Experimental Myocardial Infarction in the Cat

I. Reversible Decline in Contractility of Noninfarcted Muscle

PETER MATHES, M.D., DIETMAR ROMIG, DIETER SACK, M.D., AND WOLFGANG ERHARDT, M.D.

SUMMARY The contractile state of the noninfarcted myocardium was examined in adult cats after myocardial infarction produced by ligation of several branches of the left coronary artery. At 2 days, 7 days, and 6 weeks after infarction, and after determination of intracardiac pressures, papillary muscles were excised from the noninfarcted segment of the right ventricle and attached to a myograph for analysis of contractile function. One week after infarction there was a decline in actively developed force at $L_{\text{max}}$, caused by a decrease in the rate of force development. In addition, the response to procedures that augment myocardial contractility, such as paired stimulation and increasing the frequency of electrical stimulation, was significantly depressed. Two days after infarction, changes were less significant, although similar in direction. Six weeks after infarction, developed force at $L_{\text{max}}$ had returned to normal values. The response to procedures augmenting contractility also had returned to normal. There appears to be a distinct, reversible loss of contractility in the remaining viable myocardium in the early phase after experimental infarction.

AFTER MYOCARDIAL infarction, the uninvolved portion of the heart generally is thought to maintain function and metabolism, unless coexisting stenosis of additional vessels causes ischemia of the noninfarcted segments. However, after experimental myocardial infarction, the clearly nonischemic portion of the heart muscle shows changes in its energy metabolism as well as a reversible decline in norepinephrine content. The functional significance of these changes, however, remains largely unknown because determinations of ventricular function after infarction fail to differentiate the infarcted and the surviving areas. Theroux et al. demonstrated an increased extent of shortening of noninfarcted areas early after infarction which was caused by regional operation of the Frank-Starling mechanism; an alternate explanation could be that unloading of the ischemic segment permitted increased active shortening as well. In the present study we analyzed myocardial function after isolation of the surviving heart muscle, to exclude any effect of the infarct itself. To avoid the ill-defined changes which occur in the zone which borders the infarct, we studied right ventricular papillary muscles after left ventricular infarction. We thus attempted to determine the effect of an acute myocardial infarction on the contractile properties of surviving, noninfarcted heart muscle.

Methods

Myocardial infarction was produced in adult cats by ligation of several branches of the left coronary artery. Under general anesthesia (halothane, $N_2O$, and $O_2$), the cats were intubated, and a thoractomy was performed through the 5th left intercostal space. After opening the pericardial sac, we ligated between three and five branches of the left coronary artery which lead to the free wall of the left ventricle. Infarctions produced in this way were comparable in extent and location in different cats. Normal as well as sham-operated cats were used as controls. The sham operation consisted of opening the pericardial sac, without ligating the coronary arterial branches.

A total of 62 cats was divided into four groups, depending on the time which had elapsed after operation: group 1, controls ($n = 17$); group 2, sham-operated cats, examined 1 week postoperatively ($n = 10$); group 3, 2 days after infarction ($n = 14$); group 4, 7 days after infarction ($n = 14$); and group 5, 6 weeks after infarction ($n = 7$).

At the time of study the cats were anesthetized by intravenous injection of a mixture consisting of metamitridat, 25 mg/kg, and fentanyl, 0.025 mg/kg. The following hemodynamic parameters were determined: heart rate, mean aortic pressure, right atrial pressure, right ventricular systolic and diastolic pressure. The zero reference for intracardiac pressures was the midstich.

After hemodynamic evaluation the heart was rapidly excised and transferred to cooled saline saturated with $O_2$. Right ventricular papillary muscles were excised and transferred into the myograph chamber, which contained oxygenated Krebs-Ringer solution comprising 5.6 mm glucose and (in mEq/liter) $Na^+$, 152; $K^+$, 3.6; $Cl^-$, 135; $HCO_3^-$, 25; $Mg^{2+}$, 1.2; $H_2PO_4^-$, 1.3; and $Ca^{2+}$, 5.0. The pH of the solution was 7.4 when it was gassed with 95% $O_2$-5% $CO_2$. Studies were performed at 29°C. The water jackets of the muscle bath were attached to a constant temperature pump (F.J. Haake) adjusted to 29°C. The temperature of the bath itself was checked with an independent thermometer.

Two days after ligation of the coronary arteries, the demarcation of the infarcted area was sufficiently clear to permit a macroscopic differentiation of infarcted and noninfarcted heart muscle. In addition, the base of the removed papillary muscle was examined histologically, and only when the site of removal showed normal histology was the...
muscle included in the final evaluation. After removal of the papillary muscle, the left ventricle was cut in sections and submerged in a solution containing nitroblue-tetra-colizium-chloride, which stains normal muscle deep blue, and infarcted myocardium gray-white. The macroscopically visible area of the infarct was excised and weighed, as were the ventricles and interventricular septum.

The base of the removed papillary muscle was fixed in a Lucite clip and connected by a straight steel wire to a force transducer (Hottinger, HBM Q 11/10). The signal from the force transducer as well as its first time derivative were recorded continuously; measurements were made from recordings obtained at a paper speed of 100 mm/sec. The muscles were stimulated through field electrodes with rectangular pulses of 6-msec duration and 10% above threshold (Grass stimulator, model 588). Muscle length was determined at $L_{\text{max}}$ for calculation of cross-sectional area, the muscle was assumed to have a cylindrical shape and a specific gravity of 1.0.

After it had been mounted, the muscle was allowed to contract isometrically at lengths below $L_{\text{max}}$ until mechanical activity had stabilized; usually this occurred within 90 minutes. Then muscle length was increased in steps to a length slightly greater than $L_{\text{max}}$ and active developed force and passive tension were recorded continuously. In addition, we studied the effects of paired electrical stimulation by using pairs of stimuli at a frequency of 0.2 Hz. The interstimulus interval did not exceed the muscle’s effective refractory period by more than 30 msec. After an additional period of stabilization during which single stimuli were applied at 0.2 Hz, we studied the effects of increasing the frequency of stimulation. For each intervention studied we recorded developed force as well as the velocity of force development, along with the stimulus artifact, on a multichannel direct writing recorder with a linear frequency response up to 300 Hz. All data were analyzed by Student’s $t$-test and expressed as mean ± SE.

**Results**

**HEMODYNAMIC FINDINGS**

There was an insignificant decrease in heart rate after infarction. Mean aortic pressure decreased from a control value of 94 ± 6 mm Hg to 86 ± 8 mm Hg 2 days after infarction; subsequently there were no significant changes. Figure 1 shows right heart pressures recorded at different times after infarction. There is an insignificant increase in mean right atrial pressure in the early phase; right ventricular end-diastolic pressure increases significantly above the control value of 5.0 ± 0.5 mm Hg to 7.0 ± 1.0 mm Hg 1 week after infarction ($P < 0.05$) and shows a slight, insignificant decrease thereafter. Right ventricular systolic pressure does not change significantly after infarction, although there is a tendency for it to increase.

**VENTRICULAR WEIGHTS**

Table 1 shows the relative ventricular weights ($g/kg$) after infarction. There are no statistically significant changes, although the relative weight of the left ventricle tends to increase after infarction as a result of edema in the infarcted area. By 48 hours after infarction, the infarct comprises 21.6 ± 1.9% of the left ventricle in terms of wet weight. One week after infarction only 15.9 ± 1.7% of the left ventricle appears to be infarcted. We attribute this change to partial resorption of necrotic tissue rather than to a true difference in infarct size. Six weeks after infarction the remaining scar tissue comprises 14.1 ± 2.2% of the left ventricle.

**MYOCARDIAL MECHANICS**

Table 2 summarizes for the individual muscles the data describing force development at $L_{\text{max}}$ in relation to cross-sectional area, muscle length, and time to peak force development. The numbers in the subgroups which are identical in cross-sectional area are too small to permit statistical evaluation. In comparison to the average value for the entire group, however, a similar tendency becomes apparent in the individual subgroups consisting of muscles of similar cross-sectional areas. The average cross-sectional areas are quite similar, except for the group studied 1 week following infarction, for which values were slightly higher. Muscle length is comparable as well, except for the group studied 6 weeks following infarction, in which muscles are slightly shorter. There is little variation in the time to peak force development. The resting tension shows no significant differences between the groups under study (controls, 0.95 ± 0.15 g/mm² cross-sectional area; sham-operated cats, 1.00 ± 0.16 g/mm²; 48 hours following infarction, 0.90 ± 0.20 g/mm²; 1 week, 0.85 ± 0.18 g/mm²; 6 weeks, 0.95 ± 0.20 g/mm²).

The active force developed at the apex of the curve relating active length to force ($L_{\text{max}}$) is shown in Figure 2. For
normal muscles the average value for active force developed at \( L_{\text{max}} \) is 5.9 ± 0.16 g/mm\(^2\) cross-sectional area. There is no significant change in this value 1 week after sham operation, whereas at 1 week after infarction the force actively developed by noninfarcted right ventricular papillary muscles decreases significantly to 4.3 ± 0.24 g/mm\(^2\) cross-sectional area (\( P < 0.001 \)). Two days after infarction, developed force at \( L_{\text{max}} \) is 5.4 ± 0.33; this is an insignificant change from the control, although a tendency to decline is apparent. Six weeks after infarction, developed force at \( L_{\text{max}} \) reaches 5.35 ± 0.48 g/mm\(^2\); this represents a significant increase (\( P < 0.01 \)) from the depressed value obtained 1 week after infarction but is not significantly different from the control value. If we consider the rate of force development (Table 3), a similar tendency becomes apparent. One week after infarction the decrease in the rate of force development by noninfarcted heart muscle is significant; 6 weeks after infarction, nearly normal values for the rate of force development by the surviving heart muscle is significantly increased in comparison to the early postinfarction period.

**PAIRED ELECTRICAL STIMULATION**

Figure 3 shows the effects on force development of sustained postextrasystolic potentiation, caused by paired electrical stimulation, for the different groups under study. The normal increment in force is 3.5 ± 0.12 g/mm\(^2\) cross-sectional area; it remains essentially unchanged after sham operation (3.2 ± 0.40 g/mm\(^2\)) and decreases 2 days after infarction to 2.0 ± 0.26 (\( P < 0.01 \)) and further to 1.7 ± 0.18 at 1 week after the infarct (\( P < 0.001 \)). Six weeks after infarction the increment in force reaches a nearly normal value (3.0 ± 0.28), as does the total force developed (8.23 ± 0.90 g/mm\(^2\) cross-sectional area) (Fig. 3).

**FORCE-FREQUENCY RELATIONS**

Increasing the frequency of stimulation results in an increased force of contraction;\(^4\) in normal muscles force changes from 5.9 ± 0.16 g/mm\(^2\) at 0.2 Hz to 7.8 ± 0.24 g/mm\(^2\) at 0.6 Hz (solid line in Fig. 4). One week after sham operation, force development increases from 6.1 ± 0.13 g/mm\(^2\) at 0.2 Hz to 8.05 ± 0.40 g/mm\(^2\) at 0.6 Hz and shows no significant difference from the control value. After infarction, however, there is a diminished increment in force following an increase in the frequency of stimulation; this can be observed 2 days after infarction, and is even more prominent 1 week after infarction where there is a smaller initial increment in force development and an early flattening of the frequency-force relation (Fig. 5). Six weeks after infarction, a tendency toward normalization is observed, although control values are not reached (Fig. 4).

The rate of isometric force development shows a similar tendency (Fig. 5). Whereas normal muscles show a continuing increase in the rate of force development in response to an increase in the frequency of stimulation from 22.2 ± 1.0 g/sec at 0.2 Hz to 38.4 ± 3.0 g/sec at 0.6 Hz, the noninfarcted heart muscle 1 week after infarction shows significantly smaller increments (Fig. 5). In contrast, sham-operated cats 1 week after operation show an essentially normal increase in the rate of force development, from 20.9 ± 0.8 g/sec at 0.2 Hz to 39.4 ± 2.1 g/sec at 0.6 Hz. Six weeks after infarction, nearly normal values for the rate of force development again are reached (Fig. 5).

**Discussion**

Acute myocardial infarction is regularly followed by a depression of ventricular function.\(^**\) The performance of the ventricle depends largely on the extent of myocardial necrosis; however, the function of the surviving heart muscle, which has to compensate for the loss of viable myocardium, must be considered as well. Earlier experimental investigations demonstrated a reversible decline in cardiac norepinephrine stores at sites remote from the infarcted area;\(^2\) this change was thought to be due to a persistent increase in sympathetic activity. Hood et al.\(^*\) demonstrated an increased extent of shortening in the surviving heart muscle; this finding was confirmed by Theroux et al.,\(^*\) who demonstrated increased systolic shortening in the uninvolved myocardium early after infarction. This process was shown to be due to operation of a Frank-Starling mechanism in the noninfarcted myocardium; however, systolic unloading due to paradoxical motion of the ischemic segment also might cause increased fiber shortening in the nonischemic myocardium. In our investigation, the surviving heart muscle was isolated after myocardial infarction and studied in vitro to avoid the effects of catecholamine stimulation\(^11\) as well as the influence of the infarcted segment on the determination of relative heart weight after infarction.

### TABLE 1 Relative Heart Weight after Infarction

<table>
<thead>
<tr>
<th></th>
<th>Control</th>
<th>2 days</th>
<th>7 days</th>
<th>6 weeks</th>
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<tbody>
<tr>
<td>Total heart</td>
<td>2.86 ± 0.08</td>
<td>3.08 ± 0.14</td>
<td>3.01 ± 0.14</td>
<td>2.68 ± 0.10</td>
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<td>Left ventricle</td>
<td>1.57 ± 0.05</td>
<td>1.75 ± 0.10</td>
<td>1.63 ± 0.10</td>
<td>1.48 ± 0.05</td>
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<td>Septum</td>
<td>0.68 ± 0.03</td>
<td>0.75 ± 0.06</td>
<td>0.73 ± 0.04</td>
<td>0.68 ± 0.02</td>
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<td>Right ventricle</td>
<td>0.58 ± 0.03</td>
<td>0.59 ± 0.04</td>
<td>0.64 ± 0.03</td>
<td>0.53 ± 0.04</td>
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NS = not significant.
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<thead>
<tr>
<th></th>
<th>Controls</th>
<th>Sham-operated 48 hr after infarction</th>
<th>1 wk after infarction</th>
<th>6 wk after infarction</th>
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<tbody>
<tr>
<td>Fmax (g/mm²)</td>
<td>CSA (mm²)</td>
<td>ML (mm)</td>
<td>t₁₋FR (sec)</td>
<td>Fmax (g/mm²)</td>
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<tr>
<td>6.0</td>
<td>0.50</td>
<td>3.3</td>
<td>0.37</td>
<td>7.0</td>
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<td>5.9</td>
<td>0.51</td>
<td>5.9</td>
<td>0.43</td>
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<td>5.6</td>
<td>0.51</td>
<td>3.7</td>
<td>0.46</td>
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<td>6.2</td>
<td>0.57</td>
<td>5.5</td>
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**CSA range, 0.40-0.49 mm²**

**CSA range, 0.50-0.59 mm²**

**CSA range, 0.60-0.69 mm²**

**CSA range, 0.70-0.79 mm²**

**CSA range, 0.80-0.89 mm²**

**CSA range, 0.90-1.00 mm²**

**Mean ± SE**
of myocardial contractility. After left ventricular infarction, right ventricular papillary muscles were taken to avoid the ischemic zone bordering the necrotic area.

Hemodynamic observations showed a slight increase in right ventricular end-diastolic pressure in the early phase following infarction, but this pressure remained considerably below values observed in congestive failure. Although pressures recorded during anesthesia differ considerably from those recorded in the unanesthetized condition, available evidence indicates that there was no congestive failure, which might have been responsible for the observed depression of contractility. Two days after infarction a tendency toward a decline in maximum force development was observed in measurements on noninfarcted heart muscle, although the difference from control and sham-operated animals was not significant. One week after infarction the maximum force development by the surviving heart muscle was significantly depressed. Six weeks after infarction nearly normal levels of force development again were reached (Fig. 2). In addition to the reduction in force development, the response to procedures that augment myocardial contractility was depressed. During paired electrical stimulation (Fig. 3) the absolute increases in force and rate of force development were significantly below normal in the early phase following infarction. The force-frequency response was depressed as well (Fig. 4); the increments in force following an increase in stimulation-frequency were smaller than for controls and showed an early flattening of the curve. A similar observation was made when we examined the rate of isometric force development (Fig. 5).

Six weeks after infarction nearly normal values were reached for developed force as well as for the increments in force development in response to isotropic stimuli. Therefore, the decline in contractility of noninfarcted heart muscle appears to be a reversible process which is limited to the early postinfarction period. Similar changes in contractility have been reported for hypertrophied heart muscle caused by pulmonary and aortic constriction. In contrast to changes in resting tension observed in hypertrophied papillary muscles, there is no change in resting tension observed in the surviving heart muscle after infarction.

There appears to be a relation between the time course of the decline in norepinephrine stores in noninfarcted heart muscle and the observed changes in contractile function. Both norepinephrine stores and contractility, as determined in vitro, show a decline in the early postinfarction period and a late return to normal values. However, there appears to be

### Table 3 Rate of Isometric Force Development by Noninfarcted Heart Muscle

<table>
<thead>
<tr>
<th>After infarction</th>
<th>Control (n = 17)</th>
<th>Sham-operated (n = 10)</th>
<th>48 hours (n = 14)</th>
<th>7 days (n = 14)</th>
<th>6 weeks (n = 7)</th>
</tr>
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<tr>
<td>Velocity of force development (g/sec ± SE)</td>
<td>22.2 ± 1.0</td>
<td>20.9 ± 0.8</td>
<td>19.7 ± 1.4</td>
<td>15.8 ± 1.0</td>
<td>24.3 ± 2.4</td>
</tr>
<tr>
<td>P in comparison to controls</td>
<td>NS</td>
<td>NS</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>NS</td>
</tr>
<tr>
<td>P in comparison to 7 days after infarction</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.05</td>
<td>&lt;0.001</td>
<td></td>
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</tbody>
</table>

n = number of cats; NS = not significant.
FIGURE 4 Force-frequency relation for noninfarcted heart muscle after experimental infarction. Whereas increasing the frequency of contraction causes a proportionate increase in force developed by normal muscle, the response is significantly smaller 1 week after infarction. Six weeks after infarction, a significant increase in force development is seen, although normal values are not reached. CSA = cross-sectional area.

FIGURE 5 Rate of isometric force development in response to increasing the frequency of stimulation. Again, the noninfarcted heart muscle 1 week after infarction shows significantly smaller increments in the rate of force development than does the control, whereas 6 weeks after infarction, values return nearly to normal.

no direct relationship between intact norepinephrine stores and contractility. Reserpinized muscles, i.e., muscles completely depleted of norepinephrine, appear to have a normal contractility when examined in vitro.14, 15 Observations by Blinks,16 however, indicate that depending on the mode of stimulation, the contractile performance of the muscle may vary in relation to its catecholamine content. With field stimulation at a voltage considerably above threshold level, catecholamine release led to a significant increase in force of contraction. In our study, however, the stimulus-voltage was kept 10% above threshold, i.e., at a level which would not lead to a release of endogenous norepinephrine. The decline in norepinephrine stores thus appears unlikely to be the primary cause for the observed decline in contractility.

Although there is no clear explanation available at present, the initial decline and late recovery of contractile function indicate that the observed changes are secondary to the infarct itself rather than a direct consequence of the coronary occlusion. A significant part of the force developed by the surviving heart muscle is spent in realigning the stress in the ventricular wall,17, 18 in addition to compensating for the loss of functioning tissue. The disappearance of the paradoxical motion19 as well as the replacement of the infarcted area by noncompliant scar tissue20 reduces the force which need be generated by the noninfarcted heart muscle and thus permits a return to a normal level of function, with a subsequent return of normal contractility.

In judging the overall effect of an acute myocardial infarction on the hemodynamic performance of the heart, it appears mandatory to consider the contractile state of the surviving heart muscle. Although contractile performance in vivo may be enhanced by the Frank-Starling mechanism and by increased levels of circulating catecholamines, the basal contractile state already may be depressed.

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Modification of the Flow-Generating Capability of the Canine Heart-Lung Compartment by the Carotid Sinus Baroreceptor Reflex

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SUMMARY To quantitatively understand how the carotid sinus baroreceptor reflex modifies the Starling curve (i.e., the aortic flow (AF)-mean right atrial pressure (MRAP) relationship), experiments were performed in closed-chest, naturally breathing, and anesthetized dogs before and after vagotomy. Mean aortic pressure (MAP) was fixed at approximately 100 mm Hg and the pressure in the isolated carotid sinus (ISP) was varied from 75 to 150 mm Hg in steps of 25 mm Hg. At each ISP, MRAP was slowly increased and decreased while measuring AF by a previously implanted electromagnetic flow probe. A family of AF-MRAP relation curves specified at the different ISP’s were thus obtained. Third-order polynomials in MRAP and ISP adequately fit these curves. Whether the vagi were intact or cut, there was no significant difference between the AF-MRAP relation curves obtained at an ISP of 75 or 100 mm Hg. However, decreasing ISP from 125 to 100 mm Hg caused a 24% increase in AF, and increasing ISP to 150 mm Hg caused a 15% decrease in AF in the dogs with intact vagus nerves. For the vagotomized dogs, the same decrease or increase in ISP caused a 17% increase or a 21% decrease in AF, respectively. When MAP was allowed to change by the reflex, only insignificant changes in AF occurred. We conclude that the carotid sinus reflex significantly alters the flow-generating ability of the heart-lung compartment by as much as 40% but this becomes clearly observable only if the reflex change in aortic pressure is prevented.

CARDIAC OUTPUT, central arterial pressure, and central venous pressure are the lumped variables that couple the two major subsections of the circulatory system, i.e., the heart-lung and systemic vascular compartment. Therefore, a simplified systems analysis of reflex control of the circulation can be achieved by describing reflex control on each of the two compartments in terms of these pressure and flow variables. In our previous reports,1-3 we described carotid sinus reflex controls of the resistive and capacitive properties of the systemic vascular bed in terms of systemic arterial and venous pressures and systemic flow. What is needed then is a quantitative description of carotid sinus reflex control of the heart-lung compartment, again in terms of the three variables appropriately controlled and measured.

The literature on the arterial baroreceptor reflex control of cardiac function spans many decades and investigators used several different preparations. The emphasis of most of these studies was placed on establishing whether or not the reflex alters cardiac contractility. The quantitative aspects of the conclusions were often in conflict; some investigators2-17 presented evidence that the reflex modified the contractility of the ventricle or heart muscle, whereas others18-28 found insignificant effects on cardiac output or other indices of contractility. There are reasons for these differences. Many of these investigators used carotid arterial occlusions.8-10, 16, 18, 20-28 This, unfortunately, is not a quantitatively exact method to stimulate the receptors. Among those who used isolated carotid sinus preparations2-8, 11, 12, 14, 15, 17, 19 only several9, 8, 14, 17 controlled the arterial pressure or afterload. Even fewer investigators8, 16, 17 controlled the preload simultaneously. The studied indices of contractility include (1) peak systolic pressure in an isovolumically contracting ventricle,6, 12 (2) peak systolic force measured by an isometric strain gauge sutured on the ventricle wall,6 and (3) the maximum rate of development of ventricular pressure [dP/dt]max.14, 15, 17 Although most of the studies with these indices have demonstrated reflex changes in contractility one cannot use the reported changes in the index values to predict reflex changes in cardiac output under a given set of conditions of preload and afterload. Also, most studies analyzed either the left or right ventricle, rather than the heart-lung compartment as a whole.

For the reason stated in the first paragraph, the purpose of the present study is not to reestablish reflex change in ventricular or myocardial contractility. Instead, we are presenting a quantitative description of the extent to which the carotid sinus reflex alters the flow-generating ability of the heart-lung compartment under a variety of values of well controlled systemic arterial and venous pressures in closed-chest dogs.
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