

## Chapter 8 Cardiovascular

3 layers: pericardium-outermost layer  
myocardium-thickest & strongest-composed of cardiac muscle tissue  
endocardium-innermost layer-connective tissue

heart divided into right & left half by muscular partition called the septum

**Right atrium**-upper right chamber, receives deoxygenated blood from entire body

**Right ventricle**-lower right & receives blood from right atrium (deoxygenated).RV pumps blood to lungs via pulmonary artery to release carbon dioxide & receive oxygen

**Left atrium**-upper left chamber-receives oxygenated blood from lungs via pulmonary veins.

**Left ventricle**-lower left-receives oxygenated blood from left atrium. Thickest, most muscular section of heart & pumps oxygenated blood out through aorta to all parts of the body.

Heart-2 separate pumps-right side receives deoxygenated blood & pumps it to the lungs

Left side receives oxygenated blood from lungs & pumps it

Throughout the body.

Heart Valves:

4 valves-keep blood moving forward & prevent backflow.

2 atrioventricular (AV) valves.-located between atrium & ventricles

**Right AV** valve is located between right atrium & right ventricle & called **tricuspid**

**Left AV** valve located between left atrium & left ventricle is **mitral valve**.

Both prevent back flow of blood by rapidly closing. Small cordlike structures, **chordae tendineae** connect AV valves to the walls of the heart & work with papillary muscles to make tight seal to prevent backflow when ventricles contract.

**Semilunar valves** are located @ the points where the blood exits the ventricles.

**Pulmonary semilunar** valve is located between right ventricle & pulmonary artery.

**Aortic semilunar** valve-located between left ventricle & aorta.

Automaticity-heart to contract in a rhythmic pattern

Irritability- ability to respond to stimulus. These are the 2 characteristics that affect functions of conduction system.

Heart beat initiated in sinoatrial (SA) node-upper part of the right atrium, regulates beat of the heart, known as pacemaker.

Impulses passed to AV node-to bundle of His (AV) bundle to Purkinje fibers, which surround ventricles.

### **CARDIAC CYCLE**

Complete heartbeat

2 atria contract while 2 ventricles relax, when the ventricles contract, 2 atria

relax=**systole**.

**Diastole**-phase of relaxation.

Complete diastole & systole of both atria & ventricles constitute a cardiac cycle-average 0.8 second.

Heart sounds (LUBB & DUBB) are produced by closure of the valves.  
LUBB-first sound-long duration & low pitch, AV valves close.  
DUBB-second sound-short duration, sharp-heard when semilunar valves close.  
Murmur-swishing sound

### **BLOOD VESSELS**

3 main types: **capillaries**-(tiny, join arterioles & venules) connect **arteries** (large vessels carrying blood in a direction away from heart) to the **veins** (vessels that convey blood from capillaries to the heart). Veins return blood to the heart.

**Arterioles**-blood vessels of the smallest branch of the arterial circulation, which delivers blood to tissues.

**Capillaries**-exchange of products & by-products between the tissues & blood.

**Venules**-tiny veins-link with larger veins & return to the heart.

Artery-arteriole-capillary-venule-vein

### **CORONARY BLOOD SUPPLY**

Delivery of oxygen & nutrient-rich arterial blood to cardiac muscle tissue & return of oxygen-poor blood from this active tissue to the venous system is called-coronary circulation.

Blood flows into the heart muscle by way of 2 small vessels-right & left coronary arteries. The coronary arteries form a crown around the myocardium.

Coronary arteries bring oxygen & nutrition to the myocardium.

### **SYSTEMIC CIRCULATION :**

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### **LABORATORY TESTS**

Serum lipids-associates with vascular disease-especially CAD. Serum lipids are associated with risk of cardiovascular disease. An elevated HDL is desired, but LDL or VLDL increases risk.

ABG monitor oxygenation & acid base balance

Serum cardiac markers-certain proteins released into blood in large quantities from necrotic heart muscle after MI.

Important screening diagnostic criteria for an acute MI.

Cardiac enzyme: CK(creatine kinase) & its isoenzyme, CK-MB (creatine phosphokinase)

CK-MB also found in skeletal muscle & can be elevated by surgery, muscle trauma, & muscular disease-not specific indicator for MI. CK & CK-MB start to rise within 2-3 hours after the beginning of an MI, peak in 24 hours & return to normal within 24-40 hours.

Troponin I is myocardial muscle protein released into circulation after Myocardial injury.

Heart-2 subtypes: cardiac-specific troponin T & troponin I-sensitive markers that identify small amounts of myocardial damage. Troponin T appears in the blood 305 hours after MI & may remain elevated for up to 21 days, affected by skeletal muscle injury & renal disease.

Troponin I is sensitive & specific cardiac marker, not influenced by skeletal injury, or renal disease. Rises 3 hours after MI, peaks 14- 18 hours, returns to normal 5-7 days. Troponin I very useful in diagnosing MI.

Myoglobin-released into circulation within only a few hours after MI, lacks cardiac specific, also present in skeletal muscle. Also rapidly excreted in urine so that blood levels return to normal range within 24 hours after MI.

VLDL-contains more fat than protein

Triglycerides-main storage form of lipids

LDL-contains equal amount of fat & protein

HDL-contains more protein than fat & serves as a protection, removes cholesterol from tissues.

Diet high in saturated fat, cholesterol, & calories contributes to hyperlipidemia.

Overall serum cholesterol level of less than 200 mg/dl is desirable.

Change in diet is probably most important method of lowering cholesterol level.

Cholesterol-lowering drugs are often included in Tx. of hyperlipidemia. These drugs are divided into 5 main groups:

HTN-greater than 140/90

## **CARDIAC DYSRHYTHMIAS**

Dysrhythmia (arrhythmia)-refers to any cardiac rhythm that deviates from normal sinus rhythm. The result of an alteration in the formatin of impulses through SA node to the rest of the myocardium, Signs & SX. vary & Tx. depending on type & severity.

### **TYPES of CARDIAC DYSRHYTHMIAS**

**Sinus tachycardia**-rapid, regular rhythm-SA node. HR of 100-150 /minute

Causes: exercise, anxiety, fever, shock, meds., hypothermia, heart failure, excessive caffeine, & tobacco. Acts to increase the amount of O<sub>2</sub> delivered to cells of body by increasing the amount blood circulated through the vessels.

Sx.: palpitations, hypotension. May be asymptomatic. NSR & not usually caused by cardiac problem.

### **Sinus Bradycardia**

Slow rhythm-SA node. HR slower than 60 beats/min.

Causes: sleep, vomiting, intracranial tumors, MI, drugs (dig. Toxicity), vagal stimulation, endocrine disturbances, & hypothermia. MI-beneficial because reduces myocardial oxygen demand. Normal rate for athlete.

Sx.: fatigue, lightheadedness, & syncope, may be asymptomatic

Atropine may be prescribed to increase HR, pacemaker-temporary or permanent.

### **Supraventricular tachycardia (SVT)**

Sudden onset of a rapid HR. Originates in atria, pulse rate 150-250 beats/minute

Causes: drugs, alcohol, MVP, emotional stress, smoking, & hormone imbalance. Typically not associated with heart disease.

Sx.: palpitations, lightheadedness, dyspnea, & anginal pain.

Tx: carotid sinus pressure, Adenocard, digitalis, calcium channel blockers, beta-blockers, quinidine, cardioversion

### **ATRIAL FIBRILLATION:**

Electrical activity in atria is disorganized, atria quivers rather than contracts. Rapid production of atrial impulses, chaotically & not contracting properly.

Atrial rate 350- 6000 beats/ minute

Causes of A. Fib: cardiac surgery, HTN, pulmonary embolism, atherosclerosis, mitral valve disease, HF, cardiomyopathy, COPD, congenital abnormalities,

Clinical Manifestations: Pulse deficit, palpitations, dyspnea, angina, lightheadedness, syncope, fatigue, change in LOC, & pulmonary edema. DECREASED cardiac output.

Thrombi may form due to ineffective atrial contraction & cause embolisms.

Risk of strokes. Goal to prevent atrial thrombi from becoming embolisms in the body

### **ATRIOVENTRICULAR BLOCK**

Defect in AV junction slows or impairs conduction of impulses from SA node to ventricles.

3 types block seen: first degree, second degree, & third degree. Third degree worsening & possible complete heart block.

Causes: atherosclerotic heart disease, MI, CHF, dig. Toxicity, congenital abnormality, drugs, hypokalemia.

No sx. for first degree

Second degree block: vertigo, weakness, & irregular pulse

Third degree block: hypotension, angina, bradycardia, HR 30s, & heart failure

Atropine, pacemaker

### **PREMATURE VENTRICULAR CONTRACTIONS (PVC)**

Abnormal beats arise from right/left ventricle

Early ventricular beats that occur in conjunction with underlying rhythm.

Underlying rhythm remain same except for PVC

PVC may originate from more than 1 location in ventricles & be caused by irritability of ventricular musculature, exercise, stress, electrolyte imbalance, dig. Toxicity, MI, hypoxia.

Can lead to V-tach, V-Fib.

Bigeminy-occurs every other beat

Trigeminy-occurs every third beat

Clinical Manifestations: palpitations, weakness, lightheadedness

Tx. underlying cause, also B-blockers, Pronestyl, lidocaine

### **VENTRICULAR TACHYCARDIA**

VT occurs when 3 or more PVC occur.

Ventricular rate is greater than 100/minute

Rhythm is regular or slightly irregular

Precedes V-Fib & sudden cardiac arrest

Med. Management-IV Pronestyl or Cordarone-depress excitability of cardiac muscle

Lidocaine(Xylocaine) is used only if acute MI is considered to be cause----cardioversion

## **VENTRICULAR FIBRILLATION**

Chaotic pattern of electrical activity in ventricles, in which electrical impulses arise from many different foci. Produces no effective muscular contraction & no cardiac output.

Untreated Vfib causes most cases of sudden cardiac death in people outside of a hospital  
Ventricles quiver.

Causes:

Myocardial ischemia,MI, untreated V-Tach.,underlying heart disease, acid-base imbalance, electric shock, electrolyte imbalances (hypokalemia, hyperkalemis, hypercalcemia)

Pt in V-Fib is in full cardiac arrest, unresponsive, & without detectable B/P or carotid or femoral pulse.

DEFIBRILLATION is most effective treatment for V-Fib

CPR must be performed until defibrillator arrives to preserve oxygen

## **NSG. INTERVENTIONS & PT. TEACHING**

Assess the apical pulse (not radial) to obtain accurate pulse rate when dysrhythmias are present. Take apical pulse for 1 full minute.

Assess level of pt.'s anxiety & degree of understanding.

Explain procedures, monitor HR & rhythm, quiet environment, administer sedation, O2

## **CARDIAC ARREST**

Sudden cessation of cardiac output & circulatory process.

Severe V-Tach., V-Fib., & Ventricular asystole.

### **Artificial Cardiac Pacemakers**

Battery-operated generators that initiate & control HR by delivering electrical impulse via an electrode to myocardium.

Placed in area to be paced: r. atrium, r. ventricle, or both.

Pacemaker maintains a regular cardiac rhythm by electrically stimulating heart muscle.

Used when patients experience adverse sx. because of dysrhythmias that can't be managed by medications alone.

Nsg. Interventions:

Monitors HR & rhythm by apical pulse, EKG patterns.

V/S, LOC, insertion site

Importance of reporting signs & Sx. of pacemaker failure: weakness, vertigo, chest pain, & pulse changes. Tell pt. avoid holding electrical equipment next to pacemaker( hair dryer, battery-operated toothbrush)

## Disorders of Heart

### Coronary Atherosclerotic Heart Disease

Coronary artery disease (CAD)-describe variety of conditions that obstruct blood flow in coronary arteries.

Atherosclerosis-yellowish plaque of cholesterol, lipids, & cellular debris in inner layers of walls of arteries, primary cause of ather.heart dis.

### **ANGINA PECTORIS**

Spasmodic, cramplike, choking feeling.

Pectoris-breast of chest area.

Angina Pectoris thoracic pain & choking feeling caused by decreased oxygen or anoxia of myocardium.

Occurs when cardiac muscle deprived of oxygen.

CAD # 1 killer.

Unstable angina-unpredictable episode of severe & prolonged discomfort that appears @ rest, has never been experienced before or is worse than previous episodes. Mimics MI-tightness/crushing in chest, arms, back, neck, jaw. May be red flag that MI will occur.

#### Clinical Manifestations:

Heaviness or tightness of chest, thought to be indigestion. Substernal or retosternal.

Pain often radiates down left inner arm to little finger & upward to shoulder & jaw.

Dyspnea, anxiety, apprehension, diaphoresis, & nausea-similar to MI.

Relieved by rest or medication such as Nitroglycerin.

Feeling of impending death.

Monitor V/S

Holter Monitor

ASA. Plavix, Nitro

Surgery: CABG-coronary artery bypass graft

PTCA-percutaneous transluminal coronary angioplasty

Stent Placement-

SEE PATIENT TEACHING on PAGE 351