THE STUDY OF THE EFFECT OF VALSALVA MANEUBER ON THE LEFT VENTRICULAR DIASTOLIC DYSFUNCTION IN PATIENTS WITH CORONARY ARTERY DISEASE

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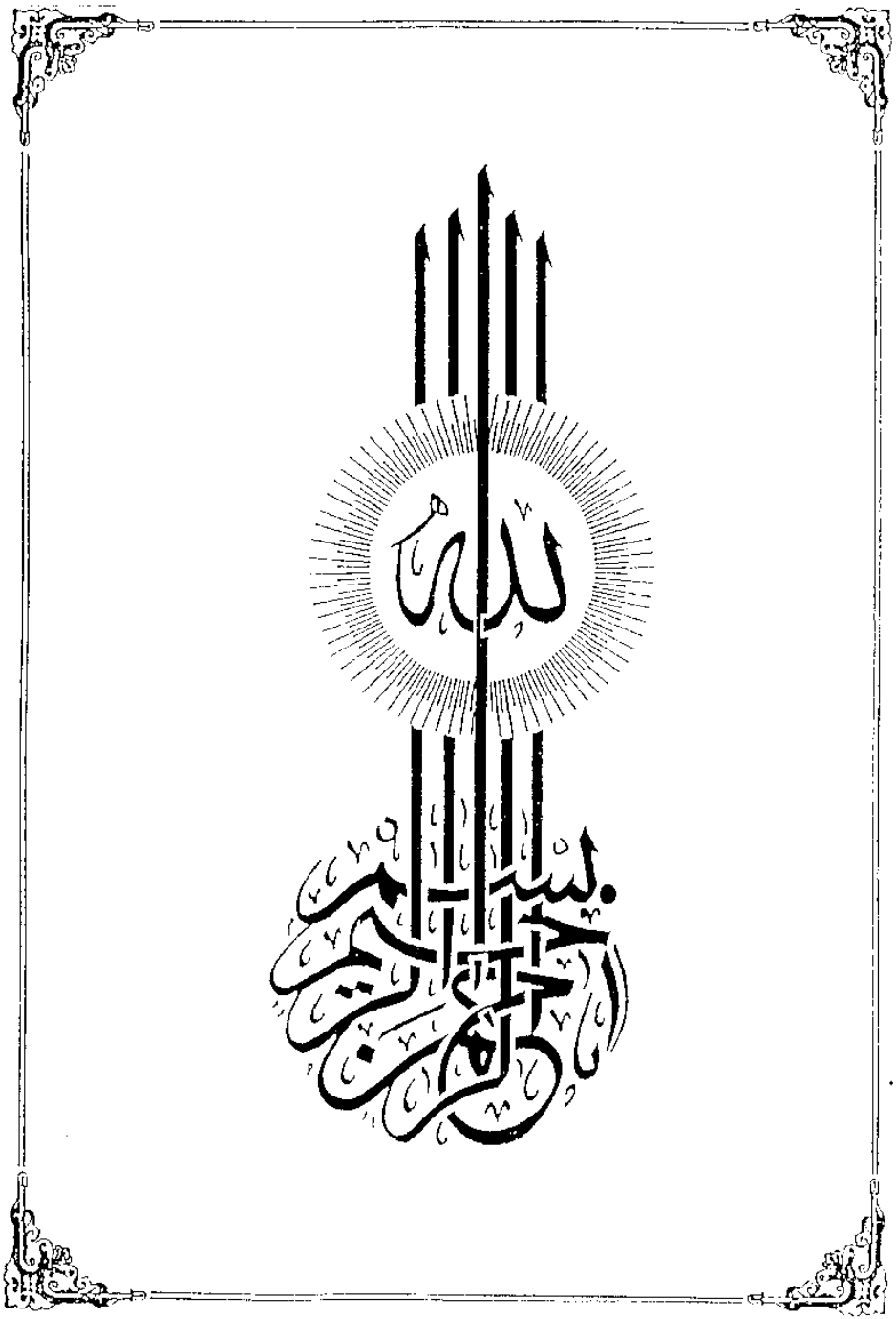

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INTRODUCTION

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There is no gold standard for detecting L.V. Diastolic abnormalities, and the trans mitral Doppler flow velocity profile is used to detect such abnormalities, yet , it is influenced by many factors such as age, heart rate, pulmonary venous return. However, some investigations have emphasized that the Doppler trans-mitral flow velocity profile used to detect such abnormalities is not only sensitive to changes in diastolic function, but also is responsive to changes in pulmonary venous return **(Stauffer JC, Gaasch WH. *Prog Cardiovasc Dis*, 1990)**

Therefore Choong et-al(**Choong CY, Herrmann HC, *J Am Coll Cardiol*, 1987**), suggested that a reduction in the left atrial pressure may change the trans mitral flow velocity profile in maneuver that mimics the abnormalities reported with impairment of L.V. diastolic functions .

Whereas, other investigators **(Nishimura RA, Abel MD et al, *Mayo Clin Proc* 1989)** , reported that an increase in the left atrial pressure may mask L.V. diastolic function abnormalities.

REVIEW OF LITERATURE

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THE VALSALVA MANEUVER .

This procedure was firstly described in 1704 as a method of expelling pus from the middle ear by straining with the mouth & nose closed .

The Valsalva maneuver consists of four phases . It may briefly reduce the coronary blood flow therefore should not be utilized in patients with acute myocardial ischaemic syndrome.

How to perform the test :

The patient is asked to blow against an aneroid manometer & maintaining a pressure of 40mmHg for 30 seconds , during the Valsalva maneuver intrathoracic pressure rises , venous return to the heart diminishes , stroke volume falls & venous pressure rises .

PHASE I :

Associated with a transient rise in systemic blood pressure as straining commences which represents transmission to the periphery of the increased intrathoracic pressure , this phase cannot, as a rule, be identified at bed side .

PHASE II :

Accompanied by a perceptible decrease in the blood pressure & pulse pressure (small pulse), & readially detectable reflex tachycardia . because the continuation of strain leads to a significant reduction of venous return .

PHASE III :

Begins with cessation of straining & is associated with an abrupt transient decrease in blood pressure equivalent to the fall in the intrathoracic pressure , phase III is generally not perceived at the bed side & is followed promptly by phase IV .

PHASE IV :

Which is characterized by an overshoot of systemic arterial blood pressure & relatively obvious reflex bradycardia ,due to the combination of the inrush into the heart of blood that had been dammed up in the venous bed, a reflex vasoconstriction & tachycardia secondary to the low perfusion pressure of the carotid & baroreceptors during phase III .

The effects of the Valsalva maneuver on the diastolic functions of the heart :

As mentioned the Valsalva maneuver causes a significant reduction in the venous return , thus decreasing the left atrial pressure as well as decreasing the left ventricle filling pressure.

The reduction in the venous return would result in an overall decrease of L.V. filling velocities, without a significant change in E/A ratio.

In patients with abnormal relaxation & high left atrial pressure a decrease in venous return induced by Valsalva maneuver would produce a reduction in initial E filling velocity . This could occur without a corresponding decrease in "A" velocity. Given that early left atrial emptying is still abnormal & that with a lower pressure regimen the emptying due to atrial contraction may actually tend to increase instead of decrease . In such case the E/A ratio would decrease & this could help differentiation between normal & so called "pesudonormal " diastolic functions measured by the Transmitral flow velocity profile .

The decrease in E velocity reflects a decrease in the early diastolic gradient between the left atrium & L.V. due to decrease in venous return .

Diastolic function :

* Factors influencing the diastolic ventricular properties

I Factors extrinsic to the ventricular chamber

a) Pericardial properties .

b) Loading of the contra lateral ventricle .

c) Coronary vascular turgor (erectile effect) .

d) Extrinsic compression by tumor, pleural pressure .

II Factors intrinsic to the ventricular chamber :

A) Passive elasticity of the ventricular wall (stiffness or compliance when the myocytes are completely relaxed)

1) Thickness of ventricular wall & composition .

2) Temperature, Osmolality .

B) Active elasticity of ventricular wall due to residual cross-bridge activation through part or all of diastole .

1) Slow relaxation , affecting early diastole only .

2) Incomplete relaxation affecting early, mid & end diastolic distinsibility .

3) Diastolic tone , contracture , rigor .

C) Elastic recoil .

D) Viscoelasticity (stress , relaxation , creep.) .

ASSESSMENT OF DIASTOLIC FUNCTION OF THE HEART

In the past, the evaluation of the myocardium has been limited to examining systolic function of the heart. Recently however investigators have demonstrated that abnormalities of diastolic function of the heart provide important contributions to the signs & symptoms experienced by patients with heart disease.

In addition abnormalities of diastolic function may precede abnormalities of systolic function in the early stages of disease. **(Rick A. Nishimura et al . 1988)**

Diastolic filling of the heart, however is a complex sequence of interrelated events.

In order to understand diastolic function each of these factors contributing to filling of the heart must be examined. (**Rick A. Nishimura et al , 1988)**

They include relaxation, passive compliance, atrial contraction, erectile effect of the coronaries, viscoelastic properties, ventricular interaction & pericardial restraint, all of which are interrelated. In addition diastolic factors are affected by changes in loading conditions & contractility & they demonstrate non uniformity in time & space. **(Rick A. Nishimura et al , 1988)**

Diseases of the heart may produce the symptoms of heart failure because of resultant elevated filling & low forward output . To treat these diseases one must have a thorough understanding of the pathophysiologic processes that lead to these abnormalities.

The major emphasis thus far has been on the evaluation of the systolic function of the pump. It has been well known that abnormal diastolic function contributes substantially to the production of symptoms in various cardiac diseases.**(Labovitz AJ, Pearson AC, *Am Heart J*, 1987)**

As many as a third of patients who have congestive heart failure have normal systolic function & thus are thought to have symptoms attributed to abnormal diastolic function.**(Dougherty AH, Naccarelli GV et al, *Am J Cardiol*, 1984)**

Investigators have demonstrated that in most diseases diastolic dysfunction preceedes the onset of systolic dysfunction.**(Aroesty JM, McKay RG, et al, Circulation. 1985)**

Unfortunately no currently available method can completely & readily assess diastolic function. The major reason has been the lack of technology for the critical examination of the diastolic filling characteristics of the left ventricle.

Despite a major research interest in understanding diastolic function for the past decade diastole is still not completely understood .**(Brustaert DL. J Am Coll Cardiol 1987)**

Diastole is a complex sequence of interrelated events & the factors contributing to diastole are highly sensitive to changes in loading conditions, heart rate & contractility. **(Brutsaert DL, Rademakers FE, et al Prog Cardiovasc Dis 1985)**

The advent of Doppler echocardiography has provided the means by which the diastolic properties of the heart can be prospectively evaluated noninvasively in a large number of patients . **(Nishimura RA, Miller FA, et al, Mayo Clin Proc, 1985)**

Some investigators have proposed that the mitral flow velocity may reflect the flow of blood into the left ventricle during diastole, which in turn is dependent on the driving pressure between the left atrium & the left ventricle. **(Rokey R, Kuo LC, et al, Circulation 1985)**

In addition Doppler echocardiography can determine the velocity of flow into right ventricle (through the tricuspid valve) the left atrium (through the pulmonary veins) & the right atrium (through the superior & inferior venae cavae) .