① Stable angina -

Also known as classical angina,

- It is most common type of angina.
- These attacks are predictable and caused by Exercise, Emotions and ↑ work load.

Classification of Anti Anginal Drugs -

① Nitrates (vasodilators) → Sublingual

[ Short acting - GTN, Isosorbid dinitrate
Long acting - Isosorbid dinitrate (oral) etc.

② Calcium channel blockers -

— Verapamil
— Amlodipine etc.

③ Beta blockers -

- Propranolol, Metoprolol, Atenolol etc.

④ $K^+$ channel opener -

- Nicorandil

— Some Others.

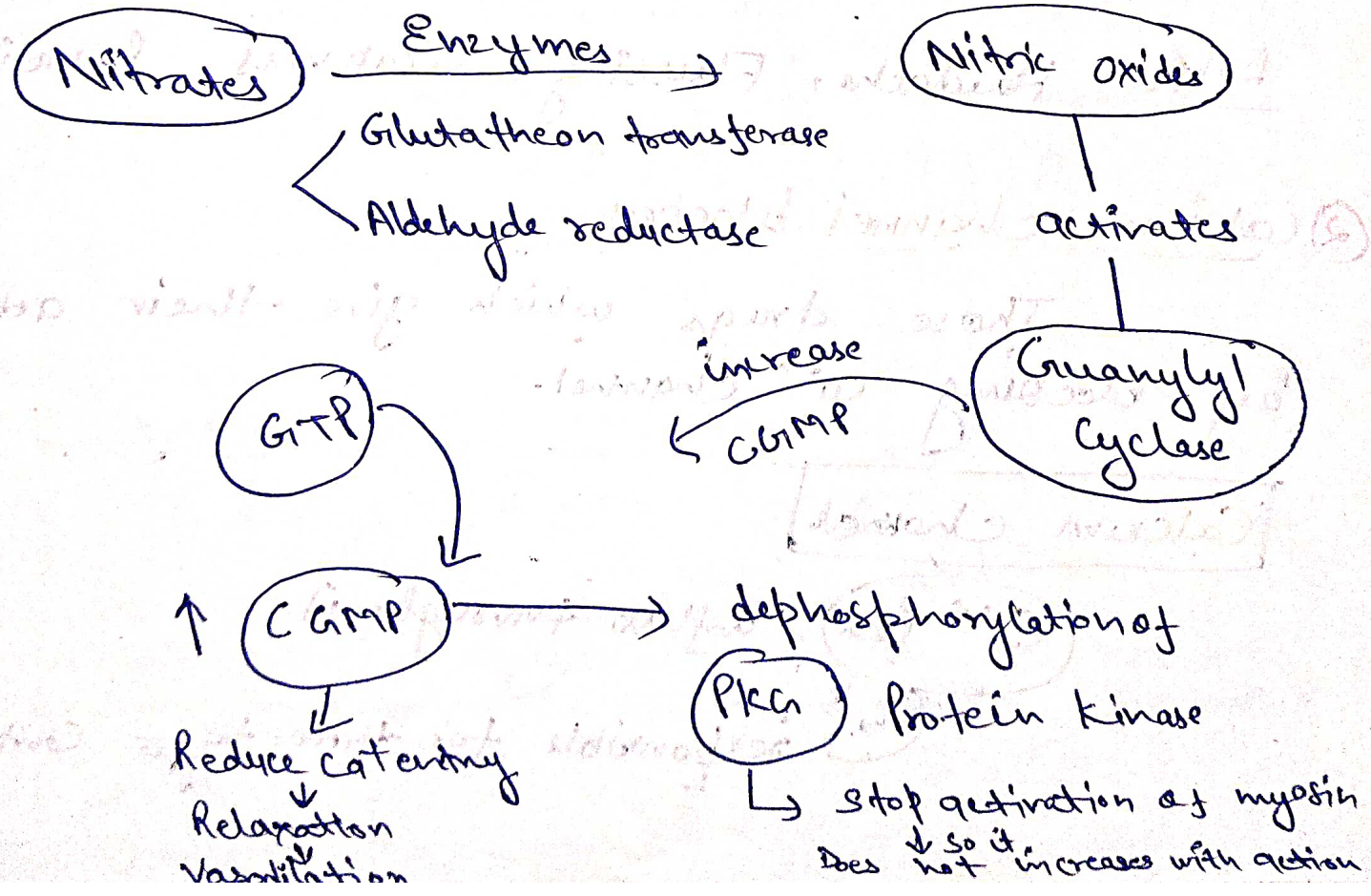
Some others also used antihypertensive drugs for lower blood pressure and for vasodilation.

Also used anticoagulant to inhibit clotting in unstable, but cause excessive bleeding. (Not ~~preffer~~ prefare).

## ① Nitrates –

Also known as organic nitrates. These drugs act as a vasodilators.

### Mechanism →



Effects -

→ ↓ Contraction
→ ↑ Relaxation
→ ↓ Preload & afterload.

Eg.- GTN / Glyceryl trinitrate / Nitroglycerin
Isosorbide dinitrate.

- Lipid soluble - well absorbed through buccal mucosa, intestine & skin.
- Short acting → it use sublingual
- Long acting → It use orally

AdR- Headache, Flussing, weakness, Sweating etc.

## (2) Calcium channel blockers

Those drugs which give their action by blocking $Ca^{++}$ channel.

**Calcium channel**

$Ca^{++}$ ion influx through it.

responsible for ~~three types~~ Contraction.

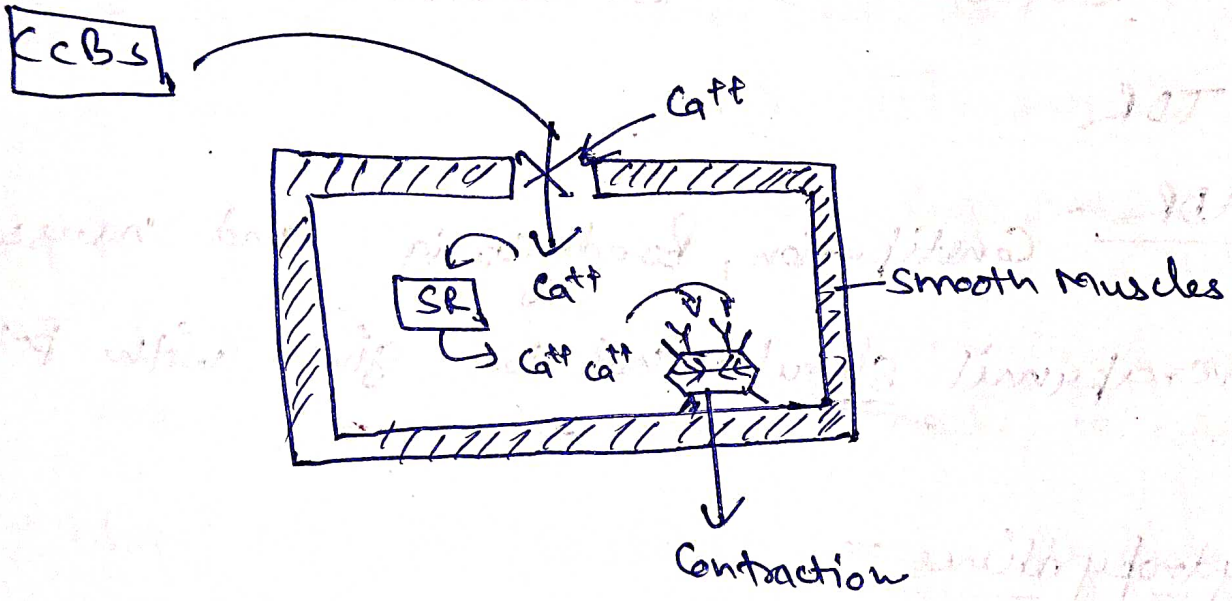
Now $Ca^{++}$ channel are of three types
- Voltage sensitive Channel ← (L type, T type, N type) — very sensitive, Rapid Depolarization
- Receptor operated channel.
- Leak channel

But these $Ca^{++}$ channel blockers give their action by blocking (voltage sensitive)

## L-type channel

## Mechanism

CCBs → binds with L-type $Ca^{++}$ channel →




→ ↓ $Ca^{++}$ entry → ↓ Contraction

↓ Contraction → Relaxation → Vasodilation

↓ Contraction → ↓ HR → work load for heart ↓

→ Also reduce ↓ peripheral vascular Resistance

→ Also ↓ preload and afterload.

Drugs — DHPs (Dihydropyridines)
↳ Lipophilic & potent

verapamil and diltiazem
↳ hydrophilic & less potent

- Verapamil –
  Same mechanism
  — Metabolism → Metabolized in liver
  ↓ TDR
  — ADR – Constipation, Bradycardia and nausea etc.
  — verapamil should not be given with β-blockers

Dihydropyridines –
Nifedipine, Amlodipine, felodipine, Nitredipine, Lacidipine, clinidipine etc

## ③ β Blockers -

These drugs does not dilate coronaries or other blood vessels instead of it, they reduce cardiac work and $O_2$ consumption, which further improves coronary perfusion.

**β- blockers**

↓ Cardiac work (HR↓, Co↓)

↓

$O_2$ demand ↓ decreases

→ It helps in classical angina

→ $\beta_1$ selective beta blocker (Atenolol, metoprolol)

Use more than non-selective (Propranolol)

(Contra - indicated in asthmatic Patients

→ long term β - blocker therapy lowers the risk of sudden cardiac death in CAD patients

→ Not used in vasospastic angina, because they can cause vasospasm (risk)

→ Combination (β blockers + Nitrates) — Better response but do not used with $Ca^{++}$ channel blockers.

eg → Propranolol, Metoprolol & atenolol etc

# Drug Combination in Angina-

① β blocker + Nitrate (long acting)

In Classical angina

— Tachycardia due to nitrate is blocked by β - Blocker

— The tendency of β blocker to reduce total coronary flow is opposed (maintained) by nitrate.

② β blocker + Slow acting DHP (Ca++ channel blocker)

In classical angina

If there are any vasospasm then DHP helps in it.

③ Nitrates + CCBs

severe vasospastic angina

— Nitrates
— CCBs

↓ Preload & afterload

↓ Cardiac work

Improves cardiac perfusion

- Absorbed orally
- Completely metabolized in liver
- Excreted in urine
- Biphasic elimination
  - Initial rapid phase $t_{1/2}$ → 1 hrs
  - later slow phase $t_{1/2}$ → 12 hrs.

Adr -

Flushing, palpitation, weakness, headache, dizziness, nausea & vomiting etc.

- 2nd line antianginal effects.
  because their efficacy and long term effects are less established.
- Administered IV during → angioplasty for acute MI.