A dissertation on

An Autopsy based Examination of heart findings in Electrocution and Sudden Cardiac Death cases.

Dissertation Submitted to

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With partial fulfillment of the regulations for the award of the degree of

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MAY 2020

DEPARTMENT OF FORENSIC MEDICINE

COIMBATORE MEDICAL COLLEGE

COIMBATORE – 641 014.

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DECLARATION

I hereby declare that the dissertation entitled "**AN AUTOPSY BASED EXAMINATION OF HEART FINDINGS IN ELECTROCUTION AND SUDDEN CARDIAC DEATH CASES**" is a bonafide research work done by me in the Department of Forensic Medicine, Coimbatore Medical College during the period from January 2018 to June2019 under the guidance and supervision of **Dr. T. Jeyasingh, M.D, D.L.O**., Professor and Head, Department of Forensic Medicine, Coimbatore Medical College.

This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University, Chennai towards the partial fulfillment of the requirement for the award of M.D., Degree (Branch XIV) in Forensic Medicine. I have not submitted this dissertation on any previous occasion to any university for the award of any Degree.

Place: Coimbatore **Dr. M. SAKTHIMANI**

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CERTIFICATE OF APPROVAL

To
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Dear Dr.Sakthimani M,

The Institutional Ethics Committee of Coimbatore Medical College, reviewed and discussed your application for approval of the proposal entitled **"Autopsy Based examination of Heart Findings in Electrocution, and sudden Cardiac Death."** No.031/2017.

The following members of Ethics Committee were present in the meeting held on 21.11.2017.conducted at MM - II Seminar Hall, Coimbatore Medical College Hospital Coimbatore-18.

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15	Mr.V.A.Shahul Hameed, +2	Lay-Person

We approve the Proposal to be conducted in its presented form.

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The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

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CERTIFICATE

This is to certify that this dissertation entitled, "**AN AUTOPSY BASED EXAMINATION OF HEART FINDINGS IN ELECTROCUTION AND SUDDEN CARDIAC DEATH CASES"** submitted by **Dr. M. SAKTHIMANI** in partial fulfilment for the award of the Degree of M.D. (Forensic Medicine) by The Tamilnadu Dr. M. G. R.Medical University, Chennai is a bonafide record of the research work done by her, under the guidance of **Dr. T. JEYASINGH, M.D (FM). DLO,** Professor and Head, Department of Forensic Medicine, Coimbatore Medical College during the academic year 2017-20 in the Department of Forensic Medicine, Coimbatore Medical College, Coimbatore. This dissertation is a record of fresh work done by the candidate **Dr. M. SAKTHIMANI,** during the course of the study (2017-2020). This work was carried out by the candidate herself under my supervision.

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ACKNOWLEDGEMENT

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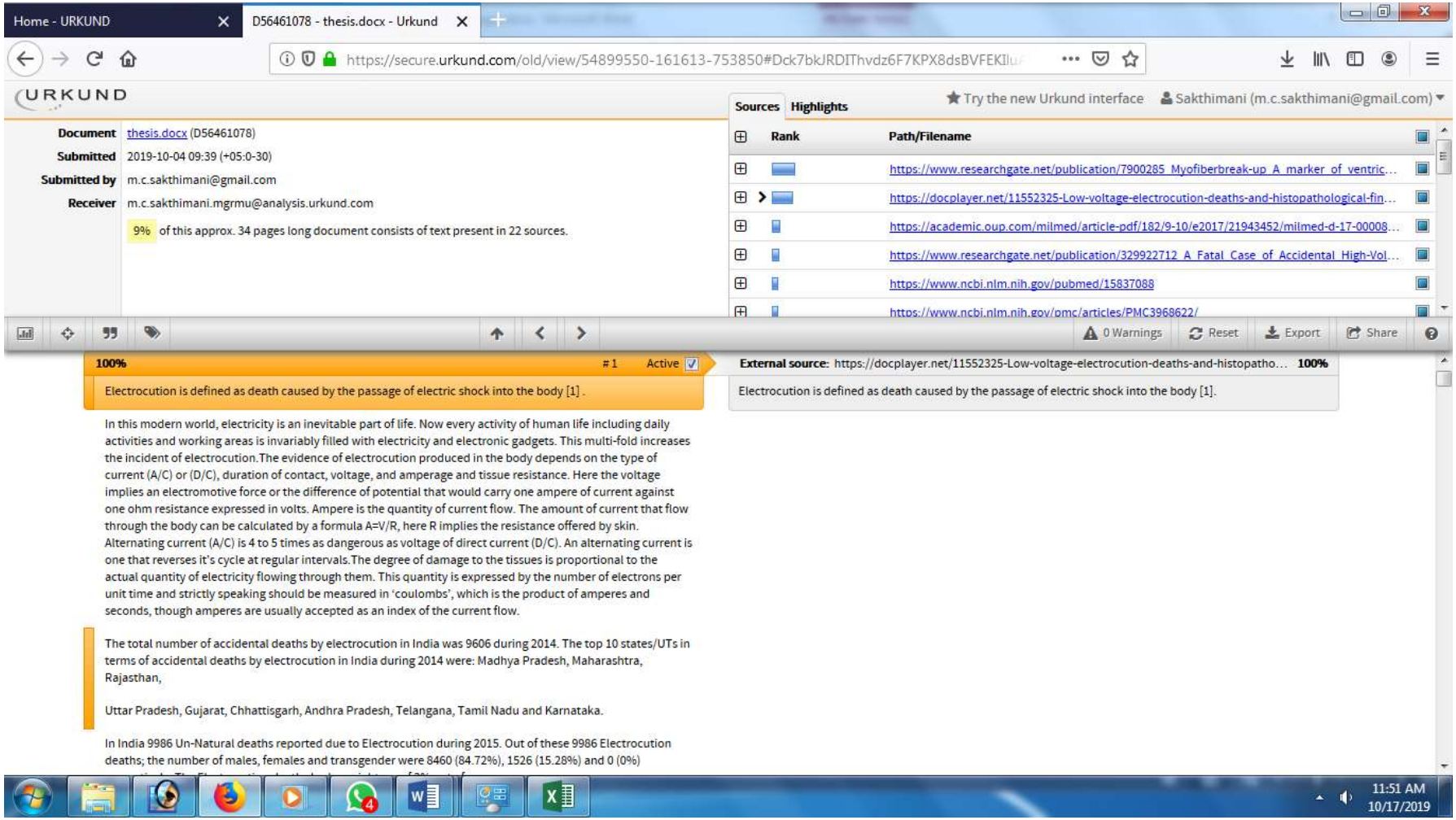
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Electrocution is defined as death caused by the passage of electric shock into the body [1] .
Electrocution is defined as death caused by the passage of electric shock into the body [1].
In this modern world, electricity is an inevitable part of life. Now every activity of human life including daily activities and working areas is invariably filled with electricity and electronic gadgets. This multi-fold increases the incident of electrocution.The evidence of electrocution produced in the body depends on the type of current (A/C) or (D/C), duration of contact, voltage, and amperage and tissue resistance. Here the voltage implies an electromotive force or the difference of potential that would carry one ampere of current against one ohm resistance expressed in volts. Ampere is the quantity of current flow. The amount of current that flow through the body can be calculated by a formula A=V/R, here R implies the resistance offered by skin. Alternating current (A/C) is 4 to 5 times as dangerous as voltage of direct current (D/C). An alternating current is one that reverses it's cycle at regular intervals.The degree of damage to the tissues is proportional to the actual quantity of electricity flowing through them. This quantity is expressed by the number of electrons per unit time and strictly speaking should be measured in 'coulombs', which is the product of amperes and seconds, though amperes are usually accepted as an index of the current flow.
The total number of accidental deaths by electrocution in India was 9606 during 2014. The top 10 states/UTs in terms of accidental deaths by electrocution in India during 2014 were: Madhya Pradesh, Maharashtra, Rajasthan,
Uttar Pradesh, Gujarat, Chhattisgarh, Andhra Pradesh, Telangana, Tamil Nadu and Karnataka.
In India 9986 Un-Natural deaths reported due to Electrocution during 2015. Out of these 9986 Electrocution deaths; the number of males, females and transgender were 8460 (84.72%), 1526 (15.28%) and 0 (0%)
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CERTIFICATE II

This is to certify that this dissertation work titled **"AN AUTOPSY BASED EXAMINATION OF HEART FINDINGS IN ELECTROCUTION AND SUDDEN CARDIAC DEATH CASES"** of the candidate **DR. M. SAKTHIMANI** with registration Number **201724203** for the award of **M.D.** in the branch of **FORENSIC MEDICINE**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **9 (Nine) percentage** of plagiarism in the dissertation.

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INTRODUCTION

INTRODUCTION

Flow of electrons through a conductor is known as electricity or current. Electrocution is defined as death caused by the passage of electric current into the body [1] .In this modern world, electricity is an inevitable part of life. Now every activity of human life including daily activities and working areas is invariably filled with electricity and electronic gadgets. This multi-fold increases the incident of electrocution. The evidence of electrocution produced in the body depends on the type of current (A/C) or (D/C), duration of contact, voltage, and amperage and tissue resistance. Here the voltage implies an electromotive force or the difference of potential that would carry one ampere of current against one ohm resistance expressed in volts. Ampere is the quantity of current flow. The amount of current that flow through the body can be calculated by a formula A=V/R, here R implies the resistance offered by skin. Alternating current (A/C) is 4 to 5 times as dangerous as voltage of direct current (D/C). An alternating current is one that reverses it's cycle at regular intervals. The degree of damage to the tissues is proportional to the actual quantity of electricity flowing through them. This quantity is expressed by the number of electrons per unit time and strictly speaking should be measured in 'coulombs', which is the product of amperes and seconds, though amperes are usually accepted as an index of the current flow.

The total number of accidental deaths by electrocution in India was 9606 during 2014. The top 10 states/UTs in terms of accidental deaths by electrocution in India during 2014 were: Madhya Pradesh, Maharashtra,

Rajasthan, Uttar Pradesh, Gujarat, Chhattisgarh, Andhra Pradesh, Telangana, Tamil Nadu and Karnataka. In India 9986 Un-Natural deaths reported due to Electrocution during 2015. Out of these 9986 Electrocution deaths; the number of males, females and transgender were 8460 (84.72%), 1526 (15.28%) and 0 (0%) respectively. The Electrocution deaths had a weightage of 3% out of the total number of Un-Natural deaths (336051) during 2015. The top 5 States/UTs in terms of number of Electrocution deaths during 2015 were: Madhya Pradesh, Maharashtra, Rajasthan, Gujarat and Uttar Pradesh. There were 651 persons died in Tamilnadu due to electrocution, out of which 549 were males, 102 were females[2].

The domestic supply of electricity in India is 220-240V. Deaths occur mostly at this voltage which is the voltage range of home electricity. It is relatively rare to be electrocuted by a voltage lower than 110 V[3]. Depending on the voltage of electric current, it is classified into Low voltage currents (less than 1000 volts) and High voltage currents (more than 1000 volts). Low voltage (household currents) is notorious in producing Ventricular fibrillation. While High voltage (Industrial currents) paralyses the respiratory center by the hyperthermic effects of the current. However it is the amperage, which is most important factor in determining the lethality of electric currents and not the voltage[4].

When the electricity flows through the body it causes potential damage to all the tissues and organs. Based on the path it may produce ventricular fibrillation or respiratory center paralysis when it flows through the heart or

brain respectively. When the voltage is high, the electricity can even throw the person several meters away or it could cause third degree thermal burns or charring of the body when the person is within the electromagnetic field. The multiple small burns are caused by arcing of the current. Dry skin offers high resistance whereas moist skin low. The electric burn mark produced during electrocution is the hallmark finding at autopsy. The identification of electrical injuries is the one of the biggest challenge in forensic field, because of the variation in its presentation. Due to decreased resistance, the electric entry and exit marks will not be prominent in electrocution happening in wet environment such as incidents at bath tub, rain, swimming pool…..etc. If the current enters over a broad surface area that offers minimal resistance, there may be no electrical burn. In such circumstances, crime scene investigation plays a major role. There are cases where this circumstantial evidence is ambiguous and remains as an artifact during investigation. Even though electric burn is a hallmark, one cannot differentiate antemortem from postmortem electrical burns. The burns indicate only that current has passed through the skin.

Electrocution is like a double edged sword, where a homicide could mimic as electrocution and vice versa which stands as an illusion in front of forensic experts.The history surrounding death is also ambiguous. A careful search for electric skin lesions in the probable areas of contact and exit will be helpful. To corroborate further, these lesions can be removed and examined under a microscope. Sub-epidermal separation, epidermal nuclear elongation, epidermal coagulation necrosis in the skin and Myofiber Breakup (MFB) in the

myocardium of the heart are the characteristic ante-mortem histopathological findings in electrocution deaths[5,6]. Death by electrocution is one of the listed causes for negative autopsy. Negative autopsy is a condition where the complete autopsy and ancillary investigations fails to demonstrate the cause of death. When the entry and exit marks as well as the circumstantial evidences are obscure, it becomes a negative autopsy. The entry and exit electrical burn mark may not be visible all the time, in those situations searching for circumstantial evidence will be helpful. This warrants the additional research in ancillary investigations where the electrocution is not evident at autopsy. To corroborate further, the suspected lesions from skin and heart can be removed and examined under a microscope. Subepidermal separation, epidermal nuclear elongation and epidermal coagulation necrosis in the skin, and Myofiber Breakup (MFB) in the myocardium of the heart are characteristic antemortem histopathological findings in electrocution deaths [5,6]. There are many studies which established the correlation between antemortem electrical burn mark with its histopathological findings, but the studies regarding histopathological findings of heart is very scant. Hence this study focuses on the histopathological findings of heart.

Ventricular fibrillation is the most common mechanism of death in electrocution. But it is not clear if, the fibrillation is due to purely electrophysiological changes or to identifiable structural abnormalities in the heart [7].

There are studies which show that Myofiber Breakup (MFB) has been significantly produced in sudden cardiac death, where the ventricular fibrillation or cardiac arrhythmias could be an attributable cause.

Sudden Cardiac death is defined as natural death due to cardiac causes in a person who may or may not have previously recognized heart disease but in whom the time and mode of death are unexpected. In the context of time, “sudden” is defined for most clinical and epidemiologic purposes as 1hr or less between a change in clinical status heralding the onset of the terminal clinical event and the cardiac arrest itself[8].

There are researches conducted on histopathology of heart to demonstrate myofiber break up as crucial evidence in determination of electrocution. But this evidence needs further delineation from the histopathology of sudden cardiac death lesions. This study is an attempt to study the cardiac histopathology of electrocution and sudden cardiac death [SCD] cases to alleviate this discrepancy, hoping this would help in reducing the rate of negative autopsies.

AIM & OBJECTIVES

AIM AND OBJECTIVES

1. Examination of Autopsy findings of heart in Electrocution and Sudden Cardiac Death cases.
2. Comparison of autopsy findings of heart in Electrocution and Sudden Cardiac Death.
3. Assessment of Electrocution and Sudden Cardiac Death related to age and gender.

STUDY DESIGN **:** Cross sectional study [Prospective study]

SAMPLE SIZE **:** 52 cases of Electrocution, 52 cases of Sudden cardiac death.

PERIOD OF STUDY **:** January 2018- June 2019

SELECTION CRITERIA

INCLUSION CRITERIA

1. Cases with history and circumstantial evidence of electrocution with electric burn mark at autopsy.
2. Sudden unexpected death with no previous history of cardiac disease and under 40 years of age.

EXCLUSION CRITERIA

1. Cases with decomposition changes.
2. Cases where the defibrillator used to revive life.
3. Cases with known history of cardiac diseases.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Cardiac anatomy:

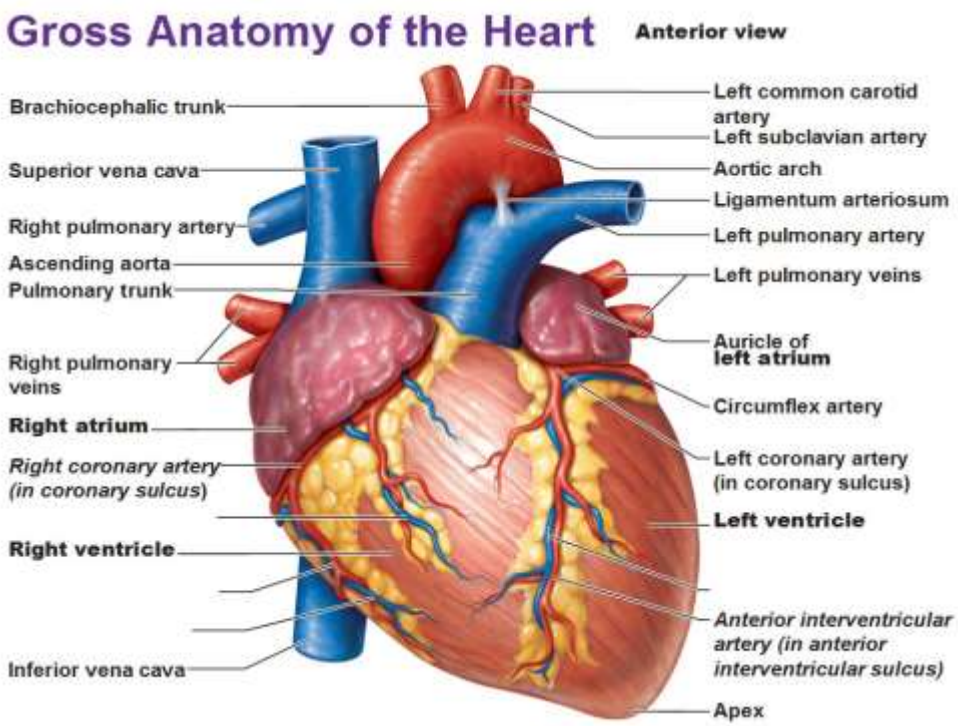


Figure 1: Shows gross anatomy of the heart

The human heart has four chambers. These are the right and left atria and the right and left ventricles. On the surface of the heart atria are separated from ventricles by an atrioventricular groove. The atria are separated from each other by aninteratrial groove. The ventricles are separated from each other by aninterventricular groove. The heart has apex, base, three surfaces and four borders.

Heart measures about 12x9cm and weight varies with body height and weight; it normally averages approximately 250 to 300 gm in females and 300 to 350 gm in males, or roughly 0.4% to 0.5% of body weight[9].

The usual thickness of the free wall of the right ventricle is 0.3 to 0.5 cm, and that of the left ventricle 1.3 to 1.5 cm, measured at the base of the papillary muscles. Increases in cardiac size and weight accompany many forms of heart disease. Greater heart weight or ventricular thickness indicates *hypertrophy*, and an enlarged chamber size implies *dilation*. An increase in cardiac weight or size or both (resulting from hypertrophy and/or dilation) is termed *cardiomegaly*[10].

Apex is formed entirely by the left ventricle. It is situated in the left fifth intercostal space 9cm lateral to the midsternal line just medial to the midclavicular line. In children below 2 years, apex is situated in the left fourth intercostal space in midclavcular line.

The base of heart is also called the posterior surface. It is formed mainly by the left atrium and a small part of right atrium.

Heart has four borders[9]:

1. The upper border is slightly oblique and formed by two atria mainly by left atrium.
2. The right border is more or less vertical and is formed by right atrium. It extends from superior vena cava to inferior vena cava.
3. The inferior border is nearly horizontal and is mainly formed by the right ventricle. A small part of it near the apex is formed by left ventricle.

4. The left border is oblique and curved. It is formed mainly by left ventricle and partly by left auricle.

Heart has three surfaces:

1. The anterior or sternocostal surface is formed mainly by the right atrium and right ventricle and partly by left ventricle and left auricle.
2. The inferior or diaphragmatic surface rests on central tendon of diaphragm. It is formed in its left two-third by left ventricle and right one-third by right ventricle.
3. The left surface is formed mainly by the left ventricle, and its upper end by left auricle.

Right atrium:

The chamber is elongated vertically, receiving the superior vena cava at the upper end and the inferior vena cava at the lower end. The upper end is prolonged to the left called the auricle, which covers the root of ascending aorta and partly the infuntibulum of right ventricle. Along the right border of atrium there is a shallow vertical groove which passes from the superior vena cava to the inferior vena cava, called the sulcus terminalis. It is produced by an internal muscular ridge called the crista terminalis. The upper part of the sulcus contains SA node or Sinoatrial node, which is the pacemaker of the heart.

A series of transverse ridges called musculipectinati arise from crista terminalis and runs downward and forwards towards the right atrioventricular orifice. The atrioventricular groove lodges the right coronary artery and small cardiac vein. The following tributaries drain into the right atrium: Superior vena cava, inferior vena cava, coronary sinus, anterior cardiac veins, venae cordisminimae (thebasian veins) and sometimes the right marginal vein.

Right ventricle:

Right ventricle is a triangular chamber which receives blood from the right atrium and pumps it to the lungs through the pulmonary trunk and pulmonary arteries. The interior shows tricuspid orifice which is guarded by tricuspid valve, pulmonary orifice which is guarded by the pulmonary valve. The inflowing part shows trabculaecorneae or muscular ridges of three types: the ridges or fixed elevations, bridges, pillars of papillary muscles. There are three papillary muscles in the right ventricle, anterior, posterior and septal. Each papillary muscles are attached by chordae tendinae to the contiguous sites of cusps. The septomarginaltrabacula or moderator band is a muscular ridge extending from the ventricular septum to the base of anterior papillar muscle, which contains the right branch of AV bundle.

Left atrium:

The left atrium is a quadrangular chamber situated posteriorly. Its appendage, the left auricle projects anteriorly to overlap the infundibulum of the right ventricle. It

receives oxygenated blood from the lungs through four pulmonary veins, and pumps it to the left ventricle through the left atrioventricular or bicuspid or mitral orifice which is guarded by bicuspid or mitral valve. The interior of the atrium is smooth walled. Musculipectinati are present only in the auricle where they form a reticulum. Apart from four pulmonary veins, few venae cordisminimae drain into the left atrium.

Left ventricle:

The left ventricle receives oxygenated blood from the left atrium and pumps it to the aorta. The interior of the left ventricle shows lower rough part with trabeculaecorneae, upper smooth part or aortic vestibule gives origin to the ascending aorta. It also two orifice, mitral orifice, aortic orifice and two papillary muscle, anterior and posterior. The walls of left ventricle is three times thicker than the right ventricle.

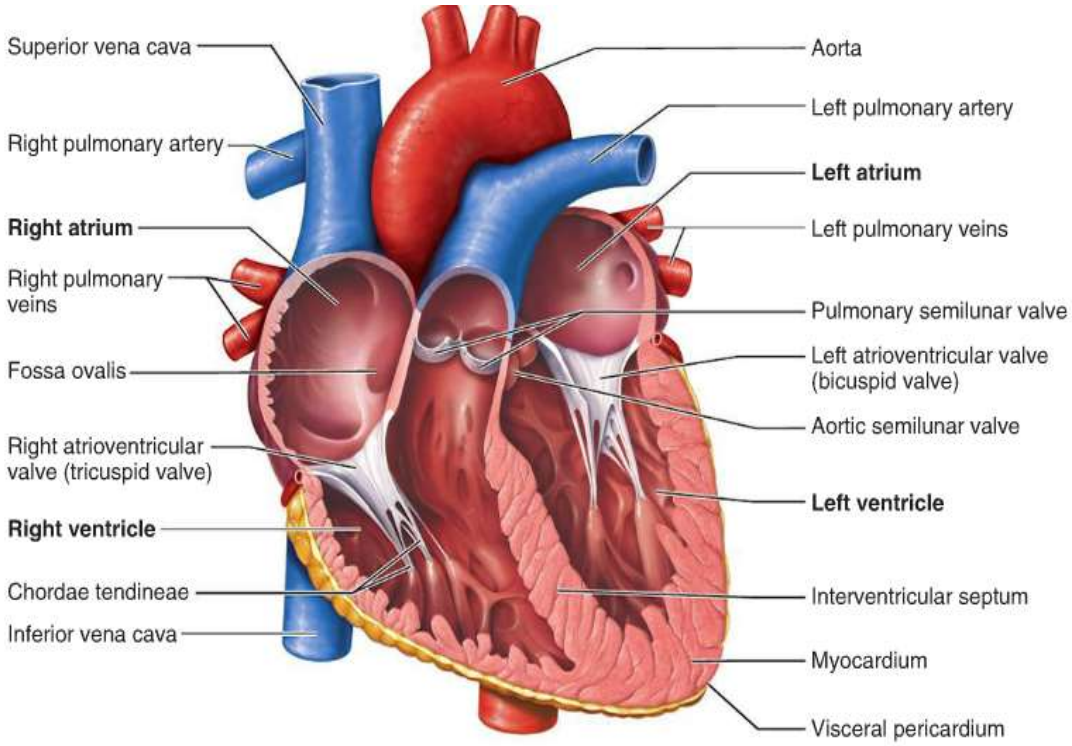


Figure 2: Shows valves of the heart

Valves of the heart:

There are two pairs of valves in the heart, a pair of atrioventricular valves and a pair of semilunar valves. The right atrioventricular valve is known as the tricuspid valve because it has three cusps. The left atrioventricular valve is known as the bicuspid valve/mitral valve because it has two cusps. The semilunar valves include aortic and pulmonary valves each has three cusps. The cusps are folds of endocardium, strengthened by an intervening layer of fibrous tissue.

Atrioventricular valves:

Both valves are made up of fibrous ring, cusps, chordae tendinae. A fibrous ring through which cusps are attached. Each cusp has an attached and a free margin, and an atrial and ventricular surface. The atrial surface is smooth, the free margin and ventricular surface is rough and irregular due to the attachment of chordae tendinae. Chordae tendinae connects the ventricular surface of free margin to the apices of papillary muscles, which pull the chordae tendinae during ventricular systole. The tricuspid valve has three cusps, the anterior, posterior or inferior and septal. It can admits tips of three fingers. The mitral or bicuspid valve has two cusps, a large anterior or aortic cusp and a small posterior cusp. The mitral cusps are smaller and thicker than those of the tricuspid valve.

Semilunar valves:

The aortic and pulmonary valves are called semilunar valves because their cusps of semilunar in shape. Each valves has three cusps which are attached directly to the vessel wall, there being no fibrous ring. The free margin of each cusp contains a central fibrous nodule from each side of which a thin smooth margin the lunule extends upto the base of cusp. Opposite the cusps the vessel walls are slightly dilated to form the aortic and pulmonary sinuses. The coronary arteries arise from the anterior and the left posterior aortic sinuses.

Blood supply of Heart:

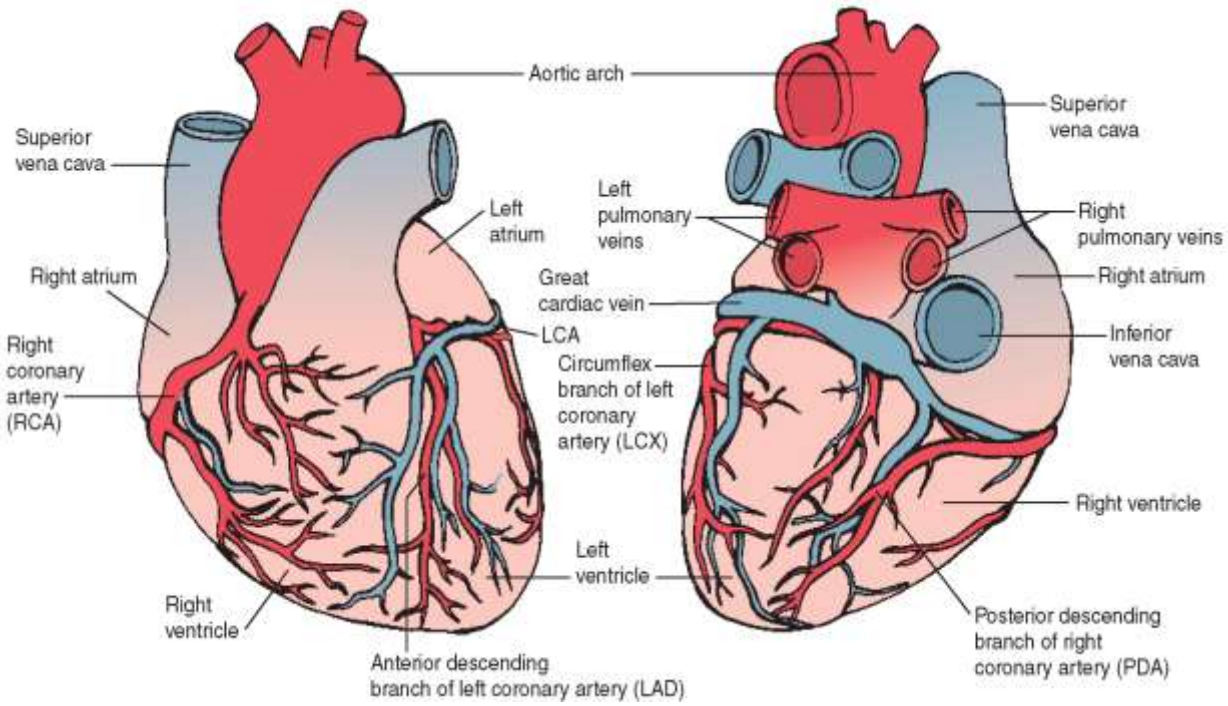


Figure 3: Shows blood supply of the heart

The Coronary arteries:

The heart is supplied by two coronary arteries, arising from ascending aorta. The blood flows through these arteries during diastole. Diameter is 1.5-5.2mm, the left coronary is larger in caliber and supplies more myocardium. These arteries are functional end arteries, though their branches anastomose, they cannot compensate for the other during thrombosis. The origin of posterior interventricular artery determines the cardiac dominance. Sympathetic stimulation dilates the intermuscular arteries and constricts the epicardial arteries.

Right coronary artery:

It arise from the anterior aortic sinus of ascending aorta, and passes between the root of pulmonary trunk and right auricle, then runs downwards in the right anterior coronary sulcus and winds around the inferior border and reach the diaphragmatic surface of the heart. Here it runs backwards and to the left in the right posterior coronary sulcus to reach the posterior interventricular groove and terminates by anastomosing with the circumflex branch of left coronary artery at the crux. The atrial branches are anterior, posterior and lateral, one of them is SA nodal artery in 60% of cases. Right conus artery forms an arterial circle called "annulus of vieussens" around pulmonary trunk with a similar branch from the left coronary artery. Right marginal artery arises as the right coronary artery crosses the right border of heart and runs along the inferior border till the apex. The ventricular branches are as anterior and posterior group, the anterior group lies on the sternocostal surface and posterior traverses the diaphragmatic surface. The posterior interventricular artery arises close to the crux of the heart and lies in the posterior interventricular groove. It gives septal branches to the posterior 1/3rd of interventricular septum and also supplies AV node. Right coronary artery supplies, right atrium, greater part of right ventricle except the area adjoining anterior interventricular groove and supplies smaller part of left ventricle adjoining the posterior interventricular groove, posterior 1/3rd part of interventricular septum, whole of the conducting system except left branch of AV bundle. SA node is supplied by left coronary artery in 40% case.

Left coronary artery:

Left coronary artery arise from the posterior aortic sinus of ascending aorta, it emerges between the pulmonary trunk and left auricle, here it gives anterior intervenricular branch which descends in the anterior interventricular groove and a circumflex branch which runs in the left anterior coronary sulcus, then curves around the left border of heart to lie in the left posterior coronary sulcus and ends by anastomosing with terminal part of right coronary artery at the crux of the heart.

Branches of anterior interventricular branch:

i. Anterior ventricular branches, the larger branch is called "left diagonal artery".
ii. Septal branches which supply the anterior 2/3rd of theinterventricular septum.
iii. Left conus artery forms the arterial ring around the pulmonary trunk along with the similar branch of right coronary artery.

Branches of circumflex branch:

i. Left marginal artery which lies along the left border of the heart till the apex of the heart.
ii. Anterior and posterior ventricular branches.
iii. Atrial branches (the anterior, posterior and lateral groups).

Area of distribution:

Left coronary artery supplies the left atrium, greater part of the left ventricle, except the area adjoining the posterior interventricular groove and supplies the smaller part adjoining the anterior interventricular groove, anterior part of interventricular septum and part of left branch of AV bundle.

In about 10% of hearts, the left coronary artery gives origin to the posterior interventricular artery where the right coronary artery is small.

Venous drainage:

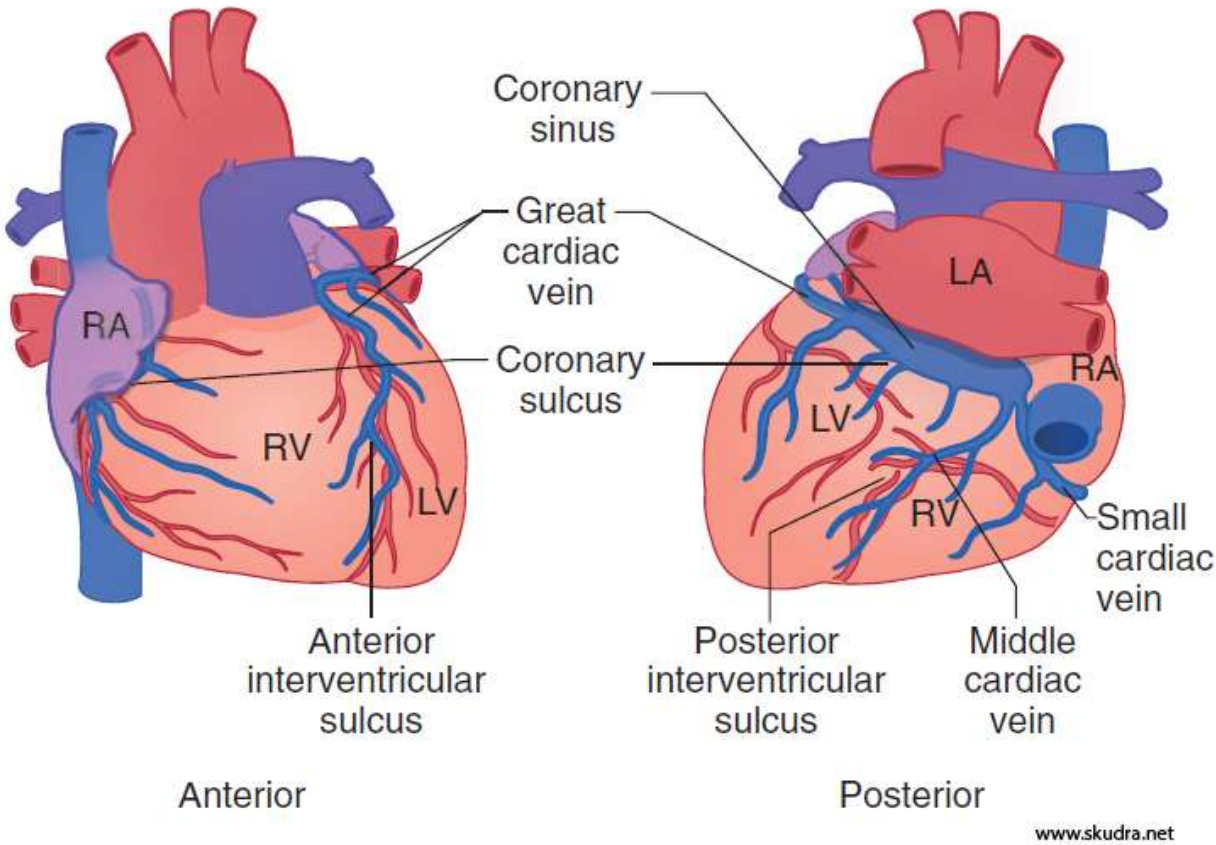


Figure 4: Shows gross venous drainage of the heart

Coronary sinus:

Coronary sinus is the largest vein of the heart. It is situated in the posterior coronary sulcus. It is about 3cm long , ends by opening into the posterior wall of right atrium.

Tributaries:

1. The great cardiac vein, first accompanies the anterior interventricular artery and passes along with left coronary artery and ends in the left end of coronary sinus.
2. The middle cardiac vein accompanies the posterior interventricular artery, and joins the middle part of coronary sinus.
3. The small cardiac vein accompanies the right coronary artery in the right posterior coronary sulcus and joins the right end of coronary sinus.
4. The posterior vein of the left ventricle runs on the diaphragmatic surface of left ventricle and ends in coronary sinus.
5. The oblique vein of left atrium of marshall is develops from the left common cardinal vein or duct of cuvier which may sometimes form a large left superior vena cava. It is a small vein running on the posterior surface of left atrium and terminates in the left end of coronary sinus.
6. The marginal vein accompanies the marginal branch of right coronary artery and may drain into the small cardiac vein or directly into the right atrium.

Anterior cardiac veins: They are three or four small veins which run parallel to one another on the anterior wall of right ventricle and drains directly into the right atrium.

Venae cordis minimae or thebesian veins or smallest cardiac veins: numerous small valveless veins present in all four chambers, which open directly into the cavity.

Lymphatics of heart:

Lymphatics accompanies the coronary arteries and forms right and left trunk, right trunk drains into the brachiocephalic nodes and left trunk drains into the tracheobronchial nodes.

Nerve supply of the heart:

Parasympathetic nerves reach the heart via the vagus and are cardio-inhibitory in nature i.e on stimulation decreases heart rate. Sympathetic nerves are derived from the upper four to five thoracic segments and are cardio-acceleratory i.e on stimulation increases heart rate. Both these branches form the superficial and deep cardiac plexuses.

Superficial cardiac plexus:

It is situated below the arch of aorta in front of right pulmonary artery. It is formed by the superior cervical cardiac branch of left sympathetic chain and inferior cervical cardiac branch of left vagus nerve. This plexus is

connected to the deep cardiac plexus, the right coronary artery and to the left anterior pulmonary plexus.

Deep cardiac plexus:

It is situated in front of the bifurcation of trachea and behind the arch of aorta. It is formed by all the cardiac branches derived from all the cervical and upper thoracic ganglia of the sympathetic chain and the cardiac branches of vagus and recurrent laryngeal nerves, except which forms the superficial plexus.

The right and left halves of the plexus distribute branches to the corresponding coronary and pulmonary plexuses. Separate branches are given to atrium.

Fibrous skeleton of the heart:

The fibrous rings surrounding the atrioventricular and arterial orifices, along with some adjoining masses of fibrous tissue constitutes fibrous skeleton. The atrioventricular fibrous rings are in the form of figure of 8. The atria, ventricles and membraneous part of interventricular septum attached to them. There is no muscular continuity between the atria and ventricles except for the bundle of His. Fibrous tissue between the atrioventricular rings behind and the aortic ring in front called trigonumfibrosumdextrum. Another fibrous tissue between the aortic and mitral rings is called trigonumfibrosumsinistrum. The tendon of infundibulum (close to pulmonary valve) binds the posterior surface of infundibulum to the aortic ring.

Musculature of heart:

Cardiac muscle fibers form the long loops which are attached to the fibrous skeleton. The atrial fibers are arranged in superficial transverse and deep vertical layer.

The ventricular fibers are arranged in superficial and deep layers.

The superficial layer:

i. Fibers start from tendon of infundibulum pass across the diaphragmatic surface, curve around inferior border to reach the sternocostal surface. Then these fibers cross the anterior interventricular groove to reach the apex, where these form a vortex and end in anterior papillary muscle of left ventricle.
ii. Fibers arise from right AV ring take same course as (2) but end in posterior papillary muscle.
iii. Fibers arise from left AV ring lie along the diaphragmatic surface, cross the posterior interventricular groove to reach the papillary muscles of right ventricle.

Deep fibers:

They are ‘S’ shaped and arise from papillary muscle of one ventricle and end in papillary muscle of other ventricle. Fibers of first layer circle right ventricle, cross through the interventricular groove, to end in papillary muscle

of left ventricle. Layers two and three have decreasing course in right ventricle and increasing course in left ventricle.

Cardiac histology[11] :

The cardiac muscle fibers exhibit cross-striations, branching, and a single central nucleus. The dark-staining intercalated disks connect individual cardiac muscle fibers. Small myofibrils are visible within each cardiac muscle fiber. Delicate strands of connective tissue fibers surround the individual cardiac muscle fibers. Atrial myocytes are generally smaller and arranged more haphazardly than their ventricular myocytes. Some atrial cells have distinctive electron-dense granules in the cytoplasm called *specific atrial granules;* these are the storage sites of *atrial natriuretic peptide*. The cardiac muscle has a vast blood supply. Numerous small blood vessels and capillaries are found in the connective tissue septa and the delicate endomysiumbetween individual muscle fibers.

Valves are lined by endothelial cells on a thin layer of collagen and elastic tissue on the atrial/arterial side, a thicker layer of dense collagen on the ventricular side, and loose myxoid connective tissue (zonaspongiosa) in between.

The conduction system is composed of specialized myocytes, with fewer intercalated disks and higher glycogen content. Masson trichrome, Verhoeff–van Giesen, and Alcian blue stains can be used to demonstrate the conduction system.

Cardiac ultrastructure[8]:

Functional integration of cardiac myocytes is mediated by structures called *intercalated discs*, which link individual cells and contain specialized intercellular junctions that permit both mechanical and electrical (ionic) coupling. Within the intercalated discs, *gap junctions* facilitate synchronous myocyte contraction through electrical coupling by permitting relatively unrestricted passage of ions across the membranes of adjoining cells. They provide a strong union between fibers, maintaining cell-to-cell cohesion, so that the pull of one contractile cell can be transmitted along its axis to the next. Along the sides of the muscle fibers next to the disks, the cell membranes of adjacent fibers fuse for considerable distances, forming gap junctions. These junctions provide low-resistance bridges for the spread of excitation from one fiber to another. They permit cardiac muscle to function as if it were a syncytium, even though no protoplasmic bridges are present between cells.

About three-fourths of the ventricular mass is composed of cardiomyocytes, normally 60-140µm in length and 17-25µm in diameter. Each cell contains multiple, rodlike cross-banded strands (myofibrils) that run the length of the cell and are composed of serially repeating structures, the sarcomeres. The cytoplasm between the myofibrils contains other cell constituents, including the single centrally located nucleus, numerous mitochondria, and the intracellular membrane system, the sarcoplasmic reticulum.

The sarcomere, the structural and functional unit of contraction, lies between adjacent Z lines, which are dark repeating bands that are apparent on transmission electron microscopy. The distance between Z lines varies with the degree of contraction or stretch of the muscle and ranges between 1.6 and 2.2µm. Within the confines of the sarcomere are alternating light and dark bands, giving the myocardial fibers their serrated appearance under the light microscope. At the center of the sarcomere is a dark band of constant length (1.5µm), the A band, which is flanked by two lighter bands, the I bands, which are of variable length. The sarcomere of heart muscle is like that of skeletal muscle, consists of two sets of interdigitating myofilaments. Thicker filaments, composed principally of the protein myosin, traverse the A band; they are about 10nm (100Å) in diameter, with tapered ends. Thinner filaments, composed primarily of actin, course from Z lines through the I bandinto the A band; they are approximately 5nm (50Å) in diameter and 1.0µm in length. Thus, thick and thin filaments overlap only within the (dark) A band, whereas the (light) I band contains only thin filaments.

The contractile process:

The sliding filament model for muscle contraction rests on the fundamental observation that both the thick and the thin filaments are constant in overall length during both contraction and relaxation. With activation, the actin filaments are propelled further into the A band. In the process, the A band remains constant in length, whereas the I band shortens and the Z lines move toward one another.

The myosin molecule is a complex, asymmetric fibrous protein with a molecular mass of about 500,000 Da; it has a rod like portion that is about150nm (1500Å) in length with a globular portion (head) at its end. These globular portions of myosin form the bridges between the myosin and actin molecules and are the site of ATPase activity. Actin has a molecular mass of about 47,000 Da. The thin filament consists of a double helix of two chains of actin molecule wound about each other on a larger molecule, tropomyosin. A group of regulatory proteins-troponins C, I, and T –are spaced at regular intervals on this filament. In contrast to myosin, actin lacks intrinsic enzymatic activity but does combine reversibly with myosin in the presence of ATP and Ca^{2+}. The calcium ion activates the myosin ATPase, which in turn breaks down ATP, the energy source for contraction. The activity of myosin ATPase determines the rate of forming and breaking of the actomyosin cross-bridges and ultimately the velocity of muscle contraction. In relaxed muscle, tropomyosin inhibits this interaction. Titin is a large, flexible, myofibrillar protein that connects myosin to the Z line; its stretching contributes to the elasticity of the heart. Dystrophin is a long cytoskeletal protein that has an amino-terminal actin-binding domain and a carboxy–terminal domain that bindsto the dystroglycan complex at adherens junctions on the cell membrane, thus tethering the sarcomere to the cell membrane at regions tightly coupled to adjacent contracting myocytes.

During activation of cardiac myocyte, Ca^{2+} becomes attached to one of the three components of the heterotrimer troponin C, which results in a

conformational change in the regulatory protein tropomyosin; the latter, in turn, exposes the actin cross bridge interaction sites. Repetitive interaction between myosin heads and actin filaments is termed cross-bridge cycling, which results in sliding of the actin along the myosin filaments, ultimately causing muscle shortening and/or the development of tension. The splitting of ATP then dissociates the myosin cross bridge from actin. In the presence of ATP, linkages between actin and myosin filaments are made and broken cyclically as long as sufficient Ca^{2+} is present; these linkages cease when [Ca^{2+}] falls below a critical level, and the troponin-tropomyosin complex once more prevents interaction between the myosin cross-bridges and actin filaments.Intracytoplasmic Ca^{2+} is a principal determinant of the ionotropic state of the heart.

Increased impulse traffic in the cardiac adrenergic nerves stimulates myocardial contractility as a consequence of the release of norepinephrine from cardiac adrenergic nerve endings. Norepinephrine activates myocardial β receptors and, through the G_s –stimulated guanine nucleotide-binding protein, activates the enzyme adenylyl cyclase, which leads to the formation of the intracellular second messenger cyclic AMP from ATP. Cyclic AMP in turn activates protein kinase A (PKA), which phosphorylates the Ca^{2+} channel in the myocardial sarcolemma, thereby enhancing the influx of Ca^{2+} into the myocyte.

Cardiac Activation:

In the inactive state, the cardiac cell is electrically polarized; i.e., the interior has a negative charge relative to the outside of the cell, with a transmembrane potential of -80 to -100 mV. The sarcolemma, which in the resting state is largely impermeable to Na^+, has a Na^+ and K^+ -stimulating pump energized by ATP that extrudes Na^+ from the cell; this pump plays a critical role in establishing the resting potential. Thus in inactive state, the extracellular Na^+ concentration is high compared to intracellular level. At the same time, in the resting state, extracellular [Ca^{2+}] greatly exceeds free intracellular [Ca^{2+}]. The action potential has four phases. During the plateau of the action potential (phase 2), there is a slow inward current through L-type Ca^{2+} channels in the sarcolemma. The absolute quantity of Ca^{2+} that crosses the sarcolemma and the T system is relatively small and by itself appears to be insufficient to bring about full activation of the contractile apparatus. However, this Ca^{2+} current triggers much larger quantities of Ca^{2+}, from the sarcoplasmic reticulum, a process termed Ca^{2+} induced Ca^{2+} release. Ca^{2+} is released from the sarcoplasmic reticulum through a Ca^{2+} release channel, a cardiac isoform of the Ryanodine receptor (RyR2), which controls intracytoplasmic [Ca^{2+}] and, as in vascular smooth muscle cells, leads to the local changes in intracellular [Ca^{2+}] called calcium sparks. A number of regulatory proteins, including calstabin-2, inhibit RyR2 and thereby the release of Ca^{2+} from the sarcoplasmic reticulum. PKA dissociates calstabin from the RyR2, enhancing Ca^{2+} release and thereby myocardial contractility.

Excessive plasma catecholamine levels and cardiac sympathetic neuronal release of norepinephrine cause hyperphosphorylationof PKA, leading to calstabin-2 depleted RyR2. The latter depletes sarcoplasmic reticulum Ca^{2+} store and thereby impairs cardiac contraction leading to heart failure, and also triggers ventricular arrhythmias.

The Ca^{2+} released from sarcoplasmic reticulum then diffuses toward the myofibrils, where it combines with troponin C, by repressing this inhibitor of contraction, Ca^{2+} activates the myofilaments to shorten. During repolarisation, the activity of Ca^{2+} pump in the sarcoplasmic reticulum, the sarcoplasmic reticulum Ca^{2+} ATPase ($SERCA_{2A}$), reaccumulates Ca^{2+} against the concentration gradient, and the Ca^{2+} is stored in sarcoplasmic reticulum by its attachment to protein, calsequestrin. This reaccumulation of Ca^{2+} is an energy (ATP) requiring process that lowers the cytoplasmic [Ca^{2+}] to a level that inhibits the actomyosin interaction responsible for contraction, and in this manner leads to myocardial relaxation. Also, there is an exchange of $Ca^{2+}$for Na^{+}at the sarcolemma, Reducing the cytoplasmic [Ca^{2+}]. Cyclic AMP dependent PKA phosphorylates the sarcoplasmic reticulum protein phospholamban; the latter, in turn, permits activation of Ca^{2+} pump, thereby increasing the uptake of Ca^{2+} by the sarcoplasmic reticulum, accelerating the rate of relaxation and providing larger quantities of Ca^{2+} in the sarcoplasmic reticulum for release by subsequent depolarization, thereby stimulating contraction.

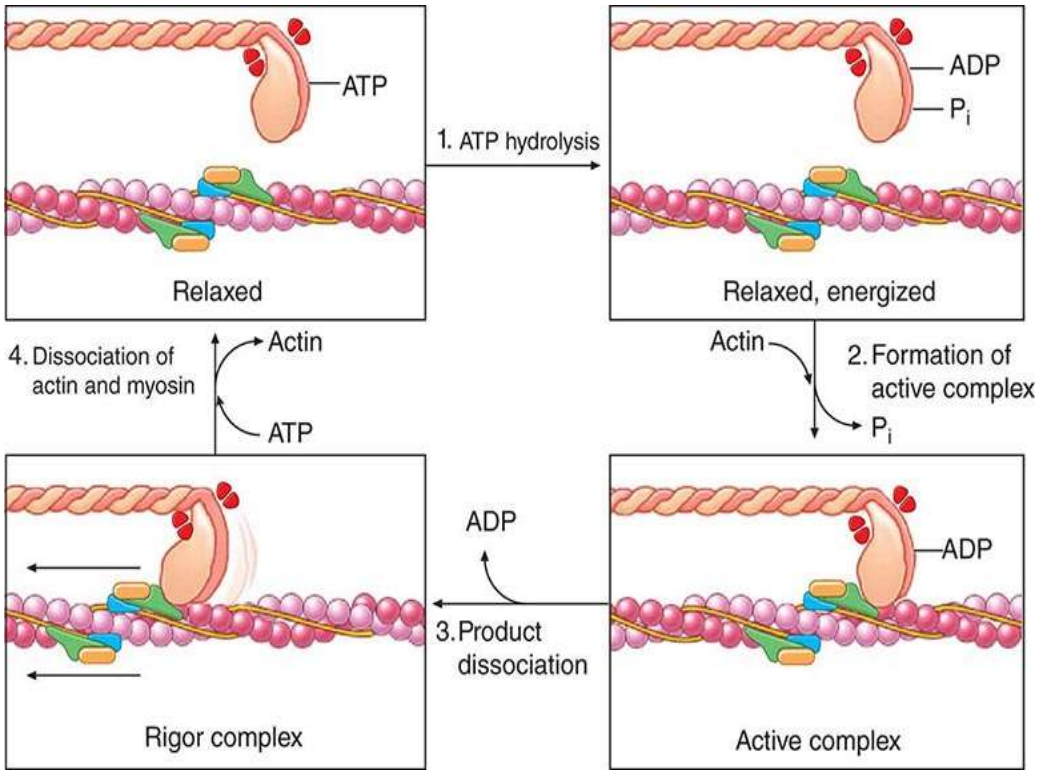


Figure 5: Shows contractile process of the heart muscle.

Conduction system of heart and electrophysiology:

Myocardial fibers have a resting membrane potential of approximately –90 mV. Stimulation produces a propagated action potential that is responsible for initiating contraction. Depolarization proceeds rapidly and an overshoot of the zero potential which is followed by a plateau before the membrane potential returns to the baseline. In mammalian hearts, depolarization lasts about 2 ms, but the plateau phase and repolarization last 200 ms or more. Repolarization is therefore not complete until the contraction is half over. The initial rapid depolarization and the overshoot (phase 0) are due to opening of voltage-gated Na^+ channels ,and the initial rapid repolarization (phase 1) is due to closure of Na^+ channels and opening of one type of K^+ channel. The subsequent prolonged plateau (phase 2) is due to a slower but prolonged opening of voltage-gated Ca^{2+}

channels. Final repolarization (phase 3) to the resting membrane potential (phase 4) is due to closure of the Ca^{2+} channels and a slow, delayed increase of K^+ efflux through various types of K^+ channels. Cardiac myocytes contain at least two types of Ca^{2+} channels (T- and L-types), but the Ca^{2+} current is due mostly to opening of the slower L-type Ca^{2+} channels.

Cardiac Conduction system:

The fibers of the conduction system are finer than the myocardial fibers, and are completely cross-striated. It has following parts[9]:

Sinuatrial node or SA node: It is horseshoe shaped and is situated at the atriocaval junction in the upper part of sulcus terminalis. It is known as the 'pacemaker' of the heart and generates impulses at a rate of 70-100 beats/min.

Atriventricular node or AV node: It is smaller than the SA node and situated at the lower and dorsal part of the atrial septum just above the opening of coronary sinus. It generates impulse at arate of 40-60 beats/min.

Atriovenricular bundle or AV bundle or bundle of His: It is the only muscular connection between the atrial and ventricular musculature. It begins as the AV node crosses the AV ring and descends along the posteroinferior border of the membraneous part of ventricular septum. At the upper border of the muscular part of the septum, it divides into right and left branches.

Right branch of AV bundle: it passes down along the right side of interventricular septum, a large part enters the moderator band to reach the anterior wall where it divides into purkinje fibers.

Left branch of AV bundle: it passes along the left side of interventricular septum and is distributed to left ventricle after dividing into purkinje fibers.

Purkinje fibers: They are large pale fibers striated only at their margins and usually possess double nuclei. They form a subendocardial plexus, and generate impulses at a rate of 20-35 beats/min.

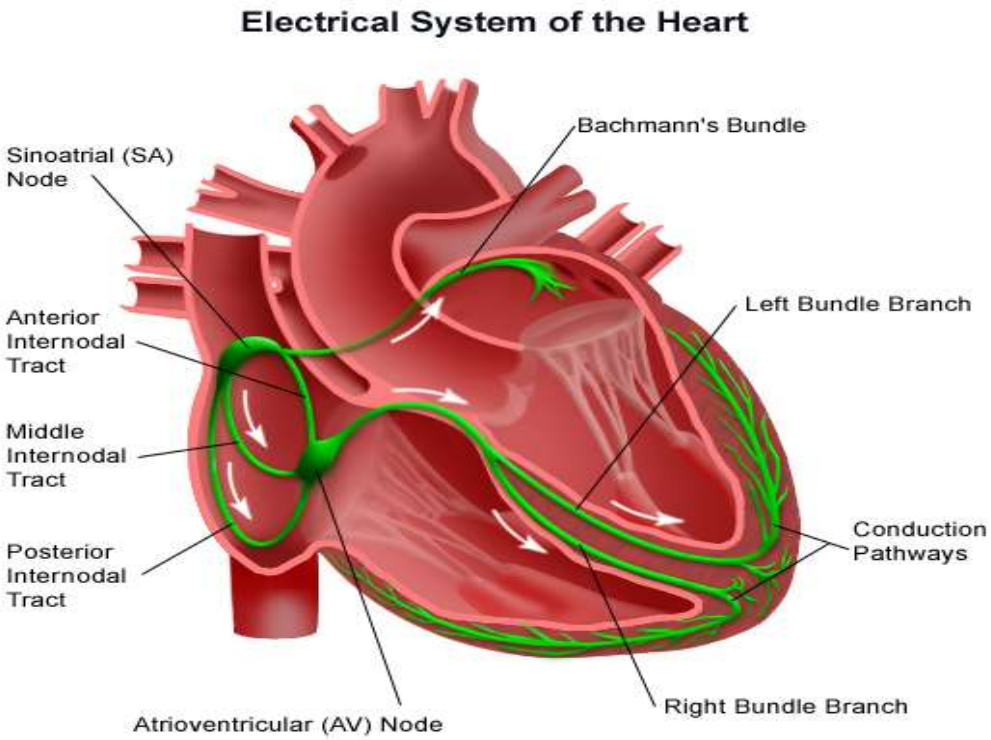


Figure 6: Shows conduction system of the heart

The physiology of conduction[12]:

The parts of the heart normally beat in orderly sequence: Contraction of the atria (atrial systole) is followed by contraction of the ventricles (ventricular systole), and during diastole all four chambers are relaxed. The heartbeat originates in a specialized

cardiac conduction system and spreads via this system to all parts of the myocardium. The structures that make up the conduction system are the sinuatrial node (SA node), the internodal atrial pathways, the atrioventricular node (AV node), the bundle of His and its branches, and the Purkinje system. The various parts of the conduction system and, under abnormal conditions, parts of the myocardium, are capable of spontaneous discharges. The SA node is the normal cardiac pacemaker, with its rate of discharge determining the rate at which the heart beats.

In the human heart, the SA node is located at the junction of the superior vena cava with the right atrium. The AV node is located in the right posterior portion of the interatrial septum. There are three bundles of atrial fibers that contain Purkinje-type fibers and connect the SA node to the AV node: the anterior internodal tract of Bachman, the middle internodal tract of Wenckebach, and the posterior internodal tract of Thorel. Conduction also occurs through atrial myocytes, but it is more rapid in these bundles. The AV node is continuous with the bundle of His, which gives off a left bundle branch at the top of the interventricular septum and continues as the right bundle branch. The left bundle branch divides into an anterior fascicle and a posterior

fascicle. The branches and fascicles run subendocardially down either side of the septum and come into contact with the Purkinje system, whose fibers spread to all parts of the ventricular myocardium.

Depolarization initiated in the SA node spreads radially through the atria, then converges on the AV node. Atrial depolarization is complete in about 0.1 s. Because conduction in the AV node is slow , a delay of about 0.1 s (AV nodal delay) occurs before excitation spreads to the ventricles. It is interesting to note here that when there is a lack of contribution of I_{Na} in the depolarization (phase 0) of the action potential, a marked loss of conduction is observed. This delay is shortened by stimulation of the sympathetic nerves to the heart and lengthened by stimulation of the vagus. From the top of the septum, the wave of depolarization spreads in the rapidly conducting Purkinje fibers to all parts of the ventricles in 0.08–0.1 s. In humans, depolarization of the ventricular muscle starts at the left side of the interventricular septum and moves to the right across the mid portion of the septum. The wave of depolarization then spreads down the septum to the apex of the heart. It returns along the ventricular walls to the AV groove, proceeding from the endocardial to the epicardial surface (Figure 30–4). The last parts of the heart to be depolarized are the postero-basal portion of the left ventricle, the pulmonary conus, and the uppermost portion of the septum.

Mechanism of cardiac arrhythmias[13]:

Cardiac arrhythmias result from abnormalities of electrical impulse generation, conduction, or both. Bradyarrhythmias typically arise from disturbances in impulse formation at the level of the sinoatrial node or from disturbances in impulse propagation at any level, including exit block from the sinus node, conduction block in the AV node, and impaired conduction in the His-Purkinje system. Tachyarrhythmias can be classified according to mechanism, including enhanced automaticity (spontaneous depolarization of atrial, junctional, or ventricular pacemakers), reentry (circus propagation of a depolarizing wavefront), or triggered arrhythmias (initiated by afterdepolarizations) occurring during or immediately after cardiac repolarization, during phase 3 or 4 of the action potential.

Abnormal automaticity:

Automaticity refers to the ability of the cardiac tissue to spontaneously generate pacemaker activity. Abnormal automaticity refers to tissues that under normal circumstances do not generate automaticity, but can become automatic in the setting of ischemia, metabolic disturbance or pharmacologic manipulation. Overall, abnormal automaticity responsible for <10% tachyarrhythmias. These latent or ectopic loci cells generate automatic, spontaneous impulses that usurb control of cardiac rhythm. These usually have a warm up and cool down period and cannot be induced by programmed electrical stimulation.

Triggered activity : it refers to pacemaker activity that is dependant on afterdepolarizations from a prior impulse or series of impulses. Afterdepolarizations are ossilations in the membrane potential. If these reaches critical threshold for depolarization of the surrounding cardiac tissue, they may trigger an action potential, thereby precipitating further depolarizations and perpetuating the pacemaker activity.

The two categories of afterdepolarizations are early and delayed.

Early afterdepolarisations [EAD] occurs before repolarisation of cardiac tissue is completed {during the phase 3 of action potential} and maybe the mechanism responsible for the ventricular arrhythmias of the Long QT syndromes (LQTSs), as well as torsades de pointes ("twisting of points") produced by class I and class II antiarrhythmetics, sympathetic discharge and hypoxia.

Delayed afterdepolarizations (DAD) occur after the repolarisation of surrounding tissue is complete (during phase 4 of action potential) and are thought to be the mechanism of triggered atrial tachycardia, arrhythmias of digitalis toxicity, and rare ventricular tachycardias responsive to calcium channel blockers.

Re-entry: Re- entry is the most common mechanism of tachy-arrhythmias. In order for re-entry to occur, three conditions must be met:

1. Two functionally distinct conducting pathways must connect to form a circuit.

2. Unidirectional conduction block occurs in one of the pathways due to difference in refractory periods. (block occurs in a pathway with longer refractory period)
3. Slow conduction occurs down the unblocked pathway (which has the shorter refractory period), allowing the blocked pathway time to recover excitability and sustain arrhythmia.

Reentrant circuits can occur in the SA node, the atrium, the AV node, between the atrium and ventricle via bypass tract, and within the ventricles itself.

The typical substrate for malignant reentry in ventricle is scar or ischemia, which can produce regions in the heart that depolarize and repolarize heterogenously. Therefore, the impulse can spread to an area that has already repolarised after being previously depolarised. This can set up a circular movement of the impulse resulting in sustained tachyarrhythmias such as ventricular tachycardia. Reentry can be typically induced by premature electrical stimulation during electrophysiologic testing.

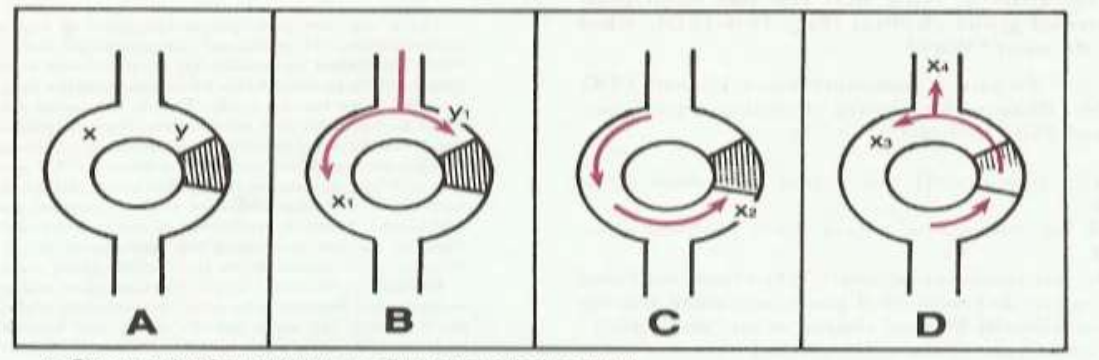


Figure 16B-19A. Theoretical illustration of a reentry circuit. The three necessary components are: (1) two separate pathways (labeled **x** and **y**); (2) unidirectional block in one of these pathways (represented by the lined area in pathway **y**); and (3) slow conduction along the other pathway.

Figure 7: Shows mechanism of re-entry in ventricular fibrillation.

Ventricular fibrillation:

Ventricular fibrillation is the chaotic rhythm that reflects no organised electrical activity and hence no cardiac output from the ventricle. It is devoid of distinct elements that makeup the usual electrical complex of ventricular activity. It is a rapid fatal rhythm, and if resuscitation is not begun within 5-7minutes death is usually certain. Ventricular fibrillation often preceeded by ventricular tachycardia. It may arise without any inciting cardiac rhythm or event. In ventricular fibrillation, the ventricular muscle fibers contract in a totally irregular and ineffective way because of the very rapid discharge of multiple ventricular ectopic foci or a circus movement. The fibrillating ventricles, like the fibrillating atria, look like a quivering "bag of worms." Ventricular fibrillation can be produced by an electric shock or an extrasystole during a critical interval, the vulnerable period**.** The vulnerable period coincides in time with the midportion of the T wave; that is, it occurs at a time when some of the ventricular myocardium is depolarized, some is incompletely repolarized, and some is completely repolarized. These are excellent conditions in which to establish reentry and a circus movement. The fibrillating ventricles cannot pump blood effectively, and circulation of the blood stops. The most frequent cause of sudden death in patients with myocardial infarcts is ventricular fibrillation.

Electricity[1]:

Current:

It is generally considered that the passage of 50–80mA across the heart for more than a few seconds is likely to cause death. The most that can be tolerated voluntarily by most people is 30mA applied to the hand, which results in painful muscle contractions. Consciousness is likely to be lost at about 40mA and, as stated, currents sustained for some seconds at over 50–80mA carry a substantial risk of death.

Voltage :

Most fatalities occur with the domestic voltage of 240. It is uncommon to encounter deaths at less than 100V, mainly because there are few sources of supply between 110V and the 12V or 24 V used in vehicle electrical systems. Extremely high voltages, such as those encountered in power transmission systems and in electronic equipment, may paradoxically be safer on some occasions, as the shock may physically fling the subject off the conductor, thus reducing the contact time below the threshold for cardiac damage.

Resistance:

The major barrier to an electrical current is the skin, which has higher resistance than internal tissues. That is why skin electric burns occur, as the resistivity causes energy transfer from the electron flow to the skin. Once inside the dermis, the semi-fluid cytoplasm, and especially the vascular system filled with electrolyte-rich fluid, passes the current through the body quite easily. The resistance of skin varies greatly depend on the thickness i.e on the soles and

finger-pads is greater than the thin skin elsewhere. The average resistance is between 500 and 10,000 ohms for areas other than the horny hand and foot pads, which may offer 1 million ohms resistance when dry. A more potent factor is the dryness or dampness of the skin. While dry palm skin may have a resistance of the order of 1 million ohms, when wetted this may fall to only 1200 ohms. Jaffe (1928) stated that sweating could reduce skin resistance from 30 000 to 2500 ohms. When the current begins to pass, there is a further marked drop in resistance, as a result of electrolytic changes in the skin, which may fall to only 380 ohms. Thus for a fixed voltage, such as the mains supply of 240V, the resultant current will be far greater if the skin is wet from sweating or external moisture. This emphasizes the dangers of bathrooms and using electrical equipment in damp surroundings.

Effect on muscle:

One effect of electricity that has practical implications is the spasm that occurs in skeletal muscle if the current reaches between 10 and 40mA at 50 cps. When, as most often happens, the entry point is in the hand, the stronger flexor muscles of the arm go into spasm and cause a ‘hold-on’ effect. This means that any object being held in the hand is involuntarily clenched and, as it is likely to be the faulty electrical appliance or a wire, the object cannot be released and the current thus continues to flow. Tingling may be felt in the skin with a current of only 1mA and ‘hold on’ can begin at a current as low as 9–10mA.

Alternating and direct current:

A current of 50–80mA a.c. can be fatal in seconds, whereas 250mA d.c. for the same time is often survived. Alternating current is four to six times as likely to cause death, partly because of the 'hold-on' effect, which is the result of tetanoid muscle spasm and prevents the victim from releasing the live conductor. Alternating current is also much more likely than direct current to cause cardiac arrhythmias. The passage of a.c. at 100mA for only one-fifth of a second is likely to cause ventricular fibrillation and arrest. High amperage d.c. (above 4A) may even cause an arrhythmic heart to revert to sinus rhythm, as in medical defibrillation.

The usual frequency of a.c. is 50 cycles/second (cps). Alternating current between 40 and 150 cps is most dangerous in terms of ventricular fibrillation, and regrettably the usual mains supply lies in the centre of this range. Above 150 cps, fibrillation is progressively less likely as the frequency increases: at 1720 cps the heart is 20 times less likely to fibrillate than at 150 cps.

Electrocution:

Electrocution is defined as the passage of a substantial electric current through the tissues that can cause skin lesions, organ damage and death[1]. Tissue damage and death can come from a variety of different mechanisms.These effects are primarily due to heating or to electric stimulation (cellular depolarization) of the tissues. Heating of tissues occurs by several different mechanisms:

(1) Current flow through the tissue itself

(2) Arcing of current through the air

(3) Burning of clothing and other nearby materials

(4) Explosion of gases or other materials ignited by the electric current.

In electrocution there must be a pathway for electrons across part of the body which, in fatal cases, contains vital structures. The current enters at one point (most often a hand being used to hold, touch or manipulate some electrical device) and then leaves the body at an exit point, usually to the earth or the neutral conductor of the electricity supply. The pathway of the current will depend mainly on therelative resistance of various potential exit points. It tends to take the shortest route between entry and best exit, irrespective of the varying conductivity of different internal tissues.

1. If a person places a finger on a 240V conductor while standing with damp shoes on a wet concrete floor, then an appreciable current will pass from hand to feet, withpossibly fatal results.
2. If, however, the person is standing on a carpeted upstairs wooden floor, the poor earth return will allow only a small current to flow and all that may be suffered is a painful muscular spasm.
3. In another case of the upstairs scene, should the neutralwire of the supply be touching the skin of the same finger a few centimeters away from the live conductor, a severe local burn may occur but no danger to life, because the high resistance through the feet to earth will prevent any significant current flow passing through the thorax.
4. Should the person upstairs happen to be turning a bathtap with the other hand, the contact with the opposite finger would allow a current

to pass to earth via the tap and metal water-pipes from hand to hand across the thorax – an extremely dangerous position.

Mode of death:

1. The most common is the passage of a current across the heart, usually when a hand is brought into contact with a live conductor, and the body is earthed either through the feet or the opposite hand. It has been claimed that the most dangerous is contact with the right hand and exit through the feet, as this causes the current to pass obliquely along the axis of the heart.

   In either way, the fatal process is a cardiacarrhythmias, usually ventricular fibrillation ending in arrest. This is caused by the passage of current through the myocardium, especially in the superficial epicardial layers and possibly across the endocardium. The current has a profound effect directly upon the myocardial syncytium, the possible dislocation of the pacemaking nodes and conducting systems being ill-understood. When death occurs from cardiac arrest, the body remains either pale or only slightly congested, the autopsy appearances being unhelpful apart from the presence of any external electrical marks.
2. Less often, the passage of a current across the chest and abdomen may lead to respiratory paralysis from spasm of the intercostal muscles and diaphragm resulting, the respiratory movements are inhibited and a congestive–hypoxic death occurs.

3. Rarely, the current passes through the head and neck, usually in circumstances when the head of a worker on overhead power lines comes into contact with the conductor. In such instances, there may be a direct effect on the brainstem so that cardiac or respiratory centres are paralysed. Either cardiac arrest or respiratory paralysis can then supervene.
4. Finally, non-electrical trauma is quite common. In industrial accidents and when working on power lines, victims of shock may be thrown from a height, or suffer violent muscular spasms that may lead to fractures and other serious injury. Fall from height and other associated trauma can result in death due to the complications of sustained injury.

Death Occurring Without Skin Burns[16]:

An electric shock can kill by a variety of mechanisms without leaving burns. Contact with the skin made over a larger area will result in less heating of the skin.

An electrothermal burn of the skin will be caused by a current flow through the skin resistance that causes enough heat to be generated. A useful model has been designed to analyze heating from electric current flow [14,15]. In the model, there are two contacts, each of one square centimeter area. One contact is energized, the other grounded. The heating is assumed to be completely concentrated in the tissue near the contact points for a depth of 1 centimeter. In this model, 2 cubic centimeters of tissue (mostly water) is heated.

The rise in temperature will therefore be 0.5OC for each calorie of heat delivered to the tissue.A calculation can be done to determine if a person receiving an electric shock adequate to cause ventricular fibrillation would also suffer an electrothermal skin burn. The minimum power required to cause ventricular fibrillation at 60 hertz, 120 volts is 100 milliamperes for a tenth of a second. It has been estimated that a skin temperature of 50°C for 20 seconds is required to cause first degree burns. Charring requires a temperature of 90°C [14]. Calculations show that the fibrillating current will raise the tissue temperature only 0.145 OC and thus not cause an electrothermal burn.

Electricity causes its physiologic effect, as opposed to its heat effect, by causing cells to depolarize or to in effect lose the resting membrane potential across the cell membrane. The amount of current required to cause damage depends on the kind of cell and where it is in the body [14]. Electrical injuries have been reported to result in numerous cardiovascular complications. The most severe of these are usually manifested at the onset of injury[17]; however, serious arrhythmias have also been reported in the immediate post injury period [18]. The documented cardiovascular effects of an electrical shock include acute myocardial necrosis, myocardial ischemia without necrosis, heart failure, arrhythmias, haemorrhagic pericarditis, acute hypertension with peripheral vasospasm and anomalous non specific ECG changes [19,20].

Ventricular fibrillation is possible from 70 mA up to approximately 4 A. For more sensitive subjects, the current in milliamperes at 60 Hz required to cause

ventricular fibrillation is 116 mA times the square root of the duration of the shock in seconds [16,21].

Sudden cardiac death[13]:

Sudden Cardiac death is defined as natural death due to cardiac causes in a person who may or may not have previously recognized heart disease but in whom the time and mode of death are unexpected. In the context of time, "sudden" is defined for most clinical and epidemiologic purposes as 1hr or less between a change in clinical status heralding the onset of the terminal clinical event and the cardiac arrest itself. An exception is unwitnessed deaths, in which pathologists can expand the definition of time to 24 h after the victim was seen to be alive and stable. It is likely that ventricular fibrillation (VF) or ventricular tachycardia (VT) is the initiating rhythm in most of the sudden cardiac arrest cases.

Cardiac arrest and Sudden Cardiac Death[8]:

Structural association and causes	**Functional contributing factors**
I. Coronary heart disease A. Coronary artery abnormalities 1. Chronic atherosclerotic lesions 2. Acute (active) lesions (plaque fissuring, platelet	I. Alterations of coronary blood flow A. Transient ischemia B. Reperfusion after ischemia

aggregation, acute thrombosis) 3. Anomalous coronary artery anatomy B. Myocardial infarction 1. Healed 2. Acute	
II. Myocardial hypertrophy A. Secondary B. Hypertrophic cardiomyopathy 1. Obstructive 2. Nonobstructive	II. Low cardiac output states A. Heart failure 1. Chronic 2. Acute decompensation B. Shock
III. Dilated cardiomyopathy-primary muscle disease	III. Systemic Metabolic abnormalities A. Electrolyte imbalance (eg., hypokalemia) B. Hypoxemia, acidosis
IV. Inflammatory and infiltrative disorders A. Myocarditis B. Noninfective inflammatory diseases C. Infiltrative diseases	IV. neurologic disturbances A. Autonomic fluctuations: central, neural, humoral B. Receptor function

V. Valvular heart disease	V. Toxic responses A. Proarrhythmic drug effects B. Cardiac toxins (eg., cocaine, digitalis intoxication) C. Drug interactions
VI. Electrophysiological abnormalities, structural A. Anomalous pathways of wolff-parkinson-white syndrome B. Conducting system disease	
VII. Inherited disorders associated with electrophysiological abnormalities (congenital long QT syndromes, right ventricular dysplasia, Brugada syndrome, catecholaminergic polymorphic ventricular tachycardia, etc.)	

Table 1: Showing causes and contributing factors of cardiac arrest in sudden cardiac death.

Ischemic heart disease[22]:

Ischemic heart disease is the leading cause of sudden death worldwide for men and women. Ischemic heart disease is the generic designation of group of syndromes resulting in myocardial ischemia- an imbalance between supply and demand of heart for oxygenated blood. More than 90% of cases occurring due to obstructive atherosclerotic lesion in coronary arteries. Thus ischemic heart disease often called as coronary artery disease (CAD). In addition to coronary atherosclerosis MI can occur in cases of coronary emboli, blockage of small myocardial vessels and low systemic blood pressure [eg: shock]. A fixed lesion obstructing coronaries, 75% or more than that is essential to cause symptomatic ischemic change in myocardium. Although only a single major coronary epicardial trunk may be affected, two or all three- the left anterior descending artery [LAD], the left circumflex [LCX] and the right coronary artery [RCA]- are often involved by atherosclerosis. In most of the cases permanent damage to the heart occurs when the perfusion of myocardium is severely reduced for an extended interval (usually 2-4 hours). Necrosis is usually complete within 6 hours of myocardial ischemia.

MI less than 12 hours old is usually not apparent in gross examination. If patient died after 2-3 hours, the infracted area can be demonstrated by triphenyltetrasolium chloride, which would detect the lactate dehydrogenase [LDH] activity in myocardium and stains bright red. The dead cells lack LDH activity so they will appear pale. By 12 to 24 hours an infarct can be identified

grossly in transverse slices as a reddish-blue area of discoloration caused by stagnated, trapped blood. Thereafter, the infarct becomes progressively more sharply defined, yellow-tan, and soft. By 10 days to 2 weeks, it is rimmed by a hyperemic zone of highly vascularized granulation tissue. Over the succeeding weeks, the injured region evolves to a fibrous scar.

During histopathological examination the coagulative necrosis can be detected by 6-12 hours. Wavy fibers can be detected at the periphery, which is produced by systolic tug of adjacent myocardium. The necrotic muscle elicits acute inflammation (between 1 and 3 days). Thereafter macrophages remove the necrotic myocytes(3 to 7 days), and the damaged zone is progressively replaced by the ingrowth of highly vascularized granulation tissue (1 to 2 weeks); as healing progresses, this is replaced by fibrous tissue. In most instances, scarring is well advanced by the end of the sixth week, but the efficiency of repair depends on the size of the original lesion. Once a lesion is completely healed, it is impossible to determine its age (i.e., the dense fibrous scar of 8-week-old and 10-year-old infarcts may look identical).

Cardiomyopathies:

Dilated cardiomyopathy (DCM)[13]:

Patientswith dilated cardiomyopathy is the second largest group patients who undergoes sudden cardiac death, accounting for 11-15% of annual deaths. The presence of reduced left ventricular Ejection fraction and syncope are the markers of sudden cardiac death in these patients.

Hypertrophic cardiomyopathy (HCM):

The incidence of sudden cardiac death in patients with HCM is 2% to 4% per year in adults and 4% to 6% in children and adolescents. Risk factors to identify sudden cardiac death are prior sudden cardiac arrest, family history of sudden cardiac death, sustained or nonsustained ventricular tachycardia (NSVT), syncope, a drop in blood pressure with exercise, and septal hypertrophy less $\geq$ 30mm. Sudden cardiac death usually results from ventricular arrhythmias, but occasionally it may be precipitated by atrial fibrillation, bradyarrhythmias or myocardial ischemia.

Arrhythmogenic right ventricular dysplasia (ARVD):

ARVD is a rare genetic disorder characterized by heart failure, ventricular arrhythmias and sudden cardiac death. Mutations involving the desmosome are manifested by fibrofatty infiltration of the right ventricle. The incidence of sudden cardiac death is approximately 2% per year and is mainly due to ventricular tachyarrhythmias. ARVD is identifiable by right bundle branch block (RBBB), T wave inversion in V1 through V3 and epsilon waves on the electrocardiogram (ECG), regional right ventricular akinesia, dyskinesia, or aneurysm on Echo or MRI and findings on endomyocardial biopsy.

The channelopathies:

Congenital Long QT syndrome:

This syndrome is characterized by prolonged repolarization resulting in an increase of the QT interval. Clinical presentation includes syncope and sudden death due to torsade's de points. LQTSs is a familial syndrome with a prevalence of about 1:2000. The two varients of the syndrome include more common dominant form (Romeno-ward syndrome) and less common recessive form (Jervellandlange Nielsen syndrome), which is associated with congenital deafness. To date, mutations at 12 different LQTSs susceptibility genes have been identified.

The most common, accounting for more than 50% of cases, is mutation of KCNQ1, which encodes the α sub unit of potassium channel, conducting the slow delayed rectifier current [I_{ks}]. The most common subtypes are LQT1, LQT2 and LQT3, which are characterized by mutations in the I_{Ks}, I_{Kr} *and*I_{Na} respectively. LQT1 and LQT2 mutations result in decreased in outward potassium current, while LQT3 mutations results in increased inward sodium current, both of which result in increased repolarization.

LQT1 patients typically have broad-based T waves and exercise induced arrhythmias, especially during swimmings. LQT2 syndrome is characterized by low-amplitude or notched T waves and auditory triggers such as sudden loud

sounds like alarm clock and strong emotions, and LQT3 is characterized by a long isoelectric ST segment and arrhythmias during sleep.

Acquired Long QT Syndrome:

Patients with genetic predisposition related to what appear to be sporadic mutations and/or single nucleotide polymorphisms can develop marked QT prolongation and associated polymorphic ventricular tachycardia (TDP) are seen more frequently in women and may be a manifestation of subclinical LQTS. Drug induced long QT and TDP frequently are potentiated by the development of hypokalemia and bradycardia. The offending drugs typically block the potassium I_{kr} channel.

Brugada syndrome:

It is a condition associated with sudden cardiac death in the setting of a structurally normal heart, characterized by an electrocardiographic pattern of RBBB and ST segment elevation in leads V1 to V3. It is inherited as autosomal dominant pattern with male predominance. It is a genetically heterogenous disease with many mutations linked to the gene SCN5A, which encodes for the cardiac sodium channel, leading to unopposed I_{to} potassium current in Right Ventricular epicardium. The diagnosis should be considered in patients who have documented ventricular fibrillation, family members with ST segment elevation, self-terminating polymorphic ventricular tachycardia, syncope and

family history of sudden death in the setting of the electrocardiographic findings noted previously. Incomplete RBBB and coved ST segment elevation in the right precardial leads is diagnostic and, although often transient, can be elicited by drug challenge. The arrhythmia commonly occurs at rest or during sleep, and the risk of sudden cardiac death is usually 30% in 3 years in untreated symptomatic patients.

Catecholeminergic polymorphic Ventricular Tachycardia:

This arrhythmia is more common in adolescents and children and may present with sudden cardiac death or stress induced syncope. While usually familial, it can occur in de novo mutations. Triggers often include, emotional or physical stress, and the arrhythmia can be polymorphic, bidirectional, and less commonly ventricular fibrillation. Two genes have been identified so far: Calsequestrine 2 [autosomal recessive pattern] and cardiac ryanodine receptor [autosomal recessive pattern].

WPW syndrome:

The basic abnormality is the presence accessory pathway of conducting tissue between the atria and ventricles, other than the normal conduction system. This accessory pathway permits the atrial impulses to bypass the normal conduction system. In the past, this accessory pathway is also known as “bundle of kent”. The risk of sudden cardiac death is higher especially if they have rapidly conducting accessory pathways, when atrial fibrillation can be

associated with very rapid ventricular rates and degeneration to ventricular fibrillation.

Myofiber break up:

The term myofiber break up includes the following histological patterns: (1) bundles of distended myocardial cells alternating with hypercontracted cells. In the latter group of cells there is also widening or rupture (segmentation) of intercalated discs. Myocardial nuclei in the hypercontracted cells have a "square" aspect rather than the ovoid morphology seen in distended myocytes. (2) hypercontracted myocytes alternated with hyper distended cells that are often divided by widened disc, (3) non-eosinophilic bands of hyper-contracted sarcomeres alternating with stretched, often apparently separated sarcomeres.

However the Two aspects of the process need further consideration: (1) whether the changes are real or artifactual and (2) whether the morphologic changes actually played a role in the initiation of the fatal arrhythmia.

The MFB changes G. Baroldi and Vittorio Fineshi[23] et al., described seem to be vital. Their absence in all hearts excised at transplantation questions the belief that they are artifactual or secondary to histological processing; the artifactual myofiber fragmentation due to the knife in cutting the histologic section being easily distinguished from MFB. On the other hand, similar changes were never described as part of rigor mortis of the myocardium[24] . Furthermore, if rigor mortis was the cause, MFB should be a regular finding in all cases autopsied after 1–24 h. In fact, when ventricular fibrillation starts,

almost immediately there is a loss of pump function with consequent severe reduction or abolishment of coronary flow[25]. The maintenance of some contractility in the absence of nutrient flow is unlikely as shown in experimental coronary occlusion where regional sudden loss of blood flow determines loss of contractility within a few seconds[26].

The morphologic aspect of MFB indicates a structural chaotic state of contraction at different level (bundles or myocells). However, the extent of this morpho-functional disruption ranges from small foci in one area to a diffuse involvement of most or all cardiac regions. One may speculate that, independent from its extent, ventricular fibrillation may be triggered (re-entry or rapid focal discharge) by a small focus or a large one, respectively in expression of short or long-lasting terminal malignant arrhythmia. In turn, successful or unsuccessful defibrillation could be related to the extent of this damage. The cases monitored electrically at the time of death showing malignant arrhythmia with unsuccessful resuscitation, had MFB extended at more than four sites.

The main cause of ventricular fibrillation and malignant arrhythmia is considered to be ischemia. It is generally accepted that any intervention that increases spatial difference of heart muscle also facilitates the induction of certain cardiac arrhythmias. Ischemia is the most likely culprit for this difference, but the findings of focal myocardial necrosis and/or fibrosis are common in cases of sudden arrhythmic death and are often assumed to be the cause of re-entry and electrical instability [23, 27, 28]. Whether MFB falls into this

same category remains to be proved, but it certainly is a possibility that must be considered.

The documented clinical cardiovascular effects of an electrical shock include immediate heart failure, acute myocardial necrosis, myocardial ischemia with or without necrosis, arrhythmia, hemorrhagic pericarditis, acute hypertension with peripheral vasospasm and anomalous but non-specific ECG changes [29]. In rapid death due to electrical shock, G. Baroldi et al., found typical cardiac lesions, characterized by a break-up of myocardial fibres that could provide the structural substrate necessary to initiate chaotic, electrical asynchronous activity and could be induced by the passage of abnormal electrical currents. The obvious limitation of the study is that autopsy findings can only rarely be correlated with terminal electrocardiographic recordings [30]. They also demonstrated that all the ECG-monitored cases with fibrillation had extensive MFB [23].

Literature review:

G. Baroldi[23] et al has conducted a study on myofiber breakup (MFB); a marker for ventricular fibrillation in sudden cardiac death. He studied heart samples from 432 cases which brought for autopsy, and divided the cases into 7 groups and named as coronary group, chagas group, intracranial hemorrhage group, transplant group, AIDS group, congestive heart failure group and cocaine group. He took 16 myocardial samples from each case and stained with haematoxylin and eosin or by specific stains and studied the pattern of

morphology and frequency of myofiberbreakup. He concluded that, if our postulates are correct finding myofiber breakup in the myocardium might allow the diagnosis of a malignant arrhythmia followed by cardiac arrest due to ventricular fibrillation even in the absence of clinical information (sudden death out-of-hospital). He also noted that, in the fifteen monitored cases with vital signs at the time of resuscitation, all had the electrocardiographic pattern of ventricular fibrillation and extensive myofiber breakup. All ourcases monitored electrically at the time of death showingmalignant arrhythmia with unsuccessful resuscitation, hadMFB extended at more than four sites.

Vittorio Fineshi[31] et al., studied morphological changes in hearts of 118 subjects, he divided the subjects into 4 study group. 1. 21 Individuals died instantaneously due to electrocution, 2. 26 individuals died of cocaine intoxication, 3. 45 individuals died of head trauma, 4. 26 healthy individuals died of carbon monoxide poisoning at home. He took 16 myocardial samples from each case and stained with haematoxylin and eosin or by specific stains when indicated and did immmunohistochemical staining by using monoclonal anti-complement C9 antibodies in all hearts. He noted that the examination of conduction system was unremarkable and there was no correlation between gender, age, heart weight, degree of coronary atherosclerosis and myofiber breakup. The frequency of myofiber breakup was maximal in electrocution deaths (90%), in respect to control groups. He also noted that myofiber breakup is an antemortemchange.They conclude that in rapid death due to electric shock, the typical cardiac lesions characterized by break-up of myocardial fibres that

could provide the structural substrate necessary to initiate chaotic, electrical asynchronous activity, possibly induced by the passage of abnormal electrical currents.

Shubha. H V and Nirmala. C[32] et al., studied the various histopathological changes in the 15 heart specimens of electrocution and compared it with15 normal heart specimens. The tissue sections were stained with eosin and haematoxylin and analysed. They recorded the following histopathological changes in electrocution deaths: Myofiber break- up(100%), separation of the myofibers (100%), hemorrhage with extravasation of RBC's, disarray of myofibers (87%), hypercontractedmyocytes with squaring of nuclei (73%), myocellular segmentation (33%), myocyte vacuolization (20%). Whereas in normal heart sections few showed similar findings in focal areas such as the myofiber break-up (33%), separation of myofibers (20%) which were not accompanied by hemorrhage and extravasation of RBC's. But none of the normal heart sections showed disarray of myofibers, hypercontractedmyocytes with square expression of nuclei, myocellular segmentation and myocyte vacuolization. The histopathological findings in the heart found consistently in electrocution cases compared to that of the normal hearts. They concluded that histopathological changes in the heart provide an additional clue in the diagnosis of electrocution deaths.

B. Viswakanth[33] et al., studied 6 cases of electrocution deaths, he analyzed the electrical skin mark as well as the myocardial samples. While

analyzing the myocardial samples, he noticed the following histopathological changes of myofiber break-up was seen in all cases (100%).

Xenopoulos[34] et al., has reported one case of fatal electrocution (110V AC) in a 19 year old male where he noticed a hemorrhage into the myocardium with patent coronary arteries. On histopathological examination he documented widespread contraction band necrosis, myolysis, coagulative changes, loss of striations and nuclear disappearance and diffuse free blood in the interstitium corresponding to the gross hemorrhagic areas, slight vacuolization between the endothelial layer and above the internal elastic membrane. He conclude that similarity of myocardial injury to reperfusion injury in our patient as well as the absence of coronary thrombus or significant atherosclerosis at autopsy makes coronary spasm a likely cause of myocardial necrosis in electrocution.

Ku[35] et al., described a case of 20 year old previously healthy male sustained an electrical injury (220V, Alternating current) while manipulating a refrigerator, he immediately lost his consciousness and underwent ventricular fibrillation, for which cardioversion was done and patient revived in a hospital. After 3 weeks of episode he was transferred to cardiology department, with no signs of entry or exit mark presented with left ventricular apical myocardial infarction. The coronary arteriograms were normal. So he hypothesized that the direct effect of electricity on coronaries resulting in coronary spasm was the probable cause for the myocardial infarction in that case. He concluded that low voltage electrical current may cause cardiac injury, and routine

echocardiographic screening may is therefore very important in patients surviving electrical injury.

Ghandour[36] et al., performed an experimental study on the cardiac histopathological and immunohistochimical changes due to electrical injury in rats. The experiment was done on 70 female rats, which were divided randomly into 4 groups [A,B,C,D]. In group A 20 rats were subjected to instantaneous antemortem electricity, in group B 20 rats were electrically injured instantaneously postmortem, in group C 20 rats were electrified upto death and 10 rats in group D acted as control group. He further subdivided the group into A1, A2, B1, B2, C1, C2 and D1,D2 by equally dividing the allotted samples. In A1, B1, C1, D1 groups the heart was collected immediately after death and in A2, B2,C2, D2 groups the heart was collected 1 hour after death. He noticedmyofiber break-up in group C and few square nuclei and thrombi in the intramyocardial vessels of group A1, few foci of intramyocardialhaemorrhage in group A2. While break-up of myocardial fibres was not seen in any of the heart specimen received from group 2. This signified that the finding myofiber break-up is antemortem in nature. He also noticed that the expression of c-fos oncogene was more in group C1, C2, A1 and A2. But there was only minimal expression in group B1 and no expression at all in group B2. He concluded that classical morphology of heart remains a gold standard to determine the death due to electrical injury in forensic cases. The immunohistochemical changes could provide additional clue for diagnosis.

Badawy[37] et al., has done an experimental study onthe effects of low-voltage electrocution on hearts of male albino rats by using histopathological and immune histochemical analysis. He used a 26 male albino rats and divided randomly into two main groups, the control group and experimental group. The 10 rats in control group were killed by cervical dislocation without electrocution and 16 rats in experimental group were electrocuted until death by a 220V alternating current applied to the skin of left forelimb and skin of right hindlimb. He found areas of interstitial hemorrhage, necrotic and fragmented cardiomyocytes, square and round nuclei, myocardial waviness and contraction band necrosis in the heart specimens of electrocuted group in comparison with control group. Also, they were showed positive reaction for caspases when compared to control group.

This study concludes that the histopathological changes and immunohistochemical findings, besides the circumstantial evidence and external marks that may found at autopsy, may provide a possible means of confirming the diagnosis of death caused by electrocution in suspected cases associated with limited external findings.

MATERIALS & METHODS

MATERIALS AND METHODS

The Electrocution and Sudden Cardiac Death medico-legal cases brought for postmortem examination in Department of Forensic Medicine, Coimbatore medical college hospital, Coimbatore were studied during the period January 2018- June 2019. Before starting autopsy, police inquest was received, the history and circumstantial evidence of electrocution and sudden cardiac death were analyzed. The postmortem examination was initiated after the body was identified by investigating police officer by two identification marks. The external examination was started with examination of cloths and body, for any physical violence, foreign bodies, electric burn mark [in cases of electrocution] and external signs of any natural diseases [in cases of sudden cardiac death]. Then autopsy was initiated by putting an "I" shaped incision. The thorax was dissected under water to look for pneumothorax and the sternum was removed by using hand-saw, both pleural cavities were examined for pleural fluids if present measured. The pericardium was opened by using scalpel; the amount of pericardial fluid was measured. The heart was grossly looked for pinpoint haemorrhages, pericarditis, and any anomalies in situ. Then the heart was removed by cutting the inferior vena cava[IVC], superior vena cava[SVC] and four pulmonary veins as close to the pericardium as possible. The heart was weighed by using a weighing machine after removing the postmortem blood clots and then the length, breadth was measured.

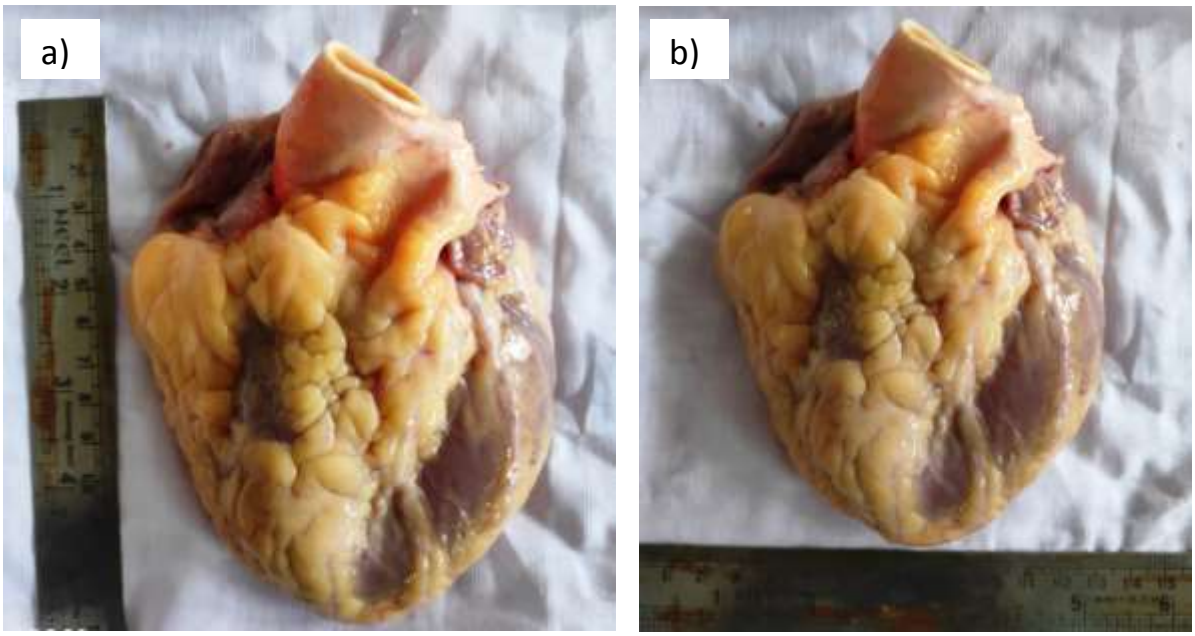


Figure 8: Shows measurement of length a) and breadth b) of heart.

The heart was fixed by using 10% formaline and sent for histopathological examination. Fixation Preserves cells and tissue components with minimal distortion and stabilizes proteins, rendering the cell and its components resistant to further autolysis by inactivating lysosomal enzymes. It also changes the tissues receptiveness to further processing and helps in thin sectioning of specimen. Tissues were fixed in 10% formalin for 2 hours.In histopathology laboratory after fixation, heart was grossly examined and was cut opened by using Inflow- outflow method and Four chamber method of dissection wherever necessary. The following were the steps used in Inflow-outflow method [38]:

1. The right atrium was opened by cutting along the free end of inferior vena cava to the tip of the atrial appendage.
2. The right atrial appendage and tricuspid valves were examined.
3. The right ventricle was opened by cutting along the free, lateral border by cutting through the tricuspid ring and continuing excision to the apex.
4. The pulmonary outflow tract was opened by cutting along the anterior border of right ventricle which starts at the apex and continues up through the pulmonary conus, valve and artery.
5. The left atrium was opened by cutting across the roof of atrium, from the orifice of right pulmonary veins to the orifice of left pulmonary veins.
6. The left atrium and mitral valves were examined.
7. The left ventricle was opened along the lateral border by cutting through the mitral valve and continuing the cut through the aortic valve and aorta.

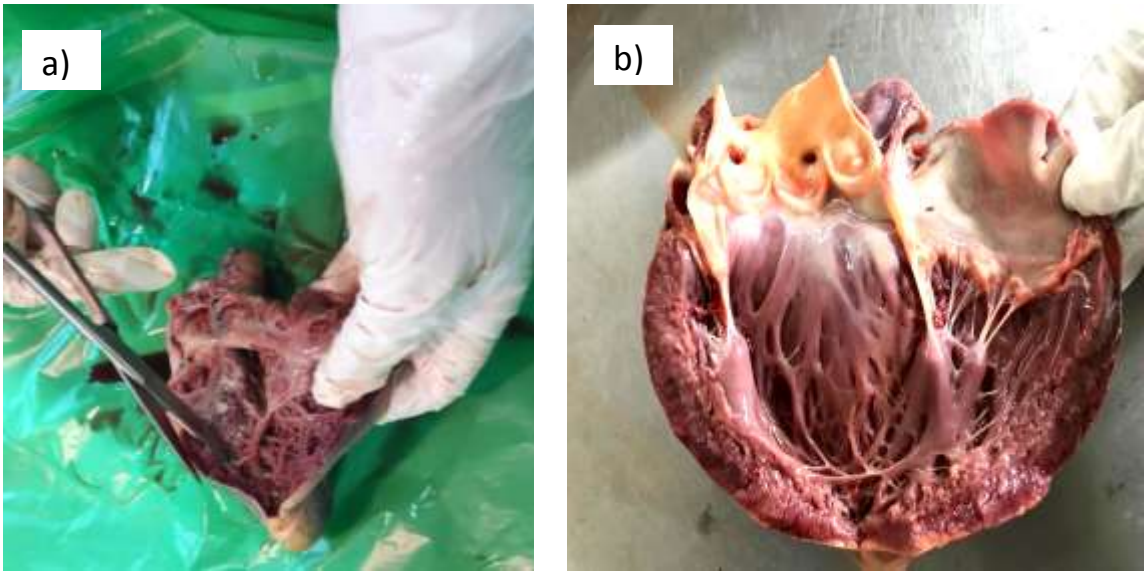


Figure 9: a), b) showing dissection of heart by inflow-outflow method.

After the heart has been opened, the thickness of the both ventricles were measured, 1cm below atrioventricular valves. Then the ventricular surface was grossly examined for any infarction, fibrosis, hemorrhage, thrombosis and dilatation. Two blocks were taken from left ventricle, two blocks from right ventricle, two blocks from the interventricular septum, aorta, pulmonary trunk and blocks from right and left coronary arteries were taken. Additional blocks were also taken from the suspected lesions of myocardium.

These tissue blocks will be further dehydrated slowly. Dehydration is a process of the removal of 'free' unbound water and aqueous fixatives from the tissue components. Dehydration is done by putting the blocks initially in 70% isopropyl alcohol followed by 90%, 95% and 100% isopropyl alcohol respectively. Tissue processed in 70% isopropyl alcohol for 1hour followed by 90%, 95% for 1 hour each and 100% isopropyl alcohol for 2 hours. After that xylene [clearing agent] were added remove the isopropyl alcohol [dehydration solution] and to make the specimen receptive for paraffin wax [infiltrating medium]. The tissue processed in xylene for 2 hours at 38°C. Then the tissue blocks were placed in paraffin wax bath for 2hours at 62°C. By using Leuckhards L mould the tissues were embedded for 1 hour at 60°C and allowed to cool for 30minutes. Then the tissues were dewaxed and rehydrated through descending order of isopropyl alcohol to water. Then the specimen was stained with ehrlich's hematoxylin for 5 minutes and washed under running water. Followed by 1% acid alcohol for 5-10 seconds, after that washed in tap water for 10-15 minutes or till the slide turns blue. 1% eosin was added for 10 minutes

and washed under tap water for 5 minutes. The tissues were dehydrated by adding alcohol, cleared and mounted. The tissue sections were observed under light microscope.

RESULTS

OBSERVATION AND RESULTS

Result for electrocution:

A larger number of victims were belongs to the age group of 31-40 years (30.8%) and majority of them were male (84.6%). The manner of death in most of the cases (94.2%) was accidental, while 3 cases were suicide. The electrocution happened in 43 cases during day and in 9 cases at night. While analyzing the source of electrocution 21.2% of victim had a direct contact with an unprotected electrical wire, 1.5 % suffered electrocution from transformer. 46.2% victims died of electrocution at their work place.

Age	Number of cases
0-10y	1
11-20y	11
21-30y	10
31-40y	16
41-50y	6
51-60y	4
61-70y	3
71-80y	0
81-90y	1
Total	52

Table 2: Shows age wise distribution of electrocution deaths.

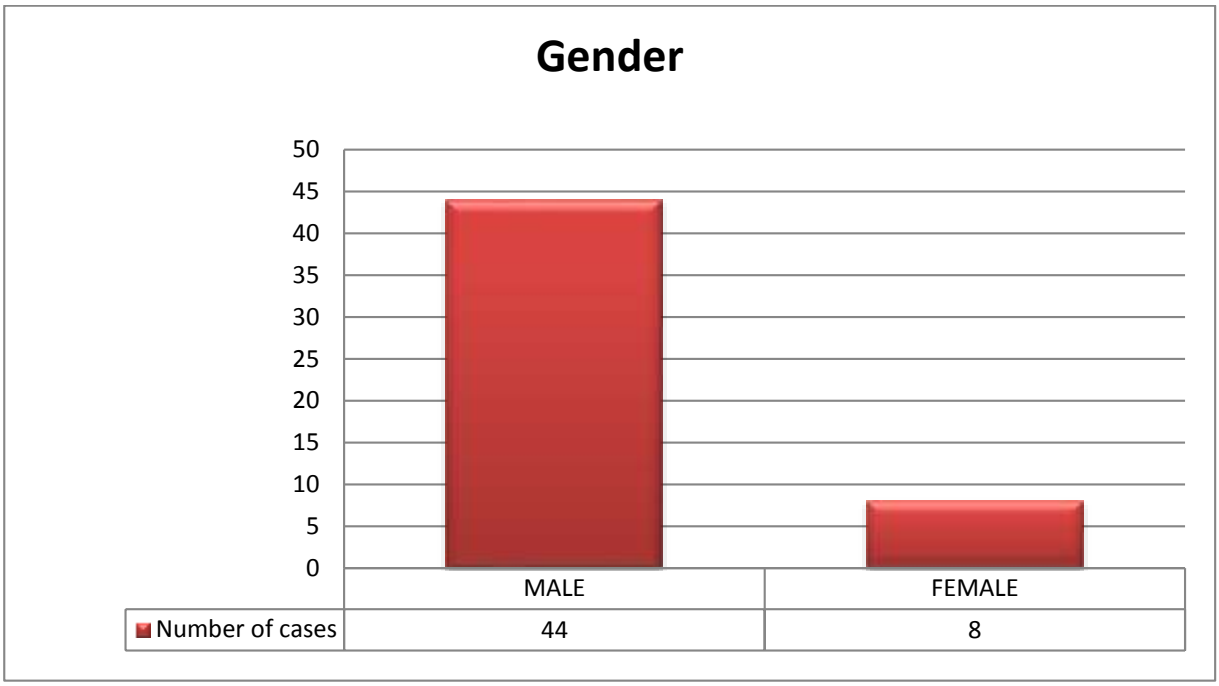


Chart 1: Shows gender wise distribution of electrocution deaths.

Source of electric shock	**Number of cases**
Electric wire	11
High voltage Transformer	8
Switch box	6
Machine used at work place (tailoring/ industrial purpose…etc)	5
Electric Fence (high voltage)	4
Water Heater	6
Cable Wire	3
Electric Post (high voltage)	3
Electric Belt (high voltage)	2
Lamp post (high voltage)	2
Electronic Battery	1
Electric water Pump	1
Total	52

Table 3: Shows source of electric shock in electrocution deaths.

During postmortem examination, both entry and exit electrical burn marks were noted in 25 cases, whereas only one electrical burn mark was present in 27 cases. Dermo-epidermal burn injuries were noted in 7 cases, 2 cases were presented with charring of entire body and one case with charring of arm. In one case victim sustained multiple injuries, when he thrown off while working in a transformer and Another victim fell off from the electric post and sustained cranio-cerebral injuries following electric shock. Both victims were died of electrocution and associated injuries.

Autopsy findings	**Number of cases**	**Percentage**
Presence of one electrical mark	27	51.9%
Presence of both Electrical Entry and Exit burn mark	25	48.1%
Dermo-epidermal burn injuries	7	13.5%
Charring of entire body	2	3.8%
Cranio cerebral injuries	2	3.8%
Charring of arm	1	1.9%
Multiple injuries	1	1.9%
Gross examination:	47	90.4%
Multiple Pinpoint hemorrhages over the surface of heart		

Table 4: Shows Autopsy findings noted in electrocution deaths.

Based on the entry and exit electrical burn marks, we could able to hypothesize the pathway of current in 15 cases. Among 15 cases, current has passed through the heart in 13 cases and through vital centres of brain in 2 cases.

Entry mark	Exit mark	Probable organ involved	Number of Cases
Arm	Arm	Heart	6
Left Arm	Right Foot	Heart	5
Chest	Foot	Heart	1
Chest	Arm	Heart	1
Face	Arm	Brain	2
		Total	15

Table 5: Shows hypothesized Pathway of current in electrocution deaths.

Gross examination of heart showed, multiple pinpoint hemorrhages in 90.4% of cases and findings were unremarkable in 5 cases (9.6%).

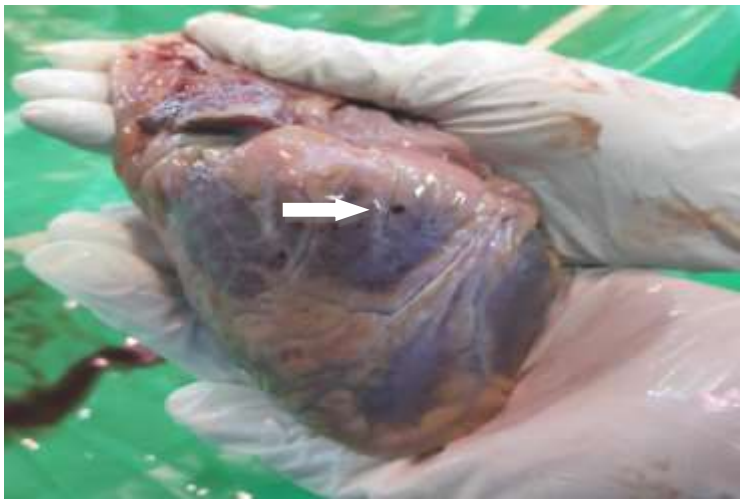


Figure 10: Shows multiple pinpoint hemorrhages over the surface of heart.

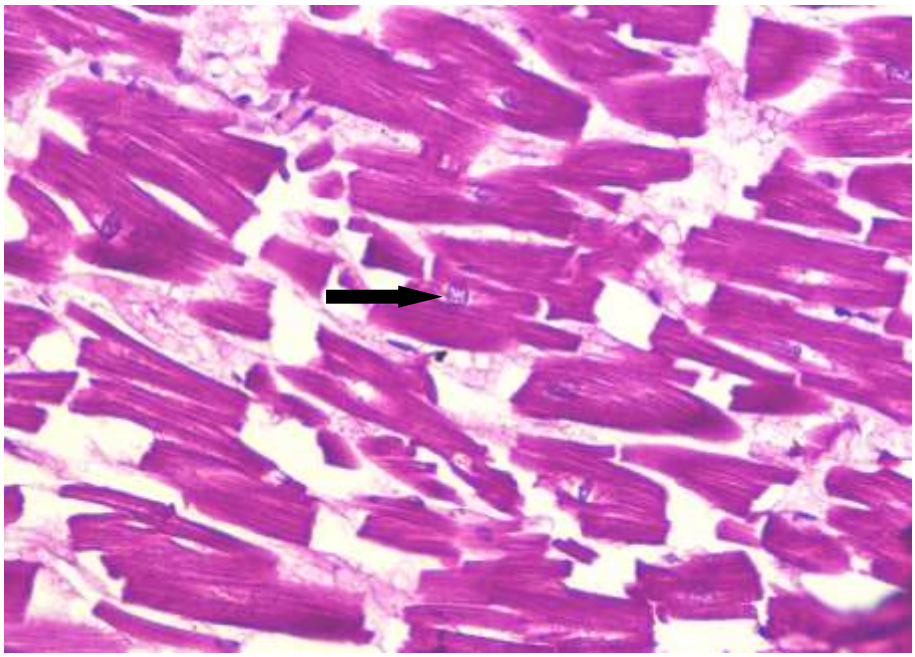
During Histopathological examination we noticed squaring of nucleus (98.1%), hyper contracted myocytes alternating with hyperdistended myocytes (98.1%), separation of myofibres (98.1%), dilated and congested blood vessels

which corresponded to the pin point hemorrhages (92.3%) and hemorrhage into the myocardium (69.2%). Acute myocardial infarction was noted in one case and myocyte disarray in another case. Healed myocardial infarct seen in 4 cases (7.6%).

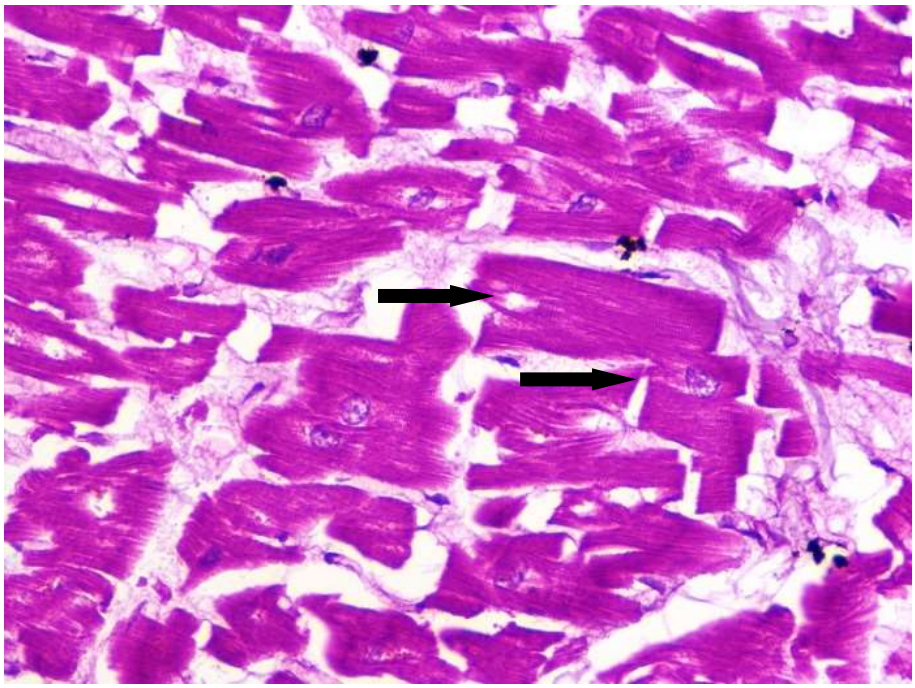
Histopathology examination findings	**No of cases**	**Percentage**
Bundles of distended myocardial cells alternating with Hypercontracted cells and Hypercontracted cells showing square expression of nucleus	51	98.1%
Non eosinophilic bands of hypercontracted sarcomeres alternating with stretched, often apparently separated sarcomeres.	51	98.1%
Hypercontracted myocytes alternating with hyperdistended myocytes.	51	98.1%
Dilated and congested blood vessels	48	92.3%
Hemorrhage into the myocardium with extravasation of RBC's	36	69.2%
Old myocardial infarct	4	7.6%
Myocyte disarray	1	1.9%
Acute myocardial infarction	1	1.9%

Table 6: Shows findings noted on Histopathological examination of heart in electrocution deaths.

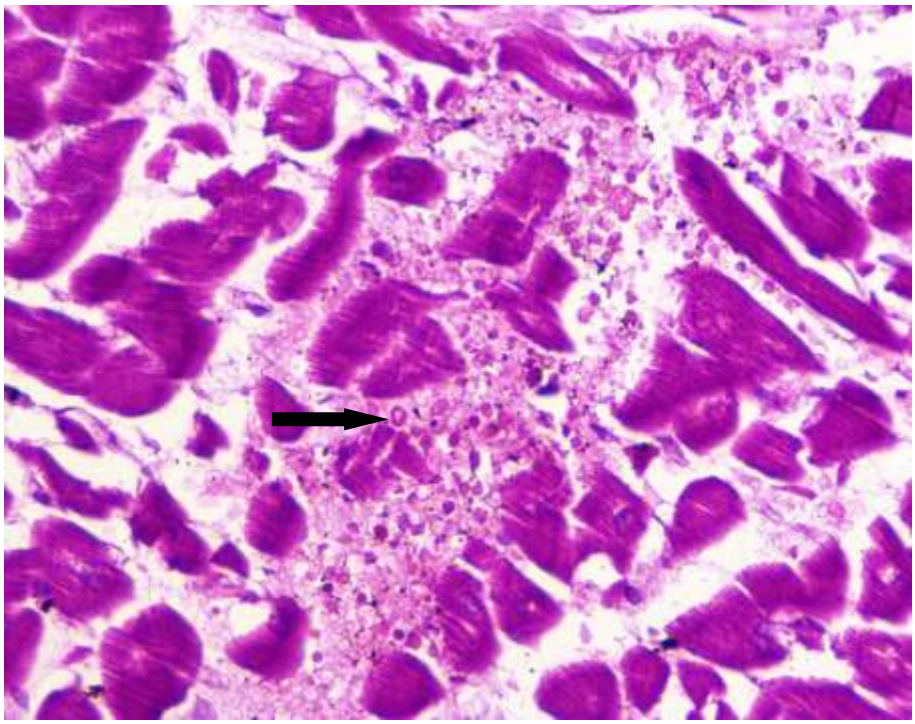
COLOUR PLATES



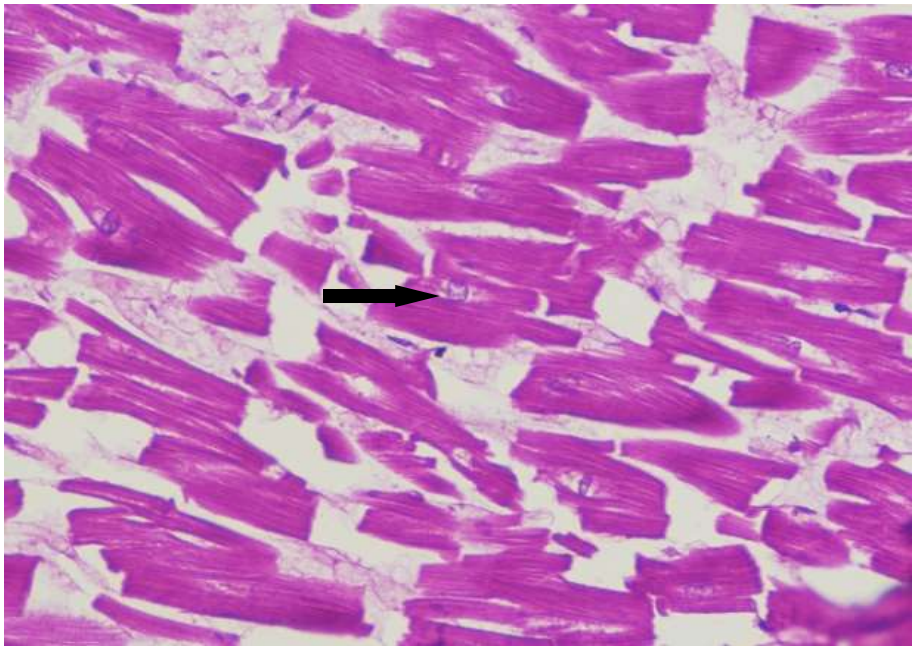
COLOUR PLATE 1: Square nuclei (arrows) in hypercontracted myocells (Eosin and Hematoxylin stain)



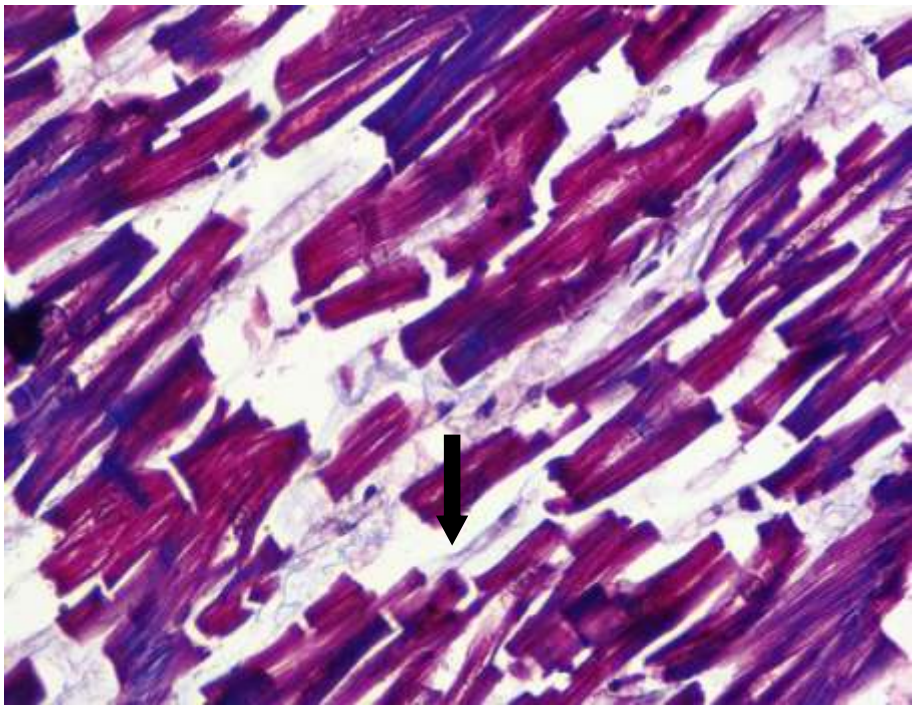
COLOUR PLATE 2: Bundles of hyper-contracted myocytes (arrows) alternated with bundles of hyper-distended myocardial cells (Eosin and Hematoxylin stain)



COLOUR PLATE 3: Bundles of hyper-contracted myocytes alternated with bundles of hyper-distended myocardial cells along with extravasated Red Blood cells (arrow) (Eosin and Hematoxylin stain)



COLOUR PLATE 4: Bundles of hyper-contracted myocytes (arrows) alternated with bundles of hyper-distended myocardial cells (Eosin and Hematoxylin stain)



COLOUR PLATE 5 : Separation of sarcomeres (arrows) in myofibres connected with contracted ones (Trichrome stain)

Result for sudden cardiac death:

Out of 52 victims, 28(53.8%) were belong to the age group 21-30 years and 18 (34.6%) were between 31-40 years of age. 17 victims had sudden loss of consciousness, 11 victims complained of chest pain and 5 had history of breathlessness, 8 were didn't wake up from their sleep, 4 had stomach pain, and one had seizures within 24 hours before death. All the victims were apparently healthy, there were no history of cardiac disease. 86.5% of Sudden cardiac death occurred in males.

AGE	Number of cases
0-10	1
11-20	5
21-30	28
31-40	18
41-50	0
Total	52

Table 7: Shows age wise distribution of sudden cardiac death.

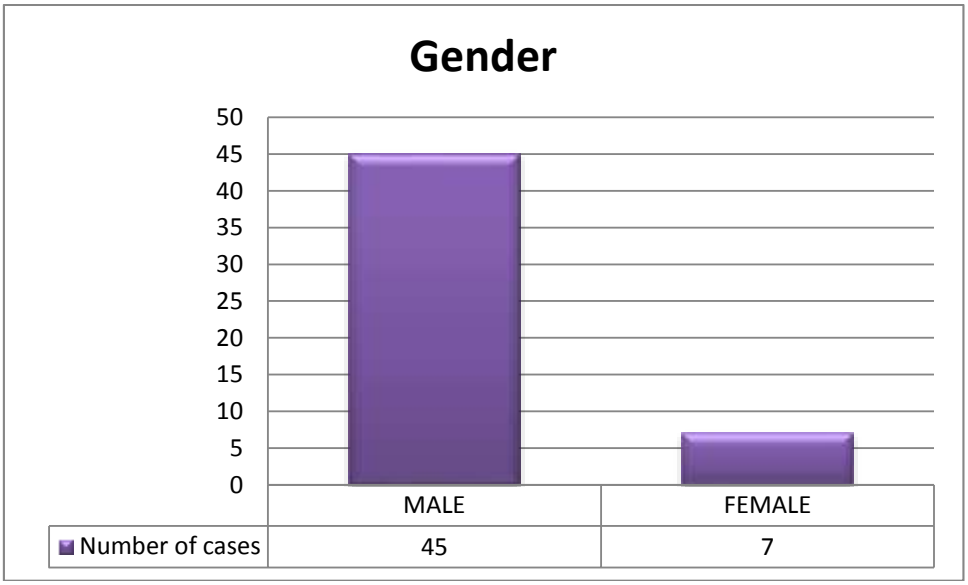


Chart 2 : Shows gender wise distribution of sudden cardiac death.

Symptoms or complaints before death	Number of deaths
Sudden loss of consciousness	17
Chest pain	11
Didn’t wake up from sleep	8
No relevant symptoms from history	6
Breathlessness	5
Stomach pain	4
Seizures	1
Total	52

Table 8:History of symptoms or complaints experienced by the victim before death in sudden cardiac deaths.

During autopsy there were no external or internal ante-mortem injuries or marks of violence noted. On gross examination of the heart almost all the specimens had a normal weight ranging from 260-340 grams, whereas in one case heart was enlarged measuring 14x11cm weighing 5 grams. In the latter case the histopathology revealed hypertrophy along with myofiber disarray suggestive of hypertrophic obstructive cardiomyopathy (HOCM).

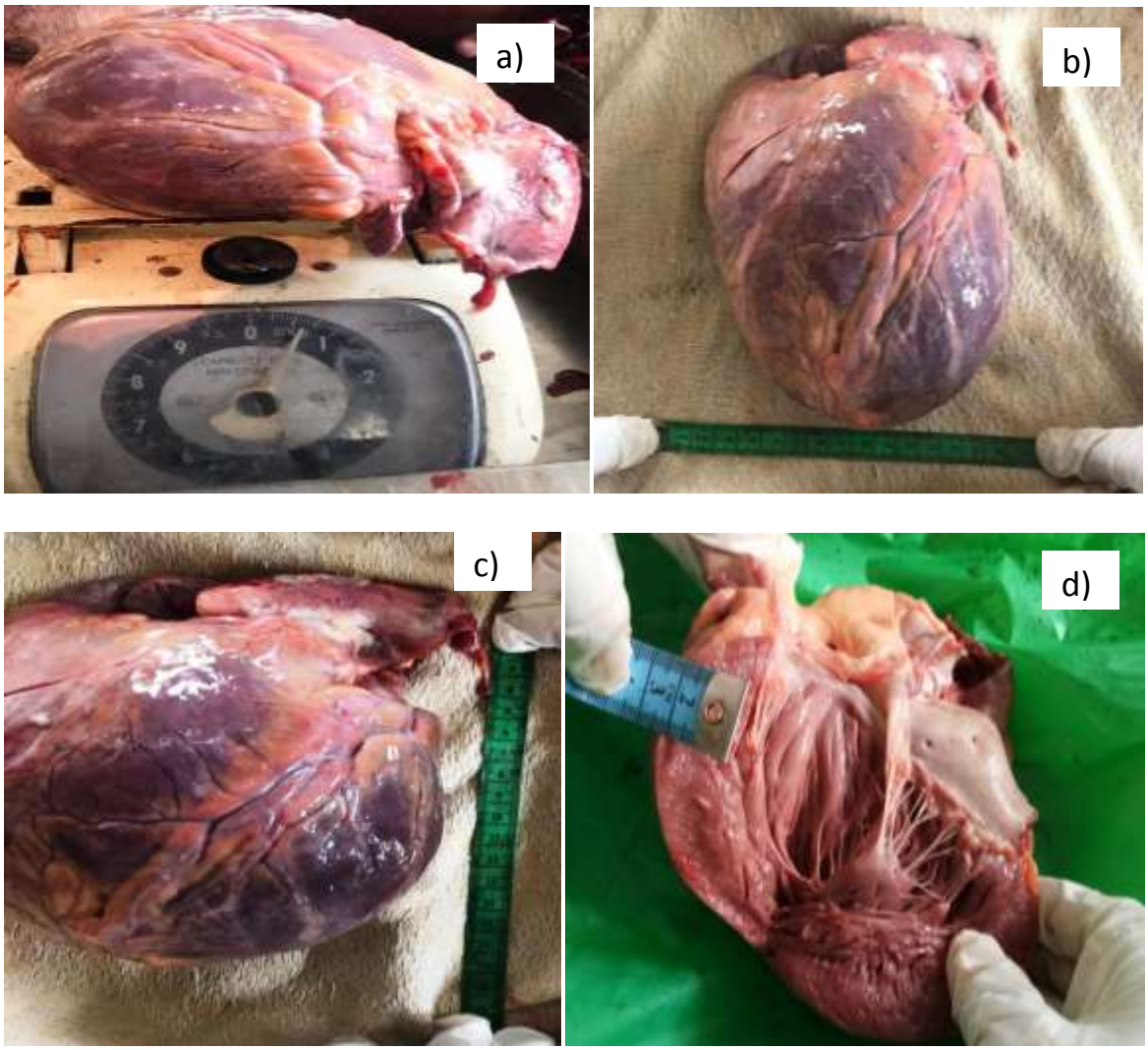


Figure 11: Shows enlarged heart measuring 14x11cm (c,b) weighing 700gms (a), left ventricular wall thickness is 2.5cm (d).

On external examination hemorrhagic areas over the surface noted in 8 cases, whitish plaques (patchy pericarditis) in 6 cases, both whitish plaques and hemorrhagic areas were noted in 6 cases and pinpoint hemorrhages over the surface in 3 cases. Occlusion of coronary artery with evidence healed Myocardial infarction noted in one case and thickened aorta noted in another case. In the latter case histopathology showed features of aortitis.

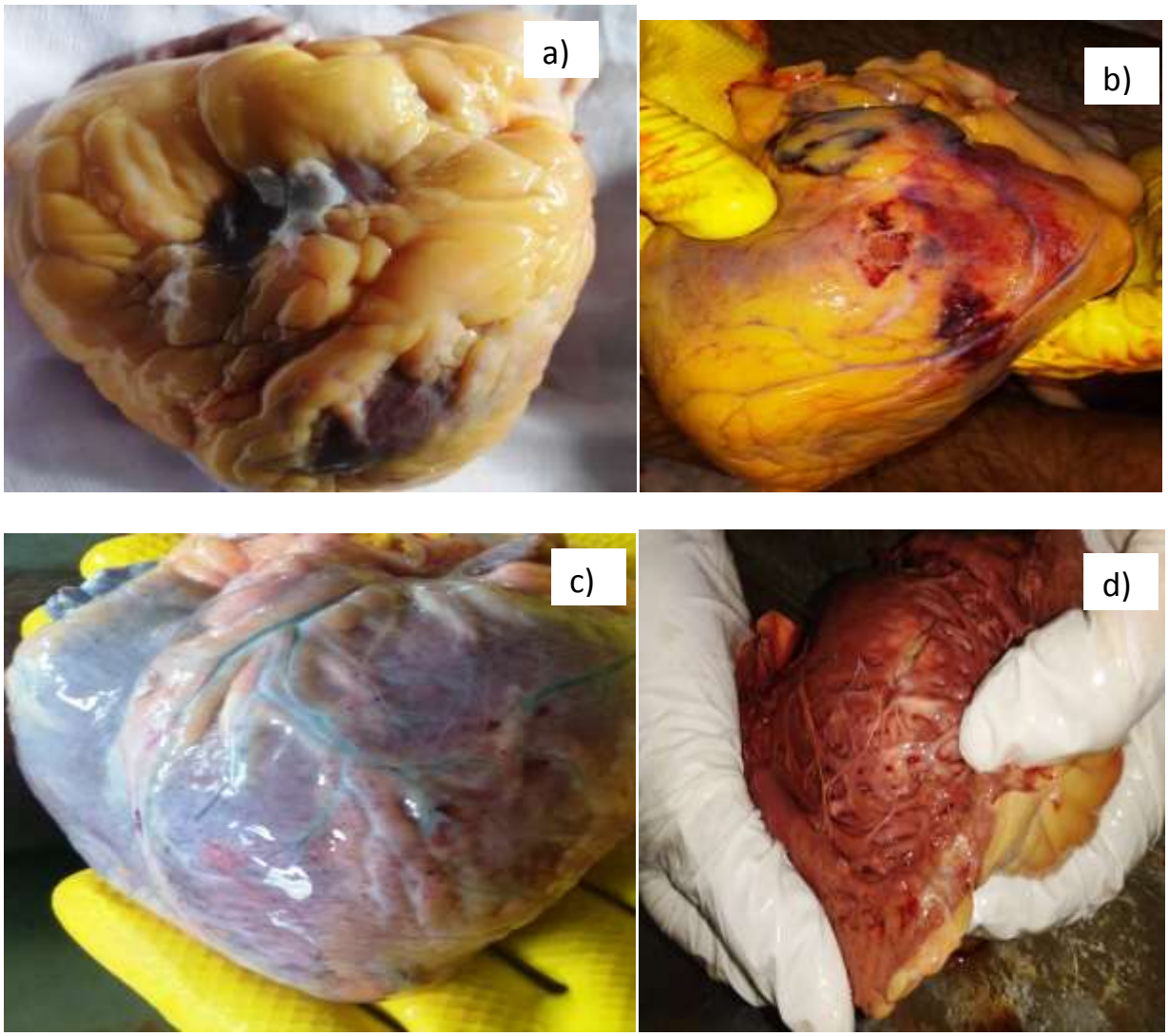


Figure 12: a)Whitish plaque (patchy pericarditis) over the surface of heart, b) haemorrhagic area over the surface of left ventricle, c)Multiple pinpoint hemorrhages over the surface of heart, d)pale area (old infarct) on cut section of heart.

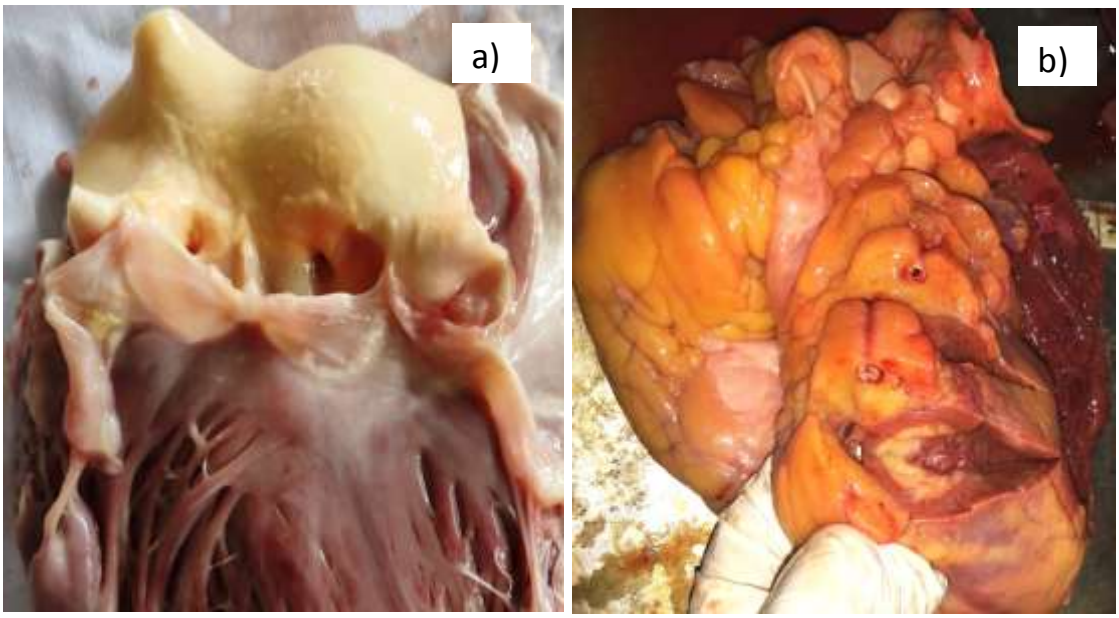


Figure 13: a)atheromatous plaque over the surface with thickened aortic valve, b) occluded left coronary artery.

Figure 14: Shows pale area (old infarct) on cut section of heart (fixed with formalin).

Gross examination findings	Number of cases
No Remarkable findings	26
Hemorrhagic area over the left ventricle of heart	8
White colour plaques over the surface of heart	6
Hemorrhagic area and White colour plaques over the surface of heart	6
Pinpoint hemorrhages	3
Occlusion of coronaries and evidence of healed myocardial infarction	1
Enlarged	1
Thickened aorta	1
Total	52

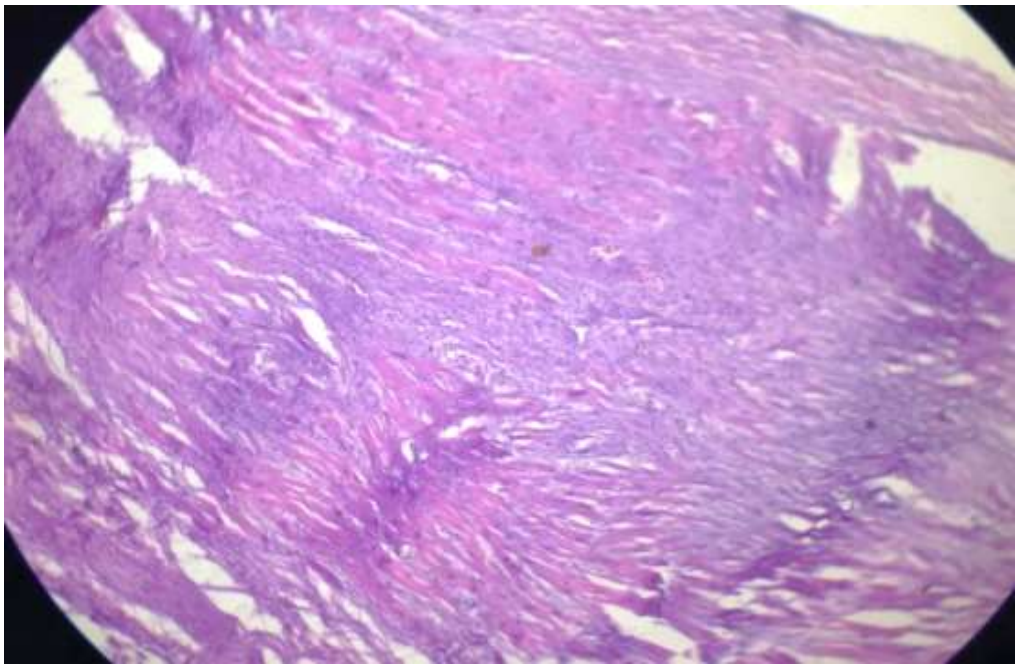
Table 9: Shows findings noted during autopsy findings of heart in sudden cardiac deaths.

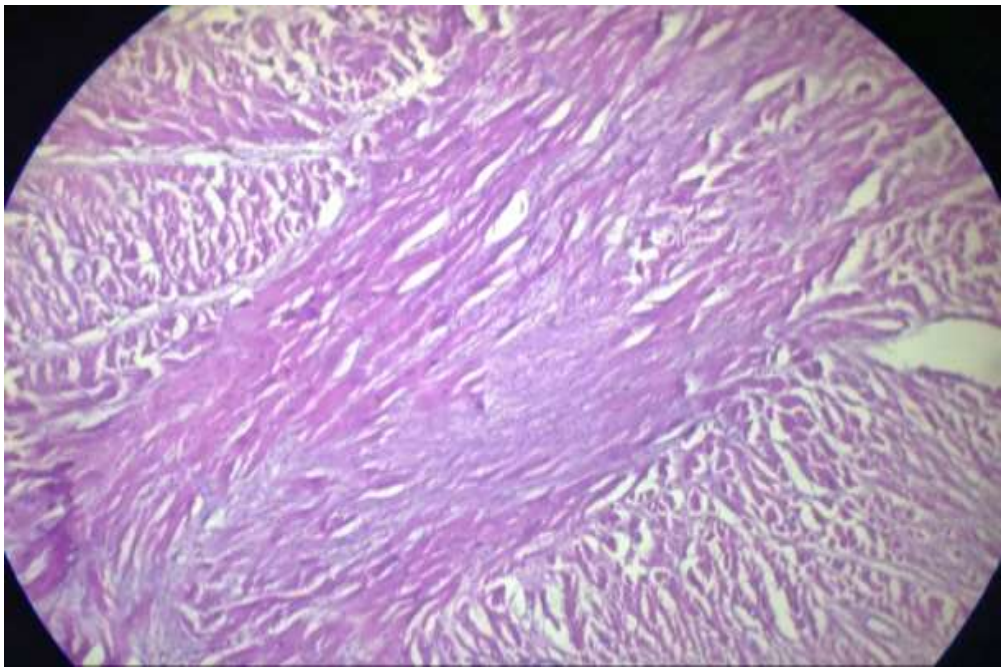
Histopathological examination of heart shows features of acute myocardial infarction in 3 cases, healed myocardial infarction in 3 cases, evidence of both acute and healed myocardial infarction in one case, whereas no remarkable changes in 39 heart specimens. There were none of the heart specimens shown features of bundles of hyercontracted myocytes with squaring of nuclei, hypercontracted myocytes alternating with hyperdistended myocytes and separation of sarcomeres.

Histopathology examination findings	**Number of cases**
No remarkable findings	39
Hypertrophy of myocardium with myofibre disarray	4
Healed myocardial infarction	3
Acute myocardial infarction	3
Cysticercosis	1
Acute and healed myocardial infarction	1
Aortitis	1
Total	52

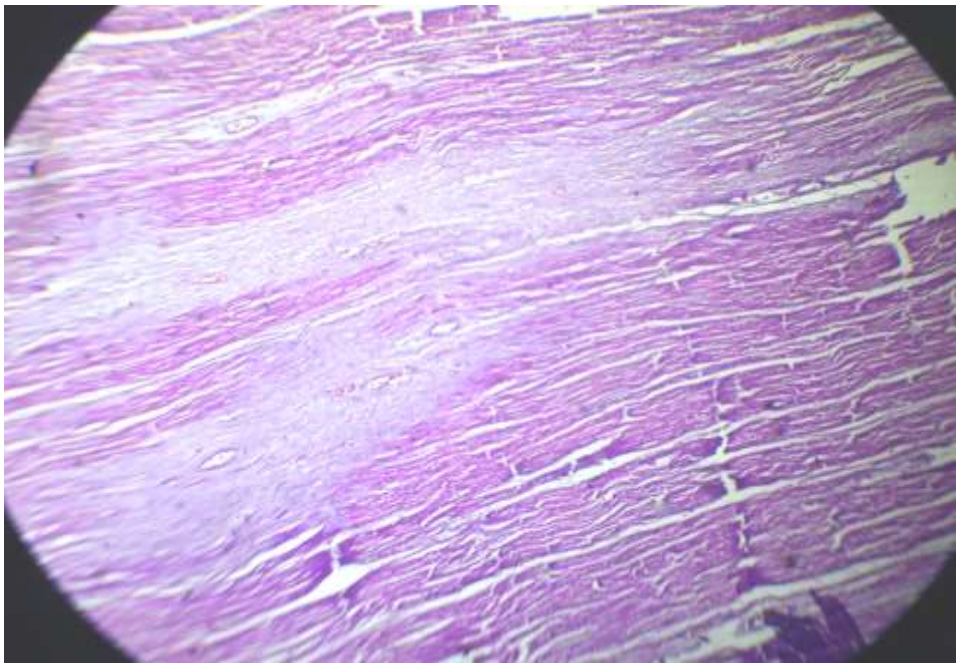
Table 10: Shows histopathological findings of heart in sudden cardiac death.

COLOUR PLATES





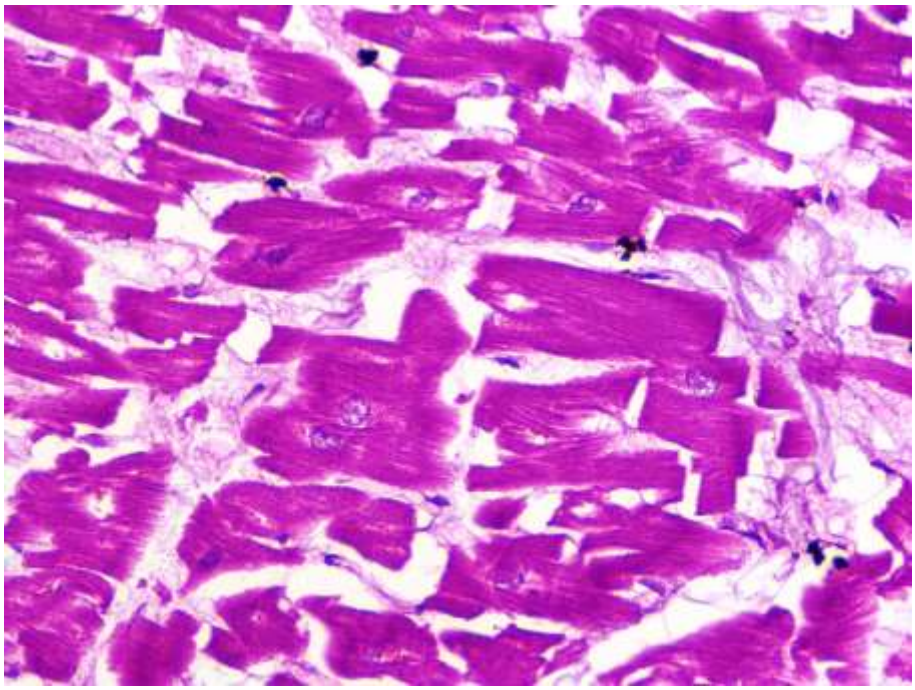
COLOUR PLATES 6a,b: showing ischemic changes in acute myocardial infarction noted in sudden cardiac death.



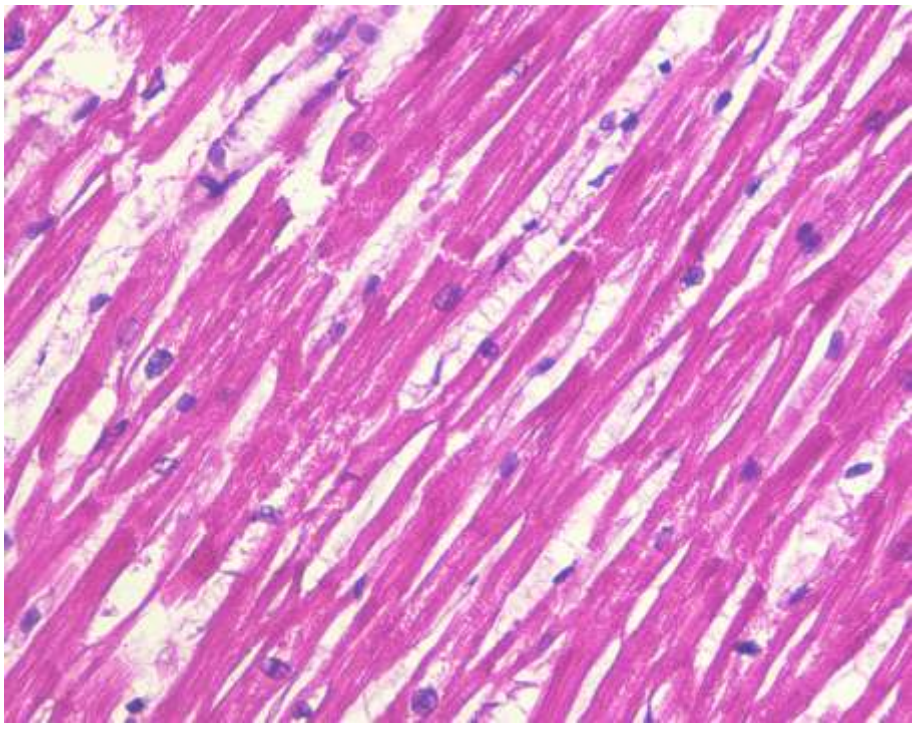
COLOUR PLATE 7: Showing evidence of fibrosis in old healed myocardial infarction.

S. No	HistopathologIcal findings of heart	Electrocution		Sudden Cardiac Death	
		Frequency	Percentage	Frequency	Percentage
1.	Bundles of distended myocardial cells alternating with Hypercontracted cells and Hypercontracted cells showing square expression of nucleus	51	98.1%	0	0%
2.	Non eosinophilic bands of hypercontracted sarcomeres alternating with stretched, often apparently separated sarcomeres.	51	98.1%	0	0%
3.	Hypercontracted myocytes alternating with hyperdistended myocytes that are often divided by widened disc.	51	98.1%	0	0%
4.	Dilated and congested blood vessels	48	92.3%	0	0%
5.	Hemorrhage into the myocardium with extravasation of RBC's	36	69.2%	0	0%
6.	Old Healed myocardial infarct	4	7.6%	3	5.7%
7.	Disarray of myofibers	1	1.9%	4	7.6%
8.	Acute myocardial infarction	1	1.9%	3	5.7%

Table 11: Shows comparison of histopathological findings of heart in electrocution and sudden cardiac death



COLOUR PLATE 8: Displays myocardium in electrocution deaths showing Myofibre break up.



COLOUR PLATE 9: Shows myocardium in Sudden cardiac death.

DISCUSSION

DISCUSSION

Our prospective study was undertaken in the department of Forensic medicine, Coimbatore medical college, Coimbatore, to scrutinize the incidence and autopsy findings of heart in electrocution and sudden cardiac deaths. A total number of 6,346 medico legal cases were brought for postmortem examination during the study period, out of which 786 deaths were of natural diseases and 68 deaths were reported as electrocution. Out of 786 natural deaths, three-fourth of the deaths (675) was constituted by male victims followed by 109 female victims and 2 transgender victims. Out of 68 electrocution deaths received, 58 victims were male and 10 were female. We elected 52 electrocution deaths and 52 sudden cardiac deaths which fulfilled our criteria.

In our study we noticed higher incidence of electrocution deaths among males as compared to females. This result is similar to the studies conducted by Shaha and Joe[39] et al, Gururaj B[40] et al, Reddy[41] et al, B. Mukherjee[42] et al, B.D. Gupta[43] et al, Giri[44] et al, Ragui[45] et al, S. Kumar[46] et al, Pathak[47] et al, Regula wick[48] et al, S von caues[49] et al, Y. Tirasi[50] et al and Massey[51] et al.

There is also higher number of electrocution deaths in the age group of 31-40 years and mean age is 34.27 in our study. The studies done by S. Kumar[46] et al, Reddy[41] et al and Pathak[47] et al., were consistent with our study. However the studies done by Shaha and Joe[39] et al, Gururaj B[40] et al, Giri[44] et al, Ragui[45] et al and S von caues[49] et al., noted higher incidence of electrocution deaths among 21-30 years of age group. On the contrary, B. Mukherjee[42] et al documented higher incidence of electrocution deaths in 11-30 years of age.

The manner of death in majority of electrocution deaths was accidental and a few were suicidal in nature. Among accidental deaths half of them were happened at work place, which implicates the necessity of safety measures at work. There is no case was reported to be homicidal. Our results were consistent with the results of S. Kumar[46] et al, S von caues[49] et al, Reddy[41] et al, B. Mukherjee[42] et al, Pathak[47] et al, Giri[44] et al, Ragui[45] et al and Y. Tirasi[50]. There were 2 homicidal electrocution deaths reported by Regula wick[48] et al and one case by Chandrakant[52] et al. In contrast M.P. Jambure[53] et al, documented a case of homicide which was concealed as electrocution by the offender.

There were more cases of low voltage electrocution deaths recorded in our study. This result is similar to the studies of Regula wick[48] et al, S. Kumar[46] et al and S von caues[49] et al. On the contrary Ragui[45] et al noted more high tension electrocution deaths in his region.

Analysis of sudden cardiac death in young adults (≤40 years) exhibited higher frequency of sudden cardiac death among males compared to females. This indicates early onset of atherosclerosis among males as compared to females. Our results are coinciding with the studies of Vartjees[54] et al and Eckarts[55] et al who have conducted study in young adults. Vartjees[54] et al and Eckarts[55] et al noticed sudden cardiac death incidence increasing over age [28,33,36], on contrary we had increasing incidence among 21-30 years and mean age is 26.88.

During autopsy gross examination of the heart, multiple pinpoint hemorrhages over the surface were invariably noted in 90.4% cases of electrocution. On the contrary, the majority of the heart specimen from sudden cardiac death displayed no remarkable changes, 26.9% shown features of myocardial infarction and 5.9% of cases had multiple pinpoint hemorrhages. Based on our data, we hypothesize that these pinpoint hemorrhages are produced in response to passage of electrical current through the heart during electrocution resulting in acute spasm of coronary vessels followed by irreversible dilatation and tetanic contraction of the muscles results in Myofibre Breakup.

Whilst histopathological examination of heart from electrocution deaths (98.1%) depicted myofibre breakup. i.e (1) bundles of distended myocardial cells alternating with hypercontracted cells. In the latter group of cells there is also widening or rupture (segmentation) of intercalated discs. Myocardial nuclei in the hypercontracted cells have a “square” aspect rather than the ovoid morphology seen in distended myocytes. (2) hypercontracted myocytes alternated with hyper distended cells that are often divided by widened disc, (3) non-eosinophilic bands of hyper-contracted sarcomeres alternating with stretched, often apparently separated sarcomeres[23,31]. Our study portrays that myofiber break up as a significant and characteristic finding of electrocution. Our results are consistent with the findings of Vittorio Fineschi[31] et al, Shubha. H V and Nirmala. C[32] et al, B. Viswakanth[33] et al, Ghandour[36] et al, Badawy[37] et al, Ku[35] et al and Xenopulos[34] et al. and contrary to the study performed by

G. Baroldi[23] et al, who documented myofibre breakup as a common finding in sudden cardiac deaths. G. Baroldi[23] et al had documented that he noticed higher incidence of Myofibre breakup in deaths occurring in patients who were on ventilator and who had resuscitative measures. To avoid bias, we excluded the cases which were undergone resuscitation by means of defibrillator and admission to hospital. For further scrutiny, we inspected the specimens from sudden cardiac death for similar findings, but none of the cases showed evidence of myofibre break up.

Acute myocardial infarction and healed myocardial infarct were very minimal among electrocution deaths;

On the other hand Acute myocardial infarction and healed myocardial infarct constituted 13.5% of sudden cardiac deaths. The autopsy and histopathological examination of more than 50% of the heart specimens from sudden cardiac death revealed no remarkable changes. Similar results were documented by Vartjees[54] et al and Eckarts[55] et al. This could be due to death occurring within 12 hours of onset of myocardial infarction or physiological dysfunctions of heart, which are difficult to detect during Autopsy and Histopathology.

SUMMARY & CONCLUSION

SUMMARY AND CONCLUSION

This prospective study of an autopsy based examination heart findings in electrocution and Sudden cardiac death was done during the period of January 2018 to June 2019 from the medicolegal autopsies conducted at the Department of Forensic Medcine, Coimbatore Medical college Hospital, Coimbatore.

Out of the 786 natural deaths and 68 electrocution death cases, 52 Sudden cardiac deaths and 52 electrocution deaths were elected based on selection criteria respectively. The hearts were dissected and sent for histopathological examination. Based on autopsy and histopathological data the following conclusions were drawn.

- There was higher incidence of electrocution death among males as compared to females. The commonest age group involved was 31-40 years and mean age is 34.27.
- There was higher incidence of sudden cardiac death among males below 40 years of age. The commonest age group involved was 21-30 years and mean age is 26.88. This indicates early onset of atherosclerosis among males and the need for earlier identification of risk factors.
- Out of 52 heart specimens from electrocution deaths, 51 specimens shown evidence of pinpoint hemorrhages at autopsy and Myofibre break up in histopathology. The myofibre breakup present in histopathology of the heart specimens in electrocution, precisely gives clues regarding cause of death and unique to the case. The findings noticed in our study

are consistent and can be relied upon as an ancillary investigation for electrocution deaths even in the absence of electrical marks.

- Out of 52 sudden cardiac death heart specimens, there were no remarkable findings in 26 cases at autopsy and 39 cases at histopathology. Out of 52 sudden cardiac death cases 3 cases showed evidence of acute myocardial infarction, 3 cases showed old infarct and one showed evidence of acute and healed infarct. This could be due to death occurring within 12 hours of onset of myocardial infarction or physiological dysfunctions of heart, which are difficult to detect during Autopsy and Histopathology.
- There were no correlations established between age, gender, coronary atherosclerosis and myofibre breakup of electrocution and Sudden cardiac deaths.
- Based on the incidence of sudden cardiac death among young adults, we recommend regular cardiac health checkups and awareness initiatives for early detection of risk factors. This also necessitates the need for research in the forensic field regarding genetic studies and invention of novel ancillary investigations for detection of physiological dysfunction of heart.
- We also recommend further systematic studies for the betterment.

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ANNEXURES

ANNEXURE I
MASTER CHART
ELECTROCUTION DEATHS

S. no	Pm.no	Age	Sex	Time of occurance	Source of electricity	Autopsy findings							Histopathology findings					
						Point of entry	Point of exit	Hypothesized pathway of current			other findings	Chemical analysis	Pinpoint hemorrhages over the surface of heart	Bundles of distended myocardial cells alternating with Hypercontracted cells and Hypercontracted cells showing square expression of nucleus	Hypercontracted myocytes alternating with hyperdistended myocytes	Non eosinophilic bands of hypercontracted sarcomeres alternating with stretched, often apparently separated	Dilated and congested blood vessels	Hemorrhage into the myocardium with extravasation of RBC's	Additional findings
1	215/18	31	female	Day	Electric belt	Present	Present	Point of entry	Point of exit	Probable Organ involved			Present	Present	Present	Present	Present		
								Centre ofChest	Right foot	Heart									
2	416/18	52	female	Day	Water heater	Present	Present	Right arm	Left arm	Heart	Charring of arm	Negative	Present	Present	Present	Present	Present	Present	
3	521/18	13	female	Day	Water heater	Present						Negative	Present	Present	Present	Present	Present	Present	
4	829/18	8	female	Night	Water heater	Present					Dermo-epidermal burn injuries	Negative			Present		Present	Present	
5	863/18	11	male	Night	Lamp post	Present						Negative	Present	Present	Present	Present	Present	Present	
6	1012/18	44	male	Day	Electric wire	Present							Present	Present	Present	Present	Present	Present	
7	1211/18	21	male	Day	Electronic Battery	Present	Present	Right arm	Left arm	Heart			Present	Present	Present	Present	Present	Present	
8	1236/18	39	male	Day	High voltage transformer	Present	Present				Dermo-epidermal burn injuries		Present	Present	Present	Present	Present	Present	
9	1297/18	18	male	Night	Switch box	Present							Present	Present	Present	Present	Present	Present	
10	1348/18	14	female	Day	Water heater	Present	Present	Face	Right arm	Brain		Negative	Present	Present	Present	Present	Present	Present	
11	1391/18	63	male	Day	Cable wire	Present							Present	Present	Present	Present	Present	Present	Healed myocardial infarct
12	1392/18	58	female	Day	Cable wire	Present	Present	Right arm	Left arm	Heart			Present	Present	Present	Present	Present	Present	Myocyte disarray
13	1407/18	16	female	Night	Electric Fence	Present	Present	Right arm	Left arm	Heart		Negative	Present	Present	Present	Present	Present	Present	
14	1427/18	26	male	Day	Electric wire	Present					Dermo-epidermal burn injuries		Present	Present	Present	Present	Present		
15	1449/18	32	male	Day	Electric wire	Present						Negative	Present	Present	Present	Present	Present	Present	
16	1470/18	31	male	Day	Switch box	Present						Negative	Present	Present	Present	Present	Present		
17	1492/18	43	male	Night	Water heater	Present	Present	Face	Right arm	Brain		Negative		Present	Present	Present	Present	Present	
18	1508/18	33	male	Day	High voltage transformer	Present							Present	Present	Present	Present	Present	Present	
19	1636/18	22	female	Day	Machine used at work place	Present							Present	Present	Present	Present	Present	Present	
20	1731/18	38	male	Day	Switch box	Present							Present	Present	Present	Present		Present	
21	1735/18	90	male	Day	High voltage transformer	Present	Present				Dermo-epidermal burn injuries		Present	Present	Present	Present	Present		
22	1972/18	19	male	Day	Electric wire	Present							Present	Present	Present	Present	Present		
23	2062/18	47	male	Day	Electric wire	Present	Present	Right arm	Left arm	Heart		Negative	Present	Present	Present	Present	Present	Present	Healed myocardial infarct
24	2129/18	60	male	Night	Switch box	Present	Present	Left arm	Right foot	Heart			Present	Present	Present	Present	Present		

ANNEXURE I
MASTER CHART
ELECTROCUTION DEATHS

25	2386/18	37	male	Day	High voltage transformer	Present	Present	Left arm	Right foot	Heart			Present	Present	Present	Present	Present	Present	
26	2418/18	27	male	Day	Machine used at work place	Present							Present	Present	Present	Present	Present	Present	
27	2481/18	35	male	Day	Machine used at work place	Present	Present	Right arm	Left arm	Heart		Negative	Present	Present	Present	Present	Present	Present	
28	2513/18	40	male	Day	Electric belt	Present	Present					Negative	Present	Present		Present	Present	Present	
29	2589/18	46	male	Day	Machine used at work place	Present						Negative	Present	Present	Present	Present	Present		
30	2669/18	20	male	Day	High voltage transformer	Present	Present				Dermo-epidermal burn injuries	Negative		Present	Present	Present		Present	
31	2740/18	18	male	Day	High voltage transformer	Present	Present				Dermo-epidermal burn injuries	Negative		Present	Present	Present	Present	Present	
32	2821/18	21	male	Day	Water heater	Present							Present	Present	Present	Present	Present	Present	
33	2857/18	30	male	Day	Electric wire	Present	Present	Left arm	Right foot	Heart		Negative	Present	Present	Present	Present	Present		
34	3015/18	47	male	Day	Cable wire	Present						Negative	Present	Present	Present	Present	Present	Present	
35	3053/18	63	male	Day	Lamp post	Present							Present	Present	Present	Present	Present	Present	
36	3076/18	19	male	Night	Switch box	Present						Negative	Present	Present	Present	Present	Present		
37	3115/18	14	male	Day	Electric Fence	Present	Present	Left arm	Right foot	Heart		Negative	Present	Present	Present	Present	Present		
38	3194/18	27	male	Night	Electric Fence	Present						Negative	Present	Present	Present	Present	Present	Present	
39	3237/18	24	male	Day	Electric post	Present	Present	Centre ofChest	Left arm	Heart	Charring of entire body		Present	Present	Present	Present	Present	Present	
40	3295/18	35	male	Day	Electric post	Present					Multiple injuries		Present	Present	Present	Present	Present	Present	
41	3376/18	38	male	Day	Electric wire	Present							Present	Present	Present	Present	Present		Healed myocardial infarct
42	3381/18	65	male	Day	Electric wire	Present							Present	Present	Present	Present	Present	Present	
43	3469/18	38	male	Day	Electric wire	Present					Cranio cerebral injuries		Present	Present	Present	Present	Present	Present	
44	3512/18	35	male	Day	Machine used at work place	Present	Present			Heart		Negative		Present	Present		Present	Present	
45	3556/18	17	male	Day	Electric water Pump	Present							Present	Present	Present	Present	Present		
46	3643/18	38	male	Day	Switch box	Present						Negative	Present	Present	Present	Present	Present		
47	3723/18	39	male	Day	Electric wire	Present							Present	Present	Present	Present		Present	healed myocardial infarct and Acute myocardial infarct
48	4010/18	28	male	Night	High voltage transformer	Present	Present				Charring of entire body		Present	Present	Present	Present	Present	Present	
49	288/19	41	male	Day	Electric post	Present	Present	left arm	Right foot	Heart	Cranio cerebral injuries	Negative	Present	Present	Present	Present		Present	
50	444/19	56	male	Day	Electric wire	Present	Present			Heart		Negative	Present	Present	Present	Present	Present		Healed myocardial infarct
51	837/19	22	male	Day	High voltage transformer	Present	Present				Dermo-epidermal burn injuries		Present	Present	Present	Present	Present	Present	
52	1340/19	33	male	Day	Electric Fence	Present						Negative	Present	Present	Present	Present	Present		

SUDDEN CARDIAC DEATHS

S.no	Pm. No	Age	Sex	Symptoms/ Complaints before death	Autopy and Histopathology Findings	
					Gross examination of heart	Histopathology of heart
1	256/18	18	male	Chest pain	Reddish Haemorrhagic area over the surface of heart	No remarkable findings
2	381/18	27	male	Sudden loss of consciousness	No remarkable findings	No remarkable findings
3	413/18	22	male	Breathlessness	No remarkable findings	Cysti cercosis
4	520/18	32	male	Sudden loss of consciousness	Whitish plaque noted over the surface of heart- Reddish Haemorrhagic area over the surface of heart	No remarkable findings
5	533/18	31	male	Chest pain	No remarkable findings	No remarkable findings
6	690/18	25	male	Sudden loss of consciousness	No remarkable findings	No remarkable findings
7	819/18	27	male	Chest pain	Reddish Haemorrhagic area over the surface of heart	Healed myocardial infarction
8	869/18	32	male	Didn't wake up from sleep	Whitish plaque noted over the surface of heart- Reddish Haemorrhagic area over the surface of heart	No remarkable findings
9	862/18	35	male	Didn't wake up from sleep	No remarkable findings	No remarkable findings
10	995/18	31	male	Didn't wake up from sleep	No remarkable findings	No remarkable findings
11	1102/18	19	female	Stomach pain	No remarkable findings	No remarkable findings
12	1189/18	24	male	Stomach pain	No remarkable findings	No remarkable findings
13	1215/18	31	male	Chest pain	Occlusion of coronaries and evidence of healed myocardial infarction	Acute and Healed myocardial infarction
14	1216/18	29	male	Sudden loss of consciousness	Reddish Haemorrhagic area over the surface of heart	No remarkable findings
15	1250/18	22	male	Stomach pain	Whitish plaque noted over the surface of heart	No remarkable findings
16	1270/18	35	male	Sudden loss of consciousness	Whitish plaque noted over the surface of heart	No remarkable findings
17	1291/18	33	male	No cmplaints/ symptoms	No remarkable findings	No remarkable findings
18	1335/18	30	male	No cmplaints/ symptoms	No remarkable findings	No remarkable findings
19	1910/18	21	female	Didn't wake up from sleep	No remarkable findings	No remarkable findings
20	1914/18	19	female	Breathlessness	No remarkable findings	No remarkable findings
21	1952/18	27	male	No cmplaints/ symptoms	Pinpoint hemorrhages over the surface of heart	No remarkable findings
22	2039/18	32	female	Sudden loss of consciousness	Whitish plaque noted over the surface of heart	Hypertrophy of myocardium with myofibre disarray
23	2103/18	9	male	Didn't wake up from sleep	Reddish Haemorrhagic area over the surface of heart	No remarkable findings
24	2203/18	21	male	Sudden loss of consciousness	No remarkable findings	No remarkable findings
25	2364/18	35	female	Sudden loss of consciousness	No remarkable findings	No remarkable findings
26	2635/18	26	male	Sudden loss of consciousness	No remarkable findings	No remarkable findings
27	2648/18	33	male	Sudden loss of consciousness	Reddish Haemorrhagic area over the surface of heart	No remarkable findings
28	2749/18	30	male	No cmplaints/ symptoms	No remarkable findings	No remarkable findings
29	2764/18	35	male	Chest pain	No remarkable findings	Healed myocardial infarction
30	2861/18	35	male	Breathlessness	Reddish Haemorrhagic area over the surface of heart	No remarkable findings
31	2863/18	21	male	Chest pain	Whitish plaque noted over the surface of heart	Acute myocardial infarction

SUDDEN CARDIAC DEATHS

32	2929/18	31	male	Sudden loss of consciousness	No remarkable findings	No remarkable findings
33	2952/18	32	female	Breathlessness	No remarkable findings	No remarkable findings
34	2991/18	26	male	Didn't wake up from sleep	Reddish Haemorrhagic area over the surface of heart	No remarkable findings
35	3010/18	32	male	Sudden loss of consciousness	Pinpoint hemorrhages over the surface of heart	No remarkable findings
36	3024/18	29	male	Chest pain	No remarkable findings	No remarkable findings
37	3046/18	19	male	Chest pain	No remarkable findings	Hypertrophy of myocardium with myofibre disarray
38	3097/18	29	male	No cmplaints/ symptoms	No remarkable findings	No remarkable findings
39	3137/18	22	male	Sudden loss of consciousness	No remarkable findings	Hypertrophy of myocardium with myofibre disarray
40	3146/18	28	male	Chest pain	No remarkable findings	Acute myocardial infarction
41	3154/18	24	male	Sudden loss of consciousness	No remarkable findings	No remarkable findings
42	3176/18	28	male	Chest pain	Whitish plaque noted over the surface of heart	No remarkable findings
43	3188/18	31	male	Sudden loss of consciousness	Whitish plaque noted over the surface of heart- Reddish Haemorrhagic area over the surface of heart	No remarkable findings
44	3246/18	21	male	Didn't wake up from sleep	Whitish plaque noted over the surface of heart- Reddish Haemorrhagic area over the surface of heart	No remarkable findings
45	3360/18	27	male	Breathlessness	Enlarged	Hypertrophy of myocardium with myofibre disarray
46	3694/18	35	male	No cmplaints/ symptoms	Pinpoint hemorrhages over the surface of heart	No remarkable findings
47	3701/18	23	male	Didn't wake up from sleep	Whitish plaque noted over the surface of heart- Reddish Haemorrhagic area over the surface of heart	No remarkable findings
48	3744/18	21	female	Sudden loss of consciousness	Thickened aorta	Aortitis
49	3813/18	25	male	Seizures	Reddish Haemorrhagic area over the surface of heart	Healed myocardial infarction
50	3909/18	30	male	Chest pain	Whitish plaque noted over the surface of heart	Acute myocardial infarction
51	3917/18	14	male	Stomach pain	No remarkable findings	No remarkable findings
52	3972/18	24	male	Sudden loss of consciousness	Whitish plaque noted over the surface of heart- Reddish Haemorrhagic area over the surface of heart	No remarkable findings

ANNEXURE II

LIST OF ABBRIVIATIONS

A/C	:	ALTERNATING CURRENT
D/C	:	DIRECT CURRENT
MFB	:	MYOFIBER BREAKUP
SCD	:	SUDDEN CARDIAC DEATH
SA NODE	:	SINOATRIAL NODE
AV	:	ATRIOVENTRICULAR
Da	:	DALTONS
ATP	:	ADENOSINE TRIPHOSPHATE
Ca^{2+}	:	CALCIUM
Na^{+}	:	SODIUM
K^{+}	:	POTTASSIUM
PKA	:	PROTEIN KINASE A
AMP	:	ADENOSINE MONOPHOSPHATE
mA	:	MILLI AMPERE
nm	:	NANOMETER
V	:	VOLTAGE
cps	:	CYCLES PER SECOND

A	:	AMPERE
VF	:	VENTRICULAR FIBRILLATION
VT	:	VENTRICULAR TACHYCARDIA
CAD	:	CORONARY ARTERY DISEASE
MI	:	MYOCARDIAL INFARCTION
LAD	:	LEFT ANTERIOR DESCENDING
LCX	:	LEFT CIRCUMFLEX
RCA	:	RIGHT CORONARY ARTERY
LDH	:	LACTATE DEHYDROGENASE
DCM	:	DILATED CARDIOMYOPATHY
HCM	:	HYPERTROPHIC CARDIOMYOPATHY
ARVD	:	ARRHYTHMOGENIC RIGHT VENTRICULAR DYSPLASIA
LQTS	:	LONG QT SYNDROME
RBBB	:	RIGHT BUNDLE BRANCH BLOCK
WPW	:	WOLF-PARKINSON-WHITE
ECG	:	ELECTROCARDIOGRAPH
AIDS	:	ACQUIRED IMMUNO DEFICIENCY SYNDROME

IVC	:	INFERIOR VENA CAVA
SVC	:	SUPERIOR VENA CAVA
HOCM	:	HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY