ASA overdose

ANESTHETIC CONSIDERATIONS:

- Avoid intubation if possible
- Aspiration risk (full stomach, decreased LOC)
- Decontamination
- Supportive management
- Noncardiogenic pulmonary edema
    - Hypovolemia
    - Electrolyte and acid-base disturbances
- Associated trauma and coingestions

ANESTHETIC GOALS:

- Supportive management of ABC's
- Prevent and treat life-threatening complications

HISTORY

- AMPLE Hx
- Sx of ASA overdose (see Pathophysiology below)

PHYSICAL

- Primary survey – ABCDE assessment
- Secondary survey – head-to-toe examination

INVESTIGATIONS

- **Labs**
    - CBC
    - Lytes, urea, Cr (reliability of renal elimination)
    - Liver enzymes, albumin, glucose
    - INR, PTT
    - ABG (respiratory/metabolic acidosis, wide anion gap)
    - Blood and urine for toxicology (ASA and other)
- **Imaging**
    - EKG (arrhythmias)
    - CXR (aspiration, noncardiogenic pulmonary edema)
    - AXR (opaque substances)

OPTIMIZATION

- **Consult Toxicology, Nephrology, ICU**
- **Decontamination**
    - **Minimize ASA absorption**
        - Charcoal:
            - 1 g/kg up to 50 g (q2-4h until passes charcoal stool)
            - Potential complications: aspiration, ileus
        - Gastric lavage:
            - No evidence that lavage changes outcome
            - Suggest limit use to < 1 hour since ingestion & lethal drug amount
        - Don't give emetics
    - **Alter ASA metabolism**
    - **Alter ASA distribution**
        - Reduce tissue uptake/sequestration – sodium bicarbonate for target pH 7.5-7.55
    - **Promote ASA elimination**
        - Urinary alkalinization – sodium bicarbonate for target urine pH >7.5 in order to promote renal clearance of salicylates
        - Hemodialysis – indicated for potentially lethal salicylic acid concentrations (>100 mg/dL), refractory acidosis, seizures, coma, renal failure, volume overload
- **Supportive management**
    - Avoid intubation
        - These patients hyperventilate and maintain a respiratory alkalosis on their own
        - Difficult to maintain degree of minute ventilation on mechanical ventilation
    - Volume resuscitation
        - Significant fluid losses from ↑metabolic rate, pyrexia, tachypnea, and vomiting
    - Supplemental glucose if altered LOC
        - Salicylate toxicity results in ↓CNS glucose levels despite normal peripheral glucose levels
    - Tx hypokalemia

ANESTHETIC OPTIONS

- **None**

- Ideally postpone surgery until resuscitated, stabilized, and acute toxicity resolved
- **Local/Regional**
  - Depending on procedure and LOC
- **General**
  - Avoid intubation if possible

ANESTHETIC SETUP
- **Drugs**
  - Standard emergency drugs
  - Sodium bicarbonate
- **Equipment**
  - Standard CAS monitors
  - Temperature probe
  - ICU ventilator

MANAGEMENT OF ANESTHESIA
- **Induction**
  - RSI
- **Maintenance**
  - Avoid hypercarbia, will require high minute ventilation
  - ARDSnet ventilation if noncardiogenic pulmonary edema
  - Maintain adequate intravascular volume
  - ?Cooling for hyperpyrexia (dDx MH, NMS, SS, etc)
- **Emergence**
  - Transfer to ICU ventilated vs extubate fully awake

DISPOSITION & MONITORING
- Transfer to ICU for ongoing supportive care

COMPLICATIONS
- Aspiration
- Hypovolemic shock
- Renal failure
- Hepatic failure
- Coma

PATHOPHYSIOLOGY
- ASA directly stimulates respiratory centre in medulla
  - Hyperventilation → ***respiratory alkalosis***
- At high levels salicylates uncouple oxidative phosphorylation → ↓ATP → anaerobic metabolism, ↑O2 consumption, ↑CO2 production, ↑heat production
  - Accumulation of organic acids (pyruvate, lactate, acetoacetate) → ***anion gap metabolic acidosis***
  - AGMA increases levels of H-salicylate (basic, non-ionized form of salicylate) which can readily cross membranes (including BBB → ↑neurotoxicity)
    - Alkalemic environment traps salicylate in blood, preventing CNS toxicity
  - ↑Metabolic rate, pyrexia, tachypnea, and vomiting → fluid loss, dehydration
    - Hyponatremia may be absent due to significant water losses
    - Marked hypokalemia present
- Pharmacokinetics
  - Absorption
    - Delayed absorption and prolonged toxicity may occur with enteric-coated or sustained-release formulations
  - Distribution
    - Metabolic acidosis favors lipid-soluble nonionized faction of drug → ↑passage of drug into tissues and brain → ↑toxicity
  - Elimination
    - Respiratory alkalosis promotes renal excretion (↑water-soluble ionized fraction of salicylic acid)
- Clinical presentation
  - VS – fever, tachypnea, tachycardia
  - CNS – tinnitus, N/V, vertigo, obtundation, seizures, low CSF glucose
  - CVS – arrhythmias, shock
  - RESP – tachypnea/respiratory alkalosis, ALI/noncardiogenic pulmonary edema
  - GI – abdominal pain, N/V, diarrhea, hepatic dysfunction
  - Metab – hypoglycemia, metabolic acidosis
  - Heme – coagulopathy
- Diagnosis
  - 6-hour post-ingestion level (in acute, single ingestion only) can be applied to the Done nomogram to assist in predicting level of toxicity
- Management
  - See 'Optimization' above

REFERENCES

- Anesthesia and Co-Existing Disease p.550-551
- Up to Date