

**Adrenergic Agonist:**

* Adrenergic against produce their effects by activating adrenergic receptors.
* Since the sympathetic nerve system acts through these same receptor, because of this similarity, adrenergic agonists are often referred to as sympathomimetics.
* Adrenergic agonists have a broad spectrum of clinical application ranging from treatment of heart failure to relief of asthma to delay of preterm labor.

**Mechanisms of Adrenergic Receptor Activation:**

* Drugs can activate adrenergic receptors by four basic mechanisms;
1. Direct receptor binding
2. Promotion of nonepinephrine (NE) release.
3. Blocked of NE reuptake
4. Inhibition of NE inactivation

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| **Mechanisms of Stimulation**  | **Examples**  |
| Direct mechanism binding to receptor to cause activation.  | EpinephrineIsoproternol Ephedrine  |
| Indirect mechanisms promotion of NE release  | EphedrineAmphetamins  |
| Inhibition of NE reuptake  | CocaineTricyclic antidepressants  |
| Inhibition of MAO  | MAO inhibitors  |

**Chemical Classification of Adrenergic Agonist**

* The adrenergic agonists fall into two major chemical classification:

 Catecholamines and non-catecholamines.

* The catecholamines and non-catecholamines differ from each other in three important respects:
1. Oral usability
2. Duration of action
3. Ability to act in the CNS

**Epinephrine (Adrenalin):**

* The first adrenergic agonists employed clinically and can be considered the prototype of the sympathomimetic drugs.
* Adrenalin is acatechnolamine acts by activation of α ₁ , α₂, β₁, β ₂ receptors.

**Therapeutic uses:**

* Epinephrine can activate all subtype of adrenergic receptors, that make epinephrine.

By activating - α ₁ - mediated (Vasoconstriction)

* Epinephrine is used to
1. Delay absorption of local anesthetics
2. Control superficial bleeding
3. Reduce nasal congestion
4. Elevate blood pressure

Activation of α2 receptors on the iris🡪 Mydriasis.

Activation of β₁🡪 overcome AV heart block and restore cardiac function in patients undergoing cardiac arrest.

Activation of β2 promotes bronchodilation in patients with asthma.

Activation of α + β 🡪 treatment of anaphylactic shock.

**Pharmacokinetics:**

* Epinephrine administered topically, by injection, and by inhalation cannot be given orally.
* **Adverse effects:**

 Hypertensive Grisis (α ₁ ), Dysrhythmias (β₁) , Anginapectoris(β₁ ), Hyperglycemia (β₂).

* **Drug interactions:**

MAO inhibitors, Tricyclic antidepressent, General anesthetics.

**Isoproterenal (Isuprel):**

* Activated β₁ and β₂ (B- slective agent).
* Therapeutic uses:

In cardiovascular: by activating β₁, the drug can help overcome AV heart block, it can restart the heart following cardiac arrest, and it can increase cardiac output during shock.

Asthma: by activating β₂ in lung, Isoprotekend can cause bronchodilation,

* Adverse effects:

Dysrhythmias and angina pectoris,

hyperglycemia in diabetics.

* Drug interactions:

MAO inhibitors, tricyclic antidepressants

* Administration:

 For therapy of asthma 🡪oral inhalation, sublinqually and intravernously.

When we used to stimulate the heart🡪 IV, IM, introcardiac injection.

**Dopamine**:

* Activated dopomine, β₁ and at high doses α₁ receptors.
* Therapeutic uses:
* shock
* heart failure by (β₁)

**Adverse effects**:

* Tachycardia, dysrthythmias and anginal pain.
* Dopamine is contro indicated for patients with tachy dysrhythmias or ventrecular fibrillation.
* Drug interactions:

 MAO inhibitors, Tricyclic-antidepressant

* Administration: IV

**Dobutamine:**

* Acts by activation of β₁ receptors.
* Action of uses: heart failure (β₁)
* Adverse effects: tachycardia
* Drug interaction: MAO inhibitors
* Administration: I.V.

**Ephedrine:**

* Acts by activation of α₁,α₂,β₁,β₂
* Therapeutic uses:
	+ Nasal congenstion, Narcolepsy,
* Adverse effects:
	+ the same adverse effects of epinephrine.