Disorders of Acid Base Balance.

Meds. 4  2010/ 2011
Acid Base Disorders

- Physiology
- Pathophysiology
- Classification of types
- Aetiology
- Case Studies
- Lab. Aspects
Introduction

- Metabolism generates 40—80 mmol of Hydrogen ions 24 h
- pH retained within 35—45 nmol/l
- Ph 7.35---7.46
- Lungs and Kidneys
- Excretion
- Control
- Compensation
COMPENSATION

LUNGS

CO₂ + H₂O

RESPIRATORY Component

Rapid Change Min s/ Hours

KIDNEYS

H⁺ + HCO⁻₃

METABOLIC Component

Slow Change Days / Weeks
Recovery of Filtered Bicarbonate in Kidneys
Generation of Additional Bicarbonate in Kidneys
Aside Cushing's Conns Syndrome

- Excess mineralocorticoids
- Increased Na reabsorption for K
- H excreted to maintain electroneutrality
- Tendency towards metabolic alkalosis
Red Blood Cell Chloride Shift
DISORDERS OF ACID BASE STATUS

- RESPIRATORY
  - ACIDOSIS: pCO2 ↑
  - ALKALOSIS: pCO2 ↓

- METABOLIC
  - ACIDOSIS: BICARB. ↓
  - ALKALOSIS: BICARB. ↑
Respiratory
Metabolic

- Impaired H\(^+\) excretion
- Increased H\(^+\) production or ingestion
- Loss of HCO\(_3^-\)

ACIDOSIS

ALKALOSIS

- Loss of H\(^+\) in vomit
- Alkali ingestion
- Potassium deficiency

Dr. John O' Mullane
Respiratory Acidosis

- Acute (mins) or Chronic (hours)
- Asthma
- Choking
- Bronchopneumonia
- Renal Compensation 3 days min.
- Note in stable Bronchitis / Emphysema Renal compensation which takes time may give pH within normal with elevated pCO2
<table>
<thead>
<tr>
<th>Causes of respiratory acidosis</th>
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</thead>
<tbody>
<tr>
<td><strong>Airway obstruction</strong></td>
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<tr>
<td>chronic obstructive airway disease</td>
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<tr>
<td>(e.g. bronchitis, emphysema)</td>
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<td>bronchospasm, e.g. in asthma</td>
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<td>aspiration</td>
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<tr>
<td><strong>Depression of respiratory centre</strong></td>
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<tr>
<td>anaesthetics</td>
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<td>sedatives</td>
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<tr>
<td>cerebral trauma</td>
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<tr>
<td>tumours</td>
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<tr>
<td><strong>Neuromuscular disease</strong></td>
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<tr>
<td>poliomyelitis</td>
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<tr>
<td>Guillain-Barré syndrome</td>
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<tr>
<td>motor neuron disease</td>
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<tr>
<td>tetanus, botulism</td>
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<tr>
<td>neurotoxins, curare</td>
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<tr>
<td><strong>Pulmonary disease</strong></td>
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<tr>
<td>pulmonary fibrosis</td>
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<tr>
<td>severe pneumonia</td>
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<tr>
<td>respiratory distress syndrome</td>
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<tr>
<td><strong>Extrapulmonary thoracic disease</strong></td>
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<tr>
<td>flail chest</td>
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<tr>
<td>severe kyphoscoliosis</td>
</tr>
</tbody>
</table>
Respiratory Alkalosis

- Hysteria
- Mechanical Ventilation
- Raised Intracranial Pressure and/or Hypoxia
- Renal Compensation takes time
Causes of respiratory alkalosis

Hypoxia
- high altitude
- severe anaemia
- pulmonary disease

Increased respiratory drive
- respiratory stimulants, e.g. salicylates
- cerebral disturbances, e.g. trauma,
- infection and tumours
- hepatic failure
- Gram-negative septicaemia
- primary hyperventilation syndrome
- voluntary hyperventilation

Pulmonary disease
- pulmonary oedema
- pulmonary embolism

Mechanical overventilation
Metabolic Acidosis Causes

- Increased Hydrogen ion production
- Impaired excretion
- Bicarb. loss from GIT - fistulae
- Renal Disease RTA
- DM
- Lactate Acidosis
- Certain Poisons ; Salicylate, MeOH, Ethylene Glycol
Clinical Effects of Metabolic Acidosis

- Compensated Hyperventilation
- Kussmaul Breathing
- Neuromuscular Irritability
- Possibility of Hyperkalaemia
- Cardiac Arrhythmias  Risk of Arrest
Metabolic Acidosis

Causes of non-respiratory acidosis

Increased H^+ formation
- ketoacidosis (usually diabetic, also alcoholic)
- lactic acidosis
- poisoning, e.g. ethanol, methanol, ethylene glycol and salicylate
- inherited organic acidoses

Acid ingestion
- acid poisoning
- excessive parenteral administration of amino acids, e.g. arginine, lysine and histidine

Decreased H^+ excretion
- renal tubular acidoses
- generalized renal failure
- carbonate dehydratase inhibitors

Loss of bicarbonate
- diarrhoea
- pancreatic, intestinal and biliary fistulae or drainage
Causes of lactic acidosis

tissue hypoxia:
  decreased perfusion
  reduced arterial $Po_2$

drugs, etc.:
  ethanol, methanol
  phenformin
  fructose, sorbitol

congenital:
  glucose 6-phosphatase deficiency
  other inherited diseases with defective
gluconeogenesis or pyruvate oxidation
Metabolic Alkalosis

- Loss of Hydrogen Ions – Vomiting
- Ingestion of Alkali
- Potassium Deficiency: H ions retained intracellularly or excreted in urine in exchange for Sodium
Clinical Effects of Metabolic Alkalosis

- Hypoventilation
- Tetany
- Ca binds Albumin as pH ↑
Metabolic Alkalosis

Causes of non-respiratory alkalosis

Loss of unbuffered hydrogen ion

gastrointestinal:
gastric aspiration
vomiting with pyloric stenosis
congenital chloride-losing diarrhoea

renal:
mineralocorticoid excess:
Cushing’s syndrome
Conn’s syndrome
drugs with mineralocorticoid activity,
e.g. carbenoxolone
diuretic therapy (not K⁺-sparring)
rapid correction of chronically raised $P_{CO_2}$
potassium depletion

Administration of alkali

inappropriate treatment of acidotic states
chronic alkali ingestion
Mixed Disorders

- Bronchitis and Renal Dysfunction
- COAD with Potassium depletion
- Salicylate OD Respir. Alk. + Metabolic Acidosis
- Interpretation must be done with extreme caution.
Lab. Measurements

- Anaerobic sample in syringe no air
- Heparinised
- On ice
- pH, pCO2, pO2, Bicarb. Derived from Henderson Hasselbalch Equation
- pH Defines disorder
- pCO2 Respiratory
- Bicarb Metabolic
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<thead>
<tr>
<th></th>
<th>Acidosis</th>
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<th>Alkalosis</th>
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<tbody>
<tr>
<td></td>
<td>Non-respiratory</td>
<td>Respiratory</td>
<td>Non-respiratory</td>
<td>Respiratory</td>
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<tr>
<td></td>
<td>acute</td>
<td>chronic</td>
<td>acute</td>
<td>chronic</td>
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<tr>
<td>$[\text{H}^+]$</td>
<td>$\uparrow$</td>
<td>$\uparrow$</td>
<td>slight $\uparrow$ or high-normal</td>
<td>$\downarrow$</td>
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<td>pH</td>
<td>$\downarrow$</td>
<td>$\downarrow$</td>
<td>slight $\downarrow$ or low-normal</td>
<td>$\uparrow$</td>
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<tr>
<td>$P_{\text{CO}_2}$</td>
<td>$\downarrow$</td>
<td>$\uparrow$</td>
<td>$\uparrow$</td>
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<tr>
<td>$[\text{HCO}_3^-]$</td>
<td>$\downarrow \downarrow$</td>
<td>slight $\uparrow$</td>
<td>$\uparrow$</td>
<td>slight $\downarrow$</td>
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<tr>
<td>Cause</td>
<td>Mechanism</td>
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<td>------------------------------------------------</td>
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<td>Low inspired oxygen</td>
<td>low alveolar $P_{O_2}$</td>
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<td>low barometric pressure</td>
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<td>low % oxygen in inspired air</td>
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<tr>
<td>Alveolar hypoventilation</td>
<td>low alveolar $P_{O_2}$</td>
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<td>respiratory depression</td>
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<tr>
<td>neuromuscular disease</td>
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<tr>
<td>Venous-to-arterial shunt</td>
<td>admixture of arterial blood (high $P_{O_2}$) with venous blood (low $P_{O_2}$)</td>
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<tr>
<td>cyanotic congenital heart disease</td>
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<tr>
<td>Impaired diffusion</td>
<td>inadequate arterial oxygenation despite normal alveolar $P_{O_2}$</td>
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<tr>
<td>pulmonary fibrosis</td>
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<tr>
<td>Ventilation/perfusion imbalance</td>
<td>blood perfuses non-aerated parts of lung and is not oxygenated</td>
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<tr>
<td>chronic obstructive airways disease</td>
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<td>Increase</td>
<td>Decrease</td>
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<td>$P_{CO_2}$</td>
<td>$P_{O_2}$</td>
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<tr>
<td>peripheral vasodilation</td>
<td>pulmonary and retinal fibrosis (only with prolonged use of high inspiratory $P_{O_2}$, particularly in infants)</td>
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<tr>
<td>headache</td>
<td>breathlessness</td>
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<td>bounding pulse</td>
<td>cyanosis</td>
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<td>papilloedema</td>
<td>drowsiness, confusion and coma</td>
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<td>flapping tremor</td>
<td>pulmonary hypotension (in chronic hypoxaemia)</td>
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<td>drowsiness, coma</td>
<td>hypoventilation</td>
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<td>late signs</td>
<td>paraesthesiae</td>
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<td>dizziness</td>
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<td>muscle cramps</td>
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<td>headache</td>
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<td></td>
<td>tetany</td>
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<td>hyperventilation</td>
<td>hypoventilation</td>
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<td>increased catecholamine release</td>
<td>paraesthesiae</td>
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<tr>
<td>hyperkalaemia</td>
<td>muscle cramps</td>
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<td>decreased myocardial contractility</td>
<td>dizziness</td>
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<td>CNS depression</td>
<td>headache</td>
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<td>tetany</td>
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<td>drowsiness, confusion and coma</td>
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<td>severe acidosis only</td>
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</table>

**Fig. 3.4** Effects of increased or decreased values of $P_{CO_2}$, $P_{O_2}$ and $[H^+]$ in the blood. Paraesthesiae, dizziness, muscle cramps and tetany are related to changes in the blood.