

Cardiovascular Effects of Beta-Blockers

Beta-blockers have numerous actions on the cardiovascular system and have been used for CHF, arrhythmias, angina, MI, hypertension and numerous other indications. They work by preventing normal ligands (epinephrine and norepinephrine) from binding to their adrenoceptor binding site. Specific actions which occur in the heart include decreasing contractility by halting Ca^{2+} influx by decreasing cAMP, and decreasing SA and AV node conduction velocity, which also slows chronotropy. By decreasing cardiac output, the blood pressure is lowered in patients, and O_2 consumption (through decreased oxygen demand) is slowed as chronotropy and inotropy are decreased. Furthermore, these drugs have been shown to decrease mortality and morbidity post-myocardial infarction.



PLAY PICMONIC

Cardiac Myocytes and Vasculature

Muscular Heart-with-vessels

Cardiac myocytes and peripheral vasculature contain α_1 - and α_2 -adrenoceptors which are activated by binding the ligands epinephrine and norepinephrine, leading to increased inotropy, chronotropy, dromotropy (electrical conduction), and thereby increasing blood pressure and cardiac output. β -blockers compete for adrenoceptor binding sites, helping to prevent these cardiac effects.

Decrease cAMP

Down-arrow cycle-AMP

β -blockers bind to G_s proteins and inhibit the formation of cAMP from ATP. This prevents intracellular flow of Ca^{2+} and leads to decreased Ca^{2+} currents by the sarcoplasmic reticulum of the heart. This translates into decreased heart contractility.

Decreases SA and AV Node Conduction Velocity

Down-arrow Silo-nose and AViator-Nose Conductor

Beta-blockers decrease sinoatrial (SA) and atrioventricular (AV) node conduction velocity, which means they decrease dromotropy. It is for this reason that they can be indicated for supraventricular tachycardia (SVT), ventricular tachycardia, atrial fibrillation, and atrial flutter. It is also for this reason that they are generally contraindicated in patients with heart block, or AV conduction delay, regardless of 1st-, 2nd-, or 3rd-degree.

Lower Blood Pressure

Down-arrow Blood Pressure cuff

These drugs typically are NOT first-line antihypertensive agents. Beta-blockers work to decrease blood pressure in multiple ways. First, their β -blockade leads to decreased chronotropy and inotropy, meaning that the heart has a lower cardiac output, contributing to lower blood pressure, and requiring less oxygen in the process. Secondly, beta-blockers decrease renin secretion, which in turn reduces extracellular volume, further lowering blood pressure, and also decreasing myocardial oxygen demand.

Decrease O_2 Consumption

Down-arrow O_2 Tank

By inhibiting the chronotropic and inotropic sympathetic effects of α -adrenergic receptors, the workload of the heart is decreased. This leads to decreased O_2 consumption (through decreased myocardial oxygen demand), making β -blockers an important medication class for treating angina.

Decreases Mortality Post-MI

Down-arrow Death at Post Mayo-heart Farting

Beta-blockers such as metoprolol, atenolol and bisoprolol are specifically used after myocardial infarctions and have been proven to decrease mortality and morbidity in these patients.