

Blood flow in the kidneys

Kidneys are the main component of the excretory system. The arrangement of blood vessels is key to its proper function.

Vascular arrangement in the kidneys

Approximately 1.2-1.3 liters of blood flow into the kidneys per minute (25% of resting minute cardiac output)^[1] from *a. renalis*, which for individual segments is divided into *aa. segmentales*. These further branch to *aa. interlobares* and on to *aa. arcuatae*. 90% flows through the cortex, 10% of the total volume through the kidney medulla. The *Vas afferens* opens into a capillary ball in the renal glomerulus, where the ultrafiltrate of the blood plasma is formed. Filtration is the result of the Starling forces acting on the vessel wall (see glomerular filtration). Renal circulation is characterized by a system of two capillary networks – glomerular and peritubular. Glomerular is high pressure (60 mm Hg^[2]), which is due to the relative proximity of the aorta. The peritubular network is low pressure (8 mm Hg)^[1] and follows the glomerular network. Here, it is necessary to point out the presence of the "vasa recta" located along the loop of Henle, whose function is to transport resorbed substances back into the circulation. Veins are called the same as arteries in the rest of the circulation.

Regulatory mechanisms of renal circulation

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

See *Glomerular Filtration* for more detailed information .

Local regulation

Under physiological circumstances, local regulation plays a major role in maintaining constant renal flow. In particular, the following mechanisms apply.

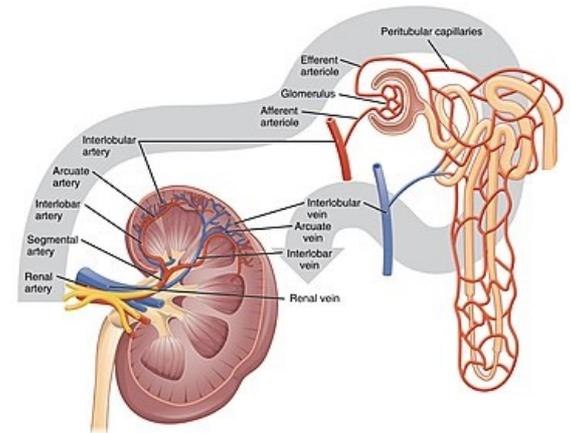
- **Myogenic autoregulation** – maintains constant blood flow. With increased blood pressure, the tension on the capillary wall increases. *Vas afferens* vasoconstriction occurs as a result of baroreceptor stimulation. The blood flow through the vessel is thus normalized. In the opposite situation, reduced blood pressure causes vasodilation with a subsequent increase in flow;
- **Tubuloglomerular feedback** – cells of the *macula densa* produce signaling molecules to which the smooth muscle in *the vas afferens* reacts and, thanks to their receptors, they can detect the amount of flow in the tubular fluid. The feedback mechanism consists in the detection of reduced flow through the cells of the macula densa (decrease in glomerular filtration) and in the production of signaling substances that have a paracrine vasodilatory effect on the receptors of *the vas afferens*. When glomerular filtration increases, 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.^[3]

Central regulation

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

- **Sympathetic** mainly acts on *the vas afferens* and with the help of its $\alpha 1$ -adrenergic receptors causes 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 medulla of the adrenal glands as well as from the sympathetic endings. 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 with their vasodilating effect on *the vas afferens* and *the vas efferens*, they try to prevent ischemia, which is threatened by the action of catecholamines and angiotensin II, whose increased activity is a stimulus for the flushing out of prostaglandins.



Other substances that potentiate blood flow in the glomeruli include: atrial natriuretic peptide, glucocorticoids , nitric oxide, kinins. Other vasoconstrictor substances are, for example: antidiuretic hormone, ATP, endothelin.

Links

External links

- Blood circulation FBLT (<http://fbt.cz/skripta/x-srdce-a-obeh-krve/2-krevni-obeh/>)
- Blood circulation in the kidneys (VIDEO) - English (<http://anatomyzone.com/tutorials/renal-circulation/>)
- Excretory system FBLT (<http://fbt.cz/skripta/vii-vylucovaci-soustava-a-acidobazicka-rovnovaha/>)

References

1. GANONG, William F. *Přehled lékařské fyziologie*. 20. edition. Galén, 2005. 890 pp. pp. 709. ISBN 80-7262-311-7.
2. KITTNAR, Otomar. *Lékařská fyziologie*. 1. edition. Grada, 2011. 790 pp. pp. 390. ISBN 978-80-247-3068-4.
3. SILBERNAGL, Stefan and Agamemnon DESPOPOULOS. *Atlas of Human Physiology*. 6th edition. Prague: Grada, 2003. 435 pp. 150. ISBN 80-247-0630-X .

Resources

- KITTNAR, Otomar. *Medical Physiology*. 1st ed. Prague: Grada, 2011. ISBN 978-80-247-3068-4.
- TROJAN, Stanislav. *Medical Physiology*. 4th ed. trans. and add. Prague: Grada Publishing, 2003, 771 pp. ISBN 80-247-0512-5.