HEART FAILURE AFTER ACUTE MYOCARDIAL INFARCTION IN CCU.

THESIS

Submitted in Partial Fulfillment for the

Degree, Master of Cardiology and Vascular diseases

By

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1976

PREFACE

It has long been my contemplation to write a concise and yet a comprehensive thesis in this subject.

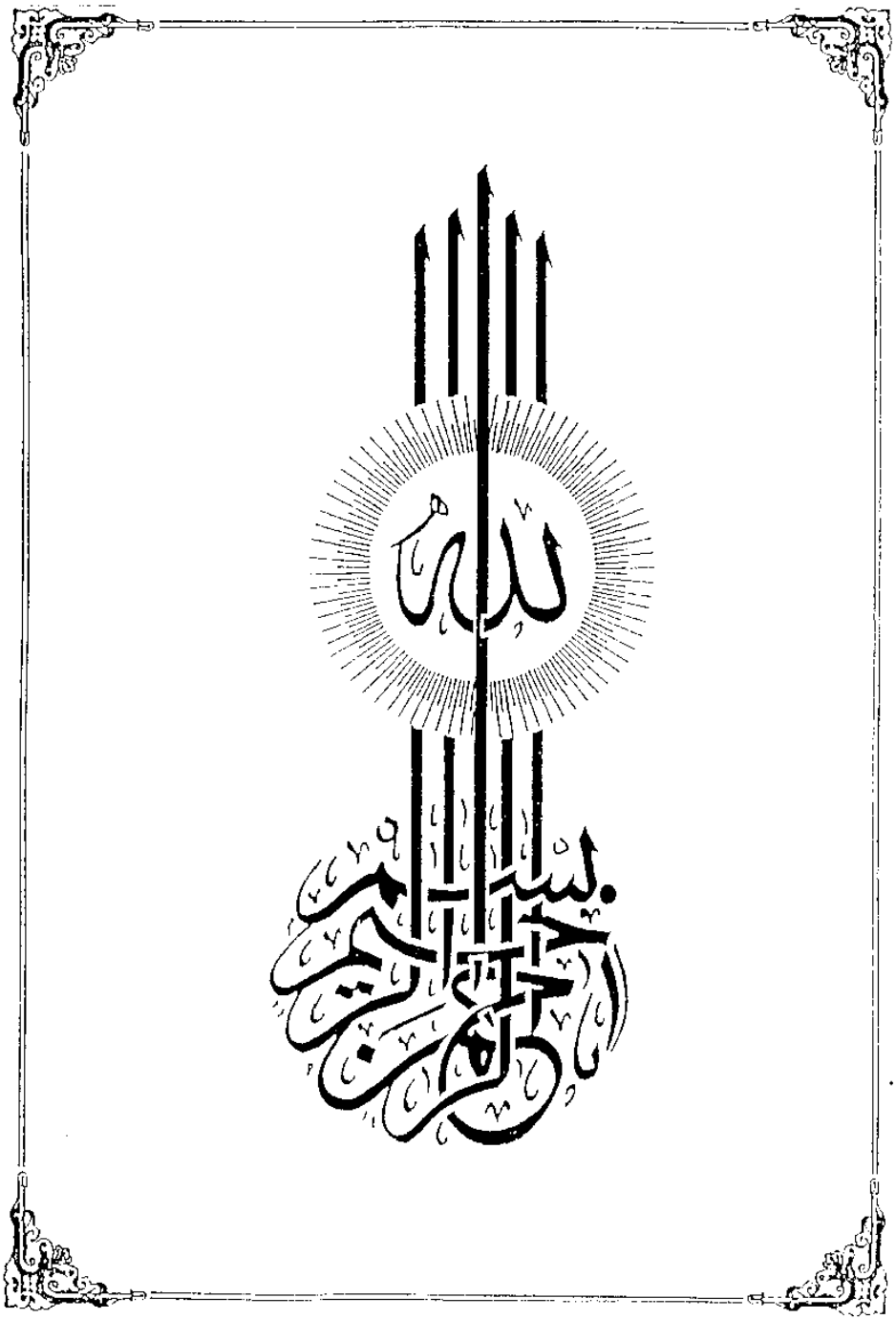
It is a tedious work and time consuming that one gets the information from large number of references to be fully assimilated.

I agree that many points are superficially touched to the extent that they are continously alarming me, but the fact that this thesis should be of moderate size keep me quiet well.

In collecting the material from the ten cases, constant observation and follow up were mandatory, but it is regrattable that many of the investigations were not done totally or at the critical time making one to lay stress on clinical judgement and the experience of those who teach me.

I hope that the scope of this thesis serves its aim of collecting materials from references and patients.

It is a pleasure to acknowledge the efforts of all those who have helped in bringing this work to light. I would like to express my grateful thanks to prof.G.M. Ziady who is a constant faithful supervisor, he inspired me and is wellcomed for any inquiry, to him I tender my best thanks



I should like to take this oppartunity of thanking those teachers of cardiology, Prof. A. Eisa, prof. M. Ateia, Prof. H. El-Demerdash Prof. M. Dayem, Prof. H. Ezz Eddin & Prof. A. Ramzi.

It is a great pleasure to acknowledge the help received from my colleagues who contributed so much, in directing me to sound way and in evaluating physical signs and laboratory data, without them no full material regarding the cases could be collected. To them I tender my best thanks.

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Cairo

July, 1976

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CHAPTER I

HEART FAILURE AFTER ACUTE MYOCARDIAL INFARCTION

Definition :

Voltaire said, "If you wish to confer with me, define your terms".

So to start, is to define the term heart failure.

In Osler's Textbook of Medicine, 1892 edition no special chapter on heart failure is found, but there is description of hypertrophy & diltation with their signs & symptons.

Osler wrote : "Extensive dilatation during severe muscular efferot results in heart strain.

For months such a man, after physical exercise, may be unfit for severe exertion or he may be permanantely incapacitated. In some way he has overstrained his heart and become broken winded". Osler continued "Exactly what has taken place in these hearts we cannot say, but their reserve force is lost and with it the power of meeting the demands in maintaining the circulation during sever exertion". Little was given by osler about coronary artery disease since he focused on valvular lesions as the underlying mechanism for dilatation.

In his book: "Mackenzie's Textbook of Heart Diseases", Mackenzie defined heart failure as inability of heart muscle to maintain the circulation and that this failure of heart muscle is due to the disturbance of the normal

adjustment of the various factors concerned in the circulation.

Recently the following definitions are applicable :

- Circulatory failure means inadequacy of the cardiovascular system of providing nutrition to the tissue cells and removing metabolic products from the cells. This may be caused primarily by either cardiac or peripheral (non cardiac) conditions.
- Cardiac Failure is a condition in which the heart is no longer able to pump an adequate supply of blood in relation to venous return and in relation to the metabolic needs of tissues of the body at that particular moment.

Harrison and Reeves defined heart failure as the condition where the heart is either unable to receive or to propel the blood that is offered it.

In most patients with clinical heart failure, cardiac output is decreased, although it can be normal or even increased. In the latter two situations, failure is presented whenever there is imbalance between the needs of the body and the supply of blood pumped by the heart. So conditions associated with high output failure, the cardiac output may be normal or higher than normal, but still it does not meet body's needs.

When abonrmal circulatory congestion occury as a result of heart failure, the term congestive heart failure is used. Congestion may be in pulmonary, systemic circulation or both, and must be of cardiac origin to validate the term CHF to distinguish it from circulatory overload or congestion e.g. increase blood volume, increase venous return ... etc. Secondary heart failure may occur in circulatary overload when there is discrepancy between supply of blood and needs of tissues.

In most instances congestive cardiac failure develops chronically with increase in total blood volume due to sodium and water retention by the kidney. Acute congestive heart failure may develop due to acute myocardial infarction of the left ventricle where there may be acute shift of blood from the systemic circulation to the pulmonary circulation due to venoconstriction before sodium and water retention occur.

- Forward failure is used to imply that most of the patient's symptoms resulted from low cardiac output, whereas backward failure has been used to imply that most of the patient's symptoms resulted from elevation of venous pressure behind the failing ventricle. Backward failure is usually chronic whereas forward

failure is usually acute. Both conditions usually coexist in patients with heart failure, this is exemplified in patient with cardiogenic shock complicating acute myocardial infarction. Clinically, these patients manifest a low cardiac output due to inadequacy of heart as a pump and a diminished peripheral blood flow. This state is usually accompained by manifestation of backward failure with congestion in both the pulmonary and systemic circularions, this is referred to power failure.

- Left heart failure and right heart failure are clinical terms where the primary impairment is of the left side of the heart or of the right side of the heart respectively. The most common cause of clinical right sided heart failure is left sided heart failure, since both sides of the heart are in a circuit and biochemical changes may contribute to contralateral ventricular failure.

Left sided failure is used in reference to symptoms and signs of elevated pressure and congestion in pulmonary veins and capillaries, right-sided failure is used in reference to symptoms and signs of elevated pressures and congestion in the systemic veins and capillaries.

- Latent heart failure is said to be present when it becomes apparent under only conditions of increased stress i. .

exercise, fever, emotion ... etc, but not apparent at rest.

- Compensated heart failure presents when failure is compensated as a result of normal compensatory mechanisms i.e. increased sympathetic dischange, increased blood volume or hypertrophy, as a result of improvement in myocardicil function due to administration of digitalis or by diuresis if fluid retention has been excessive. Diuretics relieve symptoms of congestion but seldom really compensate for ventricular function.

CHAPTER II

PHYSIOLOGICAL ASPECTS OF THE MYOCARDIUM

Ultrastructure of Myofibrils

Myocardial cell contains hundreds of parallel myofibrils between them are rows of mitochondria, the latter compose between 25 & 50% of the entire myocardial mass. The myofibril exhibits a periodic pattern of dark & light areas which are formed by a repeating morphologic & functional unit, the sarcomere. Sarcomeres, each is 1.5 - 2.2 um long depending on the degree of relaxation or contraction of myofibril, are separated from each other by Z band. The thin filament measures 1 um in length and is composed mainly of actin molecules & some tropomyosin B, a contractile protein, the thick filament measures 1.5 um in length & is composed of myosin. Overlapping of the 2 filaments gives the myofibrils their striations. The dark and light zones in the sarcomere are the optical counterpart of the relation between the thin and thick filaments. On either direction of the Z band and extending into adjacent sarcomeres is the light area, I band, composed of thin actin filaments only, the central darker area between the two peripherally located I bands is called the A band (anisotropic under polarized light). H zone is a lighter band crossing A band & contains only the thick myosin filaments, where the latter interdigitate with knoblike excrescences in the center of the H zone,

there is a thin line, M line, which probably results from optical superimpostion of the intermyosin cross bridges. So sarcomere is composed from one end to the other, Z band, I band, A band with lighter zone H & central M line, I band and the Z band. In transverse section of the myofibril, only thick filaments are observed in H zone, arranged in hexagonal pattern, in the I band only thin actin filaments which are evenly spaced can be seen, in A band lateral to H zone, six thin actin filaments surrounding one thick myosin filament forming a regular hexagonal pattern, each hexagonal group is surrounded a t its points by six thick myosin filaments, connecting bridges between thick and thin filaments can be seen.

Myosin is a protein with a long thin tail and a globular head, myosin molecule, by short periods of tryptic digestion, can be splitted into two components, H (heavy) meromyosin and L (light) meromyosin. AT pase activity of myosin is confined to globular head which is part of H meromyosin fragment. Each myosin molecule has t win globular heads at one end of the molecule, and both heads possess AT Pase activity. Between 200 - 300 molecules of myosin are arranged side by side with pairs of globular heads protruding in a spiral. Myosin has a central bare area in the middle of the thick myosin filament in which no heads project, and believed to result from reversal in the polarity

of the individual myosin molecule. In both halves of the filament the globular heads of myosin molecules are directed away from the centre. In the central region of the filament only the tails of myosin molecules are present and no projections seen. AT Pase activity of, and content of sulthydryl groups in, cardiac myosin are less than skeletal muscle myosin.

Cardiac actin, like skeletal actin, exists in two forms G actin, a globular protein and F actin, a fibrous polymer of G actin.

Tropomyosin occurs in Z band and in association with thin filament binding well to F actin. Troponin is a globular protein and forms a stable complex with tropomyosin but not with F actin unless tropomyosin is present. It is troponin which confers calcium sensitivity on natural actomyosin and myofibrils, but tropomyosin is necessary to allow the attachment of troponin to the thin filaments. Troponin binds calcium avidly and will account for the total exchangeable calcium of the contractile protein system.

Mechanism of Muscle Contraction

The sliding filament hypothesis of Huxley and Hanson for skeletal muscle is applicable to the myocardium. During contraction, myocardial sarcomere length is shortended and so myofibrils, by approaching of the two opposite Z bands without reduction in length of the two filaments, length of

A band remaind the s ame, while the width of I band decreases and the H one concomitantly decreased approximately the same amount, so that the length of the thin filament also does not change. In maximun contraction the Z line appears to fuse with the edges of the A band and the thin filaments a ppear to overlap in the center of the sarcomere. The force that a muscle can develop is related to the degree of overlap between the thick and the thin filaments, a maximun force coincides with a maximun overlap. If a muscle is sufficiently stretched to the extent of loss of overlap, no tension develops. What changed is only the relation between the two sets of filaments which possibly slide past each other. The actin filaments are pulled toward the center of the sarcomere by linkages between myosin and actin filaments during contraction. ATP ase activity of myosin and the ability of myosin to bind actin are confined to H meromyosin heads which project from thick filament in a spiral. Troponin masks the site for the interaction of F actin with myosin head, when calcium ions bind troponin, the active sites on actin are unmasked, allowing myosin heads to form cross bridges w ith F actin with subsequent great potentiation of myosin ATP ase activity. ATP is split, bridges are broken and reformed at adjacent sites on thin filament and contraction occurs. Davies suggested that calcium ions form chelate links between ATP bound in H