# TOXICOLOGY

## ETHANOL INTOXICATION

Acute alcohol intoxication is one of the most common presentations to ED.

### Pathophysiology

Ethanol is rapidly absorbed through the gastric mucosa and reaches peak concentration within 30 minutes. It interacts with the GABA-A receptor resulting in CNS inhibition. 90% is metabolised by the liver - the rest is excreted by the kidneys and lungs.

### Clinical Features

- CNS: relaxation, euphoria/dysphoria, impaired motor function, ataxia, dysarthria, sedation, coma.
- Respiratory: respiratory depression at high doses, aspiration.
- Cardiovascular: systemic vasodilation (hypotension, tachycardia), myocardial depression at high doses.
- Gastrointestinal: nausea and vomiting.
- Metabolic: hypoglycaemia (NADH production from ethanol metabolism impairs gluconeogenesis).
- Renal: diuresis (inhibition of ADH secretion).

### Treatment

Often no management is required as the patient usually sobers within 2-4 hours. The main danger is attributing a patient's condition to alcohol when in fact they have sustained a serious head injury or are medically unwell.

General supportive measures such as airway support, oxygen and fluids should be provided as indicated. Pabrinex should be given to those with signs of or at risk of Wernicke's.

Intoxicated patients with a head injury should be assessed carefully. Those with an indication for CT head should be scanned or discussed with a senior. Those who are not immediately scanned should be admitted to ward 46 - if the patient does not sober up as expected a CT can then be arranged.

Have a low threshold for taking blood, venous gas and ECG. Bloods may provide an indication that an organic illness is present and the ECG may show signs of poisoning with other drugs. Ethanol levels can be considered to confirm alcohol has been taken but should not be relied on; it does not exclude other causes of the patient's presentation.