anti-adrenergic drugs
Divisions of human nervous system

Human Nervous system

- Central Nervous System
- Peripheral Nervous System
- Autonomic Nervous System
Nervous system

Includes neurons and ganglia outside of the brain and spinal cord

Peripheral Nervous System

*Either “fight and flight” mode or “rest and digest”

*Autonomic Nervous System (involuntary)

Somatic Nervous System (voluntary)

With neurotransmitters norepinephrine and acetylcholine

Sympathetic Nervous System (adrenergic)

Parasympathetic Nervous System (cholinergic)
Sympathomimetic or adrenergic in sympathetic nervous system—neurotransmitters are ______________

Parasympathomimetic or cholinergic are used to describe parasympathetic system—neurotransmitter is ______________
Sympathetic nervous system

Fight or flight response results in:

1. Increased BP
2. Increased blood flow to brain, heart and skeletal muscles
3. Increased muscle glycogen for energy
4. Increased rate of coagulation
5. Pupil dilation
Adrenergic receptors

Alpha—A1 and A2
Beta—B1, B2, B3
Review of functions of sympathetic nervous system receptors

Alpha 1—smooth muscle contraction
Alpha 2-negative feedback causes less norepinephrine to be released so BP is reduced
Beta 1—increased heart rate
Beta 2—bronchodilation
Beta 3—actual site for lipolysis
Anti-adrenergics

Sympatholytic

Block or decrease the effects of sympathetic nerve stimulation, endogenous catecholamines and adrenergic drugs
Adrenergic Antagonists

**Alpha blockers**
- **Nonselective**
  - Irreversible (phenoxybenzamine)
  - Reversible (phenolamine)
- **Alpha$_2$-selective** (yohimbine)
- **Alpha$_1$-selective** (prazosin)

**Beta blockers**
- **Beta$_2$-selective** (butoxamine)
- **Beta$_1$-selective** (atenolol)
- **Nonselective** (propranolol)
A. Classification

Irreversible, Non selective: Phenoxybenzamine

Reversible, Non selective: Phentolamine

Alpha₁-selective: Prazosin, Terazosin, Tamsulosin

Alpha₂-selective: Yohimbine, rauwolscine
# Beta-blockers (antagonists)

<table>
<thead>
<tr>
<th>Selective $(\beta_1)$</th>
<th>Non selective $(\beta_1 &amp; \beta_2)$</th>
<th>Mixed $(\beta &amp; \alpha)$</th>
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<tbody>
<tr>
<td>Atenolol</td>
<td>Esmolol</td>
<td>Carvedilol</td>
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<tr>
<td>Betaxalol</td>
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<td>Metoprolol</td>
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Beta adrenergic blocking medications

Prevent receptors from responding to sympathetic nerve impulses, catecholamines and beta adrenergic drugs.
Effects of Propranolol

- Decreased heart rate
- Decreased force of contraction
- Decreased CO
- Slow cardiac conduction
- Decreased automaticity of ectopic pacemakers
Effects of Propranolol

- Decreased renin secretion from kidneys
- Decreased BP
- Bronchoconstriction
- Less effective metabolism of glucose. May result in more pronounced hypoglycemia and early s/s of hypoglycemia may be blocked (tachycardia)
Effects of Propranolol

Decreased production of aqueous humor in eye
May increase VLDL and decrease HDL
Diminished portal pressure in patients with cirrhosis
Beta Blockers - Indications

Mainly for cardiovascular disorders (angina, dysrhythmias, hypertension, MI and glaucoma)

In angina, beta blockers decrease myocardial oxygen consumption by decreasing rate, BP and contractility. Slow conduction both in SA node and AV node.
Beta blockers

Inhibition of renin, decreasing cardiac output and by decreasing sympathetic stimulation

May worsen condition of heart failure as they are negative inotropes

May reduce risk of “sudden death”
Beta blockers

Decrease remodeling seen in heart failure

In glaucoma, reduce intraocular pressure by binding to beta-adrenergic receptors in ciliary body, thus decrease formation of aqueous humor
Beta blockers

Propranolol is prototype

Useful in treatment of hypertension, dysrhythmias, angina pectoris, MI

Useful in pheochromocytoma in conjunction with alpha blockers (counter catecholamine release)

migraines
Beta Blockers

In cirrhosis, propranolol may decrease the incidence of bleeding esophageal varices.

Used to be contraindicated in heart failure, now are standard.

Known to reduce sudden death.

Often given with ACEIs.

Indications include: htn, angina, prevention of MI.
Adverse reactions of $\beta$-blockers

• Blockade of $\beta_1$-receptors may cause bradycardia, AV block, heart failure.

• Blockade of $\beta_2$-receptors may cause bronchospasm, cold extremities, intermittent claudication (reducing peripheral blood flow) and hypoglycemia.

• CNS effects: sleep disturbance, dreams and hallucinations (more common with lipophilic drugs which cross the BBB).
• Fatigue is probably a result of reducing of cardiac output and reduced muscle perfusion in exercise.

• Most beta-blockers raise the plasma concentration of triglycerides and lower the concentration of HDL.

• Sudden withdrawal syndrome: β-blockers should be stopped gradually.
Drug Interactions

• Propranolol + lignocaine - reduces the clearance of lignocaine by decreasing hepatic blood flow. This will lead to lignocaine toxicity.

• Beta Blockers should be used very carefully in diabetic patients who are being treated with Insulin / Sulphonylureas.
  • Propranolol masks the hypoglycemic symptoms such as tachycardia and sweating.
  • It also prevents the synthesis of new glucose molecules and breakdown of glycogen in the liver, preventing recovery from hypoglycaemia.
Thank You