Disorders of Sodium and Water Metabolism

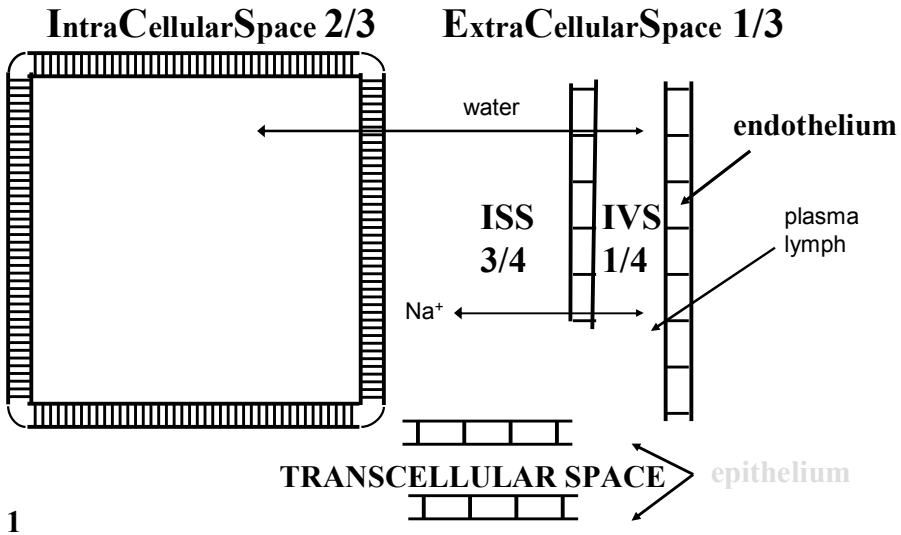
Lecture from pathophysiology

April 14, 2005

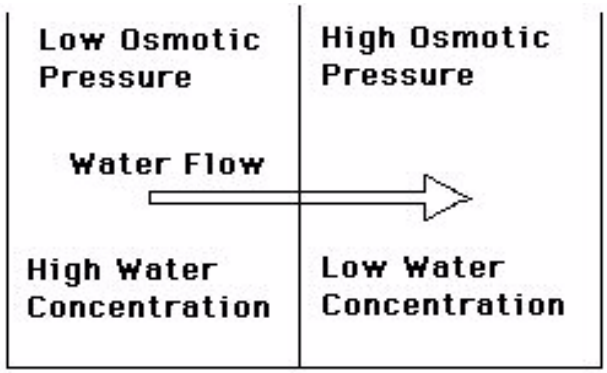
Compartments of body fluids

- Total body water averages about 60% of body weight
- Aproximate volume of body fluids compartments:
- 60% intracellular water
- 40% extracellular water
  - 31%interstitial fluid
  - 7% plasma
  - 2% transcellular fluids (saliva, bile, etc.)

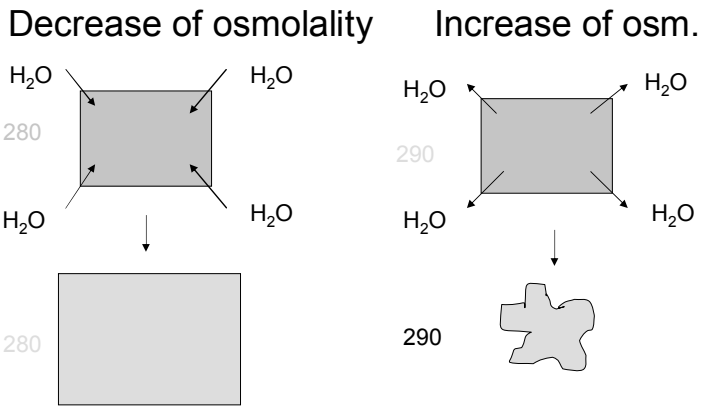
Compartements of body fluids



Osmosis



Change of the cell volume in response to change in extracellular osmolality



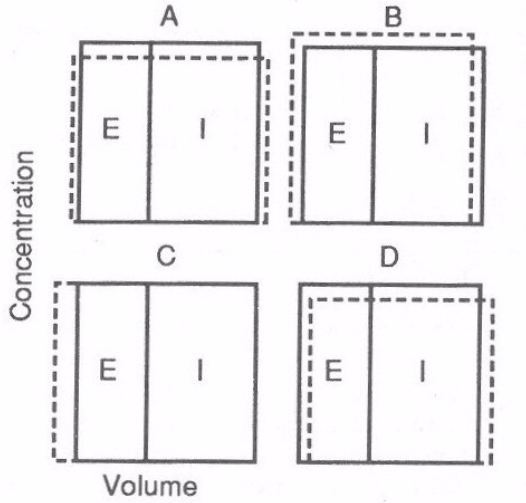
Note: Normal plasma Na concentrations → roughly normal plasma osmolality → normal osmolality of the cells. The electrolyte content in the cells is roughly fixed → normal volume of liquid in the cells (IC space)

A large quantity of water is exchanged between an organisms and the environment via kidneys and a gut → a small percentual derangement has large consequences for the whole-body water and electrolyte balance

Blood plasma

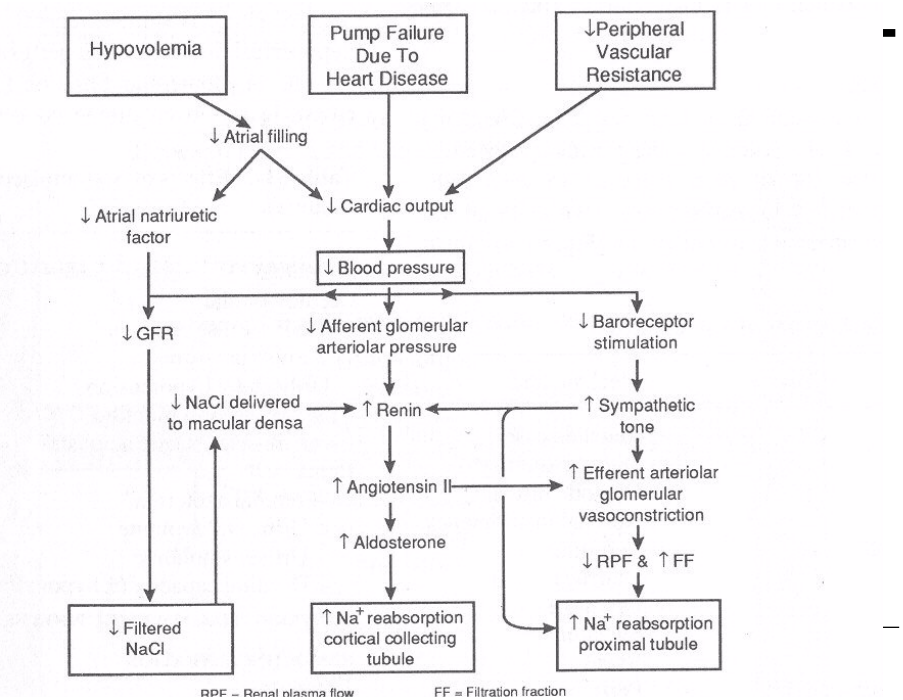
- Osmolality 280-290 mosm/kg
- Osmotic pressure 745kPa
- Onkotic pressure 3,3 kPa
- Na 135-145 mmol/l

Fluid compartment volume and osmolar changes



Normal regulation of sodium balance

- Extracellular fluid volume is controlled by the amount of sodium in the body
- The kidneys regulate the sodium excretion or retention
- The changes in osmolality are detected by hypothalamus changes in ADH secretion water secretion or reabsorption



Normal regulation of water balance

- Extracellular fluid osmolality is controlled by the amount of water in the body
- The kidneys regulate the water excretion

Water intake

- Food
- Metabolic water
- **Drinking is the most important way of water intake regulated by the thirst**

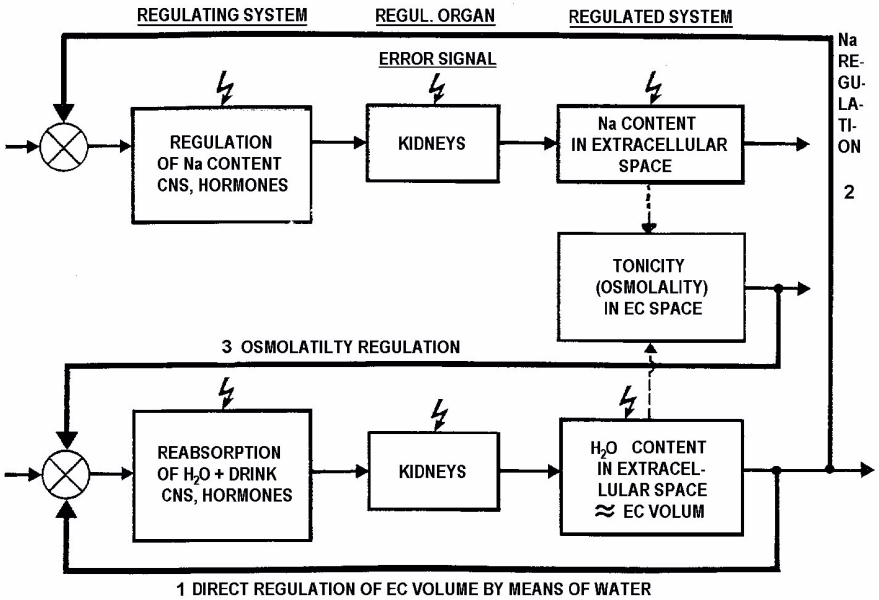
Water excretion

- Skin (perspiratio insensibilis, sweat)
- Respiratory system (perspiratio insensibilis)
- Stool
- **Urine excretion is the most important way of water loss regulation - ADH**

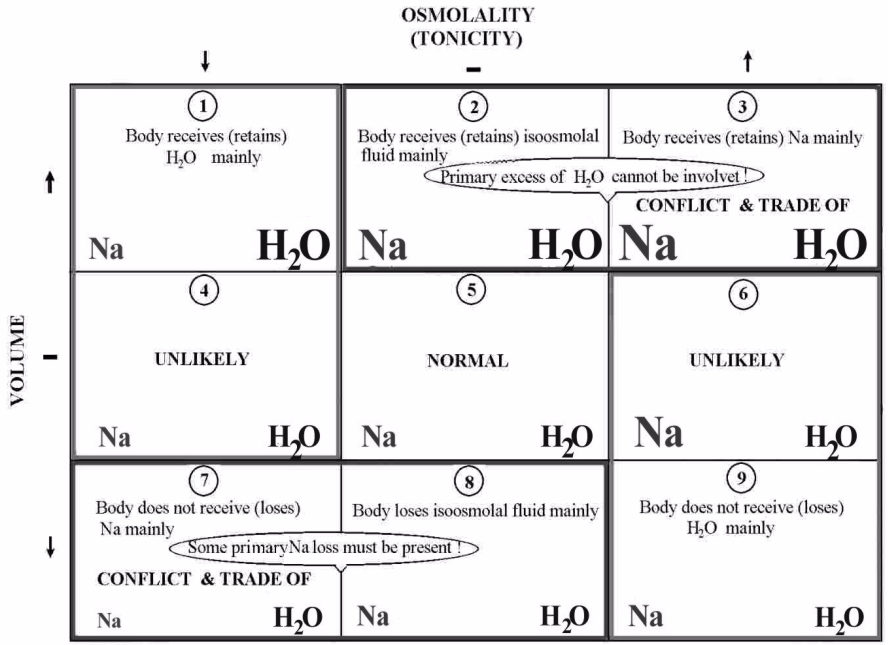
Volume and tonicity regulation

- **Tonicity is ultimately regulated by water, the circulating volume by sodium**
- Tonicity – hypothalamic osmoreceptors neurohypophysis, thirst and ADH renal water reabsorption
- Volume – baroreceptors, more sluggish feedback than osmoreceptors, under extreme conditions:

  Volume overrides tonicity

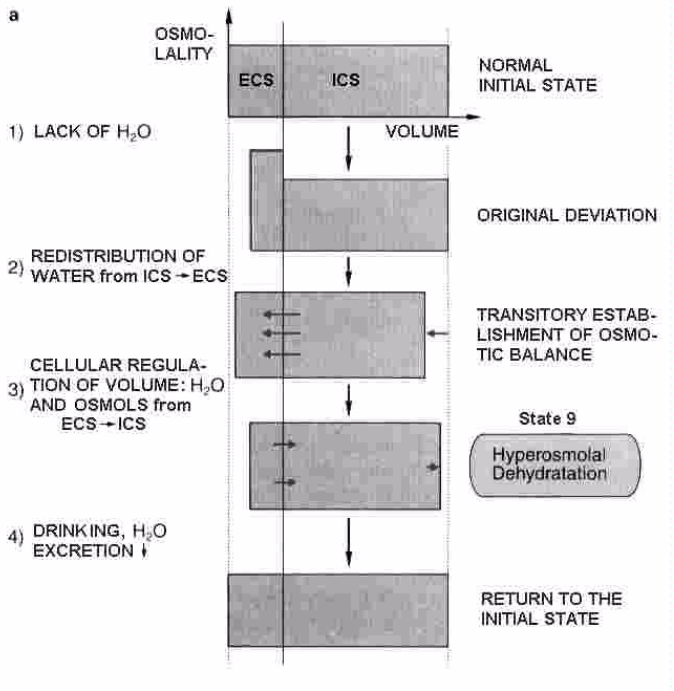


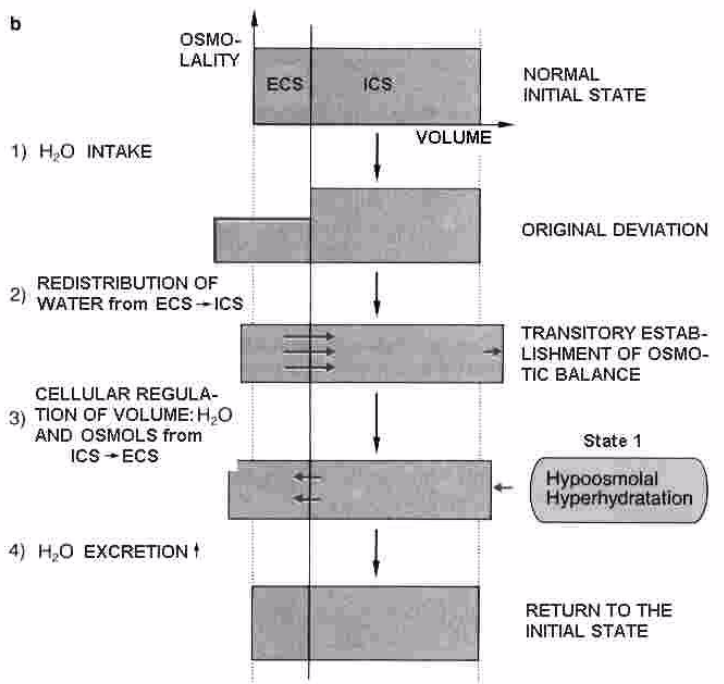
Regarding adiuretine and thirst regulation: osmoreception (feedback No. 3) is functioning more sensitively, volumoreception (feedback No. 1) more sluggish, later more forcefully, however → "volume overrides tonicity" when the large deviations of volume and tonicity from a norm take place. It is a consequence of the type of dependency of the ADH production on both these factors. A circulatory failure is apparently evaluated to be more dangerous acutely than the CNS disturbances.

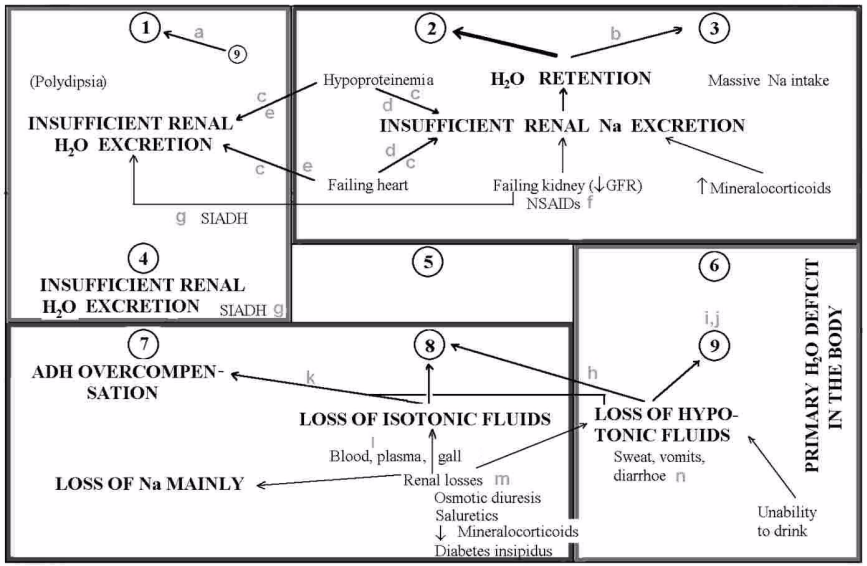


Tonicity disorders ⇔ disorders of water: states 1, 4, 6, 9

Volume disorders ⇔ sodium disorders: states 2, 3, 8, 7







Explanatory notes

a – overshooting compensation of hyperosmolality (state 9) by water
b – a trade off by means of ADH: hypervolemia does not rise so much with a considerable Na_{EC} enhancement that isoosmolality could be maintained
c – loss of effective blood volume
d – three factors of Na retention (GFR, aldosterone, 3rd factor)
e – by means of ADH
f – nonsteroid antiphlogistics (acetylosalicylic acid, sodium salicylate, phenacetin, paracetamol) depress the protective prostaglandins in the kidney → decline of GFR
g – SIADH is euvolemic clinically, hypervolemic subclinically
h – by means of thirst and ADH, some loss of salt is presupposed, however

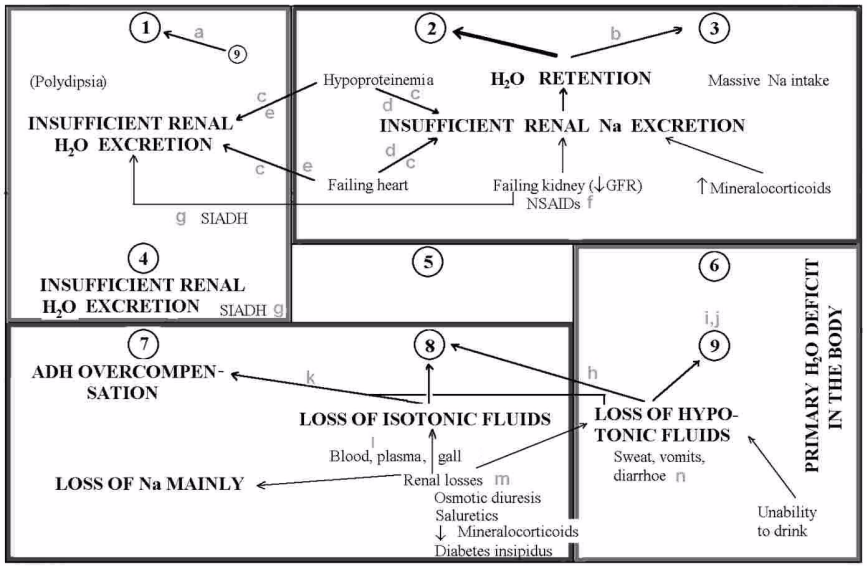
i – although body dehydration may be considerable with the loss of hypotonic fluids, loss of circulating volume used to be negligible in this condition (loss of water is compensated in 90% from stores outside the circulating volume)
j – if the water loss is much higher than loss of salt, Na_{EC} lowering may be attended by P_{Na} rise
k – an organismus has lost salt and water massively, it tries, however, to maintain predominantly the volume by the quick feedback by means of thirst and ADH in this extreme situation (salt losses are compensated only by drinking); it succeeds only partially, however, and it is paid by hypotonicity (a trade-off again);
l – Na in urine < 10mmol/L
m – Na in urine > 20 mmol/L – the urine itself is effective in the Na loss
n – with a small urine volume Na in urine > 600 mmol/L

CONDITION 3 Na

The body receives (retains) Na mainly -
hyperosmolal hyperhydratation

RdS: *massive Na intake (per os, sea water)*
RgS: *primary surplus of mineralokorticoids*
RgO: *acute glomerular diseases*
billateral parenchymatous
renal diseases with chronic
renal failure (GFR < 10mL/min)

Fig. 10 – hyperosmolal hyperhydration (state 3)
Renal failure with the GFR value higher than 10 mL/min is not connected with a deranged G-T balance → under the lowered GFR, reabsorption is lowered, too. G-T balance is disturbed in acure nephritic syndrome, however



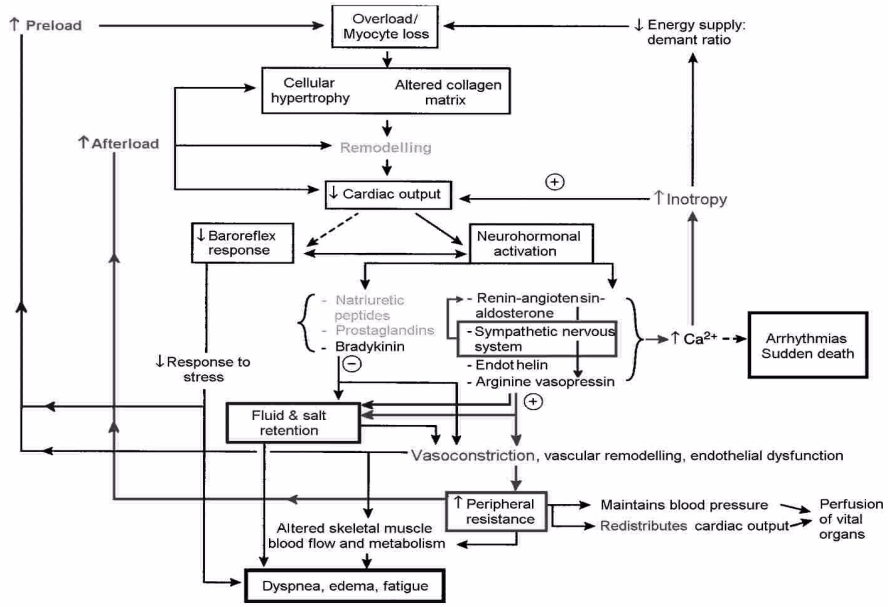
CONDITION 2 Na

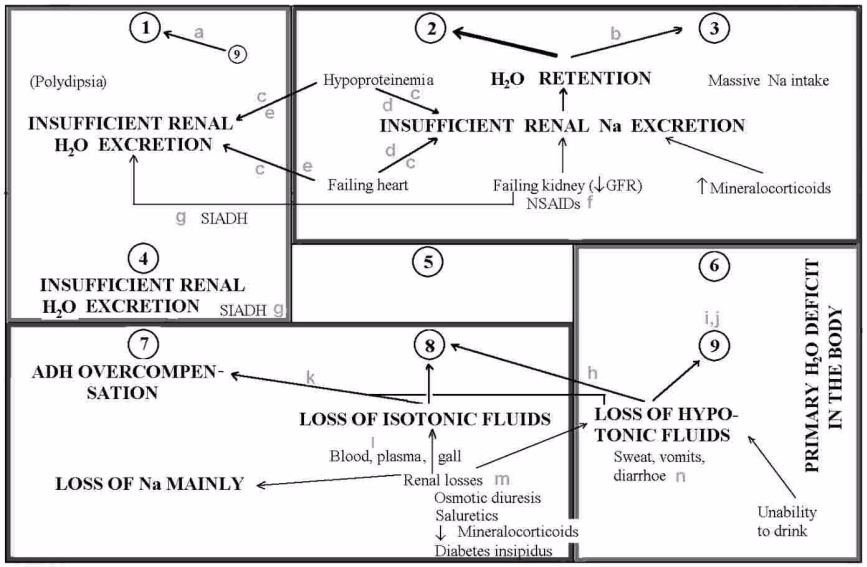
Body receives (retains) isoosmolal fluid mainly - **isoosmolal hyperhydratation**

RdS: *i.v. infusion of isoosmolal fluids*
nephrotic syndrome
cirrhosis
RgS: *cardiac failure*
RgO: *non-steroid antiphlogistics*
failing kidney (↓GFR!)
acute & chronic, esp. when isoosmotic solutions are administered

Fig. 11 – isoosmolal hyperhydration (state 2)

Heart failure: a decline of effective blood volume is signalized, RASand SAS are activated (Fig. 11), ↓GFR, "3rd factor"





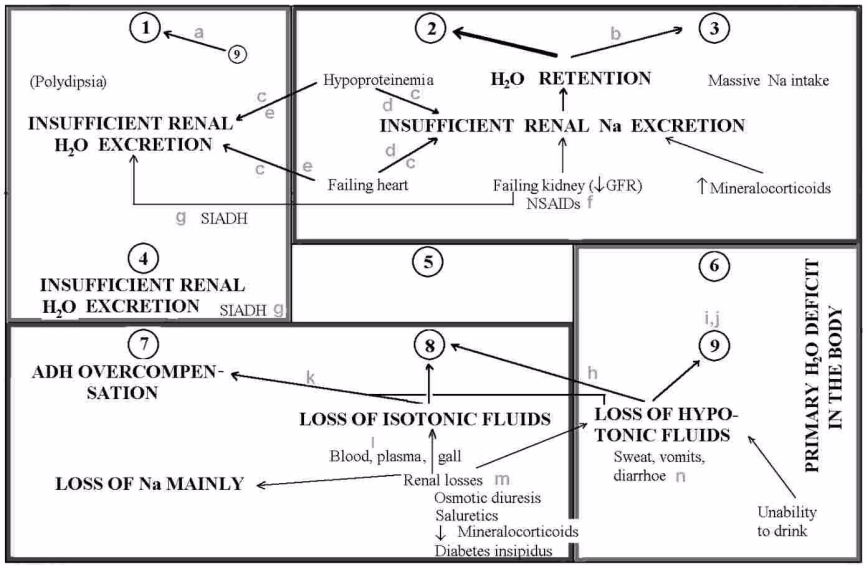
CONDITION 1 Na

The body receives (retains) H_2O mainly -
hypoosmolal hyperhydratation

RD: *infusion of glucose solutions, nephrotic syndrome cirrhosis*

RS: *psychogenic polydipsia*
renal oligo/anuria when ↑tubular H_2O reabsorption with SIADH, chlorpropamid
cardiac failure

RO: *renal oligo/anuria*
↓GFR
esp. in combination with H_2O or glucose solution administration



Consequences of hypervolemia:

Hypervolemia → enhanced left ventricle preload → enhanced cardiac output

↑cardiac output * unchanged peripheral resistance = ↑arterial pressure

↑arterial pressure → ↑hydrostatic capillary pressure → ↑filtration into the IC space → edema

CONDITION 9 Na

The body does not receive (loses) H_2O mainly -

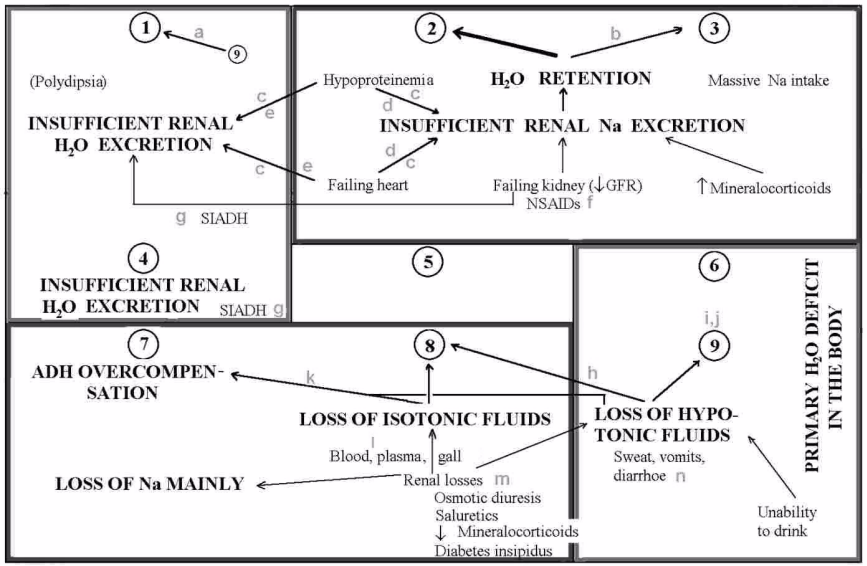
hyperosmolal dehydratation

RdS: *vomiting*
diarrhoe
sweating
insesible losses
hyperventilation, fever, hot environment
hyperglycemia in diabetes mellitus
mannitol

RgS: ↓ *thirst*
unconsciousness
newborns
diabetes insipidus (central)

RgO: *osmotic diuresis in diabetes mellitus*
diabetes insipidus (nephrogenic)
polyuria in acute renal failure

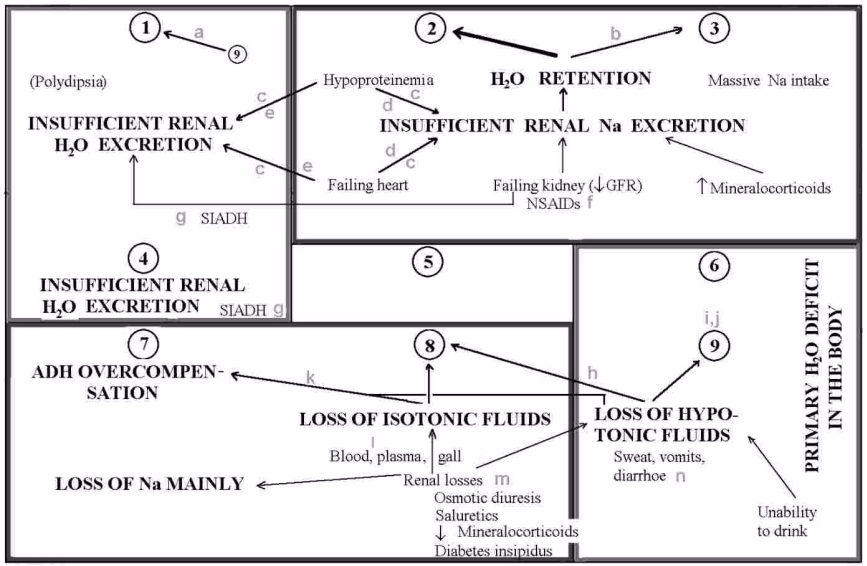
If the water supply is not disturbed and Na is normal, state 9 cannot last long



CONDITION 8 Na

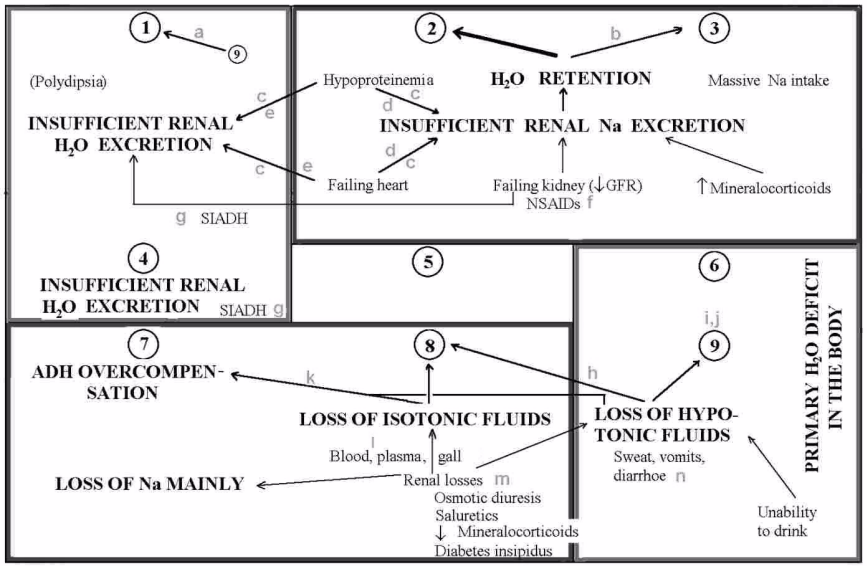
Body loses isoosmolal fluid -
isoosmolal dehydratation

RD: *loss of blood or plasma*
burns, ascites draining
diarrhoe, gall drains, fistulas
escape into interstitium or 3rd space
crushing of tissues, intestinal obstruction, pancreatitis
hemorrhage into body cavities
RO: *abusus of saluretics*
and many other renal loss types



CONDITION 7 Na

Body does not receive (loses) Na mainly -
hypoosmolal dehydratation
RD: *alimentary lack of salt in combination with loses*
RS: *primary lack of mineralocorticoids*
RO: *renal salt losses:*
polyuria in acute renal failure
loss of hypotonic fluids → trade off preferring volume
pressure diuresis in extemely enhanced blood pressure
BARTTER syndrome
abusus of diuretics



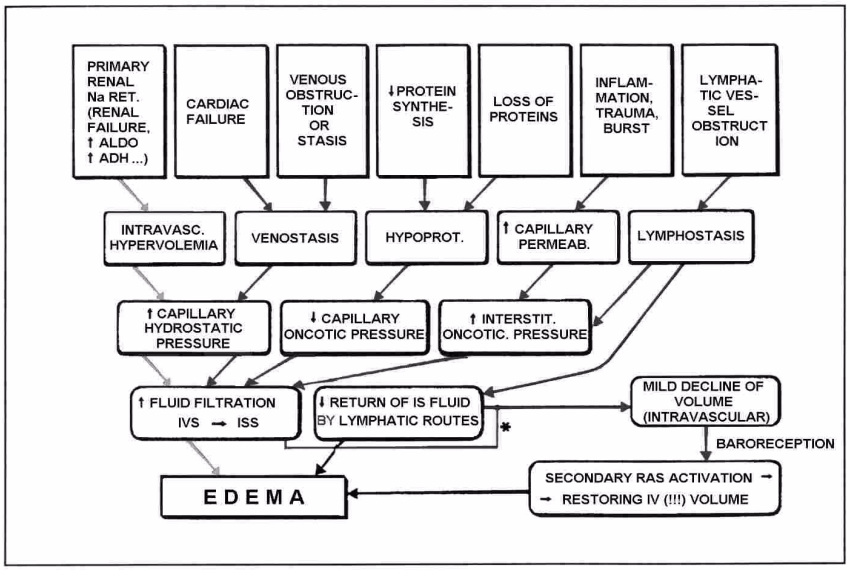
A survey of the influence of renal pathology on volume and osmolality
Fig. 17

Na AND H_2O EXCRETION IN VARIOUS PATHOLOGIC RENAL CONDITIONS

CONDITION	Na	H_2O
ACUTE GLOMERULAR DISEASES	**RETENTION**	**RETENTION**
STENOSIS OF ART. RENALIS CONSIDERABLY ENHANCED BP PRESSURE DIURESIS	**RETENTION** **↑EXCRETION**	**RETENTION** **↑EXCRETION**
PRERENAL AZOTEMIA	**RETENTION**	**RETENTION**
	AIMED AT CORRECTING BP OR VOLUME	

CONDITIOON	Na	H_2O
ACUTE RENAL FAILURE INITIAL PHASE (ANURIA, OLIGURIA) PREREN. AZOTEMIA MOST OFTEN RESTITUTION PHASE (POLYURIC) - SALT WASTING KIDNEY	**RETENTION** **↑EXCRETION**	**RETENTION** **↑EXCRETION**
CHRONIC RENAL FAILURE (TO THE ADVANCED PHASE) GFR < 10 - 20 mL/min	**WITHOUT DISTURBANCES** **RETENTION**	**WITHOUT DISTURBANCES** **RETENTION**
TUBULOINTERSTITIAL DISEASES, ADRENAL INSUFICIENCY, DIURETICS, „WASTING SALT“ NEPHROPATHY (i.g. CHRF)	**↑EXCRETION**	**↑EXCRETION**

2.2 Edematous conditions



* **with the exception of primary renal retention**

With the exception of the “primary” hypervolemia conditioned by **primary renal Na retention, RAS is activated secondarily** (possibly **secondary hyperaldosteronismus** may be elicited) → Na retention → edema

Not in Fig. : Cardiac failure → distortion of baroreception → RAS, SAS, 3rd factor activation, ↓GFR