Regulatory mechanisms of renal circulation

Like other organs, blood flow in the kidneys has its own regulatory mechanisms, which can be divided into local and central. They are used especially in changes in blood pressure, thus in maintaining constant glomerular filtration. In general, vasoconstriction reduces glomerular filtration, vasodilation increases.

Local regulation

Under physiological conditions, local regulations have a major role in maintaining a constant renal blood flow. The following mechanisms apply:

- **Myogenic autoregulation -** maintains a constant blood flow. With increased blood pressure, the tension on the capillary wall increases. As a result of stimulation of baroreceptors, vasoconstriction of the afferent arterioles occur. The blood flow through the vessel is thus normalized. In the opposite situation, reduced blood pressure causes vasodilation with a consequent increase in flow.
- **Tubuloglomerular feedback** - Macula densa cells produce signaling molecules to which the smooth muscle in the afferent arteriole responds and can detect the amount of flow in the tubular fluid. The feedback mechanism consists of the detection of reduced flow through macula densa cells (decrease in glomerular filtration) and in the production of signaling substances that act as paracrine vasodilators on vas afferent receptors . When increasing glomerular filtration, the principle is the opposite, the secreted substance is a molecule with a vasoconstrictive effect.

Local regulatory mechanisms can maintain constant glomerular filtration even with changes in blood pressure from 80 to 180 mm Hg.

Central regulation

This type of regulation is used especially in pathological conditions (e.g loss of a larger volume of fluids). With changes in arterial pressure, local regulatory mechanisms fail and central regulatory mechanisms with nervous and hormonal action apply:

- **The sympathetic nervous system** mainly acts on vas afferens and, by means of its α1 -adrenergic receptors, inducing constriction after the binding of adrenaline/noradrenaline
- **Angiotensin II** has receptors located in both the vas afferens and the vas efferens. It is involved in the stimulation of catecholamines from the adrenal medulla and from sympathetic endings. It acts contractile on mesangial cells and reduces glomerular filtration;
- **Prostaglandins** are not used in physiological situations. In pathological conditions, they are produced locally in the kidneys and their vasodilatory effect on vas afferens and vas efferens seeks to prevent ischemia, which is threatened by the action of catecholamines and angiotensin II, whose increased activity is a stimulus for prostaglandin flushing .

Other substances that potentiate blood flow in the glomeruli include: atrial natriuretic peptide, glucocorticoids , nitric oxide, kinins . Other vasoconstrictors are e.g ADH, ATP and endothelin.

References

SILBERNAGL, Stefan and Agamemnon DESPOPOULOS. Atlas of human physiology. 6th edition. Prague: Grada, 2003. 435 pp. 150. ISBN 80-247-0630-X

KITTNAR, Otomar, et al. Medical physiology. 1st edition. Prague: Grada, 2011. 790 pp. ISBN 978-80-247-3068-4 .