**A DISSERTATION ON**

# CARDIAC CONDUCTION ABNORMALITIES AND ASYMPTOMATIC MYOCARDIAL INFARCTION IN TYPE II DIABETES MELLITUS PATIENTS

*Dissertation submitted to*

**THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY**

*In partial fulfillment of the regulations*
*for the award of the degree of*

## M.D. BRANCH - I
## GENERAL MEDICINE


**GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL**
**THE TAMIL NADU DR.M.G.R. MEDICAL UNIVERSITY**
**CHENNAI, INDIA**

**MARCH 2008**

# CERTIFICATE

This is to certify that the dissertation titled **"CARDIAC CONDUCTION ABNORMALITIES AND ASYMPTOMATIC MYOCARDIAL INFARCTION IN TYPE II DIBETES MELLITUS PATIENTS**" is the bonafide original work of **DR. C.P. RAJESH,** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in MARCH 2008. The Period of study was from 2006 to January 2007.

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# DECLARATION

I, **DR. C.P. RAJESH,** solemnly declare that dissertation titled **"CARDIAC CONDUCTION ABNORMALITIES AND ASYMPTOMATIC MYOCARDIAL INFARCTION IN TYPE II DIBETES MELLITUS PATIENTS**" is a bonafide record of work done by me in the Department of Internal Medicine, Government Stanley Medical College and Hospital during 2006 to January 2007 under the guidance of **Prof. K.RAJENDRAN, M.D.,** Professor of Medicine, Government Stanley Medical College and Hospital, Chennai.

This dissertation is submitted to Tamilnadu DR. M.G.R Medical University, in partial fulfillment of the University regulations for the award of **M.D. Degree (Branch – I) in General Medicine – March 2008.**

Place : Chennai.

Date :

**(DR. C.P. RAJESH)**

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## Contents

# INTRODUCTION

Diabetes Mellitus is not a single disease with a single cause. It is a multimetabolic disorder with wide variable spectrum of clinical features, encompassing different number of illnesses that share the single phenotype of hyperglycemia.

Ubiquitous in distribution, it is a complex interaction of genetics, environmental factors and life style.

The prevalence of diabetes mellitus in India is high and the case burden is rising every year.

Diabetes affects all the organs of the body; these contribute to a very large extent to the morbidity and mortality of the disease. Hence the search for and the characterization of the abnormalities assume utmost importance.

The involvement of cardiovascular system in Diabetes Mellitus is varied. It can be involved by microvascular and macrovascular pathology. Ischaemic heart disease, hypertensive heart disease and diabetic cardiomyopathy are the ways in which cardiac tissue is usually affected. The possible mechanism being

Dyslipidemia, enhanced atherosclerosis, hypertension, diabetic macroangiopathy and metabolic alterations associated with diabetes. Adults with Diabetes have two to four times risk of developing heart diseases. There are numerous studies, which have highlighted these disorders.

There are reports showing higher incidence of involvement of conducting tissue of heart in diabetes mellitus. There is a higher prevalence of right bundle branch block and atrioventricular block that cannot be accounted by the increased incidence of ischaemic heart disease alone. Higher incidences of these blocks are seen independent of ischaemic heart disease.

Sinus node disorder in diabetes is mostly a manifestation of diabetic autonomic neuropathy. Early diabetic autonomic neuropathy is detected by estimating the heart rate variability. A depressed heart rate variability denotes a poor prognosis and often a marker for sudden cardiac death.

Population based studies regarding the involvement of conducting tissue of heart in diabetes mellitus is rare; especially in South India. This study is performed to estimate the prevalence of conduction blocks, i.e., atrioventricular, fascicular block, and asymptomatic myocardial infarction by electrocardiogram in diabetic population in Stanley Medical College and Hospital and compare it with that of general population.

# AIM OF STUDY

1. To study the prevalence of conduction blocks; namely atrio ventricular Nodal block, bundle branch blocks and fascicular blocks in electrocardiograms of asymptomatic Type II Diabetes Mellitus.

2. To compare this prevalence to that of age and sex matched asymptomatic control population.

3. To study the prevalence of electrocardiographic changes suggestive of myocardial infarction in asymptomatic Type II diabetes mellitus.

4. To compare this prevalence to that of controls.

# METHODOLOGY

This study was conducted in 150 Type II diabetes mellitus patients attending the Department of Internal Medicine, Government Stanley Medical College Hospital, Chennai and compared with 100 healthy age and sex matched controls.

## STUDY GROUPS

**Group I** - 150 patients with Type II Diabetes Mellitus.

**Group II** - 100 healthy controls.

These groups were selected on the basis of inclusion and exclusion criteria.

## INCLUSION CRITERIA

1. Type II Diabetes Mellitus
2. Healthy controls

## EXCLUSION CRITERIA

Patients with

1. Age above 50
2. Documented Ischaemic heart disease
3. History suggestive of previous angina, congestive cardiac failure.
4. Documented evidence of other cardiac disease like cardiomyopathy
5. Valvular heart disease
6. Congenital Heart Disease

7. Myocarditis
8. Alcoholism
9. Hypertension
10. Chronic obstructive pulmonary disease
11. Any abnormalities detected in the physical examination of cardiovascular system
12. Drugs – β blockers, Digoxin
13. Post menopausal females
14. Features of hypothyroidism
15. Random blood sugar > 140 mg/dL for the controls.
16. Anaemia < 10 gm/dL
17. Uremia

The cases and the control were subjected to thorough physical examination.

Blood was taken for estimation of blood sugar, serum cholesterol and renal function tests. A 12 lead ECG with rhythm strip was taken.

Diabetic population was taken from patients previously diagnosed and registered in Diabetology department. New cases were diagnosed by the revised criteria for diagnosis of diabetes mellitus. This is given in the review of literature.

## ESTIMATION OF CHOLESTEROL

### Method

***Enzymatic colorimetric test.***

Principle

This involves determining the cholesterol level by enzymatic hydrolysis and oxidation. Chinonimine is the colorimetric indicator. It is produced from 4 amino antipyrine and phenol by hydrogen peroxide under the catalytic action of peroxidase.

Cholesterol ester + $H_2O$ → Cholesterol + fatty acid

Cholesterol + $H_2O$ → Cholesterol 3 –one + $H_2O_2$

2 $H_2O_2$ + 4 amino antipyrine + phenol → chinonimine + 4 $H_2O_2$

ECG was taken in an Schiller Cardiovit AT machine. It is a 3 lead ECG. The speed of recording was adjusted to 25 mm/second. The ECG was recorded on normal standardization where an one millivolt will result in a 10 mm deflexion.

The criteria for diagnosing Atrioventricular block, bundle branch block and asymptomatic myocardial infarction is enumerated in the review of literature.

# REVIEW OF LITERATURE

Once regarded as a single disease entity, diabetes mellitus is now seen as a heterogeneous group of diseases, characterized by a state of chronic hyperglycemia, resulting from a diverse of etiologies, environmental and genetic, acting jointly.

## HISTORY

The first documented evidence of diabetes mellitus was reported in Egyptian papyrus, as a polyuric state.

In 1776 Mathew Dobson established the presence of sugar in blood and urine of diabetic patients.

## EPIDEMIOLOGY

Diabetes is a common global problem. It's prevalence is increasing and has reached epidemic proportion in developed as well as developing countries. Considerable geographic variation is seen, with most of the cases from USA, India, Europe.

Type II diabetes mellitus is more common than Insulin dependent diabetes mellitus. In Europe and North America, the ratio is 7:3 in favour of non-insulin

dependent diabetes mellitus. Type I Diabetes Mellitus is very uncommon in Chinese, Japanese and American Indians.

Diabetes is an iceberg disease, affecting atleast 300 million people through out the world. Its prevalence in most adult population is 2 – 5 percent.

By using fasting hyperglycemia as the diagnostic standard, the prevalence of diabetes in US is probably 1 – 3%. On the basis of response to a 75 g oral glucose tolerance test, the National Diabetes Data group of US estimated that 11.2% of US population as having impaired glucose tolerance.

There are an estimated 150 million Diabetes in the World, of which 25 – 50 million are in India. Epidemiological studies have established a prevalence of 2 – 3 percent in India.

A multicenter study done by India Council of Medical Research showed a prevalence rate of 1.73 percent in Indians above 15 yrs of age.

## CRITERIA FOR DIAGNOSIS

Revised criteria for diagnosing diabetes mellitus has been issued and taken from American Diabetes Association and the World Health Organization.

***The criteria is based on the premises that***

1. The spectrum of fasting plasma glucose (FPG) and the response to an oral glucose load varies in normal individuals.
2. Diabetes Mellitus defined as the level of glycemia at which diabetic specific complications are noted and not on the level of glucose tolerance from a population based viewpoint.

## CRITERIA

1. Symptoms of diabetes plus Random blood sugar > 11.1 mmol/L (200 mg/dl) or
2. Fasting plasma glucose (FPG) > 7 mmol/L (126 mg/dl)
3. 2 hr plasma glucose (FPG) > 11.1 mmol/L (200 mg/dl) during an oral glucose tolerance test.

In the absence of unequivocal hyperglycemia and acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day.

Fasting – No calorie intake for atleast 8 hrs.

## AETIOLOGY OF DIABETES MELLITUS

### Type I diabetes mellitus

***1. Genetics***

Genetic predisposition is possible permissive and not casual. Risk of diabetes is up to 5 times higher when the father is diabetic rather than mother. This risk is limited to father carrying an HLA DR4 susceptibility gene.

Risks for identical twin in 33%.

Genetic loci - Chromosome 6

***2. Viruses***

20% of persons with congenital rubella develop IDDM. Cytomegalovirus is present in genome of 20% patients. Others implicated are Coxsackie, Mumps and hepatitis.

***3. Diet***

Introduction of cow's milk before the age of 2 – 3 months is associated with an increased risk.

***4. Pancreatic pathology***

Insulitis

## TYPE II DIABETES MELLITUS

**Genetics : Probably polygenic**

- There is almost 100% concordance in monozygotic twins. Maturity onset diabetes may be associated with a mutation of glucokinase gene.

**Life style**

- Over eating; especially combined with obesity and under activity, leads to diabetes mellitus.

**Age**

- It is principally a disease of middle aged and elderly.

**Pregnancy**

- 80% of women with gestational diabetes develop permanent diabetes requiring treatment. Later in life

**Insulin resistance**

There is increased hepatic glucose production and resistance to the action of insulin in muscle.

## ETIOLOGIC CLASSIFICATION OF DIABETES MELLITUS

**1. Type 1 diabetes** (β-cell destruction, usually leading to absolute insulin deficiency)

A. Immune – mediated

B. Idiopathic

**2. Type 2 diabetes** (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)

**3. Other specific types of diabetes**

A. Genetic defects of β-cell function characterized by mutations in:

1. Hepatocyte nuclear transcription factor (HNF) 4 α (MODY 1)
2. Glucokinase (MODY 2)
3. HNF – 1 α (MODY 3)
4. Insulin promoter factor (IPF) 1 (MODY 4)

5. HNF – 1 β (MODY 5)
6. Mitochondrial DNA
7. Proinsulin or insulin conversion

B. Genetic defects in insulin action

1. Type A insulin resistance
2. Leprechaunism
3. Rabson – Mendenhall syndrome
4. Lipoartrophic diabetes

C. Disease of the exocrine pancreas – pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy.

D. Endocrinopathies – acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma.

E. Drug or chemical induced – Vacor, pentamidine, nicotinic acid, glucocorticoids, thyroid hormone, diazoxide, β-adrenergic agonists, thiazides, phenytoin, α-interferon, protease inhibitors, clozapine, and beta-blockers.

F. Infections – congenital rubella, cytomegalovirus, Coxsackie

G. Uncommon forms of immune mediated diabetes – "stiff-man" syndrome, anti-insulin receptor antibodies.

H. Other genetic syndromes sometimes associated with diabetes – Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's ataxia, Huntington chorea, Laurence – Moon –Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome.

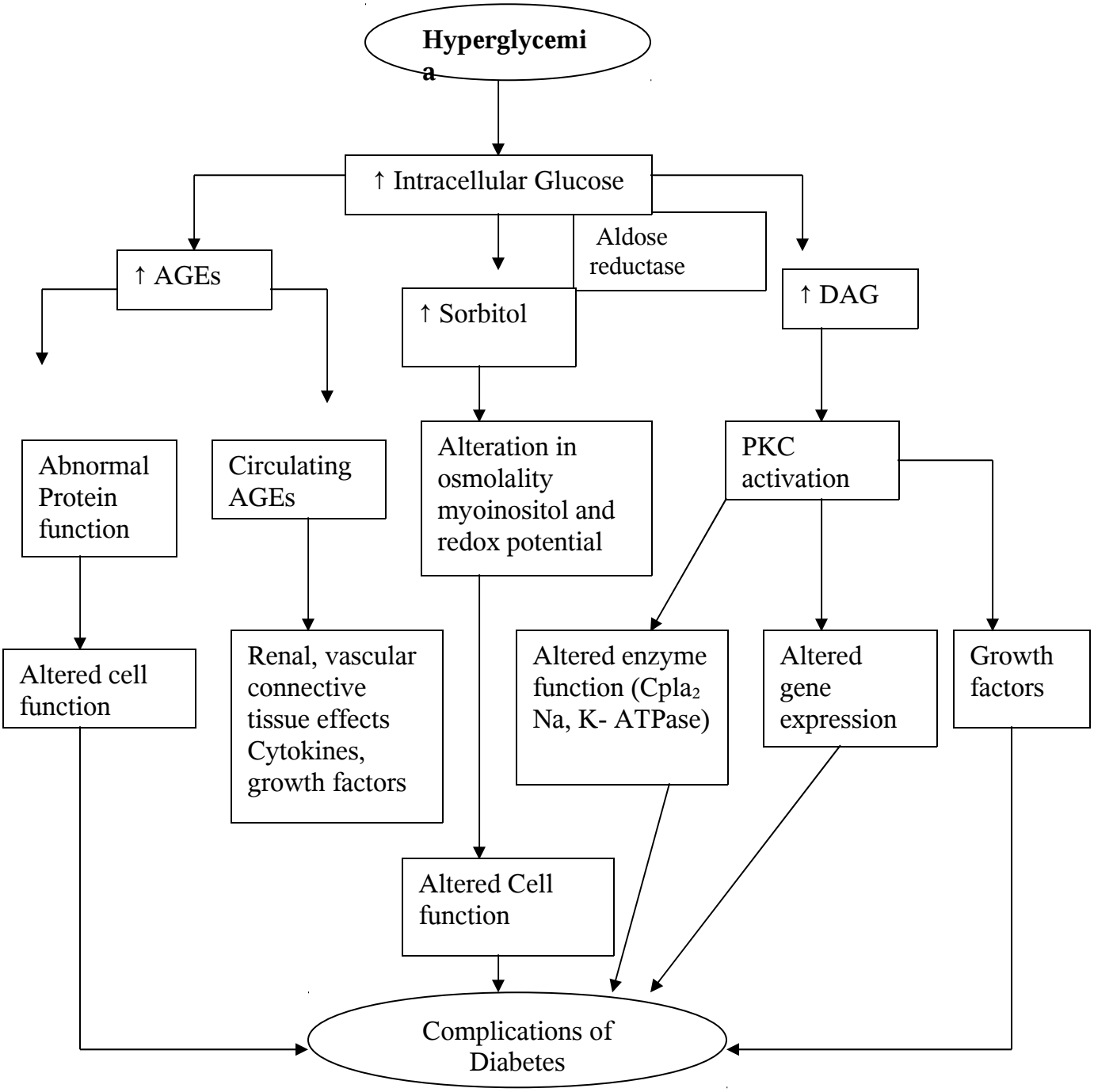
I. Gestational diabetes mellitus (GDM).

## RISK FACTORS FOR TYPE 2 DIABETES MELLITUS

- Family history of diabetes (i.e. parent or sibling with type 2 diabetes)
- Obesity (i.e. ≥ 20% desired body weight or BMI ≥ 27 kg/$m^2$)
- Age ≥ 45 years
- Race/ethnicity (e.g. African, American, Hispanic American, Native American, Asian American, Pacific Islander)
- Previously identified IFG or IGT
- History of GDM or delivery of baby over 9 pounds.
- Hypertension (blood pressure ≥ 140/90 mm Hg)
- HDL cholesterol level ≤ 0.90 mmol/L (35 mg/dL) and / or a triglyceride level ≥ 2.82 mmol/L (250 mg/dL)
- Polycystic ovary syndrome

| | | |
|---|---|---|
| BMI | = | Body Mass Index |
| IFG | = | Impaired Fasting Glucose |
| IGT | = | Impaired Glucose Tolerance |
| GDM | = | Gestational Diabetes Mellitus |
| HDL | = | High-density Lipoprotein |

## MOLECULAR MECHANISMS OF DIABETES RELATED COMPLICATIONS



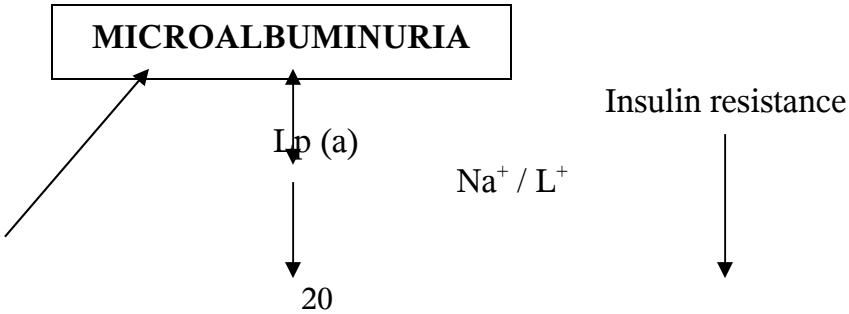

AGE = Advanced glycation end products
PKC = Protein kinase C
DAG = Diacylglycerol
$CPLA_2$ = Phospholipasae $A_2$
$Na^+$, $K^+$, ATPase = Sodium potassium ATPase

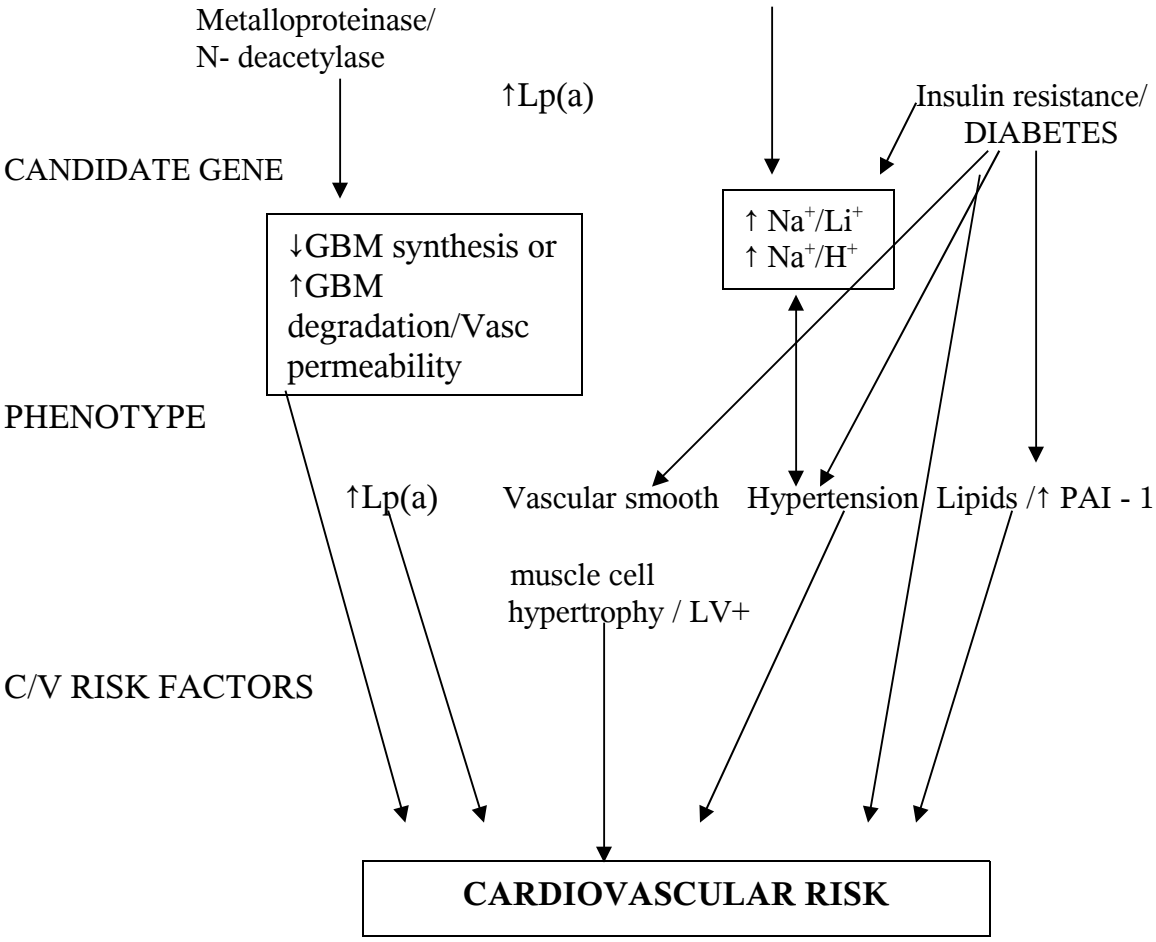
## CARDIAC INVOLVEMENT IN DIABETES MELLITUS

Heart has long been known to be involved in a number of ways by diabetes mellitus. These are:

1. Ischaemic Heart Disease
2. Hypertensive Heart Disease
3. Diabetic Cardiomyopathy
4. Conducting tissue involvement

## POSSIBLE GENETIC CONNECTIONS BETWEEN DIABETES, MICROALBUMINURIA AND CARDIOVASCULAR RISK






| | | |
|---|---|---|
| $Na^+ / Li^+$ | = | Sodium – lithium countertransport |
| $Na^+ / H^+$ | = | Sodium – hydrogen exchange |
| $LV^+$ | = | Left ventricular hypertrophy |
| GBM | = | Glomerular basement membrane |
| PAI | = | Plasminogen activator inhibitor |
| Lp | = | Lipoprotein |

## ISCHAEMIC HEART DISEASE

Ischaemia refers to a lack of oxygen due to inadequate perfusion which results from an imbalance between oxygen supply and demand. The most common cause of myocardial ischemia is atherosclerotic disease of epicardial coronary arteries.

***The spectrum of disease includes***

- Asymptomatic
- Stable angina pectoris
- Unstable angina
- Myocardial infarction
- Sudden cardiac death

***The risk factors are***

a) Non-modifiable risk factors

1. Age
2. Male sex
3. Family history of premature coronary artery disease
4. Angiotensin converting enzyme polymorphism

b) Modifiable risk factors

1. Hypertension
2. Diabetes Mellitus
3. Low HDL level
4. Cigarette smoking
5. Physical inactivity
6. Obesity

7. Post menopausal state
8. Hyperlipidemia
9. Hyperfibrinogenemia
10. Hyper homocystinemia

## PATHOPHYSIOLOGY OF ACUTE MYOCARDIAL INFARCTION

In most cases, infarction occurs when an atherosclerotic plaque fissures, ruptures or ulcerates and when conditions, local or systemic, favour thrombogenesis, so that a mural thrombus forms at the site of rupture and leads to coronary artery occlusion.

**Diagnosis**

***I. Symptoms***

- Chest pain – heaviness, squeezing or crushing type, associated with sweating, nausea, vomiting, occurs most frequently in the morning.
- Sudden onset breathlessness
- Profound weakness
- Confusional state
- Sudden loss of consciousness
- Evidence of peripheral embolism

- Arrhythmias
- Unexplained Hypotension

***II. Serum cardiac markers***

1. Creatine phosphokinase; especially MB isoenzyme
2. Cardiac specific Troponin T (cTnT)
3. Cardiac specific Troponin I (cTnI)
4. Myoglobin

## ELECTROCARDIOGRAM

It is very sensitive for detecting myocardial ischemia and infarction. Serial ECG's will show evolutionary changes in the majority of the patients.

### ELECTROCARDIOGRAPHIC LOCATION OF INFARCTION SITE

| Site | Leads |
|---|---|
| ➢ Inferior (diaphragmatic) | II, III $aV_F$ |
| ➢ Inferolateral | II, III, $aV_F$, $V_4$-$V_6$ |
| ➢ True posterior (Posterobasal) | $V_1$ (reciprocal changes) |
| ➢ Inferoposterior | II, III, $aV_F$, $V_1$ |
| ➢ Anteroseptal | $V_1$, $V_2$, $V_3$ |
| ➢ Anteroplateral | I, II, $aV_L$, $V_4$-$V_6$ |
| ➢ Extensive anterior | I, $aV_L$, $V_1$-$V_6$ |
| ➢ High anterolateral | I, $aV_L$ |
| ➢ Anterior (apical) | $V_4$-$V_6$, Reciprocal changes in $V_1$ |

| | | |
|---|---|---|
| ➢ | Posterolateral | $V_4R$ with $V_4R – V_6R$ |
| ➢ | Right ventricular | $V_1$-$V_3$ |

The evolution of a Q wave myocardial infarction can be separated into four phases; namely 1. hyperacute 2. Acute 3. subacute 4. Chronic stabilized

Changes in two contiguous leads is taken as significant.

**1. Hyper acute phase**

Usually within a few hours of onset

a. Increased ventricular activation time. It is usually delayed beyond 0.45 seconds.
b. Increased amplitude of R wave. The R wave becomes taller; especially in inferior wall infarction.
c. Slope elevation of ST segment

This is one of the earliest and most characteristic features of hyperacute phase. The segment loses its upward concavity, becomes straightened with an upward slope. Then the ST segment gets elevated.

**d. Tall and wide T wave**

T wave becomes very tall and wide; sometimes taller than the preceding R wave.

1. Points to note

**1. THE MONOPHASIC DEFLEXION** – The tall R wave, the slope elevated S-T segment and the tall and widened T wave may merge to form a single deflexion.

**2. PRINZEMETAL's** angina may have a similar picture to hyperacute MI

1. Reciprocal changes occur in leads facing un-injured surface.

**1. <u>Fully evolved phase (</u>**Acute and subacute)

***a) Evidence of myocardial necrosis.***

The basic principle in necrosis is that the QRS vectors are directed away from a necrotic or infracted myocardium. This result in (1) QS complex (2) a Qr complex (3) a loss of R wave amplitude.

***b) Evidence of myocardial injury.***

The ST segment deviates towards the surface of injured tissue. Since most myocardial infarction are dominantly epicardial with some endocardial sparing, it manifest as elevated S-T segment in the leads oriented to the surface. In addition it becomes coved or convex upwards. An ST elevation of > 1 mm in limb leads and > 2 mm in chest leads are needed for the diagnosis.

***c) Evidence of myocardial ischemia***

The T wave vector is directed away from the region of myocardial ischaemia. So the leads oriented to the ischaemia shows an inverted, deep, symmetrical and tall T waves.

**III. Chronic stabilized phase**

The S-T segment and T wave abnormalities revert to normal. The Q wave may persist indefinitely.

## COMPLETE LEFT BUNDLE BRANCH BLOCK WITH MYOCARDIAL INFARCTION

**a) Acute Myocardial infarction**

The criteria for diagnosing are (by sgarbossa)

1. ST elevation $\geq$ 1 mm concordant with QRS polarity have a high specificity and sensitivity
2. S-T elevation $\geq$ 5 mm discordant with QRS polarity, ST depression $\geq$ 1 mm in $V_1$, $V_2$, $V_3$ and sudden positive T waves in $V_5$, $V_6$ have a high specificity but low sensititivity.

**b. Old myocardial infarction**

1. A small q in lead I, $V_5$ and $V_6$
2. Sign of Cabrera and Fried land. Late notching of S waves in $V_3$-$V_5$.

3. Sign of Chapman – Notching of the upstroke of R wave in lead I, $aV_L$, $V_5$ and $V_6$

**SILENT MYOCARDIAL INFARCTION**

Population studies suggest that 20 – 60% of non-fatal infarction are not recognized by the patient. Of these half are truly silent.

The incidence is more in diabetic, hypertension, old age and those without an antecedent angina.

**Atypical presentations**

1. Congestive cardiac failure
2. Classical angina; but not severe or prolonged
3. Atypical location of pain
4. Central nervous system manifestations
5. Apprehension
6. Sudden mania or psychosis
7. Syncope
8. Overwhelming weakness
9. Peripheral embolization
10. Acute indigestion

**Myocardial Infarction in Diabetic**

The diabetic population suffers the following drawbacks

1. Increased risk of infarction by 2 – 3 times
2. The protective effect of being a premenopausal women is lost
3. Silent myocardial infarction
4. Higher morbidity
5. Less prominent circadian rhythm of attack
6. Excess cardiovascular and all cause mortality

## ANTRIOVENTRICULAR JUNCTIONAL AREA

The normal AV junction area can be divided into 3 distinct regions.

1. Transitional cell zone (nodal approaches)
2. Atrioventricular node (compact portion or Tawara's node)
3. Penetrating part of AV bundle (His bundle)

**1. Transitional cell zone**

This is the connection by which the myocardium of the right atrium anastomoses with the AV mode. This was described as outer Zone's by Tawara.

It is not a definite layer but made up of thin separate fascicles. They are subdivided into 3 groups; anterior (superior); middle and posterior (inferior).

Some workers have described fibers passing from the posterior tract to the his bundle.

**2. Atrioventricular Node**

It is an epicardial structure; lies just beneath the right atrial posterior epicardium, anterior to the ostium of the coronary sinus and directly above the insertion of septal leaflet of Tricuspid valve. It is at the apex of a triangle formed by the tricuspid annulus and the tendon of Todaro (Triangle of Koch). The AV node becomes the penetrating bundle of His at the apex of the triangle of koch where it passes below the attachment of tendon of todaro to the central fibrous body.

AV node is ovoid structure 1 by 3 by 5 mm. Histologically it is made up of thick mesh of tiny pale cells, which anastomose with each other (star cells).

By electron Microscopy, there are 4 type of cells; P cells, Transitional cells, common myocardial cells and purkinje cells.

Blood supply is by AV nodal artery, which is a branch of right coronary artery in 85 to 90% and a branch of left circumflex coronary artery in 10 to 15%.

AV node is richly supplied by adrenergic and cholinergic fibres.

**Bundle of His**

It is a cordal structure measuring 20 mm in length and 2 mm in diameter.

It is subdivided into three portions.

1. Non penetrating (Distal to AV node)

2. Penetrating (within central body and membranous ventricular septum)

3. Branching (Bifurcation at the crest of muscular ventricular septum upper connections sometimes connect the common bundle with the crest of the ventricular septum – called **paraspecific or mahaim fibers.**

Branches from both left anterior and posterior descending coronary arteries supply the upper muscular ventricular septum. Hence, this area is less likely to suffer ischemic damage.

**Bundle Branches**

They being at the crest of the Muscular ventricular septum

**Left Bundle Branch**

It forms a cascade down the left ventricular septal surface beneath the non coronary aortic cusp. Rosenbaum have postulated a bifascicular pattern. Tawara showed that left bundle radiates in a fan like pattern into 3 main divisions. Recent reconstructions studies also indicate a trifascicular division; but still the concept of a bifascicular left bundle remains useful to clinicians.

## RIGHT BUNDLE BRANCH

It is the direct continuation of the penetrating bundle. The bundle becomes a subendocardial structure in the middle and lower third of ventricular septum.

The left bundle branch receives blood from both left anterior descending and the posterior (right) descending coronary arteries.

The right bundle is supplied by both right and left anterior descending coronary arteries.

**Terminal purkinje fibres**

They connect the ends of the bundle branch to form interweaving network on the endocardial surface. They tend to be concentrated at papillary muscle tips than at the base of ventricles. They are more resistant to ischaemia than normal myocardial cell.

**Atrioventricular (Av) Block**

A delay or interruption in conduction of the atrial impulse through specialized AV conducting system. There are three degrees.

1. First Degree - Delay in conduction
2. Second Degree - Intermittent interruption of conduction
3. Third Degree - Complete interruption of conduction

**First Degree AV Block**

Delay in conduction reflected by a prolonged PR interval. The PR interval include

i). Time taken for impulse to travel from SA to AV node (usually 0.03 Sec)

ii). Time for the impulse to travel through AV node, bundle of His, bundle branches.

In first-degree block, PR interval is prolonged beyond 0.20 sec (0.18 in children) Normal is 0.12 to 0.20.

All the P waves are followed QRS complex.

**Associated with**

- Coronary artery disease
- Acute rheumatic carditis
- Drugs – Digitalis, beta blockers
- Acute Myocarditis
- Hyperkalemia
- Uremia
- Normal individuals

## SECOND DEGREE BLOCK

There is an intermittent failure of AV conduction

2 types

**Mobitz type I AV block (Wenckebach AV block)**

The transmission through conducting system becomes increasingly difficult with consecutive beats until it fails and a beat is dropped. P-R interval lengthens with successive beats until a beat fails to be conducted.

The P-R interval preceding the blocked P is the longest and that follows the blocked P is shortest.

**Mobitz type II Atrioventricular Block**

There is no preceding prolongation of PR interval. PR intervals of all the conducted impulses are normal. It is nearly always due to bilateral bundle branch block, so QRS configuration is abnormal. It frequently progress to complete block.

**Causes of second degree block**

- Acute rheumatic carditis
- Other acute carditis. Eg., Diptheritic
- Coronary artery disease
- Digitalis
- Associated with fast supraventricular rhythms

## COMPLETE ATRIOVENTRICULAR BLOCK

All the supraventricular impulses are blocked within the conducting system. The ventricles are activated by a subsidiary ectopic pace maker in the Atrioventricular node below the block or within the ventricles.

Complete block is due to a prolongation of the absolute refractory period so that is occupies the entire cardiac cycle.

**Manifestations**

1. ***AV dissociation*** – P wave bear no relationship to the QRS complexes.
2. ***A slow ventricular rate***

A pace maker situated above the bifurcation of bundle of His and distal to the block will produce a rate of 40 – 60/mt.

If the pace maker is below the common bundle, the rate if about 30 – 40/mt.

**3. *QRS configuration***

If the pacemaker is above the bifurcation of bundle of His, QRS complex is usually normal in configuration. This is called **AV junctional escape rhythm.**

If the pacemaker is below the bifurcation of common bundle, the QRS configuration is wide and bizarre because the propagation of the impulse in the ventricle occur in an abnormal fashion. This is termed **ventricular escape rhythm.**

**4. *P – P Interval***

When atrial mechanism is sinus, the P-P interval is regular. In 30% cases, the P-P interval which contains the QRS complex is shorter than the P-P interval without QRS complex. This is termed ***ventriculophasic sinus arrhythmia.***

**5. *R – R interval***

This is usually regular

Irregular R-R interval in complete heart block occur in

1. Multiple pacemaker in the AV junction or ventricles
2. Irregular discharge of a single pace maker.
3. Premature contractions (Ventricular / AV nodal)
4. Para Systole

5. Exit Blocks

6. Intermittent artificial pacemaker induced ventricular rhythm

Causes of complete heart block may be transient or permanent

**Transient**

1. Acute infections (Diptheria, other bacterial, Viral, Fungal)

2. Electrolyte imbalances

3. Trauma, Cardiac Surgery

4. Drugs - Digitalis

    4. Acute inferior wall myocardial infarction

**Permanent**

1. Idiopathic sclerodegenerative disease – Lenegre's disease

2. Lev's diseases – fibrocalcareous degeneration

3. Congenital Atrioventricular Block

4. Anterior Myocardial Infarction

5. Syphilitic Heart Disease

6. Diptheria

7. Chaga's Disease

8. Sarcoidiosis

9. Rheumatoid disease

10. Cardiomyopathies

11. Cardiac Surgery

12. Congenial Heart Disease (Corrected Transposition of great vessels, Ventricular septal defect, Ostium primum atrial septal defect)

## STOKES – ADAMS ATTACK:

A syncopal attack resulting from ventricular standstill or asystole. This occurs in third degree AV block when then subsidiary ectopic pace maker fails to discharge. This is produced in

1. The transition from second degree to complete heart block
2. When two or more ectopic pacemakers are in competition.

Syncopal attacks due to paraoxysms of ventricular flutter or ventricular fibrillation are also sometimes referred to as Stokes – Adams attack.

### Right Bundle Branch Block

The septum is activated normally from left to right. Left ventricle is activated normally, but right ventricular depolarization is delayed.

### Complete Right Bundle Branch Block

1. Lead $V_1$ reflects a tall, wide and frequently notched $R^1$ deflection.
2. The left oriented leads, $V_5$, $V_6$ and $L_1$ reflects a prominent, delayed and widened S wave
3. QRS duration > 0.12 second

a. The activation of ventricles begin in the left lower third of interventricular septum and spreads transversely from left to right through septum. This vector is no longer opposed by the normal, smaller right to left septal vector which originate from right bundle branch. So left to right septal vector marginally increase in magnitude. This results in a prominent 'r' in $V_2$.

b. Activation of the right side of the interventricular septum and the right and free wall is effected by the activation front, which arises in the left side of the septum. This crosses the 'Physiological intraseptal barrier and is conducted through ordinary myocardium'.

Consequently it is slow and anomalous and the vector directed to right and anteriorly.

The abnormal right paraseptal vector occurs simultaneously and opposite to the vector of left free wall.

This results in diminution of S wave in $V_1$, which eventually disappears. There is also attenuation of R wave in left oriented leads.

Abnormal right ventricular activation is reflected by wide $R^1$ in $V_1$ and prominent slurred and delayed S in $V_6$.

So lead $V_1$ shows a small initial R wave followed by S or s wave of left ventricular depolarization. A terminal bizarre and slurred $R^1$ wave.

**$V_5$, $V_6$ and lead I shows**

1. A small initial q wave
2. Relatively tall R wave
3. Terminal bizarre and slurred S wave

## INCOMPLETE RIGHT BUNDLE BRANCH BLOCK

Here the conduction through the right bundle branch is delayed but still possible. These manifest as

1. Diminution of the S wave in lead $V_2$. It is the earliest sign
2. Slurring of the upstroke of S wave in lead $V_2$.
3. Appearance of $r^1$ wave in lead $V_2$.
4. Amplitude of $R^1$ wave increases so that configuration is $rsR^1$ pattern.

QRS deflection is widened, but less than 0.11 sec. And the $R^1$ deflection is less than 0.04 seconds.

ST segment and T waves in an uncomplicated right bundle branch block shows secondary changes. T wave will be opposite in direction to terminal QRS deflection. ST segment will be slightly convex upwards or minimally depressed.

## CAUSES

1. Normal variant
2. Coronary artery disease
3. Acute pulmonary embolism
4. Cardiomyopathies
5. Valvular heart disease
6. Congenital heart disease – Atrial septal defect, Ebstein's anomaly, persistent Atrioeventricularis communis.

## LEFT BUNDLE BRANCH BLOCK

This is due to the delay or interruption of conduction within the left bundle branch.

There are three components of ventricular activation in complete left bundle branch block.

1. Right septal activation

The normal small right septal vector is not opposed by a concomitant, greater, left septal vector. This results in

a. A small initial positive deflection in left oriented leads.

b. A small negative deflection in right oriented leads.

2. Delayed and anomalous left septal activation.

The right septal activation process 'jump' an intraseptal physiological barrier and activates the left side of septum in an anomalous manner. The vector is directed posteriorly and to the left.

This produces a tall $R^1$ in left oriented leads and a deep S in right oriented leads.

3. Delayed and anomalous activation of the free left ventricular wall.

This results in a vector directed posteriorly, left and somewhat superiorly. This produces a tall $R^1$ deflection in left oriented leads and a deep S wave in right oriented leads.

**Electrocardiographic Manifestations**

a. QRS duration more than 0.12 Sec.

b. QRS in various leads

$V_5$, $V_6$ and lead 1 usually presents a tall and notched R wave – an $RR^1$ or M shaped complex. The intrinsicoid deflection may be 0.09 to .010 seconds.

Lead $V_1$, $V_2$ presents a widened, notched QS complex or an rS complex.

ST segment and T wave shows secondary changes, in the opposite direction of terminal segment.

## INCOMPLETE LEFT BUNDLE BRANCH BLOCK

1. The small initial q wave in lead $V_5$, $V_6$ and I disappears, resulting in a single tall R wave.
2. Small initial r wave in lead $V_1$ disappears. On further progression, a slur appears on the upstroke of QRS complex, accompanied by widening and then notching of QRS. QRS duration will be less than 0.12 second.

## CAUSES

1. Coronary Artery Disease
2. Hypertension
3. Aortic valve disease
4. Cardiomyopathy

## LEFT ANTERIOR FASCICULAR BLOCK

### Criteria for diagnosis

1. Abnormal left axis deviation (usually between $-45^o$ and $-60^o$)
2. rS complexes in leads II, III, $aV_F$ and qr complexes in lead I and $aV_L$.
3. Delayed intrinsicoid deflection in leads I and $aV_L$. (>.045 second)
4. Peak of r wave in lead III occurring earlier than peak of r wave in lead II.'
5. Peak of R wave in lead $aV_L$ occurring earlier than peak of R wave in $aV_R$.
6. Increased QRS voltage in limb leads.

## CAUSES

- Normal variant
- Coronary artery disease
- Left ventricular hypertrophy

## LEFT POSTERIOR FASCICULAR BLOCK

***Criteria for diagnosis***

1. Right axis deviation (usually $\geq +120^{o}$)
2. r in leads I and $aV_L$, q in II, III and $aV_F$.
3. Delayed intrinsicoid deflection in $aV_F$ (>.045 sec)
4. Increased QRS voltage in limb leads.
5. No evidence of right ventricular hypertrophy.

A similar sequence of ventricular activation can also occur in right ventricular hypertrophy, pleuropulmonary disease and extremely vertical anatomic heart position. So a diagnosis of 'Pure' posterior fascicular block is rarely made from ECG alone.

# OBSERVATIONS

**Group characteristics**

## 1. AGE DISTRIBUTION

Total number of cases : 150

Total number of control : 100

| S.No. | Age in years | Cases | Control |
|---|---|---|---|
| 1. | 31 – 35 | 19 | 18 |
| 2. | 36 – 40 | 58 | 43 |
| 3. | 41 – 45 | 54 | 27 |
| 4. | 46 – 50 | 19 | 12 |

## 2. SEX DISTRIBUTION OF CASES

Total number of patients : 150

Males : 78

Females : 72

| Sex | No. of cases | Percentage |
|---|---|---|
| Male | 78 | 52% |
| Female | 72 | 48% |

## 3. SEX DISTRIBUTION OF THE CONTROL

Total number : 100

Males : 55

Females : 45

| Sex | Number | Percentage |
|---|---|---|
| Male | 55 | 55% |
| Female | 45 | 45% |

## 4. DURATION OF DIABETES

| S.No. | Duration | Number | Percentage |
|---|---|---|---|
| 1. | 0 – 8 yrs | 110 | 73.4 |
| 2. | > 8 yrs | 40 | 26.6 |

## 5. COMPARISON OF PREVALENCE OF RIGHT BUNDLE BRANCH BLOCK

Total number of cases : 150

Total number of patients with RBBB : 16

Total number of control : 100

Total number of control with RBBB : 3

| S.No. | Population | Total | RBBB | Percentage |
|---|---|---|---|---|
| 1 | Case | 150 | 16 | 11% |
| 2 | Control | 100 | 3 | 3% |

## 6. RELATIONSHIP OF RBBB WITH DURATION OF DIABETES

| S.No. | Duration (Yrs) | Total | RBBB | Percentage |
|---|---|---|---|---|
| 1 | 0 – 8 | 110 | 11 | 10% |
| 2 | > 8 | 40 | 5 | 12.5% |

## 7. COMPARISON OF PREVALENCE OF LEFT BUNDLE BRANCH BLOCK

Total number of cases : 150

Total number of patients with LBBB : 2

Total number of control : 100

Total number of control with RBBB : 1

| S.No. | Population | Total Number | LBBB | Percentage |
|---|---|---|---|---|
| 1 | Cases | 150 | 2 | 1.3% |
| 2 | Control | 100 | 1 | 1% |

## 8. COMPARISON OF PREVALENCE OF LEFT ANTERIOR FASCICULAR BLOCK

Total number of cases : 150

Total number of patients with LAFB : 12

Total number of control : 100

Total number of control with LAFB : 7

| Population | Total Number | LAFB | Percentage |
|---|---|---|---|
| Cases | 150 | 12 | 8% |
| Control | 100 | 7 | 7% |

## 9. PREVALENCE OF ASYMPTOMATIC MYOCARDIAL INFARCTION

Total number of cases : 150

Total number of patients with MI : 15

Total number of control : 100

Total number of control with MI : 2

| Population | Total Number | Asymptomatic MI | Percentage |
|---|---|---|---|
| Cases | 150 | 15 | 10% |
| Control | 100 | 2 | 2% |

## 10. PREVALENCE OF ATRIOVENTRICULAR BLOCKS

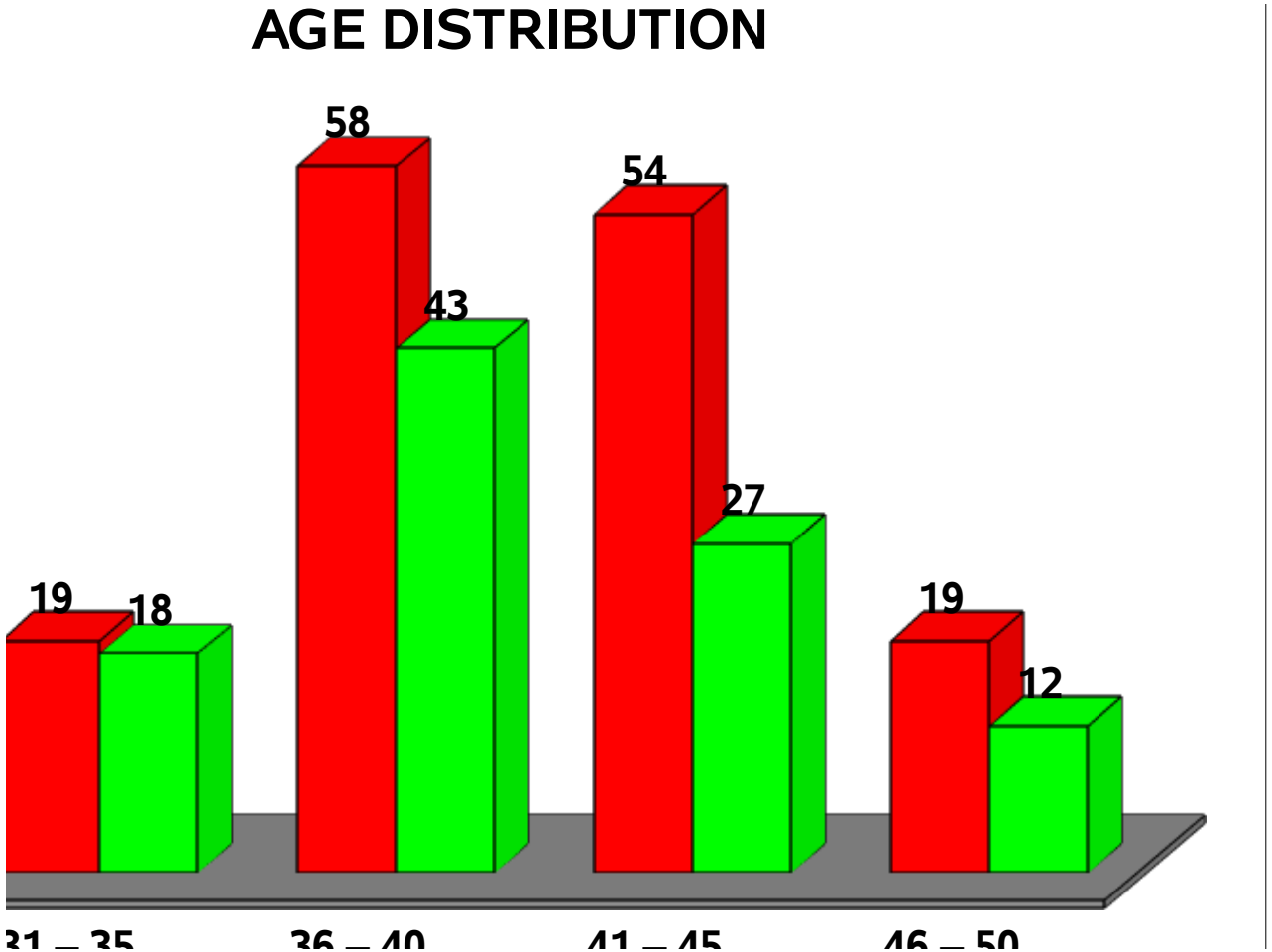
| Population | Total Number | AV Block | Percentage |
|---|---|---|---|
| Cases | 150 | 6 | 4% |
| Control | 100 | 3 | 3% |

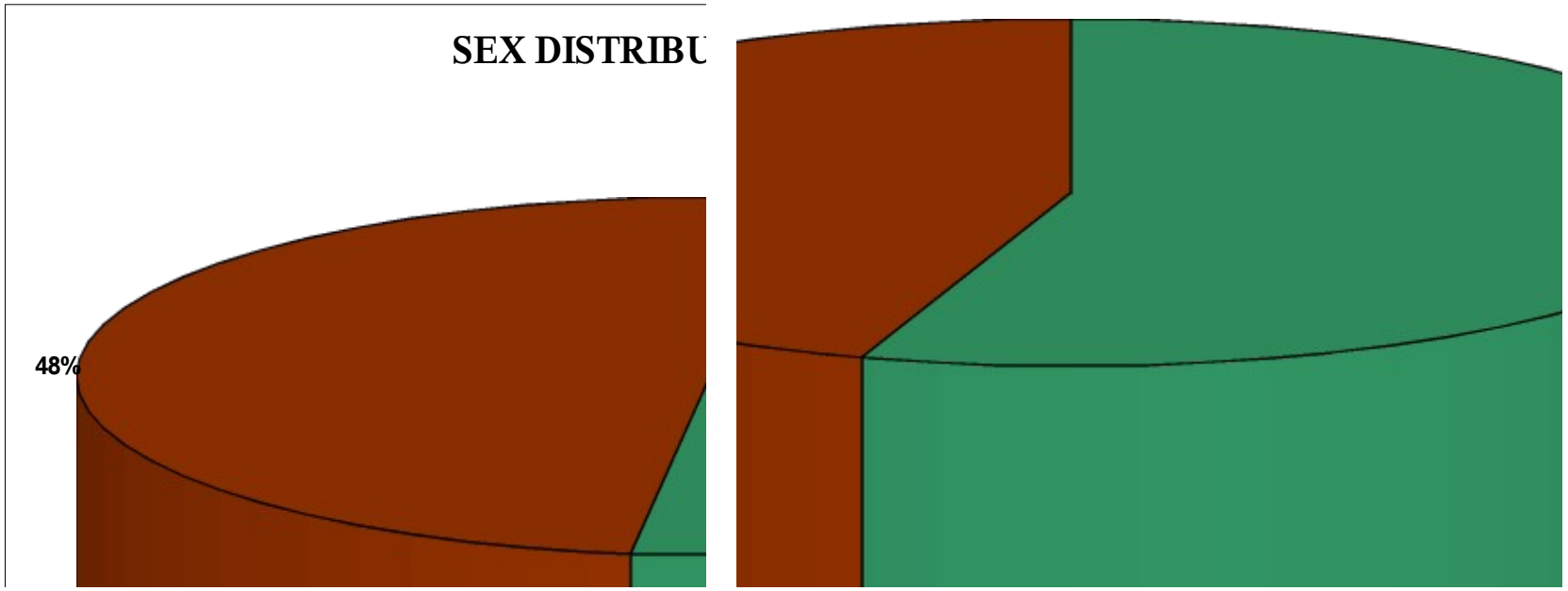
Total number of cases : 150

Total number of cases with AV Block : 6

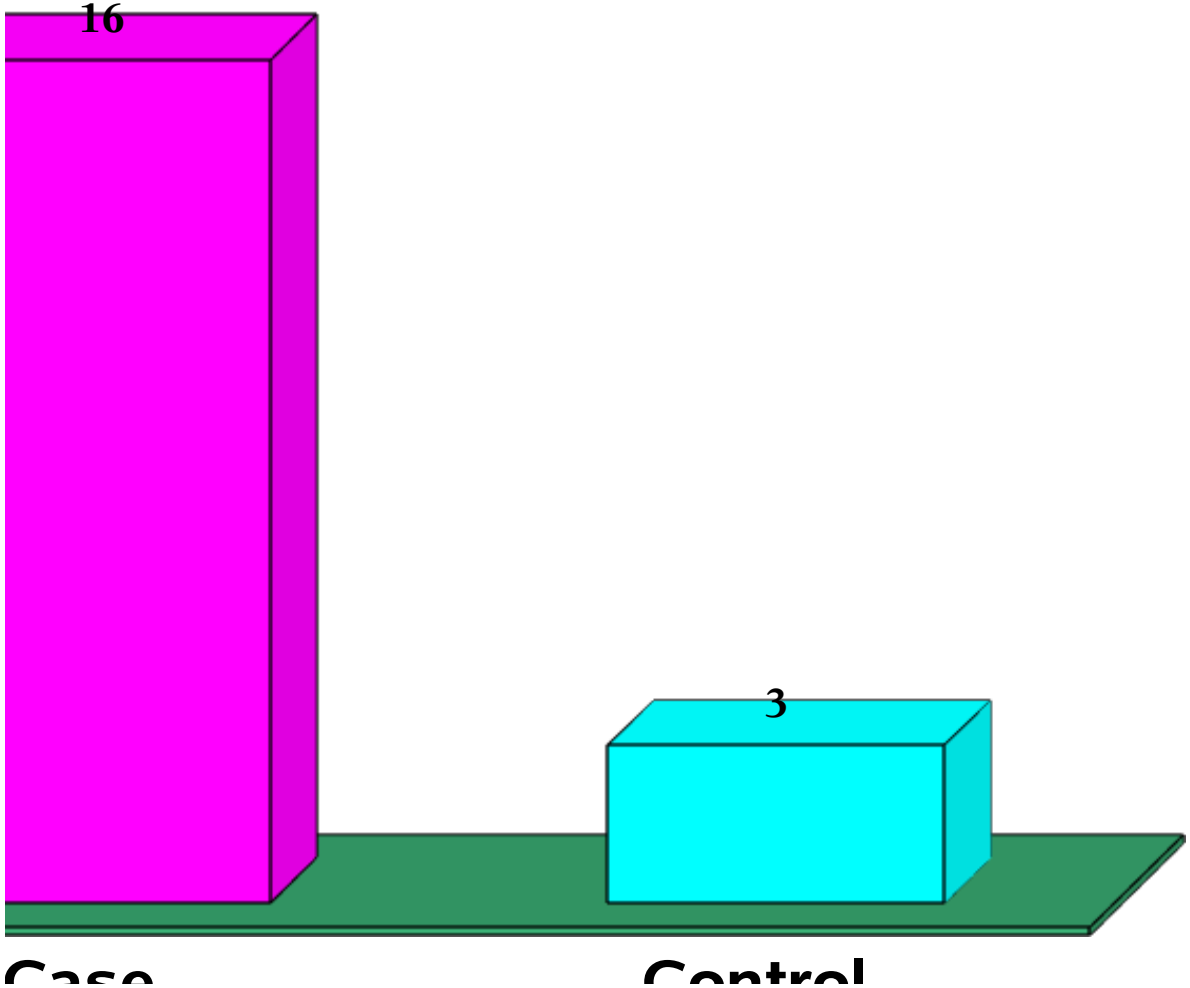
Total number of control : 100

Total number of control with AV Block : 3

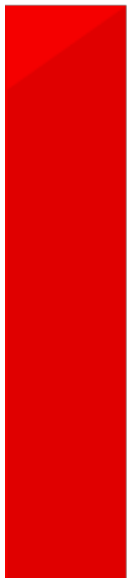

AGE DISTRIBUTION
58
54
43
27
19
18
19
12
31 – 35
36 – 40
41 – 45
46 – 50


SEX DISTRIBU
48%

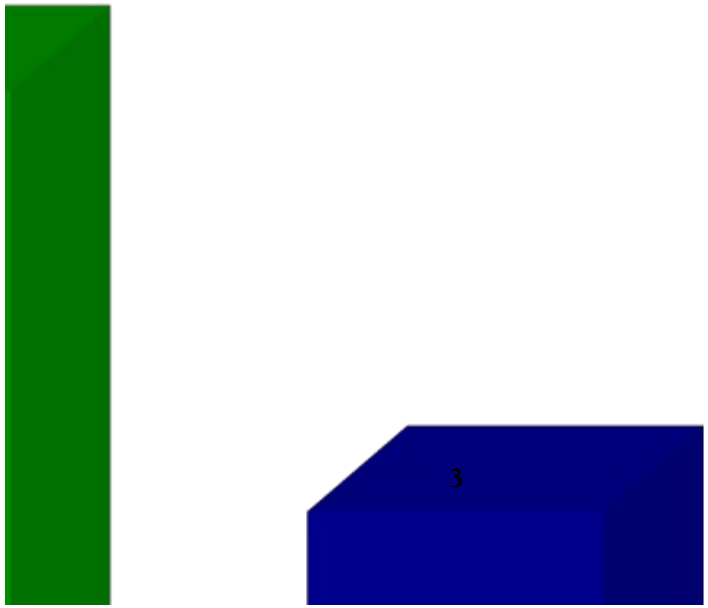
PREVALENCE OF RIGHT BUNDLE BRANCH BLOCK



## PREVALENCE OF ASYMPTOMATIC MYOCARDIAL INFARCTION



## PREVALENCE OF ATRIOVENTRICULAR BLOCKS



# RESULTS

The study population consisted of 150 non-insulin dependent diabetes mellitus. The control was made up of 100 subjects.

The study population was made of 72 females (48%) and 78 males (52%). Among the control population, there were 45 females (45%) and 55 males (55%).

Among the diabetic population, the mean age was 40.64 years with a standard deviation of 3.95 years. Among the control group, the mean age was 39.35 and the standard deviation 4.36 years.

In the diabetic population 18 males were smokers (23.07%), compared to 11(20%) males in controls. There were no female smokers in either group.

The mean cholesterol value in study group was 177.85 with a standard deviation of 15.45. The values in control group were 175.3 and 14.40 respectively.

The body mass index of diabetic females had a mean of 22.78 with a standard deviation of 1.98%, the control group females had a mean of 22.48 with a standard deviation of 1.52.

In the male diabetes, the mean of body mass index was 23.79 with a standard deviation of 1.78%. The control group males had a mean of 22.98 and a standard deviation of 1.80.

Thus the diabetic and control population had equal age and sex distribution and matched for serum cholesterol, body mass index and smoking habits, which are possible confounding factors.

Prevalence of right bundle branch blocks in diabetics was 11% (16/150) and in controls 3% (3/100). The difference in prevalence was significant with a P value of less than 0.05.

The prevalence of right bundle branch blocks in diabetics less than 8 yrs duration was 10% (11/110) and in those with more than 8 yrs duration it was 12.5% (5/40).

The prevalence of left bundle block in diabetics was 1.3% (2/150) compared to 1% (1/100) in control population. The results are not statistically significant.

The prevalence of left anterior fascicular block was 8% (12/150) in diabetic population compared to 7% (7/100) in control population. The results are not statistically significant.

No left posterior fascicular block were reported in either diabetic or control group.

The prevalence of atrioventricular block was 4% (6/150) in diabetic population and 3% (3/100) in control population. All were first-degree blocks. The results are not statistically significant.

The prevalence of asymptomatic myocardial infarction was 10% (15/150) in diabetic population and 2% (2/100) in control population.

Chi-square test was employed to test the significance of the difference between the proportions, which were compared.

# DISCUSSION

Various studies have shown that conduction abnormalities of heart and myocardial ischemia are more common in diabetics as compared to non diabetics.

In 1976, Dutta et al[1] did a study on 817 diabetics without myocardial infarction. 50 patients (6.11%) had bundle branch blocks out of which only 2 had left bundle branch block. Both these were associated with heart failure. Out of 48 cases of right bundle branch block, 19 were associated with left anterior fascicular block. None of these were associated with clinical evidence of ischemic heart disease.

Dutta et al suggested that the high incidence of right bundle branch block could be due to degenerative changes affecting the slender right bundle as a part of diabetic process. Whether this is the Lenergre's type enhanced by diabetes, or an early manifestation of diabetes cardiomyopathy remains to be evaluated.

A study of Rodriguez – Morci M. et al[2] showed that of the 1990 type II diabetics, 29.1% showed arrhythmias on ECG. Of these, right bundle branch block and left anterior fascicular block together formed 75.9%. Hypercholesterolemia and aging were additional risk factors.

Schneider JF[3] et al in a study evaluated newly detected right bundle branch blocks in Framingham study over 18 years. Cardiovascular abnormalities developed in 79% of patients. The incidence of cardiovascular mortality was three times greater than in general population. A QRS duration ≥ 130 millisec and QRS axis of 45 to 90 were markers of cardiovascular mortality.

Thraindottir I.S.[4] et al in a long prospective study involving 9135 males and 9627 females showed that in males less than 60 years, there was significant relation between right bundle branch block and diabetes.

Panja et al[5] in a study did autopsy on the hearts of 20 diabetic who died of various causes. Five of them had myocardial infarction four had complete heart block and two had right bundle branch block. All of them showed proliferative changes of small and medium seized vessels – with deposition of PAS positive materials characteristic of diabetic tissue changes. The four heart block patients had fibrosis extending to his bundle.

Panja and Dutta[6] et al in a study on 300 cases of complete heart blocks showed that diabetes was present in 14.6%.

Panja and Dutta[7] et al in an autopsy study of 15 cases of complete heart block, 8 hearts had idiopathic fibrosis extending from bundle of his to AV node. Three out of these had diabetes and had small vessel changes characteristic of diabetes.

Partiman and Bradly[9] were among the first to describe high incidence of bundle branch block in the diabetic myocardial infarction. Out of 151 survivors of myocardial infarction, the incidence of Atrivoventricular block, left and right bundle branch block were 4.3%, 6.3% and 1.5% respectively.

Strong Heart study done by Peter M.Okin et al[10] reported that rate corrected $QT_C$ interval prolongation predicted cardiovascular and all cause mortality from diabetes (hazard ratio 2.32 RR 1.35 – 3.12). They supported the use of $QT_C$ interval and ECG in clinical surveillance of diabetes.

QZ Liu[11] in a study conducted in National Institute of Diabetes reported that incidence of ECG abnormalities including blocks were higher in Type II Diabetes Mellitus patients (6.86%) as compared to non diabetic patients (2.23%). Age and sex adjusted ratios were significantly higher in diabetic patients (12.77 / 5.93). They also reported that ECG abnormalities were high in Type II DM patients treated with insulin than not treated with it.

Ozeko et al[12] compared 50 patients with diabetes and LBBB with 50 patients with diabetes only and found out during follow up. Patients with diabetes and LBBB developed more and extensive coronary artery disease than those without LBBB.

Ryomanishi et al[13] in a study of 17,361 found out that incidence of LBBB was comparable between diabetics and controls.

Roshanek Bugero et al[14] in penn diabetic study found out that diabetes accelerates the deposition of calcium in the coronary artery. In this study, the presence of pathological q waves was to the extend of 6%. There was also significant present of bundle branch blocks in diabetics.

In this study done on 150 NIDDM patients without any cardiac symptoms or hypertension, 11% (16/150) had RBBB against a control of 3% (3/100). This was significant. The incidence of left anterior fascicular block was higher in cases compared to controls.

The incidence of AV block, LBBB and left posterior fascicular block were comparable between two groups.

Jalal S et al[15] studied 200 diabetic patients. Out of these, 30 patients (15 male and female) had cardiac autonomic neuropathy. These were compared with 30 diabetics without autonomic neuropathy by 24 hour ambulatory electro cardiogram. Incidence of silent infarction was 40% (12/30) in the case population compared to 10% (3/30) in control. S.cholesterol and triglyceride were significantly higher in patients with silent ischemia. The concluded that cardiac autonomic neuropathy is a predictor to silent myocardial infarction.

Thomas K et al[16] studied the prevalence of asymptomatic cardiac ischemia in men with type 1 diabetes using myocardial scintigram and 24 hour holter monitoring. Prevalence of silent ischemia was around 20%. In the study, a diastolic blood pressure of ≥ 90 mm Hg was an independent risk factor of myocardial ischemia.

Negrusz – Kaweck M et al[17] studied the incidence of silent ischemic heart disease in diabetics. Patients with IDDM had 19.2% of silent ischemia compared to 20.22% in NIDDM patients. This was not statistically significant. They also remarked that a combination of 24 hour Holter monitoring with exercise stress test is the best combination for detecting silent ischemia.

O'Sullivan JJ, et al[18] in a study of 41 diabetic males found that silent ischemia was present in 64.7% with autonomic neuropathy against 4.1% without autonomic neuropathy. They concluded that autonomic neuropathy may prevent the development of anginal pain.

Sejil et al[19] reported that prevalence of silent myocardial infarction was higher (6.72%) in Type II DM patients when compared with general population. On follow up, while the incidence of over all cardiac mortality was same, incidence of non fatal myocardial events were more in Type II DM.

A prospective study by Andrew Neil et al[20] reported and for each mg of $HbA_1C$ decrease the risk of myocardial infarction by 8 – 21%. He recommended monitoring by measurement of $HbA_1C$ in diabetics.

Gowtham Ravipathi et at[21] studied incidence of silent ischemia in patients with elevated glycosylated haemoglobin and found out that there was a significant risk of silent myocardial ischemia in people with raised glycosylated haemoglobin.

Wackers FJ et al[22] studied the prevalence and clinical predictors of silent myocardial ischemia in asymptomatic type II DM patients (DIAD study). Out of 1123 patients studied, 113 had silent ischemia (9.8%). He also found out that odds ratio of having silent ischemia raised to 5.6 after 6 – 10 years of diabetics.

In the present study, the prevalence of silent myocardial infarction was significantly higher in the diabetic population, 10% (15/150) compared to the controls 2% (2/100).

# CONCLUSIONS

The study on 150 non-insulin dependent diabetes mellitus showed that right bundle branch block occurred in 11% (16/150) of Diabetic population. When compared to the control population, this is statistically significant.

The prevalence of left anterior fascicular block was higher in diabetics 12/150 (8%) than in control population, but this was not statistically significant.

The prevalence of asymptomatic myocardial infarction was 15/150 (10%) in diabetics compared to the control population, a statistically significant difference.

The prevalence of left bundle branch block and atrioventricular blocks is comparable in both diabetic and control population.

Cardiovascular complications form a very large percentage of diabetic morbidity and mortality.

Hence early and comprehensive evaluation of cardiovascular system should be given priority in the long-term management of diabetic patients. ECG at regular intervals can contribute significantly towards assessing the prognosis of Type II DM patients.

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# PROFORMA

## CARDIAC CONDUCTION ABNORMALITIES AND ASYMPTOMATIC MYOCARDIAL INFARCTION IN TYPE II DIABETES MELLITUS PATIENTS

Name : Age: Sex: Serial No:
Medical No:
Diabetology No:

Duration of Diabetes :

Current Treatment :

Clinical History :

Cardiac Symptoms : Chest Pain :

Breathlessness :

Palpitations :

Other Symptoms if any

**EXAMINATION FINDINGS**

Height_____Cms Weight _____Kgs.

Body Mass Index (BMI)

**GENERAL EXAMINATION**

PR : BP :

JVP : Oedema : Ascites :

**SYSTEMIC EXAMINATION**

CVS : CNS :

RS :

GIT : Fundus :

**INVESTIGATIONS**

Hb% : gm% ESR : mm/hr

FBS : B1 Urea :

2hr PPBs : Sr.Creatinine :

CXR : S. Cholesterol

ECG :

RBBB :

LAFB :

AV Block :

Myocardial Infarction :

Ref.No. /ME1/2007

Stanley Medical College,
Chennai-1 Dt. -9-2007

Sub:Medical Education—Stanley Medical College, Chennai—
Ethical Committee constituted for approval of Dissertation/
Thesis submitted—regarding.

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The Ethical Committee meeting was held on 3-9-2007 and 7-9-2007 to discuss the paper submitted for Dissertation /Thesis.

The following Members of the Ethical Committee were present and discuss in detail for the approval of the papers presented by the individual by means of power point presentation.

Dr.A.Sundaram, Dean incharge,
Dr.S.Madhavan, Prof. of Pharmacology,
Dr.Thenmozhivalli, Prof. of Microbiology,
Dr.S.Natarajan, Prof. of Medicine,
Dr.K.Balasubramanian, Prof. of Physiology
Dr.M.L.Shyamala, Prof. of Surgery,
Thiru M.Panneerselvam, Junior Administrative Officer.

**LIST OF PAPERS SUBMITTED FOR ETHICAL COMMITTEE APPROVAL**
ETHICAL MEETING

Dr. Kiruba Mohan, Prof. of Dermatology

1."N.O.C. for PMS study of pregabalin" - Dr.Parimalam Kumar
2. " A Phase IIb/III trial of LLL-3348 of lupin ltd in plaque psoriasis - Dr.A.Ramesh

Dr.M.Thirunavkarasu, M.D.(Psy)D.PM , Prof. of Psychiatry

"Prevalence, socio-demographic variables and method of suicide among various causes of death."

(2)Psychological autopsy of suicide.

V.Rohit

Effect of chewing gums (XYLITOL)

K.Chinthnidhi

Mycotic infections in immuno compromised and cancer patients.

Malavika Prasad

Profile of Hypertensive emergencies - A study of 100 cases from Dept. of medicine, GSH.
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3. Sandhya Rani.C Final MBBS,
Assessment of coverage and quality of maternal and child health services at Minjur Primary Health Centre; Block level
4.C.Muralidharan, Final year.
The implications of mobile phones on hearing loss.
5.V.Sarath Chander, 3rd MBBS
Prevalence of Deafness in children.
6.B.Madhusoothanan, 3rd year
(1) Lung functions in type 2 diabetes.
(2)Hyponatremia in intensive medical care patients in GSH.
7.S.Sathyapriya – II MBBS.,
"A study about screening tests for cases of urinary tract infections (UTIs)Using Urine samples."
8.S.Moogaambiga,
"Estended spectrum beta lactamase producing microbes.

POST GRADUATES
1.Dr.R.Arunprakas –M1. P.G.
Analysis of clinical profile of systemic lupus erythematosus
2.Dr.S.Murugananth – M.2 P.G.
Clinical Profile of infectious fevers
3.Dr.N.Loganathan – M2 P.G.
Clinical and Epidemiological profile of Human Leptospirosis in North Chennai.
4.Dr. K. Babu – M3 – P.G.
Study of Clinical Profile of patients with acute inferior wall myocardial infarction.
5.Dr. S.P.Maharajan – M3 – P.G.
Analytical study of atrial fibrillation in Govt. Stanley Medical College Hospital.
6.Dr.P.R.Sowmini – M3 - P.G.
Clinical profile of arrhythmias complicating acute anterior wall myocardial infarction.
7.Dr.E.Uma Maheswari – M4 – PG
Clinical Radiological analysis of Focal seizures with CT Scan.
8.Dr.S.Sudha Selvi, M4 - PG
Clinical profile of chronic obstructive pulmonary disease.

9.Dr.N.Jayanthi. M6- PG
Prevalence of B2 glycoprotein 1 Dependent anticardiolipin antibodies in acute ischemic stroke.
10.Dr.Lavanya. S. – MD PG
Comparative study of fasting lipid profile in chronic renal failure patients on conservative management, on dialysis and after renal transplant.
11.Dr.R.Geetha – Pharmacology

Evaluation of the sedative effects produced by antihistamines inhealthy volunteers by new techniques.

12.Dr.K.G.Devibala, Pharmacology

To evaluate the efficacy of rupatadine in controlling pruritis in lichen planus.

13.Dr.B.Anitha, Physiology

Visual Evoked potentials in hypothyroid patients.

14.Dr.M.Thirumaran, Physiology

Heart rate variability analysis in alcohol dependant individuals.

15.Dr.K.Vinod, Anaesthesia

Real time ultra sound guided catheterization of IJV – A prospective comparison with land mark guided technique.

16.Dr.Rajesh. C.P. – M6 – PG

Cardiac conduction abnormalities and asymptomatic myocardial infarction in NIDDM patients.

The papers presented to the Committee members by the Profs./Asst. Prof./Post Graduates/Under graduates were discussed across the table while their presentation.

The above papers discussed in detail with its supportive documents submitted by them and approved the above papers submitted for Ethical Committee.

| Name of the Members | Signature |
|---|---|
| Dr.A.Sundaram, Dean incharge, | |
| Dr.S.Madhavan, Prof. of Pharmacology, | 8/10/07. |
| Dr.Thenmozhivalli, Prof. of Microbiology, | |
| Dr.S.Natarajan, Prof. of Medicine, | 9.10.2007. |
| Dr.K.Balasubramanian, Prof. of Physiology, | |
| Dr.M.L.Shyamala, Prof. of Surgery, | 08/10/07 |
| Thiru M.Panneerselvam, Junior Administrative Officer. | |