Calcium channel blockers

Calcium channel blockers (calcium channel blockers) reduce the flow of calcium across the membrane into the cell by blocking the L-type calcium channel and thus causing **long-term smooth muscle dilation**. The most sensitive is the vascular muscle, especially the arterial muscle, the release of which leads to a reduction in vascular resistance. Dilation also occurs in the extravascular smooth muscle of the bronchi, GIT and uterus. In the heart are calcium-dependent "**slow response structures**" (SA and AV node), but also **contractile mechanism**s. By blocking the calcium channel, the **conductivity and inotropy of the myocardium decrease** - **the cardiac output decreases**. They dilate the coronary arteries and prevent further manifestations of ischemia. By affecting the SA and AV nodes, they suppress supraventricular tachyarrhythmias. Dilation in the systemic circulation causes a decrease in blood pressure and may lead to a reflex increase in heart rate, especially with short-acting nifedipine.

They are used therapeutically to treat angina pectoris, hypertension and arrhythmias.

Due to the fact that these drugs are significantly different in their physical and chemical properties, **their affinity for receptors of the target structure also differs.**

Drug classes

1. **First generation Ca^{2+} channel blockers = non-dihydropyridines:** nifedipine (chemically belongs to dihydropyridines, just like nicardipine), verapamil, diltiazem
2. **Second generation Ca^{2+} channel blockers = dihydropyridines:** isradipine, felodipine, nitrendipine, nisoldipine. These are dihydropyridine derivatives which, compared to nifedipine, show higher vascular selectivity (more distinct vasodilation with lower negative chronotropic and dromotropic effect) and more advantageous pharmacokinetic properties - longer action.
3. **Third generation Ca^{2+} channel blockers = dihydropyridines:** amlodipine, lacidipine, barnidipine. They are highly selective for blood vessels and have a slow onset of action, there is no reflex activation of the sympathetic nervous system. It has a long-term and antioxidant effect.

Drugs

Nifedipine

According to the chemical structure, they belong to the dihydropyridines. It mainly affects **the smooth vascular muscle**. It is therefore indicated in the therapy of **hypertension**. It is the drug of choice in the introduction to the therapy of hypertensive crisis. After oral administration, the effect appears as early as 10 minutes, with a maximum after 20-30 minutes. It is usually given in 3 daily doses. Contraindication to nifedipine is pregnancy (relaxing effect on the uterus).

Verapamil a diltiazem

It acts mainly on receptors in the myocardium. They prolong the AV conduction of excitation and slow down the sinus rhythm. They suppress supraventricular tachycardia and the ventricular response to a decrease in BP. Both are also considered suitable drugs for the prevention of cardiac ischemia, but in the acute phase of myocardial infarction they have not proved successful, probably also for the simultaneous negative inotropic effect. They can also be used as antihypertensives. Verapamil may induce AV conduction block at higher doses and is contraindicated in conductive disorders of the heart and administration of beta-blockers at a same time.

Amlodipine

In patients with heart failure, it reduces mortality, does not affect carbohydrate and lipid plasma concentrations and is administered once daily.

Comparison of pharmacodynamics of first generation Ca blockers[1]			
	NIFEDIPINE*	**DILTIAZEM**	**VERAPAMIL**
dilation of coronary arteries	++	++	++
dilation of peripheral arteries	++++	++	+++
negative inotropic effect	+	++	+++
slowing the AV conduction	no significant change	+ + +	+ + + +
heart rate	increased, no significant change	decreased, no significant change	decreased, no significant change
decrease of blood pressure	++++	++	+++
depression of SA node	no significant change	++	++
increase of cardiac output	++	no significant change	no significant change

* or others dihydripyridines

Links

Related articles

- Renin-angiotenzin-aldosteron system
- ACEI
- Angiotenzin II receptor blockers
- Hypertension
- Hypertension crisis

External links

- Blokátory kalciového kanála a EKG (TECHmED) (https://www.techmed.sk/blokatory-kalcioveho-kanala-intoxikacia/)

Source

- MARTÍNKOVÁ, Jiřina – MIČUDA, Stanislav – CERMANOVÁ, Jolana. *Vybrané kapitoly z klinické farmakologie pro bakalářské studium : Kardiovaskulární systém* [online]. ©2000. [cit. 2010-07-01]. <https://www.lfhk.cuni.cz/farmakol/predn/bak/kapitoly/prednasky/kardio-bak.ppt/>.
- LINCOVÁ, Dagmar – FARGHALI, Hassan, et al. *Základní a aplikovaná farmakologie.* 2. edition. Praha : Galén, 2007. 672 pp. ISBN 978-80-7262-373-0.

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1. MARTÍNKOVÁ, Jiřina – MIČUDA, Stanislav – CERMANOVÁ, Jolana. *Vybrané kapitoly z klinické farmakologie pro bakalářské studium : Kardiovaskulární systém* [online]. ©2000. [cit. 2010-07-02]. <https://www.lfhk.cuni.cz/farmakol/predn/bak/kapitoly/prednasky/kardio-bak.ppt/>.

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