ABG'S

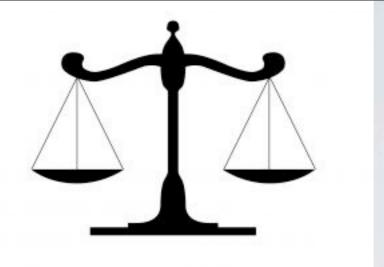
By Colin Dibble Consultant in Emergency Medicine



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OBJECTIVES

- Understand acid-base physiology & pathophysiology
- Understand oxygenation and ventilation physiology & pathophysiology
- How to perform arterial blood gas
- Interpretation of ABG's

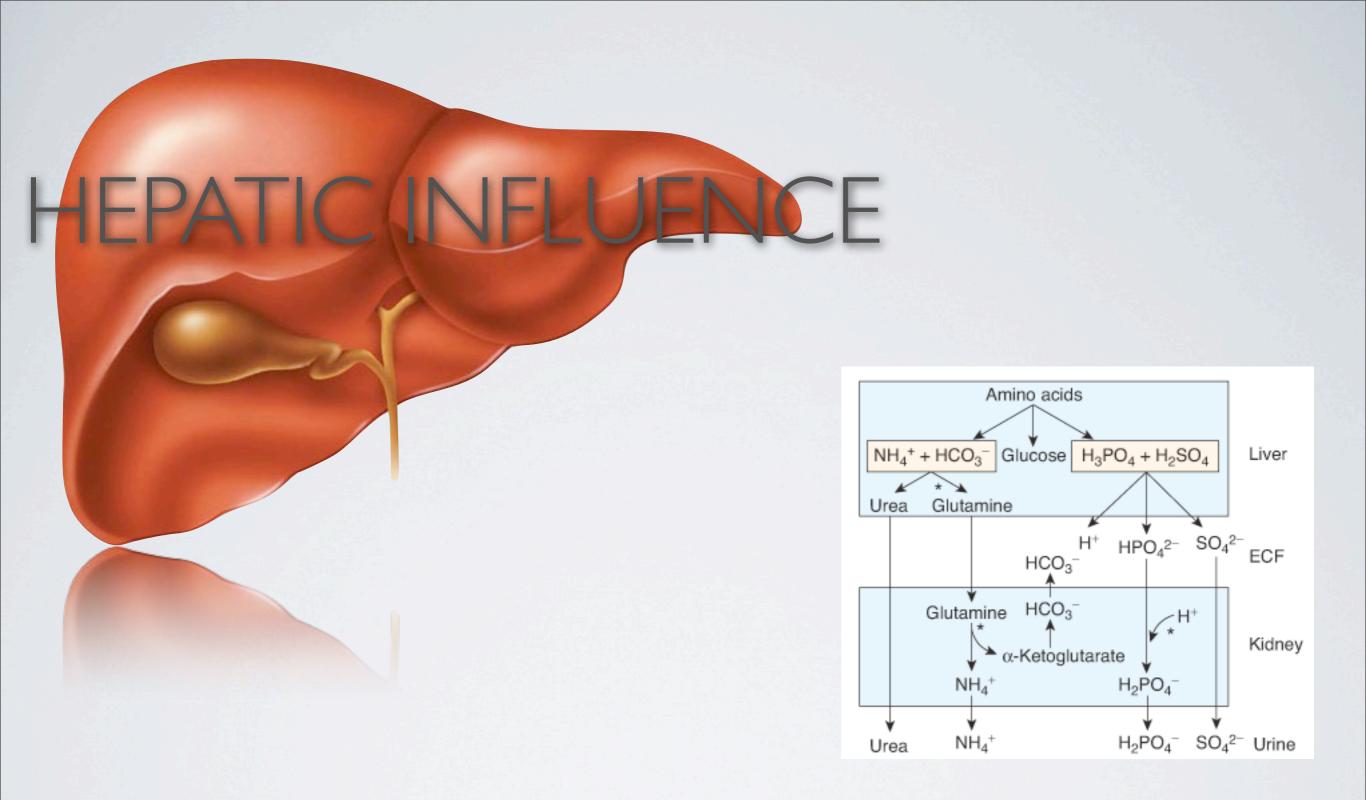


ACID-BASE

- Narrow normal range for protein function etc in body
- [H+] is represented by log= pH
- Acid [H⁺] equilibrium should be buffered (neutralised) by alkali
- Most important: bicarbonate/carbonic acid

$$pH=pK + log \frac{[HCO_3]}{[H_2CO_3]}$$

(Henderson-Hasselbalch equation)



- Neutralises bicarb & uses ammonia (urea synthesis)
- · 'third organ' of acid-base balance



RENAL ACID-BASE PHYSIOLOGY

- Controls mainly bicarbonate homeostasis
- Can increase bicarb resorption 6-10x over 4-5 days
- Excretes acid load
- Drugs that alter sodium delivery can affect bicarb resorption

Ganong's Review of Medical Physiology Twenty-Third Edition Kim E. Barrett et al McGraw Hill (2010)

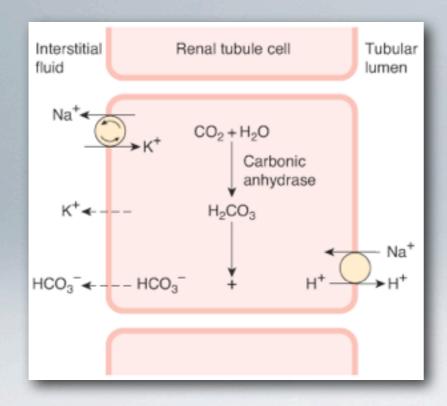


FIGURE Secretion of acid by proximal tubular cells in the kidney. H+ is transported into the tubular lumen by an antiport in exchange Na+. Active transport by Na, K-ATPase is indicated by arrows in the circle. Dashed arrows indicate diffusion

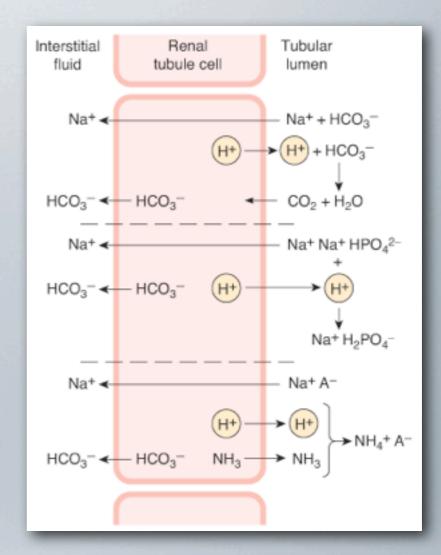
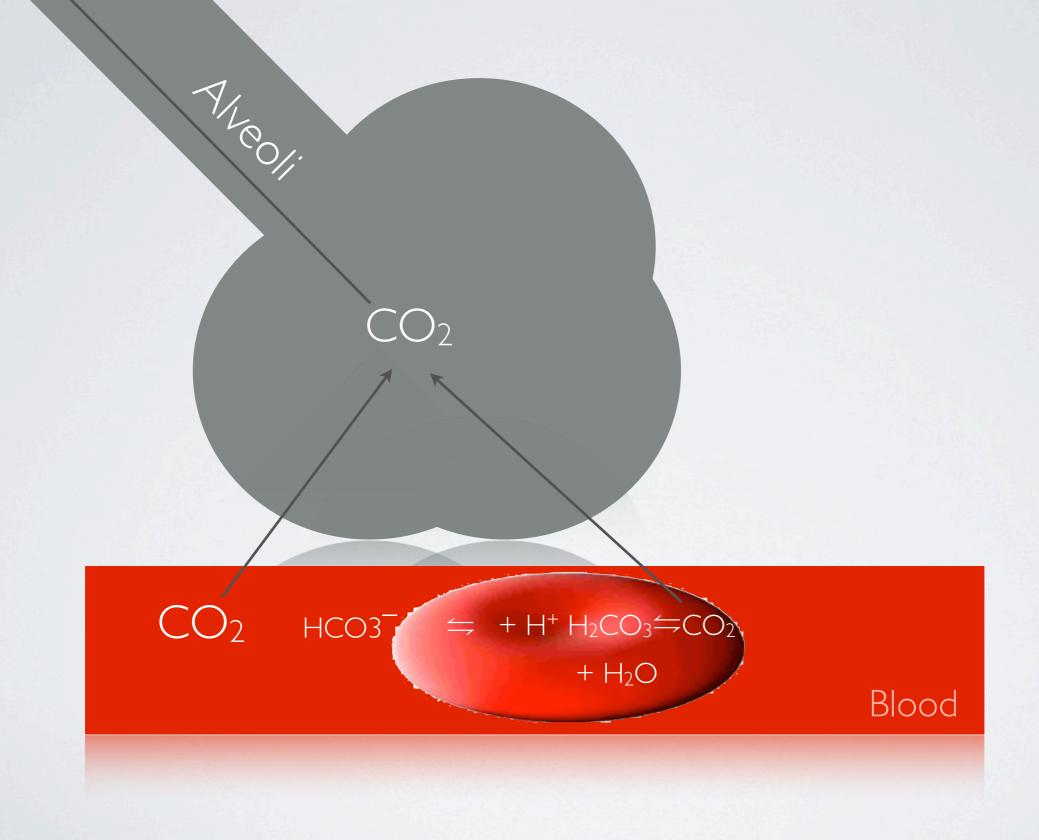
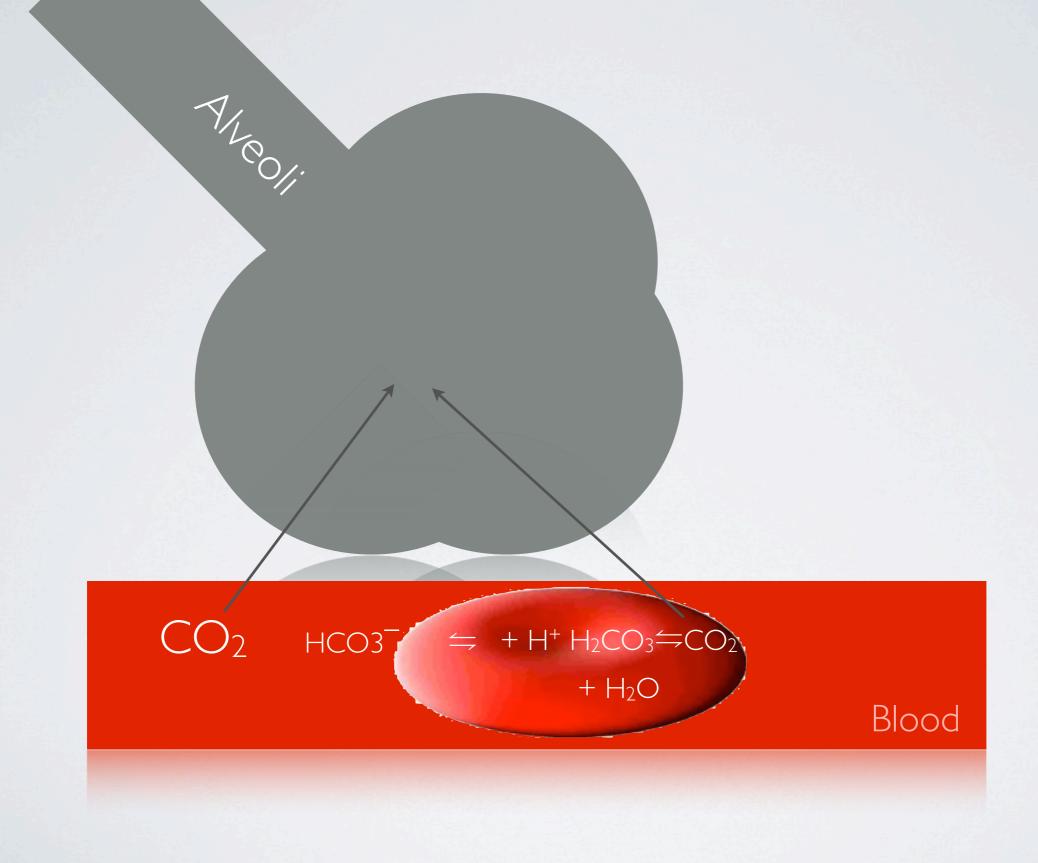


FIGURE Fate of H+ secreted into a tubule in exchange for Na+.Top:
Reabsorption of filtered bicarbonate via CO2. Middle: Formation of phosphate. Bottom: Ammonium formation. Note that in each instance one Na+ ion and one HCO3 ion enter the bloodstream for each H+ ion secreted. A , anion

LUNGS IN ACID-BASE



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ACID-BASE



- Normal pH 7.35-7.45
- Attempt to compensate back to normal pH
- Balance of acid and base (alkali)
- <7.35 = acidosis, >7.45 = alkalosis
- Clinically mainly through bicarbonate buffer system

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METABOLIC ACIDOSIS

- pH<7.35, \downarrow HCO₃, (or \downarrow BE {<-2})
- Excess [H⁺] from increased intake or production of acid or reduced excretion of H⁺
- · Reduced alkali from excess losses or reduced production
- Results in increased H⁺ delivery to renal tubules thus increased H⁺ excretion, reduced HCO₃: used up to buffer H⁺
- Increased breathing rate blows off CO₂ to compensate if able

CAUSES (raised anion gap)

- Increased production/reduced excretion
 - · Lactic acid (shock, infection, tissue ischaemia)
 - Urate (renal failure)
 - Ketones (DKA, alcoholic ketoacidosis)
 - Drugs/toxins (salicylates, metformin, ethylene glycol, methanol)

CAUSES (normal anion gap)

- Bicarbonate losses or ingestion of H+
 - Renal tubular acidosis
 - Diarrhoea
 - Drugs (acetazolamide via CA inhibition)
 - Addison's disease (competition for K⁺ excretion)
 - Pancreatic fistula
 - Ammonium chloride ingestion

METABOLIC ALKALOSIS

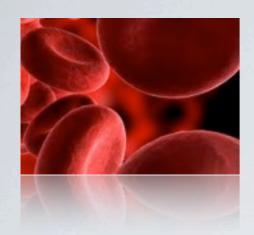
- pH>7.45, THCO₃ (<20mmol/L) (TBE>2)
- Excess alkali intake/production
- · Loss of acid (vomiting)
- K⁺ depletion (diuretics/burns)
- Reduced Cl⁻/ECF volume (diarrhoea, CF) via increased mineralcorticoid acitivity (retain Na⁺, lose K⁺ & H⁺)

RESPIRATORY ACIDOSIS

- pH<7.35, $1pCO_2 > 6 kPa$
- Reduced ventilation (COPD, drugs (e.g. Sedation), lung disease, chest trauma, neuromuscular weakness, head injury)
- Chronically: increased HCO₃ generation by kidneys and aggressive resorption to raise HCO₃ to buffer H⁺.

RESPIRATORY ALKALOSIS

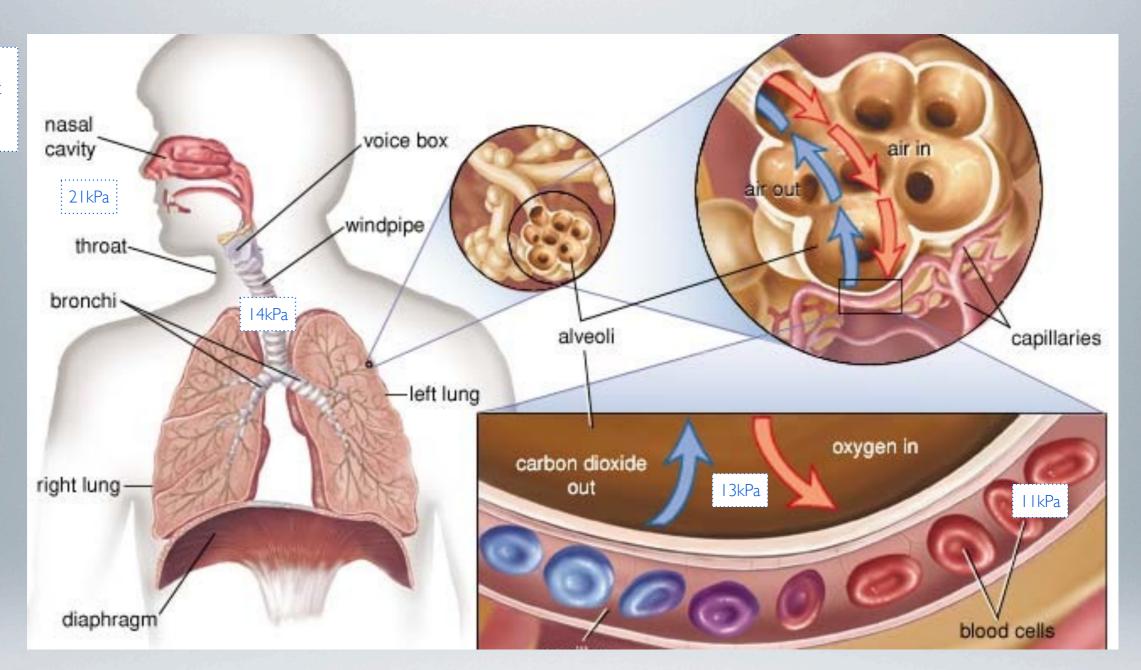
- pH>7.45, ↓pCO₂.
- Increased ventilation (anxiety, salicylates {initially}, infection, stroke, pain, iatrogenic)
- Associated with \$\square\$ free Ca⁺⁺.(taken up by proteins which have become negatively charged by releasing H⁺) thus paraesthesia and carpopedal spasms



OXYGENATION

- · 21% in air, amount of oxygen getting through to blood less
- Carried on Hb in red cells
- Reduced by: less air pressure/p0₂(e.g. Altitude), reduced tidal volume, reduced gaseous exchange surface area/increased thickness, reduced Hb binding (CO), anaemia, ↓ blood volume or perfusion (haemorrhage/failure), ↓ intracellular use (cyanide/mitochondria abnormality)

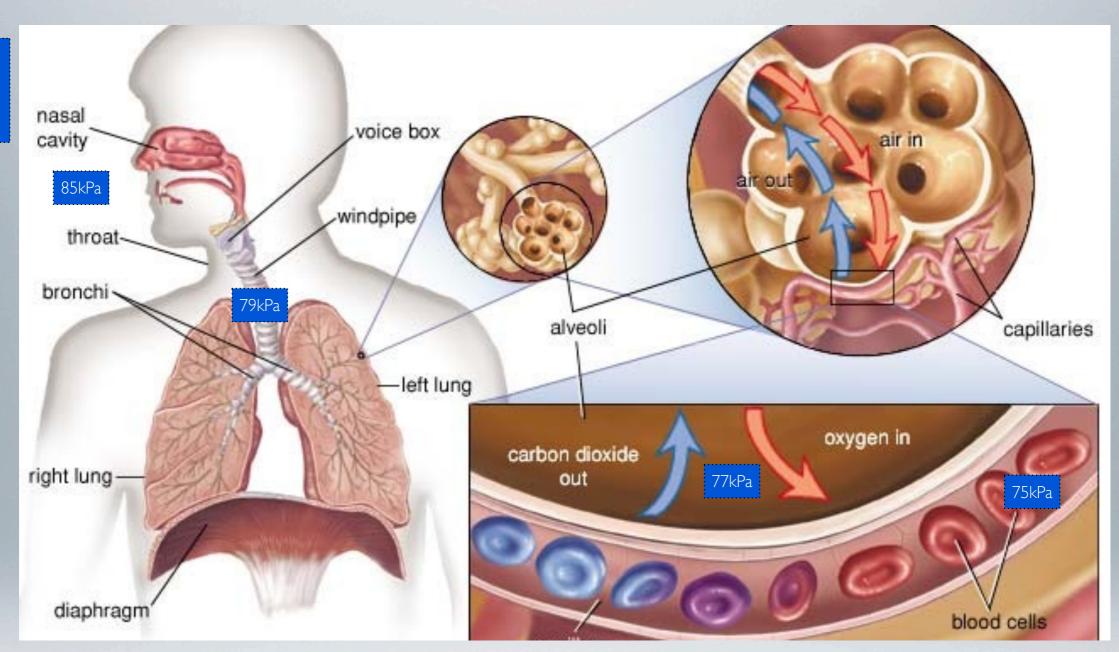
O₂ partial pressures at sea level at 37°C



A-a GRADIENT

Thus p02 should be approximately Fi02-10

O₂ partial pressures at sea level at 37°C on 85%



A-a GRADIENT

Thus p02 should be approximately Fi02-10



VENTILATION

- Movement of air in and out alveoli. C0₂ most sensitive indicator
- Reduced MV usually

 normal oxygenation but
 rapid build up of pC0₂.(see
 respiratory acidosis)
- End tidal C0₂ good estimate usually (3mmHg lower)





ABG'S

- Indication:
 - Check oxygenation and ventilation
 - Check acidosis/alkalosis
 - Monitor changes
 - Quick check Hb, Glucose, CO, Lactate, electrolytes etc

COMPLICATIONS

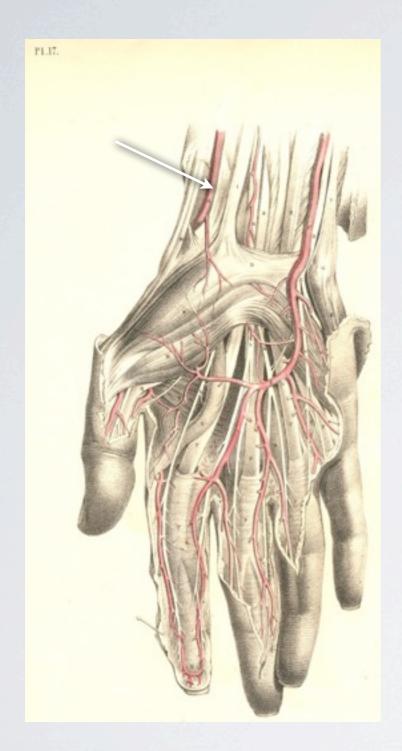
- Bleeding
- Haematoma formation
- Arterial thrombosis and ischaemia distally
- AV fistula
- Infection
- Nerve damage

PROCEDURE

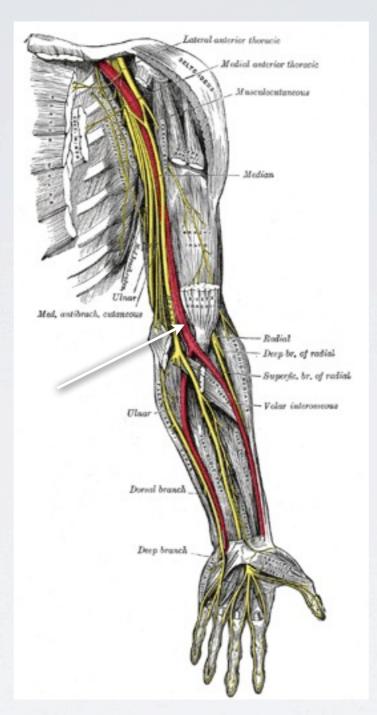
- Explain to patient and get verbal consent
- Assemble equipment
- Standard ANTT technique
- Identify anatomy (Allens test in wrist)

- 90° to skin above artery between index and middle fingers
- Slowly until self fills syringe
- Remove and press on artery
- Expel air and cap. Roll syringe

ANATOMY



Radial Artery



Brachial Artery



Femoral Artery

INTERPRETATION

- Check patient details/time etc
- Check oxygenation (p0₂ should be about Fi02-10kPa)
- Check pH
 - <7.35=acidosis
 - >7.45=alkalosis

- Check pCO₂. Does it explain abnormality? If so =respiratory. If not =metabolic.
- Check HC0₃ . Raised in metabolic alkalosis. Low in metabolic acidosis

COMPENSATION

- · The bodies way of trying to balance the pH back to normal
- The pC0₂ or HCO₃ should be the opposite of what you would expect if they were causing the pH abnormality.
 - E.g. In respiratory acidosis, the pC0₂ is high (acid). The kidneys compensate (over time) and the HCO₃ (base) rises. The HCO₃ is the opposite of what it would be if it were causing the acidosis-i.e. If it was metabolic

Acid-base disorder	рН	PaCO ₂	HCO ₃
Respiratory acidosis	+	1	N
Metabolic acidosis	Ţ	Ν	1
Respiratory alkalosis	1	1	N
Metabolic alkalosis	1	N	↑
Respiratory acidosis with renal compensation	+ *	1	↑
Metabolic acidosis with respiratory compensation	+ *	1	1
Respiratory alkalosis with renal compensation	↑ *	1	1
Metabolic alkalosis with respiratory compensation	† *	1	Ť
Mixed metabolic and respiratory acidosis	1	1	1
Mixed metabolic and respiratory alkalosis	<u> </u>	<u> </u>	↑

^{*} If the compensation is virtually complete the pH may be in the normal range – over compensation does not occur.



Scenario 1

Initial Information

A 75 year old man presents to the emergency department after a witnessed out-of-hospital VF cardiac arrest.

The paramedics arrived after 10 minutes, during which CPR had not been attempted. The paramedics had successfully restored spontaneous circulation after 3 shocks.

On arrival:

comatose (GCS 3) ventilated with 50% oxygen via tracheal tube HR 120 min⁻¹ BP 150/95 mmHg.



Scenario 1 (continued)

Arterial blood gas analysis reveals:

 FiO_2 0.5 (50%)

pH 7.10

PaCO₂ 6.2 kPa (47 mmHg)

PaO₂ 7.5 kPa (56 mmHg)

HCO₃- 14 mmol l⁻¹

BE - 10 mmol I⁻¹

Normal Values

7.35 - 7.45

4.7 – 6.0 kPa (35–45 mmHg)

> 10 kPa (75 mmHg) on air

 $22 - 26 \text{ mmol } I^{-1}$

+/- 2 mmol I⁻¹



Scenario 2

Initial Information

A 65 year old man with severe COPD has just collapsed in the respiratory unit.

On initial assessment by the ward nurse he is apnoeic but has an easily palpable carotid pulse at 90 min⁻¹.

The nurse is attempting to ventilate his lungs with a bag-mask and supplemental oxygen (with reservoir) as the cardiac arrest team arrive.



Scenario 2 (continued)

Arterial blood gas analysis reveals:

FiO ₂	0.85	(85%)	estimated	<u>Normal</u>	Values
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pH 7.10 7.35 - 7.45

PaCO₂ 18.0 kPa (135 mmHg) 4.7 – 6.0 kPa (35–45 mmHg)

PaO₂ 19.5 kPa (147 mmHg) > 10 kPa (75 mmHg) on air

 HCO_3^- 36 mmol I^{-1} 22 – 26 mmol I^{-1}

BE + 12 mmol I⁻¹ +/- 2 mmol I⁻¹



Scenario 3

Initial Information

A 75 year old woman is admitted to the emergency department following a VF cardiac arrest, which was witnessed by the paramedics. This had been preceded by 30 minutes of severe central chest pain.

A spontaneous circulation was restored after 2 shocks, but the patient remained comatose and apnoeic. The paramedics intubated her trachea, and on arrival in hospital her lungs are being ventilated with an automatic ventilator using a tidal volume of 900 ml and a rate of 18 breaths min⁻¹.



Scenario 3 (continued)

Arterial blood gas analysis reveals:

FiO_2 1.00 (100%)	Normal	Values
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pH 7.62 7.35 - 7.45

PaCO₂ 2.65 kPa (20 mmHg) 4.7 – 6.0 kPa (35–45 mmHg)

PaO₂ 25.4 kPa (192 mmHg) > 10 kPa (75 mmHg) on air

 HCO_3^- 20 mmol I^{-1} 22 – 26 mmol I^{-1}

BE - 4 mmol I⁻¹ +/- 2 mmol I⁻¹



Scenario 4

Initial Information

An 18 year old insulin dependent diabetic is admitted to the emergency department.

He has been vomiting for 48 hours and because he was unable to eat, he has taken no insulin.

On arrival:

HR 130 min⁻¹ BP 90/65 mmHg. Spontaneous breathing, RR 35 min⁻¹ Oxygen 4 I min⁻¹ via Hudson mask GCS 12 (E3, M5, V4)



Scenario 4 (continued)

Arterial blood gas analysis reveals:

FiO ₂	0.3 (30%) estimated	Normal Values
рН	6.89	7.35 - 7.45
PaCO ₂	2.48 kPa (19 mmHg)	4.7 – 6.0 kPa (35–45 mmHg)
PaO ₂	17.0 kPa (129 mmHg)	> 10 kPa (75 mmHg) on air
HCO ₃ -	4.7 mmol I ⁻¹	22 – 26 mmol I ⁻¹
BF	- 29 2 mmol I ⁻¹	+/- 2 mmol I ⁻¹

The blood glucose is 30 mmol l⁻¹ and there are ketones+++ in the urine



Scenario 5

Initial Information

A 75 year old man is on the surgical ward 2 days after a laparotomy for a perforated sigmoid colon secondary to diverticular disease. He has become hypotensive over the last 6 hours. His vital signs are:

HR 120 min⁻¹ – sinus tachycardia –

warm peripheries

BP 70/40 mmHg

RR 35 min⁻¹

SpO₂ on oxygen 92%

Urine output 50 ml in the last 6 hours

GCS 13 (E3, M6, V4)



Scenario 5 (continued)

Arterial blood gas analysis reveals:

FiO ₂	0.4	(40%)	approx
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pH 7.17

PaCO₂ 4.5 kPa (34 mmHg)

PaO₂ 8.2 kPa (62 mmHg)

HCO₃- 12 mmol l-1

BE - 15 mmol I⁻¹

Normal Values

7.35 - 7.45

4.7 – 6.0 kPa (35–45 mmHg)

> 10 kPa (75 mmHg) on air

 $22 - 26 \text{ mmol } 1^{-1}$

+/- 2 mmol I⁻¹

