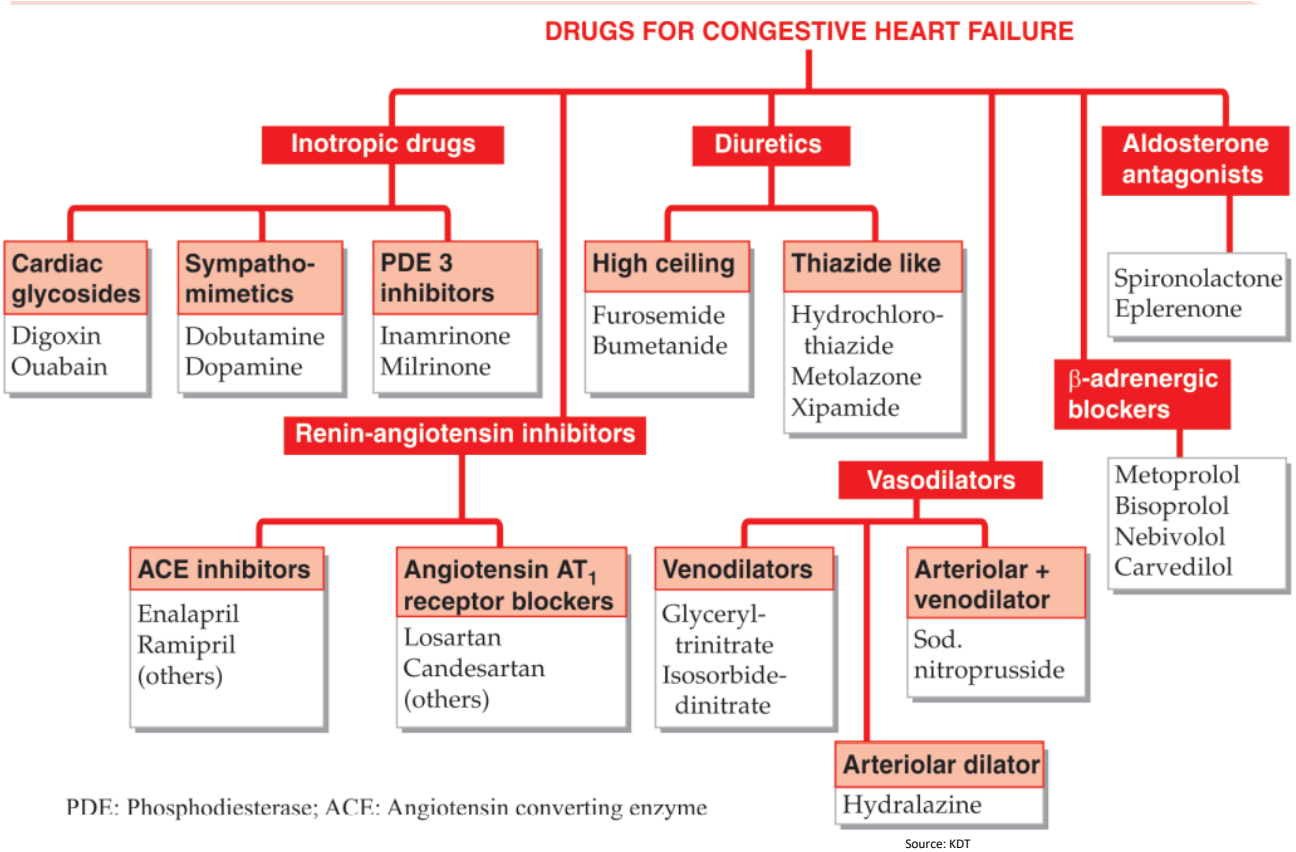
Introduction CONGESTIVE HEART FAILURE

Heart failure may be defined as the inability of the heart to maintain the cardiac output adequate to meet the metabolic demands of the body at all times. It manifests as: (1) Acute heart failure (as after MI); or (2) Chronic heart failure. Acute CHF is the condition in which heart is not able to pump the blood effectively; and failure of compensatory mechanisms to maintain the homeostasis, and it leads to increased sympathetic activity that causes increased cardiac output by stimulation of β1 adrenergic receptors in the heart. This maintains the cardiac output in short run which leads to compensation of heart failure. But, increased sympathetic activity also results in two other effects i.e. vasoconstriction due to α receptor stimulation and increased renin release from the kidney due to β1 stimulation. Elevated renin stimulates renin angiotensin aldosterone system, thus increasing angiotensin II and aldosterone (retains salt and water and is responsible for cardiac hypertrophy). Vasoconstriction of arterioles increases the after load and that of venules increases the preload, thus leading to increased workload on the heart.



Since the introduction of digitalis (in 1776) the pharmacotherapy of heart failure has made spectacular advances, resulting in better prognosis. Cardiac glycosides come from foxgloves (Digitalis spp.) and related plants. Withering wrote on the use of the foxglove in 1775.

Cardiac Glycosides are collectively known as digitalis. Compounds in this group include digoxin, digitoxin, strophanthin and ouabain etc. Cardiac glycosides are positive inotropic drugs but unlike other inotropes, these do not increase heart rate or oxygen consumption