Hyperkalaemia

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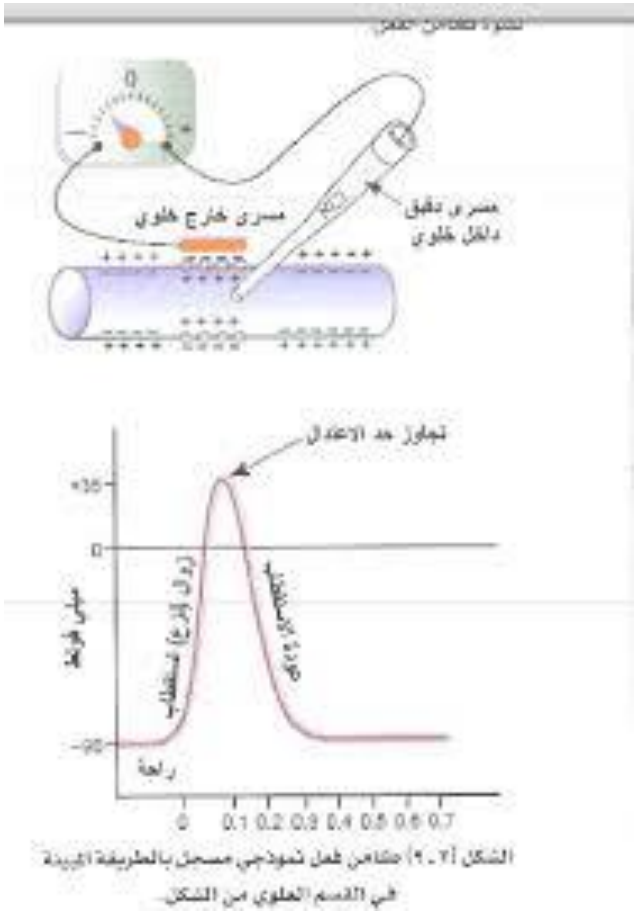
introduction

Potassium disorders are commonly encountered in clinical practice.

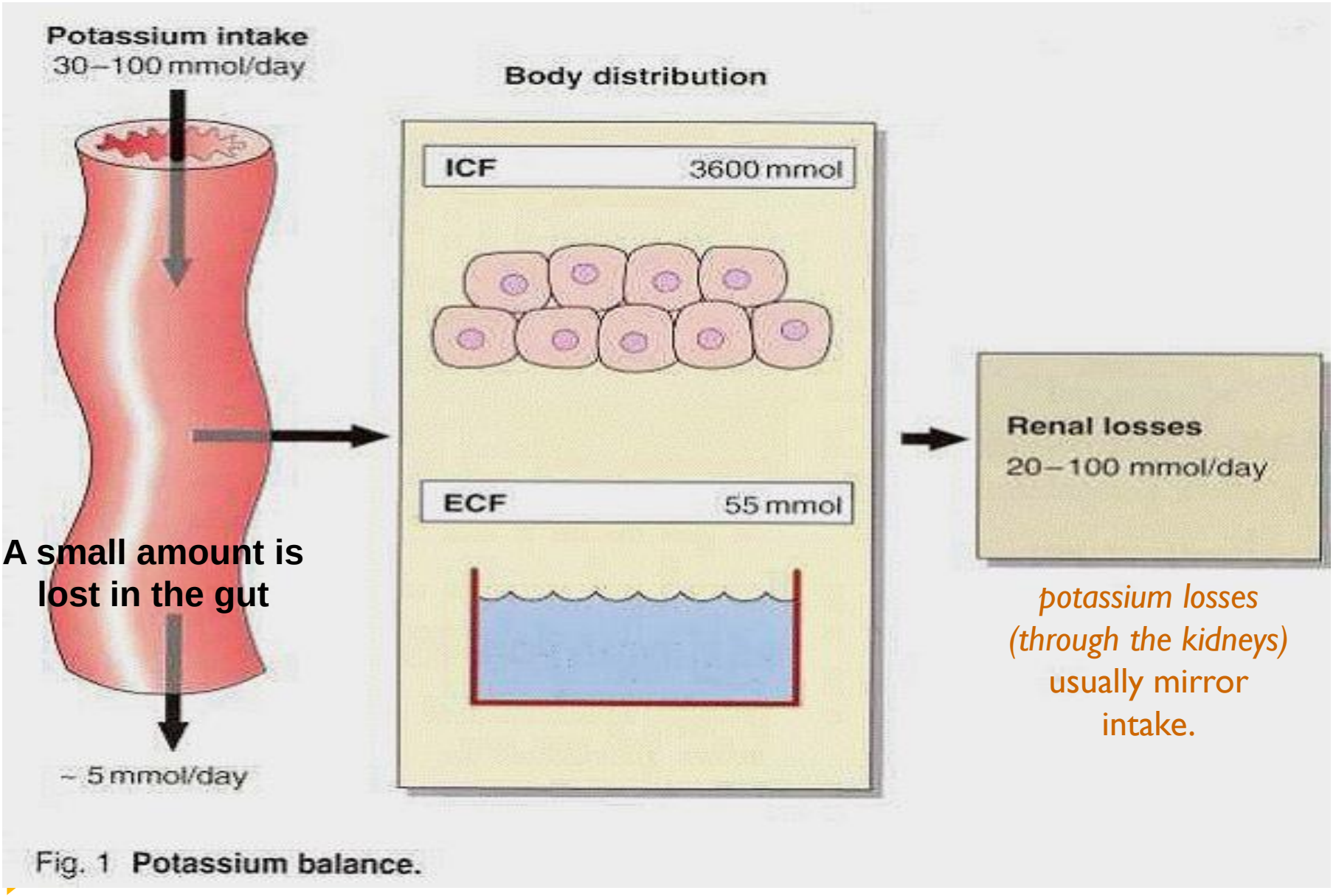
They are important because:

- of the role potassium plays in determining the resting membrane potential of cells.
- Changes in plasma potassium mean that 'excitable' cells, such as nerve and muscle, may respond differently to stimuli.
- In the heart (which is largely muscle and nerve), the consequences can be fatal,

e.g. arrhythmias.



Serum potassium and potassium balance



potassium losses (through the kidneys) usually mirror intake.

Fig. 1 **Potassium balance.**

Serum potassium and potassium balance

❖Serum potassium concentration is normally kept within a tight range (3.5–5.3 mmol/L).

❖The two most important factors that determine potassium excretion are:

✓The glomerular filtration rate.

✓ And the plasma potassium concentration.

✓An additional factor often implicated in hyperkalaemia and hypokalaemia is redistribution of potassium. Nearly all of the total body potassium (98%) is inside

✓Cells. If, for example, there is significanttissue damage, the contents of cells,

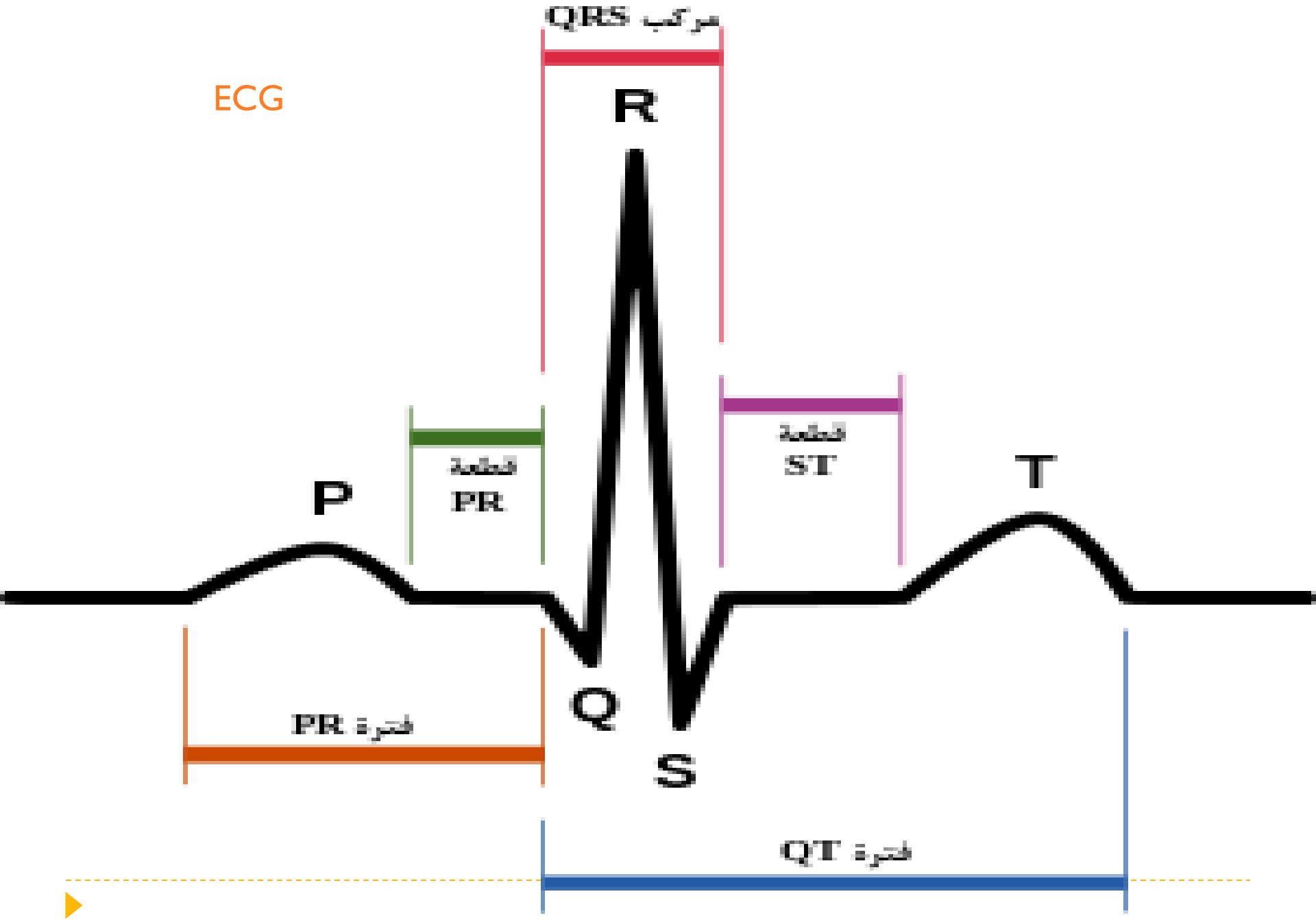
✓Including potassium, leak out into the extracellular compartment, causing Potentially dangerous increases in serum potassium .

Hyperkalaemia

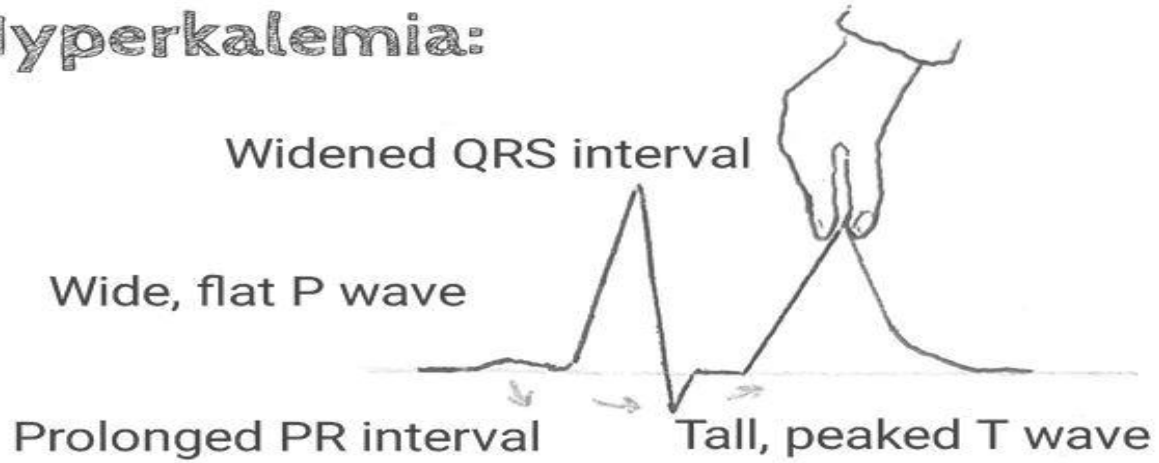
Hyperkalaemia is one of the commonest electrolyte emergencies encountered in clinical practice. If severe (>7.0 mmol/L). it is immediately life-threatening and must be dealt with as an absolute priority.

- Cardiac arrest may be the first manifestation. ECG changes seen in hyperkalaemia . include the classic tall ‘tented’ T-waves and widening of the QRS complex, reflecting altered myocardial contractility.
- Other symptoms include muscle weakness and paraesthesiae, again reflecting involvement of nerves and muscles

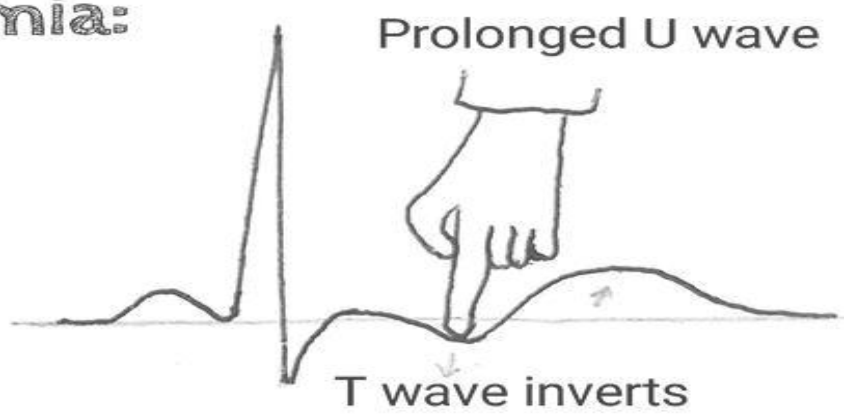
ECG



Hyperkalemia:



Hypokalemia:



" Pull and Push effects"

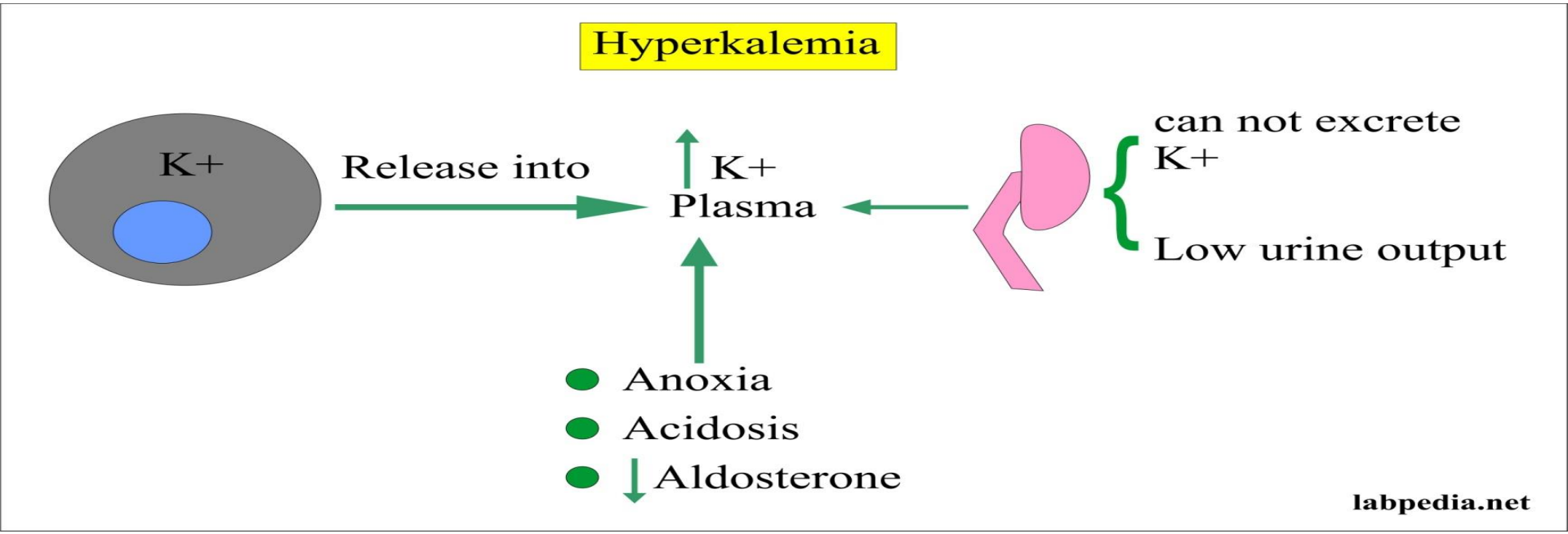
DR.ARAZOU

of Potassium on T wave of ECG

Hyperkalaemia causes

Hyperkalaemia can be categorized as due to :

1. Decreased excretion
2. Redistribution
3. Increased intake,



1.Decreased excretion

In practice, virtually all patients with hyperkalaemia will have a reduced GFR.

❖***Renal failure.*** **The kidneys may not be able to excrete a potassium load when the glomerular filtration rate is very low.**

❖and hyperkalaemia is a central feature of reduced glomerular function.

❖ It is exacerbated by the associated metabolic acidosis, due to the accumulation of organic ions that would normally be excreted.

1.Decreased excretion

❖**Hypoaldosteronism.**

❖*Aldosterone* stimulates sodium reabsorption in the renal tubules at the expense of potassium and hydrogen .

❖Deficiency, antagonism or resistance results in loss of sodium, causing a decreased GFR with associated retention of potassium and hydrogen ions.

❖In clinical practice, hyperkalaemia due to hypoaldosteronism is most often seen with the use of angiotensinconverting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) to treat hypertension.

❖spironolactone and other potassium sparing diuretics also antagonize the effect of aldosterone.

Less frequently,adrenal insufficiency is responsible.

2.Redistribution out of cells

❖Potassium release from damaged cells.

The potassium concentration inside cells (~140 mmol/L) means that cell damage can give rise to marked hyperkalaemia.

This occurs in **rhabdomyolysis** (where skeletal muscle is broken down), **extensive trauma,** or **rarely tumour lysis syndrome**, where malignant cells break down.

- Reciprocal relationship between K and H ions. to maintain electrochemical neutrality.

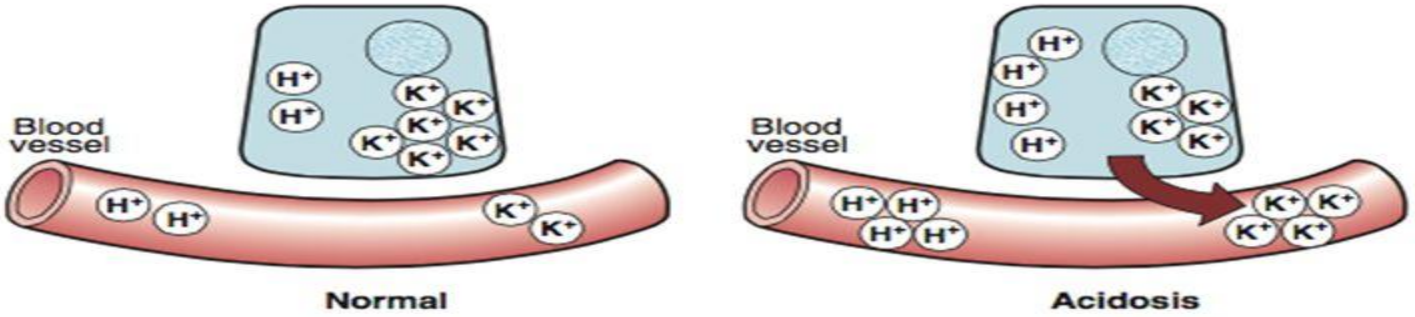
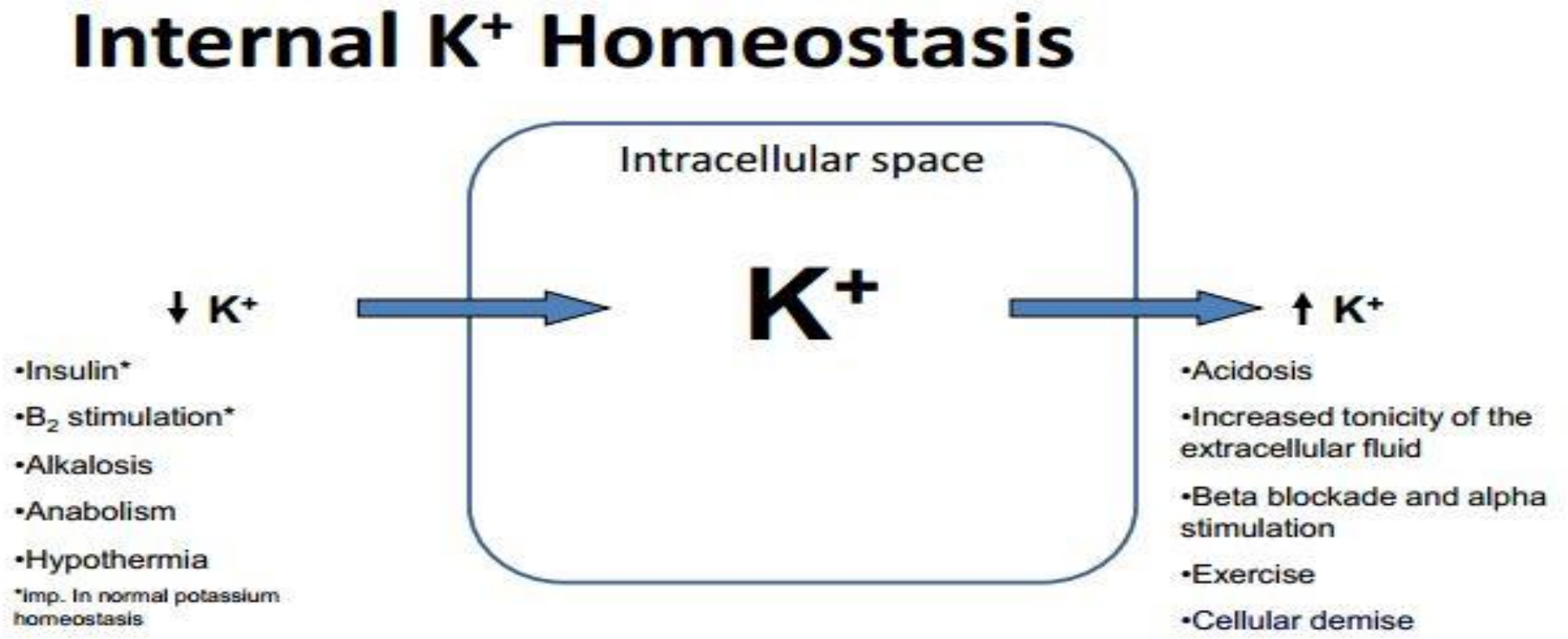


Fig 11.3 **Hyperkalaemia is associated with acidosis.**

❖**Metabolic acidosis**. There is a reciprocal relationship between potassium and hydrogen ions. As the concentration of hydrogen ions increases with the development of metabolic acidosis, so potassium ions inside cells are displaced from the cell by hydrogen ions in order to maintain electrochemical neutrality . These hydrogen ion changes cause marked alterations in serum potassium.

❖**Insulin deficiency.** **Insulin** stimulates cellular uptake of potassium, and plays a central role in treatment of severe hyperkalaemia. Where there is insulin deficiency or severe resistance to the actions of insulin, as in diabetic ketoacidosis ,hyperkalaemia is an associated feature.



❖**Pseudohyperkalaemia**. This should be considered when the cause of hyperkalaemia is not readily apparent. Indeed, it is important largely because it can lead to diagnostic dilemmas.

The commonest causes are:

(1) Delay in centrifugation separating plasma/serum from the cells/clot, especially if the specimen is chilled. This is very common in specimens from primary care.

(2) In-vitro haemolysis.

(3) An increase in the platelet and / or white cell count.

❖***Hyperkalaemic periodic paralysis***. This is a rare familial disorder with autosomal dominant inheritance. It presents typically as recurrent attacks of muscle weakness or paralysis, often precipitated by rest after exercise.

3.Increased intake

Failure to appreciate sources of potassium intake may result in dangerous hyperkalaemia, particularly in patients with impaired renal function.

❖For example, many oral drugs are administered as potassium salts. Potassium may also be given intravenously. *Intravenous potassium should not be given faster than 20 mmol/hour except in extreme cases.*

❖Occasionally, blood products may give rise to hyperkalaemia (stored red blood cells release potassium down its concentration gradient). The risk of this is reduced by using relatively fresh blood(less than 5 days old) and/or by ‘washing’ units prior to transfusing.

Hypokalaemia

Diagnosis

- Reduced intake
- Redistribution into cells
- Increased losses

causes Hypokalaemia

Reduced intake:

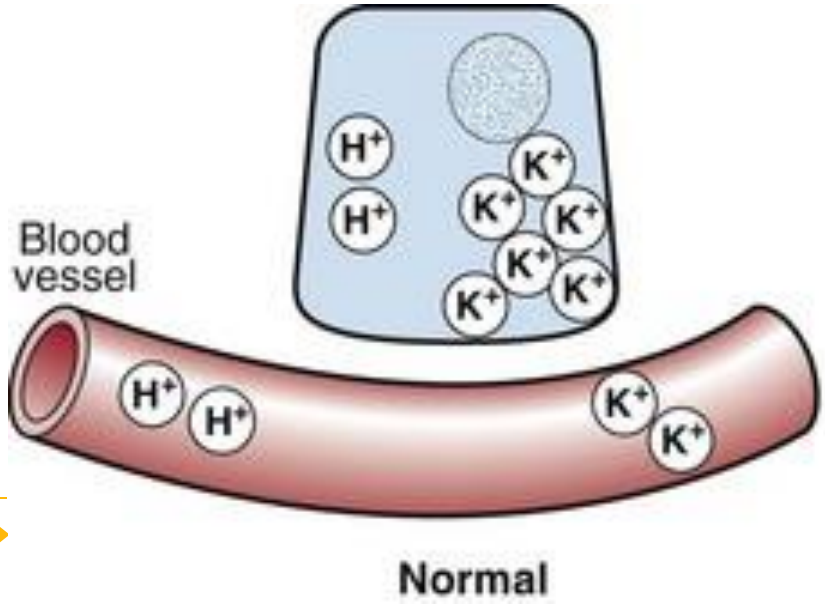
- This is a rare cause of hypokalaemia.

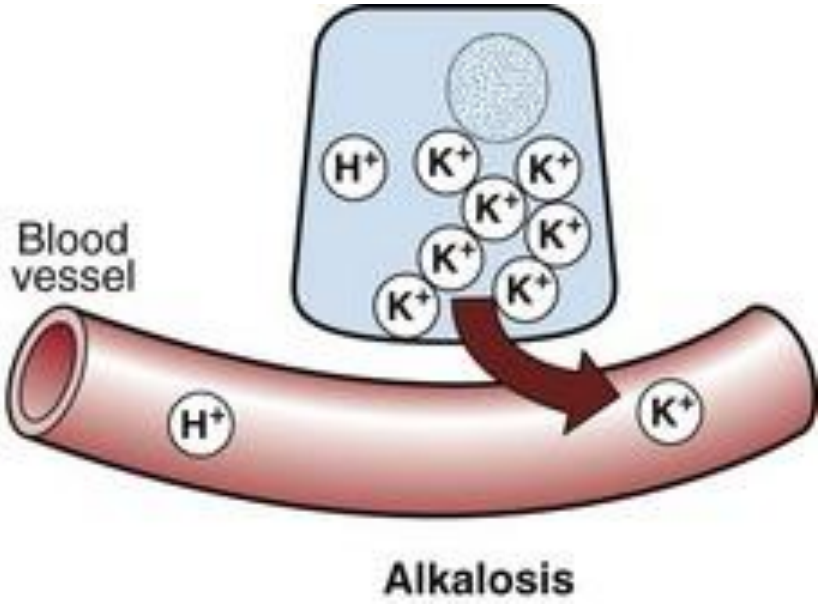
Renal retention of potassium in response to reduced intake ensures that hypokalaemia occurs only when intake is severely restricted. Since potassium is present in meat, fruit and some vegetables, marked potassium restriction is difficult to maintain.

- Hypokalaemia should, however, be a consideration when severely hypocaloric diets are prescribed to bring about rapid weight loss.

Redistribution into cells

- *Metabolic alkalosis. The reciprocal* relationship between potassium and hydrogen ions means that in just the same way as metabolic acidosis is associated with hyperkalaemia, so metabolic alkalosis is associated with hypokalaemia. As the concentration of hydrogen ions decreases, so potassium ions move inside cells in order to maintain electrochemical neutrality .





Redistribution into cells

- *Treatment with insulin. Insulin* stimulates cellular uptake of potassium, and plays a central role in treatment of severe hyperkalaemia . It should come as no surprise therefore that when insulin is given in the treatment of diabetic ketoacidosis , there is a risk of hypokalaemia. This is well recognized, and virtually all treatment protocols for diabetic ketoacidosis take this into account

- *Refeeding. The so-called 'refeeding* syndrome' was first described in prisoners of war. It occurs when previously malnourished patients are fed with high carbohydrate loads The result is a rapid fall in phosphate, magnesium and potassium, mediated by insulin as it moves glucose into cells.

❑ *β-Agonism. Acute physiological stress* can cause potassium to move into cells, an effect mediated by **catecholamines** through their actions on **β2-receptors**. β-agonists like salbutamol (used to treat asthma) or dobutamine (heart failure) predictably induce a similar effect.

❑ *Treatment of anaemia. Folic acid or* vitamin B12 for megaloblastic anaemia often produce hypokalaemia in the first couple of days of treatment, due to the uptake of potassium by the new blood cells.

Treatment of iron deficiency anaemia results in a much slower rate of new blood cell production and is therefore rarely implicated.

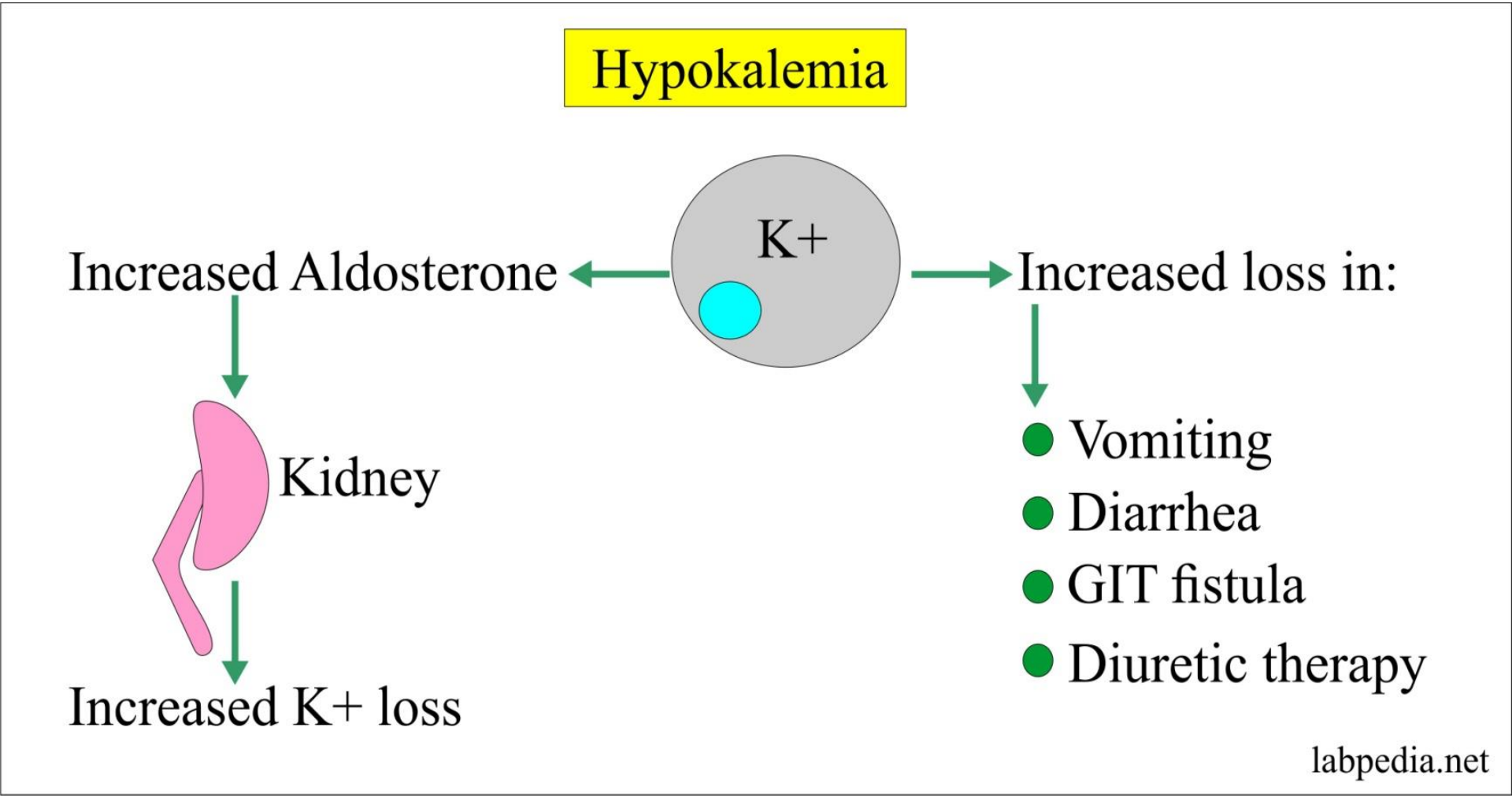
❑ ***Hypokalaemic periodic paralysis.***

❑ hypokalaemic periodic paralysis can be inherited (as an autosomal dominant trait),

❑and be precipitated by rest after exercise.

❑However, it can also be acquired as a result of thyrotoxicosis

Increased losses



Increased losses

Gastrointestinal:

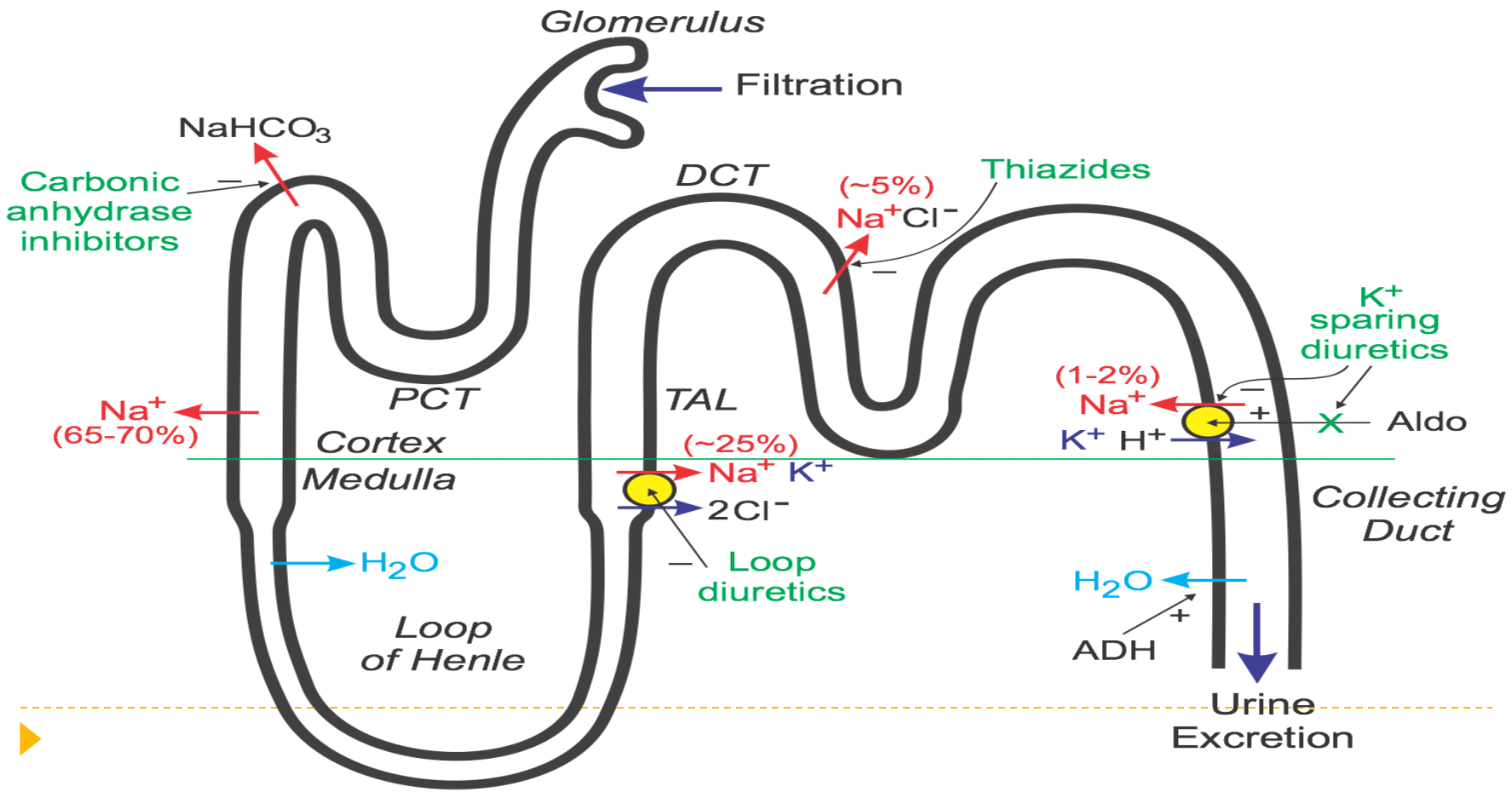
❖The common causes (diarrhoea and vomiting) are obvious, and the risk of hypokalaemia well recognized.

❖In cholera (associated with massive fluid loss from the gut), daily potassium losses may exceed 100 mmol, compared with ~5 mmol normally.

Less frequently, chronic laxative abuse may be responsible. However, this should normally be considered only when more likely causes of hypokalaemia have been excluded.

Urinary

- *Diuretics.*
- *Both loop diuretics and* thiazide diuretics produce hypokalaemia. .



- *Mineralocorticoid excess.*

- aldosterone increases sodium reabsorption in the renal tubules at the expense of potassium and hydrogen ions.

- This *mineralocorticoid* effect is shared by many steroid molecules, and hypokalaemia is a predictable and frequent consequence of mineralocorticoid excess.

- Overproduction of steroid hormones .

- Less frequently, **renal artery stenosis** drives the renin–angiotensin–aldosterone axis resulting in hypokalaemia associated with severe, refractory hypertension.

Hypomagnesaemia.

Hypomagnesaemia from any cause may lead to hypokalaemia due to impaired renal tubular absorption.

Tubulopathies.

- *The most common* causes of the tubulopathies are chemotherapeutic agents, especially platinum containing drugs.
- A small number of inherited defects in tubular function produce hypokalaemia by various mechanisms. They may need to be considered in cases of persistent unexplained hypokalaemia