

Cough

- dry cough.
- productive cough.

[dry] → no actual use for body.
 → there is no secretions, so doesn't remove anything.
 → Just irritates.

for **[dry cough]**

Anti-tussives.

⊖ cough centre of brain.

- CODEINE
- Pholcodine
- dextromethorphan.
- Noscapine.

productive cough.

- sputum ✓
- harmful bacteria goes out through sputum.
- we don't want to stop that
- we just wanna make it easier.

for productive cough

Mucokinetics.

Expectorants.

↑ secretions in Resp tract & make it dilute.
 ↓
 so easy excretion.

↓
Guafenesin
KI

Mucolytic

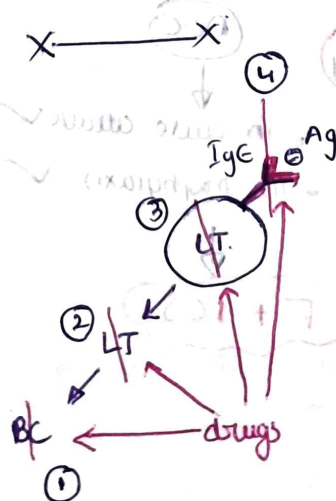
• breaks down mucous

↓
 so easy excretion.

↓
Ambroxol
Bromhexine.

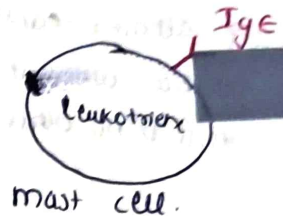
acetyl - Cysteine
Dornase - α.

dry cough ko → Band Kard do.
 productive ko → easy kar do.



Asthma.

- Type I, HS reaction
- mediated by IgE



first exposure → IgE coating.

↓
 priming of mast cell.

next exposure → Ag bind to IgE on mast cell.

↓
 degranulation.

↓
 Leukotriene release.

↓
 LT contract Bronchi.

LT → Bronchoconstriction.
 ↓
 is the probm.

Solution?

- 1) Bronchodilators.
- 2) LT ⊖
- 3) mast cell stabilizers
- 4) ⊖ Ag-Ab interaction.

Bronchodilators

- acute attack → Bronchoconstriction.
- In acute attacks Bronchodilator is must
- acute attacks, can't be treated without (BD) ... there is no other way

Symp → Bronchodilator.
para → Bronchoconstriction.

- So,
- (+) β_2 sympathetic Receptor.
- (-) m_3 parasympathetic Receptor.

(+) β_2 agonist.

Salbutamol / ALBUTEROL

Terbutaline

Salmeterol } long acting

Formoterol } long-acting

all can be given by Inhalational route.

• long acting → for prophylaxis

• short acting are also fast acting
→ for acute attacks ✓
not prophylaxis.

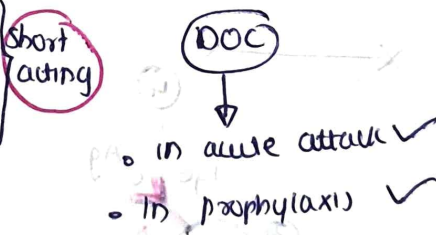
	action onset	duration of action
Salbutamol	slow	Long.
Formoterol	fast	Short.
Salbutamol	long slow	Short.
Terbutaline	slow	Short.

	onset	duration	acute attacks	prophylaxis
Salbutamol	fast	Short	✓	X
Terbutaline	fast	Short	✓	X
Salmeterol	slow	Long	X	✓
Formoterol	fast	Long	✓	✓

(DOC) → In Acute attack & prophylaxis

For formoterol + ICS
+ ICS combo.

we give it in combo with Inhalational corticosteroid



F + ICS

S/E

- T - tremor - (m/c)
- T - tachycardia - at overdose
- T - Tolerance.
- T - T-wave changes.

X Does not cause Hypoglycemia

{actually ↑ sugar}

free question

Only in acute attack.
 In p/o only both a/c & p/o.

M3 blockers.

Ipratropium Inhalation.
 Tiotropium COPD > Asthma.

* Main use: COPD. in COPD, vagus overactivity is main reason of BC. so we use M3 # -> parasymp (vagus) #
 * also used in Asthma.

In some people...

Ipratropium causes (BC)

↓
 paradoxical Bronchoconstriction.

Reasons:

- 1) Impurities in preparation -> EDTA, Benzalkonium.
- 2) Hypersaline in nebulisation sol.
- 3) presynaptic musc receptor # { Breach loose }

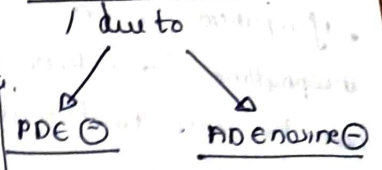
PDE Inhibitors.

Theophylline Oral, IV ✓
 Aminophylline Inhalational X

MOA

- * PDE # -> ↑ cAMP -> BD.
- * adenosine A1 receptors #.
- * ↑ HDAC -> anti-inflammatory action.

Theophylline s/e.

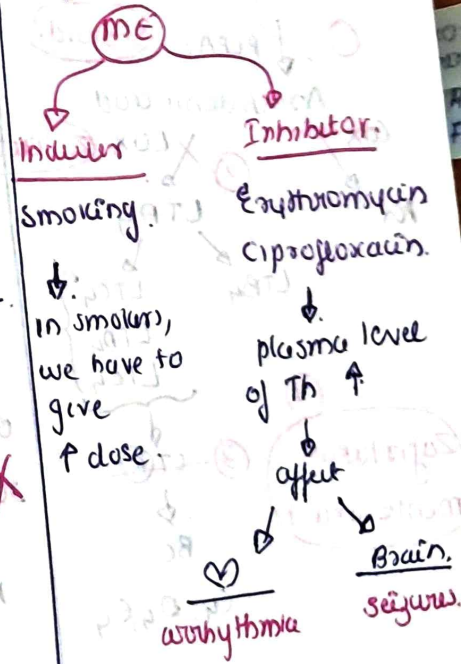


S/E: N, V, D's. Arrhythmia.
 Headache. Diuresis.
 Arrhythmia. Epileptic seizure.

freq. waked Astm. which s/e is due to waken.

Theophylline follows -

Zero order kinetics.
 (DI) microsomal enzyme degrades & metabolizes it.



due to β_2 ⊕
 -> β_1 is also ⊕ at high dose.

due to Hypokalemia.

⊕ is effect of 2 things.

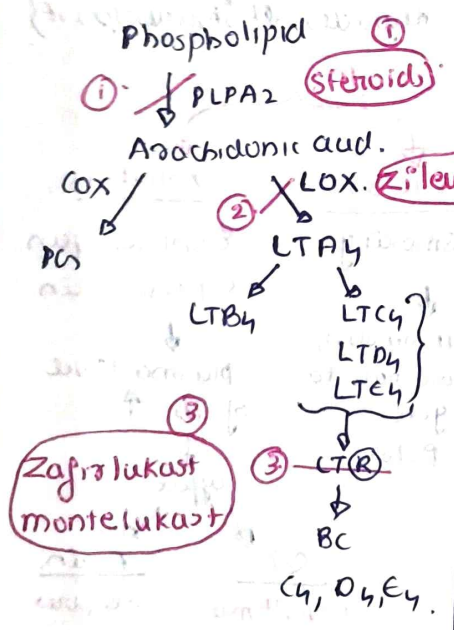
- * Insulin.
- * β_2 agonist

⊕ goes intracellularly & ↓ in serum.

DI of Th is very imp.

• if a patient is on theophylline for asthma and he comes to hospital for resp TI - a common medication prescribed for RTI is Erythromycin
 if we give him that w/o leucy proper H/O he will get seizures & arrhythmias

② Leukotriene



Zafirlukast
 Montelukast

- COX inhibition doesn't cure B.C.
- It will actually ↑ BC.
- as all AA will be conv. to CT.
- Aspirin Induced Asthma.

Steroids

- they are not B.D.
- they are anti-inflammatory
- ie, ↓ Inflammatory reaction.
- ⇒ they are given in prophylaxis.
- ⇒ ↑ B₂ sensitivity to Bronchodilators.
- ⇒ they are not B.D they are adjuvants.

Inhalation CS - ICS

- Beclomethasone
- Fluticasone
- Flunisolide
- Mometasone
- Ciclesonide
- Budesonide

only 5% reaches Bronchus. rest is in sup tract.
 ↓
 m/c s/e
 ↓
 Oral Candidiasis.

prevention ⇒ Gargling
 Candidiasis Rx
 → Topical Nystatin.

Zileuton

Inhibit Leukotriene numbers.
 ? least on inhibit

Zafirlukast
 Montelukast

Leukotriene Antagonist of LTR
 Luk as F antagonist

③ most cell stabilizers

• even after Ag-Ab interaction mast cell won't degranulate.

- CROMOGLYCATE
- NEDOCROMIL
- KETOTIFEN

In acute attack, mast cell already broken.

↓
 Not useful.

↓
 used in P/O only.

3 Inhalational group

- B₂ agonist
- steroids.
- mast cell stabilizers.

Zileuton

Inhibit leukotriene numbers.

↑ cent on inhibit

Zafirlukast
Montelukast

Leukotriene Antagonist
of LT₂R

Lux (as F) antagonist

3) Mast cell stabilizers

• even after Ag-Ab interaction mast cell won't degranulate.

• CROMOGLYCATE
• NEDOCROMIL
• KETOTIFEN
Inhalation

In acute attack, mast cell already broken.

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3 Inhalation al group

- β₂ agonist
- steroids
- mast cell stabilizers

4) Ab-Ag Interaction

Anti-Ab → Anti-IgE



OMALIZUMAB

- s/c injection.
- doesn't treat EC - NO BD.
- X acute attack.
- ✓ P/O.

IL-5



mepolizumab

Reslizumab

Global Initiative

for Asthma

(2021) GINA

Rescue therapy { acute attack }
maintenance therapy { prevention }

Both:

F+ICS

- low dose

puff.

→ acute attack

→ for maintenance