Autonomic Nervous System &
Neuromuscular Junction
Review
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Tips for Drug Learning
• Learn agents by drug classes
  ie. beta-blockers, Ca++-blockers etc
• Key points:
  - Clinical application
  - Mechanism of action
  - Important drug/drug interactions
    ie. cardiac glycosides & thiazides
  - Adverse drug reactions
    ie. beta-blockers – asthma
• Drugs of choice (DOC)
  ie. epinephrine (anaphylaxis)
• Prototype drugs
  ie. propranolol, atropine
• Do not focus on dosage or trade names

ANS – Review
1. Tissues / Organs: - receptors present,
tissue / organ response
2. Transmitters: - NE, Ach, synthesis, storage,
release, regulation
3. Eye: - miosis, mydriasis, cycloplegia,
Glaucoma: wide- vs narrow-angle,
Horner’s Syndrome
4. Drugs: - receptor selectivity,
mechanism of action
5. Can predict: - clinical application, side effects,
toxicity, treatment of toxicity
6. General: - learn by drug classes, important
adverse reactions, not dosage

Neurons of the ANS

Transmitter synthesis and release

Receptors of the ANS
Adrenoceptors  Cholinceptors
Selective agents available  Selective agents available for major
groups but not for M-receptor subtypes
**ANS - Review Tissues/Organs**

<table>
<thead>
<tr>
<th>Action</th>
<th>Tissues/Organs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathetic</td>
<td>Cardiovascular System, GI, Spleen, Liver, Tissue perfusion</td>
</tr>
<tr>
<td>Parasympathetic</td>
<td>Eye, Salivary glands, Gastrointestinal tract, PNS (ganglia, spinal nerves)</td>
</tr>
<tr>
<td>Neural</td>
<td>CNS, PNS (ganglia, spinal nerves)</td>
</tr>
</tbody>
</table>

**ANS Excess / Deficiency**

- Cholinergic excess: (ie. AchE inhibition or mushrooms) salivation, lacrimation, urination, diarrhea, emesis (sludge), miosis, |HR|, sweating, cycloplegia
- Cholinergic deficiency: (ie. Atropanicity) ↓GI motility, mydriasis, cycloplegia, |secretions|, tachycardia, delirium
- Sympathetic excess: (ie. Tyr-MAOI or Pheochrom.) ↑BP, ↑HR, pupil dilation (mydriasis), ↓GI motility
- Sympathetic deficiency: (ie. Guanethidine-block) ↓BP, ↓tissue perfusion, pupil constriction (miosis)

**Terminology – ANS**

i. clammy: → PNS excess, ie. ↑|secretion| (sweating)
ii. wheezy: → PNS excess, ie. difficulty in breathing (↑|bronchial resistance|, ↑|secretion|)
iii. flushed: → PNS deficiency ie. vasodilation, thermoregulation (atropanicity)
iv. cramps: → muscle contraction ie. abdominal (↑|PNS|)
v. palpitations: → SNS excess ie. hypertension, hypertensive crisis, MAOI-Tyramine or pheochromocytoma

**Question 1**

A 32-year old man is brought to the emergency department because of confusion, wheezing, vomiting and diarrhea for the past 6 hours. He is sweating and salivating profusely. There is generalized muscle weakness. Which of the following substances is the most likely cause of these findings?

A. Glutethimide
B. Heroin
C. Jimson weed (belladonna alkaloids)
D. Parathion
E. Phencyclidine (PCP)

**Drugs of Choice**

<table>
<thead>
<tr>
<th>Agent</th>
<th>Therapeutic use</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Epinephrine</td>
<td>Augments sympathetic activity in low dose</td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Block in angles</td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
</tr>
<tr>
<td>Timolol</td>
<td>β-blockers</td>
<td>Selectively blocks β1 receptors, reduces heart rate, decreases myocardial oxygen demand, decreases blood pressure, decreases heart rate, reduces myocardial oxygen demand, decreases blood pressure.</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>Norepinephrine, PE, MethM2, M3</td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
</tr>
<tr>
<td>Pilocarpine</td>
<td>Emergency glaucoma</td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>Emergency glaucoma</td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
</tr>
<tr>
<td>Propranolol</td>
<td></td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
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<tr>
<td>Dantrolene</td>
<td>Malignant hyperthermia</td>
<td>Increases myocardial contractility and stroke volume, decreases heart rate, produces a mixed α- and β-agonist effect on the myocardium, increases heart rate, dilates coronary arteries, constricts veins, causes vasoconstriction, increases blood pressure, decreases heart rate, increases myocardial contractility, reduces heart rate, constricts coronary arteries, dilates veins, decreases blood pressure.</td>
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**Question 2**

A 59-year old man develops excessive sweating and salivation, diarrhea, and Bradycardia while being treated with neostigmine for myasthenia gravis. Which of the following is the most appropriate therapy for these symptoms and signs?

A. Atropine
B. Carbachol
C. Endrophonium
D. Epinephrine
E. Pralidoxime

USMLE Step 1: 2003
Labetalol, Carvedilol (Propranolol)

-Blockers

β-blocker or α-blocker

Glaucoma

β₁-selective

- Open angle (wide, chronic) – treated with beta-blockers and other agents

- Closed-angle (narrow-angle) – dilated iris can occlude outflow

Increased intraocular pressure: Untreated → blindness

Glaucoma treatment

1. α-Agonist: T-Outflow
2. M-Agonists: T-Outflow
3. β-Blocker: ↓Secretion
4. α₂-Agonist: ↓Secretion
5. Prostaglandins: T-Outflow
6. Carbonic acid inhibitors: ↓Secretion

Gluconic Acid

Increased intracellular pressure

Seeley's syndrome

Diabetes mellitus, congenital heart disease

Left heart failure

Shock

Atropine

Hypertensive crisis

Tyramine effect

Albuterol

Ritodrine

β₂-selective agonists

- bronchodilation

Inhalation vs oral

- less side effects

Ritodrine

- premature labor

Horners Syndrome

Destruction of Sympathetic innervation to the iris

- loss of preganglionic fibers

- loss of postganglionic fibers

- parasympathetic innervation left unopposed

Horners Syndrome (note sagging left eyelid and miosis)
Question 3
The circles represent the size of the pupils of a patient's right and left eyes, both without treatment and with two different treatments. Which of the following is compatible with the findings shown for the left eye?

A. Blockade of α-adrenergic rec.
B. Blockade of β-adrenergic rec.
C. Blockade of muscarinic rec.
D. Inhibition of cholinesterase
E. Sympathetic denervation

Without treatment
Treatment With TYR
Treatment With EPI

Question 4
Which of the following drugs applied topically produces mydriasis without producing cyclopia?

A. Atropine
B. Neostigmine
C. Phentolamine
D. Phenylephrine
E. Pilocarpine

Drugs of Choice

<table>
<thead>
<tr>
<th>Agent</th>
<th>Therapeutic use</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phentolamine</td>
<td>M-agonist causes ↑ outflow</td>
<td>Emergency glaucoma, Pilocarpine</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Pressor agent (α1-receptors) to maintain BP but dilates renal vessels</td>
<td>D1-receptors, NE → renal vasoconstriction</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>Reverse toxic effects of AchE inhibitors ie. neostigmine, physostigmine</td>
<td>Reverse AchE inhibition</td>
</tr>
<tr>
<td>Atropine</td>
<td>Need to use before 'aging' occurs</td>
<td>Regenerate AchE enzyme</td>
</tr>
<tr>
<td>Pseudocholinesterase</td>
<td>Reversible AchE inhibitor, can cross CNS</td>
<td>Reversible atropine toxicity</td>
</tr>
<tr>
<td>Pralidoxime</td>
<td>Inhibits calcium released from SR</td>
<td>Reversible AchE inhibitor</td>
</tr>
<tr>
<td>Dantrolene</td>
<td>Inhibits calcium release from SRM</td>
<td>Malignant hyperthermia</td>
</tr>
<tr>
<td>Physostigmine</td>
<td>Inhibits calcium release from SRM</td>
<td>Malignant hyperthermia</td>
</tr>
</tbody>
</table>

Pheochromocytoma
Tumor: ↑ synthesis, Release of NE & EPI into the circulation. Result: ↑BP, ↑HR → hypertensive crisis
Treatment:
- Surgical removal for solid tumor
- α-β-blocker ie. Labetalol, Carvedilol
- α-blocker ie. phenoxbenzamine or phentolamine
- inhibitor of tyrosine hydroxylase ie. α-methyl-p-tyrosine
- β-blocker only after α-blockade

Rule of Ten
10% Pheochromocytomas are:
- Malignant
- Extra-Adrenal
- In Children
- Familial
- Present after stroke

Tyramine Interaction with MAO Inhibitors
Can cause hypertensive crisis (↑BP, ↑HR)
Aged cheese & red wine are rich in tyramine

Parkinson’s Disease
- General population 1:1000, over 60 1:75
- Tremor, stiffness, or clumsiness, usually involving one side difficulty walking, fatigue, depression
- Progressive destruction of the dopaminergic nigrostriatal pathway
- Elevated cholinergic activity

Treatment:
- MAO inhibitors:
- Dopamine agonists: bromocriptine
- L-Dopa
- Anticholinergics: benzphetamine
- Decarboxylase inhibitor: carbidopa
- Amantadine: Inhibit D-uptake, M-rec, NMDA-block, release dopamine
Drugs of Choice

**Epinephrine**
Acute hypersensitivity reaction to bee stings.

**Atropine**
Reverse toxic effects of AchE inhibitors (neostigmine, physostigmine, organophosphates).

**Pralidoxime**
Reversible AchE inhibitor, can cross CNS.

**Pilocarpine**
M-agonist causes ↑ outflow.

**β-Blockers**

- **Contraindications**:
  - Supersensitivity: Rebound effect with β-blockers, less with β-blockers with partial agonist activity (ie. pindolol). Gradual withdrawal.
  - Asthma: Blockade of pulmonary β2-receptors. β1-selective agents preferred.
  - Diabetes: Compensatory hyperglycemic effect of EPI in insulin-induced hypoglycemia is removed by block of β2-ARs in liver. β1-selective agents preferred.
  - Caution in Congestive Heart Failure: β-blockers can precipitate latent heart failure by removing compensatory increase in sympathetic effects on heart.

- **Notes**
  - Therapeutic use: Agent

- **Question 5**
The histograms show changes in HR and bronchiolar resistance produced by the administration of epinephrine alone, drug X alone, and epinephrine together with drug X. Drug X is most likely to be?

  - A. Isoproterenol
  - B. Metoprolol
  - C. Nadolol
  - D. Pindolol
  - E. Propranolol

- **USMLE Step 1: 2003**

**β-Blockers: Contraindications**

- ** Supersensitivity:** Rebound effect with β-blockers, less with β-blockers with partial agonist activity (ie. pindolol). Gradual withdrawal.
- **Asthma:** Blockade of pulmonary β2-receptors. β1-selective agents preferred.
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- **Caution in Congestive Heart Failure:** β-blockers can precipitate latent heart failure by removing compensatory increase in sympathetic effects on heart.

**Notes:** unstable CHF, depression, significant bradycardia or bronchospasm.

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**Beta-Adrenoceptor Blocking Agents (-olol)**

**Properties of several beta-receptor blocking drugs**

<table>
<thead>
<tr>
<th>Selection</th>
<th>Partial Agonist Activity</th>
<th>Local Anesthetic Action</th>
<th>Lipolytic Activity</th>
<th>Elimination Half-life</th>
<th>Approximate Benzodiazepine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amodipine</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2-4 hours</td>
<td>5</td>
</tr>
<tr>
<td>Amdolol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>3-4 hours</td>
<td>10</td>
</tr>
<tr>
<td>Bicalane</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>20</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>30</td>
</tr>
<tr>
<td>Domapim</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>40</td>
</tr>
<tr>
<td>Emoda</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>50</td>
</tr>
<tr>
<td>Epometol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>60</td>
</tr>
<tr>
<td>Labetalol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>70</td>
</tr>
<tr>
<td>Lidopim</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>80</td>
</tr>
<tr>
<td>Mepapim</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>90</td>
</tr>
<tr>
<td>Nadolol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>100</td>
</tr>
<tr>
<td>Prifelol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>110</td>
</tr>
<tr>
<td>Propranolol</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>1-2 hours</td>
<td>120</td>
</tr>
</tbody>
</table>

- *Beta agonist effect at β2 receptors. Labetalol also cause α1 and β1 blockade. Tramadol is dose-dependent.

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**Question 6**

In a hypertensive patient with congestive heart failure, which of the following drugs is CONTRAINDICATED in treatment of the hypertension?

- (A) Captopril
- (B) Chlorothiazide
- (C) Methylpria
- (D) Prazosin
- (E) Propranolol

- **USMLE Step 1: 1998**

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**Hypertension**

<table>
<thead>
<tr>
<th>Stage</th>
<th>Systolic (mmHg)</th>
<th>Diastolic (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>normal</td>
<td>&lt;130</td>
<td>&lt;85</td>
</tr>
<tr>
<td>high normal</td>
<td>130-139</td>
<td>85-90</td>
</tr>
<tr>
<td>stage 1 (mild)</td>
<td>140-159</td>
<td>90-99</td>
</tr>
<tr>
<td>stage 2 (moderate)</td>
<td>160-179</td>
<td>100-109</td>
</tr>
<tr>
<td>stage 3 (severe)</td>
<td>180-209</td>
<td>110-119</td>
</tr>
<tr>
<td>stage 4 (very severe)</td>
<td>&gt;209</td>
<td>&gt;119</td>
</tr>
</tbody>
</table>

- *Require three measurements (repeat visits) BP lowest in the morning → ↑ during the day.

**β-Blockers** - Frontline agents

**α-Blockers** - Hypertensive crisis, special circumstances

**α1-Agonists** - Useful, not frontline. Clonidine

**Reserpine** - Resistant hypertension, significant side effects, rarely used

**Guanethidine** - Resistant hypertension, significant side effects, rarely used.
Drugs of Choice

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<tr>
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<tr>
<td>Atropine</td>
<td>Acetylcholinesterase &amp; irreversible inhibition</td>
<td>No cns action</td>
</tr>
<tr>
<td>DFP, Isoflurophate, Malathion, Parathion</td>
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</tr>
</tbody>
</table>

Myasthenia gravis
Autoimmune disease

1:10,000 (250,000 USA)
- antibodies to NMJ nicotinic receptors leads to degradation
- simplified synaptic folds
- normal nerve terminal and transmitter
- wider synaptic junction

Diagnosis: Edrophonium (Tension, short acting) is used for diagnosis and determination of maintenance dose

Treatment: Neostigmine has direct (stimulates receptor) and indirect actions (inhibition of AchE). No cns activity.

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<td>No cns action</td>
</tr>
</tbody>
</table>

Deadly Nightshade

Approx 5,000 per yr
- Mainly atropine
- Devil's apple
- Stink weed
- Devil's cherries

Datura
- Mainly scopolamine & hyoscyamine
- Thorn apple
- Jimson weed

Acetylcholinesterase & Irreversible Inhibition

DFP, Isoflurophate, Malathion, Parathion

2-PAM, Pralidoxime
No cns action
Aging
30-40 min
Nerve gas secs / min
Parathion, Malathion (prodrugs)
4 – 6 hr

US Military 2-PAM / Atropine Injector

2.5 mg Atropine, 600mg 2-PAM
**Wild Mushrooms - Amanita**

10,000 cases per year
Muscarine poisoning
5,000 mushroom species
100 “bad”, 10 “deadly”

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**Drugs of Choice**

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</tr>
</thead>
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<td>Ephedrine</td>
<td>Acute hypercapnia toxicity in low arms</td>
<td>α-agonists; olefins elevate the effects of released catecholamines. MBP</td>
</tr>
<tr>
<td>Succinylcholine</td>
<td>Short-term depolarization, major effect on muscarinic receptors. MBP, hyperkalemia, malignant hyperthermia</td>
<td></td>
</tr>
<tr>
<td>Tubocurarine</td>
<td>Non-depolarizing, reversible, long-acting. MBP</td>
<td></td>
</tr>
<tr>
<td>Pancuronium</td>
<td>Non-depolarizing, reversible, long-acting. MBP</td>
<td></td>
</tr>
<tr>
<td>Vecuronium</td>
<td>Non-depolarizing, reversible, long-acting. MBP</td>
<td></td>
</tr>
<tr>
<td>Pipecuronium</td>
<td>Non-depolarizing, reversible, long-acting. MBP</td>
<td></td>
</tr>
<tr>
<td>Mivacurium</td>
<td>Non-depolarizing, reversible, long-acting. MBP</td>
<td></td>
</tr>
<tr>
<td>Clonidine, etc</td>
<td>Neostigmine or physostigmine. MBP</td>
<td></td>
</tr>
</tbody>
</table>

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**Prototype Drugs**

ie. Propranolol
Metoprolol
Tubocurarine
Succinylcholine
Clonidine
etc

---

**Neuromuscular Junction**

Succinylcholine (non-competitive):
- depolarizing (only agent), cannot reverse, short-acting, fasciculations
- atypical pseudo-AchE, hyperkalemia, malignant hyperthermia

Tubocurarine (competitive):
- non-depolarizing, reversible, long-acting, relaxed paralysis

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**Neuromuscular Blocking Agents**

<table>
<thead>
<tr>
<th>Ganglia</th>
<th>Muscarinic Receptors</th>
<th>Histamine Release</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>

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**NMJ – Onset, Duration & Elimination**

Onset, Duration and Elimination of Neuromuscular Blocking Drugs
ANS – Cardiovascular Receptors

Blood Pressure = Cardiac Output X TPR
Cardiac Output = Heart rate X Stroke volume

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Response</th>
<th>Effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>alpha₁</td>
<td>vasoconstriction</td>
<td>↑ TPR</td>
</tr>
<tr>
<td>beta₁</td>
<td>heart rate</td>
<td>↑ CO</td>
</tr>
<tr>
<td>beta₂***</td>
<td>vasodilation</td>
<td>↓ TPR</td>
</tr>
<tr>
<td>M₂ (vagus)</td>
<td>heart rate</td>
<td>↓ CO</td>
</tr>
<tr>
<td>M (vascular)**</td>
<td>vasodilation</td>
<td>↓ TPR</td>
</tr>
</tbody>
</table>

** not innervated

Cardiovascular Responses

Moderate/high doses:
- NE  ↑BP, ↑HR (reflex)
- EPI  ↑BP, ↑HR
- ISO  ↓BP, ↑HR
- ACH  ↓BP, ↓HR

- Epinephrine reversal (EPI response in presence of α-blocker ie. phentolamine)
- Norepinephrine in presence of atropine (↑BP, ↑HR)

Cardiovascular Actions – Low dose

Phenylephrine
- α-agonist, PP constant
Epinephrine
- α-β-agonist, ↑ PP
Isoproterenol
- β-agonist, ↑ PP

Epinephrine Reversal
Phentolamine
- α-antagonist
- PP, ↓BP, ↑HR (reflex)

In the presence of phentolamine, epinephrine now causes ↓BP
**Question 7**

A 65-year old woman on holidays is stung by a bee. She goes to the first aid station where she receives an intramuscular injection of epinephrine. A few minutes later she develops a pounding headache, and blood pressure is measured at 250/150 mmHg. Which of the following drugs might the patient have previously taken that could account for this unexpected effect?

A. atropine  
B. clonidine  
C. alpha-methyldopa  
D. prazosin  
E. propranolol

**Answer C**

Original workup showed high levels of the EPI metabolite and lower levels of the NE metabolite. This indicates the tumor produced almost pure EPI and little NE. This is rare but has been reported. Most Pheochromocytoma produce a mixture of EPI and NE.

A patient with a tumor like this would exhibit a dramatic ‘Epinephrine reversal response’ to any alpha-blocker, dropping the BP to shock levels. This is especially true if blood volume is low (ie. dehydrated, vomiting).

**Question 8**

A new agent (X) was tested for it’s cardiovascular actions in three anaesthetized animals.

i. Control animal  
ii. Animal treated with a ganglion blocker  
iii. Animal treated with a muscarinic blocker

Drug X caused a 50 mmHg rise in BP in the control animal, no change in BP in the ganglion blocker treated animal and a 75 mmHg rise in the muscarinic blocker treated animal. Drug X is a drug similar to?

A. Acetylcholine  
B. Atropine  
C. Mecamylamine  
D. Epinephrine  
E. Nicotine

**Question 9**

A new agent (Y) was tested for it’s cardiovascular actions in three anaesthetized animals.

i. Control animal  
ii. Animal treated with a ganglion blocker  
iii. Animal treated with a muscarinic blocker

Drug Y caused a 40 mmHg fall in BP in the control animal, a 50 mmHg fall in BP in the ganglion blocker treated animal and no change in BP in the muscarinic blocker treated animal. Drug Y is a drug similar to?

A. Acetylcholine  
B. Edrophonium  
C. Mecamylamine  
D. Pralidoxime  
E. Nicotine

**Question 14**

A 38-yr woman has symptoms of Pheochromocytoma. Urine VMA is elevated but normetanephrine level is below normal. BP is 230/150 mmHg, HR is 150 bpm. She is dehydrated and has been vomiting. She is given Phentolamine iv. Ten minutes later her BP is 40/0 and she goes into shock. She fails to respond to vasoconstrictors and dies after 6hr. What would explain the exaggerated response to Phentolamine?

A. Patient had limited ANS control of BP  
B. Low secretion of catecholamines from tumor, hence dose of phentolamine was high  
C. Patient’s tumor secreted almost pure EPI and little NE  
D. Tumor had metastasized to the vasomotor center in the medulla

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**Actions on the Eye**

- **Glucoma treatment**
  1. α-Agonist  
  2. M-Agonists  
  3. β-Blocker  
  4. α2-Agonist  
  5. Prostaglandins  
  6. Carbonic acid inhibitors

- **Outflow**

- **Secretion**
A 60-year-old asthmatic man comes in for a check-up and complains that he is having some difficulty in voiding urine. Physical examination indicates that the man has a blood pressure of 160/100 mmHg and a slightly enlarged prostate. Which of the following medications would be useful in treating both the hypertension and the enlarged prostate?

A. prazosin  
B. propranolol  
C. clonidine  
D. atenolol  
E. isoproterenol

Benign Prostate Hypertrophy (BPH)

Enlarged prostate leads to difficulty in urination
Alpha-receptor blocker (ie Prazosin) causes prostate relaxation
Relaxed prostate improves urination

Dr. Ishac, may I be excused my brain is full.

Good luck