20% of cardiac output goes to the kidney.
99% of filtered blood(serum) (which is 170L a day) in the glomerulus is reabsorbed at the tubule.
The rest of 1% is secreted as the urine.
Blood osmolality is controlled by Renin-Angiotensin-Aldosterone system(RAAS) & ADH (vasopressin)
RAAS is activated through sympathetic nerve stimuli by brain(hypothalamus?) which accepts the signal from baroreceptor on arteries.
Posterior pituitary gland incessantly monitors osmolality of the blood and control the secretion of vasopressin which increases the permeability of the distal tubules and collecting ducts to water. 2/3 of reabsorption of glucose and electrolytes (mainly natrium) is done at the proximal tubule. In a condition of the cardiac failure, the proximal tubules get into hypoxia because of narrowed efferent renal arterioles and enhanced consumption of oxygen for Na reabsorption.
There are 2 factors that make the proximal tubule hypoxia. One is enhanced oxygen consumption for Na reabsorption ordered by augmented RAAS. The other is the decreased oxygen supply to the area, because RAS system shrinks the afferent renal artery which supply oxygen to the proximal tubule.
SGLT-inhibitor suppress sympathetic nerve. So, pt. administered SGLT-2 inhibitor shows decreased heart rate & lower nighttime blood pressure.
SGLT drives reabsorption of Na & glucose at the proximal tubule using energy from fatty acid. Once the area becomes under hypoxia due to augmented RAS system and hyperglycemia, it uses glucose instead of fatty acid.
Then untrained glycolysis system must do the unaccustomed work, which leads to fibrosis of the tubule.
SGLT2-inhibitor stops this fibrosis, so the erythropoietin producing cells can work well, Therefore SGLT-2 inhibitor elevates hematocrit of the patients.

Summary: the effect of SGLT2-inhibitor
Blood glucose ↓、interstitial fluid ↓、HR ↓、nocturnal BP ↓、hematocrit ↑

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