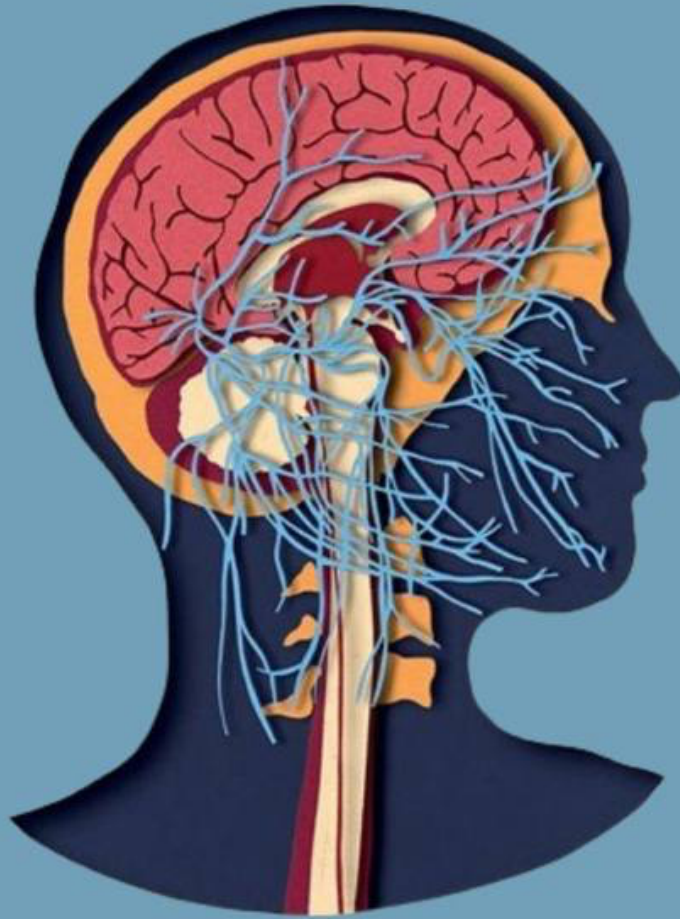


وَقُلْ رَبِّ زِدْنِي عِلْمًا



PERIPHERAL NERVOUS SYSTEM



SUBJECT : Pharma

LEC NO. : L1+2 Lippincott Qs

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#كلىنكالى_إلا_شعطة

Cholinergic agonists

All Qs are in.

STUDY QUESTIONS

Choose the **ONE** best answer.

4.1 Botulinum toxin blocks the release of acetylcholine from cholinergic nerve terminals. Which is a possible effect of botulinum toxin?

- A. Skeletal muscle paralysis
- B. Improvement of myasthenia gravis symptoms
- C. Increased salivation
- D. Reduced heart rate

Correct answer = A. Acetylcholine released by cholinergic neurons acts on nicotinic receptors in the skeletal muscle cells to cause contraction. Therefore, blockade of ACh release causes skeletal muscle paralysis. Myasthenia gravis is an autoimmune disease where antibodies are produced against nicotinic receptors and inactivate nicotinic receptors. A reduction in ACh release therefore worsens (not improves) the symptoms of this condition. Reduction in ACh release by botulinum toxin causes reduction in secretions including saliva (not increase in salivation), causing dry mouth and an increase (not reduction) in heart rate due to reduced vagal activity.

4.2 A patient develops urinary retention after an abdominal surgery. Urinary obstruction was ruled out in this patient. Which strategy would be helpful in promoting urination?

- A. Activating nicotinic receptors
- B. Inhibiting the release of acetylcholine
- C. Inhibiting cholinesterase enzyme
- D. Blocking muscarinic receptors

Correct answer = C. Activation of muscarinic receptors in the detrusor muscle of the urinary bladder can promote urination in patients where the tone of detrusor muscle is low. Inhibiting cholinesterase enzyme increases the levels of acetylcholine, and acetylcholine can increase the tone of the detrusor muscle. There are no nicotinic receptors in the detrusor muscle; therefore, activation of nicotinic receptors is not helpful. Inhibiting the release of acetylcholine or blocking muscarinic receptors worsens urinary retention.

4.3 Which of the following drugs could theoretically improve asthma symptoms?

- A. Bethanechol
- B. Pilocarpine
- C. Pyridostigmine
- D. Atropine

Correct answer = D. Muscarinic agonists and drugs that increase acetylcholine levels cause constriction of bronchial smooth muscles and could exacerbate asthma symptoms. Bethanechol and pilocarpine are muscarinic agonists, and pyridostigmine is a cholinesterase inhibitor that increases levels of acetylcholine. Atropine is a muscarinic antagonist and therefore does not exacerbate asthma. Theoretically, it should relieve symptoms of asthma (not used clinically for this purpose).

4.4 If an ophthalmologist wants to dilate the pupils for an eye examination, which drug/class of drugs is theoretically useful?

- A. Muscarinic receptor activator (agonist)
- B. Muscarinic receptor inhibitor (antagonist)
- C. Pilocarpine
- D. Neostigmine

Correct answer = B. Muscarinic agonists (for example, pilocarpine) contract the circular smooth muscles in the iris sphincter and constrict the pupil (miosis). Anticholinesterases (for example, neostigmine, physostigmine) also cause miosis by increasing the level of ACh. Muscarinic antagonists, on the other hand, relax the circular smooth muscles in the iris sphincter and cause dilation of the pupil (mydriasis).

4.5 In Alzheimer disease, there is a deficiency of cholinergic neuronal function in the brain. Theoretically, which strategy is useful in treating symptoms of Alzheimer disease?

- A. Inhibiting cholinergic receptors in the brain
- B. Inhibiting the release of acetylcholine in the brain
- C. Inhibiting the acetylcholinesterase enzyme in the brain
- D. Activating the acetylcholinesterase enzyme in the brain

Correct answer = C. Because there is already a deficiency in brain cholinergic function in Alzheimer disease, inhibiting cholinergic receptors or inhibiting the release of ACh worsens the condition. Activating the acetylcholinesterase enzyme increases the degradation of ACh, which also worsens the condition. However, inhibiting the acetylcholinesterase enzyme helps to increase the levels of ACh in the brain and thereby relieve the symptoms of Alzheimer disease.

4.6 An elderly female who lives in a farmhouse was brought to the emergency room in serious condition after ingesting a liquid from an unlabeled bottle found near her bed, apparently in a suicide attempt. She presented with diarrhea, frequent urination, convulsions, breathing difficulties, constricted pupils (miosis), and excessive salivation. Which of the following is correct regarding this patient?

- A. She most likely consumed an organophosphate pesticide.
- B. The symptoms are consistent with sympathetic activation.
- C. Her symptoms can be treated using an anticholinesterase agent.
- D. Her symptoms can be treated using a cholinergic agonist.

Correct answer = A. The symptoms are consistent with that of cholinergic crisis. Since the elderly female lives on a farm and the symptoms are consistent with a cholinergic crisis (usually caused by cholinesterase inhibitors), it may be assumed that she has consumed an organophosphate pesticide (irreversible cholinesterase inhibitor). Assuming that the symptoms are caused by organophosphate poisoning, administering an anticholinesterase agent or a cholinergic agonist will worsen the condition. The symptoms are not consistent with that of sympathetic activation, as sympathetic activation will cause symptoms opposite to that of cholinergic crisis seen in this patient.

4.7 A patient who received a nondepolarizing neuromuscular blocker (NMB) for skeletal muscle relaxation during surgery is experiencing mild skeletal muscle paralysis after the surgery. Which drug could reverse this effect of NMBs?

- A. Pilocarpine
- B. Bethanechol
- C. Neostigmine
- D. Atropine

Correct answer = C. Neuromuscular blockers act by blocking nicotinic receptors on the skeletal muscles. Increasing the levels of ACh in the neuromuscular junctions can reverse the effects of NMBs. Therefore, neostigmine, a cholinesterase inhibitor, could reverse the effects of NMBs. Pilocarpine and bethanechol are preferentially muscarinic agonists and have no effects on the nicotinic receptors. Atropine is a muscarinic antagonist and has no effects on nicotinic receptors.

4.8 A 60-year-old female who had a cancerous growth in the neck region underwent radiation therapy. Her salivary secretion was reduced due to radiation and she suffers from dry mouth (xerostomia). Which drug would be most useful in treating xerostomia in this patient?

- A. Acetylcholine
- B. Pilocarpine
- C. Echothiophate
- D. Atropine

Correct answer = B. Salivary secretion may be enhanced by activating muscarinic receptors in the salivary glands. This can be achieved in theory by using a muscarinic agonist or an anticholinesterase agent. Pilocarpine is a muscarinic agonist administered orally for this purpose. Acetylcholine has similar effects as that of pilocarpine; however, it cannot be used therapeutically as it is rapidly destroyed by cholinesterase in the body. Echothiophate is an irreversible cholinesterase inhibitor, but it cannot be used therapeutically because of its toxic effects. Atropine is a muscarinic antagonist and worsens dry mouth.

4.9 A 40-year-old male presents to his family physician with drooping eyelids, difficulty chewing and swallowing, and muscle fatigue even on mild exertion. Which agent could be used to diagnose myasthenia gravis in this patient?

- A. Atropine
- B. Edrophonium
- C. Pralidoxime
- D. Echothiophate

Correct answer = B. The function of nicotinic receptors in skeletal muscles is diminished in myasthenia gravis due to the development of antibodies to nicotinic receptors (auto-immune disease). Any drug that increases levels of ACh in the neuromuscular junction can improve symptoms in myasthenia gravis. Thus, edrophonium, a reversible cholinesterase inhibitor with a short duration of action can temporarily improve skeletal muscle weakness in myasthenia gravis, serving as a diagnostic tool. Atropine is a muscarinic antagonist and has no role in skeletal muscle function. Pralidoxime is a drug that is used to reverse the binding of irreversible cholinesterase inhibitors with cholinesterase enzyme and helps to reactivate cholinesterase enzyme. Hence, pralidoxime will not be useful in improving skeletal muscle function in myasthenia gravis.

4.10 *Atropa belladonna* is a plant that contains atropine (a muscarinic antagonist). Which of the following drugs or classes of drugs will be most useful in treating poisoning with belladonna?

- A. Malathion
- B. Physostigmine
- C. Muscarinic antagonists
- D. Nicotinic antagonists

Correct answer = B. Atropine is a competitive muscarinic receptor antagonist that causes anticholinergic effects. Muscarinic agonists or any other drugs that increase the levels of ACh are able to counteract effects of atropine. Thus, anticholinesterases such as malathion and physostigmine can counteract the effects of atropine, in theory. However, since malathion is an irreversible inhibitor of acetylcholinesterase, it is not used for systemic treatment in patients. Muscarinic antagonists worsen the toxicity of atropine. Nicotinic antagonists can worsen the toxicity by acting on parasympathetic ganglionic receptors and thus reducing the release of ACh.