

Various Diuretics and its Mechanism of Action

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DESCRIPTION

The main objective of diuretic therapy is to increase the amount of water expelled in the urine. Different types of diuretics have different mechanisms of action. Through altering how the kidney regulates sodium, this is accomplished. Water excretion will rise if the kidney excretes more sodium. The majority of diuretics cause dilation by preventing salt from being reabsorbed at various renal tubular system sections. When two diuretics are administered simultaneously, it can be much more effective than either drug alone (synergistic effect). As a result, blocking numerous nephron locations considerably increases effectiveness. This occurs because one nephron segment can make up for decreased sodium reabsorption at another nephron segment. Below is a summary of some of the mechanisms of the various diuretics.

Loop diuretics

Inhibit the thick ascending limb's sodium-potassium-chloride cotransporter. Inhibition of this pump can cause a significant rise in the distal tubular sodium concentration, a decrease in the interstitial hypertonicity, and a reduction in the amount of water reabsorption in the collecting duct because this transporter typically reabsorbs about 25% of the sodium load. Natriuresis (increased water loss) and diuresis (increased sodium loss) are caused by this changed management of sodium and water (increased sodium loss). Loop diuretics are extremely potent diuretics because they work by influencing the thick ascending limb, which is responsible for a large portion of sodium reabsorption. In addition to increasing renal blood flow and redistributing renal cortical blood flow, these drugs also stimulate the production of prostaglandins in the kidneys. Due to a very high capacity for sodium reabsorption at their site of action, loop diuretics are the most effective diuretic class. Consider the fact that effectiveness is inversely correlated with renal function, which can be severely compromised in heart failure. Furosemide is one type of loop diuretic.

Carbonic anhydrase inhibitors

These medications reduce the reabsorption of bicarbonate in the proximal tubule by suppressing the enzyme carbonic anhydrase. As a result, less sodium is absorbed and potassium is retained in the urine. Reduced sodium absorption results in less water being reabsorbed.

Thiazide diuretics

These drugs function by preventing NaCl reabsorption in the kidney's distal convoluted tubule. The sodium chloride cotransporter is suppressed, which mediates this activity. These medications are mostly used to manage kidney stones, nephrogenic diabetes insipidus, heart failure, hypertension (high blood pressure), and kidney failure. The medication hydrochlorothiazide is an illustration of this class.

Osmotic diuretics

Osmotic diuretics raise the osmolarity of the blood and the renal filtrate by inhibiting the reabsorption of sodium and water. Examples of these substances are mannitol and isosorbide. Lowering of intracranial pressure or cranial pressure. Haemorrhagic renal failure therapy. Administering medications directly to the brain.

Potassium-sparing diuretics

Some substances boost the diuresis without depleting the body of potassium. Spironolactone is one popular type of potassium-saving diuretic. This medication inhibits salt and water retention by inhibiting the entry of aldosterone into the main cells of the collecting duct and late distal tubule of the nephron. Amiloride and triamterine, epithelial sodium channel inhibitors, are more examples of potassium-saving diuretics. These directly inhibit the apical membrane of the collecting tubule's epithelial sodium channels, which are sodium channels.

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