Epicardial Coronary Artery Compliance
in the Dog

By John E. Douglas, M.D., and Joseph C. Greenfield, Jr., M.D.

ABSTRACT

The dynamic compliance of 21 coronary arteries from 17 dogs was determined in situ. Intramyocardial portions were occluded with a mixture of 200 μl glass beads and liquid silicone. To simulate in vivo pressure-volume conditions, 0.1 ml of saline was injected in 150 to 240 msec at a rate of 30 pulses/min. Silicone casts of the vessels, made at 100 mm Hg distending pressure, were used as a reference volume. The results indicate that the dynamic compliance decreases as the initial distending pressure rises. The change in volume for a 50 mm Hg pressure increment (initial distending pressure of 100 mm Hg) was 3.7 ± 0.6% (mean ± SE). In four dogs, phasic flow in the left circumflex coronary artery was measured. The volume of systolic flow per beat during the control state was of the same order of magnitude as the compliance of the vessels; however, during reactive hyperemia, systolic coronary flow markedly exceeded the dynamic compliance of the coronary vessels. In six dogs the static compliance of a segment of coronary artery was obtained radiographically. A 30.1 ± 1.4% change in volume occurred when the intravascular pressure was increased from 70 to 120 mm Hg.

ADDITIONAL KEY WORDS
coronary artery coronary artery elasticity pressure-radius relation

The pressure-volume relationships of the epicardial coronary arterial tree have not been previously described. These data are necessary to formulate any realistic dynamic model of the coronary circulation. For example, to determine the degree to which the myocardium is perfused during systole, the compliance of the epicardial coronary vessels must be known (1). Accordingly, this study was designed to determine the dynamic pressure-volume relationships of the in situ epicardial coronary arteries in the dog and to relate these measurements to volumes of these vessels obtained from casts. These data were compared to values of phasic coronary artery blood flow obtained in vivo. In addition, static pressure-volume measurements of an epicardial segment of the left coronary artery were obtained radiographically.

Methods

Seventeen mongrel dogs weighing 12 to 30 kg were killed rapidly with pentobarbital. The left circumflex coronary artery and aortic blood flows in four of these dogs had previously been measured as described below. The hearts were excised and placed in a pan of saline at room temperature. The aortic and pulmonary arteries were cut away within 1 cm of the semilunar valves and the hearts weighed. The left coronary artery was dissected from its origin for 3 mm. A 5-cm segment of high pressure, noncompliant catheter having a flame-flanged distal tip was inserted into the coronary ostia and secured with a ligature. Care was taken not to disturb the remainder of the coronary vascular bed. This technique was used to cannulate the main left coronary artery (LCA) in 10 hearts, the left circumflex coronary artery (LCCA) in five hearts, and the left anterior descending artery (LAD) in five hearts. The right coronary artery (RCA) was similarly cannulated in one of the hearts and

From the Department of Medicine (Division of Cardiology), Veterans Administration Hospital, Durham, N. C. 27705, and Duke University Medical Center, Durham, North Carolina 27708.

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Schematic diagram of the apparatus used for dynamic pressure-volume studies. Saline in syringe A was infused at a constant rate to provide various initial distending pressures in the cannulated vessel. Saline, 0.1 ml, was injected every 2 seconds with syringe B over a 150- to 240-msec period. The pressure in the system was measured by the transducer (PT).

The coronary vessels were then flushed with a small amount of saline. Subsequently, 0.2 ml of a mixture of 200 μ glass beads and white liquid silicone (without catalyst) was injected alternating with 1 to 2 ml of saline until the terminal portions of all the epicardial branches of the artery appeared to be filled with the emboli. Thus, most of the intramyocardial coronary arterial bed was eliminated from the preparation. Dynamic pressure-volume studies were performed using the apparatus schematically diagrammed in Figure 1. Each heart was allowed to float partially submerged in saline. A Statham P23 D6 pressure transducer was used to monitor pressure in the system. Syringe B was a 1 ml Luer-lock syringe with fixed delivery of 0.10 ml. Initially, infusion pump rates from 0.3 to 2.0 ml/min were selected to provide a baseline distending pressure of 40 mm Hg. After 15 seconds of equilibration at this pressure, 0.10 ml of saline was injected into the circuit via syringe B (Fig. 1). The duration of injection was varied from 150 to 240 msec to approximate the duration of cardiac ejection found in dogs during the awake state. The resulting pressure was recorded at a paper speed of 50 mm/sec on a two-channel, direct-writing oscillograph (Fig. 2). Following injection, the syringe was allowed to recoil and fill from both coronary artery reflux and the infusion pump. Pulsing frequency was 30/min. After a minimum of 15 pulses, the distending pressure was elevated approximately 20 mm Hg by increasing the rate of the infusion pump. After a 15-second equilibration period, the pulsing was repeated. This procedure was carried out until data had been obtained at a distending pressure greater than 100 mm Hg. The distending pressure was then reduced in a similar stepwise fashion and the pulsing repeated. This protocol was followed as many as four times, either immediately or after a 30-minute interval. The entire study was completed during a 3-hour period after the dog was killed. In three preparations the study was carried out with the heart and apparatus warmed to 39°C.

Pressure curves were analyzed for initial distending pressure (P), pulse pressure (ΔP) and the duration of injection (ΔT) as illustrated in Figure 2. The pressure-volume properties or compliance (C) of a distensible structure such as the coronary arterial tree may be expressed by:

\[ Q_{\text{tot}} = \frac{C \Delta P}{\Delta T} + \frac{P_{\text{tot}}}{R}. \]  (1)

This equation relates total flow \(Q_{\text{tot}}\) to the sum of a compliance variable \(C \Delta P/\Delta T\) and a resistance variable \(P_{\text{tot}}/R\). It is analogous to the electrical expression for current flow through a circuit having a capacitor and resistor in parallel. When \(\Delta P/\Delta T\) equals zero, i.e., between pulses, equation 1 reduces to:

\[ Q_s = \frac{P}{R}. \]  (2)

where \(Q_s\) is the constant flow. In our preparation, \(Q_s\) is the flow provided by the infusion pump. Since both \(Q_s\) and \(P\) were measured during the diastolic intervals, the resistance (R)
Sequential dynamic pressure records obtained from a left circumflex coronary artery (LCCA) preparation at four distending pressures. Onset of pulse occurred at a, termination of pulse at b and spring recoil at c. The pressure at a is the initial distending pressure (P) and the difference between the pressures at a and b is the pulse pressure (ΔP). The duration of injection (AT) is the time from a to b.

could be calculated for the period before each pulse. It should be noted that R is the total resistance including the resistance due to the glass beads and silicone and is not analogous to the myocardial vascular resistance. Assuming a negligible change in the relative properties of the vessel during the 150 to 240 msec between the onset and end of the pulse, this value for R was employed in the computation of C from equation 1. The value for Q вам during the pulsed injection was the sum of the flow provided by the infusion pump (Qs), and the 0.10-ml dynamic flow which was injected during the pulse (Qd). Ptot in equation 1 was the sum of the initial distending pressure (P) and the pulse pressure (ΔP). The solution for equation 1 requires that Ptot is the instantaneous value of the pressure. In these computations the peak pressure achieved during the pulse was substituted. Thus, C was computed for each pulsed injection, using in equation 1 the observed values for P, ΔP, AT and the computed values for R, Q нам, and Ptot. For each series of pulses obtained at a given initial distending pressure, an average value of C was calculated and expressed as ml/mm Hg.

To estimate the relative changes in length and diameter that occur in a segment of a coronary artery, static radiographic studies were performed on six of the 17 hearts. Following the volume measurements outlined above, the hearts were placed so that the major part of the LCCA which lies in the atrioventricular groove was in a plane parallel with the film cassette. Sodium meglumine diatrizoate (Hypaque-M, 76%) was infused through the cannula until the distending pressure was 70 mm Hg. Roentgenograms were obtained after a 15-second equilibration period. The distending pressure was increased to 120 mm Hg and the process repeated. The films were enlarged to a magnification of 7X and used for measuring length changes (distances between bifurcation points) and diameter changes at 6 to 10 representative segments along the vessel.

Silicone casts having a specific gravity of 1.165 were made of all coronary artery preparations using General Electric room temperature vulcanizing silicone (RTV-11) with 8% black silicone paste and 1% catalyst. The black coloring was used to contrast with the white silicones injected previously. The casting material had been tested and found not to change in volume during the hardening process. Casting pressure was held at 100 mm Hg while the casts were allowed to vulcanize. After 24 hours, the tissues were removed with concentrated hydrochloric acid. This process yielded a clean cast having excellent detail (Fig. 3). After trimming away the embolized beads and white silicone portions of the cast, the remaining black cast was weighed and the volume calculated from the weight and specific gravity. Thus, the cast volume provided a measurement of the total volume of the epicardial vascular bed at a distending pressure of 100 mm Hg. These volume data are listed in Table 1, column 5.

In three of the dogs, the dynamic compliance
Silicone cast of LCCA made at 100-mm Hg infusion pressure (dog 4). The white portions of the cast are the white silicone and glass bead emboli used to occlude the intramyocardial portion of the vascular bed. The black portions of the cast represent the actual segments of the artery studied in both the dynamic pressure-volume and radiographic experiments.

of the in situ right femoral arteries was measured using the same techniques as employed in the studies of the dynamic compliance of the coronary arteries except that the pulsing volume was fixed at 0.25 ml. Similar casts of the femoral arteries were made, and employed in calculations comparable to those for the coronary arteries.

Several weeks before the above studies, 4 of the 17 dogs underwent left thoracotomy during pentobarbital anesthesia, 30 mg/kg, to implant Statham electromagnetic flowmeter transducers on both the ascending aorta and the LCCA according to the method described by Alexander et al. (2). Ten to 14 days later a no. 7 Lehman catheter was inserted into the femoral artery using lidocaine local anesthesia and passed to the ascending aorta. With the animal unanesthetized, aortic blood flow and pressure and LCCA blood flow were recorded for a 5-minute control period and during reactive hyperemia which followed a 10-second snare occlusion of the LCCA. The oscillographic recordings were analyzed for the following variables. Aortic and LCCA flow were obtained by planimetry of the area beneath the aortic and LCCA flow curves, respectively. Aortic pressure was read directly from the record. Duration of ejection was defined as the interval of forward aortic flow. Stroke systolic LCCA flow was the flow during the ejection period. Standard statistical techniques were used to compare the data. All computations were carried out on an International Business Machines Model 1130 digital computer.

**Results**

Examples of the pressure recordings obtained in the dynamic pressure-volume studies at four initial distending pressures are illustrated in Figure 3. The similarity between the contours of the initial portion of these pressures and the aortic blood pressures found during life can be appreciated. In carrying out the studies, it was found that pulsing a vessel in which the pressure had been essentially zero for a 10-minute period resulted in a markedly decreased compliance when compared to that obtained after the initial distending pressure had been raised above 40 mm Hg for at least 15 seconds. Presumably this finding was due to a reduction in pressure below the critical closing pressure. Data obtained from an artery immediately after raising or lowering its distending pressure demonstrated a greater or smaller compliance, respectively, than was found if the vessel equilibrated for a 15-second period at each new distending pressure. Because of this later observation, all vessels were studied after a 15-second period at a given initial distending pressure. In addition, the first two or three pulses for each distending pressure demonstrated slight hysteresis. For this reason, the first three pulse pressure responses were not employed in final data analysis.

Table 1 summarizes the experimental data obtained in 21 coronary artery preparations. The body weights, heart weights, and final volume of the casts of the vessels studied are provided in columns 2, 3 and 5, respectively. The volume of the epicardial arterial bed for each vessel was found to correlate with the weight of the heart. The mean and standard error of the cast volumes per 100 g heart weight for the 10 LCA, five LAD, and five LCCA were 1.30 ± 0.10, 0.63 ± 0.04, and 0.75 ± 0.08 ml, respectively. To normalize the data for compliance, the values obtained at 60, 80, 100, and 120 mm Hg distending pressure were divided by the respective cast volume for each vessel and tabulated in Table 1, columns 6 to 9. These normalized values for compliance usually decreased as the initial distend-
TABLE 1

<table>
<thead>
<tr>
<th>Dog</th>
<th>Weight (kg)</th>
<th>Heart weight (g)</th>
<th>Vessel*</th>
<th>Cast vol (ml)</th>
<th>60 mm Hgt</th>
<th>80 mm Hgt</th>
<th>100 mm Hgt</th>
<th>120 mm Hgt</th>
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<tr>
<td>1</td>
<td>25</td>
<td>180</td>
<td>LCCA</td>
<td>1.31</td>
<td>19.5</td>
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<td>18.0</td>
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<td>5.4</td>
<td>5.2</td>
<td></td>
</tr>
<tr>
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<td>262</td>
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<td>3.0</td>
</tr>
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<td>228</td>
<td>LCCA</td>
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<td>9.5</td>
<td>7.8</td>
<td>6.7</td>
<td>6.1</td>
</tr>
<tr>
<td>6</td>
<td>28</td>
<td>208</td>
<td>LAD</td>
<td>1.41</td>
<td>11.8</td>
<td>10.5</td>
<td>10.1</td>
<td>9.9</td>
</tr>
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<td>208</td>
<td>LAD</td>
<td>1.39</td>
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<td>5.9</td>
<td>5.4</td>
</tr>
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<td>114</td>
<td>LAD</td>
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<tr>
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<td>117</td>
<td>LAD</td>
<td>0.86</td>
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<tr>
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<td>14</td>
<td>98</td>
<td>LCA</td>
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<td>8.3</td>
<td>8.4</td>
<td>8.4</td>
<td>7.6</td>
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<td>2.9</td>
<td>2.5</td>
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<td>1.36</td>
<td>7.6</td>
<td>5.7</td>
<td>4.4</td>
<td></td>
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<tr>
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<td>14</td>
<td>127</td>
<td>LCA</td>
<td>1.41</td>
<td>12.1</td>
<td>9.7</td>
<td>7.9</td>
<td>6.9</td>
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<td>122</td>
<td>LCA</td>
<td>1.80</td>
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<td>16</td>
<td>16</td>
<td>140</td>
<td>LCA</td>
<td>1.45</td>
<td>5.2</td>
<td>4.2</td>
<td>3.2</td>
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<tr>
<td>17</td>
<td>17</td>
<td>220</td>
<td>LCA</td>
<td>2.48</td>
<td>2.9</td>
<td>2.3</td>
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<tr>
<td>18</td>
<td>23</td>
<td>270</td>
<td>LCA</td>
<td>3.88</td>
<td>1.6</td>
<td>1.0</td>
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<td></td>
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<tr>
<td>19</td>
<td>24</td>
<td>210</td>
<td>LCA</td>
<td>2.56</td>
<td>7.2</td>
<td>6.3</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>30</td>
<td>276</td>
<td>LCA</td>
<td>2.28</td>
<td>2.0</td>
<td>1.4</td>
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<td></td>
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<tr>
<td>21</td>
<td>28</td>
<td>262</td>
<td>RCA</td>
<td>1.03</td>
<td>5.4</td>
<td>5.2</td>
<td>4.8</td>
<td></td>
</tr>
</tbody>
</table>

*Left circumflex coronary artery (LCCA); left coronary artery (LCA); left anterior descending (LAD); right coronary artery (RCA).

The blood pressures listed for columns 6 to 9 are the initial distending pressures.

For the coronary vessels: 1.6, 3.8, and 2.4 ml/mm Hg for an initial distending pressure of 100 mm Hg. The average change in volume for a 50-mm Hg change in pressure was 1.3% and can be compared to 3.7% found in the coronary vessels (Fig. 4) for the same distending pressure.

In six hearts the dimensional changes of the proximal portions of the LCCA following an increase in intravascular pressure from 70 to 120 mm Hg were determined radiographically. The mean and standard error for the static increase in radius was 10.2 ± 1.0%. The length increased (as measured from branch points) by 7.7 ± 1.4%. Assuming the vessel to be cylindrical, the volume was calculated to increase by 30.1 ± 3.5%. In all six hearts, after a 15-second period at 120-mm Hg pressure, minimal perfusion of the intramyocardial vessels was noted. Since as little as 0.5 ml of contrast media will stain the myocardium, the silicone-200µ bead mixture apparently provid-
The mean and standard error (crossed bars) of the percent change in volume following a 50-mm Hg pressure pulse for the 11 coronary arteries in which data were obtained at all four initial distending pressures.

\[ \Delta P = \text{pulse pressure.} \]

ed an effective occluding medium. In the two dogs in whom flowmeter probes had been implanted, no detectable elongation occurred in the proximal 3-cm segment of the vessel. The distal segments of these arteries, however, reacted as the LCCAs of the other four dogs, suggesting that the fibrotic reaction to the surgery in this area restricted the mobility of these arteries.

The configuration of the LCCA and aortic flow curves (Fig. 5) obtained in the four dogs were similar to those measured previously from this (3, 4) and other laboratories (1). In Table 2 hemodynamic data calculated from these recordings are summarized. In three of these dogs (2, 3, and 5), the dynamic compliance of the LCCA had been measured (Table 1). From these data the volume of blood which could have been "stored" in the epicardial portion of the LCCA during systole was calculated to be 0.02, 0.07, and 0.09 ml, respectively. In making these computations, the same distending and pulse pressures as recorded during life were employed. Using the data from Figure 4 and assuming a distending pressure of 100 mm Hg and the mean volume of the LCCA obtained from the cast, a 30 mm Hg pressure pulse would be associated with an average increase in volume of 0.06 ml. It is apparent that these values are generally comparable to the systolic flow (Table 2, column 6) which occurred during the control period, but are much less than the systolic flow during reactive hyperemia.

**TABLE 2**

<table>
<thead>
<tr>
<th></th>
<th>Heart rate (beats/min)</th>
<th>Duration of ejection (sec)</th>
<th>Blood pressure syst/diast (mm Hg)</th>
<th>Aortic flow (ml/beat)</th>
<th>Systolic coronary flow (ml/beat)</th>
<th>Diastolic coronary flow (ml/beat)</th>
</tr>
</thead>
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<tr>
<td>2</td>
<td>136</td>
<td>0.175</td>
<td>135/95</td>
<td>16.5</td>
<td>0.04</td>
<td>0.17</td>
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<td>135</td>
<td>0.170</td>
<td>130/90</td>
<td>17.9</td>
<td>0.14</td>
<td>0.30</td>
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<tr>
<td>3</td>
<td>132</td>
<td>0.180</td>
<td>170/95</td>
<td>21.6</td>
<td>0.10</td>
<td>0.34</td>
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<tr>
<td></td>
<td>136</td>
<td>0.160</td>
<td>170/90</td>
<td>19.3</td>
<td>0.38</td>
<td>1.24</td>
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<tr>
<td>5</td>
<td>75</td>
<td>0.180</td>
<td>160/80</td>
<td>18.6</td>
<td>0.06</td>
<td>0.36</td>
</tr>
<tr>
<td></td>
<td>97</td>
<td>0.160</td>
<td>155/80</td>
<td>17.7</td>
<td>0.25</td>
<td>1.14</td>
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<tr>
<td>16</td>
<td>101</td>
<td>0.160</td>
<td>140/75</td>
<td>22.4</td>
<td>0.05</td>
<td>0.28</td>
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<td></td>
<td>108</td>
<td>0.155</td>
<td>140/75</td>
<td>21.6</td>
<td>0.18</td>
<td>1.05</td>
</tr>
</tbody>
</table>

Data were obtained during a control period (C) and a hyperemic period (H) following a 10-second snare occlusion of the LCCA. The numbers in column 1 correspond with those in Table 1. Abbreviations as in Table 1.
From the top down the ECG, left circumflex coronary flow (LCCA) blood flow, ascending aortic blood flow, and aortic blood pressure are illustrated. The data were measured in vivo during snare occlusion of the LCCA. Stroke systolic and diastolic LCCA flow increased approximately fourfold during reactive hyperemia.

B.P. = blood pressure.

Discussion

The validity of the dynamic compliance measurements obtained in the present study depends on several assumptions. The first is that the properties of the vessels do not change markedly from those present during life. There are no similar data obtained in vivo to compare with these measurements; however, the present data were obtained immediately following death and it is doubtful if the elastic properties of the vessels changed markedly. The fact that in the present experiments the vessels were studied in situ and the normal tethering and restraints were not disturbed should also substantiate the results. Another possible source of error is that the intravascular portions of the epicardial vessels might not have been adequately plugged. Thus, a portion of the saline injected during the experiment might have leaked into the myocardium and given an erroneously small value for compliance. It is unlikely that this was a major source of error since the amount of fluid necessary to maintain the initial distending pressure was only 0.5 to 2.0 ml/min as compared to the 0.1 ml injected in a 150- to 340-msec period or a flow of approximately 30 ml/min. Thus, a leak of 0.5 ml/min is probably negligible. The static resistance as computed by equation 2 decreased by an average of 25% from the lowest to highest initial distending pressure. This change would be expected in a passive system and would not indicate that the leak had increased significantly. In addition, the static studies failed to demonstrate significant perfusion of the myocardium with contrast material at a pressure of 120 mm Hg. The animals were studied within a 3-hour period after death. In each preparation no difference was noted between the data obtained during the first and last series of pulses which were separated by approximately 2 hours. This would indicate that the properties of the vessels were not changing during the study. Thus, it would seem reasonable that the dynamic data presented in the present study are representative of the compliance of the epicardial portion of the coronary arteries as they existed during life.

Several investigators have examined the static pressure-volume characteristics of the coronary arteries. The results of these studies have shown that the vessels are more compliant than would be indicated by the dynamic compliance measurements obtained in the present study. Gregg and co-workers (5) injected lycopodium spores into the coronary artery of dogs to plug the small arteries and studied the static pressure-volume characteristics of the anterior descending coronary artery by infusing mercury at different pressures. From Figure 5 of their report (5) it can be estimated that the increase in volume of this vessel associated with a change in pressure from 70 to 120 mm Hg would be approximately 43%. Patel and Janicki (6) recently studied the rheologic properties of a segment of the left circumflex coronary artery in the dog. The volume distensibility data obtained in their studies would indicate a 223% change in volume for a 50-mm Hg increase in pressure (mean distending pressure 125 mm Hg). The data...
from these studies (5, 6) can be compared to the 31% change in volume for similar pressure increments noted in our static radiographic studies. The disparity between the dynamic and static pressure-volume data may be due to several factors. In the static studies by Patel and Janicki (6) and in our experiments only a segment of the more proximal portion of the coronary artery was studied and it is quite likely that the more proximal segments of the vessels are more compliant than the distal portions. The dynamic data in the present study were obtained from the entire epicardial portion of the coronary artery. Even if one assumes that the distal half of the vessel is rigid and reduces the volume of the cast by 50%, thus doubling the compliance, the values are still considerably less than those obtained in our static studies. The most likely reason for the disparity, however, is that the static technique allows sufficient time for the slow deforming properties of the vessels (stress relaxation) to be manifested.

The data obtained in the femoral artery of three dogs show that the dynamic compliance of this vessel is somewhat less than that of the coronary arteries. The average value of 1.3% change in volume for a 50-mm Hg change in pressure can be compared to a 3.7% change in volume noted in the coronary vessels for the same distending pressure. Dynamic studies of the pressure-radius relationships of the external iliac artery in the dog have been carried out by Patel and co-workers (7). From Table 1 of their report (7), a change in volume distensibility of 1.35% could be expected for a 50-mm Hg pressure increment in this vessel. The comparison of these data with the dynamic compliance obtained in the present study is remarkably good. Bergel (8) examined the static pressure-volume characteristics of the femoral artery of the dog. From Figure 3 of his report (8) one can estimate that an approximate 20% change in volume would have occurred for a 50-mm Hg increase in pressure around a mean initial distending pressure of 100 mm Hg. It is clear that the static compliance is much larger than that obtained in the dynamic situation. The similarity of the discrepancy between both the dynamic studies of Patel and co-workers (7) and the present studies and the static data obtained by Bergel (8) in the femoral artery is consistent with the discrepancy for coronary arteries and supports the validity of the dynamic coronary artery measurements given in this report.

So far as we are aware, there are no measurements of the dynamic compliance of the coronary arteries to compare with the results of the present studies. The wide variation noted in the values of compliance listed in Table 1 are somewhat surprising, but are similar to the range of values obtained in studying the in vivo dynamic vessel mechanics in other arteries (7). At least a part of the differences may be explained by variations in vessel tone at the time of study, but it is our belief that these findings are indicative of a wide range of coronary artery compliance which is present in vivo.

Our static studies were carried out primarily to evaluate the relationships between changes in radius and length in the proximal portions of the coronary artery resulting from an increase in pressure. It is obvious that the portion of vessels studied did lengthen with increasing pressure. Since, during life this would occur at a time when the heart is actually decreasing in size, the vessels must bend for this elongation to occur. At major branch points where large vessels penetrate the myocardium, it would seem reasonable that the epicardial vessel is well tethered to the surface of the heart. If the proximal segments are elongating, then large local stresses might develop at these points. In addition, the use of the opacified coronary arteries to accurately depict the movement of the myocardium would seem to be open to considerable question.

The value for systolic flow which occurred during the control state in the dogs reported in this study is similar to that noted in our laboratory (3, 4) and by others (1). The change in volume of the vessels computed from the dynamic compliance is of the same general order of magnitude. It should be...
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noted that the dynamic compliance measured in dogs with an implanted electromagnetic flowmeter transducer was somewhat less than in the others, suggesting that the area where the transducer had been placed was rendered less compliant by the fibrotic reaction. From the data presented, it would seem reasonable to assume that the major portion of blood flow during systole in the resting state is taken up by the epicardial portions of the coronary arteries. However, during augmentation of coronary flow by tachycardia, exercise, etc., or by experimental reactive hyperemia, it is obvious that the systolic flow values are too high to be accounted for by the compliance of the epicardial vessels. Thus, partial perfusion of the myocardium must take place during systole. Which portion of the myocardium is perfused is certainly conjectural. However, the small multibranching sub-epicardial vessels (class A) (9) are more likely to stay open than the larger vessels which penetrate into the sub-endocardial region since the transmural forces which tend to impede flow during cardiac systole are less in the sub-epicardial region. Thus, it would seem reasonable to conclude that a variable amount of the sub-epicardial region of the heart is perfused during systole, whereas the sub-endocardium does not receive any blood during the systolic phase of the cardiac cycle.

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References

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