Multiple Choice Questions in Pharmacology

with answers and explanatory comments

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Introduction

Multiple choice questions (MCQs) are increasingly used in examinations as they allow a wide range of knowledge to be tested and are an objective means of assessing students. There is still an opinion prevalent that MCQs measure only factual recall, and teachers may therefore be interested in attempts made in this book to formulate questions which test knowledge of concepts as opposed to drug names. We have found that such questions can be used to identify learning difficulties encountered by students, whether these be problems of remembering or distinguishing drug names, or difficulties in understanding concepts or conventional terms. For example, Questions 5 and 6 deal with receptors involved in eliciting particular responses in the eye. Questions 7 and 8 are linked with Questions 5 and 6 in that they test knowledge of the same responses, except that the stems of the questions introduce named drugs. Many of the questions on central nervous system (CNS) pharmacology have a similar design to those on the autonomic nervous system, and they incorporate ideas on the interactions of drugs with receptors.

The questions in this book have been selected from among those which have been evaluated on preclinical medical students at various stages of their course in pharmacology at The London Hospital Medical College. Some questions appear considerably easier than others; it should be remembered that they would have been presented to students at different stages of their course. The conventional construction of MCQs has been used throughout. A stem is followed by five possible alternatives, any number of which may be true or false and each one of which should be attempted. The correct response with comments appear on the facing page.

We hope that this book will help students to overcome their well-known apprehension of MCQs, and that teachers may be encouraged to use MCQs more frequently to assess learning and understanding not only in formal examinations but also in practical classes and demonstrations. We have used MCQs related to some practical classes, and students have found them more beneficial than lengthy formal laboratory reports.

Acknowledgement. We would like to thank Elaine Lockhart for her meticulous and patient typing of the manuscript.

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Contents

1 In noradrenergic neurons:
(a) tyrosine is a precursor of noradrenaline
(b) the rate-limiting step in the synthesis of noradrenaline is at tyrosine hydroxylase
(c) dopamine is decarboxylated to noradrenaline
(d) L-DOPA is converted to dopamine inside the storage vesicle
(e) isoprenaline is synthesized from noradrenaline.

2 Acetylcholine:
(a) is synthesized by acetylcholinesterase
(b) is released only from parasympathetic nerves
(c) is inactivated by re-uptake into nerves
(d) releases adrenaline from the adrenal medulla
(e) is released from sympathetic nerves to the sweat glands.

3 Blockade of nicotinic receptors in autonomic ganglia is likely to:
(a) increase salivary secretion
(b) reduce diastolic blood pressure
(c) reduce intestinal contractions
(d) paralyse striated respiratory muscles
(e) cause blurring of vision.

4 Blockade of α_1-adrenoceptors:
(a) prevents an increase in heart rate due to sympathetic stimulation
(b) causes a fall of blood pressure
(c) can lead to orthostatic hypotension
(d) can cause nasal 'stuffiness'
(e) is caused by propranolol.

1 (a) **True** L-Tyrosine $\xrightarrow{\text{tyrosine hydroxylase}}$ L-DOPA $\xrightarrow{\text{DOPA decarboxylase}}$ dopamine $\xrightarrow{\text{dopamine β-hydroxylase}}$ noradrenaline.

(b) **True** Tyrosine hydroxylase (and hence the rate of noradrenaline synthesis) is influenced by:
(i) end-product inhibition by noradrenaline,
(ii) presynaptic receptors (see Question 16),
(iii) axonal nerve traffic to terminals.

(c) **False** See (a).

(d) **False** This reaction occurs in the cytoplasm.

(e) **False**

2 (a) **False** Acetylcholinesterase inactivates acetylcholine.

(b) **False** It is also released:
(i) from postganglionic sympathetic nerves to sweat glands,
(ii) from preganglionic nerves to all autonomic ganglia,
(iii) in the CNS,
(iv) from voluntary nerves to skeletal muscle.

(c) **False** It is inactivated enzymatically:
acetylcholine $\xrightarrow{\text{acetylcholinesterase}}$ choline + acetate.

(d) **True** It acts at nicotinic receptors at this site.

(e) **True** See (b) above.

3 (a) **False** Salivary secretion is decreased by ganglion blockers (hexamethonium, mecamylamine, pempidine) acting at parasympathetic ganglia. Ganglion blockers are *not* selective for sympathetic or parasympathetic ganglia.

(b) **True** This is an effect due to blockade of sympathetic ganglia.

(c) **True** An action at parasympathetic ganglia.

(d) **False** Skeletal muscle innervation is separate from the autonomic nervous system.

(e) **True** An action at parasympathetic ganglia to the ciliary muscles and iris.

4 (a) **False** An increase in heart rate is mediated by activation of β_1-adrenoceptors.

(b) **True** Sympathetic vasoconstrictor tone is mediated by α_1-adrenoceptors.

(c) **True** The baroreceptor reflex cannot operate because the α_1-adrenoceptors in resistance arterioles are blocked and therefore vasoconstriction cannot occur.

(d) **True** Vasoconstriction (by α-adrenoceptor agonists) in the nasal mucosa causes decongestion.

(e) **False** Propranolol is a β-adrenoceptor blocker. Examples of α-adrenoceptor blockers include prazosin, phentolamine and phenoxybenzamine.

5 Excitation of α-adrenoceptors:
(a) contracts the radial smooth muscle fibres in the iris
(b) contracts the circular smooth muscle fibres in the ciliary muscle
(c) retracts the upper eyelid
(d) causes vasoconstriction of conjunctival blood vessels
(e) causes dilatation of the pupil.

6 Excitation of muscarinic receptors:
(a) contracts the radial smooth muscle fibres in the iris
(b) contracts the circular smooth muscle fibres in the ciliary muscle
(c) retracts the upper eyelid
(d) can cause vasodilatation of conjunctival blood vessels
(e) is a mechanism whereby the pupil constricts in bright light.

7 Phenylephrine eye drops:
(a) constrict the pupil
(b) move the near point for accommodation nearer
(c) retract the upper eyelid
(d) cause vasodilatation of conjunctival blood vessels
(e) may prevent the pupil from dilating in dull light.

8 Pilocarpine eye drops:
(a) constrict the pupil
(b) move the near point for accommodation further away
(c) retract the upper eyelid
(d) cause vasoconstriction of conjunctival blood vessels
(e) may prevent the pupil from dilating in dull light.

9 Blockade of β-adrenoceptors:
(a) results in an increase in heart rate
(b) results in a rise in blood pressure
(c) prevents the effect of noradrenaline on the isolated heart
(d) causes vasodilatation in skeletal muscle
(e) is caused by phentolamine.

	(a)	(b)	(c)	(d)	(e)
5	**True**	**False**	**True**	**True**	**True**
6	**False**	**True**	**False**	**True**	**True**
7	**False**	**False**	**True**	**False**	**False**
8	**True**	**False**	**False**	**False**	**True**

		Innervation	Receptor	Response
Iris	Radial smooth muscle	Sympathetic adrenergic	α-adrenoceptor	Contraction of radial muscle and dilatation of pupil
	Circular smooth muscle	Parasympathetic cholinergic	Muscarinic	Contraction of circular muscle and constriction of pupil
Ciliary body	Circular smooth muscle	Parasympathetic cholinergic	Muscarinic	Contraction of ciliary muscle resulting in less tension on the lens which becomes more spherical. The near point for accommodation moves nearer
Upper eyelid	Smooth muscle of palpebrae superioris	Sympathetic adrenergic	α-adrenoceptor	Contraction leads to retraction of upper eyelid
Blood vessels of conjunctiva	Circular smooth muscle	Sympathetic adrenergic	α-adrenoceptor	Vasoconstriction, blanching of conjunctiva
	Smooth muscle	?	Muscarinic	Vasodilatation, conjunctiva appears red (bloodshot)

Pilocarpine is a muscarinic receptor agonist.
Phenylephrine is an α-adrenoceptor agonist.

9 (a) **False** β-adrenoceptor agonists cause an increase in force and rate of contraction of the heart.
(b) **False** β-adrenoceptor blockers are used as antihypertensives (see Questions 10, 11, 12).
(c) **True** In the heart, noradrenaline acts at β_1-adrenoceptors.
(d) **False** Stimulation of β_2-adrenoceptors in skeletal muscle leads to vasodilatation.
(e) **False** Phentolamine is an α-adrenoceptor blocker. Propranolol, oxprenolol and sotalol are β-adrenoceptor blockers.

10 β-adrenoceptor blockers have the following properties.
(a) Oxprenolol has intrinsic sympathomimetic activity.
(b) When used as hypotensives, β-blockers often cause orthostatic hypotension.
(c) Propranolol can reduce skeletal muscle tremor in thyrotoxicosis.
(d) Propranolol is used in the treatment of angina.
(e) They can cause heart block or bronchospasm in susceptible individuals.

11 The following statements are true.
(a) Renin is an enzyme.
(b) Angiotensin I is converted to angiotensin II in the lungs.
(c) Angiotensin II can promote Na^+ retention.
(d) Angiotensin II is a potent vasodilator.
(e) Captopril is a competitive inhibitor of renin.

12 β-adrenoceptor blocking compounds may lower blood pressure by the following mechanisms.
(a) Decreasing sympathetically mediated renin release.
(b) Causing vasodilatation in skeletal muscle.
(c) Actions in the CNS.
(d) Decreasing noradrenaline release from sympathetic nerve endings.
(e) Reduction of circulating fluid volume.

10 (a) **True** Alprenolol, oxprenolol, pindolol and practolol are partial agonists, and are said to have intrinsic sympathomimetic activity (ISA).

(b) **False** This is less likely to occur with β-blockers than with other antihypertensive drugs such as adrenergic neuron blocking agents, probably because reflex vasoconstriction is still possible.

(c) **True** β-adrenoceptor agonists cause tremor by actions on skeletal muscle cells. Tremor caused by the release of adrenaline in thyrotoxicosis can be reduced by propranolol.

(d) **True** β-adrenoceptor blockers reduce the frequency of anginal attacks by decreasing cardiac stimulation provoked by exercise and anxiety.

(e) **True**

11 (a) **True** The decapeptide angiotensin I is formed from angiotensinogen by renin released from the kidney.

(b) **True** The converting enzyme producing the octapeptide angiotensin II is mainly present in the lungs.

(c) **True** Angiotensin II stimulates the synthesis and secretion of aldosterone by the adrenal cortex, which causes retention of Na^+.

(d) **False** Angiotensin II is a vasoconstrictor, and on a molar basis is about 40 times more potent as a pressor agent than noradrenaline.

(e) **False** Captopril inhibits the enzyme involved in converting angiotensin I to angiotensin II. Captopril is used to treat severe hypertension refractory to other treatment.

12 (a) **True** Renal sympathetic nerve stimulation or injection of β-adrenoceptor agonists causes renin secretion from the kidney.

(b) **False** β-adrenoceptor agonists cause vasodilatation in skeletal muscle. Blockade of β-adrenoceptor mediated vasodilatation may produce pain, cramps and weakness of the muscle when walking.

(c) **True** There is evidence that some β-blockers lower blood pressure in animals when injected into the cerebral ventricles and that they antagonize a central pressor action of isoprenaline.

(d) **True** Stimulation of presynaptic α-adrenoceptors leads to a decrease of noradrenaline release, whereas stimulation of presynaptic β-adrenoceptors can result in an increased noradrenaline release. Hence β-adrenoceptor blockers may lower blood pressure by this among other mechanisms.

(e) **True** This is partly due to the blockade of β-adrenoceptors involved in the release of renin.

13 Salbutamol:
(a) is devoid of any action on the heart rate
(b) contracts uterine smooth muscle
(c) causes bronchodilatation
(d) can cause skeletal muscle tremor
(e) is effective when swallowed.

14 Reserpine:
(a) prevents the active re-uptake (uptake 1) of noradrenaline from the synaptic cleft into noradrenergic neurons
(b) disrupts the storage of monoamines
(c) prevents the uptake of noradrenaline from the cytoplasm into noradrenergic storage vesicles
(d) has a selective action on noradrenergic neurons only
(e) can be used in the treatment of hypertension.

15 Guanethidine:
(a) results in decreased release of noradrenaline from noradrenergic nerves in response to sympathetic nerve stimulation
(b) can result in the release of noradrenaline
(c) can be reversed by indirectly acting sympathomimetic amines
(d) can be enhanced by a drug preventing the re-uptake of noradrenaline
(e) is used to elevate low blood pressure.

13 (a) **False** Salbutamol selectively activates β_2-adrenoceptors in the bronchioles, especially if it is given by inhalation from an aerosol. If high doses are used, then cardiac stimulation can occur.
(b) **False** Uterine smooth muscle is relaxed by β_2-adrenoceptor agonists. Salbutamol is used to prevent premature labour.
(c) **True** This is the major use of salbutamol in asthma.
(d) **True**
(e) **True**

14 (a) **False**
(b) **True**
(c) **True** Reserpine and tetrabenazine prevent the active uptake of noradrenaline into the storage vesicles. Reserpine chelates Mg^{2+}, which is necessary for the ATPase which energizes the active transport process. The Mg^{2+} is also needed to stabilize the noradrenaline storage complex. If the Mg^{2+} is chelated, then storage is disrupted.
(d) **False** Storage of other monoamines, dopamine, 5-HT and adrenaline, is also affected.
(e) **True** Depression and drug-induced parkinsonism limit its usefulness.

15 (a) **True** Guanethidine is an adrenergic neuron blocker which decreases noradrenaline release from sympathetic nerves. The exact mechanism is obscure, but a local anaesthetic action has been suggested. Other adrenergic neuron blockers include debrisoquine, bethanidine and bretylium.
(b) **True** A transient sympathomimetic action can occur if guanethidine is given intravenously.
(c) **True** These appear to displace the adrenergic neuron blocker from the nerve terminal, and then exert their amine-releasing effect.
(d) **False** Re-uptake blockers (e.g. tricyclic antidepressants such as imipramine and amitriptyline) prevent an adrenergic neuron blocker from entering the nerve terminal.
(e) **False** Whereas a transient rise in blood pressure may occur—see (b)—adrenergic neuron blockers are used to lower elevated blood pressure.

16 Stimulation of presynaptic α_2-adrenoceptors:
(a) can lead to an increase in release of noradrenaline from sympathetic nerves
(b) is part of the negative feedback control which regulates the release of noradrenaline
(c) is caused by phentolamine
(d) occurs after administration of prazosin
(e) occurs after administration of clonidine.

17 At the skeletal neuromuscular junction:
(a) (+)-tubocurarine is a competitive nicotinic receptor blocker
(b) in therapeutic doses, (+)-tubocurarine has a longer duration of action than suxamethonium
(c) the actions of (+)-tubocurarine cannot be reversed by neostigmine
(d) acetylcholine synthesis and release are impaired in myasthenia gravis
(e) pancuronium and gallamine act as desensitization blockers.

18 Suxamethonium:
(a) is metabolized by plasma cholinesterases
(b) can have its neuromuscular blocking effects reversed by anticholinesterases
(c) causes depolarization at the endplate
(d) does not cause histamine release
(e) can cause blockade of nicotinic receptors at ganglia.