Hyperkalaemia is a common electrolyte abnormality that can result in life-threatening arrhythmias and is associated with an increased risk of mortality. The strongest risk factor for the development of hyperkalaemia is CKD, the effects of which can be exacerbated by chronic conditions such as DM, obesity, and CKD itself. Medications such as ARBs, ACEi, beta-adrenoreceptor blockers (β-blockers), and aldosterone antagonists (Aldactone, spironolactone) can contribute to hyperkalaemia. The emergence of new 
K-lowering medications has led to renewed interest in pursuing therapeutic strategies that can further optimize the treatment of hyperkalaemia in patients who are already on medication. This approach is feasible in patients with CHF, and can be initiated in the acute setting but are sometimes discontinued at >6 mmol/l.

## Therapy

The pharmacologic effects of hyperkalaemia depend not only on serum K level but also on other factors such as the presence of acidosis or alkalosis, volume depletion, and the presence of organ dysfunction such as liver failure. Hyperkalaemia represents a medical emergency requiring immediate intervention to prevent cardiac arrest and death. The lack of evidence for stabilization membrane potential and lower serum K level. Acute interventions include intravenous or hyperosmotic saline, intravenous naloxone, and haemodialysis.

## Chronic interventions

Long-term interventions involve eliminating risk factors and administering therapies that facilitate K removal. Dietary K restriction (e.g. salt substitutes) is often administered to reduce K levels. The distribution of K between the intracellular and extracellular space is maintained by balancing the activity of the Na⁺/K⁺-ATPase on cell membranes. Effectors of K uptake and leak include insulin, catecholamines, mineralocorticoids, toxins, exercise and acid–base status.

## Aetiology and mechanisms

### Sources of K

Sources of K include K-rich foods; K supplements (often prescribed with diuretics); salt substitutes; GI intake causes hyperkalaemia under conditions of impaired renal excretion and/or cellular redistribution. Most K is absorbed in the small intestine and absorption increases in proportion to intake.

### Cellular distribution

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### Urinary K excretion

This has a crucial role in maintaining K homoeostasis. Healthy kidneys possess a tremendous ability to dispose of excess K, maintaining normal serum K levels even with strokes as high as 450 mmol/l per day. Most of the filtered K is reabsorbed in the proximal convoluted tubule and the loss of filtered K is largely determined by K-secretion occurring in the distal nephron and collecting duct.

### Drugs and therapy

### Cardiovascular consequences

Hyperkalaemia is generally well tolerated by most cardiac systems, although it can lead to the development of electrical conduction defects by decreasing the resting membrane potential, leading to increased cardiac depolarization. The effects of hyperkalaemia on cardiac electrical excitability, conduction, action potential amplitudes, and arrhythmias can vary from patient to patient. However, patients with cardiac disease can be affected by the increased risk of arrhythmia and sudden death.

### Cellular distribution

Most K is found in muscle cells. The physiologic effects of K (e.g. on membrane potential) depend on a normal serum concentration. A decrease in serum K is associated with a decrease in K excretion by the kidney and an increase in K excretion by muscle and liver cells. Conversely, a decrease in serum K is associated with increased renal and liver excretion. Therapy

The physiologic effects of hyperkalaemia depend not only on serum K level but also on other factors such as the presence of acidosis or alkalosis, volume depletion, and the presence of organ dysfunction such as liver failure. Hyperkalaemia represents a medical emergency requiring immediate intervention to prevent cardiac arrest and death.

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