TOXICOLOGY HANDBOOK
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Poisoning is a common emergency department presentation, and the third major injury cause of hospital admissions after falls and motor vehicle crashes. Alcohol, benzodiazepines, antidepressants, paracetamol and heroin are frequently involved, yet there are literally thousands of hazardous substances that can be ingested, as well as envenomings by terrestrial animals and sea creatures.

The challenge for the emergency physician is to be able to recognise the poisoned patient, provide supportive care, administer a specific antidote in a minority of cases, escalate management up to a full intensive care level when necessary, and know when a patient is safe to be ‘medically cleared’ pending a thorough psychiatric examination (in cases of deliberate self-harm). This presents a huge challenge to any doctor, who individually may infrequently see a severe poisoning and or can be confronted with a first case of a particular type.

Clinical Toxicology has developed rapidly as a subspecialty of Emergency Medicine in Australasia, led by a small group of expert clinicians dedicated to providing information, advice, research and teaching in this important area. The authors are in the vanguard of this group. All regularly direct and assist toxicology patient care in emergency departments, intensive care units and small rural hospitals across the country, locally as well through the national Poisons Information Centres.

Their risk assessment-based approach is maintained in this new version that builds on the success of the first edition. This handbook has been updated and expanded with the addition of many new chapters, yet it retains its award-winning format recognised for its lucidity and readability. The compact size of the book belies the true wealth of clear, practical evidence-based information covering a vast array of poisonings and their management in a logical, consistent format.

This book should live in the pocket or at the bedside, be used daily and be referred to as a prevailing standard of care not just in Australasia, but internationally. With the exception of some envenomings, the book will be just as valuable to clinicians in the UK, Europe and Asia as no doubt it will again prove to be here in Australasia. It is a truly outstanding text that will improve the care of poisoned patients to their benefit, and the readers’ edification.

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August 2010
The overwhelmingly enthusiastic response to the first edition of the *Toxicology Handbook* confirmed the need amongst emergency medical personnel for readily accessible and practical toxicology information in the context of a systematic approach to the care of the poisoned patient.

Feedback from the users of the handbook from Poisons Information Centres and Emergency Departments in urban, regional and rural settings has allowed us to expand and refine the factual information for the second edition while retaining the standardised formats and risk assessment based approach of the first edition. Routine use of the handbook by junior medical staff in our own Emergency Departments and Toxicology Units in Perth has allowed us to refine any written advice that is potentially liable to misinterpretation by inexperienced users. For the second edition we have added chapters to provide an approach to poisoning by plants and mushrooms and an approach to dealing with the issues of drug dependence, tolerance and withdrawal that frequently complicate management of the poisoned patient. We have also added new chapters for a number of important specific toxins and antidotes, and extensively revised the envenoming chapters in the light of recently published research.

Our sincere hope is that the *Toxicology Handbook* continues to contribute to excellence in the provision of care of the poisoned patient.

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# CHAPTER 1
APPROACH TO THE POISONED PATIENT

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1.1 OVERVIEW

Acute poisoning is a common emergency medicine presentation. Between 150 and 400 acute poisoning presentations annually can be expected for each 100,000 population served by an emergency department. Acute poisoning is a dynamic medical illness that frequently represents a potentially life-threatening exacerbation of a chronic psychosocial disorder. However, this is a highly heterogeneous patient population: deliberate self-poisoning, recreational drug abuse, occupational poisoning and envenoming challenge with myriad potential presentations. The clinician needs a robust and simple clinical approach that can address this heterogeneity, but which allows the development of a management plan tailored to the individual patient at that particular presentation at that particular medical facility.

Risk assessment is pivotal to that robust approach. It is a distinct cognitive process through which the clinician attempts to predict the likely clinical course and potential complications for the individual at that particular presentation. Risk assessment should wherever possible be quantitative and take into account the agent, dose and time of ingestion, clinical features and progress, and individual patient factors (e.g. weight and co-morbidities).

Toxicology management guidelines frequently focus on the agent involved. This makes adaptation of treatment recommendations to an individual patient in a particular location difficult. A risk-assessment-based approach ensures the clinician addresses potentially time-critical management priorities in an appropriate order, but avoids unnecessary investigations or interventions.

Risk assessment is secondary only to resuscitation in the management of acute poisoning. It allows subsequent management decisions regarding supportive care and monitoring, investigations, decontamination, use of enhanced elimination techniques, antidotes and disposition to be made in a sensible structured manner.

Ideally, this risk-assessment-based approach is supported by a healthcare system designed to address both the medical and psychological needs of the poisoned patient. Where the medical needs of a patient exceed local resources, a risk-assessment-based management approach ensures that this is identified early and disposition planning and communication occur in a proactive manner within that organised system.

In this handbook, the authors offer a systematic risk-assessment-based approach to the management of acute poisoning as it presents to the emergency department. Separate chapters cover the pharmaceutical, chemical and natural toxins of most importance to the practitioner in emergency departments in Australia and New Zealand. It will also be of
use to ambulance and emergency paramedic personnel and staff of general intensive care units. The approach to acute poisoning presented in this book is honed at the bedside and on the telephone. The authors collectively have directly cared for over 30 000 patients in the Western Australian Toxicology Service and offered consultation in over 12 000 acute poisonings across Australia and overseas via the Western Australian, New South Wales and Queensland Poisons Information Centres (PICs). The agents covered are carefully selected to cover all common poisonings, rare but life-threatening poisonings, poisonings where particular interventions make a difference to outcome, or which result in frequent consultations with clinical toxicologists through the PIC network. Chapters are also offered on the important antidotes and antivenoms with practical information on administration, dose and adverse effects. All chapters have a risk assessment. All chapters have special sections on ‘pitfalls’ and ‘handy tips’. These are not for show! They are designed to respond to the real questions and mistakes that regularly occur in clinical practice across Australasia.

Clinical toxicology has rightly become an area of expertise of the emergency physician but the infinite variation in presentation constantly confounds and surprises all of us. We hope that the information in this book, when combined with a structured approach, will improve the care delivered to the poisoned patient.

### TABLE 1.1.1 Risk assessment-based approach to poisoning

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<td>Circulation</td>
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<tr>
<td>Detect and correct</td>
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<tr>
<td>— hypoglycaemia</td>
<td></td>
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<tr>
<td>— seizures</td>
<td></td>
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<tr>
<td>— hyper-/hypothermia</td>
<td></td>
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<tr>
<td>Emergency antidote administration</td>
<td></td>
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</table>

#### Risk assessment

- Agent
- Dose
- Time since ingestion
- Clinical features and course
- Patient factors

**Supportive care and monitoring**

**Investigations**

- Screening—12-lead ECG, paracetamol
- Specific

**Decontamination**

**Enhanced elimination**

**Antidotes**

**Disposition**
Poisoning is most frequently the presentation of an individual suffering from exacerbation of very significant underlying psychiatric, social or drug and alcohol problems. Excellence in care of the poisoning delivered in a compassionate manner offers an opportunity to intervene and produce a happy outcome in this vulnerable group of patients.

1.2 RESUSCITATION

INTRODUCTION
Poisoning is a leading cause of death in patients under the age of 40 years and is a leading differential diagnosis when cardiac arrest occurs in a young adult.

Unlike cardiac arrest in the older population, resuscitation following acute poisoning may be associated with good neurological outcomes even after prolonged periods (hours) of cardiopulmonary resuscitation (CPR). Therefore, while poisoning is considered part of the differential diagnosis in a patient with cardiac arrest, resuscitation should continue until expert advice can be obtained. Cardiopulmonary bypass has been used successfully in a number of poisonings.

Attempts at decontamination of the skin or gastrointestinal tract never take priority over resuscitation and institution of supportive care measures.

AIRWAY, BREATHING AND CIRCULATION
Acute poisoning is a dynamic medical illness and patients may deteriorate within a few minutes or hours of presentation. Altered conscious state, loss of airway protective reflexes and hypotension are common threats to life in the poisoned patient.

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<td><strong>Breathing</strong></td>
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<td><strong>Circulation</strong></td>
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<tr>
<td><strong>Detect and correct:</strong></td>
</tr>
<tr>
<td><strong>Seizures</strong></td>
</tr>
<tr>
<td>Always generalised when due to toxicologic causes</td>
</tr>
<tr>
<td>Benzodiazepines first-line</td>
</tr>
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<td><strong>Hypoglycaemia</strong></td>
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<td>Check bedside blood sugar level (BSL) in all patients with altered mental status</td>
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<td>Treat if BSL &lt;4.0mmol/L</td>
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<tr>
<td><strong>Hyper-/hypothermia</strong></td>
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<td>Temp &gt;38.5°C prompts urgent intervention</td>
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As in all life-threatening emergencies, attention to airway, breathing and circulation are paramount. These priorities are usually managed along conventional lines. Basic resuscitative and supportive care measures ensure the survival of the vast majority of patients.

Although commonly used to describe a patient’s mental status, clinical scores such as the Glasgow Coma Scale (GCS) or Alert-Verbal-Pain-Unresponsive (AVPU) system have never been systematically validated across all poisonings. A patient’s ability to guard their airway is not well correlated to GCS. An increased risk of aspiration has been noted with GCS less than 12. Moreover, a patient’s ability to guard the airway and ventilate effectively may change within a short period of time.

In some specific situations, standard resuscitation algorithms do not apply (see Table 1.2.2).

**DETECT AND CORRECT SEIZURES**

Toxic seizures are generalised, and can usually be controlled with intravenous benzodiazepines (e.g. diazepam, midazolam, lorazepam or clonazepam). The most common causes of seizures in poisoned patients in Australasia are venlafaxine, bupropion, tramadol and amphetamines.

The presence of focal or partial seizures indicates a focal neurological disorder that is either a complication of poisoning or due to a non-toxicologic cause, and prompts further investigation.

Barbiturates are second-line therapy for refractory seizures in acute poisoning. Pyridoxine is a third-line agent that may be indicated in intractable seizures secondary to isoniazid.

Phenytoin is contraindicated in the management of seizures related to acute poisoning.

**DETECT AND CORRECT HYPOGLYCAEMIA**

Hypoglycaemia is an easily detectable and correctable cause of significant neurological injury. Bedside serum glucose estimation should be performed as soon as possible in all patients with altered mental status.

If the serum glucose is less than 4.0 mmol/L, 50 mL of 50% dextrose should be given intravenously (5 mL/kg 10% dextrose in children) to urgently correct hypoglycaemia. The result may be confirmed later with a formal serum glucose measurement.

Hypoglycaemia in acute poisoning is associated with insulin, sulfonylurea oral hypoglycaemic agents, beta-blockers, quinine, chloroquine, salicylates and valproic acid.

**DETECT AND CORRECT HYPER-/HYPOTHERMIA**

Hyperthermia is associated with a number of life-threatening acute poisonings and is associated with poor outcome.
### TABLE 1.2.2 Specific resuscitation situations in toxicology where conventional algorithms or approaches may not apply

<table>
<thead>
<tr>
<th>Life-threat</th>
<th>Mechanism</th>
<th>Agent(s)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIRWAY</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Airway compromise | Corrosive injury to oropharynx | ● Alkalis  
● Acids  
● Glyphosate  
● Paraquat | ● Stridor, dysphagia and dysphonia indicate airway injury and potential for imminent airway compromise  
● Early endotracheal intubation or surgical airway often required |
| BREATHING   |           |          |          |
| Acidosis    | Various   | ● Ethylene glycol  
● Methanol  
● Salicylates | ● Until late in the clinical course there is usually prominent respiratory compensation  
● Intubation and ventilation at standard settings may worsen acidaemia and precipitate rapid clinical deterioration, if not death.  
● Avoid normo- or hypoventilation  
● Maintain hyperventilation and consider bolus IV NaHCO₃ 1–2 mmol/kg to prevent worsening of acidaemia |
| Acidaemia   | Opioid mu receptor stimulation | ● Opioids | ● Prompt administration of naloxone may obviate need for intubation and ventilation |
| Hypoventilation | Cholinergic crisis | ● Carbamates  
● Nerve agents  
● Organophosphates | ● Rapid administration of atropine by serial doubling of atropine dose to achieve dry respiratory secretions may restore adequate oxygenation |
| Acidosis; Hypoxaemia; Multiple organ failure (MOF) | Oxygen-free radical mediated cellular injury, particularly type II pneumocytes | Paraquat | Avoid supplemental oxygen  
If hypoxia occurs, titrate supplemental oxygen to maintain oxygen saturation of ~90% or PaO₂ 60 mmHg |
| CIRCULATION | Ventricular fibrillation | Hypocalcaemia | Hydrofluoric acid ingestion or massive cutaneous burn | Defibrillation alone unlikely to be efficacious  
Bolus IV calcium (e.g. 60–90 mL 10% calcium gluconate) repeated as required every 2 minutes until defibrillation restores perfusing rhythm |
| Ventricular tachycardia | Fast Na⁺ channel blockade | Chloroquine  
Cocaine  
Flecainide  
Local anaesthetic agents  
Procainamide  
Propranolol  
Quinine  
Tricyclic antidepressants | Cardioversion or defibrillation unlikely to be efficacious  
Urgently intubate and hyperventilate  
Bolus IV NaHCO₃ 1–2 mmol/kg repeated every 1–2 minutes until restoration of perfusing rhythm  
Do not await determination of serum pH prior to intubation and NaHCO₃ boluses  
Lignocaine is third-line therapy when pH is established at >7.5  
Amiodarone and Vaughan Williams type la antiarrhythmic agents (e.g. procainamide) are contraindicated |
| Ventricular ectopy/ Ventricular tachycardia | Halogen-induced myocardial sensitisation to catecholamines | Chloral hydrate  
Organochlorines | Cardioversion or defibrillation unlikely to be efficacious  
Administer IV beta-blockers, titrate to ectopy response |

Continued
### TABLE 1.2.2 Specific resuscitation situations in toxicology where conventional algorithms or approaches may not apply—cont’d

<table>
<thead>
<tr>
<th>Life-threat</th>
<th>Mechanism</th>
<th>Agent(s)</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Refractory hypotension | Various | - Beta-blockers  
- Calcium channel blockers  
- Local anaesthetic agents | - High-dose insulin–dextrose therapy |
| Tachycardia | Central and peripheral sympathomimetic response | - Amphetamines  
- Cocaine | - Beta-blockers contraindicated  
- Administer IV benzodiazepines, titrated to gentle sedation and heart rate control |
| Supraventricular tachycardia | Adenosine antagonism | - Theophylline | - Urgent haemodialysis indicated |
| Hypertension | Central and peripheral sympathomimetic response | - Amphetamines  
- Cocaine | - Beta-blockers contraindicated  
- Administer IV benzodiazepines, titrated to gentle sedation and heart rate control  
- If further therapy necessary use agents that can be given by titratable intravenous infusion  
  - Glycerol trinitrate (GTN)  
  - Phentolamine  
  - Nitroprusside |
| Asystole Bradycardia Tachycardia | Na+/K+ ATPase pump inhibition | - Digoxin | - Usual resuscitation interventions futile  
- Digoxin-specific antibodies |
### Bradycardia
- Hypotension
- Cardiac conduction defects

| Calcium channel blockade | Calcium channel blockers | Atropine and pacing unlikely to be efficacious

- Bolus IV calcium (e.g. 60 mL 10% calcium gluconate) may provide temporary haemodynamic stability by increasing HR and BP, while other treatments are organised
- High-dose insulin–dextrose therapy

### Acute coronary syndrome
- Central and peripheral sympathomimetic response

| Amphetamines
| Cocaine |
| Beta-blockers contraindicated
| Benzodiazepines
| GTN
| Antiplatelet and anticoagulation therapy if no neurological deficits (otherwise cranial CT first)
| Reperfusion therapy along conventional lines

### OTHER

| Hyperkalaemia
| Na\(^+\)/K\(^+\) ATPase pump inhibition |
| Digoxin |
| Calcium salts are contraindicated
| Digoxin-specific antibodies |

| Hypoglycaemia
| Hyperinsulinaemia |
| Sulfonylureas |
| Difficult to maintain euglycaemia with dextrose supplementation alone
| Octreotide administration obviates need for dextrose supplementation |

| Refractory seizures
| Inhibition of GABA production |
| Isoniazid |
| IV pyridoxine 1 g per gram of isoniazid ingested, up to 5 g |

| Seizures
| Adenosine antagonism |
| Theophylline |
| Urgent haemodialysis indicated |
A temperature greater than 38.5°C during the resuscitation phase of management is an indication for continuous core-temperature monitoring. A temperature greater than 39.5°C is an emergency that requires prompt management to prevent multiple organ failure and neurological injury. Neuromuscular paralysis with intubation and ventilation leads to a cessation of muscle-generated heat production and a rapid reduction of temperature. Profound hypothermia (core temperature <29°C) may mimic or cause cardiac arrest. Clinical manifestations include coma, fixed and dilated pupils, bradycardia (usually atrial fibrillation) and hypotension. Vital signs may be difficult to elicit and the cardiac rhythm may degenerate to ventricular fibrillation or asystole. In the patient with undetectable vital signs, aggressive exogenous rewarming is indicated while CPR continues. Cardiopulmonary bypass, if available, is the most effective means. An alternative measure is pleural lavage through an intercostal catheter with large volumes of fluid warmed to 40–45°C.

**EMERGENCY ANTIDOTE ADMINISTRATION**

Administration of antidotes is sometimes indicated during the resuscitation phase of management. As with all drugs, antidotes have indications, adverse effects and contraindications. The decision to administer an antidote during resuscitation will depend on the perceived benefit compared to possible adverse effects.

Examples where early administration of an antidote is necessary to ensure a successful resuscitation include intravenous sodium bicarbonate in tricyclic antidepressant poisoning, naloxone in severe opioid intoxication, atropine in severe organophosphorus agent intoxication, and digoxin-specific antibodies for patients with suspected digoxin intoxication with cardiovascular compromise.

**References**


**1.3 RISK ASSESSMENT**

Risk assessment should occur as soon as possible in the management of the poisoned patient. Only resuscitation is a greater priority. Risk assessment is a distinct quantitative cognitive step through which the clinician attempts to predict the likely clinical course and potential complications for the individual patient at that particular presentation.
The five key components of the history and examination required to construct a risk assessment are listed in Table 1.3.1.

Risk assessment is pivotal as it allows the clinician to identify potential problems and make specific balanced decisions about all subsequent management steps (supportive care and monitoring, screening and specialised testing, decontamination, enhanced elimination, antidotes and disposition).

Provided their mental status is normal, patients with deliberate self-poisoning are generally both willing and able to give a good history from which an accurate risk assessment can be constructed. Physicians ignore the patient’s history at their peril.

If altered mental status precludes obtaining a direct history, back-up strategies are employed to gather the necessary information. These include:

1. Asking ambulance officers or family to search for agents
2. Counting missing tablets
3. Checking medical records for previous prescriptions
4. Questioning relatives about agents potentially available to the patient.

Under these circumstances, the risk assessment is less accurate and is often based on a ‘worst-case scenario’. This is commonly the case with small children where ingestions are rarely witnessed. As the clinical course progresses, the risk assessment and management plan may be refined.

In unknown ingestions, the patient’s clinical status is correlated with the clinician’s knowledge of the agents commonly available in that geographic area. For example, CNS and respiratory depression associated with miotic pupils indicates opioid intoxication in a young adult male in urban Australia, but is more likely to indicate organophosphate intoxication in rural Sri Lanka.

The agent, dose and time since ingestion should correlate with the patient’s current clinical status. If they do not, the risk assessment needs to be reviewed and revised.

Acute poisoning is a dynamic process and important decisions can often be made at particular time points. For example, following tricyclic antidepressant self-poisoning, life-threatening events occur within 6 hours

<table>
<thead>
<tr>
<th>TABLE 1.3.1</th>
<th>Steps for construction of a risk assessment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Distinct cognitive step</strong></td>
<td><strong>Quantitative</strong></td>
</tr>
<tr>
<td><strong>Takes into account</strong></td>
<td></td>
</tr>
<tr>
<td>1 Agent(s)</td>
<td>Takes into account</td>
</tr>
<tr>
<td>2 Dose(s)</td>
<td>Takes into account</td>
</tr>
<tr>
<td>3 Time since ingestion</td>
<td>Takes into account</td>
</tr>
<tr>
<td>4 Clinical features and progress</td>
<td>Takes into account</td>
</tr>
<tr>
<td>5 Patient factors (weight and co-morbidities)</td>
<td>Takes into account</td>
</tr>
</tbody>
</table>
(and usually within the first 2 hours) of ingestion. Therefore, low-risk patients can be identified on clinical grounds at 6 hours post-ingestion. In contrast, following deliberate self-poisoning with sustained-release calcium channel blockers, patients may not exhibit clinical features of poisoning during the first few hours. Indeed, the risk assessment anticipates delayed severe cardiovascular effects.

In the majority of cases, the risk assessment allows early recognition of medically trivial poisonings. This reassures attending staff, family and patient and permits the avoidance of unnecessary investigations, interventions and observation. Early psychosocial assessment and discharge planning may begin. This usually shortens hospital length of stay.

Less commonly but very importantly, risk assessment allows early identification of potentially serious poisoning and the implementation of a tailored proactive management plan. Balanced decisions about gastrointestinal decontamination can be made and appropriate investigations selected. If a specialised procedure or antidote might be required in the next few hours, early communication and disposition planning may begin.

**ROLE OF THE POISONS INFORMATION CENTRE**

The clinician’s ability to construct an accurate risk assessment relies on knowledge and experience of the toxic agents concerned. Although this is straightforward for many exposures, new or unusual agents are frequently encountered. A variety of sources of information may be used to obtain the information necessary to formulate a risk assessment. Textbooks and databases are often difficult to interpret and apply to the individual patient. When faced with a time-critical poisoning emergency, a call to the poisons information centre is the most rapid mechanism to obtain accurate information and individualised risk assessment.

The Australian Poisons Information Centre network comprises centres located in Sydney, Perth, Brisbane and Melbourne that can be accessed nation-wide by calling **131126**. The New Zealand Poisons Information Centre located in Dunedin is accessed by calling **0800-POISON (0800 764 766)**. Trained poisons information specialists with a background in pharmacy or medical science are familiar with accessing information from computerised databases and other information sources. They can assist in the identification of commercial products and their constituents and in the formulation of a risk assessment, provided the clinician is able to provide the basic dataset. Where necessary, medical callers treating an acute poisoning case are referred to an on-call clinical toxicologist who is able to offer more detailed individualised risk assessment and management advice.

**References**

1.4 SUPPORTIVE CARE AND MONITORING

Following resuscitation and risk assessment, supportive care and disposition planning can begin.

Poisoning morbidity and mortality usually result from the acute effects of the toxin on the cardiovascular, central nervous or respiratory systems. Support of these and other systems for the duration of the intoxication will ensure a good outcome for the vast majority of acute poisonings. Monitoring is essential to detect the progress of the intoxication and the timing of institution, escalation and withdrawal of supportive care and other measures.

An initial period of close observation in the emergency department is usually appropriate. During this time the patient’s clinical status is monitored closely to ensure that it correlates with the previous risk assessment. If early complications are expected (e.g. decreased level of consciousness requiring intubation in the following 2 hours), preparations can be made to secure the airway as soon as the intoxication declares itself, and before the patient is moved elsewhere. If unexpected deterioration occurs at any time, the clinician’s priorities revert to resuscitation prior to revising the risk assessment.

The duration of observation depends on the agent(s) ingested, the formulations involved (e.g. sustained-release preparations) and potential complications. For example, patients with significant beta-blocker and tricyclic antidepressant deliberate self-poisoning develop symptoms and signs of major intoxication within 2–4 hours of ingestion. In contrast, patients with sustained-release calcium channel blocker or valproic acid deliberate self-poisoning may take 6–12 hours to develop signs of major toxicity.

Disposition from the emergency department depends on the current and expected clinical status of the patient. If specific complications are anticipated, the chosen inpatient clinical area must be resourced to detect and manage them.

The accuracy and skill of the initial management and risk assessment is wasted if the subsequent plan of management is not documented and communicated to the treating team. Good practice includes the documentation of a comprehensive management plan that informs the team looking after the patient of:

1. Expected clinical course
2. Potential complications according to the individual risk assessment
3. Type of observation and monitoring required
4. Endpoints that must trigger notification of the treating doctor or further consultation
5. Management plans for agitation or delirium
6. Criteria for changing management
7. Provisional psychosocial risk assessment with contingency plan should the patient attempt to abscond prior to formal psychosocial assessment.
The needs of the vast majority of patients can be met in the emergency department, emergency observation unit or intensive care unit. The emergency observation unit is appropriate for the ongoing management of most acute poisonings, where the general supportive measures outlined below can be provided.

Criteria for admission to an emergency observation unit following acute poisoning include:

1. Ongoing cardiac monitoring not required
2. Adequate sedation achieved
3. Clinical deterioration not anticipated.

Criteria for admission to an intensive care unit following acute poisoning include requirements for:

1. Airway control
2. Ventilation
3. Prolonged or invasive haemodynamic monitoring or support
4. Haemodialysis.

### TABLE 1.4.1 Supportive care measures

<table>
<thead>
<tr>
<th>Airway</th>
<th>Breathing</th>
</tr>
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<tbody>
<tr>
<td>Intubation</td>
<td>Supplemental oxygen</td>
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<tr>
<td></td>
<td>Ventilation</td>
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<tr>
<td>Circulation</td>
<td></td>
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<tr>
<td>Intravenous fluids</td>
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<tr>
<td>Inotropes</td>
<td></td>
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<tr>
<td>Control of hypertension</td>
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<tr>
<td>Cardiopulmonary bypass</td>
<td></td>
</tr>
<tr>
<td>Sedation</td>
<td>Titrated IV benzodiazepines</td>
</tr>
<tr>
<td>Seizure control/prophylaxis</td>
<td>IV benzodiazepines</td>
</tr>
<tr>
<td>Metabolic</td>
<td></td>
</tr>
<tr>
<td>Ensuring normoglycaemia</td>
<td></td>
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<tr>
<td>Control of pH</td>
<td></td>
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<tr>
<td>Fluids and electrolytes</td>
<td></td>
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<tr>
<td>Renal function</td>
<td></td>
</tr>
<tr>
<td>Adequate hydration</td>
<td></td>
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<tr>
<td>Haemodialysis</td>
<td></td>
</tr>
<tr>
<td>General</td>
<td></td>
</tr>
<tr>
<td>Nutrition</td>
<td></td>
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<tr>
<td>Respiratory toilet</td>
<td></td>
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<tr>
<td>Bladder care (indwelling catheter)</td>
<td></td>
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<tr>
<td>Prevention of pressure areas</td>
<td></td>
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<tr>
<td>Thrombo-embolism prophylaxis</td>
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<tr>
<td>Mobilisation as mental status changes resolve</td>
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</tr>
</tbody>
</table>
1.5 INVESTIGATIONS

Investigations in acute poisoning are employed either as screening tests or for specific purposes.

Screening refers to the performance of a medical evaluation and/or diagnostic test in asymptomatic persons in the hope that early diagnosis may lead to improved outcome. In the acutely poisoned patient, screening tests aim to identify occult toxic ingestions for which early specific treatment is indicated.

The recommended screening tests for acute poisoning are the 12-lead electrocardiogram (ECG) and the serum paracetamol level.

The ECG is a readily available non-invasive tool that assists in the identification of occult but potentially lethal cardiac conduction abnormalities, such as those in tricyclic antidepressant cardiotoxicity.

Paracetamol is a ubiquitous analgesic in the western world. Deliberate self-poisoning with paracetamol is common, comprising up to 15% of adult poisoning presentations in Australasia. Life-threatening paracetamol poisoning may be occult in the early stages but progression to fulminant hepatic failure and death can be prevented by timely administration of N-acetylcysteine. Although a thorough cost–benefit analysis has never been performed, it is postulated that the cost of several thousand serum paracetamol measurements is offset by the detection of one potentially preventable paracetamol-related death or liver transplant. For this reason, it is advisable to screen for paracetamol in all cases of known or suspected acute deliberate self-poisoning. Screening is particularly important where altered mental status precludes obtaining an ingestion history directly from the patient.

The screening paracetamol level may be performed at presentation and does not need to be delayed until 4 hours after ingestion. A non-detectable paracetamol level greater than 1 hour after ingestion excludes significant paracetamol ingestion and further paracetamol levels are not required.

If paracetamol poisoning is suspected after the initial risk assessment, then a screening paracetamol level is not required. Instead, a timed

<table>
<thead>
<tr>
<th>TABLE 1.5.1 Screening tests</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>12-lead ECG</strong></td>
</tr>
<tr>
<td>- Rate</td>
</tr>
<tr>
<td>- Rhythm</td>
</tr>
<tr>
<td>- PR interval</td>
</tr>
<tr>
<td>- QRS interval</td>
</tr>
<tr>
<td>- QT interval</td>
</tr>
<tr>
<td>- Dominant R wave in aVR</td>
</tr>
<tr>
<td><strong>Serum paracetamol level</strong></td>
</tr>
</tbody>
</table>
paracetamol level should be performed as soon as possible after 4-hours post-ingestion as an additional risk assessment tool.

Serum salicylate and tricyclic antidepressant assays have been advocated as routine screening tests. Salicylate poisoning is now relatively uncommon in Australasia. Significant acute intoxication is associated with an easily recognised pattern of symptoms and acid–base disturbances and is rarely occult. Therefore, routine screening for salicylate in patients without symptoms or signs of salicylism does not comply with the rationale for screening. Serum tricyclic antidepressant levels are correlated to complications and outcome following acute poisoning. However, the major complications of tricyclic antidepressant poisoning usually occur within 2–4 hours of ingestion. The 12-lead ECG, correlated to the patient’s clinical status, reflects target organ effects more accurately and is the preferred screening test.

Many poisoned patients are young and have few medical co-morbidities. After appropriate risk assessment and the institution of supportive care they may require no further investigation beyond the screening ECG and serum paracetamol measurement. In a young and otherwise healthy patient presenting with normal mental status and vital signs, additional tests such as electrolytes, full blood picture, liver function tests and coagulation studies are not routinely indicated.

Other investigations are ordered selectively where it is anticipated that the results will assist risk assessment or management. Potential indications for specific tests in the acute poisoning patient are shown in Table 1.5.2.

For most patients and poisonings, the risk assessment and subsequent clinical course dictate management decisions. Drug concentrations do not usually assist decision making. Some of the few agents where serum levels assist in risk assessment or management decisions are shown in Table 1.5.3.

Qualitative urine screens for drugs of abuse (e.g. opioids, benzodiazepines, amphetamines, cocaine, barbiturates and cannabinoids) rarely alter the management of the acutely poisoned patient. Patients with acute intoxication with one or more of these agents may be managed according to their clinical presentation. False positives and negatives

<table>
<thead>
<tr>
<th>TABLE 1.5.2 Indications for other investigations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Refine risk assessment or prognosis</td>
</tr>
<tr>
<td>Exclude or confirm an important differential diagnosis</td>
</tr>
<tr>
<td>Exclude or confirm an important specific poisoning</td>
</tr>
<tr>
<td>Exclude or confirm a complication that requires specific management</td>
</tr>
<tr>
<td>Establish an indication for antidote administration</td>
</tr>
<tr>
<td>Establish an indication for institution of enhanced elimination</td>
</tr>
<tr>
<td>Monitor response to therapy or define an end point for a therapeutic intervention</td>
</tr>
</tbody>
</table>
occur. A positive result from a patient without corresponding symptoms of intoxication rarely alters acute medical management.

References

1.6 GASTROINTESTINAL DECONTAMINATION

Physicians have long directed great effort into attempts at gastrointestinal decontamination following ingestion of toxic substances. They have employed a variety of methods (see Table 1.6.1) in the reasonable
expectation that by reducing the dose absorbed they will also reduce the subsequent severity and duration of clinical toxicity. Unfortunately, the tendency has been to overestimate the potential benefits while underestimating the potential hazards of gastrointestinal decontamination procedures. These procedures do not provide significant benefit when applied to unselected deliberate self-poisoned patients and are no longer considered routine.

The theoretical benefits of gastrointestinal decontamination in selected poisonings have not been evaluated. The decision to decontaminate is one of clinical judgment in which the potential benefits are weighed against the potential risks and the resources required to perform the procedure (see Figure 1.6.1 and Table 1.6.2).

Employing this rationale, gastrointestinal decontamination is reserved for cases where the risk assessment predicts severe or life-threatening toxicity and where supportive care or antidote treatment alone is insufficient to ensure a satisfactory outcome. There should be reasonable grounds to believe that a significant amount of agent remains unabsorbed and is amenable to removal by the selected procedure. This requires some knowledge of the absorption kinetics of the agent(s) involved. For most ingested agents, absorption is virtually complete within 1 hour.

Gastrointestinal decontamination is never performed to the detriment of basic resuscitation or supportive care. To avoid pulmonary aspiration, the procedure is not performed without first securing the airway in a patient with a depressed level of consciousness or in whom the risk

**TABLE 1.6.1 Methods of gastrointestinal decontamination**

- Induced emesis (syrup of ipecac)
- Gastric lavage
- Activated charcoal
- Whole bowel irrigation

**TABLE 1.6.2 Gastrointestinal decontamination: risk–benefit analysis**

<table>
<thead>
<tr>
<th>Potential benefits</th>
<th>Potential risks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved clinical outcome (morbidity and mortality)</td>
<td>Pulmonary aspiration</td>
</tr>
<tr>
<td>More benign clinical course requiring lower level of supportive care</td>
<td>Gastrointestinal complications</td>
</tr>
<tr>
<td>Reduced need for other potentially hazardous interventions or expensive antidotes</td>
<td>— bowel obstruction</td>
</tr>
<tr>
<td>— perforation</td>
<td>Distraction of staff from resuscitation and supportive care priorities</td>
</tr>
<tr>
<td>— Diversion of departmental resources for performance of procedure</td>
<td>Reduced hospital length of stay</td>
</tr>
</tbody>
</table>
assessment indicates a potential for imminent seizures or decline in conscious state.

**INDUCED EMESIS (SYRUP OF IPECAC)**

Emptying the stomach by inducing emesis has a long tradition in clinical toxicology. In recent times it has been achieved almost exclusively by the administration of syrup of ipecac. This preparation contains powerful plant-derived emetics and, when administered at the recommended dose, reliably induces vomiting via central and peripheral mechanisms. The mean time from administration to vomiting is 18 minutes. For many years it was routinely recommended for home use following accidental paediatric ingestions with the intention of reducing the time to decontamination and the need for hospital referral. It is now clear that the amount of toxin removed is unreliable and decreases rapidly with time to the point that it is negligible by 1 hour. Syrup of ipecac-induced vomiting renders subsequent administration of activated charcoal more difficult. The potential benefits of syrup of ipecac theoretically outweigh the risks when it is administered promptly after ingestion of an agent in a dose likely to cause significant toxicity, that does not involve rapid onset of depressed level of consciousness or seizures and where activated charcoal is not readily available or known not to bind to the agent. Such a scenario arises so infrequently that emergency departments no longer stock syrup of ipecac and poisons information centres no longer advise it to be kept in homes with small children.

**Technique**
- Give 15 mL (children) or 15–30 mL (adults) with a glass of water
- If vomiting has not occurred within 30 minutes the dose may be repeated.

**Contraindications**
- Non-toxic ingestion
- Dose ingested known to be sub-toxic
- Seizures or decreased level of consciousness
- Risk assessment indicates potential for seizures or decreased level of consciousness within the next few hours
- Activated charcoal available within 1 hour and known to bind agent
- Infants <12 months of age
- Corrosive ingestion
- Hydrocarbon ingestion.

**Potential complications**
- Prolonged vomiting (10–20% vomit for more than 1 hour)
- Diarrhoea (20–30%)
- Lethargy (10%)
- Pulmonary aspiration if decreased mental status or seizures
- Physical injuries secondary to vomiting (rare)
  - Mallory Weiss tear
  - Pneumomediastinum
  - Gastric perforation.

**GASTRIC LAVAGE**

This technique attempts to empty the stomach of toxic substances by the sequential administration and aspiration of small volumes of fluid from the stomach via an orogastric tube. This previously widely favoured method of gastrointestinal decontamination has now been all but abandoned and few emergency departments remain experienced in its use.

The amount of toxin removed by gastric lavage is unreliable and negligible if performed after the first hour. It does not confer any clinical benefit when performed routinely on unselected patients presenting to the emergency department following deliberate self-poisoning. There are few situations where the expected benefits of this procedure might be judged to exceed the risks involved and where administration of charcoal would not be expected to provide equal or greater efficacy of decontamination.

**Technique**

- This procedure is performed in a resuscitation bay
- Do not perform in any patient with an impaired level of consciousness unless the airway is protected by a cuffed endotracheal tube
- Position the patient in the left decubitus position with 20° head down
- Measure the length of tube required to reach the stomach externally before beginning the procedure
- Pass a large bore 36–40 G lubricated lavage tube extremely gently down the oesophagus. Stop if any resistance occurs
- Confirm tube position by aspirating gastric contents and auscultating for insufflated air at the stomach
- Administer a 200 mL aliquot of warm tap water or normal saline into the stomach via the funnel and lavage tube
- Drain the administered fluid into a dependent bucket held adjacent to the bed
- Repeat administration and drainage of fluid aliquots until the effluent is clear
- Activated charcoal 50 g may be administered via the tube once lavage complete.

**Absolute contraindications**

- Initial resuscitation incomplete
- Risk assessment indicates good outcome with supportive care and antidote therapy alone
Unprotected airway where there is a decreased level of consciousness or risk assessment indicates potential for this complication during the procedure
- Small children
- Corrosive ingestion
- Hydrocarbon ingestion.

Potential complications
- Pulmonary aspiration
- Hypoxia
- Laryngospasm
- Mechanical injury to the gastrointestinal tract
- Water intoxication (especially in children)
- Hypothermia
- Distraction of staff from resuscitation and supportive care priorities.

SINGLE-DOSE ACTIVATED CHARCOAL
Activated charcoal (AC) is produced by the super-heating of distilled wood pulp. The resulting fine porous particles are suspended in water or sorbitol prior to oral or nasogastric administration. The enormous surface area provided by these particles reversibly adsorbs most ingested toxins preventing further absorption from the gastrointestinal tract.

Oral AC is generally the preferred method of decontamination. However, it does not improve clinical outcome when applied to unselected patients with self-poisoning and should not be regarded as routine. It is indicated where it is likely that toxin remains in the gastrointestinal tract (within the first hour for most agents) and where the potential benefits outweigh the potential risks. The major risk is charcoal pulmonary aspiration due to loss of airway reflexes associated with impaired level of consciousness or seizures.

There are no data to support the use of AC in sorbitol or other cathartic agent over AC in water.

Complications
- Vomiting (30% of patients given AC vomit within 1 hour)
- Mess
- Pulmonary aspiration
- Direct administration into lung via misplaced nasogastric tube (potentially fatal)
- Impaired absorption of subsequently administered oral antidotes or other therapeutic agents
- Corneal abrasions
- Distraction of attending staff from resuscitation and supportive care priorities.
**Contraindications**
- Initial resuscitation incomplete
- Non-toxic ingestion
- Sub-toxic dose
- Risk assessment indicates good outcome with supportive care and antidote therapy alone
- Decreased level of consciousness, delirium or poor cooperation (unless airway protected by endotracheal intubation)
- Risk assessment suggests potential for imminent onset of seizures or decreased level of consciousness.
- Agent not bound to AC (see Table 1.6.3)
- Corrosive ingestion.
  Note: Ileus is not a contraindication to single-dose AC.

**Technique**
- Give 50 g (adults) or 1 g/kg (children) as a single oral dose placed in a cup for self-administration
- Mixing with ice cream improves palatability for children
- In the intubated patient, AC may be given via oro- or nasogastric tube after tube placement is confirmed on chest x-ray.
  Note: If mental status precludes self-administration, AC is withheld until the patient is intubated if and when this becomes clinically necessary. The decision to intubate is based on standard criteria. Only in very rare circumstances does the risk assessment justify intubation specifically for the purpose of facilitating administration of AC.

**WHOLE BOWEL IRRIGATION**
This aggressive and labour-intensive form of gastrointestinal decontamination attempts to cleanse the entire bowel by administering large volumes of osmotically balanced polyethylene glycol electrolyte solution (PEG-ELS). It is rarely performed because risk–benefit analysis reserves this intervention for life-threatening ingestions of sustained-release or enteric-coated preparations, or agents that do not bind to charcoal and where good clinical outcome is not expected with supportive care and antidote administration and the patient presents before established severe toxicity (see Table 1.6.4).
Whole bowel irrigation has been performed on unconscious ventilated patients but this is hazardous as fluid may pool in the oropharynx and flow past the tube cuff to produce pulmonary aspiration.

**Complications**
- Nausea, vomiting and abdominal bloating
- Non-anion gap metabolic acidosis
- Pulmonary aspiration
- Distraction from resuscitation and supportive care priorities
- Delayed retrieval to a hospital offering definitive care.

**Contraindications**
- Risk assessment suggests good outcome can be assured with supportive care and antidote therapy
- Uncooperative patient
- Inability to place a nasogastric tube
- Uncontrolled vomiting
- Risk assessment suggests potential for decreased conscious state or seizure in the subsequent four hours
- Ileus or intestinal obstruction
- Intubated and ventilated patient (relative contraindication).

**Technique**
- Assign a single nurse to carry out procedure (this is a full-time job for up to 6 hours)
- Obtain sufficient supplies of PEG-ELS and make up solution as directed
- Place nasogastric tube
- Give activated charcoal 50 g (children 1 g/kg) via the nasogastric tube in non-metallic ingestions
- Administer PEG solution via the nasogastric tube at 2 L/hour (children 25 mL/kg/hour)
- Administer metoclopramide to minimise vomiting and enhance gastric emptying
- Position patient on a commode if possible to accommodate explosive diarrhoea
- Continue irrigation until the effluent is clear. This may take up to 6 hours
- Cease whole bowel irrigation if abdominal distension or loss of bowel sounds are noted

<table>
<thead>
<tr>
<th>TABLE 1.6.4  Whole bowel irrigation potentially useful</th>
</tr>
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<tbody>
<tr>
<td>- Iron overdose &gt;60 mg/kg</td>
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<tr>
<td>- Slow-release potassium chloride ingestion &gt;2.5 mmol/kg</td>
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<tr>
<td>- Life-threatening slow-release verapamil or diltiazem ingestions</td>
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<tr>
<td>- Symptomatic arsenic trioxide ingestion</td>
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<tr>
<td>- Lead ingestion</td>
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<tr>
<td>- ‘Body packers’ (see Chapter 2.17: Body packers and stuffers)</td>
</tr>
</tbody>
</table>
- Abdominal x-ray is useful to assess effectiveness of decontamination of radio-opaque substances such as iron and potassium salts
- Expelled packages may be counted in body packers.

References

1.7 ENHANCED ELIMINATION

Techniques of enhanced elimination (see Table 1.7.1) are employed to increase the rate of removal of an agent from the body with the aim of reducing the severity and duration of clinical intoxication. These interventions are only indicated if it is thought they will reduce mortality, length of stay, complications or the need for other more invasive interventions. In practice, these techniques are useful in the treatment of poisoning by only a few agents that are characterised by:
- Severe toxicity
- Poor outcome despite good supportive care/antidote administration
- Slow endogenous rates of elimination
- Suitable pharmacokinetic properties.

Accurate risk assessment allows early identification of those patients who may benefit from enhanced elimination and institution of the intervention before severe life-threatening intoxication develops. Some of these techniques require specialised equipment and staff and early identification of candidates facilitates the timely communication, planning and transport necessary to ensure a good outcome.

The final decision as to whether to initiate a technique of enhanced elimination depends on a risk–benefit analysis in which the expected benefits of the procedure are balanced against the resource utilisation and risks associated with the procedure.

Techniques of enhanced elimination are never carried out to the detriment of resuscitation, good supportive care, decontamination and antidote treatment.
Once the decision to initiate a technique of enhanced elimination is made, it is important to establish pre-defined clinical or laboratory end points for therapy.

**MULTIPLE-DOSE ACTIVATED CHARCOAL (MDAC)**

**Rationale**

Repeated administration of oral activated charcoal progressively fills the entire gut lumen with charcoal. This has the potential to enhance drug elimination in two ways:

- **Interruption of entero-hepatic circulation**
  - A number of drugs are excreted in the bile and then reabsorbed from the distal ileum. Charcoal in the small intestine binds drug and prevents reabsorption thus enhancing elimination
  - This is only significant if a drug not only undergoes entero-hepatic circulation but also has a relatively small volume of distribution

- **Gastrointestinal dialysis**
  - Drug passes across the gut mucosa from a relatively high concentration in the intravascular compartment to a low concentration in the gut lumen, which is maintained by continuing adsorption to charcoal
  - This is only effective if the drug is a relatively small molecule, lipid soluble, has a small volume of distribution and low protein binding.

**Indications**

Enhanced elimination by this technique has been proposed as clinically useful in the following scenarios:

- **Carbamazepine coma**
  - Most common indication for MDAC
  - Used in the expectation that enhanced elimination will reduce duration of ventilation and length of stay in intensive care

<table>
<thead>
<tr>
<th>TABLE 1.7.1 Techniques of enhanced elimination and amenable agents</th>
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</thead>
<tbody>
<tr>
<td><strong>Multiple-dose activated charcoal</strong></td>
</tr>
<tr>
<td>Carbamazepine</td>
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<tr>
<td>Dapsone</td>
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<tr>
<td>Phenobarbitone</td>
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<tr>
<td>Quinine</td>
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<tr>
<td>Theophylline</td>
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<tr>
<td><strong>Urinary alkalinisation</strong></td>
</tr>
<tr>
<td>Phenobarbitone</td>
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<tr>
<td>Salicylate</td>
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<tr>
<td><strong>Haemodialysis and haemofiltration</strong></td>
</tr>
<tr>
<td>Lithium</td>
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<tr>
<td>Metformin lactic acidosis</td>
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<tr>
<td>Potassium</td>
</tr>
<tr>
<td>Salicylate</td>
</tr>
<tr>
<td>Theophylline</td>
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<tr>
<td>Toxic alcohols</td>
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<tr>
<td>Valproic acid</td>
</tr>
<tr>
<td><strong>Charcoal haemoperfusion</strong></td>
</tr>
<tr>
<td>Theophylline</td>
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</tbody>
</table>
- Phenobarbitone coma
  — Rare
  — Used in the in the expectation that enhanced elimination will reduce duration of ventilation and length of stay in intensive care
- Dapsone overdose with methaemoglobinaemia
  — Very rare
  — MDAC may enhance elimination of dapsone and reduce the duration of severe prolonged methaemoglobinaemia
- Quinine overdose
  — Although MDAC might enhance drug elimination, good outcome can be expected with aggressive supportive care
- Theophylline overdose
  — Attempts at enhanced elimination with MDAC should never delay more effective elimination with haemodialysis following life-threatening overdose.

**Absolute contraindications**
- Decreased level of consciousness or anticipated decreased level of consciousness without prior airway protection
- Bowel obstruction

**Complications**
Although rare in carefully selected patients, they may include:
- Vomiting (30%)
- Charcoal aspiration, especially if there is decreased mental status or seizures
- Constipation
- Charcoal bezoar formation, bowel obstruction, bowel perforation (rare)
- Corneal abrasion
- Distraction of attending staff from resuscitation and supportive care priorities.

**Technique**
- Give an initial dose of activated charcoal 50 g (adults) or 1 g/kg (children) PO
- Give repeat doses of 25 g (0.5 g/kg in children) every 2 hours
- In the intubated patient, activated charcoal is given via oro- or nasogastric tube after tube placement has been confirmed on chest x-ray
- Check for bowel sounds prior to administration of each dose
- Cease further administration if bowel sounds are inaudible
- Reconsider the indications and clinical end points for therapy every 6 hours. MDAC should rarely be required beyond 6 hours.
URINARY ALKALINISATION

Rationale
The production of an alkaline urine pH promotes the ionisation of highly acidic drugs and prevents reabsorption across the renal tubular epithelium, thus promoting excretion in the urine. For this method to be effective the drug must be filtered at the glomerulus, have a small volume of distribution and be a weak acid.

Indications
Only two drugs of significance in clinical toxicology have the required pharmacokinetic properties for this method to be of interest in management of poisoning.

- Salicylate overdose
  - Salicylates are normally eliminated by hepatic metabolism and fail to be excreted in acidic urine. In overdose, metabolism is saturated and elimination half-life greatly prolonged
  - Urinary alkalisation greatly enhances elimination and is indicated in any symptomatic patient in an effort to reduce the duration and severity of symptoms or to avoid progression to severe poisoning and the need for haemodialysis
  - Severe established salicylate toxicity indicates immediate haemodialysis rather than a trial of urinary alkalisation
  - Note: Not generally useful in chronic salicylate toxicity due to co-morbidities.

- Phenobarbitone coma
  - May be attempted in an effort to reduce duration of coma and length of stay in intensive care
  - Not first-line as MDAC is superior.

Contraindications
- Fluid overload.

Complications
- Alkalaemia (usually well-tolerated)
- Hypokalaemia
- Hypocalcaemia (not usually clinically significant).

Technique
- Correct hypokalaemia if present
- Given 1–2 mmol/kg sodium bicarbonate IV bolus
- Commence infusion of 100 mmol sodium bicarbonate in 1000 mL 5% dextrose at 250 mL/hour
- 20 mmol of potassium chloride may be added to infusion to maintain normokalaemia
Follow serum bicarbonate and potassium at least every 4 hours
- Regularly dipstick urine and aim for urinary pH >7.5
- Continue until clinical and laboratory evidence of toxicity is resolving.

**EXTRACORPOREAL TECHNIQUES OF ELIMINATION**

A number of such techniques have been used to enhance elimination of toxins including:

- **Haemodialysis**
  - Intermittent
  - Continuous
- **Haemoperfusion**
- **Plasmapheresis**
- **Exchange transfusion.**

All of the above techniques are invasive and require specialised staff, equipment and monitoring and may be associated with significant complications. For these reasons they are reserved for life-threatening poisonings where a good outcome cannot be achieved by other means, including aggressive supportive care and antidote administration.

Haemodialysis is the most frequently used of these techniques and effectively enhances elimination of any drug that is a small molecule, has a small volume of distribution, rapid redistribution from tissues and plasma, and slow endogenous elimination. Clinical situations that involve life-threatening poisoning with agents fulfilling these criteria include:

- **Toxic alcohol poisoning**
  - Methanol
  - Ethylene glycol
- **Theophylline poisoning**
- **Severe salicylate intoxication**
  - Chronic intoxication with altered mental status
  - Late-presentation acute overdose with established severe toxicity
- **Severe chronic lithium intoxication**
- **Phenobarbitone coma**
- **Metformin lactic acidosis**
- **Massive valproate overdose**
- **Massive carbamazepine overdose**
- **Potassium salt overdose with life-threatening hyperkalaemia.**

Precise clinical indications for haemodialysis in each of these important poisonings are discussed in the relevant sections of Chapter 3. The decision to dialyse should be made early as soon as the risk assessment indicates potential lethality. In general, intermittent dialysis
achieves greater clearance rates than continuous haemodialysis techniques and is preferred where available.

References

1.8 ANTIDOTES

Antidotes are drugs that correct the effects of poisoning. Only a few antidotes exist for a limited number of poisonings and many are used only extremely rarely. Specific antidotes likely to be used in clinical practice are discussed in Chapter 4 of this book.

Like all pharmaceuticals, antidotes have specific indications, contraindications, optimal administration methods, monitoring requirements, appropriate therapeutic end points and adverse effect profiles.

The decision to administer an antidote to an individual patient is based upon a risk–benefit analysis. An antidote is administered when the potential therapeutic benefit is judged to exceed the potential adverse effects, cost and resource requirements. An accurate risk assessment combined with pharmaceutical knowledge of the antidote is essential to clinical decision making.

Many antidotes are rarely prescribed, expensive and not widely stocked. Planning of stocking, storage, access, monitoring, training and protocol development are essential components of rational antidote use. It is often appropriate for stocking to be coordinated on a regional basis in association with regional policies concerning the treatment of poisoned patients. It is frequently cheaper and safer to transport an antidote to a patient rather than vice versa.

References
A medical disposition is required for all patients who present with poisoning or potential exposure to a toxic substance. Those who have deliberately self-poisoned also require psychiatric and social review. A risk-assessment-based approach to the management of acute poisoning allows early planning for appropriate medical and psychosocial disposition. Patients must be admitted to an environment capable of providing an adequate level of monitoring and supportive care and, if appropriate, where staff and resources are available to undertake decontamination, administration of antidotes or enhanced elimination techniques.

Early risk assessment in the pre-hospital setting, usually by poisons information centre staff, often allows non-intentional exposures to be observed outside of the hospital environment. For those that present to hospital, it minimises the duration and intensity of monitoring. Frequently patients can be ‘cleared’ for medical discharge directly from the emergency department immediately following assessment or following a few hours of monitoring. No arrangements for admission to hospital need be made unless unexpected signs or symptoms of toxicity develop.

At other times the risk assessment will indicate the need for ongoing observation, supportive care or the need for specific enhanced elimination techniques or antidote administration. Under these circumstances, the patient must be admitted to an environment capable of providing a level of care appropriate for the anticipated clinical course. In many hospitals, this is now the emergency observation unit rather than the general medical ward. Where ongoing airway control, ventilation or advanced haemodynamic support is required then admission to an intensive care unit is appropriate.

**EMERGENCY OBSERVATION UNITS**

Emergency observation units (EOUs) have been established in many emergency departments in Australasia and elsewhere. These units vary in capacity, design and staffing. Ideally, they are located adjacent to emergency departments, staffed by emergency physicians and provide short-term focused goal-oriented care. They have been remarkably successful in:

- Streamlining treatment in suitable conditions
- Reducing total bed days
- Increasing patient satisfaction
- Reducing inappropriate discharges and litigation.

**TOXICOLOGY PATIENTS IN THE EMERGENCY OBSERVATION UNIT**

In most hospitals where EOU's are established, the units appear to provide an ideal environment for the management of acute poisoning beyond the initial assessment and monitoring phase. Advantages of using the EOU
to admit toxicology patients include the ready availability of appropriate resources, staff and training, 24-hour availability of experienced medical staff, an open-plan environment that facilitates observation, and an emergency department ethos that is geared towards assessment and disposition. Adequate resources must be dedicated to the EOU, particularly medical, nursing, psychiatric and social services.

Ideal design features and staffing that facilitate the management of toxicology patients in the EOU include:

- Central nursing stations with clear vision of all areas
- An environment that protects from self-harm
- Secure entrances
- Dedicated areas for private interviews
- Dedicated social work, drug and alcohol, plus outpatient liaison services
- Appropriate monitors +/- telemetry
- Dedicated resuscitation equipment
- Duress alarms
- Appropriate staff, skills and equipment
- Appropriate 24/7/365 senior staff coverage
- Dedicated psychiatric services
- Nurse–patient ratios appropriate for the acuity of patients (e.g. 1:4 for monitored ‘step-down’ patients; 1:8 for non-monitored general patients).

Criteria need to be developed for admission to the EOU following acute poisoning. Such criteria might include:

- Cardiac monitoring not required (but this can be provided in some EOU’s)
- Adequate sedation in cases of delirium
- Deterioration not anticipated (based on accurate risk assessment and initial period of observation in the emergency department).

Admission of toxicology patients to the EOU helps counter several of the difficulties encountered when poisoned patients are admitted to other areas of the hospital:

- Admissions scattered all over hospital
- Less experienced nursing staff
- Poor availability of medical staff
- Frequent security incidents/absconding patients
- Most clinicians managing patients on general medical wards are junior and have no formal or informal training in clinical toxicology
- Longer admissions.

**RETRIEVAL OF THE POISONED PATIENT**

Usually the initial receiving hospital is adequately resourced to provide an acceptable level of supportive care, monitoring and therapy for the poisoned patient. If this is not the case then transfer is necessary. Risk
assessment ensures that the need to transfer is recognised early so that appropriate planning and consultation takes place in an effort to ensure as smooth a retrieval as possible. Poisoning is unusual in that transfer frequently takes place during the most severe phase of the illness.

**Resuscitation**

The need to retrieve a patient to another centre should not distract attending staff from resuscitation and supportive care priorities. Attention to airway, breathing and circulation ensure an optimum outcome in the majority of cases. Whenever possible, the patient should be stabilised before retrieval begins. Interventions such as intubation, ventilation, initial resuscitation of hypotension, cessation of seizures, assessment of blood glucose and management of hyperthermia are completed before a patient is placed in the transport vehicle, where further assessment and detailed management are often impossible. If the referring team does not possess the necessary skills or resources to complete these resuscitation and stabilisation tasks adequately, this should be communicated to the receiving and retrieval teams, so that these resources can be brought to the patient.

**Transport**

As transport usually occurs during the most severe phase of the poisoning, the patient should never be subjected to an interval of a lower level of care during the transfer. Consideration of the mode and staffing of transport takes this into account.

**Planning**

Planning is required to ensure that any potential complications are identified and managed in a proactive fashion. Thus, if coma requiring intubation and ventilation is anticipated in the next few hours (e.g. controlled-release carbamazepine), early intubation and ventilation should

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**TABLE 1.9.1 Principles of retrieval of the poisoned patient**

- Risk assessment is vital
- Identify patients who may need retrieval to another hospital as soon as possible
- Patients should only be retrieved for specific clinical indications
- Recognise that transport may occur during the worst phase of the intoxication
- Consider bringing expertise and resources to the patient, rather than vice versa
- Assess, manage and stabilise potential resuscitation and supportive care priorities prior to transfer
- Ensure that transport does not lead to an interval of lower level of care
- Transport to a centre capable of definitive care
occur prior to transfer. Similarly, if significant hypotension is expected (e.g. calcium channel blockers), then appropriate monitoring, intravenous access and resuscitation resources should be ready prior to transfer.

**Communication**

Communication is vital. Retrieval is always to a higher level of care. Thus transport must occur to a facility with appropriate resources to manage the potential complications identified by the risk assessment. For example, if haemodialysis may be required (e.g. theophylline or salicylate poisoning), the patient must be transported to a facility capable of instituting this intervention at short notice. Ideally, communication should include the team of clinicians who will ultimately manage the patient. Consultations with other specialist teams (e.g. paediatricians, intensivists or clinical toxicologists) may also occur to assist the process. This improves continuity of care and decreases the inefficiencies and errors that may be associated with multiple handovers.

**Antidotes**

If an antidote is likely to definitively treat the patient and render them stable (e.g. N-acetylcysteine; digoxin-specific antibodies), then it is preferable to transfer the antidote to the patient, start treatment, then move the patient only if necessary.

**Psychosocial assessment**

Most episodes of acute poisoning represent an exacerbation of an underlying psychosocial disorder and the final disposition of the patient is made in this context. All patients with deliberate self-poisoning should undergo psychosocial assessment prior to discharge. Ideally, this process begins before the medical treatment of the poisoning is complete so that final disposition is facilitated.

**References**


