In the name of Allah
the compassionate , the merciful

Perioperative Fluid & Electrolyte Therapy

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Ass. Prof. of anesthesia ,
critical care consultant

Miller's anesth.,2015
Anesth.& coexisting dis.,2018

Anesthesia Grand Round , SUMS
Feb. 12 , 2019

50 little bags of chips= 1 liter 0.9% NaCl



=



Objectives:

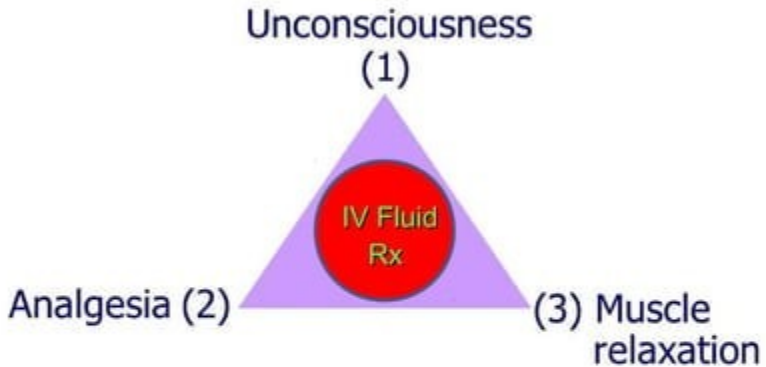
- ✓ Fluid compartments
- ✓ Osmotic/oncotic pressure / osmolality
- ✓ Fluid pharmacology
- ✓ Electrolytes' physiology
- ✓ Electrolytes' derangements
- ✓ Clinical fluid and Electrolyte management



We are approximately two-thirds water

Traditional triad

The triad of balanced general anaesthesia



Aims of periop. fluid :

1. To avoid dehydration
2. To maintain an effective circulating vol.
3. To prevent inadequate tissue perfusion



- Clinical decision about fluid Rx. :
  1- which fluid to use
  2- How much fluid to give

  - Recently, clinical studies have changed our concepts regarding both of these questions

- Despite "mega-trials" ; key questions remain unresolved



Anesthesiology 2009; 110:453-5
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Goal-directed Perioperative Fluid Management

Why, When, and How?

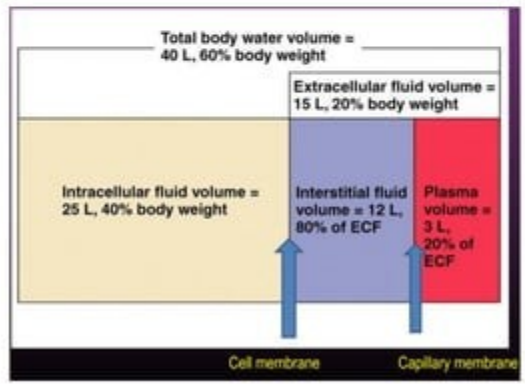
Intravenous fluid therapy



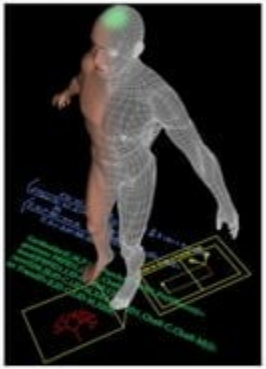
- A core of periop. practice; influences pt. outcomes
- Water ~ 60% of total body wt, vary with age & body composition
- Intracellular/functional extracellular water ~ 2:1
- During anesthesia, many of homeostatic mechanisms are disturbed

TABLE 59-1 **AGE-RELATED VARIATION IN TOTAL BODY WATER AND EXTRACELLULAR FLUID AS PERCENT OF BODY WEIGHT (MULTIPLY BY 10 FOR mL/kg)**

Age	TBW (%)	ECF (%)	Blood Volume (%)
Neonate	80	45	9
6 mo	70	35	
1 yr	60	28	
5 yr	65	25	8
Young adult (male)	60	22	7
Young adult (female)	50	20	7
Elderly	50	20	

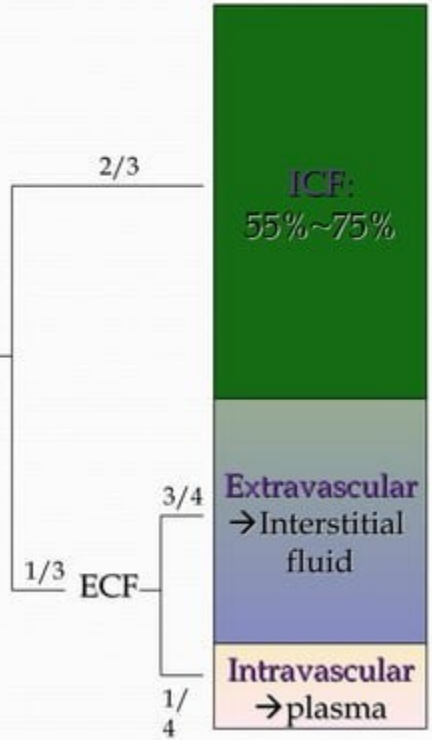


Body Fluid Compartments



X 50~70%
lean body weight

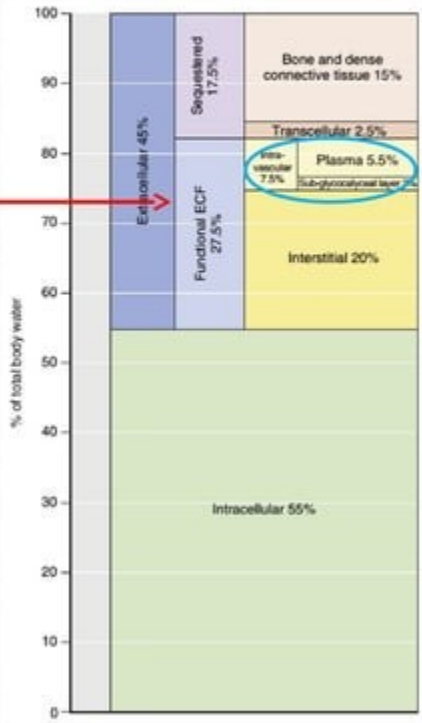




- Male (60%) > female (50%)
- Most concentrated in skeletal muscle
- TBW=0.6xBW
- ICF=0.4xBW
- ECF=0.2xBW

Total-body water (intracellular and extracellular)

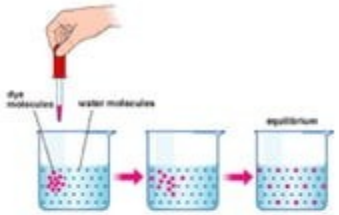
- Extracellular fluid (functional ***v/s*** sequestered)
- Functional :
  - A. Interstitial fluid (ISF):
    1. Lymphatic fluid
    2. protein poor fluid occupying cell spaces
  - B. Intravascular fluid: Plasma volume, including subglycocalyx
- Sequestered:
  - Transcellular fluid: GI tract fluid, bile, urine, csf , aqueous humor, joint fluid, pleural , peritoneal & pericardial fluid (functionally important , varying composition & regulated by active cellular transport)
  - A. Water in bone and dense connective tissue



Physicochemical Laws Governing

Fluid & Electrolyte Movement

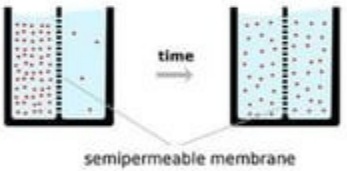
Diffusion :



- Particles **move from high to low** *concentration*

- Diffusion **cross permeable membranes** (**Fick's law**):

$$J = -DA\left(\frac{\Delta c}{\Delta x}\right)$$



J : rate of diffusion

D : diffusion coeffiient

A : cross-sectional area available for diffusion

Δc : concentration (chemical) gradient

Δx : *membrane thickness*

- Diffusion also driven by *tendency of charged solutes* to move

Physicochemical Laws Governing
Fluid & Electrolyte Movement

Osmosis

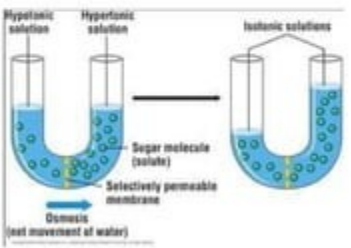
- Water cross a semipermeable mem. toward higher solute concent.

> Hydrostatic pressure required to resist movement of solvent molecules is osmotic pressure

- Depends on **number rather than type** of osmotically active particles

P : osmotic pressure
n : number of particles
R : gas constant
T : Temperature
V : volume

$$P = \frac{nRT}{V}$$



- Total plasma osmotic pressure ≈ 5545 mm Hg

- Body fluids are **not** ideal solutions (interionic interactions → ↓free particles)

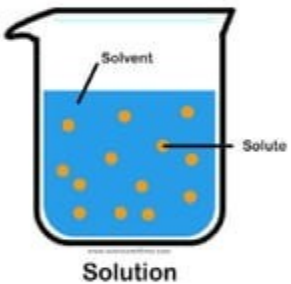
Physicochemical Laws Governing

Fluid & Electrolyte Movement

Osmolality (Chemistry)

- ✓ Number of moles (each containing 6 ×10 (23) particles) **in 1 kg** of solvent
- ✓ Nl ~ 285 to 290 mOsm/kg (ICF = ECF)
- ✓ The largest contributor : Na , Cl & bicarb.

$$\text{Serum osmolality} = \left(\begin{array}{l} (2 \times Na) + (glucose \div 18) \\ + (urea \div 2.8) \end{array} \right)$$



- Na : serum sodium (mEq/L)
  (2 × Na) : both Na and its associated anions (Cl− & HCO3−)
  glucose : serum glucose (mg/dL)
  Urea : blood urea nitrogen (mg/dL)

Osmolarity (Physiology)

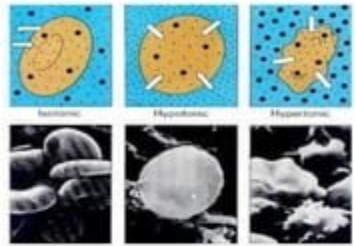
- ✓ Number of osmoles **per liter** of solution
- ✓ Unlike osmolality, this may be affected by **temperature** changes

Physicochemical Laws Governing

Fluid & Electrolyte Movement

Tonicity = *Effective osmolality*

(always in comparison to a cell)



- *Tonicity* is the *effective osmolality* of a solution through a semipermeable membrane

  e.g.:

  - Na+ and Cl− do not cross cell membranes freely → Effective osmotic force
  - Urea freely diffuses across cell membranes → Ineffective osmole
  - Glucose (taken into cells by insulin) → Ineffective osmole

The osmolarity of a solution is not an accurate predictor of its tonicity.

	OSMOLARITY		
TONICITY	Hyposmotic	Isosmotic	Hyperosmotic
Hypotonic	√	√	√
Isotonic		√	√
Hypertonic			√

- Determines in vivo distribution of fluids across a cell membrane
- Sensed by hypothalamic osmoreceptors
- Estimated by subtracting urea and glucose from osmolality

Physicochemical Laws Governing

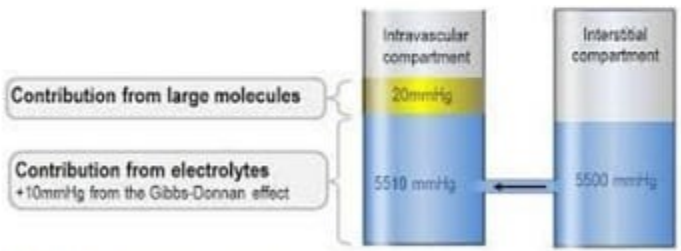
Fluid & Electrolyte Movement

Oncotic pressure (colloid osmotic pressure)

- Is an *osmotic pressure due to colloids* , mostly proteins (alb., globulin, fibrinogen)

- Of total plasma osmotic pressure of 5545 mm Hg,
  25 to 28 mm Hg is due to plasma oncotic pressure

- **Negative charge on proteins** retaining Na+ ions within plasma
  (the **Gibbs-Donnan effect**)



- Albumin is responsible for 65% to 75% of plasma oncotic pressure

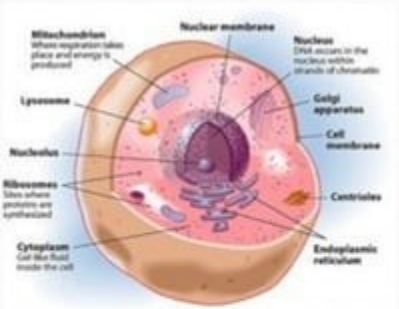
Fluid Compartment Barriers & Distribution

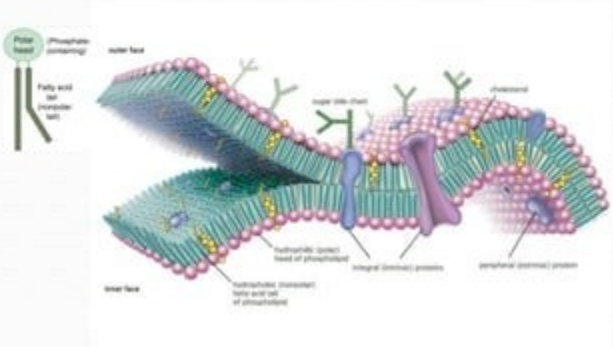
Cell membrane

A lipid bilayer membrane

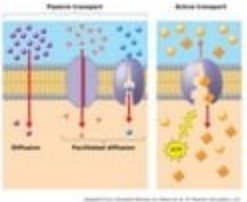
Separates intracellular & extracellular compartments

Impermeable to large hydrophilic molecules & charged particles





Fluid Compartment ; Barriers & Distribution



Cell membrane (cont'd)

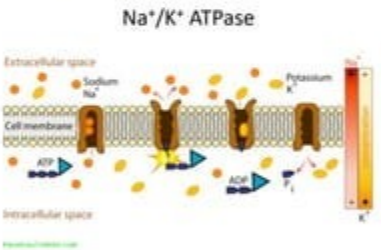
Solutes may cross cell membranes in several ways:

1- Passive diffusion (certain molecules)

2- Primary Active Transport

Transport against concent. gradient by ATPases (e.g.: Na+/K+ ATPases)

- ✓ ionic gradients are maintained
- ✓ water & solute mov.
- ✓ electrical impulse transmission in excitable tissues

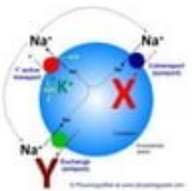


3- Secondary Active Transport

Uses concentration gradients set up by ATPases

(**cotransport** **vs** **countertransport**)

- Na^+-Ca^{2+} counter-transport on all cell membranes
- Na^+-H^+ counter-transport on proximal tubules of the kidneys $\rightarrow H^+$ homeostasis
- Na^+-Glucose co-transport mechanism

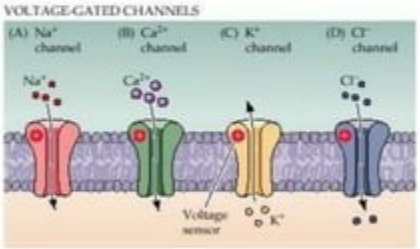


Cell membrane (cont'd)

4- Solute Channels

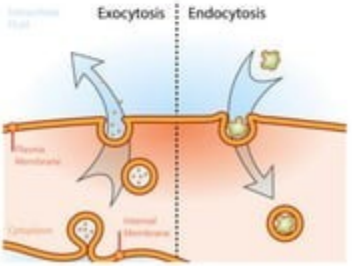
much faster than ATPases or transmembrane diffusion

(e.g.: **voltage-gated Na+ channels** & glucose transporter GLUT1)



5- Endocytosis and Exocytosis

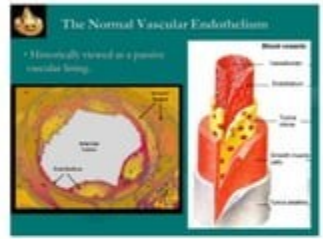
Transport of large proteins and polypeptides



Fluid Compartment ; Barriers & Distribution

Vascular endothelium

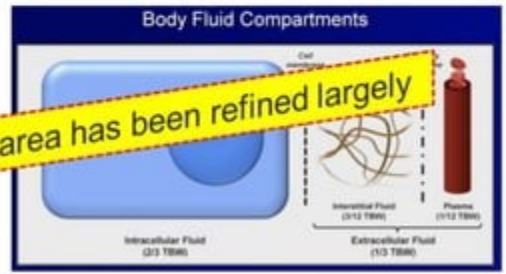
- The barrier function of the vascular endothelium → key role in periop. fluid Rx.



- Surgical tissue trauma →
  1. Loss of intravas. Vol. (blood loss)
  2. Inflammation-related shifts to other tissues

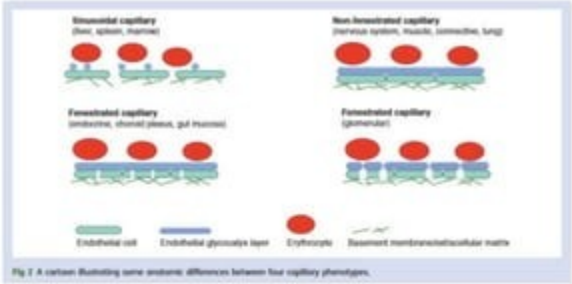
- The physiologic effect of IVF depends on fluid handling at capillary level





Our understanding of this area has been refined largely

Fluid Compartment Barriers and Distribution
Vascular epithelium



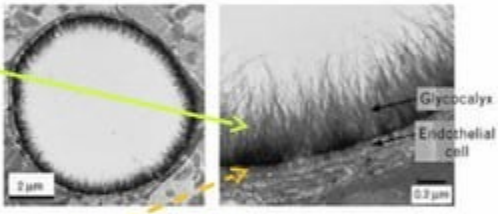
capillary structure

- Varies depending on underlying organ function

The most common type is , nonfenestrated

- Interior of endothelial cells is covered by a continuous network ;

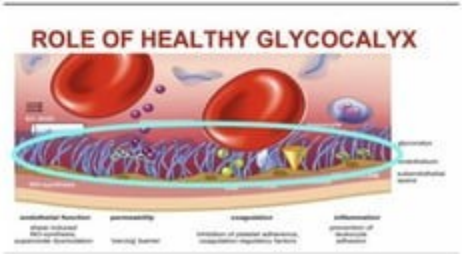
endothelial glycocalyx layer (EGL)



- ✓ covers fenestrations & intercellular clefts
- ✓ Thickness ≈ 1 µm
- ✓ Preventing plt. & leukocyte adhesion

- ❑ A semipermeable layer as endothelial barrier function

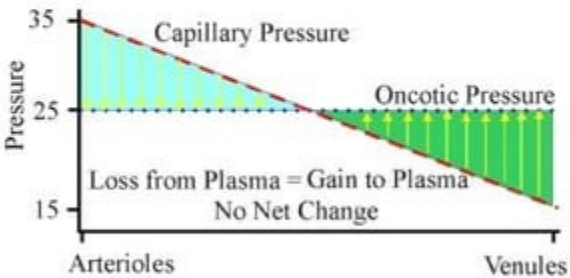
- Water & electrolytes can move freely across vas. endoth. through EGL & then intercellular clefts



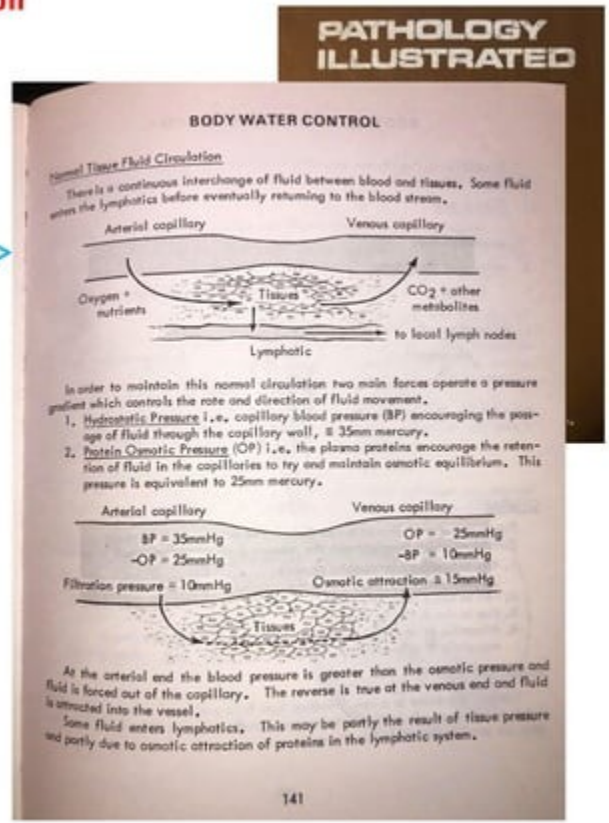
Fluid Compartment Barriers and Distribution
Vascular epithelium

capillary Function

- Movement of fluid across capillary mem ;
  Starling law



- Water not reabsorbed by capillary is removed from ISF by lymphatics



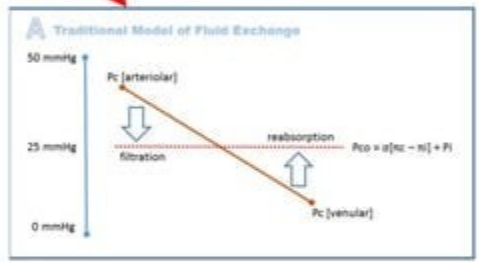
Fluid Compartment Barriers and Distribution
Vascular epithelium

REVISED

Capillary function

Revised Starling equation = Role of glycocalyx + Starling law

$$J_v = K_f([P_c - P_i] - \sigma[\pi_c - \pi_{sg}])$$



Jv : transcapillary flow

Kf : filtration coefficient

Pc : capillary hydrostatic pressure

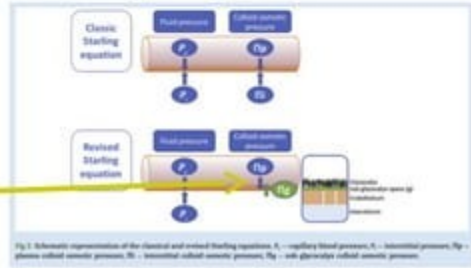
Pi : interstitial hydrostatic pressure

σ : reflection coefficient

(degree to which the tendency of a macromolecule to cross the endothelial barrier is resisted)

πc : capillary oncotic pressure

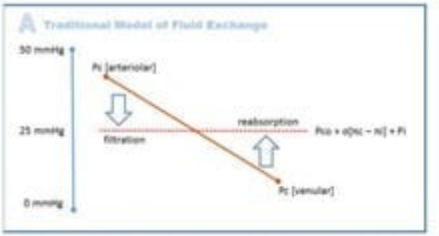
πsg : subglycocalyx oncotic pressure

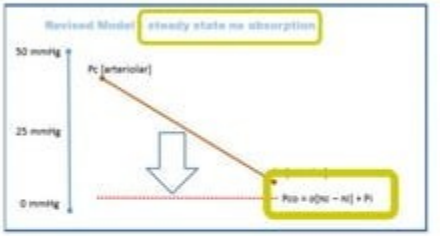


Clinical relevance



Fluid Compartment Barriers and Distribution
Vascular epithelium
Capillary function





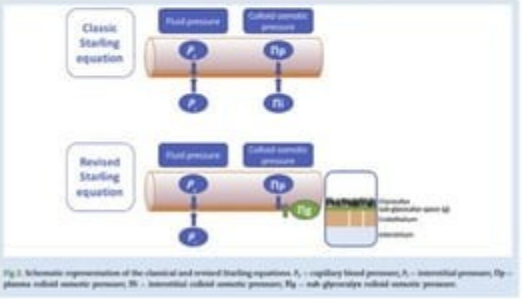
Clinical relevance :

- At steady state, continuous capillaries *do not* exhibit fluid reabsorption toward venous end (**"no-absorption" rule**)

➢Recent Measurement of all Starling forces showed→ capillary filtration (Jv) is much less than predicted (**opposing filtration**)

$$Pco = \sigma[\pi c - \pi i] + Pi$$

➢filtrate is returned to circulation by lymphatics

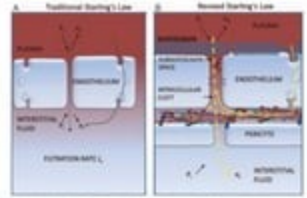


- Plasma-SGL COP difference, not plasma-ISF COP difference affects Jv.

➢If artificially raising COP (e.g.,albumin infusion) may reduce Jv but ***not*** reabsorption of fluid from ISF into plasma

$$Jv = [Pc - Pi] - \sigma[\pi c - \pi sg]$$

Fluid Compartment Barriers and Distribution
Vascular epithelium
Capillary function

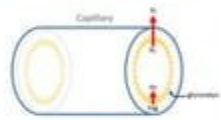


Clinical relevance

- An **exception** to no-absorption rule :

➢ in acutely **subnormal capillary pressures**; a transient **autotransfusion** occur (≈ **500 mL**)

- ✓ If subnl pressures persist , Jv approach zero **but** reabsorption does not occur
1. **Infusion of colloid** will expand plasma volume
2. **Infusion of crystalloid** will expand total intravas. vol (plasma and EGL)



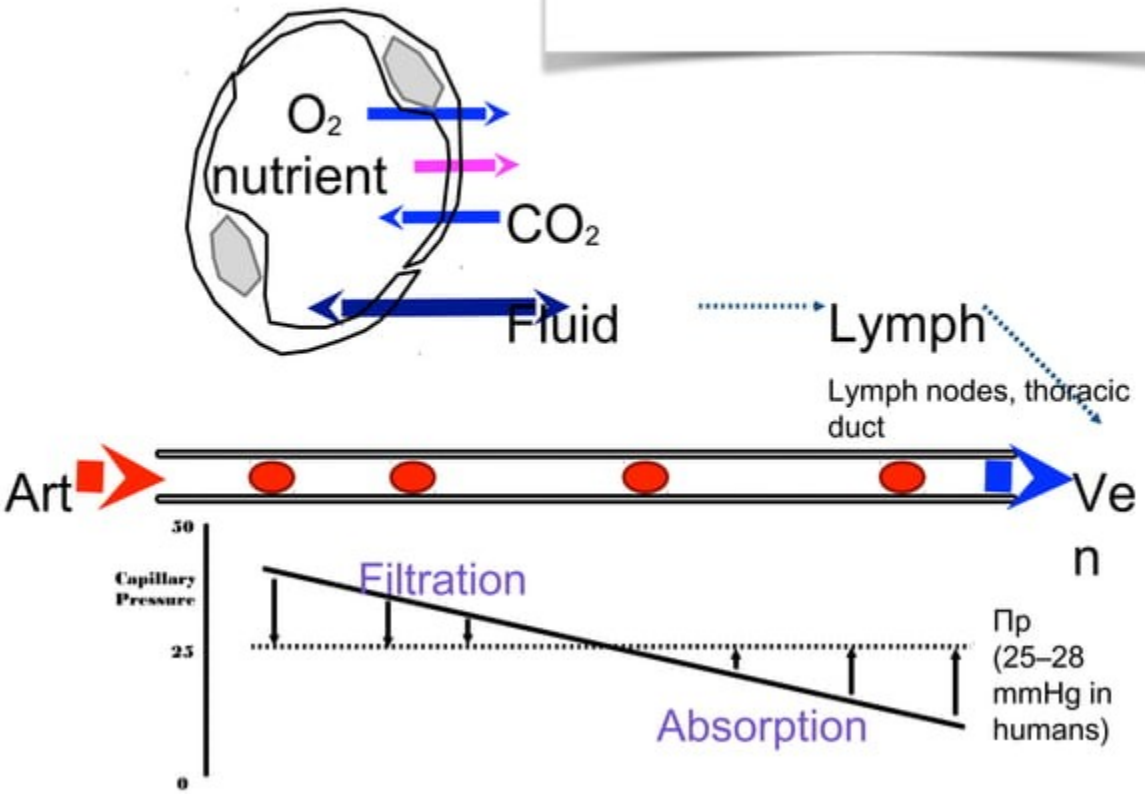
Jv remain zero in both cases until
capillary pressure rises to nl or supranl levels

➢ At **supranormal capillary pressures** :

↑Jv proportional to hydrostatic pressure

- Colloid infusion **will** maintain plasma COP but raise capillary pressure further → **↑Jv**
- Crystalloid infusion will ↑ **capillary pressure** + **↓COP** → **↑↑ Jv**


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AND
LET'S
REVIEW
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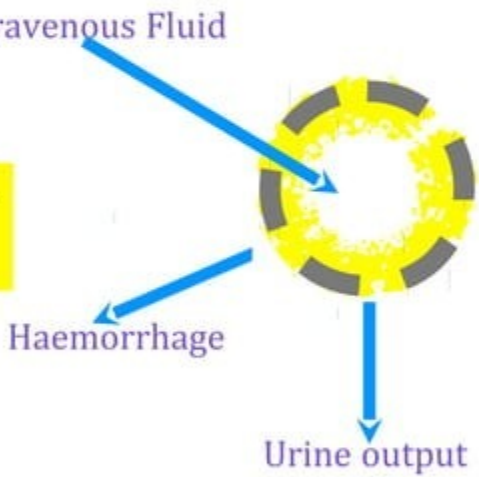

O2
nutrient
CO2
Fluid
Lymph
Lymph nodes, thoracic duct
Art
Ven
50
Capillary Pressure
25
0
Filtration
Absorption
Пр (25–28 mmHg in humans)

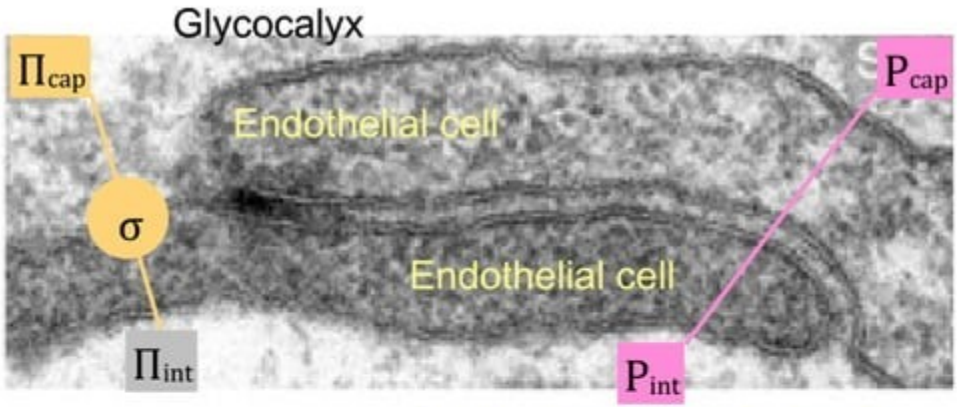
What we teach now...

Intravascular fluid exists in two forms;

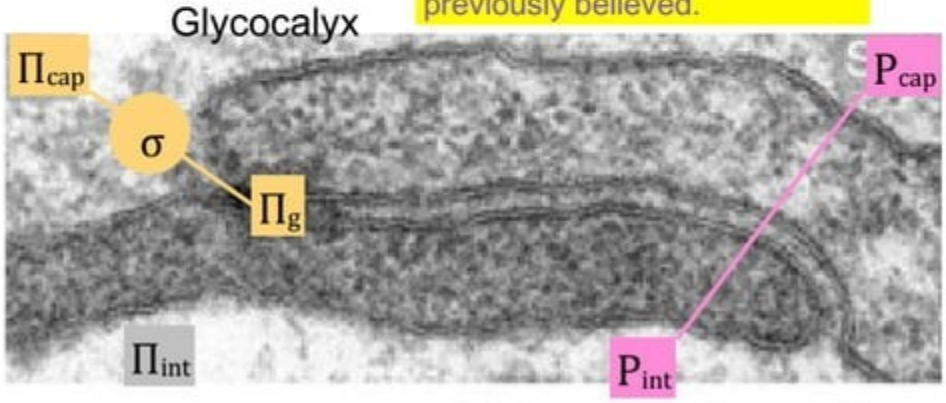
1. The free-flowing plasma

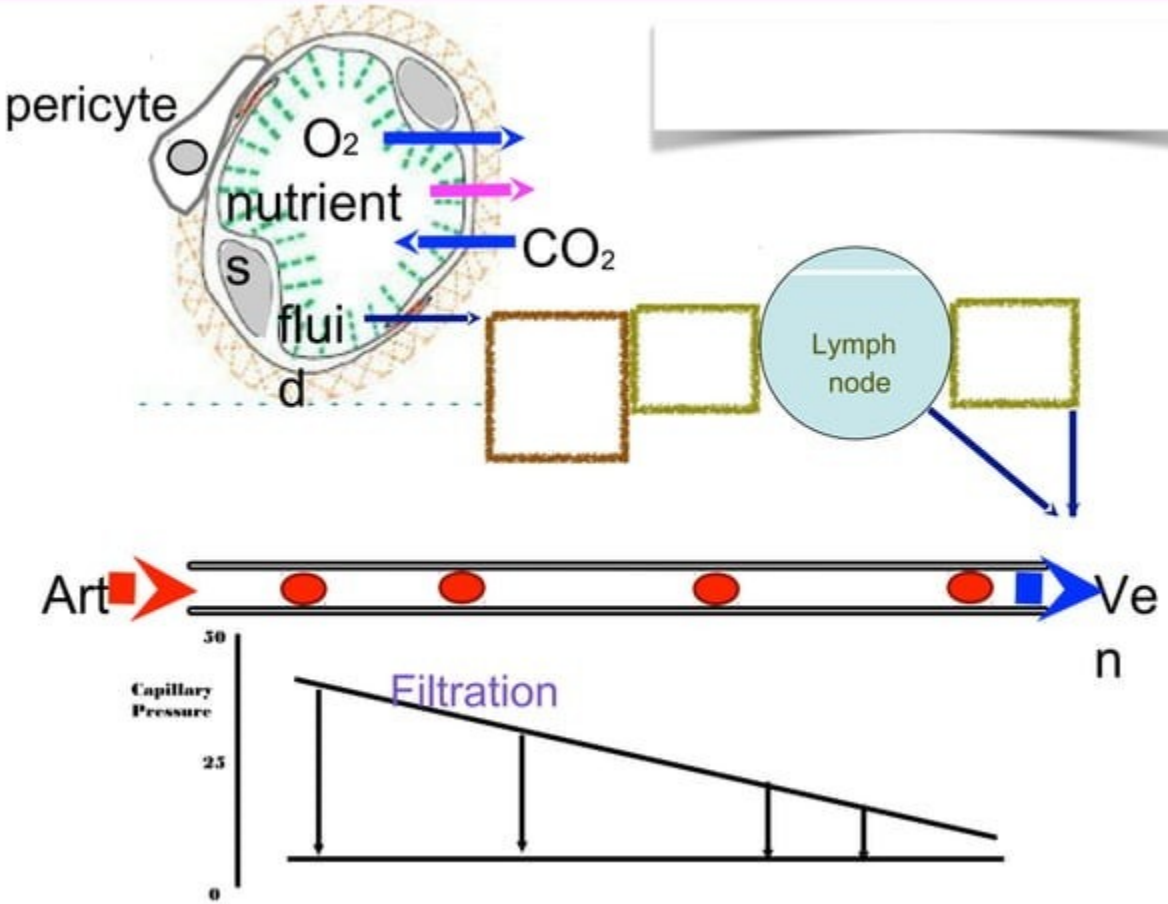
2. The gel-phase created by proteoglycans, glycoproteins & glycosaminoglycans Endothelial glycocalyx layer (EGL)





As Π_g is close to zero during filtration, J_v is much less than previously believed.

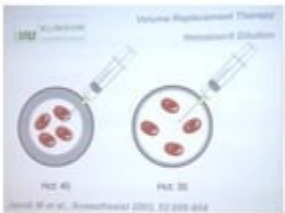


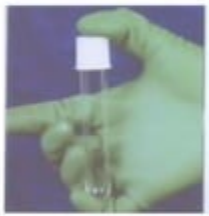

pericyte
O2
nutrient
s
flui
d
CO2
Lymph
node
Art
Ve
n
30
Capillary
Pressure
25
0
Filtration

Crystalloid Versus Colloid Intravascular Volume Effects

- Crystalloid → one fourth or one fifth of vol. within blood
- Colloids → remain largely within intravas vol.

based on RBC dilution & changes in htc **not** SGL vol.





≠



- **Colloids remain in plasma → diluting effect on htc → appear to remain** in circulation

- **Crystalloids** , in **plasma and SGL** → lower RBC dilutional effects → **appear to leave** circulation & entering ISF (remain in blood vol. within SGL)

Crystalloid Versus Colloid



"Yours is medication, and mine is coffee. They're both vital to our well-being."

Fluid Compartment Barriers and Distribution
Vascular epithelium
Crystalloid Versus Colloid Intravascular Volume Effects

Context sensitivity

- Clearance of crystalloid from intravas. vol. is slower under anesthesia than awake
- Required *crystalloid* in *low capillary pressure situations* (e.g. Resuscitation)
  ≈ 1.5:1 rather than 4:1
- The value of this ratio in periop. context is **less clear**

Example of Context Sensitivity

Table 2. Elimination Clearance of Acetated Ringer's Solution under Various Physiologic Circumstances in Adults

	Clearance (ml/min)	References
Healthy volunteers	60–110	3,8,10,11
Pre-eclampsia	125	19
Normal pregnancy	36	19
Thyroid surgery	10	17
Laparoscopic cholecystectomy	7	16
Open abdominal surgery*	21	21

* Patients received lactated Ringer's solution.

Crystalloid Versus Colloid Intravascular Volume Effects
Failure to Reduce Edema by Increasing Capillary Colloid Oncotic Pressure

- Hypoalbuminemia ; a marker of dis. severity in critical illness

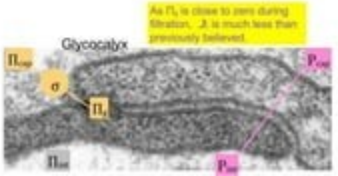


- Exogenous albumin
  - **does not** reduce peripheral or pulmonary edema
  - **does not** improve overall outcomes in sepsis

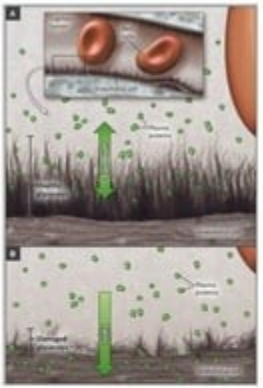


- No-absorption rule explains :
  - ↑ **COP** gradient will **not lead to reabsorption** of fluid from edematous tissues
  - Previous studies : shifts of fluid from interstitial to intravas. compartment (↓htc after albumin)
  - Now ; **compaction of glycocalyx & transfer of fluid from SGL** to plasma reduces htc.





Crystalloid Versus Colloid Intravascular Volume Effects
Failure to Reduce Edema by Increasing Capillary Colloid Oncotic Pressure



Degradation of EGL impairs endothelial barrier function

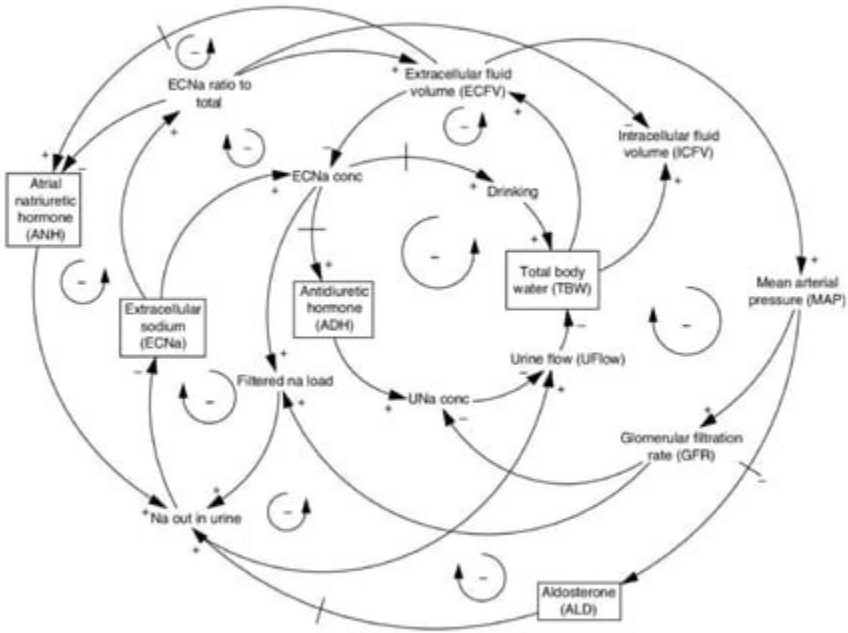
- **Physiologic insults** → glycocalyx injury & shedding
  - ✓ natriuretic peptides (acute ↑ intravas. vol.)
  - ✓ hyperglycemia
  - ✓ inflammatory mediators during surgery, trauma & sepsis
    (e.g.: CRP, bradykinin &TNF)

- **Inflammation** → EGL degradation→ ↑ No. of large pores → ↑Jv → ↑edema
  (lung, muscles & loose connective tissue)

- Impaired glycocalyx → endothelial plt. agg. & leukocyte adhesion

Maintenance of glycocalyx integrity is
a therapeutic target in
Periop. fluid management

Physiologic Control of Overall Fluid Balance



Physiologic Control of Overall Fluid Balance

Acute Disturbances in Circulating Volume

- Acute ↑↓intravas. Vol. → compensatory mech (min. to hrs)

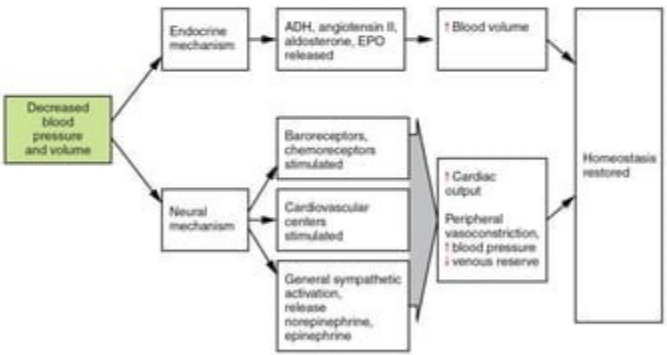
Acute blood loss

- **Rapid response**
  - ✓ ↑ effective blood vol. (venoconst. & mobilization of venous reservoirs)
  - ✓ Autotransfusion from ISF to plasma
  - ✓ ↓urine production
  - ✓ ↑C.O. & arterial pressure

Sensor organs:

Low & high-pressure barorecep. →

↑ sympath. & RAAS



- **Delayed responses :**
  - ✓ restore plasma vol. within 12 - 72 hrs
  - ✓ ↑ hepatic protein synthesis
  - ✓ erythropoiesis within 4 to 8 wk

Physiologic Control of Overall Fluid Balance

Acute Disturbances in Circulating Volume



Acute rapid fluid infusion

↑ venous & arterial press. & C.O.

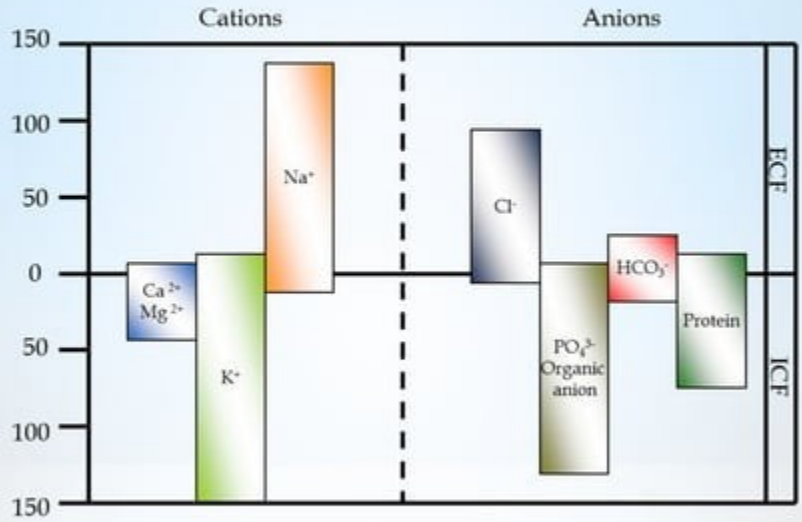
- Several mechanisms act rapidly
  - Pressure receptor– mediated venodil. & venous pooling & ↓SVR
  - At a tissue level : **arteriolar vasoconstriction**

- Multiple mechanisms then act :
  - ↓COP → ↑capillary filtration
  - ↓ADH → diuresis
  - ↑ atrial natriuretic peptide (ANP) → natriuresis
  - ↓ COP → glomerulotubular imbalance→
    - ↑GFR
    - ↓proximal tubule water and Na+ reabsorp. → ↑ U/O
    - *BP slowly restored* after acute hypervol.

- Take *several days* for a *20 mL/kg* of isotonic salt solution to be fully *excreted*


B.A.A.
1040

*Electrolyte Physiology

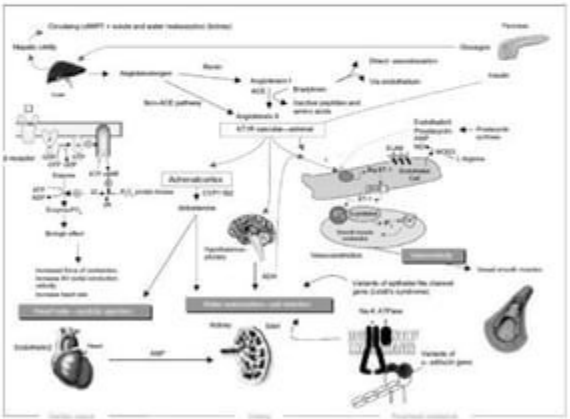


Sodium Physiology
Na ≈ 138 to 142 mEq/L

- Dominant extracellular cation
- Na^+ + its anions ≈ all osmosis of plasma & interst fluid
- Total body Na+ ≈ 4000 mmol (10% intracell)

- Intra /extracellular ratio 1:15 , by ATPases
  (vital for action potentials)

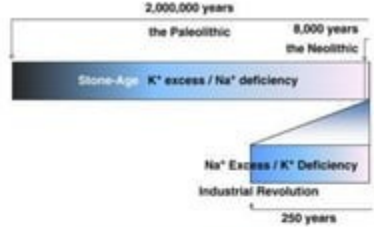
- Minimum daily req. :
  - 2 to 3 mEq/kg/day at birth
  - 1 to 1.5 mEq/kg/day in adulthood



Sodium Physiology

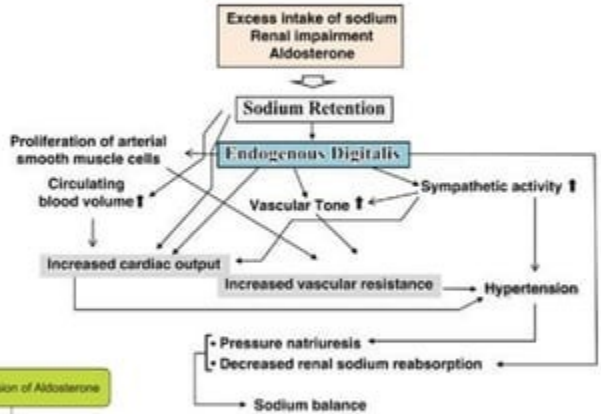
- Actively absorbed from small intestine & colon by aldosterone & glucose
- Loss is by renal , minority by feces, sweat & skin (10 meq/d each)
- Freely fitered at glomerulus, 99.5% reabsorbed at prox. convoluted tubule
- Control circulating vol. as follows:
  - Hypothalamic osmoreception: ADH release
  - Atrial volume sensing: ANP release
  - Juxtaglomerular apparatus & RAAS

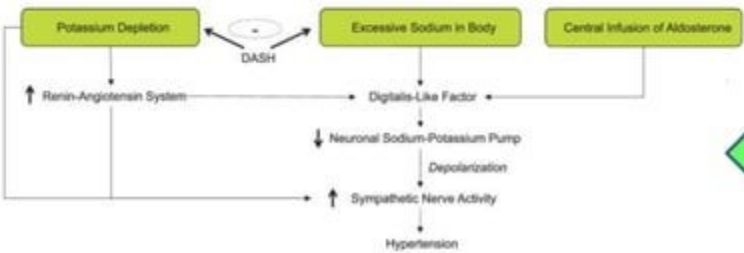
Sodium Physiology



- Excretion of ↑ Na+ relies on ***inefficient passive mechanisms***, particularly pressure-volume effect

- Chronic **↑ salt & ↓ K intake → HTN** not seen in < 50 mmol ≈ 1.3 mg/d



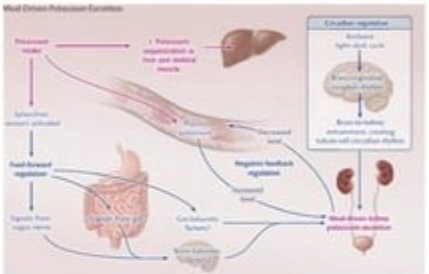


DASH : Dietary Approaches to Stop Hypertension

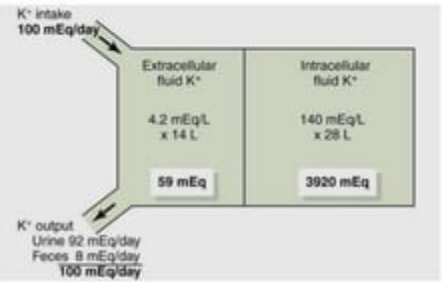
Potassium Physiology

- Dominant intracellular cation
- Total body content ≈ 4000 mmol (98% intracell. ; muscle, liver & RBCs)
- ICF to ECF balance is vital for cellular resting mem. Potential
- Daily requirement ≈ age & growth, ↑metabolic rates→↑ required K
  - ✓ Term infants require 2 to 3 mEq/kg/d
  - ✓ Adults 1 to 1.5 mEq/kg/d
- Absorbed by intestine, minimal excreted in feces
- Transmembrane potentials particularly depend on K+ permeability

Potassium Homeostasis



From N Engl J Med. Gumz ML, et al., An Integrated View of Potassium Homeostasis, 373., 60-72. Copyright © 2015 Massachusetts Medical Society. Reprinted with permission from Massachusetts Medical Society.

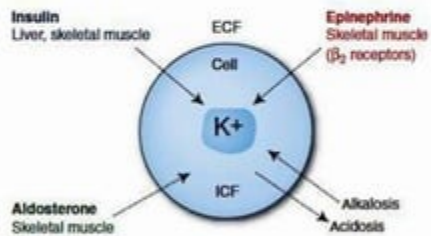


Potassium Physiology

1. Internal balance (ICF to ECF K+ distribution)
2. External balance (Renal excretion of K+)

➢ ***1. Internal balance***

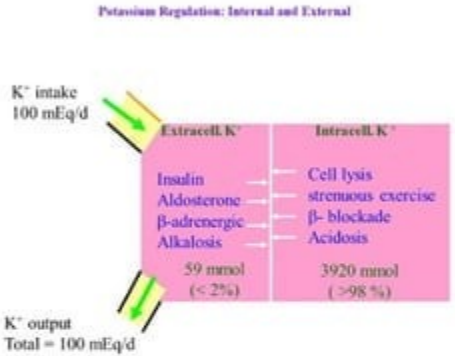
- Physiological and pathological conditions
  - Hormones : insulin, catecholamines ,aldosterone
  - Acid base imbalance
  - Changes in osmolarity



Potassium Physiology

- Insulin and beta 2 agonsists shifts K+ to cell, by:
  - ✓ ↑ Na^+,K^+-ATPase
  - ✓ ↑ 1 Na^+- 1 K^+- 2 Cl^- symporter
  - ✓ ↑Na^+-Cl^- symporter

- Aldosterone
  - ✓ ↑ uptake of K+ into cells
  - ✓ ↑ urinary K+ excretion



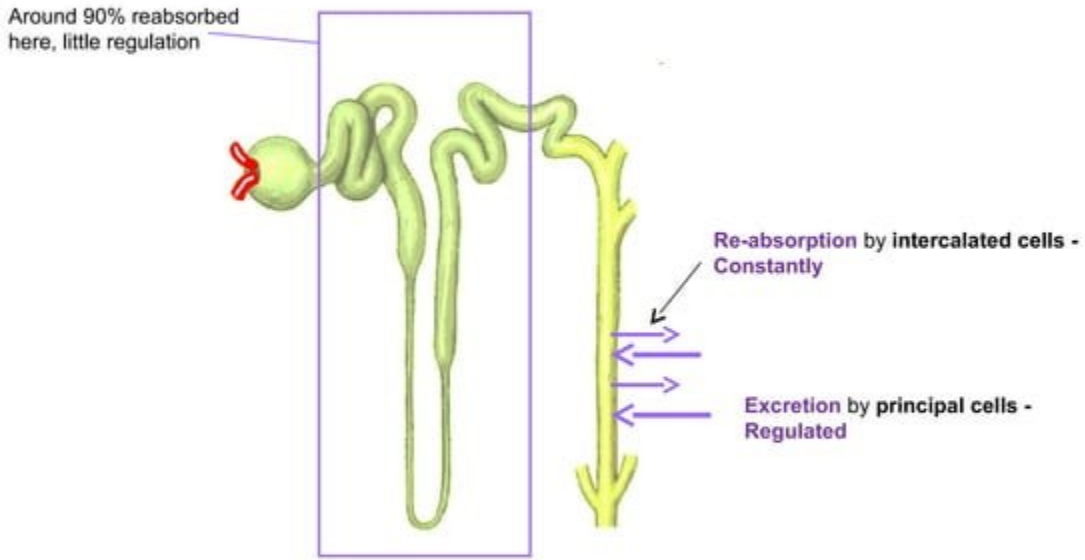
- Insulin & EPN a few min.
- Aldosterone ≈ an hr

⟹ uptake of K+ into cells

- **Hypokalemia** → ↓ skeletal mus. Na+/ K+ ATPase →"leak" of K+ from ICF to ECF
- Stimulation of α-adrenocep. **releases** K^+ from cells, esp. liver

Potassium (only 2% of body K^+ is in extracellular fluids and it has to be regulated tightly)

1. **Intercalated cells : constant reabsorption**
2. **Principal cells : regulated excretion**

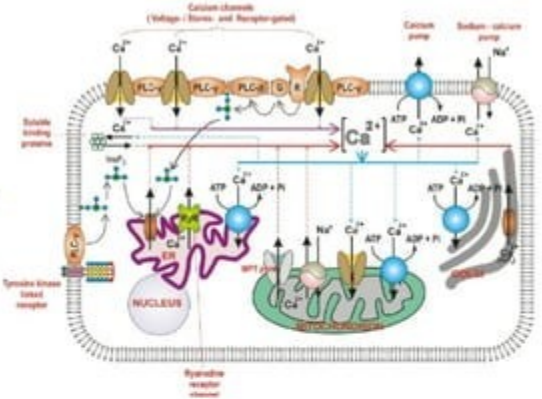


>

Data from Giebisch GH. (2002) A trail of research on potassium. Kidney Int. 62:1498-512



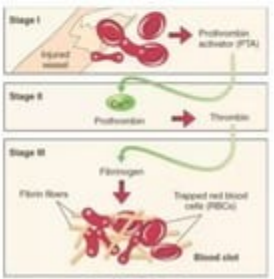
Calcium Physiology
(8.5 to 10.5 mg/dL)



- Bone : 98% of body Ca2+
- Very important intracellular 2nd messenger
- key role in
  - mus contraction,
  - neuromus transmission
  - cell division & movement
  - oxidative pathways

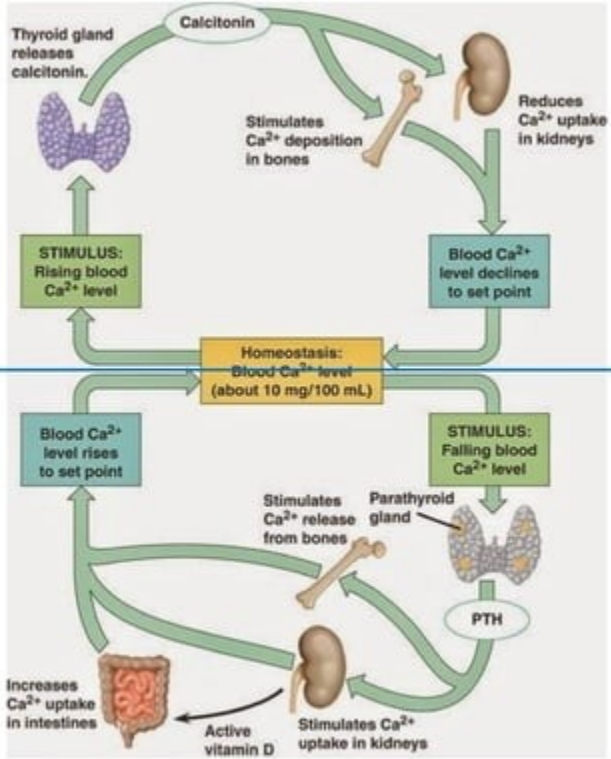
- A large ECF-to-ICF (by ATPase)
- low cytoplasmic free Ca2+ (by pumping into sarcoplasmic reticulum)
  - ↑cytoplasmic free Ca2+ (key mediator of cell death pathways)

- A key role in coagulation



Calcium homeostasis

Rising ca level



Falling Ca level

Calcium Physiology

- ✓ 50% ionized (normal range 2 to 2.5 mEq/L)
- ✓ 40% bound to proteins (mostly albumin & globulins)
- ✓ 10% complexed to anions (HCO_3^-, citrate, sulfate, PO_4^{3-} & lactate)

- ➢ Hypoalbuminemia → ↓ total serum Ca^{2+} (less effect on ionized form)

$$[Calcium]_{corrected} = [Calcium]_{measured} + \left(0.8\,(4 - [albumin])\right)$$

Correcting total serum calcium for hypoalbuminemia

- ➢ Acidemia → ↓protein binding & ↑ionized Ca (0.1 mEq/L per 0.1 ↓pH)
- ➢ Ionized Ca^{2+} should be measured when possible (Specimens taken without tourniquet because local acidosis increases ionized Ca)



Magnesium Physiology (1.5 - 2.1 mEq/L)

- primarily an intracellular anion (1% ECF)
  - ✓ 50% within bone
  - ✓ 20% within muscle
  - ✓ 30% in liver, heart & other tissues



- Total Mg2+ concentration : 1.5 to 2.1 mEq/l
  - ✓ 25% protein bound (mostly albumin)
  - ✓ 65% ionized
  - ✓ 10% complexed to phosphates, citrates & other anions

- Magnesium homeostasis

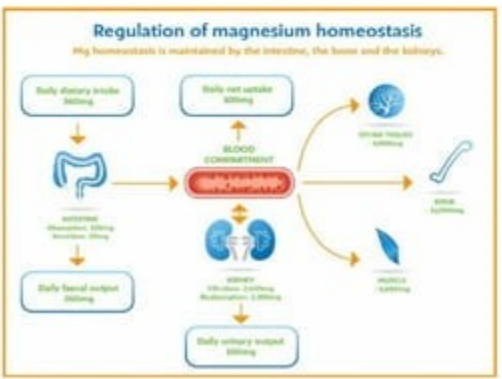


TABLE 59-5 PHYSIOLOGIC ROLES OF MAGNESIUM

System	Effect	Mechanism and Clinical Relevance
Neurologic	Reduction in pain transmission	NMDA antagonism. Mg^{2+} treatment provides effective perioperative analgesia.[236]
	Reduces neuromuscular transmission	Inhibition of neuronal Ca^{2+} influx reduces neuromuscular junction ACh release (and motor end-plate sensitivity to ACh). Hypermagnesemia potentiates the effects of neuromuscular blockade.
	Sympatholysis	Inhibition of neuronal Ca^{2+} influx reduces catecholamine release from adrenal medulla and adrenergic nerve endings. Pharmacologic use of Mg^{2+} in obtunding pressor response to intubation or during surgery for pheochromocytoma.
	Anticonvulsant	Mechanism may relate to NMDA antagonism or cerebral arteriolar vasodilation, possible mechanisms for its efficacy in eclampsia, in which vasospasm has been observed.[29]
	Cortical depression at high levels	
Cardiovascular	Vasodilation	Predominantly arteriolar, because of inhibition of Ca^{2+} influx–mediated vascular smooth muscle contraction. Mg^{2+} administration typically leads to a minor reflex increase in inotropy despite the direct action of Mg^{2+} on reducing cardiac contractility.[237]
	Antiarrhythmic effects	Mixed class IV (Ca^{2+} channel inhibition) and weak class I (Na^{+} channel inhibition) effects. Increases atrioventricular nodal conduction time and refractory periods, suppresses accessory pathway transmission, and inhibits early and delayed afterdepolarizations. Clinical use is in supraventricular tachycardias, atrial fibrillation rate control and postoperative prophylaxis, and tachyarrhythmias associated with dyskalemia, digoxin, bupivacaine, or amitriptyline.[29]
	Improved myocardial O_2 supply-to-demand ratio	Coronary vasodilation in combination with reductions in heart rate and contractility; however, no clear evidence of benefit in the setting of acute myocardial infarction.
Respiratory	Bronchodilation	Smooth muscle relaxation. Pharmacologic use of Mg^{2+} is in acute bronchospasm.
Renal	Renal vasodilation and diuresis	Ca^{2+} antagonism–related smooth muscle relaxation
Immune	Antiinflammatory	Pharmacologic doses of magnesium sulfate reduce monocyte inflammatory cytokine production.[238]
	Adaptive immunity	Mg^{2+} is required as a second messenger during T-lymphocyte activation.[239]
Obstetric	Tocolysis	May be due to smooth muscle relaxation

ACh, Acetylcholine; *NMDA*, *N*-methyl-D-aspartate.

Magnesium Physiology

Mg2+ : 3 main cellular actions

1.Energy metabolism: Mg2+ is required for **ATP phosphorylation** → defiiency impairs enzyme systems (e.g.: glucose metabolism)

2.Nucleotide and protein production: cofactor **in every step of DNA** transcription & replication & translation of messenger RNA (mRNA)

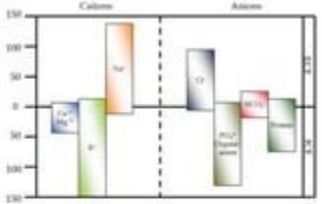
3.Ion transport:

- **Nl transmem. Potential**
- **competitive antagonism of Ca2+** → ↓ inflx of Ca2+
- **antagonizes NMDA** recep. in CNS → ↓Ca2+ entry→ ↓ **cellular actions**
  1. neurotransmitter release
  2. Mus. contraction
  3. cardiac pacemaker
  4. action potential activity
  5. pain signal transmission

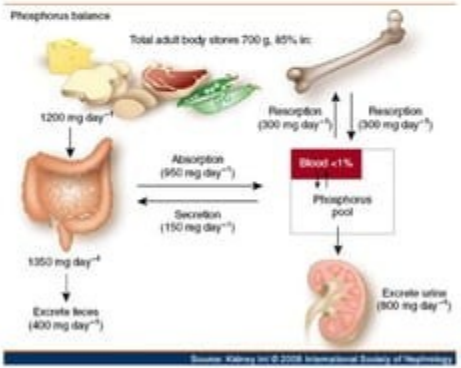
Phosphate Physiology



Phosphate Physiology (3 to 5 mg/dL)



- most abundant intracellular anion
- important biologic molecules
  - ✓ (ATP, DNA, RNA, membrane phospholipids, 2,3-DPG)
- Needed for
  - ✓ energy metabolism
  - ✓ cellular signaling
  - ✓ cellular replication
  - ✓ protein synthesis
  - ✓ membrane integrity
  - ✓ O2 delivery
- One of the key intracellular buffers
- Total body phosphorus
  - ✓ 80% to 90% in bone
  - ✓ remainder in ICF (soft tissues and erythrocytes) & ECF



Phosphate Physiology

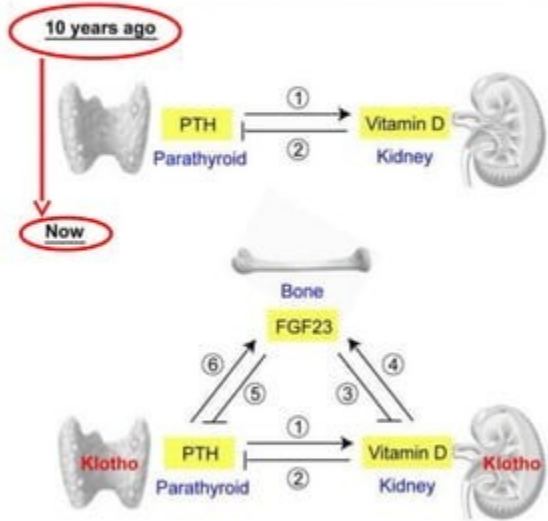
Regulation of Phosphate

- PTH & vit. D →

- Dopamine
- Adrenergic activity
- Alkalosis

} ↑cellular uptake → ↓ phosphate

Endocrine regulation of phosphate homeostasis



FGF : fibroblast growth factor
Klotho : a transmembrane co-receptor

UPDATE ON CHLORIDE

DUSKA

The 'hidden' ion?

DR. DAVID LYNESS
@GAS_CRAIC



YES, IT'S IMPORTANT



LABS MORE ACCURATE
THAN BLOOD GAS ANALYSER!

Saline resuscitated sepsis = 1/3 of acidosis = hyperchloridaemic -

Saline resuscitated DKA = 100% of acidosis = hyperchloridaemic after 8 hours -

KEY THOUGHTS

- PLASMA CHLORIDE IS NOT JUST A NUMBER
- SOME OF THE EFFECTS OF CHLORIDE ARE LINKED WITH ACIDOSIS
- (NEGATIVE INOTROPISM, VASODILATION: SID & pH MATTER)
- CHLORIDE-SPECIFIC EFFECTS IRRESPECTIVE OF pH =
- CORTICAL PERFUSION OF THE KIDNEY
- INNATE IMMUNITY
- PRO-INFLAMMATORY RESPONSE INCR IN IL-6, TNF-a AND DECREASE IN IL-10

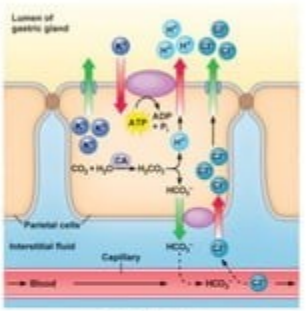
TRIALS!

TYGIT/PE:

RCT:

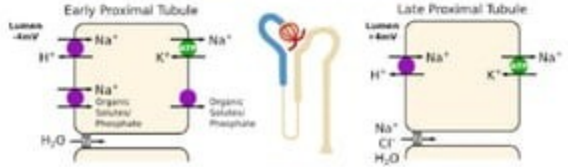
Chloride Physiology
(97 to 107 mEq/L)

- The 2nd most abundant extracellular electrolyte
- key role in
  - plasma osmolality
  - electrical neutrality
  - acid-base status **(Stewart model)**
- Responsible for :
  - 1/3 of plasma osmolality
  - 2/3 of plasma negative charge



- GI : absorbs & secretes Cl− as HCL

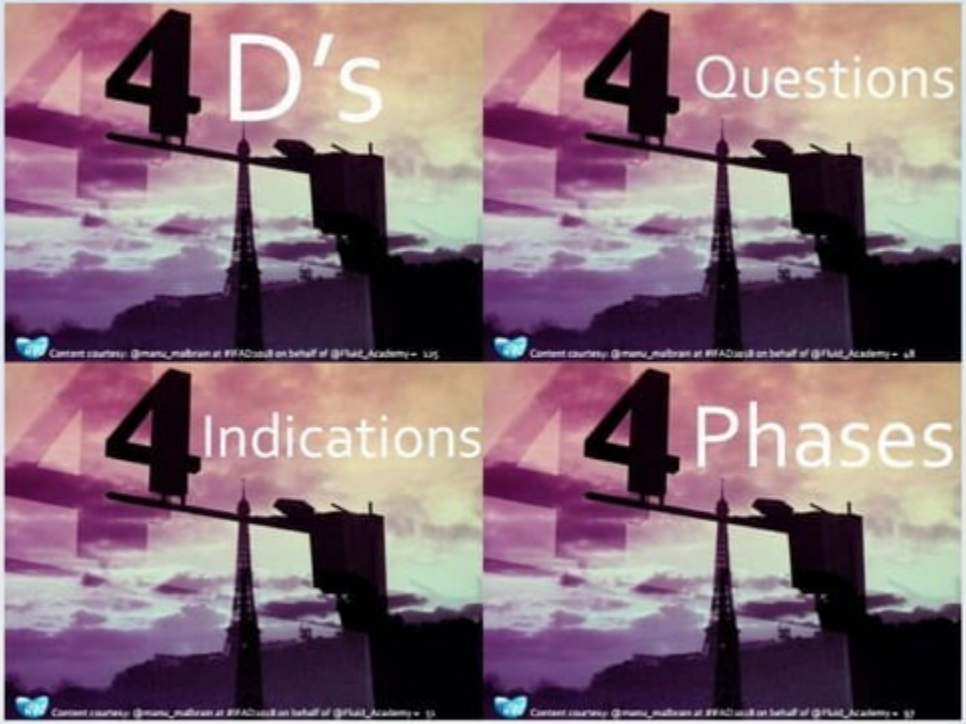
- Renal excretion
- Proximal tub. : passive reabsorption or cotransport
- Distal nephron under influence of acid-base balance ; e.g.: exchange of HCO3− for Cl−






35
Br eaking
56
Bad habits


50 little bags of chips= 1 liter 0.9% NaCl
Lay's
Classic
=
0.9%
SODIUM CHLORIDE
(Sean Bagshaw)
@drseanbagshaw
⇒ AB-normal Saline


4 D's
4 Questions
4 Indications
4 Phases

1









2


Start IV fluid
START
Stop IV fluid
STOP
Start removal
dialysis
medical
kidney
therapy
equipment
hospital
healthcare
disease
blood
patient
Stop removal

Thank you


Fluids in Surgery + Trauma
Starches ?
Gelatines ?
Saline ?
No gluc 5%
Balanced
Blood
Shelve
Fluid strategy