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Cardiovascular
(revised 2011)

Unique Characteristics of Cardiac Muscle

CV 1. Contrast the duration of the action potential and the refractory period in a cardiac muscle, a skeletal muscle, and a nerve. Sketch the temporal relationship between an action potential in a cardiac muscle cell and the resulting contraction (twitch) of that cell. On the basis of that graph, explain why cardiac muscle cannot remain in a state of sustained (tetanic) contraction.

CV 2. State the steps in excitation-contraction coupling in cardiac muscle. Outline the sequence of events that occurs between the initiation of an action potential in a cardiac muscle cell and the resulting contraction and then relaxation of that cell. Provide specific details about the special role of Ca^{2+} in the control of contraction and relaxation of cardiac muscle.

CV 3. Compare cardiac and skeletal muscle with respect to: cell size, electrical connections between cells, and arrangement of myofilaments. Based on ion permeability and electrical resistance describe role of gap junctions in creating a functional syncytium.

CV 4. Identify the role of extracellular calcium in cardiac muscle contraction. Identify other sources of calcium that mediate excitation-contraction coupling, and describe how intracellular calcium concentration modulates the strength of cardiac muscle contraction.

CV 5. Describe the role of Starling's Law of the Heart in keeping the output of the left and right ventricles equal.

CV 6. Describe the difference in the way changes in preload and changes in contractility influence ventricular force development. Compare the energetic consequences of these two separate mechanisms of force modulation.

Electrophysiology of the Heart

CV 7. Sketch a typical action potential in a ventricular muscle and a pacemaker cell, labeling both the voltage and time axes accurately. Describe how ionic currents contribute to the four phases of the cardiac action potential. Use this information to explain differences in shapes of the action potentials of different cardiac cells.

CV 8. Describe the ion channels that contribute to each phase of the cardiac action potential. How do differences in channel population influence the shape of the action potential in the nodal, atrial muscle, ventricular muscle, and Purkinje fiber cardiac cells?

CV 9. Explain what accounts for the long duration of the cardiac action potential and the resultant long refractory period. What is the advantage of the long plateau of the cardiac action potential and the long refractory period?

CV 10. Beginning in the SA node, diagram the normal sequence of cardiac activation (depolarization) and the role played by specialized cells. Predict the consequence of a failure to conduct the impulse through any of these areas.

CV 11. Explain why the AV node is the only normal electrical pathway between the atria and the ventricles, and explain the functional significance of the slow conduction through the AV node. Describe factors that influence conduction velocity through the AV node.

CV 12. Explain the ionic mechanism of pacemaker automaticity and rhythmicity, and identify cardiac cells that have pacemaker potential and their spontaneous rate. Identify neural and humoral factors that influence their rate.

CV 13. Discuss the significance of "overdrive suppression" and "ectopic pacemaker," including the conditions necessary for each to occur.

CV 14. Contrast the sympathetic and parasympathetic nervous system influence on heart rate and cardiac excitation in general. Identify which arm of the autonomic nervous system is dominant at rest and during exercise. Discuss ionic mechanisms of these effects on both working myocardium and pacemaker cells.

CV 15. Describe how cell injury, resulting in a less negative resting potential, alters ionic events in depolarization and repolarization.

CV 16. Define the following terms: decremental conduction, reentry, and circus movement.

Cardiac Function

CV 17. Draw and describe the length-tension relationship in a single cardiac cell. Correlate the cellular characteristics of length, tension, and velocity of shortening with the intact ventricle characteristics of end diastolic volume, pressure, and dP/dt.

CV 18. Define preload and explain why ventricular end-diastolic pressure, atrial pressure and venous pressure all provide estimates of ventricular preload. Explain why ventricular end-diastolic pressure provides the most reliable estimate.

CV 19. Define afterload and explain how arterial pressure influences afterload. Describe a condition when arterial pressure does not provide a good estimate of afterload.

CV 20. Define contractility and explain why dP/d*t* is a useful index of contractility. Explain how the calcium transient differs between cardiac and skeletal muscle and how this influences contractility.

CV 21. Define the difference between cardiac performance and cardiac contractility. Describe the impact of changes in preload, afterload, and contractility in determining cardiac performance.

CV 22. Explain how changes in sympathetic activity alter ventricular work, cardiac metabolism, oxygen consumption and cardiac output.

CV 23. Write the formulation of the Law of LaPlace. Describe how it applies to ventricular function in the normal and volume overloaded (failing) ventricle.

CV 24. Draw a ventricular pressure-volume loop and on it label the phases and events of the cardiac cycle (ECG, valve movement).

CV 25. Differentiate between stroke volume and stroke work. Identify stroke volume and stroke work from a pressure-volume loop.

CV 26. Define ejection fraction and be able to calculate it from end diastolic volume, end systolic volume, and/or stroke volume. Predict the change in ejection fraction that would result from a change in a) preload, b) afterload, and c) contractility.

CV 27. Draw the change in pressure-volume loops that would result from changes in a) afterload, b) preload, or c) contractility, for one cycle and the new steady state that is reached after 20 or more cycles.

Cardiac Cycle

CV 28. Understand the basic functional anatomy of the atrioventricular and semilunar valves, and explain how they operate.

CV 29. Draw, in correct temporal relationship, the pressure, volume, heart sound, and ECG changes in the cardiac cycle. Identify the intervals of isovolumic contraction, rapid ejection, reduced ejection, isovolumic relaxation, rapid ventricle filling, reduced ventricular filling and atrial contraction.

CV 30. Know the various phases of ventricular systole and ventricular diastole. Contrast the relationship between pressure and flow into and out of the left and right ventricles during each phase of the cardiac cycle.

CV 31. Understand how and why left sided and right sided events differ in their timing.

Physiology of Cardiac Defects (Heart Sounds)

CV 32. Know the factors that contribute to the formation of turbulent flow.

CV 33. Describe the timing and causes of the four heart sounds.

CV 34. Describe the expected auscultation sounds that define mitral stenosis, mitral insufficiency, aortic stenosis, and aortic insufficiency. Explain how these pathologic changes would affect cardiac mechanics and blood pressure.

The Normal Electrocardiogram (ECG) and the ECG in Cardiac Arrhythmias and Myopathies

CV 35. Define the term dipole. Describe characteristics that define a vector. Describe how dipoles generated by the heart produce the waveforms of the ECG.

CV 36. Describe the electrode conventions used by clinicians to standardize ECG measurements. Know the electrode placements and polarities for the 12 leads of a 12-lead electrocardiogram and the standard values for pen amplitude calibration and paper speed.

CV 37. Name the parts of a typical bipolar (Lead II) ECG tracing and explain the relationship between each of the waves, intervals, and segments in relation to the electrical state of the heart.

CV 38. Explain why the ECG tracing looks different in each of the 12 leads.

CV 39. Define mean electrical vector (axis) of the heart and give the normal range. Determine the mean electrical axis from knowledge of the magnitude of the QRS complex in the standard limb leads.

CV 40. Describe the alteration in conduction responsible for most common arrhythmias: i.e., tachycardia, bradycardia, A-V block, Wolff-Parkinson-White (WPW) syndrome, bundle branch block, flutter, fibrillation.

CV 41. Describe electrocardiographic changes associated respectively with myocardial ischemia, injury, and death. Define injury current and describe how it is alters the S-T segment of the ECG.

Cardiac Output and Venous Return

CV 42. Understand the principles underlying cardiac output measurements using the Fick principle, dye dilution, and thermodilution methods.

CV 43. Know how cardiac function (output) curves are generated and how factors which cause changes in contractility in the heart can alter the shape of cardiac function curves.

CV 44. Understand the concept of "mean systemic pressure," its normal value, and how various factors can alter its value.

CV 45. Define venous return. Understand the concept of "resistance to venous return" and know what factors determine its value theoretically, what factors are most important in practice, and how various interventions would change the resistance to venous return.

CV 46. Construct a vascular function curve. Predict how changes in total peripheral resistance, blood volume, and venous compliance influence this curve.

CV 47. Explain why the intersection point of the cardiac function and vascular function curves represents the steady-state cardiac output and central venous pressure under the conditions represented in the graph.

CV 48. Use the intersection point of the cardiac function curve and vascular function curve to predict how interventions such as hemorrhage, heart failure, autonomic stimulation, and exercise will affect cardiac output and right atrial pressure. Predict how physiological compensatory changes would alter acute changes.

Fluid Dynamics

CV 49. Describe the components of blood (cells, ions, proteins, platelets) giving their normal values. Relate the three red blood cell concentration estimates, red blood cell count, hematocrit, and hemoglobin concentration.

CV 50. Identify the source, stimulus for formation, and function of the hormone erythropoietin. Relate the rate of red blood cell synthesis to the normal red blood cell life span and the percentage of immature reticulocytes in the blood.

CV 51. Describe the functional consequence of the lack of a nucleus, ribosomes, and mitochondria for a) protein synthesis and b) energy production within the red blood cell.

CV 52. Discuss the normal balance of red blood cell synthesis and destruction, including how imbalances in each lead to anemia or polycythemia.

CV 53. Explain how red blood cell surface antigens account for typing of blood by the A B O system and rhesus factor. Based on these antigens, identify blood type of a "universal donor" and a "universal recipient."

CV 54. Know the factors that determine the total energy of the flowing blood and the relationship among these factors. Describe the usual reference point for physiological pressure.

CV 55. Be able to differentiate between flow and velocity in terms of units and concept.

CV 56. Understand the relationship between pressure, flow, and resistance in the vasculature and be able to calculate for one variable if the other two are known. Apply this relationship to the arteries, arterioles, capillaries, venules, and veins. Explain how blood flow to any organ is altered by changes in resistance to that organ.

CV 57. Explain how Poiseuille's Law influences resistance to flow. Use it to calculate changes in resistance in a rigid tube (blood vessel). Explain the deviations from Poiseuille's law predictions that occur in distensible blood vessels.

CV 58. Understand the relationship between flow, velocity, and cross-sectional area and the influence vascular compliance has on these variables. Explain how hemodynamics in blood vessels, especially microcirculation, deviate from theory due to anomalous viscosity, distensibility, axial streaming and critical closing behavior and the glycocalyx.

CV 59. Define resistance and conductance. Understand the effects of adding resistance in series vs. in parallel on total resistance and flow. Apply this information to solving problems characterized by a) resistances in series and b) resistances in parallel. Apply this concept to the redistribution of flow from the aorta to the tissues during exercise.

CV 60. List the factors that shift laminar flow to turbulent flow. Describe the relationship between velocity, viscosity, and audible events, such as murmurs and bruits.

CV 61. Understand the principles of flow through collapsible tubes, the Starling resistor, and what pressure gradient determines flow for different relative values of inflow, surrounding, and outflow pressures.

Arterial Pressure and the Circulation

CV 62. Describe the organization of the circulatory system and explain how the systemic and pulmonary circulations are linked physically and physiologically.

CV 63. Describe blood pressure measurement with a catheter and transducer and explain the components of blood pressure waveform. Contrast that with the indirect estimation of blood pressure with a sphygmomanometer. Explain how each approach provides estimates of systolic and diastolic pressures. Given systolic and diastolic blood pressures, calculate the pulse pressure and the mean arterial pressure.

CV 64. Describe how arterial systolic, diastolic, mean, and pulse pressure are affected by changes in a) stroke volume, b) heart rate, c) arterial compliance, and d) total peripheral resistance.

CV 65. Contrast pressures and oxygen saturations in the arteries, arterioles, capillaries, venules, and veins of both the systemic and pulmonary circulations. Repeat that process for velocity of blood flow and cross-sectional area, and volume.

CV 66. Identify the cell membrane receptors and second messenger systems mediating the contraction of vascular smooth muscle by norepinephrine, angiotensin II, and vasopressin.

CV 67. Identify the cell membrane receptors and second messenger systems mediating the relaxation of vascular smooth muscle by nitric oxide, bradykinin, prostaglandins, and histamine. Include the role of the endothelial cell.

CV 68. Identify the cell membrane receptors and second messenger systems mediating changes in cardiac performance.

The Microcirculation and Lymphatics

CV 69. Explain how water and solutes traverse the capillary wall. Use Fick's equation for diffusion to identify the factors that will affect the diffusion-mediated delivery of nutrients from the capillaries to the tissues. Define and give examples of diffusion-limited and flow-limited exchange.

CV 70. Describe how changes in capillary surface area affect the capacity for fluid exchange.

CV 71. Define the Starling equation and discuss how each component influences fluid movement across the capillary wall.

CV 72. Describe the pathway for leukocyte migration across the microcirculation, including leukocyte expression of cellular adhesion molecules, and recognition sites in the vascular endothelial cells.

CV 73. Starting at the post-capillary venule, describe the process of angiogenesis, including the stimulus that initiates new vessel growth.

CV 74. Describe the Donnan effect and its importance in capillary dynamics.

CV 75. Predict how altering pressure or resistance in pre- and post-capillary regions alters capillary pressure and the consequence of this change on transmural fluid movement.

CV 76. Using the components of the Starling equation, explain why fluid does not usually accumulate in the interstitium of the lungs.

CV 77. Describe how histamine alters the permeability of the post-capillary venules and how the loss of albumin into the interstitial space promotes localized edema.

CV 78. Describe the lymphatics, and explain how the structural characteristics of terminal lymphatics allow the reabsorption of large compounds, such as proteins.

CV 79. Contrast the structure of lymphatic capillaries and systemic capillaries, including the significance of the smooth muscle in the walls of the lymphatic vessels.

CV 80. Identify critical functions of the lymphatic system in fat absorption, interstitial fluid reabsorption, and clearing large proteins from the interstitial spaces.

CV 81. Diagram the relationship between interstitial pressure and lymph flow. Explain why edema does not normally develop as interstitial pressure increases.

CV 82. Explain how edema develops in response to: a) venous obstruction, b) lymphatic obstruction, c) increased capillary permeability, d) heart failure, e) tissue injury or allergic reaction, and f) malnutrition.

Regulation of Arterial Pressure

CV 83. List the anatomical components of the baroreceptor reflex.

CV 84. Explain the sequence of events in the baroreflex that occur after an acute increase or decrease in arterial blood pressure. Include receptor response, afferent nerve activity, CNS integration, efferent nerve activity to the SA node, ventricles, arterioles, venules, and hypothalamus.

CV 85. Explain the sequence of events mediated by cardiopulmonary (volume) receptors that occur after an acute increase or decrease in arterial blood pressure. Include receptor response, afferent nerve activity, CNS integration, efferent nerve activity to the heart, kidney, hypothalamus, and vasculature.

CV 86. Explain the sequence of events mediated by cardiopulmonary (volume) receptors that occur after an acute increase or decrease in central venous pressure. Include receptor response, afferent nerve activity, CNS integration, efferent nerve activity to the heart, kidney, hypothalamus, and vasculature.

CV 87. Contrast the sympathetic and parasympathetic nervous system control of heart rate, contractility, total peripheral resistance, and venous capacitance. Predict the cardiovascular consequence of altering sympathetic nerve activity and parasympathetic nerve activity.

CV 88. Contrast the relative contribution of neural and renal mechanisms in blood pressure and blood volume regulation.

CV 89. Outline the cardiovascular reflexes initiated by decreases in blood O_2 and increases in blood CO_2.

CV 90. Describe the release, cardiovascular target organs, and mechanisms of cardiovascular effects for angiotensin, atrial natriuretic factor, bradykinin, and nitric oxide.

CV 91. Describe the Cushing Reflex and the CNS ischemic pressor response.

Local Control of Blood Flow

CV 92. Define autoregulation of blood flow. Distinguish between short-term and long-term autoregulatory responses and the mechanisms responsible for each.

CV 93. Describe how the theory of metabolic regulation of blood flow accounts for active hyperemia and reactive hyperemia.

CV 94. Describe the contribution of myogenic tone to blood flow regulation.

CV 95. Identify the role of P_{O_2} , P_{CO_2} , pH, adenosine, and K^+ in the metabolic control of blood flow to specific tissues.

CV 96. Diagram the synthetic pathway for nitric oxide (EDRF, endothelial derived relaxing factor), including substrate and the interplay between endothelium and vascular smooth muscle.

CV 97. Discuss the circumstances and the mechanisms whereby humoral substances contribute to regulation of the microcirculation.

CV98. Discuss the interaction of a) intrinsic (local), b) neural, and c) humoral control mechanisms and contrast their relative dominance in the CNS, coronary, splanchnic, renal, cutaneous, and skeletal muscle vascular beds.

CV 99. Describe the role of angiogenesis in providing a long-term match of tissue blood flow and metabolic need.

Fetal and Neonatal Circulation

CV 100. Describe the progressive changes in maternal blood volume, cardiac output, and peripheral resistance during pregnancy and at delivery.

CV 101. Contrast the blood flow pattern in the fetus with that of a normal neonate, including the source of oxygenated blood.

CV 102. Describe the function in utero of the fetal ductus venosus, foramen ovale, and ductus arteriosus. Explain the mechanisms causing closure of these structures at birth.

CV 103. Discuss the relative differences in oxygen saturation and pressure for blood in the major blood vessels and cardiac chambers of the fetus. Explain how these values change at birth.

CV 104. Describe fetal cardiovascular responses to acute hypoxia.

CV 105. Explain the unfavorable consequences to the neonate if either the ductus arteriosis or the foramen ovale fails to close or if pulmonary vascular resistance fails to decrease.

Hemostasis and Injury, Hemorrhage, Shock

CV 106. Diagram the enzymes and substrates involved in the formation of fibrin polymers, beginning at prothrombin. Contrast the initiation of thrombin formation by intrinsic and extrinsic pathways.

CV 107. Contrast the mechanisms of anticoagulation of a) heparin, b) EGTA, and c) coumadin. Identify clinical uses for each agent.

CV 108. Describe the mechanisms of fibrinolysis by TPA (tissue plasminogen activator) and urokinase.

CV 109. Explain the role of the platelet release reaction on clot formation. Distinguish between a thrombus and an embolus.

CV 110. Explain why the activation of the clotting cascade does not coagulate all of the blood in the body.

CV 111. Describe the direct cardiovascular consequences of the loss of 30% of the circulating blood volume on cardiac output, central venous pressure, and arterial pressure. Describe the compensatory mechanisms activated by these changes.

CV 112. Explain three positive feedback mechanisms activated during severe hemorrhage that may lead to circulatory collapse and death.

CV 113. Contrast the change in plasma electrolytes, hematocrit, proteins, and colloid osmotic pressure following resuscitation from hemorrhage using a) water, b) 0.9% NaCl, c) plasma, and d) whole blood.

Coronary Circulation

CV 114. Describe the phasic flow of blood to the ventricular myocardium through an entire cardiac cycle. Contrast this cyclic variation in myocardial flow a) in the walls of the right and left ventricles and b) in the subendocardium and subepicardium of the left ventricle.

CV 115. Explain how arterio-venous O_2 difference and oxygen extraction in the heart is unique when compared with other body organs.

CV 116. Explain the mechanism whereby coronary blood flow is coupled to myocardial workload, and identify stimuli that cause increases in coronary blood flow to occur.

CV 117. Explain how sympathetic stimulation alters heart rate, contractility, and coronary vascular resistance, as well as both directly and indirectly to change coronary blood flow. Identify the relative importance of the direct and indirect SNS effects in determining coronary blood flow during exercise.

CV 118. Describe what is meant by coronary vascular reserve and the role of collateral blood vessels. Discuss physiological and pathological events that decrease coronary vascular reserve.

Cerebral, Splanchnic, and Cutaneous Circulation

CV 119. Contrast the local and neural control of cerebral blood flow. Discuss the relative importance of O_2, CO_2, and pH in regulating cerebral blood flow.

CV 120. Describe the structural components of the blood-brain barrier and how this barrier impedes the movement of gases, proteins, and lipids from the blood to neurons. Identify the differences in cerebrospinal fluid and plasma relative to protein concentration, and describe the function of cerebrospinal fluid.

CV 121. Contrast the mechanisms of the two major types of stroke, hemorrhagic and occlusive stroke.

CV 122. Contrast the local and neural control of the splanchnic circulation. Describe the role of the hepatic portal system and the hepatic artery in providing flow and oxygen to the liver.

CV 123: Describe the blood pressure in the hepatic portal vein, hepatic sinusoids, and the vena cava. Given an increase in central venous pressure, predict how hepatic microcirculatory fluid exchange will be altered, including the development of ascites.

CV 124. Describe how the GI circulation is adapted for secretion and absorption. Explain the enterohepatic circulation.

CV 125. Contrast local and neural control of cutaneous blood flow.

CV 126. Discuss the unique characteristics of skin blood flow that are adaptive for body temperature regulation.

Exercise (also see Integration)

CV 127. Describe the cardiovascular consequences of exercise on peripheral resistance, cardiac output, A-V oxygen difference, and arterial pressure.

CV 128. Describe the redistribution of cardiac output during exercise to the CNS, coronary, splanchnic, cutaneous, and skeletal muscle vascular beds during sustained exercise (distance running). Explain the relative importance of neural and local control in each vascular bed.

CV 129. Discuss four adaptations to physical training on the cardiovascular system. Explain the mechanisms underlying each.

CV 130. Contrast the effects of static vs. dynamic exercise on blood pressure.

CV 131. Contrast the neural and local control of skeletal muscle blood flow at rest and during exercise.

CV 132. Contrast the effect of phasic and sustained skeletal muscle contraction on extravascular compression of blood vessels and on central venous pressure.

CV 133. Predict the changes in cardiac output and arterial pressure during the initial and the sustained phases of the Valsalva maneuver.