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GROSS ANATOMY OF THE HUMAN HEART: AN INTEGRATED REVIEW WITH CLINICAL CORRELATIONS

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ABSTRACT

The human heart, a hollow muscular organ situated in the middle mediastinum, functions as the central pump of the circulatory system. Its gross anatomical features—including chambers, surfaces, valves, conduction system, and coronary circulation—form the foundation for understanding cardiovascular physiology and the pathogenesis of major cardiac diseases. A thorough anatomical knowledge of the heart is not only of academic significance but also of immense clinical importance, as it underpins diagnostic interpretation, interventional planning, and surgical procedures. This review presents a comprehensive overview of the gross anatomy of the heart with special emphasis on its clinical correlations. The right and left atria, ventricles, and their associated valves are described along with relevant conditions such as atrial septal defects, tricuspid and mitral valve disease, right and left ventricular hypertrophy, and heart failure. The conduction system, comprising the sinoatrial node, atrioventricular node, bundle of His, and Purkinje fibers, is explored with reference to arrhythmias and conduction blocks. Coronary circulation is detailed to highlight its relevance in ischemic heart disease, angina pectoris, and myocardial infarction. In addition, the role of applied imaging and modern diagnostic tools such as electrocardiography, echocardiography, cardiac biomarkers, cardiac catheterisation, CT angiography, and chest radiography is outlined, demonstrating how anatomical knowledge translates into clinical application. By integrating anatomical concepts with contemporary diagnostic and therapeutic advances, this article emphasizes the continuing relevance of cardiac anatomy as the cornerstone of clinical cardiology and surgical practice.

KEYWORDS: Gross anatomy, Heart, Coronary circulation, Valvular disease, Clinical relevance.

INTRODUCTION

The Heart is a hollow muscular organ located in the middle mediastinum within the pericardium, functioning as the central pump of the circulatory system. Its anatomy is crucial for understanding cardiovascular physiology and the pathogenesis of cardiac diseases. Detailed anatomical knowledge guides clinicians, cardiologists, and surgeons in diagnosis, imaging interpretation, interventions, and surgeries. Clinically, conditions like coronary artery disease, valvular defects, congenital malformations, and arrhythmias highlight the importance of precise understanding of cardiac chambers, valves, coronary circulation, and the conduction system.

AIMS AND OBJECTIVES

1. To describe the structural organization of the heart including its surfaces, borders, chambers, valves, conducting system, and coronary circulation.

2. To correlate the anatomical features of the heart with common clinical conditions such as valvular diseases, conduction abnormalities, ischemic heart disease, and heart failure.
3. To highlight the significance of anatomical knowledge in the interpretation of modern diagnostic tools such as ECG, echocardiography, CT angiography, and cardiac catheterisation.
4. To emphasize the continuing importance of cardiac anatomy as the foundation for cardiology, surgical interventions, and contemporary diagnostic and therapeutic advances.

MATERIALS AND METHODS

1. The article has been prepared as a narrative review.
2. Standard anatomy textbooks such as Gray's Anatomy, Snell's Clinical Anatomy, Moore's Clinically Oriented Anatomy and Inderbir Singh's Human Anatomy were referred for gross anatomical details.
3. Clinical reference books including Harrison's Principles of Internal Medicine, Davidson's Principles and Practice of Medicine and Robbins & Cotran Pathologic Basis of Disease were consulted for applied and pathological aspects.
4. The information collected was systematically organized into sections on anatomy and clinical relevance.

Literary Review

A. Introduction

The heart is a conical hollow muscular organ that is somewhat pyramid shaped and lies within the pericardium in the middle mediastinum. It is connected at its base to the great blood vessels but otherwise lies free within the pericardium.[1] The Greek name for the heart is **Cardia** from which we have the adjective cardia. The heart is referred to as Cor in Latin, from which the term 'coronary' is derived.[2]

B. Surfaces and Borders of the Heart

i. The heart has three main surfaces—sternocostal (anterior), diaphragmatic (inferior), and base (posterior)—along with an apex.
ii. The sternocostal surface is primarily formed by the right atrium and right ventricle, which are separated by the coronary (atrioventricular) sulcus, while the two ventricles are delineated by the anterior interventricular (IV) groove. The diaphragmatic surface consists mainly of both ventricles and part of the right atrium.
iii. The base, opposite the apex, is formed chiefly by the left atrium receiving four pulmonary veins.
iv. The apex, formed by the left ventricle, lies in the 5th left intercostal space, where the apex beat is palpable.
v. The right border is formed by the right atrium, and the left border by the left ventricle and part of the left auricle, important landmarks on chest radiographs.[3]

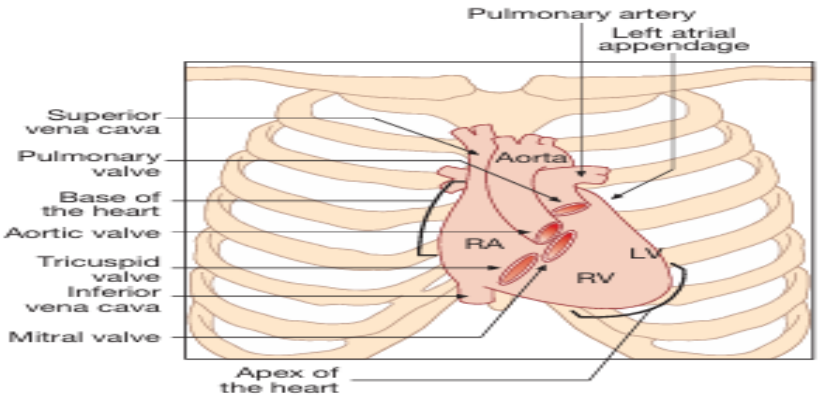

Fig.1: Position of the heart.[4]

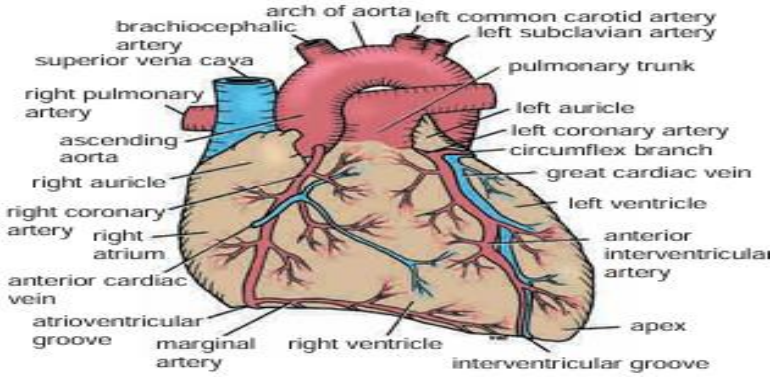

Fig.2: Anterior surface of the heart.

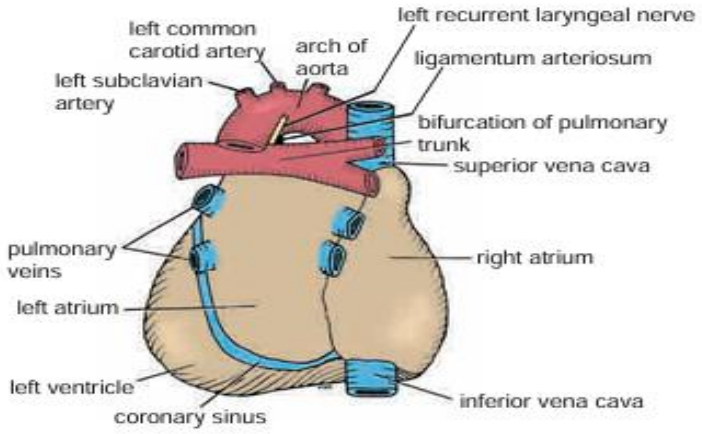

Fig.3: Posterior surface or Base of the heart.[5]

C. Chambers of the Heart

Right Atrium

The right atrium consists of a main cavity and auricle. The crista terminalis separates the smooth posterior sinus venarum from the trabeculated anterior part with musculi pectinati. Openings include the SVC (no valve), IVC (rudimentary valve), coronary sinus (valve present), and small cardiac veins. The right atrioventricular orifice, guarded by the tricuspid valve, leads into the right ventricle.[6]

Clinical Aspects

- Tricuspid stenosis → right heart failure with hepatic discomfort & edema.
- Right Atrial Enlargement (RAE**)** → pulmonary hypertension or tricuspid disease; ECG shows tall P waves.[7]
- Atrial Septal Defect (ASD) → persistent foramen ovale → RA volume overload.[8]
- RA Thrombosis → with catheters, AF, or low-flow states.[9]
- SVC/IVC obstruction → impaired venous return, engorged neck veins, edema.[10]

Right Ventricle

The right ventricle communicates with the right atrium via the tricuspid valve and with the pulmonary trunk via the pulmonary valve. Its upper funnel-shaped part is the infundibulum. The wall has trabeculae carneae, including papillary muscles (attached to cusps via chordae tendineae), the moderator band (carrying the right AV bundle branch), and muscular ridges. The tricuspid valve has three cusps (anterior, septal, posterior) anchored by chordae tendineae to prevent prolapse. The pulmonary valve has three semilunar cusps (two anterior, one posterior) with sinuses that open in systole and close in diastole.[11]

Clinical Aspects

- **RV Hypertrophy (RVH):** Pulmonary hypertension, TOF, or chronic lung disease; ECG → right axis.[12]
- **RV Failure (Cor Pulmonale):** From chronic lung disease/pulmonary HTN → venous congestion, hepatomegaly, edema.[13]
- **TOF:** Congenital → RV outflow obstruction, RVH, overriding aorta, VSD.[14]
- **Tricuspid valve disease:** Regurgitation/stenosis → RA & RV dilatation.[15]
- Right Ventricular Outflow Tract Obstruction (Pulmonary Stenosis**)** Infundibular/pulmonary valve narrowing → RVH, cyanosis.[16]

Left Atrium

The left atrium, forming most of the heart's base, lies anterior to the esophagus. Its wall is mostly smooth, except for ridges in the auricle. Four pulmonary veins (two from each lung) open into its posterior wall without valves. The left atrioventricular orifice is guarded by the mitral valve.[17]

Clinical Aspects

- Mitral valve disease**:** Stenosis/regurgitation → LA dilatation, pulmonary congestion, AF.[18]
- AF from ectopic foci near pulmonary veins → irregular rhythm, thrombus risk.[19]
- Pulmonary venous obstruction/HTN: Impaired LA drainage → congestion, dyspnea, hemoptysis.[20]

Left Ventricle

The left ventricle, the systemic pump, connects to the atrium via the mitral valve and to the aorta via the aortic valve. Its wall is three times thicker than the right ventricle, producing much higher pressure, and appears circular in cross-section. Internally, it has trabeculae carneae, two papillary muscles, and an aortic vestibule (no moderator band). The mitral valve has two cusps supported by chordae and papillary muscles. The aortic valve has three cusps with sinuses; the right and left coronary arteries arise from the anterior and left posterior sinuses.[21]

Clinical Aspects

a) Left Ventricular Hypertrophy (LVH)

- Cause: systemic hypertension, aortic stenosis (pressure overload).
- Effect: increased wall thickness, diastolic dysfunction.[22]

b) Coronary Artery Disease & Infarction

- LV most commonly affected in myocardial infarction.
- Leads to wall motion defects, aneurysm, mural thrombus.[23]

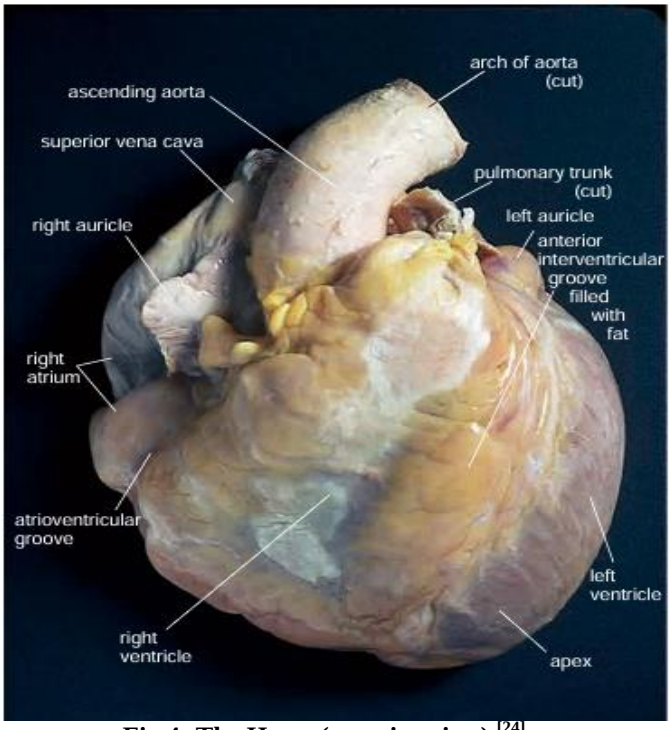

Fig.4: The Heart (anterior view).[24]

D. Conducting System of the Heart

The heart's conducting system comprises specialized muscle in the SA node, AV node, and AV bundle. The SA node, the natural pacemaker, initiates impulses that spread through the atria to the AV node (above the coronary sinus). From there, the bundle of His carries impulses into right and left branches beneath the endocardium, activating ventricular muscle via Purkinje fibers.

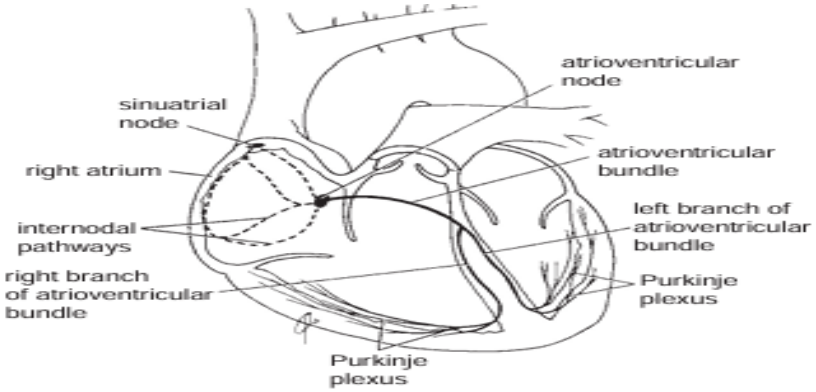

Fig.5. Conducting System of the Heart.[25]

Clinical apects

- **Conduction system failure:** SA node initiates impulse; AV node & bundle transmit to ventricles. Block/dysfunction → arrhythmias or AV dissociation, usually from coronary atherosclerosis.

- **Commotio cordis:** Sudden blunt chest blow (sports-related) → VF and sudden death, especially in youth due to compliant chest; risk highest if impact occurs during T-wave upstroke.[26]

E. Coronary Circulation/ The blood supply to the Heart

The heart is supplied by the right and left coronary arteries lying in the interventricular and atrioventricular grooves.

- The right coronary artery (from anterior aortic sinus) runs in the right AV groove, giving a marginal branch and the posterior interventricular branch, and usually supplies the SA node.
- The left coronary artery (from left posterior aortic sinus) divides into the anterior interventricular (LAD) and circumflex branches. The LAD supplies the anterior ventricles and apex, while the circumflex runs in the left AV groove. Coronary anastomoses exist but are functionally poor; thrombosis causes myocardial infarction.[27]

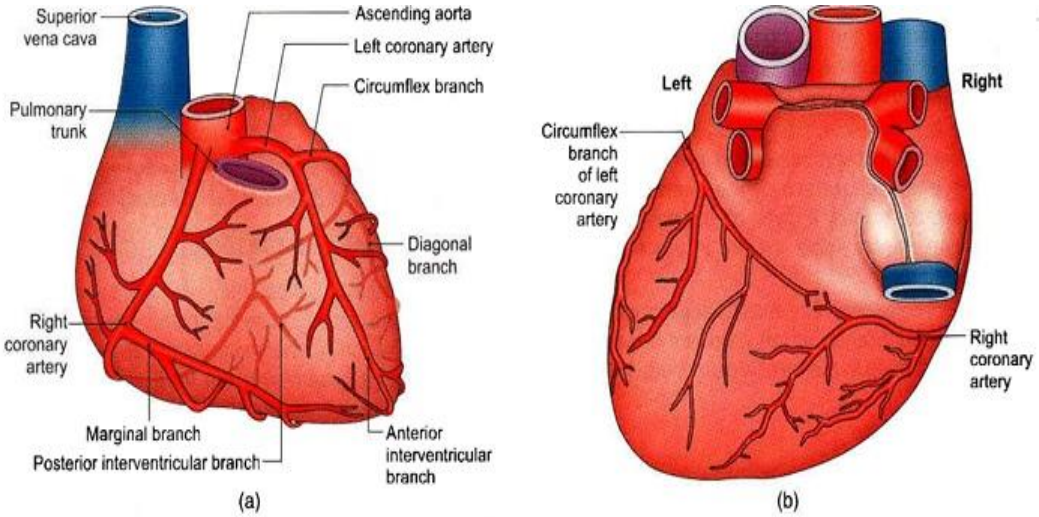

Fig.6: Arterial supply of heart: (a) Sternocostal surface, and (b) diaphragmatic surface.[28]

Clinical Apects

RCA supplies SA node (60%) & AV node (90%); its occlusion → sinus bradycardia or AV block. **RCA thrombosis** → inferior LV ± RV infarction. **LAD/Cx occlusion** → LV infarction in their territories; **left main occlusion** → usually fatal.[29]

- Thrombosis of coronary artery is a common cause of sudden death in persons past middle age. This is due to myocardial infarction and ventricular fibrillation.
- Angina pectoris: Exertional chest pain from coronary narrowing/artery spasm → myocardial ischemia. Pain radiates to left arm/forearm, relieved by rest or sublingual medication.[30]

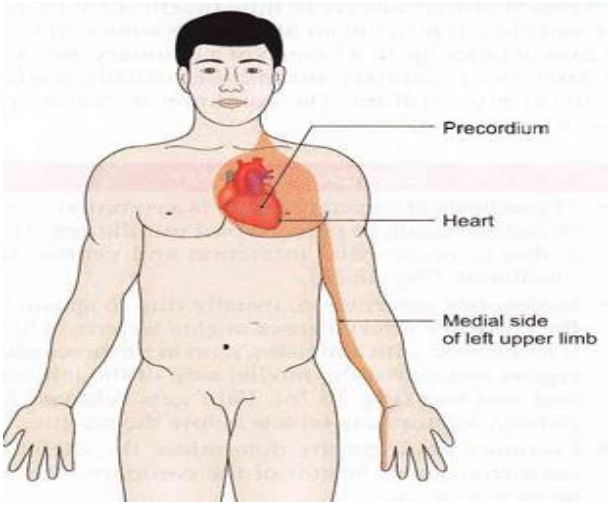

Fig.7: Pain of angina pectoris felt in precordium along medial border of left arm.[31]

Investigation or procedures to be done

i. **Coronary angiography** determines the site of narrowing or occlusion of the coronary arteries or their branches.
ii. **Angioplasty** helps in removal of small blockage. The procedure involves advancing a catheter via the femoral artery and aorta into the coronary artery, through which a small stent or an inflatable balloon is deployed

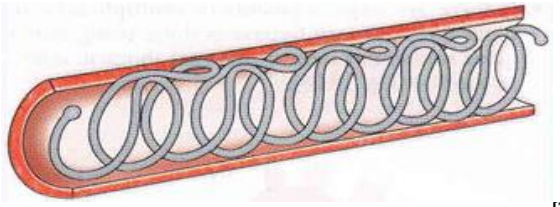

Fig. 8: Stent passed in the blocked coronary artery.[32]

iii. If there are large segments or multiple sites of blockage, coronary bypass is done using either great saphenous vein or internal thoracic artery as graft(s).

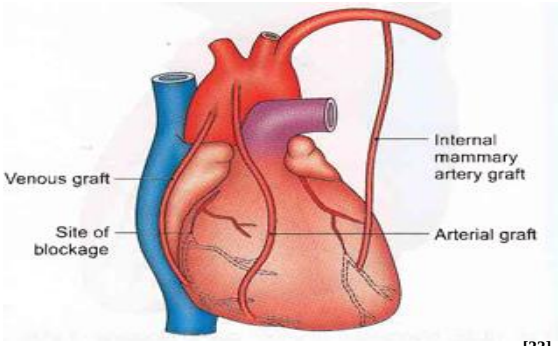

Fig.9: Grafts put beyond the site of blockage.[33]

F. Valves of the Heart

Feature	Tricuspid Valve (Right AV)	Mitral Valve (Left AV)	Semilunar Valves (Aortic & Pulmonary)
Number of cusps	3 (anterior, posterior, septal)	2 (anterior/aortic, posterior)	3 semilunar cusps each
Fibrous ring	Present	Present	Absent – cusps attached directly to vessel wall
Papillary muscles	3 (anterior largest, posterior smaller, septal rudimentary)	2 (anterior & posterior)	Absent
Chordae tendineae	Present – attach cusps to papillary muscles	Present – attach cusps to papillary muscles	Absent
Cusps size	Larger, thinner	Smaller, thicker	Thin, pocket-shaped
Phase of closure	Ventricular systole	Ventricular systole	Ventricular diastole
Special feature	Cusps oppose along serrated margins	Anterior cusp lies between mitral & aortic orifice	Sinuses of Valsalva formed; coronary arteries arise from aortic sinuses

Clinical Aspects

- 1st heart sound**:** Closure of AV valves. 2nd sound: Closure of semilunar valves.
- Stenosis: Narrowed valve orifice (e.g., mitral/aortic stenosis).
- Regurgitation: Incomplete valve closure → backflow (e.g., aortic regurgitation).

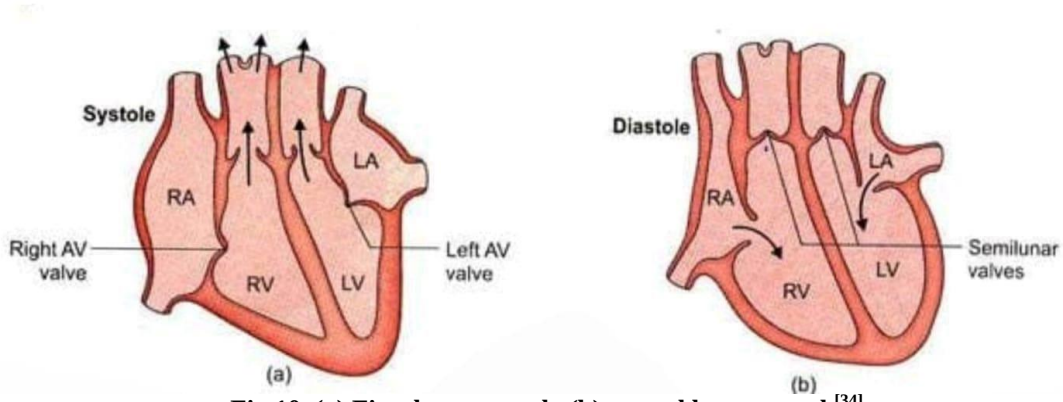

Fig.10. (a) First heart sound (b) second heart sound.[34]

- **Valvular Heart Murmurs** -Apart from the sounds of the valves closing, lub-dup, the blood passes through the normal heart silently. Should the valve orifices become narrowed or the valve cusps distorted and shrunken by disease, however, a rippling effect would be set up, leading to turbulence and vibrations that are heard as heart murmurs.[35]

G. Applied Imaging/Investigation and Modern Relevance

1. Electrocardiogram

- MI (Transmural): ST elevation → later Q waves.
- Ischaemia (Subendocardial): ST depression ± T-wave inversion.
- Mimics: LVH, electrolyte imbalance may cause similar ST-T changes.

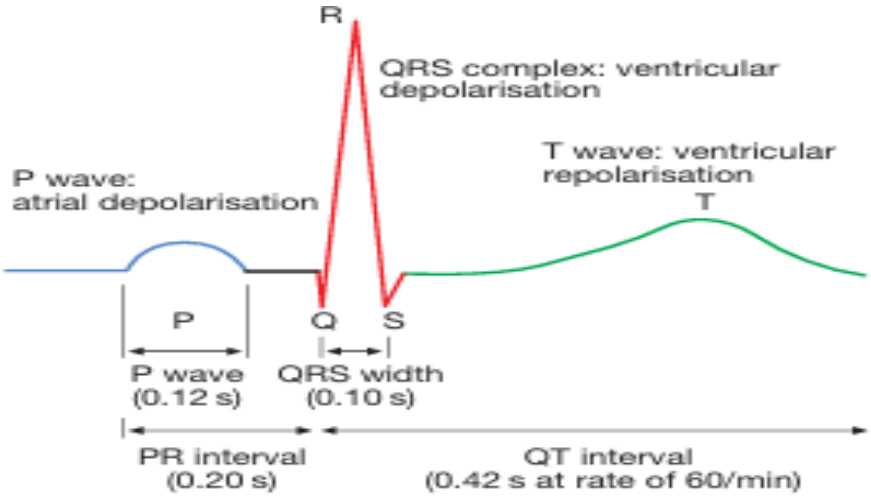

Fig.11: The electrocardiograph.[36]

2. Exercise (Stress) ECG

- **Purpose:** Detect ischaemia/CAD.
- **Method:** 12-lead ECG on treadmill/bicycle (Bruce protocol), with BP & symptom monitoring.
- **Uses:** Angina diagnosis, post-MI prognosis, post-revascularisation follow-up, exercise arrhythmias.
- **High-risk signs:** Ischaemia at low workload, BP fall, marked ST changes, exercise arrhythmia.
- **Positive test:** Angina, BP fall/no rise, ST shift >1 mm.
- **Limitations:** False +ve (young women), false –ve in CAD, not for screening.
- **Contraindications:** ACS, decompensated HF, severe HTN.[37]

3. Cardiac Biomarkers

- **BNP / NT-proBNP:** Released from LV in systolic dysfunction; ↑ levels indicate HF (diagnosis, prognosis, therapy response). NT-proBNP more reliable (longer half-life).
- **Cardiac Troponins (I & T):** Gold standard for MI; detect even minor myocyte injury. May also rise in PE, sepsis, pulmonary edema → interpret with clinical context.[38]

4. Chest X-ray in Cardiac Disease

- **Use:** Assess heart size/shape, pulmonary vessels, lungs.
- **Projection:** PA preferred; AP magnifies heart.
- **CTR >0.5 = cardiomegaly** (LV dilatation, pericardial effusion). Beware false +ve/–ve.

- **Chamber dilatation**
  - RA → right border bulge.
  - RV → enlarged heart, apex up, straight left border.
  - LA → straight left border, double shadow, wide carina.
  - LV → enlarged silhouette, prominent/rounded border.

- **Other:** Pericardial calcification, pulmonary edema, plethora (L→R shunt), pleural effusion.

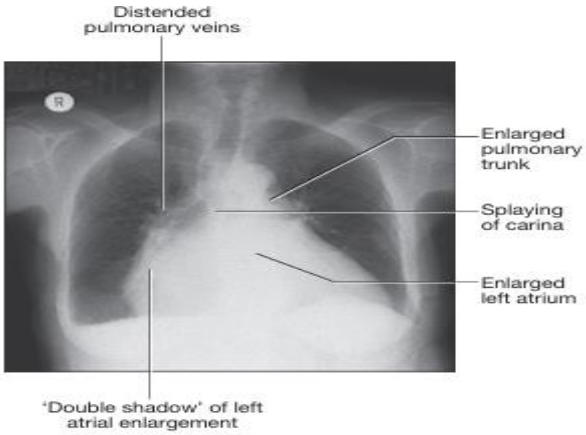

Fig.12. Chest X-ray of a patient with mitral stenosis and regurgitation (indicating enlargement of the LA and prominence of the pulmonary artery trunk).[39]

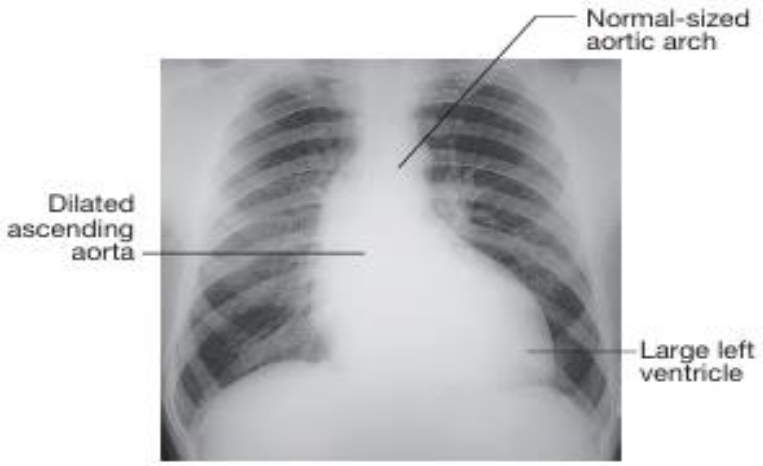

Fig.13. Chest X-ray of a patient with aortic regurgitation, left ventricular enlargement and dilatation of the ascending aorta.[40]

5. Common indications for echocardiography (Ultrasound imaging of the heart)

- Diagnose & grade valve disease.
- Detect vegetations (endocarditis).
- Identify structural disease (AF, cardiomyopathy, CHD); assess LV function.
- Detect pericardial effusion.
- **Identify thrombus / structural cause in systemic embolism.**

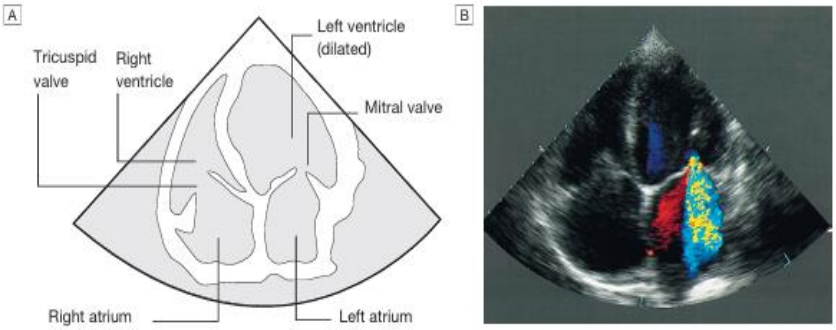

Fig.14: (A). Echocardiographic illustration of the major chambers and valves. (B) Colour - flow Doppler has been used to demonstrate mitral regurgitation: a flame-shaped (yellow/blue) turbulent jet into the left atrium.

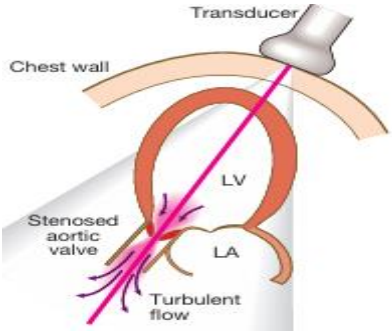

Fig.15: The aortic valve is imaged and a Doppler beam passed directly through the left ventricular outflow tract and the aorta into the turbulent flow beyond the stenosed valve.[41]

6. Computed Tomographic (CT) Imaging

- **Technique:** Multidetector CT + contrast → high-resolution vascular imaging.
- **Uses**
- **Aorta:** Dissection.
- **Pulmonary arteries:** Embolism.
- **Coronaries:** Calcium scoring (risk), CTCA (non-invasive, high NPV for CAD).
- **Myocardium:** Perfusion with volume scanners.

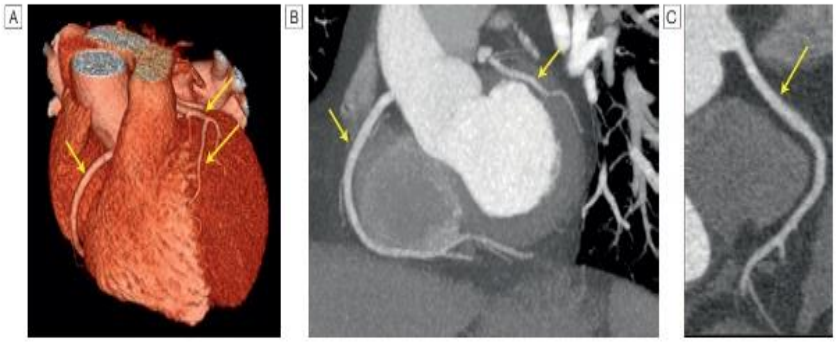

Fig.16: CT coronary angiography demonstrating normal coronary arteries (arrows).[42]

7. Cardiac Catheterisation

- **Method:** Preshaped catheter via vein/artery under X-ray → measures pressures, O_2 sat, contrast angiography.
- **Left heart cath:** Radial access; coronary angiography (gold standard for stenosis/thrombus, guides PCI/CABG); assesses LV size/function, MR, aortic root. Safe, day-case (<1/1000 serious events).
- **Right heart cath:** Measures RA/RV/PA pressures; detects shunts (O_2 step-up); cardiac output (thermodilution); PCWP (Swan–Ganz) → indirect LA pressure, critical care use.[43]

DISCUSSION

The present review emphasizes the importance of gross anatomical features of the human heart and their direct clinical relevance. While traditional anatomical descriptions focus on structural details, their integration with clinical cardiology provides a deeper understanding of pathophysiology and patient care. The description of cardiac chambers, valves, conduction pathways, and coronary circulation highlights how subtle anatomical variations may predispose to clinical disorders such as valvular heart disease, arrhythmias, or ischemic events.

Our review consolidates both anatomical and clinical aspects into a single framework, thereby bridging the gap between basic science and clinical practice. Existing literature has often presented these topics separately; however, their integration enhances diagnostic accuracy and therapeutic decision-making. For instance, knowledge of atrioventricular and semilunar valves is crucial not only in identifying murmurs but also in planning surgical interventions. Similarly, appreciation of coronary artery distribution guides cardiologists in angiographic interpretation and revascularization procedures.

The discussion also underscores the significance of modern diagnostic modalities—ECG, echocardiography, CT angiography, and cardiac catheterisation—as applied extensions of anatomical concepts. The clinical correlation of these tools demonstrates that anatomy remains the cornerstone of modern cardiology and cardiovascular surgery.

Nevertheless, this review is limited by its narrative design and reliance on standard textbooks. Future

research may incorporate advanced imaging, 3D modeling, and cadaveric correlation studies to provide more dynamic insights. Despite these limitations, the review reiterates that gross anatomy of the heart continues to be of paramount importance for medical students, clinicians, and researchers alike.

CONCLUSION

The gross anatomy of the heart underpins cardiovascular physiology and clinical practice. Detailed knowledge of chambers, valves, conduction system, and coronary circulation is vital for interpreting ECG, echocardiography, CT angiography, and catheterisation. Awareness of anatomical variations aids in diagnosing and managing coronary artery disease, valvular defects, congenital anomalies, arrhythmias, and heart failure. Thus, cardiac anatomy remains academically important and clinically relevant, linking basic science with modern imaging, interventions, and surgery.

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