Differentiation of intramyocardial fluid pressure from fiber stress

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Abstract. To characterize the complex force field generated in the ventricular myocardium, intramyocardial pressure (IMP) measurement is used as an indirect means of assessing the distribution of regional wall stress. To resolve the long term confusion associated with this measurement, IMP is divided into its two dominant components: intramyocardial fluid pressure (IFP) and intramyocardial fiber stress (IFS). The intramyocardial response to regional and global contractile function is examined in terms of changes in the magnitude and transmural gradient of IMP recording. The experimental results support the theoretical concept proposed where the hydraulic properties of the myocardium prove to have an influence on cardiac function. To gain a deeper understanding of myocardial function, cellular and subcellular components must be considered.

1. Introduction

The contractile machinery of the ventricular myocardium manifests itself primarily by muscle shortening. The contraction of muscle fibers translates into the generation of force within the myocardium (i.e., wall stress), augmenting the pressure within the ventricular cavity. During isovolumic contraction, the ventricular geometry is dynamically rearranged as the ventricular wall develops stress. The ventricle may become more spherical, decreasing the distance from apex to base. As the stress developed in the wall increases, ejection commences allowing for further and significant shortening of the myocardial fibers. Deeper insight into the nature of the stress-deformation relation in the myocardium is essential to achieve a fundamental understanding of the complex function of the heart.

The lack of reliable means to directly measure stresses in the intact ventricular wall has resulted in the development of other methods to obtain this information. For example, many investigators have measured “pressure” within the wall of the heart to obtain information about the varying state of stress.

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The determination of the true uniaxial plane stress with approximation of the network models demonstrated reasonable agreement for different models only. Peak and end-effect was.

The comparison of measured uniaxial plane stress with approximation of the true uniaxial plane stress, also from the model, demonstrated a reasonable agreement with the measured value. The experimental load was applied to an explosive chamber, and the measured value of the stress was calculated from the width of the load at the transducer and the measured width thickness by the cross-sectional area. Cross-sectional force in the loaderta's, the measured force values were divided by the cross-sectional area.

Hovever, the deviation of which the differences are observed exceed (1-2) times of the expected error limit (1-2). Hence, results of other investigations, such as Kushiyama [27], and other investigations, such as Kushiyama [27], can be developed into an analytical theory of the measurement of wall thickness and explosive pressure as well as an analytical theory of explosive pressure. Other investigations, such as Kushiyama [27], and other investigations, such as Kushiyama [27], can be developed into an analytical theory of the measurement of wall thickness and explosive pressure as well as an analytical theory of explosive pressure.

Several investigations have shown that the stress distribution in the plane of wall thickness.

2.1 Measurement of wall stress

2. Background

Supported by experimental findings:

The physical mechanisms involved in the generation of uniaxial plane stress: The conclusions are

The measured force on the uniaxial plane stress is recovered with the help of a mathematical model. A network model is also used.

Theoretical and experimental evidence of the plane stress model are presented. A crucial parameter is the ratio of the plane stress to the plane strain.

Several investigations have shown that the stress distribution in the plane of wall thickness.

X-Ray analysis of differentiation of uniaxial plane stress from plane stress
measured wall stresses were $207 \pm 19$ and $104 \pm 13$ g/cm$^2$, respectively, corresponding to calculated respective values of $198 \pm 18$ and $117 \pm 11$ g/cm$^2$ computed from the thin-wall elliptical model of Sandler and Dodge [29].

Lewartowski et al. [17] measured the "tension" within the left ventricular wall of open-chested dogs. To measure the force a resistance wire strain-gauge, glued to a plastic arch, was coupled to the ventricular wall by two pairs of stiff hooks. Wall thickness was estimated by inserting a calibrated wire across the wall during cardiac arrest, which was produced by supra threshold stimulation of both vagus nerves. Stress was computed by dividing the tension by wall thickness. These investigators compared their results with the theoretical predictions of Sandler and Dodge [29], Wong and Rautaharju [34], and Falsetti et al. [6]. The Sandler and Dodge model yielded about 30% higher values for equatorial stress than the Falsetti model, with the exception of one experiment.

The unreliable and often inexact nature of these direct measurements of stresses (due to averaging) in the intact ventricle, in addition to their invasive nature, severely impairs the current knowledge of stress distribution in the myocardium. A number of modifications made to the earlier methods of direct measurements of myocardial wall stress scarcely broadened the field. Lunkenheimer et al. [18] inserted needle force probes into the myocardium to assess myocardial stress. This results in "slipping" of the myocardial fibers on a flexible bar which evokes an electrical signal, which is postulated to be proportional to the perpendicular force produced by displacing cardiac fibers from their original stretched position. These investigators concluded that global function cannot be ascertained from measurement of local muscle function. However, this measurement procedure mitigated the desire to detect local intramyocardial forces. Kresh et al. [15] utilized the measurement of intramyocardial pressure to assess myocardial viability in patients undergoing open heart surgery.

2.2. Prediction of wall stress

To circumvent the difficulties associated with the measurement of ventricular wall stress, investigators attempted instead to calculate the stresses generated by contracting and passive cardiac muscle using measurable variables combined with mathematical models incorporating force balance considerations. This compelling desire to assess wall stress from measurements of left ventricular pressure and chamber geometry has led to the development of several analytical models in which ventricular pressure is viewed as the generator of the wall stress. The earlier models employed classical theory of elasticity utilizing Laplace-type relations; other approaches employed oversimplified boundary conditions. For instance, intramyocardial pressure distribution has been equated to radial stress, where the boundary conditions are the cavity pressure at the endocardium and (sub)atmospheric pressures at the epicardium. Others calculated circumferential and longitudinal stresses which also display a gradient from endocardium to epicardium. A number of simple geometries (i.e., spherical, cylindrical, ellipsoidal) and material properties (i.e., isotropic, homogeneous, incompressible) were assumed to represent the three dimensional structure of the ventricle. Still others utilizing finite element analyses techniques acknowledged that the ventricular geometry is irregular with material properties unlike those of a passive elastic material.

Most of the theoretical analyses seem to have contributed to the confusion by publishing what appear to be conflicting results. Therefore, we concluded that the controversial nature of the quantity measured experimentally or modeled theoretically is in part due to a lack of understanding of the nature of intramyocardial pressure generation, and that further work is required to clarify its etiology.
Experimental work.

The following section provides a summary of the various experiments conducted to measure the uniaxial compressive strength of the material. The experiments were performed at different temperatures and pressures to determine the effect of these parameters on the material's behavior. The results showed that the material exhibited significant improvement in strength under high pressure conditions.

3. Theoretical analysis

The development of a theoretical framework is necessary to understand the behavior of the material under different conditions. This framework allows for the prediction of material properties and the optimization of design parameters. The theoretical analysis presented here is based on the experimental data and provides insights into the mechanisms governing the material's response.

The theoretical analysis is divided into two main sections: (1) the uniaxial compressive strength and (2) the effect of temperature on the material's behavior. Each section discusses the relevant theories and models used to describe the material's response under the specified conditions.
4. Materials and methods

Experiments were conducted on adult mongrel dogs (30–40 kgs body weight). Each dog was sedated with morphine sulphate (2.5 mg/kg i.m.) and anesthetized with alpha-chloralose (100 mg/kg i.v.). The animals were mechanically ventilated, with a mixture of room air and low flow oxygen, via a cuffed-endotracheal tube using a positive pressure respirator. Tidal volume and respiratory rate were adjusted to maintain arterial pO2, pCO2 and pH within their normal physiological ranges. The heart was exposed via a midline sternotomy and a pericardiotomy, and supported in a pericardial cradle where lead II of the electrocardiogram was recorded.

In recognition of the observations that the ventricular wall has a high fluid content, both intracellularly and extracellularly, two pressure transducers of different design were utilized. The more commonly used transducer in this type of study is the Millar ultraminiature transducer (Millar Instruments Inc., Houston, TX). This transducer has a sensing element of 1.1 mm by 0.7 mm which is mounted laterally, approximately 2 mm from the tip of the catheter. Its design makes it sensitive to fluid pressure, as well as to any mechanical stresses exerted on the sensing element by contracting muscle bundles. The total signal recorded is considered IMP. To sense intramyocardial fluid pressure (IFP) a 4F Camino fiber-optic sensor was used (Camino Laboratories, San Diego, CA). The sensing diaphragm of this sensor is recessed inside a stiff catheter and is thus shielded from direct contact with muscle bundles. Both the Millar and the Camino transducers were calibrated by placing them in a water bath kept at 37°C and allowing them to reach steady state. Table 1 compares the specifications of the sensors.

Left ventricular cavity pressure (LVP) was measured by passing a 6F high-fidelity Millar micromanometer through the aortic valve. Root aortic pressure was measured using a 5F Millar passed through the right femoral artery. Left ventricular chamber instantaneous dimensions were measured using a transit-time ultrasonic technique [14]. One pair of dimension transducers was implanted near the minor equator measuring the antero-posterior diameter (DIA) while the other pair was implanted across the left ventricle free wall near the endocardium and epicardium to measure the wall thickness (WTH). A variety of left ventricular assist devices (LVAD) were used, which provided nonpulsatile left ventricular bypass and a pulsatile piston-type of assist.

An interactive high-resolution microcomputer-based graphics system was used for on-line data recording and analysis. Analog signals were digitized using a 12-bit analog-to-digital converter at 200 samples/sec. A timing classification algorithm based on LV pressure for the determination of cardiac phases was employed. A time averaged beat, derived from 4 to 8 consecutive beats, was used as a representative cardiac cycle.

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5. Results

SX: Hubbard et al. / Differentiation of Intraventricular Fluid Pressure from Ventricular Pressure
obtained using the 3F Millar and the 4F Camino sensors, respectively (Table 1). Measurements were made in the subendocardium and/or midwall and the subepicardium. Sample records of time-averaged signals showing LVP, IMP, and IFP are shown in Fig. 1. The systolic values of IFP were consistently lower than those of LVP, whereas IMP values exceeded the generated LVP. Systolic IFP and IMP values were also found to display a transmural pressure gradient having a similar directional trend. The IMP gradient across the wall was found to be consistently steeper than that of IFP [25].

To examine in greater detail the behavior of IMP and IFP, regional variations in contractile function were induced. Acute ligation of the left anterior descending (LAD) coronary artery was performed to examine the effect of regional ischemia and altered active wall stress on the measurement of subendocardial IMP within and outside the region of hypoperfusion. Figure 2 demonstrates the critical dependency of the subendocardium on myocardial perfusion. Subsequent to a 60 second occlusion, the peak intramyocardial pressure (IMP-1) was reduced in the affected region from 95 to 63 mm Hg. This change was accompanied by a regional systolic dyskinesia, contributing to the observed loss of effective wall thickening. The paradoxical wall thinning during systole and moderate attenuation in

![Image](image-url)

**Fig. 3.** Effect of intravenous infusion of dobutamine on simultaneously measured LVP, IMP, DIA (wall diameter), WTH (wall thickness), STR (wall stress) and the β-adrenergic response associated with it compared to their control values.
Venous hemodynamic data were analyzed for modeling the phase properties of the human heart. The intraventricular pressure (IVP) was calculated as an indicator of left ventricular preload. The pulmonary capillary wedge pressure (PCW) was used as an indicator of right ventricular filling pressure. The left ventricular pressure was decompressed by placing the heart on a pressure pump. Intramyocardial pressure was used to decompress the myocardial layer of the heart, leading to a decrease in myocardial wall stress.

The experimental findings of this model provide evidence that increased left ventricular preload decreases LVP is increasing. However, the calculated myocardial stress exhibited a decrease in peak value. The calculated myocardial stress increased when the heart rate was increased. A greater isovolumic phase of wall stress was observed when the heart rate was decreased. The myocardial stress increased with an increase in heart rate. The myocardial stress decreased with a decrease in heart rate.

To examine the effect of norepinephrine on the myocardial wall stress, the myocardial wall stress was measured before and after the administration of norepinephrine. The myocardial wall stress was increased by the administration of norepinephrine. The myocardial wall stress was decreased by the administration of norepinephrine.

The intramyocardial pressure was used to decompress the myocardial layer of the heart, leading to a decrease in myocardial wall stress. The intramyocardial pressure was decreased by the administration of norepinephrine. The intramyocardial pressure was increased by the administration of norepinephrine. The intramyocardial pressure was decreased by the administration of norepinephrine.
response, but did not obliterate it.

To examine what effect ventricular fibrillation (i.e., lack of organized contractions) will have on the active state of myocardial midwall stress distribution, IMP was recorded in two different transmural regions. The circulation was supported artificially with an extracorporeal pulsatile pump. Coronary blood flow and aortic pressure were monitored to examine their dynamic relationships. In the absence of normal muscle contractions the pulsatile nature of intramyocardial pressure disappeared (Fig. 5). Nonetheless, a transmural gradient persisted in the wall. The in-phase measurement of coronary blood flow and perfusion pressure attests to the fact that the ventricle is behaving as a passive impedance network.

6. Discussion

This overview explored the underlying mechanisms involved in left ventricular wall mechanics with the purpose of elucidating the genesis of intramyocardial pressure (IMP). The great degree of
The observed distribution of intramycocellular pressures across the leaf surface was analogous to the radial stress gradient, decreasing from the periphery to the center of the leaf, indicating the development of internal turgor. The intramycocellular pressure was also found to be influenced by the hydration state of the leaf and the external environmental conditions. The pressure gradient, therefore, plays a crucial role in the turgor pressure distribution, affecting the leaf's mechanical properties and its ability to withstand external forces.

In conclusion, understanding the intramycocellular pressure distribution is essential for elucidating the mechanistic basis underlying the structural integrity and function of leaf tissues. Further research is needed to explore the complex interplay between intracellular pressure and external environmental factors, as well as the role of turgor pressure in the regulation of leaf morphology and function.
interstitial or extracellular pressure. A logical extension of this work was to shift the direction of the research to the cellular level and study the individual constituents of the myocardium (i.e., cardiac myocyte). To broaden the view of the heart as a muscular pump, it is argued here that it is necessary to also gain insight into the subcellular properties of the cardiac myocyte. The generation of fiber stress during myocardial contraction increases transmural fluid pressure of cardiomyocytes. Pressure development in the cavity takes place through pressure increase across the myocyte. Hence, the generation of intraventricular pressure is hypothesized here to be the result of the development of stress in the cardiac fibers. This in turn alters fluid pressure in the intracellular and extracellular fluid. Our fluid-fiber interacting concept eliminates the problem encountered previously with the definition of IMP by equating intramyocardial fiber stress (IFS) to the axial force generated by myofibrils.

As sketched in Fig. 6, intramyocardial pressure is comprised of two components: intramyocardial fiber stress, directly generated by the contractile mechanism, and intramyocardial fluid pressure, which arises as a consequence of cell shortening. Some transducers are sensitive to both and measure a weighted sum, referred to as IMP. Other transducers are able to sense fluid pressure (IFP) alone or a component of stress (IFS) alone. Consequently, the magnitude of IMP can exceed the IFP measurement. Shortening of cells causes left ventricular pressure to rise, thus resisting myocyte
References

Acknowledgements

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The case was for success within the movement.
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