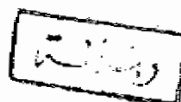


**Assessment of papillary muscle traction in mitral
valve prolapse by two-dimensional echocardiography
and its relation to late potentials by signal averaged
electrocardiogram**

A Thesis



Submitted in Partial Fulfillment for
The Master Degree in **Cardiology**

By

Dr. Mohamed Mohamed Gomaa

M.B., B.Ch.,

64033

616.1240754

M. M

Under Supervision of

Prof. Dr. Amal El-Sayed Ayoub

Professor of Cardiology

Faculty of Medicine

Ain Shams University

Dr. Mohamed Ayman M. Abdel Wahab Saleh

Lecturer of Cardiology

Faculty of Medicine

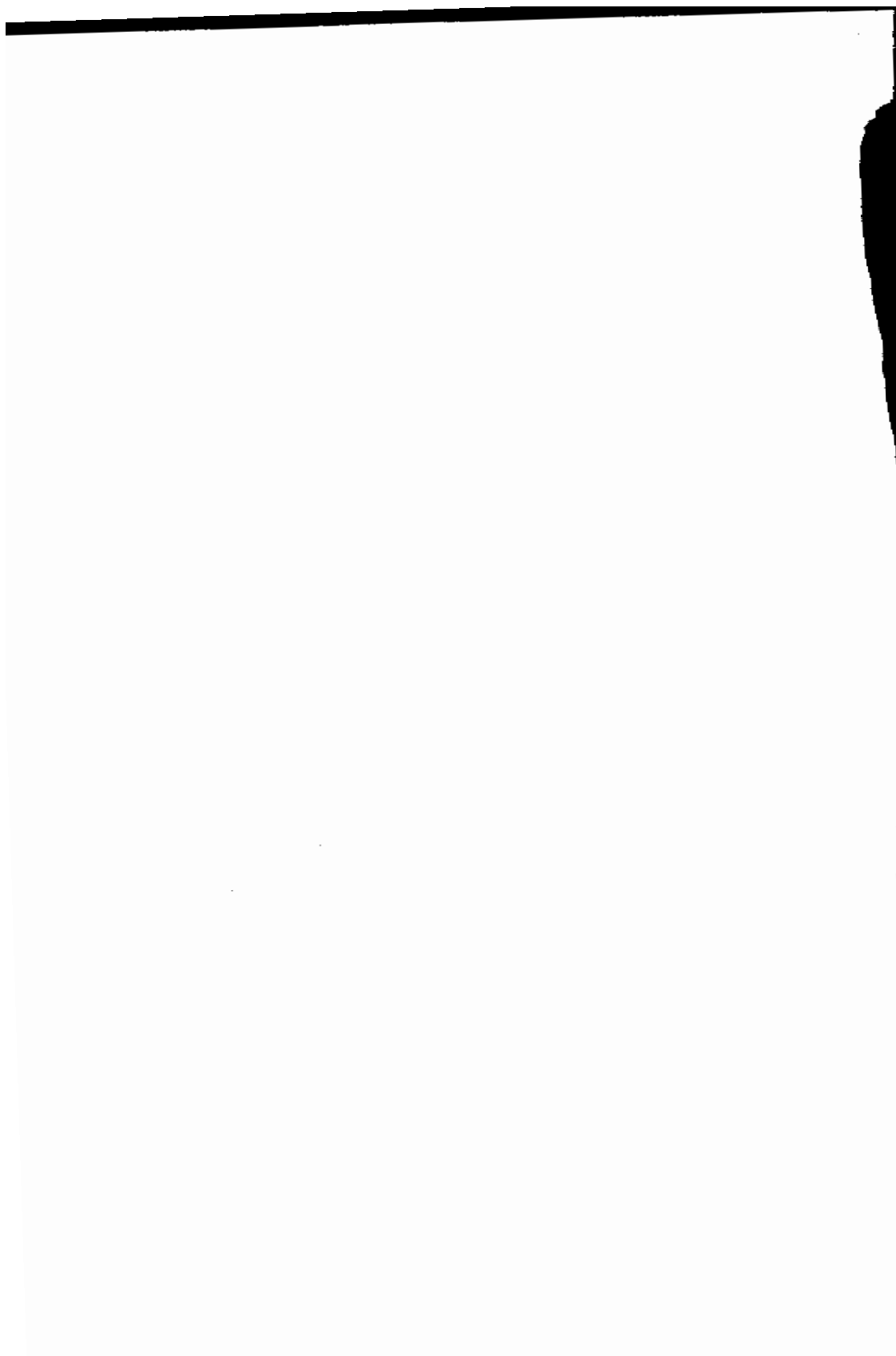
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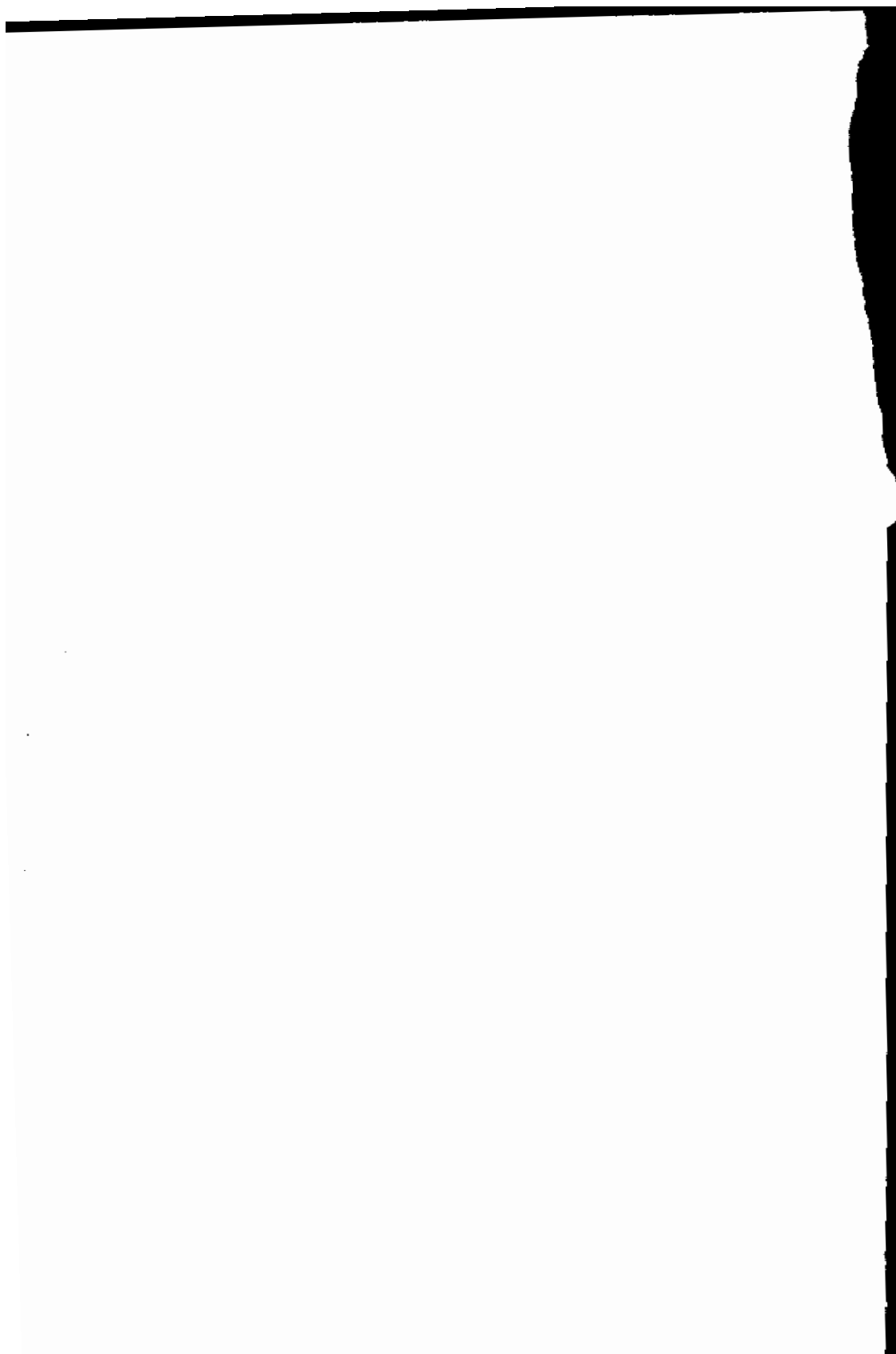


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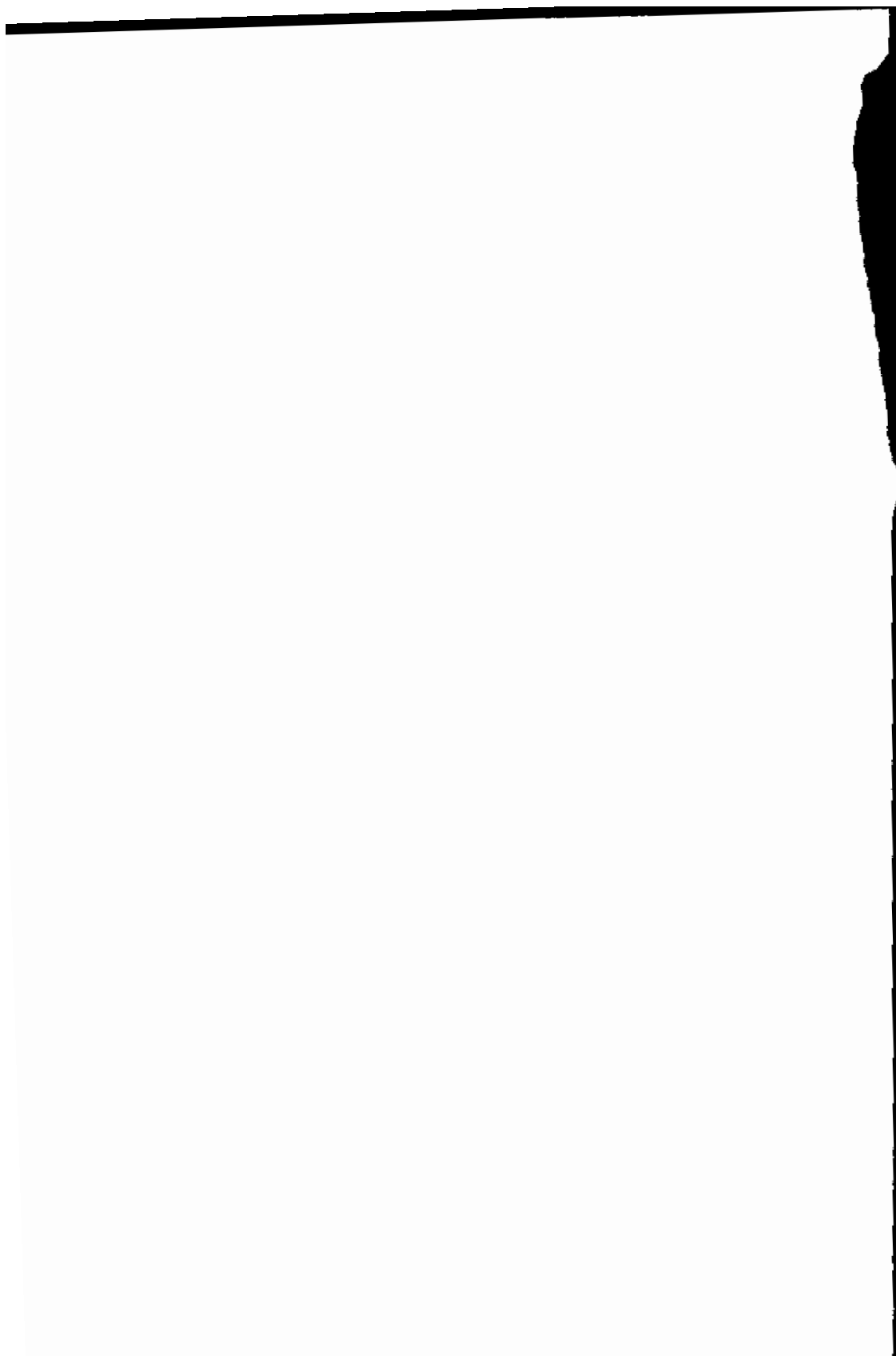
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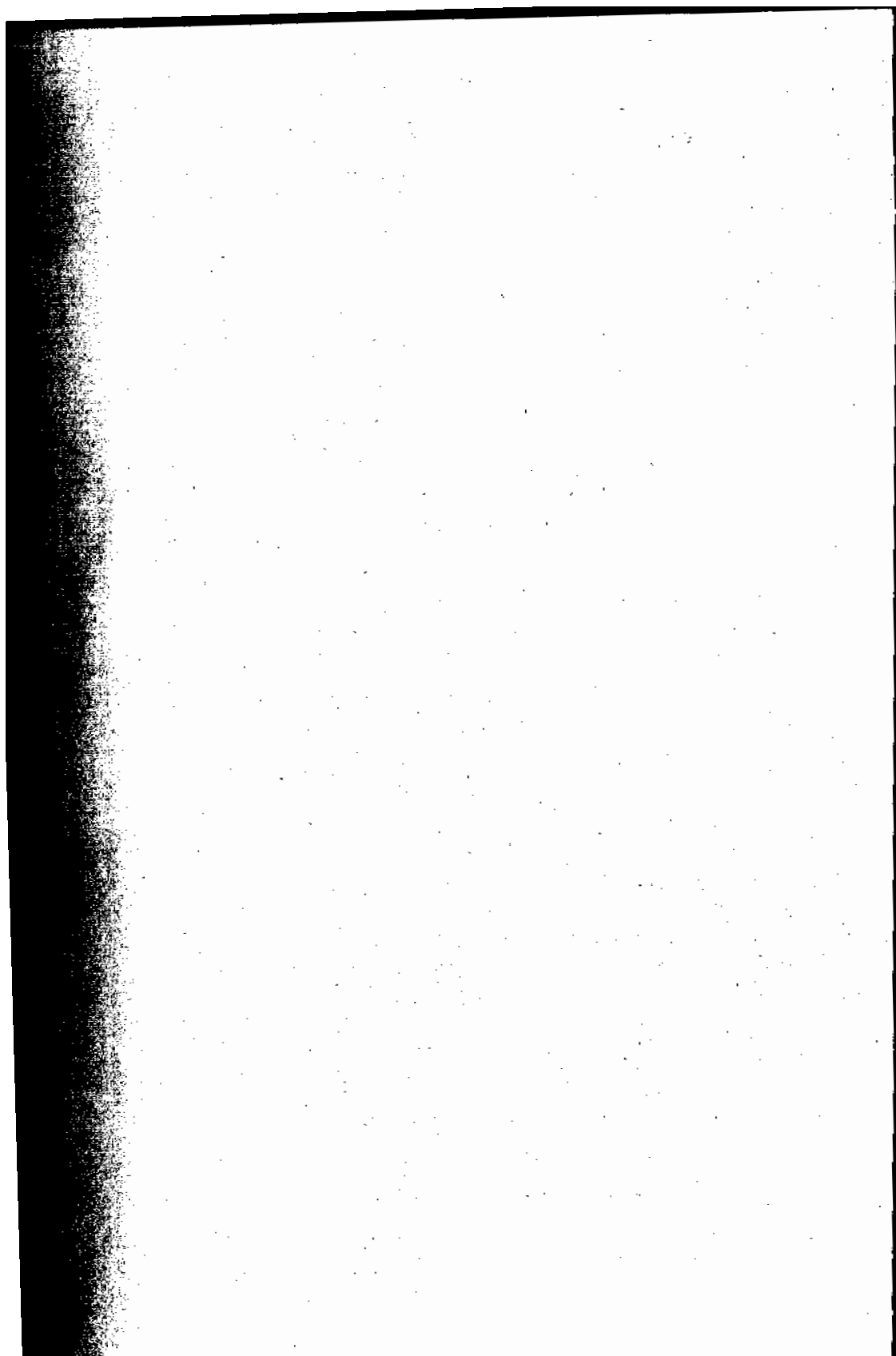
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INTRODUCTION



INTRODUCTION

Mitral valve prolapse (MVP) is considered to be one of the most prevalent cardiac valvular abnormalities affecting as much as 5-10% of the population and is detectable in patients of all ages and both sexes. MVP exists in the setting of a hereditary background. It is transmitted as an autosomal trait. It may represent one manifestation of a number of systemic connective tissue disorders and/or thoracic abnormalities. MVP can co-exist with rheumatic mitral stenosis. Both ischemic heart disease and MVP are common disorders that could co-exist not infrequently (*Braunwald, 1992*).

Previous angiographic studies of MVP are characterized by abnormal superior displacement of mitral leaflets above the level of the mitral annulus during systole. It has been postulated that this leaflet displacement may exert abnormal tension on papillary muscle tips causing their superior traction or displacement and that such traction may have adverse patho-physiologic effects. Recently, it has been observed that similar motion could be identified during echocardiographic examination of patients with classical MVP. Some studies suggested that in such patients, the papillary muscle and mitral leaflets move in parallel toward the left atrium in systole. Whereas, in normal subjects the papillary muscle moves apically

during systole in parallel with mitral annulus maintaining a relatively constant distance with respect to the annulus (*Sanfilippo, 1992*).

Late potentials have been advocated as predictors of ventricular electrical instability with clinically useful prognostic information in different cardiac disease states including coronary artery disease, dilated non-ischemic cardiomyopathy, syncope and idiopathic hypertrophic cardiomyopathy. However, extrapolation of such conclusions to other population subgroups is not necessarily appropriate. This is especially true in patients with mitral valve prolapse (MVP) who in addition to having a variety of abnormalities falsely positive, also have a higher frequency of ventricular arrhythmias (*Kuchar et al., 1986 and Pratt et al., 1986*).