



Editorial

Vasodilator Uses and Mode of Action

Loai Aljerf*

Researcher & Docent Damascus University/Faculty of Dental Medicine Syria

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Vasodilators are used to treat hypertension, heart failure and angina; however, some vasodilators are better suited than others for these indications. Some vasodilators that act primarily on resistance vessels (arterial dilators) are used for hypertension, and heart failure, and angina; however, reflex cardiac stimulation makes some arterial dilators unsuitable for angina. Venous dilators are very effective for angina, and sometimes used for heart failure, but are not used as primary therapy for hypertension. Most vasodilator drugs are mixed (or balanced) vasodilators in that they dilate both arteries and veins and therefore can have wide application in hypertension, heart failure and angina. Some vasodilators, because of their mechanism of action, also have other important actions that can in some cases enhance their therapeutic utility or provide some additional therapeutic benefit. For example, some calcium channel blockers not only dilate blood vessels, but also depress cardiac mechanical and electrical function, which can enhance their antihypertensive actions and confer additional therapeutic benefit such as blocking arrhythmias.

Classification:

1. Organic nitrates:

- (i) Nitroglycerin (glyceryl trinitrate)
- (ii) Amyl nitrite
- (iii) Isosorbide di-nitrate
- (iv) Erythryl tetra nitrate
- (v) Pentaerythritol tetra nitrate

2. Arterial Vasodilators:

- (i) Hydralazine
- (ii) Minoxidil
- (iii) Diazoxide

3. Arterial and venous vasodilators:

- (i) Sod. nitroprusside

- (ii) Prazosin
- (iii) Terazosin
- (iv) Doxazosin

Nitrates:

Nitrates relax vascular as well as all other smooth muscles by forcing an active free radical nitric oxide (NO). 'NO' binds with the guanylyl cyclase enzyme in the cytosol and activates it to produce cyclic GMP (cGMP), which activates a cGMP-dependent protein kinase resulting in a series of alterations in various enzymatic proteins.

This eventually leads to the dephosphorylation of myosin light chain (MLC). The result is relaxation of smooth muscle because phosphorylation of MLC regulates the maintenance of contractile state in smooth muscle. The pharmacological and biochemical effects of the nitro vasodilators appear to be identical to those of the endothelium derived relaxing factor (EDRF itself is nitric oxide).

Site of Action of Vasodilators

Different vasodilators may act principally on arteries, principally on veins, or on both arteries and veins. The ability of drugs to act selectively on blood vessels of different types reflects differences in the functional properties of the smooth muscle in the vessel wall. The smooth muscle of the arteriolar resistance vessels possesses intrinsic tone and, in some circumstances at least, exhibits rhythmic contractile behavior. Dilation of these arterioles reduces peripheral resistance and hence lowers blood pressure. Hydralazine is an example of a direct-acting vasodilator that is highly selective for the arteriolar resistance vessels. Most vasodilator drugs do not act solely on vessels of one type, but act to some extent both on veins and resistance vessels.

The pattern of their circulatory effects will thus depend upon their relative effects on venous capacitance and peripheral resistance. Hydralazine, minoxidil, and diazoxide dilate arterioles predominantly, whereas the nitrovasodilators dilate both arterioles and veins.

Side-Effects of Vasodilators:

There are three potential drawbacks in the use of vasodilators:

Systemic vasodilation and arterial pressure reduction can lead to a baroreceptor-mediated reflex stimulation of the heart (increased heart rate and inotropy).

This increases oxygen demand, which is undesirable if the patient also has coronary artery disease.

Vasodilators can impair normal baroreceptor-mediated reflex vasoconstriction when a person stands up, which can lead to orthostatic hypotension and syncope upon standing.

Vasodilators can lead to renal retention of sodium and water, which increases blood volume and cardiac output and thereby compensates for the reduced systemic vascular resistance.