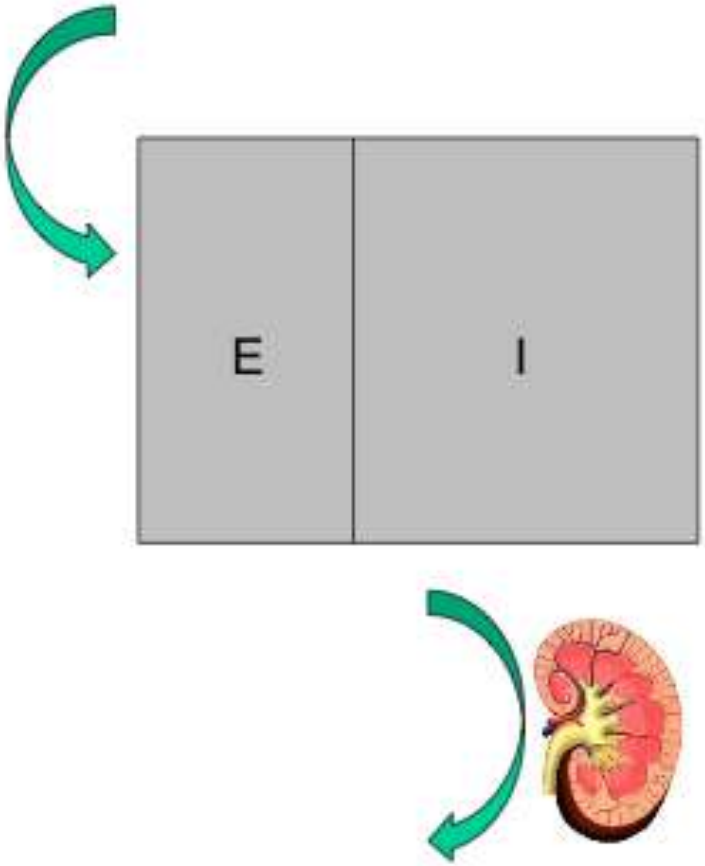
Misadventures in salt & water, as well as in acid-base balance

Entertaining you is
Friedrich C. Luft, Berlin

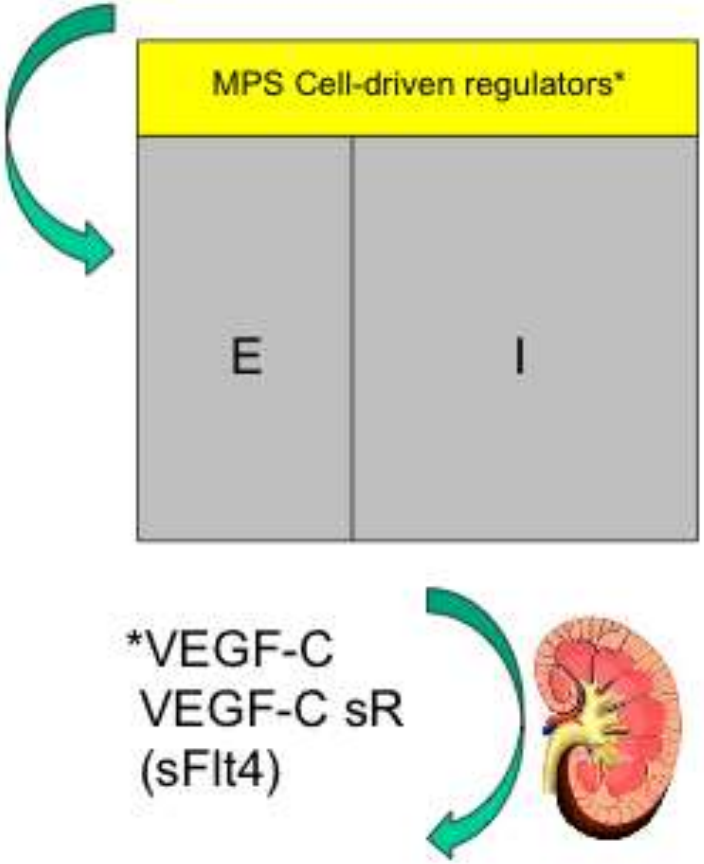
Charité

We teach this:



We found this:

Third compartment NaCl in skin, muscle, and elsewhere

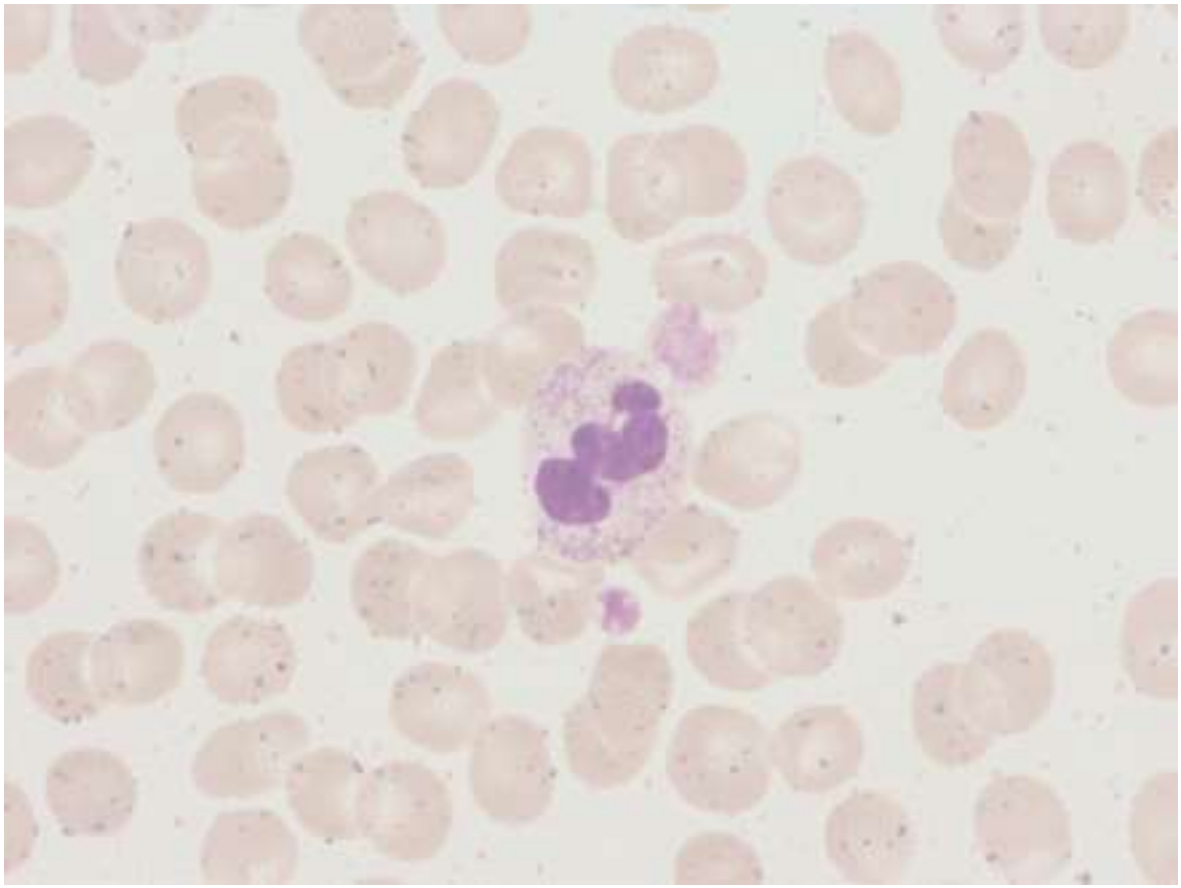


*VEGF-C
VEGF-C sR
(sFlt4)

Pflugers Arch 2015

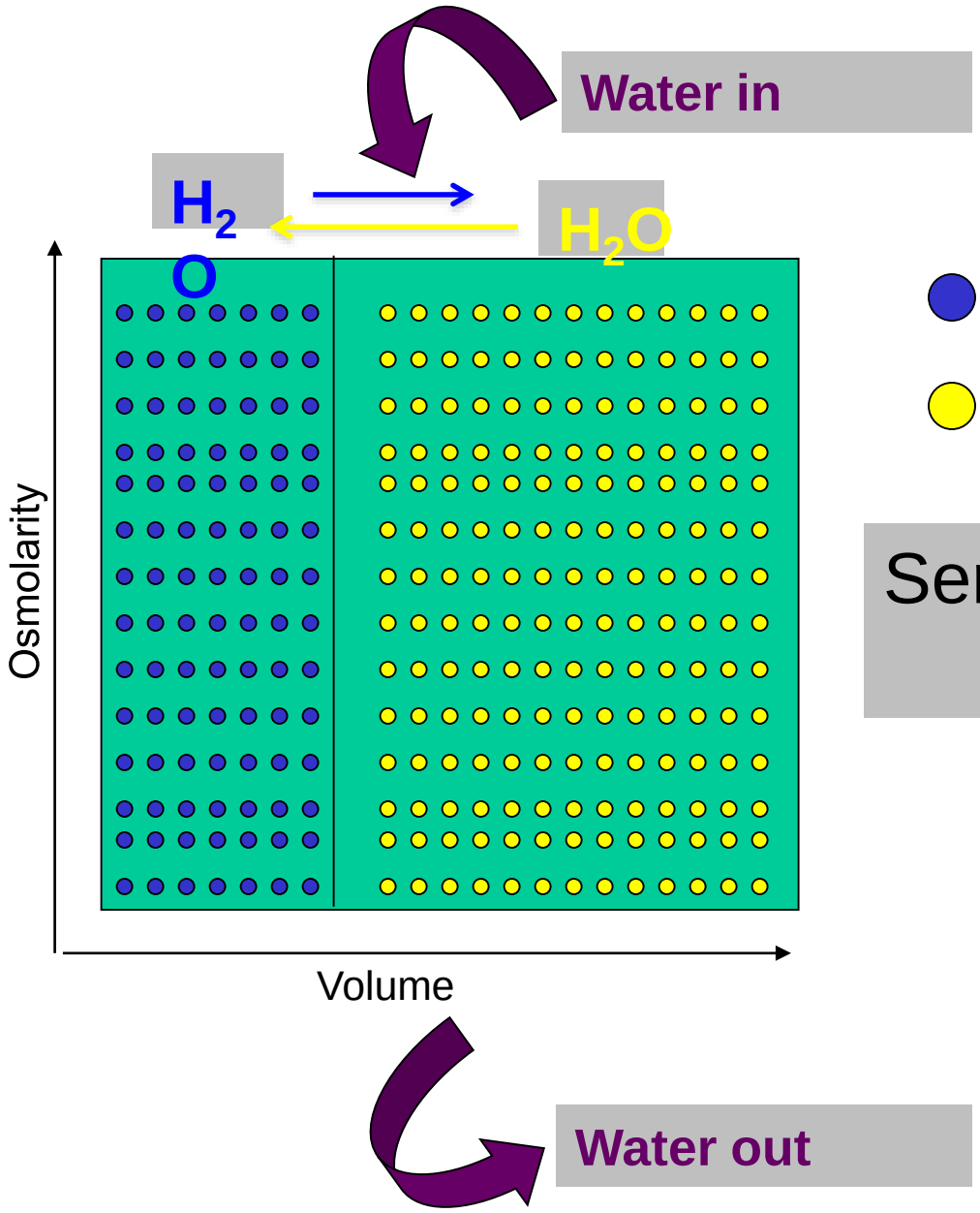
Don’t just “do something” – stand there

- 68 year-old woman presents disoriented at 18:00; had undergone tooth extraction that morning and, aside from a life-long mild bleeding tendency, had been quite normal
- BP 130/85, pulse regular, respirations 18/min no localizing findings, no edema
- Na 118, K 3.6, glucose 8, urea 4 (all mmol/L)
- What now?



An oil-immersion field showing a normal neutrophil flanked by two giant platelets (Bernard-Soulier syndrome). She had been given **desmopressin**. In addition, it had been hot so she was advised to "drink **lots of water**"

Serum-Na depends on TBW, Na and K

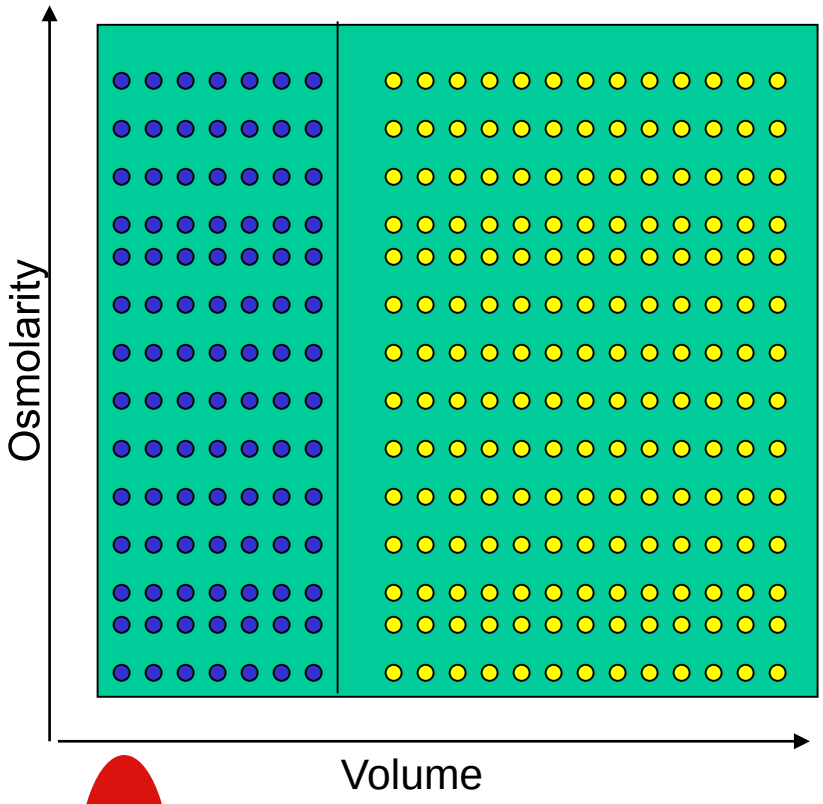


Na

K

$$\text{Serum Na} \approx \frac{Na_{exch} + K_{exch}}{\text{Total-body } H_2O}$$

Edelman formula

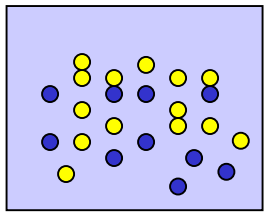


Na

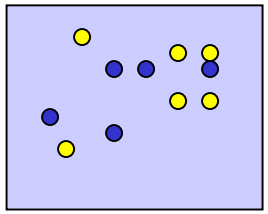
K

$$\text{Serum Na} \approx \frac{Na_{exch} + K_{exch}}{\text{total-body } H_2O}$$

$$\textbf{Clearance } H_2O \text{ (e)} = V\left(1 - \frac{UNa+UK}{SNa}\right)$$



Lots of spheres
= little H_2O
Cl_{H2O}(e) neg



Few spheres
= much H_2O
Cl_{H2O}(e) pos

When UNa+UK >SNa the Cl_{H2O}(e) neg and serum Na must fall

When UNa+UK < SNa, the Cl_{H2O}(e) pos and serum Na must rise

Actually, serum Na increased a little faster than we wanted so we infused some free water

Table 1.
Serum and urine values (mmol/L) over a timeframe of 38 h

Time	**18.00**	**06:00**	**21:00**	**08:00**
			Glucose 5% infusion	
Serum				
Sodium	118	125	138	138
Potassium	3.6	3.0	5.4	4.4
Urine				
Sodium	16	–	24	43
Potassium	7	–	9	16

Had we given 3% saline, serum Na would have increased even faster

Iatrogenic SIADH

Clin Kidney J 2013;6:96-97

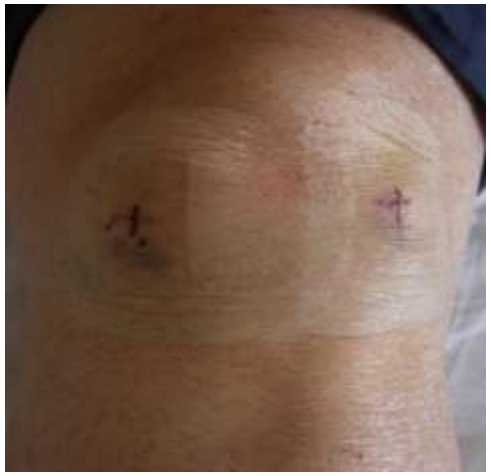
Paradoxal hyponatremia with isotonic electrolyte infusions

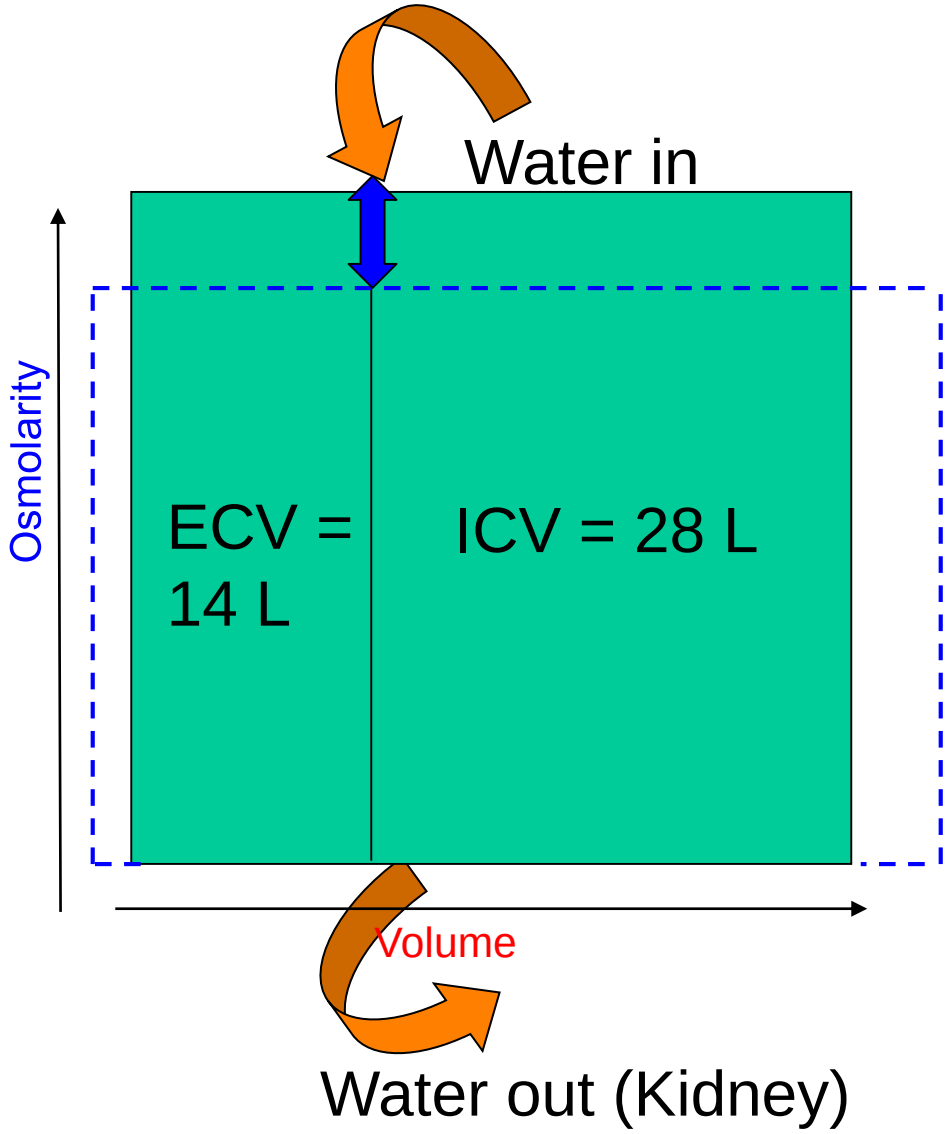
- 65 year-old woman has meniscus surgery. At that time her Na was 141 mmol/L. She is hypertensive and receives HCTZ and ACEI, as well as NSAID + opiates for pain
- Lots of nausea and vomiting post-op
- Two days later Na 120, K 3.2 in emergency
- She receives Ionosteril (Na 137) + KCl 20 mmol/l
- Two days later Na 116, K 2.8. Same therapy given again

How is it that the serum Na got worse?

- Shortly thereafter, admitted with Na 98, K 2.3, glucose 6, urea 3 (all mmol/L) and grand-mal seizure
- Exam BP 200/110 mm Hg, patient is post-ictal (comatose)
- She has no edema
- What is her plasma osmolarity?

(98+2.3)x2 + 6 + 3 = ?





At bwt 70 kg → 12 L Excess!!

Water excess = 0.6 x Bwt (kg) x (1- $\frac{\text{Na measured}}{140}$)

Appropriate reasoning with hyponatremic patients who have high ADH (Uosmo > 300)

What caused ADH release?

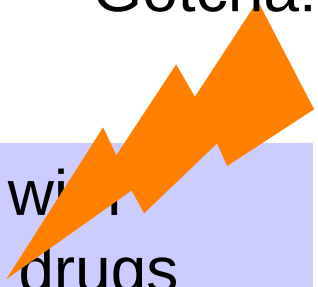
Nonosmotic stimuli with volume disturbance

- Low ECV
- High ECV (Edema)

(Heart-, Liver-, Kidney, Adrenal You will find them here)

Nonosmotic stimuli with euvolemia (SIADH)

Gotcha!



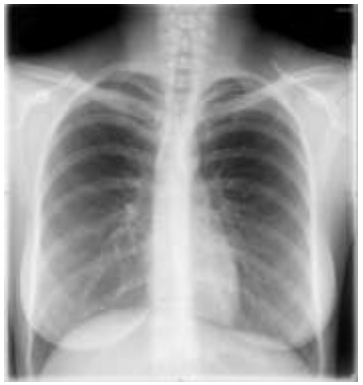
Euvolemia, wi… diseases or drugs that cause ADH release

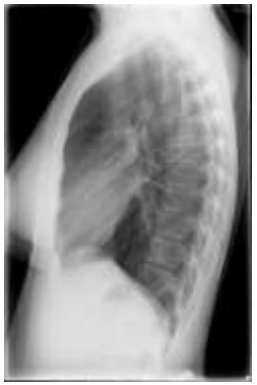
Who is teaching fluid and electrolytes?

A 22 year-old woman comes to emergency because of weakness, vomiting and abdominal discomfort. She has felt poorly several weeks but recently worse. She has Hashimoto's thyroiditis and receives replacement

Hb 14 g/dL, Na 122, K 4.8, Cl 92, glucose 2.5, urea 5 (all mmol/L). Creatinine 0.7 mg/dL. The pH was 7.17, PaO_2 95, $PaCO_2$ 28 (mm Hg), HCO_3 11

Ultrasound indicates that appendicitis "cannot be ruled out"

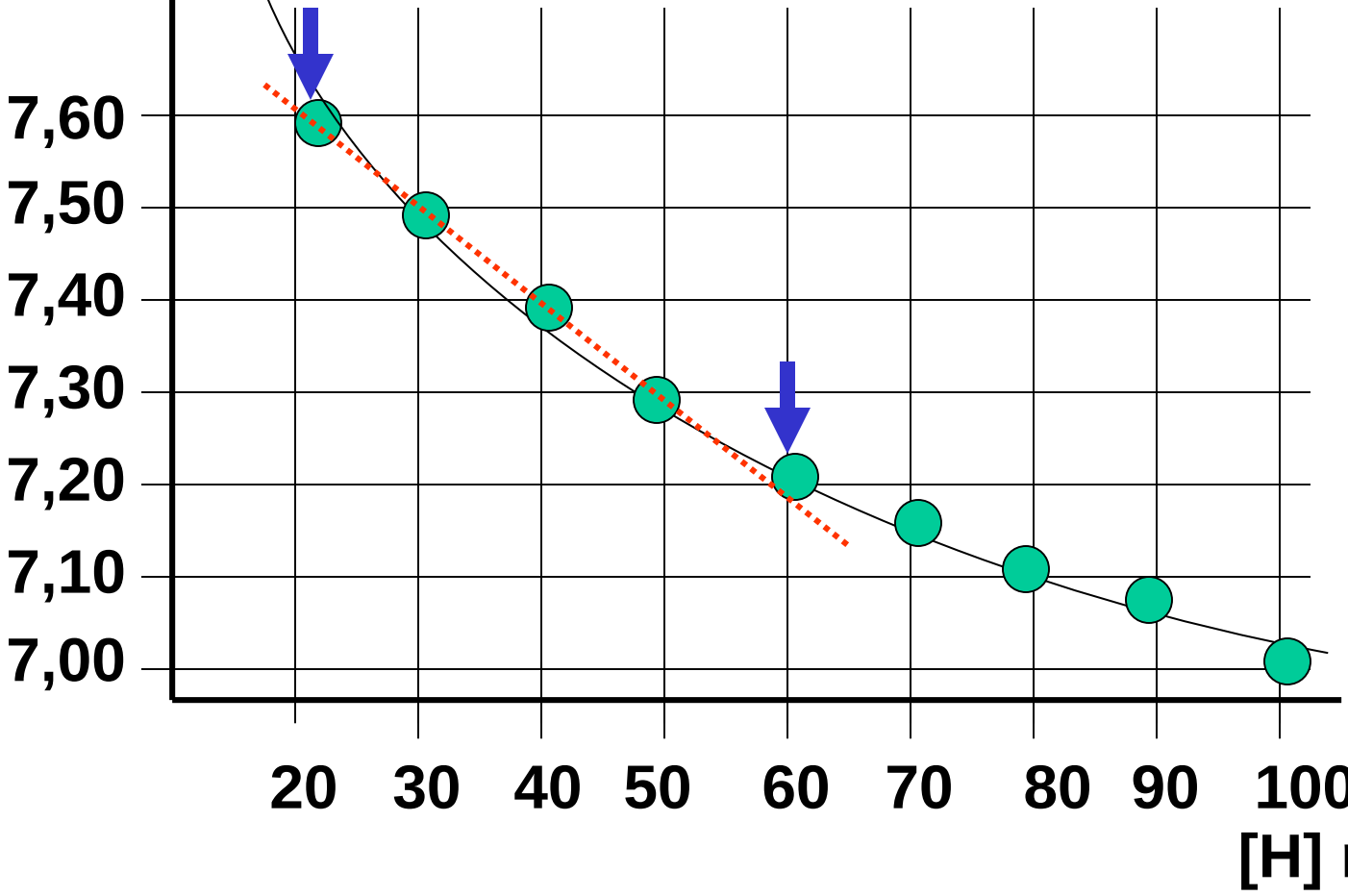




„puissance“ Hydrogen

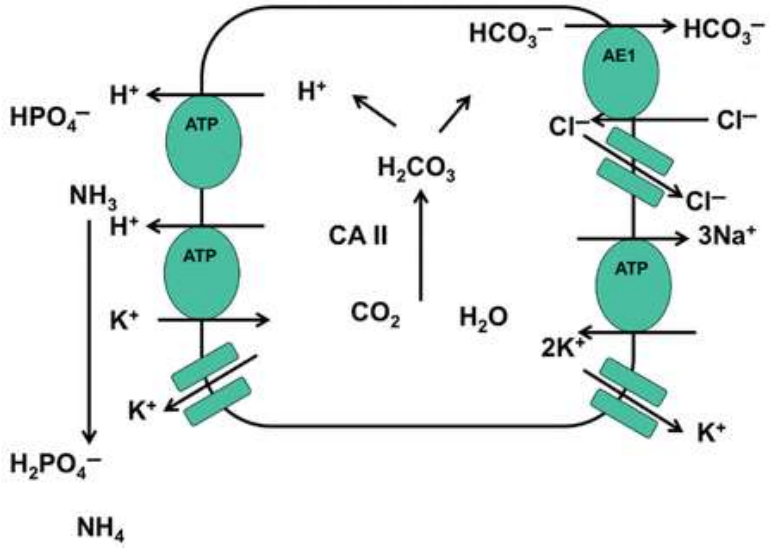
Sorensen & Hasselbalch

$[H^+] = 7{,}XY \qquad 80\text{-}XY = H^+$



Would have fired them all on the spot!

$$[H] = K \frac{H_2CO_3}{HCO_3} \quad (K = \text{Dissoziationskonstante})$$

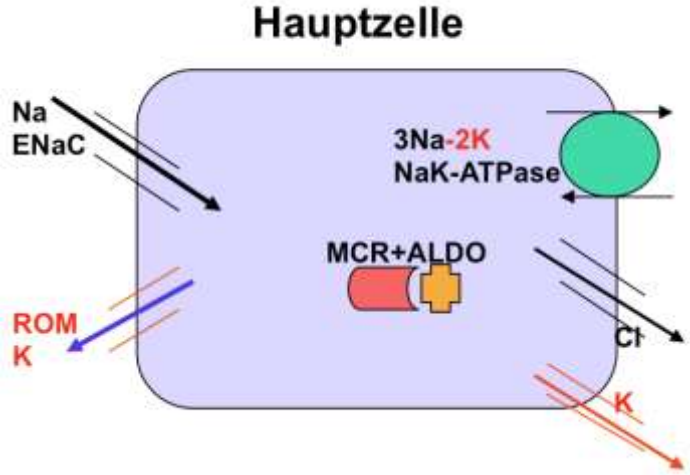


$H = (24 \times PaCO_2)/HCO_3$

$H = (24 \times 28)/11 = 61$ (nmol/L)

$AG = 122 – (92 + 11) = 19$

etc



Next day, we are called because she is no better.
BP 105/70 mm Hg. She becomes unconscious when held upright. Her blood labs are unchanged.

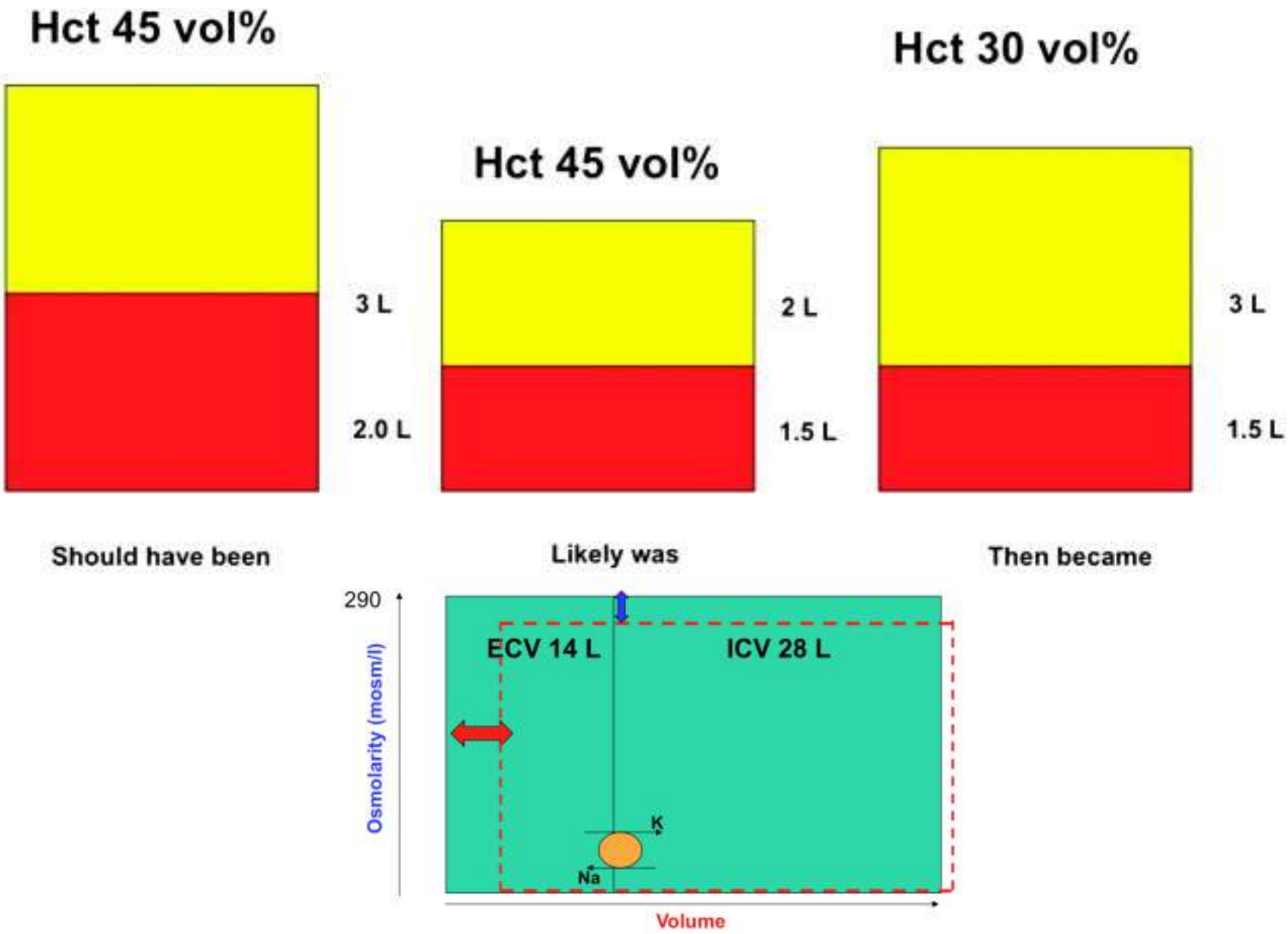
Urine Na 135, K 29, Cl 69 (mmol/L), Uosm 644

Stat cortisol 3.1 µg/dL, after ACTH 3.2 µg/dL
Aldo (took a few days) “low”
ACTH 1250 (normal <40)

Only half of Addision’s disease patients have hyperkalemia

After hydrocortisone and several liters ionosteril her Hemoglobin dropped to 10 g/dL without any blood loss

Clin Kidney J 2012



Addision's disease (polyendocrine syndrome)

Andy is a young Canadian visiting Brandenburg

His ancestors had emigrated on the “Hopewell” in 1761
Andy runs his Kawasaki into a tree and lands with our neurosurgeons. They drain an epidural hematoma.
Post op they call us because Andy “pees a lot”.

Na 166, Cl 132, K 4, HCO_3 22 (mmol/L)
Crea 230 µmol/L, urea 10, glucose 5 (mmol/L)
Urine output 8 L/24 h
IV input around 7 L/24 h

Diuresis?

Water or solute?

Can Andy concentrate his urine?

What does he require?

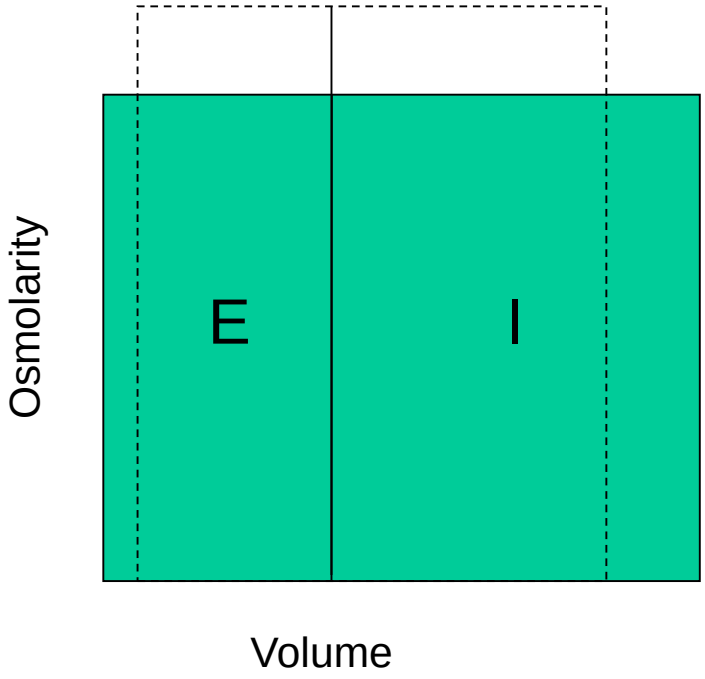
Solute diuresis

Glucose
Urea
Mannitol
Xylite
NaCl or other

Water diuresis

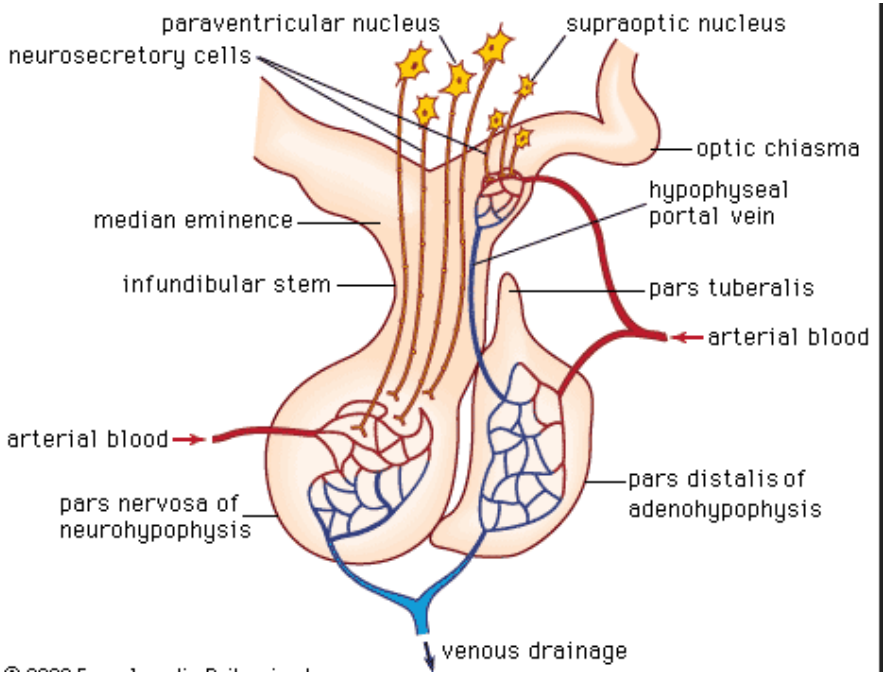
Diabetes insipidus
Central
Nephrogenic

Andy had a water diuresis!

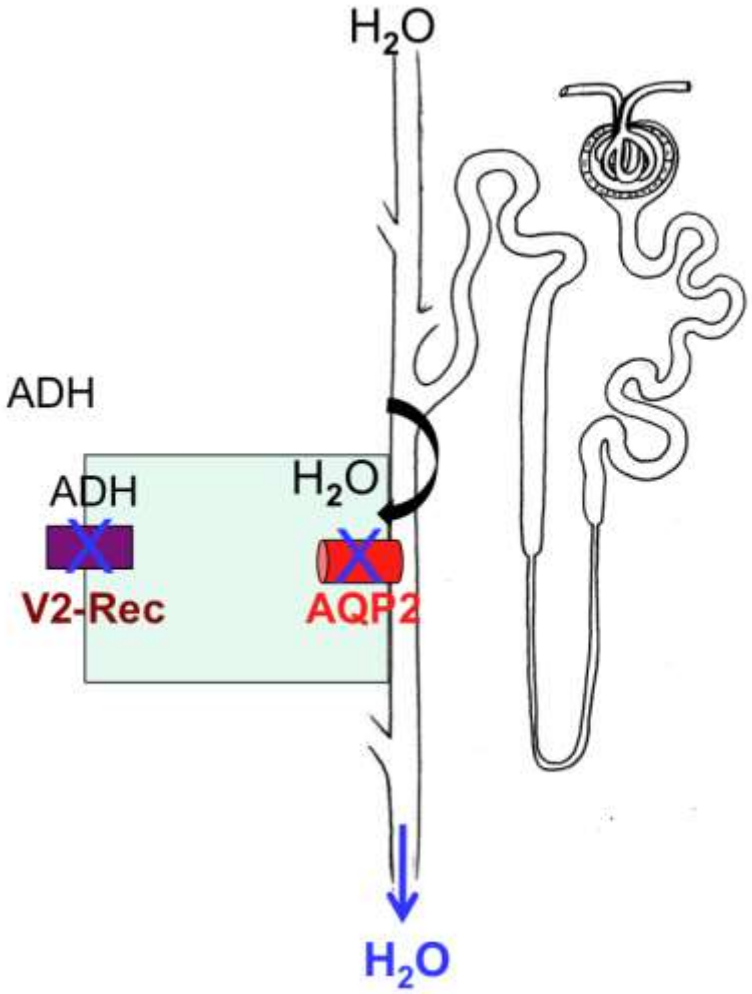


Andy had a sister who “peed” about half as much as he did

ADH release disturbed (central diabetes insipidus)



ADH is there, kidney does not react (nephrogenic DI)



Infections, tumor, trauma, postpartum

Chief has good experience with this treatment!

64 year-old patient, hypertensive, stroke, has PEG. Transferred to convalescence with HCTZ Na 134

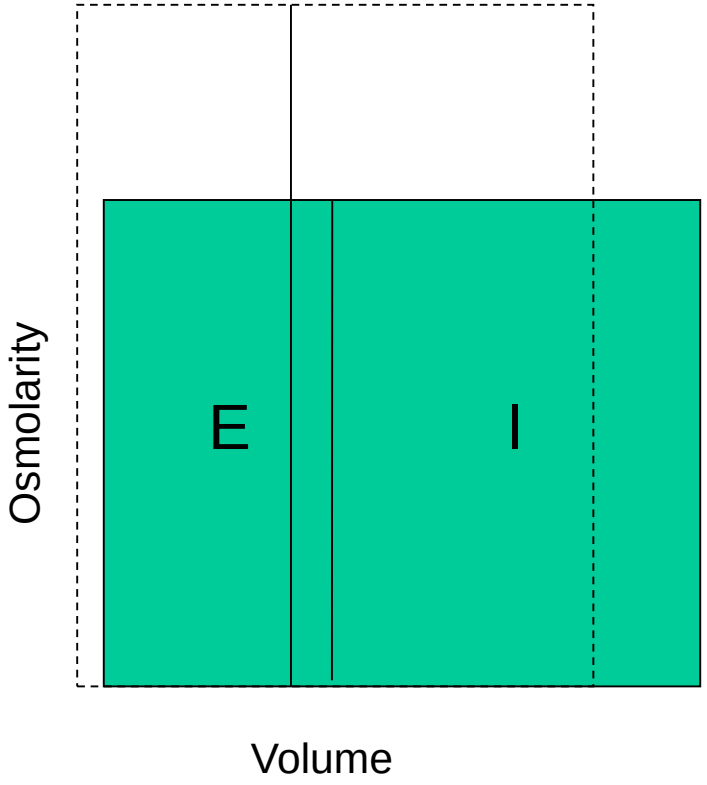
10 days later transferred by to us with Na 199, K 2.1 Cl 168 (all mmol/L), mild hyperchloremic acidosis, creatinine 299 µmol/L

BP 140/96 mm Hg. Patient comatose, findings were rales and copious edema

How do you get hypernatremia and edema?

Patient had received teaspoons of salt per PEG for “hyponatremia”, 330 mmol/day daily for 10 days.

Salt poisoning by accident or by physicians



Urine Na 133, urine K 46 (mmol/L)

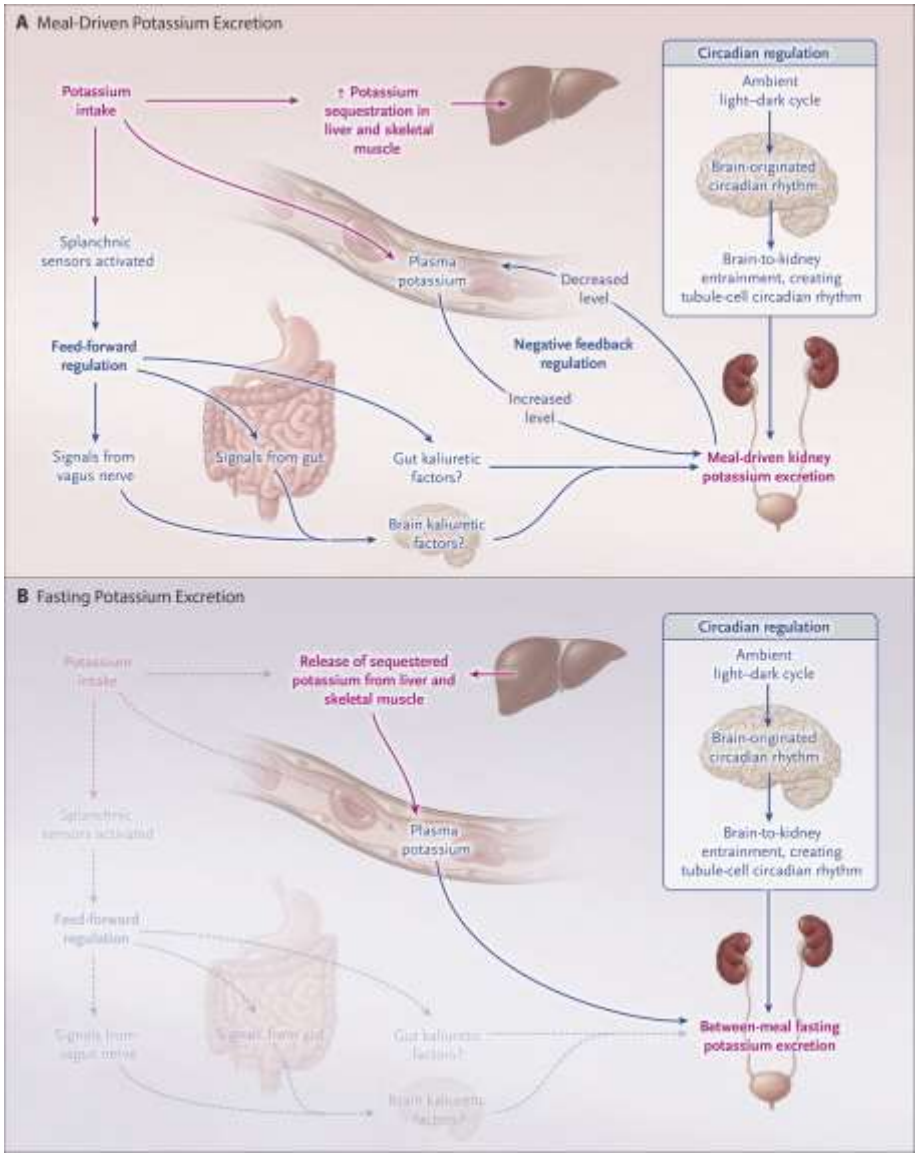
$$\text{Water „deficit“} = 0.6 \times \text{Bwt (kg)} \times \left(\frac{\text{Na measured}}{140}\right) - 1$$

= 14 L (but we could not just give this patient 14 L water)

Since patient was extracellular volume expanded we relied on slow hemofiltration and careful “free-water” (5% glucose) infusions

To our surprise, he survived little worse for wear

Salt poisoning



98% K inside cells (ICV)

2% (70 mmol) ECV

5 bananas and you die!

“Feed forwards”
“Feed backwards”

N Engl J Med 2015

Paroxysmal Hyperkalemia

50 year-old woman developed “hyperkalemic” episodes 3 years earlier. Her creatinine was 2.5 mg/dL. She had interstitial nephritis but was stable.

She could “sense” the attacks and would appear in emergency with potassium >8 mmol/L. Na and Cl would be unremarkable. 24 hour urine showed normal Na and K excretion (80 mmol/24 h)

Episodes were dramatic with tetraplegia and strange EKGs

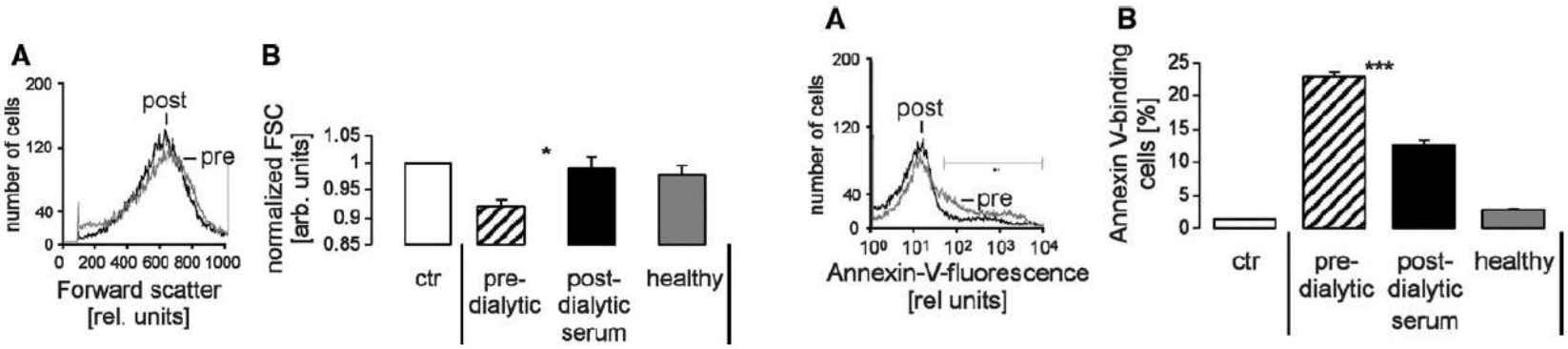
She was treated with furosemide, Resonium®, glucose, insulin, HCO_3, acute dialysis, and then a defibrillator was placed.

She had gene diagnostics for periodic paralyses of various sorts.

A diagnosis of eryptosis was considered

Finally FCL was consulted.

She was extensively worked up for eryptosis



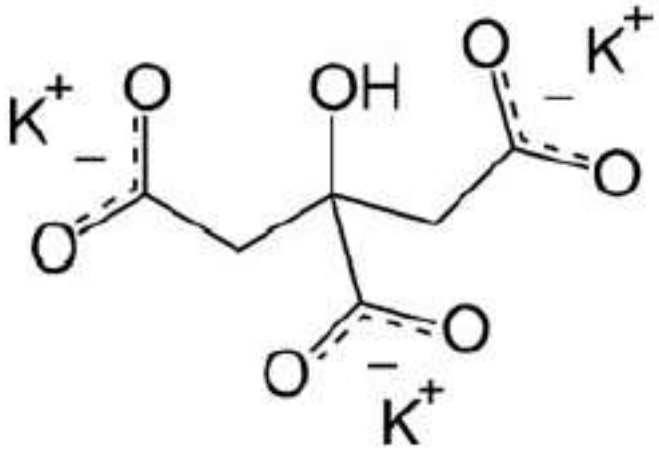
Eryptosis is erythrocyte apoptosis. It could release K

I asked about her acid-base status (it is OK!!)
But what about during the attacks?

Hmmmm?

Finally, she came in with an attack and vomited:
Vomit K 190 mmol/L, vomit Na 48 mmol/L,
vomit citrate (some time later) >5 g/L

The patient denied “everything” but the episodes stopped.



Die trinkfertige Lösung enthält: 40,0 mmol Kalium-Ionen (entsprechend 1,56 g Kalium) mindestens 13,3 mmol Citrat-Ionen

Eryptosis plus Munchhausen’s syndrome

NDT plus 2009

Our neurologists deal with tetraparesis

56 year-old patient from the “Wedding”
cannot get up from the couch

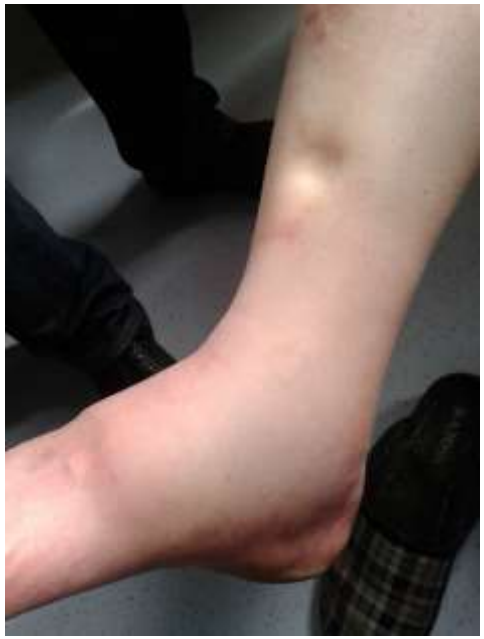
He shows up in our emergency and is seen by a surgeon

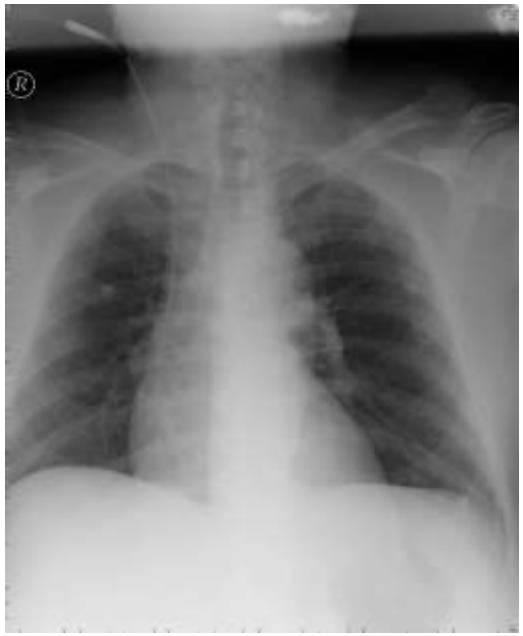
“Must be a neurological disease” They keep him for 8 days

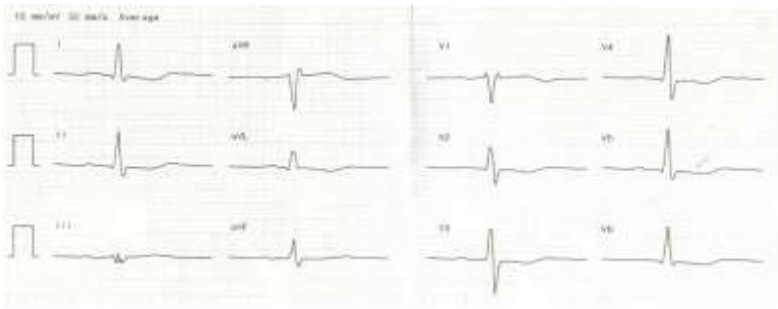
Neurologists find normal BP and tetraparesis
They do lumbar puncture, CT, MRI, EMG, multiple measurements are made. They suspect thiamine deficiency

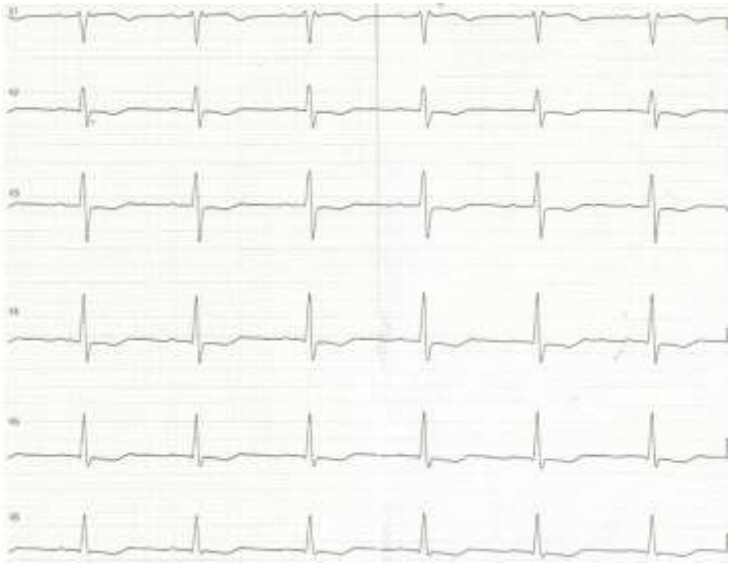
Diagnoses: sensory-motor demyelinizing polyneuropathy, carpal tunnel syndrome, alcoholism (intake 150 g/day).
Their quality-control rating was “excellent at 100%”

Patient returns to couch but is not better, returns to emergency
This time he is seen by internal medicine





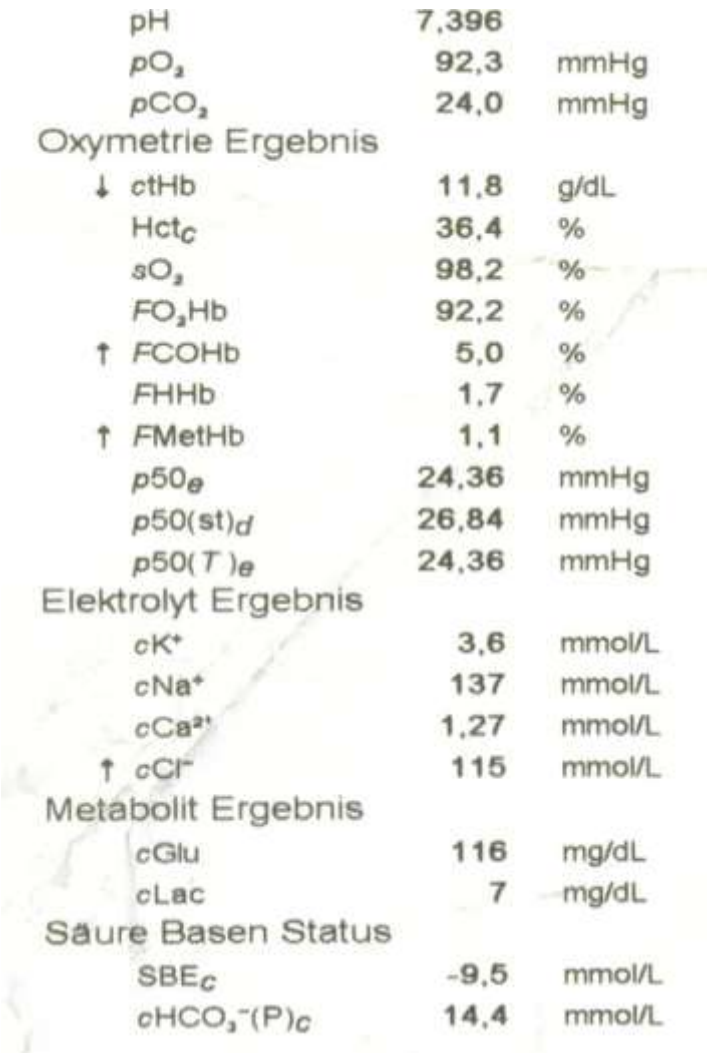




Na 145, Cl 111, K 1.5 mmol/L

Phosphate 0.44, Mg 0.86 mmol/L

Patient is sent to ICU and they pour in KCl
Next morning: “would you guys be interested in a patient with a K 1.5 mmol/L?” We allowed that we would.



	pH	7,396	
	pO_2	92,3	mmHg
	pCO_2	24,0	mmHg
Oxymetrie Ergebnis			
↓	$ctHb$	11,8	g/dL
	Hct_C	36,4	%
	sO_2	98,2	%
	FO_2Hb	92,2	%
↑	$FCOHb$	5,0	%
	$FHHb$	1,7	%
↑	$FMetHb$	1,1	%
	$p50_e$	24,36	mmHg
	$p50(st)_d$	26,84	mmHg
	$p50(T)_e$	24,36	mmHg
Elektrolyt Ergebnis			
	cK^+	3,6	mmol/L
	cNa^+	137	mmol/L
	cCa^{2+}	1,27	mmol/L
↑	cCl^-	115	mmol/L
Metabolit Ergebnis			
	$cGlu$	116	mg/dL
	$cLac$	7	mg/dL
Säure Basen Status			
	SBE_C	-9,5	mmol/L
	$cHCO_3^-(P)_C$	14,4	mmol/L

First set of blood gases

Somewhat overcompensated metabolic acidosis without broad anion gap.

Hypokalemia looks improved

Urine pH 7.0, Na 65, K 15, Cl 67 (mmol/L)

Urine anion gap was negative (ammonium chloride excretion)

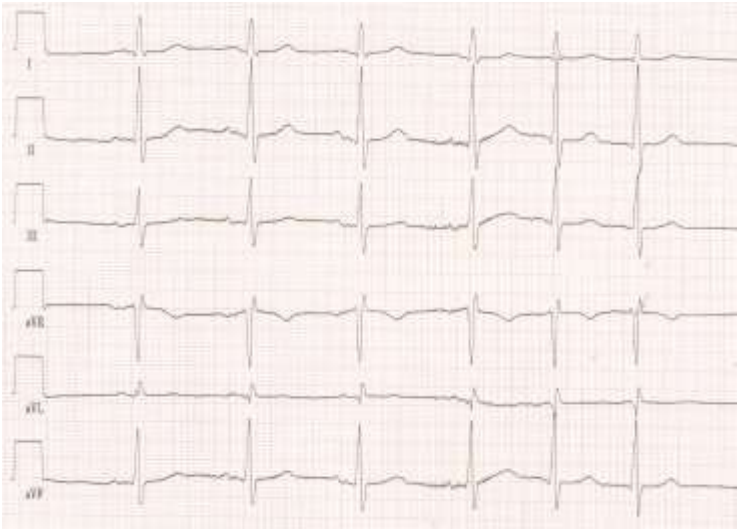
Transtubular K gradient 12

At discharge 14 days later:
Na 138, K 3.6, Ca 2.41, Cl 99 mmol/l
Crea 0.76 mg/dl, Urea 17 mg/dl,
Phos 1.18, Mg 0.78 mmol/l

BGA: pH 7.43 pO_2 84,7, pCO_2 31.5, HCO_3 22.5

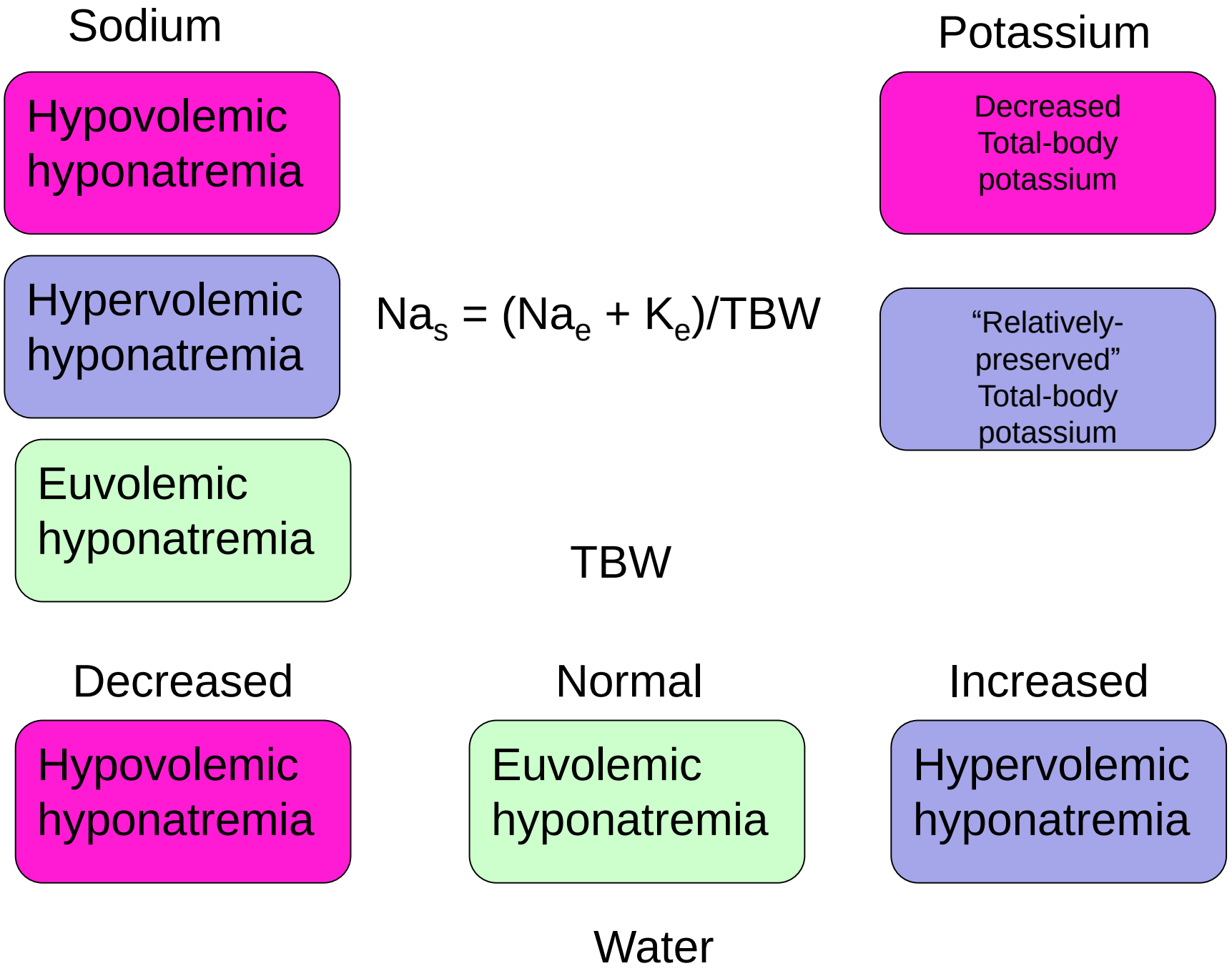
Urine: pH 7.0, Na 92, K 24, CL 110 mmol/l (negative UAG)

TTKG 4.4
Tub Phosphate reabsorption 85.5



Alcohol-associated RTA

N Engl J Med 1993


Sodium
Potassium
Hypovolemic hyponatremia
Decreased Total-body potassium
Hypervolemic hyponatremia
$Na_s = (Na_e + K_e)/TBW$
“Relatively-preserved” Total-body potassium
Euvolemic hyponatremia
TBW
Decreased
Normal
Increased
Hypovolemic hyponatremia
Euvolemic hyponatremia
Hypervolemic hyponatremia
Water

I refer you to recent N Engl J Med reviews (2014-15)

- Hyponatremia is always a defect in water excretion. Check to see if a dilute urine can be made (UNa and UK).
- ADH presence can most easily be determined by urine osmolarity. It can be calculated.
- Extracellular volume assessment seems simple but is inexact. Nonetheless, edema (yes/no), orthostatic heart-rate and BP changes must always be documented.
- How physicians think is as important as how they do not. We need genuine “instructors” in emergency and on the wards.
- Otherwise, we will be the victims of specialists