Disturbances in water management

Osmolality

Disturbances in water management:

1/ ECF is hyperosmolar

2/ ECF is isoosmolar

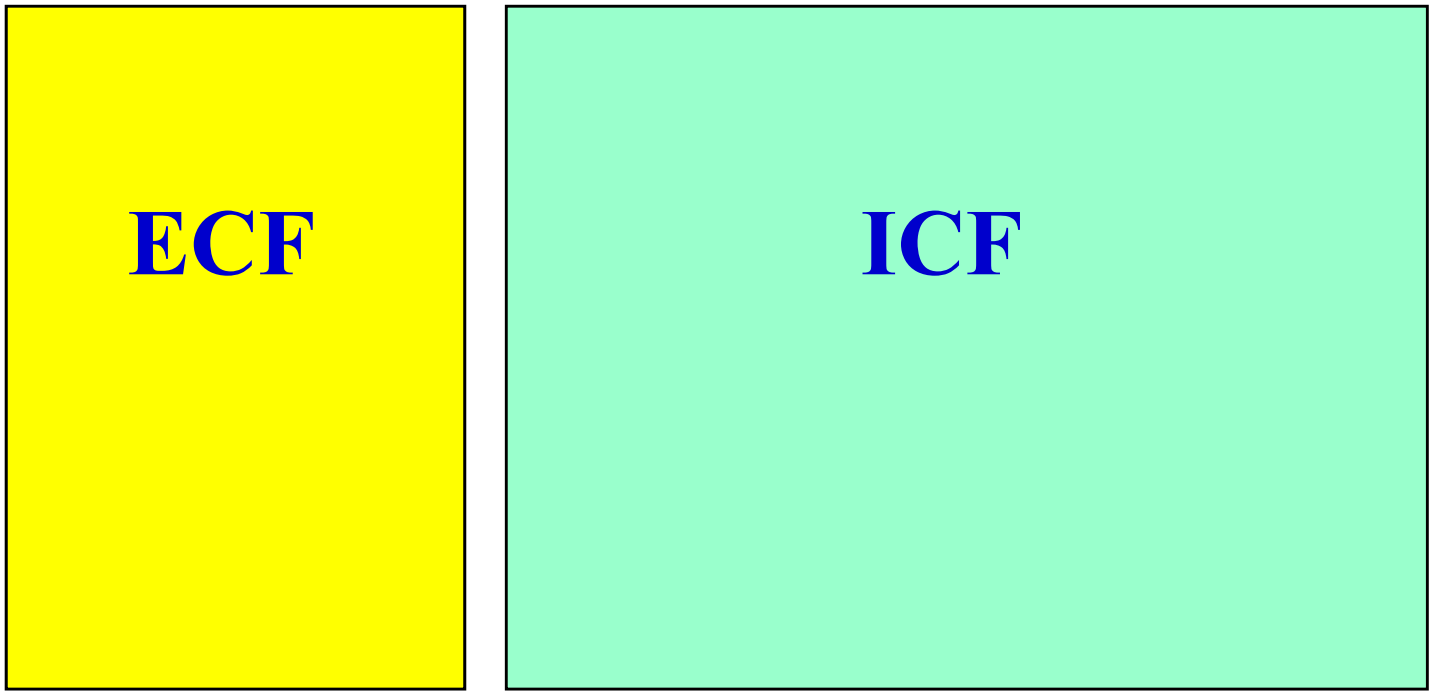
3/ ECF is hypoosmolar

Arrange of next schemas :

1/ **inicial situation (disturbance)** ⇨ **advanced situation**

2/ **the name of disturbances is according to changes in ECF:**
(„hyper-/iso-/hypo-TONIC + over-/de-HYDRATATION“)

3/



extracellular fluid **intracellular fluid**

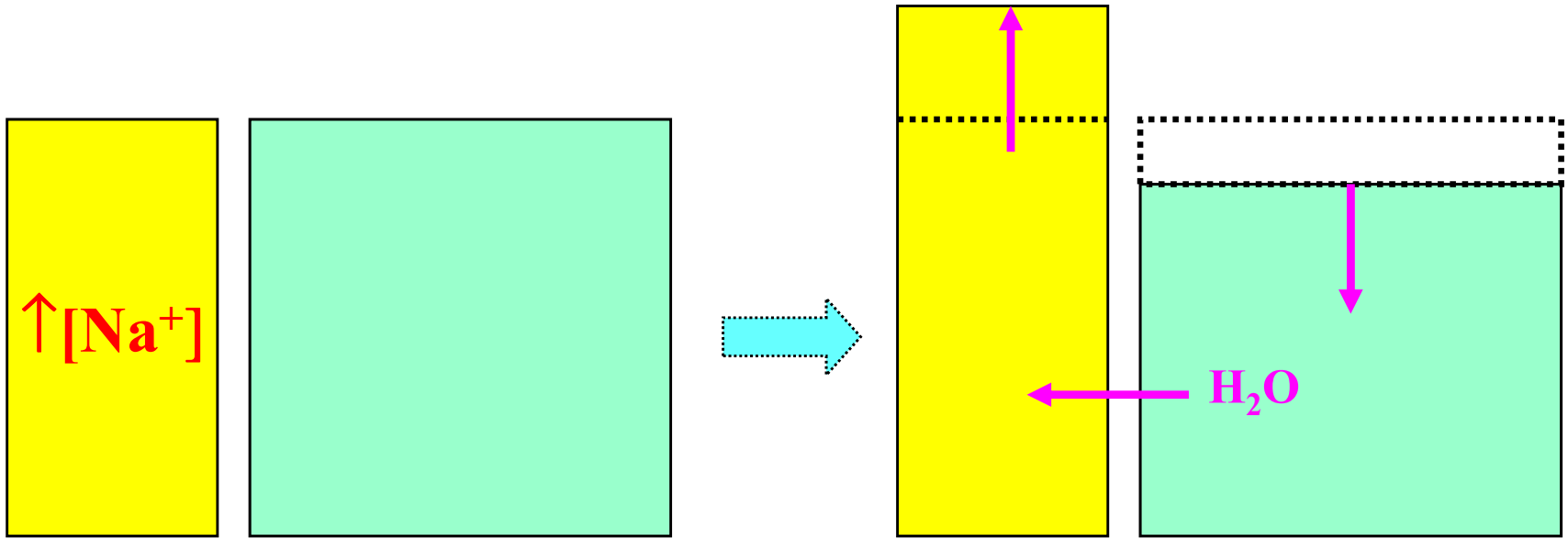
ECF is hyperosmolar :

1/ retention / supply Na^+

2/ loss of „pure“ water

Retention / supply Na^+ :

ECF is hyperosmolar



- water to the ECF → expansion of ECF
- edema – danger of pulmonary edema !
- deficit of water in ICF → disturbances of CNS

Retention / supply Na^+ : = hypertonic overhydration

causes:

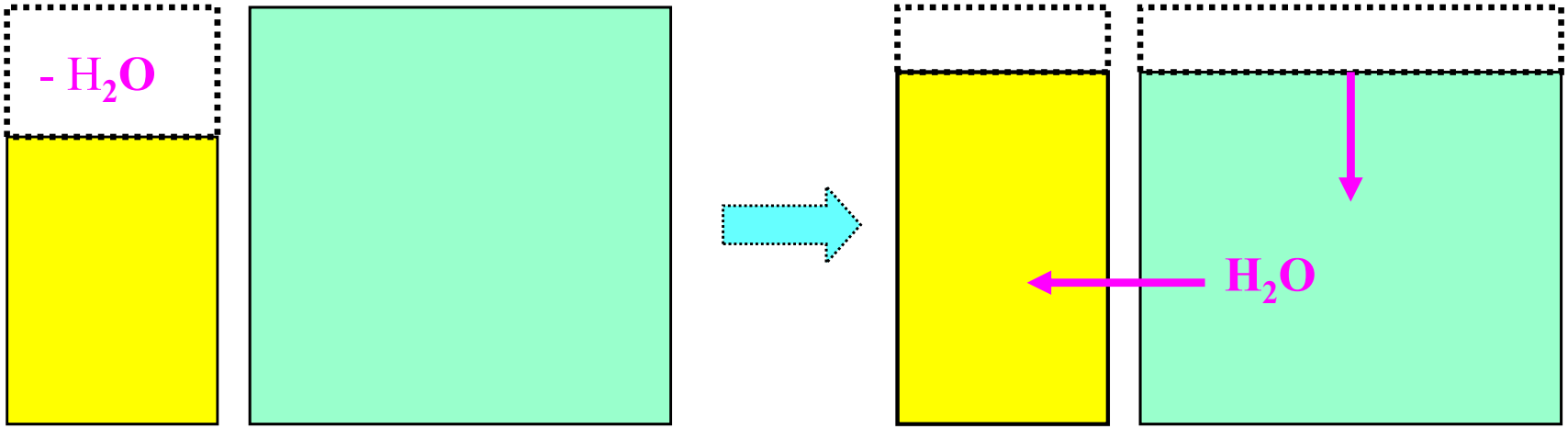
excessive administration of salt
overactivity of adrenal cortex (Conn, Cushing)
administration of steroids
cerebral „salt retention syndrome“
drinking of see-water (after shipwreck)

symptoms:

vomiting
diarrhoae
labile blood pressure
changes in central venous pressure
pulmonary oedema
restlessness

Loss of „pure“ water :

ECF **is hyperosmolar**



(normal hematocrite)

- **water to the ECF**
- **deficit of water mainly in ICF → disturbances of CNS**

Loss of „pure“ water : = hypertonic dehydration

causes:

inadequate water intake (failing feeling of thirst in old persons)
increased water losses due to sweating
osmotic diuretics
hyperventilation
chronic nephropathy
polyuric phase of akute renal failure
diabetes insipidus

symptoms:

thirst
fever
dryness
restlessness
delirium, coma

ECF is isoosmolar:

1/ loss of isotonic fluid
(→ blood circulation disturbances)
2/ isoosmotic expansion of ECF
(→ edema)

because of the same osmolality, no transfers of water between ECF and ICF,
the changes are in the volume of ECF only.

Isotonic fluid losses : = isotonic dehydration

causes:

vomiting
diarrhoea
fistulae
diuretics
drainage of ascites
burns
sedative and CO intoxication
sunstroke

symptoms:

thirst
tiredness/faiting
weakness
rapid puls
hypotonia
collapse
vomiting
muscle cramps

Isoosmotic expansion of ECF :

= isotonic overhydration

causes:

excessive administration of isotonic infuson solutions
in oliguric and anuric states
cardiac failure
nephrotic syndrome
chronic uraemia
acute glomerulonephritis
cirrhosis of liver
protein-losing enteropathy

symptoms:

oedema
effusions
dyspnoea

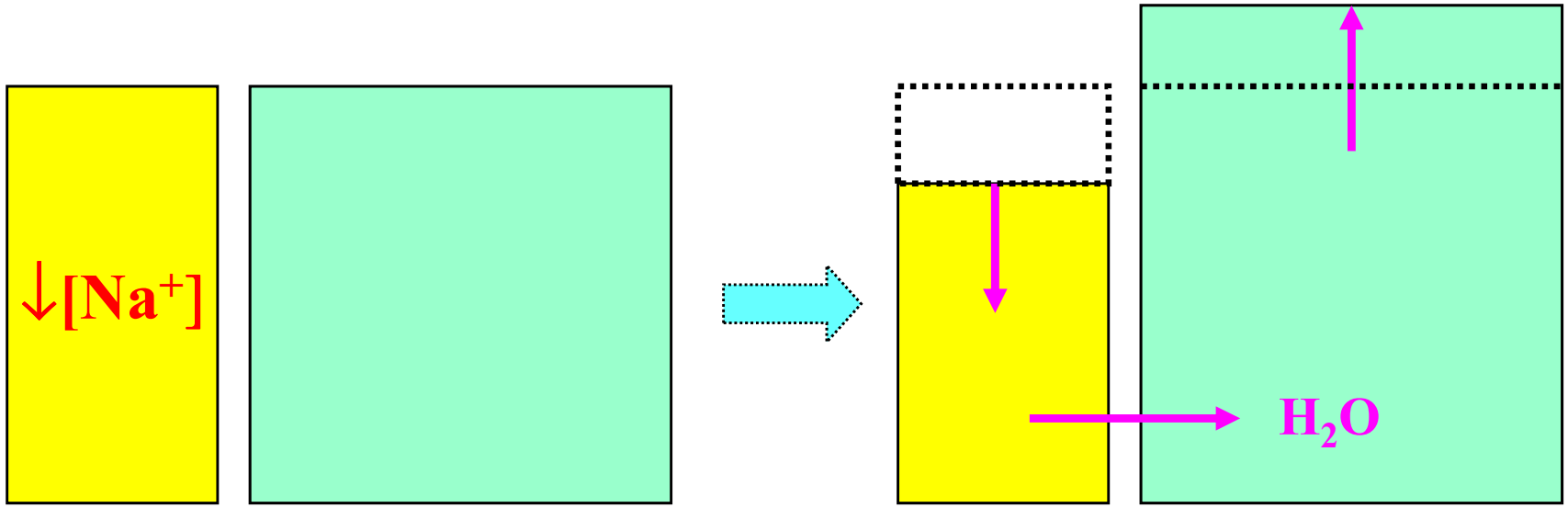
ECF is hypoosmolar:

1/ loss of „pure“ Na^+

2/ water intoxication

Loss of „pure“ Na^+ :

ECF is hypoosmolar



- leakage of water into **ICF** → ↑ intracranial pressure
- hypovolemia of **ECF** → blood circulation disturabces

Loss of „pure“ Na^+ : = hypotonic dehydration

causes:

iadequate sodium intake

after its losses through vomiting, diarrhoea and sweating

increased sodium losses due to adrenal failure

chronic diuretic therapy

diarrrhoea

fistula losses

symptoms:

tiredness/faiting

weakness

hypotonia

rapid puls

collapse

vomiting

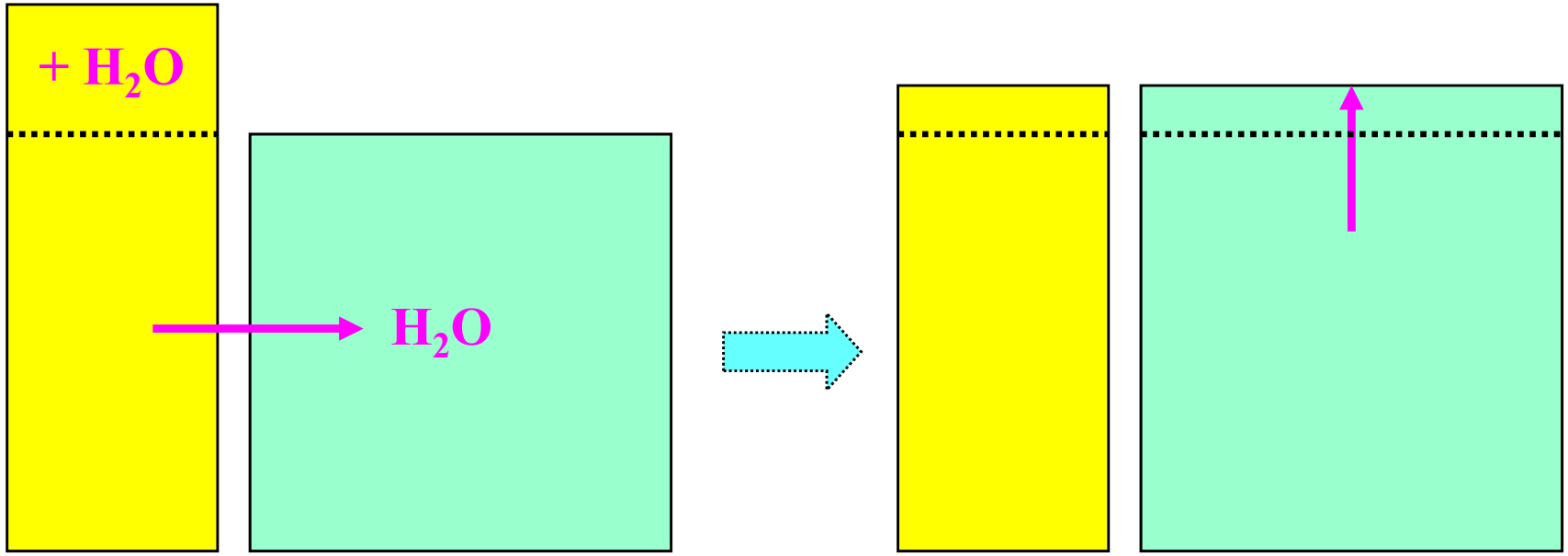
fever

muscle cramps

depressed conscinous level

Water intoxication :

ECT **je hypoosmolar**



- **leak of water into** **ICF** → ↑ **intracranial pressure**

Water intoxication : = hypotonic overhydration

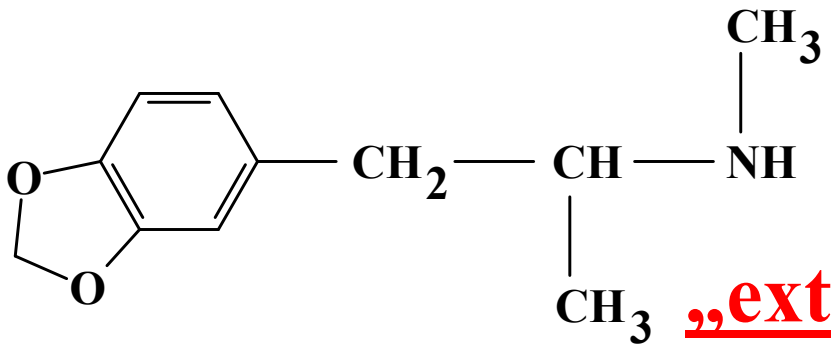
causes:

excessive administration of salt-free solutions
gastric lavage with water
increased ADH activity

příznaky:

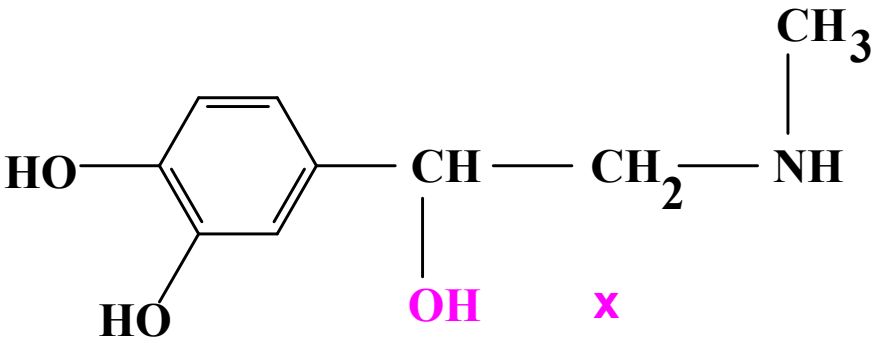
weakness
nausea
vomiting
dyspnoea
confusion
loss of consciousness

Intoxikace vodou
Water intoxication

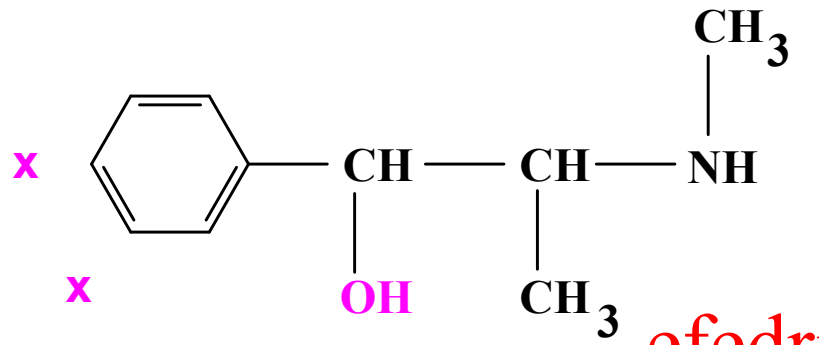


„extáze“ / „ecstasy“

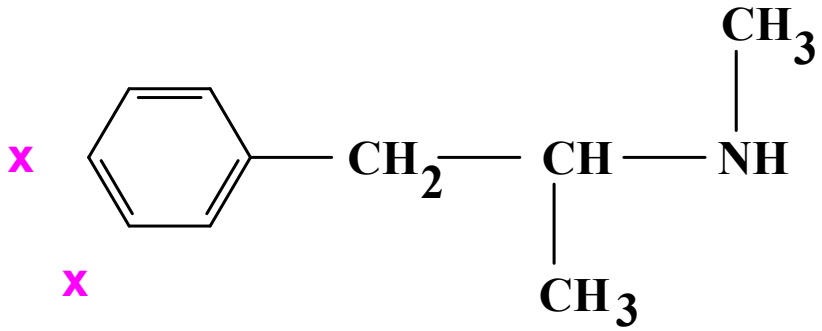
(= hypotonická hyperhydratace po požití „extáze“)



adrenalin / epinephrine



efedrin / ephedrine



„pervitin“

hlavní strukturní rozdíly extáze a příbuzných látek
the main structural differences among „ecstasy“ and similar substances

Water – losses (1):

Insensible losses:

normal temperature	550 ml . d^{-1}
37.2 °C	600
37.8	700
38.3	800
38.9	900
39.4	1,000

Sweating:

mild	300 ml . d^{-1}
medium	600
strong	1,000
(continual	2 ... 15 L . d^{-1} !!) [19]

Water - losses (2):

Breathing: 440 ml . d^{-1} (hyperventilation ?)

Urine: (diuretics ?!)

Stool: 100 ml . d^{-1} (diarrhoea ?!)

Suction, drains: ???

Water – metabolic gain:

Terminal oxidation: 300 - 500 ml . d^{-1}

Water - losses (3):

Sweat: • at the maximal load till 2 L . h^{-1} !!

• the loss of water exceedes the loss of salt

• common composition: 58 mmol Na^+. L^{-1}
10 mmol K^+ . L^{-1}
45 mmol Cl^- . L^-

1

Excretion of water : **60 % kidney (urine)**
20 % skin (sweat)
15 % lungs
5 % stool

Water – intake:

intake of water: **1/ beverages**

2/ food (solid, halfsolid)

3/ oxidation of nutrients:

1 g fat → 1.07 ml water

1 g sugar → 0.55 ml water

1 g protein → 0.41 ml water

We have to pay close attention to the intake of water in small children and in eldery persons, in which the intake of liquids is insufficient, because of absent/weakened feeling of thirst.

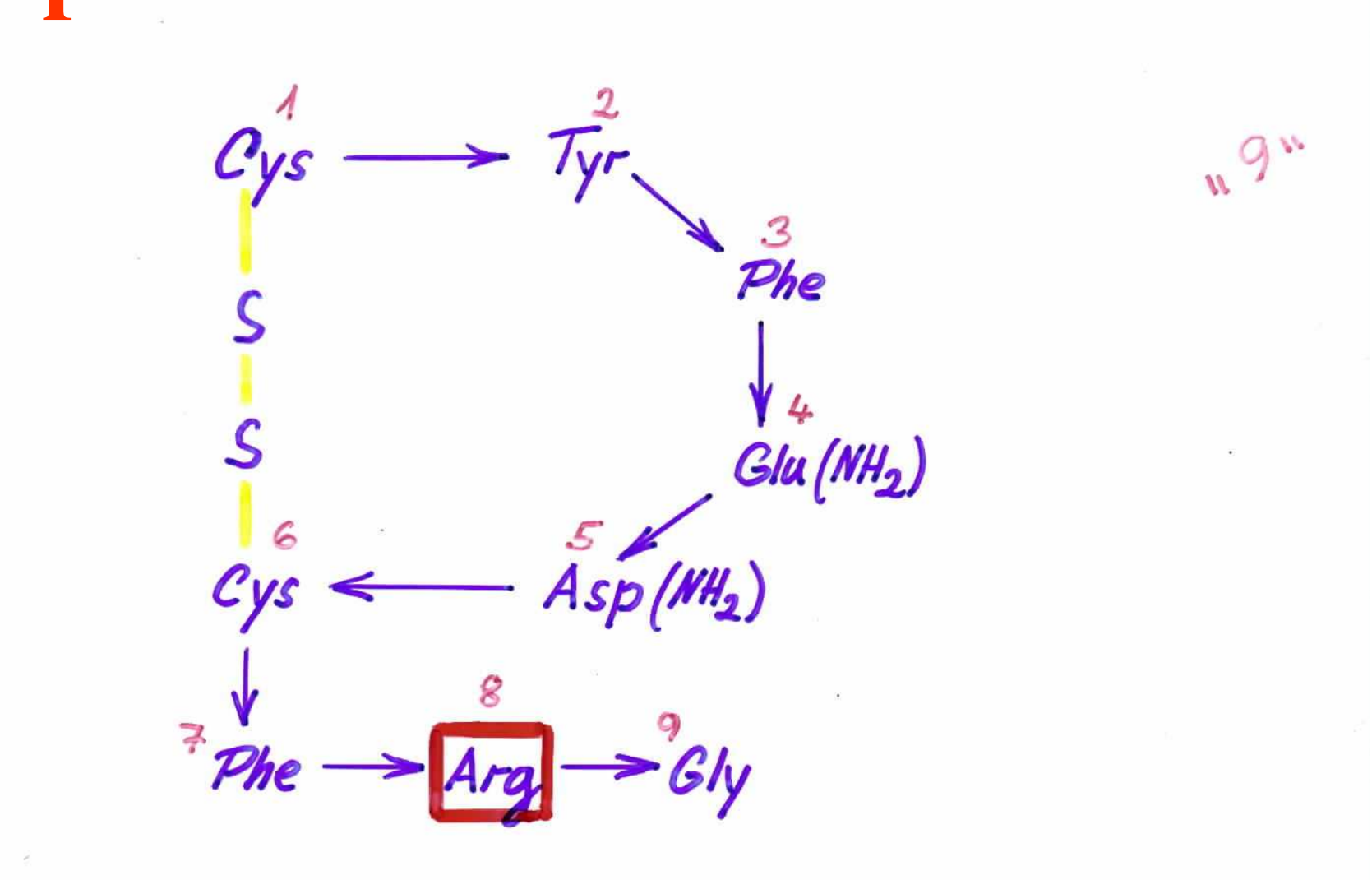
Management of water :

1/ adiuretin (antidiuretic hormone, vasopressin)

2/ RAAS (renin – angiotensin – aldosterone system)

3/ natriuretic peptides

Antidiuretic hormone (ADH) vasopressin



Angiotensins

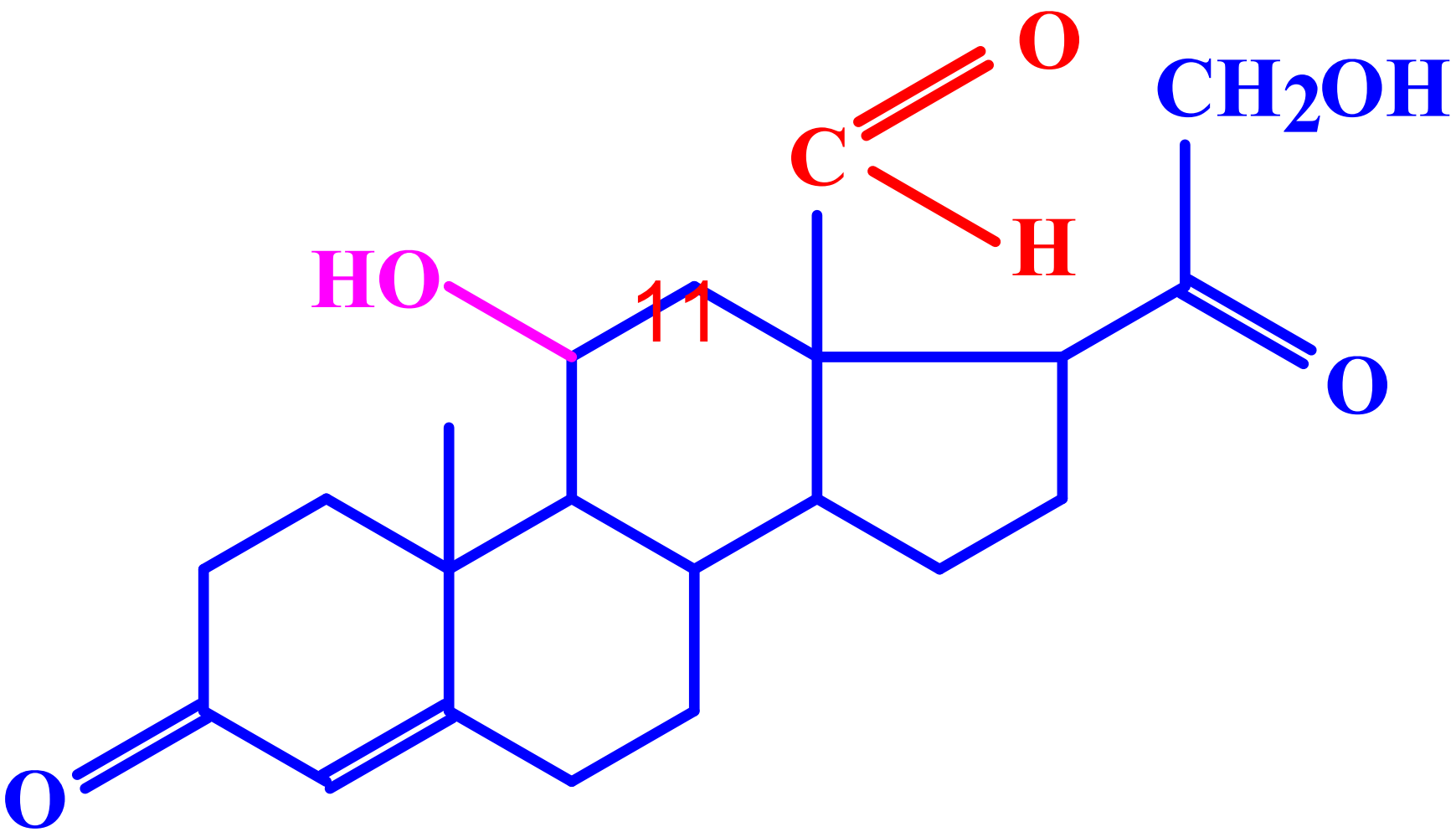
Asp → Arg → Val → Tyr → Ile → His → Pro → Phe → His → Leu

Asp → Arg → Val → Tyr → Ile → His → Pro → Phe

Arg → Val → Tyr → Ile → His → Pro → Phe

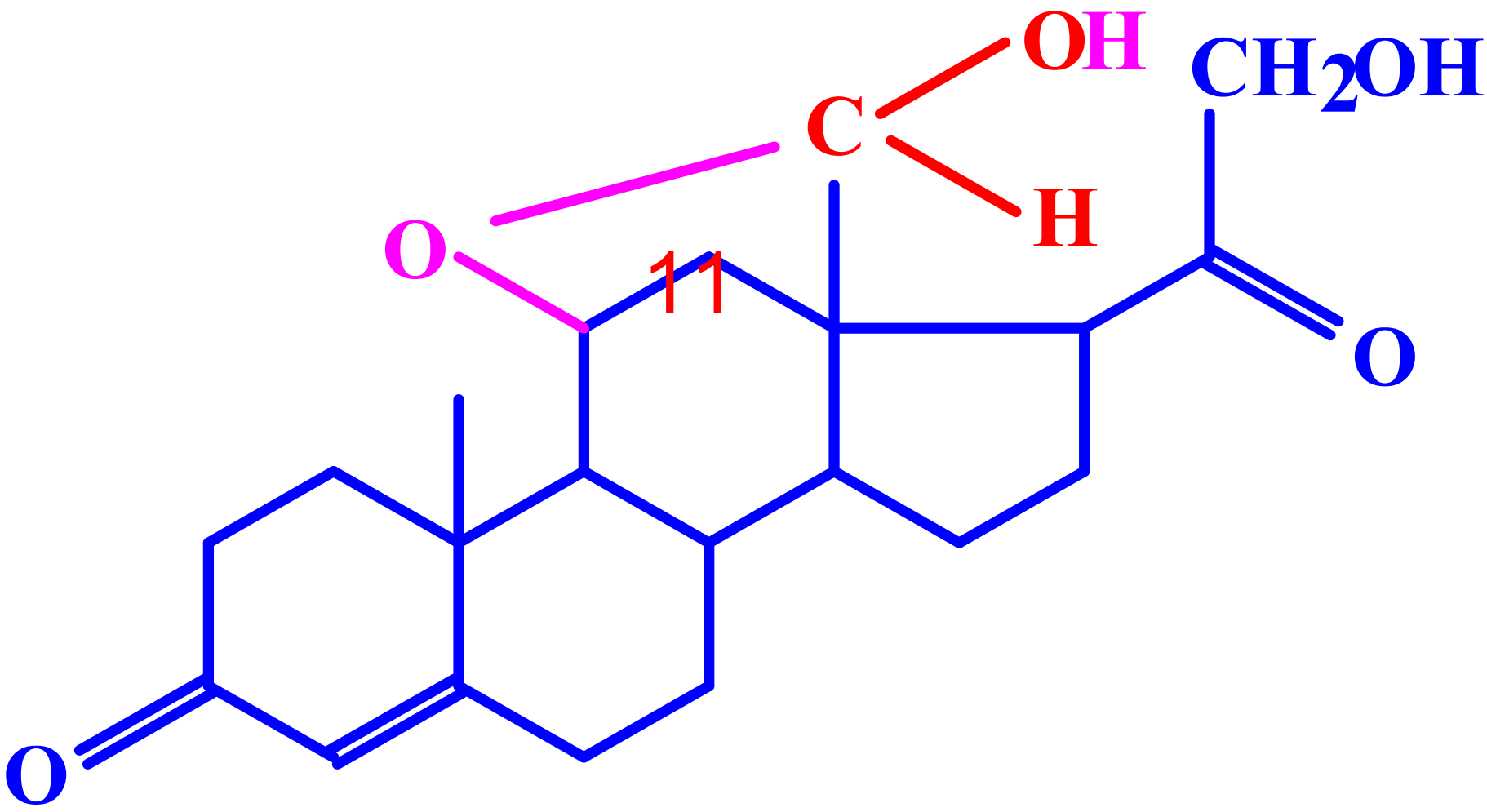
The structures of linear peptides are drawn:
angiotensin I (10 AA), angiotensin II (8 AA, two AA were split off at the carboxylic end) and angiotensin III (7 AA, is without one AA at amino end of the chain).
Angiotensin I is produced from α_2-globulin of blood plasma (angiotensinogen – the protein of liver origin), without physiological effect. Angiotensins II + III are effective vasopressoric substances (increase blood pressure), they stimulate formation and secretion of aldosterone (the main mineralocorticoid, adrenal zona glomerulosa).

aldosteron/e :



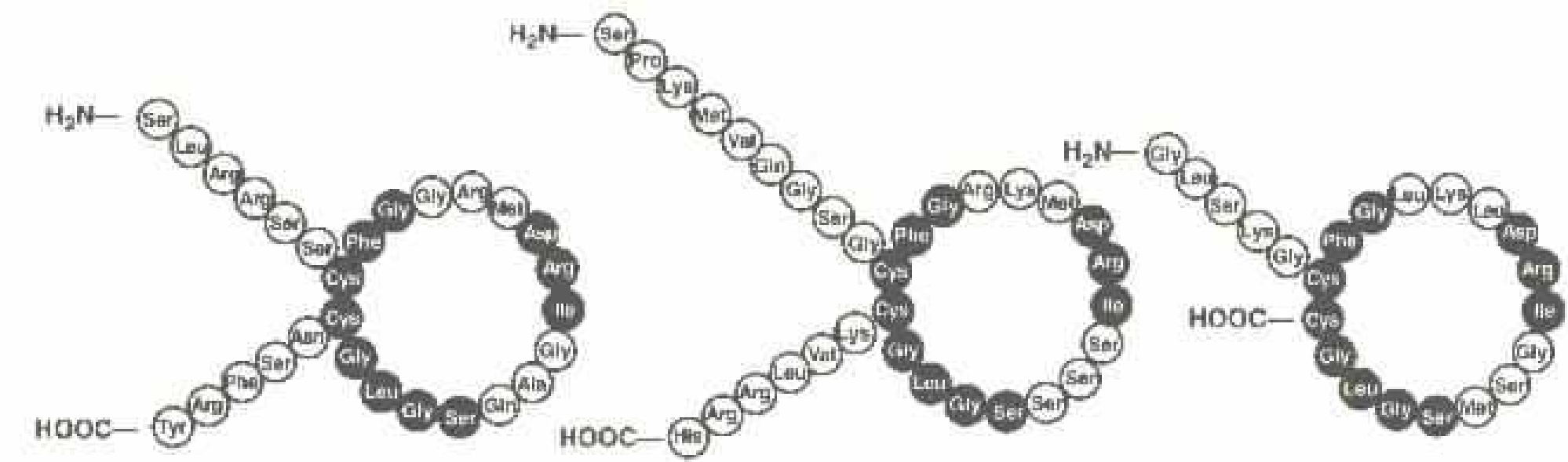
(11β,21-dihydroxy-3,20-dioxo-4-pregnen-18-al)

aldosteron/e (hemiacetal) :



(11β,18-epoxy-18,21-dihydroxypregn-4-en-3,20-dion)

Natriuretic peptides



ANP **BNP** **CNP**

28 AA **32 AA** **22 AA**

P-[pmol . l^{-1}] **[traces]**

17membered rings: (...Cys – S – S – Cys …)

„VASODILATATION, NATRIURESIS, DIURESIS“

Natriuretic peptides

Precursors : 126 AA → ANP (28 AA)
108 AA → BNP (32 AA)
53 AA → CNP (22 AA)

NP split off at the C-terminal end
- short biological half-lifes

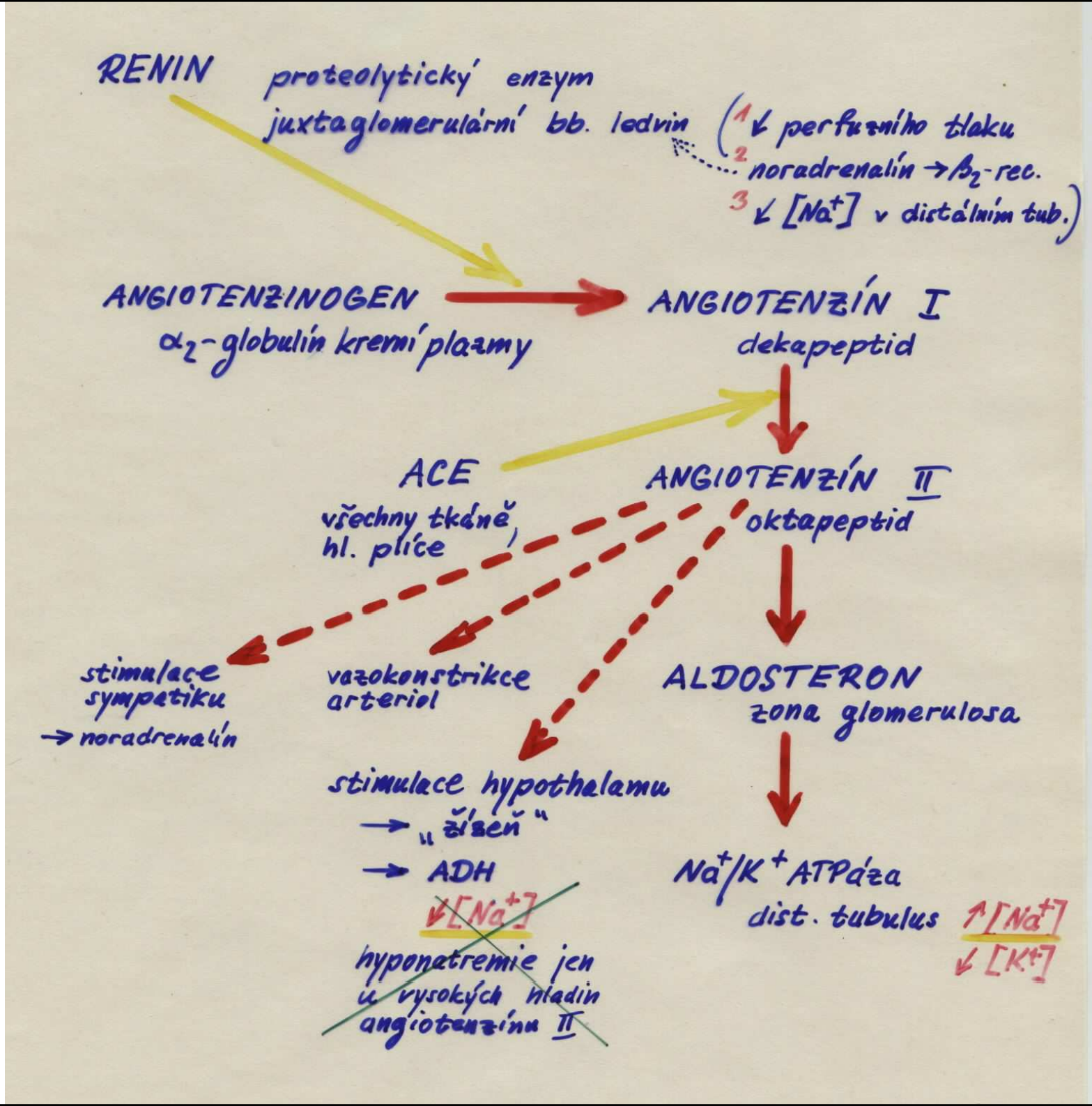
Inaktiv N-terminal parts
- longer biological half-life → frequently determinated

NP receptors : transmembrane type,
trasport cGMP

Natriuretic peptides

ANP = „atrial“ - mainly from the atrial heart wall
- the answer to increased stretch of muscle
(due to increased volume of blood)
BNP = „brain“ (first isolated from a pork brain).
Despite the name it has origine predominantly
in the heart atria.
CNP = „C-type“

NP are the protection against a liquid overload and a hypertension.
ANP + BNP are of hormone properties,
CNP is close to paracrine factor.


RENIN
proteolytický enzym
juxtaglomerulární bb. ledvin
1 ↓ perfuzního tlaku
2 noradrenalín → β2-rec.
3 ↓ [Na+] v distálním tub.
ANGIOTENZINOGEN
α2-globulin krevní plazmy
ANGIOTENZÍN I
dekapeptid
ACE
všechny tkáně, hl. plíce
ANGIOTENZÍN II
oktapeptid
stimulace sympatiku
→ noradrenalín
vazokonstrikce arteriol
ALDOSTERON
zona glomerulosa
stimulace hypothalamu
→ „žízeň"
→ ADH
↓ [Na+]
hyponatremie jen u vysokých hladin angiotenzinu II
Na+/K+ ATPáza
dist. tubulus
↑ [Na+]
↓ [K+]

The ratio $[Na^+]$ / $[K^+]$ in urine:

U-$[Na^+]$ / U-$[K^+]$ ≅ 2,4

(generally > 1)

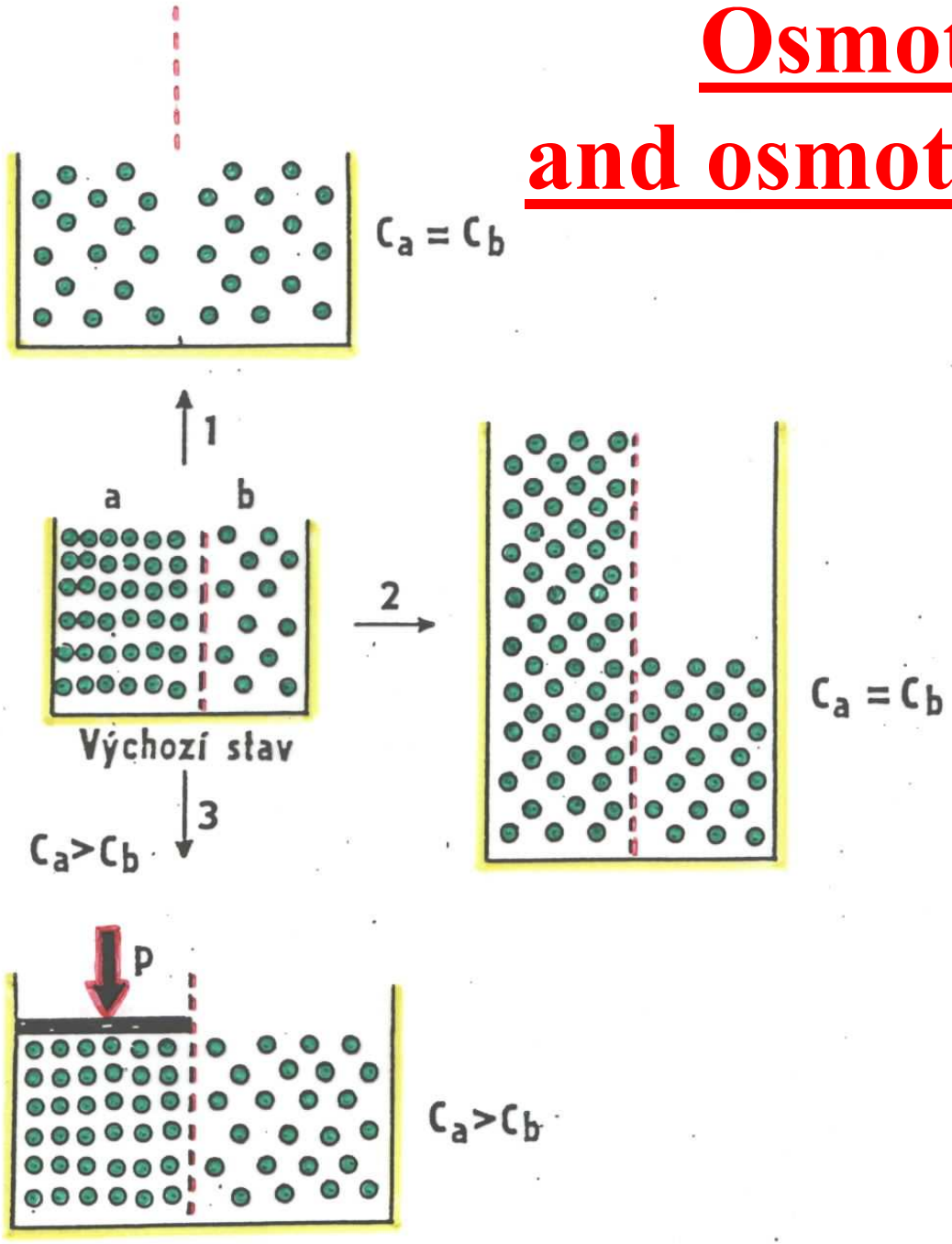
< 1 → „hyperaldosteronismus“

(accidental sample of urine is sufficient
for determination, we cannot know the volume)

OSMOLALITA

OSMOLALITY

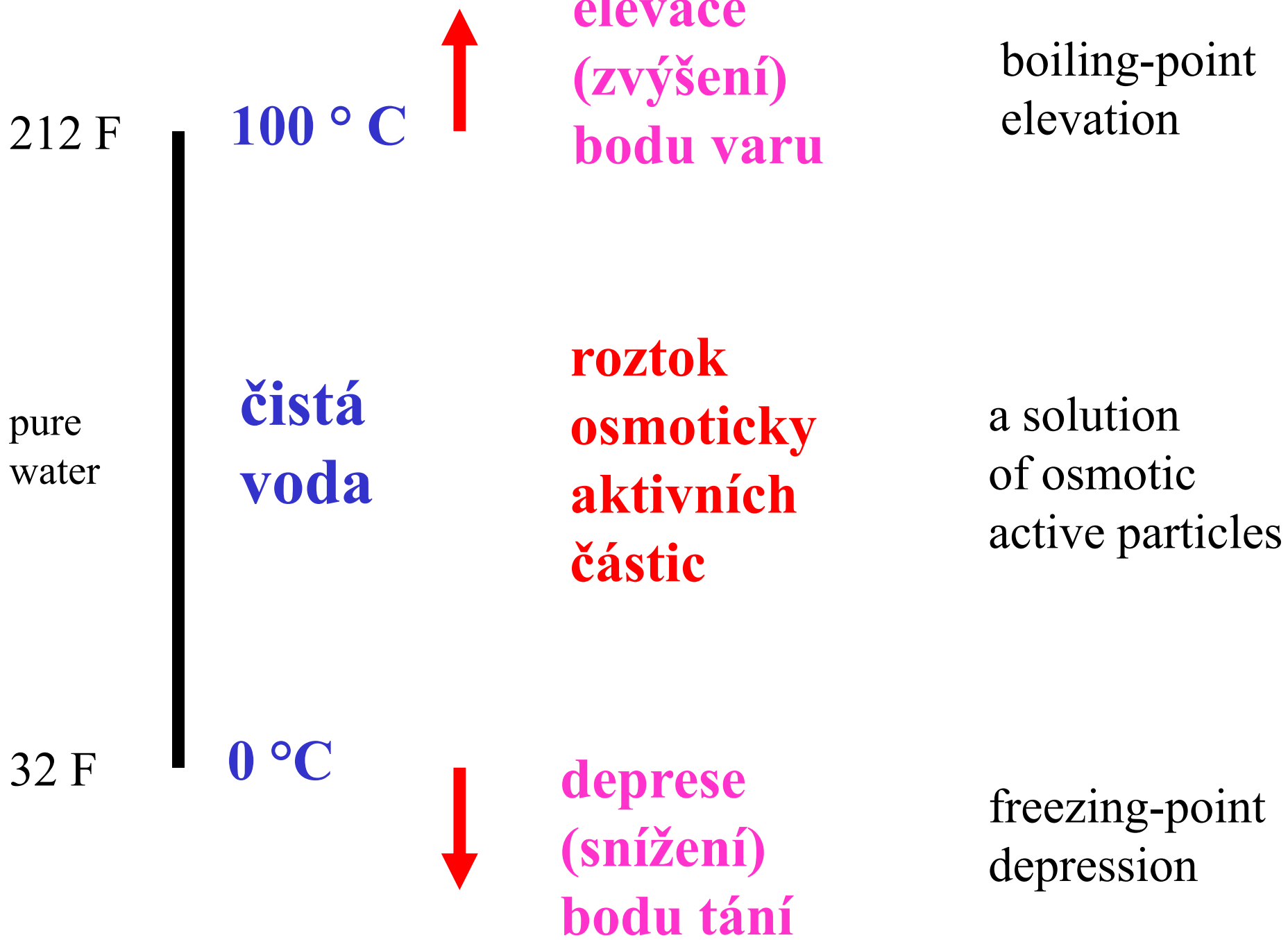
Osmotic pressure and osmotic equilibrium :



osmotic pressure: $\Pi = i \cdot c \cdot R \cdot T$ (mmol / kg)

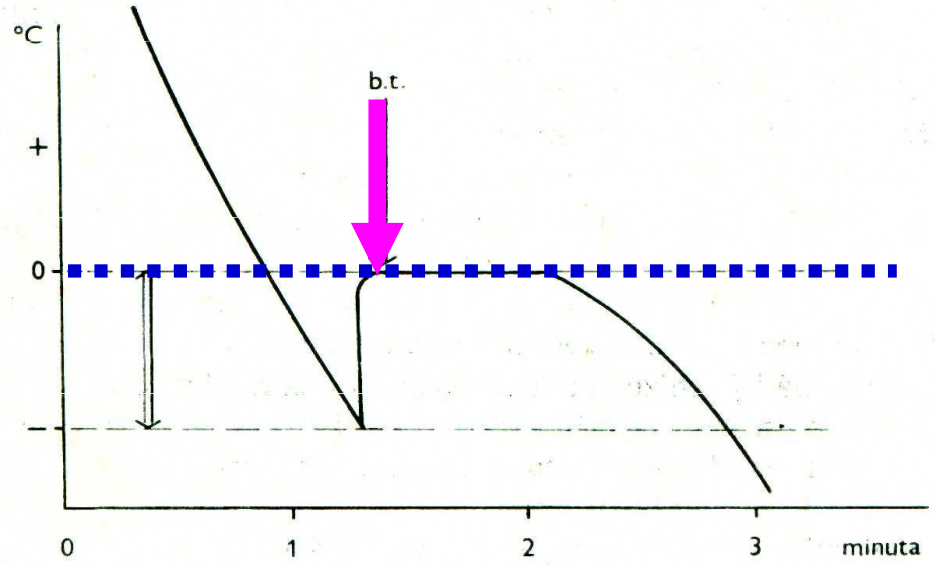
osmotic concentration: $= i \cdot c$ (mmol / L)

c = molarity
i = ionization

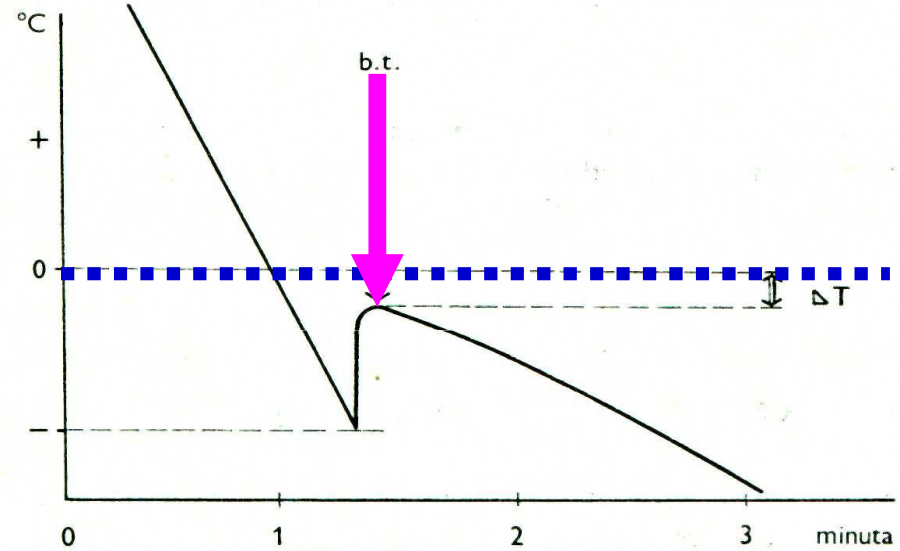

212 F
100 ° C
elevace
(zvýšení)
bodu varu
boiling-point
elevation
pure
water
čistá
voda
roztok
osmoticky
aktivních
částic
a solution
of osmotic
active particles
32 F
0 °C
deprese
(snížení)
bodu tání
freezing-point
depression

Osmometry – cryoscopic principle :

water



solution



thermistor thermometer
~ 0,001 °C

The osmolality of blood plasma :

~ 300 mmol . kg $^{-1}$

~~mosmol . kg $^{-1}$~~

man 290 ± 10 mmol . kg $^{-1}$

woman 285 ± 10 mmol . kg $^{-1}$

The blood plasma osmolality:

~ 300 mmol . kg $^{-1}$

350 mmol . kg $^{-1}$

the critical value (life threatening)

The urine osmolality:

50 – 1.400 mmol . kg $^{-1}$

The correction formula for compensation of water in hypernatraemia :

$$H_2O \text{ (litres)} = \frac{[Na^+] - 140}{140} \bullet TBW$$

60 % of body weight

The blood plasma osmolality:

Na^+, K^+, HCO_3^-, glucose, urea

P-osmolality (mmol . kg $^{-1}$) ≅

≅ $2[Na^+]$ + [glucose] + [urea]

(2 * 140 + 5 + 5 = 290)

U-osm / S-osm :

≅ 2 → normal kidney function (child and adult)

≅ 1 → isostenuria: 1/ effective diuretics
2/ renal insufficiency *)
3/ norm in the newborn

≅ 0,5 → water intoxication

≅ 0,2 → diabetes insipidus

*) insufficiency: renal < 1,2 < extrarenal

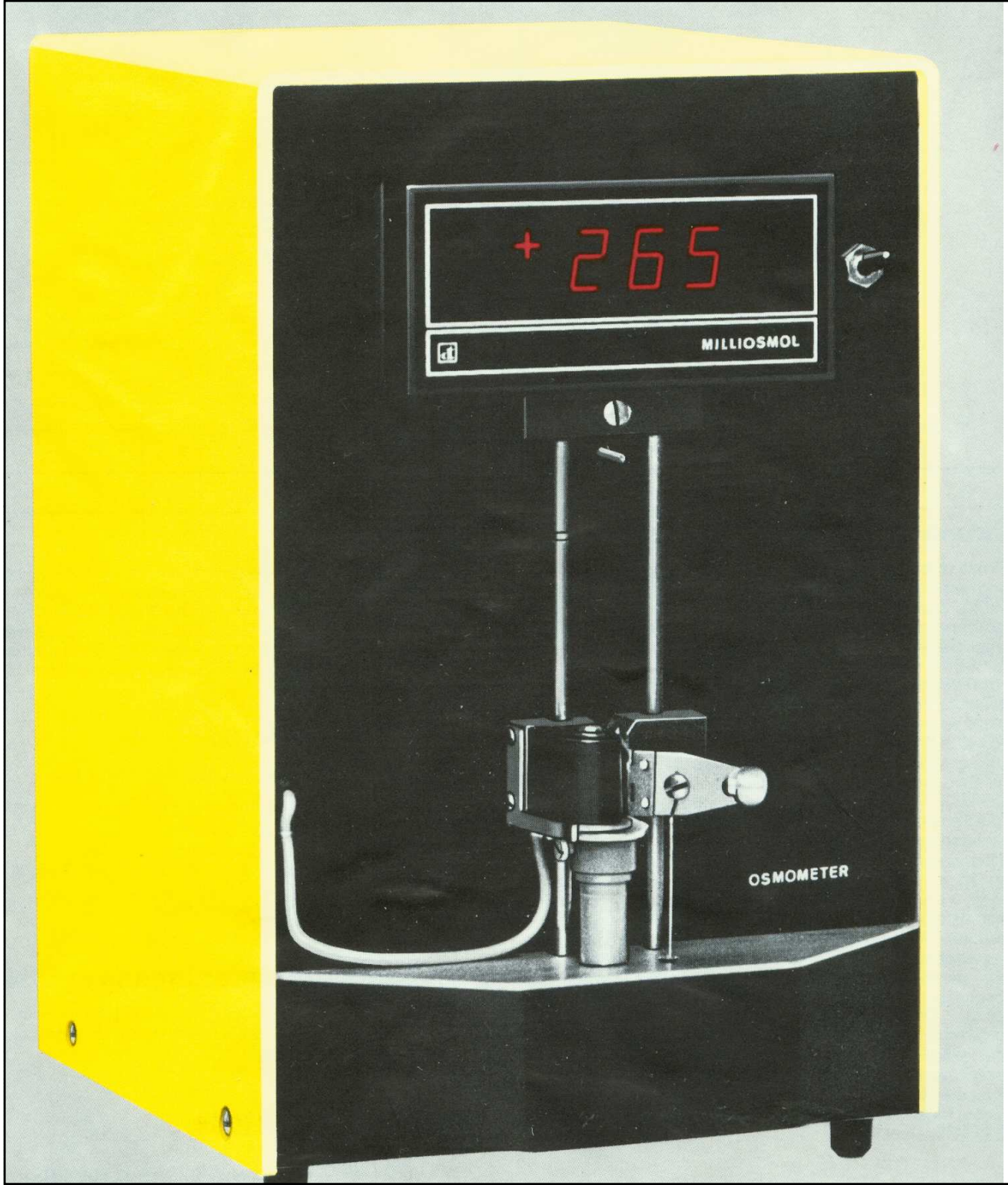
The urine osmolality:

$\sim$ 1.200 mmol . kg $^{-1}$

$\sim$ 500 $\rightarrow$ urea,

Na^+, K^+, NH_4^+

no calculation possible !

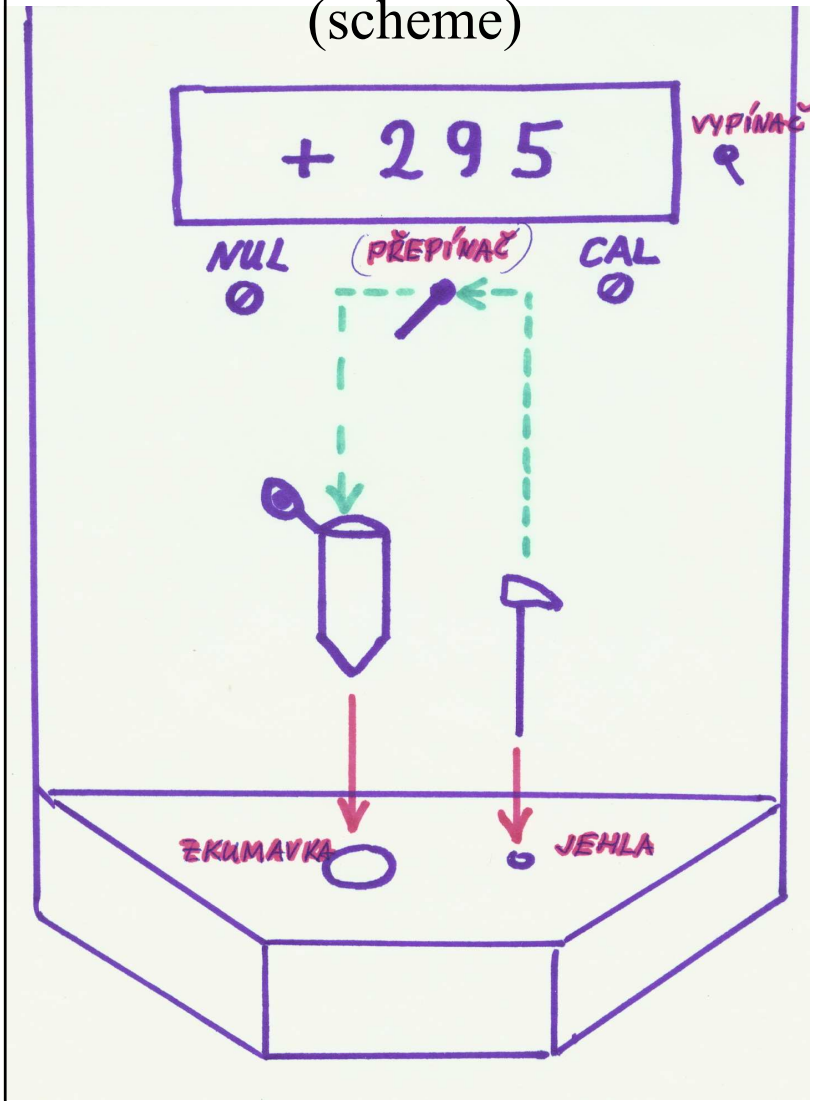


Osmometr

Osmometer

Osmometer (cryoscopic measurement) :

(scheme)



The sample cannot be measured repeatedly – freezing and unfreezing change properties of protein !

$+ 1 \text{ mol} \cdot \text{kg}^{-1} \rightarrow - 1{,}86\ ^{o}\text{C}$

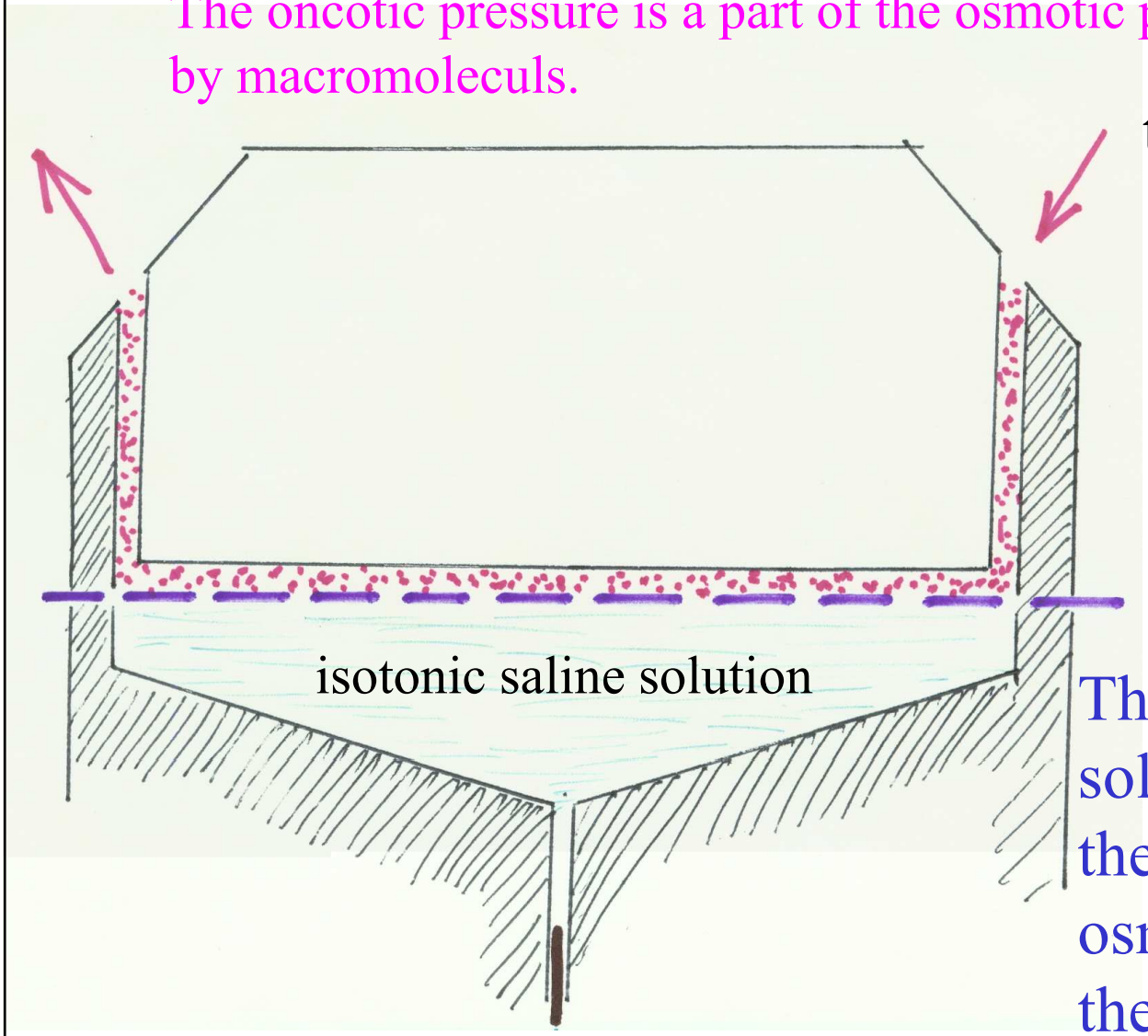
$+ 1 \text{ mmol} \cdot \text{kg}^{-1} \rightarrow - 0{,}001.86\ ^{o}\text{C}$!!

The calibration: 9,485 g NaCl / kg water =
9,485 / 58,443 = 0,161.953 mol NaCl / kg water =
= 161,95 mmol NaCl / kg water
(161,95 * **2** = 323,905 mmol / kg – at completely dissociation
161,95 * **1,86** = 301,227 mmol / kg ≅ **300** mmol / kg water

Onkometr
Oncometer

Oncotic pressure – principle of measurement :

The oncotic pressure is a part of the osmotic pressure of plasma maintained by macromoleculs.



The permeability of saline solution into sample through the membrane is given by osmosis. The sensor measures the pressure decrease of saline solution (due to decrease its volume „under“ membrane)

The oncotic pressure

= colloid osmotic pressure

= „COP“

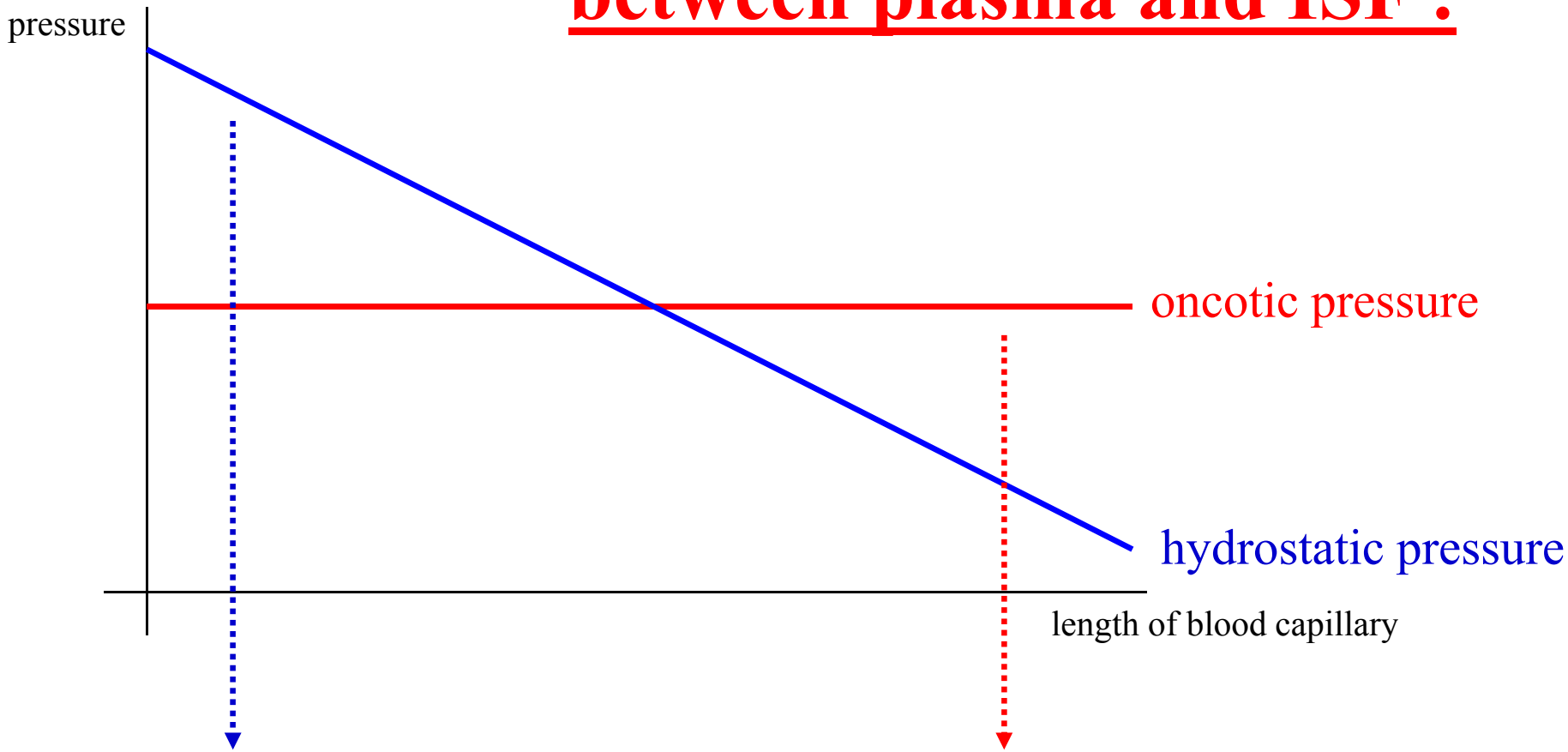
COP = 2,66 – 3,33 kPa (approximately 3 kPa)

COP = 1,33 – 2,66 kPa → danger of edema pulmonum

COP < 1,4 kPa → no survive without i.v. administration of albumin

(80 % COP of plasma ensures albumin)

Capillary – the movement of fluid between plasma and ISF :



arterial end of capillary

hydrostatic pressure exceeds oncotic pressure → filtration of fluid into ISF

venous end of capillary

oncotic pressure exceeds hydrostatic pressure → reabsorption of ISF back to the capillary

The oncotic pressure

- normal concentrations of blood proteins :

P-albumin = 35 – 50 g . l^{-1}

P-total protein = 62 – 82 g . l^{-1}

Compare: grave swelling and ascites in serious hypoproteinemias of the kwashiorkor type !