Rapid Review of ECG

Self-Assessment Questions, Case Studies and Clinical Correlation

Tapas Kumar Koley

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Preface

Welcome to the exciting world of electrocardiography (ECG), an invaluable tool in the realm of cardiovascular medicine. ECG plays a vital role in patient care. This book has been meticulously crafted to provide you with a comprehensive understanding of ECG interpretation, while also offering opportunities for self-assessment, practical case studies, and meaningful clinical correlations.

In the fast-paced environment of healthcare, the ability to swiftly interpret an ECG is a skill that can make a significant difference in patient outcomes. As healthcare professionals, we are constantly faced with the challenge of identifying and diagnosing cardiac abnormalities in a timely manner. This book aims to equip you with the knowledge and confidence needed to interpret ECGs rapidly and effectively.

To facilitate your learning, the book has been divided into seven parts. There are a total of twenty nine chapters, and we have included self-assessment questions throughout the book. These questions serve as checkpoints, allowing you to gauge your understanding of the material covered and reinforce key concepts. The answers and explanations provided will help solidify your knowledge and address any areas that may require further attention.

In addition to self-assessment questions, this book offers a range of case studies that present real-life scenarios encountered in clinical practice. Each case study provides an ECG, accompanied by relevant clinical information. By analyzing these cases, you will have the opportunity to apply your knowledge to practical situations, honing your skills in interpreting ECGs in a clinical context. The comprehensive explanations and discussions following each case will further enhance your understanding and highlight the importance of ECG interpretation in patient care.

Understanding the clinical relevance of ECG findings is crucial for accurate diagnosis and management. Therefore, throughout this book, we emphasize the correlation between ECG findings and various cardiac conditions. By exploring the connections between ECG abnormalities and underlying pathophysiology, you will develop a holistic approach to interpreting ECGs and appreciate the clinical significance of each finding.

We recognize that mastering ECG interpretation is not a solitary pursuit but rather an ongoing process. As you progress through this book, we encourage you to engage in active learning, seeking opportunities to practice your skills and seek guidance from experienced clinicians. Remember, proficiency in ECG interpretation comes with practice and exposure to diverse clinical scenarios.

We hope that this book serves as a valuable resource in your journey to becoming a proficient ECG interpreter. Our aim is to provide you with the knowledge, self-assessment tools, case studies, and clinical correlations necessary to enhance your expertise in this essential skill. We wish you success in your endeavours and hope that the knowledge gained from this book translates into improved patient care.

New Delhi, India Tapas Kumar Koley

Acknowledgments

Writing this ECG book has been a challenging yet rewarding journey, and I am profoundly grateful to all those who have contributed their time, expertise, and support to make this project a reality. Without their dedication and encouragement, this endeavour would not have been possible.

Thank you to the esteemed professors and experts in cardiology for sharing their knowledge, to my colleagues and collaborators for their support, and to the patients and participants for their valuable contributions. Special thanks to the reviewers, editors, and publishing team for their efforts. I am also thankful to my family, friends, and all the readers for their encouragement. Your support has been instrumental in making this book possible and advancing the understanding of electrocardiography.

To everyone who has been part of this project in one way or another, I offer my sincerest thanks. Your contributions have shaped this book and will undoubtedly impact the understanding and practice of ECG interpretation for years to come.

With utmost appreciation,

Tapas Kumar Koley

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About the Author

Tapas Kumar Koley, MBBS, MD Medicine graduated from Burdwan Medical College and completed his postgraduation at the Postgraduate Institute of Medical Sciences, Rohtak, India. He has several articles and international book publications and has delivered numerous presentations at conferences and CMEs. He has been an ECG tutor for All About Staffing, an affiliate of Hospital Corporation of America. He is the resource person for the National Institute of Health and Family Welfare, India, to train doctors on medical negligence. He is working as Senior Specialist Physician at Kasturba Hospital, Delhi.

Abbreviations

APC	Atrial premature complex
ASD	Atrial septal defect
AV	Atrioventricular
BBB	Bundle branch block
BPM	Beats per minute
CAH	Combined atrial hypertrophy
CVH	Combined ventricular hypertrophy
ECG	Electrocardiograph (UK)
EKG	Electrokardiograph (USA)
LA	Left atrium
LAD	Left axis deviation
LAH	Left atrial hypertrophy
LBBB	Left bundle branch block
LGL	Lown-Ganong-Levine
LV	Left ventricle
LVH	Left ventricular hypertrophy
PAT	Paroxysmal atrial tachycardia
PCI	Percutaneous coronary intervention
PDA	Patent ductus arteriosus
PSVT	Paroxysmal supraventricular tachycardia
Q-Tc	Corrected Q-T interval
RA	Right atrium
RAD	Right axis deviation
RAH	Right atrial hypertrophy
RBBB	Right bundle branch block
RV	Right ventricle
RVH	Right ventricular hypertrophy
SA	Sinoatrial
SSS	Sick sinus syndrome

SVT	Supraventricular tachycardia
VF	Ventricular fibrillation
VSD	Ventricular septal defect
VT	Ventricular tachycardia
WPW	Wolff-Parkinson-White

Part I
Basic Principles

Chapter 1
Cardiac Electrophysiology

Learning Objectives
After studying this chapter, the reader will learn about:

- Historical perspective
- Electrophysiology
- Origin of intracellular potential
- Action potential
- Generation of ECG
- Conduction of electrical impulse

Electrocardiogram (abbreviated as ECG or EKG in some countries) is the graphical recording of the change in potentials of the electrical field produced by the heart. The word ECG is derived from the German language. In German, it is elektrokardiographie. As English is more dominant in today's world, the acronym ECG is more widely used today. It is recorded by means of metal electrodes connected by cables to an ECG machine. The recorded electrical activity is depicted as a series of graph-like tracings, or waves in a paper or digitally displayed in a screen. The shapes and frequencies of these tracings reveal cardiac abnormalities.

Today ECG is one of the most important tools to study the cardiac anatomy and physiology. It helps in diagnosis of various cardiac abnormalities including myocardial infarction and ischaemia, cardiac arrhythmias, conduction defects, myocardial hypertrophy and congenital heart diseases. The fact that ECG is only a laboratory test should always be borne in mind. Normal ECG may be observed in patients with heart disease and normal healthy persons may have a nonspecific change in ECG.

Tips and Tricks of ECG

- The ECG should be read and analysed only after examination of the patient and should be interpreted based on the clinical findings.

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1.1 Historical Perspective

In May 1887, Augustus D Waller published the first single lead ECG with a capillary electrometer. He, however, failed to realize the clinical importance and significance of his discovery. Willem Einthoven, considered the father of electrocardiography, worked in The Netherlands for years, first with an electrometer and later with a string galvanometer to generate clinically relevant ECG in the early 1900s. He coined the term *elektrokardiogramm* in German and subsequently labelled the waveforms as P, Q, R, S, T and U. He described numerous abnormalities in this field and created the bipolar lead system.

The early years of studies in ECG were dominated by Einthoven and Sir Thomas Lewis, who are credited with bringing the ECG from the laboratory to the bedside of the patient. After several years of such efforts, the ECG gained importance in clinical practice and was accepted as a useful tool in cardiac evaluation. In 1924, Einthoven was awarded the Nobel Prize for his invaluable contribution to the field. Initially, the ECG machines were huge and required the help of several persons to operate them. Subsequent developments over several decades have resulted in the current computerized versions of the ECG machine. More than a century has elapsed after the first recording of ECG, yet the standard 12-lead ECG is still an essential part of complete cardiac evaluation.

1.2 Electrophysiology

The human heart lies behind the sternum in the mediastinal cavity (Fig. 1.1). Together with the blood vessels and blood, it constitutes the body's circulatory system. The top of the heart known as the base is located at the level of the second

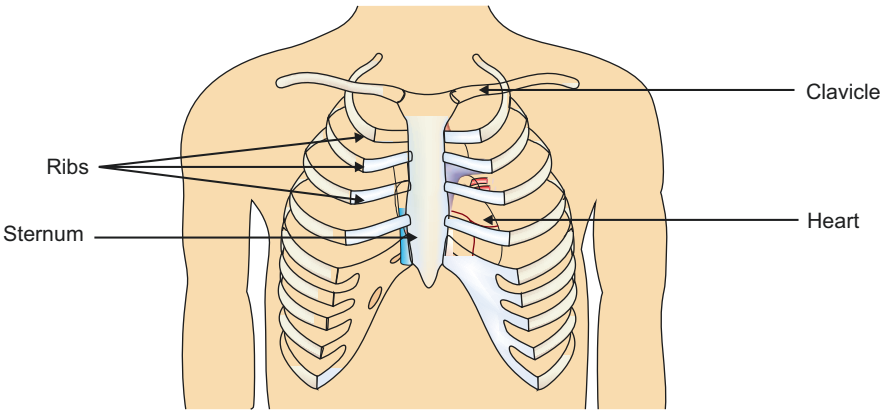

Fig. 1.1 Position of human heart in the mediastinal cavity

intercostal space, while the bottom of the heart, called the apex, is tilted to the left side of the body and rests on the diaphragm. It is well protected inside the rib cage. The heart is made up of four chambers: right atrium, left atrium, right ventricle and left ventricle. Interatrial septum separates the two atria and the two ventricles are separated by interventricular septum (Fig. 1.2). From electrophysiological angle, the heart is made up of only two chambers. The two atria form one chamber and the other chamber is formed by the two ventricles. The two atria may be considered as a single electrophysiological unit because they are activated by a single process of activation; similarly, the two ventricles may also be considered a single electrophysiological unit, as they are also activated by a single activation process. Electrophysiologically, the left ventricle together with the interventricular septum is the most important structure. The thickness of left ventricle is almost thrice that of the right ventricle, which makes the right ventricle less significant haemodynamically, electrophysiologically and electrocardiologically.

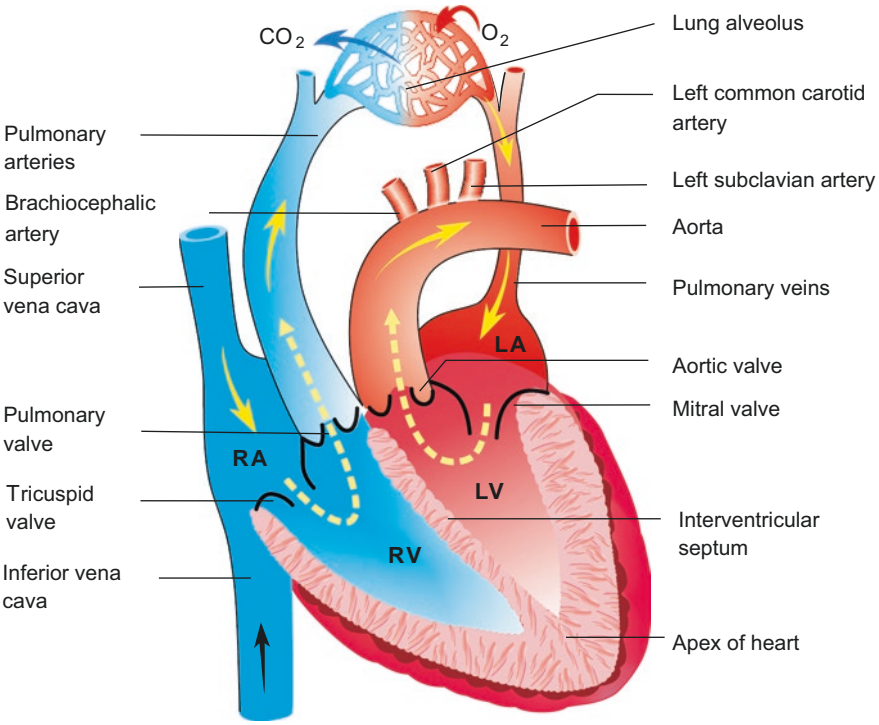

Fig. 1.2 Anatomy of human heart

1.2.1 Generation of Intracellular Potential

To understand the origin and conduction of electrical impulse, it is important to know the four primary properties of cardiac myocytes. These are:

- Automaticity: It is the ability of the cells to generate electrical impulse spontaneously. This property is specific for pacemaker cells, for example, cells of SA node.
- Excitability: It is the ability of cardiac myocytes to respond to an electric impulse, which results from shifting of ions across the cell membrane.
- Conductivity: It is the ability of cardiac myocytes to receive and further transmit electric impulse to other cells.
- Contractility: It is the ability to shorten and cause muscle contraction.

Out of these four properties, only contractility is the mechanical function of the heart, and the other three are electrical functions of the heart.

1.2.1.1 Sodium Potassium Pump

Sodium potassium pump (Fig. 1.3) maintains a potential difference of −90 mV across the cardiac cell membrane. This is known as resting membrane potential and the main factor responsible for this is the gradient of potassium ions (K^+) across the cell membrane. The extracellular K^+ ion concentration is about 5 mEq/L, and the

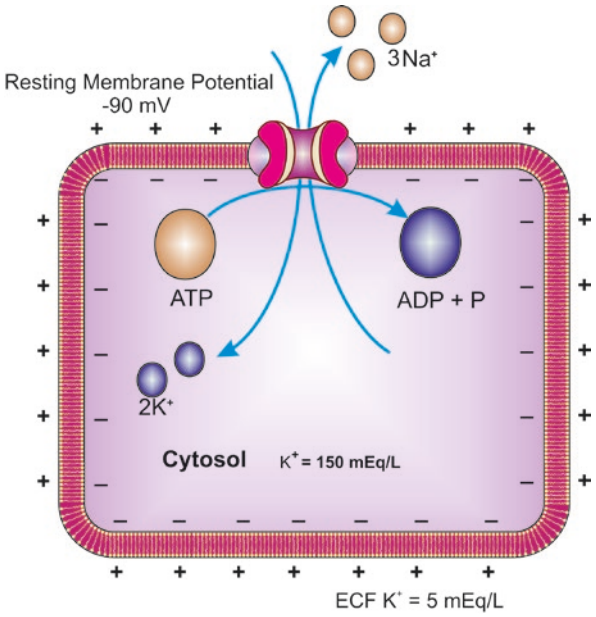

Fig. 1.3 Sodium potassium pump

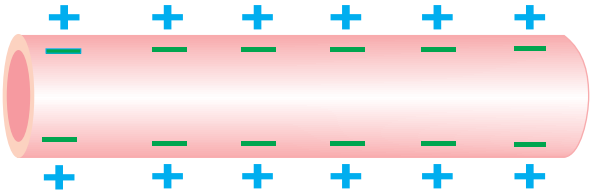

Fig. 1.4 Myocardial cell (cardiac myocyte). At resting state, the inside of cell is negatively charged and the outside is positively charged

intracellular concentration is 150 mEq/L. This 30:1 K^+ gradient is responsible for generation of resting membrane potential. Due to concentration gradient across the cardiac cell membrane and high permeability of potassium ions, outward diffusion of the positively charged potassium creates a negative electrical potential inside the cell relative to the outside. Thus, the outside of the cell is positively charged and the inside is negatively charged (Fig. 1.4). The distribution of sodium ions (Na^+) is exactly opposite of K^+ ions. Sodium and calcium ions contribute very less to resting membrane potential as the resting cardiac cell membrane permeability for these ions is very low.

1.2.1.2 Action Potential

The electrical activity of the heart depends upon the generation of action potential by the cardiac cell due to an electrical stimulus. Action potential refers to changes in membrane potential over time (Fig. 1.5). It has five phases: phase 0, 1, 2, 3 and 4. The phase 0 starts at the onset of depolarization of a cardiac myocyte (Fig. 1.6) and during this phase there is an abrupt increase in the permeability of the sodium ions. They enter into the cell (calcium ions also enter to a lesser degree) and cause a sharp rise in the potential to positivity (about +20 mV). As a result, the inside of the cell becomes positive and the outside of the cell negative. This is called the depolarization of the cell.

After depolarization, there is a gradual return to resting membrane potential. In phase 1, there is a rapid return of potential to about 0 mV. This is due to a sudden stoppage of sodium channels. In phase 2, there is a plateau. This is due to slow entry of calcium ions into the cell. In phase 3, there is a slow return to resting membrane potential. This is due to extrusion of potassium ions from the cell. As a result again the inside of the cell becomes negative and the outside becomes positive. The cell gradually returns to −90 mV (phase 4). The aim of repolarization is to bring back the cell to the resting state so that depolarization can occur again. The movement of ions is facilitated by sodium-potassium pump.

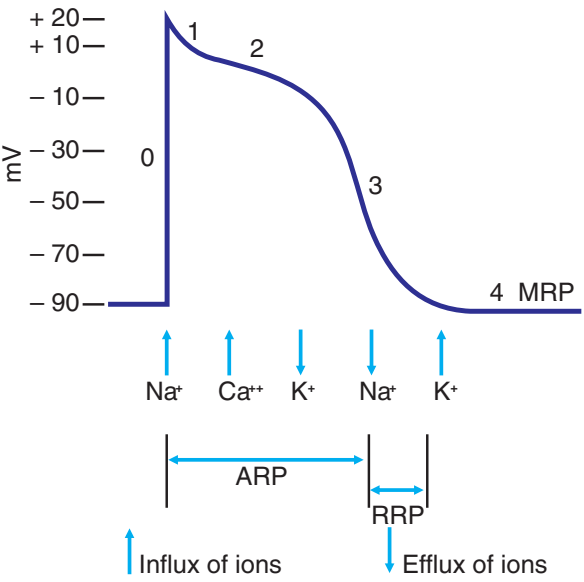

Fig. 1.5 Action potential. *MRP* membrane resting potential, *0* depolarization, *1, 2, 3* phases of repolarization, *ARP* absolute refractory period, *RRP* relative refractory period

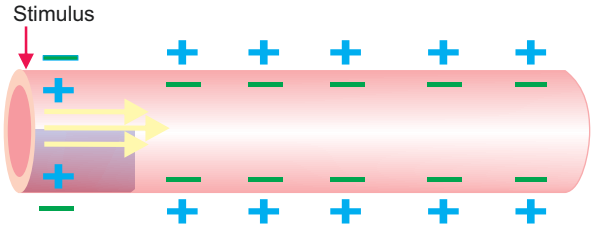

Fig. 1.6 Depolarization of cardiac myocyte after stimulus

1.2.1.3 Refractoriness of Myocardium

During the phases 0, 1, 2 and 3 of the action potential no stimulus however strong will propagate another action potential. This is known as absolute refractory period. After this there is a period during which a strong stimulus can produce a response. This is the relative refractory period. After this there is a period of supernormal excitability during which a weaker stimulus can evoke a response. This coincides with the terminal part of phase 3 and beginning of phase 4.

Tips and Tricks

- Depolarization and repolarization are electrical events and they are not same as systole and diastole.

- Depolarization just precedes systole and repolarization just precedes diastole.

1.2.1.4 Difference of Sinoatrial (SA) Node from Other Parts of Myocardium

The action potential of SA node is different from other parts of the myocardium. The pacemaker cells of SA node do not require an electrical stimulus to generate action potential, unlike other cardiac myocytes. The specialized function associated with the pacemaker cells is their spontaneous depolarization with no true resting potential. When spontaneous depolarization reaches the threshold voltage, it triggers a rapid depolarization followed by repolarization.

1. The resting membrane potential is low, about −60 to −70 mV.
2. A prepotential is present in phase 4. There is a gradual rise in resting membrane potential that is responsible for the automaticity of SA node.
3. Depolarization is slower.
4. The peak of action potential is rounded and repolarization is a slow curve, where the phases 1, 2 and 3 cannot be defined separately.

1.2.2 Generation of ECG

The basic principle behind recording an ECG is an electromagnetic force, current or vector with both magnitude and direction. A moving wavefront of depolarization is produced (Fig. 1.7) when depolarization is transmitted to the adjoining cardiac myocytes. This generates electric current which is amplified and recorded as ECG.

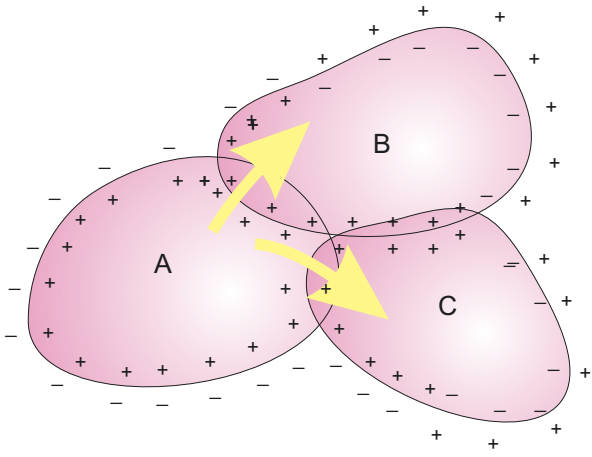

Fig. 1.7 Depolarization wavefront moving from one cardiac myocyte to adjacent cardiac myocytes

Depolarization and repolarization occur in atrial as well as in ventricular muscles and the whole process is so well synchronized that the atria and ventricles contract and relax in a rhythmic way. However, depolarization and repolarization are electrical events and they are not equal to systole and diastole.

ECG actually records these two electrical events, depolarization and repolarization, while the impulse is traveling through the conducting system of heart. The summation of potential of phase 0 of both the atria results in P wave of ECG, i.e. P wave is produced by depolarization of both the atria. Similarly, all phase 0 potentials of both the ventricles produce the QRS complex; i.e. QRS complex represents depolarization of both the ventricles. S-T segment and T wave are produced by summation of potentials of phase 1, 2 and 3 of both the ventricles. Thus, S-T segment and T wave represent repolarization of both the ventricles. However, the routinely recorded 12-lead ECG fails to record any electrical activity while the impulse is traveling through the AV node and the bundle of His.

An electrode facing the wave of depolarization records a positive or an upright deflection while an electrode from which the wave of depolarization is moving away records a negative or downward deflection (Fig. 1.8). A partly positive and a partly negative (biphasic/equiphasic) deflection is recorded when an impulse moves perpendicularly to an electrode. Similarly, a current of repolarization traveling away from the positive electrode is seen as a positive deflection and towards a positive electrode as a negative deflection. A large positive wave will be observed over the larger muscle mass and a large negative deflection will be observed over the smaller muscle mass when two muscle masses of markedly different sizes are stimulated at the centre (Fig. 1.9).

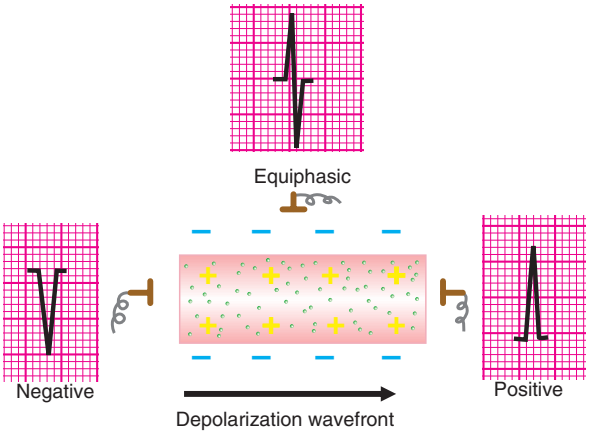

Fig. 1.8 Fundamental principle of generation of negative, positive and equiphasic deflections

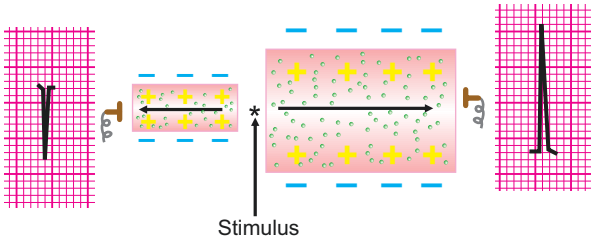

Fig. 1.9 Generation of waves in two muscle strips of markedly different sizes

Tips and Tricks

- Strong electric activity leads to tall or large waveforms.
- Weak electric activity leads to small waveform.
- No electric activity leads to straight line.

1.2.3 Conduction of Electrical Impulse

The electrical conduction system of the heart is the most important aspect of cardiac anatomy and physiology to master while learning the art of interpretation of ECG. To understand the conduction system of the heart, two types of cardiac tissue must be considered:

- Ordinary myocardium/cardiomyocytes (atrial and ventricular).
- Specialized cardiac conduction system, which includes sinoatrial (SA) or sinus node; anterior, middle and posterior internodal tracts; atrioventricular (AV) node; Bundle of His; right and left bundle branches; anterior and posterior divisions of the left bundle and the Purkinje fibre network.

The heart is a mechanical pump whose activity is governed by the electrical conduction system. The specialized conduction system is composed of myocardial tissue, whose primary function is to conduct electrical impulse rather than to contract. Both types of cardiac tissue can allow conduction of electrical impulses; but, cells in the specialized cardiac conduction system also depolarise spontaneously and act as cardiac pacemakers. However, there is a hierarchy of automaticity within the tissue of the heart. The tissue that possesses the greatest degree of automaticity (has the fastest rate of spontaneous depolarization) functions as the dominant pacemaker; it generates a spontaneous action potential that is conducted along the rest of the conduction system, activating the myocardium in a uniform fashion. The electrical impulse in heart is generated in the SA node (Fig. 1.10) located in upper part of right atrium near the opening of superior vena cava. It has the fastest rate of generation of electrical impulse as compared to any other part of the heart, and thus, acts as the main pacemaker of the heart.

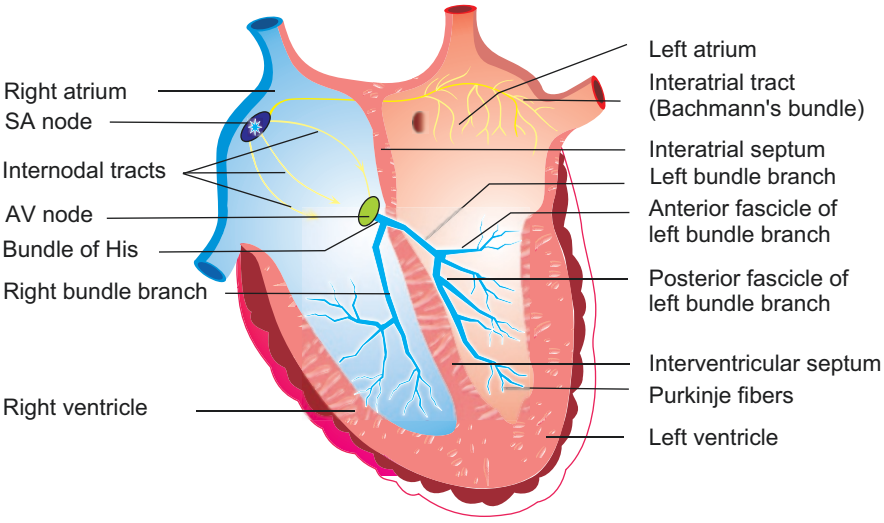

Fig. 1.10 Conducting system of heart

The right atrium is the first part of the heart to be activated. The impulse travels longitudinally, and by contiguity through three intratrial pathways to the other parts of the right atrium. These intratrial pathways are also called internodal pathways because they conduct the impulse from SA node to the AV node. As the impulse travels from the SA node along the internodal tracts, the right atrium contracts and propels blood into the right ventricle. The impulse also travels to the left atrium by interatrial tract (Bachmann's bundle) resulting in contraction of left atrium. Contraction of left atrium propels blood into the left ventricle.

AV node is situated at the lower part of the right atrium on the interatrial septum. In AV node, the impulse is delayed which gives atria the time to contract, and empty into their respective ventricles, before the ventricles are activated for contraction. This AV nodal delay is the major contributor of P-R interval.

From AV node, the impulse passes on to the Bundle of His, which starts at the AV node and then divides into two parts, the left and right bundle branch. The left bundle branch is further divided into one anterior and one posterior fascicle. The anterior fascicle spreads over to the anterior and superior surface of the left ventricle, and the posterior fascicle spreads over the posterior and inferior surface of the left ventricle. These bundles further divide into their terminal ramifications the Purkinje fibres (Fig. 1.11). A small septal fascicle originates from the left bundle and activates the interventricular septum from left to right, and this is the first part of the ventricle to be activated. Next, the impulse passes on to the other parts of the fascicles and to Purkinje fibres. The right bundle branch does not divide and innervates the right ventricle. The Purkinje fibres conduct the electrical impulse to the ventricles which are then activated. The myocardium is activated from endocardium to epicardium.

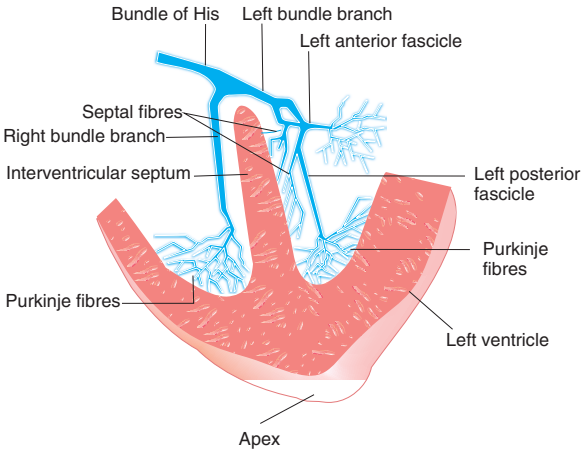

Fig. 1.11 Diagram showing the Bundle of His, the bundle branches, the fascicles and the Purkinje fibres

1.2.3.1 Velocity of Conduction

The velocity of conduction of action potential varies in the various parts of the heart. The velocity is maximum in the Purkinje fibres, and it is the minimum in the middle of AV node.

The average conduction velocity are:

- SA node: 0.05 m/s
- Atrial myocardium: 0.8–1 m/s
- AV node: 0.05 m/s
- Bundle of His: 0.8–1 m/s
- Purkinje fibres: 4 m/s
- Ventricular myocardium: 0.9–1 m/s.

Self-Assessment Questions

1. Willem Einthoven was awarded the Nobel Prize for his contribution in the field of ECG. True or false?
2. The resting membrane potential of cardiac myocyte is +90 mV. True or false?
3. Sodium ion enters the cell during phase 0 of action potential. True or false?
4. Right bundle divides into anterior and posterior fascicle. True or false?
5. The cardiac impulse is delayed at AV node. True or false?
6. **Which of the following pairs are correct?**

    a. P wave: Atrial depolarization
    b. QRS complex: Ventricular depolarization
    c. T wave: Ventricular repolarization
    d. All of the above

7. **Which of the following is the property of a cardiac cell to initiate and fire an action potential on its own without external stimulation?**

   a. Excitability
   b. Automaticity
   c. Contractility
   d. Conductance

8. **In which of the following depolarization is slow?**

   a. Atrial muscle
   b. AV node
   c. Ventricular muscle
   d. SA node

9. **Which of the following is usually the dominant pacemaker of the heart?**

   a. SA node
   b. AV node
   c. Bundle of His
   d. Purkinje fibres

10. **Which of the following is NOT a part of the specialized conduction system of the heart?**

    a. SA node
    b. Myocytes of mitral valve
    c. Bundle of His
    d. AV node

11. **The space in the middle of the thoracic cavity where the heart lies is the:**

    a. Pericardial cavity
    b. Pericardium
    c. Pleural cavity
    d. Mediastinum

12. **The velocity of conduction is maximum in:**

    a. SA node
    b. AV node
    c. Purkinje fibres
    d. Atrial myocardium

13. **During phase 0 of action potential, there is an abrupt increase in permeability of:**

    a. Sodium ion
    b. Potassium ion
    c. Calcium ion
    d. Magnesium ion