

### 2009.2.3 (ABG)

A 59 year old obese man receives 5 mg of intravenous morphine for analgesia for abdominal pain. Thirty minutes later, his GCS has fallen to 12 and investigations are performed.

			Reference Range
FiO <sub>2</sub>	0.21		
pH	7.24		7.35-7.45
pCO <sub>2</sub>	92	mmHg	35-45
pO <sub>2</sub>	45	mmHg	80-95
Bicarbonate	49	mmol/L	22-28
Base excess	10		-3 - +3
O <sub>2</sub> saturation	78	%	> 95
Lactate	1.2	mmol/L	< 1.3
Na <sup>+</sup>	142	mmol/L	134-146
K <sup>+</sup>	3.8	mmol/L	3.4-5
Cl <sup>-</sup>	86	mmol/L	98-106
Glucose	11.4	mmol/L	3.5-5.5
Haemoglobin	184	g/L	135-180
Carboxy Hb	7	%	< 6%

Describe and interpret his investigations. (100%)

Note not stated if arterial or venous blood gas

This blood gas has multiple abnormalities but the most striking are underlying metabolic alkalosis with superimposed acute respiratory acidosis, critical hypoxaemia (if arterial sample), hyperglycaemia, and polycythaemia.

#### Summary

acidaemia

marked hypercarbia

hypoventilation

likely due to CNS depression after morphine administration

no clinical suggestion of neuromuscular / chest wall pathology

critical hypoxaemia

due to hypercarbic respiratory failure in presence of normal A-a gradient

markedly raised bicarbonate with raised base excess

metabolic alkalosis

glucocorticoid excess (steroids / Cushing syndrome) possible but low potassium expected

vomiting (expect low potassium)

bicarbonate administration (possible antacids for abdominal pain)

compensatory for chronic respiratory acidosis

markedly reduced oxygen saturations

moderate hypochloraemia

likely passive shift to maintain electroneutrality

moderate hyperglycaemia

glucocorticoid excess (Cushing syndrome, steroids both possible particularly in obese patient)

absolute or relative insulin deficiency

IDDM

NIDDM (in keeping with obesity)

mild polycythaemia

chronic hypoxia suggesting degree of chronic respiratory acidosis

haemoconcentration if reduced oral intake, vomiting (abdominal pain)

mild elevation in carboxyhaemoglobin

cigarette smoking or exposure

Acid base

A – acidaemia

R – high pCO<sub>2</sub>, so respiratory acidosis suggested

M – high bicarbonate suggests metabolic alkalosis or compensation

A – anion gap  $142-86-49 = 7$  is normal

D – not relevant

assuming respiratory acidosis

expected bicarbonate

2 for 10 acutely =  $5.2 \times 2 = 10$  rise in bicarb -> 34

5 for 10 chronically =  $5.2 \times 5 = 26$  rise -> 50 expected, measured 49

clinical scenario is more in keeping with element of acute respiratory acidosis however due to

polycythaemia (chronic hypoxia)

obesity (predisposing factor)

if pre-existing metabolic alkalosis

expected pCO<sub>2</sub> =  $0.7 \times 49 + 20 = 54$

this would support clinical scenario as it allows for acute respiratory element

i.e. compensated chronic metabolic alkalosis with superimposed acute respiratory acidosis

A-a gradient

$150 - (92 \times 1.25) = 35$  expected, 45 measured

implies on FiO<sub>2</sub> >0.21 as gradient is negative, but as A-a gradient formula is an estimation this may be possible with an arterial sample.

[Need all in bold to pass](#)

Acid base disorder

Mixed acid base disturbance with acute respiratory acidosis and underlying metabolic alkalosis

Or alternatively chronic respiratory acidosis considered acceptable if supporting corrective calculations performed to justify this view despite being discrepant with scenario

Supporting calculations

Critical hypoxemia

At least 5 from

hypochloraemia

hyperglycaemia

polycythaemia

raised carboxyhaemoglobin

hypercarbia

hypoxaemia

high bicarbonate

high base excess

with reasonable interpretation of at least 3 of these