NA Imbalance

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Water and Electrolyte Distribution
- total body water -

- 70kg- BW- normal adult person.
- Men have total body water (TBW)
  - 50-60% of lean BW.
- Women have total body water
  - 45-50% of lean BW.
- Total body water is about 40L
  distributed between:

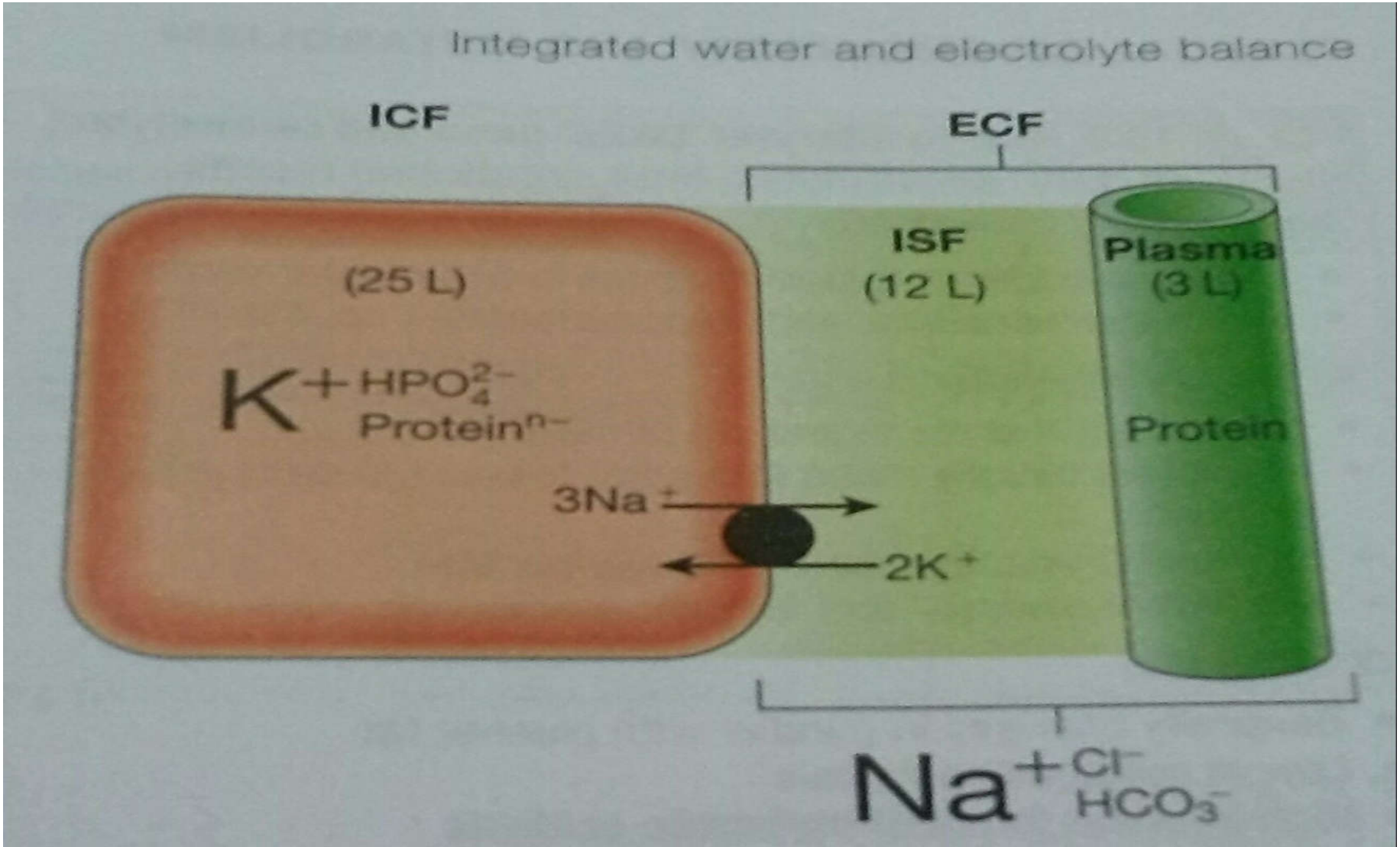
- ICFV (intracellular fluid volume)

- 25L >50% of total body water

- ECFV (extracellular fluid volume) - divided into:

- ISF (interstitial fluid) - 12L
- IVFV (intravascular fluid volume) - 3L

Water and Electrolyte Distribution
- total body water -



Water and Electrolyte Distribution
- total body water -

OSMOTIC PRESSURE –

- the ability of the main solutes (K – Na) to hold and maintain water in each compartment.
  - K : in ICFV
  - Na : in ECFV

- Na (determinant of ECFV)
  - EABV- Effective arterial blood vol.
  - BP

ONCOTIC PRESSURE –

- Plasma protein holds and maintains water in the IVFV.

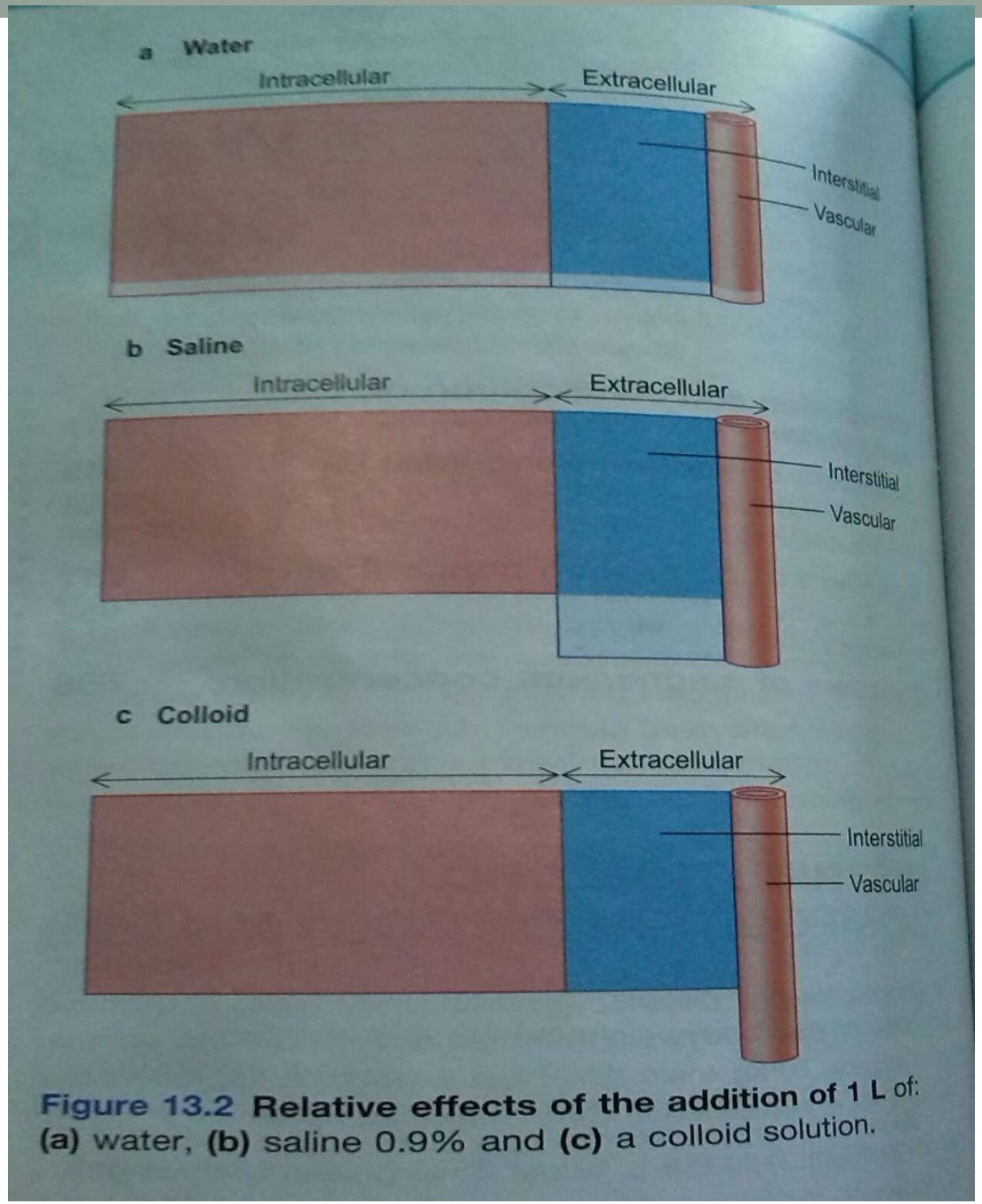


Figure 13.2 Relative effects of the addition of 1 L of: (a) water, (b) saline 0.9% and (c) a colloid solution.

Water Homeostasis

1- Total daily **water intake** is about **2600ml** including-

- 1400 ml oral water drinking
- 850ml from food intake
- 350ml Endogenous Metabolic water production.

2- Total daily **water loss** is about **2600ml** including-

- 1500ml Urine out put
- 900ml Insensible water loss from skin- resp.sys
- 200ml water loss in the stool

Water and Plasma OsmolaLity

- Normal plasma Osmolality- 280 - 296mosmol/kg.
  P. Osmolality = 2 x Na+Glucose/20+BUN/3.
- Changes in P. Osmolality-
  ADH-BNP-ANP-ALDOSTERONE H.
- Sensed and corrected by: inhibition or stimulation of
  Hypothalmic –Osmo receptors - ADH
  Vol. stretch receptors- HEART-BNP-ANP
  Pressure receptors- GREAT VESSELS

1- THIRST- Controlling oral water drinking

2- HYPOTHALAMUS- POST-PITUITARY GLAND- ADH - RENAL COLLECTING DUCTS water re-absorption or excretion Stabilizing plasma osmolality.

Water and Plasma OsmolaLity

1- Water load – more water intake –

Hypo- Osmolality < 280. Inhibits ADH

- Polyuria- WATER LOSS
- Normalizing plasma Osmolality.

2- Severe thirst or no water intake -

High plasma Osmolality >300 .

Thirst – MORE water oral drinking

Hypothalamic Osmo-receptors – ADH

- Renal collecting ducts water re-absorption.
- Oliguria- Normalizing plasma osmolality.

Water and Plasma OsmolaLity

3- High Na- salt intake-

High osmolality- Hypervolaemia.

- VOL. Receptors - Release - ANP- BNP- Natriuresis OSMO-Receptors- ADH- water reabsorption
- Suppress - Aldo. H. secretion decrease -Na- absorption.

- Normalizing P. Osmolality.

4- SIADH- persistent high ADH- maligna

water re-absorption - water intoxication

dilutional hyponatraemia- hypo-osmolality.

Hyponatraemia

- Normal plasma Na- 135-145mmol/L.
- Na-determinant - ECFV

EABV- Effective arterial blood volume .

EABV= Cardiac out put- Peripheral vascular resistance.

Drop of EABV- vascular under-filling stimulates

1- Renin- Angiotensin- Aldosterone sys.

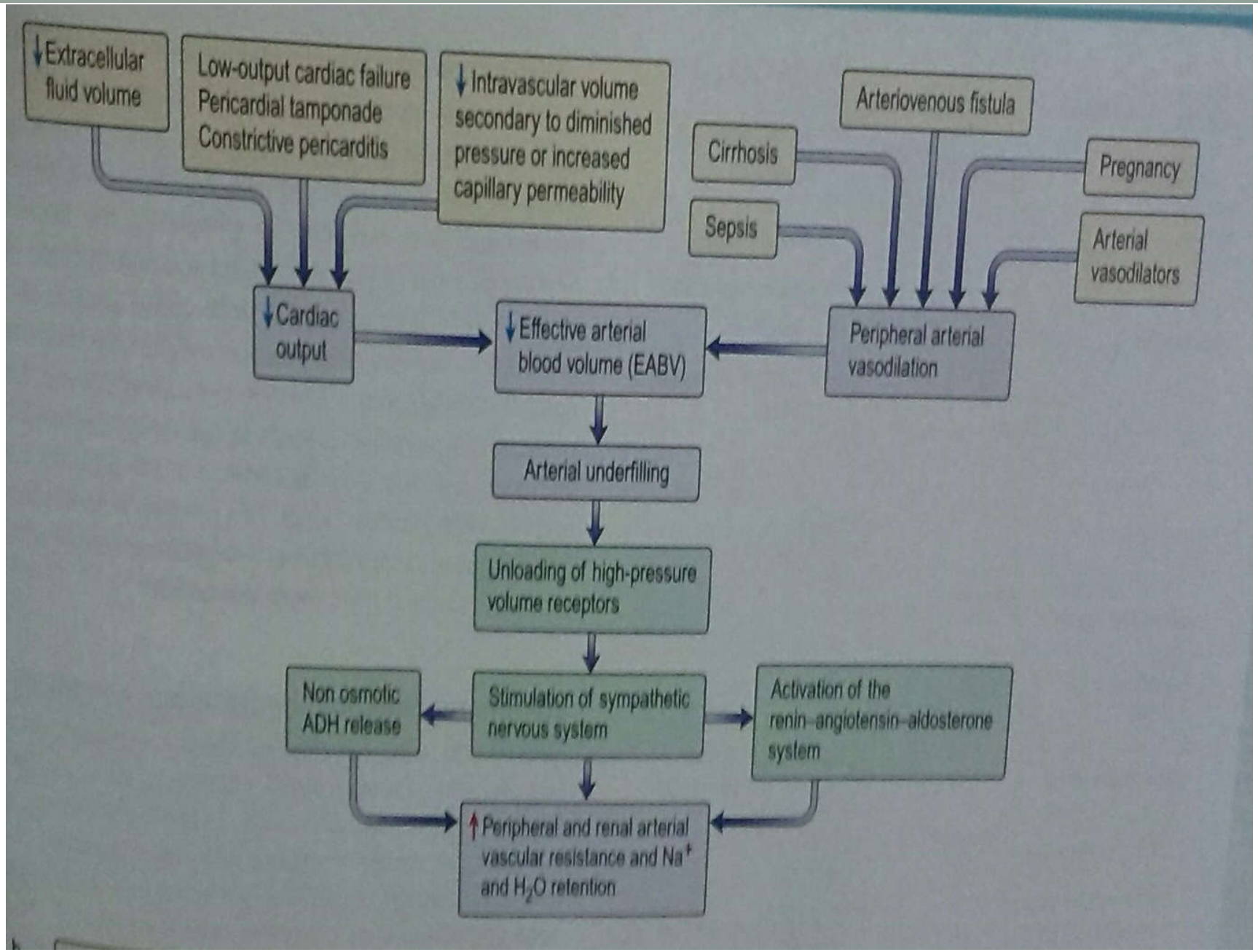
2- ADH

3- Sympathetic

Na + WATER- reabsorption maintaining plasma volume and BP.

Hyponatraemia

- S. Na- level reflects followings
  - ECFV - EABV
  - WATER STATUS- WATER PROBLEM
  - Osmolality
  - Hydration state of the body.
- Alteration of S. Na-reflects alteration of
  - Total free body water balance.
- Low S. Na-level indicates: free water excess.
- High S. Na-level indicates: relative water deficit.


↓Extracellular fluid volume
Low-output cardiac failure
Pericardial tamponade
Constrictive pericarditis
↓Intravascular volume secondary to diminished pressure or increased capillary permeability
Arteriovenous fistula
Cirrhosis
Pregnancy
Sepsis
Arterial vasodilators
↓Cardiac output
↓Effective arterial blood volume (EABV)
Peripheral arterial vasodilation
Arterial underfilling
Unloading of high-pressure volume receptors
Non osmotic ADH release
Stimulation of sympathetic nervous system
Activation of the renin–angiotensin–aldosterone system
↑Peripheral and renal arterial vascular resistance and Na^+ and H_2O retention

Hyponatraemia

- ISO-OSMOLAR- PSEUDO-HYPONATRAEMIA
  - high protein -MM- high lipids

- HYPER-OSMOLAR- DKA – MANNITOL

- HYPOOSMOLAR - (commonest)
  - **1-** Hyponatraemia with Hypovolaemia.
  - **2-** Hyponatraemia with Euvolaemia.
  - **3-** Hyponatraemia with Hypervolaemia.

- VOLUME STATUS
  - JVP- LEG-OEDAMA - SKIN TURGER
  - BP- POSTURAL HYPOTENSION-
  - BW - ASCITIS

Hyponatraemia with Hypovolaemia

- Depletional hyponatraemia
  - There is Na-loss with relatively smaller water loss.

- AETIOLOGY-

1- EXTRA-RENAL LOSS

A- GIT-

Vomiting – Diarrhea - Acute pancreatitis - Haemorrhage

B- Skin-loss – Excessive sweating - BURN.

Hyponatraemia with Hypovolaemia

2- RENAL LOSS INCLUDES

A- Diuretics

Uraemia

recovery polyuric phase of –ATN.

B- Addison's disease

Hyponatraemic Encephalopathy

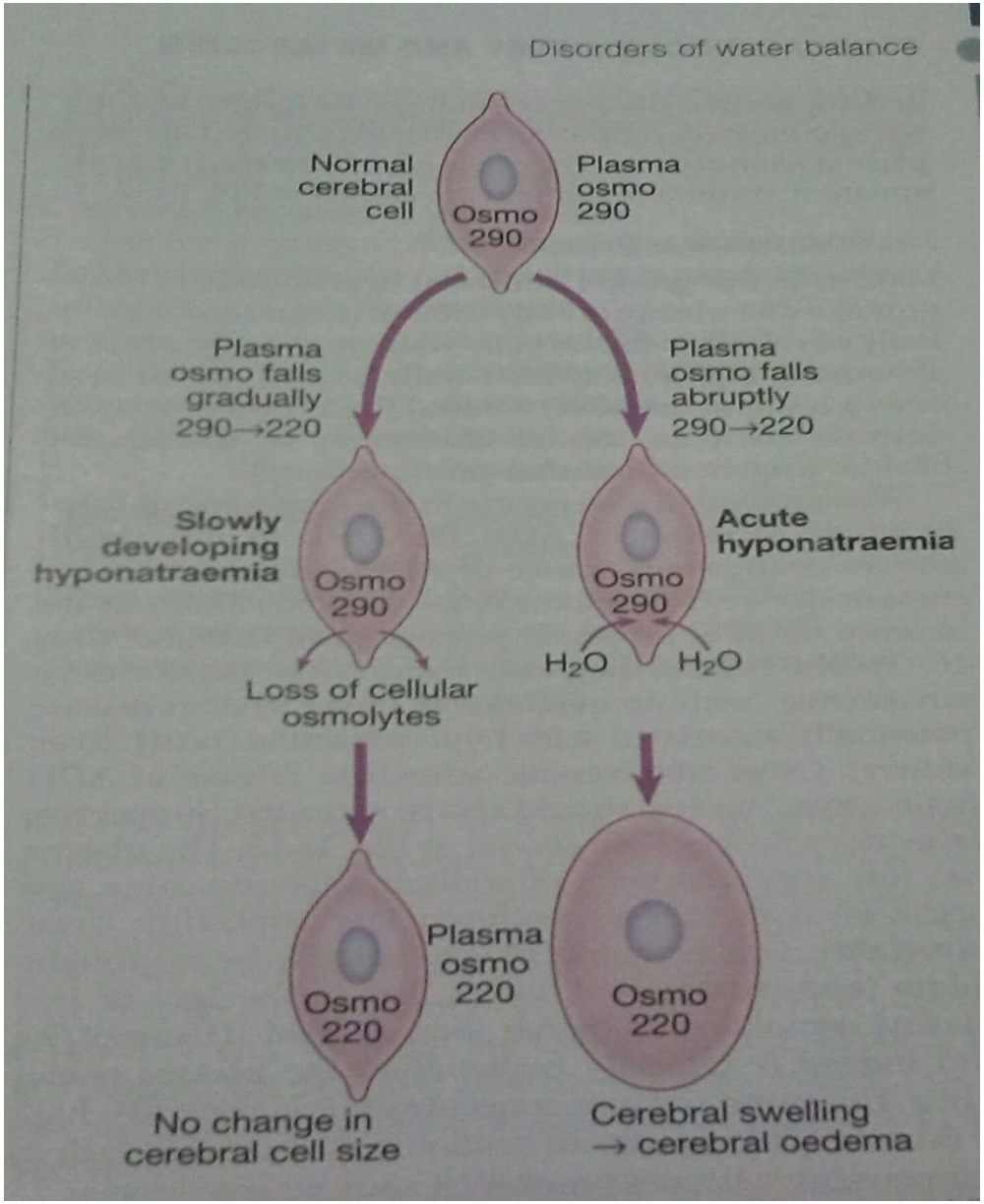
- ACUTE- RAPID RATE of onset of Hypo-natraemia
- Rather than the SEVERITY.
- Acute hyponatraemia -hypo-osmolarity.

  Causing quick shift of water from ECV- to- ICV

  Acute brain cells swelling - BRAIN EDEMA .

  CLINICALLY
- anorexia, nausea, vomiting- Headach
- Acute confusional state - Fit- coma.

Pathophysiology



Hyponatraemic Encephalopathy

- GRADUAL – LONG STANDING
- Chronic Hyponatraemia – hypo-osmolality.

  - The cerebral cells have time to adapt by slowly

    releasing intra-celluar- osmolytes from ICFV- into ECFV-

    decreasing ICF osmolality.
- Low cellular osmolality gradient

  between– ECFV- ICFV.

  protecting brain cells.

Osmotic Demyelination Syndrome (ODS)

- previously known: Central Pontine Demyelination Syn.

  - serious FATAL neurological problem due to rapid correction of hyponatraemia.

  - quickly brain cells shift water from- ICV- to ECV – causing Cerebral Cells Shrinkage- disturbing blood brain barrier function

  - allowing lymphocytes and cytokines to enter into the brain causing **demyelination and neuronal damage.**

Osmotic Demyelination Syndrome (ODS)

- Rapid correction of hyponatraemia
  - should be avoided.

- Slow correction by no more than
  8 mmol/L/24hour rise in plasma Na.

- Other risk factors of ODS should be considered and corrected
  - Hypoxia
  - Hypokalaemia
  - Hypomagnesaemia

Management of Hyponatraemia – Hypovolaemia

A- Mild -135-130 mmol/L - asymptomatic.

B- Acute Symptomatic Hyponatraemia -120-110-ho.- days.
CEREBRAL- EDEMA. SLOW CORRECTION
HYPERTONIC SALINE - 3% -100 cc-IV-BOLUS-REPEATED.
Treat underlying cause.

C- Slowly developing- Long Standing Chronic Hyponatraemia -
Over weeks –months
Cerebral cells already adapted
By reducing their intracellular osmolytes .
plasma- Na- correction should be slowly
By no more than Na- 8-10 mmol/L/24h-

- TREATMENT- decrease water intake
ADH- ANTIGONIST- WATER LOSS. treat underlying cause.

Hyponatraemia with Euvolaemia - water retention alone-

Dilutional Hyponatreamia

1- EXCESS WATER INTAKE- Primary Polydipsia
MARATHON RUNNERS – excessive water intake.

2- HYPOTHYROIDISM-

3- Iatrogenic high IV - D/W.

4- POST-OP.

- Prostectomy using urinary bladder
- Fluid irrigation Isotonic mannitol or
- Hypo-osmotic sorbitol or Glycin solution.

5- SIADH-

- Psychiatric pat, Anti-depressant, NSAID, Malignancy.

Clinical Presentation

- Often presented Acutely within <48h.
- Post-Op
- Symptoms start

  When plasma Na- <120 mmol/L.

  Severe symptoms - Plasma Na- drops <110 mmol/L.
- Causing hyponatraemic encephalopathy .
- Brain edema
- Headache, Acute confusional state, Drowsiness, Fit, Coma.

Management

1- Correct the underlying causes.

2- Water restriction- < 1L/24h.

3- Correct associated Mg- K- def.- Hypoxia.

4- Hypertonic saline 3% IV .

5- Do not give D/W.

Hyponatraemia with Hypervolaemia

- Na- retention with relatively GREATER water retention
- AETIOLOGY-

CHF- LIVER CIRRHOSIS- NEPHROTIC SYN.

Decrease Effective Arterial blood Vol- EABV

Arterial under filling Stimulate

ADH- Sympathetic H. - RENIN- Aldosterone H.

Na- and Water retention –

Vol. expantion- Generalised body Edema- ANASARCA.

- MANAGEMENT-

RESTRICT WATER IN TAKE DIURETICS

Differential Diagnosis of Hyponatraemia

1- Primary polydipsia

- Urine Na low <30 mmol/l
- Urine Osmolality low <100mmol/kg

2- Hypovolaemia

- Urine Na low
- Urine Osmolality high >250mmol/kg--concentrated

3- Diuretic

- Urine Na high >40mmol/l
- Urine Osmolality low

4- SIADH

- Urine Na High
- Urine Osmolality High

Hypernatraemia
ALWAYS HYPEROSMOLAR

1- HYPERNATREMIA WITH HYPOVOLAEMIA

- Na- loss with relatively greater water loss
- AETIOLOGY
- RENAL LOSS
- DIURETICS
- HYPEROSMOLAR HYPERGLYCAEMIC COMA
- GI-LOSS
- DIARRHEA- VOMITING – SKIN LOSS -SWEATING

Hypernatraemia

2- HYPERNATRAEMIA - EUVOLAEMIC

- Water deficit alone- DI

A- DI- Central – no ADH- or Nephrogenic type.

B- Drugs- Lithium - Tetracyclin - Amphotercin -B-

C- Iatrogenic- IV - HYPERTONIC FLUID.

- Clinical Presentation:

Polyuria- Polydipsia

Dizziness - Confusion- Weakness

Convulsion- Coma.

3- HYPERNATRAEMIA - HYPERVOLAEMIA

- Na-retention-with relatively LESS water retention

CKD- IV- RESUSCETATION- - IV- Parenteral Feeding.

Management

1- REHYDRATION- WATER REPLACEMENT

IV- N/S - 0.9%-

DW 5% or –

Hypotonic N/S- 0.45%

- Extremely slowly lowering plasma-Na- level To avoid Brain Edema.

2- CDI- DESMOPRESSIN -

NDI- HYDROCHORTHIAZIDE .

3- Stop offending drugs.