<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Action</th>
<th>Use</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atropine</td>
<td><strong>Competitive Receptor Antagonist</strong></td>
<td>Competitive Muscarinic Cholinoceptor Antagonist</td>
<td>P&amp;T Lecture 3 (Drug Receptor Interactions)</td>
</tr>
<tr>
<td>Hexamethonium</td>
<td><strong>Irreversible Receptor Antagonist</strong></td>
<td>Irreversible Nicotinic Cholinoceptor Antagonist</td>
<td></td>
</tr>
<tr>
<td>Propanolol</td>
<td><strong>Competitive Receptor Antagonist</strong></td>
<td>Competitive Beta Adrenoceptor Antagonist</td>
<td></td>
</tr>
<tr>
<td>Barbiturates</td>
<td>Anti-Convulsive</td>
<td>GABA Receptor Antagonist</td>
<td>P&amp;T Lecture 4 (Mechanism of drug action)</td>
</tr>
<tr>
<td>Pilocarpine (Alkaloid)</td>
<td><strong>Muscarinic Receptor Agonist</strong> (Directly Acting Cholinomimetic Drugs)</td>
<td><strong>Partial Agonist</strong> for many muscarinic responses (Less effective on GI Smooth Muscle and the heart) Particularly useful in ophthalmology as local treatment for GLAUCOMA Side effects: Blurred Vision, Sweating, GI Disturbance/Pain, Hypotension, Respiratory Distress</td>
<td>P&amp;T Lecture 6 (Cholinomimetics)</td>
</tr>
<tr>
<td>Bethanechol (Choline Esters)</td>
<td><strong>Muscarinic Receptor Agonist</strong> (Directly Acting Cholinomimetic Drugs)</td>
<td>Minor modification of acetylcholine produces an M3 AchR Selective Agonist that is resistant to degradation. <strong>Orally active</strong>, with limited access to the brain (t1/2= 3-4h) Mainly used to assist BLADDER EMPTYING and to enhance GASTRIC MOTILITY. Side Effects: Sweating, Impaired vision, Nausea, Bradycardia, Hypotension, Respiratory Difficulty</td>
<td>P&amp;T Lecture 6 (Cholinomimetics)</td>
</tr>
<tr>
<td>Drug</td>
<td>Classification</td>
<td>Actions</td>
<td>Side Effects</td>
</tr>
<tr>
<td>--------------</td>
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<td>----------------------------------------</td>
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<tr>
<td>Physostigmine</td>
<td><strong>Reversible Anticholinesterase</strong>&lt;br&gt;(Indirectly acting Cholinomimetic Drugs)&lt;br&gt;(Other similar drugs – Neostigmine, Donepezil ‘Aricept’)</td>
<td>Naturally occurring tertiary amine from calabar beans.&lt;br&gt;Primarily acts at postganglionic parasympathetic synapse.&lt;br&gt;Non-polar, so can cross the blood brain barrier&lt;br&gt;t1/2= 30 mins&lt;br&gt;Used in the treatment of <strong>GLAUCOMA</strong> (aiding intraocular fluid drainage) and <strong>ATROPINE POISONING</strong></td>
<td></td>
</tr>
<tr>
<td>Ecothiopate</td>
<td><strong>Irreversible Anticholinesterase</strong>&lt;br&gt;(Indirectly acting Cholinomimetic Drugs)&lt;br&gt;(Other similar drugs – Dyflos, Parathion, Sarin)</td>
<td>Potent Inhibitor of acetylcholinesterase&lt;br&gt;Slow reactivation of enzyme by hydrolysis takes several days.&lt;br&gt;Used as eye drops in the treatment of <strong>GLAUCOMA (with PROLONGED DURATION OF ACTION)</strong> since it is an irreversible inhibitor (so these enzymes become permanently inhibited until new ones are made) – so help to increase intraocular fluid drainage&lt;br&gt;<strong>Systemic Side Effects:</strong> Sweating, blurred vision, GI Pain, Bradycardia, Hypotension, Respiratory difficulty</td>
<td></td>
</tr>
<tr>
<td>Donepezil &amp; Tacrine</td>
<td><strong>Non-Polar Anticholinesterases</strong>&lt;br&gt;(so can cross Blood-Brain Barrier)</td>
<td><strong>TREATMENT OF ALZHEIMER’S DISEASE</strong>&lt;br&gt;ACh is important for learning and memory.&lt;br&gt;Potentiation of central cholinergic transmission relieves AD symptoms, but does not affect degeneration.</td>
<td></td>
</tr>
</tbody>
</table>
| Hexamethonium (Previously mentioned) | **Nicotinic Receptor Antagonists**  
(acts on sympathetic and parasympathetic ganglion fibres (Nicotinic receptor between pre- and postganglionic fibres) – hence GANGLION BLOCKADE) | Blocks ion channel  
Was used as **THE FIRST ANTIHYPERTENSIVE** (no longer clinically used) | P&T Lecture 7 (Cholinoceptor Antagonists) |
|--------------------------------------|-------------------------------------------------|-------------------------------------------------|------------------------------------------|
| Trimetaphan                          | **Nicotinic Receptor Antagonists**  
(acts on sympathetic and parasympathetic ganglion fibres (Nicotinic receptor between pre- and postganglionic fibres) – hence GANGLION BLOCKADE) | Used to cause **HYPOTENSION DURING SURGERY** because it is short acting. | Lecture 7 (Cholinoceptor Antagonists) |
| Atropine                             | **Muscarinic Receptor Antagonist**  
(acts mainly on parasympathetic target organs which all have muscarinic receptors, and sweat glands in sympathetic nervous system) | Causes **MILD RESTLESSNESS**, agitation | Lecture 7 (Cholinoceptor Antagonists) |
| Hyoscine                             | **Muscarinic Receptor Antagonist**  
(acts mainly on parasympathetic target organs which all have muscarinic receptors, and sweat glands in sympathetic nervous system) | Causes **SEDATION** | Lecture 7 (Cholinoceptor Antagonists) |
| Tropicamide                          | **Muscarinic Receptor Antagonist**  
(acts mainly on parasympathetic target organs which all have muscarinic receptors, and sweat glands in sympathetic nervous system) | Examination of the retina – tropicamide is a **MYDRIATIC** | Lecture 7 (Cholinoceptor Antagonists) |

Others – oxybutynin (muscarinic receptor antagonist)
<table>
<thead>
<tr>
<th>Adrenaline/Epinephrine</th>
<th>Directly Acting Sympathomimetics (SNS Agonist)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(acts mainly on sympathetic target organs which all have adrenergic receptors)</td>
<td><strong>Selective β1/β2 agonist</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Targets (which give rise to the clinical uses):</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Blood Vessels</td>
</tr>
<tr>
<td>• Heart</td>
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<tr>
<td>• Lungs (Trachea and bronchioles)</td>
</tr>
<tr>
<td>• Eyes</td>
</tr>
<tr>
<td>• Release of hypotensive and bronchoconstrictor mediators</td>
</tr>
</tbody>
</table>

**Clinical Uses:**

- **ALLERGIC REACTION AND ANAPHYLACTIC SHOCK**
  - Hypotensive crisis and breathing difficulties (i.v. and autoinjector delivery systems)

- **COPD (CHRONIC BRONCHITIS, EMPHYSEMA) AND ASTHMA (EMERGENCIES)**
  - Bronchodilator actions
  - Suppression of mediator release

- **ACUTE MANAGEMENT OF HEART BLOCK**
  - Increased peripheral resistance; return of blood to the heart
  - Increased heart rate and force of contraction (CO)
  - (Caution for over-stimulation of heart directly or by reflex tachycardia)

- **SPINAL ANAESTHESIA (I.V.)**
  - To maintain blood pressure

- **PROLONG DURATION OF LOCAL ANAESTHESIA (LOCAL ADMINISTRATION)**
  - Vasoconstrictor properties (α1)
    - Prolongs duration of action, and minimizes dose of local anesthesia required

- **TREATMENT OF GLAUCOMA (EYE-DROPS)**
  - Glaucoma is the second leading cause of blindness worldwide
  - Often caused by raised intra-ocular pressure leading to damage to the optic nerve
  - Adrenaline may decrease aqueous humour production

Lecture 8
(SNS Agonists)
**UNWANTED ACTIONS OF ADRENALINE:**
- **Secretions** are reduced, and thickened (dry mouth)
- **Minimal effects** on CNS
- **CVS Effects:**
  - Tachycardia, palpitations, arrhythmias
  - Cold Extremities, severe hypertension
  - Overdose – cerebral haemorrhage and pulmonary oedema
- **Gastrointestinal effects:** minimal
- **Skeletal muscle:** tremor

**Pharmacokinetics of Noradrenaline/Adrenaline**
- Administer i.v., i.m., locally/topically
- Poorly absorbed orally
- Rapid metabolism in the gut, liver and other tissues
- Duration of action: minutes

**Metabolism/Breakdown**
Adrenaline is taken up by **Uptake 1 and Uptake 2**
Adrenaline is degraded by MAO and COMT → MOPEG/VMA

<table>
<thead>
<tr>
<th>Phenylephrine</th>
<th>Directly Acting Sympathomimetics (SNS Agonist)</th>
</tr>
</thead>
<tbody>
<tr>
<td>(acts mainly on sympathetic target organs which all have adrenergic receptors)</td>
<td><strong>α1&gt;&gt;α2&gt;&gt;&gt;β1/β2 (Mainly α1 Agonist)</strong></td>
</tr>
</tbody>
</table>
| 1 | Chemically related to adrenaline  
| 2 | More resistant to COMT, but not MAO  
| 3 | Clinical usefulness:  
| 4 | **VASOCONSTRICCTOR** (i.v. **TOPICALLY** i.e. restricted to an area of the body)  
| 5 | **MYDRIATIC** (eye drops)  
| 6 | α1 contracts the radial muscle of the eye, causing mydriasis (pupillary dilatation)  
| 7 | **NASAL DECONGESTANT**  
| 8 | Colds, ‘flu, hayfever – nose drops  
| 9 | Oral administration  
| 10 | Vasoconstrictor actions (in mucous membranes) |  
|  | Lecture 8 (SNS Agonists) |
### Unwanted Effects:
- Phenylephrine has unwanted effects on the CVS (hypertension)

### Clonidine
- **Directly Acting Sympathomimetics (SNS Agonist)**
- N.B. ODD ONE OUT – DECREASES SYMPATHETIC TONE
- (acts mainly on sympathetic target organs which all have adrenergic receptors)
- \(\alpha_2 >> \alpha_1 >> \beta_1/\beta_2\) (SELECTIVE FOR \(\alpha_2\) ADRENOCEPTORS)
- **Reduces Sympathetic Tone** via \(\alpha_2\) adrenoceptor mediated pre-synaptic inhibition of noradrenaline release
- **Central Action in Brainstem** within baroreceptor pathway to REDUCE SYMPATHETIC OUTFLOW
- Clinically useful in:
  - Treatment of hypertension and migraine (oral and i.v. administration)
- Lecture 8 (SNS Agonists)

### Isoprenaline
- **Directly Acting Sympathomimetics (SNS Agonist)**
- \(\beta_1 = \beta_2 >> \alpha_1/2\)
- Less susceptible to Uptake 1 and MAO than adrenaline
- Plasma half-life of 2 hours
- Clinical Usefulness:
  - **Treatment of Heart Block**
    - Cardiogenic shock, acute heart failure or myocardial infarction;
    - i.v administration
  - **Previously Used in the Treatment of Asthma**, but now discontinued due to unwanted actions (reflex tachycardia, dysrhythmias)
- Lecture 8 (SNS Agonists)

### Dobutamine
- **Directly Acting Sympathomimetics (SNS Agonist)**
- \(\beta_1 >> \beta_2 >> \alpha_1/2\) – SIMILAR TO ISOPRENALINE
- Treats heart block
- **Lacks isoprenaline’s reflex tachycardia**
- **Administer by i.v. infusion**
- Plasma half-life of 2 minutes (rapid metabolism by COMT)
- Lecture 8 (SNS Agonists)
Salbutamol (Ventolin)

Directly Acting Sympathomimetics (SNS Agonist)

$\beta_2 >> \beta_1 >> \alpha_{1/2}$

- Synthetic catecholamine derivative
- Relatively resistant to MAO and COMT

- Clinical usefulness:
  - Treatment of **ASTHMA** (Inhalation, orally)
    - Relaxation of bronchial smooth muscle
    - Inhibition of the release of bronchoconstrictor substances from mast cells
  - Treatment of threatened uncomplicated premature labour (i.v.)

- Unwanted Effects:
  - Reflex Tachycardia
  - Tremor
  - Caution with cardiac patients, hyperthyroidism and diabetes (i.v. use)

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- Unwanted Effects:
  - Reflex Tachycardia
  - Tremor
  - Caution with cardiac patients, hyperthyroidism and diabetes (i.v. use)
### Cocaine

**Indirectly Acting Sympathomimetics**

**Uptake 1 Inhibitor**

- **CNS Effects**: Euphoria, excitement, increased motor activity, activation of vomiting centres, CNS depression, respiratory failure and death
- **CVS Effects**: Tachycardia, vasoconstriction, raised blood pressure
- **Other Effects**: Tremors, Convulsions

**Clinical Uses:**
- Local anaesthetics in ophthalmology (rare)
- Caution: Do not co-administer with adrenaline

---

### Tyramine

**Indirectly Acting Sympathomimetics**

- **Competitive Uptake 1 Inhibitor**
- **Competitive MAO Inhibitor**

- Tyramine is a dietary amino acid (e.g. cheese, red wine and soy sauce)
- Acts as a ‘false’ neurotransmitter
- Not a problem when normal mechanisms for degradation of monoamines are in operation (i.e. when MAO are fully functional)

**Tyramine Actions:**
- Acts as a weak agonist at post synaptic adrenoceptors (located on effector cells)
- Competitive **Uptake 1 Inhibitor**: competes with catecholamines for uptake 1
- Competitive **MAO Inhibitor**: Competes with noradrenaline for MAO sites
- Displaces noradrenaline from intracellular vesicles into the cytosol
- Cytoplasmic noradrenaline leaks through the neuronal membrane, to act at postsynaptic adrenoceptors

Under normal conditions, this is not a problem because of:
- Extensive first-pass metabolism
- Short half-life
- Restricted access to the CNS

**But, when monoamine oxidases are inhibited, ingestion of foods containing tyramine may cause a HYPERTENSIVE CRISIS – the ‘cheese’ reaction**
<table>
<thead>
<tr>
<th>Drug</th>
<th>Type</th>
<th>Action</th>
</tr>
</thead>
</table>
| Phentolamine | Non-Selective $\alpha_1 + \alpha_2$ Antagonist | - Causes **VASODILATION** and a **FALL IN BLOOD PRESSURE** due to a blockade of $\alpha_1$ receptors  
- However, concomitant blockade of $\alpha_2$ receptors tends to **increase noradrenaline release** (as the synapse inhibitory blockade function is stopped), enhances the **REFLEX TACHYCARDIA** that occurs with any blood pressure lowering agent.  
- Increased GIT motility and diarrhoea are common  
- No longer clinically used |
| Prazosin  | Selective $\alpha_1$ Antagonist  | - **VASODILATION** and a **FALL IN ARTERIAL PRESSURE**  
- **Less tachycardia** than non-selective antagonists, since they do not increase NA release from nerve terminals (no $\alpha_2$-actions)  
- **CARDIAC OUTPUT DECREASES**, due to a fall in venous pressure, which in turn is caused by a dilatation of capacitance vessels.  
- Hypotensive effect is dramatic.  
- Does not affect cardiac function appreciably, although **postural hypotension** is problematic. Unlike other anti-hypertensives, $\alpha_1$-antagonists causes a **modest decrease in LDL, and an increase in HDL cholesterol**. Starting to become more popular as anti-hypertensive agents. |
| Propanolol | $\beta_1 + \beta_2$ Antagonist | - First major clinical $\beta$-Receptor antagonist  
- In a subject at rest, propranolol causes **very little change in heart rate, cardiac output or arterial pressure**, but **REDUCES THE EFFECT OF EXERCISE OR STRESS** on these variables  
- Being non-selective, propranolol produces all the typical adverse effects. |
| (Anti-Arrhythmics) Non-selective $\beta$-antagonist Class II Drug | | - Reduces mortality of patients with MI, particularly successful in arrhythmias during exercise/mental stress |
| **Atenolol** | **Selective β1 Antagonist** | **Historically called cardio-selective drugs.**  
**β1-Selective** so mainly antagonises the effect of noradrenaline on the heart, but will also affect any tissue with β1-receptors.  
**Less effect on airways** than non-selective drugs, but still not safe with asthmatic patients. | Lecture 9 (SNS Antagonists) |
| --- | --- | --- |
| **Labetalol** | **Non-Selective α1+β1 Antagonist**  
But more selective towards β1 than α1 | **Dual acting β1 and α1 antagonists** but more action on β1 (but higher ratio of β1:α1 4:1)  
**This drug lowers blood pressure by a reduction in peripheral resistance.**  
**No long-term change in heart rate or cardiac output.** | Lecture 9 (SNS Antagonists) |
| **Methyldopa** | **False Transmitter**  
(Acts on adrenergic α-receptors) | **ANTIHYPERTENSIVE AGENT**  
Has two key effects:  
- **Less active than norepinephrine on α1-receptors,** and so **less effective in causing vasoconstriction.**  
- **More active on presynaptic α2-receptors** (auto-inhibitory feedback mechanism) so reduces the transmitter release below normal levels.  
**Renal blood flow** is well maintained, widely used in hypertensive patients with renal insufficiency, or cerebrovascular disease.  
Recommended in **hypertensive pregnant women,** as it has **no adverse effects on the foetus** despite crossing blood-placenta barrier.  
Adverse effects – dry mouth, sedation, orthostatic hypotension, male sexual dysfunction.  
**Therefore Methyldopa is RARELY USED** due to these actions. | Lecture 9 (SNS Antagonists) |

**False Transmitter**  
(Acts on adrenergic α-receptors)  
Taken up by noradrenergic neurones, Decarboxylated and hydroxylated to form a false transmitter: α-methyl-norepinephrine  
However, NOT DEAMINATED within the neurone by MAO, and accumulates in larger quantities than norepinephrine, and displaces norepinephrine from synaptic vesicles.
<table>
<thead>
<tr>
<th><strong>Carteolol Hydrochloride</strong></th>
<th>Non Selective Blockage of β1+β2 Receptors</th>
<th><strong>TREATMENT OF GLAUCOMA</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Levobunolol Hydrochloride</strong></td>
<td></td>
<td>Reduces the <em>rate of aqueous humour formation</em> by blocking the receptors on the ciliary body – possibly blocking the effects of <em>circulating adrenaline</em>.</td>
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<td><strong>Timolol Maleate</strong></td>
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<tr>
<td><strong>Betaxolol Hydrochloride</strong></td>
<td>Selective Blockage of β1 Receptors</td>
<td><strong>β1</strong> antagonists such as <em>betaxolol hydrochloride</em> are also shown to be effective.</td>
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<tr>
<td></td>
<td><strong>Other uses:</strong></td>
<td></td>
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<tr>
<td></td>
<td>• <strong>Anxiety States</strong> – to control somatic symptoms associated with <em>sympathetic over-reactivity</em>, such as palpitations and tremor.</td>
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<td></td>
<td>• <strong>Migraine prophylaxis</strong></td>
<td></td>
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<td></td>
<td>• <strong>Benign Essential tremor</strong></td>
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<thead>
<tr>
<th><strong>Tubocurarine</strong></th>
<th><strong>Neuromuscular Blocking Drug</strong> (Acts on the somatic nervous system – NMJ)</th>
<th>Naturally occurring 4° Ammonium Compound (alkaloid) found in South American Plant (Arrow Poison) Range of synthetic drugs now available.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td><strong>NON-DEPOLARISING (COMPETITIVE) nACHR ANTAGONIST</strong></td>
<td><strong>Method of Action:</strong> Competitive nAChR Antagonist (70-80% block necessary) <strong>Graded block:</strong> Different proportions of fibres are blocked</td>
</tr>
<tr>
<td></td>
<td><strong>Uses:</strong> Relaxation of skeletal muscle during surgical operations (= less anaesthetic)</td>
<td><strong>Effects:</strong> Tubocurarine causes <strong>FLACCID PARALYSIS</strong> in the following order:</td>
</tr>
</tbody>
</table>
|                  | **Permits artificial ventilation**                                        | • Extrinsic eye muscles → double vision  
• Small muscles of face, limbs and pharynx  
• Respiratory muscles |
<p>|                  |                                                                           | Recovery occurs in the <strong>opposite</strong> direction to paralysis <strong>Note:</strong>                                                                                                                                 |</p>
<table>
<thead>
<tr>
<th>Atracurium</th>
<th>Neuromuscular Blocking Drug (Acts on the somatic nervous system – NMJ)</th>
<th>Lecture 10 (Neuromuscular Blocking Drugs)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NON-DEPOLARISING (COMPETITIVE) nACHR ANTAGONIST</td>
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<tr>
<td></td>
<td>Actions of non-depolarising blockers can be reversed by anticholinesterases</td>
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<tr>
<td></td>
<td><strong>Neostigmine (+ Atropine)</strong></td>
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<td></td>
<td><strong>Pharmacokinetics:</strong></td>
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<td></td>
<td>Route of administration – i.v.</td>
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<tr>
<td></td>
<td>Does <strong>NOT CROSS</strong> BBB or placenta</td>
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<td></td>
<td>Onset of action 2-3 minutes</td>
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<td>Duration of paralysis: 40-60min (long)</td>
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<td>Not metabolised</td>
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<td>Excretion: 70% Urine, 30% Bile (Care required if renal or hepatic function is impaired)</td>
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<td></td>
<td><strong>Unwanted Effects:</strong></td>
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</tr>
<tr>
<td></td>
<td>• Ganglion Block, Histamine Release</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• <strong>HYPOTENSION:</strong></td>
<td></td>
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<tr>
<td></td>
<td>o Ganglion blockade leads to a decrease in Total Peripheral Resistance</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o Histamine release from mast cells</td>
<td></td>
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<tr>
<td></td>
<td>• <strong>TACHYCARDIA</strong> (May cause arrhythmias)</td>
<td></td>
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<tr>
<td></td>
<td>o Reflex</td>
<td></td>
</tr>
<tr>
<td></td>
<td>o Blockade of Vagal Ganglia</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• <strong>BRONCHOSPASM</strong> – caused by histamine release</td>
<td></td>
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<tr>
<td></td>
<td>• <strong>EXCESSIVE SECRETIONS</strong> (Bronchial and salivary) – caused by histamine release</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• <strong>APNOEA</strong> (ALWAYS assist respiration)</td>
<td></td>
</tr>
<tr>
<td>Suxamethonium</td>
<td><strong>Neuromuscular Blocking Drug</strong> (Acts on the somatic nervous system – NMJ)</td>
<td><strong>DEPOLARISING nAChR AGONIST</strong></td>
</tr>
</tbody>
</table>