/ABG

This page focuses on providing some possible **causes** for the various **disturbances** that may be seen on an ABG. Although not an exhaustive list, it attempts to outline the **main headings** for possible pathology.

It covers acid-base disturbance, respiratory failure, and a small summary for some other derangements.

Causes of disturbance

Respiratory acidosis[1]

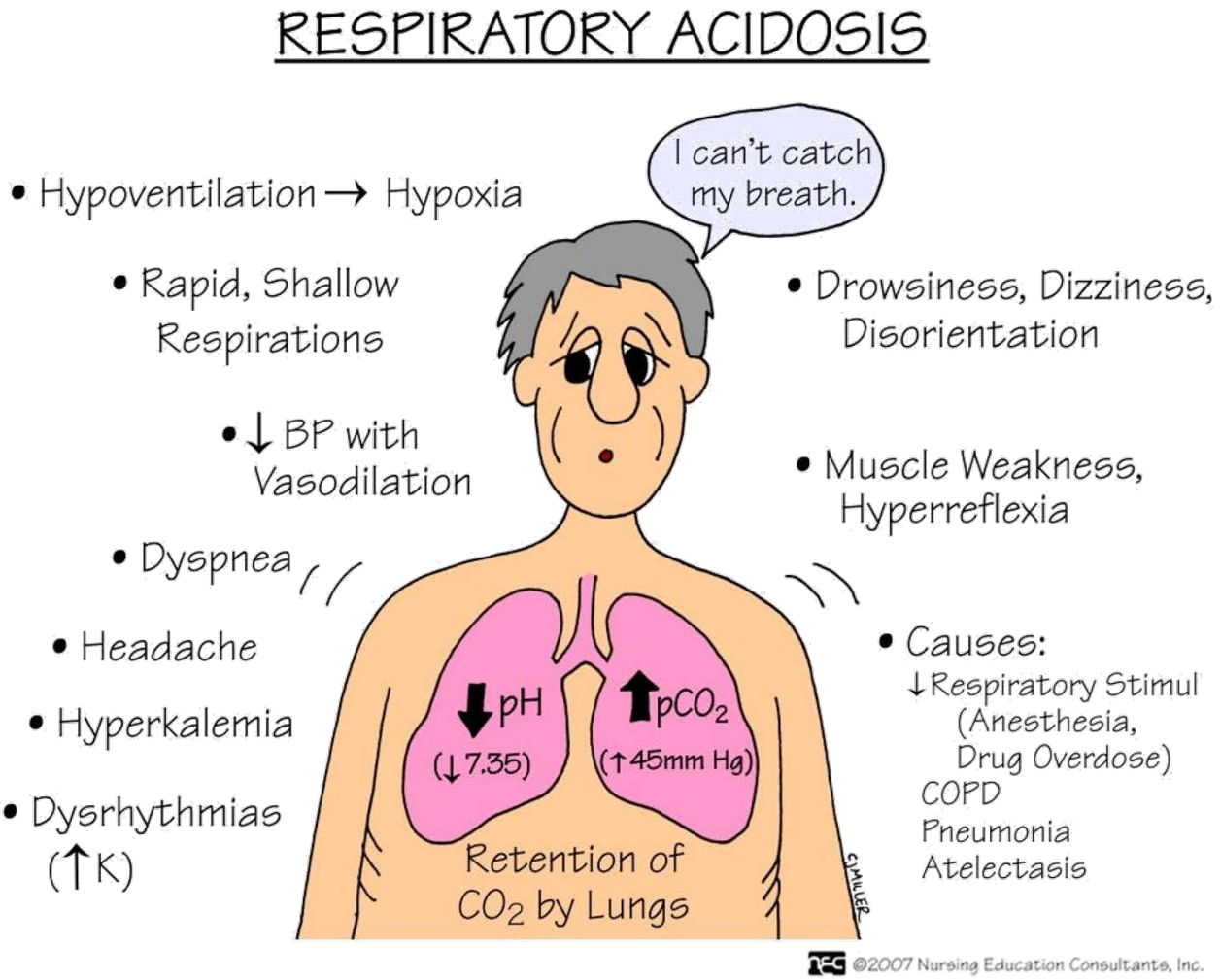
Respiratory acidosis is caused by **inadequate alveolar ventilation** leading to CO_2 **retention**.

Ventilation rate = tidal volume * respiratory rate

Therefore anything that affects **tidal volume** or **respiratory rate**, may affect the amount of CO_2 retained.

Selected etiologies of respiratory acidosis:

- Airway obstruction
    - Upper
    - Lower
        - COPD; Asthma (near-fatal); Other obstructive lung disease
- CNS depression: Opiates; Head trauma
- Sleep disordered breathing (OSA or OHS)
- Neuromuscular impairment/mechanical lung dysfunction
    - Guillain–Barré (paralysis leads to an inability to adequately ventilate); Obesity; Myasthenia gravis; deformity such as severe scoliosis.
- Increased CO2 production: rigors; seizures; malignant hyperthermia
- Iatrogenic: incorrect mechanical ventilation settings

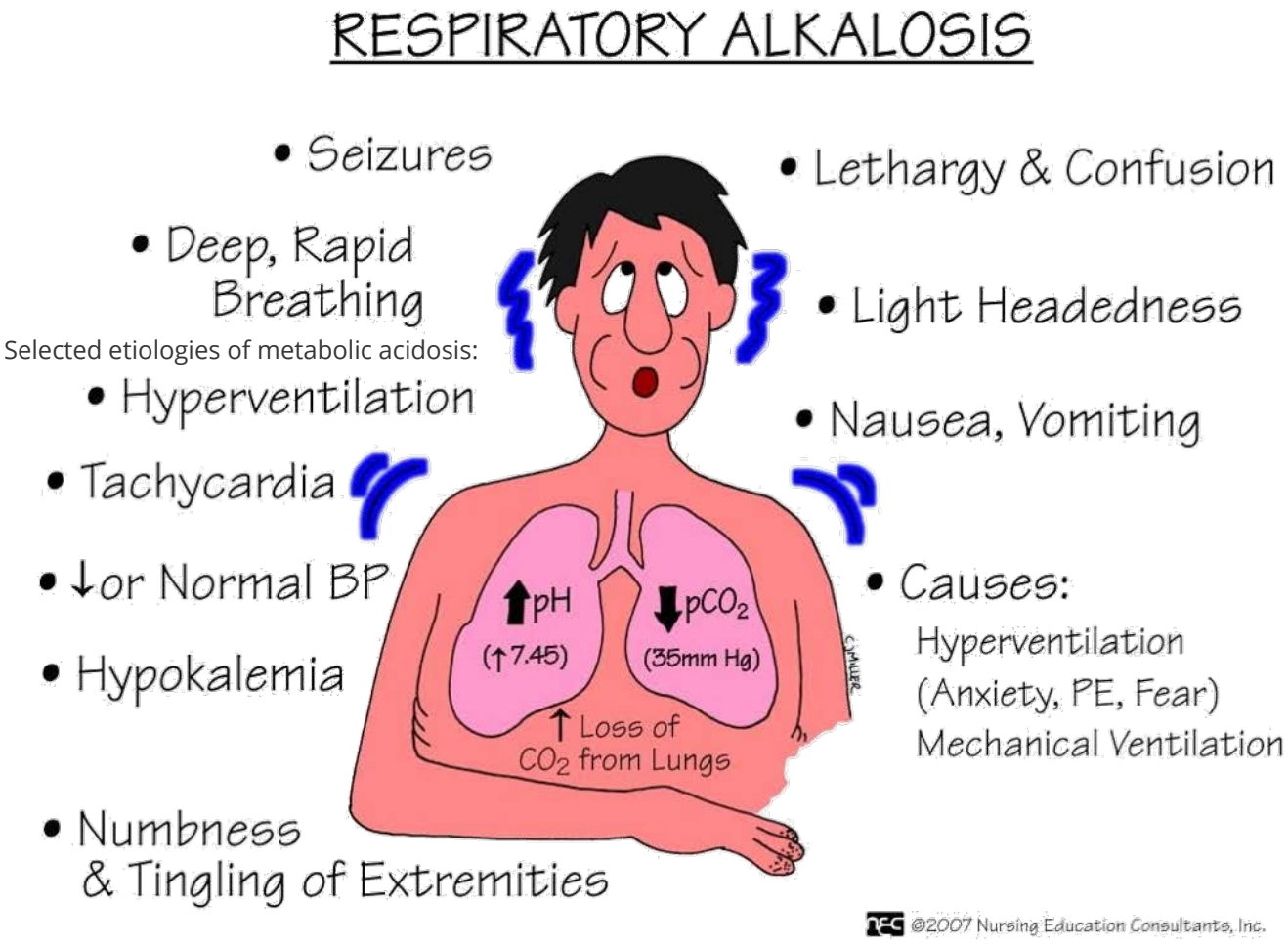


Respiratory alkalosis[1]

Respiratory alkalosis is caused by **excessive alveolar ventilation** (hyperventilation) resulting in more CO_2 than normal being **exhaled**. Often this is related to the fact that CO_2 is more easily exchanged than O_2, therefore the body may still be able to exhale excessive amounts of CO_2, even when it is **struggling** to **maintain** a normal P_aO_2. As a result, P_aCO_2 is **reduced** and **pH increases** causing alkalaemia.

Selected etiologies of respiratory alkalosis:

- CNS stimulation: anxiety (panic attack), fever, pain, fear, CVA, cerebral oedema, brain trauma, brain tumour, CNS infection – causing increased respiratory rate
- Hypoxia: lung disease, asthma (moderate/severe - hyperventilating), profound anaemia, low FiO2 – resulting in increased alveolar ventilation in an attempt to compensate
- Stimulation of chest receptors: pulmonary oedema, pleural effusion, pneumonia, pneumothorax, pulmonary embolus
- Drugs/hormones: salicylates, catecholamines, medroxyprogesterone, progestins
- Conditions: Pregnancy, liver disease, sepsis, hyperthyroidism
- Iatrogenic (excessive mechanical ventilation)



Metabolic acidosis[1]

Metabolic acidosis can occur as a result of either:

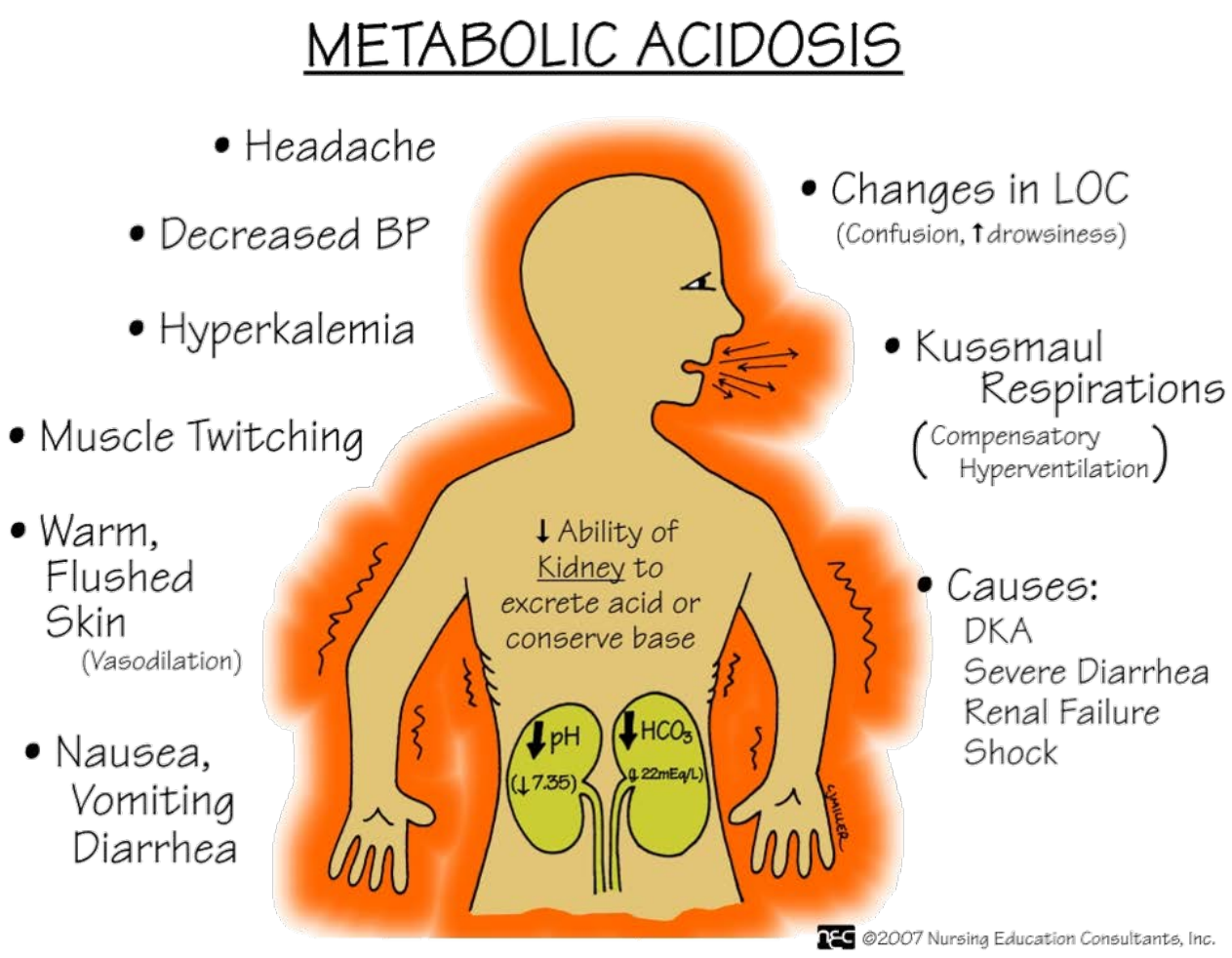
- **Increased H^+ production or ingestion**
    - HCO_3^- concentrations decrease by acting as a buffer. The HCO_3^- is consumed by the H^+ - to produce CO_2 and H_2O - resulting in a high pH.
- **GI or renal HCO_3^- loss**
    - Where a decrease in HCO_3^- is the primary pathology, causing the acidaemia.

Selected etiologies of metabolic acidosis:

- **Increased H^+ production or ingestion: (MUDPILES acronym)**
    - Methanol intoxication
    - Uremia
    - Diabetic ketoacidosis (common), alcoholic ketoacidosis, starvation ketoacidosis
    - Paraldehyde toxicity/propylene glycol
    - Isoniazid/iron
    - Lactic acidosis (metformin) (common)
        - Due to either tissue ischemia or altered cellular metabolism
    - Ethanol or ethylene glycol intoxication
    - Salicylate (aspirin) intoxication

- **HCO_3^- loss**
    - GI loss
        - Diarrhea, ileostomy, proximal colostomy, ureteral diversion
    - Renal loss
        - Proximal renal tube acidosis
        - Carbonic anhydrase inhibitor (acetazolamide)
    - Renal tubular disease (dysfunction)
        - Acute tubular necrosis
        - Chronic renal disease
        - Distal renal tube acidosis
        - Aldosterone inhibitors or absence (+Addison's disease)
        - NaCl infusion, TPN, NH_4^+ administration

Determining which one of these main headings is a fault, can be done through the use of the 'anion gap' calculation, discussed in the 'extras' page.

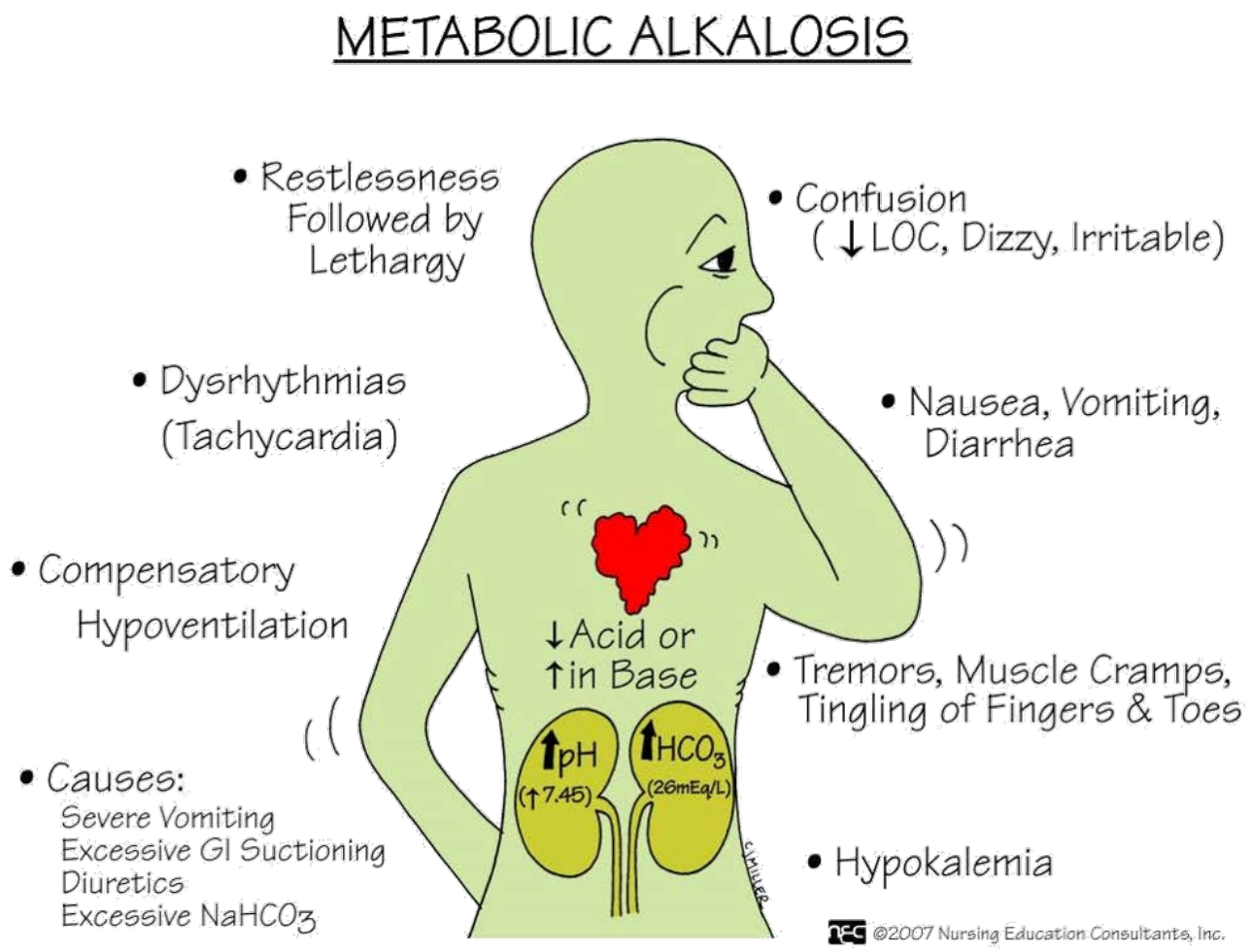


Metabolic alkalosis[1]

Metabolic alkalosis may occur as a result of **decreased hydrogen ion** concentration (by either the GI or renal system), leading to **increased bicarbonate** (as the bicarbonate buffer equation shifts to the right, to produce more H^+ and HCO_3^-), or alternatively a direct result of increased bicarbonate concentrations.

Selected causes of metabolic alkalosis

- GI loss of H^+
- Vomiting / diarrhoea (with Cl- rich fluid)
- Renal loss H^+
    - Loop and thiazide diuretics
    - Oedematous states (heart failure, cirrhosis, nephrotic syndrome)
    - Hyperaldosteronism (+Conn's syndrome),
    - Exogenous steroids
    - Severe hypokalemia (H^+/K^+ exchange)
- Burns
- Iatrogenic: alkali/bicarbonate administration (milk alkali syndrome)



Selected mixed and compensated acid-base disturbances

It must also be kept in mind that all of these conditions may occur in **simultaneously**, giving either **mixed** disorder (whereby two conditions act on the pH in the same direction), or **compensated** disorder (where the two conditions act in different directions on the pH).

A specific example a of a mixed disorder is **cardiac arrest**, whereby there is **respiratory acidosis** from respiratory arrest, and also **metabolic acidosis** from increased lactate from hypoperfusion.

An important example of a compensated disorder is **ketoacidosis with vomiting**, where there is a **metabolic acidosis** caused by increased ketoacids, as well as a **metabolic alkalosis** caused by the vomiting and loss of gastric acid.

Respiratory failure[2]

Respiratory failure can be split into Type 1 or Type 2 respiratory failure, depending on the value of P_aCO_2.

Type 1

Type 1 respiratory failure is caused by pathological processes which **reduces** the ability of the lungs to **exchange oxygen**, **without changing** the ability to **excrete CO_2**, due to the different shape of the CO_2 and O_2 **dissociation** curves.

It involves **hypoxia** (P_aO_2 <8 kP_a) with **normocapnia** (P_aCO_2 <6.0 kP_a).

It occurs as a result of **ventilation/perfusion mismatch**; where the volume of air flowing in and out of the lungs is not matched with the flow of blood to the lung tissue.

This may be due to either a **reduction in ventilation**, or a **reduction in perfusion**.

Examples of causes of type 1 respiratory failure are pulmonary embolus (reduced perfusion), pulmonary fibrosis, pneumonia, asthma/COPD, and pulmonary oedema (reduced ventilation). These may all further develop into type 2 respiratory failure.

Type 2

Type 2 respiratory failure involves **hypoxia** (P_aO_2 <8 kP_a) with **hypercapnia** (P_aCO_2 >6.0 kP_a).

Caused by a pathological process which affects the **ability** to **both exchange oxygen and excrete CO_2**. It is due to **inadequate alveolar ventilation.**

Examples of causes:

- Pulmonary problems e.g. COPD/asthma, pulmonary oedema, pneumonia
- Mechanical problems e.g. Chest wall trauma/deformity, muscular dystrophies, motor neurone disease, myasthenia gravis, Guillain–Barré
- Central problems (reduced breathing effort) e.g. Opiate overdose, acute CNS disease

Chronic type 2 respiratory failure, such as in COPD, must be managed carefully. Referred to as **CO_2 retainers,** patients rely on their **hypoxic drive** to maintain ventilation, not on P_aCO_2, therefore when exposed to **higher levels** of O_2 - as is often done when they present to A+E with increased breathlessness and low pulse oximeter readings - leads to a **decrease in respiratory drive,** and further alveolar hypoventilation, leading to extreme hypercapnia and **acidosis**. Therefore, only controlled methods of ventilation such as a **Venturi** mask should be used in these patients.

Common ABG patterns[3]

- **Hyperventilation:**
  - ↑pH ↓P_aCO_2 ~HCO_3^- ↑P_aO_2
  - Respiratory alkalosis
- **Chronic COPD:**
  - ~pH ↑P_aCO_2 ↑HCO_3^- ↓P_aO_2
  - Type 2 respiratory failure; Fully compensated respiratory acidosis
- **Acute COPD exacerbation:**
  - ↓pH ↑↑P_aCO_2 ↑HCO_3^- ↓↓P_aO_2
  - Type 2 respiratory failure; Partially compensated respiratory acidosis
- **Asthma exacerbation (life-threatening):**
  - ↑pH ↓P_aCO_2 ~HCO_3^- ~/↓P_aO_2
  - Respiratory alkalosis (wheeze → anxiety → increased respiration rate → hyperventilation → decreased P_aCO_2; on a background of increased airway resistance → decreased or normal P_aO_2, despite the hyperventilation)
- **Decreased respiratory drive:** (near-fatal e.g. exhausted asthmatic exacerbation patient, opiate overdose)
  - ↓pH ↑P_aCO_2 ~HCO_3^- ↓P_aO_2
  - Type 2 respiratory failure; Respiratory acidosis
- **Pulmonary embolism** (may vary depending on RR):
  - ↑pH ↓P_aCO_2 ~HCO_3^- ↓P_aO_2
  - Type 1 respiratory failure; Respiratory alkalosis (hyperventilation causes hypocapnia, but is unable to correct hypoxia due to VQ mismatch).
- Other important presentations include **heart failure, acute pulmonary oedema, and diabetic ketoacidosis.**

Other disturbances[2]

Lactate

- A raised lactate (lactic acid) can be caused by any anaerobic process.
- Causes of lactic acidosis:
    - Hypoxic: Increased production of lactate e.g. DKA, starvation, cardiovascular/respiratory failure, severe sepsis
    - Non-hypoxic: A failure to break down lactate e.g. secondary to metformin or poisoning

Haemoglobin (Hb)

- Haemoglobin acts as a guide but is often inaccurate. Lab samples should be sent to verify results.

Glucose

- Especially relevant in patients with decreased consciousness or seizures; or known/suspected diabetes.
- Glucose may also be disturbed in patients with sepsis.

Carbon monoxide (CO)

- >10%: indicates poisoning, commonly from poorly ventilated boilers or old heating systems.
- 10-20%: patient's may experience symptoms such as nausea, headache, vomiting, and dizziness.
- At higher levels: arrhythmias, cardiac ischaemia, respiratory failure, and seizures.

Methaemoglobin (metHb)

- Methaemoglobin is a form of haemoglobin that contains the ferric [Fe_3^+] form of iron.
- Levels of >2% are abnormal.
- Symptoms include shortness of breath, cyanosis, altered mental state, headache, fatigue.
- It may be caused by errors of metabolism or by exposure to toxins or certain medications such as nitrates.

References

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