascular event. A frequent complication of coma is respiratory depression, but watch out too for delayed onset of hypotension, hypothermia and rhabdomyolysis. ABC is logically followed by DEFG – 'don't ever forget glucose!'

Hypothermia

This condition may complicate or mimic drug overdose or poisoning, and should be suspected in every unconscious patient. The toxins may blunt the person's ability to respond appropriately to the environment, cause excessive vasodilation, inhibit shivering or decrease metabolic activity.

Hyperthermia

This can be a potentially lethal complication of intoxication by a variety of drugs or poisons. It may be caused by excessive heat generation because of sustained seizures, rigidity or other muscular hyperactivity, an increased metabolic rate, impaired heat dissipation due to suboptimal sweating (e.g. anticholinergics), or even hypothalamic disorders.

Specific hyperthermic conditions to watch out for are:

- neuroleptic malignant syndrome – due to certain antipsychotics; clues include severe rigidity and metabolic acidosis
- malignant hyperthermia – inherited disorder, typically precipitated by certain anaesthetic agents (e.g. halothane and succinylcholine)
- serotonin syndrome – clues include autonomic instability, diaphoresis and myoclonus, and may be associated with mono-amine oxidase inhibitors, serotonin re-uptake inhibitors, and even amphetamines.

Seizures

Remember

- possible airway compromise
- metabolic acidosis and hyperthermia from prolonged or multiple seizures
- use benzodiazepines before using anti-epileptic agents.

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Table I. Quick reference for poisoning clues

Always check for:

T – temperature disturbances
O – odours, e.g. pears (chloral hydrate), almonds (cyanide), carrots (water hemlock), garlic (arsenic or organophosphates)
X – extrapyramidal (tremors or dystonia) or pyramidal (hypertonia or hyperreflexia) disturbances
I – ileus
D – dry mouth or excessive salivation
R – rashes or erythema
O – overdose (beware of polypharmacy)
M – muscle tone
E – eyes (check pupils and check for nystagmus)
S – skin (flushing or sweating)

Agitation, delirium or psychosis

Functional psychosis or stimulant-induced agitation and psychosis are usually associated with an intact sensorium, and a clue may be predominantly auditory hallucinations. Metabolic encephalopathy or drug-induced delirium usually present with an altered sensorium (such as disorientation or confusion), and a clue may be predominantly visual hallucinations.

Other complications

- Dystonic reactions – torticollis, trismus, etc. can be due to antipsychotics and anti-emetics, and are usually treated with anticholinergics.
- Dyskinesias – rapid repetitive movements due to increased dopamine effects or central cholinergic blockade. Think of amphetamines, caffeine, cocaine, gamma hydroxybutyrate, ketamine and even lithium. This complication is usually treated with benzodiazepine sedation.
- Rigidity – may be due to hyperthermic conditions (see above) and also think of black widow spider, methaqualone (Mandrax) and phencyclidine (PCP). Specific treatments are required, such as antivenom and calcium for spider bite, and dantrolene for malignant hyperthermia.
- Rhabdomyolysis might be caused by prolonged immobilisation, excessive seizures or muscular hyperactivity (think of illicit stimulants and even tetanus), hyperthermia, or even direct cytotoxins such as carbon monoxide, some snake venoms and ethylene glycol. Treatment includes aggressive fluid therapy, urinary alkalinisation, and general (renal) intensive care.
- Anaphylactic (IgE-mediated) and anaphylactoid (IgG-mediated) reactions are characterised by bronchospasm, increased vascular permeability, laryngeal oedema, skin rashes and hypotension. Toxin-induced anaphylaxis may occur with antivenins, foods, insect stings, antibiotics and vaccines. Blood products, iodine contrast, opioids and even tubocurarine are commonly associated with anaphylactoid reactions. Treatment includes intramuscular adrenaline, intravenous fluid replacement, antihistamines and steroids.

The check box (Table I) is a quick reference for poisoning clues to be identified in the initial clinical examination. Check for 'T.O.X.I.D.R.O.M.E.S'.

Toxic syndromes

Toxidromes are typically a specific combination of clinical signs and symptoms characteristic (or commonly associated with, but not necessarily pathognomonic) of poisoning due to a certain type of toxin. A summary of the various syndromes is presented in Table II.

Once the patient has been safely resuscitated and the possible toxic syndrome identified (i.e. 'S – ABC-D' completed), it remains briefly to consider options under 'E' – 'evacuating the poison and dealing with the exposure'.

Evacuation

Washing or rinsing remains the gold standard for external decontamination. Use water, or soap and water. Flush eyes (especially if venophthalmia) for 20 minutes – you can use a nasal oxygen cannula as a water delivery device. The emphasis is on washing away, as opposed to neutralising (which may in theory cause an exothermic reaction). Oral mucosal irritants (e.g. many poisonous plants) may be relieved by cool calcium sources such as ice cream.

Emesis

- Always assess whether the aspiration risk outweighs the absorption risk.
- You can use digital stimulation or ipecacuanha syrup in fully alert patients who have not ingested caustics or hydrocarbons, or if no medical co-morbid conditions are present.
- Don't give too much fluid as a carrier, as this can further facilitate absorption of the toxin.

Gastric lavage

- Protect the airway first if necessary.
- Assess value in terms of anticipated gastric emptying time (of greatest benefit if performed within 1 hour of toxin ingestion).
- Never perform gastric lavage with caustics, corrosives or hydrocarbons.
- Useful for obtunded patients, following ingestion of tablets, plant seeds, salicylates, etc.

Activated charcoal

- Very effective for many ingested toxins, especially if given within 1 hour of ingestion.
- Avoid aspiration. Protect airway if necessary.
- Usual dose is 1 g/kg body weight diluted 1:4, or 5 - 10 times the weight of the poison.
- Poor adsorption of metals, pesticides, cyanide, ethanol, strong acids and alkalis, and hydrocarbons.

Laxatives

- Exercise caution when poisoning is due to corrosives, or in the presence of severe diarrhoea, electrolyte imbalance and recent bowel surgery.
- Whole-bowel irrigation is an option, e.g. with ingestion of enteric-coated tablets.

Forced diuresis and pH manipulation

- Be aware of the hazards of fluid and electrolyte management, and the risks of pulmonary and cerebral oedema. Consider only if other interventions fail, the poisoning is potentially fatal, and no renal or hepatic failure is present.
- Maximum excretion of drugs with an acid pKa (e.g. salicylates) occurs in the alkaline range. The reverse applies to those with an alkaline pKa (e.g.

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Table II. Summary of the various toxic syndromes

Syndrome	Examples	Clinical clues
Alpha adrenergic	Phenylpropanolamine, phenylephrine	Hypertension with reflex bradycardia; mydriasis
Beta adrenergic	Salbutamol, theophylline, caffeine	Hypotension, tachycardia
Mixed adrenergic (sympathomimetic)	Amphetamines, cocaine	Hypertension with tachycardia; mydriasis; sweaty skin
Sympatholytic	Methyldopa, opioids, phenothiazines	Hypotension and bradycardia; miosis; decreased peristalsis
Nicotinic cholinergic	Nicotine, succinylcholine	Unpredictable; fasciculations, paralysis
'Muscarinic cholinergic'	None	Bradycardia, miosis, sweating, hyperperistalsis, bronchorrhoea, wheezing, excessive salivation, etc.
Mixed cholinergic (acetylcholinesterase inhibition)	Organophosphates, physostigmine	Mixture of above two
Anticholinergic	Atropine, antihistamines, tricyclics	Tachycardia with mild hypertension; hot flushed dry skin; mydriasis; urinary retention; ataxia, etc.
Extrapyramidal	Haloperidol, chlorpromazine	Rigidity, opisthotonus, laryngospasm
Narcotic	Morphine, heroin, codeine	CNS depression, miosis, slow respiration, hypotension
Withdrawal	Various; alcohol, barbiturates, cocaine, benzodiazepines, etc.	Piloerection, hypertension, tachycardia, insomnia, myalgia, hallucinations, etc.
Haemoglobinopathies	Carbon monoxide, nitrites	Headache, nausea, vomiting, dyspnoea, seizures, etc.

amphetamines). The complete reverse is true for absorption.

- This technique of attempting to evacuate an absorbed toxin has dubious success, as many toxins are widely distributed and strongly protein bound.

Haemodialysis

- Only really effective if one can improve the excretion of the poison by more than 30%; typically low-molecular-weight, low-protein-binding,water-soluble toxins in a non-responsive patient with severe clinical intoxication.[3]
- Consider haemodialysis when a toxin has dangerous metabolites, such as methanol and ethylene glycol.
- Beware of hypotension, electrolyte imbalance, etc.

Haemoperfusion

- May be of value in clinical toxicity due to methaqualone, barbiturates, paraquat, methotrexate and even phenytoin.
- Main complications of this technique are thrombocytopenia and anticoagulant-related bleeding.

Conclusion

The emphasis of this review has been to provide sufficient information on the relevant toxin, and to provide general supportive intensive care for the severely poisoned patient. Selective use of relevant antidotes is appropriate. Correct referral of the patient is equally critical. If there has been any element of deliberate self-harm or illicit drug use, a difficult psychosocial case exists that should be referred to the appropriate health care professionals.

References

1. Howard A. *Emergency Management of Acute Poisoning*, Van Schaik, 2006 (recommended reference text).
2. Olson K, ed. *Poisoning and Drug Overdose*, 3rd ed. Lange, 1999.
3. Kloeck W, ed. *A Guide to the Management of Common Medical Emergencies in Adults*, 7th ed. Johannesburg: Dept of Medicine, University of the Witwatersrand, 2005: 63-79.

In a nutshell

- Safety first.
- The ABCs of resuscitation are paramount.
- The pursuit of the identity of the toxin should not take precedence over the resuscitation.
- Always have access to various poison reference tools.
- Identify toxidromes.
- Facilitate rapid and safe evacuation of potentially harmful toxins.

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