Heart Drugs

"**Angina Pectoris**" transient heart pain is caused by an inadequate blood supply to the heart, (heart suffocation), clogged (arteriosclerosis) or harden (atherosclerosis) arteries to the heart, which decreases blood, nutrient, and oxygen to the heart causing heart suffocation and pain.

Angina Pectoris transient pain episodes are usually periodic or brief. They can have a squeezing sensation in the heart, or cause an ache or heavy pain in the chest, upper body or neck.

Myocardial Infarction (heart attack) pain usually is constant and/or feels like a crushing blow to the chest. The heart pain drugs, such as **propranolol** cannot stop an attack once it begins. They can cause heart failure and asthma to worsen, can cause rash, decrease oxygen, **bradycardia** (slow down heart), peripheral vascular disease, fatigue, hallucination, edema, lethargy, diarrhea, arthralgia, low blood sugar **(hypoglycemia**), vomiting, congestive heart failure and vivid dreams.

Nitroglycerin used for heart disease causes hypersensitivity reaction, burning tongue, weakness, headache, fainting, dizziness, cutaneous vasodilation, flushing, hypotension, heart palpitations, vomiting, tachycardia, nausea, and Orthostatic hypertension. Heart pain drugs are listed as ineffective (do not work), They temporarily change the vitamin, amino acid, hormone and mineral ratio of the body's biochemistry. This shifts the body's attention away from the heart and to other problems caused by the chug's chemical stressor.

Another ineffective drug that does not work is Isosorbide Dinitrate which poisons the left ventricular-end diastolic resting heart pressure (blood preload) and weakens bodily (systemic) vascular ventricular-end diastolic resting heart pressure (blood preload) and reduces the ability of the heart to meet oxygen requirement.

"**Antianginal**" drugs for heart pain are also called Coronary Vasodilators based on the unscientific assumed belief that dilating coronary arteries will relieve pain. There are the "Antianginal" drugs for preventive management of an acute sharp rapid onset heart attack and those for long-term treatment of attacks. The "Antianginal" effects that the chugs are believed to cause can be the result of the person sitting down and resting or reduced physical exertion, anxiety, stress and other subjective measures. These subjective "Antianginal" effects can reduce the frequency and severity of the attacks without drugs. For example, a person may have heart pain then stop and rest for fear it will get worse or they fear they may die. By stopping and resting their body, they have caused an Antianginal effect Therefore, it is not clear whether the drug or the rest relieved the pain. The drug company assumes the drug stopped the attack and not the Antiangina1 activity of rest.