

Techniques Used For The Assessment Of Myocardial Contractile Force

By

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INTRODUCTION

There are several parameters which are notably successful in defining the normal physiology of the Cardiovascular system as well as the pathophysiology of heart disease. Yet there is found an inadequacy for a clear definition and early diagnosis of a functional impairment of the myocardium. The reason for this failure lies in the inadequacy of the monitored variables of the conventional haemodynamic studies. One of such parameter is myocardial contractile force which needs to be evaluated.

In the present article this important parameter has been evaluated and most recent techniques have been described for its evaluation in both experimental and clinical situations while other parameters such as cardiac output and first derivative of the ventricular pressure have been discussed elsewhere (Rashid & Ansari, in press).

Myocardial Contractility Based on Force-Velocity Relation of the Muscle

The myocardial contractile force is altered by two basic mechanisms, Frank-Starling mechanism and inotropic factor (Siegel and Sonnenblick, 1963). Frank-Starling mechanisms (Starling law of the heart) relates the force of contraction of the heart to the initial length of the muscle fibres and is *not* associated with an alteration in the velocity dependent component of muscular contraction. The inotropic factor delineates true changes in contractility and is characterized by a change in the force of contraction, as indicated by an increase in the tension at peak contraction, at a constant initial length of the muscle fibre (Furnival, Linden & Snow, 1970).

The fact that these are two active phases in the cycle of heart contraction has been derived from a study of the basic mechanical characteristics of myocardial muscle. One of these factors is an isometric phase which is dependent on the force velocity relations of the myocardium and characterizes the intact heart as a muscle independent of its pump functions. The second factor is a isotonic phase associated with ejection of blood. In following lines the force-velocity relations of the myocardium will be discussed.

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In 1938, A.V. Hill introduced the concept what has been subsequently termed the most fundamental property of muscle namely "the force velocity relation (FV). He showed that the velocity of shortening in skeletal muscle is inversely related to the magnitude of tension development i.e the greater the load, the muscle is called upon to sustain the lower the velocity of shortening. This fundamental concept of the force velocity curve from skeletal muscle has been extended to the cardiac muscle by various authors (Sonnenblick, 1962). Sonnenblick (1962) using cat papillary muscle showed that Hill's law also applies to the myocardium. In order to demonstrate Sonnenblick's hypothesis one end of the papillary muscle is attached to a lever system so that it is free to shorten. A small weight (Preload) attached at the other side of the lever stretches the muscle to a given length. Another load is then added to the preload and this is the load which will be sensed by the muscle after the contraction (as a stopper has been fixed above the tip where muscle is attached), this load is known as afterload.

When papillary muscle starts contracting against an added afterload it first develops tension until the afterload value has been reached and then proceed to shorten by lifting the total load (preload and afterload) (Braunwald, Ross and Sonnenblick, 1976a). The maximum velocity of shortening (V_{max}) in each contraction depends on the load and relation between the force developed and the velocity of contraction is expressed by the force-velocity curve (Fig. 1). When the initial muscle length is altered by changing the preload the force velocity is also shifted (Fig. 2).

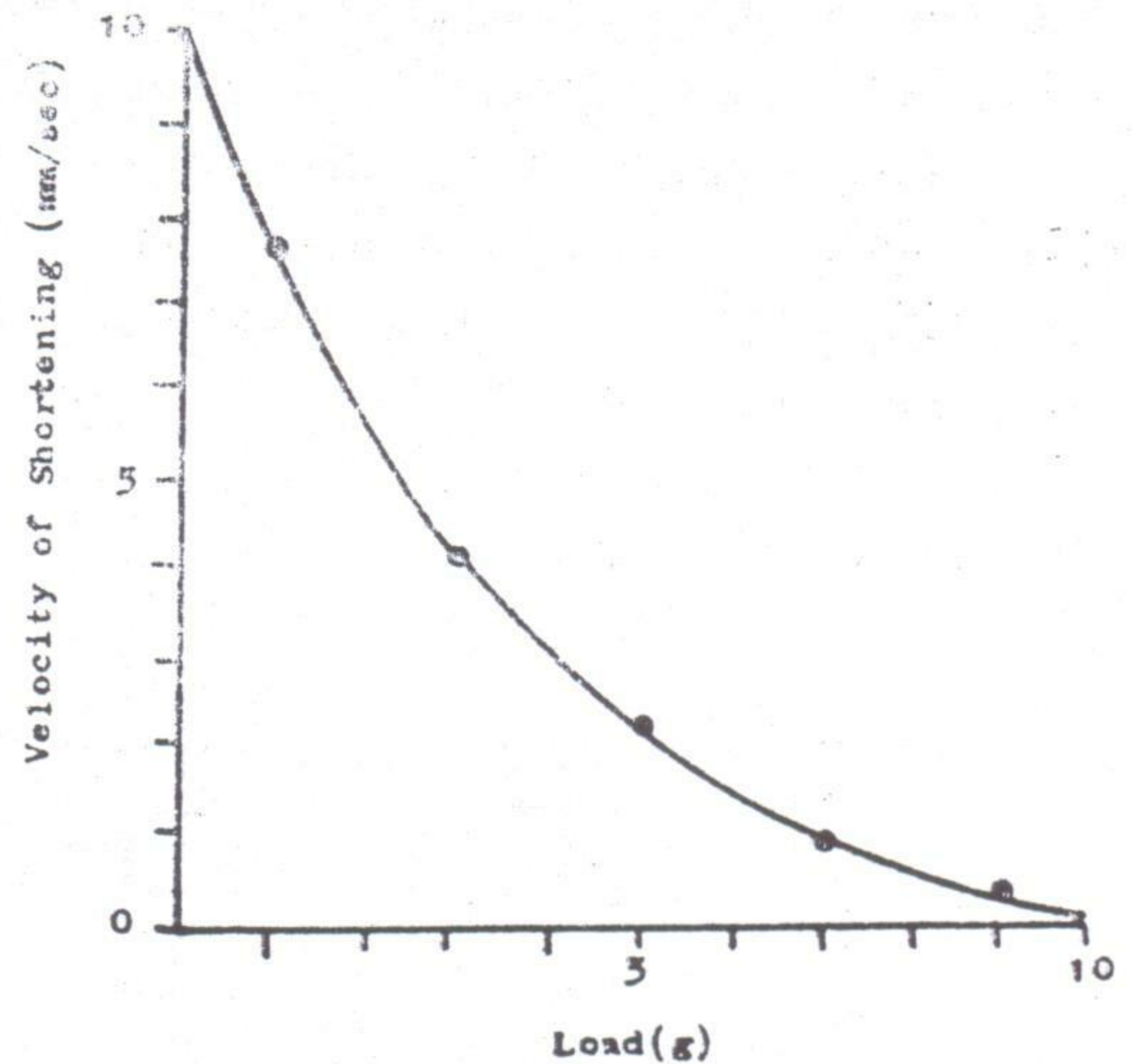


Fig. 1: Force-Velocity Relationship of Cat's Papillary Muscle.

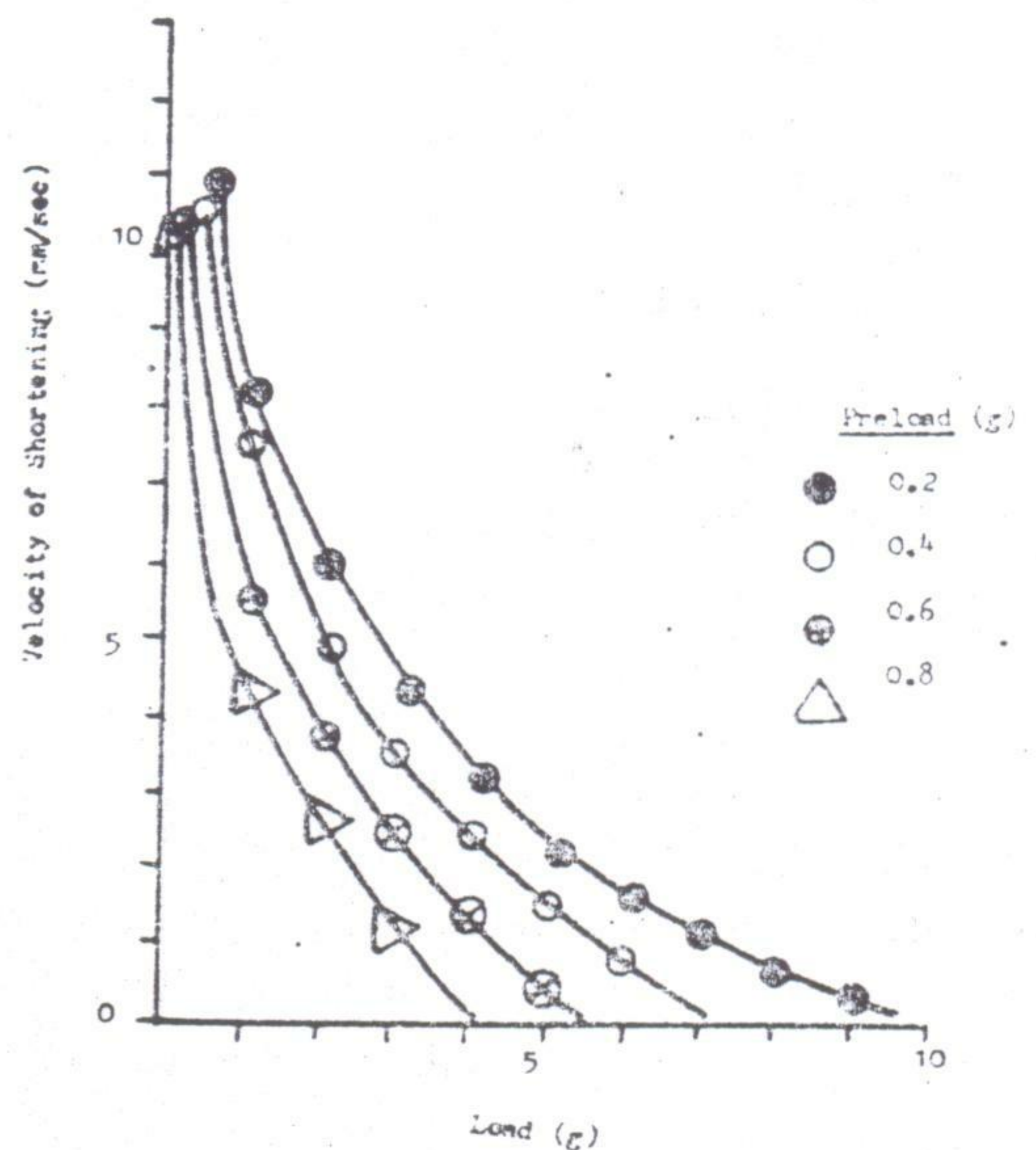


Fig. 2: The effect of increasing Pre-load on the Force-Velocity relation of the cat's Papillary Muscle.

These observations described above were made within the theoretical framework of the

physiological range, a result which is similar to the observations made in the case of isolated cardiac muscle, when measuring its V_{max} of CE (Braunwald, et. al. 1976).

The existence of the FV relationship has been proved by studies made during the ejection phase for the heart of intact dog (Levine & Britman, 1964). The isovolumic phase of ventricular systole has been chosen as the period for the determination of V_{max} (Mason, Spann & Zelis, 1970) because of the complexity of attempts to measure the force-velocity relations during ejection and of the non-isotonic phases of the heart in vivo.

A. Clinical determination of VCE and V_{max} :-

Two methods have been employed to determine the VCE in the intact human heart. One is simple but less accurate utilized by Mason, et al (1970) and the other which is complex and sophisticated have shown by Gault, Ross & Braunwald (1968).

Mason, et al. (1970) described that as the intact heart during isovolumic contraction, alterations in ventricular geometry are small and thus VCE can be assumed to equal series elastic elongation, hence VCE during isovolumic portion of ejecting beats can be determined entirely from isovolumic pressure and its rate of rise (dP/dt) without requiring knowledge of tension and this could provide a rational basis for accurate calculation of isovolumic VCE in the spherical or ellipsoidal ventricle as $dP/dt/K^* \times IP$ where, K is series elastic constant and IP intraventricular pressure. $*(K = 32/\text{muscle length at body temperature})$.

Thus it was argued that the instantaneous velocity of the contractile element (VCE) could

be determined from isovolumic ventricular pressure and its corresponding dP/dt before the ejection phase of the unloaded heart (V_{max}), the property which has shown to be a direct measure of contractility independent of loading.

Gault, et al (1968) have correlated measurements of left ventricular dimensions throughout contraction, obtained from high speed cineangiograms with high fidelity recordings of intraventricular pressure to derive myocardial wall forces, fibre length and the characteristics of fibre shortening and have used these measurements to characterize left ventricular performance in man. The method which is used is as follows:

The left ventricle was opacified with radiographic contrast material, injected into the left atrium to avoid premature beats, and cineangiograms were exposed at 60 or 80 frames per second in the right anterior oblique projection. High-fidelity left ventricular pressure pulses were continuously recorded by means of a catheter-tip micromanometer introduced by the retrograde arterial technique, and the instantaneous pressures were recorded directly on the cinefilm to allow precise time correlation with the angiogram.

In each cine frame the long axis of the left ventricle was taken as a line from the midpoint of the mitral valve plane to the apex. The radius of the minor, internal, left ventricular circumference was constructed as a perpendicular to this long axis at its midpoint. In order to compute myocardial wall tension, the long axis of the ventricle and the radius of the minor circumference were considered to represent the major axis and the minor

semiaxis, respectively, of an ellipsoid of revolution. The circumferential tensile stress or tension was then calculated throughout contraction in the circumferential slice of myocardium at the equator of the ventricle as follows:

$$\text{Tension} = P \times r_i \left(1 - \frac{2 r_i^2}{L^2}\right) / h$$

where P = intracavitary pressure (grams per square centimeters); r_i = radius of the minor circumference (centimeters); L = long axis (centimeters); and h = wall thickness (centimeters).

The velocity of fibre shortening in this same circumference was computed through contraction from the slope of the radius shortening curve as $2\pi dr_i/dt$. The instantaneous velocity of shortening was then divided by the corresponding midwall circumferential length ($2\pi (r_i + h/2)$) at each point during contraction in order to compare measurements of velocity in patients at different fibre lengths.

Using this method Gault, et al (1968) found that in the patients with normal left ventricular function, maximum tension occurred soon after the onset of ejection, and this soon then declined rapidly through the remainder of the ejection period. In contrast in patients with left ventricular disease wall tension declined less during ejection.

The most accurate estimate of myocardial contractile state presently available is one that describes the relation between tension and the velocity of shortening or the VCE (Mason, 1969).

Gault et al (1968) did not calculate this velocity throughout contraction due to the reason that coefficient of series elasticity is not known in man and may differ in the normal and abnormal left ventricle. However at one point during ejection, where tension is maximum, the rate of elastic extension is zero, and the velocity of shortening of fibre therefore be assumed to equal VCE. They used the relationship between this velocity and tension at this point during contraction as a quantitative estimate of contractile state to compare myocardial function in different patients.

Gault et al (1968) concluded that VCE was consistently reduced in patients with left ventricular disease at level of wall tension (Fig. 4) that were generally comparable with those observed in the normal group, despite the fact that the initial fibre lengths were considerably greater in the patients than in the individuals with normal function.

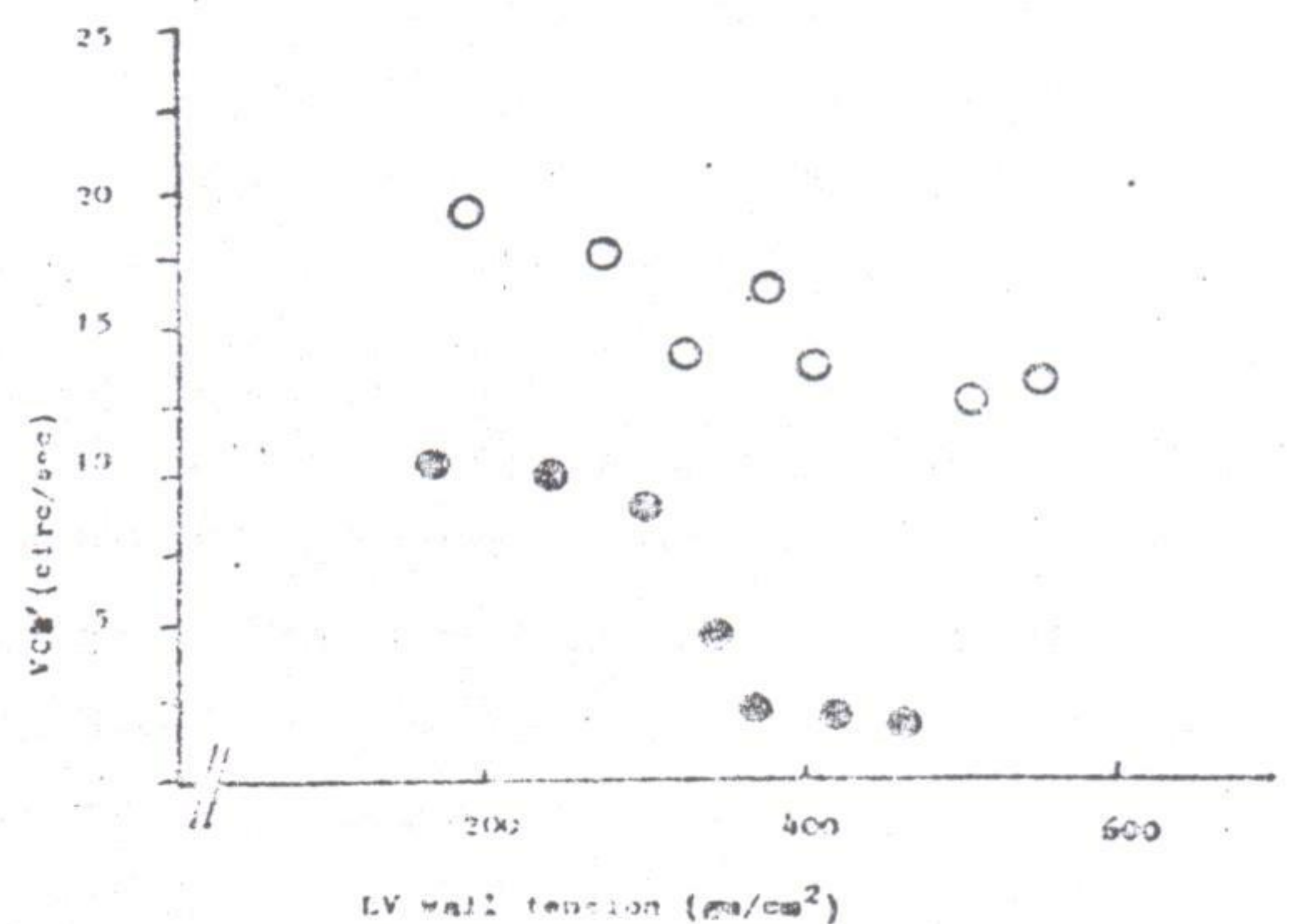


Fig. 4: Diagrammatic representation between VCE and Left Ventricular Wall Tension in normal (open circles) and diseased (closed circles) Patients.

B. *Experimental determination*:- The VCE derived during the isovolumic phase of contraction of the left ventricle in experimental animals shows an inverse relationship to the myocardial wall force irrespective of the ellipsoidal or spherical shape of the ventricle (Levine & Britman, 1964). During isovolumetric contraction the VSE and hence VCE at any instant is considered to be directly related to the rate of force development. When the contraction of left ventricle is totally isovolumetric, i.e. when ejection prevented, and the calculated VCE is plotted against the corresponding wall stress, and the end-diastolic volume is progressively increased by transfusion, the peak tension increases but there is no apparent change in the extrapolation of these FV curves to V_{max} .

Isovolumetric contractions can be produced in the dog by means of balloon fastened to the end of cannula and positioned just above the aortic valve. The balloon is suddenly inflated during diastole to occlude the aortic valve and aortic root for one contraction, thereby inducing a single essentially isovolumetric contraction (Covell, Ross, Sonnenblick, & Braunwald, 1966). From experiments it is possible to show that the heart responds to a change in preload essentially the same manner as the isolated muscle.

This method was also applied to the unanaesthetised sedated closed-chest dog (Taylor, Ross, Covell & Sonnenblick, 1967) for analysis of length-active tension, end-diastolic pressure, active tension and force velocity relationship obtained with such induced isovolumetric beats and it was found that this method was successful in detecting

acute and chronic alterations in inotropic state independently of changes in preload and afterload.

The methods for measuring the cardiac contractile force which have been discussed are not free from criticism, especially the incorporation of the findings in isolated cardiac muscle preparation (Papillary muscle to the intact heart). Brutsaert and Paulus (1977) pointed out that pragmatic usefulness being only the justification for applying isolated muscle concept to the intact ventricle is no longer a sufficient argument.

The ventricle function is as a combined muscle-pump system and not as a papillary muscle and even less a simple tension muscle. The nature of loading forces which occur during muscle shortening in the ventricular wall in intact heart are quite different from those encountered in the isolated muscle since the ventricle is not called on to sustain a weight but to eject a viscous fluid into a viscoelastic vascular system (Abbott & Gordon, 1975).

Suggestion, have been put forward to make progress in the field of measurement of ventricular function by conducting experiments that should be designed to distinguish between the relative contributions of muscular mechanisms and those of the ventricular configurational mechanisms underlying Starling's law of the heart (Brutsaert & Paulus, 1977). However, so far the index of V_{max} has been favoured both in animals (Nejad, Klein, Mirsky & Lown, 1971) and in the clinic (Mason, et al., 1970) as a suitable and accurate method determining the rate of rise of pressure in ventricles.

Summary:

Although several cardiovascular parameters are available to define the normal physiology as well as the pathophysiology of cardiovascular system yet a clear definition and early diagnosis of a functional impairment of the myocardium is lacking. The reason for this lacking may be the inadequacy of the monitoring of these variable parameters. One of the parameters among these is myocardial contractile force.

This important variable has been evaluated in both experimental as well as clinical conditions in this present paper.

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