Acid-base balance

26th May 2011

Metabolic acidosis
- Deficit of base or excess of acid
- Causes:
  - Increase in fixed acids: ketone bodies formation as in diabetes, starvation
  - Metabolites retention in renal failure
  - Anaerobic tissue metabolism
  - Loss of bases: diarrhea, ulcerative colitis, high intestinal fistula, ureterosigmoidostomy
- CF: Rapid, deep, noisy breathing
  Due to resp centre stimulation by low pH
  Urine is acidic

T/t:
- Restoration of adequate tissue perfusion
- Treat the cause
  - Na bicarbonate infusion—8.4% 50-60 mmol

Metabolic acidosis.....

Respiratory acidosis
- PCO2 is increased
- Due to impaired alveolar ventilation
- Cause: anesthetised patients, thoracic or abdominal surgery.

Metabolic alkalosis
- Condition of base excess or acid deficit
- Causes:
  - Excessive ingestion of alkali like antacids
  - Exogenous HCO3 administration
  - Acid loss like in vomiting, NG aspiration
  - Hypokalemic hyperchloremic metabolic alkalosis
  - Corticosteroids excess, cushing’s syndrome
  - Hyperaldosteronism
- CF: Cheyne-Stokes respiration with periods of apnea,
  ketony.

Treatment:
- Replacement of fluid—NaCl
- Hypokalemia—KCl supplement
- Treat underlying cause
- Severe alkalosis: acidic solution infusion eg: HCl, NH4Cl

Metabolic alkalosis.....
Respiratory alkalosis

- PCO2 decreased
- Cause:
  - Excessive pulmonary ventilation
  - Hyperventilation—high altitude, hyperpyrexia

T/t: correct underlying cause.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Initial change</th>
<th>Compensatory response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Metabolic acidosis</td>
<td>HCO3 decrease</td>
<td>PCO2 decrease</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>HCO3 increase</td>
<td>PCO2 increase</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>PCO2 increase</td>
<td>HCO3 increase</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>PCO2 decrease</td>
<td>HCO3 decrease</td>
</tr>
</tbody>
</table>

**Defn**

- A systemic state of low tissue perfusion which is inadequate for normal cellular respiration.
- One of the most common cause of death among surgical patients.
- Death—early: profound state of shock delayed: consequences of organ ischemia & reperfusion injury

**Pathophysiology**

1. Cellular:
   - Less O2 & glucose → aerobic to anaerobic metabolism
     - Lactic acid production
     - Systemic metabolic acidosis
   - Glucose pool exhausted → anaerobic respiration ceases
   - Failure of Na-K pump
   - Autodigested enzymes released from lysozymes
   - Cell lysis, release of intracellular contents into circulation.

2. Microvascular:
   - tissue ischemia
   - Activation of immune & coagulation system, complement activation, neutrophil priming
   - Capillary endothelial injury
   - Leaking through endothelial cells
   - Tissue edema
   - Further aggravation of cellular hypoxia
Pathophysiology...

3. Systemic:
   - Cardiovascular: tachycardia & vasoconstriction
   - Respiratory: tachypnea, increased minute volume
     Compensatory respiratory alkalosis
   - Renal: decreased perfusion pressure—which decreased
     glomerular pressure—which decreased
     urine output—RAA activation—vasoconstriction, Na & H2O reabsorption
   - Endocrine: adrenaline, RAA, ADH, Cortisol

Ischemia-reperfusion syndrome

- Tissue hypoxia, cellular inflammation—Aerobic metabolism—acid, K, toxic metabolites accumulation.
- Once normal circulation restored—metabolites released into circulation—direct myocardial depression, vascular dilatation, endothelial injury, acute renal injury, acute lung injury, multiorgan failure & death.
- Thus best way—reduce extent & duration of hyperperfusion.

Classification

- Hypovolemic
- Cardiogenic
- Obstructive
- Distributive
- Endocrine

Hypovolemic shock

- Most common
- Reduced circulatory volume
- Causes: hemorrhagic, poor fluid intake, excessive fluid loss, 3rd space loss

Cardiogenic shock

- Failure of heart to pump blood to the tissues.
- Causes: MI, arrhythmia, VHD, cardiomyopathy, cardiac injury, myocardial depression like in sepsis or drug.

Obstructive shock

- Mechanical obstruction of cardiac filling—reduction of preload—cardiac output.
- Causes: cardiac tamponade, tension pneumothorax, pulmonary embolism, air embolism,
Distributive shock

- Vascular dilatation with hypotension, low systemic vascular resistance, inadequate afterload.
- High cardiac output
- Causes: septic shock, anaphylaxis, spinal cord injury (neurogenic shock)

Endocrine shock

- Represent combination of hypovolemic, cardiogenic & distributive shock
- Causes: hypo/hyperthyroidism, adrenal insufficiency

Severity of shock

- Compensated shock:
  - Blood flow to kidney, brain, lungs maintained but reduced to skin, muscle, intestine.
  - Occult hypoperfusion if prolonged may lead to MOF.
- Decompensated:
  - If further loss of circulating volume persists.
  - Blood loss > 30-40 % volume is decompensated.
  - Mild, moderate, severe shock