1. This alpha-adrenergic receptor antagonist produces an irreversible receptor blockade and its principle indication is in the treatment of phaeochromocytoma.
   A. Phentolamine
   B. Phenoxybenzamine
   C. Prazosin
   D. Terazosin
   E. Reserpine

2. All of the following are possible side effects of low doses of nicotine (from smoking tobacco) EXCEPT:
   A. Increased sweating
   B. Stimulation of rate and depth of breathing
   C. Stimulation of catecholamine release from the adrenal medulla
   D. Bradycardia
   E. Nausea and vomiting

3. Epinephrine and norepinephrine must be given parenterally to elicit systemic effects. All of the following are reasons these drugs are ineffective when given orally, EXCEPT:
   A. Because of their low lipid solubility, these compounds are poorly absorbed following oral administration
   B. Both drugs complex with divalent cations found in the digestive secretions that prevent their transfer across the intestinal mucosa
   C. Both compounds produce a local vasoconstrictor effect that reduces blood flow and absorption through the intestinal mucosa
   D. These catecholamines are rapidly inactivated by enzymes in the intestines and liver

4. The enzyme that is inhibited by physostigmine is:
   A. Tyrosine hydroxylase
   B. Acetylcholinesterase
   C. Dopamine beta-hydroxylase
   D. Choline-acetyltransprose
5. The cardioselective beta-adrenergic blocking agent is:
   A. Metoprolol
   B. Timolol
   C. Propranolol
   D. Prazosin

6. Propranolol is contraindicated in, or should be used with caution in, all of the following, EXCEPT:
   A. Hypoglycemia
   B. Raynaud’s phenomenon
   C. Bronchial asthma
   D. Severe congestive heart failure
   E. Angina pectoris

7. All of the following structures respond to beta-adrenergic stimulation EXCEPT:
   A. Vasculature in the large muscles
   B. Thermoregulatory sweat glands
   C. Bronchial muscle
   D. The sinoatrial node

8. Atropine and scopolamine will block all the effects of acetylcholine listed below EXCEPT:
   A. Bradycardia
   B. Salivary secretion
   C. Bronchoconstriction
   D. Skeletal muscle contraction
   E. Miosis

9. Propranolol is indicated for use in patients with all the following conditions EXCEPT:
   A. Hypertension
   B. Angina pectoris
   C. Glaucoma
   D. Migraine headaches
   E. Hypothyroidism
10. Phenylephrine which is included in over-the-counter cold remedies:
   A. Causes vasoconstriction by stimulation of alpha-adrenergic receptors
   B. Prevents vasodilation by blocking beta-adrenergic receptors
   C. Stimulates the central nervous system to increase blood flow to the nose
   D. Reduces nasal secretions by inhibiting parasympathetic stimulation
   E. Is an antihistamine that reduces nasal mucus secretion

11. A predictably dangerous side effect of propranolol that constitutes a contraindication to its clinical use in susceptible patients is the induction of:
   A. Hypertension
   B. Cardiac arrhythmias
   C. Bronchospasm in asthmatics
   D. Respiratory depression

12. All of the following drugs are used topically to treat chronic wide-angle glaucoma. Which of these agents reduces intraocular pressure by decreasing the formation of aqueous humor?
   A. Timolol hydrochloride
   B. Pilocarpine hydrochloride
   C. Physostigmine salicylate
   D. Isoflurophate

13. The mechanism of action of the irreversible organic phosphate cholinesterase inhibitors is:
   A. Splitting of polypeptide bonds in cholinesterase
   B. Phosphorylation of the esteratic site of cholinesterase
   C. Phosphorylation of the anionic site of cholinesterase
   D. Acetylation of the esteratic site of cholinesterase
   E. Acetylation of the anionic site of cholinesterase

14. All of the following statements are accurate characterizations of ephedrine EXCEPT that it:
   A. Is a decongestant
   B. Can cause insomnia, restlessness, agitation, and tremors
   C. Can increase blood pressure
   D. Is rapidly metabolized by COMT and MAO enzymes
   E. Will relax the smooth muscles of the bronchial tree
15. A short acting cholinesterase inhibitor used in the diagnosis of myasthenia gravis is:

A. Edrophonium
B. Isoflurophate
C. Malathion
D. Physostigmine

16. All of the following are possible effects of the nicotinic ganglionic receptor antagonist mecamylamine EXCEPT:

A. Arteriolar vasodilation
B. Mydriasis and cycloplegia
C. Constipation and urinary retention
D. Tachycardia
E. Skeletal muscle weakness

17. All of the following are effects elicited by activation of the parasympathetic nervous system EXCEPT:

A. Decrease heart rate
B. Increase tone of longitudinal muscles of the intestine
C. Contraction of skeletal muscles
D. Contraction of the detrusor muscle of the urinary bladder
E. Secretion of fluids from lacrimal gland

18. Both phentolamine and prazosin:

A. Are competitive antagonists only at alpha-1 adrenergic receptors
B. Have potent direct vasodilator actions on vascular smooth muscle via beta-receptors
C. Enhance gastric acid secretion
D. Cause hypotension
E. Are used chronically for the treatment of primary hypertension

19. The major pathway for the removal of norepinephrine released from nerves into the synaptic space is:

A. Degradation by postsynaptic monoamine oxidase
B. Uptake into adrenergic nerve terminals
C. Binding with plasma membranes
D. Diffusion into the general circulation
E. Metabolism by liver enzymes
20. Timolol is:
   A. An anticholinesterase
   B. A nicotinic receptor agonist
   C. A selective alpha-adrenergic receptor antagonist
   D. A beta-adrenergic receptor antagonist
   E. An indirect acting noradrenergic agonist

21. Botulinum toxin causes paralysis because it:
   A. Increases acetylcholine release
   B. Blocks acetylcholinesterase
   C. Blocks the synthesis of acetylcholine
   D. Activates acetylcholine synthesis
   E. Prevents the release of acetylcholine

22. Which of the following is an alpha-adrenergic receptor antagonist:
   A. Metaproterenol
   B. Isoproterenol
   C. Phentolamine
   D. Amphetamine
   E. Propranolol

23. An i.v. infusion of norepinephrine will likely cause the following:
   A. An increase in total peripheral resistance
   B. An increase in heart rate
   C. A decrease in mean blood pressure
   D. A decrease in diastolic pressure
   E. An increase in kidney blood flow

Questions 24-30:

   A. Phentolamine
   B. Metoprolol
   C. Clonidine
   D. Prazosin
   E. Cocaine
   F. Pilocarpine
   G. Neostigmine
24. A long-acting acetylcholinesterase inhibitor
25. An indirectly acting sympathomimetic
26. A muscarinic receptor agonist
27. A selective alpha-1 adrenergic receptor antagonist
28. A centrally acting alpha-2 receptor agonist antihypertensive
29. A beta-receptor antagonist
30. A alpha-1 and alpha-2 receptor antagonist

31. Because of its alpha-adrenergic receptor agonist effects this compound is included in solutions of local anesthetics to prolong the duration of action of the local anesthetic:
   A. Prazosin
   B. Epinephrine
   C. Pilocarpine
   D. Phentolamine

32. Minoxidil, which activates ATP-sensitive potassium channels, can be used to treat hypertension since it acts to:
   A. Decrease cardiac contractility
   B. Reduces sodium and water retention
   C. Inhibits angiotensin-converting enzyme
   D. Vasodilate arterioles

33. As Harry Hypertensive’s physician you decide to place him on clonidine for his hypertension. You believe that because of its mechanism of action that it may be of some additional benefit to your patient. Clonidine acts by:
   A. Decreasing central sympathetic outflow by activating alpha-2 adrenergic receptors in the brain
   B. Increasing central sympathetic outflow by blocking beta-2 adrenergic receptors
   C. Direct vasodilation and reduction of total peripheral resistance by blocking alpha-1 adrenergic receptors
   D. Blockade of angiotensin II receptors
34. Many physicians find calcium channel blockers to be good drugs for the initial management of moderate hypertension because:
   A. They increase AV node conduction velocity
   B. Block peripheral alpha-1 receptors
   C. Depress CNS medullary vasomotor centers
   D. Cause direct vasodilation of blood vessels.

35. Some antihypertensive agents which act on adrenergic receptors are termed cardioselective. Their cardioselectivity occurs through the blockade of:
   A. Alpha-1 receptors
   B. Alpha-2 receptors
   C. Beta-1 receptors
   D. Beta-2 receptors

36. This drug has been shown to be efficacious in older men with benign prostatic hypertrophy, as it relaxes the smooth muscle of the prostate gland and neck of the bladder.
   A. Propranolol
   B. Clonidine
   C. Terazosin
   D. Physostigmine

37. Which of the following could be useful for treating tachycardia?
   A. Prazosin
   B. Isoproterenol
   C. Propranolol
   D. Dobutamine

38. Activation of the parasympathetic nervous system results in which of the following responses?
   A. Increase in heart rate
   B. Increased renin secretion
   C. Stimulation of gastrointestinal motility
   D. Release of epinephrine
   E. Dilation of the pupil
39. Which of the following drugs is most likely to produce orthostatic hypotension?
   A. Prazosin
   B. Isoproterenol
   C. Dobutamine
   D. Amphetamine
   E. Ephedrine

40. Which of the following side effects would NOT be experienced by a patient treated with phentolamine?
   A. Nasal stuffiness
   B. Bradycardia
   C. Impaired ejaculation
   D. Orthostatic hypotension
   F. Tachycardia

41. Terbutaline, a beta-2 receptor agonist, would be expected to cause all of the following effects EXCEPT:
   A. Mydriasis
   B. Reduced pulmonary airway resistance
   C. Tachycardia
   D. Hyperglycemia
   E. Increased blood flow in skeletal muscle

42. In children, the most dangerous toxic effect of the atropine-like drugs is:
   A. Hypotension
   B. Diarrhea
   C. Lethargy
   D. Water and sodium loss
   E. Hyperthermia

43. All of the following can be blocked by atropine pretreatment EXCEPT:
   A. Vagal reflex bradycardia
   B. Sweating induced by injection of pilocarpine
   C. Salivation induced by phystostigmine
   D. Hypertension induced by nicotine poisoning
   E. Mydriasis from pilocarpine eye drops
44. Which of the following would NOT be expected to affect accommodation (cycloplegia) when used topically in the eye:

A. Pilocarpine
B. Physostigmine
C. Timolol
D. Scopolamine
E. Atropine

45. When pupillary dilation, but not cycloplegia, is desired, a good choice is:

A. Atropine
B. Scopolamine
C. Pilocarpine
D. Phenylephrine

46. Which of the following acts directly on a receptor located on the membrane of the autonomic effector cell?

A. Cocaine
B. Tyramine
C. Amphetamine
D. Epinephrine
E. Reserpine

47. Phenylephrine

A. Increases skin temperature
B. Increases heart rate
C. Causes miosis
D. Constricts blood vessels in the nasal mucosa
E. All of the above

48. Which of the following effects of epinephrine would be blocked by phentolamine but not by propranolol?

A. Relaxation of bronchial smooth muscle
B. Cardiac stimulation
C. Vasoconstriction of cutaneous blood vessels
D. Relaxation of the uterine smooth muscle
49. Pretreatment with prazosin would be expected to block all of the following EXCEPT:

A. Increased inotropic and chronotropic effects on the heart from epinephrine
B. Bradycardia induced by phenylephrine
C. Vasoconstriction induced by norepinephrine
D. Mydriasis induced by phenylephrine

50. Regarding beta-blocking drugs:

A. Timolol reduces the production of aqueous humor
B. Propranolol is contraindicated for the treatment of angina pectoris
C. Metoprolol selectively acts at only beta-2 receptors
D. Propranolol is an effective therapy for treating asthma
E. All of the above

51. An effective drug for treating the cardiac effects of a thyroid storm would be:

A. Phentolamine
B. Prazosin
C. Clonidine
D. Propranolol
E. Ephedrine
1. Phenoxybenzamine is the only drug listed which is a non-competitive alpha-antagonist. Reserpine, is a depletor of catecholamines. Thus the answer is B.

2. Small doses of nicotine stimulate ganglionic nicotinic receptors and also sensory receptors in the lungs. These effects usually cause increased sweating, ventilatory rate, the secretion of epinephrine and norepinephrine from the adrenal medulla which contributes to elevations in heart rate, cardiac output and increased arterial pressure. These cardiovascular effects dominate the vagal mediated slowing which occurs through stimulation of parasympathetic ganglia cells. Nicotine stimulation of sensory receptors in the gut usually gives the first time smoker some nausea and possibly vomiting. Thus the answer is D. bradycardia.

3. Catecholamines are charged molecules at physiological pH and are poorly soluble in lipids. Also, they are metabolized by monoamine oxidases in the peripheral organs (intestines and liver) and are the natural agonists for activation of alpha-receptors leading to vasoconstriction in the periphery thereby reducing blood flow through the intestinal mucosa. However, they do not form complexes with divalent cations. Thus, the answer is B.

4. One of those drugs you just need to memorize: the “stigmines”, like physostigmine, neostigmine, pyridostigmine, all are selective acetylcholinesterase inhibitors. Thus, the answer is B.

5. Timolol and propranolol are pan beta-blockers (beta-1 and beta-2), while prazosin is a selective alpha-1 antagonist with no beta activity. Thus, the answer is A.

6. Beta-blockers can reduce epinephrine stimulated increases in blood sugar and could exacerbate the effects of insulin in insulin-dependent diabetics. Beta blockers also prevent catecholamine-mediated relaxation of bronchial smooth muscle and can be dangerous when given to individuals with asthma. Because they reduce cardiac output by reducing the inotropic/chronotropic effects of norepinephrine and epinephrine they can exacerbate an already reduced cardiac output in those patients with severe congestive heart failure. Lastly, blockade of beta-receptors would remove the vasodilatory effects of catecholamines in the periphery thereby unmasking only alpha-adrenergic receptor stimulation which would further constrict blood vessels in the extremities and exacerbate Raynaud’s. However, beta blockers, by reducing cardiac output and thus the work load on the heart, are useful for treating angina pectoris. Ans. E

7. To answer this question you just have to know where beta-adrenergic receptors are present. In this case they are found in bronchial smooth muscles, on cells of the SA node and on smooth muscles in vasculature. The sweat glands in contrast are a “cholinergic” effector tissue even though they are anatomically part of the sympathetic division. Ans. B
8. Parasympathetic activity (via acetylcholine) in the heart causes bradycardia, increased flow of saliva, constriction of bronchial smooth muscles and contraction of the iris sphincter muscle. Muscle contractions are mediated by acetylcholine activation of nicotinic receptors which are not blocked by the muscarinic antagonists scopolamine and atropine. Ans. D

9. Beta-blockers have been used to reduce cardiac output and work-load on the heart and thereby have been therapeutically useful in patients with hypertension and angina pectoris. Beta-blockers also reduce the production of aqueous humor which is beneficial in those with glaucoma. Also, because they can unmask or allow full alpha-adrenergic receptor stimulation which can constrict dilated dural blood vessels they have been found to produce some relief in migraine headache. Beta-blockers are effective for treating excessive catecholamine action in patients with hyperthyroidism. Ans. E

10. Alpha-1 agonists such as phenylephrine are potent vasoconstrictors, have no activity at beta receptors and do not directly affect parasympathetic effector organs. Phenylephrine does not cross the blood brain barrier and it is not an antihistamine. Ans. A

11. The rule of thumb with beta-blockers is not to use them in patients with asthma because they more often than not exacerbate the condition to dangerous levels. Ans. C

12. B, C, and D are useful for their ability to constrict the iris sphincter muscles and increase the flow of aqueous humor through the trabecular network of the Canals of Schlemm. Beta blockers (.olol’s) are useful for their ability to reduce the production of aqueous humor. Ans. A

13. The phosphate moiety forms covalent bonds with the esteratic site of the enzyme thereby preventing acetylation. There is no evidence that organophosphates split polypeptide bonds. Thus the answer is B.

14. Ephedrine is a sympathomimetic agent which is not a catecholamine derivative. As a sympathomimetic it will produce effects similar to those of norepinephrine/epinephrine including activation of alpha-1 receptors leading to vasoconstriction, stimulation of beta-1’s in the heart, and some central stimulant effects. However, because it is not a catecholamine it is not a substrate for enzyme degradation by COMT and MAO. Ans. D

15. Another one that just needs to be memorized. Edrophonium has a very short half-life which makes it useful for diagnosing myasthenia gravis. The others are too long acting and the insecticide malathion would not be a choice. Ans. A

16. Mecamylamine is an antagonist of nicotinic receptors on autonomic ganglion cells but not on nicotinic neuromuscular receptors. Thus, it will produce a number of autonomic nervous system effects but not skeletal muscle effects. Ans. E
17. When thinking parasympathetic you need to visualize the opposite of the sympathetically-mediated fight-or-flight response. Remember the cave-man relaxing and what the body is trying to do during this period of rest and rejuvenation. Also, the parasympathetic responses of the effector organs are mediated by muscarinic receptors. Skeletal muscle contractions are, however, mediated through acetylcholine activation of nicotinic receptors. Thus, the answer is C.

18. These alpha-antagonists are competitive receptor blockers and thus do not directly activate beta-receptors to produce vasodilation. However, by removing alpha activity on blood vessels a hypotensive response occurs. Gastric acid secretion is increased through cholinergic stimulation and does not involve alpha receptors. Phentolamine while being a competitive antagonist at alpha-1's, also blocks alpha-2 autoreceptors on noradrenergic terminals which then leads to increased norepinephrine release which can then enhance the reflex tachycardia that occurs with blood pressure lowering agents. Prazosin because of its selective alpha-1 blocking effects will reduce blood pressure, but not increase work-load on the heart. Because of the tachycardia and postural hypotension associated with phentolamine it is not a drug of choice for long-term treatment of hypotension. Thus, the answer is D.

19. Catecholamines, like norepinephrine, are metabolized by monoamine oxidases in the liver and inside the nerve terminal. Although a small amount may escape reuptake and diffuse into the circulation. However, the major route of inactivation is reuptake back into the nerve terminal. Ans. B

20. Remember the suffix -olol, which indicates with a high degree of certainty that it is a beta-receptor antagonist. Ans. D

21. Botulinum toxin is a potent inhibitor of acetylcholine release, and death can result from ventilatory failure due to lack of stimulation of skeletal muscle nicotinic receptors. Ans. E

22. Phentolamine is a competitive alpha-1 and alpha-2 antagonist, while the other drugs act as direct beta-adrenergic agonists (metaproterenol, isoproterenol), beta-adrenergic antagonists (propranolol), or as an indirectly acting sympathomimetic (amphetamine). Ans. C

23. Norepinephrine is a potent alpha-adrenergic agonist which through activation of alpha-1 receptors constricts smooth muscles in blood vessels resulting in an increase in total peripheral resistance, decreased perfusion of the kidney, and an increase in blood pressure. This increase in blood pressure causes a reflex bradycardia. Thus, the answer is (A).
24. G A long-acting acetylcholinesterase inhibitor
25. E An indirectly acting sympathomimetic
26. F A muscarinic agonist
27. D A selective alpha-1 adrenergic receptor antagonist
28. C A centrally acting alpha-2 agonist antihypertensive
29. B A beta-receptor antagonist
30. A A alpha-1 and alpha-2 receptor antagonist

31. Epinephrine in concentrations used in local anesthetics will act on alpha-1 adrenergic receptors to constrict blood vessels and prolong the elimination of the local anesthetic drug. Cholinergic agents (pilocarpine) and alpha-antagonists (phentolamine and prazosin) do not share this property. Ans. B

32. Minoxidal is a potent direct vasodilator of arterioles and has no effects on the contractility of heart muscle or on angiotensin-converting enzyme. A possible side effect, however, is an increase in sodium and water retention. Ans. D

33. Clonidine is an alpha-2 agonist. Alpha-2 agonists act on presynaptic autoreceptors to reduce neurotransmitter release probably through a reduction of terminal excitability. The alpha-2 agonists, like clonidine, reduce the excitability of neurons in the vasomotor centers of the medulla thereby reducing the output of the sympathetic nervous system which effectively reduces blood pressure. Ans. A

34. Calcium channel blockers have no direct effects on alpha-1 receptors nor do they affect medullary centers. They actually decrease AV node firing by reducing the influx of calcium which the AV is largely dependent upon for depolarization. In vascular tissues these drugs reduce the influx of calcium which is a primary means for depolarization (and subsequent contraction) of vascular smooth muscle cells. Thus, the answer is D.

35. As you recall from the distribution of adrenergic receptors, the heart is for all intents a beta-1 receptor organ. Thus, the beta-adrenergic blockers which have been termed cardioselective have their actions through the blockade of beta-1 receptors in the heart. Ans. C
36. The smooth muscles of the prostate and bladder neck receive noradrenergic innervation and are contracted by activation of alpha-1 noradrenergic receptors, for which terazosin (and other drugs ending in -osin) is a selective antagonist. Thus, with terazosin the smooth muscles relax. Clonidine is a selective alpha-2 agonist and is used to treat hypertension, as is the beta-blocker timolol. Physostigmine is an anticholinesterase inhibitor and is not efficacious for BPH. ANS.=D.

37. Tachycardia will occur with drugs which act to stimulate beta-1 adrenergic receptors in the heart, such as isoproterenol and dobutamine. Any beta-receptor antagonist will block these receptors and reduce heart rate and cardiac output. Prazosin is an alpha-1 antagonist and would be expected to reduce total peripheral resistance and would likely cause a decrease in blood pressure which would reflexly increase heart rate. ANS.=C.

38. Stimulation of muscarinic receptors via parasympathetic activation will slow heart rate (via the vagus) and constrict the pupil. Renin secretion and epinephrine release are on the sympathetic side of the autonomies. GI motility is increased through muscarinic parasympathetic activation. Remember that too much stimulation of muscarinic cholinergic receptors can cause SLUD, the D stands for diarrhea. ANS.=C.

39. Alpha-1 adrenergic antagonist will block sympathetic tone to the blood vessels thereby reducing total peripheral resistance and causing a drop in blood pressure. Activation of adrenergic receptors directly via agonist actions on beta-1's (isoproterenol and dobutamine) will increase heart rate and increase blood pressure, while compounds which release norepinephrine (amphetamine and ephedrine) will increase total peripheral resistance, heart rate and blood pressure. Prazosin will block alpha-1 mediated contraction of vascular smooth muscles thereby lower total peripheral resistance and also blood pressure. ANS.=A.

40. Alpha-adrenergic antagonists reduce tone to smooth muscles (blood vessels, vas deferens) and decrease total peripheral resistance (lower BP), interfere with ejaculation and will vasodilate blood vessels in the nasal mucosa. The drop in BP can precipitate a reflex increase in heart rate (tachycardia). ANS.=B.

41. Stimulation of beta-2's will dilate bronchial smooth muscles and blood vessels. The effect on blood vessels in skeletal muscle will produce increased blood flow. However, dilation of blood vessels in other organs can also decrease total peripheral resistance and decrease BP which could reflexly increase heart rate. Beta-2's when stimulated also cause some glyco- genolysis and lipolysis. Mydriasis can be produced by blockade of muscarinic receptors in iris sphincter muscles or stimulation of alpha-1 receptors on the radial muscles of the iris. ANS.=A.

42. Blockade of muscarinic cholinergic receptors will dry up secretions and decrease thermoregulatory sweating. The latter can be dangerous in babies or young children. The other effects listed are not mediated by muscarinic cholinergic receptor blockers. ANS.=E.
43. Atropine is a muscarinic receptor antagonist. The only symptom that is not mediated by activation of muscarinic receptors is the hypertension from nicotine stimulation of nicotinic receptor. ANS.=D.

44. Changes in accommodation occur through muscarinic activation of the ciliary smooth muscles. Thus drugs which affect these receptors or increase the amount of acetylcholine acting on these receptors will change accommodation. Timolol is a beta blocker and will not affect lens shape. Ans. C

45. Agents which will contract the iris radial muscle, an alpha-1 receptor effector site, will dilate the pupil but not affect accommodation. Phenylephrine is a selective alpha-1 receptor agonist. Cholinergic agents which block muscarinic receptors in the sphincter muscles of the iris and ciliary muscles will produce both dilation and loss of accommodation (cycloplegia). ANS.=D.

46. Cocaine, amphetamine and tyramine are indirectly acting sympathomimetics in that they produce their effects through the release of catecholamines. Reserpine blocks uptake of catecholamines into the vesicles and results in their metabolism and depletion of catecholamine. Epinephrine is the natural agonist acting directly on alpha-1 and 2's, beta-1 and 2's. ANS.=D.

47. Alpha-1 agonists constrict peripheral blood vessels, dilate the pupil and through increased TPR and blood pressure can cause reflex slowing of the heart. Constriction of cutaneous blood vessels by restricting blood flow to the skin causes the skin to become cool. ANS.=D

48. Phentolamine is an alpha-antagonist. The only effect described above that is mediated by alpha-receptors is contraction of the radial muscles in the iris. ANS.=C

49. Phenylephrine, acting through alpha-1 receptors on vascular smooth muscle causes an increase in TPR, an increase in BP and a reflex slowing of the heart. Phenylephrine can also activate alpha-1 receptors on the iris radial muscles to cause dilation of the pupil. Norepinephrine’s vasoconstrictor effects are also mediated by alpha-1 receptors. However, epinephrine’s cardiac effects are mediated through beta-1 receptors which would not be sensitive to blockade by the selective alpha-1 antagonist prazosin. ANS.=A

50. Beta blockers should not be given to asthmatics, but they are indicated for the treatment of angina. Metoprolol is cardioselective and acts predominantly on beta-1 receptors. Timolol is commonly prescribed for treating glaucoma because it leads to a decreased aqueous humor production. Ans.=A

51. Hyperthyroidism sensitizes the heart to beta-receptor stimulation by norepinephrine and epinephrine, probably because of an increased number of beta-receptors that are formed. Thus, the cardiac effects of a thyroid storm would be treated with a beta blocker. Only one of the drugs listed is a beta-blocker (remember the -olol). ANS.=D