

Cardiovascular Emergencies

**EMS Continuing Education
Technician through Technician-Advanced Paramedic**

**Consistent with the
National Occupational Competency Profiles
as developed by
Paramedic Association of Canada
and
“An Alternate Route to Maintenance of Licensure”
as developed by Manitoba Health**

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Disclaimer

These documents were developed for improved accessibility to “An Alternative Route to Maintenance of Licensure” for all paramedics in Manitoba. Regional implementation of Alternate Route is at the discretion of the local EMS Director.

This is a supportive document to the National Occupational Competency Profiles and “An Alternative Route to Maintenance of Licensure.” It is not the intent that this package be used as a stand-alone teaching tool. It is understood that the user has prior learning in this subject area, and that this document is strictly for supplemental continuing medical education. To this end, the Paramedic Association of Manitoba assumes no responsibility for the completeness of information contained within this package.

It is neither the intent of this package to supercede local or provincial protocols, nor to assume responsibility for patient care issues pertaining to the information found herein. Always follow local or provincial guidelines in the care and treatment of any patient.

This package is to be used in conjunction with accepted models for education delivery and assessment, as outlined in “An Alternative Route to Maintenance of Licensure”.

This document was designed to encompass all licensed training levels in the province Technician, Technician-Paramedic, Technician-Advanced Paramedic. Paramedics are encouraged to read beyond their training levels. However, the written test will only be administered at the paramedic’s current level of practice.

All packages have been reviewed by the Paramedic Association of Manitoba’s Educational Subcommittee and physician(s) for medical content.

As the industry of EMS is as dynamic as individual patient care, the profession is constantly evolving to deliver enhanced patient care through education and standards. The Paramedic Association of Manitoba would like to thank those practitioners instrumental in the creation, distribution, and maintenance of these packages. Through your efforts, our patient care improves.

This document will be amended in as timely a manner as possible to reflect changes to the National Occupational Competency Profiles, provincial protocols/Emergency Treatment Guidelines, or the Cognitive Elements outlined in the Alternate Route document.

Any comments, suggestions, errors, omissions, or questions regarding this document may be referred to info@paramedicsofmanitoba.ca , attention Director of Education and Standards.

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Introduction

Heart disease is the leading cause of morbidity and mortality among the North American adult population, and, since cardiac-related complaints comprise a significant proportion of pre-hospital incidents, it is imperative that EMS personnel have a thorough understanding of cardiovascular anatomy, physiology, pathophysiology, etiology, disease, and pre-hospital assessment and treatment.

Although this package focuses on the cardiovascular system (CVS), it is important to bear in mind the relationship between the cardiovascular and other body systems. The normal body conducts a series of activities (ingesting and distributing nutritive material, replenishing body cells and fluids, maintaining homeostasis, eliminating metabolic wastes, defending the body against pathogens, and so on), which are carried on simultaneously by a number of different systems (respiratory, digestive, endocrine, excretory, immune, etc.). The cardiovascular system plays a critical role in supporting and connecting the other body systems by distributing oxygen, nutrients and hormones throughout the body, and by directing toxins and metabolic wastes to the organs of excretion. By reducing or increasing the amount of blood in contact with the skin, the cardiovascular system helps regulate body temperature as well. The cardiovascular system also serves as a vehicle for the maintenance of homeostasis and the operation of the immune system. Successful pre-hospital treatment of cardiovascular emergencies depends on the ability of paramedics to recognize and treat the primary complaint, but overall care of the patient is enhanced when EMS personnel are aware that the potential sequelae of cardiac malfunction can affect the patient's general health in many ways.

The aim of this module is to prepare EMS personnel of all levels to respond to cardiac-related calls. An understanding of basic cardiovascular anatomy and physiology, as well as the pathophysiology of cardiovascular diseases, provides a foundation for the efficient pre-hospital assessment and treatment of cardiovascular-related complaints.

Cardiac Anatomy And Physiology

Location and Structure

The heart is a hollow muscular organ lying obliquely in the mediastinum behind the sternum, between the lungs, and just above the diaphragm. The base (top) of the heart is approximately level with the second intercostal space and the apex (bottom) is about the level of the fifth intercostal space, mid-clavicular line (Figure 1). In most people, two thirds of the heart's mass is located to the left of the sternum (this is reversed in a small percentage of the population).

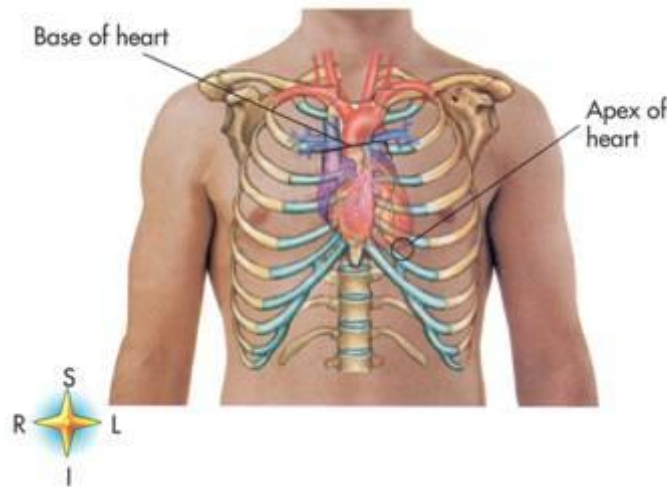


Figure 1. Location of the heart in the thoracic cavity (From Aehlert, 2002).

The heart is divided into four chambers; the upper, smaller atria and the larger, more muscular ventricles. The left and right atria and ventricles are separated by the interatrial and interventricular septa respectively. Valves control Blood flow between the four chambers. Superficially, the division between the chambers can be seen as the coronary sulci (grooves), which encircle the heart. The anterior surface is primarily the right ventricle, while the posterior is primarily the left ventricle. The normal heart is vaguely cone-shaped; about 12 cm long by 9 cm wide, and typically weighs between 250 and 350 grams. Throughout an average adult lifetime, the heart pumps about 4000 litres of blood per day through 96000 km of blood vessels.

The great vessels are the major arteries and veins, which enter and leave the heart. Blood is brought to the heart through the superior vena cava, which drains the upper body and head, and the inferior vena cava, which drains the lower body. Blood being pumped to the lungs exits the heart through the pulmonary arteries (the only arteries which carry deoxygenated blood) and returns through the pulmonary veins (the only veins that carry oxygenated blood). Finally, blood is pumped out to the body through the aorta, the largest blood vessel in the body (Figures 2 & 3).

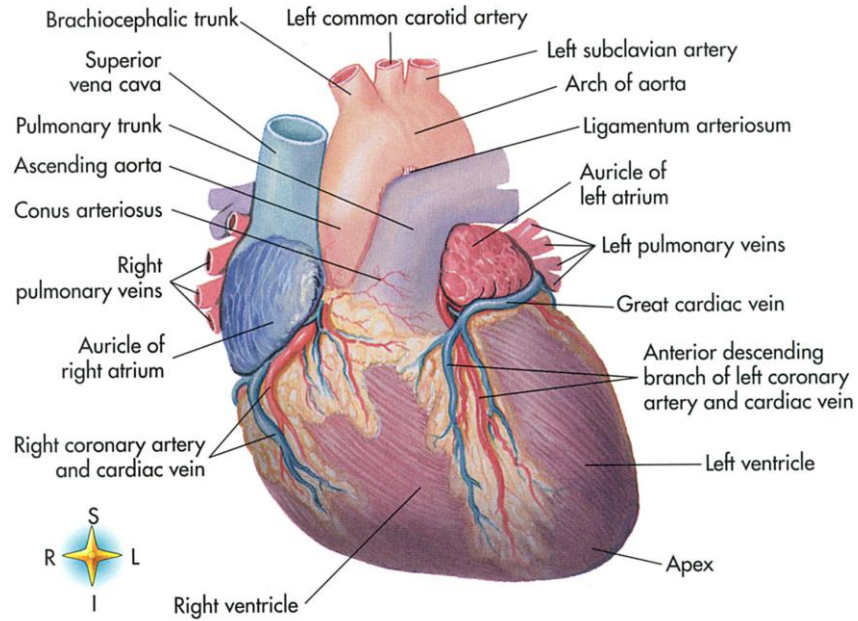


Figure 2. Anterior view of the heart showing major arteries and veins (from Aehlert, 2002).

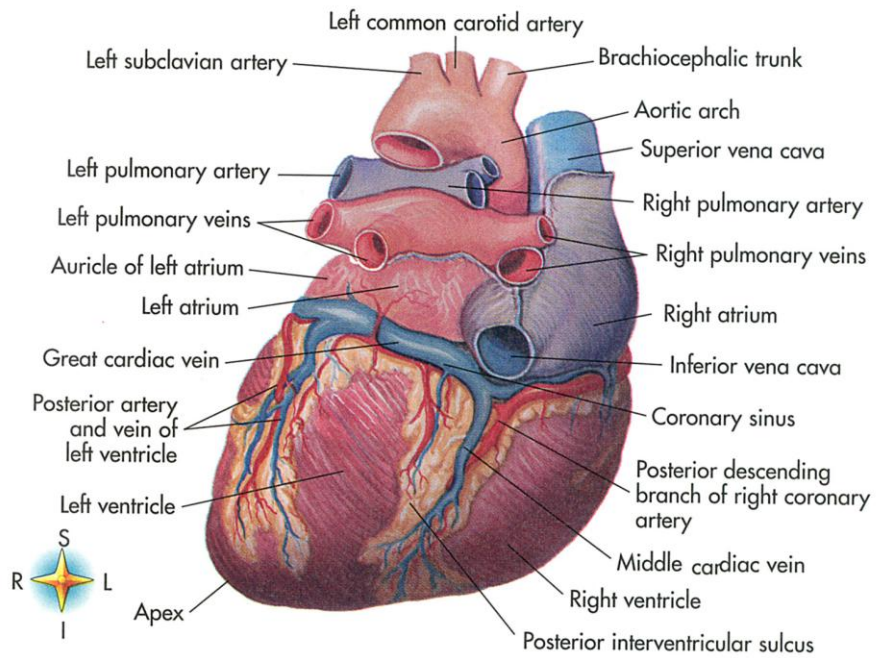
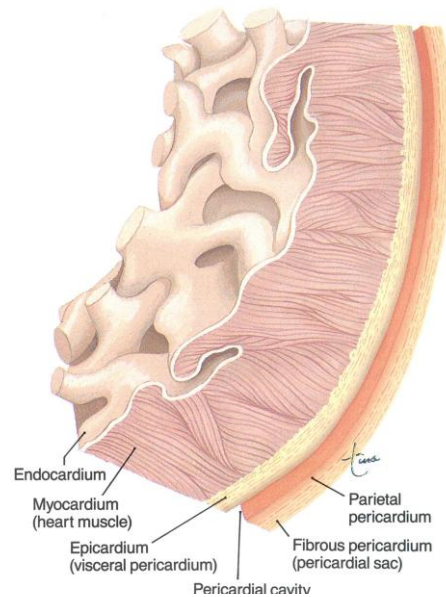


Figure 3. Posterior view of the heart showing major arteries and veins (from Aehlert, 2002).

The heart is enclosed in the double-walled pericardial sac. The tough outer *fibrous pericardium* prevents overfilling and anchors the heart to the diaphragm, sternum, and walls of the arteries, which exit the heart. The inner *serous pericardium* is a thin membrane with two delicate layers: the parietal, which lines the inside of the fibrous pericardium; and the visceral, which reflects back onto the surface of the heart. A small amount of lubricating fluid fills the slit-like space between the parietal and visceral layers of the serous pericardium.

The heart itself consists of three tissue layers: the outer layer (epicardium), middle muscular layer (myocardium) and inner lining (endocardium). The *epicardium* and the visceral layer of the serous pericardium are two names for the same tissue layer, which is connected to the underlying myocardium by a thin layer of connective tissue containing fat cells, nerves, and coronary blood vessels. The *myocardium* is the functional part of the heart. It consists of a specialized type of striated, involuntary muscle which continues into the muscular aorta and large arteries. Muscle fibers are joined to each other by specialized connections. The muscle sheets of the myocardium are attached to the “cardiac skeleton”, which is dense fibrous connective tissue that forms the central supporting structure of the heart. The *endocardium* is a thin layer, which covers all internal surfaces of the heart, including the four valves. The inner surface of the endocardium is made of flattened endothelial cells and is continuous with the lining of the blood vessels (Figure 4).

Figure 4. Heart wall showing tissue layers and pericardium (from Bledsoe et al. 2003).



Heart Valves

The four heart valves control the direction of blood flow through the chambers of the heart. The two *atrioventricular* (AV) valves are located between the atria and ventricles. The valve separating the right atrium from the right ventricle is the *tricuspid* valve. The *bicuspid* (Mitral) valve separates the left atrium from the left ventricle. The valve leaflets are connected to specialized (papillary) muscles in the ventricles. When relaxed, these papillary muscles open the valves and allow blood to flow between the atria and ventricles. The cusps of the tricuspid and mitral valves are anchored to the papillary muscles by strands of tendon called the chordae tendineae. The chordae tendineae prevent prolapse of the valves during ventricular contraction. There are two *semilunar* valves located between the ventricles and the arteries into which they pump blood. The *pulmonic* valve separates the right ventricle from the pulmonary arteries and the aortic valve separates the left ventricle from the aorta. These two valves prevent the backflow of blood into the ventricles during diastole (Figure 5).

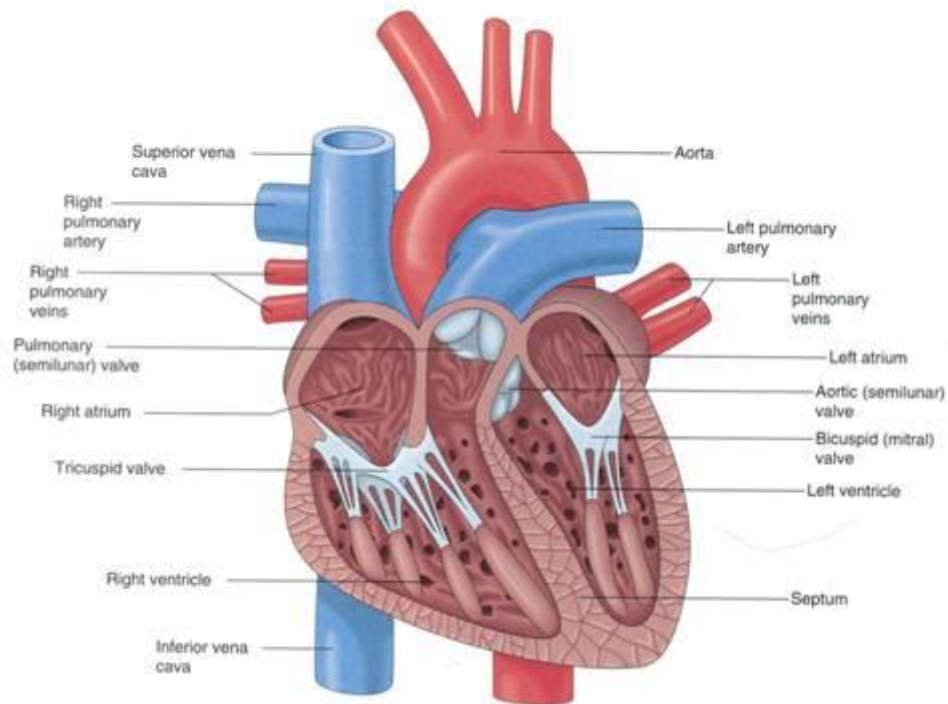


Figure 5. Internal view of the heart showing cardiac valves (from Meyers et.al., 2002).

Cardiac Cycle

The cardiac cycle refers to the repetitive pumping process that includes all the events associated with blood flow through the heart. Although the heart is a four-chambered organ, it functions as two adjacent pumps, each with an upper chamber (the atrium) and a lower chamber (the ventricle). The two sides of the heart beat simultaneously in a two-phase cycle. Each beat corresponds to a precise sequence of *contraction (systole)* and relaxation (*diastole*) phases (Figure 6).

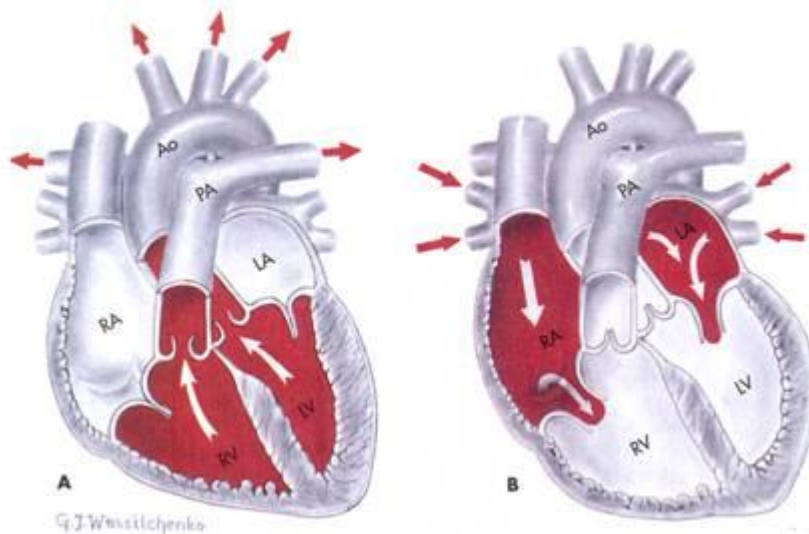


Figure 6. The cardiac cycle of systole (A) and diastole (B) (from Gould, 1997).

Deoxygenated blood enters the right atrium from the superior and inferior venae cavae. As the right atrium fills with blood, the pressure increases, triggering the tricuspid valve to open during atrial contraction (atrial systole) and allow the blood to flow into the relaxed right ventricle (ventricular diastole). It is important to remember that the majority of ventricular filling is passive. Approximately 25 ml of blood is contributed during atrial systole. This is often referred to as “atrial kick”. Approximately 105 ml of blood enters the ventricle passively, during ventricular diastole. Most healthy individuals can lose their atrial kick without significantly affecting their cardiac output. This is not necessarily true of patients with ischemic heart disease or patients with atrial fibrillation where there is a rapid ventricular response. Some patients can be very dependent on their atrial kick and the loss of even this small volume of blood can have a significant impact on their cardiac output. When the right ventricle is full, it contracts (ventricular systole) and blood is pumped out through the pulmonary valve and into the pulmonary arteries to the lungs, (pulmonary circulation) where carbon dioxide is released and oxygen is picked up through the alveolar-capillary membrane. Oxygenated blood is returned to the left atrium through the pulmonary veins. When the left atrium contracts, the same principles apply as blood passes through the mitral valve into the powerful left ventricle which then contracts and pumps blood out through the aortic valve into the aorta and on to all body tissues (systemic circulation)(Figure 7). Closure of the valves prevents back-flow of blood during the cardiac cycle. The heart sounds (“lubb-dubb”) which are heard during auscultation, result from vibrations transmitted during closure of the valves.

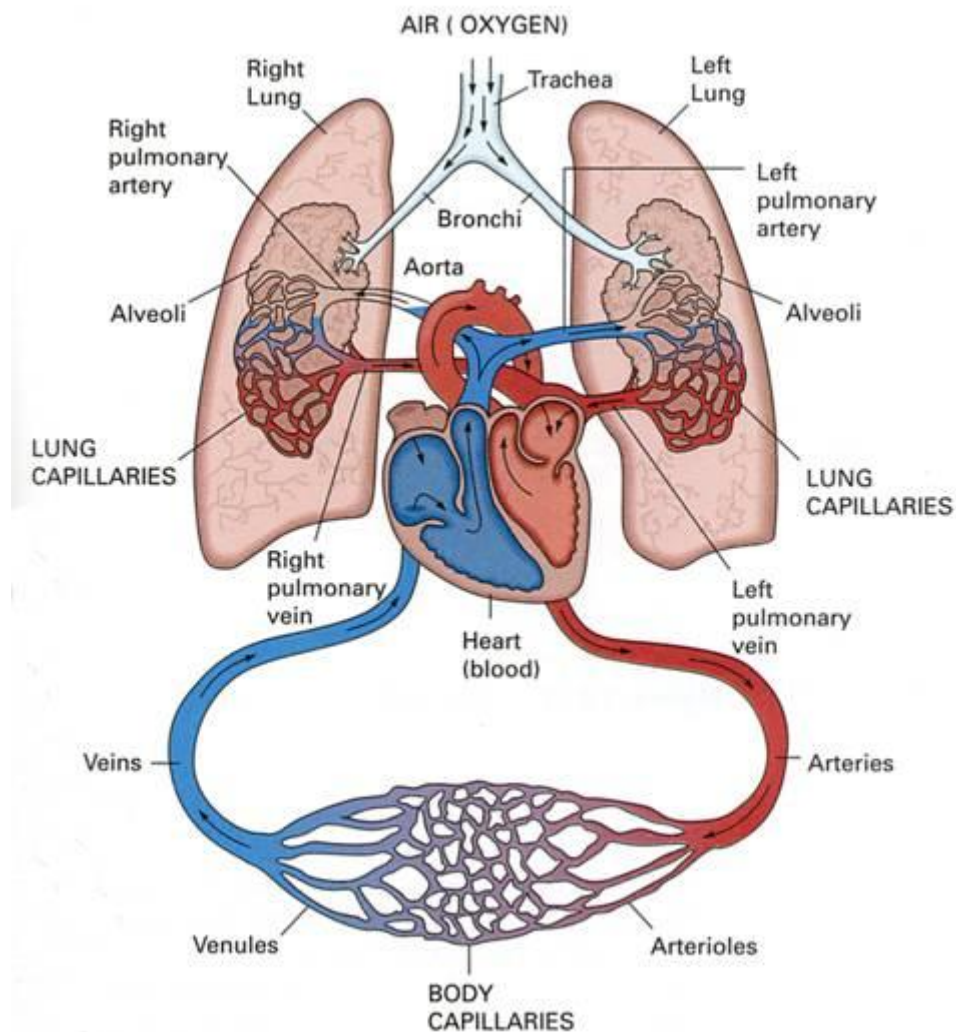


Figure 7. Circulation of blood through the cardiovascular system (from Hafen et. al, 1996).

Cardiac function can be measured by a number of parameters and a knowledge of the physiologic concepts of the cardiac cycle will help the EMS provider understand some of the complications with cardiac pump (or blood volume) related disorders.

Efficiency of the cardiac cycle can be measured by the *cardiac output*, which is the volume of blood ejected by a ventricle in one minute. The cardiac output (CO) depends on heart rate (HR, cardiac cycles or “beats” per minute) and stroke volume (SV, amount of blood pumped by a ventricle in one contraction, average is 75 ml). This relationship can be summarized by the equation: $CO = HR \times SV$. In an average healthy adult, cardiac output is approximately five litres per minute, but varies with a number of factors such as: activity level; metabolic rate; body size and temperature; or with pathologic conditions such as shock, hypovolemia, and heart failure. Cardiac reserve refers to the ability of the heart to increase output in response to increased demand. When necessary, a healthy heart may increase cardiac output by four or five times its minimum volume.

Two important parameters, which play a direct role in a patient’s cardiac output, are preload and afterload. *Preload* is defined as the degree of end-diastolic myocardial fiber stretch and is essentially a measure of venous return. When an increased amount of blood returns to the heart, as during exercise, the heart is stretched more and must contract with greater force. Certain pathologic conditions (such as left ventricular dilation, hypertrophy, or ischemic damage to the ventricular wall) will affect preload. *Afterload* is a measure of the peripheral resistance against which the ventricle must pump in order to eject blood through the semilunar valves. It is directly related to the pressure in the aorta and must be overcome with each contraction. Afterload approximates systemic blood pressure at, or shortly after, aortic valve opening. If afterload is increased, the ventricle must work harder and thus the myocardial oxygen demand will be increased.

Coronary Circulation

The myocardium requires a constant supply of oxygen and nutrients, but has very little storage capacity for oxygen; therefore, an efficient coronary circulation is vital to the functioning of the cardiovascular system as a whole. Myocardial tissue is supplied with oxygenated blood by the right and left coronary arteries. These are the first two branches of the aorta and open just distal to the cusps of the aortic valve. Many small branches extend inward into the myocardium where they further divide to become arterioles and capillaries. Since coronary arteries are somewhat constricted during systole, blood flow through the myocardium is greatest during diastole, when cardiac muscle is relaxed. This is an important concept to remember during states of hypoperfusion/hypotension or cardiac arrest, as the diastolic pressures will be minimal or non-existent and coronary circulation will be negatively impacted.

Distribution of blood flow from these arteries varies among individuals, but generally, the right coronary artery supplies the right side of the heart and the inferior portion of the left ventricle. The left coronary artery divides into two major branches: the left anterior descending artery, which brings blood to the anterior wall of the ventricles; and the circumflex artery, which nourishes the left atrium

the lateral and posterior walls of the left ventricle (Figures 2 & 3). Anastomosis (direct connections) exists between small branches of the left and right coronary arteries. These junctions have the potential to provide collateral circulation if an artery becomes obstructed. Regular aerobic exercise contributes to cardiovascular fitness by stimulating the development of collateral channels. The blood from the heart is drained by the coronary veins, the course of which generally parallels that of the arteries. Branches of the coronary veins join at the coronary sinus, from where the blood empties into the right atrium. The anterior cardiac veins empty directly into the right atrium (Figures 2 & 3).

Nervous Control of the Heart

Both the sympathetic and parasympathetic divisions of the autonomic nervous system innervate the heart. *Sympathetic stimulation* increases heart rate and contractility (adrenergic response) whereas *parasympathetic stimulation* slows the heart (cholinergic response). Autonomic control requires special nervous tissue (sensors), which detects changes in cardiac function. Two types of sensors are involved in cardiac control: *baroreceptors*, which detect changes in blood pressure; and *chemoreceptors*, which detect changes in pH, oxygen, or carbon dioxide levels in the blood. Afferent nerves carry sensory information to the cardiac control center in the medulla. The medulla interprets the information and determines what body parameters need adjustment. Efferent (motor) nerves transmit signals from the medulla to the site of action. *Neurotransmitters*, which are released from the motor nerves, bind to receptors (specialized nerve tissue) and initiate a compensatory response.

Sympathetic innervation occurs through the cardiac plexus, which is a nerve complex situated close to the aortic arch. The main neurotransmitter is *norepinephrine*, which can increase heart rate, force of ventricular contraction, blood pressure and cardiac output. Sympathetic receptor sites are divided into alpha and beta-receptors. Norepinephrine exerts its effects primarily through its actions on the beta-receptors. Stimulation of beta one receptors (found in the heart) causes increased heart rate, contractility and irritability whereas stimulation of beta-two receptors (found in the lungs and skeletal muscle blood vessels) causes dilation of blood vessels and bronchiolar smooth muscle.

Parasympathetic innervation is primarily via the vagus (tenth cranial) nerve. The main parasympathetic neurotransmitter is *acetylcholine*. The net effect of parasympathetic stimulation is to reduce the workload of the heart. This occurs because release of acetylcholine slows the heart rate, slows the rate of electrical conduction between the atria and ventricles, decreases strength of atrial contraction, and can cause a small decrease in the strength of ventricular contraction.

Three terms used in relation to the effects of nervous stimulation on the heart are inotropic, chronotropic and dromotropic. *Inotropic* refers to the strength of myocardial contraction. An increase in the strength of contraction is called a positive inotropic effect; a decrease is called a negative inotropic effect. *Chronotropic* refers to the rate of myocardial contraction. An increase in the rate is a positive chronotropic effect; a decrease is a negative chronotropic effect. *Dromotropic* is a term more concerned with the electrical conduction system of the heart, but in essence, it refers to the speed of conduction of electrical impulses through the heart. A positive dromotropic effect increases the speed of impulse conduction and a negative dromotropic effect decreases it. These terms are also commonly used when discussing the effects of medications on cardiac function.

Blood Pressure

Understanding blood pressure is important to good pre-hospital assessment of cardiac function. Blood pressure refers to the pressure of blood against the systemic arterial walls and reflects the force generated during systole. Blood pressure varies throughout the body, being higher at the proximal end of the arteries and lower in the capillaries. Blood pressure also varies during the cardiac cycle: it is at a maximum during systole and minimum during diastole. Pulse pressure is the difference between systolic and diastolic pressures. Blood pressure depends largely on cardiac output and peripheral vascular resistance (the resistance to blood flow created by friction between blood and the vessel walls), which is determined by blood vessel diameter and the tone of the vascular musculature.

It is generally agreed that elasticity of the arteries is the most important factor influencing blood pressure, but other specific variables include: size of vessel walls; blood volume and viscosity; venous return; rate and force of cardiac contractions. Blood pressure also changes in response to emotional state, activity, smoking, caffeine, and a variety of disease states. When a drop in blood pressure is sensed by the baroreceptors, the cardiac control center initiates a sympathetic response (increased vasoconstriction, heart rate, and myocardial contractility). If the blood pressure rises, the sympathetic response is suppressed and parasympathetic stimulation helps to slow the heart and lower blood pressure.

Heart Rate

Heart rate is a major determinant of cardiac output and tissue perfusion and thus is one of the most important parameters by which EMS personnel assess a patient's condition. Heart rate is commonly measured as the pulse, which is the number of beats (or cardiac cycles) per minute. Recognizing differences in heart rhythm (regular or irregular) and pulse quality (weak, thready, or strong) are also important skills for good pre-hospital care.

Heart rate is influenced by a number of factors: general cardiovascular health; blood pressure; blood volume; respiratory adequacy; tissue perfusion; extracellular ions; hormone levels; disease states; medications; stress; emotional state; age; body temperature; and general activity level. The sensors, which detect changes in heart rate, are located in the aortic arch and internal carotid arteries. Responses to changes in heart rate are directed through the sympathetic or parasympathetic pathways and result in a positive or negative chronotropic effect.

Cardiac Electrophysiology

The heart consists of striated, involuntary muscle containing two specialized types of cells, the electrical (pacemaker) cells and the mechanical (myocardial) cells. The electrical cells have three distinct properties: automaticity; excitability; and conductivity. *Automaticity* is the ability of a cell to spontaneously generate and discharge an electrical impulse. *Excitability* refers to the ability of the cell to respond to an electrical impulse. *Conductivity* is the ability of the cell to transmit or conduct an electrical impulse from one cell to another. Cardiac muscle fibers consist of myocardial cells, which are long, branching cells that fit tightly together at special junctions called intercalated disks. The intercalated disks permit rapid conduction of an electrical impulse throughout the wall of a cardiac chamber. This feature allows cardiac muscle cells to function together in a syncytium (a network of combined cells). That is, when one cell becomes excited, the impulse spreads rapidly across the myocardium, resulting in a coordinated contraction.

A specialized pathway of electrical cell fibers, which coordinates the heart, controls the mechanical pumping of the heart beat, by regulating contractions of the atrial and ventricular myocardium. This specialized pathway, or *conduction system*, is responsible for the spontaneous generation and conduction of electrical impulses in the heart. The fibers of this system have a faster rate of electrical conduction than do ordinary myocardial fibers (Figure 8).

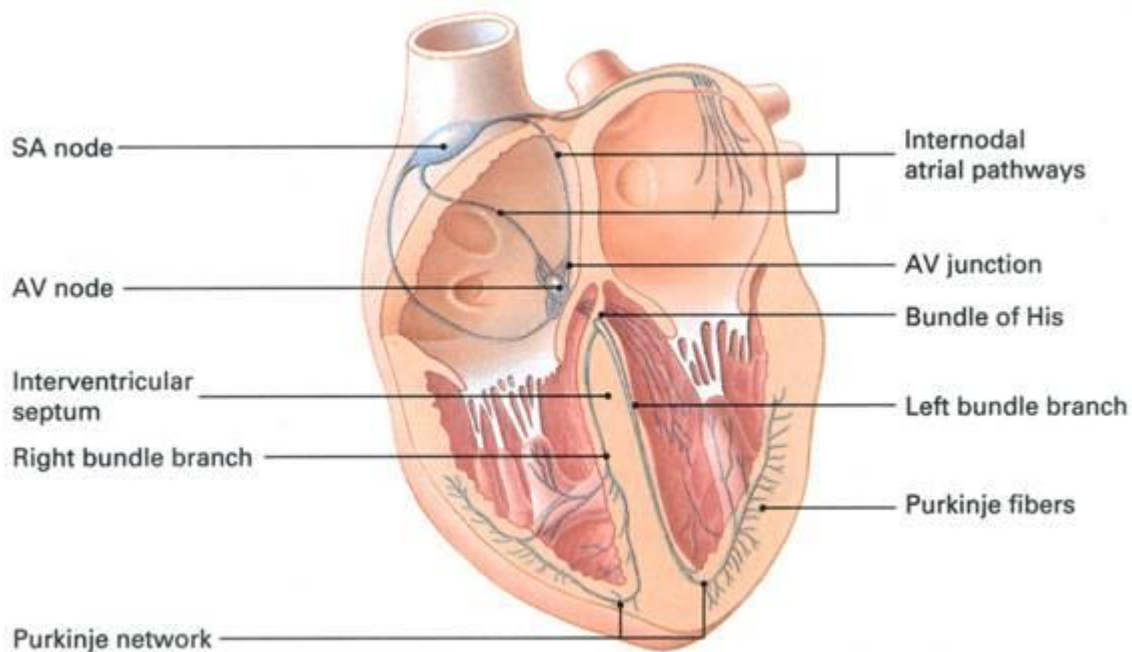


Figure 8. The cardiac conduction system (from Bledsoe et. al., 2003).

An important concept in cardiac electrophysiology is *polarization*. This refers to the resting state of myocardial cells during which no electrical activity occurs in the heart; the inside of the cell is more negatively charged than the outside as a result of the numbers and types of ions present. This difference in electrical charge across the cell membrane is referred to as the *resting potential*. Before the heart can mechanically contract and pump blood, myocardial cells must *depolarize*, which is the stimulation of the cell caused by an inward flux of positive ions; first sodium and then, more slowly, calcium. Depolarization is an electrical event, which proceeds from the endocardium outward to the epicardium. Normally, depolarization will produce a contraction. When the contraction stops, the sodium ion influx stops and potassium diffuses out of the cell. In addition, sodium is actively pumped out of the cell. This return to the polarized state, which existed before myocardial contraction is known as repolarization. Repolarization proceeds from the epicardium inward to the endocardium.

Refractoriness is another concept, which is important to the understanding of cardiac electrophysiology. It refers to the extent to which a cell is able to respond to an electrical stimulus. In cardiac muscle, the refractory period is longer than the contraction itself. Because of this mechanism, tetanic (sustained) contractions cannot be induced in the myocardium.

The cardiac cycle is initiated when an electrical impulse is generated by the *sinoatrial (SA) node*, a dense network of small conduction fibers which lies at the junction of the vena cavae and the right atrium. The impulse generated by the SA node regulates the coordinated and progressive contraction of the myocardium. After leaving the SA node, the electrical impulse spreads along internodal atrial tracts, which conduct the depolarization impulse through the atrial muscle mass to the *atrioventricular (AV) node*, which lies in the median wall of the right atrium. The AV node delays conduction long enough for the atria to contract completely and fill the ventricles before passing the electrical impulse further along the conduction system. The AV node is continuous with the bundle of His, which passes through the cardiac skeleton to the posterior margin of the interventricular septum. At this point, the *bundle of His* divides into two trunks, one to each ventricle. The trunks are called the *left bundle branch* and *right bundle branch*, depending on their location. Ultimately, the bundle branches divide into a large number of smaller fibers called the *Purkinje fibers*, which terminate in the myocardial tissue where they connect with individual mechanical myocardial cells. Because of the arrangement of this conduction system, the impulse is spread from the SA node through the atria, and then quickly to the apex of the heart. The ventricles are thus stimulated to depolarize in an inferior-to-superior direction, which effectively pumps blood out to the pulmonary and systemic circulations.

Vascular Anatomy And Physiology

Blood vessels are one of the three parts of the cardiovascular system. Arteries, capillaries and veins constitute a closed system for the distribution of blood throughout the body. There are two main circulations; the pulmonary, which allows for gaseous exchange in the lungs, and the systemic, which provides for the exchange of nutrients and wastes between the blood and all cells of the body.

Blood Vessel Structure

Blood vessels consist of three main tissue layers. The innermost lining, the *tunica* (“tunic”, or coat) *intima*, consists of a single layer of endothelial cells. The middle layer, the *tunica media*, consists of elastic fibers and smooth (involuntary) muscle cells. This layer controls the diameter of the vessel, and also gives blood vessels their strength and recoil. The tunica media is much thicker in arteries than in veins. The outermost layer, the *tunica adventitia* (or tunica externa), is a connective, fibrous tissue covering which contains elastin and collagen fibers. This layer provides the vessel with the strength required to withstand the pressures generated by the pumping force of the heart (Figure. 9).

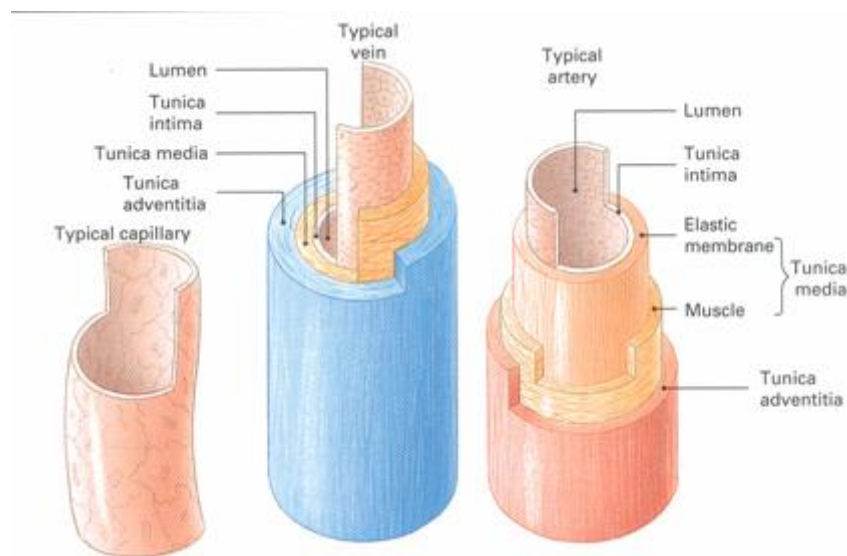


Figure 9. The structure of capillaries, veins, and arteries (from Bledsoe et. al., 2003).

Capillaries

Capillaries are the smallest blood vessels and they connect the arterial and venous sides of the circulation through a network or “microcirculation”. Their wall consists primarily of a single layer of endothelial cells, which facilitates the exchange of fluid, oxygen, carbon dioxide, electrolytes, glucose and other nutrients, and wastes between the blood and interstitial fluid. The lumen of a capillary is approximately the same diameter as a single red blood cell. Capillary networks vary in size and shape, depending on the metabolism of the tissues or organs in which they are found. For example, there is close network of capillaries in the lungs, liver, kidneys, and skeletal muscle, but a sparse network in the tendon, nerves and smooth muscle.

Arteries

The arterial system includes the largest vessels in the body and the walls of arteries have all three tissue layers. The tunica media is well developed and consists chiefly of smooth muscle cells interspersed with varying amounts of elastic and collagen fibers. Because of this structure, the large arteries are both muscular and elastic. Elasticity, especially in the aorta and its main branches, enables arteries to adjust to the changes in blood volume that occur during the cardiac cycle. For example, the aorta must expand during systole to prevent systolic pressure from rising too high, whereas during diastole, the walls must recoil to maintain adequate diastolic pressure. The walls of the arteries undergo changes with age; principally, the elasticity of the tunica intima and tunica media decreases both with normal aging and with various disease processes.

Veins

The venous system has a blood pressure about one-tenth of that in the arterial system and has a larger volume of blood (about 70 percent of the blood volume at any one time). Veins have thinner walls than arteries, with less smooth muscle and elastic fibers in the tunica media. Blood flow in the veins depends largely on skeletal muscle action, respiratory movements, and gravity. Valves in the larger veins of the extremities have an important role in maintaining the blood flow returning to the heart.

Vasa Vasorum

Arteries and veins with a diameter of more than one millimeter are themselves supplied by small blood vessels called *vasa vasorum*. The vasa vasorum enter through the adventitia and terminate in a dense capillary network in the tunica media.

Nervous Control of the Vasculature

At all times, even at rest, vascular tone is maintained by constant input from the sympathetic nervous system. The walls of blood vessels, particularly the arteries, have a rich nerve supply. Sensory nerve fibers terminate in free sensory endings within the walls of the vessels. Vasomotor nerves penetrate the adventitia and end at junctions with smooth muscle cells in the arterial media. Localized vasodilation or vasoconstriction is controlled by autoregulation, a local reflex adjustment which varies depending on the needs of cells in a particular area. For example, a decrease in oxygen or an increase in temperature may lead to local vasodilation. Local changes do not affect the systemic blood pressure. Systemic blood pressure can be increased through mechanisms such as the release of neurotransmitters like norepinephrine, which causes systemic vasoconstriction. Medications such as nitroglycerine or epinephrine are often used to treat cardiovascular complaints by inducing systemic vasodilation or vasoconstriction respectively.

Blood

Along with the heart and vessels, blood is the third major component of the cardiovascular system. Blood provides a major transport system for the body. In addition to oxygen, circulating blood delivers hormones to their site of action, nutrients to the liver and all body cells, and metabolic wastes to the liver and kidneys. The blood also plays a critical role in the immune system by carrying antibodies and white blood cells, which combat and remove pathogens and foreign material. Blood is a vehicle for the promotion of homeostasis in that it provides a mechanism for controlling body temperature; distributing core heat to peripheral tissues, and preserving core heat through peripheral vasoconstriction. Blood is the medium through which blood pressure and fluid levels are adjusted by a number of physiologic mechanisms. Blood buffers maintain serum pH within a narrow range (7.35 – 7.45). Clotting factors in circulating blood help to preserve hemostasis.

The average adult body contains about five litres of blood, which consists of water and cells. The water and dissolved solutes portion (plasma) makes up about 55% of blood volume. The remaining 45% consists of cells and formed elements: *erythrocytes* (red blood cells); *leucocytes* (white blood cells); and *thrombocytes* (platelets). Plasma is the clear, yellowish fluid remaining after all cells have been removed. Serum is the fluid and dissolved solutes remaining after cells and fibrinogen have been removed. The solutes in blood plasma include electrolytes, proteins (albumin, fibrinogen and antibodies), glucose, fats, bilirubin and gases.

Blood cells are created in the bone marrow or the spleen by the process of hemopoiesis. Erythrocytes are non-nucleated, flattened, disc-shaped cells which contain, among other things, hemoglobin. When oxygen is bound to the hemoglobin, the oxyhemoglobin thus formed gives arterial blood its bright red colour and is a critical element in tissue perfusion. Hemoglobin production is stimulated by tissue hypoxia. Hemoglobin with no bound oxygen, deoxyhemoglobin, is responsible for the dark, bluish-red colour of venous blood. A small portion of the carbon dioxide in venous blood is bound to the hemoglobin, but most carbon dioxide is carried free in the blood as bicarbonate ions. There are five main types of leucocytes, which vary in function and in physical characteristics. Their role in the immune response depends on a variety of factors, but in general it is dictated by the nature of the event; allergic response, inflammatory response, cell-mediated for humeral immunity, etc. Some leucocytes can leave the capillaries (diapedesis) when required for defensive purposes.

Pathophysiology Of Cardiovascular Illnesses And Injuries

INTRODUCTION

Diseases of the cardiovascular system are a major cause of morbidity and mortality in North America, especially in older adults. Since many disorders of the CVS share common symptoms, a basic knowledge of the pathophysiology of cardiovascular illnesses and injuries will help EMS personnel assess and treat patients with cardiovascular complaints.

Vascular Disease

Hypertension

Hypertension is a common, often asymptomatic, disorder of the vascular system characterized by a sustained elevated blood pressure persistently exceeding 140/90.

Primary or essential hypertension is the most common type and has a gradual onset over a number of years. Due to the insidious onset and mild signs, it may be undiagnosed until complications arise. Primary hypertension is idiopathic, but is known to occur in the presence of certain risk factors. Some risk factors such as obesity, hypercholesterolemia (high levels of cholesterol in the blood), stress, and sedentary lifestyle can be controlled to a certain extent, whereas other factors such as age, gender, and ethnicity cannot be controlled. Sustained hypertension is not only a disease in itself, but also contributes to, and serves as an indicator of, arteriosclerosis, congestive heart failure, angina, renal failure and stroke.

Secondary Hypertension is high blood pressure, which results from specific conditions such as pheochromocytoma, Cushing's syndrome and pre-eclampsia.

Malignant hypertension (hypertensive emergency) is an uncontrollable, severe, and rapidly progressive rise in blood pressure, which is characterized by complications such as severe headache, blurred vision and confusion and can result in a rapid decompensation of vital organ function.

Arteriosclerosis

Arteriosclerosis is a common vascular disorder characterized by degenerative changes such as thickening, calcification, and loss of elasticity of the arterial walls. As is the case with hypertension, risk factors for developing arteriosclerosis can be divided into two groups, those which can be modified (obesity, hypercholesterolemia, stress, sedentary lifestyle) and those which cannot (age, gender genetic factors).

The most common form of this disease is atherosclerosis in which *atheromatous plaque* (deposits of lipids, fibrin, cells and cellular debris) is deposited on the endothelium of large and medium-sized arteries. The coronary and cerebral arteries, aorta and peripheral arteries are principally affected, particularly at points of bifurcation where turbulent blood flow may encourage the development of atheromas. These deposits are also initiated at sites of endothelial damage. As the atheroma becomes larger, platelet adhesion often results in thrombus formation, which further contributes to the pathogenic changes. The loss of elasticity and narrowing of the arterial lumen impedes blood flow as

well as increasing blood pressure and workload on the heart. As atheromas in the coronary arteries increase in size, the reduced blood flow leads to ischemic angina and can cause myocardial infarction if the artery becomes totally occluded. Patients with arteriosclerosis are also at risk of developing aneurysms and peripheral vascular disease, as well as stroke and complications from thromboembolism (Figure 10).

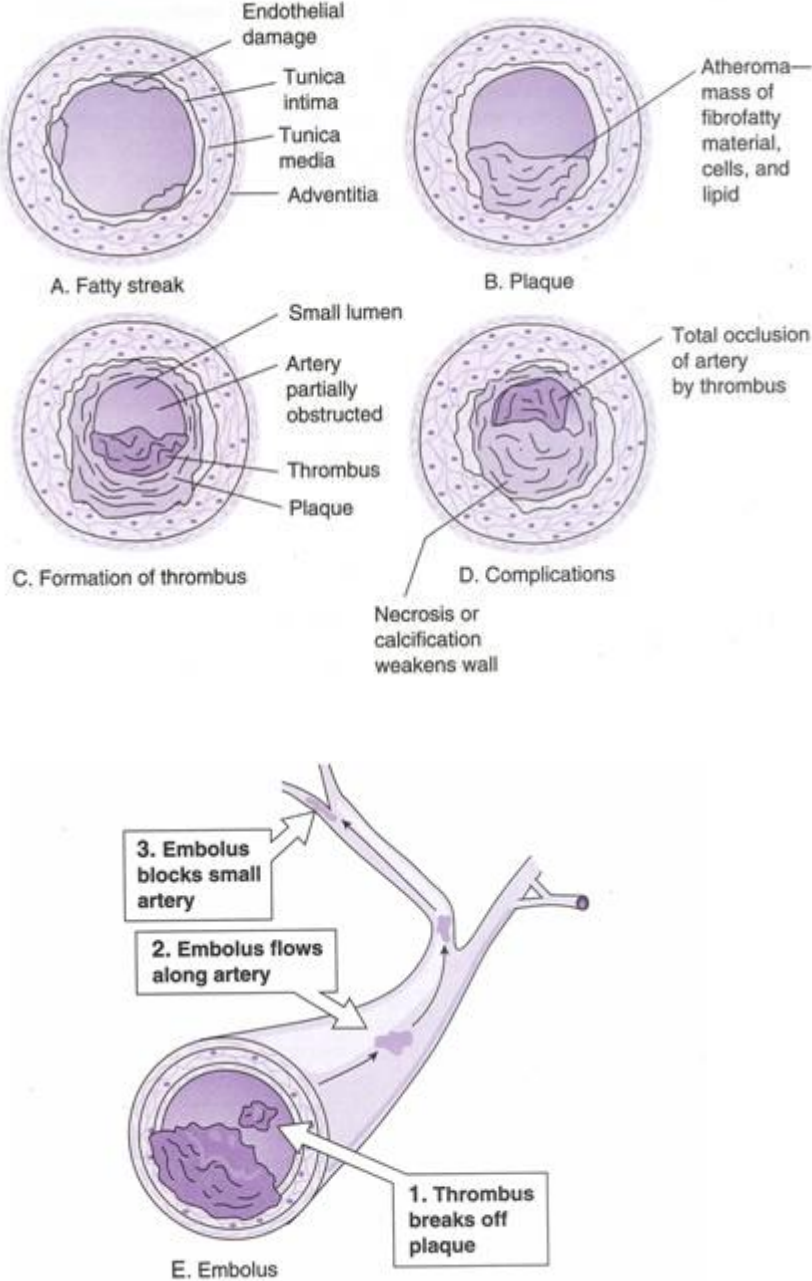


Figure 10. Development of an atheroma leading to arterial occlusion (from Gould, 1997).

Aneurysm

An aneurysm is a localized dilation or “ballooning” of an arterial wall. They are commonly classified by shape into three types: *saccular*, which is a bulging wall on one side of an artery; *fusiform*, which is a circumferential dilation along an artery; or *dissecting*, in which there is a tear in the intima, allowing blood to flow between the layers of the arterial wall (Figure 11).

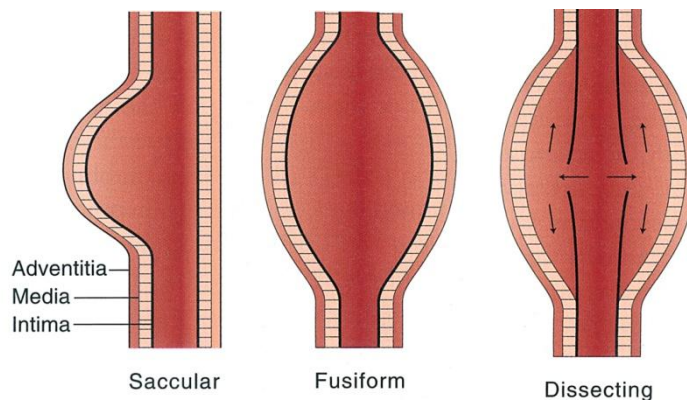


Figure 11. The three types of aneurysm (from Myers et. al., 2002).

Aneurysms most commonly occur in the thoracic or abdominal aorta, but are also found in the cerebral circulation. They usually develop at the site of an atheroma or bifurcation of a vessel where turbulent blood flow, often combined with increasing hypertension, cause a gradual dilation of the arterial wall. Thrombi frequently form in the dilated area where they may become a source of emboli, which further complicate the effects of the aneurysm.

Aneurysms are most often caused by arteriosclerosis, but may also result from hypertension, smoking, and less commonly, from syphilis, arteritis, connective tissue disorders, congenital defects and trauma (particularly motor vehicle accidents). Most aneurysms are asymptomatic until they become quite large or rupture, often with massive hemorrhage.

Deep Vein Thrombosis

Deep vein thrombosis (DVT) is a disorder involving a thrombus in one of the deep veins of the body. The most commonly affected are the iliac and femoral veins. A DVT can be asymptomatic, but often, as blood pools distal to the site, the patient develops tenderness, pain swelling, warmth, and discoloration of the skin. Valves in the blood vessels may be damaged, leading to hypertension. Long-term complications include venous insufficiency and ulceration in the lower leg. The most serious complication occurs when the thrombus (or a piece of it) becomes dislodged. In this case, the thromboembolus may travel through the circulation until it lodges in smaller vessels, where it may occlude blood flow to the heart, brain, or lungs.

Risk factors for developing DVT include; prolonged sitting, bed rest or immobility; recent surgery or trauma; fractures; first six months postpartum; estrogen therapy, and taking birth control pills. Virchow's triad (venous stasis, vessel wall injury, hypercoagulable state) is the primary mechanism for development of DVT. These three factors, especially if acting in concert, initiate activation of the clotting sequence in areas of reduced blood flow.

Peripheral Vascular Disease

Peripheral vascular disease (PVD) is caused by atherosclerotic plaque occluding the arteries which supply blood to the legs, principally the femoral and popliteal arteries. Symptoms are due to the narrowing of the vessel lumen. Patients with PVD generally have adequate blood supply to leg muscles during rest or minimal activity. However, as activity is increased, blood supply through the affected vessels becomes inadequate and the patient experiences muscle cramps. Cramps are relieved with rest. The condition of muscle cramping relieved by rest is called intermittent claudication. Acute occlusion of the peripheral arteries often involves the smaller arteries to the feet and toes and may lead to ischemic necrosis and gangrene if not treated. The etiology of PVD is unclear; however, risk factors include heredity, male sex, increasing age, smoking, hypertension, and hyperlipidemia. Diabetes, obesity, and inactivity are also thought to play a role.

Two specific types of PVD are Raynaud's Disease and Buerger's Disease (thromboangiitis obliterans).

Raynaud's disease is an idiopathic condition, more commonly seen in young women, characterized by episodic intense vasospasm of the small arteries in superficial tissue of the digits, particularly the fingers. The nose and ears are sometimes involved. The vasospasm, which is triggered by cold exposure, stress and smoking, causes a temporary ischemia, pallor, numbness and cyanosis which is followed by vasodilation, redness and throbbing pain. Normal colour and sensation are eventually restored. This condition is called Raynaud's phenomenon when it occurs secondary to conditions such as scleroderma, rheumatoid arthritis, systemic lupus erythematosus, drug intoxication, and trauma..

Buerger's Disease is an occlusive vascular disorder of the medium and small arteries, most commonly in the legs. The affected vessels become inflamed and thrombotic, which leads to fibrosis and vascular occlusion. Early signs of the condition are burning, numbness and tingling of the leg or foot distal to the lesion. Eventually, the patient develops ischemia, pain, and intermittent claudication and decreased or absent pulsation distal to the affected site. Phlebitis and gangrene may develop as the disease progresses. Young men are most commonly affected by this disorder. Smoking is a contributory factor.

Aortic Dissection

Aortic dissection is an uncommon, but potentially lethal condition resulting from a tear in the intimal layer of the aorta. This exposes the damaged vessel to the forces of blood pressure, which cleave or "dissect" the tissue layers and allow blood to flow along the length of the vessel between the layers of the arterial wall. The blood flow is usually antegrade, but occasionally it is retrograde. The most serious, sequelae of aortic dissection include aortic rupture and death through exsanguination, hemopericardium with cardiac tamponade or hemothorax. Other serious complications include heart attack or stroke, cardiac tamponade, bowel or limb ischemia, valvular regurgitation, acute heart failure, or irreversible kidney failure. There may be arterial insufficiency past the area of dissection due to the arterial lumen being compressed by the false lumen of the dissection. In such cases, renal, bowel, splanchnic, or limb ischemia may occur.

There are two main types of aortic dissections: acute and subacute. *Acute aortic dissections* involve sudden, severe symptoms and is the type usually encountered in EMS responses. As surgery is the only option, rapid recognition and transport is imperative. *Subacute aortic dissections* involve more gradual symptoms and patients may be treated with medications such as antihypertensives. When

accompanied by an aortic aneurysm, the condition is referred to as a dissecting aortic aneurysm. Dissections can be either ascending or descending, depending on whether the vessel damage occurred in the ascending or descending aorta. Ascending aortic dissection is considered the more dangerous of the two.

The major predisposing factors for aortic dissection is hypertension, but changes in the aorta produced by aging, hereditary defects of arterial connective tissue, blunt trauma, iatrogenic factors, and coarctation of the aorta (a congenital defect), may also lead to dissection. Cocaine abuse may result in a dissection as this drug can cause a rapid rise in blood pressure. The mortality rate from aortic dissection is nearly twice as high among cocaine addicts.

Acute Coronary Syndromes

Acute myocardial infarction (AMI) and unstable angina (UA) are part of a spectrum of clinical disease collectively identified as *acute coronary syndromes* (ACS). The pathophysiology common to this spectrum of disease is a ruptured or eroded atheromatous plaque. The electrocardiographic (ECG) presentation of these syndromes encompasses ST-segment elevation myocardial infarction (STEMI), ST-segment depression, and nondiagnostic ST-segment and T-wave abnormalities. A non-ST-elevation myocardial infarction (NSTEMI) is diagnosed if cardiac markers are positive with ST-segment depression or with nonspecific or normal ECGs. Sudden cardiac death may occur with any of these conditions. ACS is the most common proximate cause of sudden cardiac death.

Effective interventions for patients with ACS, particularly STEMI, are extremely time-sensitive. The first healthcare providers to encounter the ACS patient can have a big impact on patient outcome if they provide efficient risk stratification, initial stabilization, and referral for cardiology care. It is critical that basic life support (BLS) and advanced cardiovascular life support (ACLS) healthcare providers who care for ACS patients in the out-of-hospital, emergency department (ED), and hospital environments be aware of the principles and priorities of assessment and stabilization of these patients. These guidelines target BLS and ACLS healthcare providers who treat patients with ACS within the first hours after onset of symptoms, summarizing key out-of-hospital, ED, and some initial critical-care topics that are relevant to stabilization.

The primary goals of therapy for patients with ACS are to

- Reduce the amount of myocardial necrosis that occurs in patients with MI, preserving left ventricular (LV) function and preventing heart failure
- Prevent major adverse cardiac events (MACE): death, nonfatal MI, and need for urgent revascularization
- Treat acute, life-threatening complications of ACS, such as ventricular fibrillation (VF)/pulseless ventricular tachycardia (VT), symptomatic bradycardias, and unstable tachycardias

Angina Pectoris

Angina pectoris refers to any pain in the chest area. Angina is characterized by paroxysmal, often recurring, intermittent episodes of substernal chest pain. Such episodes may be triggered by physical or emotional stress. Angina is a symptom of the ischemia, which results when myocardial oxygen demand is greater than the supply. This may be caused by decreased myocardial perfusion, increased workload on the heart, or a combination of both. Angina is often a symptom of obstructive coronary artery disease processes in which the coronary arteries are unable to meet oxygen needs of the heart due to arteriosclerotic changes and hemodynamically significant stenosis. It may also result from conditions such as myocardial hypertrophy, in which the heart has outgrown its blood supply. There are three types of angina pectoris: stable; unstable; and variant (Prinzmetal's).

Stable angina is chest pain or discomfort which is often poorly localized and associated with activities which increase the myocardial oxygen demand such as physical exertion, emotional stress, respiratory infection with fever, exposure to extreme temperatures, or eating a large meal. Rest or administration of oxygen and/or sublingual nitroglycerine relieves stable angina.

Unstable angina is marked by a limitation of ordinary physical activity. It occurs at rest as well as with exertion, lasts greater than 20 minutes and often indicates new (several weeks) onset cardiac ischemia. This type of angina is often not relieved by nitroglycerine or rest, and is sometimes seen in a patient with previously diagnosed stable angina that has become unstable (severe, prolonged, or frequent). Unstable angina may or may not occur in the presence of atherosclerotic change.

Variant angina is thought to occur as a result of coronary artery spasm and often occurs in a cyclical fashion, at about the same time of day. It is not associated with exertion or stress, and almost always occurs at rest. Patients with this form of angina are usually younger than those with chronic stable or unstable angina.

It should be noted that chest pain is not necessarily due to cardiac ischemia. It may also be a symptom of esophageal spasm, gastro-esophageal reflux disease, biliary colic, pulmonary embolism, pericarditis, cardiac dysrhythmias, aortic dissection, or various traumatic injuries to the chest. Careful examination and history taking are essential to good pre-hospital treatment of patients with chest pain.

Myocardial Infarction

Infarction is cell death from a lack of oxygen. Myocardial infarction (MI) indicates the death of some portion of the myocardium and occurs as a result of a deficiency in coronary blood supply and the ensuing ischemic necrosis of the affected area. The most common cause is coronary artery atherosclerosis, which contributes to decreased blood flow in three main ways: atheromatous plaque and associated thrombi build up to occlude coronary arteries; vasospasm in the presence of partial occlusion by an atheroma leads to total obstruction; or thromboemboli can dislodge from atheromas and flow through the coronary arteries until a smaller vessel is occluded. The rupture of an unstable atheromatous plaque, and the subsequent exposure of substances that promote platelet activation and fibrin clot formation, further compromise myocardial perfusion. Nonatherosclerotic causes of MI include coronary artery spasm, drugs such as cocaine, congenital abnormalities, and conduction disorders.

The myocardial ischemia resulting from an occluded artery causes irreversible cell damage in the affected tissue. The central core of the infarcted area consists of necrotic cells surrounded by an area

of injury, ischemia, and inflammation (Figure 12). The injured cells may regain function, or they may die, thus extending the infarcted area. In non-lethal MI, the size and location of the infarct determine the severity of any detrimental effect on cardiac function. Left ventricular infarcts are usually the most debilitating. Cardiac dysrhythmias and cardiogenic shock are common complications of infarction.

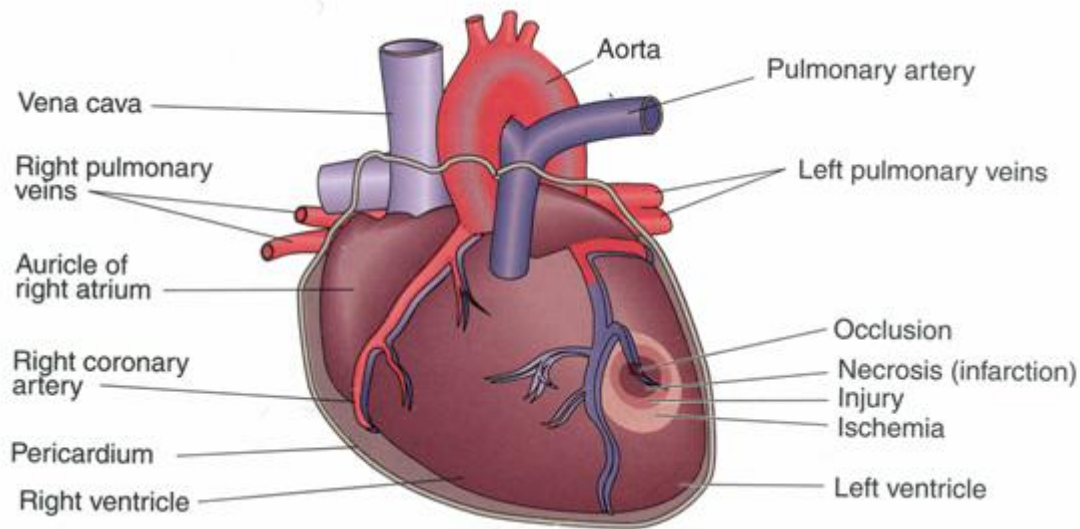


Figure 12. Myocardial infarction showing areas of damage (from Myers et. al. 2002).

Cardiac muscle is more resistant to injuries than are skeletal or smooth muscle, but shows very little regeneration after injury. As the damaged myocardium heals, the area of necrosis is gradually replaced by fibrous, non-functional granulation tissue. As a result, myocardial contractility and conductivity, as well as stroke volume and ventricular capability are decreased to a varying degree, depending on the seriousness and location of the infarct.

Inflammatory Disorders

Pericarditis

Pericarditis is an inflammation of the parietal and visceral layers of the pericardium. It may be acute or chronic and is usually secondary to another condition in the heart or surrounding structures. Cases of pericarditis are classified according to the cause or type of exudate.

Acute pericarditis may be caused by viral, bacterial or fungal infections; connective tissue diseases (lupus erythematosus, rheumatoid arthritis); renal failure; neoplastic disorders; tissue injury (MI, heart surgery, trauma); or it may be idiopathic. Inflammation of the pericardium results in increased pericardial vascularity and fibrin deposits. The rough, inflamed surfaces cause chest pain, and effusion may develop with excess fluid (serous, purulent or hemorrhagic) accumulating in the pericardial space. A small volume of fluid may have little effect on heart function, but larger amounts may result in hemodynamic compromise secondary to cardiac compression (tamponade)

Chronic pericarditis may occur in the presence of tuberculosis or radiation therapy to the mediastinum. In these cases, the inflammation causes pericardial thickening, adhesions and scar tissue between the pericardial membranes. Since the scar tissue is constrictive, the pericardium becomes tight and fibrous, thus restricting the mechanical function of the heart.

Myocarditis

Myocarditis is an inflammation of the myocardium caused by viral, bacterial, fungal or parasitic infection; serum sickness; rheumatic fever; radiation or other toxicants, such as lead. A variety of drugs, including chronic use of cocaine, may also lead to myocarditis. This disease occurs most frequently in an acute viral form, which is usually asymptomatic, mild and self-limiting. Occasionally; however, myocarditis induces necrosis of myocytes and myofibril degeneration, which can lead to right- and left-sided heart failure. If myocarditis recurs, it can result in chronic valvulitis, dysrhythmias, thromboembolism, or cardiomyopathy.

Endocarditis

Endocarditis is an infection of the endothelial lining of the heart and is most often seen in patients in whom previous structural abnormality is present. The infection may be an indolent *subacute* form or a fulminant *acute* form, depending on the virulence of the pathogen and progression of the disease. Bacteria are responsible for most episodes of endocarditis, although fungi, mycobacteria and other organisms have been implicated in some cases. The streptococcal species, which are the most common agent in subacute endocarditis, are part of the normal flora in the gastrointestinal tract and are not usually able to infect an otherwise healthy heart. The more invasive organism responsible for acute endocarditis, (*Staphylococcus aureus*), can establish infection in previously undamaged myocardial tissue.

Endocarditis is most often confined to the valves (valvular endocarditis), but does occur in the chambers (mural endocarditis). In valvular endocarditis, pathogens in the general circulation attach to, and invade, the endocardium of the heart valves and chordae tendineae, causing inflammation and the build up of vegetations on the valve cusps. Vegetations, which are fragile masses consisting of fibrin, blood cells and microbes, interfere with the opening and closing of the valves. In addition to this

interference, embolization of septic fragments may occur, causing infarction and infection in other sites such as the brain, kidney, spleen, bone, and joints. Other sequelae include progressive heart failure and dysrhythmias.

Patients with endocarditis may have a history of risk factors such as previously existing valvular disease, rheumatic heart disease, or intravenous drug use. Patients with prosthetic heart valves or long-term venous access catheters are more susceptible to developing endocarditis. This condition is uncommon in pediatric patients, but may occur in infants or children with congenital heart defects.

Valvular Disease

Valvular disease refers to any dysfunction or abnormality of any one of the heart valves and can be caused by congenital defects or can be secondary to another disease process such as endocarditis. Valvular disease may result in reduced cardiac efficiency and stroke volume, and is a common cause of heart dysfunction and mortality. Valvular dysfunction may be stenotic, incompetent or “mixed”.

Valvular Stenosis

A cardiac stenosis is any narrowing or constriction of any of the orifices or structures leading into or from the heart, or between the chambers of the heart. Stenotic valves restrict the forward flow of blood because they are unable to open fully due to narrowing, thickening, fusion or blockage. This impaired function elevates afterload, and, depending on the severity of the constriction, there may be significant hypertrophy of the atria and ventricles as they pump against the increased pressure. Eventually, the hypertrophied chambers will weaken and may result in heart failure. Dilation of the pulmonary arteries or aorta may also occur.

Aortic stenosis is a congenital narrowing of the aortic valve, or a narrowing of the aorta directly below (subaortic) or above (supra-avalvular) the aortic valve. Mitral stenosis is a constriction of the mitral valve, often resulting from rheumatic heart disease. Pulmonary stenosis is a narrowing of the pulmonary valve, which restricts the forward flow of blood from the right ventricle to the lungs.

Valvular Incompetence

Valvular incompetence, also known as valvular insufficiency or regurgitation, occurs when a valve fails to close completely, allowing blood to “regurgitate”, or leak backwards. If this happens, the heart must increase its efforts to maintain cardiac output. Eventually, the increased volume causes dilation of the cardiac chambers, which receive the reflux.

Aortic regurgitation is the leakage of blood from the aorta back through the aortic valve and into the left ventricle after ventricular contraction has been completed. Similarly, mitral regurgitation is the leakage of blood back into the left atrium. These conditions are also known as aortic insufficiency and mitral insufficiency respectively.

Heart Failure

Heart failure refers to a condition in which the heart cannot pump sufficient blood to meet the metabolic needs of the body. Heart failure may be acute, as in myocardial infarction and arrest, but it is usually chronic (congestive heart failure, CHF) occurring as a complication secondary to another condition such as chronic hypertension, valvular heart disease, pulmonary disease, and liver or kidney disease. Heart failure is most common in older patients, but does occur in young adults with a history of rheumatic fever or valvular disease, especially mitral stenosis. In infants and children, heart failure usually results from congenital heart disease but may also be caused by myocarditis and ectopic tachycardia.

Heart failure is generally classified as left- or right-sided failure, but is sometimes grouped as systolic versus diastolic, or as being secondary to another condition. Heart failure may be caused by cardiac tamponade, cardiomyopathy, or traumatic injury. It has also been known to occur in the presence of near-normal cardiac function under conditions of high demand. Whatever its etiology, heart failure ultimately leads to intravascular and interstitial volume overload, and decreased tissue perfusion.

Left-sided Heart Failure

Left ventricular failure occurs when the left ventricle fails as an effective forward pump, (systolic failure) which causes blood to back up to the pulmonary circulation. If the ventricle loses its ability to relax normally (diastolic failure) because the myocardium has become damaged or stiff, diastolic filling is compromised and cardiac output is reduced. In either case, left atrial pressure rises and is transmitted back to the pulmonary veins and capillaries. When pulmonary capillary pressure becomes too high, serum is forced across the alveolar-capillary membrane and into the alveoli, causing pulmonary edema and pleural effusion. These complications may cause tachycardia, fatigue on exertion, intolerance to cold, dyspnea, and respiratory distress. Symptoms of paroxysmal nocturnal orthopnea and nocturnal cough reflect the redistribution of excess fluid to the lungs when the patient lies down. Left ventricular failure characteristically develops in coronary artery disease, hypertension, most forms of cardiomyopathy, and with congenital heart defects (cardiomyopathy and congenital heart defects are discussed below).

Right-sided Heart Failure

Right ventricular failure usually occurs secondary to left-sided failure or tricuspid valve regurgitation. When the left ventricle fails, increased fluid pressure is transferred back through the lungs, ultimately damaging the right ventricle. Mitral stenosis, primary pulmonary hypertension, multiple pulmonary emboli, and right ventricular infarction are also causes of right-sided heart failure. When the right ventricle fails, pressure backs up in the systemic circulation, causing systemic venous congestion with dependent edema in the ankles and lower legs. Venous congestion also leads to hepatic congestion and all the complications of liver failure.

Cardiac Tamponade

The pericardial space normally contains a very small amount of fluid, which minimizes friction between the heart and surrounding structures. Cardiac tamponade occurs when the heart is compressed by blood or fluid accumulating in the pericardial space. When the heart is compressed, there is an increase in intracardiac pressure. As the amount of fluid accumulation increases, the ventricles are

prevented from expanding fully and there is a decrease in diastolic filling, which results in a reduction in stroke volume, diminished cardiac output, and hemodynamic compromise. Systemic venous return is also affected. When the right (low pressure) side of the heart is compressed, systemic venous return is impaired, causing increased pressure in the systemic veins and possible right atrial collapse.

The amount of pericardial fluid needed to impair diastolic filling depends on the rate of accumulation and on pericardial compliance. If acute, as little as 100 millimeters can result in a marked increase in pericardial pressure and can severely impede cardiac output. If chronic, several hundred milliliters can collect in the pericardial space without any significant effect on diastolic filling of the heart. This is due to adaptive stretching of the pericardium over time. The more compliant the pericardium is, the more fluid can accumulate without hemodynamic compromise.

Cardiac tamponade has a diverse etiology, but is most often associated with pericarditis caused by bacterial or viral infections. Heart surgery, thoracic dissecting aortic aneurysm, end-stage lung cancer, uremia, acute MI and various traumatic wounds to the heart or chest can all lead to cardiac tamponade. It may be considered both an injury and a symptom.

Clinical manifestations depend on whether there is an acute or chronic onset, but the classic presentation is “Beck’s Triad” of hypotension, jugular venous distension, and muffled heart tones. Patients may also experience dyspnea, palpitations, chest pain, light-headedness, or shock.

Cardiomyopathy

Cardiomyopathy is a general term describing disease of the myocardium. It is often associated with inadequate heart pumping or other heart function abnormalities. Classification of the different types of cardiomyopathies varies, but it is generally accepted that *primary cardiomyopathy* describes myocardial disease, which cannot be attributed to a specific cause. *Secondary cardiomyopathy* is due to specific disease process such as hypertension, viral infections, diseased heart valves, atherosclerosis, or congenital heart defects. Secondary cardiomyopathy may also be associated with other diseases such as inherited disorders of the muscle, or electrolyte abnormalities. When cardiomyopathy results in a significantly enlarged heart, the mitral and tricuspid valves may not be able to close properly, resulting in heart murmurs.

The most common form of this disease is dilated (congestive) cardiomyopathy. Other forms include hypertrophic, restrictive, alcoholic, or peripartum cardiomyopathy

In *dilated cardiomyopathy*, the heart cavity is enlarged and dilated. The weakened myocardium cannot pump efficiently and most patients develop congestive heart failure. Dysrhythmias may also occur. Since blood flows more slowly through an enlarged heart, mural thrombi form more easily. If a thromboembolus is dislodged from the right ventricle, pulmonary embolism may occur. If the thromboembolus is dislodged from the left ventricle, the embolus may occlude the cerebral, renal, peripheral or coronary arteries.

In *hypertrophic cardiomyopathy* there is a disproportionate growth, or “hypertrophy”, of the left ventricle or of the interventricular septum. If the septum enlarges, blood flow may become obstructed. This disease is most common in young adults and up to 70 percent of patients have a family history. Some patients may be asymptomatic.

Restrictive cardiomyopathy is a disorder affecting diastolic function; the result is inadequate filling and reduced cardiac output. Alcoholic cardiomyopathy begins about ten years after sustained, heavy

alcohol consumption. It can occur with typical signs of heart failure, with atrial fibrillation or other heart rhythm disturbances. Peripartum cardiomyopathy is a dilated type, which appears in women during the last trimester of pregnancy, or after childbirth. In children, cardiomyopathy may be asymptomatic except for a murmur.

Cardiac Conduction Disorders

The term “cardiac rhythm” describes the rate and regularity or irregularity of the heartbeat. It also indicates the site of origin of electrical impulses (sinus rhythm, ventricular rhythm, etc.). Cardiac dysrhythmias (also known as arrhythmias) are alterations in the normal heart rate or rhythm. They result from many different types and degrees of malfunction in the heart’s normal conduction of electrical impulses (caused by inflammation, scar tissue, etc., of the conduction system itself). Dysrhythmias may also result from systemic causes such as electrolyte imbalances, fever, hypoxia, stress, infection, drug toxicity, or caffeine/tobacco use.

Dysrhythmias may be benign and asymptomatic, or may produce a variety of signs and symptoms, depending on the nature and severity of the alteration of normal cardiac impulse conduction. Whatever the cause, symptomatic dysrhythmias reduce the efficiency of the cardiac cycle. A significant increase in heart rate prevents adequate filling during diastole, and a very slow rate reduces output to the tissues, including the brain and the heart itself. Irregular contractions are inefficient because they interfere with the normal filling and emptying cycle.

Cardiac dysrhythmias are generally classified by site of origin and, in general, the lower the dysrhythmia originates in the cardiac conduction system, the more lethal are the effects on cardiac function. There are many abnormal conduction patterns. Recognition and interpretation of cardiac dysrhythmias is an advanced skill which normally requires at the very minimum certification in Advanced Cardiac Life Support (ACLS) and should include further advanced education and training. As such, discussion of the identification and significance of the various cardiac dysrhythmias is beyond the scope of this ARML section. A brief discussion of dysrhythmias according to their potential pathophysiologic effects is presented below.

Benign Dysrhythmias

Benign dysrhythmias do not induce cardiac dysfunction and generally originate in the SA node or in the atria, although they may also be ventricular in origin. The transmission of an electrical impulse from the SA node is normally between 60 and 100 beats per minute and may be affected by physical conditioning, medications, disease, or conditions that delay the emergence of the impulse from the SA node. Variations in rhythms originating from the SA node or atria include differences in rate and/or regularity. Whether or not cardiac compromise occurs depends largely on the health circumstances of each patient. For example, a slow heartbeat (sinus bradycardia, 40 to 60 beats per minute) may be normal in well-conditioned adults, or during sleep. However, sinus bradycardia is also a common dysrhythmia associated with acute myocardial infarction. Another example of normally benign dysrhythmias is a premature atrial contraction. This is an extra contraction or “ectopic” beat of the atria, which usually arise from a focus of irritable atrial muscle cells outside the normal conduction

pathway. This type of dysrhythmias may be asymptomatic in some people, whereas others may feel palpitations (rapid, irregular or abnormally strong heartbeats).

Life-threatening Dysrhythmias

Life-threatening dysrhythmias are those which compromise cardiac function and are at risk of deteriorating into lethal dysrhythmias. They may develop from previously benign dysrhythmias, or they may result from cardiac ischemia, drug toxicity, congenital defects, or traumatic injury. Generally, life-threatening dysrhythmias originate at or below the AV node. This includes junctional rhythms, some heart blocks, and ventricular rhythms. When the dysrhythmia results in significant cardiac compromise, tissue perfusion becomes inadequate and the myocardium itself may be further damaged, leading to cardiogenic shock or cardiac arrest. An example of a life-threatening rhythm is a third-degree heart block, in which there is no transmission of impulses from the atria to the ventricles. The ventricles, which contract spontaneously at 20 to 40 beats per minute, generate the pumping action. In such cases, cardiac output is greatly reduced and the patient may experience syncope and cardiac arrest. Another life-threatening dysrhythmia is ventricular tachycardia; a rapid beat which originates from an ectopic focus in either ventricle and which reduces cardiac output because ventricular filling time is inadequate.

Lethal Dysrhythmias

Lethal dysrhythmias generate no cardiac output and will result in death unless timely interventions are successful in reestablishing a perfusing heart beat. Lethal dysrhythmias include pulseless ventricle tachycardia (a rapid ventricular rhythm which produces no pulse), ventricular fibrillation (a chaotic, unorganized depolarization of the ventricles in which there is no effective myocardial contraction and no pulse), or pulseless electrical activity (electrical activity is generated in the heart but there is no corresponding mechanical activity and therefore no pulse). Pulseless ventricular tachycardia and ventricular fibrillation are the two lethal dysrhythmias that are recognized by Automatic External Defibrillators (AEDs). The absence of electrical activity in the heart is known as asystole (“flat line”).

Congenital Heart Defects

Congenital heart defects arise during the first eight weeks of embryonic development and can be a major cause of death in the first year. Both genetic and environmental factors (rubella, fetal alcohol syndrome, maternal diabetes) contribute to cardiac abnormalities. Congenital heart defects are often associated with problems elsewhere in the body.

Many congenital heart defects do not produce serious hemodynamic compromise; the direction and amount of abnormal blood flow determine the effects on an individual. All significant defects do however cause a reduction in tissue perfusion. EMS personnel should note that multiple defects may be present at the same time. History is important as risk of a child having a congenital heart defect increases with a history of heart defects in parents or siblings, maternal diabetes, German measles, toxoplasmosis or HIV infection in the mother, the mother’s use of alcohol or use of certain drugs

during pregnancy, or in the presence of certain chromosomal defects such as trisomy 13 (Down's Syndrome).

Left-to-Right Shunt Defects

Ventricular Septal Defect

Ventricular septal defect is the most common form of congenital heart defect and is seen more often in males than in females. In this condition, there is a hole in the interventricular septum. The clinical significance of a ventricular septal defect is directly related to the size of the opening between the ventricles. In a small defect there tends to be minimal pulmonary vascular congestion and chamber enlargement; however, significant left-to-right blood flow will increase the volume in the pulmonary circulation, causing pulmonary hypertension and decreased cardiac output.

Atrial Septal Defect

The atrial septal defect is an opening between the right and left atria, and is a result of the failure of the foramen ovale to close after birth. As blood enters the left atria from the pulmonary vein, the pressure gradient causes a shift of volume to the right side and eventually causes right atrial and ventricular enlargement. This condition is more common in females and is usually asymptomatic, although a small percentage of those affected develop congestive heart failure. The presence of an atrial septal defect in children is normally identified when taking a history, but should be suspected when signs of CHF are present in young children. Since most children with an atrial septal defect have it surgically corrected when they are school-aged (or younger if CHF develops), signs of cardiac compromise may be caused by factors other than this type of birth defect. Obtaining a good history is very important.

Patent Ductus Arteriosus

A ductus arteriosus is a connection between the pulmonary artery and the aorta of the normal fetal heart. The ductus arteriosus, like the foramen ovale, normally closes off shortly after birth. If this structure remains patent postpartum, the condition is called patent ductus arteriosus. This allows blood from the right side of the heart to bypass the lungs before being pumped to the systemic circulation.

Obstructive Defects

Obstructive heart defects result from a complete or partial blockage of blood flow. The blockage is often caused by a structural deformity.

Coarctation of the Aorta

Coarctation of the aorta is a localized narrowing of the aorta near the aortic arch. Because of this defect, the left ventricle has more resistance to pump against and, as a result, the left ventricle may become hypertrophied and eventually fail. In addition, there is a risk for stroke due to the increased pressure exerted on the blood vessels of the brain.

Two obstructive defects involving the heart valves - aortic and pulmonary stenosis - are discussed under "Valvular Disease" above.

Cyanotic Defects

Tetralogy of Fallot

Tetralogy of Fallot is one of the most common forms of complex congenital heart defects and causes cyanosis (patients are sometimes called “blue babies”). This condition is comprised of four separate defects: ventricular septal defect; pulmonary stenosis; right ventricular hypertrophy; and dextraposition, or “overriding”, of the aorta. The restricted outflow from the right ventricle leads to high pressure in that chamber and resultant hypertrophy. The higher pressure in the right ventricle causes a right-to-left shunt through the ventricular septal defect. This flow of unoxygenated blood through the left ventricle and into the systemic circulation is promoted by the aorta, which is positioned over the interventricular septum. The end result is that the pulmonary circulation receives a small amount of unoxygenated blood and the systemic circulation receives a larger amount of mixed oxygenated and unoxygenated blood, which results in the cyanotic appearance of the patient. Open-heart surgery is needed to correct this defect.

Transposition of the Great Vessels

In this congenital defect, the aorta and pulmonary artery are “transposed” in that the aorta originates from the right ventricle and the pulmonary artery from the left ventricle. Because of this reversal, the aorta carries low-oxygen blood from the right ventricle to the body whereas the pulmonary artery carries oxygen-rich blood to the lungs. The principle pathologic effect of this condition is an increased workload on the heart and pulmonary system, and decreased perfusion of body tissues. Infants born with this defect are extremely cyanosed at birth and if they are to survive, they must also have some communication between the right and left sides of the heart, such as a septal defect. As with Tetralogy of Fallot, transposition of the great vessels must be corrected by cardiac surgery.

Shock

Shock is covered in greater detail in the “Bleeding and Shock” section (year two) of the ARML program. A review of shock with an etiology relating to blood volume and cardiac function is presented below.

Shock is a general term describing inadequate perfusion of the tissues with oxygenated blood. Types of shock are classified by cause, but the general pathophysiology is the same. One or more of the major cardiovascular factors (blood volume, effectiveness of the heart as a forward pump, and peripheral vascular resistance) is involved in the pathophysiologic cascade of shock. In the presence of actual or relative hypovolemia, blood pressure falls within the vasculature. If compensatory mechanisms are inadequate and the pumping force of the heart declines, blood flow slows and venous return is reduced. Patients in shock have compromised cardiac output and reduced blood flow through the microcirculation. This hypoperfusion leads to a reduced oxygen and nutrient supply for the cells and results in anaerobic metabolism and lactic acid build-up. The acidosis causes vasodilation, which further exacerbates inadequate perfusion by increasing the capacity of the vasculature, thereby reducing peripheral vascular resistance. This, in turn, leads to lower pressure and sluggish blood flow that furthers the cycle of cardiac decline.

Hypovolemic Shock

Hypovolemic shock is caused by a loss of blood volume and is a common cause of shock in the pre-hospital setting. Acute blood loss from blunt or penetrating trauma occurs in patients who have experienced a significant mechanism of injury, such as gunshot wounds, crush injuries, or vehicular accidents. Acute blood loss may also occur in medical conditions such as ruptured esophageal varices or a ruptured aneurysm. In some cases, blood loss may be chronic, as in colon tumors, which bleed when stool passes.

Hypovolemic shock may also be caused by a loss of plasma from the vascular compartment. This can occur with the extravasation of fluid in patients who have been severely burned. In cases of prolonged diarrhea or vomiting, fluid moves from the circulation to the gastrointestinal tract, which can lead to hypovolemic shock through dehydration. Infection processes such as peritonitis may also cause fluid shifts leading to hypovolemic shock.

Cardiogenic Shock

Cardiogenic shock is literally shock of cardiac origin and is the physiologic end point of all other causes of shock. Regardless of its etiology, cardiogenic shock is the failure of the heart as a forward pump and is most often associated with cardiac impairment caused by acute infarction, dysrhythmias, or valve problems. It may also occur with conditions such as traumatic injury, infectious diseases (for example, endocarditis), heart failure due to drug toxicity (for example, cocaine or beta-blockers), cardiac tamponade, tension pneumothorax, or pulmonary embolism.

In cardiogenic shock, the body’s compensatory mechanisms may actually worsen cardiac compromise. Increases in heart rate and force of contraction will increase myocardial oxygen demand and may thereby exacerbate myocardial ischemia. Fluid retention, coupled with the inability of the ventricles to completely fill in the presence of significant tachycardia, may lead to pulmonary congestion and

increasing hypoxia. Ischemic necrosis of the myocardium results in a reduced cardiac output and subsequent hypoperfusion. Eventually, cellular ischemia will lead to lactic acidosis, which can cause irreversible myocardial cell membrane damage and ultimately, irreversible loss of pump function.

Traumatic Injuries

Myocardial Contusion

Traumatic thoracic injury has the potential to cause significant damage to the heart and lungs. Secondary impact between the heart and the inside of the thoracic cage can cause further injury or even infarction. Damage to the myocardium can range from a minor contusion to life-threatening myocardial damage. Nonpenetrating injuries involve compression of the chest, usually as a result of significant falls; crush injuries, explosions, or deceleration injuries (especially against the steering wheel in motor vehicle accidents). Penetrating injuries, those in which the chest cavity is pierced, include stabbings and gunshot wounds. Alone or in combination, these forces can cause a myocardial contusion.

A myocardial contusion may have any of the following effects: bleeding into the pericardial space, producing cardiac tamponade; bleeding within the heart muscle; conduction disturbances; congestive heart failure; damage to heart valves; cardiac rupture; or a general weakening of the myocardium. Patients may exhibit dysrhythmias, chest pain, shock, or cardiac arrest. Alternatively, injuries to the heart can be severe even if there is no external sign of chest trauma. In non-severe cases, the myocardium may regain its function without permanent scarring.

Aortic Disruption

Traumatic aortic disruption, or rupture of the aorta, is most often due to blunt (deceleration) injury to the thorax. The rupture itself is caused by the shearing and rotational forces of high speed front- and side-impact automobile accidents and by falls from great heights. Either of these mechanisms can tear the aorta away from the heart, or rupture it somewhere along its length. The tear usually occurs at points of attachment to the heart, or because the aorta is crushed between the spinal column and the sternum. The most common site for aortic disruption is just superior to the origin of the great vessels. Other locations are the descending thoracic aorta, the ascending aorta, the aortic arch and the abdominal aorta. Blunt injury to the aorta is mostly confined to the thoracic aorta, except in the case of seat-belt injuries, which involve the abdominal aorta. Aortic disruption can also be caused by penetrating (gunshot or stabbing) injuries.

A complete tear through all layers of the aorta leads to rapid exsanguination, hypovolemic shock, and death. In partial tears, the tough outer (adventitial) layer of the aorta may remain intact, resulting in a posttraumatic “pseudoaneurysm” which is contained by the adventitia, and occasionally by the mediastinal structures. In such cases, rupture may occur at any time. Smaller leaks may not be immediately fatal, but can cause hemothorax, pericardial tamponade or hypotension. In cases of aortic trauma, prompt surgical intervention is the only option for patient survival. If the mechanism of injury is significant, EMS personnel must maintain a high index of suspicion for aortic disruption and initiate rapid transport to a facility with surgical capabilities, whenever possible.

Peripheral Vascular disruption

Peripheral vascular injuries may result from penetrating or blunt trauma to the extremities. If not recognized and treated rapidly, injuries to major arteries and veins may lead to the loss of limb or life through ischemia or hemorrhage. Many of the serious cases of peripheral vascular disruption are caused by penetrating injuries, particularly gunshot wounds, and, to a lesser extent, stab wounds. Serious burns may also cause significant peripheral vascular compromise. With significant blunt trauma (vehicular accidents, crush injuries, etc.), the risk of neurovascular disruption and extensive soft tissue damage is increased by the presence of fracture or dislocation of adjacent structures.

Death due solely to extremity vascular disruption is uncommon, except in cases of exsanguination or development of a necrotizing myofascial infection. Some of the significant sequelae to peripheral vascular injury include: compartment syndrome resulting from muscle ischemia if limb perfusion is compromised for more than six hours; and extensive associated musculoskeletal, nerve, and skin injuries.

Assessment Of Patients With Cardiovascular Complaints

When responding to a call involving a cardiac complaint, EMS personnel must perform a rapid and thorough patient assessment to determine the most appropriate course of treatment. Injuries and medical complications involving the cardiovascular system are often serious and can be life threatening. Patients experiencing a cardiac event may not present with chest pain; however, chest pain should be viewed as cardiac in origin until proven otherwise. If the patient has a cardiac illness or injury, a rapid and accurate assessment will help identify the chief complaint and permit early initiation of appropriate treatment and early determination of a load and go situation.

Assessment Of Cardiovascular Illness

In accordance with the Manitoba Health Emergency Treatment Guidelines, the initial approach to the patient should always include consideration of *scene safety* and the use of *personal protective equipment and body substance isolation* techniques where required.

Initial Assessment

As with all patients, the *initial assessment* of a cardiac patient should be completed rapidly upon initial patient contact (if no immediate life-threatening injuries and conditions requiring interventions are found during the survey). As with all patients, the initial assessment includes assessment of airway, breathing and circulation.

For cardiovascular patients, particular care should be paid to *assessment of circulation*:

- is there a pulse (note rhythm, rate, quality);
- initiate CPR if indicated;
- assess for major external bleeding (control bleeding if indicated).

The initial assessment should only be interrupted when:

- a life-threatening condition is identified and immediate life saving interventions must be initiated;
- scene conditions require that the patient be moved immediately due to danger to EMS personnel or to the patient.

Load and go should be immediately initiated if:

- the patients vital signs are abnormal or unstable;
- the patient exhibits dyspnea or respiratory distress;
- the patient continues to have chest pain unrelieved either by oxygen or nitrates.

Following the Initial assessment:

- place the patient in a low or high Fowler's position if tolerated or in a position of comfort (do not allow the patient to exert him/herself);
- reassure the patient;
- connect the patient to a cardiac monitor.

Focused Assessment

The *focused assessment* of a cardiac patient includes a thorough assessment of vital signs (including but not limited to pulse, respirations, blood pressure, LOC/GCS, skin condition, sP02 and cardiac monitoring), focus history taking and exam, paying particular attention to the cardiovascular, respiratory and central nervous systems. With any cardiac patient, be prepared to deal with respiratory and cardiac compromise, including respiratory and or cardiorespiratory arrest

Record and repeat vital signs at regular intervals (5 – 15 minutes) or when there is a change in the patient's status.

Obtain and record pertinent *current and past medical history*:
information relating to chest pain;

Onset

- What was the patient doing when the symptoms started
- Was the onset sudden or gradual
- Where they at rest or involved in some form of activity

Provoke (precipitating)Palliative (alleviating),

- Does anything the patient do make the pain better or relieve them in any way
- Does anything the patient do make the symptoms worse in any way

Quality of the pain

- Subjective description of the pain by patient
- Dull, heavy, squeezing, crushing, tearing or other descriptors (often ischemic chest pain is felt more as a discomfort than actual "pain
- Is it constant, throbbing, intermittent

Region and radiation of the pain

- Where is the pain; mid sternal, epigastric, sub sterna, sub xyphoid, left sided...
- Does the pain radiate anywhere: neck, arms, jaw.
- Referred pain can sometimes give clues to underlying medical causes

Severity of pain

- More of an objective measurement, but still individualistic
- Usually done using the 1 to 10 scale
- Can be “comparative” or “imaginative”
- Compare pain now to time of onset, post med administration and with movement and or breathing/cough
- Other pain scales, such as the Wong Baker faces pain scale when a patient is unable to verbalize a score

Time

- How long has the pain been going on
- Is it worse, better or same, different symptoms
- Has it occurred before (Hx of same or similar pains before)

Take note of any other associated signs and symptoms, such as dyspnea, diaphoresis, nausea or vomiting. If this is the result of a traumatic injury, take note of the MOI.

- attempt to obtain information relating to pertinent past medical history;
 - smoking,
 - -hypertension,
 - cholesterol status,
 - diabetes mellitus,
 - angina, myocardial infarction, coronary artery bypass surgery,
 - other risk factors for cardiovascular disease,
 - familial history of cardiovascular disease.
 - -gender
 - -age
 - -obesity, sedentary lifestyle,
 - -stress

Record any *medication(s)* taken by the patient, and note effects/reactions to medication(s).

Identify any *allergies*.

If not already on route, initiate transport:

- on scene times should be kept to a minimum (preferably under ten minutes);
- treat other conditions on route;
- notify the receiving health care facility of the patient’s status as soon as possible;
- continue to monitor and treat the patient en route.

Note: Gender and extremes of age may falsely suggest chest pain of non-cardiac etiology;

- men and women at any age can experience chest pain due to cardiac disease,
- elderly and diabetic patient often present with atypical symptoms.

Assessment Of Cardiovascular Injuries

Patients with significant *traumatic injuries* may deteriorate rapidly from blood loss and damage to internal organs. EMS personnel must consider that injuries to the thorax often involve significant damage to the cardiovascular and respiratory systems. Any such injuries must be identified in the initial assessment and treated immediately. Ongoing assessment is vital to identifying any changes in the patient's condition.

Scene assessment is very important since the cause of injury to the patient (vehicular accidents, violence, toxic spills, etc.) may still present a danger to the safety of responding emergency personnel. Other emergency response agencies (police, fire, hazardous materials teams, etc.) may need to be called to stabilize a scene before patient treatment can begin.

Once the patient can safely be approached, scene assessment should be ongoing:

- ensure continued safety of patient and EMS personnel;
- identify the mechanism(s) of injury. Certain mechanisms (laceration or chest compression, for example) may alert EMS personnel to expect cardiovascular injuries).

Personal protective equipment and body substance isolation techniques must be used.

Initial assessment

Assess for airway and breathing: Signs of respiratory compromise (agitation, restlessness, inadequate respiratory effort and ventilation, decreased level of consciousness) often reflect underlying cardiac malfunction.

Assess for signs of cardiovascular injury. In the presence of compression injuries, or blunt or penetrating trauma to the chest, assess the patient for immediate threats to life:

- major external bleeding;
- impaled object in the chest;
- signs and symptoms of progressively worsening shock;
- pericardial tamponade or myocardial contusion
- any significant chest injury which could result in injury to the heart and great vessels.

Load and Go should be initiated as soon as an immediate threat to life is identified

Continue to assess the patient for signs of cardiovascular injury. Assess for:

- symmetry of chest expansion;
- bleeding, especially pulsatile bleeding;
- open wounds;
- bruising;
- swelling;
- deformity
- hypotension;
- flat or distended neck veins;
- complaints of chest pain;
- heart sounds
- Do not remove objects impaled in the chest unless they interfere with CPR.

Focused assessment

The focused assessment should be completed en route if the patient's condition requires immediate transport. Assess for:

- dyspnea;
- tracheal deviation;
- jugular venous distention;
- distended veins in face and eyes;
- signs of internal bleeding;
- open wounds;
- bruising and swelling;
- air entry;
- heart sounds

Note proximity of wounds/bruises, etc., to major vascular structures.

On scene times should be kept to a minimum. Reassessment and treatment should continue en route.

ADDITIONAL ASSESSMENT NOTES

Additional signs of cardiovascular malfunction may be obtained during the physical exam.

A pulse deficit is a difference between the apical pulse (auscultated over the heart) and the peripheral pulse (palpated at the radial or carotid arteries). This is typically found with dysrhythmias that do not allow the ventricles to fill prior to contraction. Because of the dysrhythmia, there will not be a peripheral pulse to correspond to every apical pulse.

Pulsus paradoxus is defined as a decrease of greater than 10 mm Hg in systolic blood pressure with inspiration and is typically seen in patients with pericardial tamponade.

Pulsus alternans is a term used to describe a regular alteration in amplitude of pulse. The heart rate does not change, but the power behind each pulse changes, producing a difference in palpated pulse strength. This sign is associated with patients in congestive heart failure with left ventricular dysfunction.

Signs And Symptoms Of Cardiovascular Complaints

Cardiovascular illness or injury generally results in some degree of reduced tissue perfusion and hypoxemia, and therefore any cardiovascular compromise will exhibit certain common clinical signs. Careful assessment and history taking, as well as understanding the underlying pathophysiology of cardiovascular complaints, will help EMS personnel recognize and appropriately treat patients with cardiovascular illness or injury in the pre-hospital setting.

Cardiovascular Illness

Signs and symptoms associated with cardiac compromise vary widely, depending on the patient's age, gender, general health status, and nature of the heart disease.

Myocardial Infarction may or may not be accompanied by chest pain. If there is no pain associated with a cardiac event, it is known as a "silent" heart attack. When the presentation and or pain is not described "classically", the pain is described as atypical. Up to 30% of patients with AMI identified in longitudinal studies are clinically unrecognized. If pain is present, it may vary greatly from one individual to the next, and it may or may not be intensified by exertion. Typical chest pain can take many forms and is most typically described as dull, aching, burning, squeezing, crushing, or as chest tightness. Despite the many variables, the most common signs and symptoms of a heart attack include:

- chest pain, sometimes radiating to the neck, jaw, and arm(s);
- epigastric pain or discomfort, often described as indigestion;
- sudden onset of diaphoresis;
- dyspnea;
- tachycardia, bradycardia, or irregular heartbeat;
- abnormal blood pressure;
- fatigue;
- lightheadedness or dizziness;
- anxiety or irritability;
- feelings of impending doom;
- nausea and/or vomiting.

It should be noted that women often experience heart attacks differently than men. Women have smaller, fewer, and more flexible blood vessels and, as a result, often manifest heart disease differently. As a result, doctors, EMS providers, and women themselves have largely ignored their unusual symptoms. Some studies show that women have a 50% higher chance of having a pre-hospital myocardial infarction than do men. Sometimes women exhibit the signs and symptoms described above, but in addition, they may experience:

- unusual fatigue or shortness of breath during everyday activities, particularly those
- requiring use of the upper arms or upper body, like brushing the hair or vacuuming;
- nausea and dizziness which are often ignored because there is no chest pain with them;
- lower back pain without any history of back pain or trauma.

Elderly and diabetic patients are also at risk for atypical presentations or silent MI's.

Angina presents with many of the same signs and symptoms as myocardial infarction, but the chest pain is typically relieved with rest and nitroglycerine

Congestive heart failure is a more chronic condition, associated with a number of heart diseases such as cardiomyopathy or valvular insufficiency. The signs and symptoms usually develop over time and may include the following:

- rapid, shallow respirations;
- dyspnea (often severe, with the patient exhibiting one- or two-word dyspnea);
- wheezing or rales during breathing;
- anxiety;
- cyanosis;
- desire to sit upright;
- chest pain may or may not be present;
- pedal and lower extremity edema;
- tachycardia;
- distended neck veins;
- intolerance to cold.

Acute left ventricular failure often results in acute pulmonary edema, in addition to some of the above signs.

Whether acute or chronic, failure of the heart as a forward pump will ultimately result in cardiogenic shock. While difficult to diagnose in the field, cardiogenic shock should be suspected in the presence of unexplained hypotension, impaired mental function and signs of pulmonary vascular congestion

Heart failure in infants and young children is usually the result of congenital heart disease. Feeding difficulties are often the first sign, followed by failure to gain weight or meet developmental guidelines. Additional signs of heart failure in pediatric patients include:

- cough, tachypnea and/or noisy respirations, flared nostrils, and wheezing;
- cyanosis;
- clubbed fingers;
- intolerance to exercise or exposure to cold weather;
- toddlers and older children frequently assume a squatting position.

With right-sided heart failure, hepatomegaly and ascites may be present. It is important to conduct a thorough physical exam and to get as accurate a history as possible from the caregivers.

Other cardiovascular illnesses may present with a variety of the signs and symptoms listed above, or they may present with symptoms that are more characteristic of the specific disease conditions.

Inflammatory disorders such as pericarditis, present with varying signs, depending on the underlying problem and its effects on the patient; however, tachycardia, chest pain, dyspnea, cough, distended neck veins, pulsus paradoxus and abdominal discomfort may be present.

Aneurysms are often asymptomatic until they become very large or rupture. In thin patients, a palpable mass may be felt in the abdomen of patients with an abdominal aortic aneurysm. Dissecting aneurysms cause severe pain and loss of pulses. Aneurysm in certain sites may cause signs and symptoms not typically associated with the cardiovascular system; patients may exhibit dysphagia from a large aneurysm pressing on the esophagus, or pain if a spinal nerve is compressed by an aneurysm.

Once an aneurysm has ruptured, signs and symptoms often manifest themselves rapidly because of massive blood loss from the vascular compartment. These symptoms range from severe back pain and weakness, low blood pressure, dizziness, nausea, pale skin and diaphoresis, to unconsciousness and sudden death.

Peripheral vascular disease and deep vein thrombosis often present with pain, edema, cyanosis, localized swelling. Pain is often associated with poor tissue perfusion, leading to ischemia. Edema of the extremities is commonly caused by poor venous return leading to congestion of blood and fluids in the tissues.

Cardiovascular Injury

Traumatic injuries to the heart and vessels often cause similar symptoms to those seen in patients with cardiovascular illness. Being aware of the *mechanism of injury* and getting a good *history* are very important when caring for patients with cardiovascular injury. EMS personnel must be aware of the potential for cardiovascular injury in trauma patients who do not initially present with signs and symptoms of cardiac compromise. Injuries to the heart can be severe even if there is no external sign of chest trauma. Mechanisms of injury which should alert responders to watch for signs of cardiovascular injury include: *non-penetrating* injuries such as compression or “crush” injuries, explosions, deceleration forces (motor vehicle accidents or falls); or *penetrating injuries* such as stabbings or gunshot wounds.

Patients with non-penetrating cardiovascular injuries resulting in conditions such as myocardial contusion or cardiac tamponade, may exhibit the following signs and symptoms:

- anxiety, restlessness
- discomfort, sometimes relieved by sitting upright or leaning forward
- dyspnea
- tachypnea
- fainting, light-headedness
- chest pain
 - radiating to the neck, shoulder, back or abdomen
 - sharp, stabbing
 - worsened by deep breathing or coughing
- swelling of the abdomen or other areas
- cyanosis
- palpitations, tachycardia
- additional symptoms may include:
 - weak or absent pulse
 - drowsiness
 - dizziness
 - low blood pressure.

Patients with penetrating cardiovascular injuries, such as stabbings or gunshot wounds, which involve significant blood loss will show signs and symptoms associated with hypovolemic and cardiogenic shock, as described below.

Internal blood loss from blunt trauma (sports injuries, falls, vehicular accidents) will often cause relative hypovolemia and these patients should also be watched for signs of hypovolemic shock and cardiac failure.

Shock

Hypovolemic or cardiogenic shock can result from cardiovascular illness or injury. Some of the first signs of shock are thirst and agitation or restlessness; the EMS provider should be careful not to overlook these signs when initially approaching and assessing a patient. As shock progresses, the characteristic signs of compensatory shock begin to appear:

- anxiety, altered mental status;
- cool, moist, pale skin;
- tachycardia;
- oliguria;
- tachypnea.

As shock progresses the following signs appear:

- increasing dyspnea, progressing to deep, labored breathing;
- pallor with cyanosis to lips and fingertips;
- dilated, sluggish pupils;
- lethargy and confusion, progressing to significantly altered mental status;
- weakening, thready pulse, or absent peripheral pulses;
- low blood pressure;
- unresponsiveness.

EMS personnel should also bear in mind that there are age-related differences in the signs and symptoms of shock and that these differences underscore the need to thoroughly evaluate the signs and symptoms of shock in pediatric and geriatric patients. Children can compensate for blood loss relatively well, and then suddenly decompensate rapidly. Delayed capillary refill in children under six years of age can be considered a sign of shock. It is important to be alert to any possibility of cardiovascular compromise in children. The elderly adjust more slowly to changes in blood pressure; certain cardiovascular diseases play a role in this slower adjustment. Certain medications (for example, beta-blockers) can blunt the body's natural response to increase the heart rate in the face of hypovolemia. Elderly patients who sustain traumatic injury should be assessed very carefully for other signs and symptoms of shock as tachycardia may never develop.

Peripheral Vascular Injury

Blunt or penetrating trauma to the extremities may cause injuries to the blood vessels in the affected limb(s). Examples of such injuries are: musculoskeletal injuries with vascular compromise; lacerations due to violence or suicide attempts; and crush injuries of the extremities. Signs and symptoms of peripheral vascular injuries include:

- active or pulsatile hemorrhage;
- hematoma;
- signs of limb ischemia (pallor, pulse deficit, paralysis, pain);
- diminished or absent pulses distal to the injury;
- hypotension or shock.

Diagnostics

Vital Signs

Recording vital signs is an integral part of patient assessment, but relating them to underlying cardiovascular pathophysiology can give an indication of the underlying causes of a patient's current condition.

Respiration

Some degree of *respiratory distress* usually develops with cardiac compromise. Respiratory difficulty, considered in concert with other signs and symptoms, may indicate decreased cardiac function. Consider cardiovascular involvement in patients exhibiting abnormal respiratory signs.

In some of the chronic cardiac illnesses, such as congestive heart failure or congenital heart defects, fatigue develops and the patient often exhibits increasing shortness of breath. The pulmonary backup effects of cardiac congestion include dyspnea and orthopnea (difficulty in breathing when lying down as increased fluid accumulates in the lungs in the recumbent position). Cough is commonly associated with this condition, and paroxysmal nocturnal dyspnea may be present. Acute pulmonary edema indicates advanced heart failure. Getting a thorough respiratory history, including symptoms such as night-time awakenings (because of breathing difficulties or coughing), or hemoptysis (producing a frothy, blood-stained sputum) is important diagnostic information for assessing and treating patients with cardiac complaints. The more acute causes of cardiovascular compromise, such as myocardial infarction or hypovolemic shock, are often associated with respiratory signs (rapid, shallow breathing, for example) which have a relatively sudden onset.

Pulse

Early assessment of the *rate, rhythm, and quality* of a patient's pulse is important in assessing cardiovascular function, as well as for ongoing assessment of a patient's condition in that it establishes a baseline for comparison with later readings. EMS providers must be aware of the hemodynamic consequences of various cardiovascular illnesses and injuries and be able to relate these to observed signs and symptoms (for example, that pulse will increase with hypovolemia, or be slow in a patient with bradycardia). Additional diagnostic benefit can be gained by knowing the relationships between pulse measurements and other hemodynamic signs. For example,

- the presence or absence of pulses taken at different sites can give information about blood pressure (radial pulse is only felt if the systolic blood pressure is above 80 mm Hg, femoral is palpable at 70 mm Hg or higher, and carotid is palpable at 60 mm Hg or higher).
- Another example is that the presence or absence of a palpable pulse can be correlated with ECG readings to help identify cardiac rhythms.

It should also be noted that some variations in pulse are normal in some patients and do not indicate cardiovascular problems. For example, the normal, regular pulse may vary with inspiration and expiration (sinus dysrhythmia), especially in children.

Blood Pressure

Blood pressure is an important diagnostic sign of potential cardiovascular illness or injury. Compensatory mechanisms may keep blood pressure relatively normal in early stages of hemodynamic compromise. In patients without significant underlying pathological conditions, such as heart disease or traumatic injury, systolic blood pressure may be maintained until cardiac output decreases significantly, or until one quarter of the blood volume is lost. EMS personnel should obtain a blood pressure reading early in patient assessment. Comparing the initial reading to later ones can help monitor changes in patient condition.

It should also be considered whether normal pulse and blood pressure measurements taken while the person is supine differ (by more than 20 mm Hg) from those taken while seated or standing. This is known as an orthostatic difference in these vital signs and may indicate that a patient is hemodynamically compromised. Thorough patient assessment is also important in establishing what are the normal values for each individual. A low blood pressure may be normal for some patients and does not necessarily indicate a cardiovascular problem. Blood pressure must be considered in light of other diagnostic means, as well as the overall patient condition, when making treatment decisions.

Skin Condition

Skin condition can be a good indicator of some cardiovascular conditions, especially shock and hypoxemia. Initial and ongoing assessment of a patient's skin condition can reflect cardiac output and tissue perfusion, or changes in these over time. For example, skin is cool and moist in poorly perfused areas because of the compensatory mechanism of peripheral vasoconstriction. Cyanosis indicates generalized or local hypoxemia, depending on its location. Any worsening of these signs can alert EMS providers to changes in a patient's hemodynamic status, possibly requiring initiation of a load and go decision. Alternately, a reversal of these symptoms with treatment (oxygen, medication, positioning, etc.), reflects an improvement in a patient's condition.

Skin condition may also show changes in the presence of localized vascular compromise. In patients with peripheral vascular disease, the changes in the appearance of the skin on the feet and legs (marked pallor or cyanosis when the legs are elevated and redness when they are dangling/dependent) may be diagnostic. Such changes should be considered in conjunction with other signs and symptoms such as pain and swelling, and relevant history, such as the presence bed rest, leg trauma or surgery.

Pulse Oximetry

Pulse oximetry is a widely used and simple, noninvasive monitoring procedure for the evaluation of adequate perfusion. A pulse oximeter measures the amount of oxygen bound to hemoglobin by transmitting red and infrared light through the arterial blood in the capillary beds. The light is transmitted via a probe with two light-emitting sensors. Infrared light is absorbed by hemoglobin bound with oxygen (oxyhemoglobin) and red light is absorbed by hemoglobin that is bound to something other than oxygen. The differences in these two measurements indicate the percentage of available hemoglobin sites that are carrying oxygen molecules.

The primary use of pulse oximetry is to assess respiratory function, but when properly used and interpreted, pulse oximetry can help EMS personnel evaluate a patient's circulatory status. Heart rate shown on the oximeter should correlate with the palpable pulse. Certain conditions (hypovolemia, hypothermia, ambient light conditions, etc.) may interfere with accurate pulse oximetry. A more detailed review of pulse oximetry may be found in the "Respiratory Emergencies" section of the ARML program.

Electrocardiogram

An electrocardiogram (ECG) is a recording of the electrical activity of the myocardium and shows transmission of electrical impulses through the cardiac conduction system. The information gained from an ECG is useful in the initial diagnosis and monitoring of many cardiovascular conditions. ECG monitoring augments patient assessment and care by enabling EMS personnel to obtain a baseline recording of the electrical activity of the heart, interpret cardiac dysrhythmias, and evaluate the possible effects of cardiovascular illness or injury on cardiac function. The ECG can provide information about the orientation of the heart in the thoracic cavity, conduction disturbances, presence of ischemic damage, and cardiac response to the effects of medications. ECG monitoring may also be used to evaluate pacemaker function and to obtain a baseline recording before, during, or after a specific procedure.

An ECG does not show the adequacy of the mechanical (pumping) ability of the myocardium. The effectiveness of the heart's mechanical activity is evaluated by assessment of the patient's pulse and blood pressure. This information should be considered in conjunction with ECG recordings when assessing and treating patients with cardiovascular complaints.

Electrical activity of the heart can be detected through electrodes placed at specific locations on the chest wall and/or extremities. Electrodes used in pre-hospital monitoring are typically disposable disks consisting of an adhesive ring with a conductive substance in the center. Each pair of electrodes (one positive and one negative) is referred to as a "lead". A lead detects the average current flow in a portion of the heart at a specific time and records it on ECG graph paper (Figure 13). ECG paper is made of various sized boxes measured in millimeters. The horizontal axis records time (25 mm/sec), which is stated in seconds and the vertical axis records voltage (amplitude) of the ECG deflections or "waveforms".

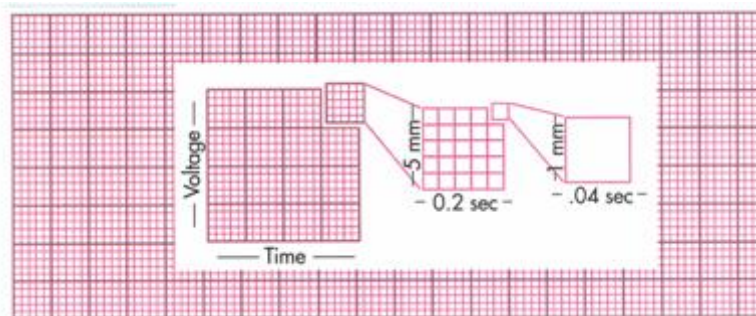


Figure 13 . Electrocardiogram paper. The horizontal axis represents time and the vertical axis represents amplitude of the wave (voltage) (from Aehlert, 2002).

The ECG waveforms correspond to electrical events in the cardiac cycle. Electrical impulses originating in the SA node produce various waves on the ECG as they spread throughout the heart. The positive and negative electrical impulses are shown as upward or downward deflections on a visual display, or on the ECG graph paper (Figure 14). As an impulse leaves the SA node and causes a wave of depolarization of the atria, it is recorded as a small wave known as the P wave. The PR interval corresponds to the duration of the conduction through the atria, the delay at the AV node, and the passage of the impulse through the bundle of His and the bundle branches. The next event is ventricular depolarization, which as recorded on the ECG as the QRS complex. The T wave is the final waveform in a normal cardiac electric cycle. It represents ventricular repolarization and the heart returning to its regular resting state. The QT interval represents both ventricular depolarization and repolarization. There is no visible wave for atrial repolarization as it is hidden in the QRS complex. The absence of electrical impulse at any time causes no reaction on the ECG monitor and is recorded as a straight line known as the isoelectric line.

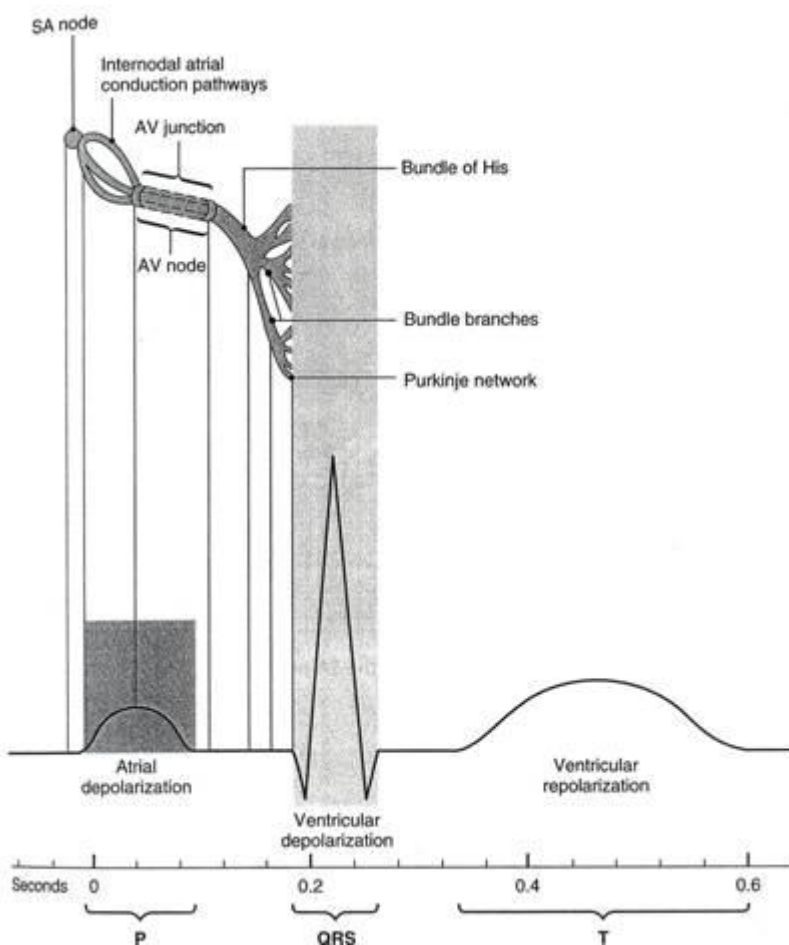


Figure 14. Cardiac conduction shown as waveforms on an ECG (from Bledsoe et. al., 2003).

Recognizing and interpreting ECG recordings is an advanced skill usually acquired in an Advanced Cardiac Life Support course, and as such, is beyond the scope of this package. A brief overview of the use of three- and twelve-lead electrocardiograms in pre-hospital care is presented below.

Three Lead Electrocardiogram

Three-lead ECGs are traditionally used most commonly in the pre-hospital setting, although twelve-lead ECGs are becoming more available in the field. Three-lead ECGs give one or two views of the heart from the frontal plane and can be used to identify a number of cardiac dysrhythmias, but are of limited diagnostic ability in terms of location, extent, and age of myocardial injury.

The bipolar Lead II (with a positive electrode, a negative electrode, and a ground) views the inferior surface of the left ventricle and is most frequently used in three-lead monitoring. Bipolar leads detect the difference in electrical potential at one site relative to the electrical potential at another site. The positioning of positive and negative electrodes in Lead II most closely resembles the pathway of current flow in normal atrial and ventricular depolarization (Figure 15). Monitoring through Lead II gives the rate and regularity of the heartbeat (Figure 16), as well as allowing EMS personnel to see the origin of certain dysrhythmias. This information, when considered in relation to clinical signs and symptoms, overall assessment, and patient history, can be useful in determining possible treatments in the field.

Figure 15. Placement of positive and negative electrodes in Lead II (from Aehlert, 2002).

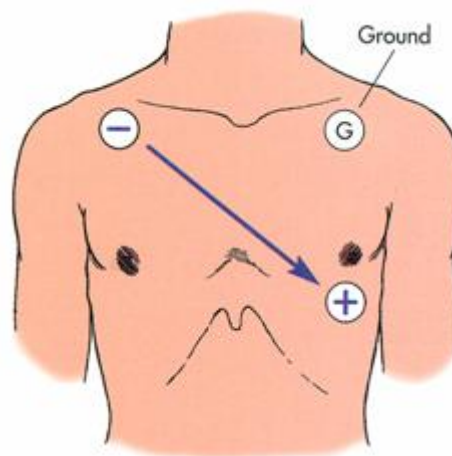


Figure 16. Three-lead ECG (Lead II) showing a normal sinus rhythm (from Aehlert, 2002).

Twelve Lead Electrocardiogram

A standard twelve-lead ECG provides views of the heart in both frontal and horizontal planes and views the surfaces of the left ventricle from twelve different angles. Multiple views of the heart can provide important information such as: identification of ST segment and T wave changes associated with myocardial ischemia, injury and infarction; recognition of bundle branch blocks and QRS complex abnormalities; and identification of ECG changes associated with certain medications and electrolyte imbalances.

A twelve-lead ECG is obtained by using electrodes placed on the body in specific locations (Figure 17). In addition to the standard bipolar leads used in a three-lead ECG, there are augmented (unipolar leads which record the difference in electrical potential relative to zero rather than to the electrical potential of another site) and precordial (bipolar chest leads) leads. A twelve-lead monitor simultaneously records each lead and provides a read-out in a conventional four-column format (Figure 18). Each column consists of three rows: the standard limb leads are in the first column, the augmented leads in the second column, and the precordial leads in the third and fourth columns.

Figure 17. Electrode placement for a twelve-lead ECG. Electrodes are applied at specific locations on the chest wall and on the extremities to view the heart's electrical activity from different angles and planes (from Aehlert, 2002).

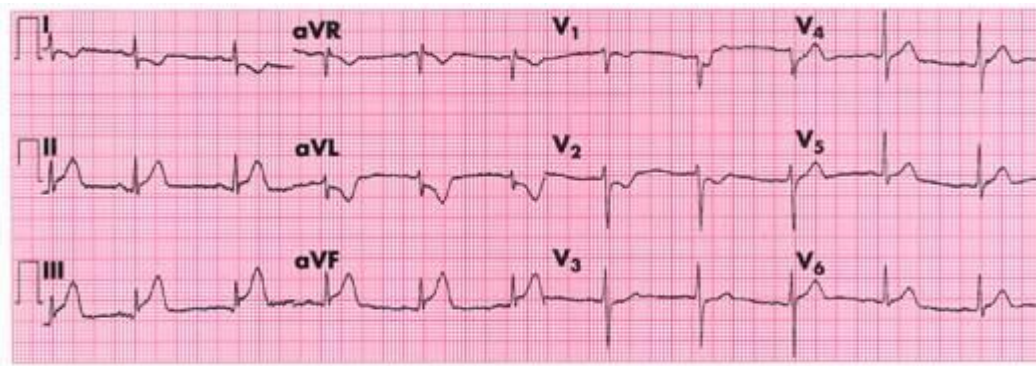


Figure 18. Example of a twelve-lead ECG; note four column format (from Aehlert, 2002).

Therapeutics

Defibrillation

Defibrillation is the therapeutic process of passing an electric current through a fibrillating heart with the aim of terminating lethal cardiac dysrhythmias. The most frequent initial rhythm in sudden cardiac arrest is ventricular fibrillation, and the most effective treatment is defibrillation. The defibrillator itself is an electrical capacitor that stores energy for delivery to the patient at a desired time. Defibrillation therapy completely depolarizes the myocardium, producing momentary asystole. This stops the chaotic electrical discharge from ectopic foci, and allows the SA node an opportunity to restore an organized cardiac rhythm.

Early defibrillation is a critical step in improving survival for victims of cardiac arrest. From the onset of cardiac arrest, the chance for survival diminishes by approximately 10% for every minute delay in defibrillation. Therefore, the less time a patient remains in cardiac arrest, the greater the chance that the natural pacemaker function of the SA node can be restored. In addition, timely defibrillation may prevent a viable rhythm from deteriorating to a more lethal rhythm (for example, coarse ventricular fibrillation to fine ventricular fibrillation or asystole). The importance of early defibrillation is emphasized by its inclusion in the “Chain of Survival” (Figure 19), which was developed by the American Heart Association in 1992 to visually present the series of steps necessary to increase the chance of survival following cardiac arrest. The Heart and Stroke Foundation of Canada expanded the Chain of Survival to include components required to improve the chance of survival for patients with cardiac disease. Without early defibrillation, other components of the “chain” are less likely to positively influence survival of cardiac patients.



Figure 19. The Chain of Survival (The Heart and Stroke Foundation of Canada).

Manitoba Health maintains that early defibrillation (within five minutes of collapse) is a high-priority goal of EMS care. While this may not always be possible, especially in many rural areas of Manitoba, every attempt should be made by EMS personnel to initiate timely use of defibrillation therapy for patients in cardiac arrest.

In addition to early initiation, the success of defibrillation depends on: condition of the myocardium; heart size and body weight; paddle size and placement; paddle-skin interface; paddle contact pressure; and number of shocks already delivered to the patient.

In 2005, the AHA and ECC recognized the importance of early, aggressive and uninterrupted CPR in their guidelines, more so than ever before. Prior to these guidelines, most of the efforts of a healthcare

provider were to provide defibrillation as early as possible in most types of arrest situations. We now recognize that, especially in states of unwitnessed arrest, good, basic CPR for at least 5 cycles (2 minutes of CPR) is of the utmost importance. It is also imperative to try and limit times where CPR is interrupted. Rhythm analysis should be quick, medications, if indicated should be drawn up and ready to administer before CPR is interrupted, if interrupted at all, pulse checks should be brief. Airway management, such as intubation, double lumen airways, patient reassessments should be performed in a way that minimizes interruption of chest compressions etc. If at all possible, during patient movements to stretchers or the ambulance etc...CPR should be maintained at all times if possible. The goal is to “Push Hard and Push Fast” and dramatically reduce the times were CPR is not being done. CPR should be continuous if at all possible.

Immediate defibrillation is now only indicated if an arrest is witnessed by a healthcare provider trained in defibrillation and the defibrillator is connected. In all other circumstances, at least 5 cycles of CPR should be initiated prior to rhythm analysis. Furthermore, pulse checks are no longer done immediately after delivery of a shock; 5 cycles of CPR should be done prior to pulse checks.

Automated External Defibrillation

An Automatic External Defibrillator (AED) recognizes the presence of lethal dysrhythmias (pulseless ventricular tachycardia and ventricular fibrillation) (Figure 20), and delivers an electric shock through disposable, adhesive electrode pads attached to the patient’s chest.

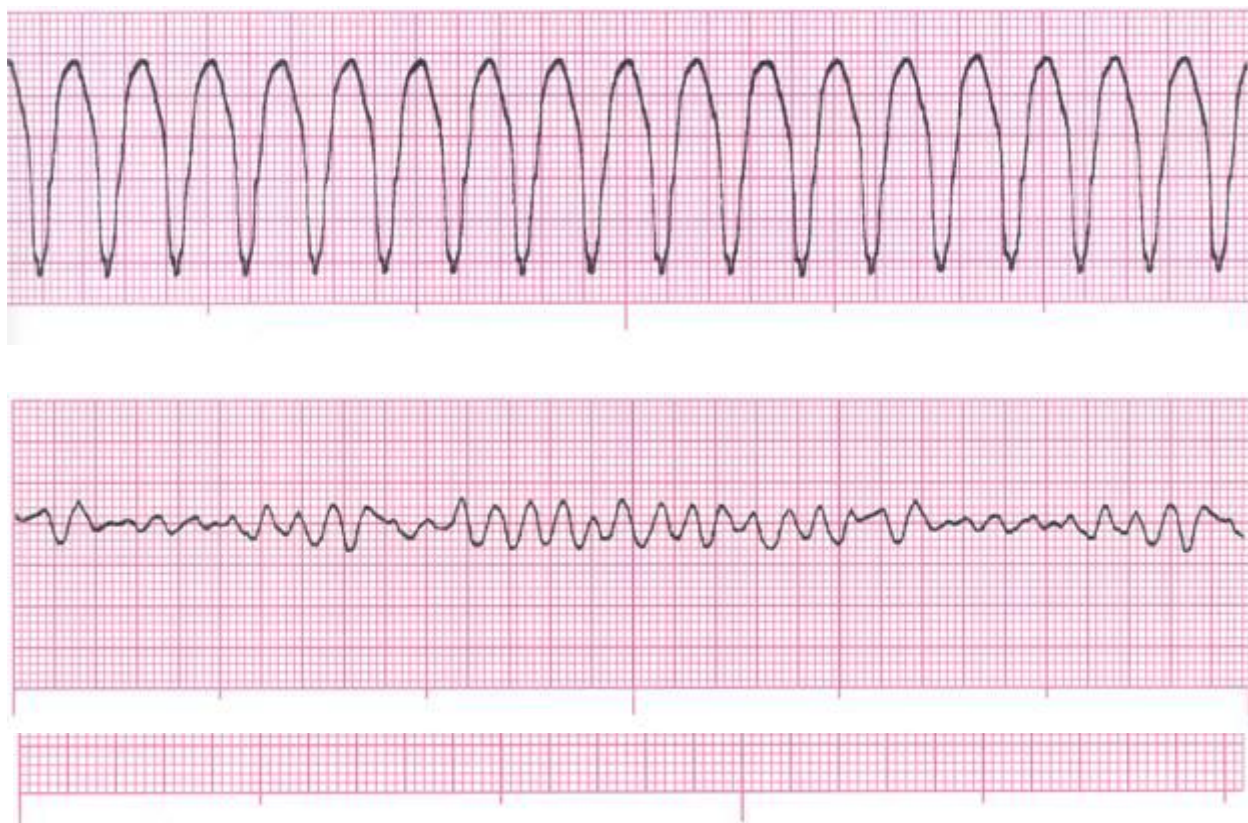


Figure 20. Lethal dysrhythmias recognized by AEDs: top – ventricular tachycardia; middle – coarse ventricular fibrillation; and bottom – fine ventricular fibrillation (from Aehlert, 2002).

There are two types of automated defibrillators; fully automated and semi-automated. A fully automated defibrillator performs all functions by computer; it analyses rhythms, selects an energy level, charges the machine, and shocks the patient. The operator applies the adhesive pads and turns on the machine, then makes certain that no one is in contact with the patient. A semi-automatic defibrillator requires the operator to manually activate the analysis and defibrillating function when advised to do so by the machine. In this case, the operator makes the final decision to deliver a shock to the patient.

Indications for defibrillation:

- minimum age of patient 1 year old
for ages 1-8 years, pediatric pads are preferred but not essential
- cardiac arrest, characterized by no pulse, not breathing, and has no visible signs of circulation

Contraindications for defibrillation:

- patient with massive trauma or penetrating injury requiring surgical intervention. For such patients, analysis and defibrillation are permitted until the first “no shock advised” is obtained, at which time an immediate load and go must be initiated.

Procedure:

- scene survey, including hazards, mechanism of injury, safety, infection control routine precautions
- establish unresponsiveness and absence of respiratory effort and pulse
- assess that the patient meets the criteria for application of the AED
- initiate at least 5 cycles of CPR
- direct colleague to place the AED next to the victim;
- power on the AED
- expose the chest and prepare chest for pad application
- attach pads and defibrillator cable
- if age 1-8: attach pediatric pads if available
- analyze the cardiac rhythm using the AED
- stay clear of the victim during an analysis
- shock if the AED advises to do so
- stay clear of the victim during delivery of shock(s)
- continue with 5 cycles of CPR (about 2 minutes) before doing a pulse check

Return of Circulation Post Shock Delivery

If the victim regains a pulse after defibrillation, standard life support measures should be initiated;

- following confirmation of a pulse, ventilation should be assessed and assisted if required
- a minimum of twelve breaths per minute by bag-valve-mask
- do not turn off AED, as some AED's will recognize a change in rhythm and alert the operator
- once the victim has stabilized, vital sign assessment and level of consciousness should be monitored en-route
- high-flow oxygen should be administered by appropriate device if victim is spontaneously breathing
- the AED should be used to reanalyze the cardiac rhythm if the pulse is lost at any time
- if a palpable pulse is present, proceed with appropriate airway management techniques and continually monitor patient's pulse, not the patient's cardiac rhythm
- if at any time the patient becomes pulseless: immediately reanalyze patient to determine if defibrillation is indicated
- the decision when to initiate transport is based on factors such as distance to hospital, level of training and certification of EMS crew, and other regional factors. Transport may be initiated prior to completion of 9 rhythm analyses using the AED

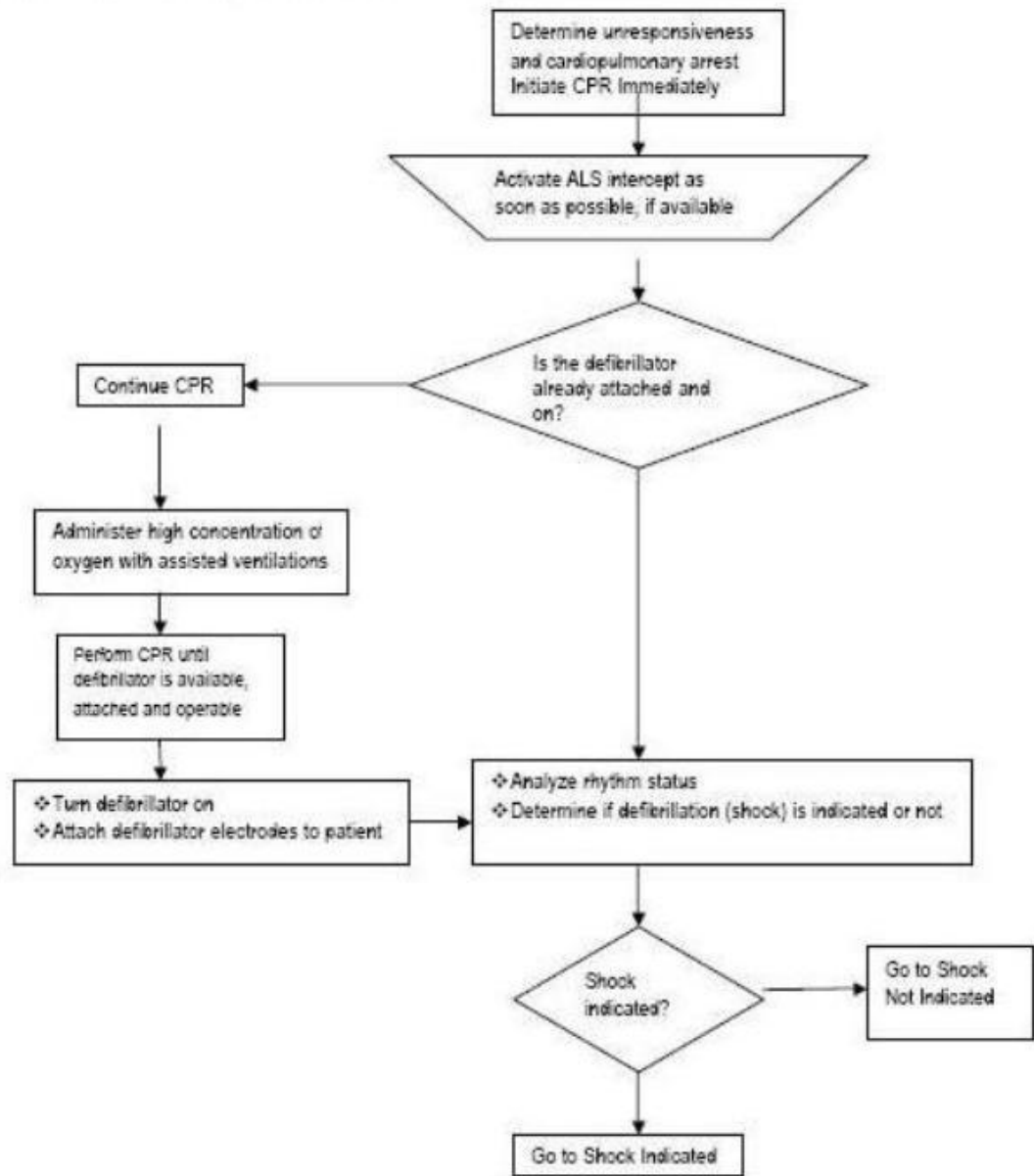
Special Situations

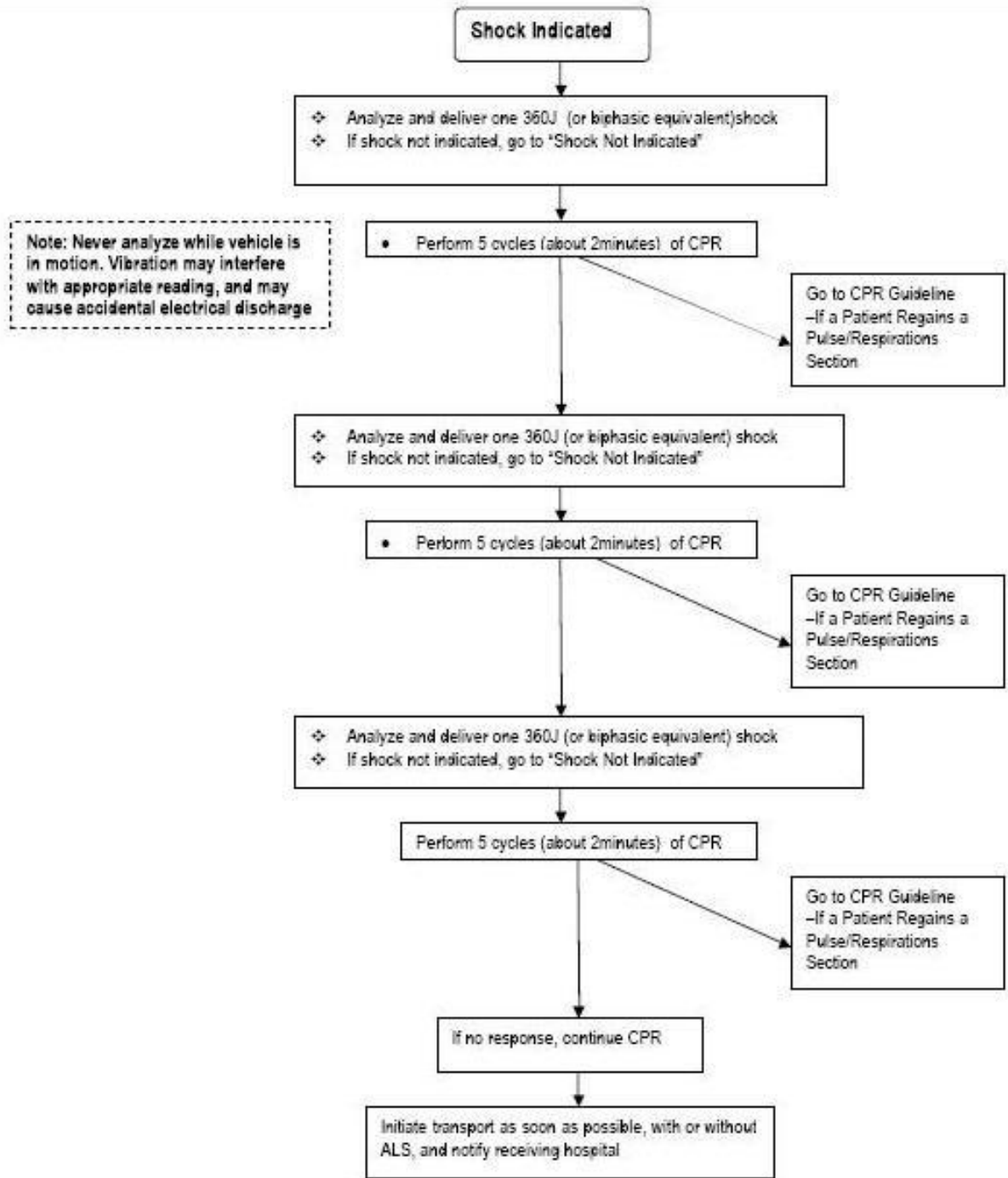
- agonal respirations
 - personnel may have to wait for agonal respirations to cease before being able to analyze the cardiac rhythm using the AED
 - during this time, the personnel must perform CPR until agonal respirations stop
- pacemakers / implantable defibrillators
 - for patients with known Automated Internal Cardiac Defibrillators (AICD), attach the AED taking care to avoid defibrillator pads adjacent to the area where the AICD is located
- hypothermia
 - an initial “analyze – shock” if indicated should be attempted
- trauma
 - if the victim is in cardiac arrest due to blunt trauma, an initial “analyze - shock” may be attempted, if indicated
- health care directives; determination of death guidelines; response to an expected death at home Guideline

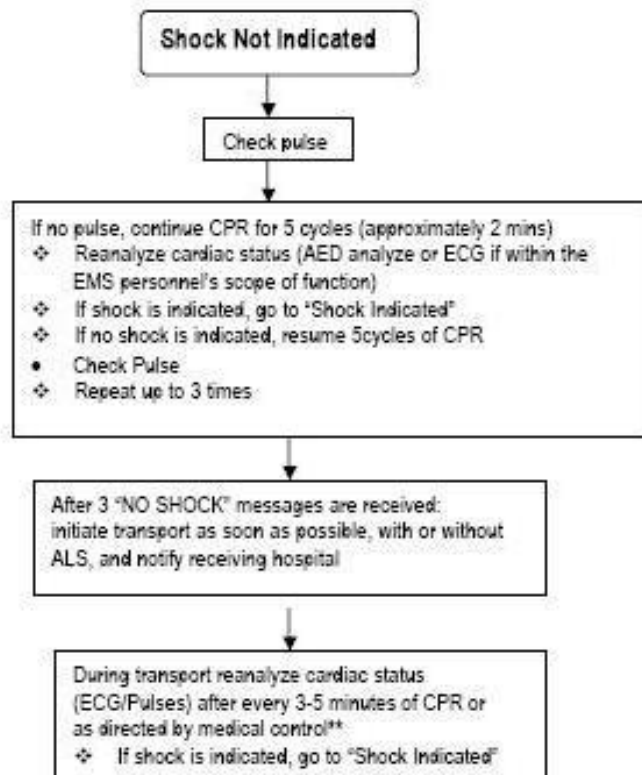
Environments Requiring Caution

- victims with medication patches
 - remove any medication patches on their chest
- moving vehicles
 - if the AED states during transport that you should check the patient, immediately stop the vehicle, and analyze per algorithm
 - never analyze while vehicle is in motion. Vibration may interfere with appropriate reading, and may cause accidental electrical discharge
- wet surfaces
 - move victim to a dry surface (eg. non-metallic spine board)
- metal platforms and other conductive surfaces
 - position defibrillator pads to avoid contact with metal objects (e.g. body piercing or jewelry)
- electrical interference
 - remove/disconnect source

Table 1: Algorithm for AED Use







Manual Defibrillation

In manual defibrillation, the operator must recognize the presence of a lethal cardiac dysrhythmias and then select the desired electrical charge based on presenting rhythms, patient's age and condition, or other pertinent factors. Relevant local protocols should be followed to determine when manual defibrillation is appropriate, the strength of initial and subsequent charges, number of shocks to be delivered, etc.

Cardioversion

Cardioversion is the act of converting a dysrhythmia to a normal rhythm via the delivery of an electric shock that has been timed to the heart's electrical activity. It differs from defibrillation in that the electrical discharge is synchronized with the R wave of the underlying rhythm. This is to avoid the vulnerable relative refractory period, thereby decreasing the chance of causing a lethal, non-perfusing rhythm such as ventricular fibrillation. It is designed to interrupt an ectopic pacemaker activity with a view to allowing the SA node to reestablish a normal, perfusing heartbeat. Because of the efficiency in delivering the shock, synchronized cardioversion may require less energy to terminate dysrhythmias, and thus reduce the chance of developing new, problematic dysrhythmias. The particular energy requirements are dependent on the type of dysrhythmias being treated. Ventricular dysrhythmias require more energy than do atrial ones.

Although cardioversion is usually performed in the hospital, appropriately qualified EMS providers can perform it in the pre-hospital setting. The primary indications for cardioversion are: rapid atrial fibrillation; atrial flutter; supraventricular tachycardia; and perfusing ventricular tachycardia. The conscious patient who is hemodynamically compromised should be cardioverted after receiving an appropriate amount of sedation. Cardioversion has limited success in patients with long-standing atrial fibrillation or with some other types of heart problems, such as hypertrophic cardiomyopathy.

Temporary Pacing

A pacemaker is an artificial pulse generator that delivers an electrical current to the myocardium to stimulate depolarization. Pacemaker systems are usually named according to where the electrodes are located and the route the electrical current takes to the heart. A pacemaker system consists of a pulse generator and pacing leads. The pulse generator contains electronic circuitry to analyze the patient's intrinsic rhythm, and timing circuitry to pace the stimulus output. The pacing lead is an insulated wire that carries the electrical impulse to the patient and back to the pacemaker. An exposed portion of the lead is the electrode, which is placed in contact with the patient. Temporary pacing can be accomplished through transcutaneous, transvenous, or epicardial means.

Transcutaneous pacing

Transcutaneous pacing (TCP) delivers pacing impulses to the heart via electrodes placed on the patient's thorax. This procedure is also called "temporary external" or "non-invasive" pacing. TCP involves attaching two large bipolar pacing electrodes to the skin of the patient's chest wall. As with defibrillation, the electrical current must be strong enough to overcome the resistance of the chest wall.

TCP is recommended as the initial pacing method in emergency cardiac care because it is effective, quick, safe, and is the least invasive pacing technique currently available. Indications for emergent TCP include severe symptomatic or hemodynamically unstable bradycardia which is unresponsive to atropine or when atropine is not immediately available. TCP may be used until transvenous pacing can be initiated or until the cause of the bradydysrhythmias can be reversed (as in drug overdoses, hypoxia, or hyperkalemia). Studies have shown that the patients most likely to benefit from TCP are those who have a palpable pulse, but with a heart rate too low to maintain adequate perfusion. These patients are often conscious, and may be alert, but have hypotension, chest pain, or pulmonary edema as a result of extreme bradycardia. In cases of witnessed asystolic arrest, TCP may be effective if implemented immediately.

Complications of TCP include pain from electrical stimulation of the skin and muscles, failure to recognize that the pacemaker is not capturing, and failure to recognize the presence of underlying, treatable ventricular fibrillation. Tissue damage, including third-degree burns, has been reported in pediatric patients. Prolonged pacing has been associated with pacing threshold changes, leading to capture failure.

Transvenous pacing

Transvenous pacing stimulates the endocardium of the right atrium or ventricle (or both) via a bipolar electrode introduced into a central vein. It can be used in cases of symptomatic bradycardia, bradycardia with escape rhythms unresponsive to drug therapy, overdrive pacing of tachycardia refractory to pharmacological therapy or electrical countershock, and bradysystolic cardiac arrest.

Complications of transvenous pacing include bleeding, infection, pneumothorax, cardiac dysrhythmias, lead displacement, fracture of the pacing lead, hematoma at the insertion site, or perforation (of the right ventricle, inferior vena cava, pulmonary artery, or coronary arteries) because of improper placement of the pacing lead.

Intra-Aortic Balloon Pumps

An intra-aortic balloon pump (IABP) is a device that increases blood flow to the myocardium and decreases the heart's workload through a process called counterpulsation. The primary goal of IABP treatment is to increase myocardial oxygen supply and decrease oxygen demand. This therapy can reduce the heart's workload by up to 20 percent. The IABP is used for a short periods of time, such as:

- before, during, or after open-heart surgery;
- acute angina attacks;
- emergency situations such as myocardial infarctions or cardiogenic shock;
- supporting cardiac function in patients waiting for a heart transplant
- support and stabilization of high-risk patients undergoing angioplasty and angiography.

A catheter or "balloon" is inserted into the femoral artery and, using an ultrasonic detecting device, is slid into position below the aortic arch. It is then connected to a pump that uses helium (which has the fastest ability to diffuse) or carbon dioxide (which has fewer consequences if the balloon bursts) to inflate and deflate the balloon. The inflation and deflation cycles are timed electronically from an ECG signal or from an arterial pressure wave. Because the balloon is placed in the aorta, patients with

aorta-related conditions, such as aortic dissection or aortic regurgitation, are not eligible for this therapy.

The counterpulsation process of IABPs works with the normal pumping action of the heart. As the left ventricle contracts, it must pump blood through the aorta against the peripheral vascular resistance. If the balloon is suddenly deflated at this time, a “vacuum” is created, thereby assisting ventricular emptying and systemic perfusion. During diastole, the balloon inflates, helping to push blood forward through the systemic circulation and into the coronary arteries.

Complications, which may have ramifications during EMS transport of patients with IABPs, include:

- damage to the aorta;
- hemorrhaging at the insertion site;
- infection;
- limb ischemia or visceral ischemia;
- tearing or bursting of the balloon, releasing gas into the bloodstream.

Pharmacology

Acetylsalicylic Acid (Aspirin)

Class: Analgesic, antipyretic, anti-inflammatory, platelet aggregation inhibitor.

Mechanism of Action:

- interferes with prostaglandin synthesis. Reduction of tissue prostaglandins may be responsible for the analgesic and anti-inflammatory effects.
- acts on hypothalamic heat-regulating center to cause peripheral vasodilation
- inhibits thromboxane A₂ synthesis, preventing clot formation.

Indications:

- chest pain consistent with acute myocardial infarction
- mild to moderate pain, fever and inflammation, osteoarthritis
- prevention of thrombosis, and thrombotic complications in patients with atrial fibrillation and in procedures such as angioplasty.

Contraindications:

- hypersensitivity
- active peptic ulcer, active gastrointestinal bleed

Precautions:

- pediatric patients, esp. patients with chickenpox, influenza, or flu-like illnesses (strong association with development of Reyes syndrome).
- decreased renal function, bleeding tendencies, anemia, vitamin K deficiency, severe hepatic disease.

Adverse Effects:

- gastric ulceration, hemorrhage, dyspepsia, heartburn, epigastric distress, nausea, vomiting, diarrhea, hepatotoxicity, thrombocytopenia.
- tinnitus, hearing loss

Interactions:

- concomitant use with anticoagulants increases the risk of bleeding
- decreases effect of antihypertensive drugs, NSAIDS, increases hypoglycemic effects of oral antidiabetic agents

Special Considerations:

- geriatric, frail or debilitated patients are more susceptible to adverse effects of ASA
- consult physician for use during last trimester of pregnancy

Dosage and Administration:

- 160 mg po (chewed)

Alteplase (Activase – rt-PA)

Class: Fibrinolytic

Mechanism of Action:

binds to fibrin in thrombi and converts plasminogen into plasmin
initiates local fibrinolysis and limited systemic proteolysis

Indications:

lysis of occlusive coronary artery thrombi in evolving transmural AMI
to improve ventricular function following AMI
to decrease incidence of congestive heart failure
acute ischemic stroke within three hours of onset
lysis of pulmonary thromboemboli

Contraindications:

hypersensitivity
active internal bleeding, history of stroke, patients receiving other IV thrombolytic agents
recent intraspinal to intracranial surgery, aneurysm, uncontrolled hypertension
recent traumatic CPR, recent severe trauma or surgery.

Precautions:

IM injection and non-essential handling of the should be avoided during and immediately following treatment
minimize arterial or venous puncture

Adverse Effects:

hypersensitivity
peripheral or systemic bleeding; intracranial hemorrhage
reperfusion dysrhythmias following AMI

Interactions:

additional bleeding risk in patients already taking anticoagulants
specific interaction with other drugs has not been studied

Special Considerations:

should be administered as soon as possible after onset of AMI
pre-hospital administration may only be useful with relatively long transportation times
risks of therapy may be increased in the elderly

Dosage and Administration:

administered as an IV infusion as per physician's orders.

Amiodarone Hydrochloride (Cordarone)

Class: Antidysrhythmic,

Mechanism of Action:

calcium channel blocker, sodium channel blocker, beta blocker, potassium channel inhibitor, decreases cytokines

Indications:

refractory unstable ventricular tachycardia,
life-threatening ventricular dysrhythmias,
cardiac arrest secondary to ventricular dysrhythmias.

Contraindications:

hypersensitivity
cardiogenic shock, sinus bradycardia, second- or third- degree AV block

Precautions:

history of MI, liver disease, low blood platelets, hypo- or hyperthyroidism, electrolyte imbalances, pulmonary dysfunction.

Adverse effects:

pulmonary toxicity
hypotension, bradycardia, ventricular tachycardia, cardiac arrest
corneal microdeposits, visual disturbances
hypersensitivity
dizziness, blurred vision, unsteadiness (geriatric patients)
photosensitization

Interactions:

May enhance the effects of other medications such as antihypertensives, antidysrhythmics, atropine-like drugs, cyclosporine, digoxin, procainamide, and anticoagulants
may blunt blood sugar benefits if taken with insulin or oral antidiabetic drugs
potential for interaction may persist long after discontinuation of amiodarone because of its long half-life

Dosage and Administration:

- Adult: 300 mg IV slow push
 - 150 mg IV dose may be repeated once in cases of refractory ventricular fibrillation or pulseless ventricular tachycardia
 - maximum total dose by IV route: 450 mg

Atropine Sulfate

Class: Anticholinergic

Mechanism of Action:

inhibits acetylcholine at the parasympathetic neuroeffector junction, blocking vagal effects on the SA and AV nodes; this enhances conduction through the nodes and increases heart rate
antimuscarinic effects on bronchial smooth muscle

Indications:

symptomatic bradycardia, slow PEA, ventricular asystole
narrow complex type II second degree and third degree heart blocks

Contraindications:

unstable cardiovascular status in acute hemorrhage, tachycardia, myocardial ischemia, asthma, myasthenia gravis, hypersensitivity
glaucoma, hypersensitivity
obstructive uropathy, gastrointestinal obstructive disease, paralytic ileus, intestinal atony

Precautions:

use cautiously in patients with Down syndrome, hepatic or renal impairment, hypertension, hyperthyroidism, children with brain damage, and febrile patients

Adverse Effects:

coma, headache, restlessness, ataxia, insomnia, dizziness, confusion in elderly patients.
palpitations, bradycardia (low doses), tachycardia (high doses), aggravated AV block
blurred vision, mydriasis, increased intraocular pressure, dilated pupils
thirst, constipation, bloating, nausea, vomiting
anaphylaxis, urticaria,

Interactions:

antacids decrease absorption of anticholinergics
opioids increase risk of severe constipation, paralytic ileus
antihistamines, antipsychotics, benzodiazepines, tricyclic antidepressants intensify antimuscarinic effects

Special Considerations:

little effect when bradycardia is caused by intrinsic defect of sinoatrial node
may reverse sinus bradycardia when secondary to extracardiac causes

Dosage and Administration:

symptomatic bradycardia - 0.5 mg IV, repeat q 5 min prn, maximum total dose 0.04 mg/kg
PEA/asystole - 1.0 mg IV, repeat q 3 – 5 min prn, maximum total dose 0.04 mg/kg

Clopidogrel Bisulfate (Plavix)

Class: Platelet Aggregation Inhibitor

Mechanism of Action:

specifically inhibits the binding of adenosine diphosphate (ADP) to its platelet receptor thereby inhibiting ADP-mediated activation of platelet aggregation
platelet ADP receptors are irreversibly modified, therefore platelets are affected for their life span

Indications:

secondary prevention/reduction of atherothrombotic events (MI, ischemic stroke, cardiovascular death and/or refractory ischemia) in patients with atherosclerosis documented by stroke, acute coronary syndromes, unstable angina or peripheral arterial disease

Contraindications:

hypersensitivity
active bleeding (peptic ulcer, intracranial hemorrhage)
significant liver impairment, cholestatic jaundice

Precautions:

prolongs bleeding time in patients with active gastrointestinal bleeds
use with caution in patients at risk of increased bleeding from recent surgery, trauma or other pathological conditions

Adverse Effects:

gastrointestinal hemorrhage, abdominal pain, nausea/vomiting
rash

Interactions:

risk of bleeding when used with anticoagulant drugs, however significant adverse interactions have not been detected in patients receiving a variety of concomitant medications

Special Considerations:

benefits shown only when patients were concomitantly treated with ASA in addition to other standard therapies.

Dosage and administration:

75 mg one daily, or for patients with acute coronary syndrome, 300 mg loading dose and then 75 mg daily, as per physician's orders.

Enoxaparin (Low Molecular Weight Heparin) (Lovenox)

Class: Anticoagulant

Mechanism of Action:

inhibits clotting by its ability to catalyze the inhibition of factor Xa
does not bind significantly to plasma proteins, endothelial cells or macrophages

Indications:

unstable angina, prevention of ischemic complications of unstable angina and non Q-wave MI
prophylaxis and treatment of deep vein thrombosis
prevention of extracorporeal clotting during hemodialysis and hemoperfusion

Contraindications:

hypersensitivity to heparin or pork products
patients with severe hypotension, uncontrolled active bleeding, hemorrhagic stroke

Precautions:

avoid in patients with heparin-induced thrombocytopenia

Adverse effects:

hemorrhagic complications at higher doses and in patients with chronic alcoholism, use of platelet-inhibiting drugs, renal failure, advanced age
hematoma at injection site
neurologic injury when used with spinal or epidural puncture
osteoporosis with prolonged use

Interactions:

increased risk of bleeding with concomitant use of oral anticoagulants, platelet inhibitors, NSAIDS

Special Considerations:

no intramuscular injections due to risk of hematoma

Dosage and Administration:

administered via deep subcutaneous injection once or twice daily as per physician's orders

Epinephrine

Class: Sympathomimetic

Mechanism of Action:

stimulates alpha- and beta-adrenergic receptors
increases rate and strength of ventricular contraction
produces vasoconstriction and bronchodilation

Indications:

pulseless ventricular tachycardia; ventricular fibrillation, PEA, asystole
bronchospasm
anaphylaxis
cardiac arrest

Contraindications:

shock (other than anaphylactic shock)
cardiac dilation or dysrhythmias, coronary insufficiency
bronchospasm in patients with coronary artery disease or suspected intracranial hemorrhage

Precautions:

elderly patients and those with hypertension, hyperthyroidism, diabetes and cardiac disease.
use cautiously in patients sensitive to sympathomimetic amines or sulfites, and in patients with psychoneurosis or Parkinson's disease

Adverse effects:

anxiety, headache, fear, disorientation, hallucinations, insomnia, nausea, vomiting
hypertension, cardiac dysrhythmias, tachycardia, anginal pain, palpitations
cerebral hemorrhage, subarachnoid hemorrhage
hemiplegia

Interactions:

may interfere with treatment of anaphylaxis if patient is on beta-blockers
potentiated effects if administered concomitantly with other sympathomimetic drugs
CNS stimulants may increase pressor effects of epinephrine.
diuretic agents may decrease vascular response to epinephrine
antagonizes effects of vasodilators, adrenergic blockers, antidiabetic medications

Special considerations:

do not use epinephrine if its colour is pinkish or darker than slightly yellow or it contains a precipitate

Dosage and Administration:

1.0 mg IV, repeat q 5 min prn

Furosemide (Lasix)

Class: Diuretic

Mechanism of Action:

inhibits sodium and chloride reabsorption, increases potassium excretion
diuretic effect of furosemide is exerted even when glomerular filtration is impaired

Indications:

acute pulmonary edema, hypertension
edema associated with CHF, renal failure, liver cirrhosis

Contraindications:

hypersensitivity
hypotension, hypovolemia, acute AMI
hepatic impairment, hepatic coma
hypokalemia, anuria
pregnancy, jaundiced neonates

Precautions:

may cause electrolyte depletion, potassium supplements may be required with long-term therapy
may patient alertness
use with caution in diabetic, geriatric and pediatric patients

Adverse Effects:

hypersensitivity
cardiac arrest or dysrhythmias, necrotizing angiitis, orthostatic hypotension
electrolyte depletion/disturbances, dehydration, obstructed micturition
dizziness, dry mouth, blurred vision, hearing loss, vertigo, paresthesia, tinnitus
nausea, vomiting, anorexia, gastric irritation, diarrhea and constipation, pancreatitis, ischemic hepatitis,
hyperglycemia
aplastic anemia, eosinophilia, leucopenia and thrombocytopenia

Interactions:

use with caution in patients with sulfonamide sensitivity
NSAIDS may attenuate the effect of furosemide
incompatible with diazepam, diphenhydramine and thiamine

Special Considerations:

use during pregnancy only if potential benefits clearly outweigh possible risks to fetus.
may exacerbate pancreatitis or lupus erythematosus

Dosage and Administration:

40 mg IV, repeat once q 15 min, maximum total dose 160 mg

- if patient is taking a total daily dose greater than 40 mg:
 - dose equivalent to patient's total daily dose (maximum 160 mg)

Heparin Sodium (Unfractionated Heparin)

Class: Anticoagulant

Mechanism of Action:

inhibits clotting process by accelerating the ability of antithrombin III to neutralize thrombin and activated coagulation factor X
blocks conversion of fibrinogen to fibrin
heparin-AT III complex inactivates activated coagulation factors IX, XI, XII and plasmin

Indications:

acute deep vein thrombosis
IV therapy in acute MI patients, treatment of AF pulmonary embolism
diagnosis and treatment of DIC (disseminated intravascular coagulation)
prevention of clotting during dialysis

Contraindications:

hypersensitivity
shock, active bleeding, blood dyscrasia, or bleeding tendencies (hemophilia, thrombocytopenia)
acute ulcer or ulcerating carcinoma
severe liver damage
labour and immediate postpartum period

Precautions:

may increase risk of hemorrhage in third trimester and immediate postpartum period
use with caution in presence of severe hepatic or renal disease

Adverse effects:

hemorrhage, bruising, thrombocytopenia, hyperkalemia, elevated AST and ALT levels
hair loss, osteoporosis may develop with long-term use

Interactions:

may increase risk of bleeding when used with other platelet aggregation inhibitors (salicylates, NSAIDs)
IV nitroglycerine may reduce heparin's anticoagulant effect and necessitate higher doses

Special Considerations:

avoid IM injection of other medications to decrease risk of hematoma formation

Dosage and Administration:

Adult: loading dose: 60 - 80 units/kg IV; maintenance dose: 14 – 18 units/kg/hour IV
Pediatric: loading dose: 50 units/kg IV; maintenance dose: 7.5 units/kg/hour IV

Lidocaine Hydrochloride (Xylocard)

Class: Antidysrhythmic

Mechanism of Action:

depresses ventricular excitability and increases ventricular stimulation threshold during diastole
does not produce significant decrease in arterial pressure or cardiac contractile force

Indications:

pulseless ventricular tachycardia, ventricular fibrillation, ventricular ectopy in the presence of AMI, wide complex tachycardia

Contraindications:

hypersensitivity to amide anesthetics
Adams-Stokes syndrome
severe degrees of sinoatrial, atrioventricular or intraventricular block

Precautions:

constant EKG monitoring is essential during the administration of lidocaine
watch for circulatory collapse in unconscious patients

Adverse Effects:

hypersensitivity
drowsiness, numbness and tingling, blurred vision, disorientation, euphoria, convulsions, coma
apnea, respiratory depression and arrest
cardiovascular manifestations are usually depressant (bradycardia, hypotension, asystole)

Interactions:

none in prehospital setting
concomitant use of metoprolol, nadolol and propranolol may increase plasma levels of lidocaine

Special Considerations:

dosage should be decreased in patient with liver disease, CHF and in the elderly

Dosage and Administration:

Initial: 1.0 – 1.5 mg/kg loading dose, subsequent: 0.5 – 0.75 mg/kg; repeat q 5 – 10 min prn;
maximum total dose: 3.0 mg/kg
patient > 75 yrs, known liver disease, CHF: 0.5 mg/kg; maximum total dose: 3.0 mg/kg

Morphine Sulfate

Class: opioid analgesic

Mechanism of Action:

- alleviates pain through CNS actions
- suppresses fear and anxiety centers in brain
- depresses brain stem respiratory centers
- increases peripheral venous capacitance and decreases venous return
- decreases preload and afterload, decreasing myocardial oxygen demand

Indications:

- analgesia for moderate to severe acute and chronic pain (use with caution)
- severe CHF, pulmonary edema
- chest pain associated with acute MI

Contraindications:

- head injury, exacerbated COPD, depressed respiratory drive, hypotension, suspected hypovolemia
- undiagnosed abdominal pain, decreased level of consciousness
- patients who have taken MAOIs within past 14 days

Adverse Reactions:

- respiratory depression, hypotension, decreased level of consciousness, nausea, vomiting
- bradycardia, tachycardia, syncope, facial flushing, euphoria, bronchospasm, dry mouth

Drug Interactions:

- potentiates sedative effects of phenothiazines
- CNS depressants may potentiate effects of morphine
- MAOIs may cause paradoxical excitation

How Supplied:

- 10 mg in 1 ml of solution, ampules

Dosage and Administration:

- adult: 1-3 mg IV, IM, SC (may repeat q 5 min. to a max. of 10 mg)
- pediatric: 0.1-0.2 mg/kg dose, IV, IO, I'M, SC (may repeat q 5 min. to max. of 5 mg)

Duration of Action:

- onset: immediate
- peak effect: 20 minutes
- duration: 2-7 hours

Special Considerations:

- pregnancy safety: category C
- morphine rapidly crosses the placenta
- safety in neonate not established
- use with caution in geriatric population and those with COPD, asthma
- vagotonic effect in patient with acute inferior MI (bradycardia, heart block)
- naloxone should be readily available as antidote.

Nitroglycerine

Class: vasodilator

Mechanism of Action:

- systemic relaxation of vascular smooth muscle
- reduces myocardial oxygen demand, primarily through peripheral action

Indications:

- chest pain suspected to be of myocardial origin, CHF, PE, HTN

Contraindications:

- hypersensitivity
- hypotension, uncorrected hypovolemia, severe anemia, CNS hemorrhage and increased ICP
- concomitant use of sildenafil

Precautions:

- use in patients with CHF or AMI requires careful hemodynamic monitoring

Adverse Reactions:

- hypotension, headache, syncope, facial flushing, paresthesia,
- dyspnea
- nausea

Interactions:

- caution with other medications that potentially cause hypotension

Special Considerations:

Some individuals may occasionally exhibit marked sensitivity to the hypotensive effects of nitroglycerine and severe responses (pallor, retrosternal discomfort, perspiration and collapse) may occur even with therapeutic doses.

Dosage and Administration:

- 0.4 mg SL spray; repeat q 5 min prn; maximum total dose: 3 sprays.
 - Note: in the event of prolonged transport times, additional doses of nitroglycerine may be administered with orders from physician via on-line medical control or by prior expressed written instructions from the Medical Director.

Oxygen

Class: gas

Mechanism of action: distributed through blood circulation and directly used by all tissues

Indications:

- altered level of consciousness
- chest pain, cardiac emergency
- dyspnea, hypoxemia, respiratory distress, suspected carbon monoxide toxicity
- multiple trauma

Contraindications:

- none

Precautions:

- none

Adverse Effects:

- none

Interactions:

- none

Special Considerations:

- oxygen should not be withheld or minimally administered to patients with chronic obstructive disease

Dosage and Administration:

- by nasal cannula: 2 – 6 litres per minute, inhaled
- by non rebreath mask: 12 – 15 litres per minute, inhaled

Streptokinase (Streptase)

Class: Fibrinolytic

Mechanism of Action:

activates plasminogen in two steps: plasminogen and streptokinase form a complex that exposes the plasminogen-activating site; plasminogen is then converted to plasmin by cleavage of the peptide bond, which leads to fibrinolysis both within a thrombus and on its surface

Indications:

acute evolving transmural myocardial infarction
pulmonary embolism , deep vein thrombosis
lysis of coronary artery thrombi in AMI

Contraindications:

hypersensitivity
previous hemorrhagic stroke, other strokes within previous year
known intracranial neoplasm
active internal bleeding, uncontrollable clotting disorders
suspected aortic dissection

Precautions:

avoid intramuscular injections, venipunctures and unnecessary handling

Adverse Effects:

bleeding, reperfusion dysrhythmias
bronchospasm, pulmonary edema
hypersensitivity
fever

Interactions:

risk of bleeding complications is patients already taking anticoagulants
efficacy is inhibited/reduced by antifibrinolytic drugs

Special Considerations:

unless otherwise indicated, low-dose ASA should be administered with streptokinase for treatment of AMI

Dosage and Administration:

administered via controlled-infusion device as per physician's orders.

Tenecteplase (TNKase)

Class: Fibrinolytic

Mechanism of Action:

modified form of human tissue plasminogen activator (tPA) that binds to fibrin
converts plasminogen to plasmin

Indications:

lysis of occlusive coronary artery thrombi in evolving transmural AMI
pulmonary embolism
ischemic stroke

Contraindications:

active internal bleeding, aneurysm, severe uncontrolled hypertension,
intracranial hemorrhage, intracranial neoplasm
history of CVA, intraspinal or intracranial trauma or surgery within two months

Precautions:

arterial and venous punctures should be minimized
use cautiously in patients with recent surgical history, bleeding disorders, hypertension, or any
conditions which may lead to left heart thrombus

Adverse Effects:

hypersensitivity
cerebral hemorrhage, other peripheral or systemic bleeding
dysrhythmias, hypotension, edema

Interactions:

increased risk of bleeding with concomitant use of ASA, coumadin, anticoagulants, heparin

Special Considerations:

treatment should be initiated as soon as possible after onset of symptoms
standard management of myocardial infarction should be implemented concomitantly with
tenecteplase

Dosage and Administration:

administered via IV infusion as per physician's orders.

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