Effects of Acute Valvular Regurgitation on the Oxygen Consumption of the Canine Heart

By Charles W. Urschel, M.D., James W. Covell, M.D., Thomas P. Graham, M.D., Richard L. Clancy, M.D., John Ross, Jr., M.D., Edmund H. Sonnenblick, M.D., and Eugene Braunwald, M.D.

ABSTRACT

The effects on myocardial oxygen consumption and mechanics of acute, simulated aortic and mitral regurgitation were studied in open-chest, anesthetized dogs to determine how changes in the mechanical performance of the ventricle alter oxygen consumption. When regurgitation was induced acutely with effective stroke volume (total stroke volume less regurgitant volume) and heart rate held constant, left ventricular end-diastolic volume, total stroke volume, the ejection fraction, left ventricular wall tension, and the extent of shortening of the contractile element and the circumferential fibers all increased. With volumes of regurgitation approaching effective systemic blood flow, oxygen consumption increased only moderately, despite the increases in tension and shortening. When valvular regurgitation was induced while peak ventricular wall tension was held relatively constant, stroke volume doubled and the extent of both contractile element and circumferential fiber shortening increased. Contractile element work in generating tension was unchanged; that which led to fiber shortening increased substantially. Myocardial oxygen consumption did not increase significantly. Thus, marked increases in the efficiency of the contractile elements and myocardial fibers occurred. The low energy cost per unit of work expended in shortening as opposed to that used for tension development therefore allows the excess stroke volume of valvular regurgitation to be maintained at only a small added oxygen cost to the ventricle.

ADDITIONAL KEY WORDS aortic insufficiency mitral insufficiency cardiac energetics contractile element work myocardial tension

Although the effects of valvular regurgitation on cardiovascular dynamics have been studied intensively for many years (1, 2), relatively little information is available concerning the effects of these lesions on myocardial energy requirements. Although acutely induced aortic and mitral regurgitation have been shown to increase myocardial oxygen consumption (MVO₂) (3-8), the mechanism by which these increases are mediated has not been defined.

It has recently become possible to define with greater precision the relative importance of the various determinants of MVO₂. These include the tension developed by the myocardium (6-9), the contractility of the myocardium as reflected in the velocity of shortening of the unloaded fibers (V_major) (9-12) and myocardial shortening against a load (7, 13-15). Also the effects of acutely induced, simulated, aortic and mitral valvular regurgitation on myocardial mechanics have been examined (16) and such an approach now permits an analysis of the effects of these lesions on MVO₂.

Methods

A bilateral thoracotomy was performed in 11 dogs (avg wt, 20.7 kg) anesthetized with sodium...
Blood was drained from both venae cavae (SVC, IVC) into a disk oxygenator (Oxy.) and pumped with a roller pump through a heat exchanger into the cannulated pulmonary artery (PA). A drain from the right atrium (RA) and ventricle (RV) allowed collection of coronary venous effluent. A stainless steel cannula was placed into the apex of the left ventricle (LV). By opening clamp A, blood was shunted from the left ventricle into the left atrium (LA) during systole (simulated mitral insufficiency). By opening clamp B blood was permitted to flow from the aorta (Ao) to the left ventricle during diastole. Aortic and shunt flows were measured with electromagnetic flow transducers (EMF). Peripheral resistance was controlled by a balloon catheter in the aorta. Stim = electrical stimulator. A.S. = aortic sampling site. SG = Statham P23Db strain gauge.

**Figure 1**

Experimental preparation. Blood was drained from both venae cavae (SVC, IVC) into a disk oxygenator (Oxy.) and pumped with a roller pump through a heat exchanger into the cannulated pulmonary artery (PA). A drain from the right atrium (RA) and ventricle (RV) allowed collection of coronary venous effluent. A stainless steel cannula was placed into the apex of the left ventricle (LV). By opening clamp A, blood was shunted from the left ventricle into the left atrium (LA) during systole (simulated mitral insufficiency). By opening clamp B blood was permitted to flow from the aorta (Ao) to the left ventricle during diastole. Aortic and shunt flows were measured with electromagnetic flow transducers (EMF). Peripheral resistance was controlled by a balloon catheter in the aorta. Stim = electrical stimulator. A.S. = aortic sampling site. SG = Statham P23Db strain gauge.

To prevent reflex changes in contractility, both vagus nerves were sectioned in the neck and dl-propranolol, 0.25 to 0.50 mg/kg, was administered intravenously. These doses of the drug were sufficient to prevent the augmentation of left ventricular dP/dt produced by 0.5 μg of isoproterenol injected into the pulmonary artery.

The effects of simulated valvular regurgitation on MVO₂ were studied in two ways. The first was a comparison between the control state and either

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aortic insufficiency or mitral insufficiency in which regurgitation was produced but left ventricular tension was allowed to vary, while heart rate and effective cardiac output (total left ventricular output minus regurgitant flow) were maintained constant. Since left ventricular tension rose with both interventions, a second type of comparison was made in which the effects of aortic insufficiency or mitral insufficiency were determined while an attempt was made to maintain peak tension constant. In these studies an analog computer was used to provide an indication of left ventricular wall tension during the course of the experiment. This was accomplished by first producing aortic insufficiency or mitral insufficiency and then, during the subsequent control observation, adjusting central aortic pressure, with the inflatable balloon in the descending aorta (Fig. 1), until peak left ventricular tension was matched to the level existing in the presence of regurgitation.

Coronary blood flow was measured by timed collection of the right heart effluent. In all experiments after coronary blood flow and other hemodynamic measurements were stable for 3 to 5 minutes, blood samples were obtained simultaneously from the coronary venous line and central aorta, and were analyzed for oxygen content by the method of Van Slyke and Neill (50). In 8 experiments a Guyton arteriovenous oxygen analyzer (21) was used for continuous monitoring of the approximate coronary arteriovenous O₂ difference (this analyzer is not accurate when arterial PO₂ is very high—399 mm Hg in these experiments) before blood samples were withdrawn for manometric determination of O₂ content. MVO₂ was calculated as the product of coronary blood flow and the coronary arteriovenous O₂ difference. MVO₂ represents the sum of that utilized by the working left ventricle and the empty, nonworking right ventricle. The latter is small (12) and was considered not to change when mitral or aortic regurgitation were induced. Therefore MVO₂ is expressed as ml/min/100 g of left ventricle and related to left ventricular force, velocity, power, and work.

The standard deviation of duplicate determinations of MVO₂ in this preparation has been shown to be 0.20 ml O₂/100 g (11). Recordings of left ventricular pressure, dP/dt, aortic flow, regurgitant flow, aortic pressure and calculated wall tension were made during the period when samples were withdrawn.

Following completion of the experiment, the heart was arrested with potassium chloride, and passive pressure-volume curves of the left ventricle were determined as described elsewhere (22). The left ventricle, including the septum, was then dissected free and weighed.

Calculations

Left ventricular pressure and its derivative (dP/dt), aortic flow, and regurgitant flow were determined at 10-msec intervals during representative contractions from each experiment. Left ventricular end-diastolic volume was determined from the pressure-volume curve of the potassium or anoxia-arrested heart, and instantaneous internal ventricular volume at a given point in ejection was calculated as initial volume less integrated flow to that point. The validity of this method is based on the assumption that ventricular diastolic compliance is unchanged by the procedure used. To ensure that this is the case, the length of a segment of the left ventricle was measured with a continuously recording caliper gauge in seven dogs, and it was observed that neither aortic nor mitral insufficiency altered the relationship between left ventricular end-diastolic pressure and segment length (16). Preliminary data from studies examining the relationship between left ventricular end-diastolic volume determined by the passive-pressure volume curve and by biplane cineangiography show that there is an excellent correlation between the two techniques. In 24 observations, carried out over a wide range of filling pressures and volumes (30 to 100 ml), left ventricular end-diastolic volumes, determined by the pressure-volume curve technique correlated with (r = .84), and averaged 1.6 ± 5 (SEM) ml less than, those obtained by cineangiography.

The calculations of wall tension were carried out in the same way as those previously presented in detail elsewhere (9, 12, 16, 23). The left ventricle was considered to be a thick-walled sphere. Assuming an even distribution of left ventricular muscle, external volume was taken as the sum of internal volume and muscle volume. The radii of the external and internal volumes were computed, ventricular wall thickness being their difference. Assuming that force is evenly distrib-
uted across the ventricular wall, i.e. identical at all radii, it can be shown (24) that the force per unit cross-sectional area (stress or tension, \( T = \frac{g}{cm^2} \)) is equal to \( \frac{P R_i}{2h} \), where \( R_i = \) internal radius, and \( h = \) ventricular wall thickness (24, 25). In brief, this derivation assumes that the total force to be supported by the ventricular wall is equal to the product of the left ventricular pressure and the internal surface area of the left ventricle. It is appreciated that this equation, derived for a thin-walled sphere, will—because of the thickness of the left ventricular wall—provide a relatively constant overestimation of true wall stress (23). It is also appreciated that the relationship of \( \frac{P R_i}{2h} \) to true wall stress may be affected by stress distribution across the ventricular wall which cannot be defined at present.8

As discussed in detail previously (23), we use the terms tension and stress interchangeably to represent force per unit cross-sectional area.

The equation used for calculating the mean velocity of circumferential fiber shortening was modified from that of Fry et al. (25). For this derivation the ventricular wall is assumed to be a series of concentric thin-walled spheres. The instantaneous circumferential fiber-shortening velocity (\( V_{CF} \)) in cm/sec at the midwall can be calculated as

\[
V_{CF} = \frac{Q}{2R_i R_e}
\]

where \( Q = \) instantaneous flow rate out of the ventricle in cm3/sec, \( R_e = \) external radius in cm, and \( R_i = \) internal radius in cm. \( V_{CF} \) and tension describe the velocity of shortening and the force necessary to approximate the ends of a hypothetical band of muscle (1.0 cm2 cross-sectional area) encircling the ventricle at the midwall radius. All subsequent calculations describe the behavior of this hypothetical band.

Contractile element velocity (\( V_{0E} \)) equals \( V_{CF} \) plus the velocity of elongation of the series contractile element and circumferential fiber work. Contractile element work (solid circles) and circumferential fiber-shortening work (open squares) are shown as a function of time during a single cardiac cycle in the control period. Prior to the onset of ejection (arrow 1), contractile element shortening performs work only by stretching the series elastic component. From the onset of ejection until peak tension (arrow 2), the contractile element continues to stretch the series elastic, as well as performing circumferential fiber work. Area A represents the total work performed by the contractile elements in generating tension. Following peak tension (arrow 2), circumferential fiber work is derived both from contractile element work and energy released in the recoil of the stretched series elastic component (area C). Fiber-shortening work therefore comprises a fraction of contractile element work, "effective" contractile element work, area B, and the recoil of the series elastic component, area C.

![Figure 2](image-url)
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elastic component \( V_E \) of the Hill muscle model (26). \( V_E = \frac{dT}{dE} \), where \( kT \) equals the modulus of elasticity of the series elastic component of the muscle (27). The normalized value for \( k \) used in this study, i.e. 28, is based upon 23 observations on isolated heart muscle (27).

Contractile element work was determined by integrating the instantaneous contractile element power curve, calculated as the product of \( V_E \) and tension, at 10-msec intervals. Contractile element work can be considered to be the sum of two separate components: work which results in stretching of the series elastic component and work which results in shortening of myocardial fibers. The first component was calculated as the product of \( V_E \) and \( T \) and the latter, termed "effective

![Graphs showing calculated ventricular mechanics during experiments with mitral insufficiency (MI) and aortic insufficiency (AI) in which tension was not controlled. A-C are from an experiment with mitral insufficiency and panels D-F are from similar experiments with aortic insufficiency. CE = contractile element. CEAl = extent of shortening of the contractile element in centimeters. Arrows denote the onset of ejection.](http://circres.ahajournals.org/)

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Effects of Mitral Insufficiency and of Aortic Insufficiency (Means ± 1s)

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>Mitral insufficiency</th>
<th>Aortic insufficiency</th>
<th>Control</th>
<th>Mitral insufficiency</th>
<th>Aortic insufficiency</th>
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<tr>
<td><strong>Aortic systolic pressure (mm Hg)</strong></td>
<td>106 ± 5</td>
<td>105 ± 4</td>
<td>121 ± 10</td>
<td>123 ± 7</td>
<td>106 ± 6</td>
<td>114 ± 6</td>
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<tr>
<td><strong>Aortic diastolic pressure (mm Hg)</strong></td>
<td>75 ± 5</td>
<td>71 ± 5</td>
<td>90 ± 8</td>
<td>65 ± 6*</td>
<td>96 ± 5</td>
<td>81 ± 6*</td>
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<tr>
<td><strong>Total stroke volume (ml)</strong></td>
<td>13.8 ± 1.2</td>
<td>13.9 ± 1.2*</td>
<td>11.3 ± 1.1</td>
<td>12.2 ± 2.4*</td>
<td>11.8 ± 1.0</td>
<td>24.0 ± 2.0*</td>
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<tr>
<td><strong>Regurgitant stroke volume (ml)</strong></td>
<td>10.8 ± 1.0</td>
<td>10.8 ± 1.0</td>
<td>11.6 ± 1.2</td>
<td>11.6 ± 1.2</td>
<td>11.5 ± 1.4</td>
<td>13.0 ± 1.7</td>
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<td><strong>LV end-diastolic volume (ml)</strong></td>
<td>25.7 ± 2.5</td>
<td>33.7 ± 2.8*</td>
<td>33.7 ± 3.6</td>
<td>43.5 ± 2.8*</td>
<td>29.5 ± 2.6</td>
<td>34.6 ± 2.3*</td>
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<td><strong>Fraction ejected</strong></td>
<td>0.53 ± 0.03</td>
<td>0.51 ± 0.04*</td>
<td>0.38 ± 0.05</td>
<td>0.52 ± 0.06*</td>
<td>0.63 ± 0.04</td>
<td>0.70 ± 0.05</td>
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<tr>
<td><strong>Peak LV wall tension (g/cm²)</strong></td>
<td>91 ± 7</td>
<td>109 ± 6*</td>
<td>120 ± 12</td>
<td>149 ± 18*</td>
<td>118 ± 9</td>
<td>110 ± 7</td>
</tr>
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<td><strong>Resting LV wall tension (g/cm²)</strong></td>
<td>4.8 ± 0.4</td>
<td>11.0 ± 1.1*</td>
<td>7.8 ± 1.3</td>
<td>13.8 ± 2.9*</td>
<td>8.4 ± 1.2</td>
<td>11.7 ± 1.2*</td>
</tr>
<tr>
<td><strong>Total CE work (g-cm)</strong></td>
<td>24.4 ± 0.13</td>
<td>2.93 ± 0.16*</td>
<td>2.95 ± 0.14</td>
<td>2.68 ± 0.19*</td>
<td>2.38 ± 0.13</td>
<td>2.55 ± 0.19*</td>
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<tr>
<td><strong>CE work on SE (g-cm)</strong></td>
<td>53 ± 5</td>
<td>59 ± 5*</td>
<td>68 ± 4</td>
<td>50 ± 6*</td>
<td>64 ± 7</td>
<td>60 ± 7</td>
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<tr>
<td><strong>Effective CE work (g-cm)</strong></td>
<td>99 ± 10</td>
<td>127 ± 12*</td>
<td>87 ± 11</td>
<td>173 ± 28*</td>
<td>98 ± 9</td>
<td>140 ± 14*</td>
</tr>
<tr>
<td><strong>Periodontal fiber shortening (cm)</strong></td>
<td>1.59 ± 0.11</td>
<td>2.74 ± 0.15*</td>
<td>1.11 ± 0.14</td>
<td>1.59 ± 0.20*</td>
<td>1.39 ± 0.16</td>
<td>2.80 ± 0.28*</td>
</tr>
<tr>
<td><strong>Periodontal fiber shortening work (g-cm)</strong></td>
<td>103 ± 12</td>
<td>168 ± 15*</td>
<td>104 ± 11</td>
<td>208 ± 24*</td>
<td>117 ± 11</td>
<td>154 ± 17*</td>
</tr>
<tr>
<td><strong>Regurgitant fiber shortening work/CE work</strong></td>
<td>0.77 ± 0.02</td>
<td>0.90 ± 0.01*</td>
<td>0.68 ± 0.03</td>
<td>0.82 ± 0.02*</td>
<td>0.74 ± 0.02</td>
<td>0.90 ± 0.02*</td>
</tr>
<tr>
<td><strong>Coronary blood flow (ml/min)</strong></td>
<td>2.9 ± 0.5</td>
<td>3.1 ± 0.5*</td>
<td>3.14 ± 0.22</td>
<td>3.30 ± 0.21</td>
<td>3.00 ± 0.17</td>
<td>3.09 ± 0.07*</td>
</tr>
<tr>
<td><strong>MVO₂ (ml/min/100g LV)</strong></td>
<td>12.4 ± 1.7</td>
<td>12.0 ± 1.8</td>
<td>112 ± 19*</td>
<td>135 ± 17</td>
<td>100 ± 8</td>
<td>140 ± 10</td>
</tr>
<tr>
<td><strong>Heart rate (beats/min)</strong></td>
<td>141 ± 5</td>
<td>141 ± 4</td>
<td>139 ± 4</td>
<td>139 ± 4</td>
<td>146 ± 3</td>
<td>143 ± 3</td>
</tr>
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</table>

*P < 0.01, †P < 0.05, comparing data from the period with insufficiency with that from the control period (33).
contractile element work," was the difference between total contractile element work and contractile element work performed upon the series elastic component. Figure 3 illustrates this separation of contractile element work into these two components and further illustrates that external fiber-shortening work is the sum of effective contractile element work and an additional increment of work regained from the recoiling series elastic component. The percent of total contractile element work which appears as fiber-shortening work will therefore depend on the degree to which energy stored in the series elastic component before the development of peak tension is regained during the latter portion of ejection.

Wall tension was estimated during the course of the experiment by analog computer techniques. Left ventricular end-diastolic volume and muscle volume were estimated from normalized data. Following completion of the experiment, when actual values for left ventricular weight and the ventricular pressure-volume curve were available, final calculations were made with a digital computer.

Contrast element and circumferential fiber efficiencies were calculated as:

\[ \text{minute work} \times 10^{-3} \times \text{contractile element or circumferential fiber} \]
\[ \text{[CBF} \times \text{arteriovenous } \text{O}_2 - 1.5 \times (\text{LV wt/100})] \times 2.06 \]

where CBF is the coronary blood flow, 1.5 is the MVO$_2$ of the arrested left ventricle (LV) in ml/min/100 g (9, 25, 29), and 2.06 is the work equivalent of oxygen in kg/ml O$_2$ (30).

### Results

**Valvular Regurgitation with Tension Allowed to Vary but Constant Effective Forward Blood Flow**

**Mitral Insufficiency.**—In the seven dogs in which mitral insufficiency was induced (average regurgitant volume = 10.8 ml/stroke) aortic systolic and diastolic pressures were unchanged (Table 1). Total stroke volume (aortic flow plus left ventricle-atrium flow) increased from an average of 13.3 to 23.7 ml. Left ventricular end-diastolic volume rose

![Graph](http://circres.ahajournals.org/)

**FIGURE 4**

Calculated changes in MVO$_2$ during experiments with mitral insufficiency (M1) and aortic insufficiency (A1) plotted as a function of changes in effective contractile element work (ΔCEEW) and extent of contractile element shortening. Tension was held constant during these experiments. The dashed horizontal lines at MVO$_2$ of ±0.5 ml/min/100 g LV illustrate the range of ±2 s of the differences between duplicate determinations of MVO$_2$. 
from 0.53 to 0.71. Peak left ventricular wall tension increased from 91 to 108 g/cm² (Fig. 3A, Table 1). The extent and velocity of shortening of the contractile elements (Fig. 3B) and circumferential fibers, as well as the contractile element work and circumferential fiber work increased substantially (Fig. 3B, Table 1). Associated with these changes in myocardial mechanics, MVO₂ increased only slightly, from 8.9 to 10.1 ml/100 g LV/min (P<0.01). Consequently, calculated contractile element and circumferential fiber efficiencies increased substantially, by 22% (P<0.05) and 40% (P<0.01) of control, respectively. Contractility was unchanged in all experiments, as defined by extrapolation of the isovolumic portion of the force-velocity relation to zero tension (Vₘₚₑₓ) (Fig. 3C, Table 1).

**Aortic Insufficiency.**—When aortic insufficiency (average regurgitant volume = 11.6 ml/stroke) was induced in six dogs, diastolic aortic pressure fell, while total stroke volume rose from 11.3 to 22.2 ml (Table 1). As was observed with mitral insufficiency, the left ventricular end-diastolic volume and the fraction ejected rose, while peak tension increased from 120 to 146 g/cm² (Fig. 3D). Contractile element and myocardial fiber shortening velocity (Fig. 3E) and work increased in a manner similar to that observed in mitral insufficiency.

MVO₂ increased from 10.9 ml/100 g LV/min to 12.7 (P<0.01) and calculated contractile element and myocardial fiber efficiencies increased by 26% and 51% of control, respectively (P<0.01). As with mitral insufficiency, extrapolated Vₘₚₑₓ remained constant, demonstrating that contractility was unchanged (Fig. 3F).

**Discussion**

In an earlier investigation on the acute hemodynamic effects of simulated aortic and mitral regurgitation (16), it was observed that these lesions do not affect the fundamental contractile state of the myocardium but that the profound changes in ventricular dynamics which occur can be explained by the marked alterations in the instantaneous load induced by these lesions. In contrast to this earlier study, in the present experiments the effects of valvular regurgitation were studied in a right heart bypass preparation. This experimental design permitted elucidation of the effect of regurgitation per se both on ven-
tricular dynamics and on myocardial energetics, while maintaining net effective forward flow constant.

In the experiments in which ventricular afterload was allowed to vary, it was observed that the induction of regurgitant flows of magnitudes approximating the forward cardiac output resulted in modest elevations of the ventricular preload (left ventricular end-diastolic volume) and in peak left ventricular wall tension, as well as relatively large increases in the total stroke volume and ejection fraction. In addition, the calculated work performed by the contractile elements rose with both valvular lesions. As already indicated, the latter variable may be considered to be composed of work performed by the contractile elements in stretching the series elastic component as well as in shortening the myocardial fibers. Work expended on the series elastic component is a function of wall tension, and since the latter rose with the induction of valvular regurgitation, a modest increase in work performed in stretching the series elastic component occurred, both with mitral insufficiency and aortic insufficiency (23% and 21%, respectively). Effective contractile element work, that fraction which leads directly to fiber shortening, increased more substantially in mitral insufficiency and aortic insufficiency, by 48% and 99%, respectively.

It has been noted previously that with both valvular lesions the effectiveness of energy transfer from the contractile element across the series elastic component to the myocardial fibers is increased (16). This results primarily from the lower impedance to ejection which reduces ventricular size in late systole, lowering wall tension at this time and allowing the energy stored in the stretched series elastic component to be regained as fiber shortening. As a consequence, circumferential fiber work rose substantially, by 63% and 100% with mitral insufficiency and aortic insufficiency, respectively.

The major finding of this investigation is that these striking changes in left ventricular dynamics are associated with only minimal alterations in myocardial energetics, \( MVO_2 \), rising by an average of only 16% with mitral insufficiency and 17% with aortic insufficiency. Left ventricular wall tension has been shown to be an important determinant of \( MVO_2 \) (6-9, 31) and since developed wall tension increased moderately when regurgitation was induced, the elevations of \( MVO_2 \) may be attributed in part to these increases in wall tension. On the basis of previous estimates of the oxygen cost of left ventricular tension development (9), it may be calculated that the increases in developed tension resulting from aortic insufficiency and mitral insufficiency would, by themselves, increase \( MVO_2 \) by average amounts of 0.8 ml and 0.5 ml/min/100 g LV, respectively, leaving average increments of only 1.0 ml/min/100 g LV and 0.7 ml/min/100 g LV which must be accounted for on another basis. Since myocardial contractility, as reflected in the maximum velocity of shortening of the unloaded muscle (\( V_{max} \)), was unaltered by the production of valvular regurgitation (16) (Fig. 3, C and F), it appears likely that the small increments in \( MVO_2 \) which were not attributable to increases in myocardial tension were related to the marked increases in effective contractile element work.

It has been appreciated for many years that a given augmentation of external cardiac work produced by increasing stroke volume is associated with a much smaller energy requirement than a similar augmentation of work produced by increasing aortic pressure (32). Recently, studies performed on isolated papillary muscles (14, 15) have shown that the energy costs of contractile element work associated with fiber shortening are far smaller than those of equivalent levels of contractile element work performed on the series elastic component. The results of the present investigation with the intact heart are consistent with these concepts, since effective contractile element work increased substantially with both mitral insufficiency and aortic insufficiency and the associated increments in \( MVO_2 \) are not attributable to increases in tension which were very small. As a consequence, when
efficiency was calculated as the ratio either of contractile element work or myocardial fiber work to MVO₂, large increases were observed, both with aortic insufficiency and mitral insufficiency.

To identify with greater precision the contribution of contractile element shortening to MVO₂, a second group of experiments was performed in which valvular regurgitation was produced, but an attempt was made to maintain peak left ventricular wall tension constant. Under these circumstances, the average values of MVO₂ did not change significantly, with either mitral insufficiency or aortic insufficiency. In these experiments there were no changes in the calculated work performed by the contractile elements in stretching the series elastic components. However, effective contractile element work increased markedly, by an average of 46% for mitral insufficiency and an average of 56% for aortic insufficiency, and myocardial fiber shortening work also rose strikingly, by an average of 57% for mitral insufficiency and 64% for aortic insufficiency. The calculated efficiencies of the contractile elements and myocardial fibers were, again, greatly augmented.

In conclusion, the results of this investigation emphasize the importance of the instantaneous load on the myocardium to the relationship between the mechanical and energetic aspects of cardiac contraction. At a constant preload, acutely induced valvular regurgitation reduces the afterload on the ventricle and thus allows a greater fraction of the contractile activity to be manifest in the shortening rather than the tension developed by the myocardial fibers. Since, as we have emphasized by this investigation, the energy expended by the contractile elements in shortening the myocardial fibers is very small relative to the energy cost of stretching the series elastic component, i.e., developing tension, the total energy costs of acutely induced mitral and aortic valvular regurgitation are relatively modest, and can be explained in part by the increases in tension which are induced.

References
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