

SECTION 7 - THE CARDIOVASCULAR SYSTEM

EXERCISE 7.1 EFFECTS OF DRUGS ON THE FROG HEART

Approximate Time for Completion: 2-3 hours

Introduction

This exercise is designed to introduce students to the effects of drugs on the heart. Students expose the heart of a freshly killed frog and then record the effects of calcium, digitalis, pilocarpine, atropine, potassium, epinephrine, caffeine, and nicotine placed directly on the beating heart. From the recording, students determine the effect of these drugs on the cardiac rate (bpm), the strength of contraction (amplitude in mm), and the electrical conduction velocity (distance between atrial and ventricular peaks in mm). The design of the recording apparatus and the preparation setup should be discussed. Advise students to run a new control strip before adding each drug, then compare the drug effects with this control. Observation is very important. Atropine must be added while under the influence of pilocarpine or the desired effect will be missed, and the preparation might croak. Potassium is also very potent and may stop the heart in systole. If the frog heart stops, it can sometimes be started again by relieving the tension on the heart, rinsing off all drugs with Ringer's and gently massaging the heart with forceps; or by applying an electric shock (about 30 V) to the heart.

Materials

1. Frogs, dissecting instruments, and trays
2. Copper wire or bent pin, thread
3. Recording apparatus; Physiograph, transducer coupler, and myograph transducer (Narco); or kymograph, kymograph paper, and kerosene burner
4. Alternatively, you can use computerized data acquisition and analysis systems, such as those provided by Biopac and iWorx.
5. Drugs — all drugs should be added to Ringer's solution as the solvent (see recipe in exercise 5.1): Calcium chloride (2.0 g/dl); digoxin (or digitoxin, 0.2 g/dl); pilocarpine (2.5 g/dl); atropine (2.5 g/dl); potassium chloride (2.0 g/dl); epinephrine (adrenaline chloride, 0.01 g/dl); caffeine (0.2 g/dl); and nicotine (1.0 g/dl; or 6.16 ml of liquid nicotine/dl). Drugs can be obtained from Aldrich Chemical Company or Sigma Chemical Company.

Textbook Correlations: Chapter 12 – Cardiac Muscle

Chapter 13 – Structure of the Heart, Electrical Activity of the Heart

Answers to Questions

1. d
2. a
3. f
4. c
5. b
6. e
7. digitalis glycosides
8. atropine
9. abnormally high potassium levels in the blood
10. Hyperkalemia may cause the production of ectopic pacemakers and result in fibrillation. These effects are produced because a rise in plasma potassium concentration promotes the entry of K^+ into cardiac fibers resulting in depolarization and myocardial hyperexcitability.
11. A sympathomimetic drug mimics or duplicates the actions of the sympathetic nervous system. In this way, a sympathomimetic drug causes the heart to beat stronger (increased contractility) and faster (increased cardiac rate) and promotes a net vasoconstriction of the blood vessels (raises blood pressure).

12. By poisoning the Na^+/K^+ pump, digitalis increases the force of contraction (increased Ca^{2+}) and slows the rate of impulse conduction from the atria to the ventricles. The latter effect reduces the excitability of the ventricles that respond by beating more slowly. As a result of these actions, digitalis may be used in congestive heart failure to increase the pumping strength of the heart and it may be used to treat atrial flutter and fibrillation by decreasing the overall excitability of the heart.
13. Bathing the heart with Ca^{2+} increases the amplitude of the heart's contraction recording. The heartbeat is stronger yet the cardiac rate should slow. This occurs because unlike skeletal muscle contractions that are all or none, an increase in intracellular Ca^{2+} in cardiac muscle promotes the formation of a greater number of myosin cross bridges. The extra influx of Ca^{2+} lengthens the duration of the myocardial action potential at the calcium plateau and, consequently the duration of each contraction-relaxation cycle. The added intracellular calcium also increases the overall tension in the cardiac fibers even while relaxing between beats.
14. Theophylline, like caffeine, is a mild central nervous system (CNS) stimulant that also acts directly on the myocardium to increase both the strength of contraction and the cardiac rate. Theophylline inhibits the activity of the enzyme phosphodiesterase, which breaks down cyclic AMP second messenger molecules. As a result, rising cAMP concentration in heart cells mimics the action of epinephrine on the heart. Similarly, epinephrine and rising levels of cyclic AMP cause the smooth muscle fibers of the bronchioles to relax and dilate. Theophylline, therefore, duplicates and enhances the effects of epinephrine by promoting the rise in cyclic AMP levels in the bronchiolar smooth muscle, thereby relieving the stress of breathing in asthmatics.

EXERCISE 7.2 ELECTROCARDIOGRAM (ECG)

Approximate Time for Completion: 1 hour

Introduction

This exercise is designed to introduce students to the concept and method of measuring the electrical activity of the heart—the electrocardiogram. Students should be able to set up the monitor and discuss the physiological events that produce the basic ECG recording. A discussion of abnormal ECG tracings will enhance the concepts presented and will be of interest to most students. This exercise could be combined with exercises 7.3 and 7.4 for a laboratory period devoted to the electrical activity of the heart. If time is limited, the data collected in this exercise can be used to perform exercise 7.4 at home.

Materials

1. Electrocardiograph or other strip chart recorder, such as a Physiograph (Narco), or Lafayette Instrument Co., Inc. recorder with ECG module
2. Alternatively, you can use computerized data acquisition and analysis systems, such as Cardiocomp (Intelitool, Inc.) and those provided by Biopac and iWorx.
3. Electrode plates, rubber straps, electrolyte gel or paste
4. Alternatively, simulated electrocardiograph exercises may be performed using the *Physiology Interactive Lab Simulations*: Electrocardiogram and Heart Function (exercises 4 and 6).

Textbook Correlations: Chapter 13 – Electrical Activity of the Heart and the Electrocardiogram

Answers to Questions

1. Sinoatrial (SA) node
2. atrioventricular bundle (bundle of His)
3.
 - a. depolarization of the atria
 - b. depolarization of the ventricles
 - c. repolarization of the ventricles

4. gap junctions
5. tachycardia; bradycardia
6. first-degree AV block
7. standard limb
8. QRS
9. True
10. The SA node fibers serves as the pacemaker of the heart because they open special ion channels and undergo spontaneous depolarization faster than any other fibers in the heart. Consequently, the SA node sets the pace, generating action potentials that are rapidly conducted to other myocardial cells via intercalated discs (gap junctions). Although capable of spontaneous depolarization, other myocardial cells depolarize at a slower rate and thus normally follow the SA node rhythm. The depolarized cells of the atria respond first by contracting as a unit, followed shortly by the ventricles that similarly depolarize and contract. Furthermore, the SA node pace is influenced by autonomic neurotransmitters acetylcholine (slows) and norepinephrine (speeds), and by epinephrine (speeds).
11. Depolarization spreads through the atria producing the P wave. After a delay at the AV node, the depolarization then spreads down the bundle of His to the Purkinje fibers and the ventricular myocardium. This produces the QRS wave. Repolarization of the ventricles in the reverse direction produces the T wave. In third-degree, or complete AV node block the ventricles are not depolarized by impulses from the atria. The atria continue to beat according to the pace set by the SA node (supraventricular) while the ventricles beat according to the pace set by an ectopic pacemaker within the ventricles, responsible for the slower rate and the lack of response to the demands of exercise that raise the atrial pacing only.
12. Supraventricular tachycardia is characterized by an abrupt rapid atrial beat that drives a rapid ventricular beat for short intervals for periods of time. The ECG is normal in appearance and the ventricles are able to pump blood during this brief condition. In ventricular tachycardia however, the ventricles are not paced by the atria but rather by excitable ectopic foci in the ventricles that cause the ECG to have widened and distorted QRS complexes that can mask normal P waves. Ventricular tachycardia is more serious because it can degenerate into ventricular fibrillation and because the ventricular pumping is very erratic.
13. In third-degree AV node block none of the impulses from the atria reach the ventricles. No longer subservient to the SA node rhythm, the ventricle fibers beat at a much slower rate (20-45 beats per minute) and do not respond to any demands from the cardiovascular control center in the medulla oblongata. The atria however, are still responding to the medulla and will continue to beat at a normal pace (sinus rhythm).
14. At the time of the initial examination a P-R interval of 0.24 seconds defines a first-degree AV node block. A year later the AV conduction block has progressed with the patient now in third-degree AV node block as demonstrated by the appearance of ectopic pacemakers in the ventricles, the very slow rate of beat, and the lack of response to the demands of exercise. An artificial pacemaker may be installed to compensate for the complete conduction block.
15. Paroxysmal atrial tachycardia is also known as supraventricular tachycardia in which an ectopic focus above the ventricles results in spontaneous rapid running of the heart (150-250 beats per minute) that begins and ends abruptly. The ectopic focus extrasystoles often produce a subjective feeling in the chest, called palpitations. This condition is often treated successfully with digitalis. Digitalis decreases the excitability of the AV node and thus maintains the ventricular rate within the normal range, despite the excitability that may be occurring in the atria.

EXERCISE 7.3 EFFECTS OF EXERCISE ON THE ELECTROCARDIOGRAM

Approximate Time for Completion: 15 minutes

Introduction

This exercise shows the effects of exercise on the electrocardiogram. Since the setup is the same, this exercise should be performed while recordings for exercise 7.2 are being made.

Materials

1. Electrocardiograph or other strip chart recorder, such as a Physiograph (Narco), or Lafayette Instrument Co., Inc. recorder with ECG module
2. Electrode plates, rubber straps, electrolyte gel or paste
3. Alternative equipment: *Cardiocomp* (Intellitool, Inc.); Biopac; or iWorx. For Biopac, the pulse transducer and electrodes can be used for Student Lab Lesson 7.
4. Simulated electrocardiograph exercises may be performed using the *Physiology Lab Simulations: Electrocardiogram and Heart Function* (exercises 4 and 6).

Textbook Correlations: Chapter 13 – Pressure Changes During the Cardiac Cycle, The Electrocardiogram;
Chapter 14 – Regulation of Cardiac Rate

Answers to Questions

1. QRS
2. T
3. P
4. sympathetic (cardiac)
5. parasympathetic (vagus)
6. ischemia
7. At the beginning of the exercise, there is a decrease in the activity of the parasympathetic (vagus) innervation of the heart. This causes an increase in cardiac rate that is further raised during greater levels of exercise by increased sympathetic nerve (cardiac) activity. As a result of the faster rate, the period of diastole is shortened so that the time between the T wave of one ECG tracing and the P wave of the next tracing is significantly reduced. At higher heart rates the increased conduction velocity shortens the period of systole as shown by a decrease in the QRS-T interval.
8. During the P wave depolarization of the atria, pressure in the ventricles changes little. During the Q to R the septum is depolarizing and starting to contract with only slight changes in the ventricular pressure. The R to S interval completes depolarization of the lateral walls of the ventricles and produces the sharp rise in ventricular pressure. The S to T segment represents the peak depolarization and contraction resulting in maximum pressure developed by the ventricular fibers. During the peak of the T wave the ventricle fibers are undergoing repolarization and beginning diastole. The pressure in the ventricles during the T wave falls sharply, returning to the near zero value.
9. At rest the delivery of blood to the heart may be adequate for the needs of the myocardium despite the possibility that there may be some occlusion affecting coronary blood flow. After moderate exercise, the myocardial demand for oxygen is raised so that the myocardium may become ischemic as a result of the coronary occlusion. This myocardial ischemia may thus be revealed in the ECG taken during exercise primarily as an abnormally elevated or depressed S-T segment (ventricular repolarization) that was not present during the resting ECG. Variable answer, yet regular treadmill ECG tests are highly recommended in persons over 40 years of age, or sooner if heart disease runs in the family.
10. Electrical excitation of the cardiac fibers (ECG tracing) results in their mechanical contraction (pulse). More rapid discharge of the SA node, more rapid conduction of impulses, and a faster rate of contraction all result in an increased cardiac rate with exercise. The reverse occurs while resting. Increased cardiac rates are mainly due to shortening of the ventricular diastole and only secondarily due to a shortening of ventricular systole.

EXERCISE 7.4 MEAN ELECTRICAL AXIS OF THE VENTRICLES

Approximate Time for Completion: 15 minutes

Introduction

This exercise is designed to emphasize the use of the ECG to monitor the electrical activity of the heart and to showcase the versatility of the ECG in the determination of the mean electrical axis of the ventricles. This exercise also lends itself to further discussion of leads I and III, which should help the students, better visualize Einthoven's hypothesis and triangle. This exercise could be combined with exercise 7.2 and 7.3 for a laboratory period about the electrical activity of the heart or could be assigned as a take-home exercise using data from exercise 7.2.

Materials

1. Electrocardiograph or other strip chart recorder, such as a Physiograph (Narco), or Lafayette Instrument Co., Inc. recorder with ECG module; alternatively, the Biopac or iWorx systems may be used.
2. Electrode plates, rubber straps, electrolyte gel or paste
3. Alternatively, simulated electrocardiograph exercises may be performed using the *Physiology Interactive Lab Simulations*: Electrocardiogram and Heart Function (exercises 4 and 6).

Textbook Correlations: Chapter 13 – The Electrocardiogram

Answers to Questions

1. P
2. Q to the R
3. Leads I and III
4. left
5. His; right
6. When one ventricle is hypertrophied, a longer time will be required to depolarize the thicker muscle of that ventricle relative to the other, normal ventricle. In this way, hypertrophy changes the orientation (axis) of the heart in the chest and will change the QRS portion of the ECG. These changes in the ECG recorded at leads I (horizontal) and III (about 120 degrees) are used to note the change in the mean electrical axis of the ventricles. Whenever a longer time than normal is needed to depolarize one ventricle, the mean electrical axis will deviate in the direction of the affected ventricle. Hypertrophy of the left ventricle may occur as a result of hypertension or narrowing of the aortic semilunar valve. Right ventricular hypertrophy may be secondary to narrowing of the pulmonary semilunar valve or to such congenital conditions as a septal defect or the tetralogy of Fallot.
7. If there is a block in one branch of the atrioventricular bundle of His, it will take longer for action potentials to travel into the region of the ventricles served by that branch. A block in the left branch, for example, will cause the left ventricle to be depolarized more slowly than the right resulting in a shift in the mean electrical axis to the left. Bundle branch blocks are the result of the interruption of conduction along one branch of the bundle of His, such as the progression from first to third-degree AV conduction block.
8. The spread of action potentials into the ventricles from the AV node produces the QRS wave. As the impulses spread down the interventricular septum into the myocardium one region of the ventricles is depolarized (septum) while other regions (lateral walls) are not, creating a potential difference in the ventricles. This potential difference is seen by an upward deflection of the ECG from the baseline (Q to R). When the entire mass of the ventricles is depolarized and all regions have the same polarity, there is no potential difference in the ventricles and the ECG pattern returns to the baseline (R to S).
9. Both obesity and pregnancy present strains on the cardiovascular system that could result in hypertrophy of cardiac fibers. In both cases, the heart has to work harder to circulate blood through excess adipose tissue and to circulate extra blood volume to a growing fetus. The extra load on the left ventricle could cause left heart hypertrophy and shift the mean electrical axis to the left.

EXERCISE 7.5 HEART SOUNDS

Approximate Time for Completion: 15-30 minutes

Introduction

This exercise is designed to introduce students to the normal and abnormal sounds produced by the closing of heart valves. After this exercise, students should be able to properly use a stethoscope to monitor heart sounds. It is helpful to correlate the ECG with the mechanical forces that result in opening and closing of the cardiac valves. The use of a Physiograph allows a phonocardiogram to be obtained with the heart sounds correlated with the ECG waves. In this way, the sequence of both electrical and mechanical events in cardiac function can be presented and their interrelationships can be discussed.

Materials

1. Stethoscope
2. Physiograph, high-gain couplers, microphone for heart sounds (Narco)
3. Alternatively, the Biopac system may be used per Student Lab Lesson 17
4. Simulated electrocardiograph exercises may be performed using the *Physiology Interactive Lab Simulations: Electrocardiogram and Heart Function* (exercise 5).

Textbook Correlations: Chapter 13 – Cardiac Cycle and Heart Sounds, The Electrocardiogram

Answers to Questions

1. auscultation
2. simultaneous closure of the AV valves
3. systole
4. simultaneous closure of the semilunar valves
5. end of systole or the beginning of diastole
6. murmurs
7. end of the QRS
8. peak of the T wave
9. The QRS wave represents ventricular depolarization. When the ventricles depolarize they are stimulated to contract, causing pressure to rise in the ventricles and the AV valves to close simultaneously, with vibrations that produce the first heart sound. The T wave represents ventricular repolarization. When the ventricles repolarize electrically they begin to relax mechanically, causing the aortic and pulmonary semilunar valves to close simultaneously, with vibrations that produce the second heart sound.
10. As explained above heart sounds are normally produced by pressure changes causing two valves of the heart to close simultaneously, producing vibrations that can be heard with a stethoscope. The first heart sound, “lub,” is caused by the closure of the right and left atrioventricular valves. The second heart sound, “dub,” is caused by the closure of the aortic and pulmonary semilunar valves. Heart murmurs are caused by structural defects in the valves that affect the pattern of blood flow through the heart. Abnormal patterns of blood flow produced by defective valves can cause abnormal vibrations and thus abnormal heart sounds that can be heard by auscultation. Heart murmurs may be caused by an irregularity in a valve, a septal defect, or the persistent fetal opening (foramen ovale) between the right and left atria after birth.
11. Splitting of the heart sounds refers to hearing two separate components to the lub (first sound) and/or the dub (second sound). This is caused by deep inspiration promoting an increase in venous return and the separate closure of the two AV valves or separate closure of the aortic and pulmonary valves to handle the increased volume.
12. A defective valve produces abnormal flow patterns that can only be detected only by auscultation. Electrocardiography does not detect valve defects and palpation with the finger is too qualitative to permit accurate detection of any changes in the peripheral pulse at the radial artery.

13. None of these techniques can be used to detect the beating of the atria. The atrial beat occurs during the P to R interval and is not noticeable on the ECG, and auscultation and palpation of the radial artery are not sensitive enough to detect the small pressure changes generated by the atrial contraction.

EXERCISE 7.6 MEASUREMENT OF BLOOD PRESSURE

Approximate Time for Completion: 1 hour

Introduction

This exercise is designed to introduce students to the theory and techniques for measuring blood pressure. This exercise provides an opportunity to explain the mechanics of measuring blood pressure: that the occlusion of the cuff by pressures greater than the systolic pressure prevents the flow of blood through the cuff, but that blood can flow when the systolic pressure is greater than cuff pressure. After this exercise, students should be able to define important terms, to explain the concepts underlying the measurement of blood pressure, and to perform a simple blood pressure measurement. Remind the students that sphygmomanometer pressures above 200 mmHg are very painful for the individual being monitored.

Materials

1. Stethoscope
2. Sphygmomanometer
3. Alternatively, the Biopac system may be used with Student Lab 16.

Textbook Correlations: Chapter 14 – Blood Pressure, Hypertension

Answers to Questions

1. systolic pressure
2. diastolic pressure
3. vibrations due to the turbulent flow of blood in a partially compressed artery
4.
 - a. 168
 - b. 112
 - c. 56
 - d. approximately 130
5. Hypertension
6. cardiac output; total peripheral resistance
7. sphygmomanometer
8. Vibrations due to the turbulent flow of blood in a partially compressed artery cause Korotkoff sounds. Before the cuff is inflated, arterial blood flows in a smooth layered, or laminar pattern. The absence of turbulence results in silent blood flow. When the pressure in the sphygmomanometer cuff partially compresses the artery, the narrowing of the lumen creates turbulent blood flow and the vibrations produced are characterized as Korotkoff sounds.
9. As the inflated cuff is allowed to deflate slowly, the initial turbulent flow through the cuff will begin producing the first (Korotkoff) sound when the cuff pressure is equal to the systolic pressure. When the cuff pressure equals the diastolic pressure the artery is no longer compressed, laminar flow is restored and no more sounds are detected.

10. The position of the arm directly affects the measurement of diastolic pressure partially due to the effect of gravity. When the arm is below the heart level the diastolic pressure measured by the cuff is raised. When the arm is positioned above the heart level, the diastolic pressure is lowered. This helps to explain why the cuff should be placed at the heart level on the arm of the subject during blood pressure measurements. The greatest drop in diastolic pressure should occur immediately upon standing. Theoretically, the highest diastolic pressures should be read when the subject is lying down or immediately upon standing with activation of the baroreceptors reflex response. However, there are many factors that can vary results from the expected - such as anxiety, sympathetic drive, respiratory movements (laughter), and others.
11. During exercise the increase in heart rate and stroke volume (cardiac output) raises the systolic pressure more than it raises the diastolic pressure. However, both pressures can rise to as high as 200/100 mmHg. The pulse pressure (systolic minus diastolic) in this case would increase to 100 mmHg while the mean arterial pressure (1/3 pulse pressure plus diastolic pressure) would rise accordingly. The exercise-induced vasodilation causes the diastolic pressure to rise the least.
12. Regular aerobic exercise should lower the resting blood pressure. Exercise often results in a lowering of the resting cardiac rate and an increase in the resting stroke volume. Athletes have a greater degree of inhibition of the SA node by the vagus nerve and an increased blood volume. Working together these factors increase in cardiac output, improve the delivery of oxygen to the tissues, and result in a lowered blood pressure.

EXERCISE 7.7 CARDIOVASCULAR SYSTEM AND PHYSICAL FITNESS

Approximate Time for Completion: 30 minutes

Introduction

This exercise is designed to introduce students to the effects of physical activity on the cardiovascular system. Students will apply the measurement of blood pressure techniques learned in exercise 7.6 and will learn how to measure pulse rate in various body positions. This exercise provides students the opportunity to improve their technique of measuring blood pressure in the reclining and standing, as well as sitting positions.

Materials

1. Sphygmomanometer
2. Stethoscope, clock with second hand
3. Chair, stair, or platform 18 inches high
4. Optional: pulse and blood pressure may be monitored with the Biopac system using the pulse transducer and blood pressure cuff.

Textbook Correlations: Chapter 12 – Adaptations of Muscles to Exercise Training
Chapter 14 – Circulatory Changes During Exercise

Answers to Questions

1. falls
2. lower
3. endurance training – Results from higher levels of inhibitory activity by the vagus nerve.
4. Aerobic capacity is the maximal oxygen uptake or the maximum rate of oxygen consumption by the body.
5. Lactate threshold is that percent of one's aerobic capacity when the muscle's anaerobic respiration production of lactic acid (lactate) rises significantly (normally at about 50-70% of one's aerobic capacity).
6. a higher maximum cardiac output, and thus higher rates of oxygen delivery to the muscles in endurance athletes.

7. Endurance training increases the cardiac output through a higher blood volume and a stronger contraction of the ventricles, which thus ejects more blood per beat. The increase in oxygen demand also stimulates an increase in blood volume. The higher stroke volume and larger blood volume allow the same cardiac output to be achieved at a slower heart rate and thus endurance improves with training and training improves performance.
8. Persons who are physically fit may have a higher stroke volume and, therefore, a greater cardiac output than those who are unfit. Higher cardiac output increases the rate of blood flow and the delivery of oxygen to the exercising muscles. Conditioned muscles also have an increased ability to extract oxygen from the blood because of biochemical and metabolic changes within the muscle cells. At a given level of physical exercise, therefore, the physically fit have a slower rate of increase in the cardiac rate and a faster rate of return to the resting cardiac rate after exercise. Since mean arterial blood pressure is directly related to cardiac rate and to cardiac output, the fit person will have a slower rise in blood pressure and a correspondingly faster return to resting pressures after exercise.
9. Both aerobic capacity and lactate threshold declines with age beyond about age 29. This results in part from the decrease in the maximum cardiac rate with age, accompanied by a decrease in cardiac output and thus in the maximal rate of oxygen delivery to the tissues. Also, ageing alters the biochemical and metabolic processes in muscle tissue such that the anaerobic metabolism and lactic acid production occur sooner at lower levels of activity, thus lowering the lactate threshold.
10. Myocardial ischemia is inadequate blood flow to the heart muscle via the coronary circulation. The heart requires a continuous supply of oxygen and has only a limited ability to respire anaerobically. If oxygen delivery is inadequate for an extended period, myocardial ischemia may occur that is often accompanied by pain, angina pectoris. Severe ischemia may result in a myocardial infarction (heart attack). Since physical activity raises the myocardial demand for oxygen, the presence of myocardial ischemia with consequent changes in the S-T segment of the ECG, may be evident during a medically supervised treadmill test that was not observed at rest. Regular exercise in a healthy person helps the heart maintain a higher cardiac output and greater blood volume at a lower workload than that seen in the sedentary person. Regular exercise, therefore, along with physical checkups, healthy eating habits, avoidance of smoke, and healthy blood lipid levels would all contribute to promoting the adequate flow of blood through the coronary blood vessels and to minimizing the risk of myocardial ischemia.