

How much is too much??

Dr Chris Hill
Christchurch ED



Exercise induced hyponatremia

- Case
- Who and why?
- Physiology
- How much?
- Prevention
- The future

Case: Mr H

- A 43-year-old male collapsed on the third day of an 8-day guided trek
- Complained of abdominal pains and leg cramps for 24 hours leading up to the collapse
- He collapsed suddenly and had a generalised seizure
- Evacuation to a hospital was impossible until the following day owing to the remoteness of the Trail
- Best Glasgow Coma Score of 10 until the following morning
- Evening before and day of the collapse, fellow trekkers and guides encouraged him to drink large amounts of water
- He was evacuated by helicopter and taken to Port Moresby Hospital
- Na 107 mmol/L (135–145 mmol/L).
- Other investigation results included potassium 5.8 mmol/L (3.2–4.5 mmol/L), urea 12.8 mmol/L (3.0–8.0 mmol/L), and creatinine 108 umol/L (70–120 umol/L)

Who and why?

- EAH is a modern phenomenon that has emerged with the increase in popularity of ultra-endurance events
- Encouragement to drink as much fluid as possible to avoid heat illness and improve performance.
- EAH is usually a dilutional hyponatremia, with an absolute increase in total body water.
- The primary aetiology is the consumption of hypotonic fluids in excess of body fluid losses.

Who and why?

- Low body weight
- Female gender, (three times more common in women than in men in the 1997 New Zealand Ironman triathlon)
- Greater than 4 hours of continuous exercise
- Slow performance pace, race inexperience,
- Excessive drinking behaviour,
- Altered renal excretory capacity,
- Hot environmental conditions
- NSAID use prior 24 hours



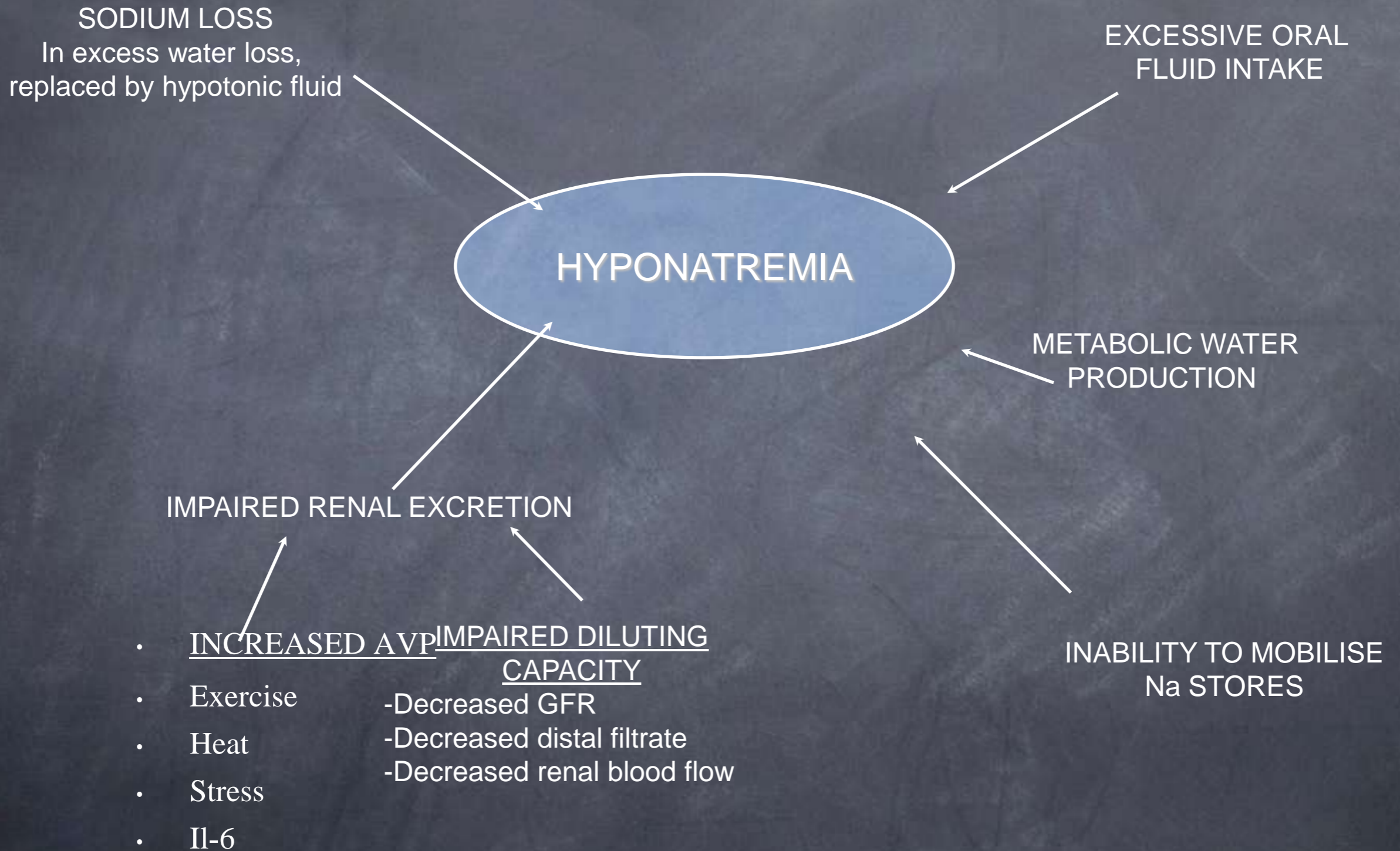
Who and Why?

- 2002 Boston Marathon **13%** of 488 runners studied had hyponatremia
- **0.6%-1% had critical hyponatremia** (Na of 120 mmol/L or less).
- 330 athletes ultramarathon race. In this study, 58 (**18%**) **were hyponatremic** (Na 135 mmol/L)
- **11 had severe hyponatremia** (Na 130 mmol/L).
- Studies of other endurance events have reported the incidence of hyponatremia to be up to 29%
- In collapsed athletes **6 to 30%** of these athletes had serum sodium values below normal

Symptoms

- Similar to dehydration, need good history
- Anorexia
- Nausea and vomiting
- Difficulty concentrating
- Confusion
- Lethargy
- Agitation
- Headache
- Seizures

Simple physiology



Simple physiology

- maximum water excretory capacity of the kidneys is between 750 and 1500 ml/h
- In combination with fluid losses from sweating and insensible losses (which may be in excess of 500 ml/h), most athletes should be able to consume fluids in excess of 1500 ml/h before retaining weight and increasing TBW
- Non osmotic increase in AVP secondary to stresses, exercise etc
- Mobilisation of osmotically inactive sodium in some athletes

Simple physiology

- Poor blood flow to gut reversed on completing event and increased reabsorption fluid
- Arterial Na much lower than venous

So how much?



So how much?

- The first is to drink only according to thirst and no more than 400 to 800 ml/h
- Currently, there is insufficient evidence to support the suggestion that ingestion of sodium prevents or decreases the risk for EAH
- Neither is there any evidence that consumption of sports drinks can prevent the development of EAH . Again, most commercial sports drinks are hypotonic with a sodium content of 10 to 20 mmol/L (230 to 460 mg/L)

Management: Mild symptoms

- Most cases will go unnoticed
- Na 130-135 for fluid restriction
- Observe until spontaneous diuresis
- Avoid 0.9% saline as exacerbates

Management: Severe symptoms

- Na <120 mmol/L) or symptomatic
- First is the assumption that all EAH is acute (<48 h)
- The second consideration is that no cases of osmotic demyelination syndrome have been reported with the treatment of EAH
- **100 ml of 3% saline over 10 min**
- Case series by Ayus *et al*, six of seven marathon runners were treated with hypertonic saline for hyponatremia, cerebral oedema, and noncardiogenic pulmonary oedema.
- **All six of the athletes who received hypertonic saline made a full recovery.** Of the five athletes who had **follow-up MRI 1 yr after treatment were normal.** The one athlete who was **not treated with hypertonic saline died**

Management: Seizure

- A, B, C, D (on't ever forget glucose)
- Hypertonic saline push
- 3ml/kg of 3% over 3min and repeat if necessary
- Usual seizure management

So How did he do??

- He was treated with **3500 mL of 0.9% saline** over approximately 36 hours.
- His condition improved, and he was transferred to a tertiary referral.
- On arrival at this hospital, his sodium had increased to **132 mmol/L**, and he was alert but still mildly confused.
- He remained in the hospital a further 6 days.
- His creatine kinase peaked at 18600 U/L (<200 U/L).
- **CT of his head was normal.**
- An electroencephalogram suggested a toxic or metabolic encephalopathy, while neuropsychological testing was consistent with **mild hypoxic brain injury**.
- He showed no clinical signs of **central pontine myelinolysis**.
- No magnetic resonance imaging was performed. His cognitive function improved during his admission.
- **He returned to work 2 weeks after discharge**

Our role

- Education
- Recognition
- Observation
- Don't make things worse

The technology in the wilderness

- I-stat
- Blood gas and electrolytes on the move
- \$NZ8-9,000



Any questions?

