EXERCISE ECHO-DOPPLER EVALUATION OF DIABETIC PATIENTS WITH NORMAL RESTING AND EXERCISE ECG

Thesis

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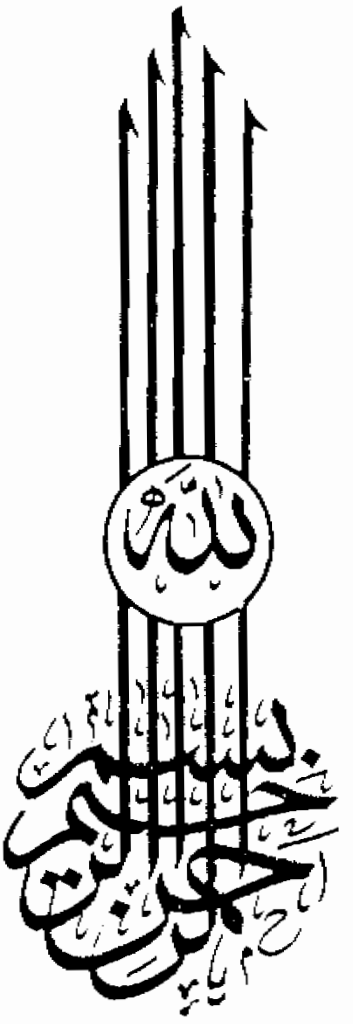

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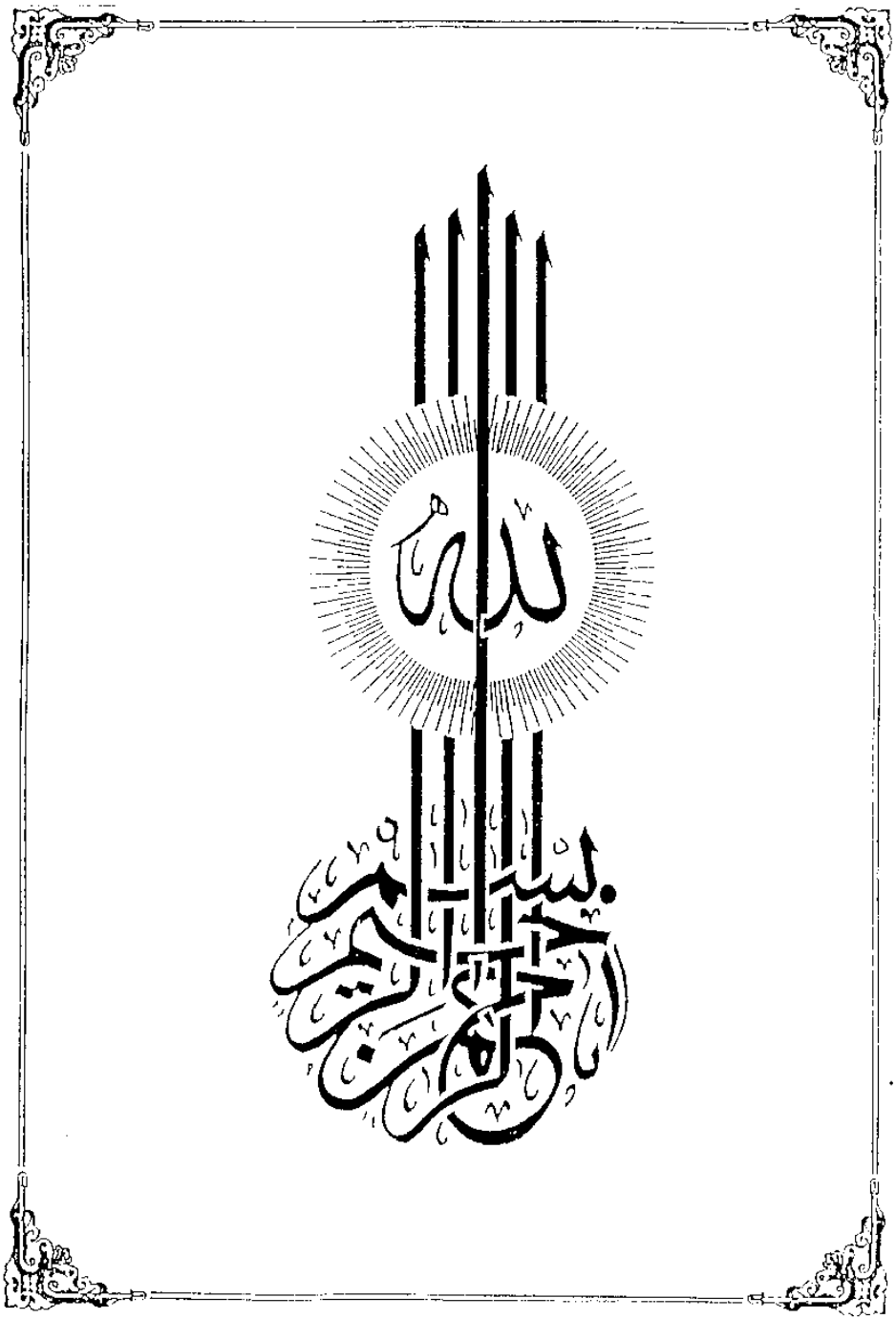

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INTRODUCTION

The biochemical definition of diabetes mellitus is a lack of insulin effect upon tissues, resulting in abnormalities of carbohydrate, protein and fat metabolism. Superimposed upon the biochemical abnormality of diabetes is the clinical syndrome resulting from long term disordered metabolism. The main features of the syndrome are a specific microangiopathy, neuropathy and more frequent and accelerated macrovascular disease (Nattras, et al;1988).

Recently, there is increasing evidence that diabetic have abnormalities of the left ventricular function in absence of clinical heart disease (Sanderson, et al., 1978). Whatever this results from small vessels disease of the myocardium, the metabolic effects of diabetes or coronary artery disease is unknown, but Framingham study showed that diabetics suffered an incidence of heart failure in excess of that predicted from atherogenic risk Factors (Kannel, et al; 1974).

For many years attempts to quantify the function state of the L.V. has been focused on the systolic events of the cardiac contraction. Same patients may have marked systolic ventricular dysfunction or failure at a time when they do not have significant, if any, elevation of ventricular diastolic pressure. On the other hand, same patients have marked elevation of left ventricular diastolic pressure and pulmonary

congestion (Diastolic dysfunction of the left ventricle) at a time, when the systolic or pumping function of the ventricle is well mentained or even greater than normal.

Recently, there has been an increasing interest in the assessment of the L.V. diastolic function in the deterioration of myocardial contractile function. The L.V. diastolic filling characteristics have been used as indicators of L.V. diastolic function (Rocky, et al., 1985).

It is well known that under resting condition L.V. function may normal.'It there has been no permanent myocardial damage and if the ventricle is not ischaemic at the time of examination the echocardiographic study will not reflect the ventricular dysfunction that may be present under ischaemic condition . Thus, there has been considerable interest in combining echo-doppler with interventions that will produce sufficient stress for ischaemia to occur. The technique that seem to have most promise at the moment is the immediate postread-mill exercise echo-doppler (Berberich, et al; 1984).

Exercise echo-doppler is completely non-invasive and requires no radiation exposure. It has been useful in patients with suspected ischaemic heart disease for the evaluation of chest pain and determination of L.V. performance.

AIM OF THE WORK

Early detection and assessment of the degree of L.V. diastolic dysfunction by post-exercise echo-doppler in diabetic patients with normal resting and exercise ECG.

DEFINITION

Diabetes is a disorder in which the level of blood glucose is persistently raised above the normal range. It occur either because of insulin deficiency or because of the presence of factors which oppose the action of insulin. Hyperglycaemia results from insufficient insulin action. There are many associated metabolic abnormalities, notably the development of hyper ketonaemia when there is a severe lack of insulin, together with alternations of fatty acids, lipids and protein turnover. Diabetes is a permanent condition in all but a few special situations in which it can be transient (Peter, et al., 1990).

Classification of diabetes Mellitus

[I] ***Spontaneous diabetes mellitus***

(A) Type I or insulin-dependent diabetes (formerly called juvenile-onset diabetes).

(B) Type II or insulin-independent diabetes (formerly called maturity-onset diabetes).

[II] ***Secondary diabetes***

(A) Pancreatic disease (pancreaprival diabetes, e.g., pancreatectomy, pancreatic insufficiency, haemohromatosis).

(B) Hormonal: excess secretion of counterregulatory hormones (e.g., acromegaly, cushing's syndrome, pheochromocytoma).

(C) Drug induced (e.g., potassium-loosing diuretics contra insulin hormones, psychoactive agents, diphenyl hydantoin)

(D) Associated with complex genetic syndromes (e.g., ataxia telangiectasia, laurance-Moon -Biedl syndrome, myotonic dystrophy, friedreich's ataxia).

[III] ***Impaired glucose tolerance*** : (formerly called chemical diabetes, asymptomatic diabetes, latent diabetes, and subclinical diabetes): normal fasting plasma glucose, and 2-h value on glucose tolerance test > 140 mg/dl but < 200 mg/dl

[IV] ***Gestational diabetes:***

Glucose intolerance which has its onset in pregnancy. (Shatrir, et al., 1987).

Aetiology of insulin dependent diabetes:

Genetic influences are important in IDDM but are not crucial, since only about 36% of identical twins of diabetic patients and about 10% of non-identical twins and sibs of diabetic patients develop the disease (Leslie et al., 1986).

As regard the environmental factors causing IDDM Attention has been focused on viruses and toxins. Wild viruses exist which have specific beta cell tropism and diabetogenic potential in animals and diabetes is frequently found in patients with congenital rubella syndrome (Rubenstein, et al., 1982).

Of toxins, only N-nitroso derivatives such as alloxam, streptozocin and the rat poison Vacor are known to induce diabetic ketoacidosis (Karam, et al., 1980).

The interaction of genetic and non-genetically determine factors result in destruction of the insulin secreting beta cells of the pancreatic islets. At diagnosis about 80% of islets contain no beta cells and the islets may be heavily infiltrated with lymphocytes (Foulis, et al., 1986).

Aetiology of non-insulin dependent diabetes

*** Genetic factor**

The genetic basis of NIDDM is strong, although environmental factors must contribute to the time of onset of the syndrome and account for occasional discordance between twins (Taylor, 1989)

*** Diet:**

Observations that the incidence of NIDDM decreases during times of good scarcity have led to assumptions about the role of diet as an aetiological factor (Mann, 1987).

Such deductions confuse the effect of calorie restriction leading to a prolonged subclinical period with an effect upon initiation of the disease process. Dietary therapy dose not restore insulin sensitivity to normal (Hollenbeck, et al., 1987).

At the present time there is no evidence that dietary factors perse underlie the initiation of NIDDM although they are likely to affect the rate of progression to clinical syndromes (Taylor, 1989).

Obesity:

Obesity has been ascribed great significance in the aetiology of NIDDM by many writers, However there is much evidence that obesity is irrelevant and little or no direct evidence that it plays an aetiological role. Deliberate our feeding of lean subjects to achieve a doubling of body fat content (20-30% increase in body weight) produces hyperinsulinaemia and insulin resistance but no impairment of glucose tolerance.

This concurs with the observation that the majority of obese subjects have normal glucose tolerance. The additional effect of obesity on the insulin insensitivity in NIDDM is small and contrary to popular belief, there are no differences in the relative contributions of decreased insulin secretion and insulin resistance in obese and lean NIDDM subjects (Reaven, et al.,1982). Upper body obesity is associated with greater risk of ischaemic heart disease, decreased hepatic insulin extraction and hyperinsulinaemia (Kopelman, et al., 1980).

Ageing and exercise:

The prevalence of NIDDM increases markedly with age. It may be considered that this represents progressive loss of beta cell capacity and the association of syndromes of premature ageing with NIDDM is of interest (Rimoin, et al., 1982). Ageing per se is not associated with decreased insulin secretion (Dudl, et al., 1977). nor with decreased insulin sensitivity provided that adequately nourished, physically active subjects are studied (Brougghton, et al., 1987). If the decreasing level of physical fitness in the elderly population as a whale are considered, then the effect of lack of exercise on insulin sensitivity may Fasten the appearance of hyperglycaemia as it may at any age. Curiansly, this important factor has received scants attention to date.