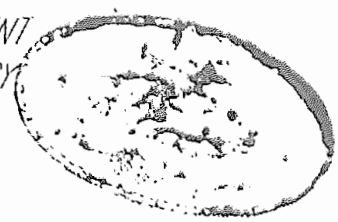
END DIASTOLIC FLOW VELOCITY JUST BENEATH THE AORTIC ISTHMUS ASSESSED BY PULSED DOPPLER ECHOCARDIOGRAPHY A NEW PREDICTOR OF THE AORTIC REGURGITANT FRACTION

THESIS

SUBMITTED FOR PARTIAL FULFILLMENT OF MASTER DEGREE IN CARDIOLOGY



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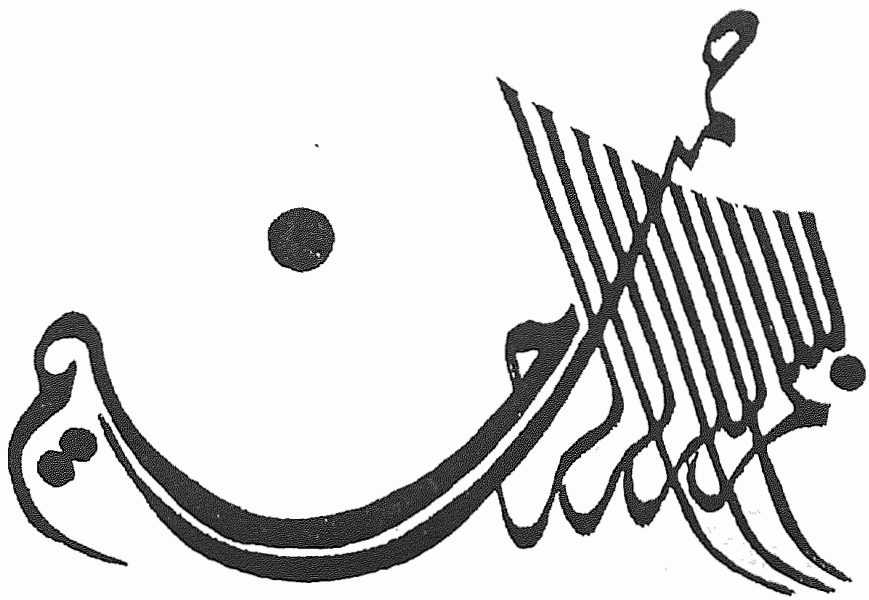

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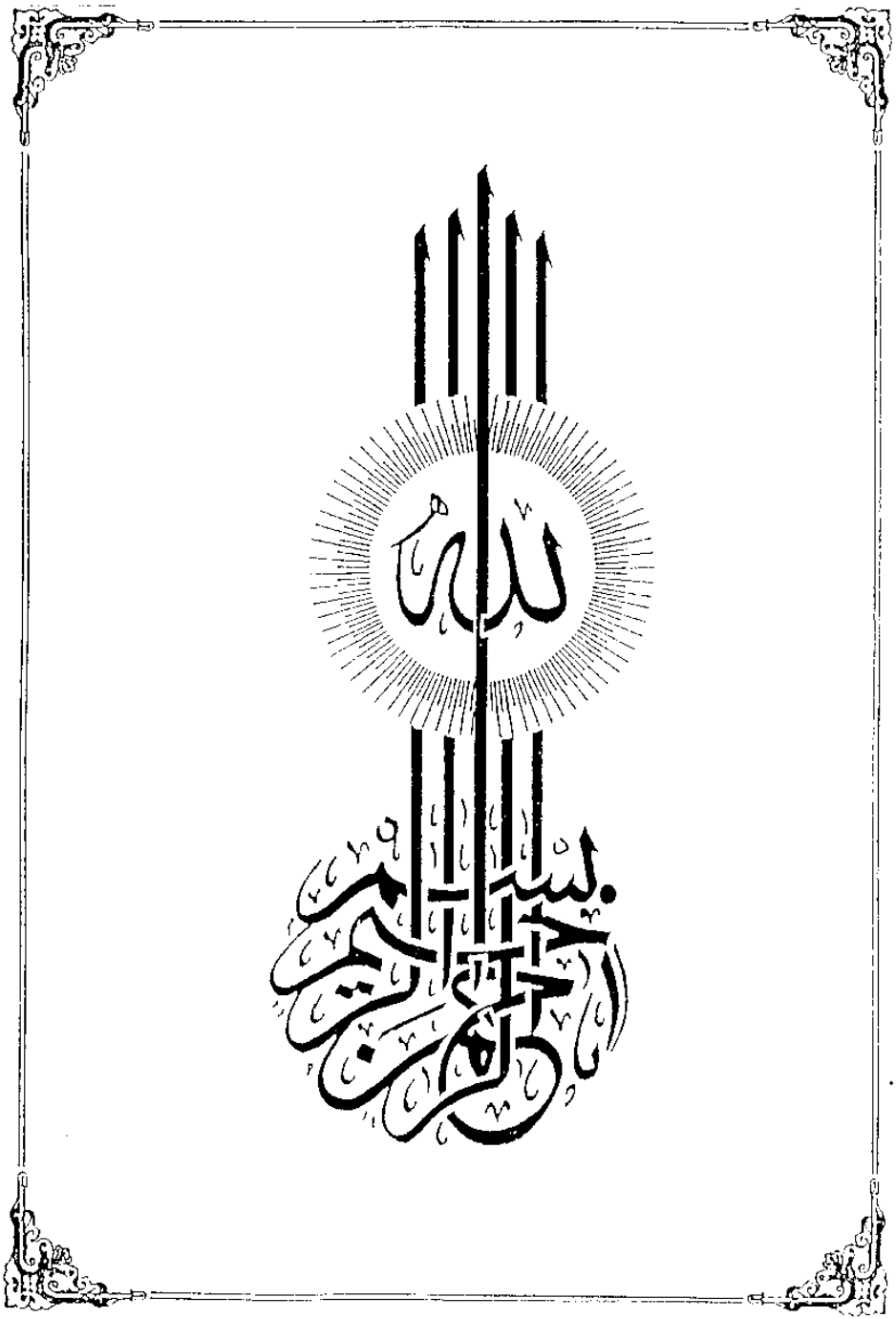

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Contents

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INTRODUCTION AND AIM OF THE WORK

INTRODUCTION

Pulsed Doppler echocardiography is highly sensitive & specific in detecting aortic insufficiency . it is also useful in estimating the severity of the aortic insufficiency. Since its introduction numerous indexes have been proposed as indicators of the severity of aotic incompetence . Development of each new index aimed at achieving more sensitivity & better specificity in diagnosing Ao. I. . Accuracy and simplicity were the goals for the developing pulsed Doppler echocardiography over the past few years.

One of these indexes is end diastolic flow velocity which is measured just below the aortic isthmus at the peak R wave on a simultaneously recorded ECG. In normal individuals , there is no reverse flow at the end diastole beneath the aortic isthmus . In patients with aortic regurgitation , the end diastolic flow velocity can be measured .It was found to be correlated well with angiographic grade of regurgitation & regurgitant fraction .

This technique has the advantage of being a simple direct measurement of a flow velocity simultaneous with the peak of R wave , avoiding complex calculations of other indexes which reduce their sensitivity and add more difficulty in assessing the severity of aortic incompetence.

AIM OF THE WORK

The aim of the work is to investigate the usefulness of measuring end diastolic aortic flow velocity just beneath the aortic isthmus with the use of pulsed Doppler echocardiography in assessing the severity of aortic incompetence and in estimating the aortic regurgitant fraction.

REVIEW OF LITERATURE

METHODS OF ASSESSMENT OF AORTIC INCOMPETENCE

I) Clinical Manifestations :

History :

Ao.I results either from intrinsic disease of valve cusps or from diseases of the ascending aorta which takes the form of either dilatation or laceration of the vessel. The onset & course of the underlying disease determine the presenting manifestation of Ao.I. The slowly developing Ao.I. & the compensatory mechanisms of the left ventricle enable the patient to remain asymptomatic for many years. But, prior to symptoms of limited exercise performance, patients may be aware of palpitations & the circulatory sensation of a large stroke volume with rapid diastolic runoff which can be appreciated as prominent neck vein pulsations & an awareness of the heartbeat when the patient turns on the left side[1].

Symptoms of reduced cardiac reserve or myocardial ischaemia develop, most often in the 4^{th} or the 5^{th} decade and usually only after considerable cardiomegaly and myocardial dysfunction have occured.

When symptoms do develop, exertional dyspnea, orthopnea and paroxysmal nocturnal dyspnea are the principal complaints. Syncope is rare, and although angina pectoris is not so frequent, nocturnal angina, often accompanied by diaphoresis, which occurs when the heart rate slows and the arterial diastolic pressure falls to extremely low levels, may be particularly

troublesome. These episodes are occasionally accomanied by abdominal discomfort, presumably caused by splanchnic ischaemia[2].

Chest pain experienced with severe Ao.I is atypical to angina pectoris, as it occurs at rest as well as persists for a longer duration than coronary artery disease[1].

Chest pain may develop due to significant increase in myocardial O_2 consumption-as a result of the markedly large left ventricular stroke volume which increase the mechanical pressure-volume work of the ventricle, together with compensatory left ventricular hypertrophy - particularly when associated with very low diastolic aortic pressure which may reduce the coronary blood flow[3].

Patients with severe Ao.I often complain of a disagreable thoracic pain due to pounding of the heart against the chest wall .

Usually symptoms may occur like neck and abdominal pain due to stretch of the carotid sheath from large left ventricular stroke volume with a similar mechanism accounting for the abdominal pain.

Postural dizziness is another symptom which is attributed to disturbances in cerebral circulation with marked pressure changes during the rapid diastolic runoff [1].

In acute Ao.I, sudden volume overload is imposed on left ventricle whih is unable to dilate acutely and adapt the increased diastolic filling,

resulting in marked increase in left ventricular end diastolic pressure and minimal ventricular dilatation with subsequent pulmonary venous hypertension and acute pulmonary oedema,which is manifested clinically as sudden onset of cardiovascular collapse with weakness, severe dyspnea and hypotension; angina is uncommon[4].

Physical Findings :

Examination of a patient with chronic aortic regurgitation may reveal abnormalities associated with the underlying mechanism, like yellowish linear streaks in the skin which is seen in the Ehlers- Danlos syndrome. Also, Marfan's syndrome is characterized by asthenic body habitus, long extremities, arachnodactyly of the fingers and may be sublaxation of the lens or high arched palate. Another finding is the bluish discoloration of the sclera which is seen with osteogenesis imperfecta[1].

As regards the arterial blood pressure, the systolic pressure is elevated while the diastolic pressure is abnormally low. The popliteal systolic pressure exceeds the brachial systolic pressure by more than 60 mmHg [Hill's sign]. Korotkoff sounds often persist to zero even though intraarterial pressure rarely falls below 30 mmHg. Phase IV of Korotkoff sounds, i.e. muffling of these sounds correlates with the diastolic blood pressure. As heart failure develops, peripheral vasoconstriction may occur and arterial diastolic pressure may rise. However, this finding should not be interpreted as a reduction in the severity of aortic regurge.

A variety of auscultatory findings provide confirmation of a wide pulse pressure. [Traube's sign] "also known as pistol shot sounds" refers to booming systolic & diastolic sounds heard over the femoral artery, [Muller's sign] consists of systolic pulsations of the uvula, [Duroziez's sign] consists of systolic and diastolic murmur (to and fro murmur) can be heard over the femoral artery when it is lightly compressed with a stethoscope. [Quincke's sign] which is capillary pulsations can be detected by pressing a glass slide on the patient's lip or by transmitting a light through the patient's fingertips.

As regards the pulse, it is of water - hammer or collapsing type with abrupt distension and quick collapse [Corrigan's pulse]. This pulse is readily visible in the carotid arteries and can be best appreciated by palpation of the radial artery with the patient's arm elevated. A bisferiens pulse may be present and is more readily recognized in the femoral and brachial than in the carotid arteries. In patients with chronic severe aortic regurge, the head frequently bobs with each heartbeat [de Musset's sign][2].

Blood pressure and pulse pressure are considered to be good indicators for the severity of chronic aortic regurgitation. If the pulse pressure does not exceeds 50% of the peak systolic pressure or if the diastolic pressure is above 70 mmHg, the aortic regurge will not be haemodynamically severe unless left ventricular failure has developed[1].

When the diastolic blood pressure is <60 mmHg and the pulse pressure is >60 mmHg, the aortic imcompetence is considered to be significant, while it is considered to be severe when the pulse pressure is > 100 mmHg [5].

The apical impulse is diffuse and hyperdynamic and is displaced laterally and inferiorly; there may be systolic retraction over the parasternal region. A rapid ventricular filling wave is often palpable at the apex, as is a systolic thrill at the base of the heart or suprasternal notch and over the carotid arteries, resulting from the augmented stroke volume[2].

The jugular venous pressure is usually normal except in late cases when pulmonary hypertension and right ventricular failure occur giving rise to increased jugular venous pressure. A carotid shudder is palpable or may be recorded[6].

In chronic severe aortic regurge, a soft S_1 and prolongation of the P-R interval are frequently present. A_2 is soft or absent, and P_2 may be obscured by the early diastolic murmur. Thus, S_2 is variable; it may be absent or single or exhibit narrow or paradoxical splitting. A systolic ejection sound is frequently audible. A S_3 gallop correlates with an increased left ventricular end-systolic volume and has been suggested as a sign useful in considering patients with severe regurgitation for surgical treatment[7].

The aortic regurgitant murmur is one of high frequency that begins immediately after A_2. The murmur is heard best through the diaphragm of the stethoscope while the patient is sitting up and leaning forward, with the breath held in deep expiration. In severe aortic regurge, the murmur reaches an early peak and then has a dominant decrescendo pattern through diastole.

The severity of regurgitation correlates better with the duration than with the intensity of the murmur. In severe aortic regurgitation, equilibration

of aortic and left ventricular pressure in late diastole abolishes this component of the regurgitant murmur.

When the murmur is musical ("cooing dove" murmur), it usually signifies eversion or perforation of an aortic cusp. The diastolic murmur is best heard along the left sternal border in the third and fourth intercostal spaces when regurgitation is due to primary valvular cause, but it is often more readily audiable along the right sternal border when it is mainly due to dilatation of the ascending aorta[8].

A mid-and late-diastolic apical rumble, the Austin Flint murmur, is common in severe aortic regurge and may occur in the presence of a normal mitral valve. This murmur appears to be created by rapid antegrade flow across a mitral orifice[9], that may be being narrowed by the rapidly rising left ventricular diastolic pressure caused by severe aortic reflux[10].

A short, midsystolic murmur, grades1 to 4/6, related to the increased ejection rate and stroke volume, may be audible at the base of the heart and transmitted to the carotid vessel. It may be pitched and less rasping than the murmur of aortic stenosis but is often accompanied by a systolic thrill[2].

In acute aortic regurgitation, patients often appear gravely ill, with tachycardia, severe peripheral vasoconstriction and cyanosis, and sometimes pulmonary congestion and oedema[11]. The peripheral signs of aortic regurge are often not impressive and certainly not as dramatic as in patients with chronic regurge[12] .