ELECTROLYTE IMBALANCE

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OUTLINE....

Pathophysiology, Clinical Features, Aetiology and Management of.....

A. Hyponatraemia

B. Hyperkalaemia

HYPONATRAEMIA

- The most common electrolyte disorder(occur in up to 6% of hospitalized patients)
- Serum sodium concentration of <135 mEq/L
- Due to an excess of body water relative to body sodium content
- Changes in total body water is regulated by thirst, arginine vasopressin(AVP) and the kidney

HYPONATRAEMIA

- Three types:
  1. Hypovolaemic Hyponatraemia
  2. Hypervolaemic Hyponatraemia
  3. Euvolaemic Hyponatraemia

HYPONATRAEMIA

Hypovolemic

- Gastrointestinal solute loss (diarrhea, emesis)
- Third-spacing (ileus, pancreatitis)
- Diuretic use
- Addison disease
- Salt-wasting nephritis

HYPONATRAEMIA

Euvolemic

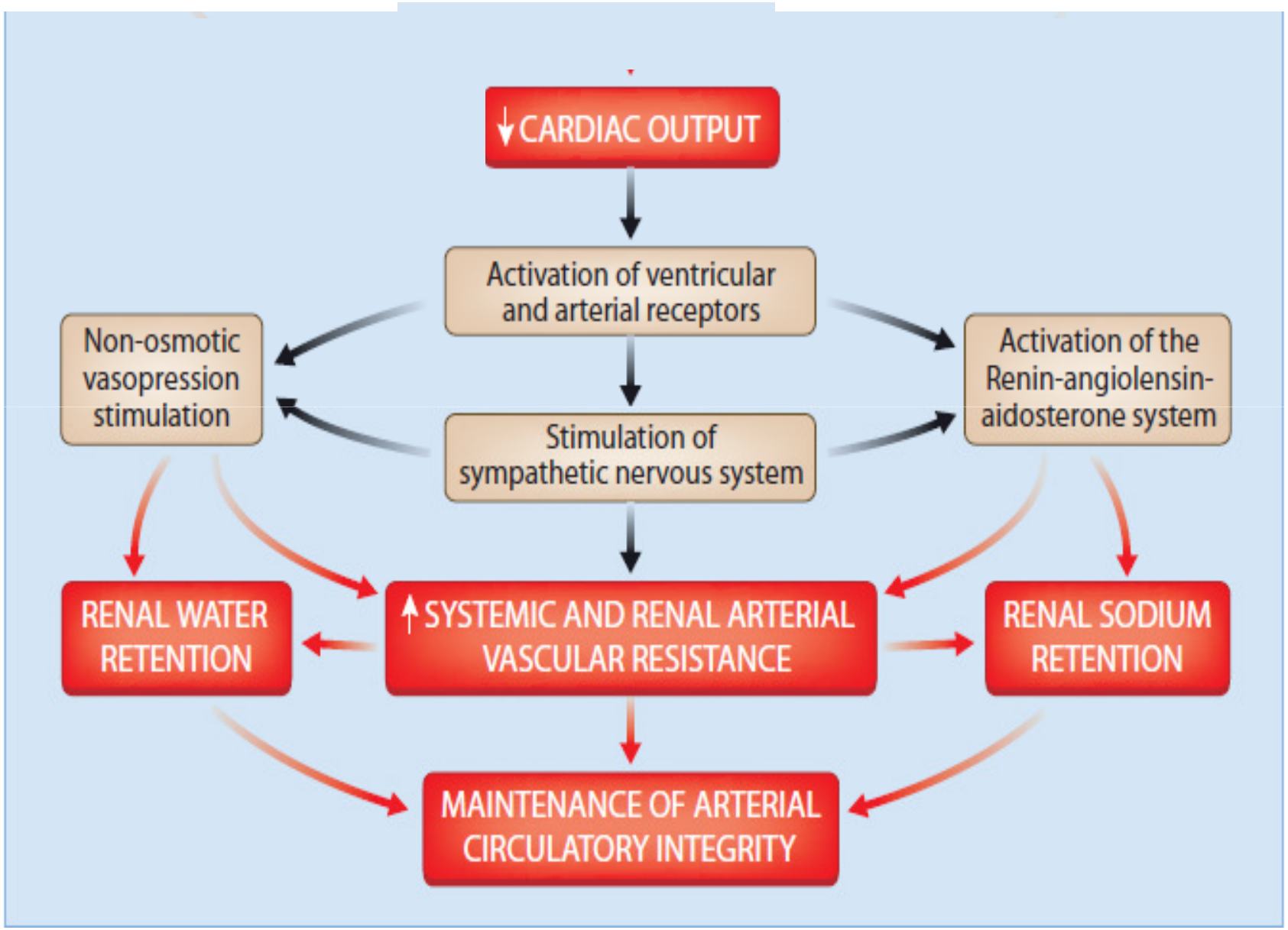
- Syndrome of inappropriate antidiuretic hormone secretion (SIADH)
- Diuretic use
- Glucocorticoid deficiency
- Hypothyroidism
- psychogenic polydipsia

HYPONATRAEMIA

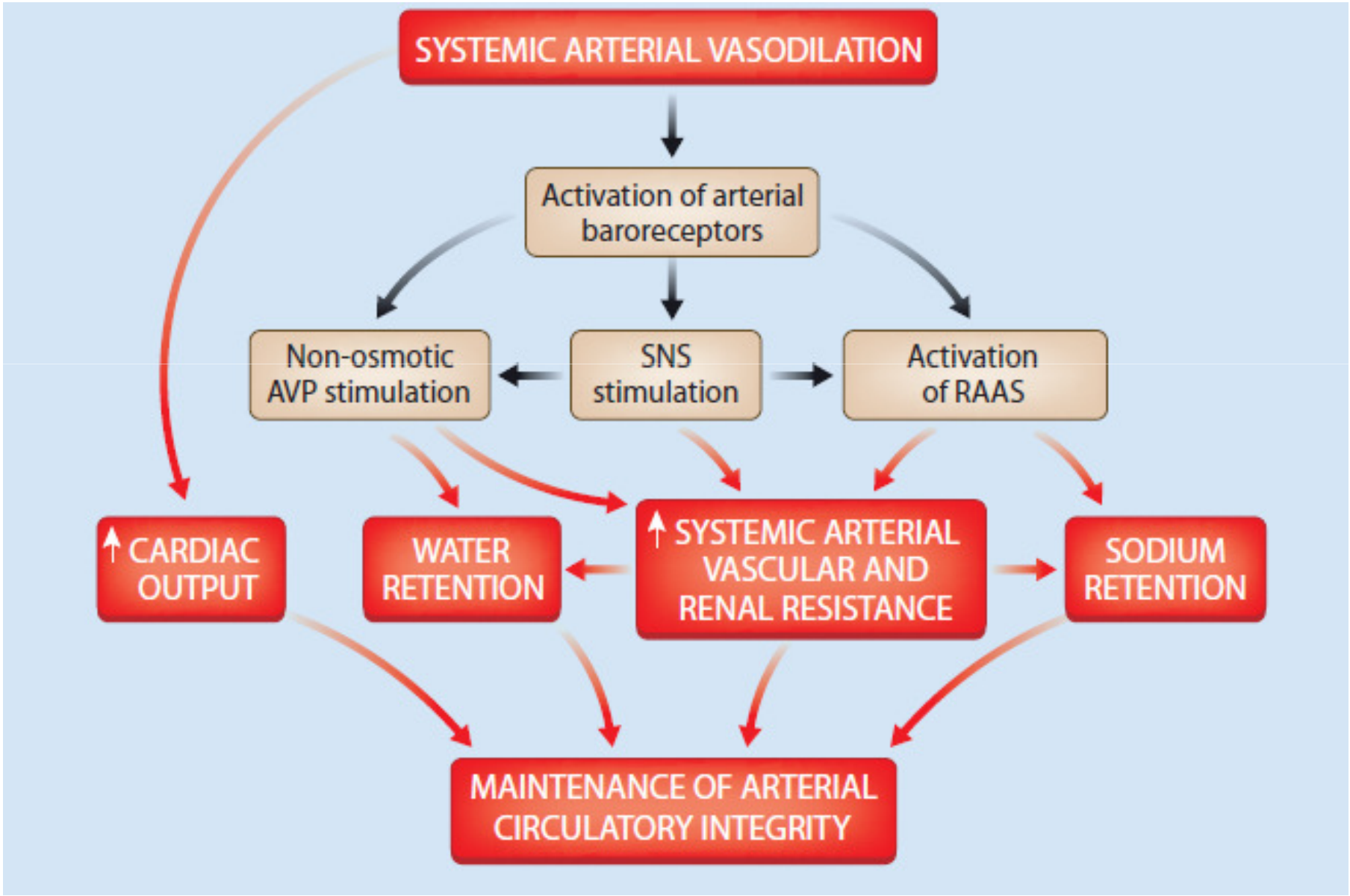
Hypervolemic with decreased effective circulating blood volume

- Decompensated heart failure
- Advanced liver cirrhosis
- Renal failure

CARDIAC FAILURE



CIRRHOSIS


SYSTEMIC ARTERIAL VASODILATION
Activation of arterial baroreceptors
Non-osmotic AVP stimulation
SNS stimulation
Activation of RAAS
↑ CARDIAC OUTPUT
WATER RETENTION
↑ SYSTEMIC ARTERIAL VASCULAR AND RENAL RESISTANCE
SODIUM RETENTION
MAINTENANCE OF ARTERIAL CIRCULATORY INTEGRITY

CLINICAL FEATURES

- CNS symptoms – Sodium <125 mEq/L
  - Disorientation
  - Restlessness and agitation
  - Apathy
  - Psychosis
  - Seizures
- Others – nausea,vomiting, headache, muscle cramps

COMPLICATIONS OF SEVERE HYPONATRAEMIA

- Due to hyponatraemia induced cerebral oedema,
  I. Respiratory arrest
  II. Coma
  III. Brainstem herniation
  IV. Death

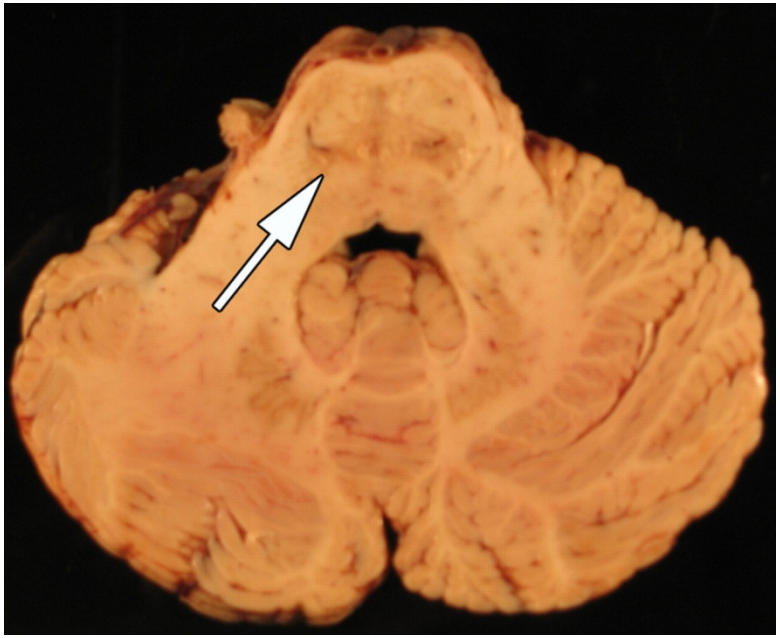
TREATMENT

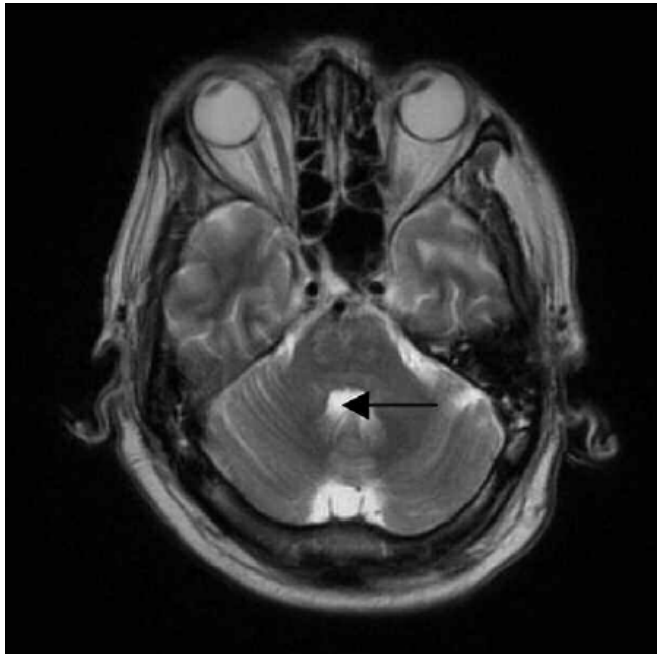
- Rapid correction of hyponatraemia can cause **OSMOTIC DEMYELINATION**

- Goal - to raise the serum sodium level by 1.5 to 2 mEq/L/hour (<= 12 mEq/L for 24 hours)

OSMOTIC DEMYELINATION

- Characterized by confusion, quadriplegia, pseudobulbar palsy, and ‘locked-in syndrome’





TREATMENT OPTIONS

- Hypertonic saline (3%)
- Loop diuretics e.g. Frusemide
- For hypervolemic and euvolemic hyponatremia:
  - Fluid restriction
  - Demeclocycline
  - AVP antagonists e.g. Conivaptan

TREATMENT OPTIONS

- **Demeclocycline:**

Inhibits AVP action at the distal renal tubules

Render AVP ineffective even in the presence of increased AVP levels

Loss of free water in urine

TREATMENT OPTIONS

- **Arginine vasopressin receptor antagonists :**

  Act as an antagonist of vasopressin receptors

  ↓

  Block the effect of vasopressin

  ↓

  Excretion of free water in urine

POTASSIUM BALANCE

- The ratio of ICF:ECF K^+ concentration ~38:1
- Maintained by basolateral Na^+, K^+-ATPase pump
- To maintain the steady state, K^+ ingestion should be matched with excretion
- K+ secretion at distal convoluted tubule and cortical ducts – main contributor to K^+ excretion

HYPERKALAEMIA

- Plasma K^+ concentration >5.0 mmol/L
- Chronic hyperkalemia - always due to decreased renal K^+ excretion
- Hyperkalemia partially depolarizes the cell membrane and prolonged depolarization impairs membrane excitability

CLINICAL FEATURES

Impaired cell membrane excitability causes,

- Nervous system :

Weakness and flaccid paralysis

- Heart :

ECG changes → ventricular fibrillation/asystole

HYPERKALAEMIA- CAUSES

- Renal failure
- Decreased K^+ secretion
  - Adrenal insufficiency, drugs (ACE inhibitors, NSAIDs, heparin)
- Resistance to aldosterone:
  - tubulointerstitial disease, drugs (K^+-sparing diuretics,)

TREATMENT

1. Discontinuing exogenous K^+ intake and drugs reducing K^+ excretion

   e.g. Angiotensin Converting Enzyme Inhibitors

2. Minimizing membrane depolarization and excitability
   - Calcium gluconate

TREATMENT

3. Shifting K^+ into cells
   - Insulin (with glucose to prevent hypoglycemia)
   - $NaHCO_3$
   - β_2-adrenergic agonists e.g. Salbutamol

4. Promoting K^+ loss
   - Diuretics
   - Cation-exchange resin e.g. Sodium polystyrene sulfonate
   - Dialysis

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