## Introduction

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| **Adrenergic drugs** | release acetylcholine at the post synaptic level. Only post ganglionic sypathetic nerves are adrenergic as they use epinephrine as neuron transmitters. |
| **Function** | Norepinephrine is a neurotransmitter of adrenergic synapse, coming from tyrosine → DOPA → Dopamine, stored in vesicles until it needs to be released, when impulses from CNS send the signals. They use exocytosis so the norepinephrine gets into the synaptic cleft. In synaptic cleft, norepinephrine needs to act on post synaptic receptors. |
| | Norepinephrine is released from the presynaptic membrane 1) Most of it is taken back w/out any changes to be reused (uptake 1),<br>2) Taken by factors cells AKA non neuronal uptake (uptake 2)<br>3) Diffuse<br>4) Degrade (by enzyme present in the synaptic left, minor). |

## Introduction (cont)

Norepinephrine can act on presynaptic receptors, regulate release of Norepinephrine from sympathetic nerve endings (AKA alpha 2 receptors), regulates -ive feedback mechanism (uptake 1)

## Drugs - based on potency

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| **α-adrenoceptors** | Norepinephrine → epinephrine → isoprenaline |
| **β-adrenoceptors** | Isoprenaline → epinephrine → norepinephrine |
| **α,β adrenoceptors** | Epinephrine + norepinephrine |

## Groups of drugs

| Subtypes | Mechanism of action |
|---|---|
| **α1 adrenoceptors** | **Postsynaptic** - mediate effect on sympathetic nerve system (smooth muscle tissues + organs + blood vessels)) |
| **α2 adrenoceptors** | **Presynaptic** - out of synapse (mediat effects on catecholamines released from adrenal medulla) |
| **β1 adrenoceptors** | **Postsynaptic** - mediate effect on sympathetic nerve system (heart) |
| **β2 adrenoceptors** | **Presynaptic** - out of synapse (mediate effects of catecholamines ccirculating in blood leads to vasodilation) |

## Adreno-positive drugs

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| **α, β-adrenomimetics** (natural - used intravenously) | **Epinephrine, norepinephrine** |
| **α-adrenomimetics** (selective) | **α1-adrenomimetics** = **phenylephrine** (increases BP locally + effective orally)<br>**α2-adrenomimetics** = **Naphazoline**, **clonidine** (decreases BP, antihypertensive drug) |
| **β-adrenomimetics** (selective) | **β1,β2-adrenomimetics** = **isoprenaline**<br>**β1-adrenomimetics** = **dobutamine** (if patient doesnt have hypoxia or ischemic disease)<br>**β2-adrenomimetics** = **salbutamol** (short acting drug), **phenoterol**, **salmeterol** (slow/-prolonged acting drug) |
| **Sympathomimetics** (indirect binding on receptors) | **Ephedrine** (acts on sypathetic endings, stimulates release of norepinephrine, indirectly produces efftecs on post synaptic receptors) |

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Published 23rd August, 2023.
Last updated 23rd August, 2023.

## Adreno-negative drugs

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| **Adrenoblockers/-adrenoceptors antagonists** (direct binding on receptors) | **α, β-adrenoblockers** (natural) - **Labetalol** **α-adrenoblockers** - **α1,α2-adrenoblockers** (non-selective) - **Phentolamine**, dihydroergotamine **α1-adrenoblockers** - **α1a-adrenoblockers** - **tamsulosin** **α1b-adrenoblockers** - **prazosin** |
| | **β- Adrenoblockers / adrenoceptor antagonists** **β1,β2-Adrenoblockers** (non-selective) - **Propranolol**, timolol **β1-adrenoblockers** - **atenolol, metrolol** |
| **Sympatholytics** (indirect binding on receptors) | **Reserpine**, guanethidine |

## Indications + side effects

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| α adrenomimetics | Pharmacological effects - Vasoconstriction of blood vessels<br>Mydriasis<br>Decrease NA peripheral |

## Indications + side effects (cont)

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| β adrenomimetics | **Indications** - Bronchial asthma, uterine relaxation (preserving pregnancy)<br>**pharmacological effects** - cause dilation of brochial passages, Vasodilation in muscle and liver, Relaxaion of uterine muscle, Release of insulin |
| α adrenoblockers | **Side effects** - hypotension Tachycardia |
| β adrenoblockers | **Mechanism of action of antihypertensive action of beta adrenoblockers** - decrease cardiac output Decrease renin secretion reduce central sympathetic activity (selective BB))<br>**Side effects**<br>**Side effects β2 AB** - *Heart insufficiency*, *bronchoconstriction*, *hypoglycemia*, fatigue, dizziness, nausea, diarrhea<br>Side effects β1 AB - |

## Specific drugs

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|---|---|
| **Phentolamine** (anatgonises α1+2 adrenoceptors) | Decreases BP caused by adrenaline, affects -ive feedback mechanism (α2-adrenoceptors) in the synapse<br>**Indications** - Pheochromocytoma (adrenal gland tumour) + endarteritis (inflammation of arteries, legs<br>**Side effects** - Orthostatic (standing) collapse (severe drop in BP) + tachycardia reverse effects of adrenaline on BP |

## Specific drugs (cont)

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|---|---|
| **Tamsulosin** (Selectively anatgonises/-blocks α1A-adrenoceptors) | **Indication** - benign prostate hyperplasia (increased cell production in a normal tissue or organ)<br>Relax smooth muscle of prostate gland<br>Help to decrease these symptoms |
| **Prazosin** (Selectively atangonises/blocks α1B-adrenoceptors in bood vessels) | Does not affect the -ive feedback mechanism in synapse<br>**Indication** - arterial hypertension<br>**Side effect** - othostatic collapse |

## Specific drugs

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| **Propanolol** - Non-selective β1,β2-adrenoblocker | Decrease heart output, decrease activity of SA + AV nodes, decrease BP due to action on:<br>Heart - decreases heart output<br>Kidney (propanolol decreases production production of renin in the kidney<br>CNS (decrease sympathetic activity on PNS)) |
| **Atenolol, Metoprolol** (Selective β1-Adrenoblocker) | Cardioselective in therapeutic doses<br>drugs of choice in cardiac patients |