VAQ 2010.1.3 (ABG)

A 45 year old man with type 1 diabetes mellitus is brought in by ambulance with an altered conscious state.

Serum biochemistry and arterial blood gas

			Reference Range
FiO_2	0.50		
pH	7.05		7.35-7.45
pCO_2	66	mmHg	35-45
pO_2	247	mmHg	80-95
Bicarbonate	18	mmol/L	22-28
Base excess	-14		-3 - +3
O_2 saturation	99	%	> 95
Na^+	131	mmol/L	134-146
K^+	5.0	mmol/L	3.4-5.0
Cl^-	92	mmol/L	98-106
Urea	15	mmol/L	3-8
Creatinine	227	micromol/L	45-90
Glucose	50.9	mmol/L	3.5-5.5

Describe and interpret his investigations (100%)

These investigations demonstrate a high AG metabolic acidosis and co-existent respiratory acidosis with marked hyperglycaemia. Urea/creatinine assay suggests renal impairment, likely to be hypovolaemic prerenal impairment. He has hypercapnoeic respiratory failure with a normal A-a gradient suggesting that there is no significant lung pathology. The overall picture is consistent with severe diabetic keotacidosis with sedation/obtundation causing hypoventilation.

Acidosis
Hypercapnoea
Hyperoxia (but supplemental oxygen)
Low bicarb suggests metabolic acidosis
Marked negative BE suggest metabolic acidosis

Na 131; corrected = 46/3 = 15 + 131 = 146 is normal -> pseudohyponatraemia

Potassium ‘high normal’

Slight hypochloraemia – corrects for glucose to essentially normal range

Markedly raised urea
- prerenal impairment i.e. hypovolaemia
- protein load e.g. dietary, GI bleed

Moderately raised creatinine
- Renal impairment
  - pre/renal/post
    - likely prerenal hypovolaemia
    - may have co-existing diabetic nephropathy
- Significant crossreactivity in some assays with ketone bodies – may be artefactual in which case likely more normal creatinine and more suggestive of prerenal hypovolaemia

Markedly raised glucose
- absolute or relative insulin deficiency (IDDM / NIDDM)
- stress response, steroids, iatrogenic dextrose administration unlikely to cause this degree of hyperglycaemia

A – acidosis
R – pCO2 raised BUT low bicarbonate suggesting metabolic acidosis also
M – low bicarbonate, highly negative BE support metabolic acidosis
A – 131-92-18 = 21 is a high anion gap ; supported by normal/low chlorine

Causes of anion gap acidosis
C – cyanide – not suggested
U – uraemia – likely to have small or no contribution at these levels
T – toluene – not hypokalaemic / history does not support
E – ethanol – not suggested
D – diabetic ketoacidosis overwhelmingly most likely
I – iron/isoniazid – not suggested
M – methanol – not suggested
P – paraquat / propylene glycol – not suggested
L – lactate – may contribute
E – ethylene glycol – not suggested
S – salicylates – not suggested, no resp alkalosis

This is likely to be diabetic ketoacidosis +/- some lactic acidosis (can measure to check)

D – 21-12 / 24-18 = 9/6 = 1.5 supports pure AGMA

A – expected pCO2 = 1.5×18+8 = 35, measured 66 -> uncompensated with primary respiratory acidosis
- hypoventilation
  - CNS
    - loss of hypoxaemic drive
    - depressed respiratory centre and conscious state
    - sedative / narcotics
  - neuromuscular
    - muscular pathology e.g. muscular dystrophy
    - impaired neuromuscular transmission
    - paralysing agent
    - demyelination e.g. GBS

A-a gradient = (713×0.5 = 356) – (66×1.25 = 82.5) = 273 = gap 26.5
- Age 45 ; expected gradient = age/4+4 = 15

This is a mildly raised A-a gradient and is suggestive of little or no significant oxygen exchange deficit.