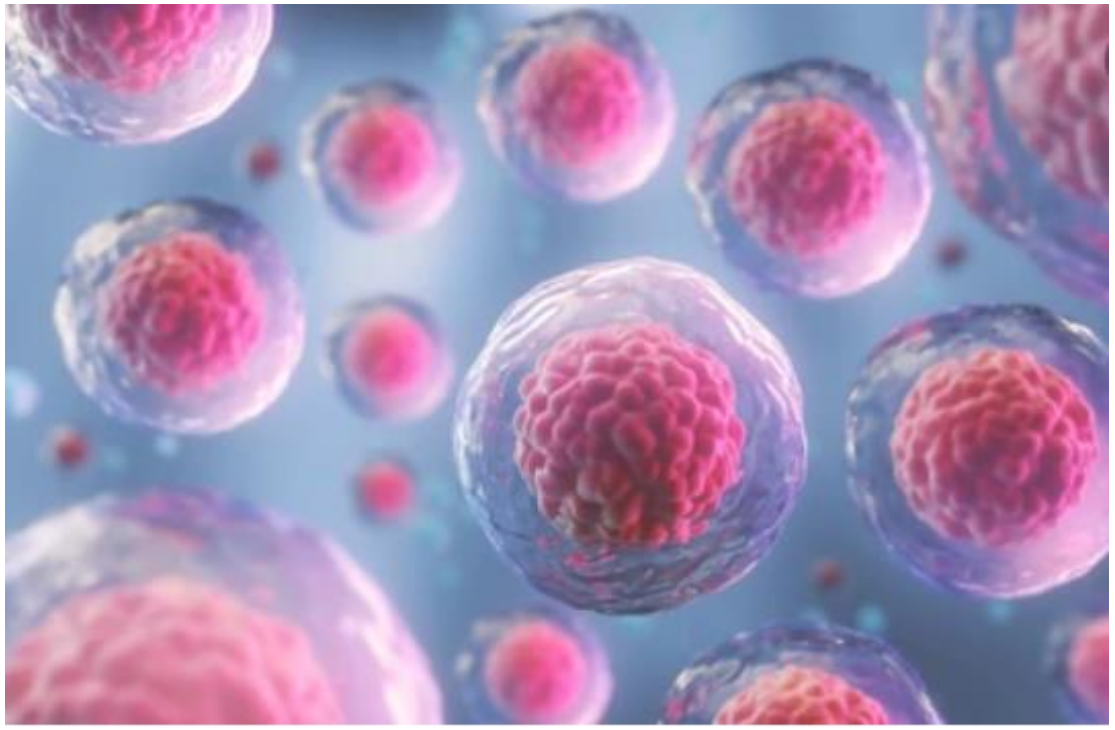
Composition of body fluids and its most common disorders.

Adriana Rybnikářová

Foto: NeuPaddy/CC0 Creative Commons | Zdroj: Pixabay

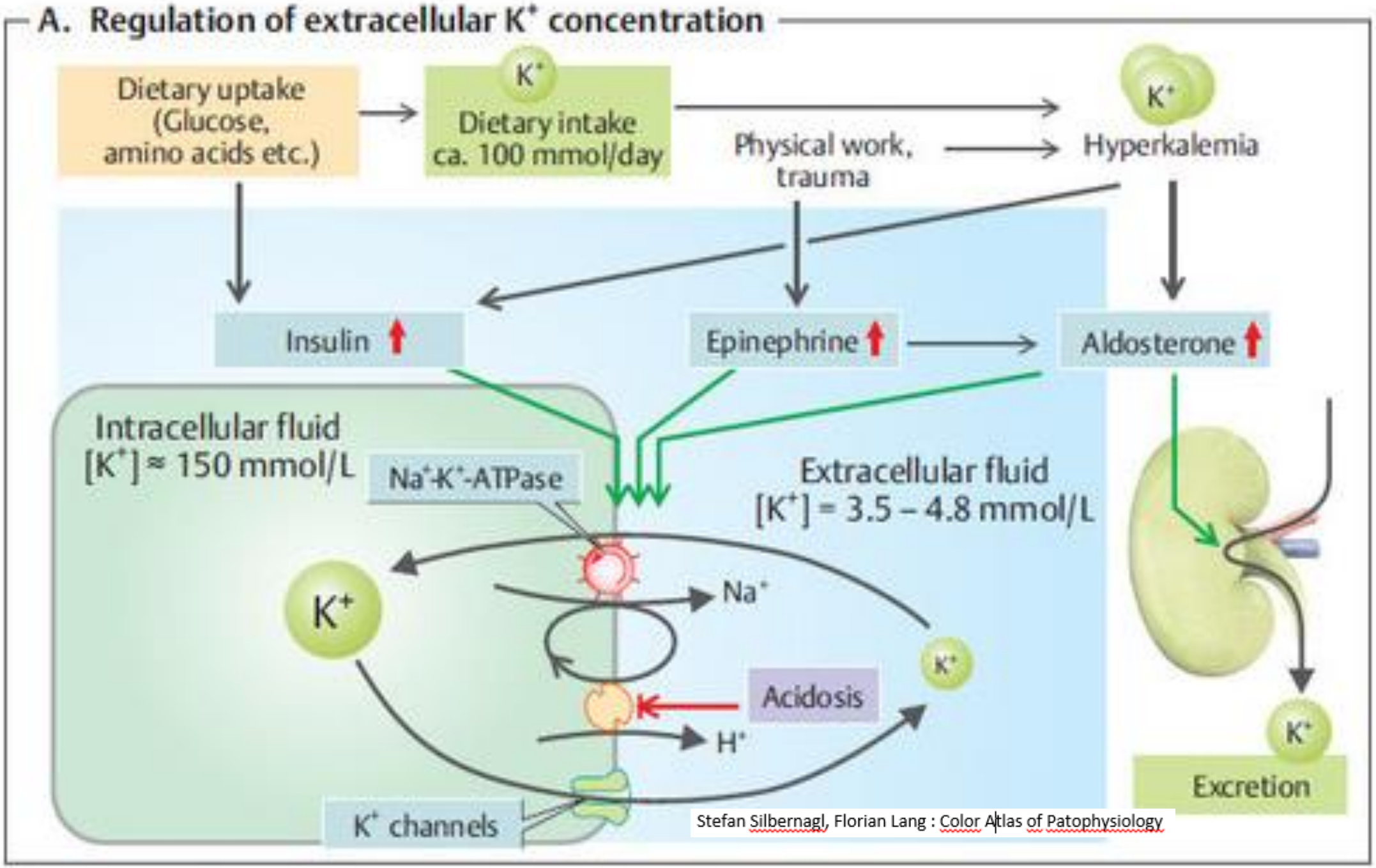
Kalium (Potassium)



https://www.shutterstock.com/cs/search/cells| Pixabay

- **Intracellular cation**

- **98% inside the cells to 2% outside the cells**



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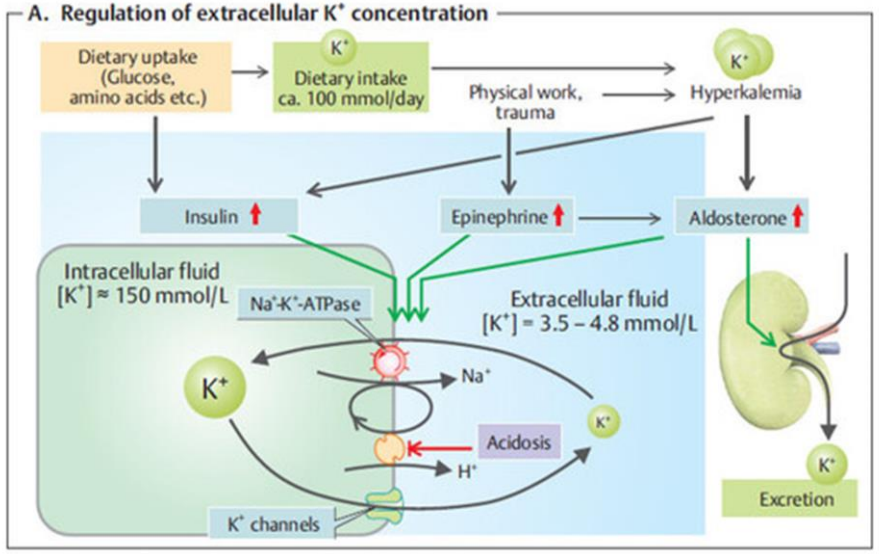
Decide, how will change the kalium level in plasma in acidosis.

- (A) hyperkalemia
- (B) hypokalemia
- (C) normal level

Membrane



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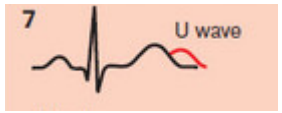
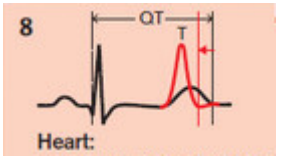
- Protons/Kalium Exchange
- (Acidosis –hyperkalemia)
- Hormons (Insulin, Epinephrine, Aldosterone)
- Osmolarity

Low



High

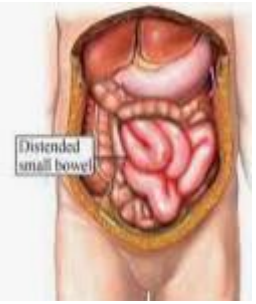


- Arytmia
- Paralysis
- (paralytic ileus)
- (paralysis of resp.)

Kalium

- **Arytmia**

Death (heart arrest)



Fatigue, Hypotonia,
Obstirpation

. Paresthesia, Fatigue

https://sites.google.com/site/totallyradpatho/home/paralytic-ileus

https://www.healthdirect.gov.au/neuromuscular-system

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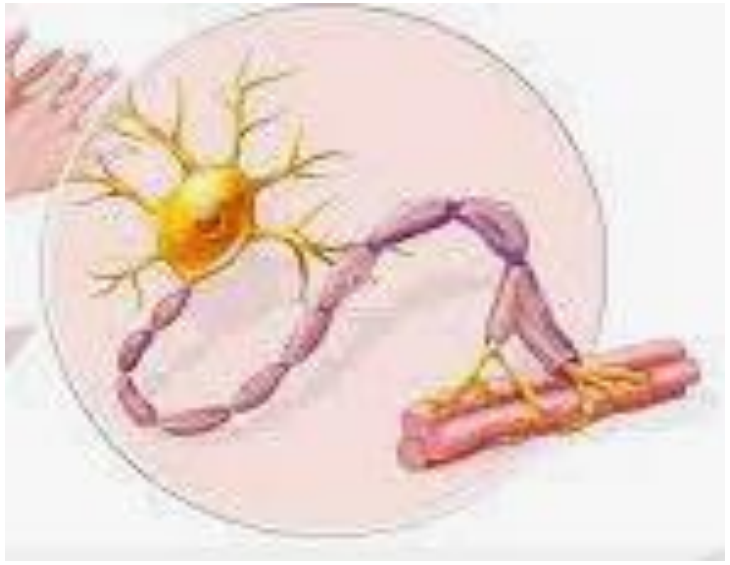

Neuromuscular exitability =

$$\frac{Na^{+} \cdot K^{+}}{Ca^{2+} \cdot H^{+} \cdot Mg^{2+}}$$

https://www.healthdirect.gov.au/neuromuscular-system

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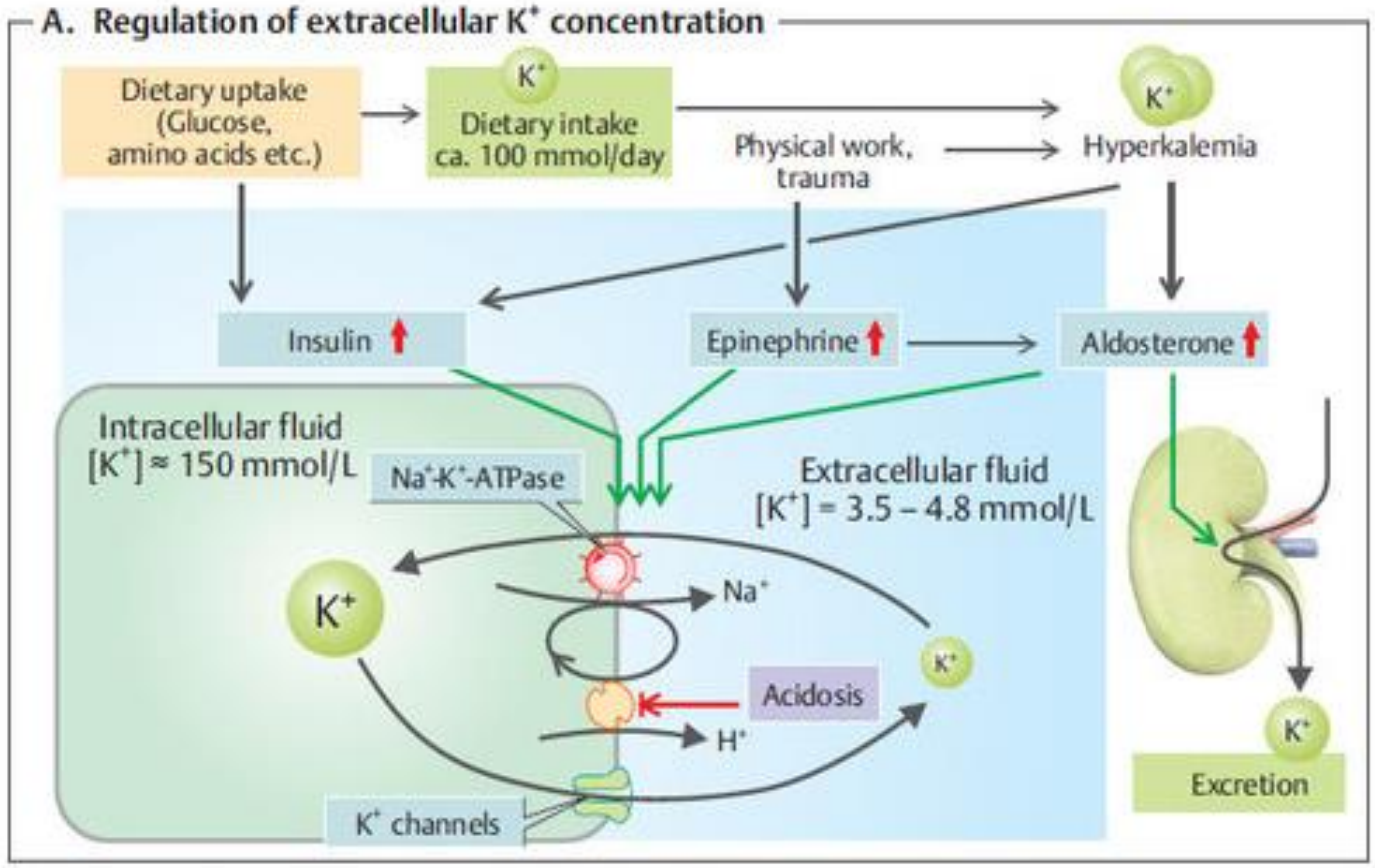
- (A) hyperkalemia
- (B) hypokalemia
- (C) normal level

Synchronicity

- H+↑ causes K+↑
- H+↓ causes K+↓
- K+↑ causes H+↑
- K+↓ causes H+↓

- Synchronous changes- enterance of H^+ into the cell is compensated by exit of kalium

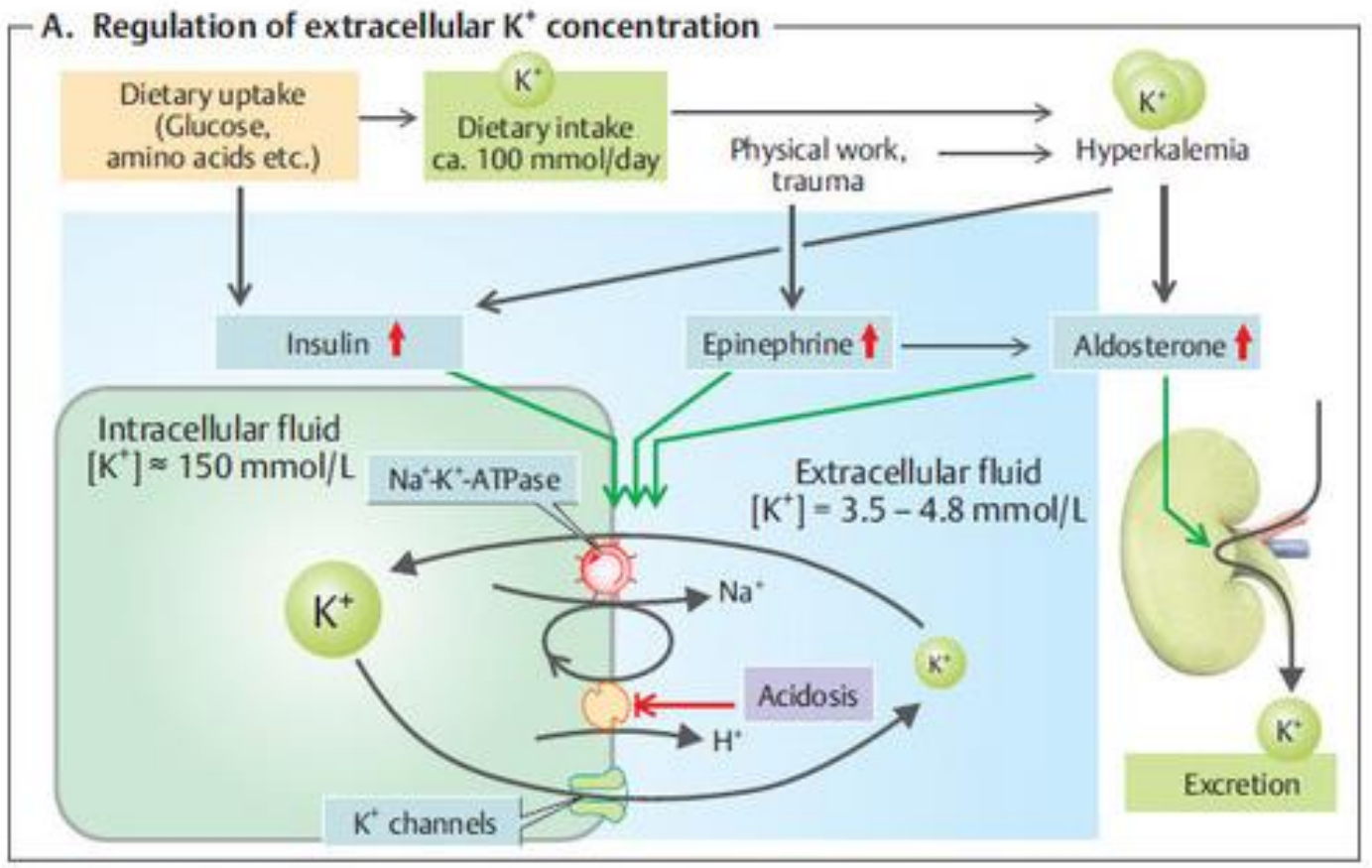
Decide- Hyperkalemia (A), Hypokalemia (B) or no change (C)?



https://www.healthdirect.gov.au/neuromuscular-system

- Acute metabolis alkalosis
- Anorexia nervosa (alkalosis)
- Metabolic acidosis

Decide- Hyperkalemia (A), Hypokalemia (B) or no change (C)?



https://www.healthdirect.gov.au/neuromuscular-system

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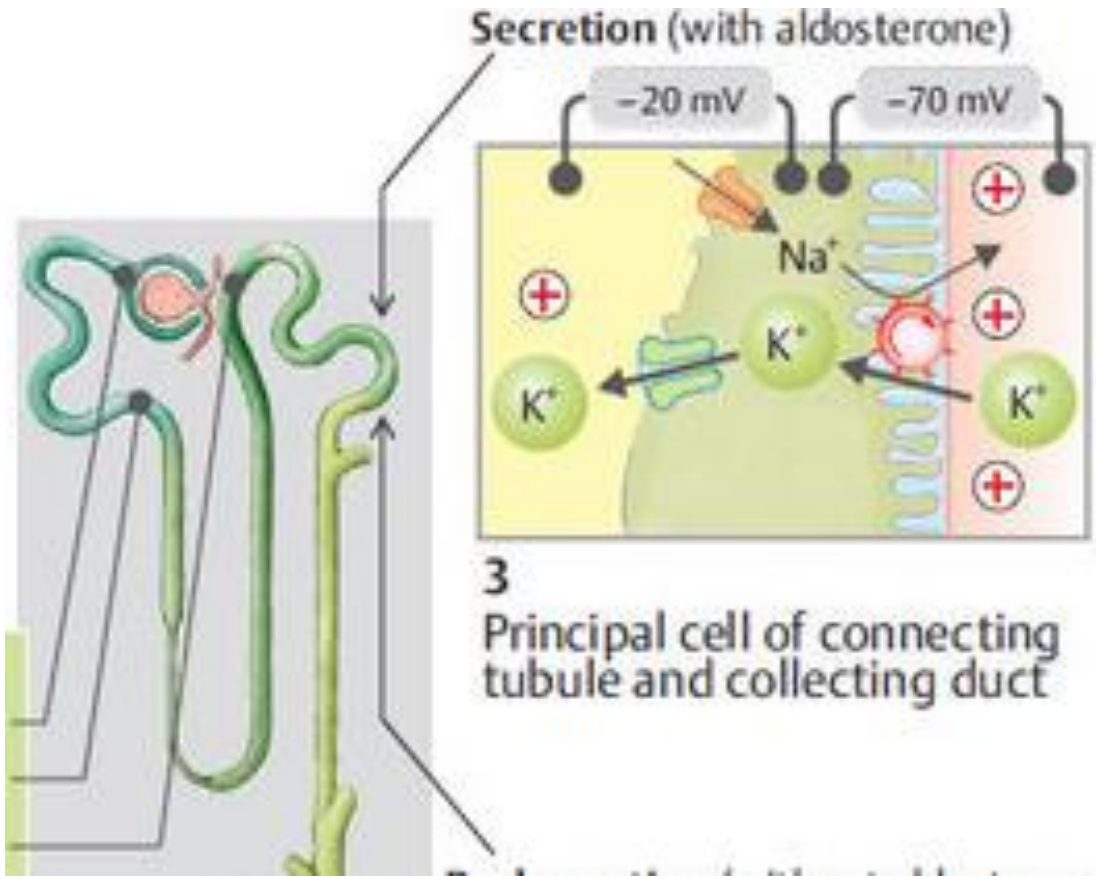
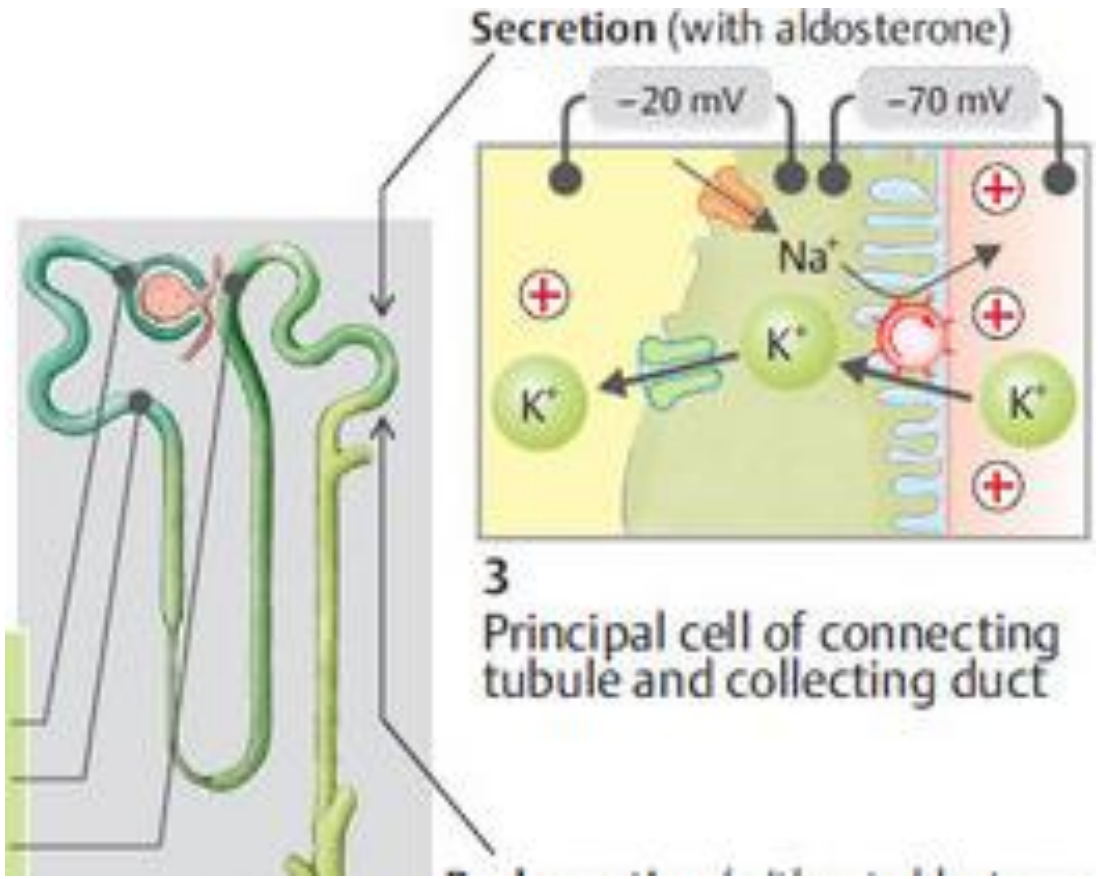


https://www.healthdirect.gov.au/neuromuscular-system

- Polyuric renal failure
- Acute anuric renal failure

- **Urinary excretion of K^+ increases with the urinary flow**
- FE-3-200%

Decide- Hyperkalemia (A), Hypokalemia (B) or no change (C)?



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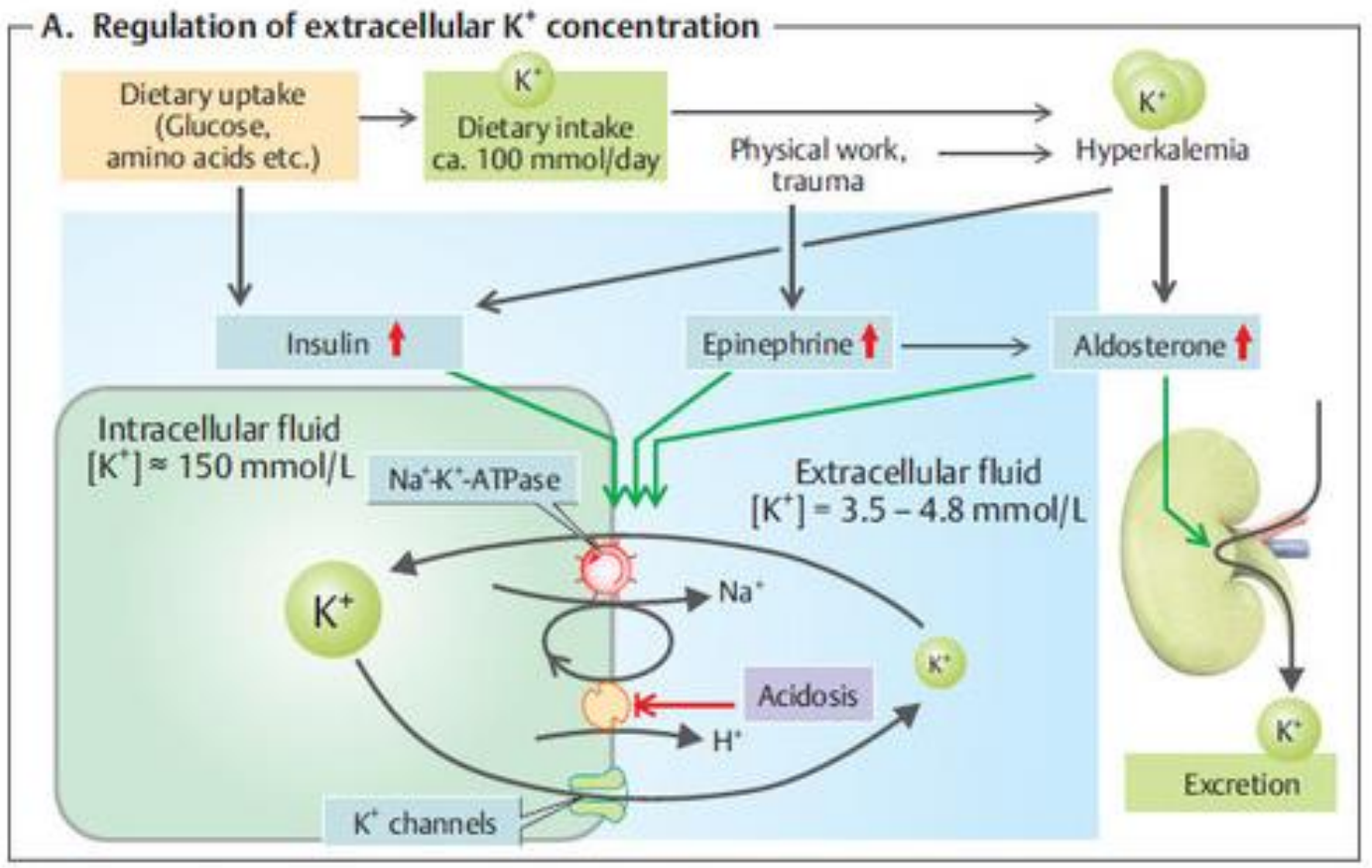
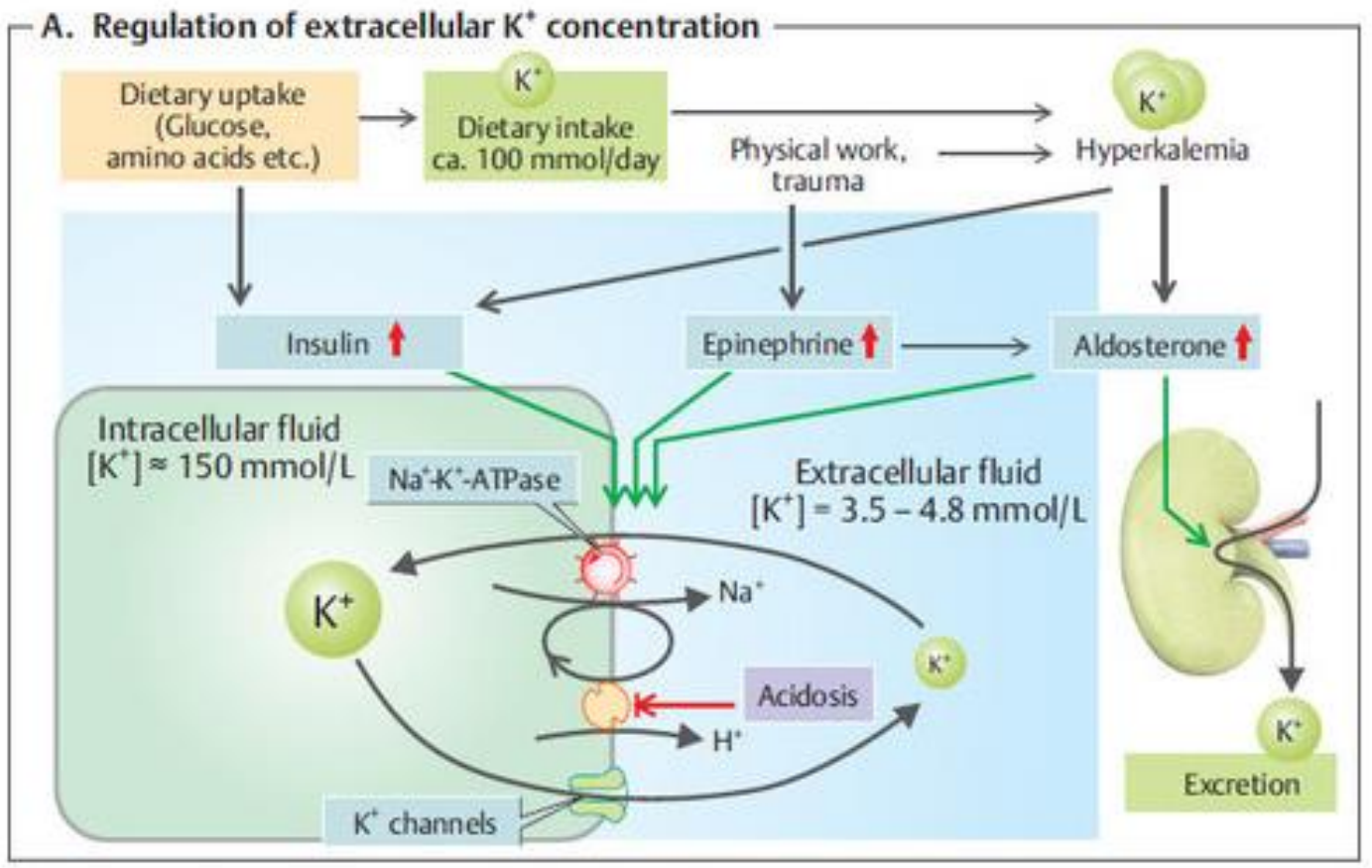


https://www.healthdirect.gov.au/neuromuscular-system

- Anemia after causal B12 treatment
- Insulin
- Burns
- Rhabdomyolysis
- Cytostatic therapy

Decide- Hyperkalemia (A), Hypokalemia (B) or no change (C)?



https://www.healthdirect.gov.au/neuromuscular-system

- Anemia causal B12 treatment ↓
- Insulin ↓
- Burnst ↑
- Rhabdomyolysis ↑
- Cytostatic therapy ↑

- **Anabolism-Catabolism**

Case report

- A young man was trapped underneath a car in a road traffic accident, and suffered multiple fractures. Despide adequate fluid intake over the next 36 hours, he was noted to be oliguric. The following results were obtained. Why is the potassium high?

Serum	Result	Reference range
Urea	22.1	2.5–6.6 mmol/L
Na^+	133	135–145 mmol/L
K^+	6.1	3.6–5.0 mmol/L
Creatinine	214	60–120 µmol/L

http://clipart-library.com/broken-leg-cartoons.html

Case report

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- Rhybdomylysis
- Acute renal failure-myoglobine precipitation in the distal nefrons

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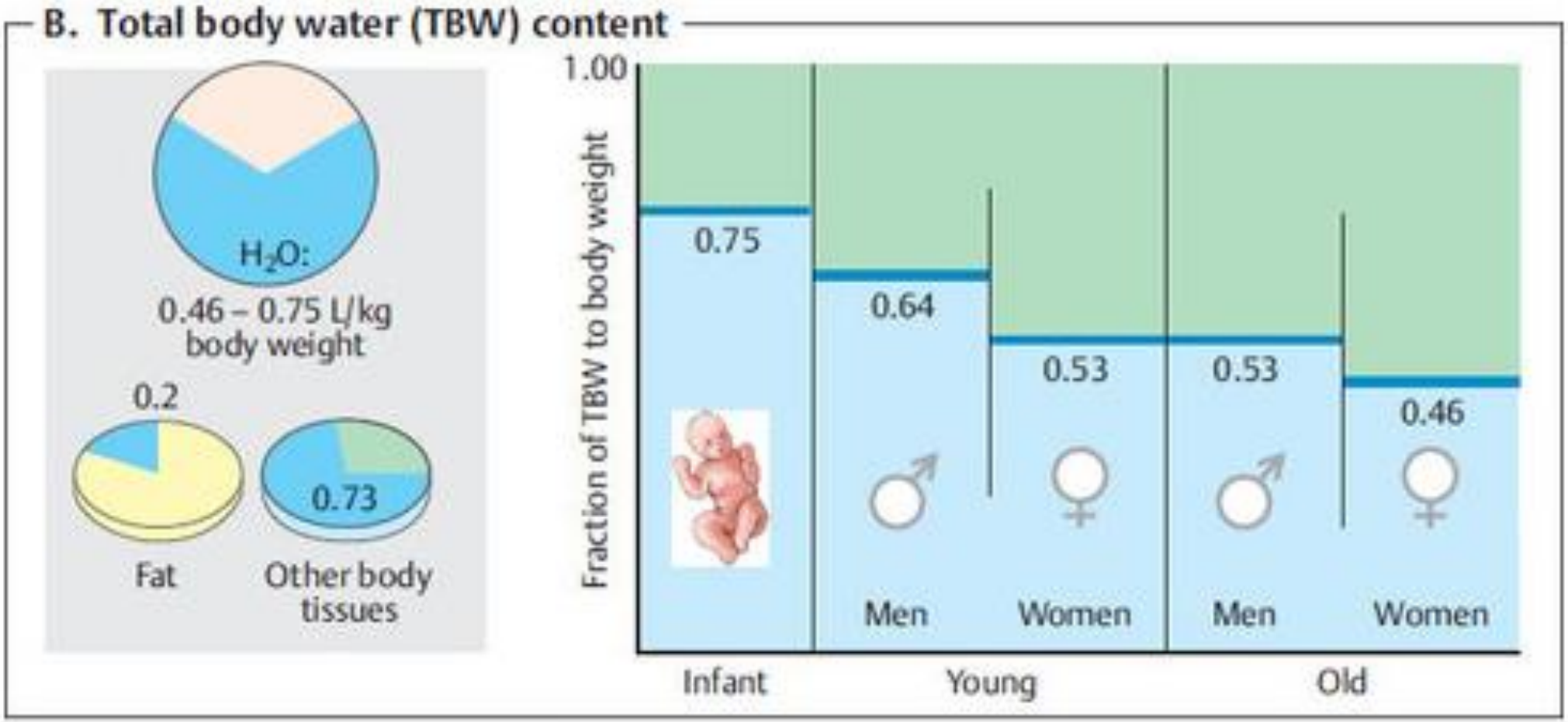
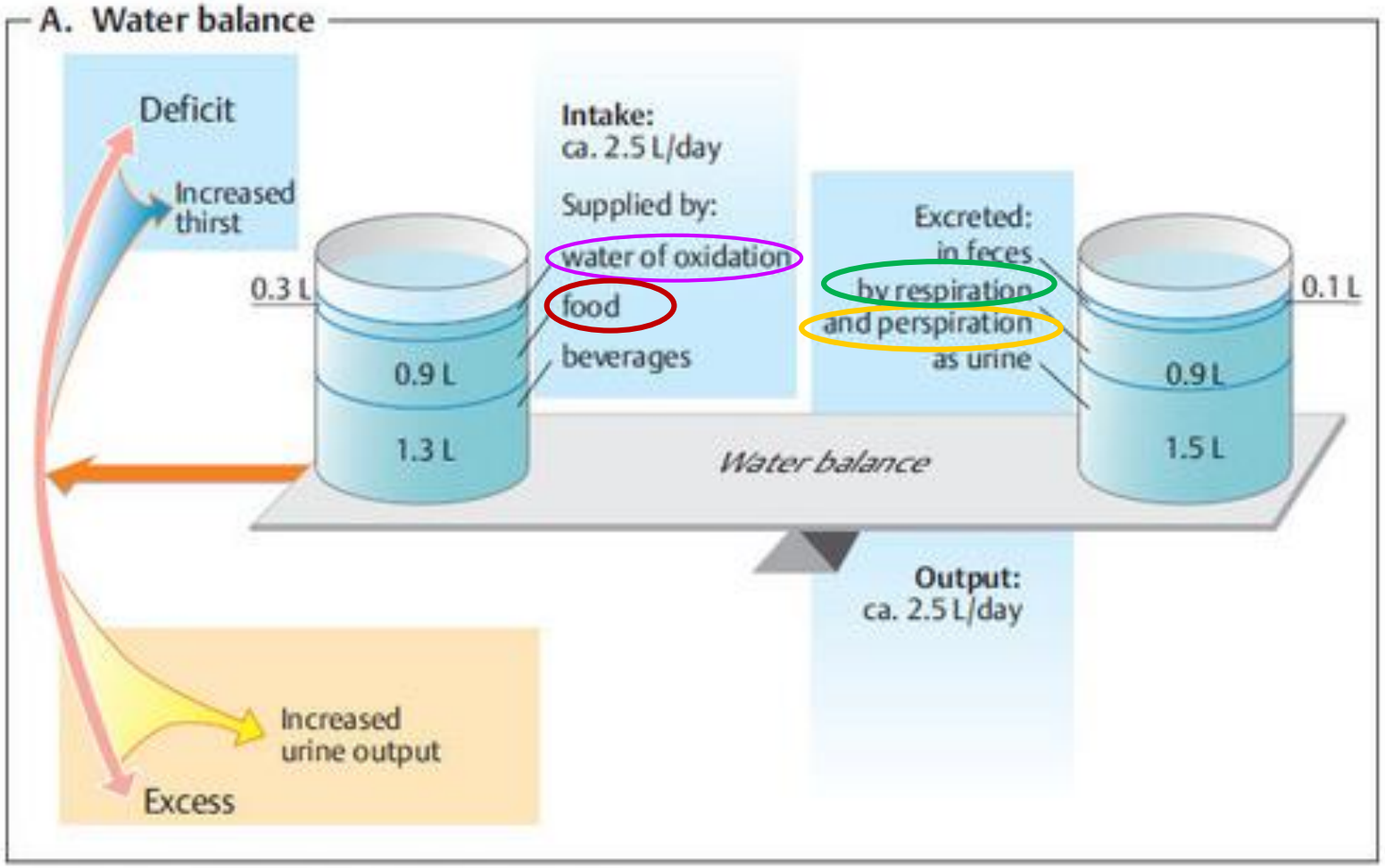
Kalium – What to remember?

- Intracellular : Anabolism/catabolism
- Membranes : A- Aldosterone (K+ goes inside the cell)
- E- Epinephrine
- I- Insulin
- O - osmolarity
- U = H+, synchonous
- Excretion : kidney: K+(aldosterone, urinary flow)

Water balance

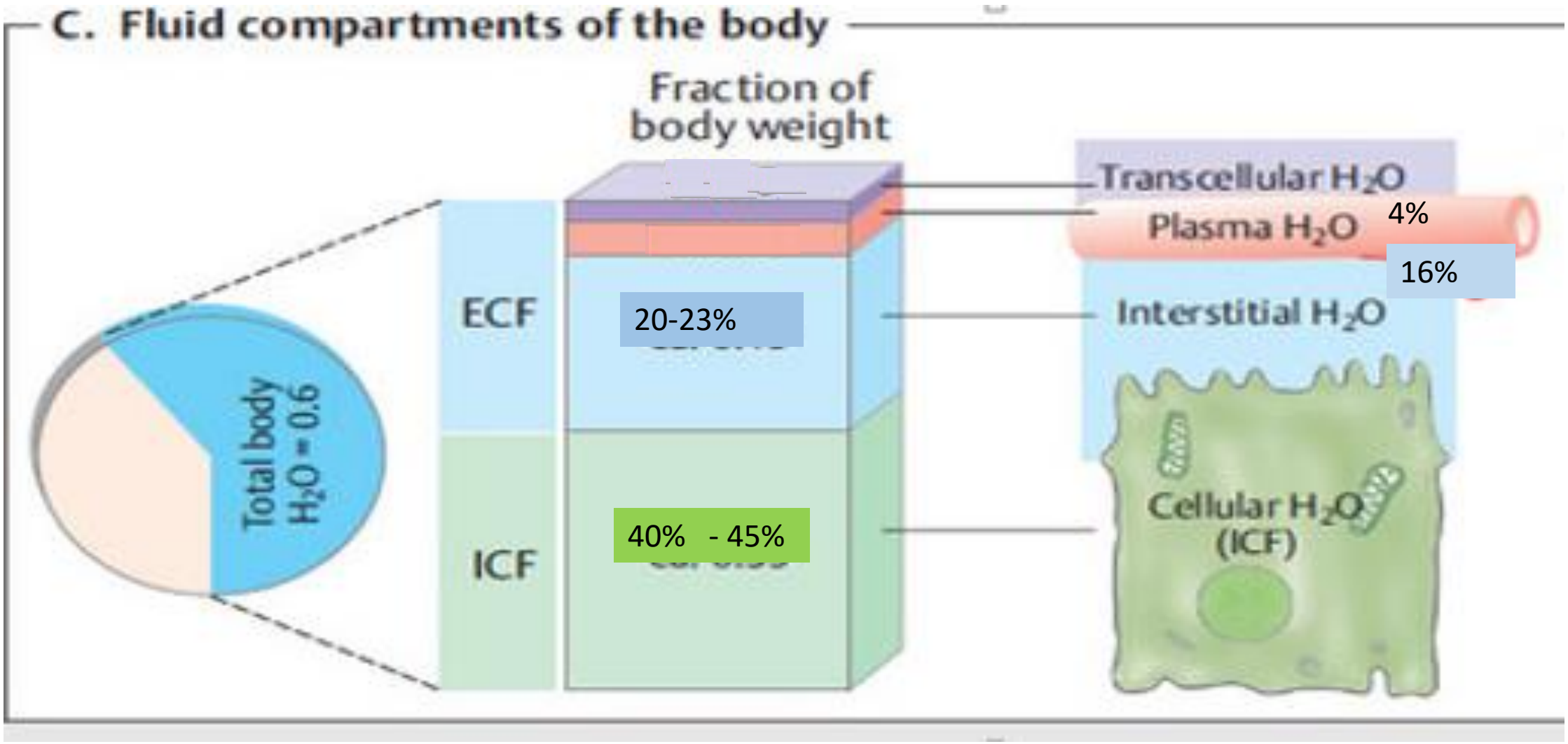


Water Balance



- Cave:
  Starvation- higher water intake is necessary

(+1000ml)


C. Fluid compartments of the body
Fraction of body weight
Total body H_2O = 0.6
ECF
ICF
20-23%
40% - 45%
Transcellular H_2O
Plasma H_2O
4%
16%
Interstitial H_2O
Cellular H_2O (ICF)

Renal excretion

- Oligouria: -400mL/day
- Anuria: -100mL/day
- Polyuria: +2.5 L /day

- Osmolarity of urine:
- 2(Na^+ + K^++ NH_4^+)+ urea – estimation

- Excretion of Na^+

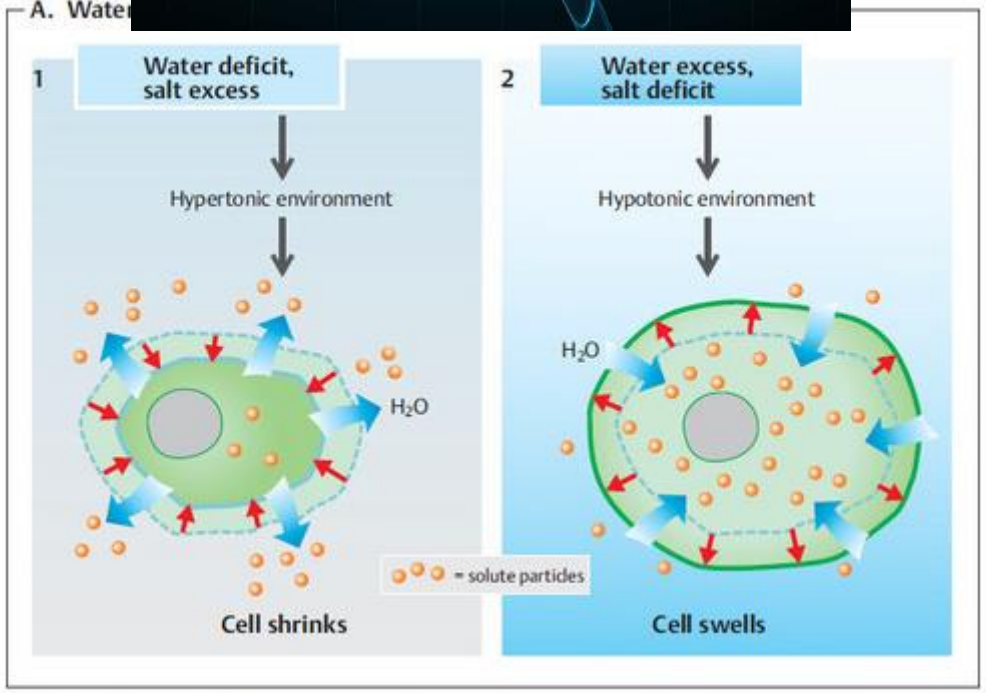


Foto: Shutterstock

From: https://www.denik.cz/veda-a-technika/neurologove-myslenka-mozek.html

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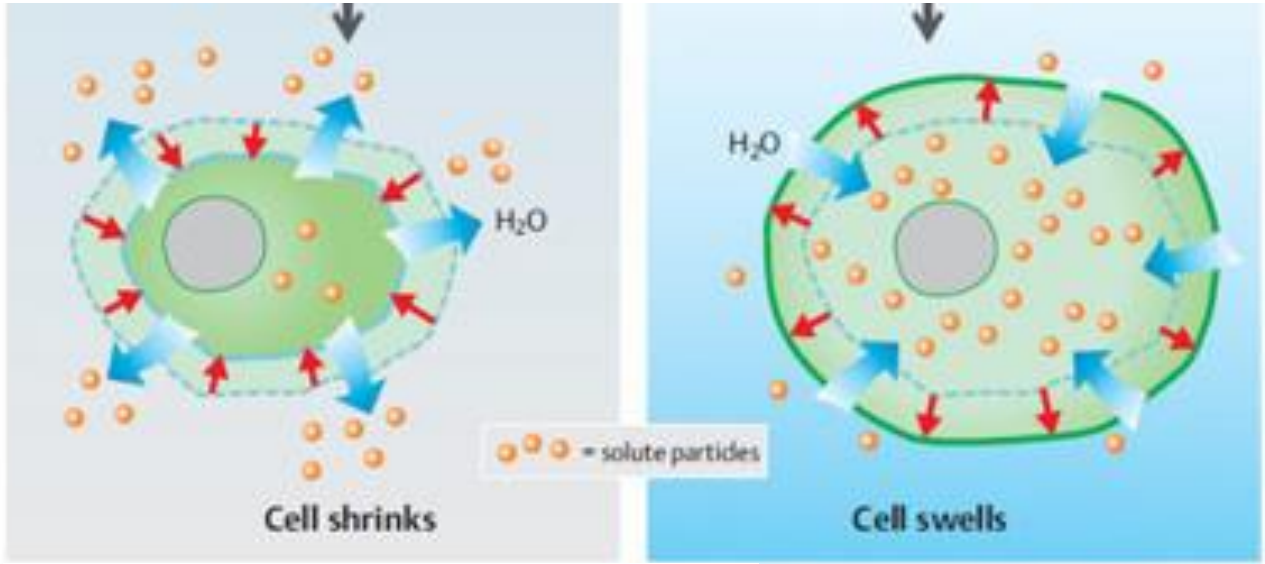
Osmolarity

- Serum:
- 275–295 mmol/kg H_2O
- Limited space- CNS

- Urine : 300-900mmol/L
- Limits:50-1200mmol/L

Adaptation



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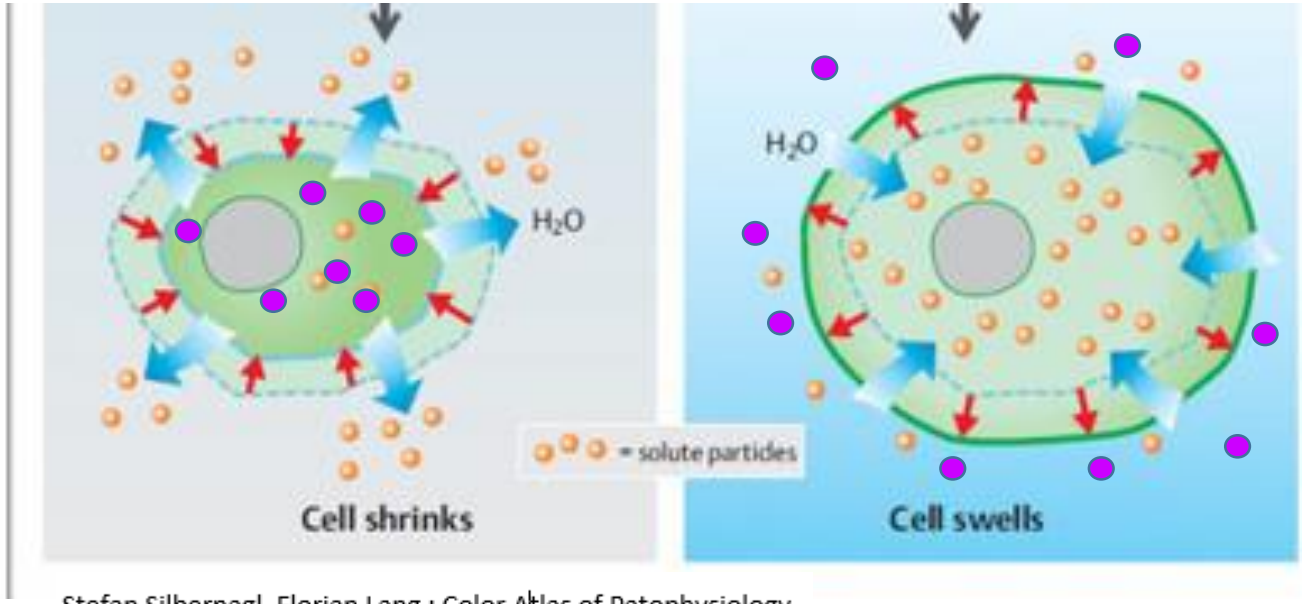
- Time: after 48 hours
- Ex.: Hyponatremia- secretion of osmotic active particles
- Cave: demyelinisation (pons) after a quickly iatrogenic therapy (Shrinkage to subnormal volume)

Adaptation



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Sodium (Natrium)

Extracellar cation

Hyponatremia: under 135mmol/L

Hypernatremia: over 150mmol/L

Foto: Shutterstock

From: https://www.denik.cz/veda-a-technika/neurologove-myslenka-mozek.html

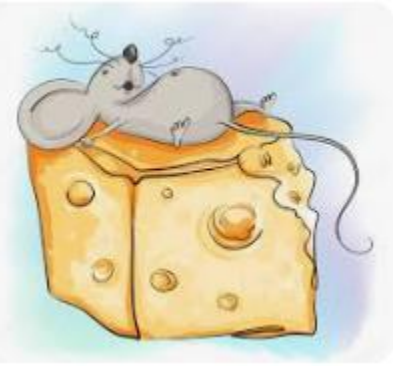
CAVE: Desorientation, Convulsions, Coma

Under 120mmol/L or over 160mmol/L

Water in ECT

Depletion of water

- Orthostatic hypotensia
- Diff.15-20mmHg,freqvency ++15-20



- Oligouria

(-400ml/day) + osmolarity over 600mm/L

- Central venous pressure

- (Thirst,Hematocrit, Albumine only progress, Albumine)

cz.depositphotos.com/91140860/stock-illustration-funny-cartoon-mouse-lies-with.html

Expansion

- Edema

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- Weight gain ,
- CAVE : ather 2-4L surplus-edema

Differential diagnosis Sodium- Water balance

	Hypovolemic	Euvolemic -clinically	Hypervolemic
Hyponatremia	Kidney GIT Burst	ADH↑ Endocrinal	CDK Albumine deficit Heart (right) insuficiency
Sodium normal	Kidney GIT Bleeding Burst		CDK Albumine deficit Heart (right) insuficiency
Hypernatremia	Kidney, GIT	ADH↓	Infusion of hypertonic solutions - saline

Differential diagnosis Sodium- Water balance

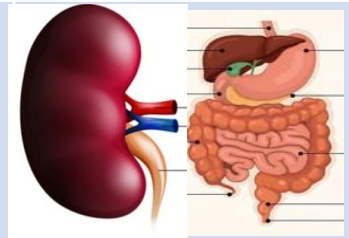
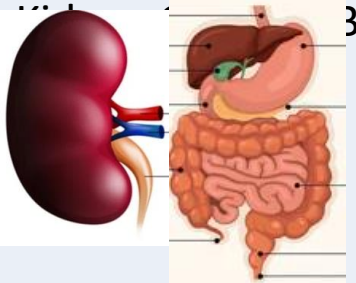
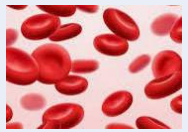
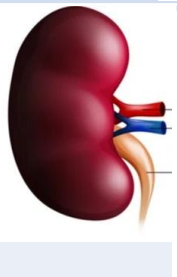
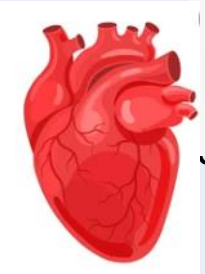
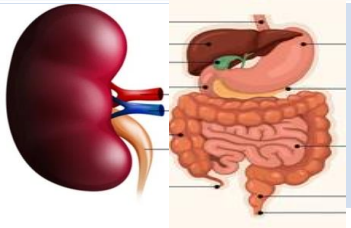
	Hypovolemic	Euvolemic -clinically	Hypervolemic
Hyponatremia	 Burst	ADH↑  Endocrinal	  
Sodium normal	 Bleeding 		  
Hypernatremia		ADH↓ 	Infusion of solutions - 

Foto: Shutterstock

https://www.raumedic.com/application-areas/infusion

How can you decide between renal and non renal dehydratation?

Renal

- Diuretic
- Osmotic (ketoacidosis, glucose)

GIT

- Vomiting,
- Diarrhoea

- In hypovolemic hypernatremia by extrarenal losses of water and solutes (as vomiting, diarrhoea) is renal anti- regulation present mediated by ADH (osmosis, volume) and RAA. There is renal anti-regulation present with typical findings as: oligouria V (urine)↓, increase of osmolarity↑ and decrease of sodium in urine.↓

Renal

- Renal
- Diuretic
- Osmotic (ketoacidosis, glucose)

- GIT extra renal (oligouria, incerase of osmolarity in urine, low sodium in urine)
- Vomiting,
- Diarhoea

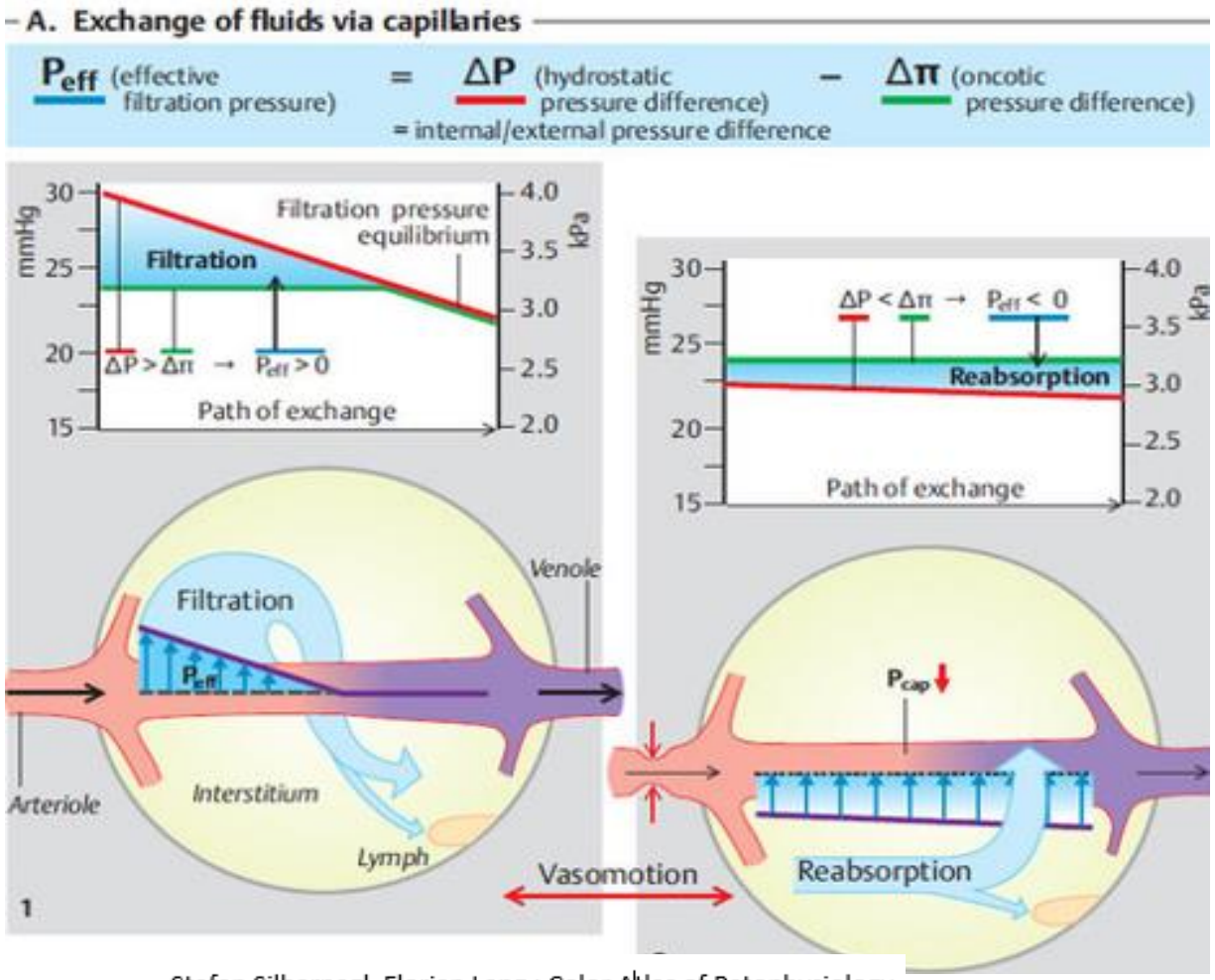
Hypetonic dehydratation

- Renal
- Diuretic
- Osmotic (ketoacidosis, glucose)

- GIT
- Vomiting
- Diarrhoea

Hypertonic dehydratation-old patients, kids
After drinking hyponatremic or normonatremic

What is the mechanism of edema by hypoproteinemias and a right heart failure?



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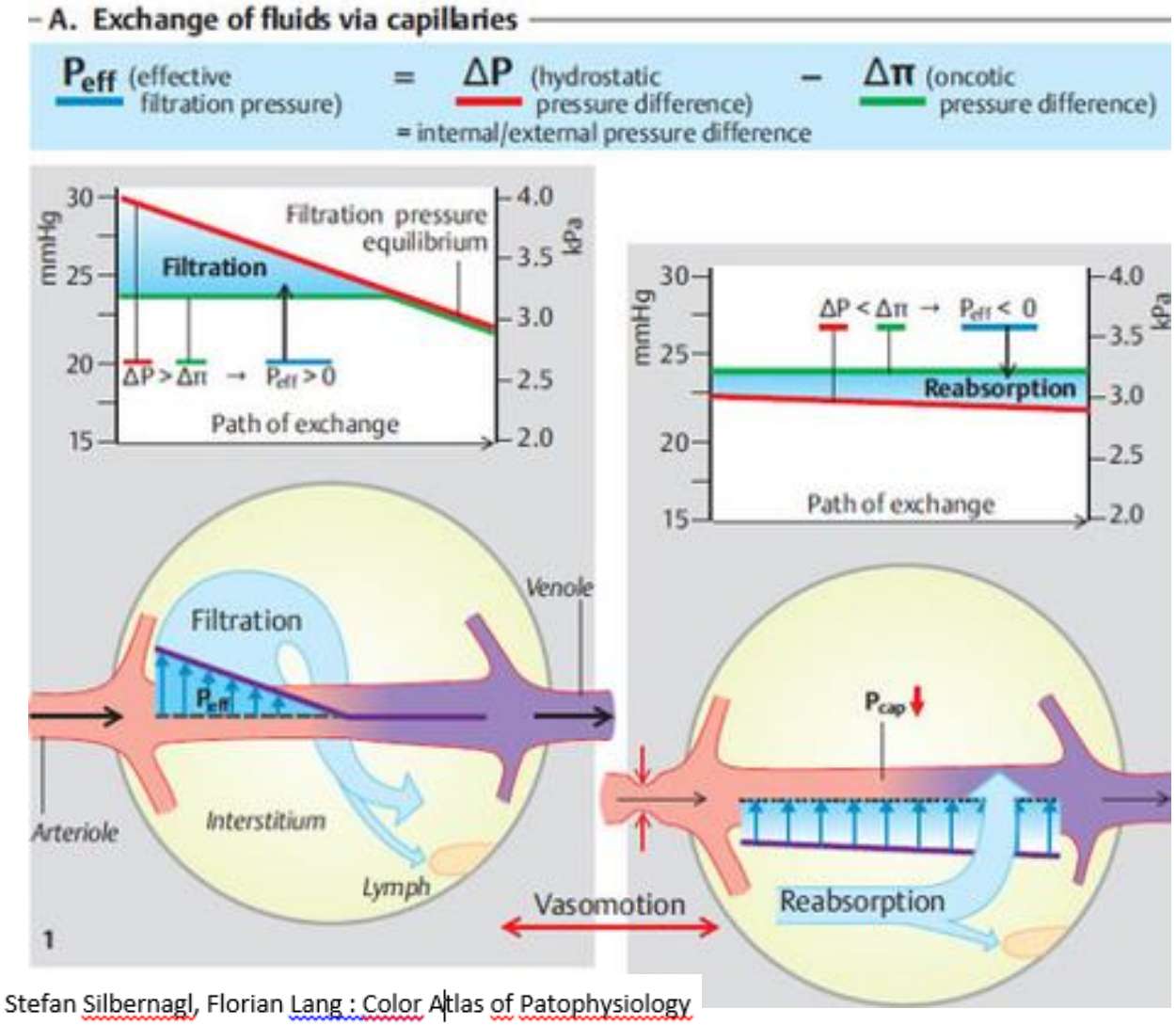
Causes of edema (Pressure, Proteins)



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- ↑ Increased venous pressure (heart failure)
- ↓ Decreased concentration of plasma proteins (cirrhosis, nephrotic syndrome)
- ↑ Increased permeability
- ↓ Lymph drainage blockade

Edema – Salt end water retention in Hypoalbuminemia and right heart failure

- Effective circulating volume loss
- RAA activation
- ADH

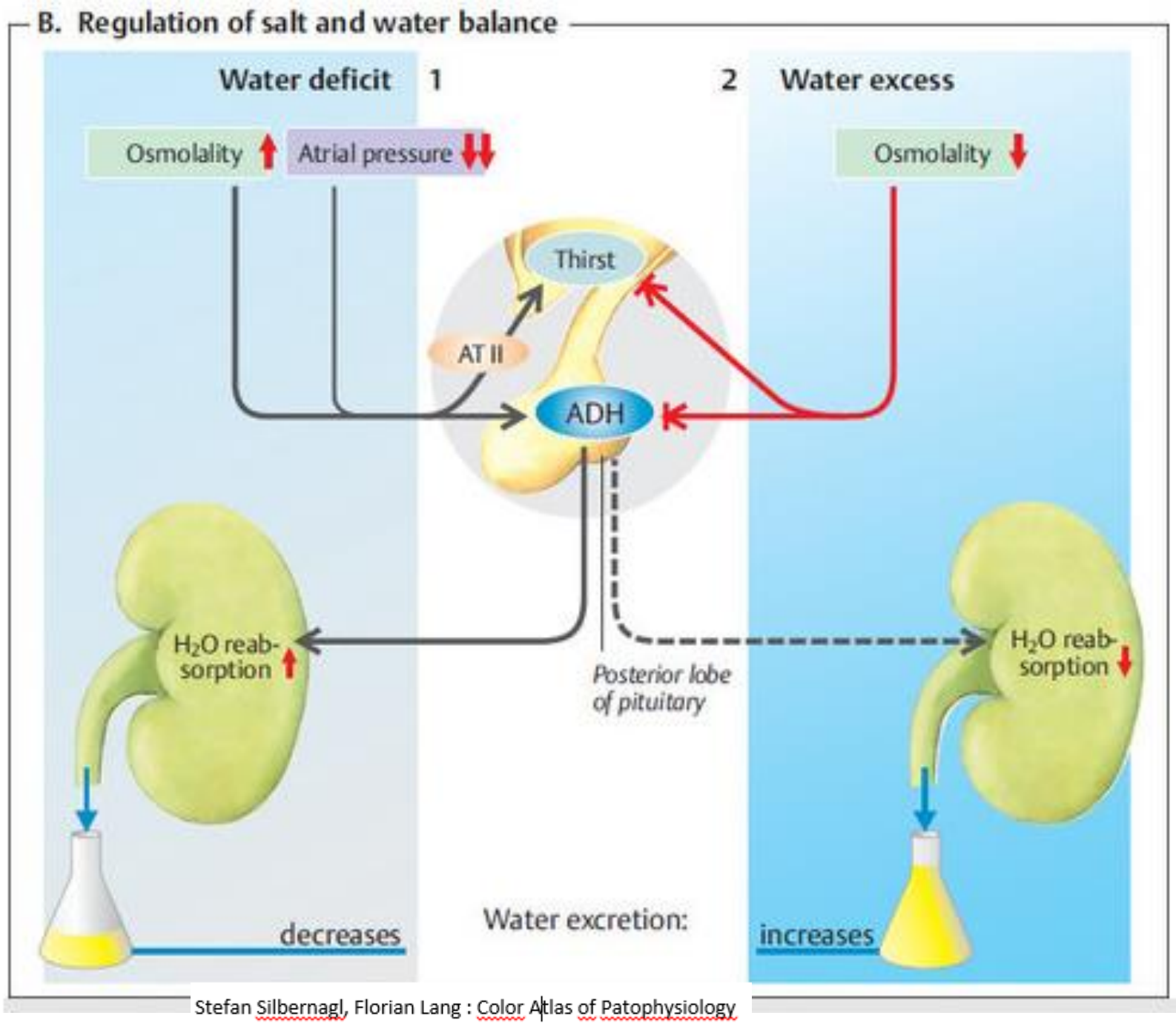
ADH

Stimulation:

- Osmolarity
- Atrial pressure

Reaction:

- AD reabsorbtion



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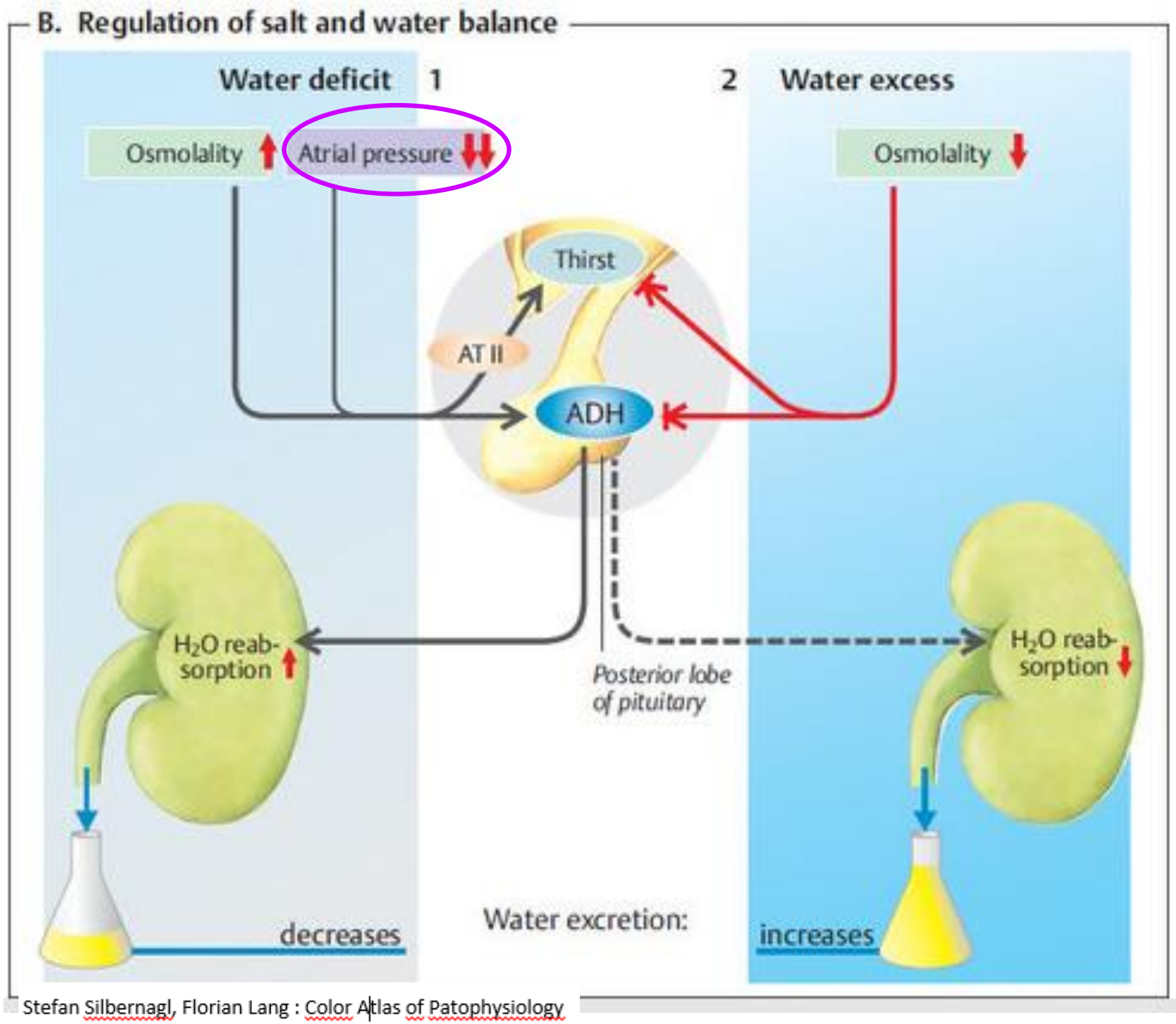

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RAA

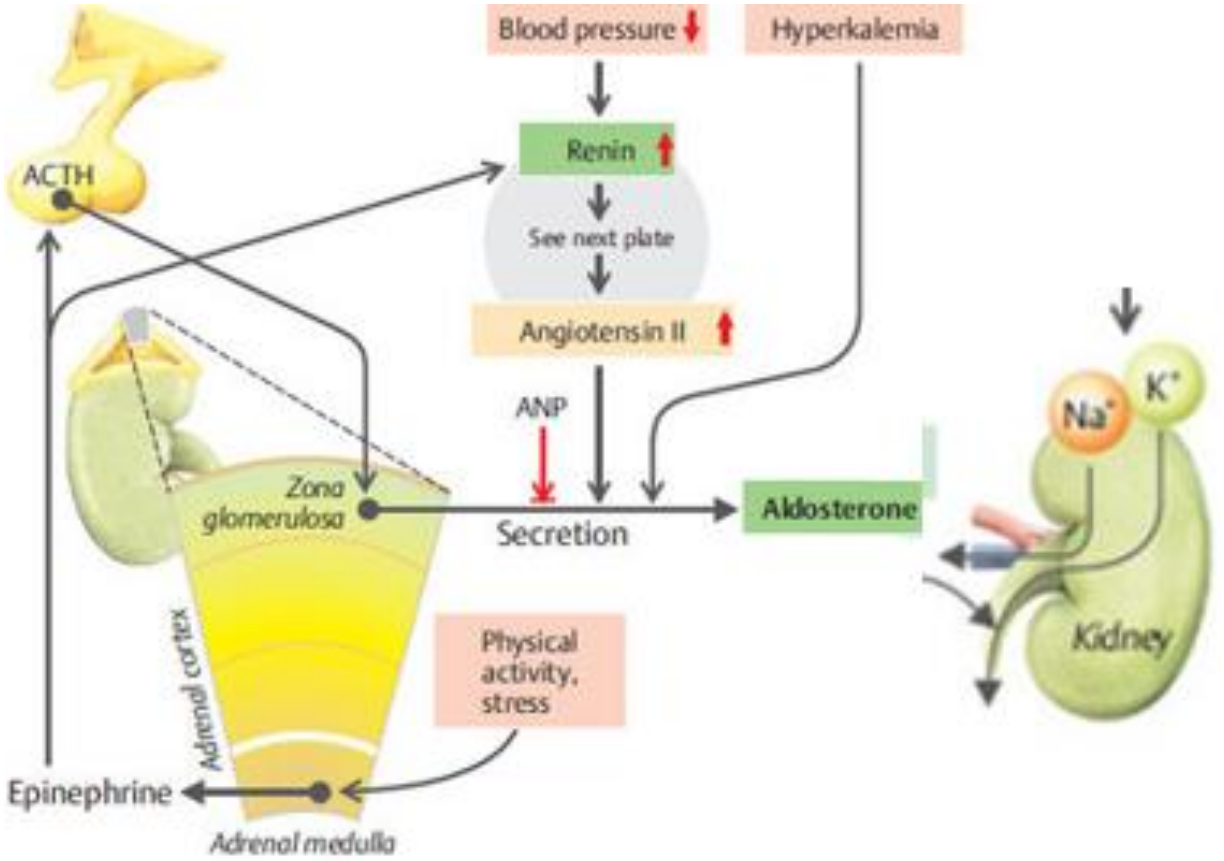
- Renin Inhibition:

Angiotensinogen II

Aldosterone

Prostaglandins

Increased reabsorbtion Na+, Cl-, macula densa



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- Renin Stimulation:

Pressure (vas afference)

Sympaticus activity in the kidney

Katecholamines in blood

RAA

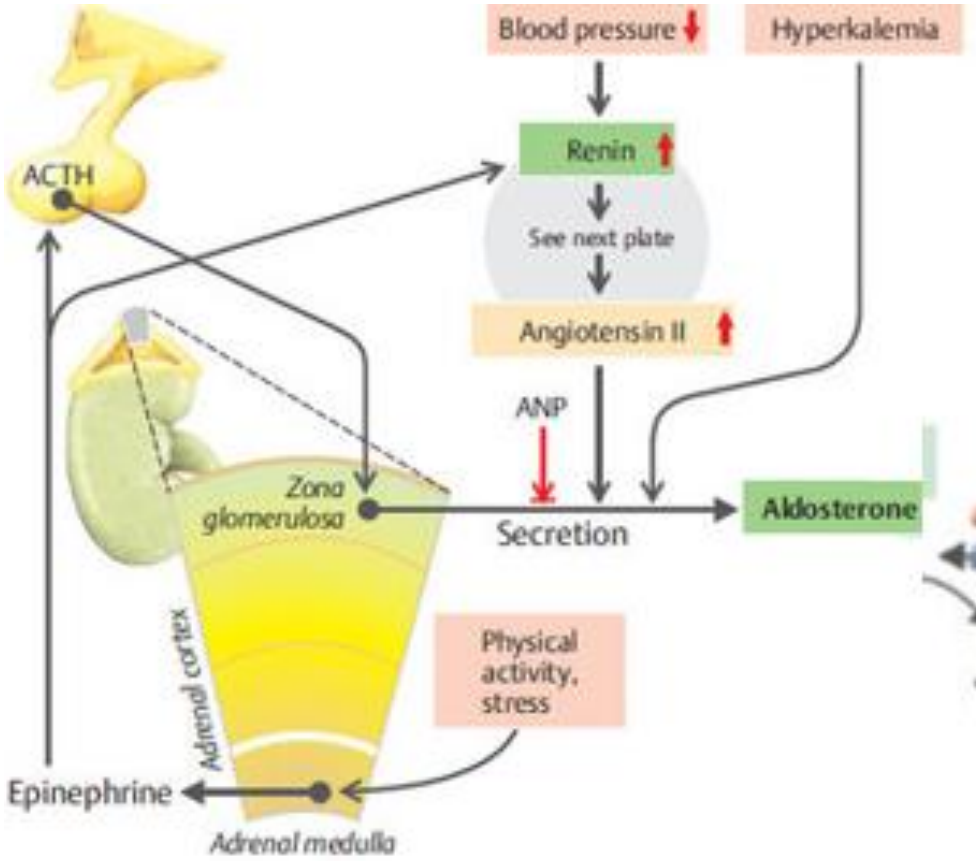
- Renin Inhibition:

Angiotensinogen II

Aldosterone

Prostaglandins

Increased reabsorbtion Na+, Cl-, macula densa



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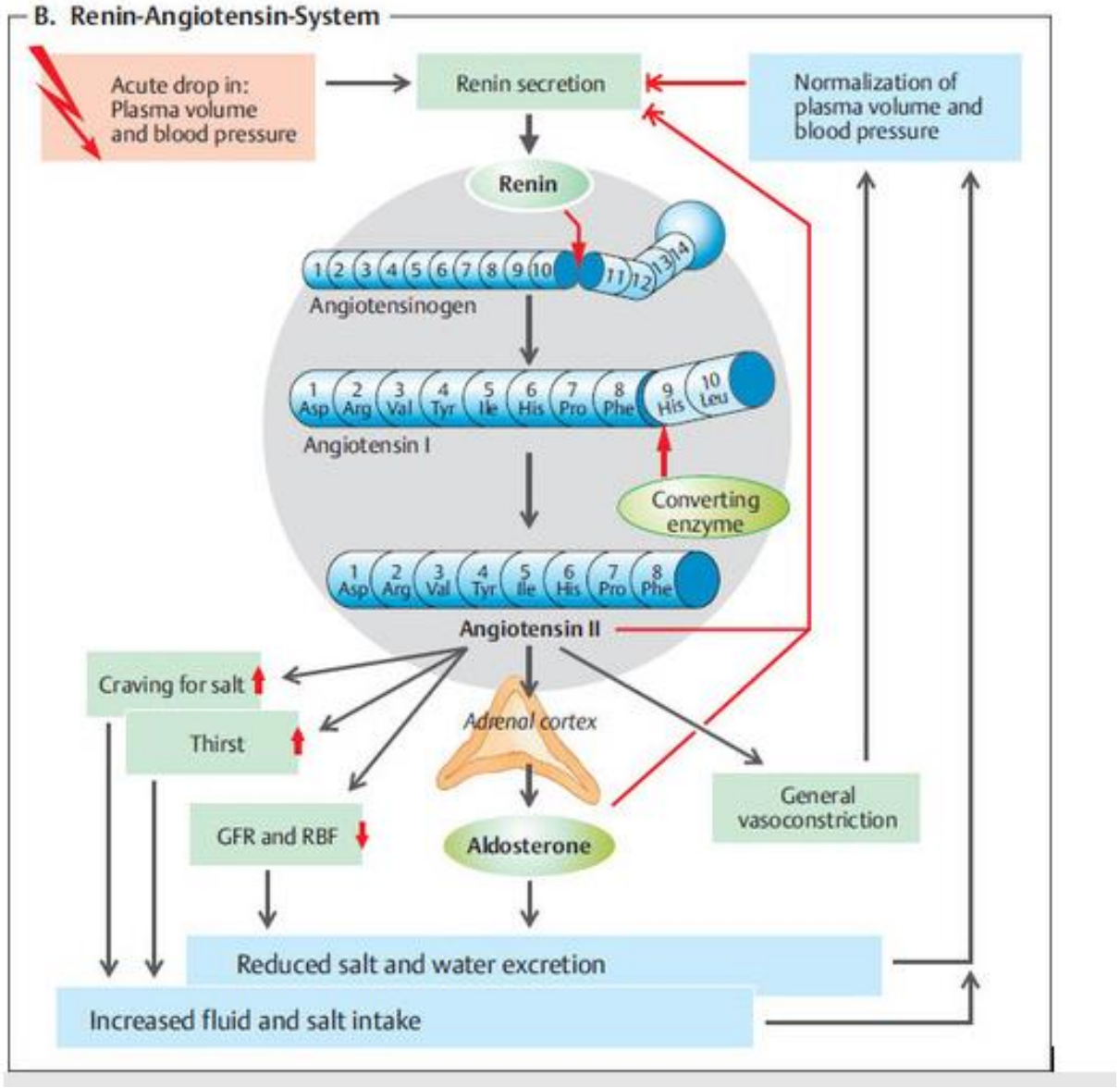

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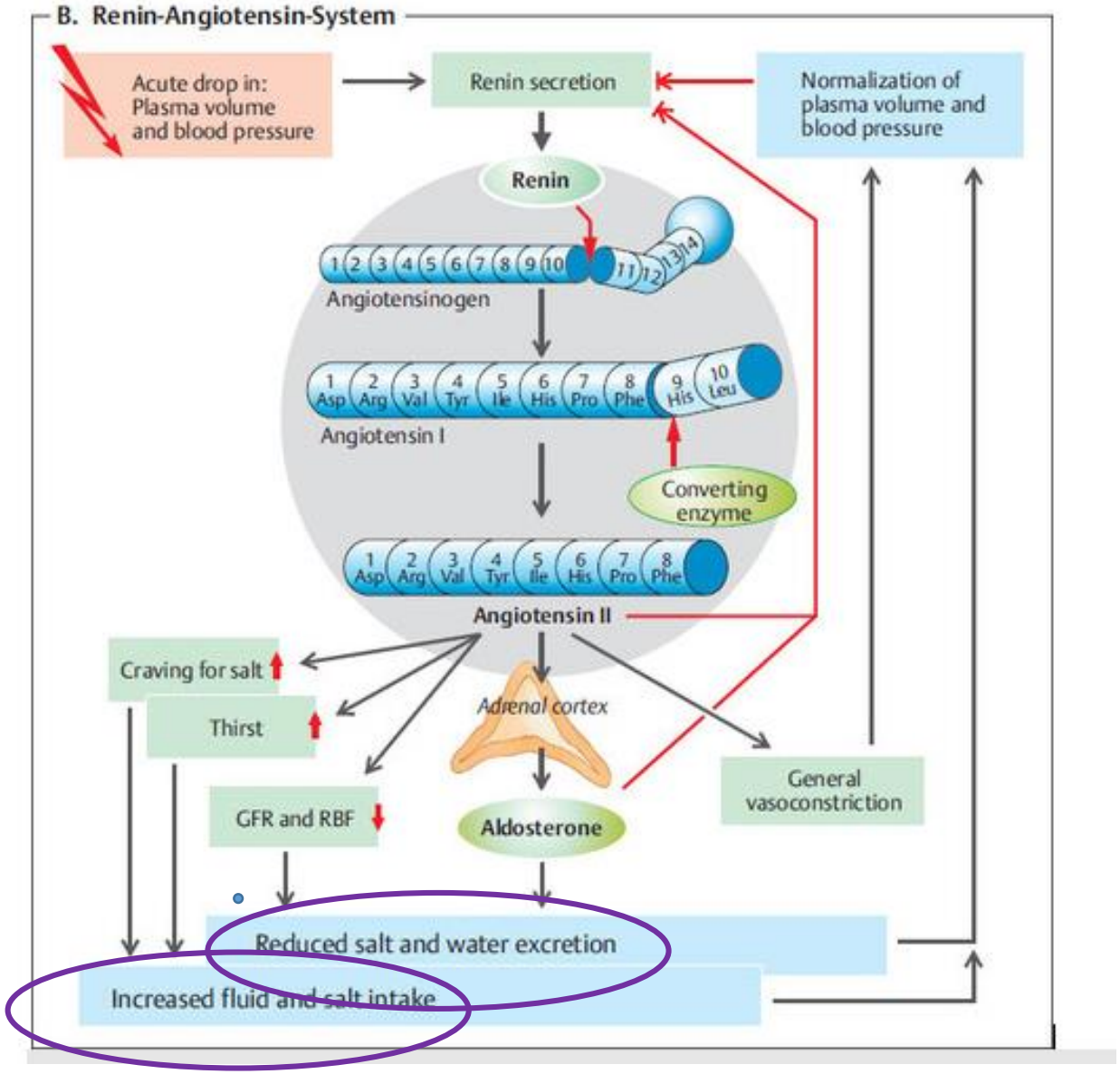
Angiotensinogen II

Activated by Renin + ACE

Effects:

Aldosterone

Vasoconstriction



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Angiotensinogen II

Activated by Renin + ACE

Effects:

Aldosterone

Vasoconstriction

Hyponatremia – CNS trauma

CWS

BNP - dehydration

Therapy:

Primary cause+

Slowly Na+ suplementation, when symptoms

Cave: Slowly

Brain diseases: Subarachnoidal bleeding, trauma, tumors, trombosis of veins in brain

SIADH

- ADH- no clinical features of hyperhydratation (no edema)
- Therapy:
- Primary cause + volume restriction, when symptoms
- CAVE: Slowly

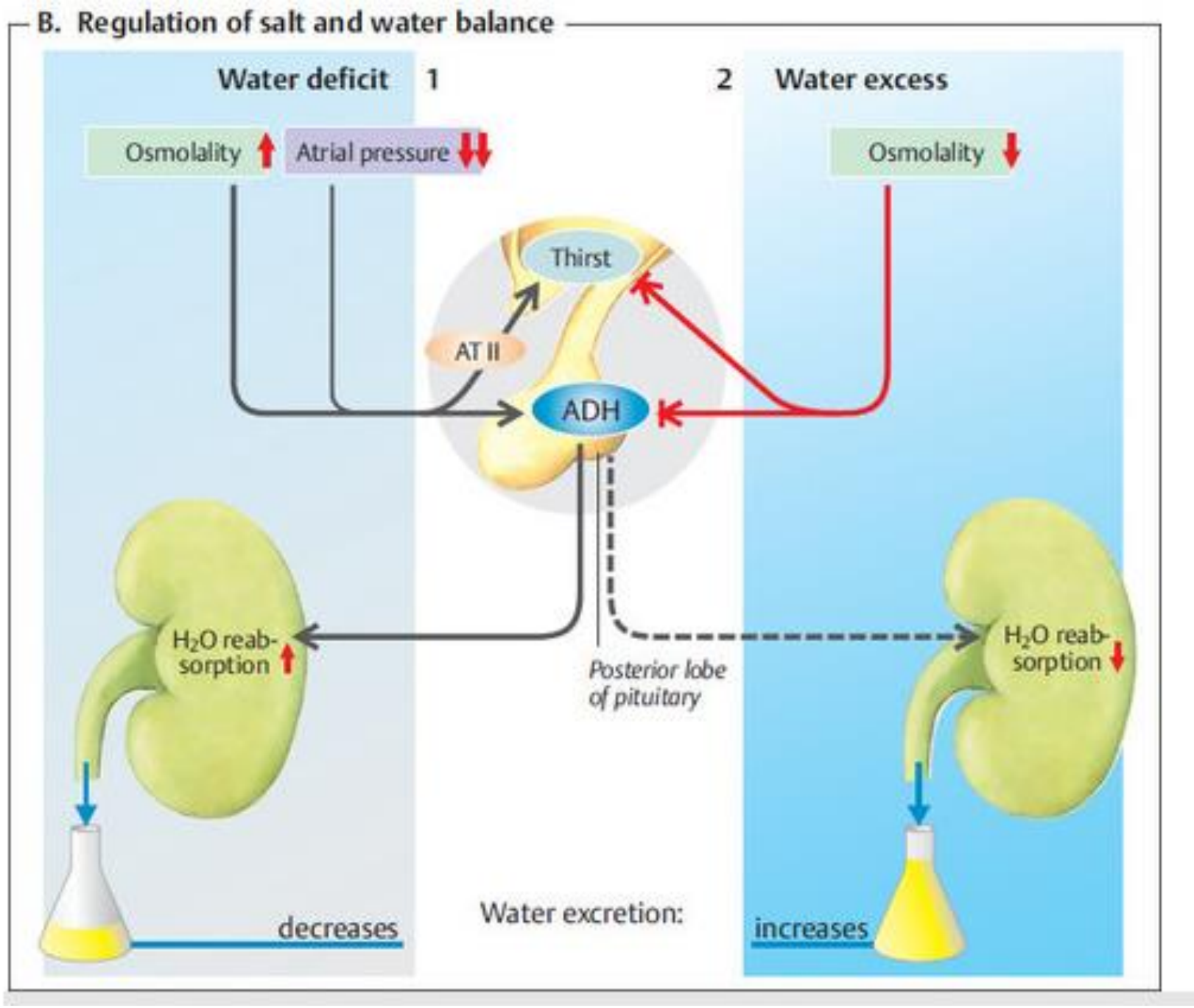
Other causes of inappropriate secretion of ADH: medicaments, tumors, lungs diseases

Foto: Shutterstock

ADH

- Stimulation:
- Osmolarity
- Atrial pressure

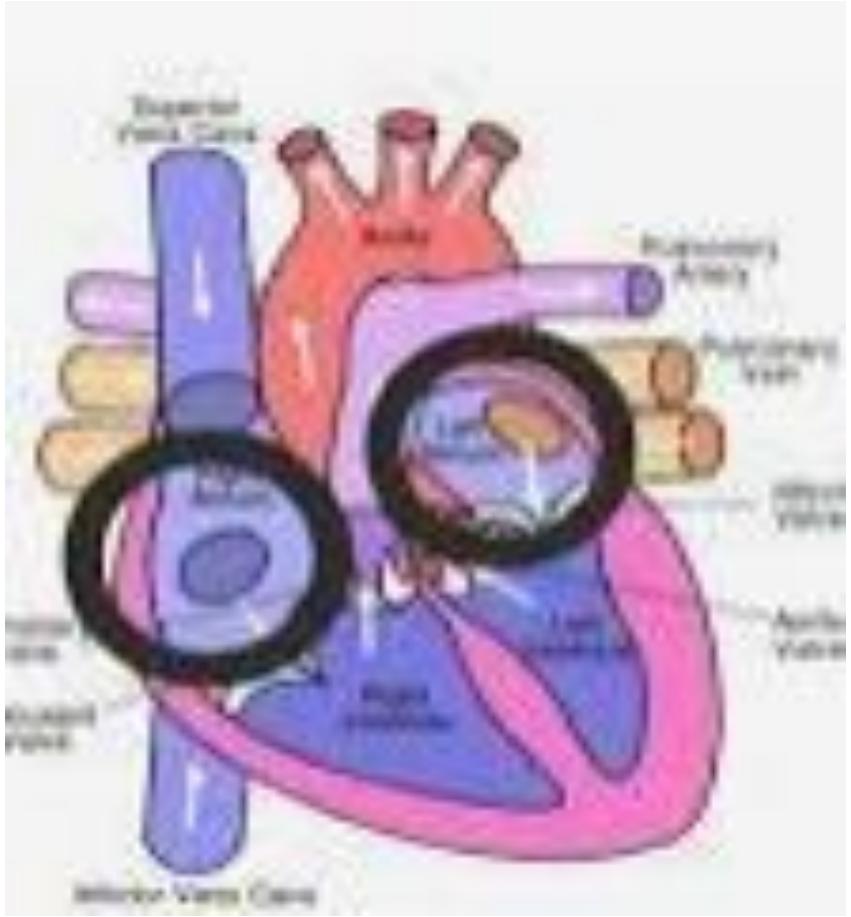
- Reaction:
- water reabsorbtion



ANP (Atrial natriuretic peptide) (or BNP in brain and ventriculus)

Stimulated by

- Baroreceptors in atrium



Effects:

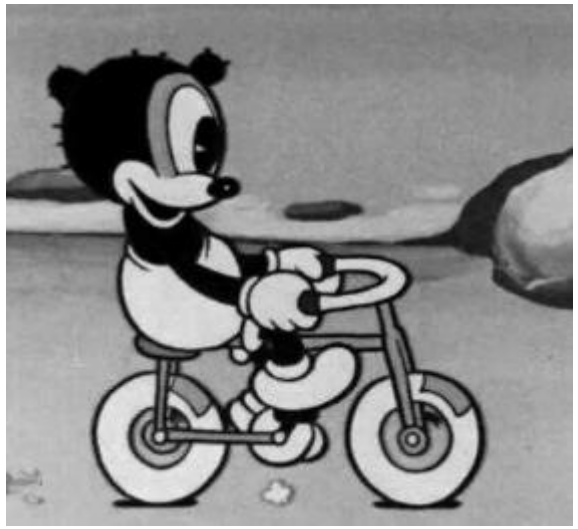
- Vasodilatation
- Excretion of sodium and water (kidney)

https://www.youtube.com/watch?v=bWZqN1my5sQ

Case study

- Bike, accident, broken 3 teeth
- Disorientated, he speaks very quickly
- After leaving vomiting

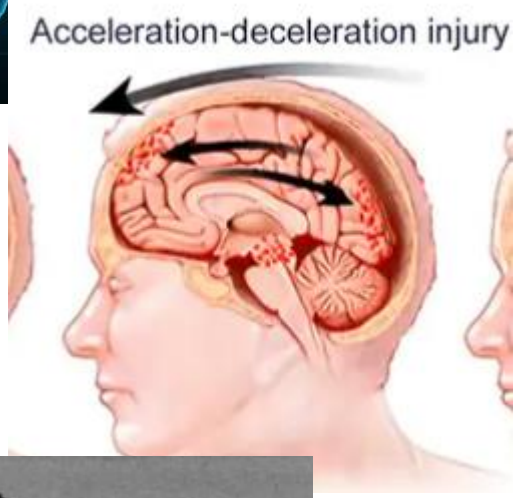
Case study



- Bike, accident, broken 3 teeth
- Disorientated, speaks very quickly.
- After leaving vomiting
- What water balance disease is suspected ?

Case study

- Bike, accident, broken 3 teeth (other side- contusion in brain)
- Disorientated, speaks very quickly, hyponatremia or contusion/commotion
- After leaving vomiting contusion, commotion (intracranial pressure)



ADH

Water reabsorbtion,
Na + dilution,
hyponatremia

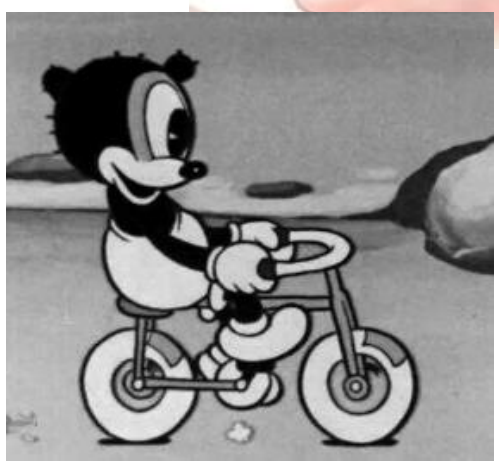


Foto: Shutterstock

https://www.mayoclinic.org/medical-professionals/trauma/news/optimizing-care-for-adults-with-traumatic-brain-injury/mac-20527270

Hyponatremia- SIADH or CWS Contusion

ADH

Water reabsorbtion,
Na + dilution,
hyponatremia

- Bike, accident, broken 3 teeth (other side- contusion in brain)
- Disorientated, speaks very quickly, hyponatremia or contusion/commotion
- After leaving vomiting contusion, commotion (intracranial pressure)

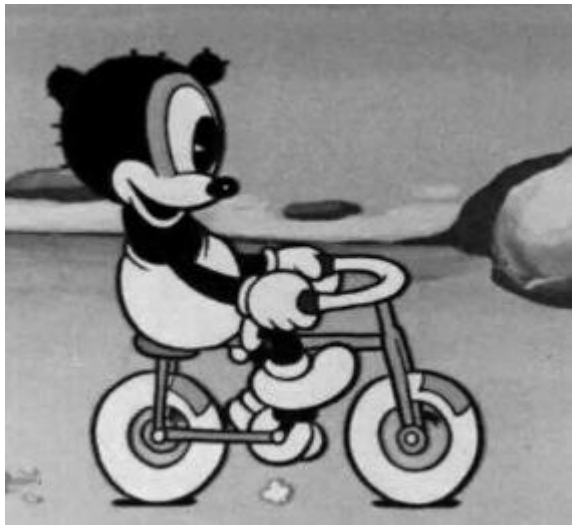


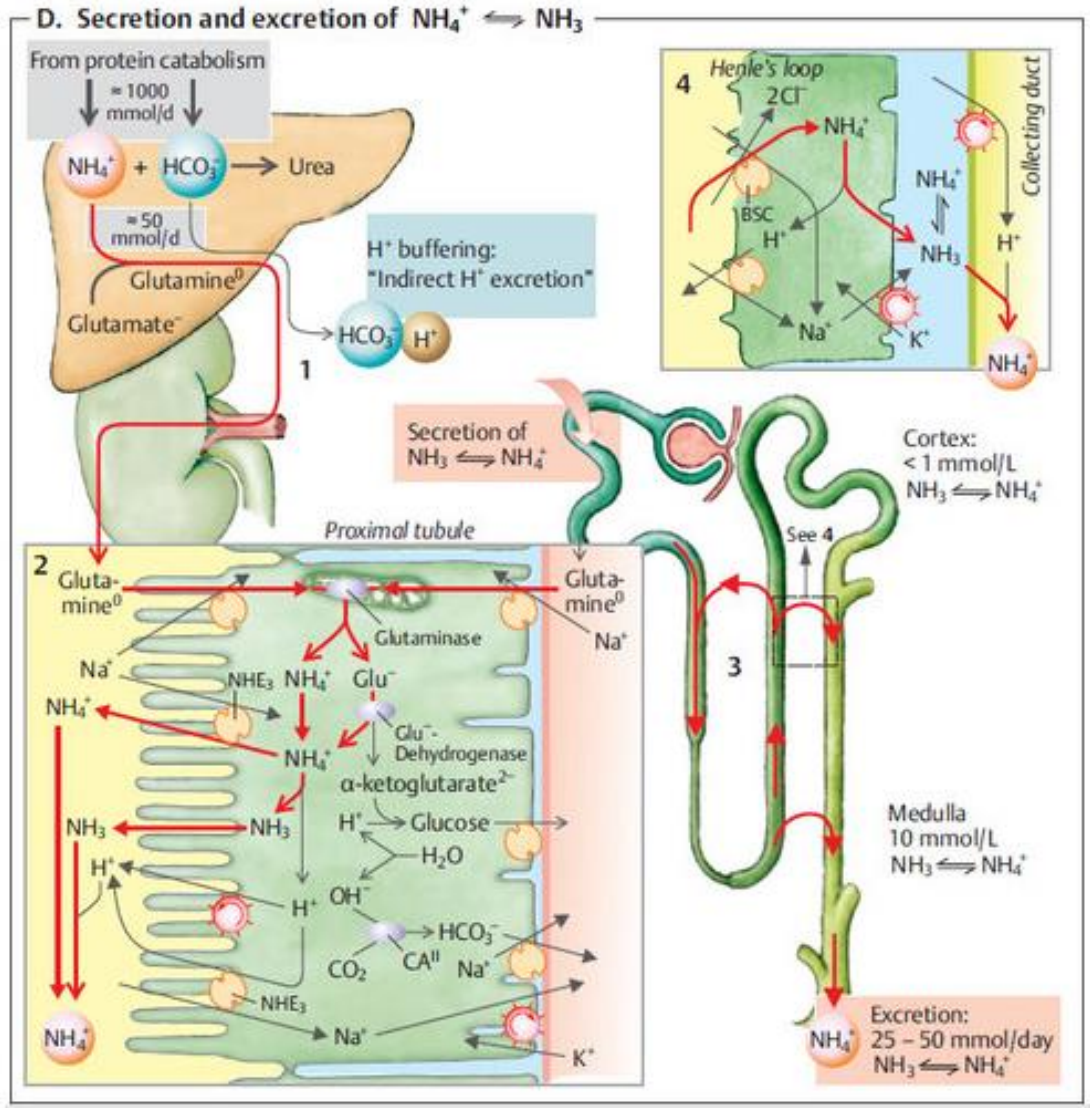
Foto: Shutterstock

What to remember ?

- Cerebral trauma :hyponatremia
- CSW-BNP orthostatic hypotensia
- SIADH- ADH without edema

Table differential diagnosis + effects aldosterone, ANF, RAA

Differentiation according compensatory reaction



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Acid base balance

pH= 7,4 +/- 0,02

Why?

pH influences

- (1) properties of proteins – enzyme activity

- (2) structure of cell components permeability of membranes - distribution of electrolytes

Protection

- Time dependency:
- Buffers
- Respiratory: Lungs, ventilation (12 hours)
- Metabolic: Kidney 3-5 days
- Liver

uniformwares.com/history-of-watches/

https://www.youtube.com/watch?v=WwkGoQQRTIo

Blood buffers

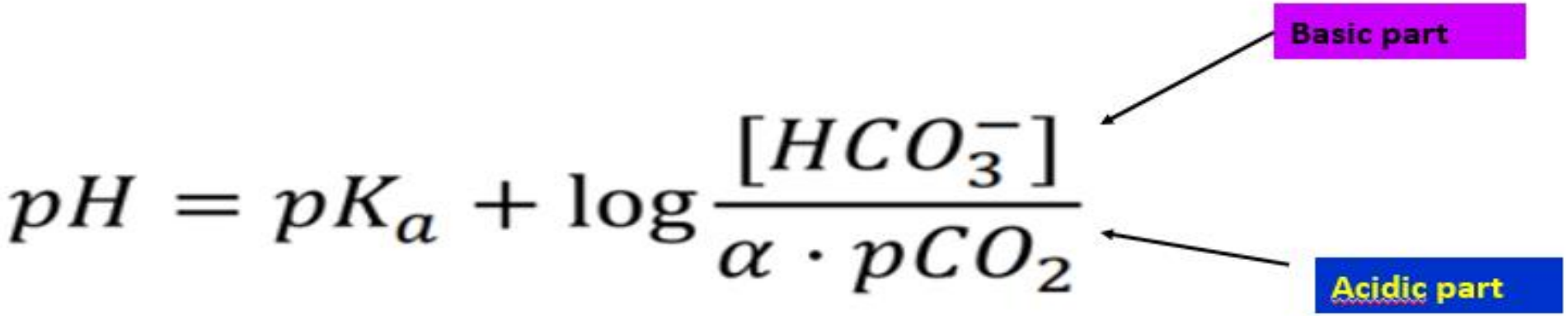
$$CO_2 + H_2O \rightleftharpoons HCO_3^- + H^+$$

- Hydrocarbohydrate 53%
- Haemoglobin 35%
- Others-plasma proteins, phosphate-organic inorganic

$$HbH \rightleftharpoons Hb^- + H^+$$
$$Oxy\text{-}HbH \rightleftharpoons Oxy\text{-}Hb^- + H^+$$

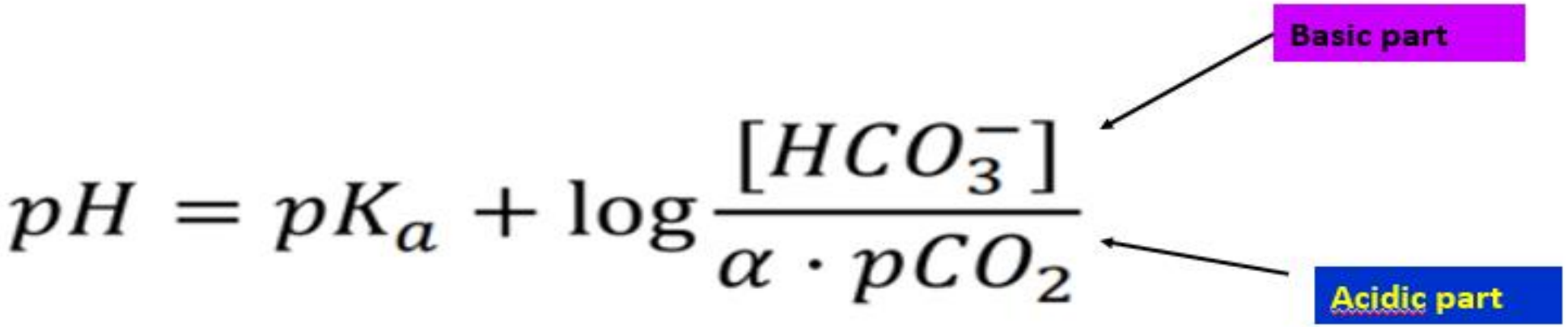
https://www.youtube.com/watch?v=LtNJy-rLpao

Henderson-Hasselbalch equation



- pKa = 6,1
- [HCO3 -] = 24 mmol.l-1
- α = 0,224 mmol.l-1 / kPa , pCO2 = 5,3 kPa

How will change the pH, when bicarbonate will increase ?



- Normal ratio is 20 to 1
- A) will be more acidic, pH decreases
- B) will be more acidic, pH increases
- C) no change

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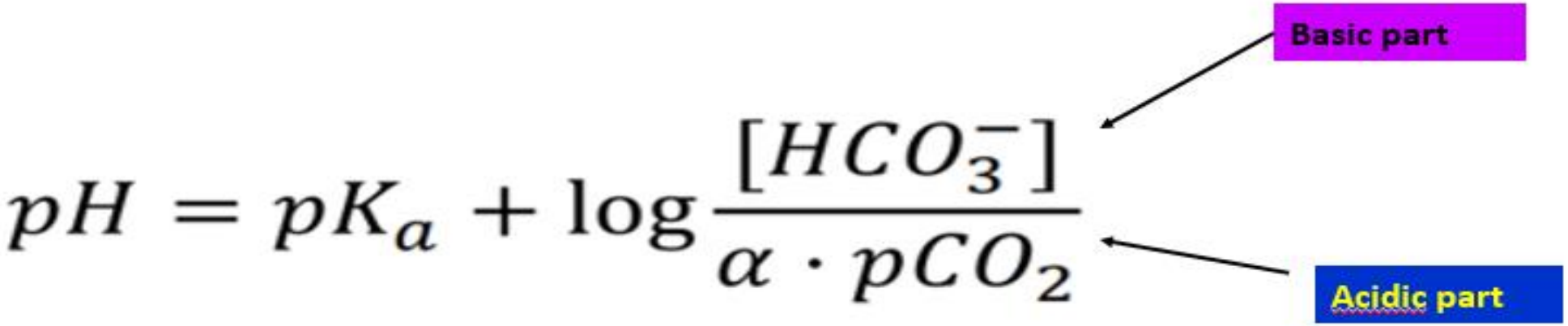
How will change the pH, when CO_2 will increase ?



- Normal ratio is 20 to 1
- A) will be more acidic, pH decreases
- B) will be more acidic, pH increases
- C) no change

Dissociation of weak acid-Buffer

More weak acid added, more HA is dissociated

More Base(A-) added, less HA is dissociated

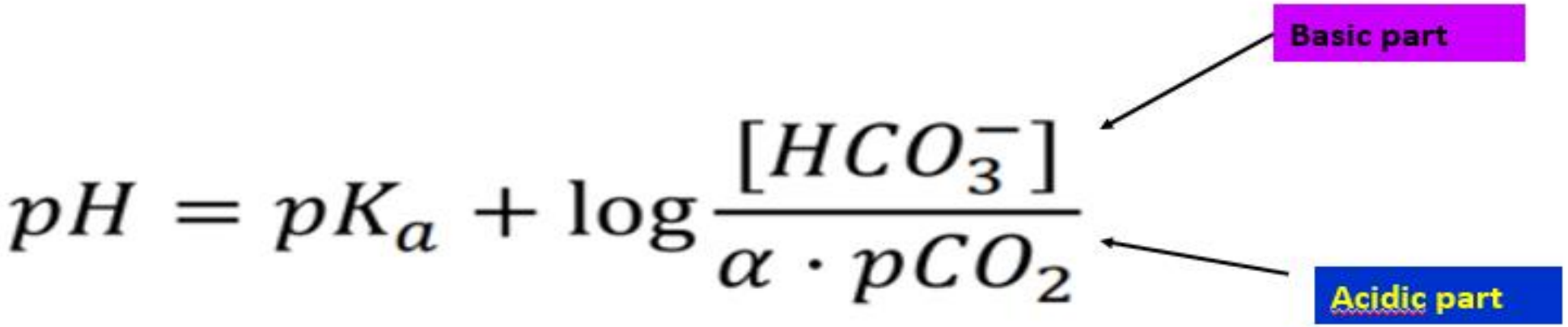
pH depends on the ratio

How does a buffer solution work?

- When adding H^+s, where they dissapear?

- When adding OH^-s, where they dissapear?

Henderson-Hasselbalch equation



- pH is determined by ratio of bicarbonate and carbondioxide

Metabolism H+ creation

Anaerobic glycolysis

Glucose= 2 $Lactate^-$ + $2H^+$

Lipolysis

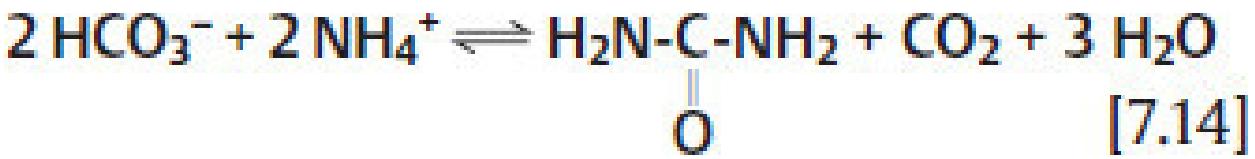
TAG=3 FFA^- +glycerol + $3H^+$

Ketogenesis

Palmitate + 0_2= $acetoacetate^-$ + 3 $hydroxybutyrate^-$ + 2 H_2O + $3H^+$

Metabolism H+ creation

Urea synthesis:



[7.14]

Utilisation of sulphur aminoacids

Utilisation of basic aminoacids

Metabolism H+ usage

- Gluconeogenesis from lactate:

$Lactate^- + H^+ = Glucose$

- Complete oxidation of lactate:
- $Lactate^- + O_2 + H^+ = CO_2 + H_2O$

- Utilisation of keton bodies:
- $3\text{-hydroxybutyrate}^- + H^+ = CO_2 + H_2O$

- Utilisation or organic anions:
- $Citrate^- + H^+ = CO_2 + H_2O$

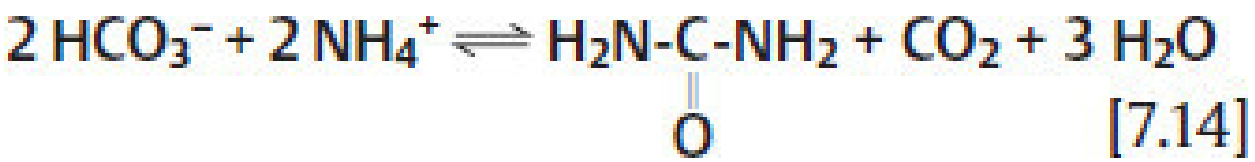
Metabolism H^+ usage

Utilisation of neutral aminoacids

Utilisation of acidis aminoacids

Metabolism H^+ creation

Urea synthesis:

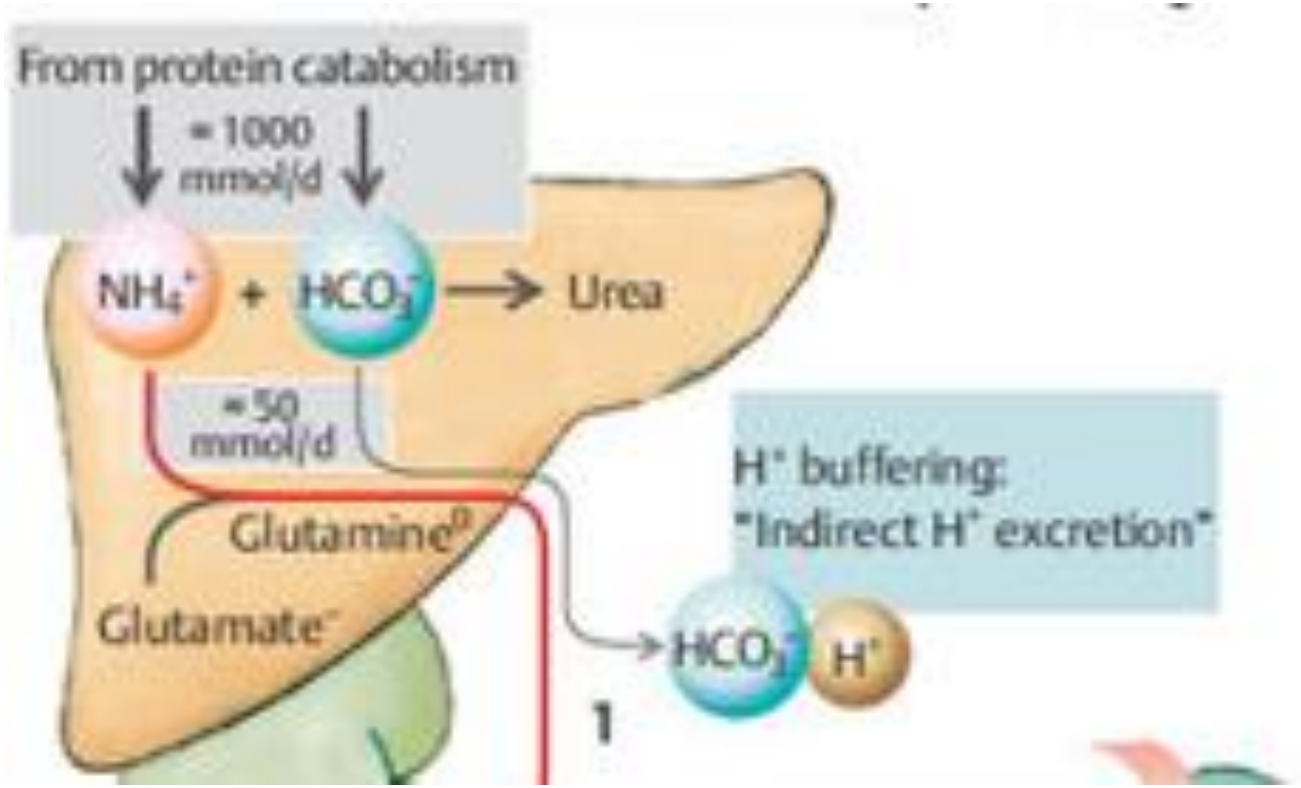


[7.14]

Utilisation of sulphur aminoacids

Utilisation of aminoacids with 2 dicarboxyls

Liver



- Compensation:
- In acidosis- H+ - to amonium cation to glutamine to kidney
- In alkalosis – use of bicarbonate to create urea

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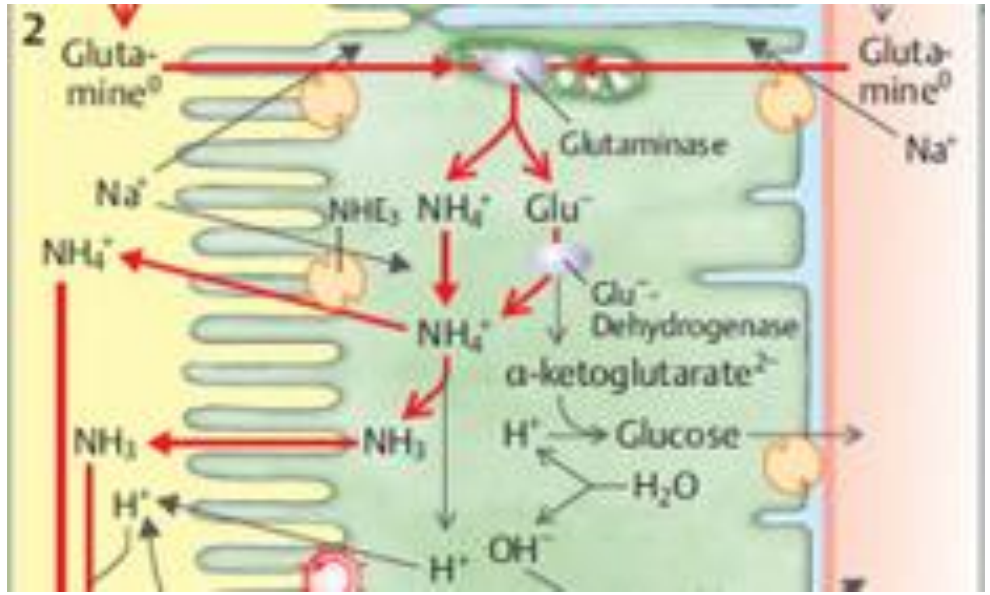
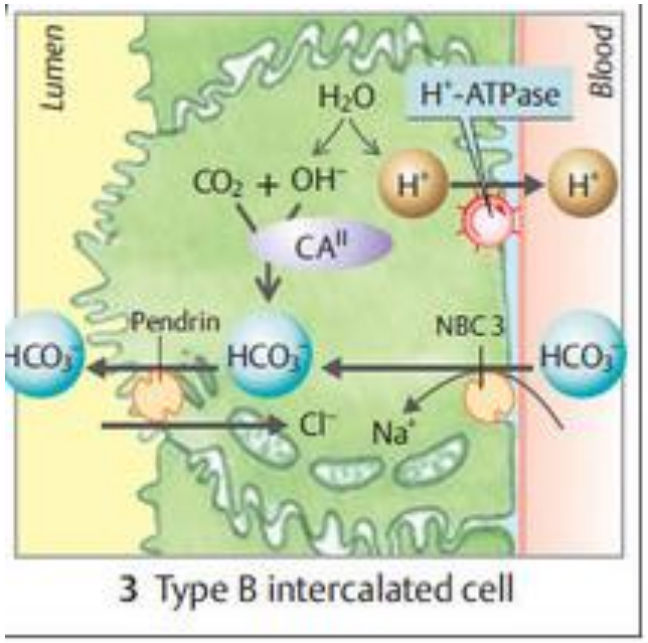
Kidney: Decide which process does compensate Alkalosis ?





H+ secretion:

- H+ (under 1%)
- As NH_4^+
- As phosphates

For reabsorbtion of HCO_3^-

HCO_3^- secretion

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How will pH influence K+ and ionized Ca^{2+} ?

Ion exchance-Synchronicity

- Ionized Ca^{2+} and H^{+}
- K^{+} and H^{+}

Hypochloremic alkalosis

- Primary cause:
- Compensation:
- Ions:
- Cave: dissociation of oxygen is more difficult- tissue hypoxia

Metabolic alkalosis

Dehydratation

Hypoalbuminemia

Vomiting

Ketoacidosis-Metabolic acidosis

- Primary Cause
- Compensation:
- K+ loss

Metabolic acidosis

Anion gap (AG) +

Diabetic Ketoacidosis
Lactate acidosis
Starvation
Uremic acidosis
Intoxications

AG normal
(chlorides suplement bicarbonate)

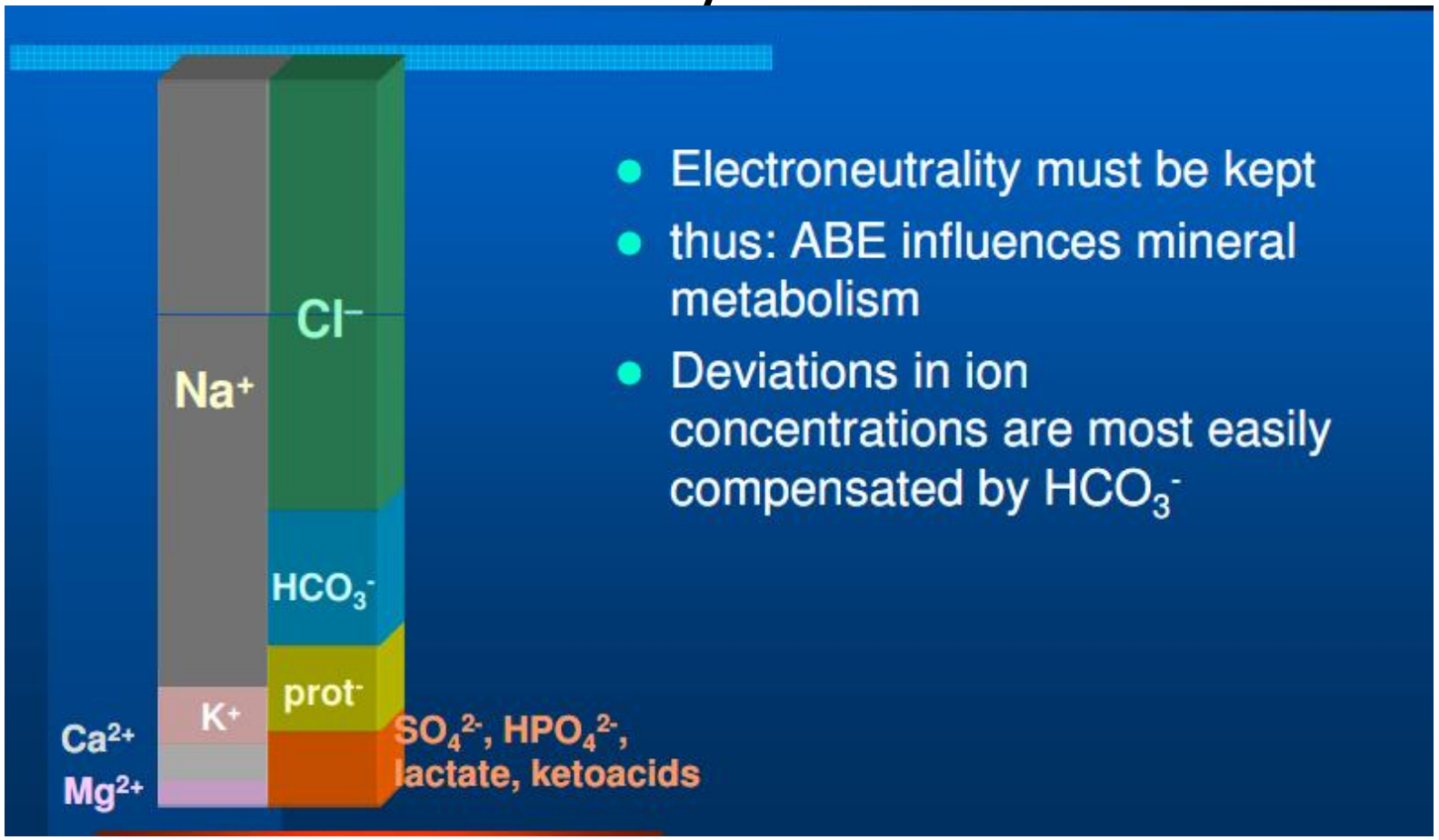
- GIT (diarrhoea)
- RTA (renal tubular acidosis)

Metabolic acidosis AG

G	Glycols (ethylene glycol and propylene glycol)
O	Oxoproline
L	L-Lactate
D	D-Lactate
M	Methanol
A	Aspirin
R	Renal Failure (Uremia)
K	Ketoacidosis

https://knowmedge.com/blog/medical-mnemonics-anion-gap-acidosis-goldmark/

Electroneutrality

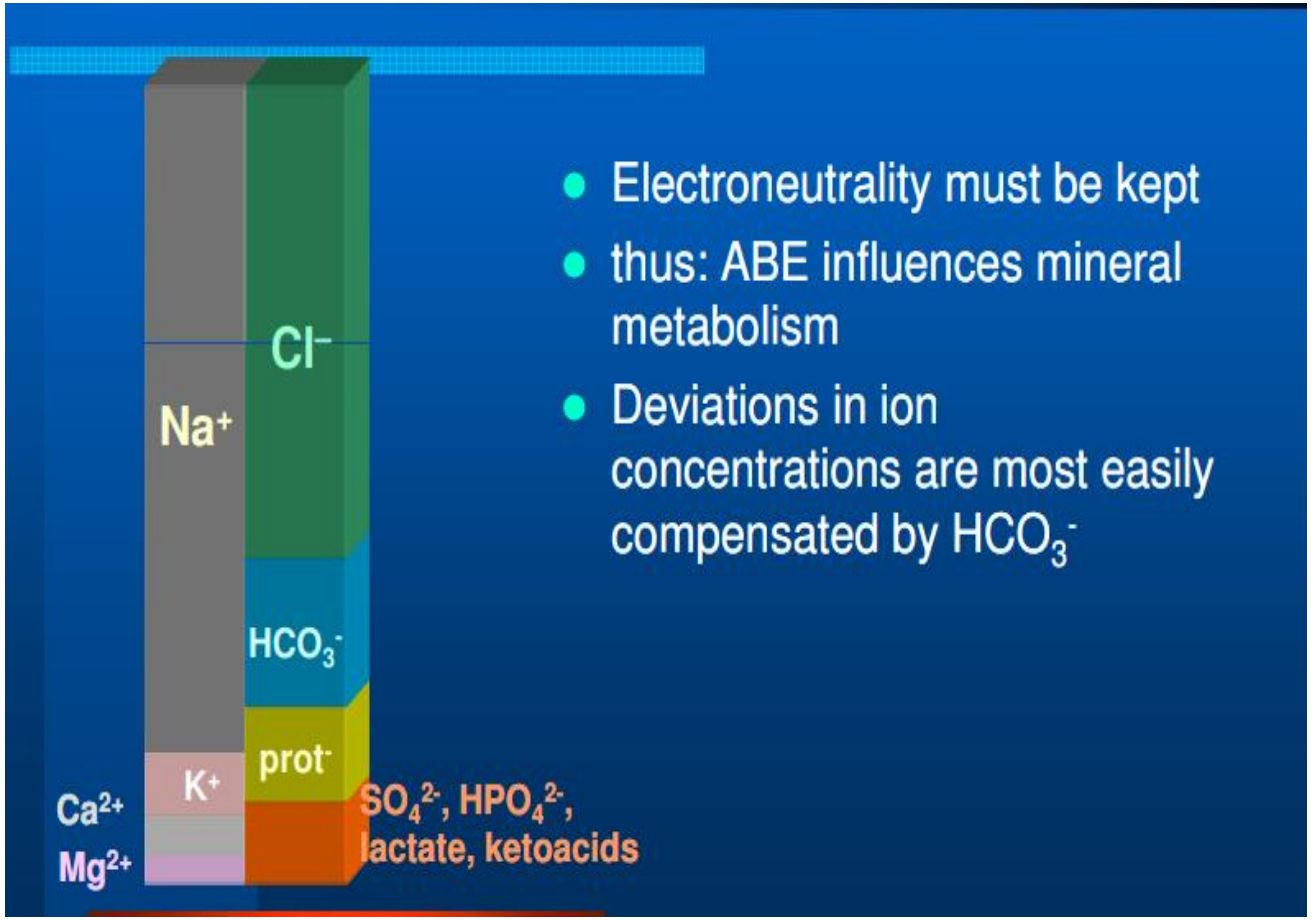


Picture: from Dr. Vejražka

Electroneutrality

- Strong iont diference:
- SID= Cations-Cl^-- UA^-
- Approximation:
- SID= Na^+-Cl^-
- 34mmol/L

- Anion GAP:
- AG= $Na^+ + K^+$-Cl^--HCO_3^-
- 12mmol/L



Picture: from Dr. Vejražka

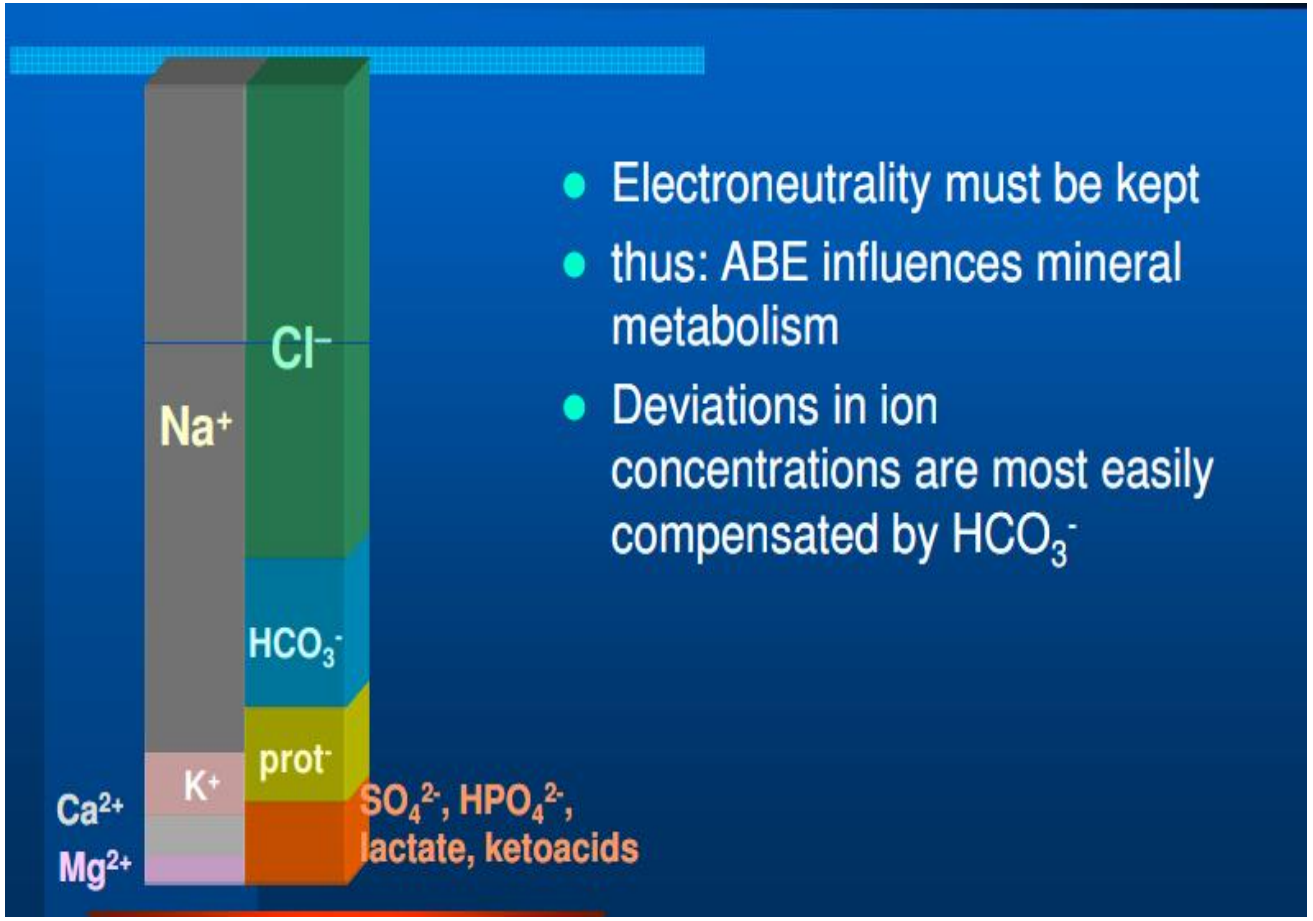
Albumine

- 10g/l = 3mM
- Albumine:15 mM

Electroneutrality

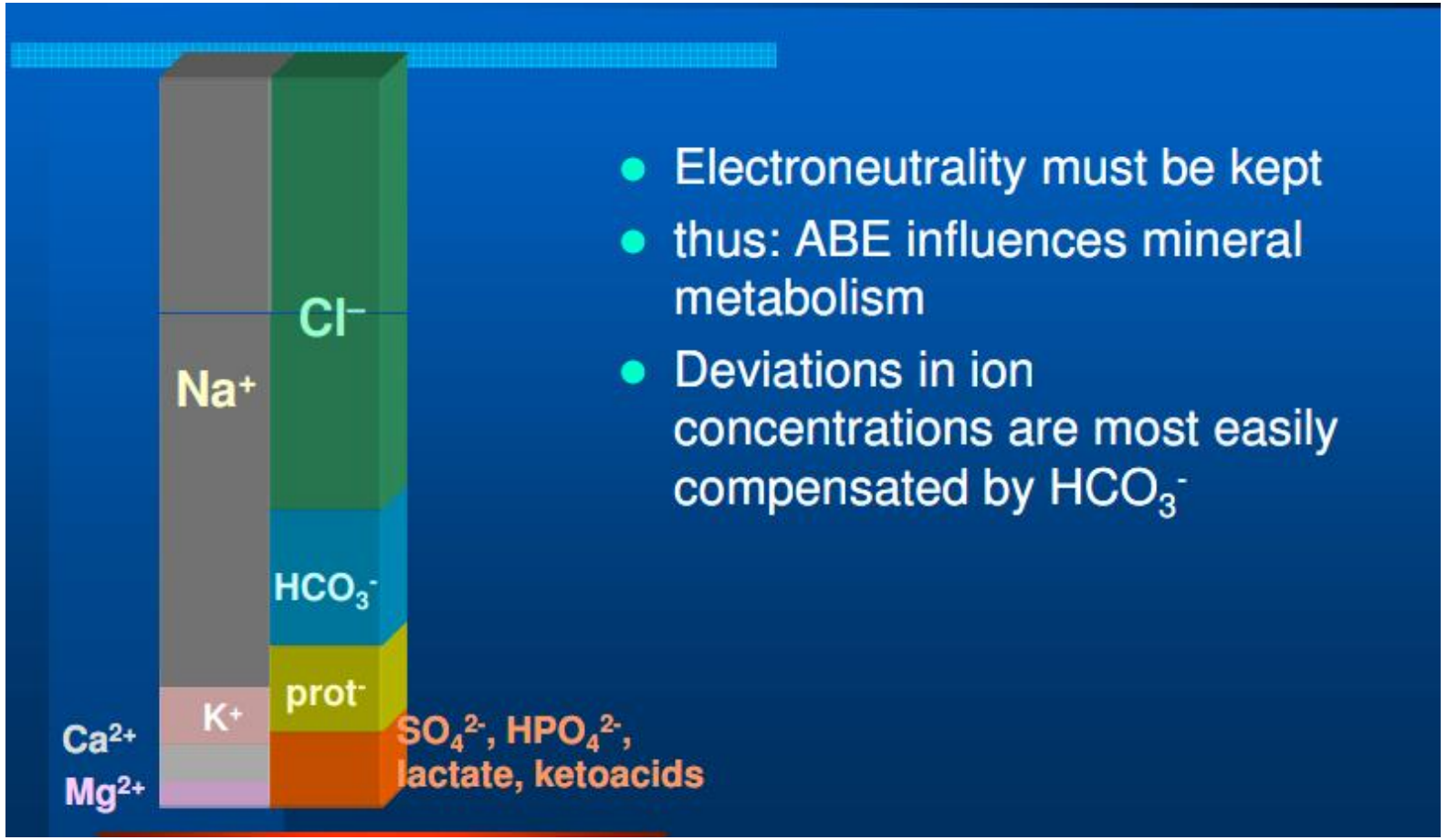
- Strong ion difference:
- SID= Cations-Cl^- -UA^-
- Approximation:
- SID= Na^+-Cl^-
- 34mM/L

- Anion GAP:
- AG= $Na^+ + K^+$-Cl^--HCO_3^-
- 12mmol/L



Picture: from Dr. Vejražka

Electroneutrality



Picture: from Dr. Vejražka

Respiratory acidosis

Shutterstock.com

- Causes: pulmonary infection

- Compensation:
- excretion of protons into urine

Respiratory acidosis

Shutterstock.com

- Causes:
- Obstructive or restrictive pulmonary disease
- Brain: depresion of breathing centre (trauma, tumor)

Respiratory alkalosis

https://tenor.com/view/tantrum-gargamel-azrael-smurfs-no-gif-21416353

- Causes: stress hyperventilation
- Ion Ca2+ low cramps
- Vasoconstriction – loosing consciousness
- Therapy : calming down, breathing into paper/plastic bag

How can you distinguish hyperventilation in respiratory alkalosis by emotional stress from Kussmaul breathing as a compensation of metabolic acidosis ?

Respiratory alkalosis

- Stress, pain
- Hypoxia
- Salycilate
- Brain(tumor, trauma)

https://www.newindianexpress.com/lifestyle/books/2022/oct/30/himalaya-exploring-the-roof-of-the-world-demystifying-the-high-and-mighty-2512316.html

What to remeber?

	Primary cause	pH	Compensation
Respiratory acidosis	CO_2 ↑	Acidemia ↓	Kidney: HCO_3^- ↑
Respiratory alkalosis	CO_2 ↓	Alkalemia ↑	Kidney HCO_3^- ↓
Metabolic acidosis	HCO_3^- ↓	Acidemia ↓	Respiration: CO_2 ↓ Hyperventilation Kussmaul Breathing
Metabolic alkalosis	HCO_3^- ↑	Alkalemia ↑	Respiration CO_2 ↑

- Metabolic : synchronic :)