Cardiac function, coronary flow, and oxygen consumption in stable left ventricular hypertrophy

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Malik, Asrar B., Tomio Abe, Hugh O'Kane, and Alexander S. Geha Cardiac function, coronary flow, and oxygen consumption in stable left ventricular hypertrophy. Am. J. Physiol. 225(1): 185-191. 1973.—We have compared cardiac function, coronary flow, and oxygen consumption of the canine left ventricle in stable hypertrophy (LVH) with those of the normal left ventricle (N). LVH was produced by banding the ascending aorta early in life. Nine dogs with LVH and 13 normal dogs were anesthetized with 30 mg/kg sodium pentobarbital. Coronary flow to the left ventricle (QLv) was measured by the dye-dilution technique, and pressures were monitored in the aorta and left ventricle. Oxygen contents of the arterial and LV effluent blood were determined and LV oxygen consumption (~LvO~) was calculated using the Fick equation. The results indicate that various indices of left ventricular function such as Q, minute work, stroke work, external mechanical efficiency, peak L.V.dP/dt, ratio of peak L.V.dP/dt to L.V.EPD, and ratio of peak L.V.dP/dt to isovolumic pressure were in the normal or above-normal range in LVH. However, the mean QLv of 74 ± 12 ml/min per 100 g LV wt in LVH was significantly lower than 101 ± 8 ml/min per 100 g LV wt in N as a result of a decreased flow in late systole, probably due to increased myocardial systolic compression. Despite the lower mean QLv in LVH, ~LvO~ was maintained at normal levels (9.40 ± 1.60 ml/min per 100 g LV wt in N and 9.13 ± 1.75 ml/min per 100 g LV wt in LVH) through greater oxygen extraction by the hypertrophied myocardium.

METHODS

Left ventricular hypertrophy (LVH) was produced by banding the ascending aorta in 7-week-old puppies. The band was adjusted to provide a 15–20 mm Hg peak systolic pressure gradient across the constriction. All long-term survivors (70% of the operated dogs) developed electrocardiographic, radiographic, and anatomic evidence of LVH which reached a stable stage within 6 months after banding (29). The present study was carried out in nine dogs with LVH 9–18 months after banding and in 13 normal mongrel dogs. The animals, varying in weight between 12 and 28 kg, were anesthetized with 30 mg/kg sodium pentobarbital intravenously, intubated with an endotracheal tube, and ventilated with a Harvard respirator supplied with oxygen. A left thoracotomy was performed and 50 cm 0 F (id 1.73 mm) Teflon catheters were positioned in the aorta distal to the band and in the left ventricle via the left atrial appendage.

It has been suggested that cardiac hypertrophy is an adaptive response of the heart to a sustained increase in work load that enables it to adjust its mass to meet the increased work requirements. This process of cardiac growth may be separated into three stages: 1) developing cardiac hypertrophy, a period of active accumulation of myocardial components; 2) stable cardiac hypertrophy, a steady-state in the synthetic and degradative rates; and 3) hypertrophy with failure which may follow prolonged or severe increase in work load (27). The isolated hypertrophied myocardium has been extensively studied, but there is little information regarding the functional changes occurring in the stage of stable hypertrophy in the intact heart.

In light of the studies available in the literature, the question whether stable cardiac hypertrophy is associated with decreased myocardial contractility has been controversial (31). Bing et al. (5) demonstrated that the maximum velocity of shortening (Vmax) of the isolated left ventricular trabecular carnea from the hypertrophied hearts of aortic-constricted rats was depressed 1–4 weeks after the constriction. Spagnu et al. (36) have found a depression in Vmax of patients with left ventricular hypertrophy and no evidence of cardiac failure. On the other hand, several studies have shown normal or enhanced cardiac function and contractility in animals and human subjects with hypertrophied non-failing hearts and in isolated hypertrophied myocardium from nonfailing hearts (1, 4, 8, 13, 16, 23).

In addition to the variability of data on the cardiac contractility, there is little reliable information concerning changes in coronary flow and myocardial oxygen consumption of the stable hypertrophied myocardium in the intact animal (2). We have produced stable left ventricular hypertrophy in dogs by banding the ascending aorta 7 weeks after birth and then allowing the animals to gradually develop hypertrophy in response to increasing stenosis (29). The hypertrophy was stabilized within 6 months after banding as evidenced by no further increase in left ventricular weight. This model was used to study the performance, coronary blood flow, and oxygen consumption of the intact hypertrophied left ventricle and the results were compared with the normal left ventricle.
age. Since the main left coronary artery in the dog is too short for placement of a flow probe, segments of the left circumflex and left anterior descending coronary arteries were dissected free from the epicardial tissue at their origins for placement of electromagnetic flow probes (Micron Instruments, Inc.). Small branches arising proximal to the probe sites were ligated without any electrocardiographic changes. The diameters of the flow transducers ranged from 2.5 to 3.5 mm. A small-bore catheter was advanced from a small epicardial vein into the great coronary vein to sample the effluent blood from the left heart. The epicardial vein was ligated around the catheter. Blood flows in the left circumflex and left anterior descending coronary arteries were recorded on a Beckman Type R Dynograph using Micron Instruments model RC1000, square-wave, electromagnetic, blood flowmeters. Total left coronary flow (QLV) was calculated from the sum of flows in the two arteries and was considered to represent left ventricular coronary flow. Although the flowmeter is capable of giving zero-flow output without occluding the vessels, the accuracy of the electronic zero was verified in each instance by temporarily occluding the coronary arteries proximal and distal to the site of the flow probes. There were no branches between the flow probes and the sites of occlusion. The zero-flow deflections were flat and the mechanical zero was equivalent to the electronic zero in all cases. Left ventricular pressure (LVP), the first derivative of LVP (LVP/dt), aortic pressure, and ECG were simultaneously recorded on a Hewlett-Packard model 7700 recorder. Pressures were obtained using Hewlett-Packard model 1830C pressure transducers and were referred to the midplane of the left ventricle. The catheter-manometer system gave a 90% response to a square wave of pressure in 0.01 sec, and the frequency response of the system was greater than 35 Hz. All the determinations were recorded at paper speeds of 25 mm/sec and 100 mm/sec. LVP/dt was continuously calculated using the Hewlett-Packard 8814A derivative computer with an R-C differentiating circuit having a time constant of 8.25 X 10^-8 sec. Mean pressures were derived by electronic integration of pulse pressures. Cardiac output (Q) was determined by the indicator-dilution technique on a Beckman Instruments Cardiac-Densitometer. The indicator, indocyanine green (Cardio-Green), was injected into the left ventricle and its concentration was sampled in the aorta.

Blood samples were collected anaerobically from the aortic and the LV venous catheters for blood gases, pH, oxygen content, hemoglobin, lactate, and pyruvate determinations. Blood gases and pH were determined on the Instrumentation Laboratory model 113 blood gas and pH analyzer. Lactates and pyruvates were determined enzymatically (25). Oxygen contents were obtained from the hemoglobin and oxygen tension values and by the Van Slyke method. The values with both methods were not significantly different, which confirmed previous observations (19). The hemodynamic determinations and blood samples were taken simultaneously and in duplicate to ensure that the values were reproducible. The difference between consecutive cardiac output measurements was less than ±3%. At the end of the study, the animals were sacrificed, the hearts were examined, and the left and right ventricles were separated (17) and their individual weights determined.

Left ventricular minute work (W) in kilogram meters/ min (kg-m/min) and stroke work (SW) in gram-meters (g-m) were obtained by the formulas: W = (SW X HR)/ 1,000 and SW = (SV X (LVPm - LVEDP)) X 0.0144, where HR = heart rate (beats/min); SV = stroke volume (ml); LVPm = mean ventricular systolic pressure (mm Hg); LVEDP = LV end-diastolic pressure (mm Hg), and 0.0144 = a constant which takes into consideration the conversion of millimeters Hg to centimeters H2O (1.36), milliliters of blood to grams (1.06), and centimeters to meters (34). Both SW and W were normalized per 100 g LV wt. Peak LVP/dt determinations were affected by variations in preload and afterload; an attempt was made to correct for these factors. LVP/dt was divided by LVEDP as well as by isovolumic pressure (IP), i.e., the difference between maximum isovolumic pressure and LVEDP (37). Extern mechanical efficiency (EME) of the left ventricle was expressed as the ratio of left ventricular work to the work equivalent of left ventricular oxygen consumption. EME was calculated by the formula: EME = (W/QLV) x 2.059, where W = LV minute work (in kg-m/min); QLV is LV oxygen consumption (in ml/min); and 2.059 is the work equivalent (kg-m/ml) of oxygen consumption at a cardiac respiratory quotient of 0.89 (6). QLVm (mil/min) was obtained from the product of QLV (mil/min) and the arteriovenous difference in oxygen content across the left ventricle. Both QLVm and QLV were also normalized per 100 g LV wt.

RESULTS

The data obtained in the LVH group were compared with those in the normal (N) group with the use of the two-tailed Student t test. The stability of LVH was confirmed by normal LVEDP and absence of any evidence of pulmonary edema or cardiac decompensation. Mean left ventricular weight was 120 ± 10 g in LVH and 91 ± 6 g in N, while the right ventricular weights were not significantly different (Fig. 1). The ratio of LV weight to RV weight was significantly higher in LVH (Fig. 1). The right ventricular weight-to-body weight ratio in N of 2.7 X 10^-6 ± 0.16 was not significantly different from the right ventricular weight-to-body weight ratio in LVH of 3.0 X 10^-3 ± 0.25. However, the left ventricular weight-to-body weight ratio in LVH of 6.6 X 10^-3 ± 0.51 was significantly greater (P < 0.01) than the value of 4.2 X 10^-3 ± 0.36 in N. These data indicate the selectivity of the left ventricular hypertrophy.

The comparison of the two groups is summarized in Tables 1 and 2.

Cardiac function. Cardiac output, normalized for body weight in kilograms, was significantly greater in LVH than in N. Heart rates in N and LVH were not significantly different. Stroke volume in LVH of 96.0 ± 4.8 ml was significantly greater (P < 0.05) than the mean value in N of 16.5 ± 2.8 ml.

The phasic left ventricular pressures and dP/dt in the two groups are shown in Fig. 2. The mean and peak left ventricular systolic pressures in LVH were higher than in N; LVEDP in the two groups were not significantly different. Aortic pressures in N and in LVH distal to the band were similar. The peak systolic gradient across the band averaged
was observed only in the hypertrophied group (Fig. 3).

The derived parameters of cardiac function, such as SW, W, EME, and peak (LVdp/dt)/LVEDP, were all significantly higher in LVH than in N. However, SW and W normalized per 100 g LV wt, and peak (LVdp/dt)/IP were not significantly different in the two groups.

Coronary flow. Table 2 shows the peak diastolic and late systolic phasic flows in the left anterior descending and the left circumflex arteries as well as the mean left coronary flow in the two groups. Flows are expressed per 100 g LV wt. The results indicated that mean left coronary flow per 100 g LV wt was significantly lower (P < 0.05) in LVH as a result of the differences in the systolic flow pattern and no change in the diastolic pattern. Representative pulsatile flows in the left circumflex artery of normal and hypertrophied left ventricles are shown in Fig. 3. Retrograde flow in late systole was observed only in the hypertrophied group (Fig. 3).

Metabolism and oxygen consumption. Table 2 shows the oxygen content of arterial blood (CaO₂) and effluent blood from the LV (CLvO₂), the arteriovenous difference in oxygen content across the LV, and the LV oxygen consumption per 100 g LV wt. The data indicate a significantly lower (P < 0.05) CLvO₂ in LVH which results in a significantly wider (P < 0.05) arteriovenous difference in oxygen content. There was a significantly higher (P < 0.05) oxygen extraction ratio in LVH (0.52 ± 0.03 in N and 0.65 ± 0.02 in LVH). The lactate-to-pyruvate ratio in the effluent blood from the left ventricle in LVH was 27.3 ± 2.8, a value significantly greater (P < 0.05) than that of 19.1 ± 2.3 in N.

DISCUSSION

Myocardial hypertrophy occurs in response to chronic increase in cardiac work. It has been suggested that hypertrophy is an adaptive mechanism which allows the heart to handle the increased work load (30). However, the basic question whether hypertrophy is associated with any change in cardiac function has been controversial. Most previous studies have considered the mechanical properties of the isolated hypertrophied myocardium. Kerr et al. (18) observed an increase in the maximum tension developed per unit weight of papillary muscle from the hypertrophied left ventricle of rats. In a similar preparation, Grimm et al. (16) noted that the peak tension developed was not significantly different from normal papillary muscles. Studies by Spann et al. (35) and Bing et al. (5) on isolated strips of cardiac muscle from hypertrophied hearts have indicated a depression of Vmax and the tension developed. Thus results obtained from previous studies on isolated muscle preparations have been equivocal regarding the contractile state of the myocardium in stable hypertrophy. Fisher and Kaveler (12) have pointed out that the most sensitive index of contractility, the maximum contractile capability of the isolated muscle, was not studied by previous workers with the exception of Spann et al. (35). Studies using the isolated muscle preparation also suffer from problems such as the accumulation of metabolic wastes in the water bath and the lack of adequate oxygenation because of increase in diffusion distance. Fisher and Kaveler (12) were able to avoid these difficulties by determining the maximum rate of increase of force in the in situ hypertrophied right ventricular papillary

| Table 1. Cardiac function in dogs with normal and hypertrophied left ventricle |
|---------------------------------|-----------------|------------------|
|                                | Normal          | Left Ventricular Hypertrophy |
| Cardiac output, ml/min per kg body wt | 109 ± 9         | 155 ± 12         |
| Heart rate, beats/min         | 138 ± 8         | 163 ± 10         |
| Stroke volume, ml             | 16.5 ± 2.8      | 26.0 ± 4.8       |
| Mean LV systolic pressure, mm Hg | 74 ± 4          | 92 ± 5           |
| LVEDP, mm Hg                  | 11 ± 3          | 11 ± 2           |
| Peak LV systolic pressure, mm Hg | 154 ± 10        | 194 ± 10         |
| Peak LVdp/dt, mm Hg/sec       | 2,619 ± 221     | 4,166 ± 324      |
| LV stroke work, g-m/min       | 15.1 ± 2.5      | 22.2 ± 1.9       |
| LV stroke work, g-m/100 g LV wt | 17.2 ± 2.9      | 18.1 ± 3.5       |
| LV minute work, kg-m/min      | 2.13 ± 0.25     | 3.32 ± 0.29      |
| LV minute work, kg-m/min per 100 g LV wt | 2.77 ± 0.32 | 2.82 ± 0.38 |
| External mechanical efficiency, % | 16.0 ± 1.6 | 22.3 ± 2.0 |
| Peak (LV dp/dt)/LVEDP, sec⁻¹  | 277 ± 52        | 422 ± 41         |
| Peak (LV dp/dt)/IP, sec⁻¹     | 99.1 ± 3.5      | 28.9 ± 2.2       |

All values are means ± se. P values refer to the comparison between the two groups using the Student t test.
TABLE 2. Coronary flow and oxygen consumption in dogs with normal and hypertrophied left ventricles

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Left Ventricular Hypertrophy</th>
<th>P</th>
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<tbody>
<tr>
<td>Peak diastolic flow, ml/min per 100 g LV wt</td>
<td></td>
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<tr>
<td>LAD</td>
<td>73 ± 8</td>
<td>62 ± 12</td>
<td>NS</td>
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<tr>
<td>CIRCUM</td>
<td>84 ± 11</td>
<td>83 ± 17</td>
<td>NS</td>
</tr>
<tr>
<td>Late systolic flow, ml/min per 100 g LV wt</td>
<td></td>
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<td></td>
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<tr>
<td>LAD</td>
<td>22 ± 7</td>
<td>2 ± 2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>CIRCUM</td>
<td>12 ± 3</td>
<td>-3 ± 2</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Mean left coronary flow, ml/min</td>
<td>93 ± 7</td>
<td>80 ± 10</td>
<td>NS</td>
</tr>
<tr>
<td>Mean left coronary flow, ml/min per 100 g LV wt</td>
<td>101 ± 8</td>
<td>74 ± 12</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Arterial oxygen content, ml/100 ml blood</td>
<td>17.2 ± 0.9</td>
<td>18.4 ± 1.1</td>
<td>NS</td>
</tr>
<tr>
<td>LV venous oxygen content, ml/100 ml blood</td>
<td>8.6 ± 0.7</td>
<td>6.9 ± 0.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Arteriovenous difference, ml/100 ml blood</td>
<td>8.9 ± 1.1</td>
<td>11.1 ± 0.7</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LV oxygen consumption, ml/min per 100 g LV wt</td>
<td>9.40 ± 1.60</td>
<td>9.13 ± 1.75</td>
<td>NS</td>
</tr>
</tbody>
</table>

All values are mean ± 1 se. P values refer to the comparison between the two groups using the Student t test.

FIG. 2. Phasic left ventricular pressures and dp/dt in dogs with normal and hypertrophied left ventricles. Paper speed, 25 mm/sec.

FIG. 3. Phasic left circumflex coronary flow and phasic aortic pressure in a dog with a normal left ventricle and a dog with left ventricular hypertrophy. Aortic pressures in dog with left ventricular hypertrophy were obtained proximal to band. Paper speed, 50 mm/sec.

Meerson and Pshennikova (28) found cardiac function was depressed in the hypertrophied left ventricle of rabbits. Spann et al. (36) demonstrated that V_{max} was depressed in conscious patients with hypertrophied nonfailing hearts, while Levine and associates (22) found no change in the force-velocity relations in a similar group of patients. However, the interpretation of V_{max} as a reflection of contractility is subject to criticism, as its determination in the intact left ventricle is based on assumptions. In addition, the possibility that the reduction in contractility was secondary to heart disease rather than to hypertrophy itself could not be ruled out in studies on patients. Spann et al. (35) have suggested that contractility of the hypertrophied muscle is depressed, but pump fraction was not altered. The cardiac output in cats with hypertrophied right ventricles was higher than in the normal cats; however, isolated muscle studies using hearts of the same cats, demonstrated a depression in tension development and in V_{max}. It was proposed that despite the decrease in myocardial contractility, the pump function of the hypertrophied heart is maintained by a combination of the Frank-Starling mechanism and increased sympathetic stimulation (35).

There have been no studies to the authors knowledge that have measured both cardiac muscle and pump function during the stage of stable ventricular hypertrophy in the intact animal. The canine left ventricle offers distinct advantages for a comprehensive study of its function, coronary flow, and oxygen consumption because of its configuration and the anatomy of the coronary circulation. We have produced an experimental model of stable left ventricular hypertrophy in the adult dog (29).

The present study shows that the cardiac muscle and pump functions in the hypertrophied left ventricle were not depressed in comparison to the normal left ventricle. Minute work, stroke work, cardiac output, and mechanical efficiency of the hypertrophied left ventricle were all higher than those of the normal left ventricle, although the end-diastolic pressures were similar. However, both minute and stroke work, when normalized per 100 g left ventricular weight, were not significantly different in hypertrophy. The indices of the contractile state of cardiac muscle, such as peak LVdP/dt and the ratio of peak LVdP/dt to LVEDP, were also higher in the hypertrophied left ventricle. However, the ratio of peak LVdP/dt to peak isovolumic pressure...
was not altered. Thus, various parameters of cardiac function in the intact hypertrophied left ventricle were in the normal or above-normal range. Although these studies were carried out in the anesthetized, open-chest dog, they are consistent with previous observations on the performance of the hypertrophied right ventricle in the conscious dog (13). Acute reduction of the cross-sectional area of the aorta of an adult dog to a degree comparable to that produced in the present study results in depressed cardiac function (20), suggesting that ventricular hypertrophy associated with a chronic increase in afterload is an adaptation that allows the heart to meet the increased work requirements.

Coronary circulation in cardiac hypertrophy has been most extensively studied morphologically because of the difficulties in measuring coronary flow in vivo. Postmortem studies of hearts in which hypertrophy was produced by pressure or volume overload indicate that the ratio of capillaries to muscle fibers did not change (30). However, there was an increase in the distance from the capillaries to the midportion of the muscle fibers because of increase in the diameter of the latter, which suggests an impairment of oxygen delivery to the hypertrophied muscle. Badeer (2) has cautioned against making conclusions regarding adequacy of coronary flow in living tissue from postmortem studies on segments of the coronary circulation.

Most measurements of coronary flow in the intact hypertrophied heart to date have been carried out with the use of nitrous oxide saturation and desaturation techniques (2). A meaningful comparison of coronary flows to normal and hypertrophied myocardium can be made only on the basis of unit weight of tissue. West et al. (30) found that, on this basis, the coronary flow to the left ventricle in stable hypertrophy was normal. Bing et al. (6) and Rowe et al. (32) came to similar conclusions in patients with cardiac enlargement from various forms of cardiovascular disease. However, the nitrous oxide method lacks precision; there is a ± 12.4% variation when compared with a more direct method of measuring flow with the use of a rotameter (14).

In the present study, flows were measured electromagnetically in the left circumflex and left anterior descending arteries, and it was assumed that they represented total flow to the left ventricle. Septal flow which may account for 11–14% of normal left coronary flow (9) was not measured in either group. Mean flow per 100 g LV wt to the hypertrophied left ventricle was significantly lower than the mean flow to the normal left ventricle. A decrease in mean right coronary artery flow also has been observed in adult dogs with congenital pulmonic or subpulmonic stenosis (24). These findings support the hypothesis (10) that the hypertrophied heart may fail by depriving the hypertrophied myocardial fibers of oxygen and other nutrients subsequent to the decrease in mean coronary blood flow. The decrease in flow may also account for the necrosis and degeneration of myocardial fibers which are often observed in the advanced stage of hypertrophy (10).

Coronary flow is determined by three mechanical factors: 1) aortic pressure or coronary perfusion pressure, 2) alterations in myocardial tissue oxygen content secondary to changes in contractility, and 3) myocardial systolic compression (3). In the present study, coronary perfusion pressure was higher than normal as a result of the elevated aortic pressure proximal to the band. In addition, there was evidence of myocardial hypoxia in hypertrophy, as suggested by a decrease in the oxygen content and an increase in the lactate-to-pyruvate ratio in the left ventricular venous blood. Hypoxia of the hypertrophied myocardium may result from a combination of impaired oxygen supply due to decreased coronary flow and increased oxygen requirement due to increased contractility in hypertrophy. Flow to the normal left ventricle is likely to increase under these circumstances. The observed decrease in mean flow to the hypertrophied left ventricle may therefore be due to increased systolic myocardial compression of the coronary vasculature. This is supported by the observations that late systolic flow in the left anterior descending and circumflex coronary arteries was retrograde in the hypertrophied left ventricle, while the diastolic flow pattern was comparable in both groups. The present observations are supported by the findings of Lovensohn et al. (24) who reported similar restriction of right coronary blood flow during systolic in dogs with right ventricular hypertrophy.

Heart rate, tension developed in the myocardial wall, and contractile state of the heart are the important direct determinants of myocardial oxygen consumption (7). Myocardial oxygen consumption is also influenced by the work performed during external shortening against a load (7). The role of external work as a determinant of myocardial oxygen consumption over and above that associated with tension development, heart rate, and contractility is analogous to the effect of external work on the oxygen consumption of skeletal muscle described by Fenn (11). In the present study, oxygen consumption of hypertrophied and normal left ventricles per 100 g LV wt was not significantly different. This is to be expected since the contractile state, heart rate, and external work normalized per 100 g LV wt were not significantly different in the two groups. The finding that the myocardial oxygen consumption in hypertrophied left ventricles was in the normal range suggests that the tension development in the hypertrophied ventricle was also normal. This conclusion is not consistent with observations of Laks and co-workers (22) who demonstrated a decrease in right ventricular wall tension in isolated hearts obtained from dogs with stable right ventricular hypertrophy. The difference may have been due to postmortem changes in the isolated heart preparation used by these workers. On the other hand, myocardial oxygen consumption in cardiac hypertrophy with failure was reported to be increased, presumably due to an increase in the developed tension in the dilated, failing heart (21).

Our data also demonstrate that, despite the decreased coronary flow in stable hypertrophy, myocardial oxygen consumption is maintained by increased oxygen extraction from the blood, resulting in lower venous oxygen content. The increase in oxygen extraction by the hypertrophied cardiac muscle is comparable to that observed in the isolated hypertrophied cardiac muscle from conditioned rats (33). Our results indicate that in open-chest, anesthetized dogs, oxygen extraction by the hypertrophied left ventricle is greater than normal. However, oxygen extraction by the normal left ventricle in this study was not as high as reported by Gregg et al. (15) in conscious dogs. The lower values in the present study may be due to the open-chest, anesthetized preparation, or to selective sampling of...
All these factors aid in maintaining left ventricular function.

References