SNS COLLEGE OF ALLIED HEALTH SCIENCE





DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE TECHNOLOGY

COURSE NAME: PHARMACOLOGY

TOPIC: ANTIANGINAL DRUGS

UNIT: 1

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INTRODUCTION

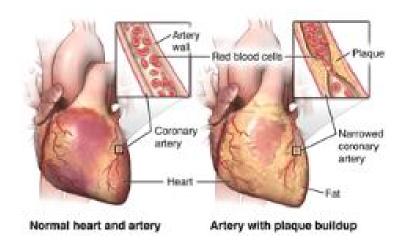


• **DEFINITION { DEFINE STAGE } :**

- Angina pectoris is a clinical syndrome of chest pain or discomfort caused by transient myocardial ischemia — an imbalance between myocardial oxygen supply and demand.
- Antianginal drugs are used to restore this balance.

GOALS OF THERAPY:

- Relieve acute symptoms of an angina attack.
- Prevent further attacks and improve exercise tolerance.
- Reduce the risk of myocardial infarction (MI).



CLASSIFICATION





Nitrates:
e.g., Nitroglycerin,
Isosorbide Dinitrate.
Primarily act as
vasodilators to reduce
preload.



Beta-Blockers:
e.g., Metoprolol, Atenolol.
Decrease heart rate and
contractility to reduce
oxygen demand.



Calcium Channel Blockers:
e.g., Amlodipine, Diltiazem.
Reduce afterload and/or
heart rate.



Other Agents:
e.g., Ranolazine, Ivabradine.
Newer agents with unique
mechanisms of action.

NITRATES



MECHANISM OF ACTION:

- Converted to Nitric Oxide (NO), which increases cGMP.
- leads to smooth muscle relaxation (vasodilation), (importantly veins)
- reduces venous return (preload) and, myocardial oxygen demand.

PHARMACOKINETICS (NITROGLYCERIN SL)

- Dosage: 0.3 0.6 mg Sublingual
- Onset: 1-3 minutes (Rapid)
- Half-life (t1/2): 2-3 minutes
- Duration: 10-30 minutes







INDICATIONS: Used for acute angina attacks (Sublingual) and for long-term prophylaxis (oral, transdermal forms).



ADVERSE EFFECTS: Most common is a throbbing headache.

Also causes flushing, postural hypotension, and reflex tachycardia.



CONTRAINDICATIONS: Severe hypotension. Critically, DO

NOT use with PDE5 inhibitors (e.g., Sildenafil) due to risk of life-threatening hypotension.



BETA BLOCEKERS



MECHANISM OF ACTION:

- Competitively block β1-adrenergic receptors in the heart.
- This decreases heart rate (negative chronotropy) and contractility (negative inotropy),
- reducing myocardial oxygen demand, (especially during exertion.)
- PHARMACOKINETICS (METOPROLOL):
- Bioavailability: ~50% (variable, high first-pass)
- Metabolism: Hepatic (CYP2D6)
- Half-life (t1/2): 3-7 hours
- Excretion: Renal (as metabolites)





- **INDICATIONS:** First-line for prophylaxis of exertional angina. Also used for hypertension and post-myocardial infarction.
- ADVERSE EFFECTS: Bradycardia, fatigue, lethargy. Nonselective β-blockers (like Propranolol) can cause bronchospasm in asthma patients.
 - **CONTRAINDICATIONS**: Severe bradycardia, high-degree AV block, acute decompensated heart failure, and severe asthma (for non-selective agents).



CALCIUM CHANNEL BLOCKERS



MECHANISM OF ACTION:

- Block L-type calcium channels.
- Dihydropyridines (e.g., Amlodipine): Potent peripheral vasodilation, reducing afterload.
- Non-Dihydropyridines (e.g., Diltiazem): Also decrease heart rate and contractility.
- PHARMACOKINETICS (AMLODIPINE) :
- Bioavailability: 64-90%
- Metabolism: Hepatic (extensive)
- Half-life (t1/2): 30-50 hours (very long)
- Onset: Slow (suitable for prophylaxis)







INDICATIONS: Prophylaxis of angina, especially vasospastic angina. Also used for hypertension and arrhythmias (Non-DHPs).



ADVERSE EFFECTS: (Amlodipine) Peripheral edema, flushing, headache. (Diltiazem/Verapamil) Constipation, bradycardia.



CONTRAINDICATIONS: (Non-DHPs) Severe bradycardia, AV block, acute heart failure. (DHPs) Use with caution in severe hypotension.



OTHER AGENTS (RANOLAZINE)



MECHANISM OF ACTION:

- Selectively inhibits the late inward sodium current Na in myocardial cells,
- preventing Ca2+ overload, improving relaxation and perfusion.

PHARMACOKINETICS:

- Administered as 500 -1000 mgBID
- Highly metabolized by CYP3A4 and CYP2D6.
- Short T1/2: 2-3 hours,
- with renal excretion.





INDICATIONS: Chronic stable angina (used in combination with other antianginals).

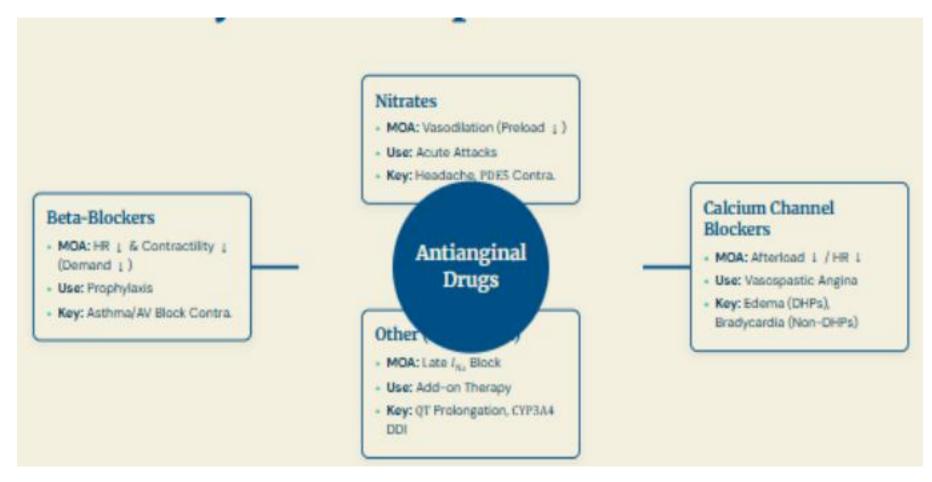
ADVERSE EFFECTS: Constipation, nausea, dizziness, and QT interval prolongation.

CONTRAINDICATIONS: Liver cirrhosis, co-administration with potent CYP3A4 inhibitors (e.g., Ketoconazole).





SUMMARY



KEY DEPICTION AND REFERENCES



https://www.drugs.com/drug-class/antianginal-agents.html

https://www.slideshare.net/slideshow/antianginal-drug-88309899/88309899

https://www.thecardiologyadvisor.com/ddi/antianginal-drugs/

https://pubmed.ncbi.nlm.nih.gov/30165445/



