

Exercise walk-through

11.3

Autonomic pharmacology

University Medicine

Autonomic pharmacology: one map

- 1 **Rows** $\alpha 1$ constricts/dilates pupil · $\beta 1$ heart · $\beta 2$ airway · M3 secretes and slows.
- 2 **Predictions** $\beta 1$ blocker $\downarrow$ rate; $\alpha 1$ blocker $\downarrow$ pressure (postural); $\beta 2$ agonist opens airways; M blocker dries.
- 3 **Toxidromes** Cholinergic = wet, slow, miosis · anticholinergic = dry, blind, fast · adrenergic = fast, tight, sweating.

Receptor-to-effect map

the map, read upside down, is the toxidrome — every finding is a receptor row

α1	vasoconstriction · mydriasis	agonist $\uparrow$ pressure; blocker $\downarrow$ (postural drop)
β1	heart rate and contractility	agonist $\uparrow$ output; blocker $\downarrow$ rate
β2	bronchodilation · vasodilation	agonist opens airways
M3	glands · gut · miosis · slow heart	agonist = wet and slow; blocker = dry, fast

CHOLINERGIC

wet and slow: salivation, miosis, bronchorrhoea, bradycardia, cramps

ANTICHOLINERGIC

dry, blind, fast: dry mouth, fixed dilated pupils, tachycardia, retention

ADRENERGIC

fast and tight: tachycardia, high pressure, mydriasis, sweating

One map, four receptor rows — every autonomic drug is an arrow into it.

Exercises 2.1-2.3

2.1 Which receptor does a bronchodilator β -selective agonist avoid? [3]

2.2 Name three anticholinergic findings. [3]

2.3 Why does an α 1 blocker cause postural hypotension? [3]

Exercises 2.1-2.3

2.1 Which receptor does a bronchodilator β -selective agonist avoid? [3]

2.1

- β_1 – selectivity spares the heart ✓.
-

2.2 Name three anticholinergic findings. [3]

2.2

- Dry mouth, fixed dilated pupils, tachycardia, urinary retention (any three) ✓.
-

2.3 Why does an α_1 blocker cause postural hypotension? [3]

2.3

- α_1 keeps venous and arterial tone; blocked, standing pools blood below the heart ✓.

Exercise 3.1

3.1 Four fictional INN teaching drugs: a β_1 blocker, an α_1 blocker, a β_2 agonist, an M blocker.

Predict two effects of each straight off the receptor map (β_1 blocker: rate and contractility $\downarrow$; α_1 blocker: pressure $\downarrow$ with postural drop; β_2 agonist: airway opens, little cardiac effect; M blocker: dry mouth, blurred vision) ✓. Explain why a cardioselective β_1 blocker is preferred over non-selective in a patient with airway disease ✓. [8]

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3.1

- β_1 blocker: heart rate and contractility fall ✓.
- α_1 blocker: pressure falls, postural drop ✓.
- β_2 agonist: bronchi open, cardiac effect minimal ✓.
- M blocker: secretions dry, accommodation blurs ✓.
- β_1 selectivity avoids β_2 airway blockade in airway disease ✓.

Exercise 3.2

3.2 Two fictional overdoses: patient A has pinpoint pupils, sweating and slow gut; patient B is dry, dilated and tachycardic.

Assign each toxidrome (cholinergic; anticholinergic) with the receptor rows that generate every finding ✓. Explain why patient A's bronchorrhoea is the dangerous feature for ventilation ✓.

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3.2

- A: cholinergic – M rows firing (miosis, secretions, gut) ✓.
- B: anticholinergic – M rows silenced (dry, dilated, fast, retention) ✓.
- Bronchorrhoea floods the airway and impairs ventilation – the dangerous row ✓.