Cardiovascular Assessment

A Home study
Course
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P.O. Box 480
Hayward CA 94543-0480
Office: 510-888-9070 Fax: 510-537-3434

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Introduction

As all nurses know, the goal of continuing education is to help each nurse to maintain the highest level of nursing skill. This course was designed with that objective in mind. This test was meant to be used by those nurses who need to advance and/or improve their skill level in the aria of cardiovascular assessment.

Therefore, this course goes beyond the basic" theory that all nurses would be expected to know from their basic training in nursing school. This course presents the theory needed to perform a complete and detailed cardiovascular assessment. We hope that all medical/surgical nurses who participate in this course will have the opportunity to use this theoretical knowledge in the workplace, and to improve their clinical assessment skills.

You will be able to maximize your learning experience by first studying the course objectives. The objectives will point out those topics that are of the greatest importance. The test at the end of this course will be based on course objectives.

This is a comprehensive course dealing with major aspects of assessment of the adult patient. The beginning of the course points out the basic anatomy and physiology of the cardiovascular system that all nurses are quite familiar with.

Preface

Assessment of the cardiovascular system is one of the most important areas of the nurse's daily patient assessment. This course is designed to be used with the guidelines already in effect at your institution.

As a guide, this course could be used alone. It presents a foundation of relevant anatomy and physiology concepts needed as a background for performing a precise and definite nursing assessment. At the end of this program a brief tool is presented for your use at the bedside. One of the most powerful tools that the nurse should use is the power of observation. Always be observant for even a slight deviation from the norm. If you detect something that is abnormal, further assess that area, and report your findings.

Be as objective as possible. When you are unsure, investigate it further. Assess with every tool possible; inspection, palpation, etc. Report your findings as clearly as possible. Charting your results clearly is essential for others to be able to assess the problem, and good documentation is also essential for the treatment of the patient as well as for the nursing care.

Lastly, follow through with your findings. Especially if there is an abnormality, report your findings to the appropriate person(s). Chart those finding and the fact that you did report it. Take credit and responsibility for your actions. It is your legal and moral duty. Accurate charting and reporting will protect you and the patient. In other words . . . accurate and thorough assessment and reporting are essential tools to keep in mind throughout this course.

Objectives

Long term objectives:

- To increase awareness of the importance of the cardiovascular assessment techniques and obtain a thorough knowledge base in the areas of assessment and patient care, in order to better care for all your patients; especially those with a cardiovascular impairment.
- To improve the skill level of hands on assessment techniques of the cardiovascular system, by increasing the theoretical knowledge base of the nurse participant.
- To better understand the legal and moral ramifications of performing a complete and thorough assessment; reporting of abnormal findings, the emergency procedures, and the accurate charting of all results, whether normal or abnormal.

At the end of the course, each participant will be able to:

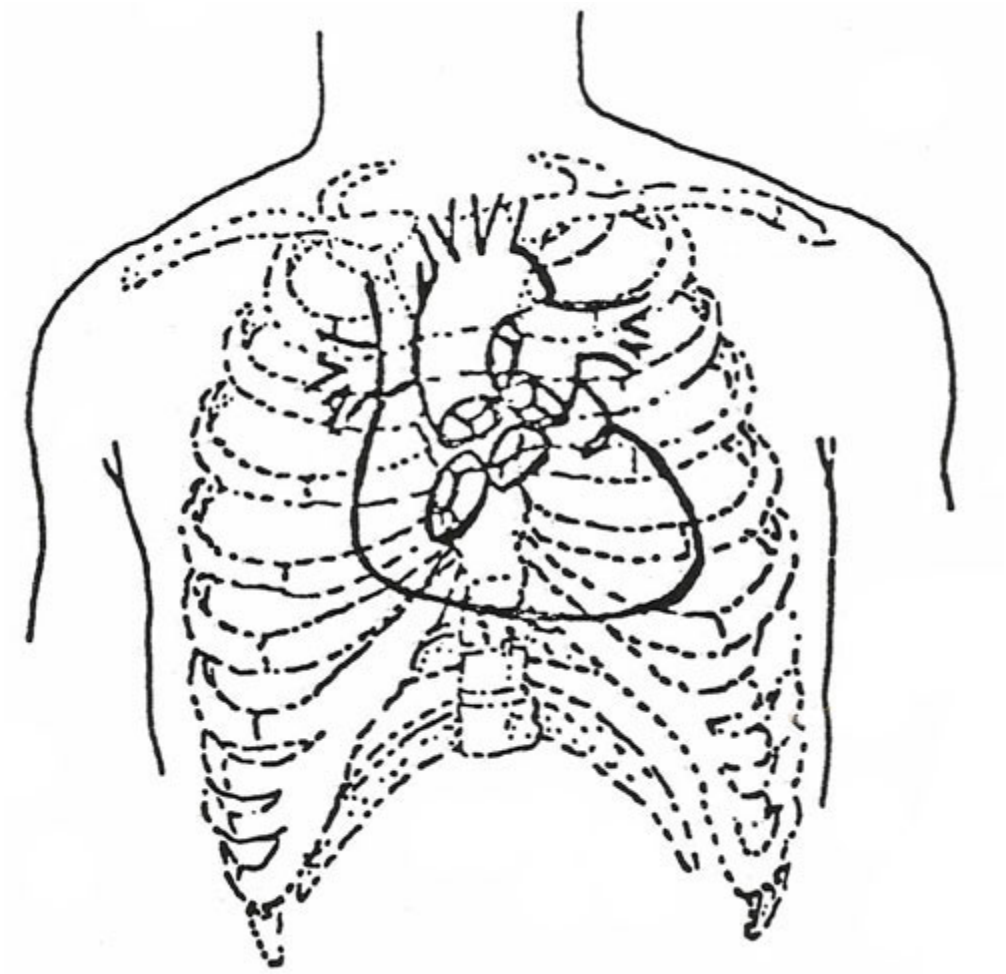
- Name and describe the seven parts of the thorax.
- Name and describe the two major parts of the gross muscular structure of the heart.
- Name and describe the four chambers of the heart as they relate to pumping action.
- Name and describe the four valves of the heart and their position in the heart.
- Discuss all the important parts of the cardiovascular system as identified in the outline of the test; including heart sounds and EKG interpretation.
- Perform with 70% accuracy on an objective examination covering all areas of the course.
- Name and discuss at least two major aspects of the physiology of the CV system which relates to the disease process and the resultant abnormal findings on examination.
- Name and discuss the normal and abnormal findings of assessment of the bony thorax and gross findings of the heart and great blood vessels, including peripheral vascular disease, hypertension, and the condition of shock.
- Discuss the definition and importance of inspection of the CV system including the gross anatomical structures.

Part I: Anatomy and Physiology Update

The Thorax

The first part of this course is a review of the anatomical structures and the physiology of the cardiovascular system in the relationship to the nursing care of the patient with a related disorder. If you need to review the structures and/or function which we will be discussing, please refer to any basic anatomy textbook. However, it is not required that you use any other reference. All information needed to pass the test at the end of this course will be included in the text.

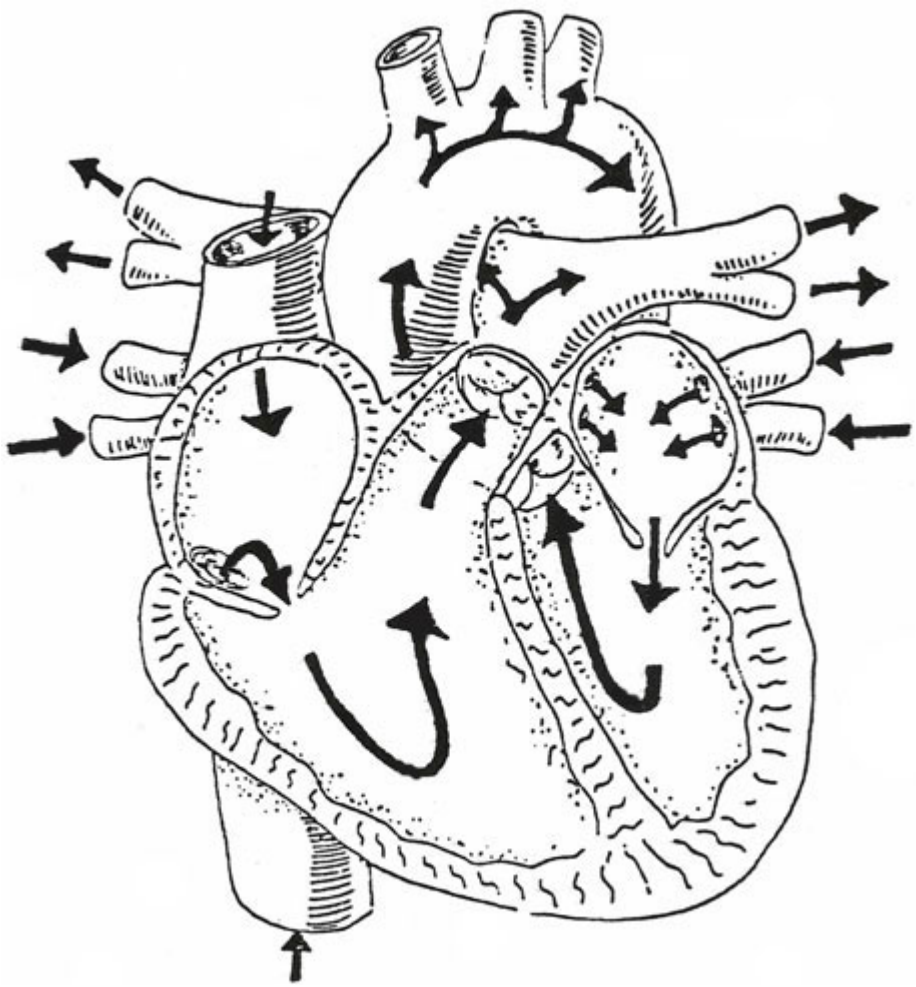
We will list the most important structures of the thorax and entire CV system only so that you can relate these to the clinical approach that we will use. Following is an illustration of the thorax and the heart in relationship to other structures which will be noticed upon visual examination. The thorax has a characteristic shape, size, and movement.



Be sure you can identify the following:

- Sternum – mid chest, flat, non-protruding.
- Ribs – slope of ribs, intercostals spaces, costal margins.
- Heart – heartbeat in some cases can be visible as a pulsation in the thorax at lower costal margin.
- Shoulder – patient's shoulder should be relaxed, and at a 90 degree angle to the patient – look for abnormal angles and musculature which might indicate overuse of accessory muscles.
- Neck veins – should not normally be visible.
- Clavicle – clavicular line horizontal with no protrusions during breathing.
- Respirations – normal respirations should look unlabored and comfortable.

The Heart



Gross Structures

1. Musculature – pericardium, fibrous and serous epicardium, visceral serous pericardium, myocardium, heart muscle.
2. Muscle cell (microscopic structures) central nucleus, sarcoplasm, sarcolemma, intercalated discs.

Chambers – Right Side of Heart

1. Right atrium – the thin walled atrium, low relative pressure receives blood from superior and inferior vena cavae, the coronary sinus and Thebesian veins. The outflow of blood through tricuspid valve.
2. Right ventricle – relatively thin muscle wall, crescent shaped, papillary muscles, chordate tendineae, low pressure. Outflow through the pulmonic valve to the pulmonary artery.

Left Side of Heart

1. Left atrium – thicker muscle, medium pressure of blood, inflow of blood through four pulmonary veins. Outflow is through mitral valve.

2. Left ventricle – largest muscle mass, high pressure blood flow, papillary muscles, spring like pump action. Outflow of blood through the aortic valve and the aorta.

Cardiac Anatomy:

The human heart is a hollow, four chambered, muscular pump. It is the major organ in the mediastinum. The pericardium is the outermost layer of the heart. It consists of parietal and visceral layers. The pericardial sac, normally containing 5 to 20 cc of fluid, protects the myocardium and prevents friction during the pumping action of the heart.

Muscle tissue, the myocardium, makes up the walls of the heart chambers. The left ventricular myocardium is 5 to 7 times thicker than the right. The inner surface of the myocardium is lined with endocardium, as are the cardiac valves and blood vessels.

The heart is divided into chambers by intraventricular and intra-atrial septa. Fibrous tissue separates the atria from the ventricles on the right and left sides of the heart. The tricuspid and mitral valves, together called the atrioventricular (A-V) valves, allow for the passage of blood from the atria to the ventricles.

Heart Valves

Antrioventricular Valves

1. Tricuspid – has three leaflets, controlled by papillary muscles, chordate tendineae.
2. Mitral Valve – two cusps, controlled by papillary muscles and the chordate tendineae.

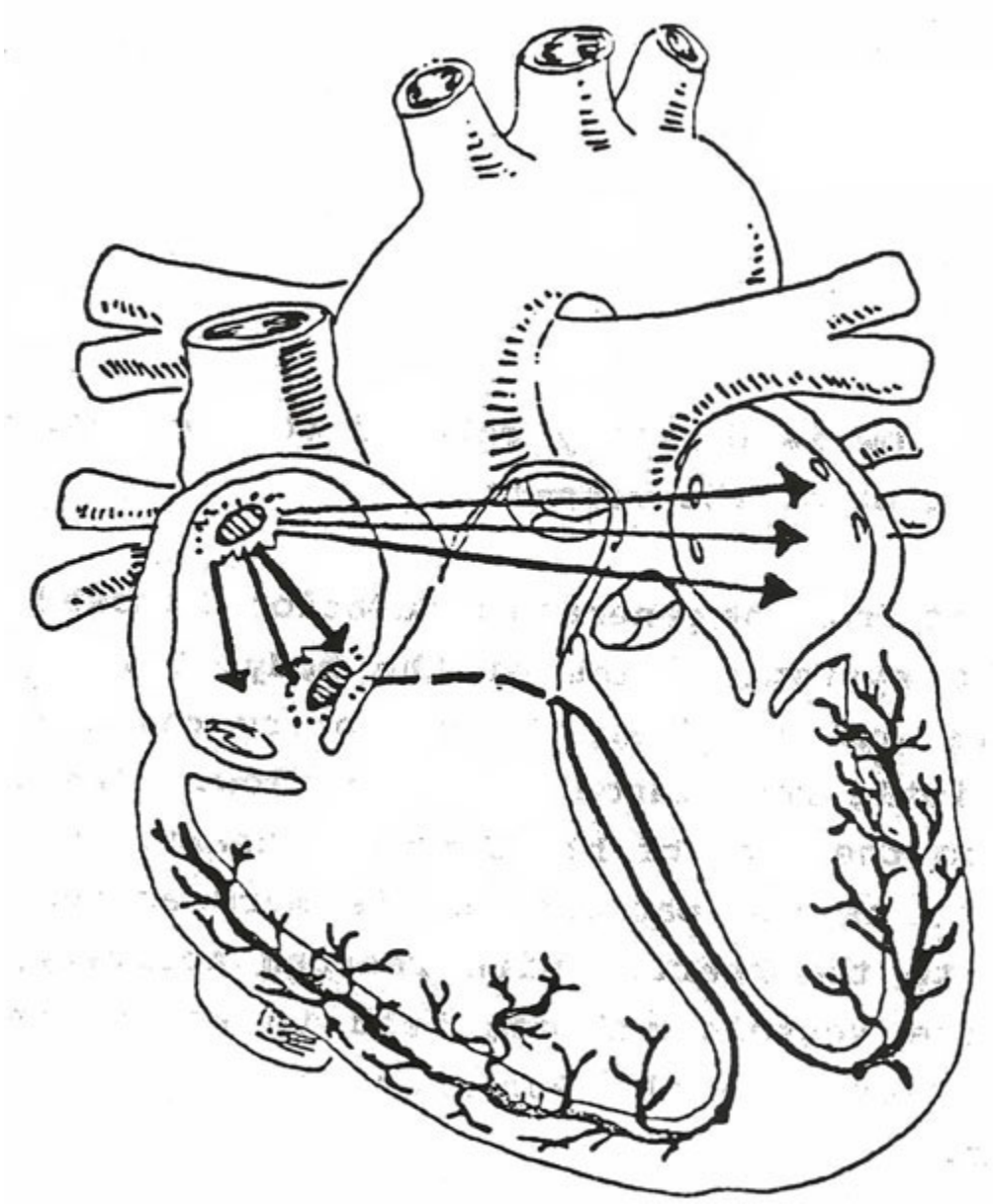
Semi lunar Valves

1. Pulmonic Valve – Three leaflet valves, formed by fibrous ring, tendinous tubercle midpoint free edges.
2. Aortic Valve – three leaflets, also formed by fibrous ring, tendinous tubercle midpoint free edges.

Vasculature of the Heart

1. Right Coronary Artery – most branches of this artery anastomose distally with left anterior descending.
2. Left Coronary Artery – Divides into two main branches, left ant. Descending and left circumflex artery.
3. Great Cardiac Vein - largest system, forms coronary sinus, drains left ventricle primarily.
4. Anterior Cardiac Veins – empty directly into right atrium.
5. Thebesian Veins – smallest system, empty into right atrium.

Conduction System of the Heart



1. SA (Sino-atrial node)
2. Atrial preferential pathways – anterior intermodal, middle, posterior intermodal.
3. AV (Atrio-ventricular node)
4. Bundle of HIS
5. Left Bundle Branch
6. Right Bundle Branch
7. Purkinje Fibres

Contractility of Heart Muscle

Electrical conduction in the heart is unique and remarkable.

Heart muscle possesses the following properties:

1. Automaticity – pacemaker ability.
2. Conductivity – each cell has the ability to conduct impulses to the next cell.
3. Contractility – ability to contract (make each cell shorter or longer).
4. Irritability – each cell has the ability to contract on its own, to send out impulses to other cells without it first being stimulated from another source.

These properties make the myocardium different from other muscle cells I the body. The normal activity of the heart conducts impulses from one point (AS node) to another point (individual muscle cells), thus stimulating a uniform and effective contraction of the heart.

Various factors affect the activity of the cardiac muscles. The availability of oxygen, after load, nervous control, muscle condition, and other factors can affect the four of the contractions of the heart. Drugs can also affect the contraction of the heart. Certain drugs depress the heart activity and others can cause excitation. The nurse should be aware of all the factors which influence heart activity.

Blood Flow through the Heart

Blood flow through the heart is shown in the illustration. Blood is shown as it enters the heart, circulates, and then leaves the heart. In relation to the physical assessment performed by most nurses, keep in mind the changes in circulation which will be assessed. Impeded flow may cause extra heart sounds and/or physical changes. Also, reduced flow will usually cause changes that can be assessed by the nurse.

Physical Characteristics Important to Blood Flow:

1. Diameter of the blood vessels.
2. Cross-section areas of the chambers and vessels.
3. Length of the vessels.

Quantities of Blood:

1. Heart – 18%	5. Arterioles – 2%
2. Pulmonary vessels – 12%	6. Capillaries – 5%
3. Large arteries – 8%	7. Small veins – 25%
4. Small arteries – 5%	8. Large veins – 25%

Velocities of Blood Flow:

The velocity of blood flow is directly related to the amount of circulating blood volume and the area of the vessels.

Blood returns to the heart from the general circulations. Almost 50% of all blood in the body is in the systemic veins of the body. This system includes small veins and venules and blood in the pulmonary circulation. The small veins usually offer little resistance to blood flow. The large veins do offer much resistance to the flow of blood to the heart. This is an important nursing implication, as the patient who is more active will have better flow of blood back to the heart. With reduced activity, the blood tends to pool in the large vessels and can lead to severe venous stasis. Blood returns to the heart via the superior and inferior vena cavae, and into the right atrium.

From the right atrium blood flows to the right ventricle and is then propelled into pulmonary circulation. After blood is aerated with fresh oxygen, it is returned to the left side of the heart into the left atrium.

From the left atrium the blood is ejected into the left ventricle. The left ventricle then pumps the blood out of the heart into the general circulation. The aorta is the first vessel to carry blood, and, at the same time, the coronary arteries are being fed oxygenated blood to circulate trough the heart.

The above is only a brief outline of the circulation of blood. Be sure you can trace the blood through the heart. Be sure that you can name all the valves and chambers of the heart as blood flows through. You should also be able to list the major arteries of the body. Later, when we are performing the assessment, it will be necessary for you to know these vessels and their location.

Physiology of the Cardiovascular System

Blood Pressure

Blood pressure is determined by many factors in the body. Normal blood pressure is determined by the cardiac output, the velocity, the resistance of the blood vessels, and by other factors. Systolic pressure refers to the initial force of contraction of the heart. Diastolic pressure refers to the pressure of the blood vessels after the initial force of contraction of the heart. In other words, the diastolic pressure is due to the elasticity of the arteries as they “snap” back after the initial “stretch” due to the systolic pressure. Blood pressure is probably one of the most important measures of the overall cardiovascular system that exists.

Take the patient’s blood pressure lying down and then standing up. If there is difference of more than 15 – 20 mm Hg, this may be an indication of one or more problems. Persons with hypertension that is poorly controlled may have orthostatic hypertension. This may also indicate aortic disease or cerebrovascular disease.

The pulse pressure is the difference between the two pressure readings (systolic and diastolic). The pulse pressure is very important because it can indicate certain major problems in the cardiovascular system. Shock can be diagnosed, in part, by blood pressure readings and the pulse pressure. The pulse pressure is normally determined by the general condition of the heart, arteries, and the amount of circulating blood.

Autonomic Nervous System Control

Autonomic nervous system control of the cardiovascular system includes control of the heart rate, cardiac output, blood vessels and amount of blood volume. Cardiac muscle is under the influence of the sympathetic and parasympathic nervous systems. The sympathetic (cervical) system secretes norepinephrine and innervates the cardiac plexus. It increases the SA node rate, it increase the AV node conduction, and increases the contractile force of the myocardium. The parasympathetic division (cholinergic fibres) secretes acetylcholine and also innervates the cardiac plexus. This branch decreases the SA node, AV node, and the contractility of the heart. Together, they work to regulate the heart rate, blood pressure, and other vital cardiovascular functions. Blood vessel contraction is controlled in a like manner. The sympathetic division

causes vasoconstriction in most blood vessels, thus raising the blood pressure and circulating volume. The parasympathetic division causes vasodilatation for most vessels.

The Pressoreceptor System

The pressoreceptor system originates in the arch of the aorta. Here are located sensitive nerve endings which help to control blood pressure and heart rate. Receptors are also located in the carotid sinus, vena cavae, and the pulmonary arteries. When these receptors sense low pressure, the signal is fed to the medulla, where then the sympathetic division impulse is increased, causing the pressure to rise (either due to increased pulse rate of increased contractility). The stimulation acts to bring the pressure back to normal by either vasoconstriction or vasodilatation; by increased heart rate or decreased rate; by increased contractility or decreased contractility. Whichever combination occurs, the result will be the same, to bring the pressure back to normal.

Summary (History and Chief Complaint)

Before you go to the patient's room, you should read pertinent data that has already been recorded about the patient. In many hospitals, the patient's old charts from previous admissions are available. Read the old charts and obtain a summary of important data such as:

- Previous admissions to the hospital.
- Current and past drugs taken.
- Reasons for admission.
- Social supports.
- Allergies.
- Discharge information.
- Chronic medical problems.

Next, interview the patient to discover "History of Present Illness."

The *History of Present Illness* means: what contributed to their coming to the hospital? You will ask for present symptoms, and other recent symptoms that would be pertinent to this present illness. Perhaps they had some symptom at home that is now gone. You should question them in detail to confirm any historical data that you found in the old chart. Do not record any information from the old chart as fact until confirmed by the patient in your interview.

When you enter the room to interview your patient, the first thing to do is introduce yourself and explain that you will be interviewing them. Try to make them relax as much as possible. If they are just being admitted to the hospital, they will probably be very apprehensive. Use a blend of subtle humor and calm, soothing conversation to help the patient relax. One important consideration is not to rush them. Often you are interviewing the patient under rushed conditions. Rushing them will make them even more anxious. Give them time to answer questions. Do not interrupt them when they are trying to answer; let them finish before you ask the next question.

We will discuss charting and recording later. However, be sure that you record patient's statements accurately. Put what they say in quotes when applicable.

This is extremely important to remember: BE ACCURATE! Many times the patient is unfamiliar with medical terms, and they might express a symptom in lay terms. Quote them as much as possible when you record their statements or comments. You might not understand what the patient means why they describe something in lay terms.

For example, the patient might describe that they feel “crawly” when they took penicillin last year. Perhaps “crawly” means they were having an allergic reaction or maybe not. In any case, bring it to the attention of the physician and record what they say in quotes.

Once you have completed the history and details of the present illness, ask the patient to describe any symptoms that seem important to you. Ask them to describe in detail, their symptoms, pain or bowel habits, whatever is important and relates to their diagnosis. For example, eating habits for a patient who has abdominal pain ask:

- How many meals do you eat each day?
- Do you eat meats?
- Do you eat dairy products?
- Do you drink coffee?

Then ask:

- How many of these foods do you eat?
- What time of day do you eat them?

You might get interesting responses to the detailed questions that might explain some of their symptoms. Record the responses and ask more detailed questions if necessary. Remember to have the patient fully explain all symptoms in detail.

Since we’re focusing on the cardiovascular system for this course, we will ask the patient to give us those pertinent details. First ask the patient if s/he has been told they have any of the following conditions:

- “Heart Attack” or “Heart Trouble”
- Myocardial infarction.
- Angina or “hear pain” or angina pectoris.
- Congenital heart problems or heart problems you were born with.
- Rheumatic fever or rheumatic heart disease.
- Coronary heart disease.
- Heart arrhythmias or heart murmurs.

You should use the precise medical terms mentioned above. Many patients might remember the term if they heard it. Some patients have been hospitalized so many times that they will be able to sue the exact term for their illness. Now remember that you looked at their chart (if it was available). That will give you an edge, but remember to confirm the problems with them directly. Some patients will not know the medical term for any past problems they had. In that case, you should ask them to describe the condition. Use some of the terms mentioned above.

Next, ask what medications they have taken for their heart problems. Of course, they might have answered this question and others in the above general medical history. However, have them give more details, since we are now concerned primary with the cardiovascular part of the history. Find out the following for each drug:

- Drug name and dosage.
- What s/he takes it for.
- Frequency.
- Does it work or help?
- When was the last dose taken?

Next, assess their use of alcohol and nicotine. Get details of the type of alcohol and/or "recreational" drugs used and the frequency, and their smoking habits. Alcohol, cocaine, nicotine, and other drugs can directly affect the heart, causing arrhythmias and other cardiac conditions.

Assess social factors that might affect the patient should major surgery be required. If the patient is facing a severe debilitating condition the physician would need to know the patient's educational background, and the resources they have at home and at the job. Will this illness affect their job or career? These are questions the nurse should anticipate will be important later on in their hospitalization.

Recording the Findings:

There are many different ways to record your observations. Some facilities utilize narrative charting forms. Other facilities use problem-oriented charting. Still other facilities have computerized medical records. However it does not matter how you record the findings; just be sure that you do record the findings. This will include vital signs, history, and any pertinent data.

Part II: Assessment Techniques

Inspection

As you prepare to begin the actual assessment, you already have obtained and recorded the patient history and you arm yourself with pertinent data such as their chief complaint and allergic history.

Also keep in mind to allow a certain amount of time in order to complete a thorough exam. Many nurses do not have large blocks of time for completion of the assessment but you must be as thorough as possible. If this is an admission assessment, you *must* allow enough time to be complete. If this is an on-going assessment, not as much time will be required.

Begin Exam

- Patient undresses, but allow for privacy.
- Have the patient sit upright and inspect the thorax from the front.
- Now inspect from the back of the patient.

You will inspect for posture and symmetry of the thorax, color of the skin, gross deformities of the skin or bone structure, the neck, face, eyes, and any abnormal contours. Breathing patters will also be noted. Be especially aware of the presence of cyanosis. Central cyanosis is a condition which will cause the lips, mouth, and conjunctiva to become blue. Peripheral cyanosis will cause blue discoloration mainly on the lips, ear lobes, and nail beds. Peripheral cyanosis might indicate a peripheral problem of vasoconstriction, and would generally be less severe than central cyanosis, which could indicate heart disease and poor oxygenation.

Thorax

Inspect for symmetry of thorax, point of maximum intensity (PMI). PMI is easier to find if the patient will lay on the left side. PMI may also be palpated. Check skin color of thorax.

Eyes

Arcus Senilis is a light gray ring surrounding the iris, common in older patients; in younger patients it might indicate a type of lipid metabolism disorder, which is a precursor to coronary artery disease.

Xanthelasma is yellowish raised plaques on the skin surrounding the eyes. Can also appear on the elbows. This is a possible indication, or sign of hypercholesterolemia, often a precursor to coronary artery disease (atherosclerosis).

Palpation

Palpation, or touching, is the next part of the exam. In the stop above, if we noted any abnormalities, we will now palpate and evaluate them further.

Skin: temperature, texture, moisture, lumps, bumps, tenderness.

Examination of extremities for edema might also indicate a cardiovascular problem. Examine the feet, ankles, sacrum, abdomen, trunk, and face for edema. If you notice puffiness of frank edema, then palpate the area for pitting edema. Most facilities recognize the following scale:

+1 Pitting Edema	=	0 to ¼ inch indentation
+2 Pitting Edema	=	¼ to ½ inch indentation
+3 Pitting Edema	=	½ to 1 inch indentation
+4 Pitting Edema	=	More than 1 inch indentation

Breathing: lay hands the chest at different locations and feel the respiratory patterns, feel the ribs elevate and separate during normal breathing.

Pre-Cordial Areas you can feel the pounding of the heartbeat, normal and abnormal pulsations o the chest wall; PMI, as mentioned above.

Arteries: Assess all pulses

You undoubtedly assessed the apical pulse earlier when you took the patient's vital signs, if not, now is the time. Assess the following pulses:

1. Apical heart rate – monitor for a full minute, note rhythm, rate, regularity.

2. Radial pulse – monitor for a full minute. Note the rhythm, rate, and the regularity. Note any differences from right to left radial, a large difference might indicate arterial blockage or even enlarged ventricles. If pulse is regular but volume diminishes from beat to beat, this might indicate left-sided heart failure and is called **pulses alternans**. If the volume of the pulse diminishes on inspiration,

might indicate constrictive pericardial disease, the condition is **called pulsus paradoxus**.

3. Carotid, brachial, femoral, popliteal, posterior tibialis, and dorsalis pedis pulses – when checking these pulses do it the same way as the others mentioned in this section; right then left side. When you check the carotid, press gently and do not rub.

   Do not palpate carotid on persons with known carotid disease or bruits; listen with stethoscope instead; and do not palpate both carotid pulses at the same time.

Carotid Artery:

- Plateau pulse – slow rise and slow collapse pulse; may be caused by aortic stenosis, slow ejection of blood through a narrowed aortic valve.

- Decreases amplitude (grade point pulse) – due to hemorrhagic shock, pulse is weak due to decreased blood volume.

Bounding Pulse - (Grade IV) can be due to hypertension, thyrotoxicosis, others; associated with high pulse pressure, the upstroke and downstroke of the pulse waves are very sharp.

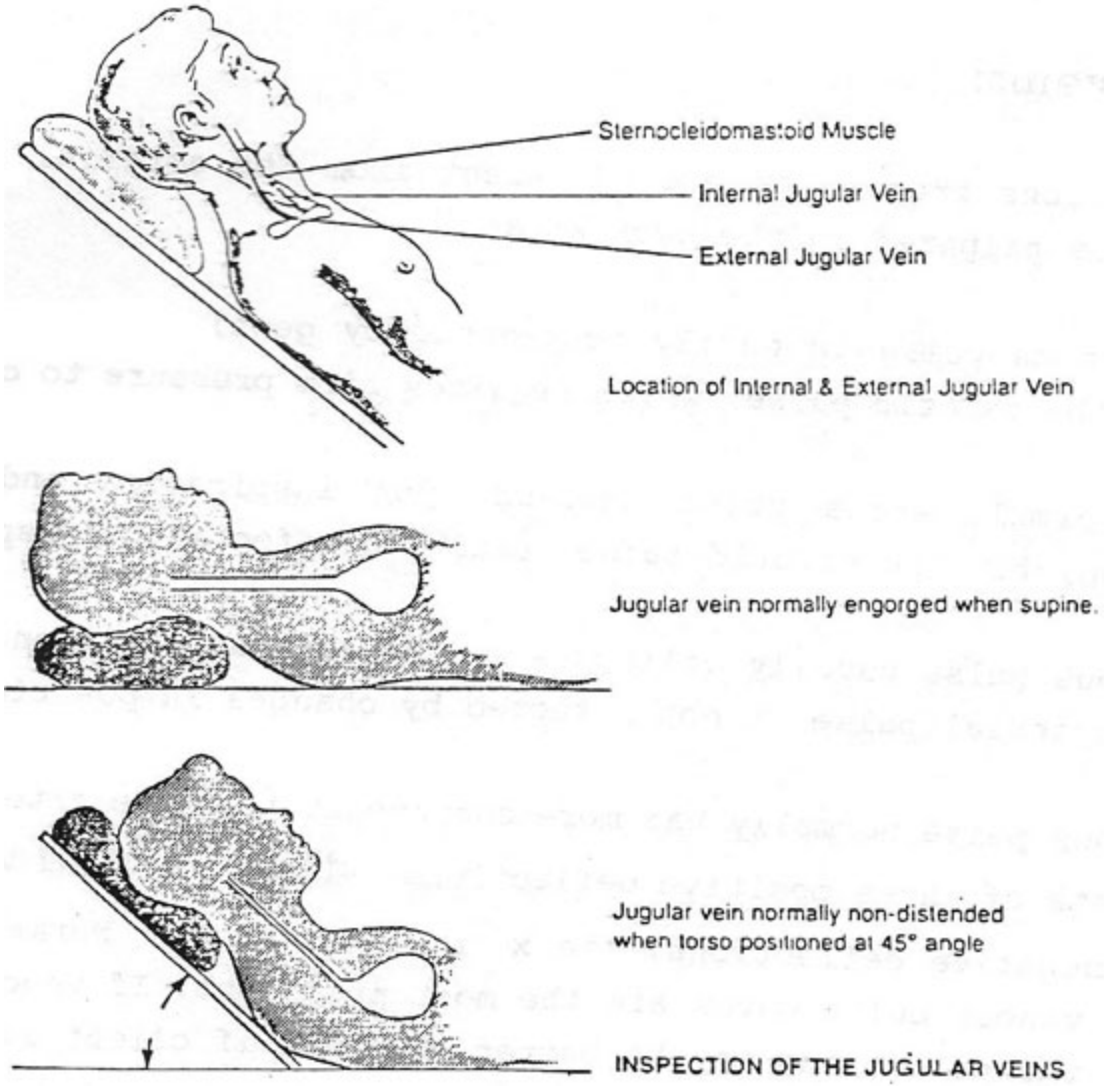
It is common to use +1, +2, etc. when recording pulses:

- 0 = absent
- +1 = diminished or decreased
- +2 = normal pulses
- +3 = full pulse or slight increase in pulse volume
- +4 = bounding pulse or increased volume

Veins – neck, arms, legs, etc.

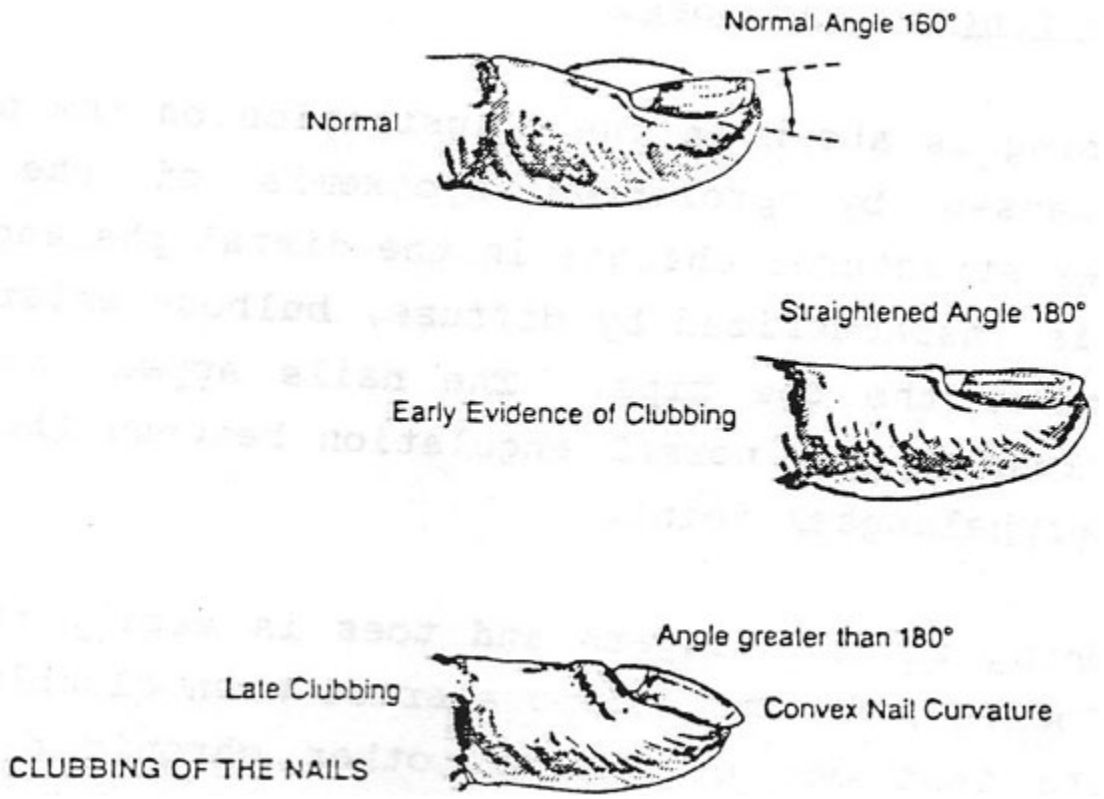
Veins: Central Venous Pressure (CVP):

In order to assess the patients CVP, start by having the patient sit in bed and then lean backwards at about a 45 degree angle. Let the patient relax for a few seconds while you look for the internal jugular vein. In most persons in which the vein's pulsating is visible, the vein will be seen to pulsate at the level of the sterna notch (Angel of Louis). If the level of pulsation is more than 3cm above the level of the sterna notch, it is a sign that the CVP is elevated. An elevated CVP may be indicative of right sided heart failure, obstruction of the superior vena cava, or constrictive pericarditis. Normal pressure in the venous circulation runs from 5 to 12 centimeters of water pressure. The CVP would usually be measured by placing a catheter into a large vein and attach it to manometer or strain gauge. See figures below:



If you find evidence of elevated CVP, may further confirm the findings that you just saw. The hepato-jugular reflux test may be used. This test is performed by placing your hand in the aria of the right upper quadrant of the abdomen. Once you have placed your hand on the abdomen, exert firm pressure directly into the abdomen for one full minute, and at the same time, observe the jugular vein. If the pulsation you observed begins to definitely rise over the highest level of pulsation seen, then this confirms that the CVP is elevated.

Clubbing of the Fingers and Toes



Clubbing is caused y prolonged hypoxemia of the extremities. Hypoxemia causes structural changes in the distal phalanges over time. Nail clubbing is characterized by diffuse, bulbous enlargement of the finger tips and/or the toe tips. The nails appear shiny and curve downward with loss of the normal angulations between the nail bed and the distal interphalangeal joint.

Clubbing of the fingers and toes is easily recognized upon inspection. However, do not become alarmed when clubbing present. It may indicate that one of several other chronic diseases may be present. Clubbing only indicates that there is possibly a chronic lack of oxygen to the extremities and may be caused by many different factors.

Jugular Veins:

The pulsations from veins are different from the arterial pulsations that can be palpated in the neck area:

- The venous pulse is easily compressed by gentle pressure in contrast to the carotid pulse, which requires firm pressure to obliterate.

- The normal venous pulse descends upon inspiration and rises on expiration, but the carotid pulse remains unaffected by respirations.

- A venous pulse usually collapses in the sitting position, while the carotid arterial pulse is not affected by changes in position.

- A venous pulse normally has more components than the arterial pulse. If consists of three positive deflections, the **A, C, and V waves**, and two negative deflections, **the x and y descents**. Normally this when the venous pulse waves are the most prominent. If venous pressure is high, the pulsations may e better observed if client is sitting.

A Wave - The predominant wave in the neck reflects the pressure transmission caused by atrial contraction begins just before the fist heart sound; it can be palpated by feeling the jugular pulse, while ausculating the apex of the heart. The wave also occurs just prior to the carotid pulsation.

C Wave - This is a reflection of the onset of right ventricular contraction. Begins at the end of the first heart sound and is usually not visible in the neck veins.

V Wave - Represents atrial filling with the Antrioventricular valve closed. It is very small and is considered a passive filling wave.

X Descent - Is a wave following the c wave. Represents atrial diastole.

V Wave - is followed by a y descent, a negative wave produced when the tricuspid valve opens, allowing blood to pour into the right ventricle.

Hair

Observe and feel the consistency and texture of the person's hair. Very fine hair shafts may indicate hyperthyroidism. Very course hair shafts present might indicate hypothyroidism. Both conditions of the thyroid may have adverse effects on heart and cardiovascular systems.

Percussion

This technique has a very limited place for nurses in assessment of patients. Percussion refers to "tapping" the chest wall with the fingers in order to elicit sounds which indicate abnormalities. We will discuss and demonstrate the technique but remember that it is a very limited tool. Much information obtained by percussion can be more easily determined by auscultation.

The technique for percussion involves hyper extending the fingers of one hand and placing the middle distal phalanx firmly on the chest wall. Hold your opposite hand close to the hand on the patient. Retract the middle finger of that second hand; strike the finger firmly at the top of the distal phalanx. After striking the finger, quickly remove it and then move to another area and repeat the same motions.

Percuss the precordial area of the chest, listening for a resonant sound which indicates normal tissue beneath the finders. When percussion over the lung tissue, the sound will be resonant, a semi-hollow, medium pitched sound will be flat or "dull" in pitch. These are normal sounds. If the patient is sensitive or indicates pain or difficulty breathing, stop the percussion and go on with the other parts of your assessment.

Auscultation

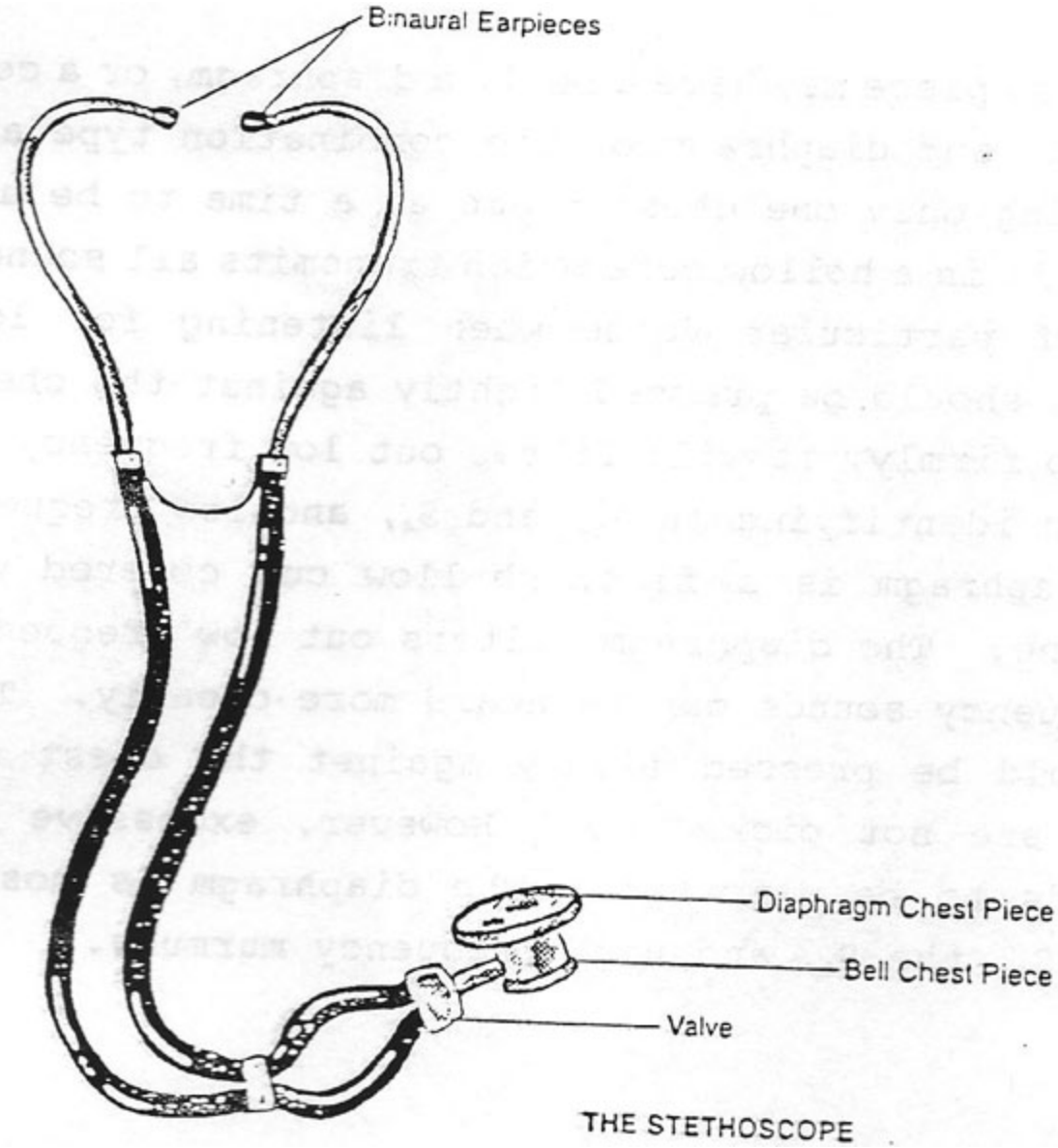
Auscultation is defined as listening to the sounds produced by the body with or without the use of a stethoscope. Some sounds may be loud enough to hear without the use of the stethoscope. Although when we think of auscultation and the cardiovascular system, most

nurses think of listening to the chest and heart through the stethoscope. The heart sounds heard are due to the closure of the heart valves under pressure of the blood flow. The stethoscope can also be used to listen to other sounds that could be of significant importance to the cardiovascular system.

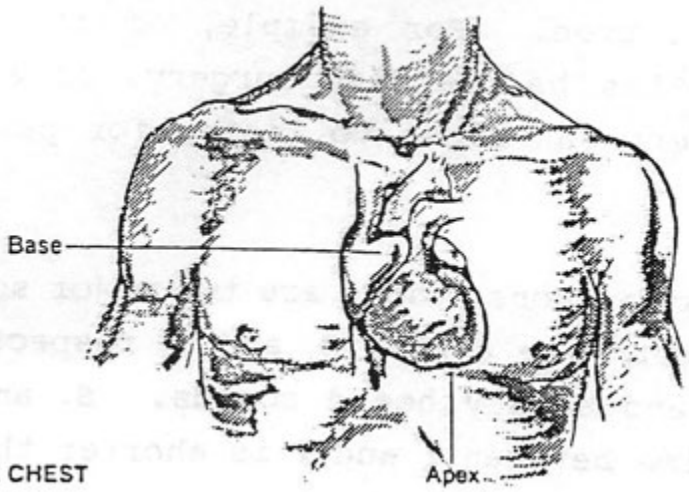
Use of the Stethoscope:

The chest piece may have a bell, diaphragm, or combination of the two. The bell and diaphragm of the combination type are connected by a valve allowing only one chest piece at a time to be used.

The bell is a hollow cone which transmits all sounds within the chest. It is of particular value when listening against the chest wall. If it is pressed too firmly, it will filter out low frequency sounds. The bell is useful in indentifying an S3and S4, and low frequency sounds to that higher frequency sounds can be heard more clearly. The diaphragm chest piece should be pressed firmly against the chest wall so that external sounds are not picked up. However, excessive pressure may cause some sounds to be dampened. The diaphragm is most useful for indentifying the S1, the S2, and high frequency murmurs.



THE STETHOSCOPE



POSITION OF HEART WITHIN THE CHEST

Heart Sounds

Auscultation of heart sounds should usually follow the general medical assessment and the general assessment of the cardiovascular system. The nurse should first think about the results of the general assessment and then proceed to listen to the heart sounds. In many cases, the first part of the assessment will give you a clue of what to listen for upon auscultation. For example, if the patient states in his history that he has cardiac surgery, a valve replaced, etc., then it will alert the nurse to listen for particular sounds or murmurs.

In most persons, there are two major sounds that can be heard. The “lub” and “dub” are called S1 and S2, respectively and are the two most prominent and easily heard sounds. S1 and S2 follow each other closely. The time between a 1 and 2 is shorter than the time from end of S2 to the beginning of the next cycle and S1 of the next beat. The time interval between S1 and S2 also corresponds to systolic pressure of the cardiac cycle.

The additional heart sounds may be audible in the cardiac cycle; these are S3 and S4. S3 is the sound of early, rapid diastolic filling of the ventricles. It is not often heard in adults but is heard very commonly in children. S4 is the last heart sound and like S3, it is rarely heard in the adult except in disease conditions such as congestive heart failure of multiple sclerosis.

Origins of Heart Sounds:

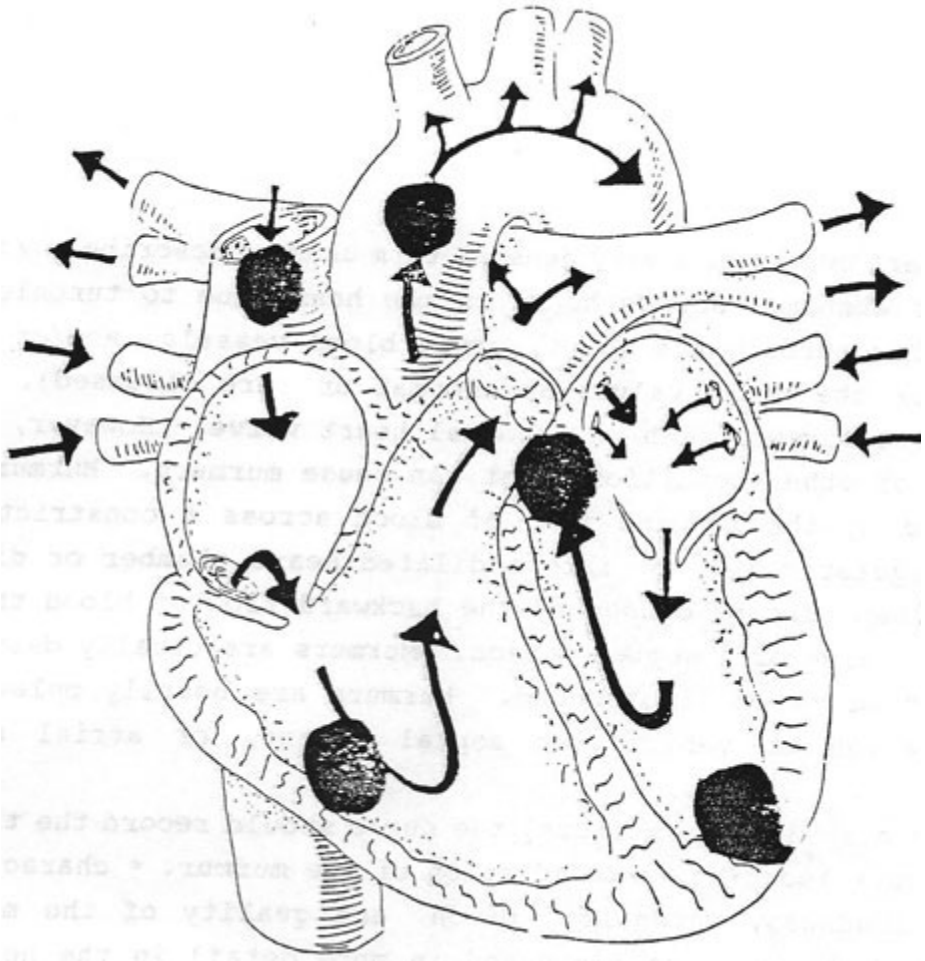
S1	=	Closing of the atrioventricular valves (Mitral and Tricuspid) corresponds with the carotid pulse; ventricular systole heard loudest at the mitral and tricuspid areas.
S2	=	Closing of the aortic and pulmonic valves heard loudest at the aortic area.

Each of the two major heard sounds is made up of the rushing of blood and of the two valves closing at the same time. Normally, the pairs of valves open and close at the same time, causing one clear and distinct sound. At certain times when the valves may close at slightly different times, or one valve may close very slightly slower than the corresponding valve.

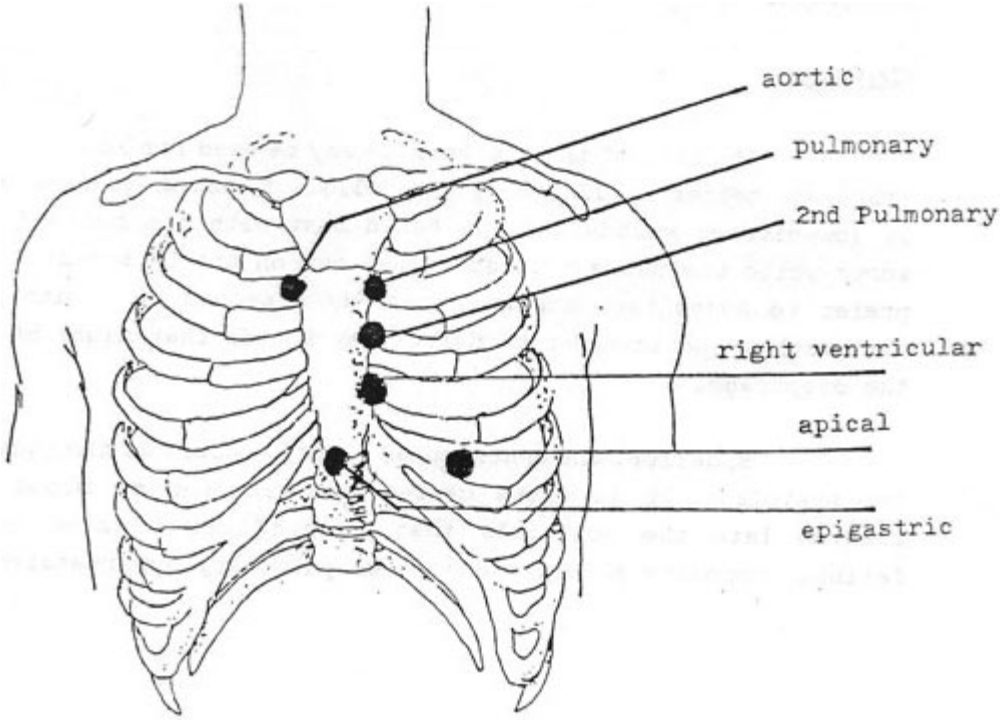
This causes one of the heart sounds to be distinctly “split” or having an “echo” sound. This may be naturally occurring phenomenon, called “physiological splitting” or if may be due to a disease called pathological splitting. When S1 valve closures can be heard separately, there may be a conduction defect present or even a mechanical defect. Of course, even young and healthy children and some adults can still have normal splitting of S1 and S2.

In review, the two common heart sounds are S1 and S2. They each are made up of two distinctly separate sounds of two valves closing in unison. When assessing the patient for heart sounds, the nurse must first obtain a patient history; inspection, palpation, and possibly percussion will also usually be performed before you begin to auscultate.

Ideally the nurse must know that patient’s heart rate and the regularity of rhythm before auscultation is performed. S1 and S2 are two “normal” heart sounds that may sometimes be heard in the cardiac cycle. Splitting is usually a normal situation arising from asynchronistic closure of two valves responsible for each of S1 and S2.



The places on the chest are marked.



The corresponding areas of the heart.

In beginning to auscultate the sounds have the patient lie comfortably on his/her back at about a 45 degree angle. Have them put their hands at their side and then explain what you are going to do. You may have to tell some patients to relax and to breathe normally as anxiety may sometimes make them breathe rapidly and noisily and interfere with your procedure.

First, start at point number one above the aortic area. Then proceed to the pulmonic, 2nd pulmonic, right ventricular, apical, and then epigastria area. Each of these areas allows for the clearest heart sound for that valve it is named for. The aortic region, for example, is the best place to listen to the aortic valve, etc. Even through the valve is not actually located at the precise area.

Heart sounds are generally easy to hear; but sometimes due to the patient and other conditions, it may be difficult to hear clearly. Use the diaphragm of the stethoscope and place it gently on the chest in the areas indicated. The diaphragm will be best for listening to the high-pitched sounds of the S1 so auscultate using the diaphragm at all points. Do not “drag” the stethoscope, as excess noise will be generated by this action. Have the patient breath normally and put them in a prone position. Sometimes the sounds may be better heard in a sitting position. Try both ways if you have difficulty hearing the sounds.

Murmurs

A heart murmur is a very general term used to describe any one of the verity of abnormal sounds heard in the heart due to turbulent or rapid blood flow through the heart, great blood vessels, and/or heart valves (whether the heart valves are normal or are diseased). Most nurses associate murmurs with an abnormal heart valve. However, there are a variety of other conditions that can cause murmurs. Murmurs can also be caused by the forward flow of blood

across a constricted or otherwise irregular valve, or into a dilated heart chamber or dilated vessel. They can also be caused by the backward flow of blood through an incompetent valve or a septal defect murmurs are usually described as a “rushing” or “swooshing” sound. Murmurs are usually related to defect in valves or ventricular septal defect, or atrial septal defect.

When ausculatining murmurs, the nurse should record the timing, characteristics, location, and radiation of the murmur. Characteristics include: loudness, intensity, pitch, and quality of the murmur. These assessment factors are discussed in more detail later in the course.

Gallops:

The bell of the stethoscope may be used for low frequency sounds (they are better amplified by the bell). S3 and S4 gallops are generally low-pitched sounds and are heard best with the bell of the stethoscope while the patient is stretched out on his left side. Many nurses prefer to auscultate the heart sounds a second time with the bell of the stethoscope in order to detect any sounds that might be missed with the diaphragm. S3 gallop, the ventricular gallop, occurs at the end of ventricular systole. It is often caused by the sound of blood prematurely rushing into the ventricle that is stiff or dilated due to heart failure, coronary artery disease, or pulmonary hypertension.

Clicks

Sounds described as “clicks” are extra sounds often heard in those patients with mitral valve prolapsed, aortic stenosis, or those with prosthetic heart valves. Opening “snaps” are usually caused by mitral stenosis or stenosis of the tricuspid valves.

Rubs

Sounds referred to as “rubs” occur when the visceral and parietal layers of the pericardium rub together. The sound is produced when inflammation is present due to uremic pericarditis, myocardial infarction, or other inflammatory condition.

Discussion of Heart Sounds

The loudness and intensity of heart sounds are important when you are listening. S1 and S2 are heard at different levels of loudness, depending upon where you listen on the chest. The loudness of S1 is mainly determined by the position of the heart valves when ventricles contract. If valve leaflets are wide open at the time of contraction, the sound is very loud.

The loudness of the sound is also affected by the pressure of the blood. It is this pressure that “slams” the valves shut and generates the sound. If you recal that the interval between S1and S2 corresponds to the systolic phase, then a murmer that is heard between S1 and S2 wuld be called a systolic murmur. Then a diastolic murmur would be called a murmer heard between S2 and S1, which corresponds to the diastolic phase of the cardiac cycle.

Next, these two murmurs, systolic and diastolic, can further be pinpointed by descriving exactly when in the phase it occurs. The murmur can be described as:

Early systolic	Midsystolic	Late systolic
Early diastolic	Middiastolic	Late diastolic

These above terms describe murmurs in the exact position that they fall I the phase. For example, an early systolic murmur would be “timed” as occurring early in the phase of systole; and so on for all the phases. Another term called holosystolic (also called pansystolic), is used to describe a murmur heard throughout the entire systolic phase (S1 to S2). Similarly, holodiastolic will be used to refer to the murmur heard throughout the entire diastolic phase (S2 to S1).

The timing of the murmur above is very difficult to assess in some patients. In other patients, the timing will be very easy to assess. An important factor is that the nurse has experience in listening to a variety of “normal” variation of normal heart sounds. You must first listen to many different normal heart sounds. Once you have some experience at differentiating normal S1 and S2 sounds, then you will be able to identify abnormal sounds, and to determine the timing of those abnormal sounds.

The valves are sat their widest when blood is actually filling into the ventricle. As the ventricle fills and the atria empty, the leaflets of the valve begin to close or to narrow. At that point, when the atria are empty, the ventricle is contracting, and slams the valve shut. This is the dynamic force behind the loudness and intensity of the heart sounds.

Other factors affect closure. Exercise, fever, anemia, and other factors and affect heart rate and force of the closure of the valves. Loudness, of course, is also affected. Note the following changes due to disease:

1. Mitral Stenosis loud S1 and delayed closing of valve.
2. Heart block varying intensity of S1 and S2 due to incomplete emptying of the atria, leaflets may be partially open at some times, completely open at others.
3. Atrial flutter; fibrillation sounds vary in intensify for the same reason as above.
4. Bundle Branch Block S1 maybe be widely split (two separate sounds make up S1) S2 can also be affected.
5. Normal Respirations physiological splitting of S2
6. Pulmonic Stenosis delayed emptying of right side can cause splitting of S2.
7. Atrial-Spetal Defect causes delayed emptying of right side as in above condition, splitting of S2 will occur.
8. Hypertension increases back pressure on aortic valve, causes increase in loudness of S2.

These are some disease conditions and the resulting change in the heart sounds. The term murmur refers generally to any “extra” or unusual heart sounds. Most nurses will not be expected of fully diagnose all murmurs and/or abnormal heart sounds. However, the nurse should be able to recognize whether or not the two normal sounds are present, and if they are not, what sounds that are present, should be described carefully.

When charting heart sounds from your nurses’ notes, chart only the sounds that are abnormal. Chart basic information such as heart rate, rhythm, intensity and abnormal sounds. Describe carefully their location in the cycle. Describe any coincidental factors that may be influencing the rhythm such as respirations or movement of the patient. For murmurs, chart where it occurs I the cardiac cycle, loudness, pitch, the location of the where it is heard the best, and other locations where it can be heard. Also record the general type of sound heard and if anything makes the sound change in any way. For example, if you reposition the patient, does the sound change. If the patient happened to breathe deeply and the sound changed; all these world be notable events.

Following is a guide to auscultation of sounds of the heart. It is only a guide, and should be used with existing guidelines at your facility. The methods of charting are different at each hospital, so are responsibility levels for each type of nurse. Always use terms which are acceptable at your facility. If a heart sound or murmur is accompanied by adverse clinical symptoms, results should be reported.

Guide to Ausculation of Heart Sounds

Step 1	Prepare patient	Have patent relax, remove clothes from waste up, cover with gown, provide for privacy.
Step 2	Vital signs	Record vital signs, TPR and blood pressure, note any abnormalities.
Step 3	Heart Rate	Listen to apical pulse and record the rate, even though you already took the radial pulse rate from the above step not any abnormal sounds while listening to apex.
Step 4	Rhythm	Determine the regularity of rhythm; regular or irregular? a. Is the irregularity due to respirations? b. Ausculate at the points mentioned previously. c. Listen especially for any murmurs you previously heard. d. Timing-describe where the sound is heard in the cardiac cycle.
Step 5	Sounds	Describe carefully the sound head that is abnormal. Is it a “rubbing sound” or a “clicking sound” a “swooshing” or other? Chart the sound just as you heard it. This course is not designed to make you a cardiologist, but the nurse should be able to recognize and chart anything abnormal in the cardiac cycle.

Ausculation Criteria

Patient Position:

INSERT S1: Supine or lying with upper body slightly elevated.
Use diaphragm of stethoscope.

S2 Supine or lying with upper body slightly elevated.
S2 Use diaphragm of stethoscope.

Ausculation Site(s):

S1: Start at mitral or tricuspid area; loudest at apex (mitral).
S2: Start at mitral or tricuspid area; loudest at base; (higher frequency than S1).

Split Sites:

S1 S1 split best heard at lower left sterna border.
S2 Listen for S2 split in the pulmonic and secondary aortic area. Ask the patient to breathe deeply through the nose to accentuate the split.

Differentiating S1 from S2

S1 occurs with the downstroke of the R wave on the EKG. It also occurs simultaneously with the carotid impulse upstroke.

S2 Follow the T wave on the EKG and occurs as the carotid pulse wave fades.

Loudness of Heart Murmurs

Once you have determined the timing and other gross characteristics of the murmur – you should determine and record the loudness. Loudness is graded on a numerical scale as shown below. Grade I is the softest and Grade VI is the loudest. *Remember, however, that you will see other scales used to measure loudness. Some authorities use a scale of I, II, III, IV. Some others use a scale of only I, II, III; so be aware of the scale that is generally used at your facility and apply the same principles to it.

Grade I	Soft
Grade II	Medium Soft
Grade III	Loud
Grade IV	Medium Loud
Grade V	Louder
Grade VI	Loudest

All of the ratings are very subjective.

Intensity of heart murmurs:

Crescendo begins softly and becomes louder.
Decrescendo begins loudly and becomes softer.
Crescendo/Decrescendo begins softly, peaks at a certain intensity, and then becomes soft again.
Decrescendo/Crescendo begins loudly, becomes softer, and then becomes loud again.
Holosystolic Stays the same intensity through systole and diastole, also called pansystolic murmur.
Holodiastolic The same intensity throughout diastole; pandiastolic murmur.

Further characteristics of murmurs include the quality and the pitch. The quality of a murmur may be described as harsh, blowing, musical, rumbling. The pitch may be described as high or low pitched. Other terms may also be used, such as: dull-sounding, sharp, and others. In many cases, the nurse may simply describe how the murmur sounds to you. Be very descriptive and try to use established terms. However, you should also be precise in location and changes I the sounds that you hear. Do not be afraid to say “it is a swooshing murmur when the patient is lying on left side, than has a “tapping” sound with sitting upright.” Describe exactly what you hear.

Types of Murmurs

The heart murmur associated with mitral stenosis is caused by the flow of blood across the constricted mitral valve during the period of rapid ventricular filing. There are two periods of this rapid ventricular filling in the cardiac cycle. These are: early diastole, shortly after the opening of the atriventricular valves; and immediately prior to the onset of systole just s the atria

contract. This explains why there are two murmurs heart in mitrial stenosis. The first is an early to middiastolic murmur and a presystolic murmur. There is also another distinct sound heard with the condition of mitral stenosis. This sound is referred to as the opening snap of mitral stenosis. Normally, this opening of the mitral valve is silent. In the presence of mitral stenosis, there is a sharp, high-pitched click that can be ausculated. The click is best heard between the apex and the lower left sterna border.

The murmur of aortic insufficiency is caused by the backward flow of blood from the aorta into the left ventricle across an aortic valve that is incompetent. This backward pressure exerted by the blood in the aorta is the greatest, just after the closure of the aortic valve and progressively fall thereafter thought diastole. The sound produced is described as regurgitation. The murmur usually beings immediately after S2 and can progress throughout the entire diastole. This murmur is best heard at the base of the heart and will often radiate to the apex. The murmur will usually be very high pitched and will usually have a “blowing” quality; although at many times has a very harsh quality to the sound and may be very loud.

The third heart sound S3 was discussed earlier as being normal in some adults an in children. In the case of a pathological S3, it may be noted with ether vent of damage to the myocardium. This heart sound, when ausculated, sounds like the gallop of a horse. Indeed, it is described as a gallop. The S3 creates and extra heart sound that can be rapid and very distinctive. The S3 G (S3 Gallop) is caused by early diastolic vibrations that are probably the result of ventricular dispensability associated with the myocardial damage.

The S3 G can be best heard at the apex as a short, low-pitched sound. It may possibly be palpated, since it is associated with the ventricle and can create a “thrust” from the heart. A gallop rhythm is very suggestive of myocardial damage, as stated, and the possible presence of early congestive heart failure.

Cardiac Arrhythmias:

An arrhythmia is described as an abnormal heartbeat. An arrhythmia might be caused by one or more of several different factors:

a. SA node – variation of the rate of discharge of the node.
b. Conduction defect – an abnormality of the conduction of the electrical impulses from the sinoatrial node through the heart.
c. Myocardial irritability – an initiation of the contraction of the heart muscle by an impulse on a random area of the heart, other than the SA node.

The “average” person’s heart rate is usually 70 – 100 beats per minute (BPM). However, we all know that some persons may have a normal heart rate that is slightly higher or lower then these normal ranges. Of course metabolism, exercise, and other factors will affect a person’s normal heart rate. A rapid heart rate is called tachycardia, and a slower than normal rate is called Bradycardia.

When assessing a patient’s heart rate, you must consider that the heart rate is also controlled by the sympathetic and parasympathetic nervous systems. The sympathetic nervous innervations increases the rate at which the SA node fires. The parasympathetic nervous innervation decreases the rate of firing and subsequently the heart rate and produce a normal heart rate. However, in disease conditions, either or both of these nerves may dominate and produce a fast or a slowed heart rate.

The normal heart beat is also referred to as sinus rhythm, or being initiated in the sinoatrial nods. Very specifically, if the rate is normal and the heart beat is a sinus rhythm, it is called "normal sinus rhythm." Using this terminology, it would be easy to see why a rapid but otherwise normal (sinus) rhythm, is called sinus tachycardia. Conversely, a slow, but otherwise normal (sinus) rhythm is called sinus Bradycardia. Both of these arrhythmias are not normal, but they are usually not severe or life-threatening. The nurse might easily understand that sinus tachycardia could be caused as a normal reaction to anxiety. In fact, just being sick and in the hospital might produce anxiety and also sinus tachycardia. The nurse would need to assess the patient very carefully if a rapid heart rate is present; especially if there are no adverse clinical signs or symptoms present. Of course, an EKG tracing and interpretation would be necessary to absolutely confirm that only sinus tachycardia is present, and that there is now immediate danger to the patient.

Factors that affect the sympathetic nervous system are: anxiety, fear, fever, extreme physical exercise, other. These conditions will generally cause an increase in the sympathetic stimulation of the SA node and subsequently, tachycardia.

Factors that affect the parasympathetic nervous system will induce a slowed heart rate. The parasympathetic nervous system exerts its influence through the vagus nerve. This stimulation of the vagus nerve will cause Bradycardia.

In summary, prepare the patient; take vital signs and history of the patient before ausculating the heart. Compare findings to normal heart sounds. Then, report findings, especially if the patient has adverse symptoms, such as cyanosis or some other important symptoms.

Part III Recording the Physical Assessment Findings

As an introduction to charting, it should be known that there are many different ways to record an assessment. Some hospitals have their own form for recoding findings, and other facilities, a narrative or "story" form. This guide for charting will present one method. If your facility uses a different method of charting, you may still derive some benefit from this exercise. You can study terminology and the presentation, and then apply it to your facility. Even if your facility uses a "checklist" style charting, you still may have to have to record certain observations that do not exactly fit those checklists. Therefore, remember to observe and carefully describe and record your findings for each patient.

Narrative Style:

Begin with:

Vital signs, radial pulse, BP, temperature, respirations, and history. "Patient is a 78 year old mail, in no acute distress, reports a "heart attack" 5 years ago and has been in good health since then; came into the ER today feeling weak, dizzy, and pounding in the chest."

Next the general medical exam:

Patient is alert, oriented, no respiratory difficulty, no complaints of pain now, skin turgor good, skin color good, skin is warm and dry, no problems voiding, and no bowel

movement for 2 days. Takes Dioxin and Lasix QD, dosage unknown, lungs sound slightly congested but no dyspnea, as stated above.

Cardiovascular:

Peripheral pulses all present and strong, neck veins slightly distended when laying down, heart rate regular and strong, thorax normal shape, no masses, no tenderness, heart sounds clear and strong, with faint murmur between INSERT S1 and S_2, sounds like clicking noise, MD was notified, no treatment because patient has had condition for many years.

This is a sample of a fairly healthy patient. Some facilities might want the cardiovascular system charted first in the nurse's notes section. Others will want <u>all</u> cardiovascular findings together in one place o the chart. In the above example, we placed skin color together with the other skin findings. Skin color could be considered a cardiovascular sign. It does not matter where you put it; just remember to include all pertinent findings. How do you know what is pertinent? That is a difficult question, but always remember to include all findings that you would expect to be abnormal if the patient did have a definite cardiovascular problem.

Assessments such as skin color, respiratory difficulty, poor pulses, poor heart sounds, and low BP, etc. this is why it is important to have the history and the general medical exam reviewed by the nurse before you concentrate on your cardiovascular exam. Once you know general findings, it is easier to review the cardiovascular system.

As you finish recording your findings remember to include all actions that you took for your patient. If you start your exam and the patient was having a severe asthma attack, you would say, "wait," I have to do my cardiovascular assessment first. You would take the appropriate emergency measures first. Remember to chart all such treatments for emergency measures. Legally, you might be held responsible, even if you did take appropriate measures, if you did not chart "notified MD" you could be responsible for some adverse occurrence.

Charting is a method of recoding that you <u>did</u> take the appropriate action for the situation; "notified MD and no treatment at this time." this charting protects the patient, and protects the nurse. It lets everyone know that you performed the correction action in response to your abnormal findings. If you are ever in doubt as to how you should chart something; remember to be as objective as possible. Chart the findings (be descriptive), and then chart what you did about it. That is how good charting protects you and the patient.

Part IV Cardiovascular Drugs Update

We are including this section, because it is vitally important that all nurses be up-to-date on the cardiovascular drugs. When assessing the heart, the nurse must take into consideration the effects of drugs upon the cardiovascular system. This section will be concerned with the most common drugs used in a cardiac arrest situation. Each drug will be presented along with its uses and other helpful information.

During a code, or cardiac arrest, it is not unusual for even the most experienced nurse to have fears concerning the administration of these potent drugs. Since this is a life and death situation, the nurse must learn not to be overwhelmed by the numbers of new drugs being used today. Be sure to study the drugs before the code and not during. Some of these drugs will be

new to you and others have been around for a long time. It wouldn't hurt to study these drugs, see if they have any new uses of which you are not aware.

Lidocaine HCL	Dopamine HCL	Sodium Bicarbonate
Isoproterenol HCL	Procainamide HCL	Epinephrine HCL
Calcium Chloride	Atropine Sulfate	Verapamil (Isoptin)
Bretylium Tosylate	Dobutamine HCL	

1. Sodium Bicarbonate: corrects metabolic acidosis during a cardiac arrest. It is administered by IV push in a dose of 50ml, D5W solution, 44.6mEg of sodium bicarbonate. Metabolic acidosis occurs after the heart stops, due to a buildup of the acid waste materials in the body. This condition will be corrected by regularly administering (approx every 10 minutes) the sodium bicarbonate.

   Defibrillation will be more affective if the body pH is adjusted, so will other drugs be more affective. Arterial blood gas results will tell you the patient's pH and if it needs correcting. Do not administer too much sodium bicarbonate, as alkalosis may occur. If alkalosis does occur, the patient can develop arrhythmias and other problems.

2. Lidocaine: is used for reducing the irritability of the heart muscle. Specifically it treats the PVC's and other ventricular arrhythmias. The drug is usually administered intravenously, but in an emergency it can be delivered via ET tube, intratracheally. The usual dose is 50 to 100 mgm IV push, followed by a continuous IV drip. The continuous drip helps to overcome reappearance of the arrhythmia.

   It is usually mixed in the following ratio: Lidocaine 2Gms/50ml of D5W, delivers 4mg per ml 60 gtts/min gives 4mg/min

3. Epinephrine: is a potent stimulant. It increases the contractility of the myocardium and stimulates spontaneous contraction. It is administered IV push or via the ET tube when necessary. The dosage is 0.5mg to 1.0 mg (5 to 10 ml) of a 1:10,000 solution. This drug in the smaller dose above can be given intracardiac as well. This drug will also in certain conditions, make the myocardium susceptible to defibrillation.

4. Dopamine: is used for the treatment of insufficient cardiac output and for hypotension. Dopamine is often preferable during codes because it also acts as a vasodilator, brining better circulation to the brain, myocardium and the kidneys. The main action of this drug is due to its alpha receipt stimulation effect. This increases blood pressure and cardiac output. The usual dosage is 2 to 5 mcg/kg/min given in a continuous IV drip. The dosage can be as high as 50 mcg/kg/min. Dopamine is mixed: 400mg (2x 200mg vials) in 500ml of D5W. This results in 800 mcg/ml. 15gtts/min of this solution would result in 200 mcg/min. The blood pressure should be monitored closely, every 5 to 10 minutes at the beginning of the infusion and while the dosage is being adjusted. Once stabilized, the blood pressure need be taken only as often as clinical signs warrant, approximately every half hour or one hour. Urine output should also be monitored; dopamine will cause greater kidney perfusion, but output will still need to be monitored for persistent renal failure.

5. Atropine: is a cardiac stimulant. It is used for severe cases of Bradycardia. The drug is administered by IV push, slowly. The action is that of blocking the vagus nerve. The smallest dose to give is 0.5 mg, if a smaller dose is given it may have the opposite effect

and cause further slowing of the heart. The dose is then repeated every 5 minutes for up to 4 doses (2.0mg). Atrpine will usually not be given if the patient has had an acute MI. With atropine the heart rate is increased which causes increased oxygen demands upon the myocardium. MI patients usually cannot tolerate added oxygen demands.

6. Isoproterenol: Isuprel is used to combat sinus Bradycardia; infused in a drip solution containing 2mg (2-1mg ampoules) in 500ml of D5W which gives dilution of 4mcg/ml. dosage range is 0.5 mcg/min to 5mcg/min. and then the patient is titrated as the patient improves. Very similar to atropine, the oxygen demands must be considered when using Isuprel.
7. Procainamide: is usually administered IV push at a dosage of 100 mgm pushed at a rate of 20mg/min so as not to be given too rapidly the drug is used to stop PVC's and is the second choice if Lidocaine fails. The above dosage can be repeated every 5 minutes, up to a dose of 1 gram. If adverse effects occur, such as hypotension or widening of the QRS complex, then the drug should be stopped immediately.

8. Calcium Chloride: this drug is administered IV push at a dose of 5 to 10 m. at 1ml/min. It is used to stimulate the heart to make a more forceful contraction. In cases of asystole, the drug can be used to start spontaneous contractions. The drug can also be used in cases of electromechanical dissociation, a condition in which electrical impulses are being produced by the heart, but the heart does not respond to them. On EKG, it seems like the heart s should be beating just fine. However, the patient has not effective contractions. Calcium Chloride can be given in order to make the heart respond effectively.

9. Verapamil: is one of the newer drugs which are in common use today. It is a calcium channel blocker (also classified as slow channel blacking agents or calcium antagonists). Verapamil and others in this group are used for slowing conduction of the heart's electrical impulses and treating such arrhythmias as atrial flutter, atrial fibrillation, atrial tachycardia and for supraventricular tachycardia.

   The drug is also used for angina and the group of drugs being investigated for treating other disorders; such as hypertension and cardiomyopathy.

   The drugs in this class have many different and varied chemical structures but they all perform the same function they inhibit calcium flux across the cell membrane the drugs do not seem to affect sodium flux or any other electrolyte in eh cells. Calcium, as you k now, is important for the contraction of all muscle cells, especially cardiac cells. Some cells are more dependent upon calcium than other cells. They do not all utilize it equally. Therefore, cardiac muscle cells react differently to each of the drugs in this class. There are many different effects which can occur because of this action; it depends upon:

   1. Which cardiac cells are affected by the slowing
   2. The chemical structure of the particular blocker drug being used
   3. The dosage
   4. The route of administration
   5. The extent to which that particular cardiac cell depends upon calcium

   Verapamil and some of the others in the class affect the SA node and the AV node. It depresses the SA node and also slows conduction through the AV node. The overall effects of Verapamil are:

1. Negative chronotropic effect (slowed SA rate)
2. Negative dromotropic effect (slowed AV conduction)
3. Prolonged PR interval on EKG
4. Decreased myocardial contractility

Another effect of this group is usually coronary artery dilation. The blood flow through these arteries is increased by relaxing the smooth muscle of the vessels. Therefore, the drug can be useful for treating angina. In addition, myocardial oxygen consumption is reduced because both the preload and after load are decreased. Of course, just the fact that the heart rate is reduced, however the work load on the heart.

The side effects of these drugs can be severe. They are related to the way in which the drubs work. The drugs will have a systemic effect on the body. Peripheral circulation is also affected, resistance is decreased and peripheral blood flow is increased.

Possible side effects are:

More serious:
1. Hypotension
2. Bradycardia
3. AV heart block
4. Dizziness
5. Headache

Less serious:
1. Dysestbesias
2. Constipation
3. Pedal edema
4. Vertigo
5. Flushing

Dosage of Verapamil: 60 to 80 mg PO Q8 hours

75 to 150 mcg/kg IV…or continuous infusion at: 0.005 mg/kg/minute

10. Nifedipine: (Procardia) is another calcium blocker drug. Actions and side effects are very similar to Verapamil.

    Dosage: 10 to 30 mg PO Q4 to Q8 hours; 10mg SL

    Caution must be observed when administering this drug. If given to patients with refractory angina pectoris, the “coronary steal syndrome” may be set off. This syndrome has been observed in patients taking this drug and also taking combinations of nitrates and beta blockers at the same time. The syndrome is characterized by multiple episodes of chest pain, about 30 minutes after taking a dose of nifedipine. It is used when the combination of drugs reduces the coronary perfusion pressure too greatly. This can cause blood to be diverted into the extremely dilated systemic arterioles. By slightly reducing the dosage of the drug, less dilation will occur and the syndrome is usually relieved. Symptoms of the syndrome could include, (in addition to chest pain), hypotension and reversible myocardial ischemia.

11. Diltiazem: (Cardizem) is another calcium channel blocker.

    Dosage: 60 to 90 mg PO Q8 hours
    75 to 150 mcg/kg IV

    Note: each calcium antagonist drug has its own particular uses.

    Each MD will use one o the drugs as they have had success with treatment. In the near future you will see many more uses for the drugs, as research progresses. Before you administer any of the drugs be sure to read the literature concerning that particular drug, including its side effects.

12. Bretylium: is also one of the new drugs in common use today. It works directly on the heart to slow the refractory period allowing the heart to have a longer recovery period between beats. This drug is used for some of the life-threatening arrhythmias in which there is no response to Lidocaine. Some physicians prefer Bretylium of Lidocaine for its effect upon the heart.

    Dosage: 1 to 8 mg/kg per minute IV (push or infusion)
    (given IV push slowly, 1 or 2 mg/kg/minute)

    It is used to control severe ventricular arrhythmias such as ventricular tachycardia and or ventricular fibrillation. Bretylium is supplied in ampoules of 10ml which contain 500 mg and can be sued for IM or IV injection.

13. Bobutamine: is similar to dopamine in that they both increase contractility of the myocardium. It can be used to treat hypotension and/or shock. Dobutamine (Dobutrex) works directly on the heart muscle to increase cardiac output, whereas dopamine works indirectly via the kidneys. Dobutrex, thereby, does a better job increasing the cardiac output; but dopamine treats hypotension better.

    Dosage: 2.5 to 10 mcg/kg/min is administered by infusion only and must be reconstituted from a powder just before use.

Summary

Arrhythmia	Drug, in order of use
1. Sinus Bradycardia	1. Atropine
2. Complete heart block	1. Atropine 2. Isoproterenol
3. PVC's	1. Lidocaine 2. Procainamide 3. Bretylium
4. Ventricular Tachycardia	1. Lidocaine 2. Procainamide 3. Bretylium
5. Ventricular Fibrillation	1. Lidocaine 2. Procainamide 3. Bretylium (sodium bicarbonate may be

	administered concurrently other drugs may be used to treat underlying arrhythmias)
6. Ventricular Asystole	1. Epinephrine 2. Calcium chloride 3. Atropine (sodium bicard and other drugs may also be used)

Presented here are the most basic drugs used today in code or emergency cardiac situation. This list will vary with each hospital and each physician. However, once you as the nurse becomes familiar with these drugs, it will be easier for you to respond to that tense situation. During a code, try to anticipate the next drug that will be given. The nurse giving drugs during a code should have several bottles of D5W ready to be missed with infusion drips if necessary.

Be aware that there are also pre-mixed infusion available today with the more common infusions such as dopamine and Lidocaine and others. If your hospital used these pre-mixed solutions have them handy for use. Also ask the MD how often the sodium bicarbonate is to be administered during the code. Some doctors will want to be reminded every 5 to 10 minutes so he/she can evaluation the patient for the need of the "bicarb" and MD's will say to give it automatically every 5 to 15 minutes during the code.

Make sure that you know the protocol at your facility for administering the various drugs. For instance, the proper strength for missing drugs and infusing them, not to mention the correct route.

Remember that some prefer to use Bretylium before Lidocaine, however more MD's will try Lidocaine first.

Other Cardiovascular Drug Updates

1. Vascor, Bepridil, a new calcium channel blocker is up for approval by the FDA. It will be marketed by McNeil Pharmaceuticals as a treatment for chronic stable angina pectoris. It has a much longer effect than all other calcium blockers so far. The patient will take the drug only once a day in 300mg to 400mg doses. So far, studies show that the drug significantly reduces the frequency of angina attacks and the consumption of nitroglycerin tablets. The side effects are nausea, dyspepsia, diarrhea, dizziness and nervousness, not unlike the side effects of many of the other calcium blockers. The company also reports that this drug may be safely used in combination with other drugs commonly used by these angina patients (AJN, 91).

2. Rare reaction to Verapamil One patient using the calcium channel blocker, Verapamil, (Calan, Isoptin), developed myoclonic dystopia from the drug. The symptoms were uncontrolled, irregular, symmetrical jerking movements of the arms and legs with accompanying twisting movements of the trunk (AIM). The patient was also taking nitroglycerin, a diuretic and a potassium supplement drug at the same time. It is unknown the exact cause for the problem but another calcium blocker as substituted for the Verapamil and the problem stopped.

3. Norpace-Induced liver damage Norpace is commonly used antiarrhythmic agent. It is used for ventricular arrhythmias such as PVC's. The normal side effects and include antcholinergic reactions such as dry mouth, blurred vision, urinary retention and also

constipation. Some patients experience sever hypotension and congestive heart failure. Liver enzyme abnormalities have been reported and even Norpace-induced cholestatic jaundice. Recently, a case of direct hepatocellular toxicity has been reported. It is the first case ever reported. The treatment was to withdraw the medication.

4. Long-term Amidarone therapy – amiodarone is a very new antiarryhthmic and antianginal drug. The drug contains 75 mg of iodine per 200mg tablet and recent studies have shown it can cause thyroid dysfunctions. These include hyperthyroidism (most commonly), goiter or hypothyroidism. The extent of the problem depends upon the "normal" intake of iodine by the person from the environment (foods). Persons who already have sufficient iodine intake would be prone to develop hyperthyroidism faster and more severely. The problem is that the drug causes T4 levels to increase and causes T3 levels to decrease. Patients on short-term therapy seem to be affected the most. The hormone levels seem to g back to normal in about 3 months, even if ht patient continues the drug. More studies are being done on this new drug to determine why there is such a fluctuation in the hormone levels.

5. Levarterenol (Levophed) this drug is also called Norepinephrine, a naturally occurring catecholamine. It is used really today because there are several other drugs which are preferred by most physicians. However, in some areas of the country, this drug is still used quite extensively. It is a potent peripheral vasoconstrictor. An alpha-receptor stimulating agent, it results in an increase in the blood pressure. The drug is also a powerful beta-stimulating agent which works mainly upon blood vessels. It also causes coronary vasoodilatoin. Bevophed is also used in peripheral vascular collapse, manifested by hypotension. However, this drug is used only in the absence of significant peripheral vasoconstriction. Levophed works will in hypotension. But it will also cause renal and mesenteric vasoconstrtion. This is why a drug such as dopamine usually is preferable over levophed.

   For the use of this drug, see the directions packed with the drug. It usually supplied in ampoules of 4ml of a 0.2% solution. Each ampoule contains 8.0 mg of Levophed. It is usually mixed by adding two ampoules in a liter of D5W. This produces a concentration of 16mg/L, or 16 uGu/ml.

6. Verapamil (Calan, Isoptin) another calcium antagonist, is used for flowing arrhythmias such as atrial flutter or fibrillation or supraventricular tachycardia. Recently, it has been used for angina.

   Nursing Implications:
   a. Dose: 60 to 80 mg PO Q8 hours OR 75 to 150 mcg/kg IV
   b. Possible headache, hypotension, AV block, constipation
   c. Can also be given as a continuous IV drip at 0.005 mg/kg/minute

Verapamil Update: Verapamil Decreasing Efficacy

A 68 year old man suddenly stopped responding to his ordinary doses of the calcium channel blocker Verapamil. For two years he successfully used oral Verapamil, 240 mg/day, to prevent the episodes of the supraventricular tachycardia.

Then he was hospitalized with fever and leucopenia. Intravenous gentamicin and carbenicillin were started; he continued taking the oral Verapamil. On the first hospital day, supraventricular

tachycardia developed, and reverted to sinus rhythm only after injection of 2.5 mg of Verapamil. His oral Verapamil dosage was increased to 360 mg/day, but tachyarrhythmia episodes recurred. On the second and third hospital days, 5mg and 10mg of IV Verapamil, respectively, were needed to reverse the arrhythmia.

By the fourth hospital day, arrhythmia could not be controlled even by 30 mg of IV Verapamil; infused over one hour, followed by 0.5 mg of dioxin. Finally, reversion to the sinus rhythm was achieved with the IV administration of the antiarrhythmic, amiodarone, cardarone, 150 mg. The man was placed on a regimen of oral amiodarone, 400mg/day and his heart remained in sinus rhythm during a one-month follow up (AJN, 86).

When a drug or hormone is administered repeatedly, resistance to its effects can build up gradually. The phenomenon is known variously as tachyphylaxis, refractoriesness, desensitionzation and tolerance.

Pediatric Doses

1. Sodium bicarbonate: 1ml (1mEq) kg; repeat dose after pE and base deficit determinations' are made.
2. Epinephrine: 0.1 ml/kg of 1:10,000 solution, maximum 0.1 ml to 1:10,000 solution – 0.01mg or 10 ug.
3. Atropine: 0.01 – 0.02 mg/kg IV
4. Lidocaine: infants: 0.5 mg/kg, children: 5.0mg or approx 1 mg/kg may be repeated as needed.
5. Calcium Chloride: IV infusion: minimal dose of 1ml/5kg (20mg/kg) intracardiac: 1 ml/5kg diluted 1:1 with saline.

Part V: Cardiovascular Assessment in Specific Disease Conditions

Chest Tubes

Assessment of patients with chest tubes and/or underwater drainage systems is extremely important. The principle of this type of drainage is simple. The end of the tube from the thoracic cavity is placed below the level of the water in the closed bottle. The water prevents air from entering the thorax, yet allows for drainage of the pleural space. Remember the dynamics of breathing; pressure is increased during expiration and pressure is reduced during inspiration. See the following illustrations if you still have questions regarding the principles of a closed drainage system.

Assessment of the Patient

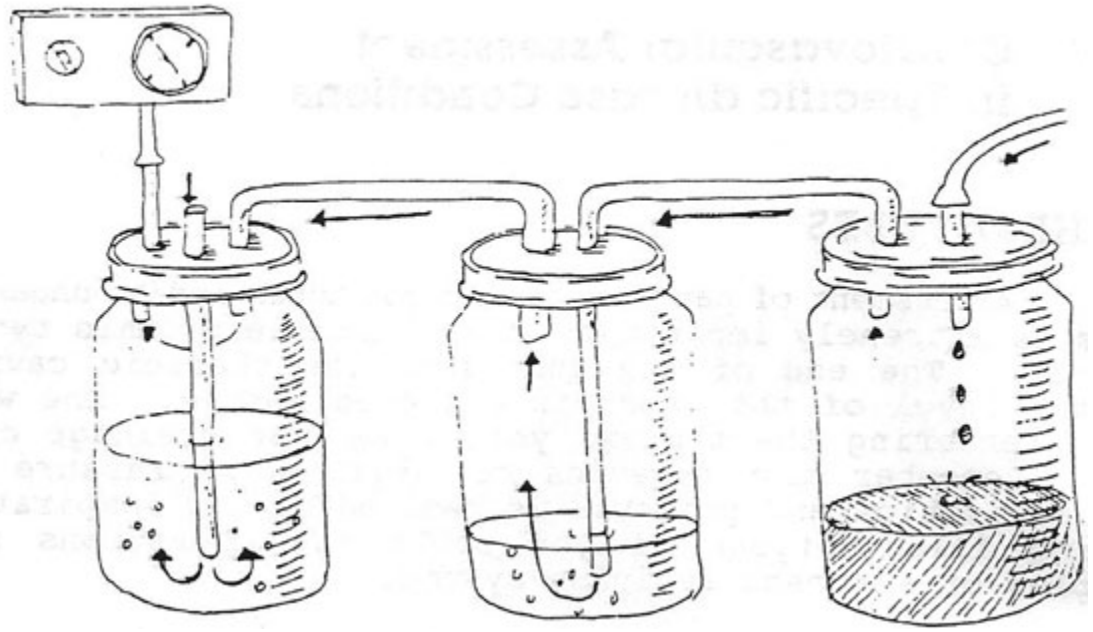
A patient will usually need chest tubes after any type of surgery that enters the thorax, or for treatment of atelectasis, etc. We will try to confine our discussion to the cardiovascular system, but the lungs must also be assessed carefully.

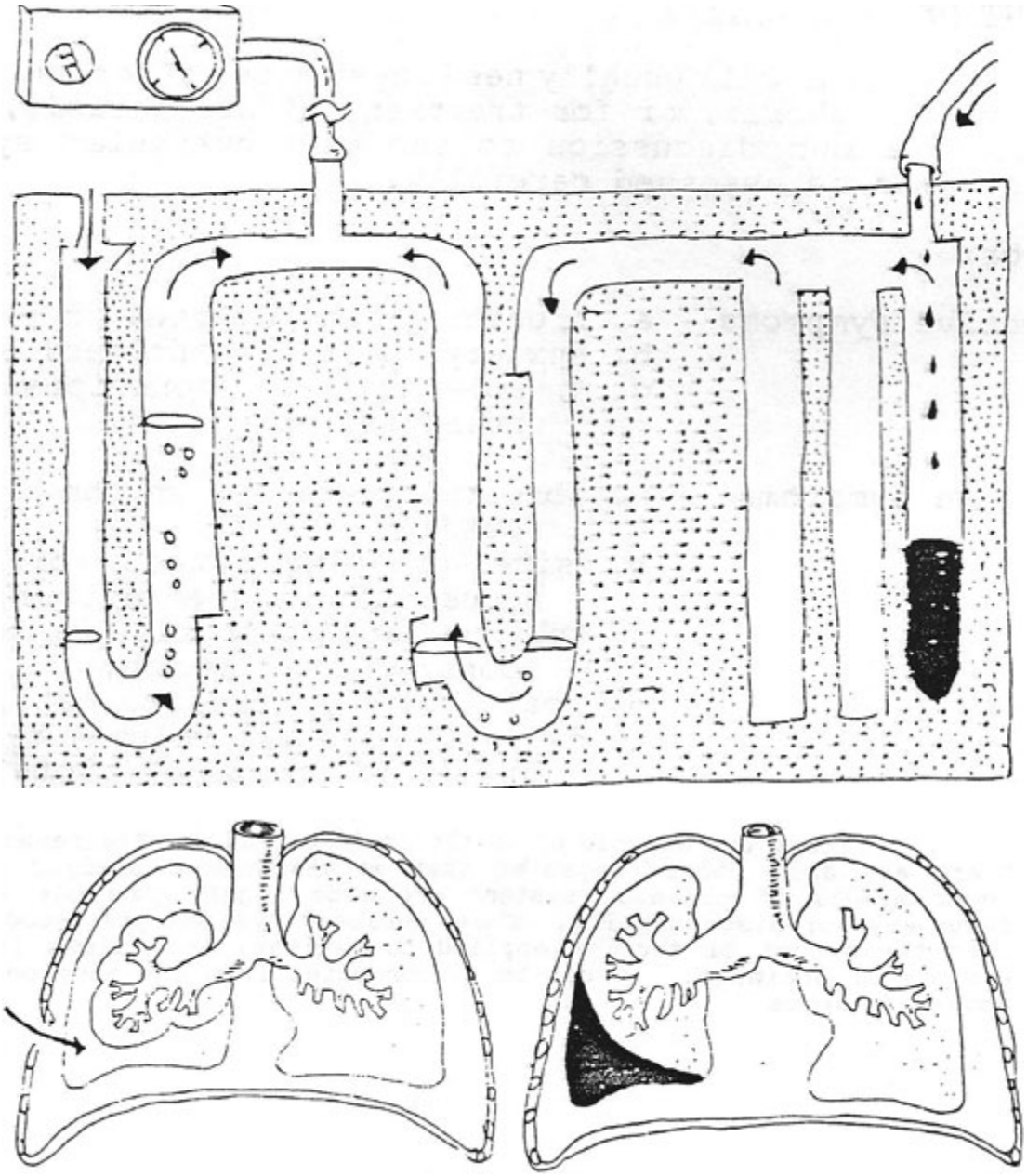
Assess for:

1. Subjective symptoms
    a. Breathing, any dyspnea or pain
    b. Anxiety does patient feel uncomfortable
    c. Neuro-level of consciousness, level of understanding

2. Objective symptoms
    a. Breathing rate, rhythm, depth, breath sounds.
    b. Site dressing intact, drainage, subcutaneous emphysema (crepitus)
    c. Tubing taped properly, kinks, no dependent loops, check suction
    d. Heart sounds regular, rate easily heard
    e. Drainage measure volume, type, color, note any solid drainage (clots)
    f. Suction set a proper level, bubbling gently and continuously.
    g. Other assess entire cardiovascular system, skin color, pulses.

Study the example of bottle drainage and the Pleurevac system. There are also other companies that manufacture a similar product. Newer models of drainage systems are made highly portable and with fewer ways of disconnecting. These enclosed systems give good control over the amount of suction applied to patient, and allows for large amounts of drainage. It can be disconnected from the suction with no adverse effects.





Peripheral Vascular Disease

When we discuss peripheral vascular diseases, we will limit the topics to the most common ones. Many of the diseases of the vessels in the extremities will not be observed by most nurses, as these patients are usually treated in the doctor's office and rarely reach the hospital.

Peripheral vascular problems can be divided into two main sections dealing with arterial problems and then venous problems. As you know, peripheral vascular disease is defined clinically as ischemia to a part due to decreased circulation. In this section we will concentrate on assessment concerning peripheral vascular disorders dealing with both the arterial system and the venous system. Below are some criteria to use for assessing these conditions. Keep in mind that these criteria below are primarily for the acute disease conditions. Chronic occlusive disorders will tend to cause chronic problems with pain and sensory and/or motor systems.

Chronic disorders are usually asymptomatic at rest, and then symptoms appear or get worse on exertion.

Arterial Vessel Assessment:

Begin by performing the routine assessment that you would for any patient. Begin with vital signs, routine pulse determinations, including pedal pulses, and then proceed to the more specific assessment below (Sexton 85, Taggert 85)

a. Pain

   the most prevalent sign of acute arterial problems is pain. Question patient as to the type, location, severity of pain. In chronic cases, pain will get worse upon exertion; symptoms might be intermittent in nature. Pain is usually cramping in nature, but gait is usually not affected.

b. Skin

   color is usually pale and the skin would be cold in acute conditions of ischemia. Nail blanching response in distal bed will be poor. Assess or atrophic changed in the skin; thickened nails; hair loss. Often in arterial disease, the leg can have rubor, a blue-red discoloration when the leg is in a dependent position. Elevate leg 12 inches above heart for 30 seconds, assess for pallor of toes, sole, heel, or leg.

c. Sensory

   test sensory function by touch, pressure, and/or nail blanching. Assess the amount of loss of sensation, if any. Assess for numbness or tingling which will probably be present in acute disease. Numbness is prevalent also in chronic disease, and gets worse on exercise.

d. Motor

   inability to move extremity can be a serous complication. Remember that ischemic conditions can progress rapidly.

Venous Disease Assessment

Peripheral venous problems usually develop from increased venous pressure. These conditions may include: valve damage from inflammation or stretching, dilation from defective vein walls, thrombus formation secondary to endothelial lining damage, venous stasis, or hypercoagulability. The symptoms of peripheral venous vascular disease will usually also correspond to the extent of the damage of the vessels. Also remember that today there are many sophisticated ways of diagnosing peripheral vascular disease. However, they still do not replace a through hands on assessment from the nurse.

Acute disease assessment:

Acute peripheral venous disease is usually associated with conditions such as immobility, dehydration, blood dyscrasias, and malignancies. Acute phlebitis and acute thrombophlebitis are the most common problems. One of the major indicators of peripheral venous conditions is

edema. Increased hydrostatic pressure within a vein can cause a fluid shift into the interstitial space, edema is the result. Assessment should include recording any edema and the amount present. First, you inspect both legs for symmetry in color, temperature, and size. In some cases, you may need to measure the exact diameter of the leg at various points in order to detect if the problem might be getting worse. Measure the leg at several different points with the same tape measure and at the same points every day. You may have to mark the exact locations on the leg to be sure that you are measuring the same place every day.

Continue your assessment of the patient by gently palpating the legs for nodules, lumps, or inflamed veins. Assess for Homan's sign as well as for general feeling of malaise, fever, or fatigue which are often present with inflamed veins.

Chronic disease assessment:

The symptoms seen with chronic peripheral venous disease are similar to those of the acute type. Those with chronic disorders however, will tend to have other medical problems and tend to have both legs involved. Some symptoms might be: chronic pain and edema, cramping, fatigue in legs after standing or sitting for short periods, there is often more discomfort at the end of the day. Also assess for feelings of burning and itching of the legs that usually is due to a buildup of catabolic wastes. This might also lead to an exzematoid dermatitis. Their legs feel heavy and tight. Assess also for skin ulcers, pigmentation, and trophic changes.

In Summary:

It is sometimes very difficult to differentiate between peripheral arterial and venous conditions. When there are many systemic and chronic medical problems present, along with edema and/or fatigue in the extremity, chances are the problem is venous. When the symptoms include numbness, tingling and/or sensory and/or motor changes, the indications might indicate an arterial problem. Whichever is the case; the nurse must still assess the patient very carefully and keep in mind the immediate nursing measures that should be taken.

Hypertension and Shock

Hypertension and shock are two additional disorders which will be discussed in this section. You might even say that these disorders are not precisely named as peripheral vascular diseases, but for our purposes, will be considered under this heading. They are very common disorders and often not assess properly.

Hypertension: in review, there are two basic types of hypertension, primary and secondary. Primary or essential hypertension is idiopathic. Secondary hypertension may or may not have a treatable cause. In secondary type, there is a medical problem causing the symptom of hypertension. When assessing these patients, keep in mind the pathologies that server hypertonic cause to other body systems such as kidneys, brain, liver, and others.

Assessment in Hypertension:

1. Cardiovascular: apical pulse, listen for any murmurs heart rate and rhythm, assess pulses and blanching in extremities.
2. Pulmonary: assess thorax, lung sounds for any congestion.
3. Renal (GU): Assess urinary output hourly in sever hypertension, specific gravity.

4. Neurological: assess for level of understanding, level of consciousness, any signs of neuro degeneration.

Perform your complete assessment for these patients, but particularly these items mentioned above. Nursing care for patients may include drug therapy, rest, comfort measure, sedation when necessary, and discharge planning and teaching. The patient must make changes in lifestyle and in many instances and needs the support of the nursing staff and family. Today patients are not in the hospital for very long, the nurse much being discharge planning and teaching immediately upon admission. The nurses' assessment of the family situation should include interviews with the family and to include them in the teaching of the patient.

Assessment in Cardiovascular collapse: SHOCK:

This section will deal with assessing the patient at risk for developing the symptoms of sock. If a patient is in shock, he is seriously ill. We will present the evaluation of the cardiovascular signs of impending shock, so that the nurse can hopefully prevent this life-threatening disorder. One of the most important factors for the prevention of shock is an adequate baseline of information. We have discussed throughout this test, the importance of history and general assessment. We much always have a baseline of information in order to asses shock in the patient. We must know the person's "average" blood pressure and TPR. We must know the person's "average" response to disease.

This means, do they respond appropriately to questions/ do they have "normal" intelligence? Do they have "normal" skin color, (normal for them), etc.? once we determine a baseline, the nurse can very easily assess the cardiovascular system for changes. the nurse will then be able to assess the patient for deterioration of the body systems.

Guide to Assessment of Shock

The following guide will include assessment for the prevention of shock, and it will contain guidelines for assessing the patient already in shock. This nurse should be able to assess both situations. Shock is defined as "cardiovascular collapse." There are many different types of shock, originating from many conditions. For example, allergy can lead to shock, anaphylaxis. Hemorrhage can cause shock, due to blood loss, hypovolemia shock. Whatever the condition, shock is a disease by itself, and it can be also be considered as a symptom that there is another condition present. The initial problem leads to shock, then, like a snowball downhill, gathers momentum and gets worse.

Cardiovascular collapse means that the body's response to the primary condition, leads to loss of tone of the entire cardiovascular system. The heart action weakens, arteries lose elasticity, blood pressure falls, pulse increased and weakens, there is mental confusion, changes I level of consciousness (LOC), and many other possible related symptoms.

Clinical Assessment of Shock:

As shock develops in a person, many different and varied symptoms can be seen. Some types actually cause symptoms which are confusion, because they do not follow the usual patterns of shock. In this text, we will concentrate on "what to look for" if your patient begins to develop shock or who is at risk of developing shock.

Stages of Shock:

State I: Baseline Shock – this is beginning shock, the patient "baseline" must be obtained and compared to the next stage of shock; the patient in this stage is essentially stable.

State II: Early Shock – compared to baseline, the patient's blood pressure is going down, and perfusion to the vital organs decreases.

State III: Late Shock – complete collapse and poor perfusion to vital organs, next to death.

Again, this is only a guide. Some authorities call early shock "compensated shock" due to the fact that the patient has only early symptoms and can still easily be treated. Late shock is also called by some "decompensated shock" due to the fact that it is so severe that it usually cannot be reversed, except by extraordinary means.

Circulatory Effects of Shock

Most forms of shock cause an immediate decline of perfusion to the vital organs. Peripheral vessels are constricted and therefore flow is decreased. Some types of shock may be slightly different as to pattern of blood flow, but most types follow this pattern of declining peripheral blood flow.

Due to peripheral shutdown, many and varied symptoms of shock may occur. There are some common symptoms which have been mentioned, but other symptoms can occur due to reduced blood flow to the organs of the body. F or example, neurological changes may occur. They are very non-specific in most cases, so a baseline is very important. Have any changes occurred in your patients mental status? If so, it may be due to reduced peripheral flow of blood. The same may be true for the renal system and the hepatic system and all others.

Assessment

1. Cardiovascular – skin color pale, cool and moist, blanching poor, increased heart rate, weak pulse, assess all pulses and check vital signs frequently, CHF may develop.
2. Renal – decreased urinary output, concentrated urine, increased BUN, increased serum ceratinine, increased specific gravity, in very late stages, burin may become very dilute, indicating near death.
3. Neurological – restlessness, agitation, disorientation, drowsy, even coma may develop, the rain is usually affected last due to the tremendous circulation to the rain, however late state shock can cause thee and other symptoms.

The above is a guide to assessing the patient if you suspect shock. If you do suspect shock, you must institute emergency measures. Each facility has a procedure, but here are some general guidelines:

For Suspected Shock (What Should You Do?)

1. Elevate legs

   Many hospital beds today have controls to elevate the food of the bed, if not, prop legs upon pillows, this puts more blood into circulation and may raise the blood pressure.

2. Keep warm (place blankets on the patient)

   You will help to maintain perfusion of the peripheral vessels, thereby protecting vital functions of the body.
3. Vital signs

   Take and record as soon as possible.

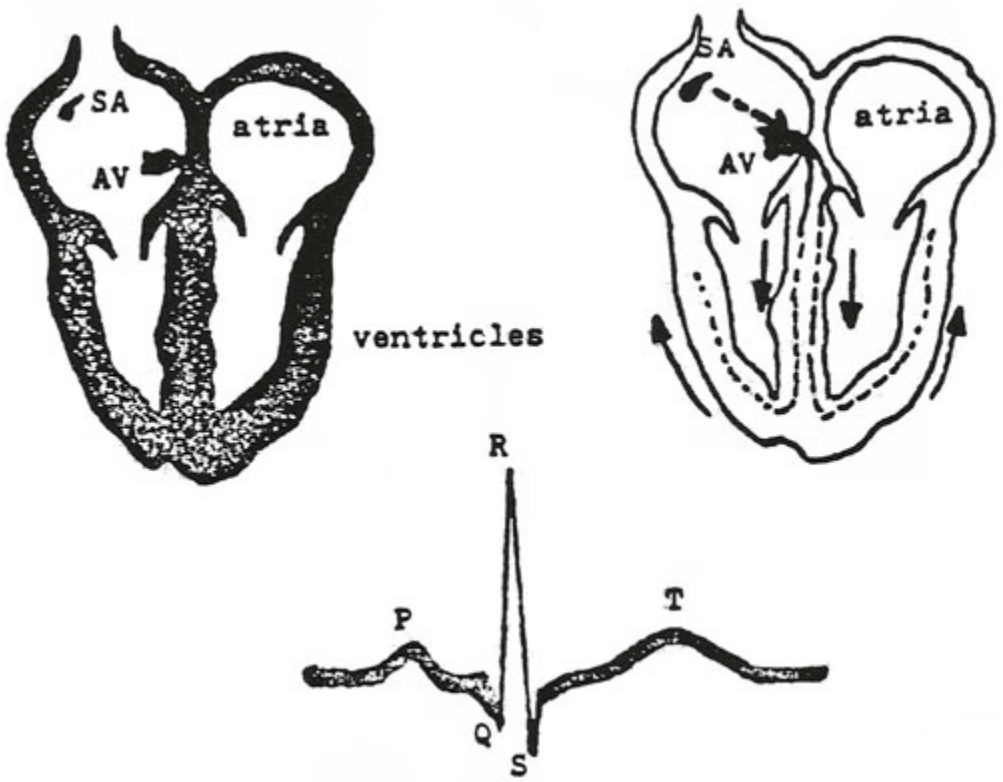
4. Oxygen

   Supplemental oxygen 1 – 3 L/min only, since perfusion is reduced, person will need supplemental oxygen.

5. Extended care

   Those nurses in the ICU will usually have a protocol, or specific orders to give the patient drugs or other treatments in the case of shock; floor nurses will at this point call the doctor and await orders for these treatments, meds, etc., remember to take vital signs frequently, stay with the patient, have the crash cart in the room, help to calm other patients if in the room, and be ready for life-support if needed.

Electrical Activity of the Heart Related to the Normal EKG



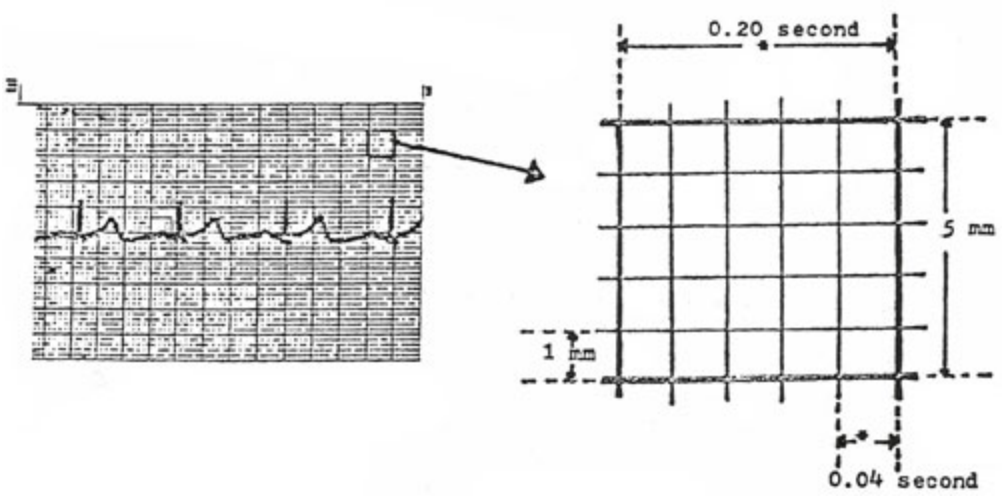
Represented in the diagram is the electrical pathway of the impulses through the heart. Each wave on the EKG is related to a portion of those impulses. When the heart muscle is stimulated by electrical impulses, blood is ejected from the corresponding chamber of the heart. Below are listed the waves of the EKG and the impulses which is the origin:

P Wave: impulse going through the atria
QRS Complex: impulses going through the ventricles.
T wave: Repolarization (recovery phase) no heart contraction

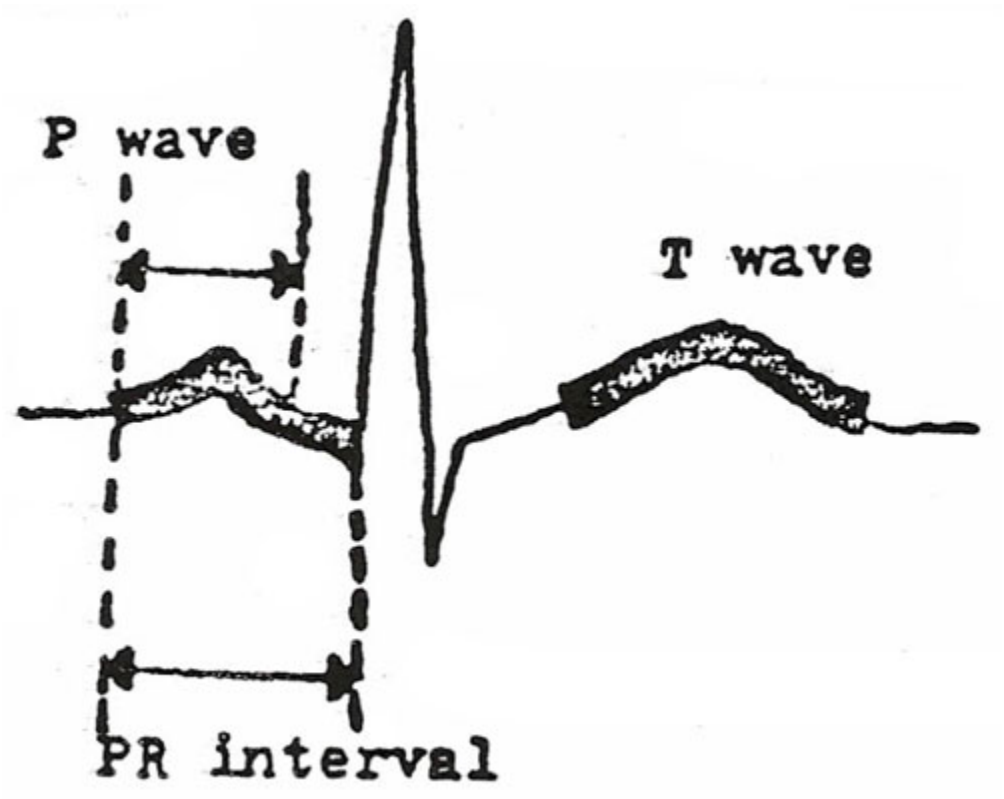
The SA node is referred to as pacemaker of the heart. This is where the electrical impulses originate, which eventually stimulate the entire cardiac cycle. The SA node is located near the top of the atrium and is also called the “normal physiological pacemaker.” The impulse then spreads form the atria to the AV node. The QRS complex being in the AV node which is located on the septum of the heart near the superior aspect of the ventricle. Both SA and AV nodes are innervated by the autonomic nervous system. Branches of the vagus nerve cause heart rate to slow or to increase. The T wav, as mentioned earlier, represents repolarization. The heart muscle is readying for the next contraction. There is electrical activity at this time, but it is related to recovery, not to an impulse to contract the heart muscle.

The EKG Paper

In order to begin understanding the interpretations of EKG’s one must have knowledge of the EKG paper. Shown in this illustration is one of the large blocks of the EKG paper. The time intervals are shown as well as the measurements of each block.



Measuring each wave of the EKG:



P Wave – 3 small blocks tall and 3 small blocks wide.

T Wave – up to 10 small block high in the precordial leads; up to 5 small blocks high in the remaining leads.

PR Interval – Up to 5 small blocks in length (0.20 seconds) will be lengthened if there is scarring to the area of the atrium and AV node area.

QRS Complex – consists of Q wave, R wave, S wave the QRS complex refers to the ventricular impulse and the contraction.

ST Segment – Begins at the end of the S wave and ends at the beginning of the T wave; if it is elevated, can mean an MI if it is depressed can mean hypoxia to the myocardium.

The Locations of the EKG Leads and Their Significance:

All leads of the EKG record the same electrical impulses of the heart muscle; however, each lead placed in a different area of the body records the electrical activity from a slightly different "angle". This means that by using the EKG tracing from different positions, various EKG waves will be accentuated. The diagnosis of arrhythmias may be made easier by examination of different leads. The 12-ead EKG tracing is standard. Six leads are recorded by placing wires on each limb. Si leads are recorded by placing wires on the chest in different positions.

Limb Leads: I, II, III, IV, V, VI

Lead IV also called AVR
Lead V also called AVL
Lead VI also called AVF

Chest Leads: V1, V2, V3, V4, V5, V6

For diagnosis of most arrhythmias, lead II is most commonly used. Lead II and the chest leads, most consistently show the most clear P wave, which can be diagnostic of many common arrhythmias.

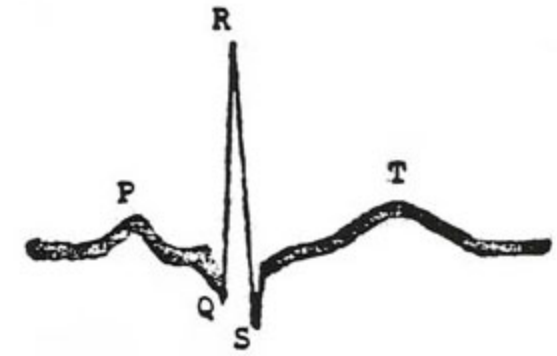
The following leads are listed to show their relationship to areas of the myocardium:

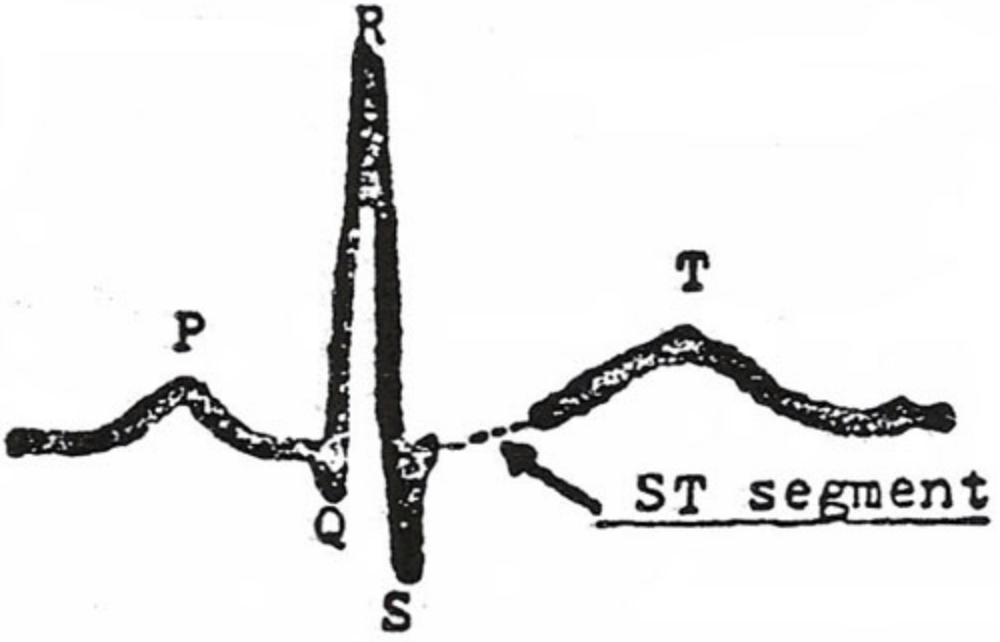
V1, AVR – right side of heart
V2, V3, V4 - transition between right and left sides of heart
V5, V6, I, AVL – left side of heart
II, III, AVF – inferior aspect of the heart

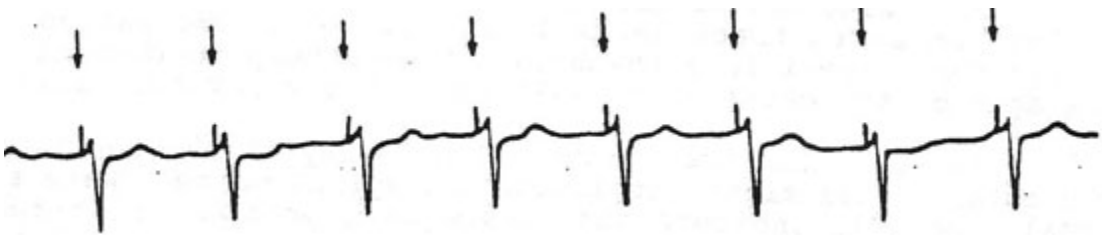
If changes in the EKG tracing are seen in a group of the above leads, this disease can be localized to a particular area of the heart. In the case of an MI which shows changes in leads V1 and AVR only, the damage ot he heart is in the right side. If the MD can thus localize the damage to the heart they can usually diagnose other possible problems in the heart. Valvular problems may show up as a specific change in one or more leads of the EKG tracing. Blockages in one of the major arteries of veins, may show up as an altered deflection in the EKG.

Following is a diagram of the chest and the placement of leads on the chest, so as to trace leads I and II. When the patient is being monitored for a specific arrhythmia, it will help to connect the wires to the spot on the chest which will show that the arrhythmia most clearly.

In assessing the electrical activity of the heart, the nurse should obtain vital signs, including the apical pulse. Rate and rhythm abnormalities will indicate that there is a problem which requires an EKG tracing. EKG interpretation is a lengthy course which would be too long to discuss here.







Assessment of the MI Patient

As a review, we remember that an MI myocardial infarct is death of the heart muscle tissue. The area of infarct can be small or large depending upon the amount of the blood supply which was cut off.

The treatment for the MI patient is divided into two phases. First, the acute stage where the patient is in the ICU. The second phase of medical treatment is the rehabilitation stage. The person is placed on the nursing care until where rehabilitation starts.

Assessment During the Acute Phase (Day 1 – 4)

1. Assess for possible complications of the MI
    a. Arrhythmias
    b. Congestive heart failure
    c. Carcinogenic shock
    d. Mitral regurgitation
    e. Ventricular septal rupture
    f. Pericarditis
    g. Ventricular aneurysm
    h. Dressler's syndrome (post MI syndrome)
2. Progressive activity
    Starting with self-care items, then progress as per the individual's capability, MD will order activity levels.

Assessment During the Rehabilitation Phase (semi acute 4 – 10 days):

1. Up to bathroom and assess patient for any arrhythmia which is still a concern at this time, bedside activities only also assess vital signs regularly, especially after activity.
2. Daily care – assess short walks in room and hallway, can usually do all hygiene activates at this time.

Assessment during late Rehabilitation Stage:

1. Counseling on discharge – does the patient return to their same job and lifestyle?
2. Patient teaching – the patient should be taught what to look for; any adverse symptoms should be reported to the physician immediately, careful assessment before discharge is important.

These are only guidelines to assessing the MI patient. Each person will progress differently and must be assess on their own merits. Their individual programs will be worked out with their cardiologist. The nurse should be familiar with each individual's program so that it can be followed carefully. Continually asses for the above complications which can occur at any time during the rehabilitation phase of the MI patient. Complications such as arrhythmias, CHF, shock, and angina will always slow the recovery of the patient. These persons with complications will have to be assessed even more carefully to prevent life threatening further complications.

Assessment of Patient with CHF

Congestive heart failure, or cardiac decompensation, whatever the cause, results in lowered cardiac output. In addition to this problem, blood backs up behind the heard causing hypertension and a variety of similar conditions. In assessing this patient, first keep in mind

emergency procedures in case of a crisis. CHF can lead to pulmonary edema very quickly so be prepared to act quickly.

Assessment of CHF:

1. Vital Signs

   Baseline vital signs are important here as well as for our other assessments, including an apical pulse; history is also important.
2. Cardiovascular
   Assess heart rhythm, and strength of the heartbeat. Assess pulses, skin color, tugor and blanching.
3. Respiratory
   Assess lung sounds for congestion, rales
4. General Medical
   Neuro assessment, lethargy, restlessness, skin color, extremities for reduced circulation

Drugs for treating CHF:

1. Digoxin
   Used to strengthen the force of contraction and increases cardiac output, can be used to reduce all the symptoms of even severe CHF, watch for signs of toxicity – nausea and gastritis.
2. Diuretics
   Either thiazide type or loop type; helps to reduce fluid volume over load and reduce pulmonary symptoms.
3. Vasodilators
   Can help reduce volume that ventricles must pump, thus reducing eh workload. (arterial dilators)

The above guide assumes that the patient is stable. If the patient is in a crisis situation, such as pulmonary edema, they will need emergency care in the form of intravenous digoxin and/or diuretics. In addition, the patient will require supplemental oxygen (1-3 L/min), sometimes needs oxygen via ventilator and endotracheal tube, under pressure, and in high concentrations. In this acute state of emergency it is important to continually monitor the vital signs and lab test values. The patient must be maintained with adequate oxygen and blood supply until the heart can begin again to pump sufficient supplies of blood into circulation, and the lungs can clear of the excess fluid. Digoxin is a cardiotonic drug that increased the force of the contraction of the heart, increasing cardiac output.

With most vasodilators, the blood pressure will be lowered and the pulse rate will be increased as will the respiratory rate be increased. The nurse must relax the patient as much as possible and make them comfortable as possible so as to reduce the work load of the heart. Pulmonary assessment in these emergencies is also important as cardiovascular assessment.

Artificial Pacemakers

There are many different types of cardiac pacemakers. They can be external or internal artificial pacemakers. Earlier in the text, it was mentioned that pacemakers can be used to treat many of the Brady (slow) arrhythmias. The EKG tracing illustrated, shows a normally functioning pacemaker. The pacemaker can be the demand type, where the pacemaker will automatically

start again if the patient's rate falls below a certain preset rate. There are also pacemakers which are preset to give a constant pulse, no matter what the patient's heart rate is. Note the pacemaker "spikes" on the sample EKG. Each of these spikes represents an electrical stimulation of the heart.

Cardiovascular Disease and the EKG

Condition	Possible Arrhythmias
1. Myocardial Infract	Heart block, PAC's, PVC's, tachycardia
2. Congestive Heart Failure	Tachyarrhythmias (sinus tachycardia, ventricular tachycardia), PVC's
3. Cardiogenic Shock	Tachyarrhythmias, PAC's, PVC's, Cardiac Standstill
4. Pericarditis	Tachycardia, PVC's, Ventricular arrhythmias
5. Open Heart Surgery	Heart block, tachyarrhythmias, other conduction defects.

The above is merely a guide to possible complications with certain cardiac related disorders. There can also be others. If the patient is being monitored in the ICU or other such unit, any of the arrhythmias will be detected rapidly. If they are on a medical/surgical unit, and not monitored, the nurse must be acutely aware of arrhythmias. Look for indications as change in level of consciousness, dizziness, pallor, confusion, lowered urinary output, sudden development of edema and other signs/symptoms which might indicate an arrhythmia. Of course, nothing will take the place of careful and accurate recording of vital signs. A sudden or even insidious change in the vial signs can be the first indication of an arrhythmia.

Interpretation of the EKG

This is perhaps the most important section of this course. Interpretation of the EKG must be performed in a logical sequence in order to best serve the patient. There are many different approaches to the interpretation of the EKG, but in this section we will present a "common sense" approach. If you are responsible for a patient who is being monitored, you want to be able to recognize any arrhythmia which may be life-threatening. You want to be able to do that as fast as possible; it may save the life of your patient. As you read through these steps, remember that they may not apply to every patient in every situation, but always be deliberate and methodical when you interpret the EKG.

Step I: Assessment

a. Assess patient symptoms, if any, and vital signs.
b. Assess leads to the patient (must be in proper place)
c. Assess obvious abnormalities of EKG (rate, rhythm)

In this phase, the nurse must quickly note any adverse symptoms. The symptoms will determine if the arrhythmia is severe or not. The vital signs are important. At the same time, you are assessing the patient, look at the EKG to determine if there are any gross arrhythmias, such as no rhythm, or grossly irregular rhythm. After these are determined, the nurse can then take the appropriate action. If the patient needs immediate resuscitation or if they can wait for treatment.

Step II: EKG Components

a. Examine the individual components of the EKG tracing.
b. Are any of the components missing
c. Are any of the components present but altered

Examine each of the waves of the EKG. Make sure that there is a P wave, QRS complex, etc. if all of the components are present, are they normal in configuration. They should all be identical in shape, and in timing.

Step III: Determine the Arrhythmia

a. Atrial arrhythmia
b. Ventricle arrhythmia

Once you have identified the abnormal component on the EKG, you then name the arrhythmia. If the abnormality is in the atria (P wave), then identify the arrhythmia. If the abnormality is in the ventricle, then identify the arrhythmia.

Step IV: Action

a. Immediate action
b. Long-term action/intervention

As we stated above, if the arrhythmia is immediately life-threatening, then immediate action must be taken. However, in the most nursing situations, the action will involve notifying the physician and then treating the arrhythmia with the appropriate drug. As you proceed through each of the above steps, you must continually be aware of the changes in the patient's condition and of possible intervention. Each hospital will have a different protocol for dealing with arrhythmias. Always consider your hospital's policy and procedure and use your common sense when dealing with these potentially fatal arrhythmias.

Cocaine Use and Nursing Assessment

The US Food and Drug Administration has classified cocaine as a schedule II narcotic (controlled substance with a high potential for abuse). (Swinyard 1985) Cocaine has several approved medical uses, and is mostly used as a local anesthetic. Cocaine is also classified as a sympathomimetic agent (Gay 1982). However, cocaine today has become a highly abused drug, and there is not much known about its effects on humans.

There have now been many reports published about its effects and yet there is still much controversy about the drug. Cocaine is a tropane alkaloid (benzoylmethylecogognine), of the evergreen shrub, Erythroxylon coca. It is extensively grown in Bolivia and Peru. The strong sympathomimetic effects of cocaine have been compared to amphetamines. It causes the "fight and flight" reaction; tachycardia, dilated pupils, increased muscle contractility, increased blood glucose, and peripheral vasoconstriction.

Cocaine has been found to primarily block the uptake of neurotransmitters at the nerve terminals, thereby potentiating sympathetic stimulation and central nervous system effects of euphoria. This is due to the effects of dopamine and serotonin. (Langer 1974) (Gropetti 1976) of course, the response to cocaine is a phenomenon unique to each individual. Even the

feelings of dysphoria experienced when the effects of the cocaine wearing off, are different for many individuals. When assessing patient who have taken cocaine, the nurse should remember that the drug has quite variable effects upon individuals. Physical tolerance and withdrawal symptoms do not occur with cocaine. However, it is still considered to be very addictive due to its effect as the drug is wearing off. During this time, the person experiences extreme dysphonic sensations, which leads to a craving of the drug. Therefore, the need for cocaine could be considered an obsession rather than a physical addiction. Many authorities consider this a psychological addiction rather than a physical additions. Cocaine will usually cause the following:

Central Nervous System: euphoria, a sensation of "soaring", elation, laughing, talkativeness, flighty, irritability, apprehensiveness, unable to sit still, nausea, vomiting, headaches, cold sweats, vertigo, twitching of small muscles especially of face, fingers, feet, tremors, generalized tics, possible psychosis, hallucinations, core body temperature rises. Advanced effects may be unresponsive, decreased responsiveness to all stimuli, incontinence. Cocaine may have the opposite effect: depression, flaccid paralysis of muscles, coma, pupils fixed and dilated, loss of reflexes, respiratory arrest, cocaine has also been known to precipitate CVA's.

Cardiovascular System: initially the pulse may be irregular, and then become very slow, later. Pulse may raise dramatically, hypertension, skin pallor caused by vasoconstriction, PVC's, increased respiratory rate and depth. Advanced symptoms may include more increase in pulse and blood pressure, then blood pressure can fall due to ventricular arrhythmias that can occur, pulse becomes rapid, weak and irregular, peripheral and then central cyanosis, Cheyne-Stokes respirations, gross pulmonary edema, may lead to MI or ventricular fibrillation and death.

Dosage and Absorption of Cocaine

As with all other drugs, the dosage and the route of administration will greatly influence the effects of the cocaine. The maximum therapeutic dose of cocaine for local anesthesia is 200mg to 300mg. (Perman 1979) cocaine can be in the form of a powder, paste, liquid, or crystal and may be administered by oral, parenteral, intranasal routes. In fact, newer forms of abusing cocaine include the inhalation of concentrated forms of "street" cocaine, "freebasing" that can be "smoked" like a cigarette or just inhaled.

Many street forms of cocaine will also contain other drugs such as amphetamines, caffeine and other stimulants or Lidocaine (Allred 1981). However, cocaine is not taken orally very frequently, because the effect is diminished due to the acidity of the stomach and the alkalinity of the small intestine; absorption is poor from these environments.

Nurses today are most likely to encounter patients with cocaine toxicity in the intensive care unit of the hospital. However, it is certain that other patients, some of whom may be seriously ill, may be suffering from the effects of cocaine use. Every nurse will need to remember that there is a very wide prevalence of cocaine use today and be able to recognize and assess those signs and symptoms of the use of cocaine.

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Course Test

1. Prior to beginning the cardiovascular assessment, the nurse should obtain the:
    a. Patient history
    b. Lab test results
    c. Closing of both the aortic and pulmonic valves
    d. Closing of both the mitral and tricuspid valves

2. S1, the first heart sound, is made up of:
    a. Closing of the pulmonic valves
    b. Closing of the tricuspid valves
    c. Closing of both the aortic and pulmonic valves
    d. Closing of both the mitral and tricuspid valves

3. The timing of a heart murmur may be described as:
    a. Early
    b. Late
    c. Middle
    d. All of these

4. When interviewing the patient, you should ask questions using lay terminology and
    a. You should not use medical terms.
    b. You should use precise medical terms

5. The first step to perform, when determining the EKG rhythm is:
    a. Examine the individual components of the EEG
    b. Take action to correct any arrhythmia
    c. Assessment
    d. Name the arrhythmia

6. All but one are cardiovascular signs of shock, that one not a classical symptom is:
    a. Tachycardia
    b. Skin cool and moist
    c. Hypotension
    d. Cyanosis
    e. Blanching poor

7. The first step in the inspection process is:
    a. Have patient sit upright
    b. Allow for privacy
    c. Inspect from the back of patient

8. The significance of the 5^{th} intercostals space and the midclavicular line is:
    a. Aortic area
    b. Pulmonic area
    c. Ventricular area
    d. Apical area
    e. Epigastric

9. A pressoreceptor is located in which area(s) of the body?
    a. Aortic arch
    b. Abdominal aorta
    c. Vena cava
    d. Coronary artery
    e. All of these

10. A light gray ring surrounding the iris and is common in older patients, is called:
    a. Xantheiasma
    b. Cataract
    c. Hordeolum
    d. Cyanosis
    e. Arcus senilis

11. An example of a cardiotonic drug is:
    a. Lidocaine
    b. Apresoline
    c. Amyl nitrite
    d. Inderal
    e. Digoxin

12. A drug used to treat sinus Bradycardia is:
    a. Lidocaine
    b. Bretylium
    c. Atropine
    d. Digoxin
    e. None of these

13. Edema described as +4 is edema having more than a one inch indentation
    a. True
    b. False

14. Mitral stenosis usually causes a very loud S2 sound
    a. True
    b. False

15. Physiological splitting of S2can be caused by normal respiratory patterns
    a. True
    b. False

16. Each large block of EEG paper represents two seconds of time
    a. True
    b. False

17. The QRS complex refers to the electrical conduction going through the atria
    a. True
    b. False

18. A possible complication of a myocardial infarction is shock
    a. True
    b. False

19. CVF will tend to cause slow arrhythmias such as sinus Bradycardia, in patients
    a. True
    b. False

20. Very late stage shock will cause oliguria and concentrated urine
    a. True
    b. False

21. In secondary hypertension, the cause of high blood pressure is not known.
    a. True
    b. False

22. Pulsus paradoxus might be indicative of constrictive pericardial disease.
    a. True
    b. False

23. The first step to examination of the heart is auscultation
    a. True
    b. False

24. A bounding pulse is described as having very sharp upstroke and downstroke wave
    a. True
    b. False

25. Isuprel combats shock, but also increases the demand for oxygen by the heart.
    a. True
    b. False

26. Levophed is a patient vasoconstrictor used for combating shock.
    a. True
    b. False

27. S3 heart sound is not normally heard, but can be normal in most situations.
    a. True
    b. False

28. Murmurs can be described by indicating where they fall in cardiac cycle.
    a. True
    b. False

29. A sign that CVF might be elevated is any distention of the jugular vein.
    a. True
    b. False

30. The a wave is the predominant wave in neck, it reflects ventricular pressure.
    a. True
    b. False

31. In beginning to auscultate the heart, have the patient lie on their back at about
    a. 30 degree angle
    b. 45 degree angle
    c. 60 degree angle
    d. 90 degree angle

32. The diaphragm of the stethoscope is best for hearing
    a. High pitched sounds
    b. Low pitched sounds

33. S3 and S4 gallops are usually low pitched sounds, and heard best with the
    a. Diaphragm of the stethoscope
    b. Bell of the stethoscope

34. Holodiastolic murmurs describes murmurs heard:
    a. S1 to S3
    b. S3 to S4
    c. S1 to S2
    d. S1 to S4
    e. S2 to S1

35. Mitral stenosis will usually cause the following heart sound:
    a. Loud S2
    b. Splitting of S2
    c. Loud S1
    d. Splitting of S1
36. Hypertension will usually cause the following heart sound:
    a. Loud S2
    b. Splitting of S2
    c. Loud S1
    d. Splitting of S1

37. Describing where the sound was heard in the cardiac cycle is called:
    a. Pitch
    b. Loudness of volume
    c. Timing
    d. Splitting

38. If the intensity of the heart murmur begins softly and becomes louder, it is:
    a. Medium
    b. Decrescendo
    c. Holosystolic
    d. Crescendo

39. Narrative charting of your cardiovascular assessment is also called:
    a. long form
    b. problem form
    c. story form
    d. short form

40. Tachycardia can be caused by increased stimulation of sympathetic nervous system. _____ can cause this.
    a. watching television of eating dinner
    b. resting in bed
    c. fear or fever
    d. walking downstairs or listening to music

41. Usually, the first drug used to treat PVC's is
    a. atropine
    b. Bretylium
    c. Lidocaine
    d. Isuprel

42. The most common symptom of acute peripheral arterial disease is:
    a. Spasms
    b. Occlusion
    c. Pain
    d. Pallor
    e. Tingling

43. In chronic peripheral arterial disease, numbness of the extremity get worse:
    a. After resting
    b. Upon eating
    c. Upon exercise
    d. At night

44. The usual cause of peripheral venous disease is likely to be:
    a. Strain at work
    b. Increased venous pressure
    c. Poor nutrition

45. One of the major indicators of the presence of peripheral venus disease is:
    a. Pain in the area
    b. Increased blood pressure
    c. Edema

46. With cocaine use, the initial response of the cardiovascular system is:
    a. Irregular pulse
    b. Slowed pulse rate
    c. Dilated arteries.