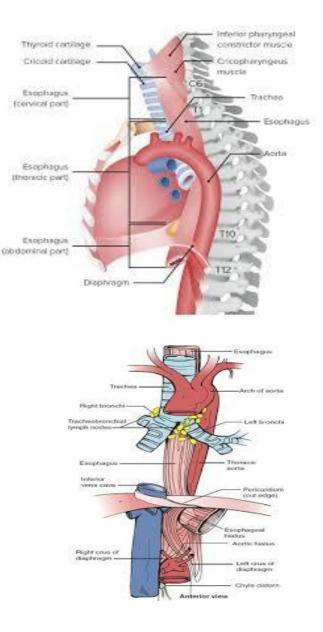
ESOPHAGUS

The esophagus is a tubular structure about **25-cm long** that is continuous above with the laryngeal part of the pharynx opposite the sixth cervical vertebra. It passes through the esophageal hiatus of the diaphragm at the level of the 10th thoracic vertebra to join the stomach.

In the neck, the esophagus lies in front of the vertebral column. In the thorax, it passes downward and to the left through the superior and then the posterior mediastinum.



Relations of the thoracic part of the esophagus from above downward are as follows:

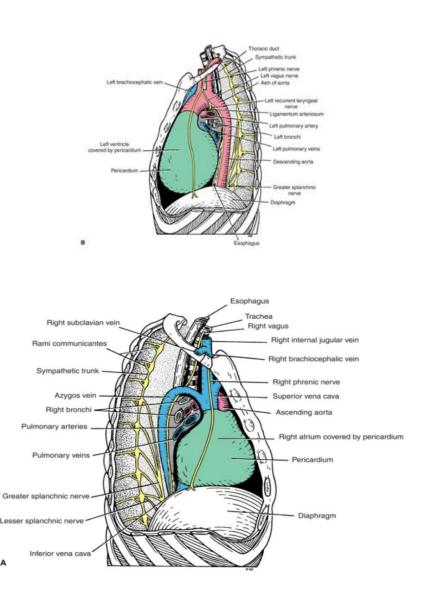
Anteriorly: The trachea and the left recurrent laryngeal nerve; the left

principal bronchus, and the pericardium.

Posteriorly: The bodies of the thoracic vertebrae, the thoracic duct, the azygos veins, and at its lower end, the descending thoracic aorta.

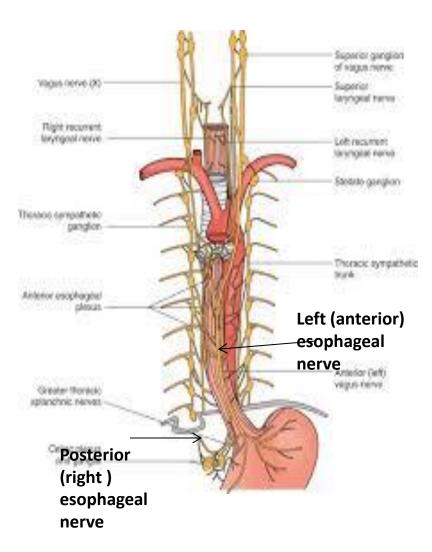
Right side: **The mediastinal pleura** and the **terminal part of the azygos v**ein.

Left side: The left subclavian artery, the aortic arch, the thoracic duct, and the mediastinal pleura.



Inferior to the level of the roots of the lungs, the vagus nerves leave the pulmonary plexus and join with sympathetic nerves to form the esophageal plexus. The left vagus lies anterior to the esophagus, and the right vagus lies posterior.

At its opening in the diaphragm, the esophagus is accompanied by the two vagi, branches of the left gastric blood vessels, and lymphatic vessels. Fibers from the right crus of diaphragm pass around the esophagus in the form of a sling. In the abdomen, the esophagus descends for about 1.3 cm and then enters the stomach. It is related to the left lobe of the liver anteriorly and to the left crus of diaphragm posteriorly.



Blood supply

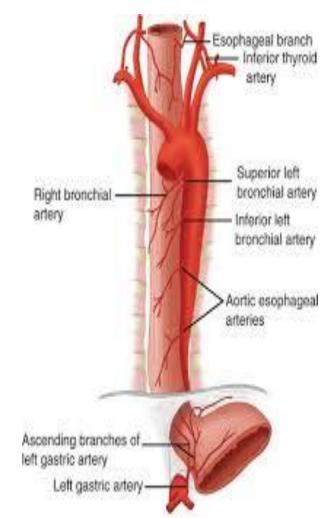
The upper third of the esophagus is supplied by the inferior thyroid artery, the middle third by esophageal branches from the descending thoracic aorta, and the lower third by branches from the left gastric artery. The veins from the upper third drain into the inferior thyroid veins, from the middle third into the azygos veins, and from the lower third into the left gastric vein, a tributary of the portal vein.

Lymph Drainage

Lymph vessels from the upper third of the esophagus drain into the **deep cervical nodes**, from the middle third into the **superior and posterior mediastinal nodes**, and from the lower third into **nodes along the left gastric blood vessels** and the **celiac nodes**.

Nerve Supply

The esophagus is supplied by parasympathetic and sympathetic fibers via the **vagi** and **sympathetic trunks**, respectively. In the lower part of its thoracic course, the esophagus is surrounded by the **esophageal nerve plexus**.



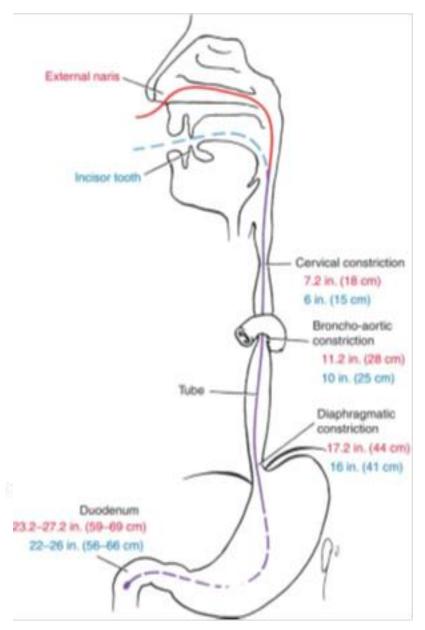
CLINICAL NOTES

Esophageal Constrictions

The esophagus has three anatomic and physiologic constrictions. The first is where the pharynx joins the upper end, the second is where the aortic arch and the left primary bronchus cross its anterior surface, and the third occurs where the esophagus passes through the diaphragm into the stomach.

These constrictions are of considerable clinical importance because they are sites where Swallowed foreign bodies can lodge or through which it may be difficult to pass an esophago-scope. These constrictions are also the common sites of carcinoma of the esophagus. Their respective distances from the upper incisor teeth are **15 cm**,

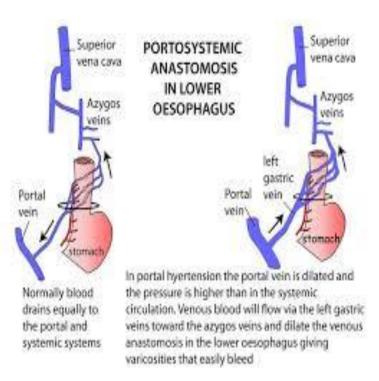
25 cm, and 41 cm, respectively.



The approximate respective distances from the incisor teeth

Portal–Systemic Venous Anastomosis

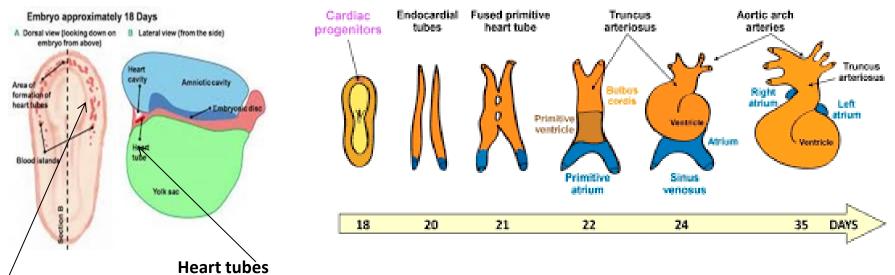
An important portal-systemic venous anastomosis occurs at the lower third of the esophagus Here, the esophageal tributaries of the azygos veins (systemic veins) anastomose with the esophageal tributaries of the left gastric vein (components of the hepatic portal system). when the portal vein become obstructed (e.g., in cirrhosis of the liver), **portal hypertension** develops, resulting in dilatation and varicosity of the portalsystemic anastomoses. Varicose esophageal veins may rupture during the passage of food, causing hematemesis (vomiting of blood), which may be fatal.



Embryology Notes

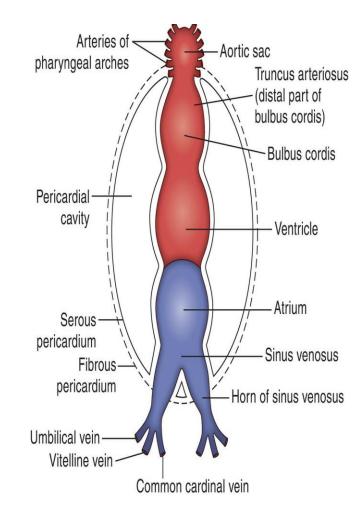
Heart Tube Development

About the 15th day of gestation, clusters of cells arise in the mesenchyme at the cephalic end of the embryonic disc. These clusters of cells form a plexus blood vessels that fuse to form the right and left endocardial heart tubes. The paired tubes soon fuse to form a single median endocardial tube. As the head fold of the embryo develops, the endocardial tube rotate on a transverse axis through almost 180°, so that they come to lie ventral to (in front of) the esophagus and caudal to the developing mouth.



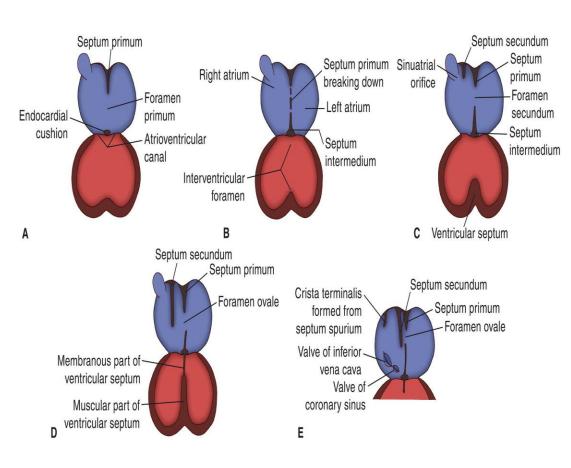
Blood Clusters

The endocardial tube starts to bulge into pericardial cavity and becomes the surrounded by a thick layer of mesenchyme, which will differentiate into the myocardium and the visceral layer of the serous pericardium. This establishes the **primitive heart tube**, with the cephalic end as the arterial end and the caudal end as the venous end. The heart begins to beat during the 3rd week. The heart tube then undergoes differential expansion, resulting in the formation of four dilatations, separated by grooves . From the arterial to the venous end, these dilatations are called the **bulbus cordis**, the ventricle, the atrium, and the sinus venosus.

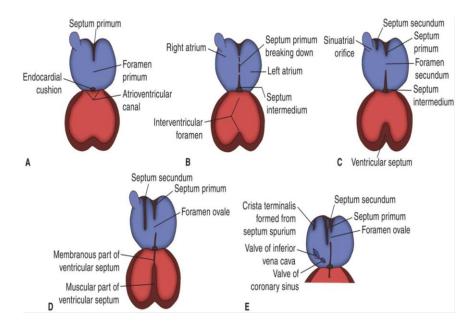


Atria Development

The single primitive atrial canal divides into two separate right and left atria; firstly by septum intermedium develops from the floor A, which divides the canal into right and left halves. Meanwhile, another septum, the septum primum, develops from the roof of the primitive atrium and grows down to fuse with the septum intermedium **B**.

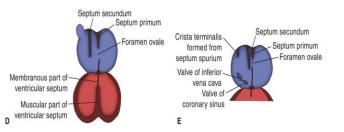


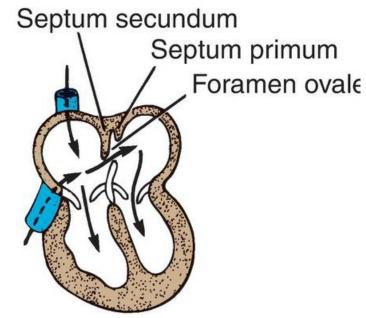
Degenerative changes occur in the central portion of the septum primum. This breakdown in the septum primum forms a second foramen, the foramen secundum **C**, which allows the right and left atrial chambers to communicate. Another, thicker, septum (the **septum secundum**) grows down from the atrial roof on the right side of the septum primum C&D. The lower edge of the septum secundum overlaps the foramen secundum in the septum primum but does not reach the floor of the atrium and does not fuse with the septum intermedium E. The space between the free margin of the septum secundum and the septum primum is now known as the foramen ovale.



Before birth, the foramen ovale allows oxygenated blood that has entered the right atrium from the inferior vena cava to pass into the left atrium. However, the lower part of the septum primum serves as a flaplike valve to prevent blood from moving from the left atrium to the right **atrium**. At birth, owing to raised blood pressure in the left atrium, the septum primum is pressed against the septum secundum and fuses with it, and the

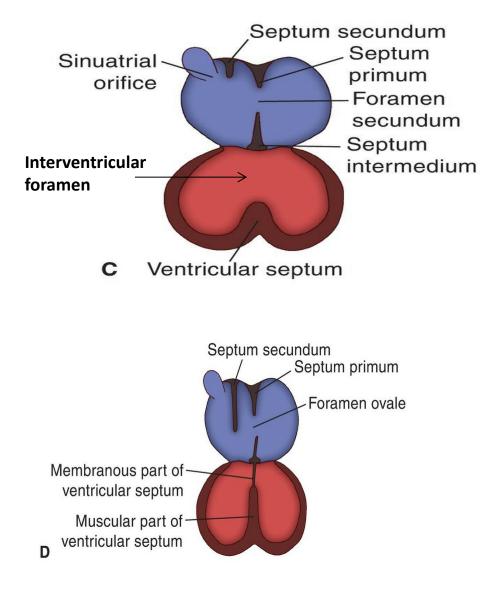
foramen ovale is closed. Thus, the two atria are separated from each other. The lower edge of the septum secundum seen in the right atrium becomes the **annulus** ovalis, and the depression below this is called the fossa ovalis.





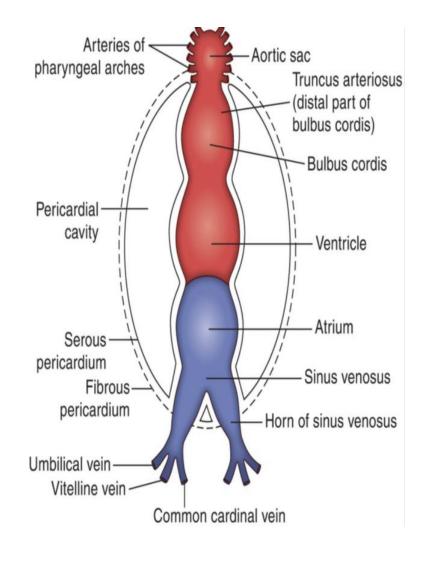
Ventricle Development

A muscular partition projects upward from the floor of the primitive ventricle to form the ventricular septum C&D. The interventricular foramen is the space bounded by the crescentic upper edge of the septum and the endocardial cushions closes as the result of proliferation of the bulbar ridges and the fused endocardial cushions (septum intermedium).



Development of the Roots and Proximal Portions of the Aorta and the Pulmonary Trunk.

The distal part of the bulbus cordis is termed the truncus arteriosus. The spiral aorticopulmonary septum divides the truncus to form the roots and proximal portions of the aorta and pulmonary trunk. With the establishment of right and left ventricles, the proximal portion of the bulbus cordis becomes incorporated into the right ventricle as the definitive conus arteriosus or infundibulum and into the left ventricle as the **aortic vestibule**. Just distal to the aortic valves, the two coronary arteries arise as outgrowths from the developing aorta.

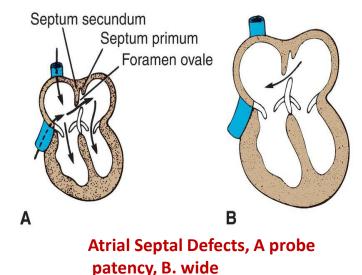


Atrial Septal Defects

After birth, the foramen ovale closes completely as the result of the **fusion of the septum primum with the septum secundum**. In 20% to 25% of hearts, a small opening (a **probe patency**) persists, but this is usually of such a minor nature that it has no clinical significance. Occasionally, the opening is much larger and results in oxygenated blood from the left atrium passing over into the right atrium.

Ventricular Septal Defects

Formation of the ventricular septum is complete when the membranous part fuses with the muscular part. Ventricular septal defects (VSDs) are the most common congenital cardiac malformation and may occur in either the membranous or muscular part. They are more common in the muscular part of the septum but are generally more serious defects when occurring in the membranous part. Blood under high pressure passes through the defect from left to right, causing enlargement of the right ventricle. Larger VSDs can shorten life if corrective surgery is not performed.

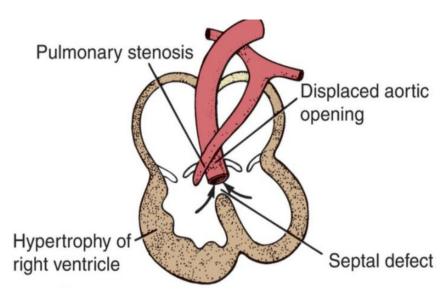


RA Reart with VSD

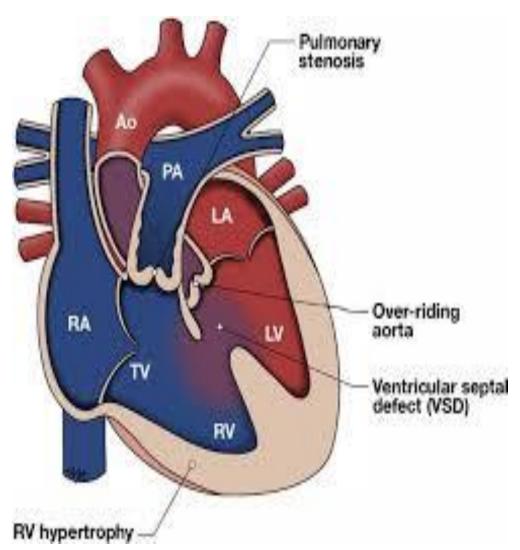
Ventricular Septal Defects

Tetralogy of Fallot

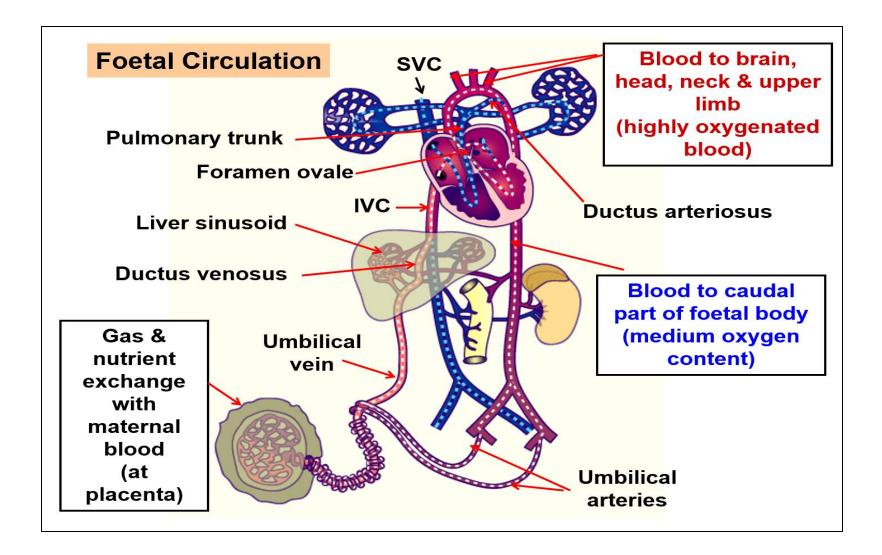
Normally, the bulbus cordis divides into the aorta and pulmonary trunk because of formation of the the spiral aorticopulmonary septum. This septum forms by the fusion of the bulbar ridges. If the bulbar ridges fail to fuse correctly, unequal division of the bulbus cordis may occur, with consequent narrowing of the pulmonary trunk resulting in **interference** with the right ventricular outflow. The resulting congenital anomaly, tetralogy of Fallot, which is the most common defect in the conotruncal region. The four anatomic abnormalities include 1) stenosis of the pulmonary trunk (narrowing of the right ventricular outflow), a 2) large ventricular septal **defect** (mainly in the membranous part), 3) overriding aorta (exit of the aorta immediately above the VSD instead of from the left ventricular cavity only), and 4) severe hypertrophy of the right ventricle (because of the high blood pressure in the right ventricle).



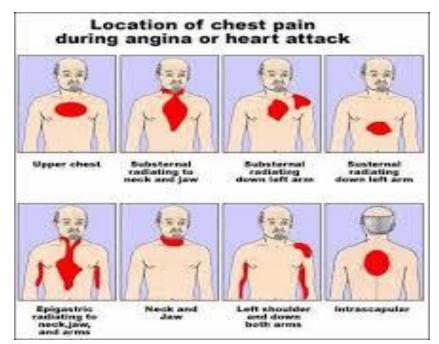
The defects, although not necessarily fatal, cause congenital cyanosis. Once the diagnosis has been made, most children can be successfully treated surgically. Most children find that assuming the squatting position after physical relieves activity their breathlessness. This happens because squatting reduces the venous return by compressing the abdominal veins and increasing the systemic arterial resistance bv kinking the femoral and popliteal arteries in the legs; both of these mechanisms tend to decrease the right-to-left shunt through the ventricular septal defect and improve the pulmonary circulation.



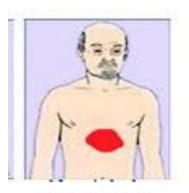
FETAL CIRCULATION



Cardiac Pain Oxygen deficiency and the accumulation of metabolites, are assumed to cause the pain originating in the heart as the result of acute myocardial ischemia. The nature of the pain varies considerably, from a severe crushing pain to nothing more than a mild discomfort. The pain is not felt in the heart but is **referred to** the skin areas supplied by the corresponding spinal nerves. The skin areas supplied by the upper four intercostal nerves and by the intercostobrachial nerve (T2) are therefore affected. The intercostobrachial nerve communicates with the medial cutaneous nerve of the arm and is distributed to the skin on the medial side of the upper part of the arm.



Myocardial infarction involving the inferior wall or diaphragmatic surface of the heart often gives rise to discomfort in the epigastrium. One must assume that the afferent pain fibers from the heart ascend in the sympathetic nerves and enter the spinal cord in the **posterior roots of the** seventh, eighth, and ninth thoracic spinal nerves and give rise to referred in the T7 to 9 thoracic pain dermatomes in the epigastrium. Because the heart and the thoracic part of the esophagus probably have similar afferent pain pathways, it is not surprising that painful acute esophagitis can mimic the pain of myocardial infarction.



Chest pain radiates to epigastrium



Epigastrium pain radiates to arms and neck

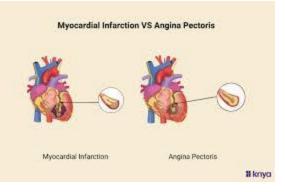
Coronary Artery Disease

The myocardium receives its blood supply through the right and left coronary arteries. A sudden block of one of the large branches of either coronary artery will usually lead to necrosis of the cardiac muscle (myocardial infarction) in that vascular area, and often, the patient dies.

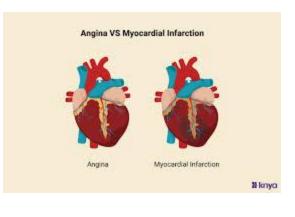
Arteriosclerotic disease of the coronary arteries may present in three ways, depending on the rate of narrowing of the Lumina of the arteries:



(1) general degeneration and fibrosis of the myocardium occur over many years and are caused by a gradual narrowing of the coronary arteries. (2) Angina pectoris is cardiac pain that occurs on exertion and is relieved by rest. In this condition, the coronary arteries are so narrowed that myocardial ischemia occurs on exertion but not at rest. (3) **Myocardial** infarction occurs when coronary flow is suddenly reduced or stopped and the cardiac muscle undergoes necrosis.

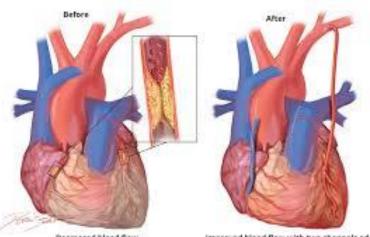


ANGINA VS MI



ANGINA VS MI

Myocardial infarction is the major cause of death in industrialized nations. Coronary bypass surgery, coronary angioplasty, and coronary artery stenting are now commonly accepted methods of treating coronary artery disease. Coronary bypass surgery involves getting a segment of blood vessel and using that to bypass a blockage in a coronary artery. Many such procedures use the great saphenous **vein** from the lower limb as the donor vessel because of its size and surgical ease of access. Emerging techniques increasingly use the internal thoracic **artery** from the neighboring chest wall to re-vascularize the heart wall.



Decreased blood flow

improved blood flow with two channels added

Coronary Artery Lesions, Infarct Location, and ECG signature

| CORONARY ARTERY | INFARCT LOCATION | ECG SIGNATURE |
|--|---|---|
| Proximal LAD | Large anterior wall | ST elevation: I, L, V1 to 6 |
| More distal LAD | Anteroapical Inferior wall if wraparound LAD | ST elevation: V2 to 4 ST elevation: II, III, F |
| Distal LAD | Anteroseptal | ST elevation: V1 to 3 |
| Early obtuse, marginal | High lateral wall | ST elevation: I, L, V4 to 6 |
| More distal marginal branch, circumflex | Small lateral wall | ST elevation: I, L, or V4 to 6 or no abnormality |
| Circumflex | Posterolateral | ST elevation: V4 to 6; ST depression: V1 to 2 |
| Distal RCA | Small inferior wall | ST elevation: II, III, F; ST depression: I, L |
| Proximal RCA | Large inferior wall and posterior wall Some lateral wall | ST elevation: II, III, F; ST depression: I, L, V1 to 3 ST elevation: V5 to 6 |
| RCA | Right ventricular Usually inferior | ST elevation: V2R to V4R; some ST elevation: V1; or ST depression V2, V3 ST elevation: II, III, F |

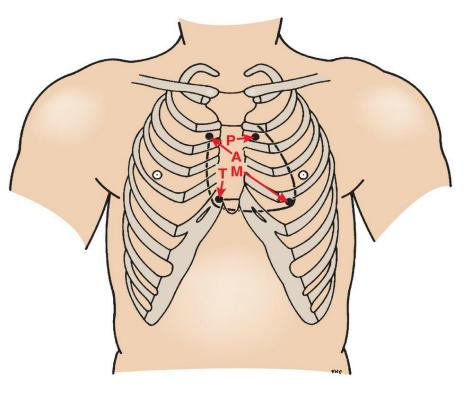
SurfaceProjectionsandAuscultation of the Heart

Valves

The cardiac valves are located deep to the sternum. **Each valve has an anatomical projection to a place on the thoracic wall** that immediately overlies its position. However, the sounds produced by the valves are projected to auscultation areas that are removed from the anatomical projections and widely separated from each other.

This difference is because blood carries sound along the direction of its flow. Thus, each auscultation area is located superficial to the heart chamber or great vessel into which the blood flows after passing each valve. Table

5.7 summarizes the anatomical and auscultation projection areas for each valve.



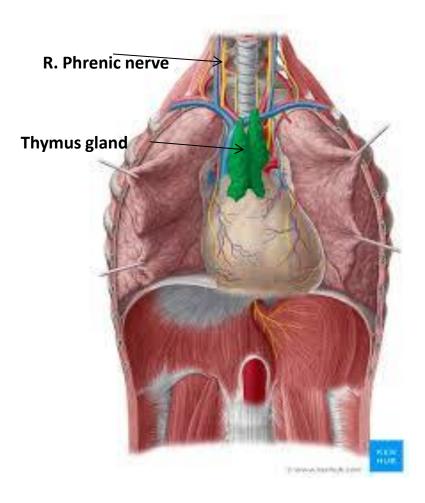
Valvular Heart Disease

Inflammation of a valve can cause the edges of the valve cusps to stick together. Later, fibrous thickening occurs, followed by loss of flexibility and shrinkage. Narrowing (stenosis) and valvular incompetence (regurgitation) result, and the heart ceases to function as an efficient pump. In rheumatic disease of the mitral valve, not only do the cusps undergo fibrosis and shrink but the chordae tendineae shorten as well, preventing closure of the cusps during ventricular systole.

| VALVE | ANATOMIC LOCATION | OPTIMAL AUSCULTATION AREA |
|-----------|--|--|
| Tricuspid | Behind the right half of the sternum opposite the fourth intercostal space | Over the right half of the lower end of the body of the sternum |
| Bicuspid | Behind the left half of the sternum opposite the fourth costal cartilage | Over the apex beat, that is, at the level of the fifth left intercostal space, 3.5 in. (9 cm) from the midline |
| Pulmonary | Behind the medial end of the third left costal cartilage and the adjoining part of the sternum | Over the medial end of the second left intercostal space |
| Aortic | Behind the left half of the sternum opposite the third intercostal space | Over the medial end of the second right intercostal space |

THYMUS

The thymus is a flattened, bi-lobed structure lying between the sternum and the pericardium in the anterior mediastinum. l† reaches its largest size elative to the size of the body in the newborn infant, at which time it may extend up through the superior mediastinum in front of the great vessels into the root of the neck. The thymus continues to grow until puberty but thereafter undergoes involution. It has a pink, lobulated appearance and is the site for development of T (thymic) lymphocytes.



Thank you