

APPROACH TO THE POISONED PATIENT

POISONING IS A LEADING CAUSE OF DEATH IN PATIENTS UNDER 40 AND SHOULD BE A LEADING DIFFERENTIAL WHEN CARDIAC ARREST OCCURS IN A YOUNG ADULT

ACUTE POISONING IS A DYNAMIC ILLNESS → I.E. THINGS CAN CHANGE FAST

ATTEMPTS AT DECONTAMINATION SHOULD NEVER TAKE PRIORITY OVER RESUSCITATION EFFORTS

RESUSCITATION FOLLOWING PROLONGED EFFORTS IN THE ACUTELY POISONED PATIENT MAY BE ASSOCIATED WITH GOOD NEUROLOGICAL OUTCOMES

SELF POISONED PATIENTS, ONCE MEDICALLY STABLE, ALSO NEED PSYCHIATRIC AND SOCIAL INTERVENTION

The approach to the poisoned patient initially is similar to that of any critical illness, with a few caveats

RESUSCITATION:

- **AIRWAY, BREATHING AND CIRCULATION AS PER NORMAL:**
 - Altered conscious state, loss of airway reflexes and hypotension are common threats to life in the poisoned patient
- **DETECT AND CORRECT SEIZURES:**
 - GENERALISED in the toxic patient
 - IV BENZOS ARE FIRST LINE
 - Barbiturates are second line
 - Pyridoxine third line in intractable seizures due to ISONIAZID
 - Phenytoin contraindicated
 - Common causes in poisoned patients in Australia:
 - Venlafaxine
 - Bupropion
 - Tramadol
 - Amphetamines
- **DETECT AND CORRECT HYPOGLYCAEMIA:**
 - BSL ASAP
 - Easily detectable and correctable cause of neurological injury
 - In acute poisoning, hypoglycaemia associated with:
 - Insulin
 - Sulfonylureas
 - Beta-blockers
 - Valproate

- Salicylates
 - Quinine/chloroquine
- DETECT AND CORRECT HYPER/HYPOTHERMIA:
 - Hyperthermia in acute poisoning associated with poor outcome
 - If $\geq 39.5 \rightarrow$ urgent intervention
- EMERGENCY ANTIDOTE ADMINISTRATION (IF APPLICABLE)

RISK ASSESSMENT:

- SHOULD OCCUR AS SOON AS POSSIBLE
- Distinct cognitive step, quantitative
- Should take into account:
 - Agent(s) \rightarrow beware sustained release preparations
 - Dose(s)
 - Time since ingestion
 - Clinical features and progress
 - Patient factors (weight, comorbidities)
- IGNORE PATIENT HISTORY AT YOUR PERIL
- If patient has altered mental state \rightarrow alternative sources include:
 - Ambulance
 - Family
 - Count missing tablets
 - Check medical records for prior prescriptions
 - In either situation \rightarrow THINK WORST-CASE SCENARIO
- POISON'S CENTRE \rightarrow 131126

SUPPORTIVE CARE AND MONITORING:

- MOST PATIENTS CAN BE TREATED IN EITHER ED, OBSERVATION UNITS OR ICU
 - Think ICU if requirements include:
 - Airway control or ventilation
 - Prolonged or invasive haemodynamic support/monitoring
 - Haemodialysis

INVESTIGATIONS:

- TWO RECOMMENDED SCREENING TESTS FOR ACUTE POISONING:
 - 12-lead ECG
 - Readily available, non invasive
 - Assists in identifying occult conduction disturbance
 - Serum paracetamol level:
 - Deliberate self-poisoning common, up to 15% presentations in Australasia
 - Life-threatening poisoning may be occult in early stages and hepatic failure/death readily preventable with N-Acetyl Cysteine

- Non-detectable level greater than one hour post-ingestion excludes significant ingestion
 - If paracetamol poisoning is suspected after risk assessment, delay first level until four hours
- QUALITITATIVE URINE DRUG SCREENS:
 - For drugs of abuse
 - RARELY alters management of acutely poisoned patient
- USEFUL DRUG LEVELS:
 - Carbamazepine
 - **Digoxin**
 - Ethanol
 - Ethylene glycol
 - Iron
 - **Lithium**
 - Methanol
 - Methotrexate
 - **Paracetamol**
 - Phenobarbitone
 - Salicylate
 - Theophylline
 - Valproic acid

GASTROINTESTINAL DECONTAMINATION:

- The tendency throughout the recent past has been to OVERESTIMATE BENEFITS and to UNDERSTATE POTENTIAL HARMS
 - THUS IT IS NO LONGER CONSIDERED ROUTINE
- BENEFITS:
 - More benign clinical course with potentially improved outcome
- RISKS:
 - Aspiration
 - GI complications → perforation, obstruction
 - Distraction from resuscitation
- There should be reasonable grounds to believe that a significant amount of agent remains unabsorbed and is amenable to removal by the selected process
- FOUR METHODS:
 - INDUCED EMESIS
 - GASTRIC LAVAGE
 - ACTIVATED CHARCOAL
 - WHOLE BOWEL IRRIGATION
- INDUCED EMESIS:
 - With IPECAC SYRUP → plant derived emetics induces vomiting by central and peripheral means
 - Amount of toxin removed is unreliable and ↓s rapidly with time
 - Subsequent charcoal administration more difficult

- Serious complications → Mallory-Weiss tear, pneumomediastinum, gastric perforation, aspiration
- GASTRIC LAVAGE:
 - Attempts to empty stomach by sequential administration and aspiration of small volumes of fluid via NG/OG
 - All but abandoned
 - Amount of toxin removed after first hour of administration negligible
 - Aspiration, hypoxia, laryngospasm, water intoxication and hypothermia all complications
- ACTIVATED CHARCOAL → SINGLE DOSE:
 - Enormous surface area reversibly ADSORBS most ingested toxins preventing further absorption from GI tract
 - If applied widely, does not improve outcome
 - It is useful where the toxin remains in the GI tract, i.e. within the first hour of consumption
 - Major risk is aspiration → potentially fatal
 - Subsequent antidote administration rendered ineffective
 - Certain agents bind poorly:
 - HYDROCARBONS AND ALCOHOLS (inc ethanol, methanol and ethylene glycol)
 - METALS → Lithium, iron, potassium, lead, arsenic, mercury
 - CORROSIVES → i.e. acids or alkalis
 - 50g dose in adults, 1g/kg in kids
- WHOLE-BOWEL IRRIGATION:
 - Attempts to cleanse the bowel by administering large volumes of osmotically active polyethylene glycol solution
 - Reserved for overdose of ENTERICALLY COATED or SUSTAINED RELEASE PREPARATIONS
 - Useful in:
 - Iron overdose (>60mg/kg)
 - Slow release potassium chloride ingestion (>2.5mmol/kg)
 - Slow release verapamil/diltiazem ingestion
 - Symptomatic arsenic ingestion
 - Lead ingestion
 - Body packers
 - Continue irrigation until effluent clear and pro-kinetic agent to minimize vomiting and enhance gastric emptying

ENHANCED ELIMINATION:

- Aims to increase the rate of removal of an agent from the body with the aim of reducing severity and duration of intoxication
- Only useful in limited circumstances:
 - Severe toxicity
 - Poor outcome despite good care/antidote

- Slow endogenous rates of elimination
- Suitable pharmacokinetics
- FOUR METHODS:
 - MULTIPLE DOSE ACTIVATED CHARCOAL
 - URINARY ALKALINISATION
 - HAEMODIALYSIS/FILTRATION
 - CHARCOAL HAEMOPERFUSION
- MULTI-DOSE CHARCOAL:
 - Enhances elimination by:
 - Interruption of enterohepatic circulation
 - GI dialysis → passing from high concentration in bloodstream to “low” concentration in GI lumen due to binding of drug to charcoal
 - Don’t use in bowel obstruction
 - Consider in overdose of:
 - Carbamazepine → MOST COMMON, only in coma
 - Dapsone
 - Phenobarbitone
 - Quinine
 - Theophylline → don’t delay dialysis
- URINARY ALKALINISATION:
 - Promotes ionization of highly ACIDIC DRUGS and prevents reabsorption across renal tubular epithelium
 - Drug must be filtered at the glomerulus, be a weak acid and have a small volume of distribution
 - Useful in:
 - SALICYLATE OVERDOSE → normally excreted by liver, but this is overwhelmed in OD. Do not delay dialysis in severe intoxication
 - PHENOBARBITONE COMA → multi dose charcoal superior
 - Complicated by alkalaemia, ↓K, ↓Ca
 - 1-2mmol/kg NaHCO₃ bolus, then infusion of 100mmol in 1L 5% dex over four hours
- HAEMODIALYSIS:
 - Enhances elimination of any drug that has small V_d, rapid redistribution from tissues to plasma and slow endogenous elimination
 - Consider in:
 - TOXIC ALCOHOLS
 - SALICYLATE INTOXICATION
 - THEOPHYLLINE
 - LITHIUM
 - Other:
 - Phenobarb, metformin lactic acidosis, massive valproate/carbamazepine OD, K salt OD

ANTIDOTES:

DRUGS THAT CORRECT THE EFFECTS OF POISONING

DECISION TO ADMINISTER IS BASED UPON RISK/BENEFIT ASSESSMENT