


CARDIAC REHABILITATION PHASE IV



# Medication

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# Medication

Drugs can be taken in a number of different ways:

- Orally – Most drugs for the heart are taken orally (by mouth) either as tablets or capsules which you swallow.
- Sublingually – When you place a tablet under the tongue and let it dissolve.
- Aerosol spray – When you spray the drug directly under your tongue.
- Self-adhesive patch – You place a patch containing the drug on your skin and the drug is absorbed over a period of time.
- Intravenously – When a drug is injected directly into a vein (a 'bolus' injection), or in a diluted form through an intravenous drip.
- Intramuscularly – When a drug is given by an injection into a muscle such as the buttock or thigh.
- Subcutaneously – When a drug is given by an injection just under the skin. (Subcutaneous means 'under the skin'.)

(Source: BHF: Medicines for the heart. Heart Information Series Number 17, 2006)

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# Medication groups

## Diuretics

- Reducing the circulatory volume by increasing the excretion of water and sodium ions. This reduces venous return to the heart and, therefore, right-ventricular output. This reduces the workload placed upon the heart.
- Largely replaced by modern Angiotensin converting enzyme (ACE) inhibitors but still play a major role.
- Three main types are used: thiazides, loop diuretics and potassium-sparing.

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**Vasodilators**

- This group comprises a number of chemically unrelated compounds, which all have different modes of action. However, they all have the ability to cause dilation of peripheral blood vessels — both arteries and veins.

**Nitrates**

- The main effect of a nitrate is to cause venodilation, thereby reducing the amount of blood returning to the heart and, therefore, the heart's workload.
- Fast-acting nitrates can be administered under the tongue (sub-lingually) to relieve acute angina. The fast acting forms may come as a dissolvable tablet, a tablet for chewing or an aerosol spray. Slow-acting forms come as orally administered drugs or impregnated sticking plasters or 'patches'.

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**2. Calcium Channel Blockers**

- Muscle fibres require calcium to contract, they enter the cell across the cell membrane via so called "channels". The net effect of blocking these channels is decreased myocardial contractility and, therefore, less oxygen requirement; they also cause relaxation of the smooth muscle in arterioles, therefore reducing peripheral resistance and opening up coronary circulation.

**Angiotensin-Converting Enzyme (ACE) inhibitors**

- ACE inhibitors act by inhibiting the conversion of Angiotensin I to Angiotensin II, which therefore inhibits formation of the most powerful naturally occurring vasoconstrictor known. The dual action is also inhibiting the release of Anti-diuretic Hormone (another vasodilator) with its Sodium and water retaining properties leads to decreased peripheral resistance and decreased cardiac preloading. Their greatest effect is thus on the peripheral circulation and can be viewed as protective to the heart in reducing its work-load.

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**Angiotensin-II Receptor Antagonists**

- These drugs block the receptors of Angiotensin II and therefore reduce vasoconstriction. As a direct alternative to ACE Inhibitors, they do not cause a dry persistent cough, as a possible side effect.

**Alpha-adrenoceptor Blocking Agents**

- Alpha receptors exist as further sub-divisions Alpha 1 and Alpha 2. Their modes of action are different and they have opposite effects. Alpha 1 receptors facilitate vasoconstriction and it is this action which needs to be blocked to produce beneficial effects for the cardiovascular system, i.e., vasodilation.

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**Beta-adrenoceptor Blocking Agents**

- Beta-receptors are activated by circulating adrenaline and noradrenaline at sympathetic nerve endings and are found in greatest concentration in the heart, bronchi and peripheral blood vessels. The receptors are subdivided into  $\beta_1$  and  $\beta_2$  receptors:  $\beta_1$  receptors are found in the heart and  $\beta_2$  receptors in the bronchi and peripheral vasculature: blood vessel walls. Modern  $\beta$ -blockers are relatively cardio-selective but will still have some effect on  $\beta_2$  receptors.

**Potassium-channel Activators**

- These drugs cause vasodilation of the large coronary arteries and the smaller blood vessels, thereby increasing cardiac perfusion or restoring the oxygen supply to the heart in clients with angina. They also dilate the veins in the circulatory system, thereby reducing the preload to the heart.

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**Anti-platelet drugs**

- The action of these drugs is to reduce platelet aggregation (or stickiness) and, as such, may inhibit thrombus (blood clot) formation in the arterial circulation.

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**Medication groups**



Hypertension	Angina	Cardiac Rehabilitation *CHF
<ul style="list-style-type: none"> <li>Beta-blockers</li> <li>Calcium Channel Blockers</li> <li>Diuretics</li> <li>ACE Inhibitors</li> <li>Angiotensin-II Receptor Antagonists</li> <li>Anti-platelets</li> </ul>	<ul style="list-style-type: none"> <li>Nitrates</li> <li>Beta-blockers</li> <li>Calcium Channel Blockers</li> <li>Potassium Channel Activators</li> <li>Anti-platelets</li> <li>ACE-inhibitors</li> <li>Statins</li> </ul>	<ul style="list-style-type: none"> <li>Nitrates</li> <li><b>Beta-blockers*</b></li> <li>Calcium Channel Blockers</li> <li><b>Diuretics*</b></li> <li><b>ACE Inhibitors*</b></li> <li>Angiotensin-II Receptor Antagonists</li> <li>Potassium Channel Activators</li> <li>Anti-platelets</li> </ul>

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Any questions?

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