VAQ 2011.2.2 (ABG)

A 3 month old girl is brought to your emergency department after three days of diarrhoea and vomiting. She appears very unwell and lethargic, with sunken eyes, a sunken fontanelle and dry mucous membranes. Her serum biochemical results are as follows

			Reference Range
Venous Blood Gas			
FiO_2	50	%	
pH	7.12	mmHg	(7.35-7.45)
pCO_2	12	mmHg	(40-52)
pO_2	103	mmHg	
O_2 Saturation	98	%	
Base Excess	-25.0	mmol/L	(-3 - +3)
Bicarbonate	4	mmol/L	(24-32)
Lactate	3.6	mmol/L	(0.5-2.0)
Electrolytes			
Sodium	155	mmol/L	(135-145)
Potassium	3.0	mmol/L	(3.5-4.8)
Chloride	136	mmol/L	(95-110)
Urea	15.4	mmol/L	(3-8)
Creatinine	45	mcmol/L	(50-120)
Glucose	6.1	mmol/L	(3.0-6.0)

Describe and interpret her blood test results (100%)

This child has a marked, compensated normal anion gap acidosis with some high AG contribution (lactataemia), with severe hypernatraemia, hyperchloraemia, hypokalemia, and uraemia consistent with GI bicarbonate, fluid, and potassium losses. Her clinical dehydration would be consistent with this being a hypovolaemic hyperchloraemia due to water>salt losses and requiring hypotonic fluid resuscitation with careful potassium supplementation.

Acidaemia
A – acid
R – resp component NOT acidic (i.e. not primary resp process)
M – low bicarbonate with markedly negative BE consistent with metabolic acidosis
A – 155 – (136+4) = 15 which is near normal, consistent with hyperchloraemia (NAGMA/hyperchloraemic acidosis) but the raised lactate suggests a co-existent degree of lactic acidosis (i.e. degree of HAGMA)
D – 3/20 = consistent with pure NAGMA
A – expected pCO2 (though venous sample) = 4×1.5+8 = 14, measured 12 i.e. appropriate compensation
i.e. **mixed high AG and normal AG but predominantly normal AG metabolic acidosis with respiratory compensation**

low pO2 in venous sample not interpretable
pCO2 low despite venous sampling
mild elevation lactate
- anaerobic metabolism
- **hypoperfusion** (c/w clinical picture)
- cytochrome toxins (drugs – not suggested)

marked hypernatraemia
- **hypovolaemia**
  - **water > salt losses**
    - **GI losses**
      - **vomiting / diarrhoea (c/w clinical)**
      - fistula (not suggested)
    - GU losses
      - osmotic diuresis
      - drugs (mannitol) – not suggested
      - glycosuria (not c/w euglycaemia)
      - central / nephrogenic diabetes insipidus
      - lithium / renal disease (not c/w normal creatinine, clinical picture)
    - Sweating / exercise (not c/w clinical)
- euvolaemia /hypervolaemia (not c/w clinical picture)

moderate hypokalaemia
- inadequate intake
  - anorexia / alcoholism (likely some degree of anorexia during acute illness but small contribution)
  - transcellular shift
  - acidaemia implies 'true' potassium even lower
- **excess losses**
  - **GI**
    - **vomiting, diarrhoea (c/w clinical)**
  - GU
    - osmotic losses, drug effect, mineralocorticoid XS (not c/w clinical picture)

marked hyperchloraemia
- almost always passive shift to maintain electroneutrality – normal AG

marked uraemia with normal creatinine
- ratio suggests pre-renal azotaemia

consistent with intravascular volume depletion / hypoperfusion and c/w clinical picture
glucose essentially normal
Although mineralocorticoid XS (Conn syndrome) can cause hypernatraemia and hypokalaemia, this is not consistent with the clinical picture of a gastrointestinal illness and acute water and salt losses.