



The electrolytes: sodium and potassium

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The electrolytes

An electrolyte

- is the ionized (or ionizable) constituents of a living cell, blood or other organic matter
- when ionized it carries a net electrical charge
- sodium(Na^+), potassium(K^+), calcium(Ca^{++}), magnesium, chloride(Cl^-), phosphate, and bicarbonate (HCO_3^-)....



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Why do vets care?

- All higher life forms require a subtle and complex electrolyte balance between the intracellular and extracellular environments

Intracellular fluid		
Na+	10	mmol/L
K+	145	mmol/L

Extracellular fluid		
Na+	140	mmol/L
K+	4	mmol/L



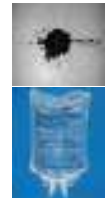
Why do vets care?

- Serious electrolyte disturbances may lead to cardiac and neurological complications, and most are medical emergencies
- Knowledge of blood electrolyte concentrations may help to make a diagnosis or dramatically influence treatment



Why do vets care?

- Fluids lost from the animal have an unknown concentration of electrolytes
- Fluids added to the patient have a known concentration but an unknown effect
- Drugs given to the patient can dramatically alter the concentration of electrolytes



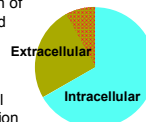
Evaluation of electrolytes

- Rapid diagnosis
- Refine patient management
- The situation can change very quickly
- Ability to measure this in-house is essential



Disorders of sodium and water

- Volume and concentration of body fluids are maintained within a narrow range by regulation of sodium and water loss
- The kidney plays a crucial role: balancing the excretion of salt and water with their intake



Role of the kidneys

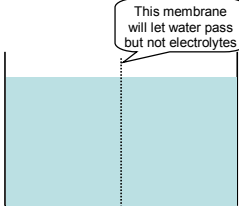
- The kidneys are the prime site for sodium and water homeostasis
- Numerous endocrine mechanisms are integrated in the nephron



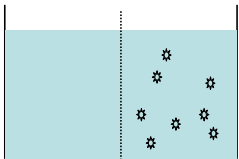
Do you remember osmosis?



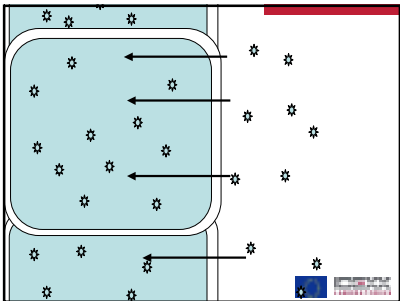
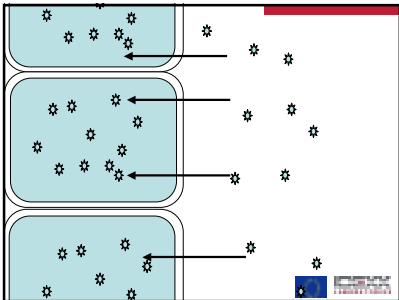
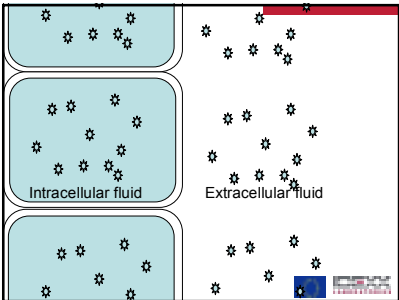
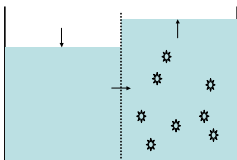
We start with a bucket of water...



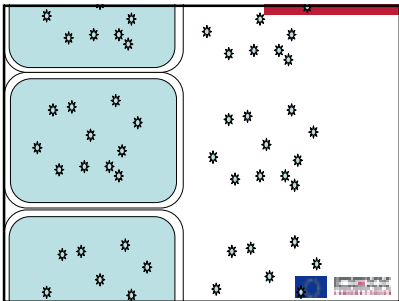
...we add a little salt..

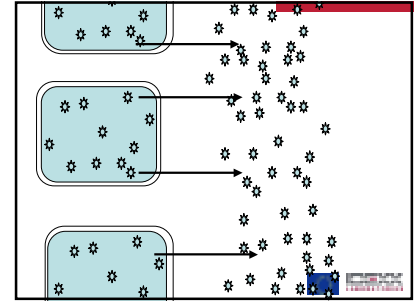
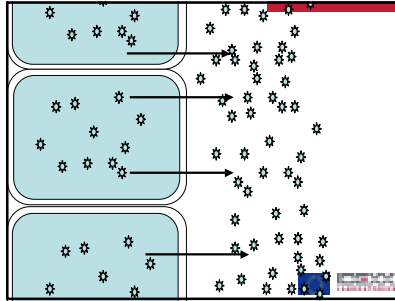
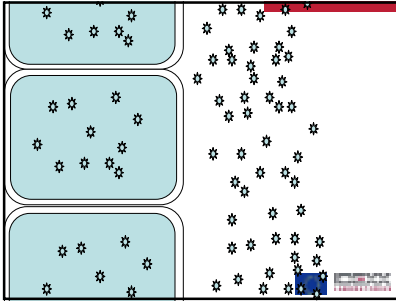


...and the magic happens.



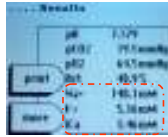
Let us start again...





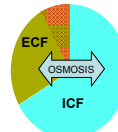
Blood sodium concentrations

- Indication of the amount of sodium relative to the amount of water in the ECF
- Provides no information about the total body sodium
- Not an indicator of dehydration – the vet must use other signs



Dehydration

- Loss of body water
- Loss will occur from the ECF
- Fluid lost from the body will have an unknown concentration of electrolytes



	ECF		ICF	
	Vol	Conc	Vol	Conc
Fluid lost from patient				
Low electrolyte concentration	↓	↑	↓	↑
Similar to ECF	↓	↔	↔	↔
High electrolyte concentration	↓	↓	↑	↓

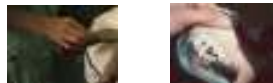
What is the point?

- Serum sodium concentrations **do not** help the vet decide if the patient is dehydrated
- A knowledge the patient's hydration status and sodium concentration will help the vet to decide what the likely underlying mechanism was and how best to provide intravenous fluid therapy



What about the intravascular volume?

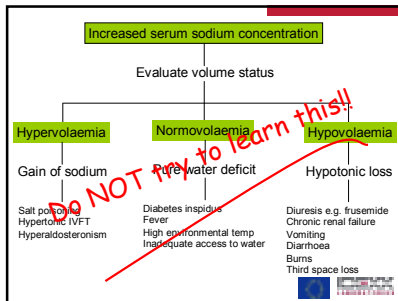
Recognition of the patient's volume status allows refinement of the differential diagnosis list



Signs of hypovolaemia

	Mild	Moderate	Severe
Heart rate	120-150	150-170	170-220
MM colour	Pinker	Pale	White/grey
CRT	~1 sec	~2 secs	~3 secs or more
Pulse amplitude	↑	↓	↓↓↓
Peripheral pulse	Easy	Hard	Absent





Hypernatraemia

- Hypervolaemia
- Normovolaemia
- Hypovolaemia

Hypernatraemia (common causes)

- Hypervolaemia
- Normovolaemia
 - Hyperthermia
- Hypovolaemia
 - Renal failure (acute or chronic)
 - Vomiting and diarrhoea

Hyponatraemia

- Hypervolaemia
- Normovolaemia
- Hypovolaemia

Hyponatraemia (common causes)

- Hypervolaemia
 - Congestive heart failure, severe hepatic disease, renal disease (nephrotic syndrome)
- Normovolaemia
 - Hypotonic fluid administration
- Hypovolaemia
 - Hypoadrenocorticism, vomiting and diarrhoea, diuretics

Clinical signs

- More likely if the abnormality has developed rapidly
- Principally neurological
 - HyperNa⁺ leads to cerebral dehydration
 - HypoNa⁺ leads to cerebral oedema



Does this require specific treatment?

- This electrolyte abnormality will (almost always) correct itself as the underlying disease is treated
- If hypovolaemic, restore volume using a fluid with a Na⁺ concentration close to that of the patient's plasma
 - hyperNa⁺ → 0.9% NaCl
 - hypoNa⁺ → Hartmann's or LRS

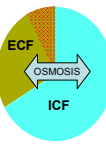
Once the volume is restored...

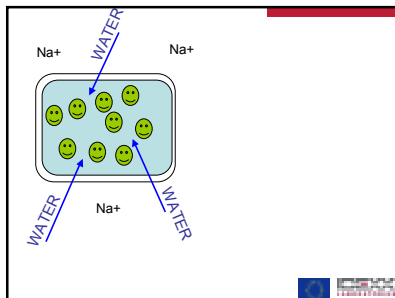
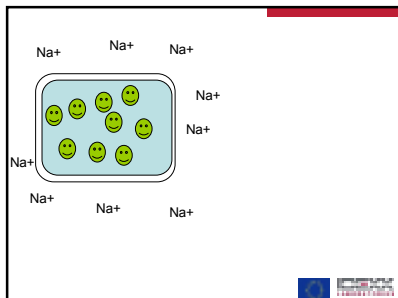
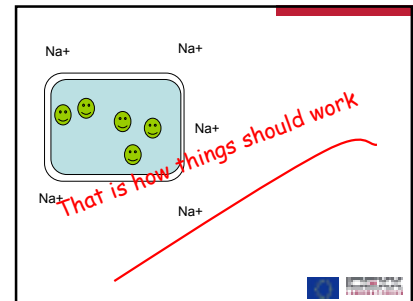
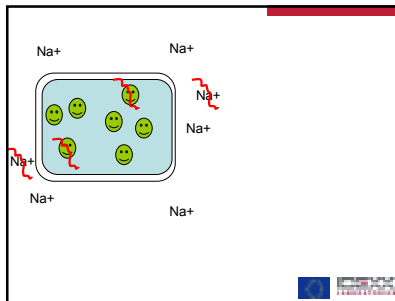
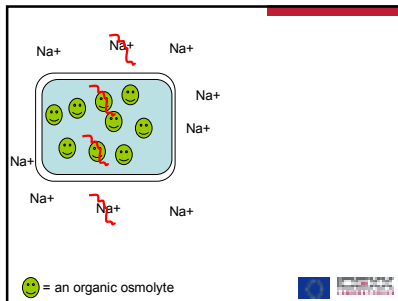
- Hypernatraemic patients should be encouraged to drink water
 - if they cannot, or will not, drink then administer free water (5% glucose, 0.45% NaCl with glucose)
- Monitor every 2 hours (at least)



Do not change the plasma sodium too quickly

- A change in total brain water of >10% is incompatible with life
 - smaller changes associated with neurological symptoms
- Organic osmolytes
- Severe neurological consequences if serum sodium concentration changed too quickly
- Serum sodium concentration should be monitored serially
 - change of < 0.5 mmol/L/hour





What have I learnt about sodium?

- Blood sodium concentrations do not tell you anything about body sodium concentration
- Blood sodium concentration is not an indicator of dehydration
- An abnormality does not usually require specific treatment
- Serum sodium concentration should be monitored serially**
 - change of < 0.5 mmol/L/hour

Chloride

- Chloride is a common electrolyte in the extracellular fluid
- Mean plasma Cl⁻ 110mmol/L
- Mean gastric juice Cl⁻ 140mmol/L
- Associated with the regulation of acid-base

Results: venous	
pH	7.48
pCO ₂	48mmHg
HCO ₃ ⁻	36mmol/L
Na ⁺	149mmol/L
Cl ⁻	89mmol/L
K ⁺	3.21mmol/L

Law of electroneutrality

- Total positive charges of the electrolytes are equal to the total negative charges of the electrolytes
- Body "allows" the concentration of chloride to vary in a passive manner

Plasma electrolytes			
Cation	mmol/L	Anion	mmol/L
Na ⁺	142	Cl ⁻	103
K ⁺	4	Bicarb ⁻	27
Ca ⁺⁺	5	HPO ₄ ²⁻	2
Mg ⁺⁺	2	SO ₄ ²⁻	1
Trace	1	Organic acids	5
		Protein	16
Total	154	Total	154

Law of electroneutrality

The anion gap

Plasma electrolytes			
Cation	mEq/L	Anion	mEq/L
Na ⁺	142	Cl ⁻	103
		Bicarb ⁻	27
		UA ⁻	
UC ⁺			
Total	154	Total	154

Unmeasured cations

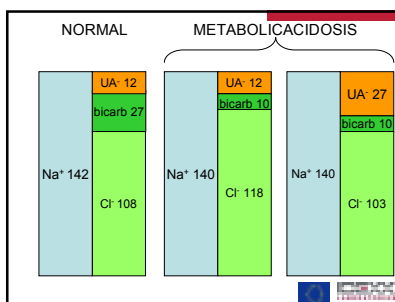
Unmeasured anions

Law of electroneutrality

Plasma electrolytes			
Cation	mEq/L	Anion	mEq/L
Na ⁺	142	Cl ⁻	103
		Bicarb	27
UC ⁺		UA ⁻	
Total	154	Total	154

Anion gap

$$[Na^+] + [UC^+] = ([Cl^-] + [bicarb] + [UA^-])$$

$$[UA^-] - [UC^+] = [Na^+] - ([Cl^-] + [bicarb])$$


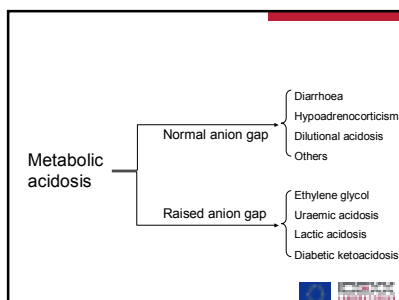
Why is the anion gap useful?

- Differentiating the general causes of metabolic acidosis
- Beware hyperalbuminaemia
- Anion gap greater than 16 mEq/L strongly suggests increased fixed acids in the blood

METABOLIC ACIDOSIS

Na ⁺ 140	UA ⁻ 12	bicarb 10	Cl ⁻ 118
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Na ⁺ 140	UA ⁻ 27	bicarb 10	Cl ⁻ 103
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Clinical approach to chloride disorders

- [Cl⁻] may change as a result of changes in water balance
 - observed chloride corrected for dilution/concentration
 - evaluation of [Cl⁻] must be done in conjunction with [Na⁺]

Dog:

$$Cl^- \text{ (corrected)} = Cl^- \text{ (measured)} \times 146 / Na^+$$

Cat:

$$Cl^- \text{ (corrected)} = Cl^- \text{ (measured)} \times 156 / Na^+$$

Na ⁺ 142	UA ⁻ 12	bicarb 27	Cl ⁻ 108
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Clinical approach to chloride disorders

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$$Cl^- \text{ (corrected)} = Cl^- \text{ (measured)} \times 156 / Na^+$$

Na ⁺ 142	UA ⁻ 12	bicarb 27	Cl ⁻ 108
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Na+	156 mmol/L (138 - 148)	High
K+	5.3 mmol/L (3.5 - 5.0)	High
Cl-	118 mmol/L (105 - 117)	High

Increased from the middle of reference interval by 13 units

Increased by 7 units

Na⁺ 156 mmol/L (138 - 148) High

K⁺ 5.3 mmol/L (3.5 - 5.0) High

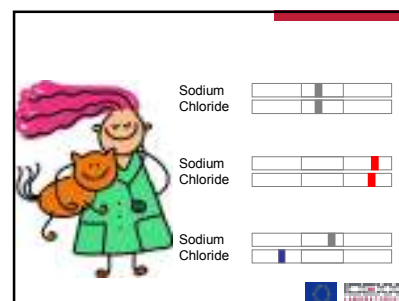
Cl⁻ 118 mmol/L (105 - 117) High

148 - 138 = 10

10 / 2 = 5

138 + 5 = 143

156 - 143 = increase of 13 units



Corrected hypochloraemia

- Usually associated with metabolic alkalosis
 - vomiting of stomach contents
 - some diuretics
- If concurrent hypokalaemia use KCl
- Otherwise NaCl (0.9%)



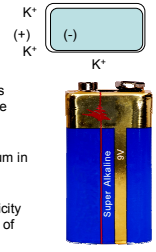
Corrected hyperchloraemia

- Beware potassium bromide treatment
 - measured as chloride
- Associated with:
 - metabolic acidosis with normal anion gap
 - IVFT with 0.9% NaCl
 - fluids 'spiked' with KCl
- Treat with sodium bicarbonate if the pH below 7.2

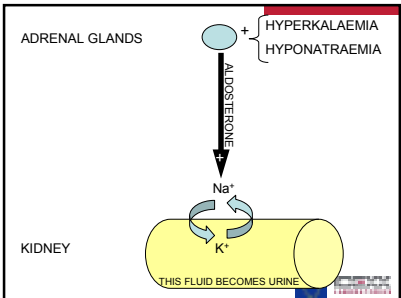
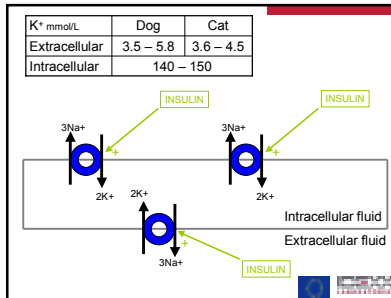
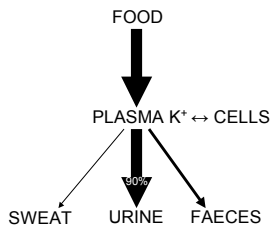


Potassium

- Just like a 'battery' some cells within the body have a voltage
 - ve inside the cell membrane
 - +ve outside the cell membrane
- The concentration of potassium in the extracellular fluid can dramatically alter this voltage
- Important role in the automaticity of the heart and transmission of signals in the nerves



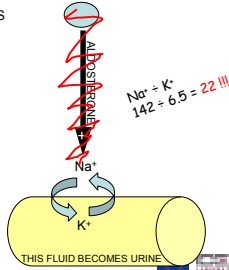
How is potassium balance maintained?



ADRENAL GLANDS

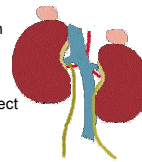


KIDNEY

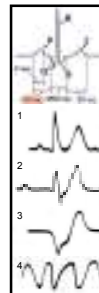


Hyperkalaemia

- Long term hyperkalaemia uncommon if renal function normal
- Increased oral intake is an unlikely cause
- Clinical manifestations reflect changes in cell membrane voltage
 - muscle weakness
 - changed electrocardiogram (ECG)



Hyperkalaemia



- ECG changes
- Some vets consider they do not need to measure potassium if they have an ECG machine
- The waveform cannot be used to predict a plasma potassium concentration

Causes of hyperkalaemia

- Urinary bladder rupture
- Urinary tract obstruction
 - treatment must focus on removing obstruction and restoring urine flow
- Kidney failure



Causes of hyperkalaemia

- Drugs
 - some diuretics
 - ACE inhibitors
- Iatrogenic ("vet induced")
 - potassium-rich fluid therapy ('drips')
 - excessive oral potassium supplementation



Causes of hyperkalaemia

- Significant tissue destruction
- Massive amounts of intracellular potassium released into the circulation
- Chemotherapy
- Severe trauma



Causes of hyperkalaemia

- Metabolic acidosis
- Intracellular translocation of hydrogen ions



Treatment of hyperkalaemia

- Mild (5.9 to 6.4 in dogs, 4.6 to 6.4 mmol/L in cats)
 - intravenous fluids with low potassium
- Moderate (6.5 to 7.5 mmol/L)
 - as above with insulin
- Severe (>7.6 mmol/L)
 - calcium gluconate or
 - sodium bicarbonate



Clinical features of hypokalaemia

- May have no clinical signs
 - not usually apparent until serum K^+ < 3 mmol/L
- Muscle weakness
- Cardiac muscle dysfunction (arrhythmias)



Causes of hypokalaemia

- Iatrogenic causes
 - some diuretics
 - aggressive IVFT
 - excessive insulin
- Chronic vomiting
- Chronic kidney failure
- Monitor in any patient that is:
 - NOT eating
 - receiving intravenous fluid therapy



Cats and hypokalaemia

- Cats seem particularly prone to developing hypokalaemia
- Many cats receiving standard intravenous fluids benefit from K^+ supplementation
- Cats with chronic renal failure may benefit from oral supplementation



Useful tips when measuring $[K^+]$

- Tourniquet released after maximum of one minute to avoid venous stasis
 - haemoconcentration and drives potassium out of cells
- Avoid 'fighting' with patient
- Analysis without delay



Causes of hyperkalaemia

- Laboratory artefact: occurs either during or after sampling
- NOT a problem for the animal
 - haemolysed blood sample (especially puppies and Akita dogs)
 - leucocytosis
 - thrombocytosis
 - Tri-K EDTA



Case example: ketoacidotic cats

- A cause of hyperkalaemia AND hypokalaemia
- Body K depleted
 - polyuria and insulin deficiency, vomiting
- Ketoacidotic patients
 - in general acidosis is associated with movement of K⁺ ions from ICF to ECF
 - rapidly reversed when insulin therapy and IVFT commenced
- Potassium monitoring is vital



Case example: congestive heart failure

- Diagnosed with congestive heart failure three years ago
- 'Accumulated' drugs over that time: frusemide, enalapril and spironolactone
- Presented collapsed with neurological signs

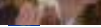


	Plasma	Na ⁺	K ⁺
Frusemide		↓	↓
Spironolactone		↓	↑
ACEi		↓	↑



When to test

- Any patient that presents in an emergency
- Any sick patient
- Any patient receiving intravenous fluid therapy (particularly those receiving large volumes quickly)
- Any patient that isn't eating



When to test

- Any sick patient prior to anaesthesia
- Any patients
 - receiving multiple medications for heart failure
 - with chronic renal failure
 - receiving potassium supplementation
- Any patient worth taking blood from?



Any questions?

