ANALYTICAL STUDY OF ATRIAL FIBRILLATION IN GOVERNMENT STANLY MEDICAL COLLEGE HOSPITAL, CHENNAI

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 **"ANALYTICAL STUDY OF ATRIAL FIBRILLATION IN GOVERNMENT STANLEY HOSPITAL, CHENNAI"** is the bonafide original work of **DR. S.P.MAHARAJAN,** 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 January 2007 to June 2007.

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DECLARATION

I, **DR. S. P. MAHARAJAN,** solemnly declare that dissertation titled **"ANALYTICAL STUDY OF ATRIAL FIBRILLATION IN GOVERNMENT STANLEY MEDICAL COLLEGE, CHENNAI"** is a bonafide record of work done by me in the Department of Internal Medicine, Government Stanley Medical College and Hospital during January 2007 to June 2007 under the guidance of **Prof. A.K. GEETHADEVI, 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.

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ACKNOWLEDGEMENT

I would like to thank our beloved Dean, Govt. Stanley Medical College and Hospital, **Dr. MYTHILI BHASKARAN, M.D.,** for permitting me to utilize the hospital facilities for this dissertation.

I extend my sincere thanks to **Prof. S. NATARAJAN, M.D.,** Professor and Head of the Department of Medicine, Govt. Stanley Medical College and Hospital for his guidance during the study.

I also extend my sincere thanks to my Chief **Prof. A.K. GEETHADEVI, M.D.,** Professor of Medicine, Government Stanley Medical College & Hospital for his constant support and excellent guidance during this study.

I express my sincere thanks to **Dr. R.SUBRAMANIAN, M.D., D.M.,** Professor of Cardiology and **Dr. M.SOMASUNDARAM, M.D., D.M.,** Additional Professor of Cardiology for permitting me to utilize the facilities in the Intensive Coronary Care Unit for the purpose of this study.

I thank the Assistant Professors of my unit **Dr.S.CHANDRASEKAR, M.D.,** and **Dr.S.NALINIKUMARAVELU, M.D.,** for their valid comments and suggestions and guidance throughout the study.

Finally, I thank all the patients for their extreme patience and co-operation.

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ETHICAL COMMITTEE CLERARANCE

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Ethical Committee constituted for approval of Dissertation/
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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,
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Dr.M.L.Shyamala, Prof. of Surgery,
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LIST OF PAPERS SUBMITTED FOR ETHICAL COMMITTEE APPROVAL
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Dr. Kiruba Mohan, Prof. of Dermatology

1."N.O.C. for PMS study of pregabalin" - Dr.Parimalam Kumar
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Study of Clinical Profile of patients with acute inferior wall myocardial infarction.
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13.Dr.B.Anitha, Physiology

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14.Dr.M.Thirumaran, Physiology

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15.Dr.K.Vinod, Anaesthesia

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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.

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INTRODUCTION

Atrial fibrillation is a condition of increasing clinical and economic importance.It is the most common arrhythmia encountered in clinical practice.AF is a supraventricular tachyarrhythmia characterized by uncoordinated atria activation with consequent deterioration of atrial mechanical function.

Atrial fibrillation is associated with substantial mortality and morbidity.It is caused by many cardiac and non cardiac conditions.AF coexists with common cardiovascular conditions ,such as hypertension,heart failure,coronary heart disease and diabetes mellitus,and with an increasing older general population af will become an increasing health care burden.

AF is increasing in incidence and prevalence.. The estimated prevalence of AF in general pop[ulation is 0.4%..The prevalence and incidence increases with advancing age,affecting approximately 5% of individuals older than 65 years and nearly 10% of those aged older than 80 years. In the Framingham study, yearly incidence rates for persons age 50 – 59 were approximate 1 – 9 and 0 – 9 per 1000 person years in men and women respectively.

Over 38 years of follow up, the Framingham study found an over all incidence rate of approximately 3 per 1000 years in men and 2 per

1000 years in women aged 55 – 64 years. The incidence of AF doubled, for every decade increment in age in the Framingham heart study cohort.

Men are at moderately higher risk of AF than women, however the onset of AF in women occurs later in life.

Prognostically the prevalence of AF is associated with five-fold increase in morbidity risk and a two-fold increase in mortality risk. Most complications and Death associated with AF are due to complications associated cerebrovascular embolic events.

Pharmacological treatment modalities form the mainstay of treatment. Recent research has highlighted new approaches to both pharmacological and non pharmacological management strategies.Newer antiarrhythmic agents have been developed and others are being evaluated for their potential use in atrial fibrillation.

AIM OF STUDY

1. To analyse the etiological factor, clinical presentation and complications of 50 cases of Atrial fibrillation in Government Stanley Medical College Hospital, Chennai-1.

REVIEW OF LITERATURE

HISTORY

Perhaps the earliest description of atrial fibrillation is in The Yellow Emperor's Classic of Internal Medicine (Huang Ti Nei Ching Su Wen). The legendary emperor physician is believed to have ruled China between 1696 and 2598 BC. The poor prognosis associated with chaotic irregularity of the pulse was clearly acknowledged by most of the ancient physicians, but in recorded history, William Harvey in 1628 was probably the first to describe "fibrillation of the auricles" in animals.

In clinical practice and with the aid of Laennec's recently invented stethoscope, Robert Adams reported in 1827 the association of irregular pulses with mitral stenosis; in 1863, Etienne Marey published a pulse tracing from such a patient. Other early descriptions of atrial fibrillation and its importance were published early this century by Sir James Mackenzie and Heinrich Hering.

The discovery of the therapeutic properties of digitalis leaf (Digitalis purpurea) in 1785 by William Withering brought some relief to patients with severe heart failure. It is interesting that Withering recorded a patient who had a weak, irregular pulse that became "more full and more regular" after five draughts containing Fol Digital Purp oz iv. In 1935 Jean Baptiste Bouilland said that he considered digitalis to be "a sort of opium for the heart."

The main diagnostic breakthrough was the invention of the electrocardiograph by William Einthoven in 1900. A close friend of Einthoven, Sir Thomas Lewis at University College Hospital, London, was the first to record an electrocardiogram in a patient with atrial fibrillation.

The exact mechanisms and importance of atrial fibrillation remained controversial (Lewis and Mackenzie had disagreed about these issues) until 1970, when Bootsma and coworkers, with the aid of computers, concluded that the totally irregular response of the ventricles was due to the effect of "randomly spaced atrial impulses of random strength reaching the atrioventricular node from random directions."

The epidemiological importance of atrial fibrillation as an important precursor of cardiac and cerebrovascular death was investigated in detail in the Framingham study by William Kannell and colleagues in 1982. Over the past 10 years, awareness has increased of the hazards of sustained non-rheumatic atrial fibrillation and the benefits of prophylaxis against thrombosis in preventing cerebral thromboembolism.

EPIDEMIOLOGY

Atrial fibrillation is common in the community, affecting up to 5% of people aged 75 or over. It is a major reason for emergency admissions and cause of cardiovascular deaths. Thus most clinicians in hospital and

general practice will participate in managing such patients. As the prevalence of the condition increases with age, atrial fibrillation will become increasingly common in the increasingly aging population.

Epidemiological studies have shown that atrial fibrillation is fairly uncommon in people aged under 50 years but is found in 0.5% of people aged 50-59, increasing to 8.8% at age 80-89. Furthermore, the arrhythmia may be either chronic or paroxysmal. In the Framingham study, hypertension, cardiac failure, and rheumatic heart disease were the commonest precursors of atrial fibrillation. Up to a third of patients with atrial fibrillation, however, may have idiopathic or "lone" atrial fibrillation, where no precipitating cause can be identified and no evidence of structural heart disease exists.

Atrial fibrillation is more common in hospital practice than in general practice, being present in up to 7% of emergency medical admissions to district general hospitals. The commonest causes in Western countries include coronary heart disease, hypertension, and rheumatic and non-rheumatic valve heart disease. The commonest presenting features included heart failure, stroke, chest pain (including myocardial infarction or angina), and respiratory diseases. By contrast, in developing countries rheumatic heart disease is by far the commonest cause of atrial fibrillation

PATHOLOGY

The incidence of AF is increased with age and is prevalent in those aged 60 or greater. Microscopic and Macroscopic alternation in the atria begin in the 1st year of life. By 4th / 5th decades small fat spots appear in the right atrium in the region of AV-node and septum. These changes associated with aging will result in loss of myocardial fibers and fatty metamorphosis, and connective tissue and Focal hemorrhages in sinus node and Atrial Structures.[1]

One of the most Important pathological study of Atria in patients with AF was done by Davies and Pomerance.[3] In nearly 75 patients chronic AF there was sinus node muscle loss, internodal tract muscle loss and Atrial dilatation. In case of Rheumatic heart disease the LA is enlarged in almost all cases. In some case of tight mitral stenosis Aschoff bodies were found with in the Atrial myocardium. In other cases spinde shaped, or triangular scar lying between the muscle bundles and surrounding blood vessels represent the healing of Aschoff bodies.

MECHANISM OF ATRIAL FIBRILLATION

The basic mechanism

Two chamber concept

In an electrophysiological scene the heart consist of only two chambers. One formed by the atria and the other formed by the ventricles.

The two electrophysiological chambers are separated from each other by an electrically inert conductions barrier. Conduction barrier formed by the fibrous AV-ring. Communication across the barrier under normal circumstances is only possible through the specialized conduction system formed by the AV-nodes, bundle of his, the bundle branch and their ramifications.

Each chamber is activated by single, coordinated, uniform and progressive excitation process, which effects a total and almost simultaneous depolarization of the chambers. All the fibers are rapidly brought to the same state of excitability; consequently, the fibers of the chamber will at any given movement be in the same or almost the same electrophysiological state.

They will all be in a state of excitability or all in a state of potential responsiveness, and when all the fibers of a chamber are uniformly in one state, they are said to be in phase with each other. In myocardial fibrillation, the single, uniform activations process is lost and the phase state is transformed into a complex out of state. A physiological fragmentation tissue islet in varying states of refractoriness, excitation and responsiveness occur.[3]

The predisposition to and development of the state is favoured by the alternation of the fundamental event.

1. Uneven recovery of the chamber an out of phase state. Where one part of the chamber is responsive and the other part of the chamber is refractory a case of physiological asymmetry.

2. Premature stimulation of the chamber by an impulse that originates in or is induced into, the chamber before activation or recovery is complete.

THE GENESIS OF PHYSIOLOGICAL ASSYMETRY

The development of physiological asymmetry with in the biatrial chamber is favored by[3]

a) Mode of atrial activation
b) Prolongation of conduction
c) Unequal conduction
d) Increase in chamber size
e) Chamber asymmetry
f) Aberrancy in refractoriness
g) Unequal refractory period

a. Mode of Atrial Activation

Activation of the actual chamber occurs longitudinally and by contiguity, spreading from its point of origin in the SA node to engulf the whole chamber. Thus with longitudinal form of atrial activation proximal part are activated before the distal. This sequential and longitudinal form of activation thus favors an out of phase state and hence induces atrial fibrillation.

Prolongation of Conduction

A prolongation of conduction time when associated with long activation, increase the initial and terminal discrepancy.

UNEQUAL CONDUCTIONS

Unequal conduction c in the bit actual chamber will also potentiate, simple out of phase due to the difference in intramural terrain and disease process such as local ischaemia.

INCREASE IN CHAMBER SIZE

The larger the size of the chamber, the greater the potential out of phase state and more readily will fibrillation be initiated and maintained. As it is common in mitral regurgitation / tricuspid regurgitation which are associated with mamixal left atrial enlargement.

CHAMBER ASSYMETRY

The bilateral chamber is asymmetric and the pacemaker is eccentrically placed. Consequently, activation, recovery occurs soon at a point close to the pacemaker, then a point remote from the pacemaker. This bizarre or assignment shape of biatrial chamber will therefore in presence of long activation predispose toward the unequal recovery of the chamber.[4]

ABREVATION OF REFRACTORINESS

A short refractory period when associated with a long or relatively long conduction time will also predispose to the maintenance of fibrillation.

UNEQUAL REFRACTORINESS

Unequal refractoriness of the biatrial chamber will also aggravate the terminal out of phase discrepancy and predispose to atrial fibrillation.

SHORTENED REFRACTORY PERIOD

Any condition, which shortens the refractory period, will facilitate the perpetuation of fibrillation.

The following condition shorter the refractory period.

1. Acetyl choline
2. Vagal stimulation
3. Low K+ Concentration
4. High K+ Concentration
5. Digitalis toxicity

All the factors facilitate the precipitation and perpetuation of fibrillation. AF is easily maintained in cases of TR / MR due to large atria.

CAUSES OF PREMATURE ATRIAL STIMULI

The development of an out of phase state with in the biatrial chamber predisposes to the prescription of fibrillation by premature stimuli.

Those are of 4 basic sources of premature stimuli.

1. Atrial Extra systoles
2. Atrial Tachycardia
3. Reciprocal stimuli
4. Circus movement

Atrial extra systole are premature impulses which arise from an ectopic atrial pacemaker and then particularly if multiple and multifocal in origin will favour the initiation of fibrillation.[5]

ATRIAL TACHYCARDIA

Tachycardia shortens the refractory period but with very fast rates, the refractory period is reduced to a critical level, when it cannot shorten any further. It then occupies virtually the complete diastolic interval. When this occurs, successive impulses will tend to fall in the terminal out of phase period and may then initiate fibrillation.[6]

RECIPROCAL STIMULI

A premature stimulus of atria is also facilitated by the presence of a bypass within the AV conduction pathway. The rapidly returning reciprocal impulses constitute an early stimulus to the bi-atrial chamber and may consequently initiate the fibrillation in the WPW syndrome and LGL syndrome[6].

CIRCUS MOVEMENT STIMULUS

Circus movement stimuli a form of activation where in the excitation wave travels in a circular path around the bi-atrial chamber.

The Phenomenon occurs when the time taken for the excitation wave to travel around the bi-atrial chamber exceeds the refractory period. When this comes about the tissue ahead of the excitation front is always fully responsible and excitation is there by precipitated as a circular movement around the atria.[1]

The mechanism is facilitated by the association of a long conduction time and short refractory period. A circus movement may be responsible in some cases of atrial flutter.

Atrial flutter frequently precedes AF. The flutter constitute a source of early stimulation by virtue of a circus movement of rapidly

repitive stimulation and the flutter rate increases further before the conversion to Atrial Fibrillation.[6]

The aforementioned principle indicates that in the presence of longitudinal activation, the initiation of AF is formed by enlarged of bi-actual chamber, prolongation of conduction, inequality of conduction, decrease in refractory period.

BASIC MECHANISM OF ATRIAL FIBRILLATION

It is recognized that AF is initiated through one of several triggers. These triggers induce premature atrial complex or repetive activity occurring in the atrial body or in the myocardial sleeve of the pulmonary Vein.[7]

These triggers can initiate a self-sustained Atrial fibrillation in a vulnerable substrate (Focal trigger) or continue to fire and drive the atria in atria fibrillation by means of Fibrillatory conduits. (Focal driver)

There is recent evidence that the focal trigger is in the pulmonary Vein.[7]

MULTIPLE WAVELETS

It was proposed that atrial Fibrillation was caused by multiple random recentrant wavelets.

MOTHER ROTOR THEORY

A rotor is defined as stably rotating pathway of reaction that surrounds a pivotal point. A curved wave front radiates from the rotor to the surrounding tissues.[7]

ECG MANIFESTATION OF ATRIAL FIBRILLATION

Atrial Fibrillation is characterized electrocardiographically by grossly disorganized atrial actually that is irregular in respect to both rate / rhythm. There is no visually discernible timing pattern to the atrial electrical activity on the surface ECG.

Atrial fibrillating waves are best seen in VI, LII and LIII.

A fine atrial fibrillation waves virtually no imprint on the base line ("Straight line AF"). Coarse AF – fibrillatory waves are the size of respectable flutter waves, but are irregular.

One of the most challenging aspects in ECG is the distinction between aberrant intra ventricular conduction and Ventricular ectopy in the presence of Atrial Fibrillation.

Aberrant conduction tends to occur when a long ventricular cycle is followed by a short cycle. This long short cycle sequence with the short cycle terminated by an aberrantly conducted beat is called **ASHMAN's PHENOMENON.**

A series of short cycle if short enough may generate runs of consequent aberrant beats initiating VT.

Thus additional criteria for distinction between aberrancy and ectopy are required. In general an initial QRS Vector similar or identical to that of narrow QRS and a typical RBBB pattern in association with a long-short cycle sequence strongly favours aberrancy over ectopy.

LBBB aberrancy also occurs but is less common. Atrial fibrillation alters intraventricular conductions only through the following mechanism.

1. Functional bundle – branch block / Aberrancy.
2. Loss of Delta waves in WPW with normal pathway conduction during AF.

In patients with pre existing bundle branch block who develop AF with rapid ventricular response, the distinction from VT may be different.

AV CONDUCTION IN AF

Conduction of the impulses from the fibrillating atria may be complicated by

1. Physiological 2º A V – block
2. High Degree A V – block
3. Complete A V – block

AF WITH PHYSIOLOGICAL 2º AV – BLOCK

A – V node offers block because of its inherent refractory period. When AV node is bombarded by a large number of stimulus from the atrial, many, which fall on the refractory period of the AV node are blocked, and conduction occurs only when the node recovers. Because of this irregularity of the atrial impulses, the block also occurs irregularly and so the QRS complexes are placed at irregular intervals. In this type of block the vent of block the ventricular rate is between 120 – 180 / mt.

At times, the distributed pattern of the QRS complex in atrial fibrillation suggests a wenckeback form of AV conduction. This is suggested by a progressive diminution in RR interval followed by a long pause.[8]

AF WITH HIGH GRADE AV – BLOCK

This is characterized by idionodal (or) idioventricular escape beats that occurs regularly. Hence the long interval of QRS complexes during fibrillation remains constant.

AF WITH COMPLETE HEART BLOCK

Characterized by slow and regular ventricular rate. These beats are due to idionodal or idioventricular rhythms. In the former the QRS complexes are narrow, while in the latter the complexes are wide.

TYPES OF ATRIAL FIBRILLATION

Fast AF : When the ventricular rate is more than 120 / mt.

Show AF : When the QRS Complexes occur at the rate of 60 – 120 /mt

These terms are misnomers, service the atrial rate is very high in both case and the term fast and slow here applies to the ventricular rate.

AF WITH REDUCED VENTRICULAR RATE

This occurs in

1. Complete heart block with idionodal / idioventricular rhythm.
2. After administration of drugs like verapamil.
3. Functional or Ventricular Tachycardia.

DURATION

PAROXYSMAL

PERSISTENT

PERMANENT

Basic classification of the types of atrial fibrillation (AF)

Lone AF

Lone AF occurs in the absence of cardiac or other conditions predisposing to AF.

Acute AF

Acute AF generally refers to AF lasting less than 48 hours.

Paroxysmal AF

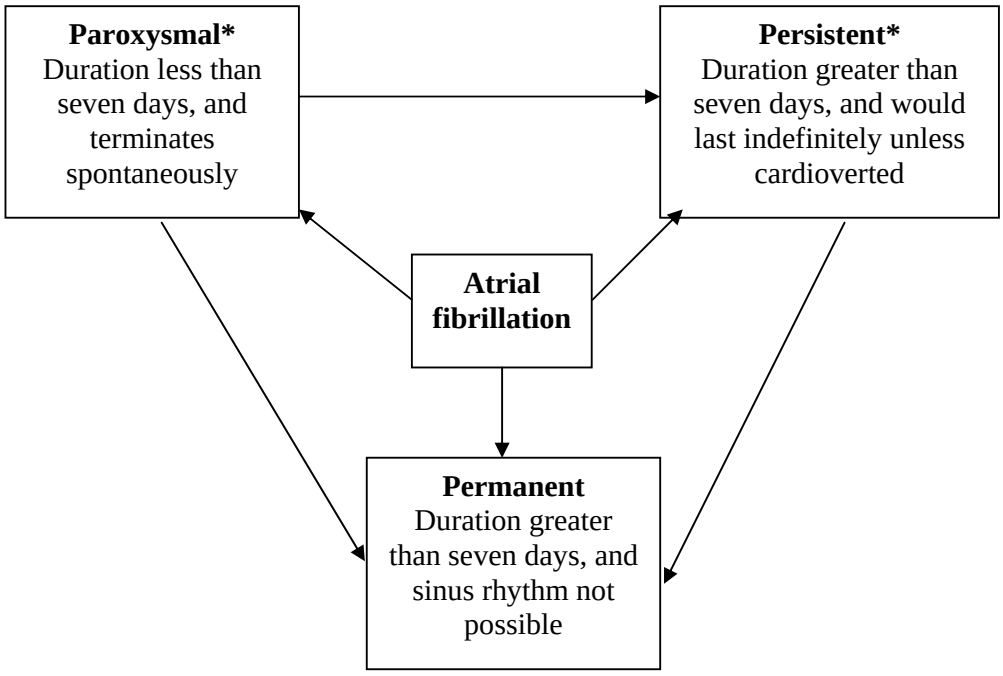
Paroxysmal AF generally is characterized by recurrent, transient episodes that revert to sinus rhythm spontaneously or with treatment.

Persistent AF

Persistent AF does not convert to sinus rhythm with out intervention or cardioversion.

Permanent AF

Permanent AF is persistent despite cardioversion.



AETIOLOGY OF ATRIAL FIBRILLATION

CVS disorders

1. Rheumatic heart disease
2. Coronary artery heart disease
3. Systemic Hypertension
4. Cardiomyopathy
    a. Dilated
    b. Hypertrophic
5. Congenital heart disease
    a. Atrial septal Defect
    b. Leutembackers syndrome
    c. Tricuspid Artesia
    d. Ebsteins anomaly
6. Sick Sinus
7. Diffuse myocardial disease – myocarditis
8. Pericardial disease
    a. Constrictive pericarditis
    b. Pericardial effusion
9. Pre – Excitation syndromes
    a. WPW
    b. LGL

10. Syphilitic heart disease
11. Bacterial Endocarditis
12. Atrial myxoma
13. Metabolic
    a. Hyperthyroidism
    b. Hyperkalemia
    c. Uremia

NEUROLOGIC DISEASE

Meningitis

Emotional Stress

MISCELLANEOUS CAUSES

During Anesthesia

Drugs & chemicals

a) Alcohol
b) Smoking
c) Coffee
d) Digitalis
e) Adrenaline

Vomiting spells

Lone Atrial Fibrillation

RHEUMATIC HEART DISEASE

Causes of Mitral, Tricuspid Valve disease is frequently associated with AF because,

1. Enlargement of Atria
2. Increased Atrial conduction time
3. Difference refractoriness of Atrial myocardium
4. Atrial ectopic

Rheumatic heart disease especially mitral stenosis is particularly important as it increases the risk of embolisation in patients with chronic AF 5 fold. Transthoracic echo may show left atrial thrombus, but transesophagial Echo is more sensitive than this.[9]

CORONARY ARTERY HEART DISEASE

CAHD is the most common cause of AF in western countries. In addition fast ventricular rate due to fibrillation may cause angina, leading to cardiac ischaemia and heart failure.[1]

AF may complicate MI and as a marker of extensive myocardial infarction and poor prognosis. If it occurs within 24 hours if an infarct it is usually self-limiting. AF is also a marker of underlying ventricular dysfunction. Many years after MI ventricular scaring and dilatation predispose to AF / CCF.

Hypertensive heart disease

Hypertension accounted for about 1/3 of the cases in Framingham study. Hypertension contributes to the complication of stroke.[15]

AF may be due to LA dilatation, which occurs in SHT and decreased LV compliance. In additions SHT is associated with CAHD which is an add on feature for AF.

Congestive Cardiac Failure

CCF from any cause may predispose to AF. This favoured by associated enlarged atria, and the consequent distention of atrial walls.

Congenital heart Disease

AF is common in two forms of congenital heart disease – ASD, Ebsteins anomaly.

Cardiac Surgery

AF is the most frequent atrial arrhythmia noted after cardiac surgery. Patients undergoing CABG is one of the group to have one episode of AF in the post OP period. Post OP discontinuation of Beta blockers taken regularly before surgery increases the risk of AF.

Hyperthyroidism

AF is not uncommon in thyrotaoxicosis.

This is an important curable cause of AF. About 10 – 15% of patients with untreated thyrotoxicosis develop AF. It is more in males, and increased with age.

Thyroid affects the circulatory system and myocardium. There appears to be an interaction between the excess thyroid hormone and catacholamine action, including potentiation of catacholamine effects and increase in number of cardiac Beta-receptors.[3]

NEUROGENIC CAUSE OF AF

AF is facilitated by increased sympathetic tone. Atrial refractory period is decreased by Acetylcholine.

Adregenic form of AF has also been described the characteristic are:

1. Onset during day time
2. Preceded by emotional stimulus
3. Exercise

EXCESS ALCOHOL INTAKE

AF due to an excess alcohol often occurs after holidays or at week ends. (**Holiday heart Synd)**

Alcohol thus precipitates AF in health people and chronic excess intake of alcohol can be associated with a dilated heart. (Alcohol heat muscle disease) and AF.

PNEUMONIA

Pneumonia is commonly associated with AF in 7% cases of pneumonia as a precipitation of AF and occurs predominantly in elderly patients.

IDIOPATHIC / LONE AF

Some patients with AF have not predisposing factor or Cardiac lesion. The condition in this patient is classified as lone or idiopathic AF.

FAMILIAL AF

Extremely uncommon, is seen in relatively young age less than 20 years. The gene responsible for AF was located on chromosome 10q in the region of 10q 22.

CLINICAL FEATURES OF AF

The haemodynamic consequences of AF are due to two factors.

1. Loss of Atrial systole – may impair Ventricular filling in the non-complaint ventricle e.g.: AS / LVH

2. A rapid ventricular rate will encroach upon the diastolic filling period of the Left ventricle and the diastolic flow time of the coronary time.

ATRIAL CONTRACTION IMPORTANT	DIASTOLIC CONTRACTION IMPORTANT
Aortic Stenosis	Mitral stenosis
Hyp. Cardiomyopathy	CAHD
SHT / LVH	DCMP
Restrictive Cardiomyopathy	WPW syndrome
DCMP / CCF	Nodal Conduction

The symptoms are:

1. Palpitation
2. Dyspnoea
3. Fatigue
4. Angina
5. Presyncope
6. Syncope

Physical Findings

Pulse is irregularly irregular.

Absence of ‘a’ waves in the “JVP”

An Apex – radial pulse deficit appears, as each contraction may not be sufficiently strong to transmit an arterial pulse to the peripheral artery.

Cardiac auscultation will reveal the Variable intensity of S1 due to fluctuating ventricular filling period and sign of underlying heart disease like MS.

COMPLICATIONS

Heart failure

The sudden onset of AF may precipitate overt hear failure particularly of LV functions is already compromised by co-existing heart disease. In HF patients AF may be a marker of increased mortality and may also enhance the substantial risk of embolisation.

THROMBOEMBOLISATION

AF predisposes to the formation of intracardiac thrombus, which may result in stroke, Thromboembolisastion. The commonest site of thrombus is LA appendage. RA thrombus with pulmonary embolisation is a rare complication.

STROKE

The following factors are associated with high risk stroke in patients with AF[11].

1. Age > 65 yrs
2. SHT
3. RHD

4. Prior Stroke / TIA
5. DM
6. CCF
7. LA dimension > 50mm

INVESTIGATIONS

ECG

AF should be documented fully with a conventional 12 lead ECG. This may provide a clue to the etiology or electrophysiological features that may cause this arrhythmias e.g. : IHD / LVH.

It was thought that coarse fibrillating waves favor the etiology of RHD or hyperthyroidism while fine fibrillating waves are associated with SHT. This not true.

A 24 hours holter monitoring may be needed to document paroxysmal atrial fibrillation or sick sinus syndrome.

CHEST RADIOGRAPHY

A chest X ray is useful in most cases of AF. In a young patient it may provide a clue to congenital heart disease such as ASD.

X ray film can given information regarding the size of the heart, whether the patient has CCF.

BLOOD TEST

Full blood counts: Especially if anticoagulants are indicated
Urea, Electrolytes
Thyroid function test
ESR /ASO – titer – Rheumatic Fever
MI – Enzyme studies – CPK, CPK-MB, Troponin–T

ECHO

An Echo is an important test to obtain in patients with Atrial Fibrillation. It allows the evaluation of Atrial, R/L, Ventricular function. Presence of congenital and valvular lesions.

Transesophageal echo is valuable to investigate atrial thrombi prior to cardio version.

EXERCISE TESTING

Exercise testing is necessary in some patients with IHD and AF to clarify the seriosity of underlying cardiac ischaemia.

ELECTROPHYSIOLOGICAL STUDY

In patients with AF due to prexicitation syndrome, electrophysiological studies may be needed to document the characteristic of conduction from the atria to the ventricles and the presence of accessory pathway. This may lead to a cure of the conduction by transcathedral ablation of accessory pathway[9].

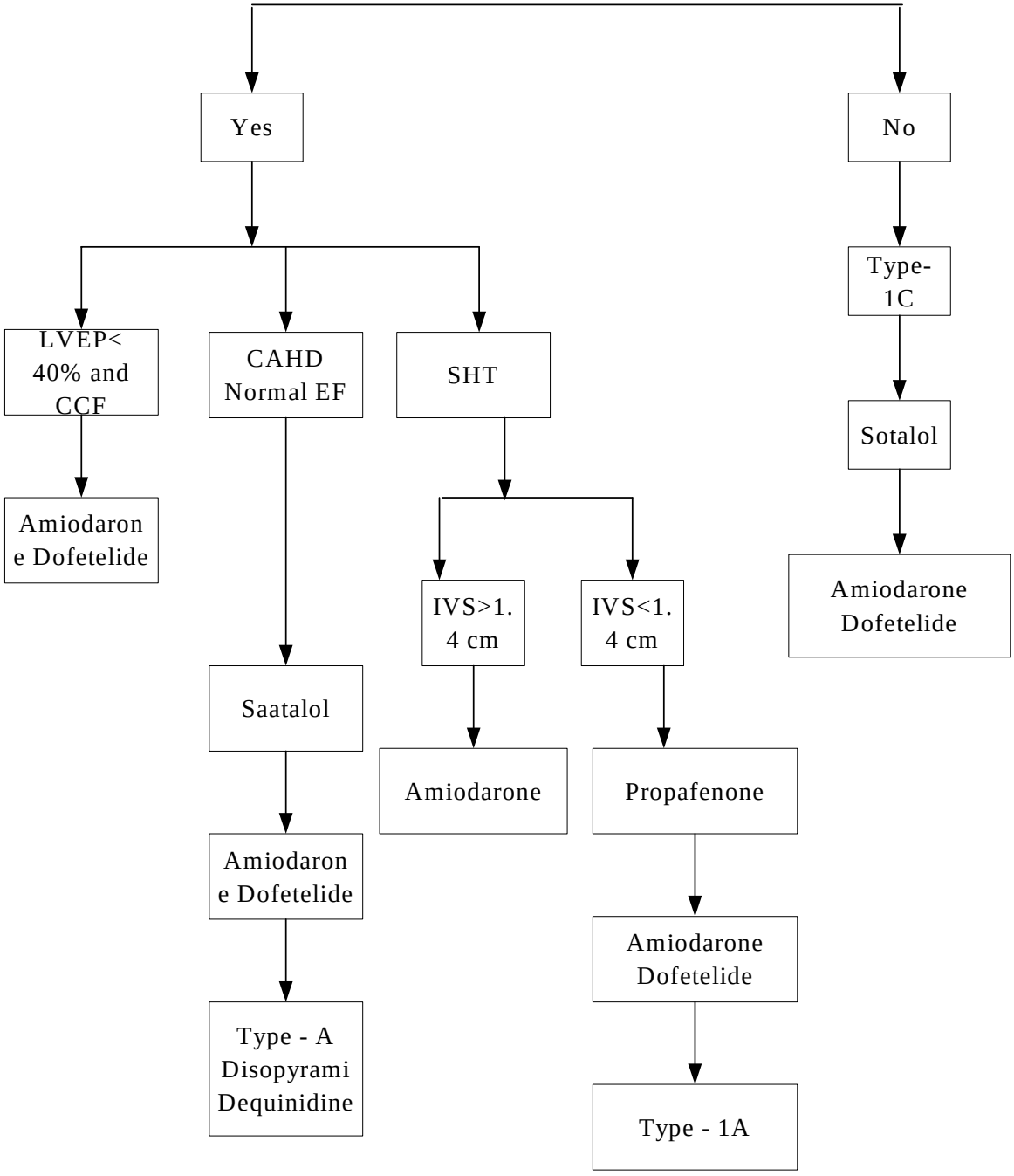
MANAGEMENT OF ATRIAL FIBRILLATION

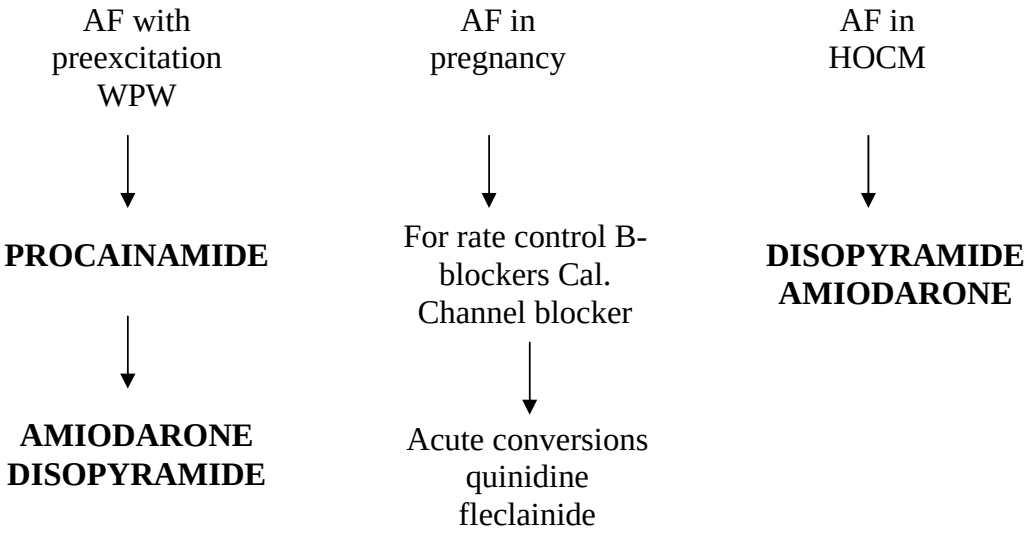
Management of AF includes following strategies

- Identification and treatment of the underlying reversible causes like hypoxaemia with acidosis in patients with acute respiratory failure, hyperthyroidism and carbon monoxide poisoning.
- Anticoagulation
- Attempt to convert it to sinus rhythm and maintain it
- Attempt to slow the ventricular rate by using AV nodal blocking drugs and in drug refractory cases by AV nodal ablation followed by placement of a pacemaker while allowing the arrhythmia to continue.

The following are the recommendations for treatment for Atrial Fibrillation.

STRUCTURAL HEART DISEASE





ACUTE TREATMENT OF ATRIAL FIBRILLATION

Pharmacological cardio version appears to be most effective with in 7 days after the AF especially within 48 hours.

If AF with haemodynamic compromose, ongoing angina – electrical cardio version is preferable[9].

If electrical cardio version is fails to restore, sinus rate, pre treatment with anti arrhythmic like ibutulide will decrease the atrial defibrillation threshold.

GUIDELINES FOR THROMBOTIC THERAPY IN AF

Based on a guideline formed by the 6th consensuses Conference of Anti Thrombotic therapy of the American College of Chest Physicians.

HIGH RISK

H/o Prior stroke / TIA	Anti-coagulants
Poor LV function	Maintain INR 2 – 3
Age 71 years	± Aspirin
Rheumatic Mitral Valvular Disease	

MODERATE RISK

65–75 Yrs DM/CAHD	Anti coagulant maintain INR .5– 2.5

LOW RISK

< 65 years	Aspirin alone
No clinical / ECHO	
Evidence of CVS disease	

ASPIRIN Vs ANTI COAGULATION IN PATIENTS WITH HIGH RISK CATEGORY OF AF

SPAF – I trial (stroke prevention in AF) found that Anti Coagulation was significantly more effective than aspirin in decreasing the rate of all strokes and other cardiovascular events.

ANTI COAGULATION COMPARED WITH ANTI COAGULATION WITH ASPIRIN

NASPEAF (National Study of Prevention of Embolism in AF) compared the efficacy of Aspirin + anti coagulation (Target INR 1.5 – 2.4) Vs anti coagulation targeted at INR (2 – 3) preliminary reports suggested that the former is more effective than the later.

RATE Vs RHYTHM CONTROL

PIAF trial (Pharmacological intervention in AF) has come with the conclusion.

1. Quality of life is same in both
2. Exercise tolerance is better in rhythm controlled patients.
3. Hospital admission and adverse effects are high in rhythm controlled patients.

Non-pharmacological methods for treating atrial fibrillation

- Rhythm control strategies
    - Surgical ablation
    - Catheter ablation
    - Single and multiple atrial pacing

- Atrial defibrillators

- Rate control strategies
  - AV node function modification
  - AV node ablation and permanent pacing

$CHADS_2$ INDEX (Stroke Risk in Patients with Nonvalvular AF Not Treated with Antiocoagulation)

$CHADS_2$ risk	**Score**
Cardiac tailure	1
Hypertension	1
Age > 75 Yrs	1
Diabetes mellitus	1
Prior stroke or TIA	2

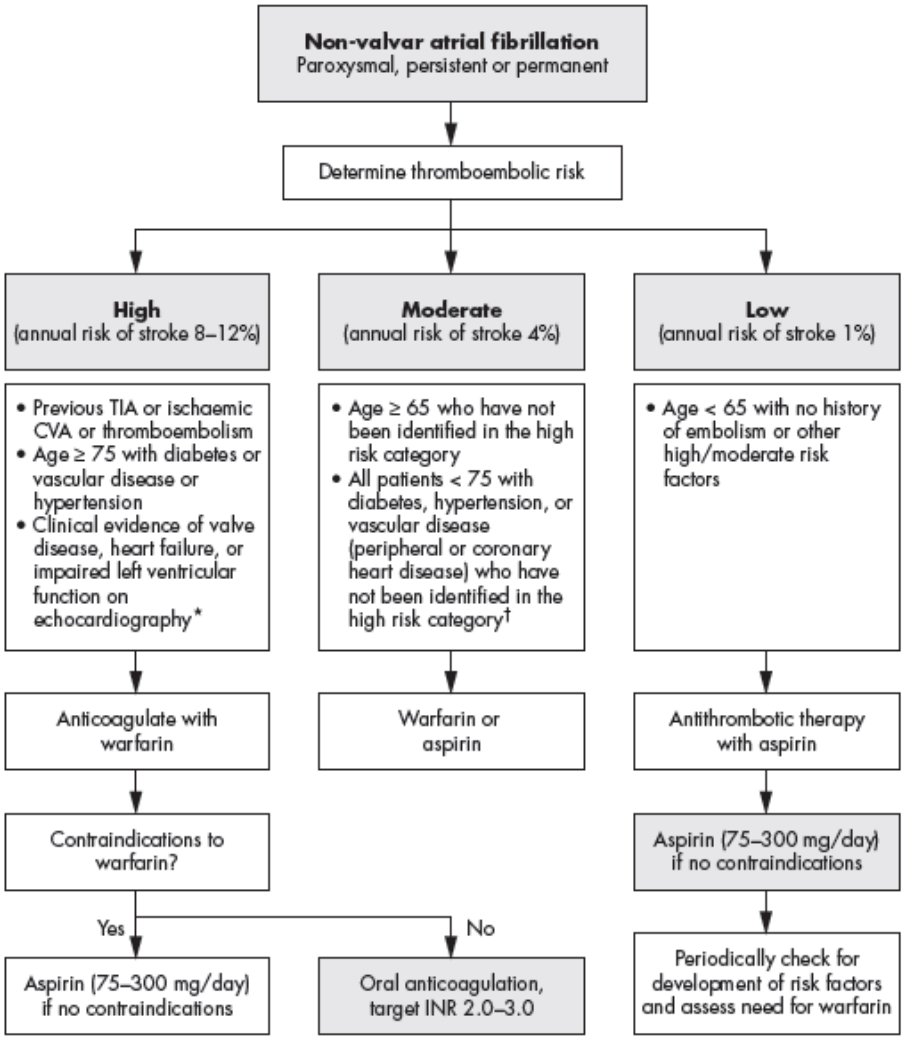
Risk category and recommended anti thrombotic therapy according to $CHADS_2$ score

CHADS Score	Risk category	Recommended anti thrombotic therapy
0	Low	Aspirin 81-325 mg PO daily
1	Moderate	Aspirin or warfarin
2	Moderate	Previous CVA/TIA/embolism/ - Yes = Warfarian - No = Aspirin or warfarian
3	High	Warfarin (INR 2.0 – 3.0)
4	High	
5	High	
6	High	

Appropriate anticoagulation in various AF populations

Patients features	Antithrombotic therapy
Age<60 yr, no heart disease (lone AF)	Aspirin (81-325 mg/d) or no therapy
Age<60 yr, heart disease but no RFs	Aspirin (81-325 mg/d)
Age 60-74 yr, no RFs	Aspirin (81-325 mg/d)
Age 65-74 yr with diabetes mellitus or CAD	Oral anticoagulation (INR 2.0-3.0)
Age 75 yr or older, women	Oral anticoagulation (INR 2.0-3.0)
Age 75 yr or older, men, no other RFs	Oral anticoagulation (INR 2.0-3.0 or aspirin (81-325 mg/d)
Age 65 or older, heart failure	Oral anticoagulation (INR 2.0-3.0)
LV ejection fraction <35% or fractional shortening <25% and hypertension	Oral anticoagulation (INR 2.0-3.0)
Rheumatic heart disease (mitral stenosis)	Oral anticoagulation (INR 2.0-3.0)
Prosthetic heart valves; prior thromboembolism	Oral anticoagulation (INR 2.0-3.0 or higher)
Persistent atrial thrombus on TEE	Oral anticoagulation (INR 2.0-3.0)

Algorithm for ANTITHROMBOTIC THEARPY IN NON VALVULAR AF



MATERIAL AND METHODS

This study was conducted at Government Stanley Medical College Hospital, Chennai during the period of January 2007 to June2007. Fifty cases of patients admitted with atrial fibrillation were recorded. No patient had been counted to if he/she got admitted again after discharge. Paediatric age group (13 Yrs and Less) was not included in this study.

The Diagnosis of AF:

This was made on clinical grounds and confirmed by ECG / ECHO.

Clinical Grounds:

Irregularly irregular pulse:

If the patient was not in failure, he or she was exercised and the persistence of the irregularity noted.

Pulse deficit:

Simultaneous counting of the pulse rates by one observer and the heart rate by another for one full minute.

Absence of “a” waves in JVP.

On Auscultation varying intensity of SI.

ECG Recording

A 12 lead ECG was taken for all the cases. It was standardized to produce a deflection of 10 mm/1 mv input and paper speed was set at 25 mm/Sec. The ECG features of AF are:

1. Absence of regular rhythm / p waves.
2. Atrial Activity reflected by a variable irregularity compounded deflection forming the base line “f” waves.
3. Marked variation in ‘RR’ interval.
4. Variation in QRS complex configuration in ECG.

Other findings like Ventricular Enlargement RVE, RBBB, evidence of ischaemia/infarction also noted.

In all cases complete history was taken and general examination done. Further the patients was subjected the investigations according to the suspected etiology.

Rheumatic Heart Disease

1. Features of RHD
2. Features of CCF
3. Presence of Valvular heart disease
4. Features of Infective Endocarditis
5. Chest X-Ray PA view
6. CT Scan for any embolic complication
7. ECHO

CORONARY ARTERY HEART DISEASE:

1. History / ECG
2. HT / DM
3. Auscultation for S3 / S4
4. Ophthalmic Examination
5. Chest X-ray PA view for cardiomegaly / Pulmonary congestion
6. Serum enzymes – CPK – CPK-MB in case of infarction

Systemic Hypertension:

1. Blood Pressure
2. Fundus examination
3. ECG for LVH, LVE, LAE
4. Chest X-ray PA view – Cardiomegaly
5. Urine analysis
6. Urea – Creatinine – Sugar

CHEST X-RAY PA VIEW

Chest X-ray PA view was taken and evaluated for evidence of valvular heart disease, congenital heart disease, pericardial effusion, COPD, pneumonia, etc.,

ECHO

M-mode, 2 D ECHO was done for all the patients and the presence of thickening of the valves, calcification, and valve closure were noted. The size of the valve ring and the chamber of the heart were noted. The presence of clot in the atria/atrial appendage was also noted.

Diagnosis of Complication of AF

AF complicating CCF, complete history, examination done. ECG, Chest X-ray PA for cardiomegaly and pulmonary congestion. ECHO evaluation of ventricular function was noted.

AF complicating stroke patient neurological examination done. CT scan brain taken and the feature were noted. The presence of thrombus in atria using ECHO noted.

OBSERVATIONS

TABLE 1

AETIOLOGY

CAUSES	NUMBER OF CASES	%
Rheumatic Heart Disease	30	60%
Ischaemic Heart Disease	8	16%
Cardiomyopathy (Dilated)	8	16%
Syst hypertension	2	4%
Misc. Holiday heart synd	2	4%

TABLE 2

AGE DISTRIBUTIONS

CAUSES	NUMBER OF CASES	%
13 – 20	--	0%
21 – 30	10	20%
31 – 40	15	30%
41 – 50	9	18%
51 – 60	13	26%
61 and above	3	6%

TABLE 3

SEX DISTRIBUTIONS

AETIOLOGY	MALES	FEMALES
Rheumatic Heart Disease	19	11
Ischaemic Heart Disease	5	3
Cardiomyopathy	--	8
Systemic Hypertension	1	1
Miscellaneous	2	--
%	54%	46%

TABLE 4

AGE AND ETIOLOGICAL INCIDENCES

AETIOLOGY	AGE	
	RANGE	MEAN
Rheumatic Heart Disease	21 – 60 Yrs	35.7 yrs
Ischaemic Heart Disease	50 – 67 Yrs	58.3 yrs
Cardiomyopathy	42 – 58 Yrs	52 yrs
Systemic Hypertension	44 – 52 Yrs	48 yrs

TABLE 5

TYPES OF VALVULAR LESION CAUSING RHEUMATIC HEART DISEASE WITH ATRIAL FIBRILLATION

Valvular Lesion	Number of cases	%
Rheumatic M.S.	11	37%
MS + MR	15	50%
MS + MR + AS + AR	4	13%

TABLE 6

CLINICAL MANIFESTATION OF ATRIAL FIBRILLATION

Clinical Manifestation	Number of cases	%
PALPITATIONS	40	80%
DYSPNOEA	30	60%
ANGINA	7	14%
FATIQUE	5	10%
SYNCOPE	4	8%

This table is taken into consideration the fact that persons with palpitation also has co-existing other clinical manifestation like Dyspnoea/Angina.

TABLE 7

LA SIZE IN PATIENTS WITH RHD WITH ATRIAL FIBRILLATION

LA SIZE	NUMBER OF CASES	%
3 – 3.5 cm	2	6%
3.5 – 4 cm	4	14%
> 4 cm	24	80%

TABLE 8

COMPLICATION OF ATRIAL FIBRILLATION

COMPLICATION	NUMBER OF CASES	%
Precipitation of cardiac failure	40	80%
Stroke (Thromboembolic Complication)	3	6%
Angina	7	14%

Atril fibrillation per se does not cause cardiac failure in a majority of case but it precipitates failure in a heart which is compromised haemodynamically due to rheumatic heart disease or when the pumping action is compromised in case of DCMP and CAHD.

DISCUSSION

ETIOLOGY

In our study, we found that the commonest etiology causing atrial fibrillation was rheumatic heart disease, which constituted 60% of the cases followed by dilated cardiomyopathy. This is in sharp contrast to the study by WARREN J.MANNING, STEPHENGORDON (NEJM volume 328 March 1993) were hypertension was the commonest etiology contributing 46%. Rheumatic Heart Disease contributes to only 4% of the cases. But our study coincided a study from Ethiopia in which rheumataic heart disease contributes about 66% of the cases.

AGE/SEX DISTRIBUTION

In our study, we found that the majority of the cases were in the 4^{th} and 5^{th} decade, but in Framingham study and study by GODTFREDSENJ (AF, molndal Sweden: AB hassle 1982) which was common in the 6^{th} and 7^{th} decade. This can be attributed to the fact that rheumatic heart disease accounted for majority of the cases in our study. And also in our study there was no major sex difference in the distribution of atrial fibrillation.

In our study atrial fibrillation in rheumatic heart disease occurred at a mean age of 35.7 years. This is low when compared to the Framingham study in which the mean age was in the 6^{th} decade.

VALVULAR LESIONS IN RHEUMATIC HEART DISEASE WITH ATRIAL FIBRILLATION

In our study, the predominant lesion in rheumatic heart disease causing atrial fibrillation was mitral valvular disease. This correlates well with the Framingham study.

LEFT ATRIAL SIZE IN PATIENTS WITH RHEUMATIC HEART DISEASE WITH ATRIAL FIBRILLATION

In out study, it was found that the majority of the cases of rheumatic heart disease with atrial fibrillation had a left atrial diameter of greater than 4 cm. This coincides with the study by SANOOP, JOHNSON (INDIAN HEART JOURNAL Sep 2003) in which atrial fibrillation was common when the left atrial size was above 4 cm.

COMPLICATIONS OF ATRIAL FIBRILLATION

In our study, the precipitation of cardiac failure was the major complication constituting to 80% of the cases. This is due to the fact that atrial fibrillation precipitates heart failure in a haemodynamically compromised state as in rheumatic heart disease due to structural abnormality and compromise in pumping action in case of DCMP and CAHD. Since the majority of the cases in our study come under the above category precipitation of covert cardiac failure was the major complication. Atrial fibrillation per se only contributes little to cardiac failure. The % of stroke was 6%, this correlated with the study by p.Peterson and godtfredson (European heart journal Vol.9 No.3 March 1998).

CONCLUSION

- The Commonest etiology causing atrial fibrillation was Rhenmatic heart disease, which contributed to 60% of the cases.

- The majority of the cases of Atrial fibrillation were in the 4^{th} or 5^{th} decade.

- There were no significant sex differences in the distribution of Atrial fibrillation but in Dilated cardiomyopathy had a predominant female preponderance.

- Rheumatic heart disease patients with atrial fibrillation had a mean of 35.7 years, while patients with ischaemic heart disease had a mean of 58.3 years.

- In Rheumatic heart disease the predominant lesion causing AF was mitral valve lesion.

- The commonest clinical manifestation causing Atrial fibrillation was palpitations followed by dyspnoea.

- A Left atrial size of >4 cm predisposes to Atrial fibrillation in Rheumatic Heart Disease.

- The major complication of atrial fibrillation was precipitation of cardiac failure.

- Atrial fibrillation perse does not cause cardiac failure in a majority of cases. But it may precipitate overt cardiac failure in a haemodynamically compromised heart as in rheumatic heard disease and pump failure as in DCMP and CHD. Since majority of the cases in the study were rheumatic heard disease precipitation of cardiac failure was the major complication.

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CASE REPORT 1

Name	:	Mr. Selvaraj
AGE / SEX	:	40 / M
I.P. No.	:	033540
Address	:	Redhills
Presenting Complaints	:	
Dyspnea	:	Yes. For the past 6 months
Palpitation	:	Yes. For the past 6 months
Chest Pain	:	No
Syncope	:	No
Weakness of limbs	:	No
Fatigue	:	No

Past History

DM	:	No
HTN	:	No
CAD	:	No
Hyperlipedemia	:	No
COPD/BA	:	No
RHD	:	Yes
Treatment History	:	On irregular penicillin prophylaxis

General Examination:

Consciousness	:	Conscious
Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy:	:	No
Pedal Edema	:	Yes
JVP	:	Normal

Vitals:

Pulse Rate	:	60/mt Heart Rate 90/mt
Pulse Deficit	:	30/mt
BP	:	90/70 mmHg
Temperature	:	98.4^{o}F
Respiratory Rate	:	18/min

Examination of Systems:

CVS	:	S1-varying intensity S2-loud pulmonary component No - S3, S4 Murmurs–Mid-Diastolic and Pasystolic murmur in mitral area
RS	:	Normal vesicular breath sounds
CNS	:	Clinically normal
Abd	:	Soft, hepatomegaly present

Investigations

Labs	:	
CBC	:	Normal
RFT	:	Normal
LFT	:	Normal
Sr. Electrolytes	:	Normal
Free T4 and TSH	:	
ECG	:	'P' waves absent, varying RR Interval Fibrillatory waves present
ECHO		
Valvular Abnormality	:	Rheumatic Heart Disease, Severe (MS), Mod (MR)

LA dilatation	:	LA size 4.8 cm
LA clot / Vegetation	:	No Calcified Mitral valve. EF 42%
X-Ray Chest	:	C-T ratio 60%. LA Enlarged.
		Right ventricular configuration. MPA prominent. Grade I pulmonary venous hypertension.
Diagnosis	:	RHEUMATIC MITRAL STENOSIS AND REGUGITATION WITH ATRIAL FIBRILLATION.

CASE REPORT 2

Name	:	Mr. Kannapan
AGE / SEX	:	55 / M
I.P. No.	:	026778
Address	:	Washermenpet

Presenting Complaints

Dyspnea	:	Yes. For the past 2 days
Palpitation	:	No
Chest Pain	:	Yes. For the past 2 days
Syncope	:	No
Weakness of limbs	:	No
Fatigue	:	Yes. For the past 1 day

Past History

DM	:	No
HTN	:	No
CAD	:	No
Hyperlipedemia	:	No
COPD/BA	:	No
RHD	:	No
Treatment History	:	No history of any intake of medications

General Examination:

Consciousness	:	Conscious
Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy:	:	No
Pedal Edema	:	No

JVP	:	Raised

Vitals:

Pulse Rate	:	68/mt Heart Rate 94/mt
Pulse Deficit	:	26/mt
BP	:	100/70 mmHg
Temperature	:	98.4^{o}F
Respiratory Rate	:	18/min

Examination of Systems

CVS	:	S1-varying intensity S2-heard; S3-heard S4-No
RS	:	Normal vesicular breath sounds
CNS	:	Clinically normal
Abdomen	:	Soft, No organomegaly

Investigations

Labs	:	
CBC	:	Normal
RFT	:	Normal
LFT	:	Normal
Sr. Electrolytes	:	Normal
Free T4 and TSH	:	Normal
ECG	:	ST elevation in II, III, AVF ST **de**pression in L1, A**VL**, V2-V6 Varying RR interval Fibrillatory waves seen Controlled ventricular response

ECHO	:	Severe hypokinesia of inferior wall and LV posterior wall. EF 40%. No clots.
X-Ray Chest	:	Normal
Diagnosis	:	INFERIOR WALL MYOCARDIAL INFARCTION WITH ATRIAL FIBRILLATION

CASE REPORT 3

Name	:	Mrs. Raggamal
AGE / SEX	:	50 / F
I.P. No.	:	033640
Address	:	Bharathi Nagar
Presenting Complaints		
Dyspnea	:	Yes. For the past 5 months
Palpitation	:	No
Chest Pain	:	Yes
Syncope	:	No
Weakness of limbs	:	No
Fatigue	:	Yes. For the past 6 months
Past History		
DM	:	Known Diabetic for 5 years. On T.Glibenclamide 5mg 1 bd
HTN	:	No
CAD	:	No
Hyperlipedemia	:	No
COPD/BA	:	No
RHD	:	No
Treatment History	:	Known case of DCMP on Aspirin and Digoxin and Frusemide
General Examination:		
Consciousness	:	Conscious
Pallor, Icterus, Clubbing, Cyanosis,	:	No

Lymphadenopathy:		
Pedal Edema	:	Yes
JVP	:	Raised

Vitals:

Pulse Rate	:	154/mt Heart Rate 194/mt
Pulse Deficit	:	40/mt
BP	:	90/60 mmHg
Temperature	:	98.4^{o}F
Respiratory Rate	:	20/min

Examination of Systems:

CVS	:	S1-varying intensity S2-heard; S3, S4- No Murmurs–Pansystolic murmur in Mitral area.
RS	:	Normal vesicular breath sounds heard. Basal crepitations heard
CNS	:	Clinically normal
Abdomen	:	Soft, Hopatomegaly present

Investigations

Labs	:	
CBC	:	Normal
RFT	:	Normal
LFT	:	Normal
Sr. Electrolytes	:	Normal
Free T4 and TSH	:	Normal

ECG	:	Absent p waves/varying RR Interval. Rapid ventricular response
ECHO	:	Dilated cardiomyopathy. EF 30%. No clots. Moderate MR present
X-Ray Chest	:	Cardiomegaly Grade I Pulmonary Venous Hypertension
Diagnosis	:	DILATED CARDIOMYOPATHY WITH ATRIAL FIBRILLATION

CASE REPORT 4

Name	:	Vasanth
AGE / SEX	:	67 / Male
I.P. No.	:	036230
Address	:	Mannadi
Presenting Complaints	:	
Dyspnea	:	Yes. For the past 2 days
Palpitation	:	Yes. For the past 2 days
Chest Pain	:	Yes. For the past 1 day
Syncope	:	No
Fatigue	:	No.
Weakness of limbs	:	No.

Past History

DM	:	Known Diabetic for past 10 years
HTN	:	No
CAD	:	No
Hyperlipedemia	:	No
COPD/BA	:	No
RHD	:	No
Treatment History	:	Oral Metformin 500 mg bid

General Examination:

Consciousness	:	Conscious
Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy:	:	No
Pedal Edema	:	No
JVP	:	Raised

Vitals:

Pulse Rate	:	80/mt Heart Rate 110/mt
Pulse Deficit	:	30/mt
BP	:	94/60 mmHg
Temperature	:	98.4^{o}F
Respiratory Rate	:	18/min

Examination of Systems:

CVS	:	S1-varying intensity S2-heard; S3-heard S4-No Murmur - No
RS	:	Normal vesicular breath sounds. Basal crepitations present.
CNS	:	Clinically normal
Abdomen	:	Soft, No organomegaly

Investigations

Labs	:	
CBC	:	Normal : CPK-MB raised.
RFT	:	Normal
LFT	:	Normal
Sr. Electrolytes	:	Normal
Free T4 and TSH	:	Normal
ECG	:	ST elevation in V1-V6 Absent p waves Varying RR interval. Non progression of r wave in V1–V5

ECHO	:	Regional wall motion abnormalitay present. Severe hypokinesia of lower 2/3 of IVS, LV anterior wall, LV Apex. EF. 36%
X-Ray Chest	:	Cardiac shadow normal. Round glass appearance of both lung field seen
Diagnosis	:	ACUTE ANTERIOR WALL MYOCARDIAL INFARCTION WITH ATRIAL FIBRILLATION

CASE REPORT 5

Name	:	Mr. Sekar
AGE / SEX	:	27 / M
I.P. No.	:	020629
Address	:	Thiruvotriyur

Presenting Complaints

Dyspnea	:	No.
Palpitation	:	Yes. For the past 8 months
Chest Pain	:	No
Syncope	:	No
Fatigue	:	No.
Weakness of limbs	:	No.

Past History

DM	:	No
HTN	:	No
CAD	:	No
Hyperlipedemia	:	No
COPD/BA	:	No
RHD	:	Known case of Rheumatic Heart disease on penicillin prophylaxis

General Examination:

Consciousness	:	Conscious
Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy:	:	
JVP	:	Raised

Vitals:

Pulse Rate	:	98/mt
Pulse Deficit	:	34/mt
BP	:	100/70 mmHg
Temperature	:	$98.4^{o}F$
Respiratory Rate	:	18/min

Examination of Systems:

CVS	:	S1-varying intensity S2-Loud; S3, S4 – No Murmurs-Mid diastolic and pan systolic murmurs heard
RS	:	Normal vesicular breath sounds heard.
CNS	:	Clinically normal

Investigations

Labs	:	
CBC	:	Normal
RFT	:	Normal
LFT	:	Normal
Sr. Electrolytes	:	Normal
Free T4 and TSH	:	Normal
ECG	:	Absent p waves/varying RR Interval. Fibrillatory waves present. Rapid ventricular response
ECHO	:	Rheumatic heart disease Mitral stenosis and mitral regurgitation. EF. 56%. No clots.

X-Ray Chest	:	Cardiomegaly present Straightening of left heart border. Rvapex Grade I Pulmonary Venous Hypertension
Diagnosis	:	RHEUMATIC MITRAL STENOSIS AND REGURGITATION WITH ATRIAL FIBRILLATION

PROFORMA

CLINICAL PROFILE OF ATRIAL FIBRILLATION

Name :

AGE / SEX :

I.P. No. :

Presenting Complaints :

Shortness of Breath :

Palpitation :

Chest Pain :

Syncope :

Fatigue :

Weakness of limbs :

Past History

DM :

HTN :

CAD :

Hyperlipedemia :

COPD/BA :

RHD :

Treatment History

General Examination:

Consciousness :

Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy :

Pedal Edema :

JVP :

Vitals:

Pulse Rate	:	
Pulse Deficit	:	
BP	:	
Temperature	:	
Respiratory Rate	:	

Examination of Systems:

CVS	:	S1 S2 S3 S4 Murmurs
RS	:	
Abdomen	:	
CNS	:	

Investigation

LABS	:	
CBC	:	
RFT	:	
LFT	:	
Sr. Electrolytes	:	
Free T4 and TSH	:	
ECG	:	
ECHO	:	
Valvular Abnormality	:	
LA dilatation	:	
LA clot / Vegetation	:	
X-Ray Chest	:	
DIAGNOSIS	:	

ABBREVIATIONS

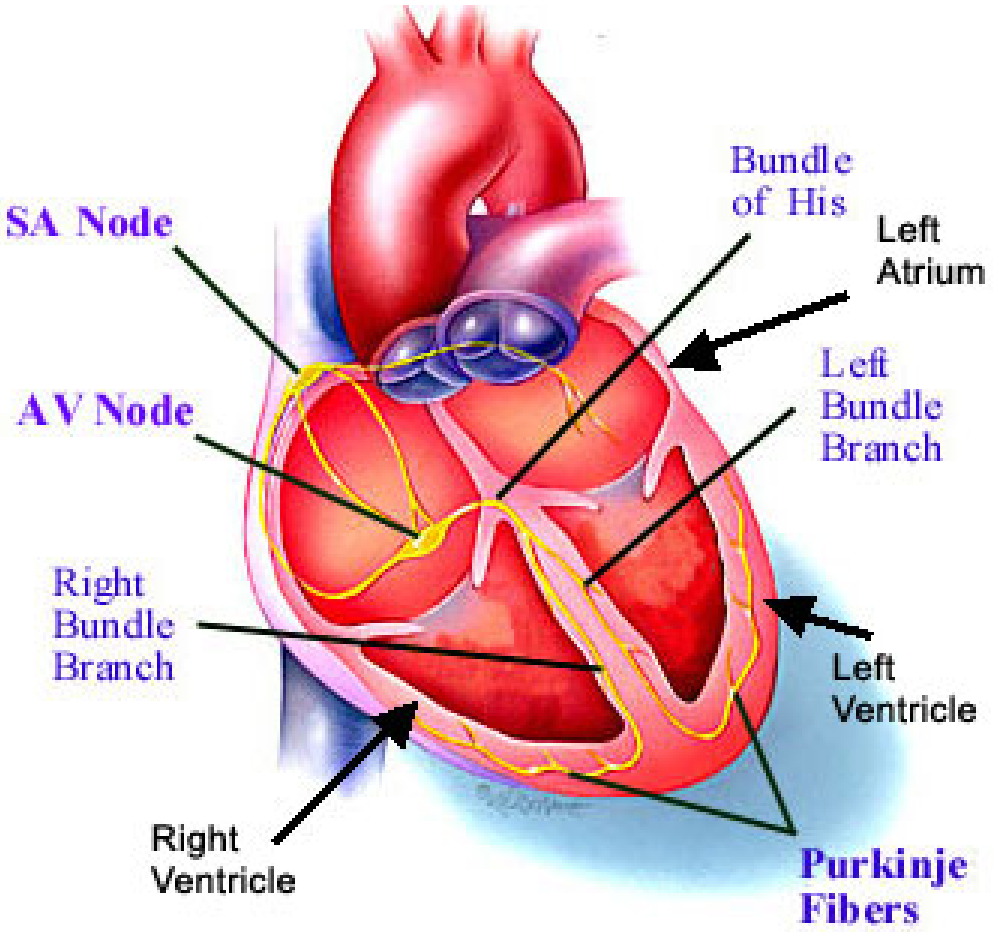
SHT	-	Systemic hypertension
VT	-	Ventricular tachycardia
RBBB	-	Right bundle block
LBBB	-	Left bundle block
CCF	-	Congestive cardiac failure
DCMP	-	Dialated cardiomyopathy
TIA	-	Transient ishemic attack
CVA	-	Cerebro vascular accident
AF	-	Atrial fibrillation
CAHD	-	Coronary artery disease
LV	-	Left ventricle
LA	-	Left atrium
RF	-	Risk factor
INR	-	International normalized radio
MS	-	Mitral stenosis
MR	-	Mitral regurgitation
AS	-	Aortic stenosis
AR	-	Aortic regurgitation
WPW	-	Wolf parkinson white syndrome
HOCM	-	Hypertrophic obstructive cardiomyopathy
JVP	-	Jugular venous pressure

BP	-	Blood pressure
ECG	-	Electro cardiogram
CABG	-	Coronary artery bypass grafting
ASD	-	Atrial septal defect
DM	-	Diabetes mellitus
LVH	-	Left ventricular hypertrophy
COPD	-	Chronic obstructive disease
BA	-	Bronchial asthma
CVS	-	Cardio vascular system
RS	-	Respiratory system
CNS	-	Central nervous system
CBC	-	Complete blood count
RFT	-	Renal function test
LFT	-	Liver function test
TSH	-	Thyroid stimulating hormone

KEY TO MASTER CHART

N	-	Normal
PALP	-	Palpitations
DYS	-	Dyspnea
FAT	-	Fatigue
SYN	-	Syncope
ANG	-	Angina
JVP	-	Jugular venous pressure
LA	-	Left atrium
CM	-	Centimeter
RHD	-	Rheumatic heart disease
CVA	-	Cerebrovascular accident
MS	-	Mitral stenosis
MR	-	Mitral regurgitation
AS	-	Aortic stenosis
AR	-	Aortic regurgitation
DCMP	-	Dilated cardiomyopathy
SHT	-	Systemic hypertension
CAHD	-	Coronary artery heart disease

NORMAL CONDUCTION SYSTEM OF HEART



MULTIPLE FOCI (AREAS) SEND OUT IMPULSES WHICH CAUSES THE ATRIA TO FIBRILLATE