# The Cardiovascular System: The Heart Outline

18.1 The heart has four chambers and pumps blood through the pulmonary and systemic circuits (pp. 664–670; Figs. 18.1–18.5)

- A. The right side of the heart receives oxygen-poor blood returning from body tissues and pumps it to the lungs to release $CO_2$ and pick up $O_2$. (p. 664; Fig. 18.1).
- B. The left side of the heart receives oxygenated blood from the lungs and pumps it out to the body to supply oxygen to tissues. (p. 664; Fig. 18.1).
- C. The heart has two receiving chambers, the right and left atria, and two pumping chambers, the right and left ventricles. (p. 664; Fig. 18.1).
- B. Size, Location, and Orientation (pp. 664-665; Fig. 18.2)
  1. The heart is the size of a fist and weighs 250–300 grams.
  2. The heart is found in the mediastinum and two-thirds lies left of the midsternal line.
  3. The base is directed toward the right shoulder and the apex points toward the left hip.
- C. Coverings of the Heart (p. 666; Fig. 18.3)
  1. The heart is enclosed in a double-walled sac called the pericardium.
     - a. The superficial pericardium is the fibrous pericardium that protects the heart, anchors it to surrounding structures, and prevents the heart from overfilling.
     - b. Deep to the fibrous pericardium is the serous pericardium, consisting of a parietal layer that lines the inside of the pericardium, and a visceral layer (epicardium) that covers the surface of the heart.
  2. Between the visceral and parietal layers is the pericardial cavity, containing a film of serous fluid that lubricates their movement against each other.
- D. Layers of the Heart Wall (pp. 666–667; Figs. 18.3–18.4)
  1. The epicardium is the visceral pericardium of the serous pericardium.
  2. The myocardium is composed mainly of cardiac muscle and forms the bulk of the heart.
     - a. Within the myocardium exists a network of connective tissue fibers, the cardiac skeleton, which reinforces the myocardium, supports the heart valves and provides electrical insulation between areas of the heart.
  3. The endocardium lines the chambers of the heart and is continuous with the endothelial linings of the vascular system.
- E. Chambers and Associated Great Vessels (p. 667; Fig. 18.5)
  1. There are partitions that separate the heart longitudinally: the interatrial septum divides the atria, and between the ventricles lies the interventricular septum.
  2. The right and left atria are the receiving chambers of the heart and only minimally contract to propel blood into the ventricles.

a. There are three veins that enter the right atrium: the superior and inferior vena cavae, which return blood from the body, and the coronary sinus, which returns blood from the myocardium.
b. Four pulmonary veins enter the left atrium from the lungs.
3. The ventricles are the discharging chambers of the heart: the right ventricle pumps blood into the pulmonary trunk, while the left ventricle pumps blood into the aorta.

18.2 Heart valves make blood flow in one direction (pp. 671–673; Figs. 18.5–18.8)

A. There are two atrioventricular (AV) valves between each atrial-ventricular junction: the tricuspid valve in the right and the mitral valve in the left (pp. 671-673; Figs. 18.5–18.8).
1. When the heart is relaxed, the AV valves hang loosely down into the ventricles.
2. When the ventricles contract, blood is forced upward against the flaps of the AV valves, pushing them closed.
3. Each flap of the AV valves are anchored to papillary muscles in the ventricles by collagen strings called chordae tendinae, which prevent eversion of the valves into the atria.

B. Aortic and pulmonary semilunar (SL) valves are located at the base of the arteries exiting the heart and prevent backflow of blood into the ventricles (p. 673; Figs. 18.5–18.8).
1. When ventricular pressure rises above aortic and pulmonary pressure, the SL valves are forced open, allowing blood to be ejected from the heart.
2. When the ventricles relax, blood flows backward toward the heart, filling the cusps of the SL valves, forcing them closed.

C. There are no valves at the entrances of the vena cavae or pulmonary veins, because the intertia of blood and the collapse of the atria during contraction minimizes backflow into these vessels (p. 673).

18.3 Blood flows from atrium to ventricle, and then to either the lungs or the rest of the body (pp. 673–676; Figs. 18.9–18.10; Focus Figure 18.1)

A. The right side of the heart pumps blood into the pulmonary circuit; the left side of the heart pumps blood into the systemic circuit. (pp. 673-674; Focus Figure 18.1)

B. Equal volumes of blood are pumped to the pulmonary and systemic circuits at the same time, but the two sides have different workloads. (p. 675; Fig. 18.9)
1. The right ventricle is thinner walled than the left and is a short, low-pressure circulation.
2. The left ventricle is three times thicker than the right, enabling it to generate a much higher pressure, so that the force can overcome the much greater resistance in the systemic circulation.

C. Coronary Circulation (pp. 675-676; Fig. 18.10)
1. The heart receives no nourishment from the blood as it passes through the chamber, so a series of vessels, the coronary circulation, exist to supply blood to the heart itself.

| Coronary Arteries | | |
|---|---|---|
| Main Vessel:serving the myocardium | Branches from Main Artery | Part of Heart Served |
| Left Coronary Artery | Anterior Interventricular Artery | Interventricular septum and anterior walls of both ventricles |
| | Circumflex Artery | Left atrium and posterior walls of left ventricle |
| Right Coronary Artery | Right Marginal Artery | Lateral right side of the heart |
| | Posterior Interventricular Artery | Posterior interventricular sulcus/ Posterior aspect of the heart |

| Coronary Veins | | |
|---|---|---|
| Main Vessel: returns to the right artium | Branches into Main Vein | Location |
| Coronary Sinus | | Posterior aspect of the heart |
| | Great Cardiac Vein | Anterior interventricular sulcus |
| | Middle Cardiac Vein | Posterior interventricular sulcus |
| | Small Cardiac Vein | Right inferior margin |
| Anterior Cardiac Veins | | Anterior aspect of the heart |

18.4 Intercalated discs connect cardiac muscle fibers into a functional syncytium (pp. 676–679; Fig. 18.11; Table 18.1)

A. Microscopic Anatomy (pp. 676-678; Fig. 18.11)

1. Cardiac muscle is striated and contraction occurs via the sliding filament mechanism.
2. The cells are short, fat, and branched, and each cardiac muscle fiber has one or two large, pale, centrally located nuclei.
3. Intercellular space is filled with a matrix of loose connective tissue that connects the muscle to the cardiac skeleton.
4. Cells are connected to each other at intercalated discs, containing desmosomes for structural strength, and gap junctions that allow electrical current to travel from cell to cell.
5. Cardiac muscle cells have large mitochondria that occupy 25–35% of the total cell volume, have myofibrils arranged in sarcomeres, but have a less extensive sarcoplasmic reticulum, when compared to skeletal muscle.

B. How Does the Physiology of Skeletal and Cardiac Muscle Differ? (pp. 678–679; Table 18.1)

1. Some cardiac muscle cells are self-excitable and initiate their own depolarization, as well as depolarizing the rest of the heart.

2. All fibers of the heart contract as a unit, or not at all because, unlike skeletal muscle motor units, gap junctions electrically tie all cardiac muscle cells together.
3. Release of $Ca^{++}$ from the sarcoplasmic reticulum is triggered by and influx of $Ca^{++}$ from the extracellular fluid, not by the depolarization wave, as in skeletal muscle.
4. The heart's absolute refractory period is longer than a skeletal muscle's, preventing tetanic contractions.
5. Cardiac muscle has more mitochondria than skeletal muscle, indicating reliance on exclusively aerobic respiration for its energy demands, and is capable of switching nutrient pathways to use whatever nutrient supply is available, including lactic acid.

18.5 Pacemaker cells trigger action potentials throughout the heart (pp. 679–685; Figs. 18.12-18.17)

A. Setting the Basic Rhythm: The Intrinsic Conduction System (pp. 679–681; Figs. 18.12–18.13)
   1. The heart does not depend on the nervous system to provide stimulation; it relies on gap junctions to conduct impulses throughout the heart and the intrinsic conduction system.
   2. The intrinsic conduction system consists of cardiac pacemaker cells have an unstable resting potential and produce pacemaker potentials that continuously depolarize, initiating the action potentials that are conducted throughout the heart.
   3. Impulses pass through the cardiac pacemaker cells in the following order: sinoatrial node, atrioventricular node, atrioventricular bundle, right and left bundle branches, and the subendocardial conducting network.
      a. The sinoatrial node is located in the right atrium and is the primary pacemaker for the heart.
      b. The atrioventricular (AV) node, in the interatrial septum, delays firing slightly, in order to allow the atria to finish contracting before the ventricles contract.
      c. The atrioventricular (AV) bundle is the only electrical connection between the atria and the ventricles and conducts impulses into the ventricles from the AV node.
      d. The right and left bundle branches conduct impulses down the interventricular septum to the apex.
      e. The subendocardial conducting network penetrates throughout the ventricular walls, distributing impulses throughout the ventricles.

B. Modifying the Basic Rhythm: Extrinsic Innervation of the Heart (p. 682; Fig. 18.14)
   1. The autonomic nervous system modifies the heartbeat through cardiac centers in the medulla oblongata:
      a. The cardioacceleratory center projects to sympathetic neurons throughout the heart, increasing both heart rate and contractile force.
      b. The cardioinhibitory center sends impulses to the parasympathetic dorsal vagus nucleus in the medulla oblongata, which stimulates the vagus nerve to the heart, slowing the heartbeat.

C. Action potentials in cardiac muscle cells are generated by the following mechanism (pp. 682–683; Fig. 18.15):

1. When cardiac muscle cells are stimulated, voltage gated $Na^+$ channels allow $Na^+$ to enter the cell, resulting in rapid depolarization.
2. Depolarization opens slow $Ca^{++}$ channels, allowing $Ca^{++}$ to enter the cell, even as $K^+$ exits, producing a plateau phase of the action potential that delays repolarization.
3. After 200 msec, $Ca^{++}$ channels are inactivated, $K^+$ channels open, and the cell rapidly repolarizes back to the resting membrane potential.

D. Electrocardiography (pp. 683–684; Figs. 18.16–18.18)
1. An electrocardiograph monitors and amplifies the electrical signals of the heart and records it as an electrocardiogram (ECG).
    a. A typical ECG has three deflections: a P wave, indicating depolarization of the atria; a QRS complex, resulting from ventricular contraction; and a T wave, caused by ventricular repolarization.

18.6 The cardiac cycle describes the mechanical events associated with blood flow through the heart (pp. 685–688; Figs. 18.19–18.20)

A. Systole is the contractile phase of the cardiac cycle and diastole is the relaxation phase of the cardiac cycle. (p. 685)

B. A cardiac cycle consists of a series of pressure and volume changes in the heart during one heartbeat. (pp. 685–688; Fig. 18.19)
1. Ventricular filling occurs during mid-to-late ventricular diastole, when the AV valves are open, semilunar valves are closed, and blood is flowing passively into the ventricles.
2. The atria contract during the end of ventricular diastole, propelling the final volume of blood into the ventricles.
3. The atria relax and the ventricles contract during ventricular systole, causing closure of the AV valves and opening of the semilunar valves, as blood is ejected from the ventricles to the great arteries.
4. Isovolumetric relaxation occurs during early diastole, resulting in a rapid drop in ventricular pressure, which then causes closure of the semilunar valves and opening of the AV valves.

C. Heart Sounds (pp. 687-688; Fig. 18.20)
1. Normal
    a. The first heart sound, lub, corresponds to closure of the AV valves, and occurs during ventricular systole.
    b. The second heart sound, dup, corresponds to the closure of the aortic and pulmonary valves and occurs during ventricular diastole.
2. Abnormal
    a. Heart murmurs are extraneous heart sounds due to turbulent backflow of blood through a valve that does not close tightly.

18.7 Stroke volume and heart rate are regulated to alter cardiac output (pp. 688–692; Figs. 18.21–18.22)

A. Cardiac output is defined as the amount of blood pumped out of a ventricle per minute, and is calculated as the product of stroke volume and heart rate. (pp. 688–689; Fig. 18.21)
1. Stroke volume is the volume of blood pumped out of a ventricle per beat, and roughly equals 70 ml.
2. The average adult heart rate is 75 beats per minute.
3. Cardiac output changes with demand; cardiac reserve is the difference between the resting and maximal cardiac output.

B. Regulation of Stroke Volume (pp. 689–690; Fig. 18.22)
1. Stroke volume represents the difference between the end diastolic volume (EDV), the amount of blood that collects in the ventricle during diastole, and end systolic volume (ESV), the volume of blood that remains in the ventricle after contraction is complete.
2. The Frank-Starling law of the heart states that the critical factor controlling stroke volume is preload, the degree of stretch of cardiac muscle cells immediately before they contract.
3. The most important factor determining the degree of stretch of cardiac muscle is venous return to the heart.
4. Contractility is the contractile strength achieved at a given muscle length; contractile strength increases if there is an increase in cytoplasmic calcium ion concentration.
5. Afterload is the ventricular pressure that must be overcome before blood can be ejected from the heart and does not become a significant determinant of stroke volume except in hypertensive individuals.

C. Regulation of Heart Rate (pp. 690–691)
1. Sympathetic stimulation of pacemaker cells increases heart rate and contractility by increasing $Ca^{++}$ movement into the cell.
2. Parasympathetic inhibition of cardiac pacemaker cells decreases heart rate by increasing membrane permeability to $K^{+}$.
3. Hormones such as epinephrine and thyroxine increase heart rate.
4. Ion imbalances can interfere with the normal function of the heart.
5. Age, gender, exercise, and body temperature all influence heart rate.

D. Homeostatic Imbalance of Cardiac Output (p. 692)
1. Congestive heart failure occurs when the pumping efficiency of the heart is so low that blood circulation cannot meet tissue needs.
2. Pulmonary congestion occurs when one side of the heart fails, resulting in pulmonary edema.

Developmental Aspects of the Heart (pp. 692–694; Figs. 18.23–18.24)

A. Before Birth (pp. 692–693; Figs. 18.23–18.24)
1. The heart begins as a pair of endothelial tubes that fuse to make a single heart tube with four bulges representing the four chambers.
2. The foramen ovale is an opening in the interatrial septum that allows blood returning to the pulmonary circuit to be directed into the atrium of the systemic circuit.
3. The ductus arteriosus is a vessel extending between the pulmonary trunk and the aortic arch that allows blood in the pulmonary trunk to be shunted to the aorta.

B. Heart Function Throughout Life (p. 694)
1. Regular exercise causes the heart to enlarge and become more powerful and efficient.
2. Sclerosis and thickening of the valve flaps occurs over time, in response to constant pressure of the blood against the valve flaps.
3. Decline in cardiac reserve occurs due to a decline in efficiency of sympathetic stimulation.
4. Fibrosis of cardiac muscle may occur in the nodes of the intrinsic conduction system, resulting in arrhythmias.

5. Atherosclerosis is the gradual deposit of fatty plaques in the walls of the systemic vessels.