UNDERSTANDING BLOOD GAS

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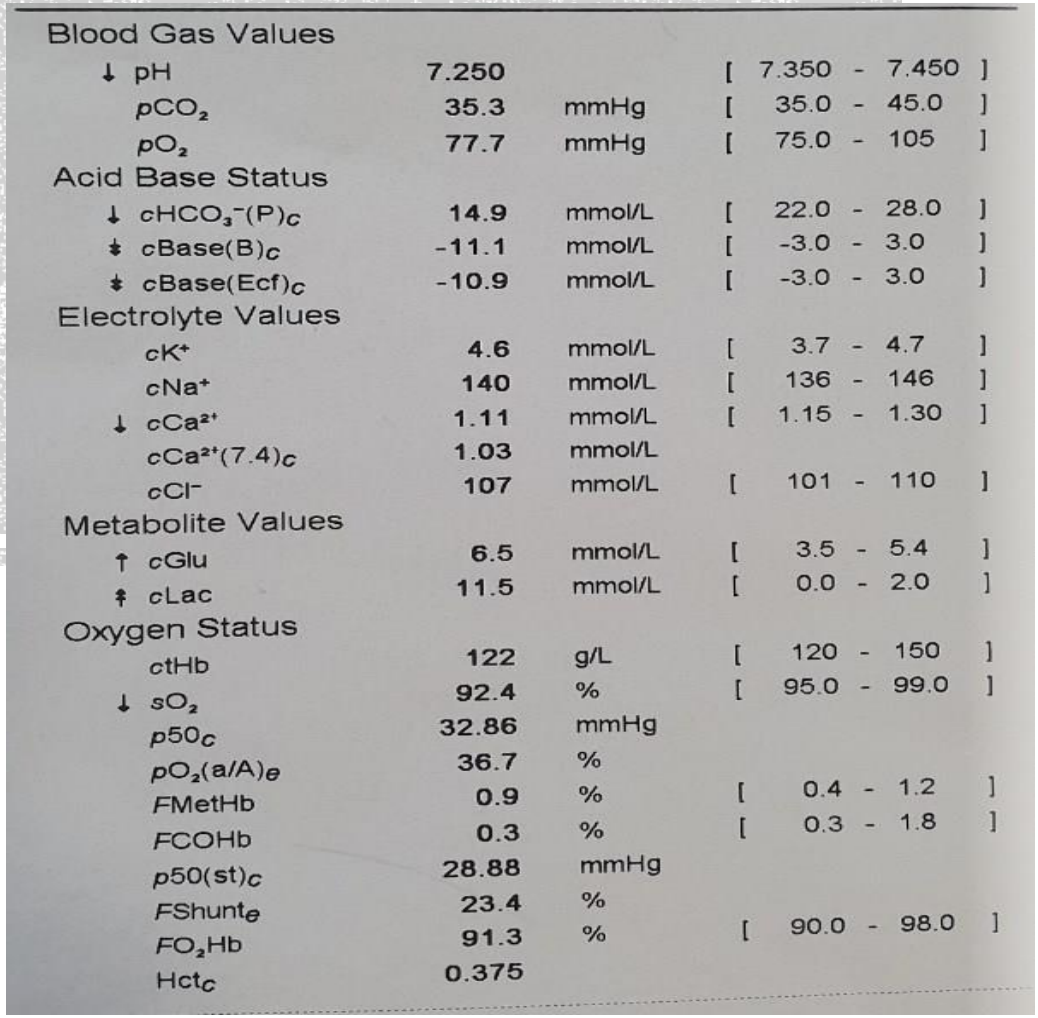
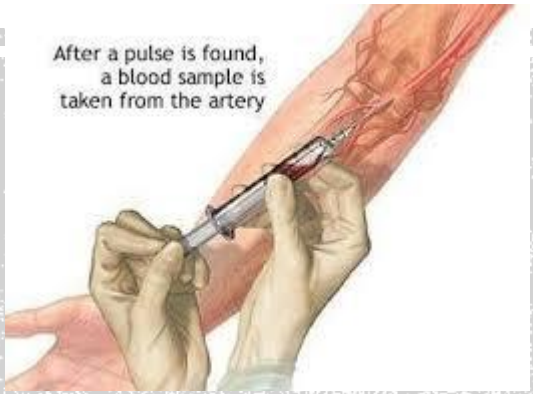


Blood Gas Values			
↓ pH	7.250		[7.350 - 7.450]
pCO_2	35.3	mmHg	[35.0 - 45.0]
pO_2	77.7	mmHg	[75.0 - 105]
Acid Base Status			
↓ $cHCO_3^-(P)_c$	14.9	mmol/L	[22.0 - 28.0]
↕ $cBase(B)_c$	-11.1	mmol/L	[-3.0 - 3.0]
↕ $cBase(Ecf)_c$	-10.9	mmol/L	[-3.0 - 3.0]
Electrolyte Values			
cK^+	4.6	mmol/L	[3.7 - 4.7]
cNa^+	140	mmol/L	[136 - 146]
↓ cCa^{2+}	1.11	mmol/L	[1.15 - 1.30]
$cCa^{2+}(7.4)_c$	1.03	mmol/L	
cCl^-	107	mmol/L	[101 - 110]
Metabolite Values			
↑ cGlu	6.5	mmol/L	[3.5 - 5.4]
↕ cLac	11.5	mmol/L	[0.0 - 2.0]
Oxygen Status			
ctHb	122	g/L	[120 - 150]
↓ sO_2	92.4	%	[95.0 - 99.0]
$p50_c$	32.86	mmHg	
$pO_2(a/A)_e$	36.7	%	
FMetHb	0.9	%	[0.4 - 1.2]
FCOHb	0.3	%	[0.3 - 1.8]
$p50(st)_c$	28.88	mmHg	
$FShunt_e$	23.4	%	
FO_2Hb	91.3	%	[90.0 - 98.0]
Hct_c	0.375		

Understanding ABG



WHAT YOU SHOULD ALREADY KNOW:
ABG (Hep.syringe) is more accurate than VBG (Lithium Hep. Tube)
ABG is more accurate than VBG
Glass tube/syringe is preferable but poses safety risk
Preferable : Branchial Artery, , Radial Artery, Femoral Artery
VBG transportation: RT in less 20 min. NO ICE!!
Sample is temperature dependent (gas solubility increases when cold)
Familiar with ABL90 operations at NICU, ICU, CCU

OBJECTIVE

- Significances of Acid Base Balance
- Understand Acid Base Equation
- What is acidic and basic
- Important ABG components
- Respiratory and Renal mechanisms
- Determine if pH is Respiratory or Metabolic- driven (R.O.M.E)
- Determine if non/partial/ full compensation has occurred
- Relate the diseases/ symptoms with acid base imbalances

Determine the causes of Acid Base Imbalance

SIDS

HEAD INJURY

Cough syrup

DRUGS

PAINKILLER

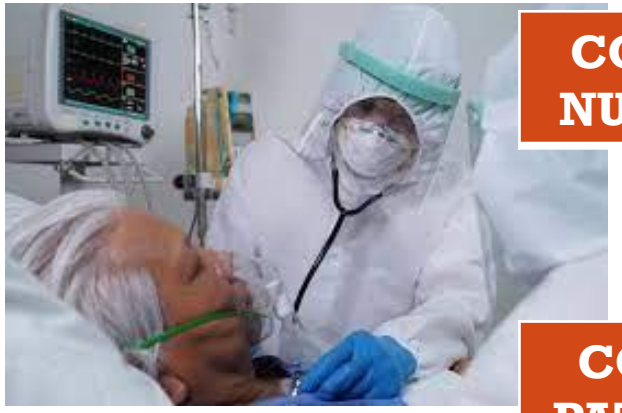
ASTHMA

VOMITING

SPORTS

HIGH ALTITUDE

RENAL PATIENT



COVID NURSES

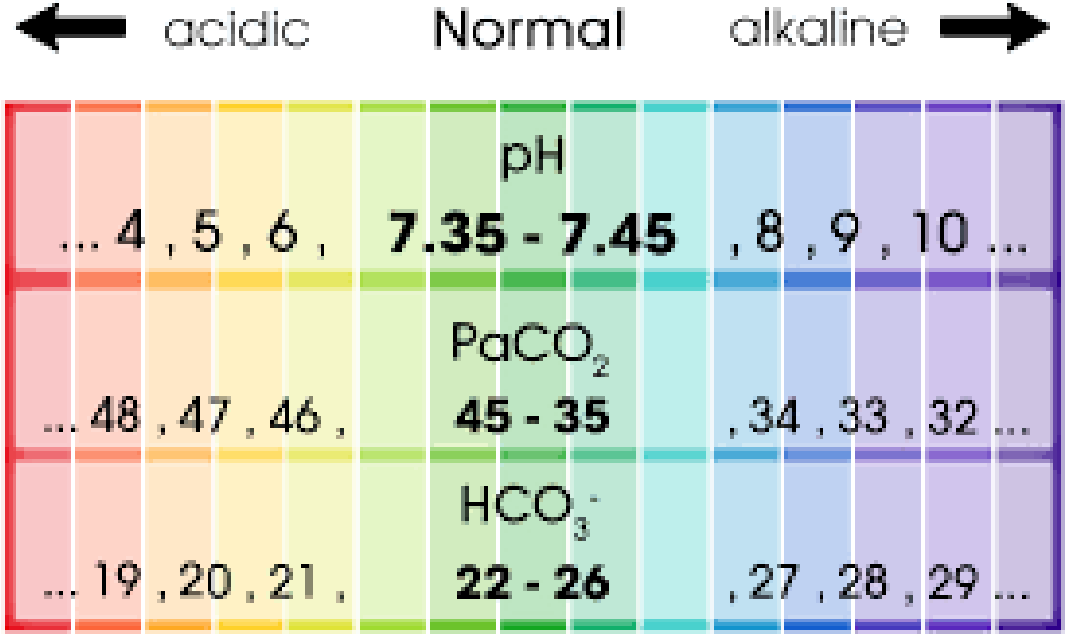
COVID PATIENT

ACID-BASE BALANCE

MAINTAINING PH REQUIRES A DELICATE BALANCE BTW CO2 (LUNGS) AND BICARBONATE (KIDNEYS/ METABOLIC SYSTEM)

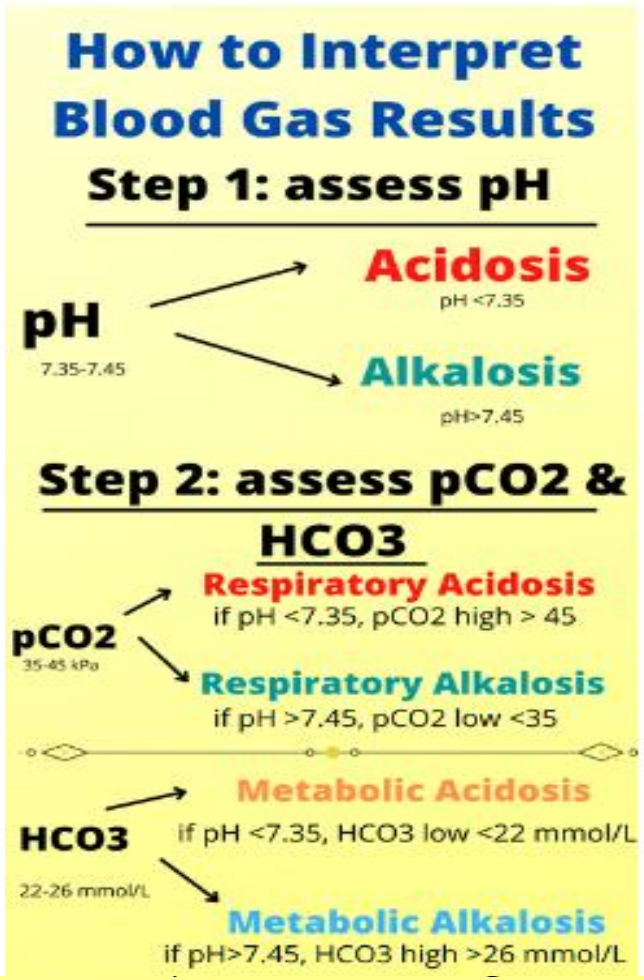
$$CO_2 + H_2O \rightleftharpoons H_2CO_3 \rightleftharpoons HCO_3^- + H^+$$

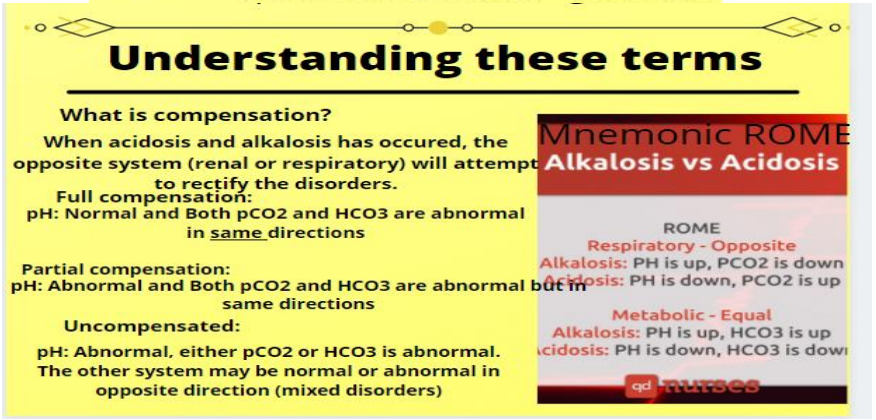
CARBON DIOXIDE + WATER	CARBONIC ACID	BICARBONATE + HYDROGEN ION



INTRO: SOME PARAMETERS OF ABG

- pH
- PaCO2 and PaO2 — the partial pressures of CO2 and oxygen
- $HCO3^-$ — bicarbonate
- Base excess (BE) — a measure of the excess or deficiency of base in the blood; by definition, it is the amount of base (in mmol) that would correct one litre of blood to a normal pH (if an excess, its +ve, this is the amount of base needed to be removed for a normal pH, or if a deficit (-ve), the amount required to be added)
- Lactate — the end product of anaerobic glycosis (a rise indicates poor oxygenation and perfusion of tissues)





ABG Reference Interval

- **pH:** 7.35-7.45
- **Partial pressure of oxygen (PaO2):** 75 to 100 mmHg
- **Partial pressure of carbon dioxide (PaCO2):** 35-45 mmHg
- **Bicarbonate (HCO3):** 22-26 mEq/L or mmol/L

R.O.M.E Acronym

	pH	CO2	HCO3
Respiratory acidosis	↓	↑	Normal
Respiratory alkalosis	↑	↓	Normal
Compensated respiratory acidosis	↓	↑	↑
Compensated respiratory alkalosis	↑	↓	↓

** Source: American Thoracic Society*

The acronym **ROME** is used to help nurses remember the relationship between pH and CO2.

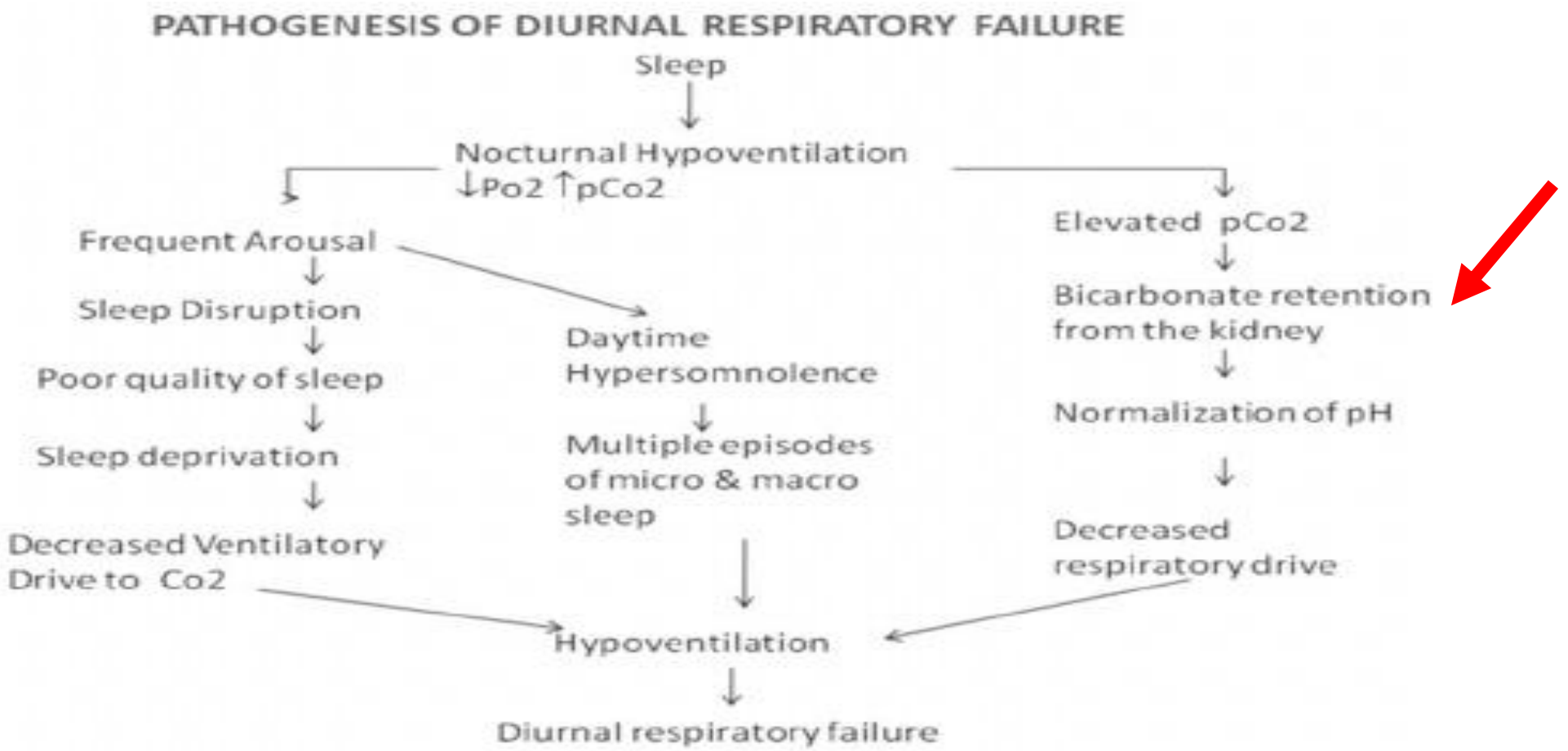
Respiratory **O**pposite -- In respiratory disorders, the pH and CO2 arrows move in opposite directions.

Metabolic **E**qual -- In metabolic disorders, the PH and CO2 arrows will move in the same direction.

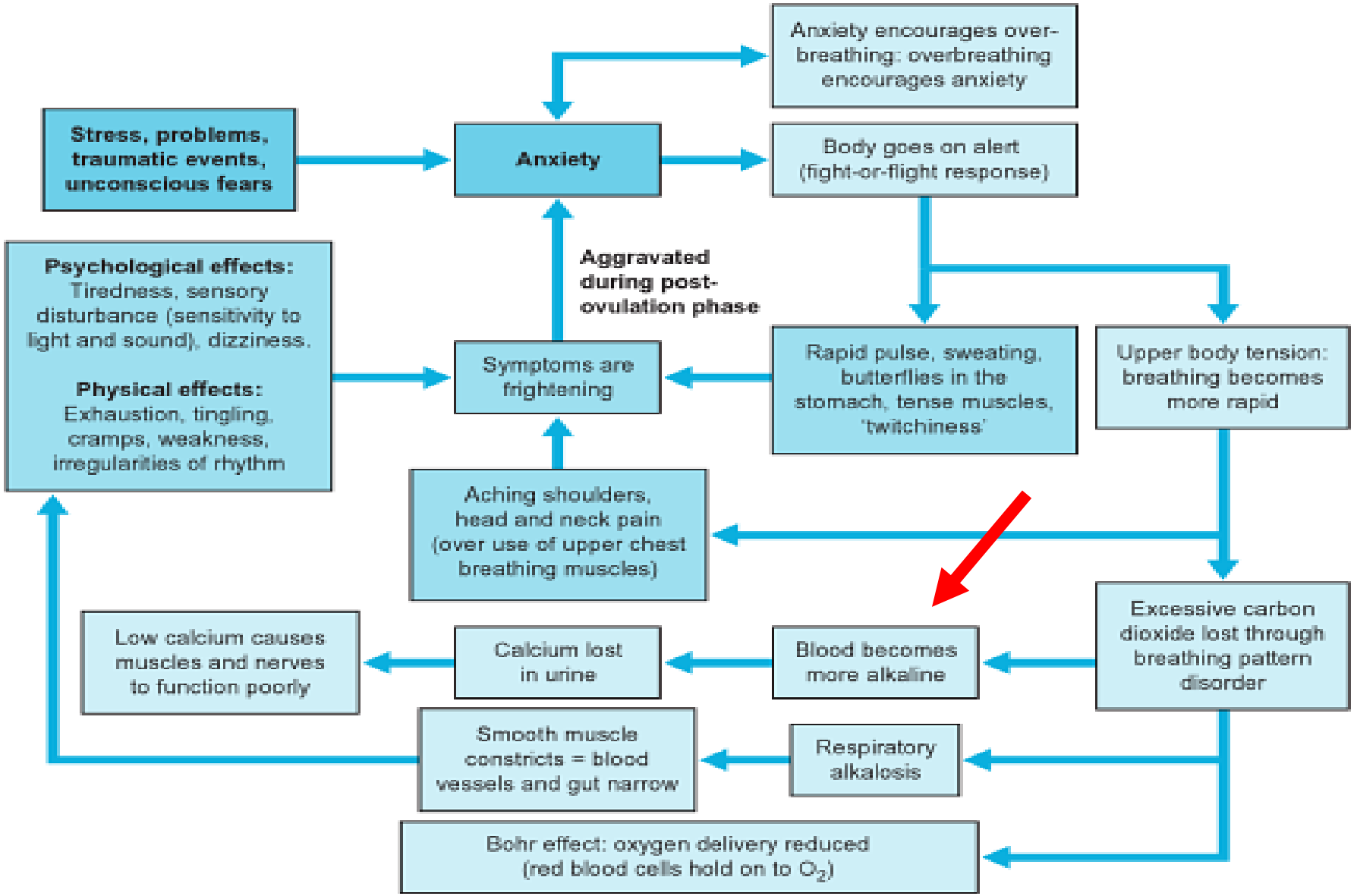
EFFECTS OF HYPO AND HYPERVENTILATION

AS DEMONSTRATED BY SLEEP HYPOVENTILATION AND ANXIETY DISORDER:

The Effects of Hypoventilation in Panic Attack



The Effects of Hyperventilation in Panic Attack



Hyperventilation (Resp Alkalosis) was implicated in causing low ionised Ca (hypocalcaemia) which results in cramps in laboured mothers

2. Case report

An otherwise healthy 30 year old full term parturient weighing 50 kg and 5 feet in height, G_4P_2 presented in labour in a highly anxious state. Anaesthesia consultation was sought because of non-progression of labour. She was hyperventilating with a respiratory rate of 30/min. Pulse was 96/min and blood pressure was 130/80 mmHg. After some time, she complained of circumoral numbness and her both wrists were in extreme flexion (carpal spasm). Arterial blood gas analysis revealed pH – 7.68, $PaCO_2$ – 24 mmHg and plasma HCO_3^- – 30 mmol/L. ECG showed prolonged QT interval. Samples of blood were drawn for estimation of serum calcium, proteins and parathormone levels. An impression of severe anxiety induced alkalosis causing hypocalcaemia was made. ***No anxiolytic was given,*** the patient was reassured, and infusion of 10 ml of 10% calcium gluconate was given over 10 min. The patient improved symptomatically over next 15–20 min. Due to non-progress of labour, caesarean section was planned by obstetrician. ***Foetal heart*** **sound varied from 120–140/min**. Systemic examination revealed normal heart sounds and chest was clear bilaterally. Airway assessment was unremarkable and spine was normal. Haemoglobin, bleeding time, clotting time, urine examination and INR were normal. Standard monitors were attached. Spinal anaesthesia was planned for the procedure. It was given in L_3–L_4 interspace with 25 G Quincke babcock needle using 1.8 ml of 0.5% hyperbaric bupivacaine. Adequate sensory block was achieved up to T4.

Ionised serum calcium level was low (0.7 mmol/L), while serum proteins were 7.2 gm/dl, and parathormone levels were 20 pg/ml In the recovery room, repeat arterial blood gas analysis was done which revealed pH – 7.36, $PaCO_2$ – 36 mmHg and HCO_3^- – 23 mol/L. Subsequent calcium concentrations were 1.3 mmol/L, well within normal range. Postoperatively USG of parathyroid was done which was normal. An endocrinologist was consulted who did not suspect any pathological cause for the episode of hypocalcaemia. The final clinical impression was that the patient was having hypocalcaemia due to respiratory alkalosis due to hyperventilation caused by severe anxiety because of outcome of pregnancy per se, labour pains and non-progression of labour.

3. Discussion

Respiratory alkalosis secondary to hyperventilation is probably the most common cause of acute ionised hypocalcaemia, and this appears to be the most likely cause in our case. Binding between calcium and protein is enhanced when serum pH increases, resulting in decreased ionised calcium. Ionised calcium should be measured whenever true hypocalcaemia is suspected [2]. Normal range of total serum calcium is 8.0–10.2 mg% or 2.2–2.5 m mol/L. About half of total calcium is ionised calcium (normal 4.0–4.6 mg% or 1.0–1.5 m mol/L), which is physiologically active. Ionised calcium may be roughly estimated by the following formula:

RESPIRATORY ACIDOSIS

- Respiratory acidosis is caused by **inadequate alveolar ventilation** (hypoventilation) leading to **CO_2 retention**.
- A **respiratory acidosis** would have the following **characteristics** on an **ABG**:
- ↓ pH
- ↑ CO_2
- **Causes** of **respiratory acidosis** include:
- Respiratory depression (e.g. opiates)
- Guillain-Barre: paralysis leads to an inability to adequately ventilate
- Asthma
- Chronic obstructive pulmonary disease (COPD)
- Iatrogenic (incorrect mechanical ventilation settings)
- CCHS

RESPIRATORY ALKALOSIS

- Respiratory alkalosis is caused by **excessive alveolar ventilation** (hyperventilation) resulting in more CO_2 than normal being exhaled. As a result, $PaCO_2$ is reduced and pH increases causing alkalosis.
- A **respiratory alkalosis** would have the following **characteristics** on
- ↑ pH
- ↓ CO_2
- **Causes** of **respiratory alkalosis** include: [3]
- Anxiety (i.e. panic attack)
- Pain: causing an increased respiratory rate.
- Hypoxia: resulting in increased alveolar ventilation in an attempt to compensate.
- Pulmonary embolism
- Pneumothorax
- Iatrogenic (e.g. excessive mechanical ventilation)

METABOLIC ACIDOSIS

Metabolic acidosis can occur as a result of either:

- **Increased acid production** or **acid ingestion**.
- **Decreased acid excretion** or **rate of gastrointestinal** and **renal HCO_3– loss**.
- A **metabolic acidosis** would have the following **characteristics** on an **ABG**:
- ↓ pH
- ↓ HCO_{3-}
- ↓ BE

ANION GAP

- The **anion gap** (AG) is a derived variable primarily used for the evaluation of metabolic acidosis to determine the presence of **unmeasured anions**. To work out if the metabolic acidosis is due to **increased acid production** or **ingestion** vs **decreased acid excretion** or **loss of HCO_3–** you can calculate the anion gap. The normal anion gap varies with different assays but is typically between **4 to 12 mmol/L**.
- **Anion gap formula:** Anion gap = $Na+ – (Cl- + HCO_{3-})$
- An **increased** anion gap indicates **increased acid production** or **ingestion**:
- Diabetic ketoacidosis (↑ production)
- Lactic acidosis (↑ production)
- Aspirin overdose (ingestion of acid)
- A **decreased** anion gap indicates **decreased acid excretion or loss of HCO_3–**:
- Gastrointestinal loss of HCO_3– (e.g. diarrhoea, ileostomy, proximal colostomy)
- Renal tubular acidosis (retaining H^+)
- Addison's disease (retaining H^+)

METABOLIC ALKALOSIS

- Metabolic alkalosis occurs as a result of **decreased hydrogen ion concentration**, leading to **increased bicarbonate**, or alternatively a direct result of increased bicarbonate concentrations.
- A **metabolic alkalosis** would have the following **characteristics** on an **ABG**:
- ↑ pH
- ↑ HCO_{3-}
- ↑ BE
- **Causes** of **metabolic alkalosis** include:
- Gastrointestinal loss of H^+ ions (e.g. vomiting, diarrhoea)
- Renal loss of H^+ ions (e.g. loop and thiazide diuretics, heart failure, nephrotic syndrome, cirrhosis, Conn's syndrome)
- Iatrogenic (e.g. addition of excess alkali such as milk-alkali syndrome)

MIXED ACIDOSIS/ALKALOSIS

- It's worth mentioning that it is possible to have a **mixed acidosis** or **alkalosis** (e.g. respiratory and metabolic acidosis/respiratory and metabolic alkalosis).
- In these circumstances, the **CO_2** and **HCO_3^-** will be moving in **opposite** directions (e.g. ↑ CO_2 ↓ HCO_3^- in mixed respiratory and metabolic acidosis).
- Treatment is directed towards correcting each primary acid-base disturbance.

MIXED RESPIRATORY AND METABOLIC ACIDOSIS

- A **mixed respiratory and metabolic acidosis** would have the following **characteristics** on an **ABG**:
- ↓ pH
- ↑CO_2
- ↓HCO_3–
- **Causes** of **mixed respiratory and metabolic acidosis** include:
- Cardiac arrest
- Multi-organ failure

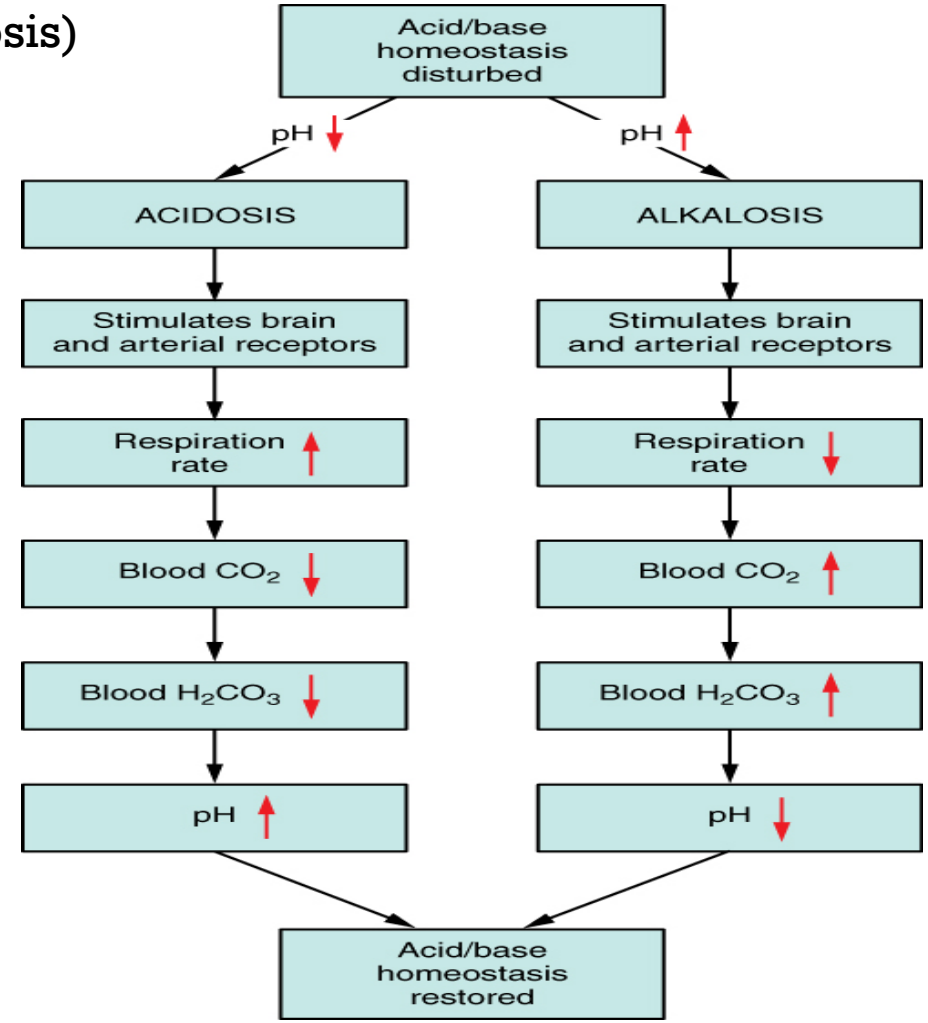
Mixed respiratory and metabolic alkalosis

A **mixed respiratory and metabolic alkalosis** would have the following **characteristics** on an **ABG**:
↑ pH
↓ CO_2
↑ HCO_3–
Causes of **mixed respiratory** and **metabolic alkalosis**:
Liver cirrhosis in addition to diuretic use
Hyperemesis gravidarum
Excessive ventilation in COPD

EXAMPLES OF COMPENSATION

Respiratory Compensation

e.g. after consumption of aspirin (acidosis) or antacid (alkalosis)



Metabolic Compensation

e.g following hypoventilation (acidosis) or hyperventilation (alkalosis)

Renal Compensation During Acidosis

The renal response to acidemia is 3-fold:

(1) increased reabsorption of the filtered HCO 3 –

(2) increased excretion of titratable acids

(3) increased production of ammonia

Renal Compensation During Alkalosis

The tremendous amount of HCO 3 – normally filtered and subsequently reabsorbed allows the kidneys to rapidly excrete large amounts of bicarbonate, if necessary

Compensation

- Respiratory acidosis/alkalosis (changes in CO_2) can be metabolically compensated by increasing or decreasing the levels of HCO_3– in an attempt to move the pH closer to the normal range.
- Metabolic acidosis/alkalosis (changes in HCO_3-) can be compensated by the respiratory system retaining or blowing off CO_2 in an attempt to move the pH closer to the normal range.

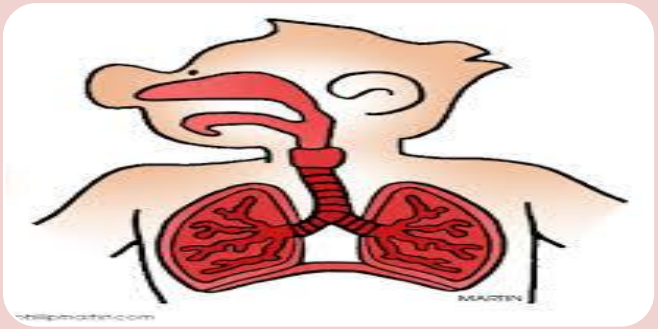
Rate of compensation

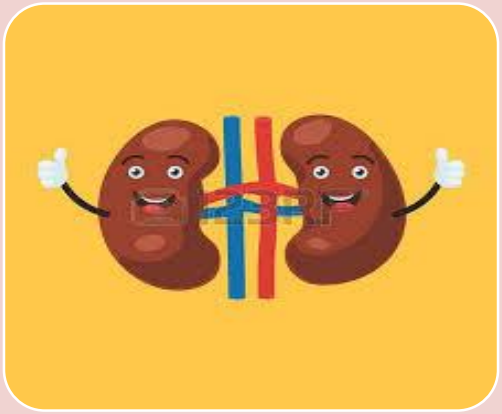
Respiratory compensation for a metabolic disorder can occur **quickly** by either increasing or decreasing alveolar ventilation to blow off more CO_2 (↑ pH) or retain more CO_2 (↓ pH).

Metabolic compensation for a respiratory disorder, however, takes **at least a few days** to occur as it requires the kidneys to either reduce HCO_3– production (to decrease pH) or increase HCO_3– production (to increase pH). As a result, if you see evidence of metabolic compensation for a respiratory disorder (e.g. increased HCO_3-/base excess in a patient with COPD and CO_2 retention) you can assume that the respiratory derangement has been ongoing for at least a few days, if not more.

It's important to note that 'over-compensation' should never occur and, therefore, if you see something that resembles this you should consider other pathologies driving the change (e.g. a mixed acid/base disorder).

RESPIRATORY AND RENAL MECHANISMS, COMPENSATION





RESPIRATORY MECHANISM

Body controls pH by increasing or decreasing rate and depth of breathing and thereby the amount CO_2 expelled

If pH ↑→Hypoventilation → CO_2↑ →correct pH

If pH is ↓ →Hyperventilation→ CO_2 ↓ →correct pH

RENAL MECHANISM

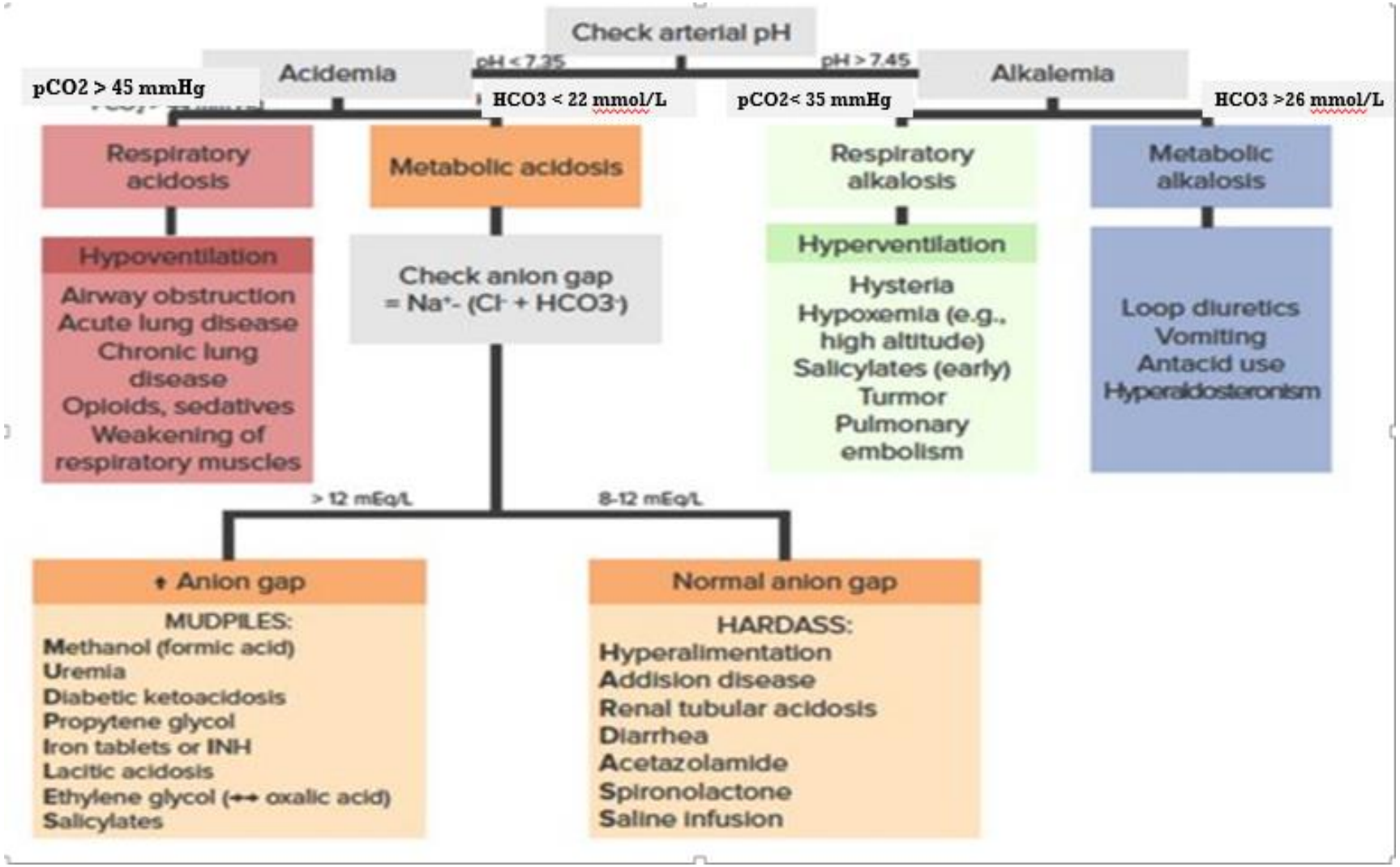
Kidneys control ph by adjusting the excretion of H^+ and renal tubular reabsorption of HCO_3^- in response to metabolic acid production.

COMPENSATION

When acidosis or alkalosis occurs (either through respiratory or renal mechanism), the opposite system will attempt to rectify this imbalance, this is termed "COMPENSATION".

e.g. if kidneys fail to excrete metabolic acids, ventilation is adjusted in order to eliminate more CO_2.

SUMMARY


Check arterial pH
Acidemia
pH < 7.35
pH > 7.45
Alkalemia
pCO2 > 45 mmHg
HCO3 < 22 mmol/L
pCO2< 35 mmHg
HCO3 >26 mmol/L
Respiratory acidosis
Metabolic acidosis
Respiratory alkalosis
Metabolic alkalosis
Hypoventilation
Airway obstruction
Acute lung disease
Chronic lung disease
Opioids, sedatives
Weakening of respiratory muscles
Check anion gap
= Na+ - (Cl- + HCO3-)
Hyperventilation
Hysteria
Hypoxemia (e.g., high altitude)
Salicylates (early)
Tumor
Pulmonary embolism
Loop diuretics
Vomiting
Antacid use
Hyperaldosteronism
> 12 mEq/L
8-12 mEq/L
↑ Anion gap
MUDPILES:
Methanol (formic acid)
Uremia
Diabetic ketoacidosis
Propytene glycol
Iron tablets or INH
Lacitic acidosis
Ethylene glycol (↔ oxalic acid)
Salicylates
Normal anion gap
HARDASS:
Hyperalimentation
Addision disease
Renal tubular acidosis
Diarrhea
Acetazolamide
Spironolactone
Saline infusion

CAUSES OF ACID BASE IMBALANCES

Box 1: Some causes of acid-base disturbance[3,4]

	METABOLIC	RESPIRATORY
Acidosis	Tissue hypoxia (eg, sepsis) Renal tubular acidosis Severe diarrhoea INCREASED ANION GAP: Diabetic ketoacidosis Salicylate/metformin poisoning Lactic acidosis Uraemia	Hypoventilation (eg, severe asthma exacerbation) Respiratory depression from drugs (eg, opioids, benzodiazepines) Chronic obstructive pulmonary disease
Alkalosis	Severe vomiting Cushing's disease Antacid abuse Diuretics Nasogastric tube suction	Altitude sickness Hyperventilation CNS trauma/disease Hepatic failure Anxiety

WORKED EXAMPLE 1

- A 17-year-old patient presents to ER complaining of a tight feeling in their chest, shortness of breath and some tingling in their fingers and around their mouth. She doesn't have significant past medical history and are not on any regular medication. An ABG is performed on the patient (who is not currently receiving any oxygen therapy).

An ABG is performed and reveals the following:

- pO2: 105 mmHg (82.5 – 97.5 mmHg)
- pH: 7.49 (7.35 – 7.45)
- pCO2: 27 mmHg (35 – 45 mmHg)
- HCO_3^-: 24 (22 – 26 mEq/L)

CONTINUE-

- PaO_2 of 14 on room air is at the upper limit of normal, so the patient is not hypoxic.
- **pH**
- A pH of 7.49 is higher than normal and therefore the patient is **alkalotic.**
- The next step is to figure out whether the respiratory system is contributing the alkalosis (e.g. ↓ CO_2).
- **$PaCO_2$**
- The CO_2 is low, which would be in keeping with an alkalosis, so we now know the respiratory system is definitely contributing to the alkalosis, if not the entire cause of it.
- The next step is to look at the HCO_3– and see if it is also contributing to the alkalosis.
- **HCO_3–**
- HCO_3– is normal, ruling out a mixed respiratory and metabolic alkalosis, leaving us with an isolated respiratory alkalosis.
- **Compensation**
- There is no evidence of metabolic compensation of the respiratory alkalosis (which would involve a lowered HCO_3-) suggesting that this derangement is relatively acute (as metabolic compensation takes a few days to develop).
- **Interpretation**
- Respiratory alkalosis with no metabolic compensation.
- The underlying cause of respiratory alkalosis, in this case, is a panic attack, with hyperventilation in addition to peripheral and peri-oral tingling being classical presenting features.

RESPIRATORY ALKALOSIS WITH NO METABOLIC COMPENSATION.

WORKED EXAMPLE 2

- A 16-year-old female presents to hospital with drowsiness and dehydration. She has no previous past medical history and on no regular medication.

An ABG is performed on room air reveals the following:

- PaO2: 105 mmHg (82.5 – 97.5 mmHg)
- pH: 7.33 (7.35 – 7.45)
- PaCO2: 22.5 mmHg (35 – 45 mmHg)
- HCO3–: 17 (22 – 26 mEq/L)

INVESTIGATION

- PaO2 of 14 on room air is at the upper limit of normal, so the patient is not hypoxic.
- A pH of 7.33 is lower than normal and therefore the patient is acidotic.
- The next step is to figure out whether the respiratory system is contributing the acidosis (i.e. ↑ CO2).

PaCO2:

- The CO2 is low, which rules out the respiratory system as the cause of the acidosis (as we would expect it to be raised if this was the case).
- So we now know the respiratory system is NOT contributing to the acidosis and this is, therefore, a metabolic acidosis.
- The next step is to look at the HCO3– to confirm this.

HCO3: is low, which is in keeping with a metabolic acidosis.

Check for Compensation

- We now know that the patient has a metabolic acidosis and therefore we can look back at the CO2 to see if the respiratory system is attempting to compensate for the metabolic derangement.
- In this case, there is evidence of respiratory compensation as the CO2 has been lowered in an attempt to normalise the pH.
- An important point to recognise here is that although the derangement in pH seems relatively minor this should not lead to the assumption that the metabolic acidosis is also minor.
- The severity of the metabolic acidosis is masked by the respiratory system's attempt at compensating via reduced CO2 levels.
- Interpretation
- Metabolic acidosis with respiratory compensation.

CONT-

- The underlying cause of the metabolic acidosis, in this case, is diabetic ketoacidosis.

METABOLIC ACIDOSIS WITH PARTIAL RESPIRATORY COMPENSATION.

END OF PRESENTATION

THANK YOU AND GOOD LUCK IN POST-CME EXAMS