Complete the following.

1. ________________ drugs affect the rate of the heart and can either increase its rate or decrease its rate.

2. ________________ drugs affect the force of contraction and can be either positive or negative.

3. ________________ drugs alter the rhythm or electrical conduction through the heart muscle.

4. __________ dromotropic drugs enhance the electrical conduction of signals in certain parts of the heart and __________ dromotropic drugs slow conduction.

5. The sympathetic nerves release ___________________ and cause adrenergic responses.

6. The parasympathetic nerves release _______________ and cause cholinergic responses.

7. The __________________ is related to contraction and relaxation of the heart.

8. The action potential is controlled at the cellular level by _______ transporting in and out of cells.

9. The pacemaking and conduction throughout the heart are driven by action potentials that are dependent on ________________, ________________, and ________________ activity, and their passages through certain ionic channels within the membranes of the myocardial cells.

10. Potassium moves out of the cell while sodium moves in for a process called ________________.

11. Potassium moving back into the cell and sodium back out is a process called ________________.

12. ________________ is needed for the actual muscle contraction to occur.

13. Calcium influences actin and ____________, which control cardiac cell length and muscle contraction.

14. The time when cells cannot be excited after electrical stimulation is called the ________________ ________________.
15. The mechanisms of cardiac arrhythmias include disorders of ___________________ and/or conduction.

16. ___________________ is the ability to generate pacemaking activity and is normal when pacemaker activity originates in the SA node.

17. Arrhythmias occur mainly because the pacemaker __________________________________ somewhere other than the SA node or the impulse does not follow normal conduction pathway and so abnormal conduction occurs.

18. Cells can become excited if there is an oxygen ____________, as in ischemia or infarction, or if an electrolyte imbalance occurs.

19. Because underlying arrhythmias are variable by nature, ___________________ or arrhythmias induced by the drugs themselves can be difficult to diagnose, so awareness among healthcare professionals is important.

20. Arrhythmias can even be a sign of antiarrhythmic drug ________________.

21. ________________ treatment is warranted when hemodynamic compromise occurs, when an increase in myocardial oxygen demand occurs, or when the arrhythmias may lead to malignant ventricular arrhythmias.

22. Common __________________________ arrhythmias are atrial fibrillation or flutter, paroxysmal supraventricular tachycardia, and autonomic atrial tachycardias.

23. Common __________________________ arrhythmias include premature ventricular contractions, ventricular tachycardia, torsade de pointes, and ventricular fibrillation.

24. Goals for drugs used for cardiac arrhythmias are to restore and maintain ____________ ________________ ________________.

25. An ________________ ________________ is the electrical stimulation of cardiac contraction beginning at another point or outside the normal conduction pathway.

26. Antiarrhythmics’ mechanism of action is both pharmacologic and ______________________________.
27. Antiarrhythmic drugs are described by ______________ classes, which subclasses for Class I. These drugs _______________ calcium, potassium, and sodium channels, _______________ the repolarization phase, or _______________ B-Adrenergic activity.

28. _______________ is an unclassified antiarrhythmic that inhibits the sodium/potassium exchange pump in the heart.

29. Digoxin does not convert atrial fibrillation to normal sinus rhythm, but it does _________ the ventricular rate.

30. _______________ Side effects of digoxin include anorexia, visual disturbances, fatigue, and life-threatening _______________.

31. _______________ is an unclassified antiarrhythmic used to convert superaventricular tachycardia to a sinus rhythm and at times it aids in the _______________ of the rhythm disorders.

32. Pharmacologic treatment for heart failure consists of removal of sodium and water with _______________, increasing contraction of the heart with positive _______________ activity and _______________ vascular resistance with vasodilators.

33. Basically, drugs for heart failure improve heart _______________ and decrease the heart’s work.

34. Digoxin is one of the oldest inotropic drugs known. Inotropes increase the peak tension produced by the heart during _______________ _______________.

35. The _______________ action of digoxin increases cardiac contractility, so the heart pumps more strongly.

36. _______________ _______________ _______________ _______________ decrease blood levels of angiotensin II which is a vasoconstrictor, and therefore blocking its production induces vasodilation.

37. ACE inhibitors decrease aldosterone secretion, salt and water retention, and vascular resistance so they are beneficial to patients with _______________ _______________ _______________ because they decrease afterload and attenuate the remodeling or structural changes that can occur in the congested heart due to heart failure.

38. Side effects of ACE inhibitors include dry, nonproductive cough, _______________ of renal function, proteinuria, and _______________.
39. Angiotensin II receptor blockers (ARBs) are used for ___________________ and for heart failure.

40. ARBs may have a role in patients who are ________________ of ACE inhibitors. Also, the blockade of ACE inhibitors can be incomplete so ARBs and ACE inhibitors may be used ________________.

41. Beta-blockers are also used for heart failure and may be beneficial by slowing the heart rate to allow more time for complete __________________ filling, blocking the deleterious effects of __________________ on the failing heart, reducing myocardial oxygen ________________, and controlling blood pressure.

42. ACC/AHA guidelines recommend that Beta blockers be prescribed for all patients with stable ________________ ______________ ______________.

43. ________________ can greatly reduce the afterload forces on the heart. They decrease afterload by dilating arterial vessels, dilating venous vessels and decrease preload, and affect both arterial and venous vessels.

44. ________________ ________________ respond better to hydralazine and Isosorbide for heart failure management.

45. Calcium-channel blockers are not recommended for use in patients with ________________- ________________.

46. ________________ therapy is recommended for all patients with fluid retention and heart failure to get rid of excess volume that increases the workload of the heart.

47. Patients with heart failure should restrict ________________ in the diet to 2 to 3 grams or less to prevent the accumulation of fluid in the lungs and lower extremities.

48. Aldosterone antagonists are beneficial in heart failure because of their ________________ of neurohormones that may contribute to adverse cardiac enlargement and fibrosis and by inhibiting ________________ loss that may contribute to arrhythmias.

49. Patients with acute heart failure are usually prescribed IV medications such as ________________ and Milrinone. Both drugs ________________ cardiac output and decrease left ventricular filling pressure.
Cardiac Agents

50. Other IV medications that may be used to treat acute heart failure include dopamine, ________________, and nitroglycerin.

51. Nesiritide is a recombinant manufactured drug identical to endogenous ________ causing vasodilation and natriuretic effects.

52. Some of the same drugs used for the treatment of heart failure are also used for the treatment of ____________.

53. Pharmacologic treatment of angina is aimed at _______ ___________ and prevention of recurrent pain.

54. Prophylactic treatment of angina is given by calcium entry blockers, ________________, or nitrates.

55. Nitroglycerin given sublingually relieves pain with angina because it reduces _______________ and afterload by dilating both veins and _______________, thus decreasing cardiac workload.

56. Beta blockers given to myocardial infarction patients can __________ infarct size and decrease the incidence of arrhythmias.

57. Calcium channel blockers ___________ the calcium influx into muscle that initiates contraction. They are also ________________, so these drugs can be used as an antianginal drug.

58. Acute coronary syndromes include unstable ____________ and myocardial infarction. ST segment elevation ACS is treated with early _______________ therapy with either primary percutaneous coronary intervention (PCI) or administration of a fibrinolytic agent.

59. Patients with non-ST segment elevation MI receive additional ________________ and/or glycoprotein IIb/IIIa receptor blocker.

60. Metabolic syndrome __________ the risk of myocardial infarction or stroke and doubles the mortality.

61. The risk of coronary heart disease is two to four times greater in patients with ______________ than in nondiabetics.

62. Drugs used to treat ______________ include bile acid resins or sequestrants, HMG-CoA reductase inhibitors, niacin, gemfibrozil, fish oil, or ezetimibe.
63. Statins interrupt the biochemical rate-limiting step in __________________________.

64. Bile acid resins decrease cholesterol __________________________.

65. Niacin reduces __________________________ of LDL.