ISCAP Interventional Society for Cathlab Allied Professionals

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Cardiac Catheterisation

Manual - Module 1

EDUCARE

ISCAP Interventional Society for Cathlab Allied Professionals

The ISCAP Catheterisation Manual

Endorsed by
The South African Society of Cardiovascular Intervention (SASCI)
The Society for Cardiovascular Angiography and Interventions Foundation (SCAI)

SASCI
South African Society of Cardiovascular Intervention

SCAI
The Society for Cardiovascular Angiography and Interventions Foundation

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Foreword

Where once we were a murmur, we now have the opportunity to find our own rhythm and become the true heart of the cath lab.

Though the quality of South African Cardiology has always been on a par with the rest of the world, the training of professional nurses, technologists and radiographers (Allied Professionals), in the highly specialised field of cardiovascular intervention has been neglected. Our country has lacked guidelines that describe the requirements for a cardiovascular interventional laboratory to be managed successfully. There continues to be no official course to provide credentialing in the subject to the registered nurse.

The national Interventional Society of Cath Lab Allied Professionals (ISCAP) aims to uphold a high standard of cardiovascular interventional laboratory practice and improve the standing of the nursing and allied professional working within that environment. By these means our members will gain recognition as important participants in patient management within the cardiovascular interventional laboratory.

This second edition of the Cath Lab Manual is the continuation of this process. The Manual has been written for all those who work in the cardiovascular interventional laboratory, both as an introductory aid for the novice and as a reliable reference for the experienced practitioner.

By ensuring that educational material such as this is available on line and in hard copy , we are enabling ourselves to assume greater responsibility for our staff's development and our own job satisfaction. We also hope that the overall morale will also be enhanced.

CPD points will be attainable for those who wish to complete the questions at the end of each Chapter. There will also be a component whereby we can share information and experiences on line.

We need to equip staff with the knowledge and specific skills necessary for invasive physiology and anatomical assessment, also for the diagnosis and management of coronary and structural disease.

We trust you will find the Manual helpful. We look forward to hearing your comments and criticisms, so as to contribute to the greater value of this ongoing process of sharing information and thus learning from each other.

If you want to go quickly, go alone. If you want to go far go together

~ African proverb

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Contents

Module 1

1.1 The Historical Developments of Coronary Angiography

Like any scientific field, coronary angiography and its associated developments, follow a very long and interesting history. Surrounded by ludicrous experiments on themselves and animals, early physiologists craved the need to explain the functioning of the human body.

Watson (2005) states that the Egyptians led the way, and around 3000 BC, they performed the first urinary bladder catheterisation using a metal pipe.

Scientists, around 400 BC used hollow reeds, pipes and tubes to examine the heart valves of a cadaver heart. In 1711, Hales constructed a "device" (more of a contraption) and performed the first cardiac catheterisation on a horse. His device consisted of brass pipes, a glass tube and the trachea of a goose! The history of coronary angiography teaches us that the journey from invention to innovation is a complex process. From the beginning this process has involved a large number of pioneers who did not accept dogma and were willing to attempt new technologies, and this will continue in the future.

Hildner (as cited in Eeckhout, et al: 2012) presented the evolution of cardiac catheterisation into 5 eras. He also divides the evolution of cardiac catheterisation into three phases namely, the historical perspective, cardiac catheterisation in animals, and cardiac catheterisations in humans.

In 1844, French physiologist, Claude Bernard catheterized a horse to evaluate intra-cardiac pressures. Both the right and left ventricles were entered by a retrograde approach from the jugular vein and carotid artery. His scientific methodology to study cardiac physiology using the cardiac catheter was indeed an enormous technical innovation (Baim & Grossman, 2000).

Werner Forssman is acclaimed as the first person to pass a catheter into the heart of a living person. This person was himself! At the age of 25, he passed a 65 cm catheter via his left antecubital vein, and under fluoroscopy guidance, advanced the catheter to his right atrium. He walked to the radiology department where the placement was confirmed by a chest roentgenogram. He received an official reprimand for his dangerous experiment. For the following 2 years, Forssman conducted 6 catheterisations on himself. He also managed to insert a catheter into the right atrium of a dying septic patient for the administration of fluids and medication. This technique went largely unnoticed by the medical community (Baim & Grossman, 2000).

Eras of Cardiac Catheterisation	
1929 – 1938	The beginning
1938 – 1948	Initial clinical application
1949 – 1958	Left heart access
1959 – 1977	Coronary angiography
1977 – 1990	Therapeutic techniques
1990 – present	Interventional Procedures

Cornerstones of Cardiac Catheterisations and Cardiac Techniques

1846	First cardiac catheterisation in animals. Bernard
1861	First measurement of intracardiac pressures in animals, Chauveau and Marey
1907	1st roentgenographic atlas of the human coronary arteries
1929	First cardiac catheterisation (self-experiment), Forssmann
1930	First clinically used cardiac catheterisation, Klein
1933	Parasternal left ventricular puncture, Reboul and Racine
1939	Beginning of clinical use of cardiac catheterisation in animals. Cournand and Richards
1941	The cardiac catheter was used as a diagnostic tool for the 1st time
1942	Catheterisation of right ventricle, Cournand and Maurice
1944	Catheterisation of pulmonary artery, Cournand and Maurice
1948	Radner described transradial catheterization using radial artery cut-down
1949	Retrograde catheterisation, Zimmerman
1953	Seldinger technique – sheath access
1956	Apical left ventricular puncture, Brock
1958	The first clinical implantation into a human of a fully implantable pacemaker
1959	Transeptal left atrial access, Ross
1962	External defibrillator was invented
1964	Charles Dotter introduced transluminal angioplasty Melvin Judkins
1967	First Coronary artery bypass surgery was done using saphenous vein grafts By Rene Favaloro
1969	First use of artificial heart in human by Denton Cooley
1970	Bedside catheterisation and monitoring of right heart pressures, Ganz and Swan Preformed femoral caheters, Drs Judkins & Amplatz
1974	K. Lance Gould did first animal studies on FFR
1975	Andreas Gruentzig developed a double lumen balloon catheter
1977	Andreas Gruentzig performs first cath lab Percutaneous Transluminal Coronary Agioplasty (PTCA) on an awake human
1979	First use by Peter Rentrop of intracoronary streptokinase, a clot-dissolving drug to stop a heart attack in progress
1981	The first catheter ablation in humans was performed by Dr. Melvin Scheinman
1982	Over-the-wire coaxial balloon systems introduced, brachial guiding catheters & steerable guide wires are developed
1984	Percutaneous Mitral Balloon Valvuloplasty has since become the treatment of choice for Mitral Stenosis
1986	Puel and Sigwart implanted the first coronary stent in a human patient
1987	Palmaz and Schatz presented the first balloon expandable stent
1988	Rotational Atherectomy was developed – rotoblator procedure
1989	First radial diagnostic series was done - Campeau
1990	Intravascular Ultrasound (IVUS) used for the first time

Cornerstones of Cardiac Catheterisations and Cardiac Techniques	
1994	FDA approves commercial use of coronary stents. First bare metal stent designed by Julio C. Palmaz and Richard Schatz.
1994	Dr Fujimoto did first Optical Coherence Tomography (OCT) scan on patients in Massachusetts on retina of eyes
1995	First radial Interventional program - Kiemeneii
1996	0.014 wire used with the Radi machine to measure Fractional Flow Reserve (FFR)
1999	First drug eluting stent implanted in humans
2001	Percutaneous Left Atrial Appendage Transcatheter Occlusion device (PLAATO) was the first percutaneous LAA device manufactured with the first human implant
2002	The first-in-man Transcatheter Aortic Valve Implantation (TAVI) was performed
2003	The first Drug-eluting-stent, the Cypher stent is manufactured
2003	First radial program in South Africa
2009	Optical Coherence Tomography (OCT) used to visualize coronary morphology
2012	Fractional Flow Reserve (FFR); then Instant wave-free ratio (IFR)
2015	FDA approves first bioabsorbable polymer drug eluting stent

Baim & Grossman (2000) continued that in the1930's, a few physicians performed right heart catheterisations, and measured cardiac output via the Fick's method. O. Klein reported 11 right heart catheterisations, including penetration of the right ventricle, in his attempts to measure cardiac output.

Padillo and his co-workers performed 2 right heart catheterisations.

Butler (2007) asserts that more investigations were done by Andre Cournand and Dickinson Richards in 1941, at the Bellevue Hospital in New York, and they used catheters to study human circulation and pulmonary physiology. In 1947, Dexter reported his studies on congenital heart diseases. He superseded his predecessors by advancing the cardiac catheter to the distal pulmonary artery to measure oxygen saturation. In conjunction with Werkö, they reported that this "wedge" position is a good estimate of pulmonary venous pressure as well as left atrial pressure. During this period, cardiac catheterisation was used to investigate problems in cardiovascular physiology: McMichael (England), Lenégre (Paris), Warren, Stead, Bing, Dexter, Cournand (USA).

Zimmerman and Limon-Lason performed a retrograde left-sided heart catheterisation in 1950. This was followed by the development of the percutaneous femoral artery technique by Seldinger in 1953. Until the end of the 1950's, only non-selective visualizations of the proximal coronary vasculature could be evaluated. This was done by injecting contrast medium into the aortic root. A fellow working under the supervision of Dr. Mason Sones (in 1959) was performing an aortic angiogram. Accidentally, he engaged the catheter into the ostium of the right coronary artery while injecting the contrast medium. Because of this, Dr. Sones can be regarded as the father of coronary arteriography. Prior to this event, it was feared that direct contrast injection into the coronary

arteries would result in ventricular fibrillation and death. This patient developed a brief period of asystole. Following this, Dr. Sones performed catheterisations by performing a one inch incision in the antecubital fossa to isolate the brachial artery. This approach became known as the Sones technique (Freed: 1992).

The first selective coronary angiogram on an adult patient was done by Dr. Sones at the Cleveland Clinic in 1952. The first nurse that was present was Lucile Van Derwyst, his assistant. The practice of Dr. Sones grew exponentially, and eventually he had 6 catheterisation laboratories. Mrs. Van Derwyst trained and supervised more than 40 nurses. She also became a pioneer, developing a new era for nursing practice (Watson: 2005).

More advances occurred in the following years. In 1959 Ross & Cope performed a trans-septal catheterisation. This technique was refined by Ross & Brockenbrough, and mastered by Dr. Mullins. During 1964, Charles Dotter and Melvin Judkins were starting to treat patients with peripheral vascular disease. They inserted stiff dilators, gradually increasing the size, in an attempt to improve blood flow. Dotter called this technique transluminal angioplasty (Baim & Grossman: 2000).

In 1967, Dr. Judkins, a radiologist, started to use pre-formed curved catheters and cannulating with them using the Seldinger technique. He formed the catheters himself, and through trial and error he found the most appropriate shapes to engage the coronary ostia. This period also marks the emergence of bypass surgery. Dr. Rene Favaloro performed a saphenous vein graft at the Cleveland Clinic (Watson: 2005).

In 1970, Swan & Ganz introduced the flow-directional balloon tip catheter for monitoring right heart pressures and cardiac output monitoring via the thermodilution method.

In this time the intra-aortic balloon catheter was developed, but this device could only be inserted after a surgical cut-down (Baim & Grossman: 2000).

After employing Dotter's techniques of transluminal andioplasty, Andreas Roland Gruntzig (1939-1985) began toying with the idea of adding a balloon to the Dotter catheter.

The field of interventional cardiology was born in September 1977 when Andreas Gruentzig performed the first PTCA in Zurich (Baim & Grossman: 2000), Switzerland, on a 37 year male patient with a LAD stenosis. The first PTCA case was performed in 1978 in America by Myler in San Francisco and Stertzer in New York (Nguyen, et al:2008). During this time, Gruentzig conducted his first demonstration course in Zurich, Switzerland, attended by 28 pioneering physicians.

During 1980, Gruentzig conducted the last of five demonstration courses in Zurich. Predecessors like Sones, Judkins and Dotter attended. He moved to Atlanta where he became the Director of Interventional Cardiology at Emory University. Guiding catheters were introduced during this time as well (Nguyen: 2008).

The over-the-wire coaxial balloon systems were introduced in 1982. This also marks the time for the development of brachial guiding catheters & steerable guide wires.

From 1986 – 1993 coronary atherectomy devices were introduced. Jacques Puel and Ulrich Sigwart implanted the first coronary Wallstents in Toulose, France. A large number of new interventional devices were invented and perfected: these devices included rotational atherectomy devices (Rotablator), intravascular ultrasound (IVUS) and stents (Freed: 1992).
In 1994, the Palmaz-Schatz stent was approved by the F.D.A. for use in the United States. Coronary

stenting became commonplace. Apart from coronary stenting, over one million angioplasties were performed worldwide, making angioplasty the most common medical intervention in the world (Wikimedia.org).

The origin of the word 'stent' comes from Charles Thomas Stent (1807-1885) an English dentist. He is known for his work in denture making. He improved the denture base compound making it practical for dental use and called it "Stent".

The first coronary stent was placed within a patient in France in 1986 by Jacques Puel.

Ulrich Sigwart from Switzerland is seen as the one who revolutionized modern day stenting.

In 2002 the first drug eluting stent received FDA approval. The Cypher stent (Johnson & Johnson). It combined a proven stent design with a pharmaceutical agent that inhibits smooth-muscle proliferation. It marked a major advance in the battle to reduce restenosis. Boston Scientific had its Taxus drug- eluting stent approved. Many studies have been done demonstrating the vastly improved outcomes from drug-eluting stents.

We are entering into an era of the bio-resorbable vascular scaffolds (BVS), whereby there is a temporary platform, by which the drug is eluted, and the scaffold then slowly dissolves. The development of this device is ongoing at present.

The term percutaneous coronary angioplasty has given way to the term percutaneous coronary intervention (PCI), which is a more general term that includes stenting.

PCI has become the most frequently performed therapeutic intervention in the entire field of medicine. The invention of balloon catheters was the catalyst that allowed this field of medicine to grow to the size it is today.

In 1989, Campeau et al revisited Radner's idea of using the radial artery as an access site. They reported on percutaneous entry into the distal radial artery for selective coronary angiography in 100 patients. In 1992, prompted by the need to avoid the frequent femoral bleeding associated with the anticoagulation used at the time of stent implantation, Kiemeneij et al used Campeau's work as the basis for developing transradial coronary interventions. More recently, the radial approach has been shown to confer mortality benefits for STEMI patients and a reduction in mortality, myocardial infarction and stroke for patients undergoing the procedure at high radial volume centres. Radial access use has been growing steadily but despite these encouraging results, still vaaries greatly among operators, hospitals, countries and continents. (Consensus document : European Association of Percutaneous Cardiovascular Interventions and Working groups on Acute Cardiac Care. Euro Intervention 2013.)

1.2 Cardiac Anatomy and Physiology

The cardiovascular system is responsible for circulating blood throughout the body. This system maintains homeostasis by supplying oxygen and nutrients to the body cells, and the removal of carbon dioxide and other waste products to the lungs and kidneys for excretion.

The heart acts as a pump and the hollow blood vessels are the delivery routes. Blood serves as the transport medium. This remarkable pump beats 37 million times a year.

The circulatory system is equipped with a myriad of compensatory mechanisms which can complicate a cardiac catheterisation procedure and alter the patient's cardiovascular status in seconds.

A general overview of the macroscopic structure of the heart will be discussed.

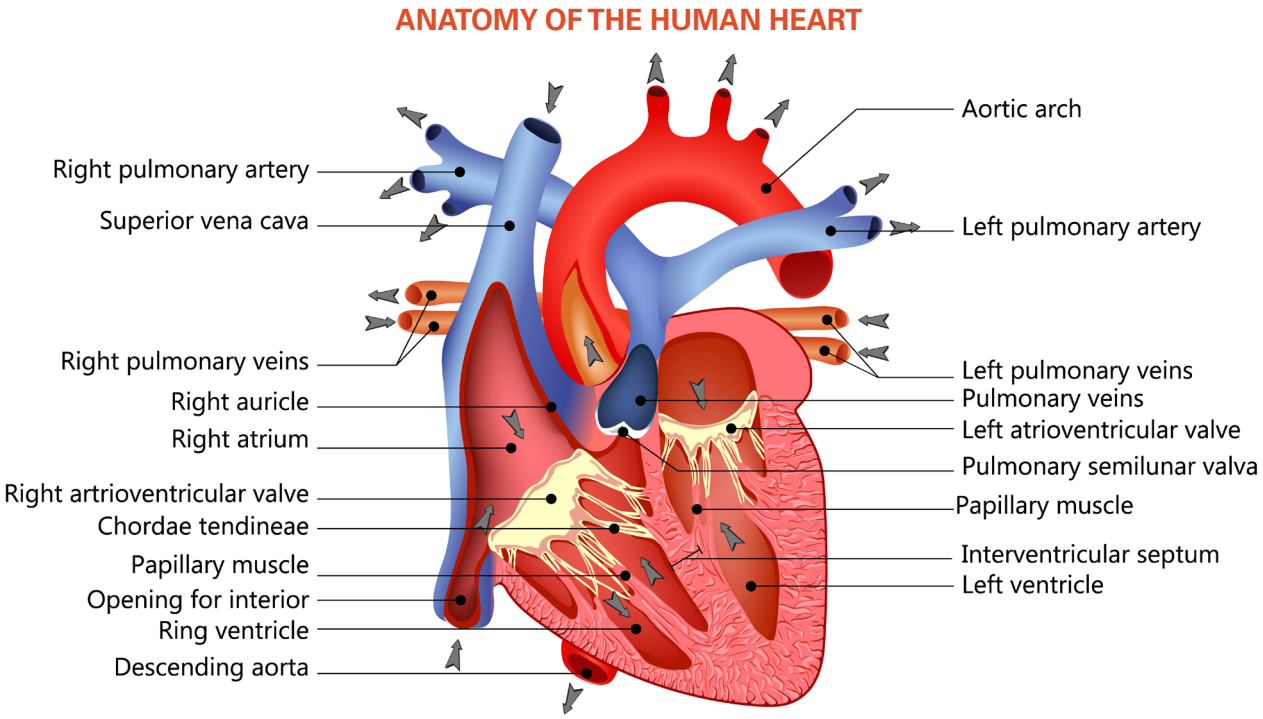

Figure 2.1 The internal view of the heart

THE MACROSCOPIC STRUCTURE OF THE HEART

Hurst (1994), Watson (2005), Butler (2007), Marieb & Hoehn (2010), Askari (2011), Marieb & Hoehn (2010), Askari (2011), and Di Mario (2011) elaborated on the macroscopic structure of the heart as follows:

A. POSITION OF THE HEART

- The heart lies in the thoracic cavity in the mediastinum between the lungs
- It lies obliquely, a little more to the left than the right
- It presents a base above, and an apex below
- The apex is about 9 cm to the left of the midline at the level of the 5th intercostal space
- The base extends to the level of the 2nd rib

B. STRUCTURES ASSOCIATED WITH THE HEART

- INFERIORLY: the diaphragm and the inferior vena cava
- SUPERIORLY: great blood vessels: aorta, superior vena cava, pulmonary artery and pulmonary veins
- POSTERIORLY: oesophagus, trachea, left & right bronchus, descending aorta, thoracic vertebrae
- LATERALLY: lungs, the left lung overlaps the left side of the heart
- ANTERIORLY: sternum, ribs and intercostal muscles

C. SIZE & WEIGHT OF THE HEART

- Average human heart is about the size of the clenched fist of that individual
- ±12 cm in length and 8 – 9 cm in breadth at the broadest part
- In men, the weight averages 310 grams, and in women 255 grams
- There are no significant differences in ventricular wall thickness between men and women
- Pathological conditions such as hypertension will increase the weight of the heart muscle secondary to ventricular hypertrophy

D. LAYERS OF THE HEART

The heart consists of 4 layers: pericardium, epicardium, myocardium & endocardium.

PERICARDIUM

- This doubled-layered sac surrounds the heart and the origin of the great vessels
- There are 2 layers with a potential space between each layer, filled with fluid that serves as a lubricant that prevents friction as the heart contracts
- Ligaments anchor the outer pericardium to the diaphragm and great vessels so that the heart is maintained in a fixed position within the thoracic cavity
- The outermost pericardium is a thick, fibrous envelope that is tough and inelastic (fibrosa)
- Inside the fibrous layer is an inner serous sac, the serosa, that is divided into 2 layers
- The outer layer forms an inner, serous lining to the tough, outer pericardium (fibrosa). Together, they form the parietal pericardium.
- The serosa directly covering the heart is known as the visceral pericardium or epicardium
- The fibrous, outer pericardial sac is non-compliant and unable to adapt to a rapid increase in either cardiac size or the amount of fluid in the sac.

EPICARDIUM

- There is a layer of adipose tissue beneath the visceral pericardium on top of the heart
- The coronary arteries lie on top of the epicardium

MYOCARDIUM

- This is the mid-wall, thick muscular layer
- This layer includes all of the atrial and ventricular muscle fibres necessary for contraction
- The fibres of the myocardium do not have the

same thickness throughout the ventricular walls
- The left ventricle is much thicker than the right ventricle and atria
- The fibres are organized so that the force of contraction is most efficient in ejecting blood towards the outflow tract in a wringing motion from the apex towards the base

ENDOCARDIUM

- It is the thin innermost layer of endothelium and connective tissue lining the inside of the heart
- This smooth, glistening membrane permits smooth blood flow of blood inside the heart
- The endothelial layer is continuous with that of the blood vessels

E. THE CARDIAC CHAMBERS

- The heart consists of 4 chambers: right and left atria & right and left ventricles
- The right and left side of the heart is separated by the septum
- The atria are thin-walled and normally low-pressured chambers
- The right atrium receives blood from the inferior and superior vena cava and the left atrium receives blood from 4 pulmonary veins. The right atrium empties blood into the right ventricle via the tricuspid valve and the left atrium empties blood into the left ventricle via the mitral valve
- Atrial contraction (also referred to as "atrial kick") contributes ± 20% of blood flow to ventricular filling, whereas the remaining 80% occurs passively during diastole
- The ventricles are the primary pumping forces of the heart
- The right ventricle is about 3 mm thick, whereas the left ventricle is 10 – 13 mm thick
- The right ventricle pumps blood into the low-pressured pulmonary circulation (a mean pressure of 15 mmHg)
- The left ventricle must eject its volume into the aorta (with a mean pressure of 80 - 100 mmHg) and is considered as the pump component

F. THE CARDIAC VALVES

- Cardiac valves are composed of flexible, fibrous tissue covered with endothelium
- A normal valve has a translucent rose petal appearance
- The valve structure allows blood to flow in one direction only
- The opening and closing of the valves depends on the relative pressure gradients on either side of the valve
- The 4 cardiac valves lie in an oblique plane of collagen, referred to as the fibrous skeleton
- There are also 4 adjacent rings of connective tissue that contain and support the cardiac valves

THE ATRIO-VENTRICULAR VALVES

- These valves are situated between the atria and the ventricles
- The tricuspid valve is situated between the right atrium and right ventricle. The tricuspid valve has 3 cusps which arise from a fibrous ring around the valve, known as the anulus. The cusps are named according to their anatomical location, namely: anterior, posterior and medial
- The divisions between each cusp are known as the commissures. They do not normally extend to the annulus and therefore the leaflets are continuous with each other
- The mitral (or bicuspid valve) is situated between the left atrium and ventricle. There is an anterior and a posterior leaflet.
- The atrio-ventricular valves are open during ventricular diastole (filling) and prevent back flow of blood into the atria during ventricular systole (contraction)
- The chordae tendinae and papillary muscles, attached to the tricuspid and mitral valves, give

the valves stability and prevent leaflet eversion during systole. Papillary muscles arise from the ventricular myocardium and derive their blood supply from the coronary arteries. The chordae tendineae are fibrous, avascular structures covered by a thin layer of endocardium

THE SEMI-LUNAR VALVES

- These are the aortic and pulmonic valves
- They are located at the root of the respective artery behind which is a dilated area known as the Sinus of Valsava into which the blood in its regurgitation towards the heart enters and thereby closes the valves.
- Each valve has 3 cusps
- During systole, the semi-lunar valves open, allowing blood flow out of the ventricles
- As systole ends and the pressure in the outflow arteries exceeds that of the ventricles, the semi-lunar valves close, thus preventing blood regurgitation back into the ventricles
- The leaflets are thinner than those of the AV-valves and they do not have chordae tendinae. When the valves are closed, they have an umbrella-like feature. A centrally placed nodule of dense connective tissue is found on the free margin of each valve. On each side of the nodule is a thinned-out area, called the lanula.
- The pulmonary valve is divided into the anterior cusp, right cusp and left cusp
- The aortic valve is divided into the right coronary cusp, left coronary cusp and posterior cusp (non-coronary cusp)

G. THE MAJOR CARDIAC VESSELS

- The major vessels are: aorta, pulmonary veins, pulmonary arteries and the systemic vasculature.

AORTA

- Is the largest artery in the body
- It carries oxygenated blood from the left ventricle to the systemic circulation
- Just above the aortic valve, there are two openings that represents the origin of the right and left coronary artery system
- These openings are referred to as the coronary ostia (single opening is an ostium)

VENA CAVAE

- The superior vena cava (SVC) returns the deoxygenated blood from the upper body to the heart
- Inferior vena cava (IVC) returns blood from the abdomen and legs to the heart
- IVC and SVC drain into the right atrium

THE PULMONARY ARTERY

- The main pulmonary artery divides into the left and right pulmonary artery that diverts blood to the respective lungs
- It carries deoxygenated blood from the right ventricle to the pulmonary arteries
- The pulmonary artery is the only artery that carries deoxygenated blood

THE PULMONARY VEINS

- There are usually four pulmonary veins from the lungs which transport oxygenated blood to the left atrium
- These veins connect into the back wall of the left atrium. There are no valves that inhibit the blood flow into the left atrium
- Blood flow is accomplished by simple hydrostatic pressure gradients: the pressure must be lower in the left atrium than in the pulmonary circulation for flow to occur in a forward direction

THE SYSTEMIC CIRCULATION

- The vascular structures act as conduits to carry vital oxygen and nutrients to each cell and also to carry away waste products
- The vascular system acts not only as a conducting system for the blood but also as a control mechanism for the pressure in the heart and vessels
- It is a complex interplay between the heart and the blood vessels that maintaining adequate pressure and velocity within this system for optimal functioning

H. CORONARY BLOOD SUPPLY

Hurst (1994), Watson (2005), Butler (2007), Marieb (2010), Askari (2011), and Di Mario (2011) elaborated on the coronary blood supply as follows:

- Blood travelling within the heart passes through the heart without delivering oxygen and nutrients to the heart muscle
- The heart receives its blood supply from its own circulatory system made up of the coronary arteries and the cardiac veins
- The heart is the first organ to be perfused with oxygenated blood as it leaves the left vetricle via the aorta
- The coronary arteries supply oxygen to the heart muscle mainly during diastole
- Arteries branch into capillaries where gas exchange occurs, merging into cardiac veins. The cardiac veins drain into the coronary sinus.
- The coronary sinus empties into the right atrium
- After leaving the base of the aorta, the coronary arteries traverse the outside of the heart, above the epicardium, in the natural grooves (sulci) between the chambers
- To perfuse the thick heart muscle, branches from these main arteries arise at acute angles, penetrating the muscular wall and eventually feeding the endocardium

CORONARY ARTERIES

- Originate in Sinuses of Valsalva, at the root of the aorta just above the aortic valve
- Fill during diastole
- Right coronary artery (RCA)
  - Originates from the right anterior aortic cusp, slightly inferior to origin of the left.
  - Winds around in the right AV groove towards the crux (a point on the diaphragmatic surface of the heart where the anterior (right) atrioventricular (AV), the posterior AV groove (left) and the inferior interventricular groove meet.)
  - First branch is usually the conus artery arising from the os or within the first few millimetres in 50% of patients. In other patients, it arises from a separate ostium above the RCA os.
  - The second branch is usually the sinoatrial node artery. Supplies the sinoatrial (SA) node in 59% of hearts.
  - The mid portion of the RCA usually has several acute marginal branches supplying the anterior wall of the right ventricle.
  - Forms the posterior descending artery (PDA) in the posterior interventricular groove in about
  - 80% of hearts are the right coronary artery dominant
  - Supplies the AV node in approximately 90% of hearts.
  - Supplies the right atrium and right ventricular heart muscle.
  - Supplies the inferior and posterior wall of the ventricle.
  - Supplies the posterior portion of the interventricular septum.
- Left main coronary artery arises from the superior portion of the left aortic sinus and bifurcates into 2 major coronary arteries: left anterior descending (LAD) and circumflex (CX)

- LAD
  - Travels down the anterior interventricular groove towards the apex.
  - Supplies the anterior part of the interventricular septum via the septal branches.
  - The septal branches interconnect with similar branches of the PDA making the septum the most densely vascularised area of the heart.
  - Supplies the anterior and lateral walls of the LV via the diagonal branches. In 90% of patients there are 1-3 diagonal branches.
  - Supplies the bundle of His.
- CX
  - Winds around the left posterior AV groove towards the inferior interventricular septum.
  - It is dominant in 15% of patients, supplying the PDA.
  - Supplies the SA node in 38% of hearts. 3% of patients have supply from the RCA and CX.
  - Supplies the AV node in 10% of hearts.
  - Supplies the lateral free wall of LV via the obtuse marginal branches (OM).
  - There are usually 1-3 OM branches.
- Ramus
  - In some patients, there is a ramus intermedius branch off the left main beween the LAD and CX supplying the lateral wall beween the diagonal branches and the OM.

I. PHYSIOLOGICAL SHUNTS

A shunt occurs when arterial and venous blood are mixing

In the heart, there is a specific situation where this is a normal physiological process: the the besian veins are small vessels that connect capillary beds directly with the cardiac chambers via irregular endothelium-lined sinuses within the myocardium. The thebesian veins add a small quantity of deoxygenated blood into the left ventricle

J. VENOUS DRAINAGE

Marieb (2010) reflects on the venous drainage by stating that there are two major groups of veins in the heart: those that drain into the coronary sinus and those that drain directly into the cardiac chambers. The veins that drain directly into the cardiac chambers are the thebesian and the anterior cardiac veins.The anterior cardiac veins drain blood from the anterior aspect of the heart directly into the right atrium.

- There are three main tributaries of the coronary sinus: the great, middle and small cardiac veins. The entrance to the sinus is guarded by the crescent-shaped thebesian valve.
- The great cardiac vein: this vessel follows the initial course of the LAD in the anterior interventricular groove, thereby draining the anterior wall and anterior septum, before entering the left atrio- ventricular (AV) groove alongside the circumflex. It picks up the venous drainage of the obtuse marginal vessels on the lateral surface. It terminates in the coronary sinus.
- The middle cardiac vein: this vessel follows the posterior descending artery in the posterior interventricular groove, and drains the inferior wall and posterior septum.
- The small cardiac vein: this vessel traverses the right ventricular wall, with the marginal branches of the RCA, before entering the AV groove and terminating in the coronary sinus.
- There are several other branches draining into the coronary sinus that are important for biventricular pacing: The postero-lateral vein, the anterior interventricular vein with lateral branches and in some cases, separate lateral veins.

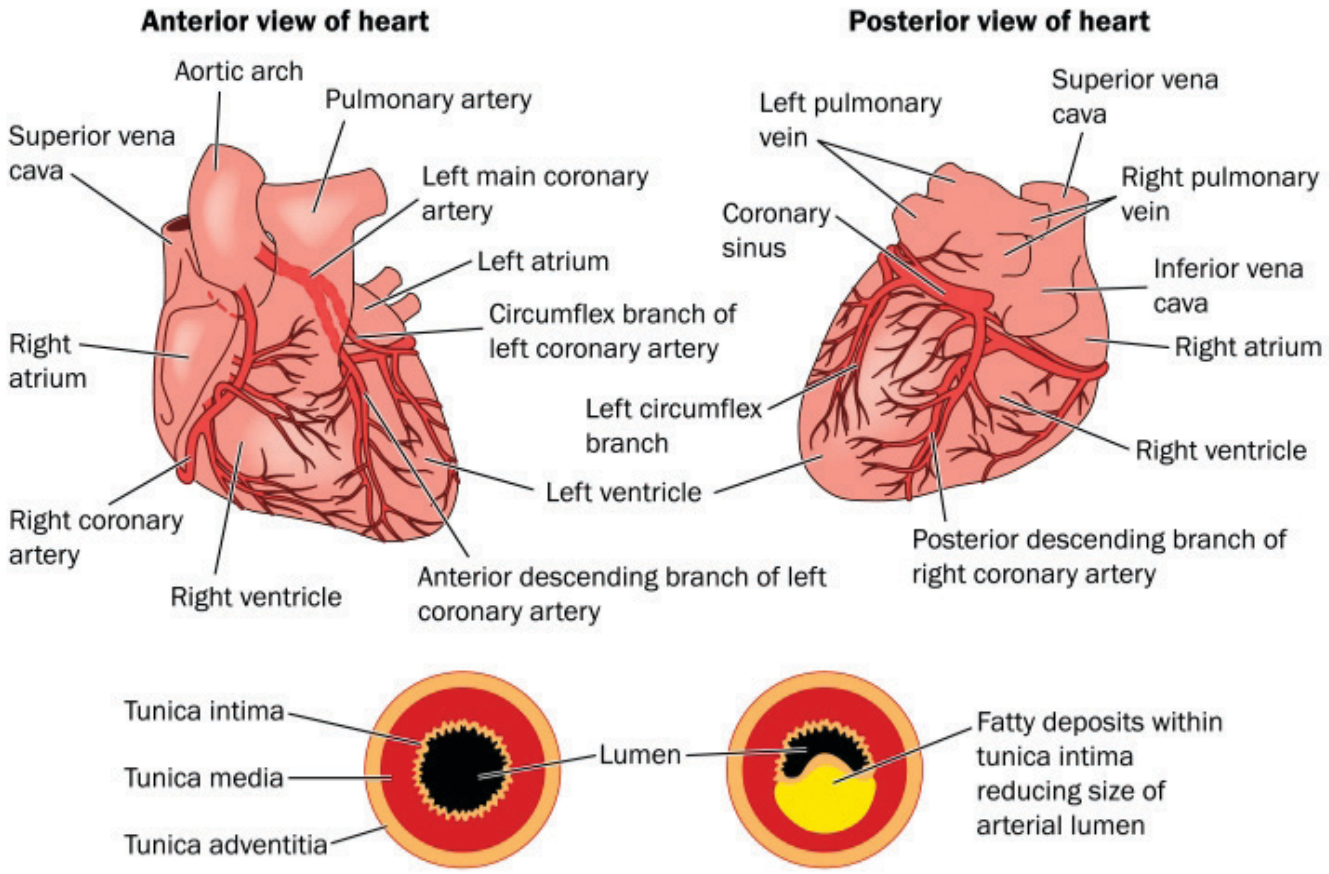

Figure 2.2: Coronary Arteries - Anterior and Posterior view

K. THE CONDUCTION SYSTEM

Hurst (1994), Watson (2005), Butler (2007), Marieb (2010), Askari (2011) and Di Mario (2011) elaborated on the cardiac conduction system as follows:

- The heart has an intrinsic system whereby the cardiac muscle is automatically and electrically stimulated to contract without the need for a nerve supply from the brain
- There are 3 main areas of impulse propagation and conduction:
  a. SA node
  b. AV node
  c. Bundle of His

THE SA NODE (SINOATRIAL)

- Is considered the natural pacemaker of the heart, because it has the highest degree of automaticity, producing the fastest intrinsic heart rate
- The node is a spindle-shaped structure located near the entrance (inflow tract) of the superior vena cava in the high posterior right atrial wall
- The node contains two types of cells: specialised pacemaker cells found in the node centre and border zone cells. Once the central nodal cells depolarize, the impulse is conducted through the nodal border zone toward the atrium
- Atrial depolarisation occurs both cell to cell and through three specialised conduction pathways (anterior, medial & posterior) that exit the SA node
- There is also an intra-atrial pathway, called the Bachmann's bundle, that travels from the right to the left atrium
  – AV-node acts as back-up pacemaker if the SA-node fails (rate ± 60 bpm)
  – AV-node can conduct retrograde (backward impulses through the node

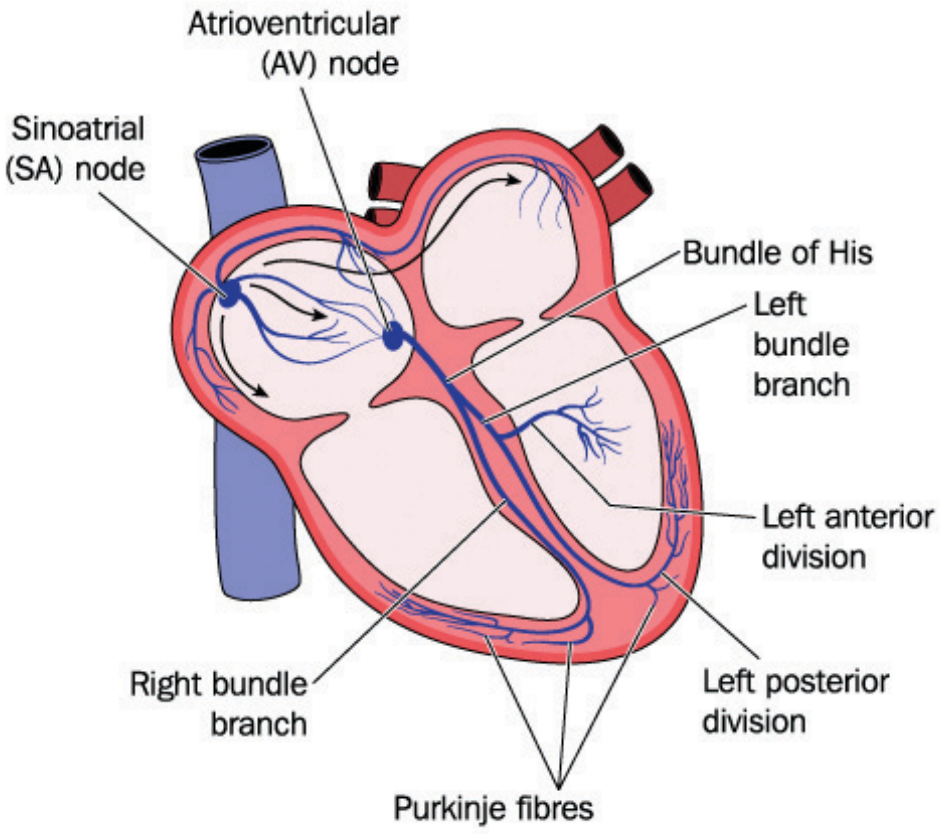

Figure 2.3: The electrical (conduction) system of the heart

THE AV NODE (ATRIOVENTRICULAR)

- Is located posteriorly on the right side of the inter-atrial septum on the floor of the right atrium
- The AV-node performs 4 essential functions:
  - Delays the conducted impulse to provide time for ventricular filling
  - AV-node controls the number of impulses that are transmitted from the atria to the ventricles (this prevents rapid irregular atrial rates from destabilising the ventricles)

THE BUNDLE OF HIS; BUNDLE BRANCHES & PURKINJE FIBRES

- The bundle of His, bundle branches & Purkinje fibres run through the sub-endocardium down the right side of the interventricular septum
- About 12 mm from the AV-node, the Bundle of His divides into the Left and Right Bundle Branches
- The Right Bundle Branch continues down the right side of the interventricular septum toward the right apex
- The Left Bundle Branch is thicker and takes off from the Bundle of His at almost a right angle
- It traverses the septum to the sub-endocardial surface of the left interventricular wall, where it divides into a thin anterior branch and a thick posterior branch
- The Right Bundle Branch and the two divisions of the Left Bundle Branch eventually divide into the Purkinje fibres
- The Purkinje fibres have the fastest conduction velocity of all heart tissue

L. NERVE SUPPLY TO THE HEART

Urden et al (2006) reflects on the nerve supply as follows:

- In addition to the intrinsic impulses generated within the conduction system, the heart is influenced by autonomic nerves originating

in the cardiovascular centre in the medulla oblongata. These consist of parasympathetic and sympathetic nerves, with antagonistic actions to one another

- The Vagus nerve (parasympathetic) supplies mainly the SA & AV nodes and atrial muscle. Parasympathetic stimulation reduces the rate at which impulses are produced, thus decreasing the rate
- Vagal stimulation slows conduction through the AV node.
- The sympathetic nerves supply the SA & AV nodes and the atria and ventricles. Sympathetic stimulation increases the rate and the force of the heart beat

THE MICROSCOPIC STRUCTURE OF THE HEART MUSCLE

Hurst (1994), Watson (2005), Butler (2007), Marieb (2010), Askari (2011) and Di Mario (2011) elaborated on the microscopic structure of the heart as follows:

A. CARDIAC FIBRES

- Are found in a lattice-work arrangement
- These fibres (myofibrils) divide, rejoin and then separate again, but they retain distinct cellular walls and possess a single nucleus
- Cardiac myofibrils run on a longitudinal axis, and the fibres appears striated
- The areas separating each of the myocardial cell are called intercalated discs, which are continuous with the sarcolemma (cell membrane)
- The point where a longitudinal branch of the

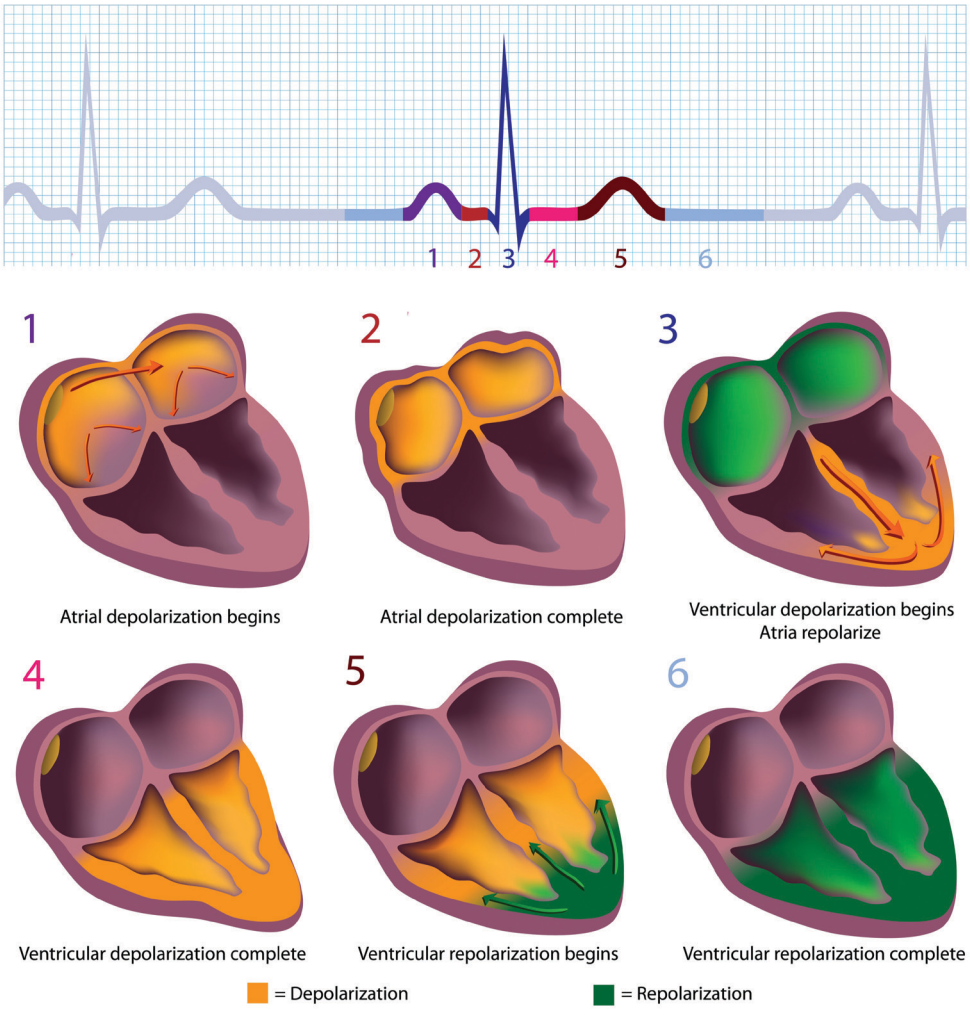

Figure 2.4: The progression of cardiac action potential through myocardium

cell meets another cell branch is a tight junction (gap junction) which promotes electrical flow at a faster rate than the sarcolemma which shows some impedance to electrical flow

- Because of this, depolarisation occurs from cell to cell with relative ease
- The cardiac muscle is a functional syncytium in which depolarisation starts in any cardiac cell and is quickly spread to all of the heart

B. CARDIAC CELLS

- Each cell contains 2 types of intracellular contractile proteins: actin and myosin filaments
- These proteins are organized in a longitudinal arrangement: myosin filaments appear thick, and the actin filaments appear thin
- The actin filaments are connected with Z-bands on one end, leaving the other end free to interact with the myosin cross-bridges
- The ends of the myosin filament which overlap with the actin, have tiny projections. For contraction to occur, these projections interact with the actin to from cross-bridges
- The portion of the muscle fibre between two Z-bands is called a sacromere
- The sarcoplasmic reticulum is another important intracellular structure that is necessary for contraction. Calcium ions are stored in the sarcoplasmic reticulum and is released for use after depolarisation
- Deep invaginations into the sacromere are called transverse tubules (T-tubules)
- T-tubules are essentially an extension of the cell membrane and thus allow depolarisation to rapidly penetrate the interior structures of the cell. This better co-ordinates muscle contraction.
- Cardiac cells abound with mitochondria (which contain respiratory enzymes necessary for oxidative phosphorylation)
- This enables the cell to keep up with the tremendous energy requirements of repetitive contraction

C. TRANSMEMBRANE POTENTIALS (ELECTRICAL ACTIVITY)

- Electrical potentials across cell membranes are present in all body cells
- Nerve and muscle cells are specialised for conduction of electrical impulses along their membranes
- This electrical potential is called transmembrane potential, and refers to the relative electrical difference between the interior of a cell and that of the fluid surrounding the cell
- Ionic channels are pores in the cell membrane that allow for passage of specific ions at specific times or signals
- Transmembrane potentials form the basis for electrical impulse conduction and muscle contraction.

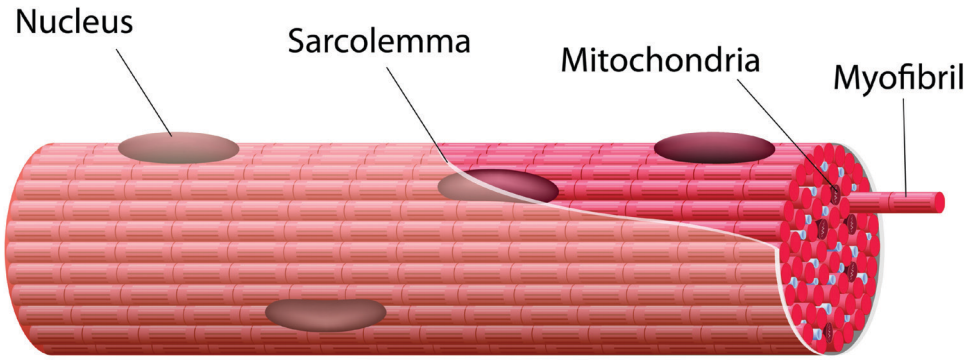

Figure 2.5 Cardiac muscle cell

Table 2.1: Overview of the phases of action potential

Phase	Description	Ionic Movement	Mechanisms
0	Upstroke	Na+ moves into the cell	Fast Na+ channels open
1	Overshoot		Fast Na+ channels close
2	Plateau	Na+ & Ca2+ in and K+ out	Multiple channels (Ca2+, K+, Na+) open to maintain membrane voltage
3	Repolarisation	K+ moves out of the cell	Ca2+ & Na+ channels close. K+ channels remains open
4	RMP	Na+ out & K+ moves in	Na+/K+ pump

Huether & McCance (2001) describe the phases of action potential as follows:

Table 2.2: Description of the phases of action potential

Phase	Description
0	As the membrane is depolarised, sodium rapidly enters the cell, causing the interior of the cell to become more positive. At -65 mV, the membrane reaches threshold, the point at which the inward sodium overcomes the efflux of potassium. This is reflected in the overshoot where the charges is 20 – 30 mV
1&2	When the influx of Na+ is terminated, a brief period of partial repolarisation occurs as the action potential slope returns to zero (phase 1). The plateau that follows is phase 2 where slow Na+ & Ca2+ channels open. K+ diffuses out to balance the influx of Na+. The entered Ca2+ causes contraction
3	This is the repolarisation phase, and depends on 2 processes: 1. Ca2+ & Na+ channels are inactivated 2. there is continued efflux of K+ out of the cell. This tries to re-establish the RMP. A gradual descent is seen where the interior of the cell becomes more negative than the outside
4	The action potential returns to resting membrane potential of -80 to -90 mV. The excess Na+ that entered the cell during depolarisation is now removed from the cell in exchange for K+ by means of the N+/K+ pump. This mechanism returns the intracellular concentrations of Na+ and K+ to the levels before depolarization. This is essential for the next depolarisation

D. RESTING MEMBRANE POTENTIAL

Guyton & Hall (2001) elaborate as follows:

- When a myocardial cell is at rest, the resting membrane potential is -90 mV
- This means that the interior of the cell is relatively negative compared to the exterior medium
- This negativity is created by an uneven distribution of positively charged ions and negatively charged ions
- When at rest, the intracellular K+ is high (135 mEq/L) and the intracellular Na+ is low (10 mEq/L)
- Conversely, the extracellular K+ is relatively low (4 mEq/L), and the Na+ is high (145 mEq/L)

- These differences cause a chemical gradient, and an electrical gradient, which causes positively charged ions to move to an area of relative negativity

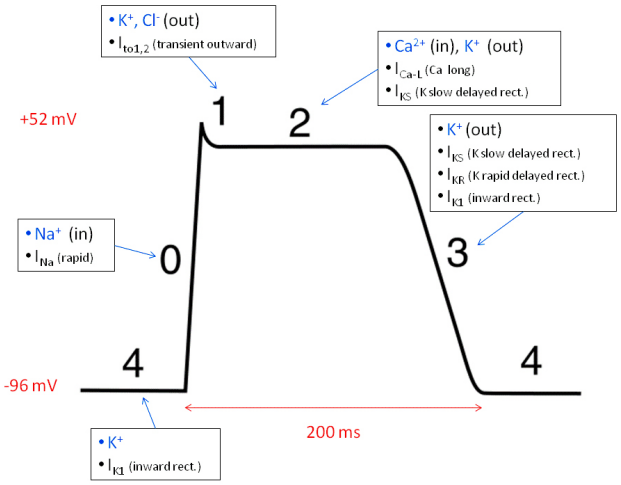

Figure 2.6: Action potential of cardiac muscle

E. THE PHASES OF THE ACTION POTENTIAL EXCITATION-CONTRACTING COUPLING

Thibodeau & Patton define the excitation-contraction coupling concept as follows:

- The electrical activity is the stimulus for mechanical contraction
- As the myocardial cell is depolarized, some calcium enters the cytoplasm through the cell membrane (via calcium channels in phase 2)
- The majority of calcium enters the cytoplasm from stores in the Sarcoplasmic Reticulum
- The cytoplasmic calcium binds with troponin and tropomyocin (molecules which are present on the actin filaments) resulting in contraction, and this spreads throughout the myocardium, causing myocardial contraction
- Once contraction has occurred, calcium is taken back to the Sarcoplasmic Reticulum and the cytoplasmic concentration of calcium falls, leading to muscle relaxation
- Both contraction & relaxation are active processes because they require energy from ATP (adenosine triphosphate) and because calcium is removed from the cell by way of the Na+/Ca2+ pump.

F. THE CARDIAC CYCLE

Lichtenthal (2002) states that the cardiac cycle refers to one complete mechanical cycle of the heart beat, beginning with ventricular contraction and ending with ventricular relaxation.

a. VENTRICULAR SYSTOLE

- This is the ejection phase of the cardiac cycle
- As the ventricles are electrically depolarized, the septum and papillary muscles tense first
- This provides a stable outflow tract and competent atrio-ventricular valves
- The ventricles begin to tense, and this increases the pressure in the ventricles.
- When intraventricular pressure exceeds that of intra-atrial pressure, the mitral and tricuspid valves close. Left ventricular volume is fixed - isovolumic contraction (Braunwald reference)
- As the ventricular tension increases, the intra-ventricular pressure exceeds those of the aorta and pulmonary arteries, causing the aortic and pulmonic valves to open
- The blood ejected rapidly from the ventricles with each contraction is called the stroke volume
- More than half of the total ventricular volume is ejected. The blood that remains is the residual or end-systolic volume
- The ejection fraction is the ratio of stroke volume to the left ventricular end-diastolic volume
- The normal ejection fraction is greater than 50%

b. VENTRICULAR DIASTOLE

- As more myofibres relax, the rate of the ejection of the blood is reduced (phase of reduced ejection).
- Isovolumetric relaxation occurs between the closure of the semi-lunar valves and the opening of the AV valves

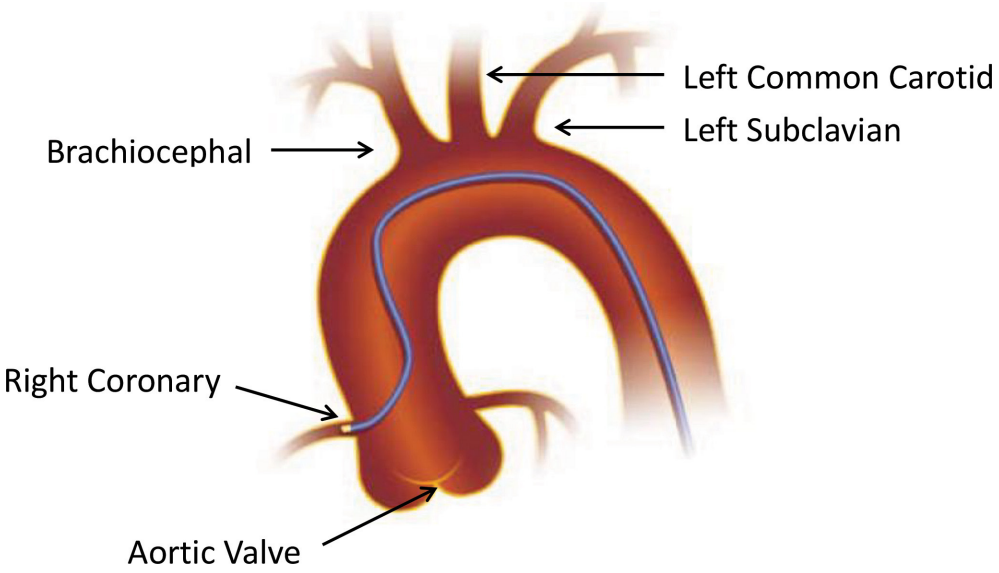

Figure 2.7: Aortic Arch with Brachiocephalic, Right Coronary Ostium, Aortic Valve Cusps, Left Common Carotid, Left Subclavian

- Immediately after this, is the rapid filling phase, in which the AV valves open and the majority of ventricular filling takes place
- As pressures in the atria and ventricles equalise, LV filling virtually stops.
- The last part of ventricular filling is atrial contraction, also called "atrial kick", which provides approximately 20% of the total ventricular filling
- With this, the cycle is complete and begins once again with systole

G. THE CONTROL OF PERIPHERAL CIRCULATION

Clochesey (1993) and Urden (2006) assert that the peripheral circulation is controlled by 2 mechanisms: intrinsic (local) control and extrinsic control.

a. INTRINSIC CONTROL

- This control is most influential at the arteriolar level
- The arterioles are the major resistance vessels because of the amount of smooth muscle in the vessel wall and they possess the ability to increaseor decrease the vessel
- Local factors influencing this balance are: medication, locally released catecholamines, medication, locally released catecholamines, histamine, acetylcholine, serotonin, angiotensin, adenosine and prostaglandins
- Other factors that influence circulation are temperature and carbon dioxide

b. EXTRINSIC CONTROL

- Is mediated by the autonomic nervous system and peripheral vascular reflexes
- The Autonomic Nervous System exerts antagonistic control over most organ systems via sympathetic (constrict) and parasympathetic fibres (dilate).
- Stimulation of the vasomotor centre in the medulla oblongata enhances sympathetic outflow, and inhibits parasympathetic outflow. This leads to an increase in blood pressure and heart rate
- Peripheral vascular reflexes include emotional states and temperature

H. REGULATION OF HEART RATE

Urden (2006) and Alspach (1991) state that heart rate is regulated by the autonomic nervous system (ANS) and intrinsic regulators:

a. AUTONOMIC NERVOUS SYSTEM

- Parasympathetic and sympathetic nervous system create the balance between the fight-and-flight and relaxation

- Parasympathetic fibres are mostly near the SA and AV nodes. This specifically involves the left & right vagus nerves: stimulation of the vagus nerve causes bradycardia, as a result of hyperpolarisation of phase 4 of the Action Potential (causes the slope to take longer to reach threshold)
- Sympathetic nerve fibres have a greater impact on the ventricles: acceleration and contractility is the main focus
- Sympathetic stimulation increases the heart rate and conduction through the AV node.

b. INTRINSIC REGULATION

- This includes the reflex network that serves as the feedback mechanisms to the brain. These reflexes work to maintain even blood flow, oxygenation and perfusion.
- These reflexes are: baroreceptors, right atrial receptors, natriuretic peptides, RAAS (renin angiotensin-aldosterone system) and the respiratory cycle.

c. BARORECEPTORS

- Baroreceptors are located in the aortic arch and carotid sinuses. They are more sensitive to wall stress (stretch) than absolute pressure. When an increase or decrease in pressure is noted, the autonomic nervous system is activated to increase or decrease heart rate
- The Baroreflex also initiates changes in venous tone to alter cardiac output according to need: vasoconstriction will increase blood return to the heart to augment stroke volume

d. CHEMORECEPTORS

- Arterial chemoreceptors, or the carotid and aortic bodies, are located in the carotid arteries at the bifurcation and in the aortic arch
- Their main function is to maintain homeostasis during hypoxemia
- Chemoreceptors signal changes in oxygen tension, drop in blood pH, and increase in carbon dioxide
- Stimulation of these chemoreceptors causes an increase in respiratory rate and depth and selectve constriction of resistance vessels to increase blood flow to the heart and brain.

e. NATRIURETIC PEPTIDES

- The atrial myocardium secretes atrial natriuretic peptide and the ventricular myocardium secretes brain natriuretic peptide
- These are secreted in response to chamber stretch
- Both peptides causes vasodilatation, natriuresis, inhibit the sympathetic nervous system and RAAS (apparatus in the kidney as response to decreased renal blood flow. Renin converts the protein angiotensinogen to angiotensin I, which gets converted to angiotensin II in the pulmonary vascular bed (by the enzyme ACE (angiotensin converting enzyme)). Peripheral vasoconstriction is the result, but also the release of aldosterone, which retains sodium, and thus water
- Respiratory influences
- Heart rate varies slightly with the respiratory cycle: rate accelerates on inspiration and decreases with exhalation
- Left ventricular-stroke volume decreases du- ring normal inspiration (sympathetic and vagal tone may be responsible for this)
- The above can be ascribed to:
- Decreased intrathoracic pressure leading to an increase in venous return to the right atrium and ventricle
- Bainbridge reflex (activation of stretch receptors in the lungs)
- Decreased left ventricular compliance resulting from increased right ventricular return
- Increased impedance to left ventricular outflow related to pleural pressure changes.

I. CARDIAC OUTPUT

Alspach (1991), Clochesey (1993), Braunwald (2001), Lichtenthal (2002) and Carlson (2009) elaborate on cardiac output as follows:

- Cardiac output (CO) is defined as the volume of blood ejected from the ventricles over 1 minute.
- The determinants for cardiac output is thus the heart rate (HR) and the stroke volume (SV):

  CO = HR X SV
- Normal CO: 4 – 8 L/minute
- Stroke volume as a value is influenced by 3 factors: preload, afterload and contractility

  Preload
  - Urden, et al (2006) states that preload is a function of the volume of blood presented to the left ventricle and also the compliance (ability of the ventricle to stretch) of the ventricles at the end of diastole. It can be assessed by the left ventricular end-diastolic volume (LVEDV)
  - The pressure distending the LV just before contraction is the LV end-diastolic pressure (LVEDP). This is a measure of the compliance.
  - Factors affecting preload: venous return, blood volume and atrial kick. Factors affecting the ventricular compliance include ventricular wall thickness and stiffness

  Afterload
  - Alspach (1991) defines afterload as the text ventricular wall tension or stress during systole. It is commonly described by the terms, systemic vascular resistance and peripheral vascular resistance. The increased afterload increases the work load of the heart, and thus the myocardial oxygen demand. An increase in afterload is caused by: aortic stenosis, vasoconstriction and obstructive hypertrophy.

  Contractility
  - This term refers to the heart's contractile force. Clochesey et al (1993) refer to the term inotropy. Inotropy can be negative or positive. Negative inotropy can be caused by certain drugs (like beta blockers) and positive inotropy by a sympathomimetic, like adrenaline. Contractility can also be enhanced by increasing the Starling's mechanism, using fluid replacement. The Frank Starling law of the heart states that the stroke volume of the heart increases in response to an increase in the volume of blood filling the heart (the end diastolic volume or preload) when all other factors remain constant. The increased volume of blood stretches the ventricular wall, causing the muscle to contract more forcefully, thus increasing stroke volume (SV) (the so-called Frank Starling mechanism). This occurs up to a point of maximal stretching, above which the stroke volume will not increase further. A good analogy is stretching a rubber band: The more you stretch it, the stronger it will recoil, up to a point where it becomes "overstretched".

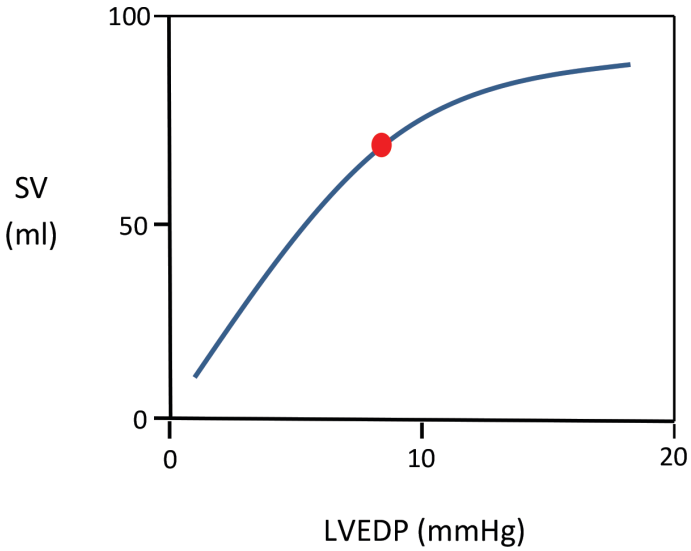

Figure 2.8: Frank-Starling mechanism.

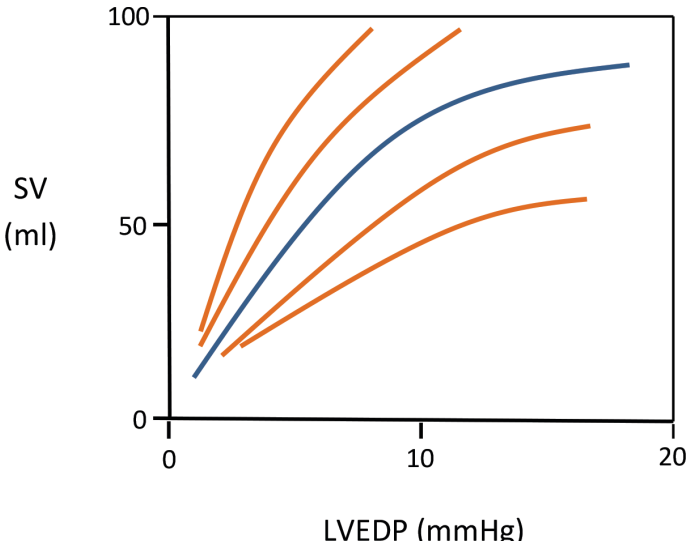

Figure 2.9: Family of Frank-Starling curves.

1.3 Basic ECG Interpretation

Electrocardiography is an imperative diagnostic component on how the heart generates and conducts electrical impulses.

Learning how to identify ECG changes and understanding the implications and impact on the patient's condition is considered a critical skill which a catheterisation laboratory nurse must possess to function effectively in this environment.

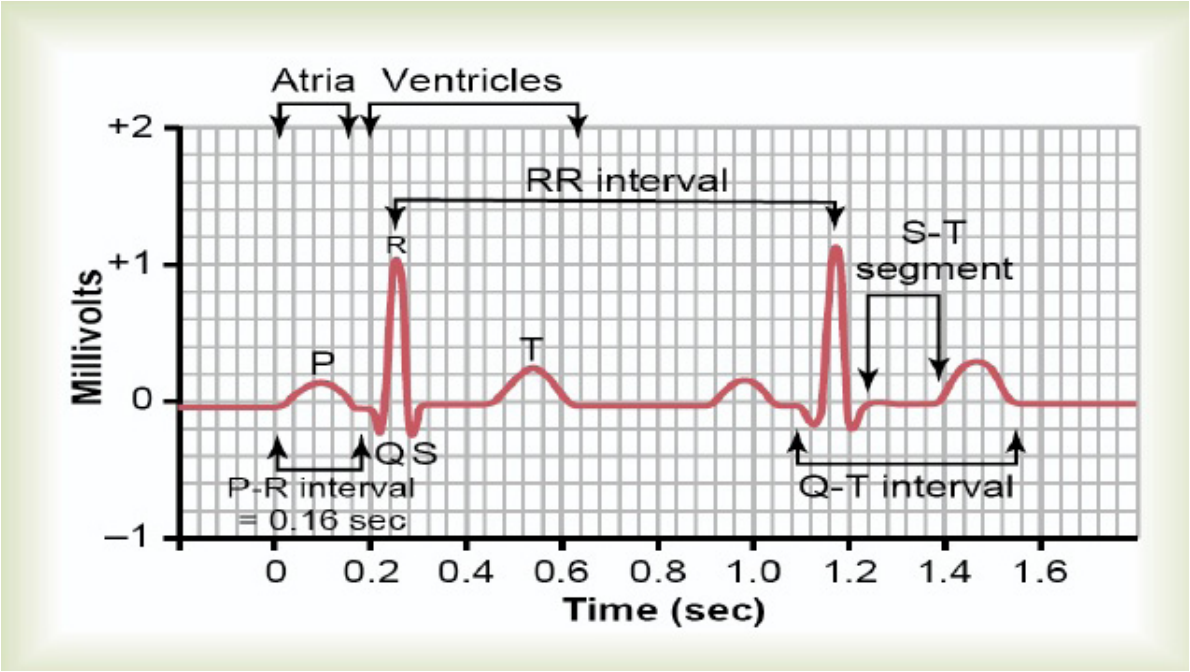

Figure 3.1: A rhythm strip with associated ECG components

Table 3.1: Cardiac conduction and its relationship to the ECG cardiac cycle

COMPONENT	DESCRIPTION	REPRESENTS	VALUE
P wave	First waveform	Atrial depolarisation	< 0.10 sec < 2.5 mm
PR interval	Beginning of P wave to beginning of QRS complex	The impulse through the atria, AV, the Bundle of His, the bundle branches and Purkinje fibres	0.12 – 0.20 sec
QRS complex	Contains 3 waves: Q wave; R wave; S wave (may not all be present)	Depolarisation of ventricles	0.06 – 0.10 sec
Q wave	First negative deflection after P wave	Depolarisation of septum	< ⅓ of QRS
R wave	First positive deflection following P or Q wave	Depolarisation of ventricles	< 12sec
S wave	Negative deflection following R wave	Depolarisation of ventricles	N/A
T wave	First waveform after S wave	Repolarisation of ventricles	< 6 mm in limb leads < 10 mm in precordial leads
U wave	Waveform following T wave	Repolarisation of Purkinje fibres	N/A
QT interval	Beginning of Qwave to the end of the T wave	Duration of ventricular depolarisation and repolarisation	≤0.46 sec
ST segment	Following the S wave to the beginning of the T wave	Initial ventricular repolarisation	On iso-electric line < 0.12 sec

ECG PAPER

ECG paper usually moves at a speed of 25 mm/second. At that speed the following are standard intervals on ECG paper:

- 1 tiny square: 0.04 seconds
- 1 large square (5 tiny squares): 0.20 seconds
- In a 6 second strip there are 30 large squares
- In a 1 minute strip there are 300 large squares
- In a 1 minute strip there are 1500 tiny squares

The horizontal axis measures time (in seconds). The vertical axis measures voltage or amplitude. A small square measures 0.1 mV in voltage and 1 mm in amplitude. A large square measures 0.5 mV in voltage and 5 mm in voltage (Hampton: 2003).

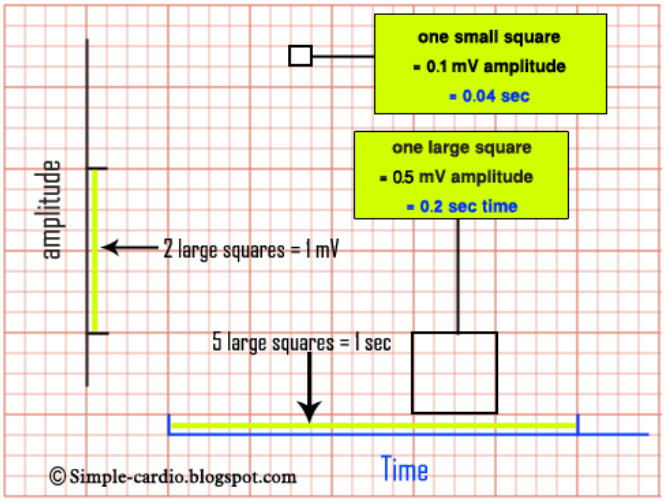

Figure 3.2 ECG paper

HEART RATE ESTIMATION

Hampton (2003) states that there are a few strategies available to determine the heart rate on an ECG or a rhythm strip. The heart rate is recorded on the ECG by most ECG machines, but it is useful when rate recording is not present.

First method:

- To estimate heart rate, memorise the rate intervals: 300, 150, 100, 75, 60, 50, 43 and 38.
- Pick a QRS-complex that falls on a dark thick line.
- Using the above "count- down" , the patient's heart rate can be estimated (see figure below)

Second method:

- Count the number of QRS-complexes on a 6-second strip and multiply it by 10.

Third method:

- Divide 300 by the number of large squares between 2 consecutive QRS-complexes

Fourth method:

- Divide 1500 by the number of tiny squares between 2 consecutive QRS-complexes

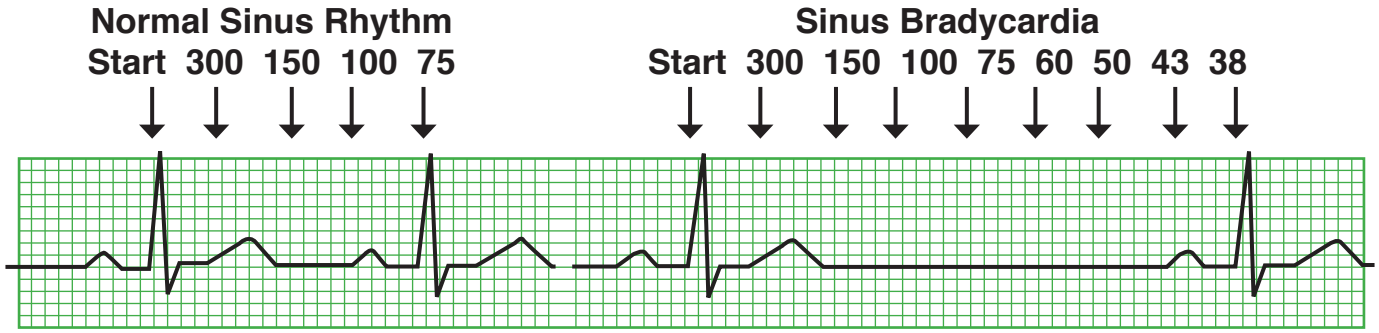

Figure 3.3 Normal Sinus Rhythm vs Sinus Bradycardia

ABNORMAL CARDIAC RHYTHMS

All the rhythm strips were obtained from: ISCAP

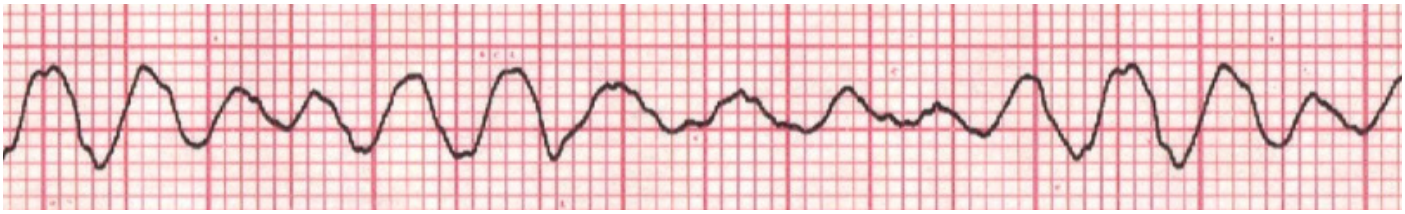

Figure 3.4: Ventricular Fibrillation

This represents a severe derangement of the heartbeat which is usually terminal within 3-5 minutes unless corrective measures are undertaken promptly. It occurs in a variety of clinical situations but is most commonly associated with coronary artery disease.

Defining criteria: unable to determine a QRS-complex. Irregular undulations occur with varying contour and undulations. There are no distinct QRS, ST segment or T waves.

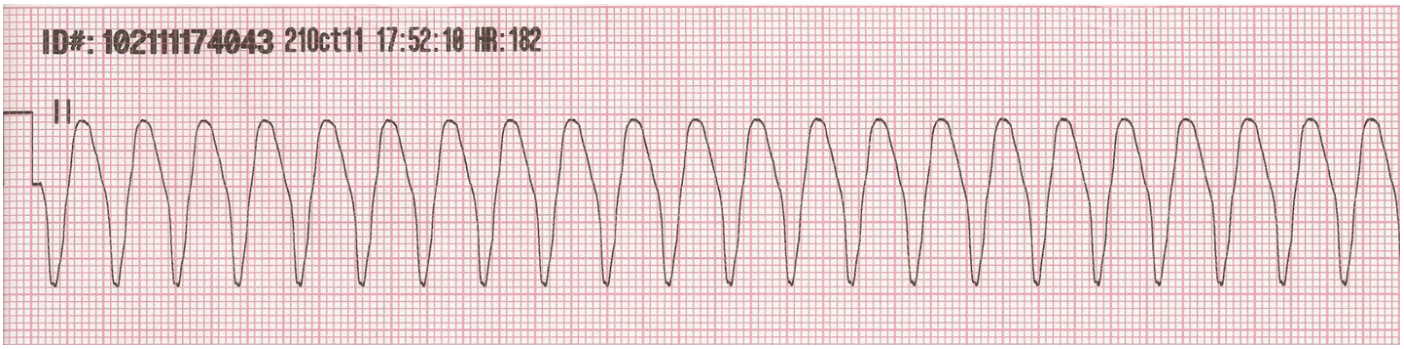

Figure 3.5: Ventricular Tachycardia

Impulse conduction is slowed around areas of ventricular injury, infarct or ischemia. These areas are sources of ectopic impulses. These areas can cause the impulse to take a circular course, leading to the re-entry phenomenon and rapid repetitive depolarizations. This is the most common mechanism. The ventricular rate is typically 120 – 250 beats. The rhythm is regular. The QRS-complex is wide with a largeT- wave of opposite polarity from the QRS-complex.

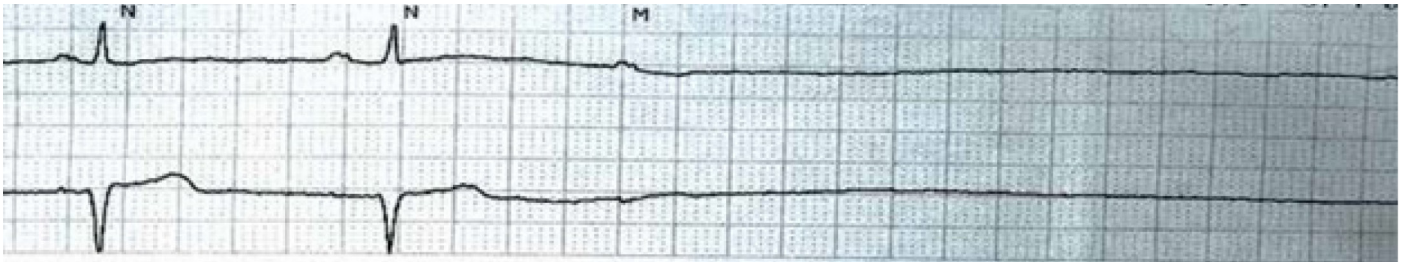

Figure 3.6: Asystole

In asystole, no ventricular or atrial activity is seen or less than 6 complexes per minute.

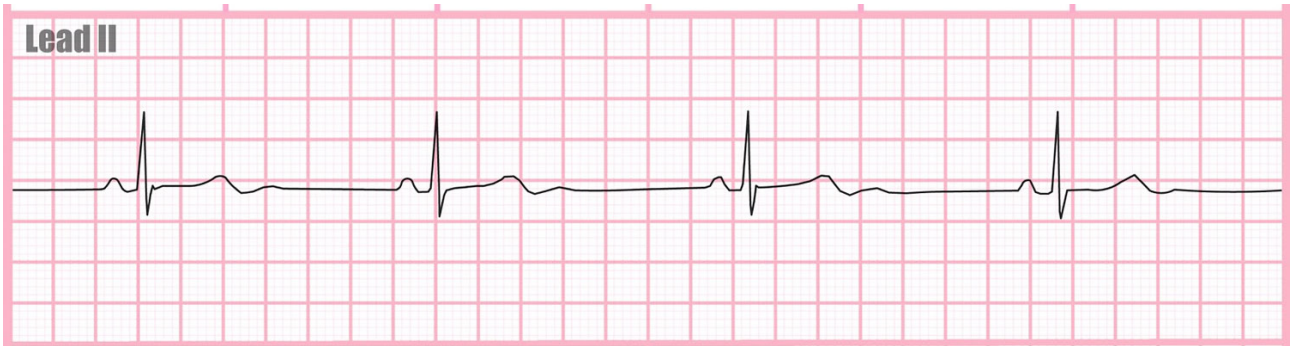

Figure 3.7: Sinus Bradycardia

In sinus bradycardia, the impulses originate from the SA node at a slow rate. The rate is lower than 60 b/min, PR-interval is regular in every complex. Every P-wave is followed by a QRS-complex.

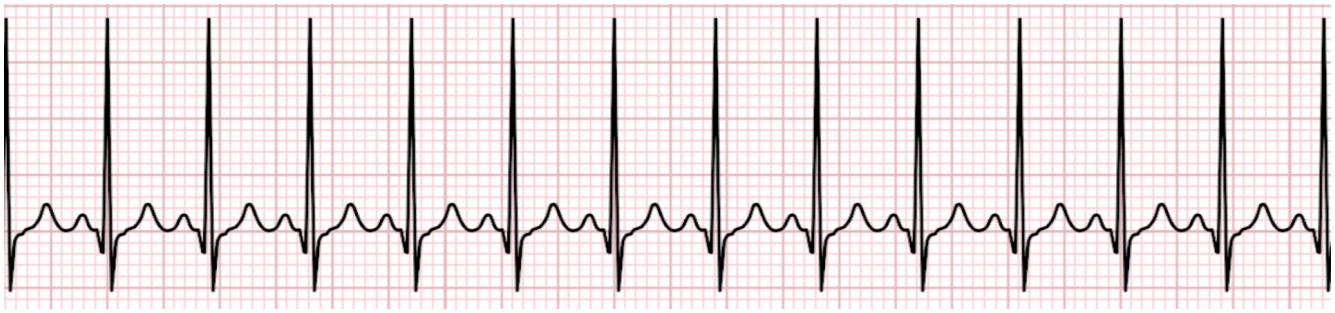

Figure 3.8: Sinus Tachycardia

In sinus tachycardia, the impulses originate from the SA node at a fast rate. The rate is higher than 100 b/min. R-R interval is regular. Every P-wave is followed by a QRS-complex.

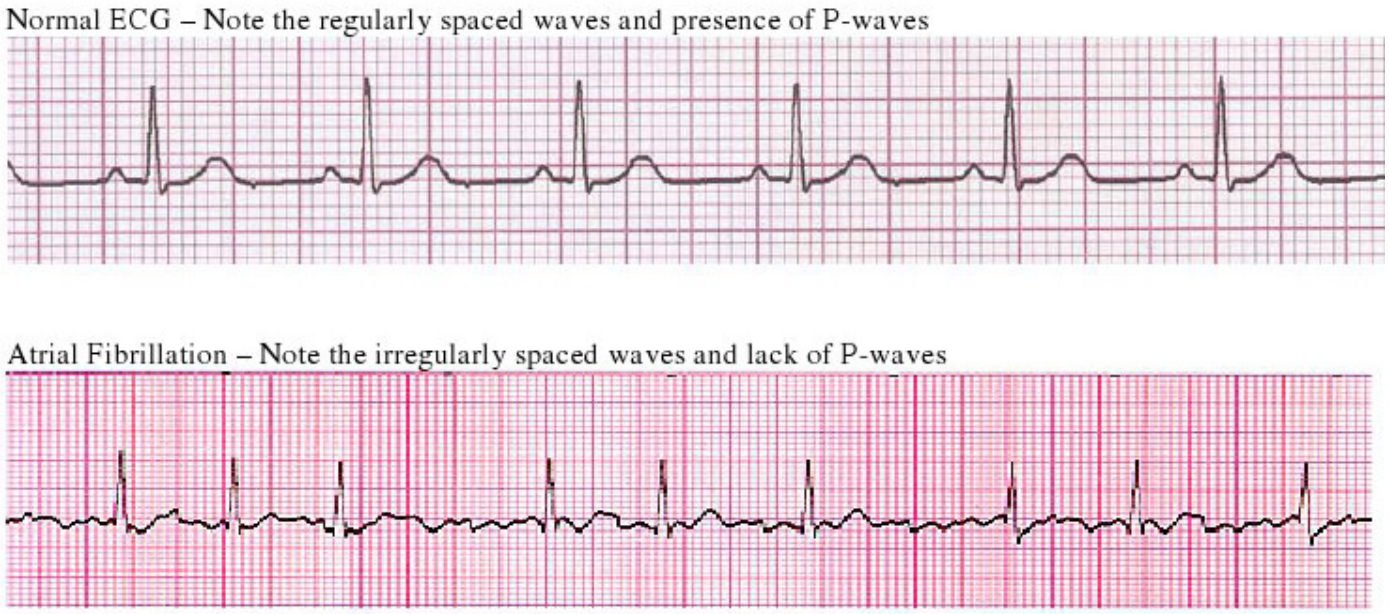

Figure 3.9: Atrial Fibrillation

Atrial impulses are faster than the SA node impulses. The impulses take multiple, chaotic, random pathways through the atria. Fine fibrillation waves are noted with no discernible P-waves. Atrial undulations occur between 300 – 400 per minute. The R-R interval is irregular.

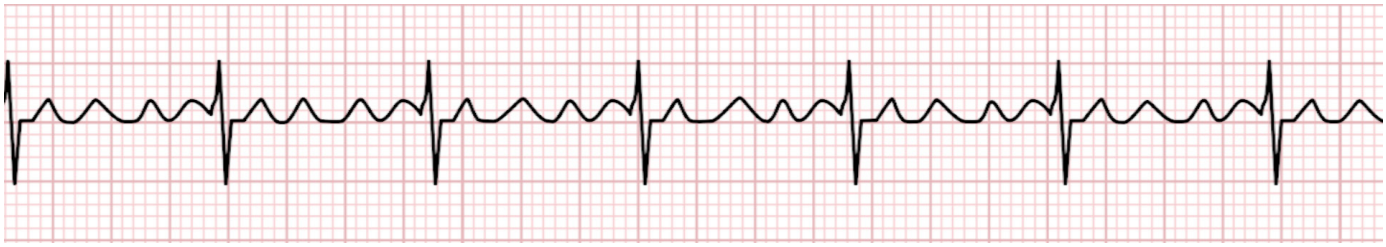

Figure 3.10: Atrial Flutter

During atrial flutter, the impulses take a circular course around the atria, setting up flutter waves. The atrial rate ranges between 220 – 350 beats per minute. The ventricular response is rarely above 150 beats/minute due to AV node conduction limits. The baseline appears as a "saw tooth" pattern.

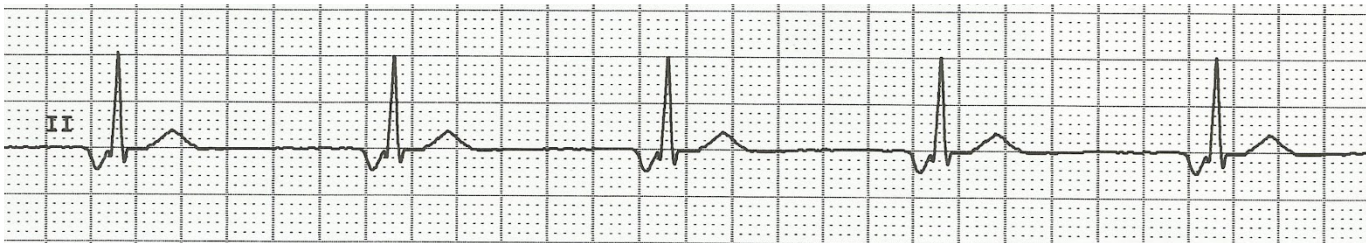

Figure 3.11: Junctional rhythm

A junctional rhythm occurs when the pacing rhythm of the heart originates in the AV node or first part of the bundle of His. This may occur as an escape rhythm if there is severe sinus bradycardia or sinus arrest. It may also occur due to increased automaticity of the AV node, when it is called nodal tachycardia. The atria are depolarised in a retrograde manner. If the retrograde depolarisation is rapid, the retrograde P wave occurs at the same time as the QRS complex and no retrograde P waves are visible (Upper ECG strip).

When retrograde atrial repolarisation takes a bit longer, a retrograde inverted P wave is visible in the ST segment (lower ECG strip).

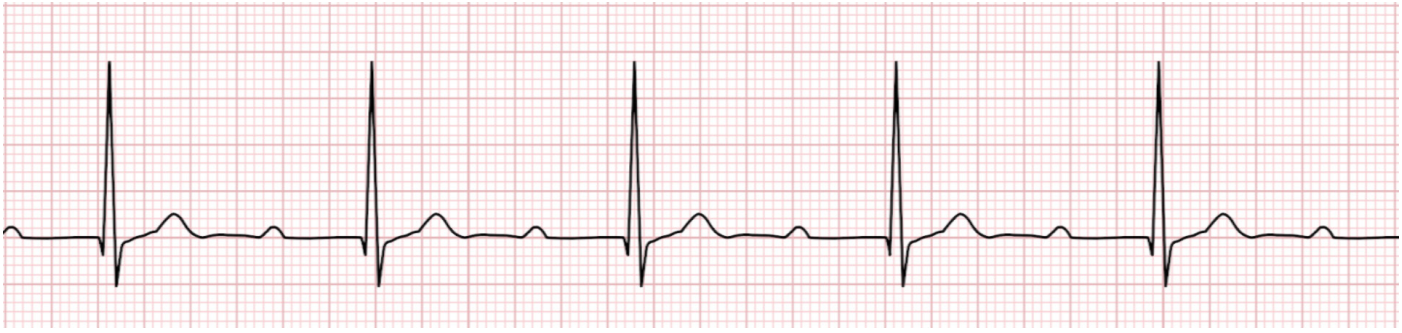

Figure 3.12: First Degree Heart Block (AV-Block)

During 1st degree AV block, impulse conduction is slowed at the AV node or the His-Purkinje system for a fixed interval. The PR-interval is prolonged (> 0.20 seconds). Every P-wave is followed by a QRS-complex.

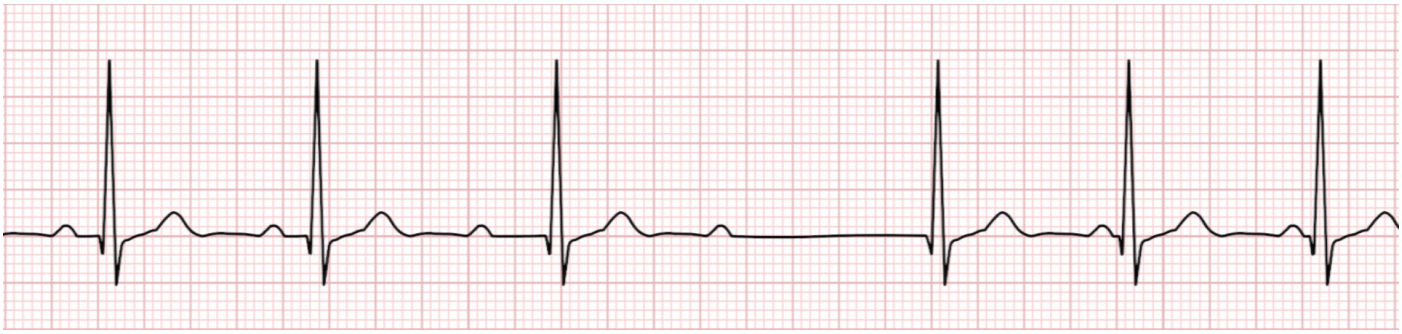

Figure 3.13: Second Degree Heart Block: Wenckebach (Mobitz type I)

This dysrhythmia is associated with AV node pathology. Impulse conduction is progressively slowed at the AV node (causing a progressively prolonging PR-interval) until one sinus impulse is completely blocked and a QRS-complex fails to follow.

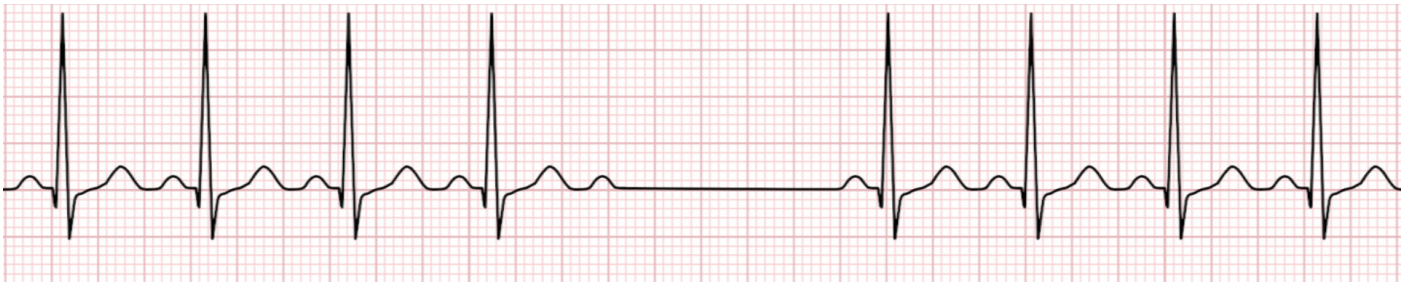

Figure 3.14: Second Degree Heart Block: Mobitz type II

In 2nd degree AV block, the site of the block is usually below the AV node (infranodal) at the bundle of His or at the bundle branches. Impulse conduction through the AV node is normal. Some P-waves are not conducted, thus the absent QRS-complexes. The atrial rate is regular, but the ventricular rate is irregular. Narrow QRS-complexes indicate a high block relative to the AV node, and wide QRS-complexes implies low block relative to the AV node.

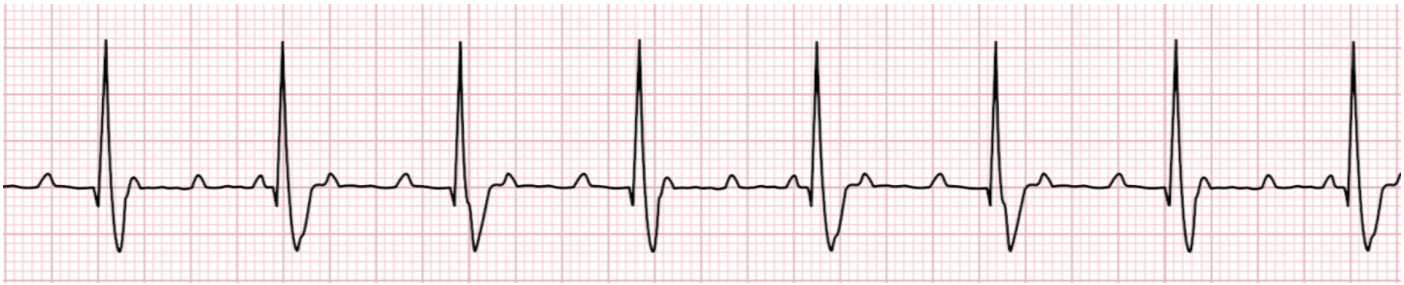

Figure 3.15: Third Degree Heart Block

No impulses pass between the atria and the ventricles. The atria and the ventricles depolarize independently, with no relationship between the two. The atrial rate is usually 60 – 100 beats per minute and the ventricular rate depends on the rate of ventricular escape (20 – 60 beats per minute).

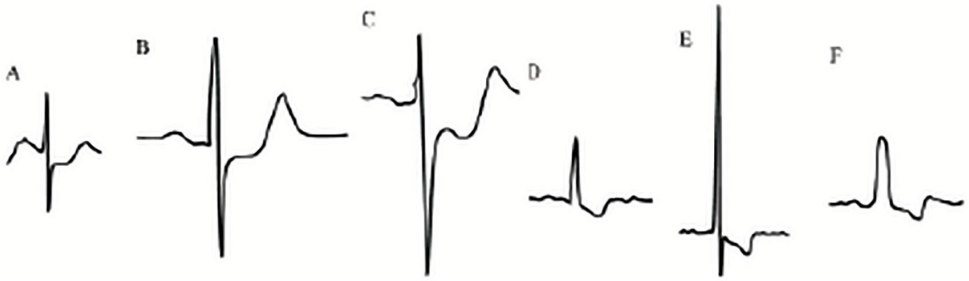

Figure 3.16: ST-segment changes

a. The various causes of electrocardiographic ST segment depression.
b. ST segment depression related to non-infarction ischaemia, horizontal in morphology.
c. Reciprocal ST segment depression in lead III in a patient with acute anterior wall AMI.
d. Lead V2 STD attributable to posterior wall AMI.
e. Digoxin effect. (E) Left ventricular hypertrophy.
f. Left bundle branch block.

An elevated ST-segment is indicative of myocardial injury or infarction. Inverted T- waves occurs when myocardial ischemia is present. The presence of a pathological Q-wave indicates a trans-mural myocardial infarction, and may take months or years to disappear.

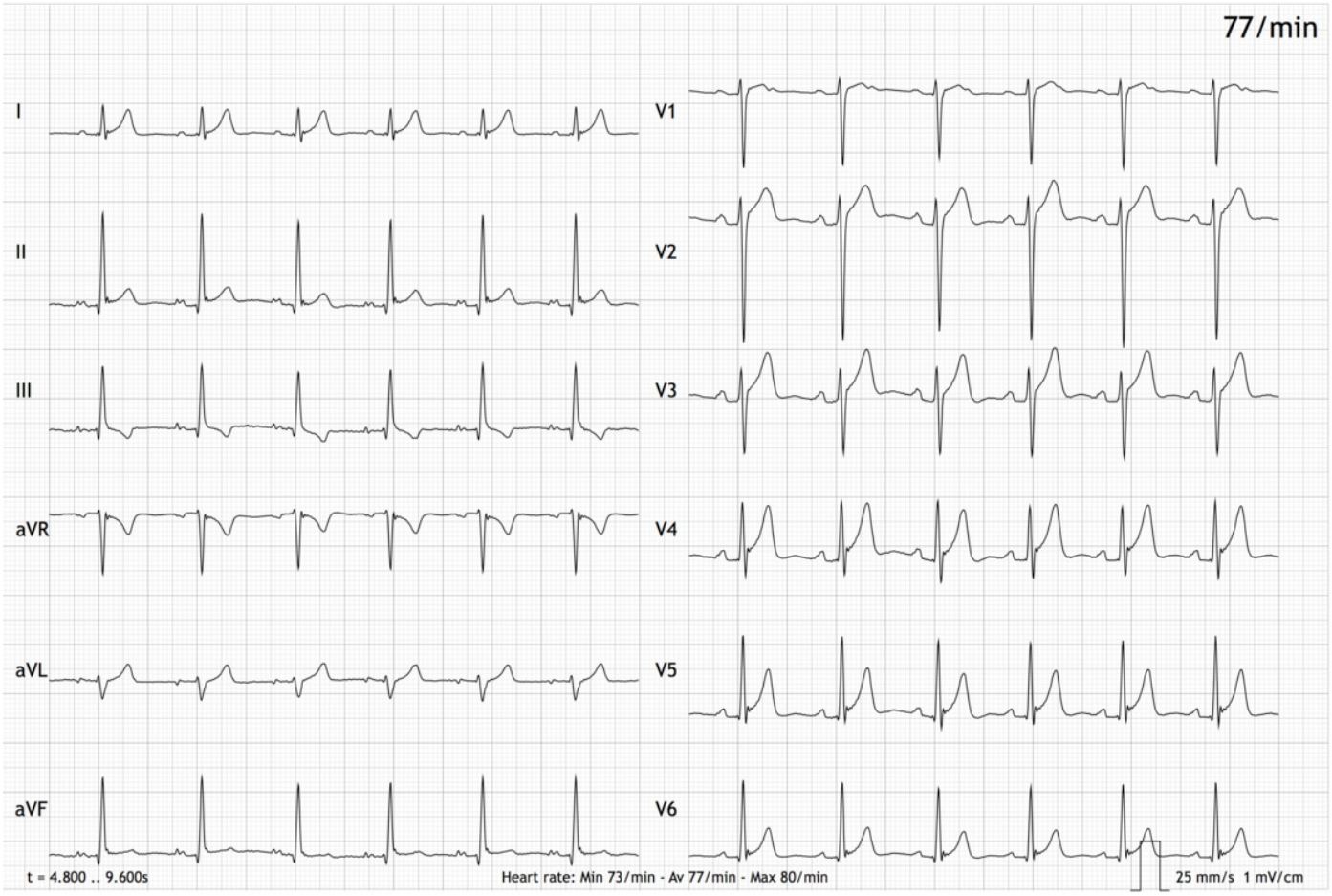

Figure 3.17: The 12-lead ECG

Table 3.2: A Guideline for interpreting a 12-lead ECG
Compiled by Dr. W. Kloeck and Dr. M. Wells

Rate	300 150 100 75 60 50 43 38 33 30 Is atrial and ventricular rate the same? Bradycardia/Tachycardia/accelerated rhythm?
Rhythm	Is every QRS-complex preceded by an identical P-wave? Is atrial and ventricular rate the same? Regular/irregular? PAC/PJC/PVC/Escape beats?
QRS axis	Normal if S1 and aVF are both positive (Normal: -30° to +90°) Exact axis is 90° to most iso-electric and equiphasic lead
P wave	Present/absent? Retrograde? Multifocal? Configuration? (especially S2 and V1)
PR(PQ) interval	Short or long? (Normal: 0.12 – 0.2) AV block? (1st, 2nd Type I/Type II, 3rd degree) Regular/irregular block? Ratio?
QRS complex	Width? (Normal <0.12) Height? (Electrical alternans?) LBBB? (V1 "W" or "U"-shaped / V6 "M"-shaped) RBBB? (V1 "M"-shaped / or V6 "W" or "U"-shaped) Pathological Q-wave? (>0.04 wide & deeper than ¼ of R-wave) Other waves? (Delta wave, Osborne wave, Pacing spikes) Small complexes: tamponade, hypothyroidism, COAD, obesity, myocarditis)
ST segment	Elevation ≥ 1mm?/depression ≥0.5mm? (measured 0.04s after J-point) Shape? (saddle/ camel-hump)
T wave	Tall? (>5mm in limb leads; >10mm in precordial leads) (Symmetrically peaked with narrow base: ↑K+ / ↑Mg2+) (Asymmetrical with broad base: ischemia) Inverted? Dynamic? T-wave alternans?
U wave	Present? (Prominent in ↓K+)
QT interval	QTc = (QT-interval ÷ √R-R interval). Normal: 0.36 – 0.46s Normal QT-interval is < than ½ of the R-R interval
Hypertrophy	LA (P-wave broad (>0.12s) & notched in S2 / biphasic in V1) RA (P-wave tall & peaked in S2 (>2.5MM) &V1) LV (LAD) S-wave in V1 & R-wave in V5/V6 >35mm; R in aVL>12mm RV (RAD) & tall R-wave in V1 & aVR Strain pattern? (Asymmetrical ST-depression & T-wave inversion) LV strain: S1, aVL,V4-V6; RV strain: S2,S3, aVF, V1-V3
Ischaemia	ST elevation ≥ 1mm/ ST depression ≥0.5mm in contiguous leads? Pathological Q-waves? Dynamic T-wave inversion? RV infarction (look at aVR & V4(R) Posterior infarction? (Tall R-wave in V1 – "inverted mirror image") Poor R-wave progression? Additional views? (V7-V9)
Metabolic	K+ (↑: tall peaked T-waves with narrow base. T>R-wave in 2/ more leads): ↓K+: big "U"-waves. Mg2+: ↑- peaked T-wave; ↓: long QT-time Ca2+: ↑ - short QT-time; ↓: long QT-time

1.4 Coronary Pathology

Coronary artery disease is a progressive atherosclerotic disorder that results in intracoronary narrowing or complete coronary artery occlusion. This results in an imbalance between myocardial oxygen supply and demand (Woods: 2005).

This chapter focuses on the following pathologies: coronary artery disease, angina, myocardial infarction and ventricular remodelling.

A. CORONARY ARTERY DISEASE

The pathophysiological process of coronary artery disease is: the development of atherosclerosis, atherosclerotic plaque rupture and plaque regression.

DEVELOPMENT OF ATHEROSCLEROSIS

Woods (2005) states that atherosclerosis is a chronic inflammatory disorder that is characterised by an accumulation of macrophages and T lymphocytes in the tunica intima. A high level of low density lipoprotein (LDL) is one of the triggers of vascular inflammation. Other major causes of endothelial injury are hypertension, diabetes mellitus and smoking.

The inflammation injures the wall, allowing the LDL cholesterol to move into the vessel wall below the endothelial surface. In addition, blood monocytes adhere to endothelial cells and migrate into the vessel wall. Within the artery wall, some monocytes differentiate into macrophages that unite with, and then internalize, LDL cholesterol. The foam cells that result are the marker cells of atherosclerosis.

Elevated LDL cholesterol levels promote low-level endothelial inflammation that allows lipoproteins to infiltrate the tunica intima. Once infiltrated under the endothelium, the LDL tends to stay within the vessel wall rather than return to the circulation. HDL enters the vessel wall, helps efflux cholesterol from cells, and then returns to the circulation.

PLAQUE RUPTURE

When the "mature" plaque develops, it is not uniform in composition: it has a lipid liquid centre filled with procoagulant factors and a connective tissue fibrous cap that covers the top of the fluid lipid centre.

The abrupt rupture of this cap allows procoagulant lipids to flood into the vessel lumen causing platelet activation and local formation of a blood clot. As the enlarging clot blocks blood flow through the coronary artery, myocardial ischaemia will occur unless there is adequate collateral circulation from other coronary vessels. Plaques likely to rupture are saturated with macrophages and other inflammatory cells. These vulnerable plaques are usually non-obstructive and are located at bends or branch points in the arterial tree. It is not yet known what causes the fibrous cap to erode and then rupture. As deep fissures in the cap expose the procoagulant factors to the blood plasma, an unstoppable cycle is initiated: when platelets are exposed to collagen, necrotic debris, von Willebrand factor and thromboxane, a thrombus is formed that can occlude the coronary artery (Braunwald et al: 2001).

Highly fibrotic plaques do not rupture. The type of atherosclerotic plaque that is prone to rupture has a weak fibrous cap and a large amount of liquid cholesterol within the core (Watson: 2005).

PLAQUE REGRESSION

A reduction in blood cholesterol decreases atherosclerotic plaque size by decreasing the amount of liquid cholesterol within the plaque core. Lowering cholesterol levels will not change the dimensions of the fibrous calcified portions of the plaque. However, lowering cholesterol levels reduces vascular inflammation and makes vulnerable plaque less likely to rupture (Urden: 2006).

B. ACUTE CORONARY SYNDROME

The American College of Cardiologists (2012) ascribes acute coronary syndrome (ACS) to the array of clinical presentations of coronary artery disease and includes: stable angina, unstable angina, variant angina, silent ischaemia and myocardial infarction.

ANGINA

Angina is caused by myocardial ischaemia and is not a separate disease, but rather a symptom of coronary artery disease, usually caused by a blockage or spasm of a coronary artery that results in a lack of oxygen supply to the myocardium.

STABLE ANGINA

Stable angina is predictable and caused by similar precipitating factors each time, usually exercise. Stable angina is the result of fixed lesions (blockages) of more than 75% of the coronary lumen. Ischaemia and chest pain occur when myocardial oxygen demand from exertion exceeds the fixed blood oxygen supply.

UNSTABLE ANGINA

Unstable angina is defined as a change in a previously established pattern of angina. Unstable angina is more intense than stable angina. Unstable angina is an indication of atherosclerotic plaque instability. It can signal atherosclerotic plaque rupture and thrombus formation that can lead to myocardial infarction.

VARIANT ANGINA (PRINZMETAL'S ANGINA)

Variant angina is chest pain caused by arterial spasm. Spasm occurs with or without atherosclerotic lesions. Smoking, alcohol and drug abuse may precipitate coronary artery spasm.

SILENT ISCHAEMIA

In this type of angina, all the evidence indicating ischaemia is present, but the patient is asymptomatic.This is usually evident in the diabetic population suffering from autonomic neuropathy.

C. MYOCARDIAL INFARCTION (MI)

This refers to irreversible myocardial necrosis that results from an abrupt decrease or total cessation of coronary blood flow to a specific area of the myocardium. There are 3 mechanisms that are responsible for an acute reduction in oxygen supply to myocardium:

- Plaque rupture
- New coronary artery thrombosis
- Coronary artery spasm

Myocardial tissue can best be salvaged within the first 2 hours after onset of anginal symptoms (American Heart Association, 1999).

PATHOPHYSIOLOGY ASSOCIATED WITH MI

During the pathophysiological process of myocardial infarction, there is differentiation between ischaemia, injury and infarction (Alspach:

1991; Clochesey: 1993; Braunwald: 2001; Carlson: 2009).

ISCHAEMIA

The outer region of the infarcted myocardial area is called the zone of ischaemia. It is composed of viable myocardial cells. Repolarisation in this zone is temporarily impaired but will eventually restore itself to normal. Repolarisation of the cells in this area is presented as ST - segment depression and inverted T- waves on the ECG in the zone of ischaemia.

INJURY

The infarcted zone is surrounded by injured but still potentially viable tissue. Myocardial cells in this area do not fully repolarise because of a blood flow deficiency. This is represented by ST-segment elevation on the ECG.

INFARCTION

The area of dead myocardial muscle is called the zone of infarction. This is represented by ST -segment elevation and later pathological Q - waves on ECG.

This indicates a lack of depolarisation from the cardiac surface involved in the MI. A patient may suffer from an MI without the presence of a Q - wave (NQWMI) or NSTEMI (non ST - segment elevation myocardial infarction) and the diagnosis is made by elevated cardiac enzyme profile as well as a troponin leak. As healing occurs, the dead cells in this area are replaced by scar tissue.

It is important to be aware that instead of infarction, sometimes myocardial cells that have been subjected to severe ischaemia may become stunned or hibernate if blood flow is re-established in time. Myocardial stunning occurs when, after a brief period of severe ischaemia and an initial period of cardiac dysfunction, normal contractility gradually returns over a period of hours to days. The hibernating myocardium refers to severe ischaemic myocardium that experiences a return of normal functioning over a period of days to weeks after re-establishment of coronary blood flow.

The location of the infarction is determined by the coronary artery that supplies an area of myocardium. Infarction most commonly affects the left ventricle and the interventricular septum. However, the right ventricle can also suffer a myocardial infarction.

Areas of myocardial infarction include: anterior wall, left lateral wall, inferior wall and the posterior wall.

Fuster et al (2011) states that a right ventricular infarction occurs when there is a blockage in the proximal section of the RCA proximal to the right ventricular branch. Right ventricular ischaemia can be demonstrated in 50% of inferior wall MI's. Only 10% - 15% shows hemodynamic instability. If a massive right ventricular MI occurs, the patient may suffer from cardiogenic shock, which carries over 50% mortality in this population group. A V4R lead is placed to diagnose a right ventricle infarction. ST-segment elevation in this lead can only be seen in the acute phase of infarction. Posterior wall infarction can be suspected when tall R-waves are seen in V1 and V2.

COMPLICATIONS ASSOCIATED WITH MYOCARDIAL INFARCTION

Hurst & Alpert (1994) identified the following complications to be associated with a myocardial infarction:

- Brady-arrhythmias
- Tachy-arrhythmias
- Atrio-ventricular block
- Ventricular aneurysm
- Ventricular septal rupture
- Papillary muscle rupture
- Cardiac wall rupture
- Pericarditis
- Heart failure

D. VENTRICULAR REMODELLING

Sidebotham (2007) defines ventricular remodelling as an alteration in ventricular structure that occurs as part of normal growth or due to a pathological process such as hypertension, valvular disease, MI or cardiomyopathy. The primary feature of remodelling is hypertrophy.

Ventricular hypertrophy is an adaptive response to a change in loading conditions that helps to attenuate ventricular dilatation, reduce wall stress, and stabilise contractile function. Ventricular hypertrophy is initiated by myocardial stretch and various neuro-endocrine processes.

Chronic pressure overload such as that which occurs with hypertension and aortic stenosis typically results in concentric hypertrophy, in which ventricular wall thickness is increased out of proportion to the increase in chamber size.

Chronic volume overload such as that which occurs with aortic and mitral regurgitation typically results in eccentric hypertrophy, in which wall thickness is increased in proportion to chamber size.

Following MI, ventricular remodelling (hypertrophy, dilation, and impaired contractility) can occur in sites adjacent to or remote from the zone of infarction.

Fuster et al (2011) mention that the signal for ventricular remodelling is complex, but it involves:

- Alterations in ventricular loading conditions
- Activation of neuro-endocrine pathways (sympathetic nervous system, RAAS and natriuretic peptides)
- Inflammation within infarcted and non-infarcted myocardium

Ventricular remodelling can eventually result in irreversible cardiac dysfunction due to myocardial fibrosis. If appropriate therapy is introduced early enough, remodelling can be interrupted or reversed.

Treatment is directed to the underlying cause: valvular disease and hypertension control. Pharmacological antagonists include: ACE-inhibitors, β-blockers and aldosterone antagonists. This is proven to be partially effective.

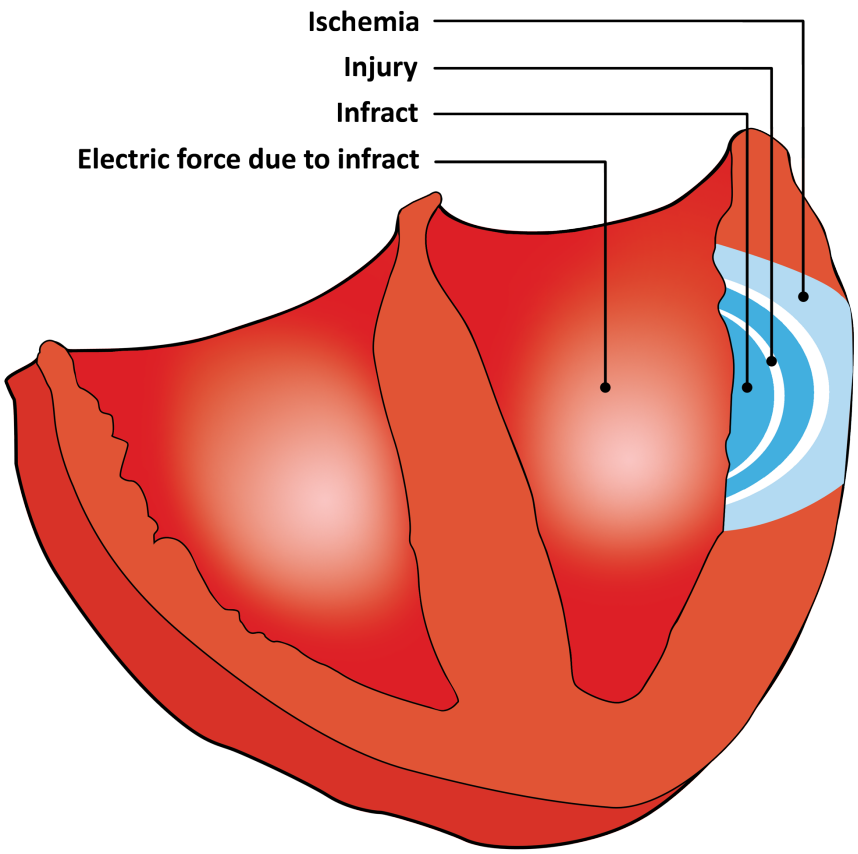

Figure 4.2: A Schematic Indicating the Zones of Ischemia, Injury and Infarction

Table 4.1: Correlation between ventricle surface, ECG leads and coronary arteries involved.

Surface of the left ventricle	ECG leads	Involved coronary artery
INFERIOR	II, III, AvF	Right coronary artery
LATERAL	V5 – V6, I, AvL	Circumflex artery
ANTERIOR	V2 – V4	Left anterior descending
ANTERIOR LATERAL	V1 – V6, I, AvL	Left main artery
SEPTAL	V1 – V2	Left anterior descending
POSTERIOR	V1 – V2 (Tall R wave and marked ST depression)	Circumflex or RCA

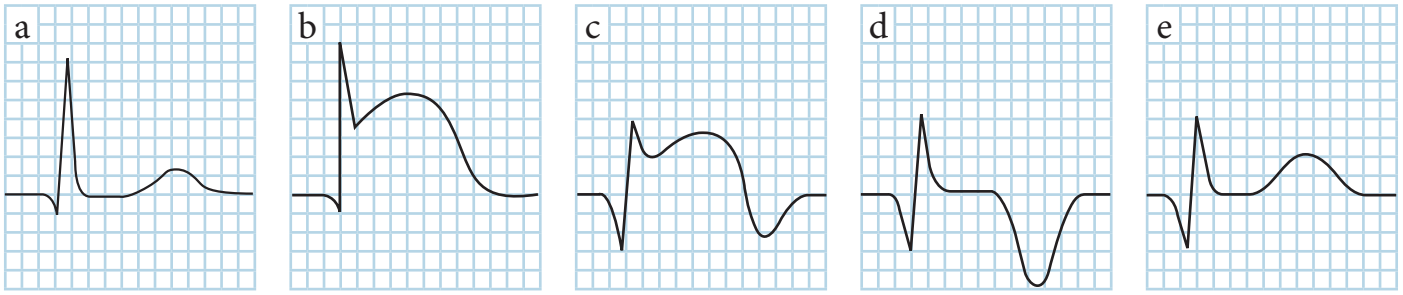

Figure 4.2: A Schematic Indicating the Zones of Ischemia, Injury and Infarction

a. **Normal ECG**
b. **ST-segment elevation (hyper-acute)**
c. **ST-segment elevation with an inverted T-wave**
d. **Inverted T-wave**
e. **Pathological Q-wave**

Note the progressive loss of R wave amplitude from b to c as a Q wave develops. This indicates a loss of cardiac muscle due to necrosis.

This Module is Linked to a CPD Accredited Online Questionaire at www.sasci.co.za

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