Evaluation of Left Ventricular Performance

Circumferential Fiber Shortening and Tension

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SUMMARY

In the first part of the study, the influence of changes in preload and afterload on mean circumferential fiber shortening velocity, Vcf, was investigated in the absence of any extrinsic cause for change in the contractile state of the left ventricle (LV) of seven dogs. In five of seven dogs in which afterload was controlled and contractility was unchanged, there was a significant negative correlation between LV end-diastolic pressure and mean Vcf expressed in circumferences per second \( r = -0.88 \) to \(-0.97\). In one dog there was a positive correlation \( r = +0.847 \) and in another dog there was no significant correlation. Correlation as a group was poor.

In five of the above seven dogs in which afterload was decreased from a control level while preload was controlled and contractility was not extrinsically altered, mean Vcf increased in all instances. Because of the influence of afterload on Vcf increased in all instances. Because of the influence of afterload on Vcf and the inconsistent relation between Vcf and preload, we controlled preload and afterload during assessment of Vcf in reflecting a change of LV contractility.

Since mean left ventricular hydraulic output power, MLVP, quantitates practically all of the mechanical power of the left ventricular musculature, MLVP was utilized to represent the contractile state of the left ventricle under conditions in which preload and afterload were controlled. Mean and peak Vcf, Vcf at peak tension, extent of circumferential fiber shortening, and peak tension were compared with MLVP to test their usefulness in reflecting a change in the contractile state of the LV.

Twenty dogs were divided into four groups of five each. Group I was exposed to cardiopulmonary bypass alone. Dogs of groups II, III, and IV were exposed to a total of 45 minutes of myocardial ischemia during bypass. After bypass, groups I, II, III and IV retained an average of 89.3, 79.9, 82.5 and 69.2% of the control or original mean Vcf, and 87.1, 63.9, 53.4 and 33.5% of the control MLVP, respectively. Although an effect of ischemia was detected by changes of peak tension and extent of fiber shortening, the statistical evidence was less satisfactory than observed for MLVP. It was concluded that mean Vcf, peak Vcf, and Vcf at peak tension are inadequate indices to detect acute changes in the contractile state of the LV produced by prior myocardial ischemia. These same changes in contractility are readily determined by MLVP.

Additional Indexing Words:
Preload
Hydraulic output power
Afterload
Myocardial contractility

MEAN CIRCUMFERENTIAL FIBER SHORTENING VELOCITY (Vcf) has been utilized to assess left ventricular function in man.1, 2, 3, 4, 5 This approach has practical advantages because the measure can easily be calculated from left ventricular cineangiograms, or indicator dilution techniques.1, 6 Both Karliner2 and Cooper6 and their associates have observed significant reduction of mean circumferential fiber shortening velocities of patients with left ventricular myocardial disease compared to patients in whom mechanical performance was considered to be normal. Mean velocity of fiber shortening was calculated as the difference between end-diastolic and end-systolic circumference divided by the systolic ejection period. More recently, Peterson et al.4 obtained in man peak velocity of shortening by means of an electromagnetic probe bonded to the end of a cardiac catheter. They reported peak velocity in circumferences per second provided the best separation of groups with normal and abnormal left ventricular function. Recently, Eckberg et al.4 have reported that mean Vcf of patients with chronic severe mitral regurgitation was decreased relative to normal values.

The relationships between myocardial force,
velocity, and fiber length to determine the contractile state of the heart have evolved from investigations of cardiac muscle strips, and these principles have been applied in the analysis of function of the intact heart. Important determinants of cardiac performance are considered to be preload, afterload, contractility, and heart rate.

Prior to investigating circumferential fiber shortening velocity as an index of cardiac contractile state, we performed an initial study to evaluate the influence of a change of preload and afterload on mean Vcf while no agents were given extrinsically that would alter the contractile state of the heart. We recognized that alteration of arterial pressure during the performance of such studies may effect contractility through reflex stimulation.

Subsequently, the contractile state of the left ventricle (LV) was altered while preload and afterload were controlled to test the ability of velocity of circumferential fiber shortening and myocardial wall tension to reflect a change in contractility. Several levels of LV contractility were produced by exposing the heart for varying intervals to myocardial ischemia similar to that used clinically and experimentally.

In order to have an alternate index of change in contractility, we reviewed the mechanical energy expenditure of the left ventricle. From mathematical analysis the mechanical energy required to increase left ventricular pressure from its end-diastolic level to that just prior to aortic valve opening is negligible although significant oxygen consumption occurs. During the ejection phase of systole, the mechanical power developed by the left ventricle may be divided into four components: 1) that power associated with pumping of blood from the left ventricle, 2) the rate of changes of kinetic energy of the left ventricular wall and of the blood contained therein, 3) the rate of energy lost because of friction between muscle fibers and 4) the power utilized to change the potential energy of blood ejected from the heart. Of these items only the first is believed to be associated with a significant quantity of mechanical power. On this basis we suggest the measurement of mean left ventricular hydraulic output power (MLVP) represents practically all of the mechanical power produced by the left ventricle. If preload, afterload, and heart rate are controlled, hydraulic output power would seem to provide an index of the change in the contractile state of the left ventricle.

The purpose of this study was to evaluate the ability of mean velocity of circumferential fiber shortening, peak velocity of circumferential fiber shortening, fiber shortening velocity at peak tension, extent of fiber shortening, and peak tension to reflect a change in the contractile state of the left ventricle produced acutely by myocardial ischemia.

**Methods**

Twenty-seven mongrel dogs weighing 13 to 24 kg were studied. Sodium pentobarbital (25 mg/kg) was used to anesthetize each animal. A satisfactory plane of anesthesia was maintained with morphine sulfate. The animal’s temperature was maintained at 37°C by external sources. After passage of an endotracheal tube, ventilation was accomplished with a positive pressure ventilator. The pH of arterial blood was maintained within physiological limits during the study. The chest was opened by a median sternotomy incision. The pericardium was opened to expose the heart. Heparin (200 units/kg) was infused intravenously prior to cannulations of vessels. Aortic pressure was obtained via the right internal mammary artery. Left ventricular pressure was recorded from a micromanometer transducer on the tip of a catheter referenced against an external transducer.

Sodium pentobarbital was used to anesthetize each animal. Aortic blood flow was quantitated from an electromagnetic flow probe placed around the root of the aorta. A catheter was inserted in the left atrium for purposes of injection of contrast media. A large bore catheter was placed in the superior vena cava for transfusion of warm heparinized blood to control left ventricular end-diastolic pressure. Systemic vascular resistance was controlled by regulating a screw clamp on tubing which connected canulae in both femoral arteries and the right atrium.

In order to avoid the transient negative inotropic effects of radiopaque contrast material on the left ventricular function, several simultaneous measurements of left ventricular volume and end-diastolic pressure were obtained one hour prior to the recording of data for hydraulic output power, circumferential fiber shortening, and tension. Left ventricular end-diastolic pressure was increased by transfusion of blood to three or four specific levels from 5 to 15 mm Hg. A separate injection of 10-12 ml of radiopaque contrast media (75% Hypaque M or Renografin) was made into the left heart at each of these levels of left ventricular end-diastolic pressure while cineangiocardioiaphy at 60 frames/second was performed of the right anterior oblique view of the dog’s chest. A cine record of metallic beads embedded in a sheet of plastic placed at the same level as the dog’s heart permitted determination of left ventricular end-diastolic volume from the dimensions measured from the cine recording. Both end-diastolic and end-systolic volumes were calculated to confirm the accuracy of measurement with stroke volume obtained from the electromagnetic flow probe. Left ventricular end-diastolic pressure and volume were plotted opposite one another to provide a continuum of this relationship.

At the completion of the entire study the heart was rapidly excised and the mitral valve sutured closed. In——

1SF-1 pressure transducer. Statham Instruments, Inc. 254 Carpenter Road, Hato Rey, Puerto Rico.
crements of saline were infused passively via polyvinyl chloride tubing inserted in the aorta while ventricular pressures were recorded serially. Volume was determined by the amount of saline infused into the empty left ventricle. Duplicate measurements were made for confirmation of accuracy. Left ventricular wall thickness was measured directly by incising the left ventricular wall. Good correlation was obtained between the angiographic measurements of the LV volume and volume of the excised heart after completion of the function studies.

Left ventricular end-diastolic pressure was equated to preload. Mean aortic pressure was divided electronically by mean aortic blood flow to determine systemic vascular resistance. Vascular resistance was considered to be afterload. A total of 27 dogs was used in this study. In an initial study of seven animals left ventricular end-diastolic pressure was increased in a stepwise manner. Mean Vcf was determined, while afterload (systemic vascular resistance) was regulated to approximately the same value for each end-diastolic pressure. During these measurements, no extrinsic intervention was employed that would change contractility. In another study of five of the above seven dogs, systemic vascular resistance was varied, while LV end-diastolic pressure was controlled and while no agents were given extrinsically that would alter the contractile state of the heart.

Subsequently, a separate investigation of 20 dogs, four groups of five dogs each, was performed. End-diastolic pressure of these dogs was controlled to an arbitrarily selected level in the range of 9 to 12 mm Hg before and after cardiopulmonary bypass with or without myocardial ischemia. Specific data were recorded to determine nine mean left ventricular hydraulic power, systemic vascular resistance, instantaneous aortic blood flow, the integral of blood flow, mean velocity of circumferential fiber shortening, peak Vcf, peak tension at the endocardial equator of the ventricle (assumed to have an ellipsoid configuration), Vcf at peak tension, and extent of circumferential fiber shortening. The left ventricular volume used in the determination of circumferential fiber shortening, shortening velocities, and latitudinal tension was determined from the pressure-volume curve plotted for each dog. In the group of 20 dogs the end-diastolic pressure was controlled by blood transfusion to an exact filling pressure used during the angiographic study. After control data were obtained, each dog was placed on total cardiopulmonary bypass. Using an in-line heat exchanger body temperature was lowered to 32° C.

Group I, five dogs, was subjected to cardiopulmonary bypass of the same total duration as dogs of groups II, III, and IV, but with no interval of myocardial ischemia. All dogs of groups II, III, and IV were exposed to a total of 45 minutes of interruption of coronary blood flow. Myocardial ischemia was produced by clamping the root of the ascending aorta. In dogs of group II, the aortic clamp was released for 5 min after each 15 min of myocardial ischemia. Group III dogs were permitted only 45 sec of coronary flow each 15 minutes of cardiac anoxia. Dogs of group IV were subjected to 45 min of uninterrupted ischemia. During ischemia, the LV was decompressed by an apical catheter. After release of the aortic clamp, body temperature was restored to 37° and bypass discontinued.

After 30 min, measurements were repeated as described above. For purposes of comparison of data, LV end-diastolic pressure and systemic vascular resistance were regulated to the same levels as recorded in each individual dog during the control study.

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**Data Collection**

Signals were recorded on a 12 channel* recorder and transmitted to an analog† and digital‡ computer. Mean left ventricular hydraulic output power, MLVP, was calculated in units of gram-meters/minute by the equation:

$$MLVP = \frac{1}{N} \int_0^N (P_v - P_{ed}) Q dt$$

where $P_v$ = instantaneous left ventricular pressure; $P_{ed}$ = left ventricular end-diastolic pressure; $Q$ = instantaneous aortic blood flow; $N$ = interval in time from R wave to R wave of the electrocardiogram.

Systemic vascular resistance was calculated by dividing mean aortic pressure by mean aortic blood flow.

A prolate ellipsoid configuration was assumed to approximate the left ventricular cavity with negligible change in length of the major axis. End-diastolic volume (EDV) was calculated as indicated below:

$$EDV = \frac{4A^2}{3\pi a}$$

where $A$ = measured cineangiographic area of left ventricular cavity; $a$ = semi-major axis.

Analog signals of left ventricular pressure and aortic flow were digitized. From values of end-diastolic volume, wall thickness, and semi-major axis, tension was calculated in units of kilodynes per cm² by the equation below:

$$T = \frac{P_b (1-b^2/2a^2)}{h}$$

where $T$ = latitudinal tension; $P_b$ = instantaneous left ventricular pressure; $h$ = wall thickness; $a$ = semi-major axis; $b$ = semi-minor axis.

The greatest value of tension calculated was considered peak tension. The associated velocity of circumferential fiber shortening was noted at the same point in time.

The semi-minor axis, $b$, as a function of time, was calculated in centimeters by the equation:

$$b = \sqrt{\frac{3(EDV-v)}{4\pi a}}$$

where $v$ = sequential accumulative measured stroke volume obtained from aortic flow signal.

Fiber shortening velocity was determined from the rate of change of $2\pi b$.

Mean velocity of circumferential fiber shortening, mean Vcf, was obtained from the difference between end-diastolic and end-systolic circumferential velocity divided by the systolic ejection period measured from the aortic flow tracing. Mean Vcf was quantitated in centimeters per second and was also divided by end-diastolic circumstance to normalize the data to permit comparison of dogs in units of circumferences per second.

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*Electronics for Medicine DB-12. Electronics for Medicine, Inc., White Plains, N.Y.
†Applied Dynamics AD-4. Box 1488, Ann Arbor, Michigan 48106.
‡Digital IBM 1130. White Plains, N.Y.
The extent of shortening of the semi-minor axis was determined from the difference in end-diastolic and end-systolic circumference of the midwall of the left ventricle and expressed as a percentage of end-diastolic fiber length.

Results

When preload of seven dogs was serially increased and systemic resistance controlled to the same level, mean Vcf, expressed in circumferences per second, varied as seen in figure 1. These changes occurred in the absence of any alteration of the contractile state of the heart. Using the Pearson r, 18 a negative correlation between LV end-diastolic pressure and mean Vcf was observed in five of seven dogs (r = -0.87 to -0.94). In one dog there was a positive correlation (r = +0.867) and in another dog there was no significant correlation. However, there is no correlation of mean Vcf to LV end-diastolic pressure when data from all seven dogs are combined (r = 0.094). When mean Vcf was expressed in cm/sec there was no significant correlation of mean Vcf and LV end-diastolic pressure in five of seven dogs, positive correlation in one (r = +0.92) and negative correlation in another (r = -0.78). In these seven dogs in which LV end-diastolic pressure was serially increased from 15 mm Hg, heart rate declined to an average of 92% of that observed at the low end-diastolic pressure or from an average of 152 to 138 beats per minute. There was no significant correlation between LV end-diastolic pressure and heart rate of these seven dogs.

Under conditions in which LV end-diastolic pressure was controlled and there was no extrinsic intervention that would alter the LV contractile state of five dogs, decreasing systemic vascular resistance always resulted in an increased mean Vcf (table 1). The data of 11 sets of paired values of mean Vcf and systemic resistance of the five dogs of table 1 were analyzed for correlation. Each pair was at the same LV end-diastolic pressure. The Pearson r value was -0.842.

During one experiment LV end-diastolic pressure was increased from 7 to 17 mm Hg, while systemic vascular resistance was controlled successively to 2600, 2900, and 3200 dyne sec cm^-5. The results are summarized in figure 2, which illustrates that mean Vcf at all levels of LV end-diastolic pressure was consistently raised with each decrease in systemic resistance. In this dog the correlation of LV end-diastolic pressure and mean Vcf in circumference/sec at the same systemic vascular resistance levels of 2600, 2900, and 3200 dyne sec cm^-5 was high (r = -0.91) at 2600 but not significant at higher levels.

In another experiment LV end-diastolic pressure was increased from 4.6 to 13.3 mm Hg while systemic resistance was allowed to decrease spontaneously from

![Figure 1](image)

**Figure 1**

Mean circumferential fiber shortening velocity (Vcf) plotted against left ventricular (LV) end-diastolic pressure in seven dogs. No intervention was employed to change the contractile state. Systemic resistance was controlled for each dog to the same value for all LV end-diastolic pressures. Sec = second.

<table>
<thead>
<tr>
<th>Example #</th>
<th>LVEDP mm Hg</th>
<th>Flow ml/min</th>
<th>MLVP g Ml/min</th>
<th>Rs Dyne sec cm^-5</th>
<th>Mean Vcf cm/sec</th>
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</table>

Rs versus mean Vcf (r = 0.842).

Abbreviations: LVEDP = left ventricular end-diastolic pressure; Flow = aortic blood flow; Rs = systemic vascular resistance; MLVP = mean left ventricular hydraulic power; Mean Vcf = mean circumferential fiber shortening velocity.

CIRCUMFERENTIAL FIBER SHORTENING & TENSION

Mean Vcf plotted versus LV end-diastolic pressure of one dog. Systemic vascular resistance was maintained at successively increased levels of 2600, 2900 and 3200 dyne sec cm⁻² while LV end-diastolic pressure was serially increased.

6000 to 2700 dyne sec cm⁻². Under these conditions mean Vcf increased from 6.4 to 14.4 cm/sec (table 2).

Mean Left Ventricular Output Power (MLVP) and Mean Circumferential Fiber Shortening Velocity (Vcf)

Table 3 and figure 3 summarize the data obtained from dogs of groups I, II, III, and IV. Under conditions in which LV end-diastolic pressure and systemic vascular resistance were controlled on the same level before and after bypass, dogs of group I retained 87.1% of original mean left ventricular hydraulic output power and 89.3% of original mean Vcf following only cardiopulmonary bypass. Dogs of group II retained 63.0% of original MLVP and 79.0% of original mean Vcf. Group III dogs had an average of 53.0% of the control MLVP and 82.5% of control mean Vcf. Group IV dogs retained only 33.5% of the control MLVP but 69.2% of mean Vcf.

Mean Vcf of groups I, II, III, and IV before bypass averaged 16.3, 17.3, 21.5 and 20.8 cm/sec, and 1.19, 1.23, 1.46 and 1.50 circumferences per second respectively. After bypass groups I, II, III and IV mean Vcf averaged 15.2, 13.6, 17.5 and 14.4 cm/sec and 1.03, 0.96, 1.29 and 1.06 circumferences per second respectively. The average of the values of mean Vcf obtained during the control study in these 20 dogs was 1.34 circumferences per second.

By means of an analysis of variance a significant over-all effect of ischemia was noted in the retention of MLVP (F = 39.1, df = 3, 16; P < 0.01) but no significant effect (F = 1.73, df = 3, 16; NS) was noted on mean Vcf in cm/sec or circumferences/second.

Group I, exposed to no ischemia, differed statistically (Duncan’s New Multiple Range Test) from groups II, III, and IV (P < 0.01) in the retention of MLVP. Group II differed from group IV (P < 0.01) in the retention of MLVP, but not significantly from group III. Group III similarly differed from group IV (P < 0.01).

Peak Circumferential Fiber Shortening Velocity (Vcf)

Dogs of groups I, II, III, and IV retained 90.1, 82.0, 89.0 and 76.4% of the control peak Vcf values. Before bypass peak Vcf of groups I, II, III, and IV was 1.59, 1.77, 2.07 and 2.06 circumferences per second respectively. After bypass the peak Vcf of these same groups was 1.42, 1.44, 1.87, and 1.57 circumferences per second.

Table 2

Data Obtained in One Dog During Performance of a Ventricular Function Curve with Usual Variation of Preload and Afterload

<table>
<thead>
<tr>
<th>LVEDP</th>
<th>MLVP</th>
<th>Flow</th>
<th>R₀</th>
<th>Mean Vcf</th>
</tr>
</thead>
<tbody>
<tr>
<td>mm Hg</td>
<td>g M/min</td>
<td>ml/min</td>
<td>dyne sec cm⁻²</td>
<td>cm/sec</td>
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<td>1450</td>
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See table 1 for abbreviations.

Circulation, Volume XLIX, May 1974
Table 3

Summary of Percentage Changes from Control of Measures of Left Ventricular Function Following Bypass With or Without Myocardial Ischemia

<table>
<thead>
<tr>
<th>Dog group*</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>IV</th>
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<tr>
<td>MLVP</td>
<td>87.1±10.5</td>
<td>63.9±7.1**</td>
<td>53.4±7.7**</td>
<td>33.5±5.9**</td>
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<td>Mean Vcf</td>
<td>89.3±14.6</td>
<td>79.9±10.7†</td>
<td>82.5±6.4†</td>
<td>69.2±10.1†</td>
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<tr>
<td>Peak Vcf</td>
<td>90.1±16.1</td>
<td>82.0±12.8†</td>
<td>89.8±4.3†</td>
<td>76.4±7.0†</td>
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<tr>
<td>Vcf at To</td>
<td>101.5±8.2</td>
<td>80.7±16.6†</td>
<td>89.5±4.5†</td>
<td>82.5±13.9†</td>
</tr>
<tr>
<td>To</td>
<td>90.7±11.5</td>
<td>87.7±8.7†</td>
<td>76.3±7.8‡</td>
<td>70.1±8.1***</td>
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<tr>
<td>Ext of S</td>
<td>82.1±16.1</td>
<td>65.7±12.3†</td>
<td>69.8±7.9†</td>
<td>47.2±7.2***</td>
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<td>Cardiac output</td>
<td>93.1±9.8</td>
<td>79.0±10.3‡</td>
<td>69.5±6.8**</td>
<td>51.4±3.3**</td>
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<tr>
<td>Heart rate</td>
<td>109.3±12.3</td>
<td>110.9±14.9†</td>
<td>106.3±7.8†</td>
<td>113.0±10.1†</td>
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<td>113.3±15.2‡</td>
<td>116.8±26.1†</td>
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</tbody>
</table>

Values given as mean and standard deviation.

*Group I exposed to bypass with no ischemia. Groups II, III, and IV subject to 45 minutes of myocardial ischemia. N = 5 for all groups.

†No significant change from Group I.

‡P < 0.05. Group significantly differs from Group I.

**P < 0.01. Group significantly differs from Group I.

Abbreviations: N = number of dogs; MLVP = mean left ventricular hydraulic output power; Vcf = circumferential fiber shortening velocity; To = peak tension; Ext of S = extent of circumferential fiber shortening; EDV = left ventricular end-diastolic volume; LVEDP = left ventricular end-diastolic pressure; R0 = systemic vascular resistance.

respectively. There was no significant (F = 1.75, df = 3.16; NS) effect of ischemia on peak Vcf in cm/sec or circumferences/second.

Circumferential Fiber Shortening Velocity (Vcf) at Peak Tension

Percentages of Vcf at peak tension retained by dogs of groups I, II, III, and IV were 101.5, 80.7, 89.0 and 82.5. Before bypass Vcf at peak tension of groups I, II, III, and IV was 1.41, 1.61, 1.91 and 1.86 circumferences/sec respectively. After bypass of these groups Vcf at peak tension was 1.32, 1.26, 1.71 and 1.52 circumferences/sec respectively. There was no significant (F = 3.20, df = 3.16; NS) effect of ischemia on Vcf at peak tension on cm/sec or circumferences/sec.

Peak Tension

The percentage of the control values of peak tension of groups I, II, III, and IV was 90.7, 87.7, 76.3 and 70.1. Although a significant effect of ischemia was noted (F = 5.38, df = 3.16; P < 0.05), the only significant intergroup differences found* were between groups I and III (P < 0.05) and groups I and IV (P < 0.05).

Extent of Circumferential Fiber Shortening

The percentage of the control values of extent of circumferential fiber shortening noted in groups I, II, III, and IV were 82.1, 65.7, 69.8 and 47.2 respectively. A significant effect on the extent of circumferential fiber shortening was observed (F = 7.94, df = 3.16; P < 0.01). In extent of circumferential shortening, group I differed significantly only from group IV (P < 0.01). Group II differed from group IV (P < 0.05) and group III differed from group IV (P < 0.01).

Cardiac Output

The percentage of the control cardiac output retained by the dogs of groups I, II, III, and IV were 93.1, 79.0, 69.5 and 51.4 respectively. Group IV differed from groups I, II, and III (P < 0.01). Group I differed from group II (P < 0.05) and from group III (P < 0.01). There was no significant difference between groups II and III.

There was no significant change in heart rate, systemic vascular resistance, or left ventricular end-diastolic pressure or volume observed after cardiopulmonary bypass of dogs in groups I, II, III, and IV (table 3).

Discussion

Tension-velocity relations have been used to analyze left ventricular contractile state.* Because of the complexity of determining maximum contractile element velocity (V max) attention has been turned to indices of contractility that are more readily measured.
and that require no assumptions with regard to a muscle model. Mean velocity of circumferential fiber shortening (Vcf) of the left ventricle is one such index. There has been little emphasis previously on the influence of preload and afterload on mean Vcf. Gault et al. suggested that mean Vcf may be influenced by resting fiber length. Urschel et al. have observed an increase in peak Vcf in response to a decrease in afterload produced by mitral insufficiency in the dog.

From figure 1 it is apparent that the inherent levels of mean Vcf vary from dog to dog. In response to increasing preload, values of mean Vcf are observed to vary unpredictably either in a positive or negative direction. There was poor correlation between mean Vcf and LV end-diastolic pressure for the seven dogs as a group, although there was a significant negative correlation of the relationship in five of this group when analyzed on an individual basis. From figure 2 and table 1 mean Vcf is observed to be dependent on the level of systemic vascular resistance. When preload and afterload are both permitted to vary (table 2), it is seen that there is no specific value of Vcf that characterizes the contractile state of a given heart.

With this background we concluded it was necessary to control preload and afterload in order to assess the ability of velocity of fiber shortening to reflect a change in the contractile state of the left ventricle (LV). Based on the premise that mean left ventricular hydraulic output power (MLVP) summates practically all of the mechanical power produced by the left ventricle, MLVP is suggested as a means quantitating the magnitude of change in the contractile state of the myocardium when preload and afterload are controlled. In order to determine the discriminability of mean Vcf to quantify changes of the contractile state, the LV was subjected to cardiopulmonary bypass or to bypass plus varying intervals of myocardial ischemia. The method of changing the contractile state of the LV was selected since it simulates the circumstances that occur in the operating room during surgical treatment of many forms of congenital and acquired cardiac disease. The four groups of dogs retained four different levels of contractility following bypass, distinguished from one another by the percentage retention of original MLVP. It is evident from this study (table 3) that mean Vcf failed to reflect the severity of depression of left ventricular function, especially following 45 minutes of uninterrupted myocardial ischemia. From statistical analysis, extent of fiber shortening and peak tension appeared better than mean Vcf in reflecting the effects of anoxia on the LV.

This investigation suggests that the use of velocity of circumferential fiber shortening in the evaluation of patients' cardiac function following exposure to myocardial ischemia during cardiopulmonary bypass may be of questionable value. It is uncertain if the findings of this study apply to chronic heart disease, which may result in a change in the LV end-diastolic pressure-volume relationship. The use of MLVP in the evaluation of contractility in man is limited by the impracticality of controlling preload and afterload and by the problems related to valvar deformities. The application of MLVP would necessitate the establishment of a range of normal values for specific levels of preload and afterload.

In the study of Peterson et al. patients with clinically impaired heart function had an increased left ventricular end-diastolic pressure and volume relative to their patients with normal function. The greater peak tension of patients with impaired heart function observed by Peterson et al. may be accounted for in part by the greater LV end-diastolic volume. In contrast, we studied the dog's heart acutely impaired by ischemia, and we controlled LV end-diastolic pressure to the same level as present in the control condition. We observed no significant change in end-diastolic volume, and a decrease in peak tension after ischemia. Because of the above, our study is not thought comparable to the studies of Karliner, Peterson, Eckberg and their associates.

Exposure of the dog's heart to myocardial ischemia appeared to be manifested in part by a decrease in LV wall tension. The exclusive use of either fiber shortening velocity or wall tension provided only an incomplete assessment of the contractile state of the LV musculature. Mean Vcf is derived from the change in circumference of the minor axis of the heart divided by the systolic ejection period. If both the change in circumference and the ejection interval decrease nearly proportionately as a result of ischemia, mean Vcf will not reflect impaired contractility. Although the amplitude of Vcf was not greatly altered by ischemia, its time interval was decreased so that the extent of shortening more nearly reflected the severity of impairment of LV function than mean Vcf.

Because of the dependence of Vcf on afterload and because of the observed variability of mean Vcf in response to changes in preload, it is recommended that isolated values for Vcf be interpreted with caution. It is concluded that discrete changes of the contractile state of the left ventricle produced acutely by ischemia did not result in significant changes of mean Vcf. Specific levels of ventricular impairment which are readily detected by mean left ventricular hydraulic output power under conditions of controlled preload and afterload were not satisfactorily reflected.
in the values for mean Vcf, peak Vcf, or Vcf at peak tension.

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