ECHOCARDIOGRAPHIC ASSESSMENT OF ATRIOVENTRICULAR PLANE DISPLACEMENT IN ACUTE MYOCARDIAL INFARCTION

THESIS
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By

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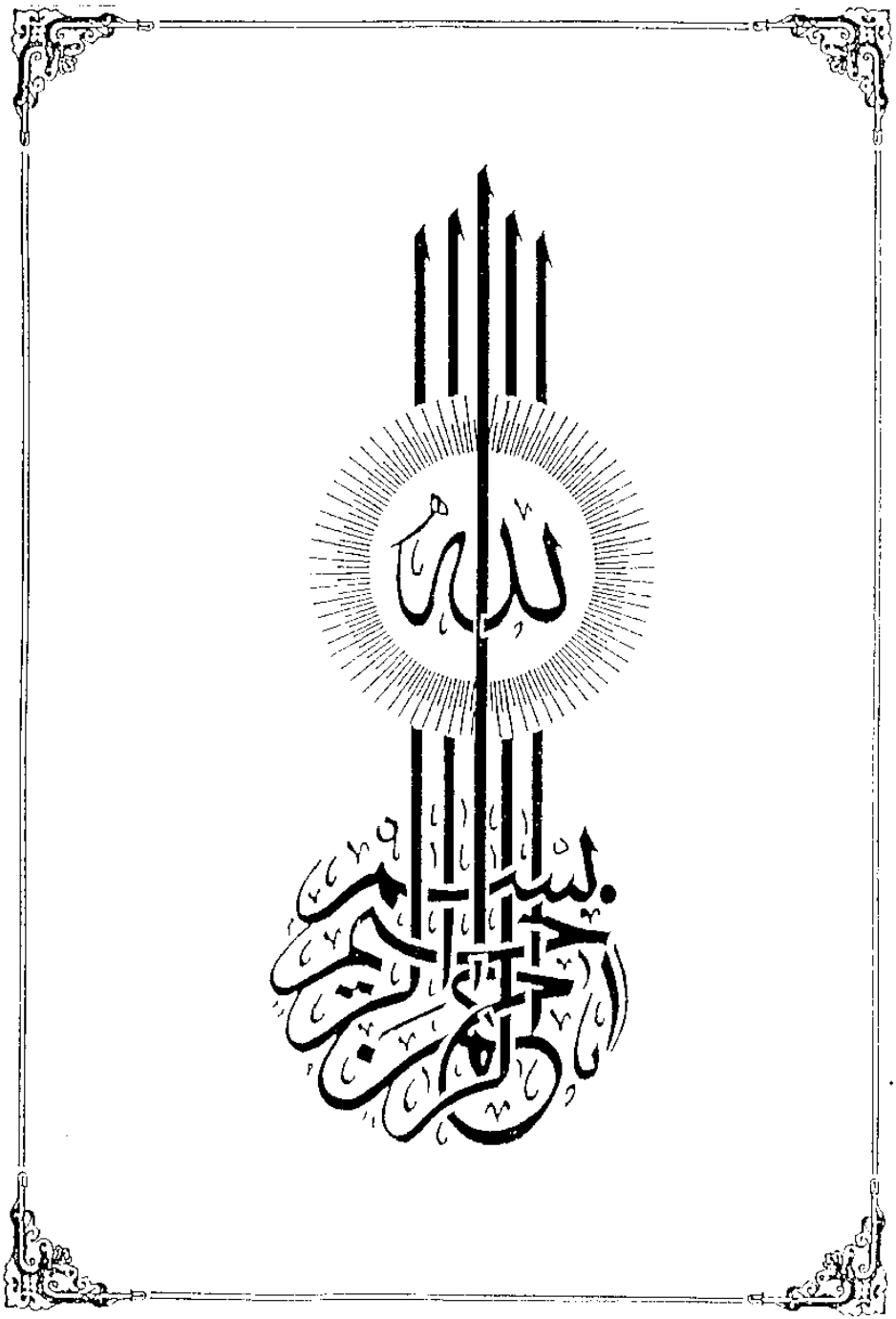

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INTRODUCTION

INTRODUCTION

Myocardial infarction generally results in abnormalities of left ventricular contraction causing global and/or regional impairment of left ventricular (L.V) function. (Asinger et al., 1988).

Wall motion abnormalities can be visualized with the aid of echocardiography, radionuclide angiography and cine-angiography. Echocardiography remains one of the most reliable methods for studies of local wall motion. (Hecht et al., 1981).

Conventional echocardiographic methods for evaluating regional wall motion abnormalities look at different segments of the heart. Proper analysis of regional contraction requires a great deal of experience and technical skill that limits the clinical availability of the methods. (Lindvall et al., 1984).

Studies in both humans and animals show that the mitral annulus changes its size, shape and position during the cardiac cycle. Left ventricular (L.V). Contraction results in shortening along both the short and long axis of the left ventricle. With each systole, the mitral annulus moves toward the apex in a cephalocaudal direction. (Hoglund et al., 1988).

Recent echocardiographic studies have shown that atrioventricular (AV) valve plane displacement towards the apex in systole plays an important role in the pump function of the left ventricle. (Pai et al., 1991).

The clinical application was reported by Hoglund et al, (1988) in a series of healthy subjects who showed a similar magnitude of contraction at different sites of the AV plane. In that study the displacement was the same at four sites situated about 90 degrees apart inthe AV plane.

In acute myocardial infarction a reduced AV plane displacement was found at the site corresponding to the site of myocardial infarction. (Hoglund et al., 1989).

The method of recording the AV plane displacement is simple and the magnitude of the AV plane displacement can be used to assess left ventricular systolic function in patients with acute myocardial infarction, coronary artery disease and severe chronic congestive heart failure. (Alam et al., 1990).

AIM OF THE WORK