Right and left heart pressures in acute myocardial infarction

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AUTHORS' SYNOPSIS In dogs the effects of acute myocardial infarction (AMI) on right and left heart pressure relationships were studied. Right atrial pressure was a poor predictor of left heart pressures. Pulmonary artery end-diastolic, left atrial, and left ventricular end-diastolic pressures tended to equalize in normals (except with tachycardia) and after AMI. In a previously scarred left ventricle, however, after superimposed AMI and marked elevations in left ventricular end-diastolic pressure, prominent atrial impact waves resulted in this being appreciably higher than the other two pressure measurements.

Although pressure relationships between the right and left sides of the heart have long been of interest to cardiovascular physiologists, increasing utilization of right heart pressures in monitoring patients with acute myocardial infarction for indications of left-sided events underscores the need for a better appreciation of the uses and limitations of such measurements.

While central venous pressure tends to rise with elevations of left atrial and ventricular end-diastolic pressures, use of the central venous pressure as an indicator of the level of left heart pressures in acute myocardial infarction has been unrewarding (Forrester, Diamond, McHugh, and Swan, 1971; Hamosh and Cohn, 1971). Since, in the absence of mitral stenosis and pulmonary vascular obstruction, the pulmonary artery end-diastolic pressure has been shown to reflect the left ventricular end-diastolic pressure in stable patients studied at catheterization (Kalman, Herbert, Conroy, and Kossman, 1966), this has been recommended as a better indicator of left ventricular end-diastolic pressure in acute myocardial infarction (Rapaport and Scheinman, 1969; Hunt, Pombo, Potanin, Russell, and Rackley, 1970). However, this, too, has been reported frequently to underestimate left-ventricular end-diastolic pressure, especially when the latter is significantly elevated in acute myocardial infarction (Lal, Loeb, Sinno, Rahimtoola, Chuquimia, Rosen, and Gunnar, 1971).

Since the dog is frequently used in experimental studies of acute myocardial infarction, and can readily have pressures measured simultaneously in multiple chambers before and after acute infarction, the present study was undertaken in this model. Our purpose was to define right and left heart pressure relationships in the dog before and after myocardial infarction with and without shock, compare these findings with what is known in man, and attempt to determine various factors affecting and altering such relationships.

Methods
Mongrel dogs were anaesthetized with morphine sulphate (3 mg/kg) and sodium pentobarbital...
(15 to 20 mg/kg) and placed in the right lateral decubitus position. Cuffed endotracheal tubes were inserted and respiration assisted by means of a Harvard volume type respirator to assure adequate ventilation (Kleinman and Radford, 1964), verified by frequent determinations of arterial pH and pO₂. Five groups of dogs were studied:

- **Group I. Normal dogs.**
- **Group II. Dogs with acute myocardial infarction without shock.**
- **Group III. Dogs with acute myocardial infarction with shock.**
- **Group IV.** A non-shock group specifically studied for left ventricular-right atrial pressure relationships before and after acute myocardial infarction.
- **Group V.** Dogs with acute myocardial infarction imposed upon a previously scarred left ventricle.

**Normal dogs**

In eight dogs catheters were advanced under fluoroscopy via neck and femoral vessels to the left ventricle, aortic root, right atrium, and pulmonary artery with ECG monitoring. Since the injudicious use of positive pressure ventilation has been shown to reduce cardiac output and alter cardiovascular pressures (Morgan, Crawford, and Guntheroth, 1969), these dogs were studied with and without artificial respiratory assistance. No significant differences in heart rate, pressures, or cardiac output resulted. To increase intracardiac pressures in six dogs, blood volume was expanded with three 7 ml./kg increments of donor dog blood. Fifteen minutes were allowed for stabilization of hemodynamics after each transfusion before repeat measurements.

Statham pressure transducers were used for measurement of left ventricular (P23Gb) and other pressures (P23Db). These were placed at mid-thoracic level and averaged during the course of a respiratory cycle. In addition to aortic pressure, left ventricular end-diastolic, (LVEDP) pulmonary artery and right atrial pressures were measured simultaneously at a high sensitivity (1 mm Hg equal to 4 mm paper) with equisensitive transducers. To prevent clotting, catheters were flushed with heparinized saline. Cardiac output was determined with indocyanine green as previously described (Weisse, Saffa, Levinson, Jacobsen, and Regan, 1970).

Dye curves, pressures, and ECG were recorded on an Electronics for Medicine DR-8 Recorder. Arterial hematocrit determinations were performed on all dogs to assure normal levels.

**Myocardial infarction – no shock (30 dogs)**

After control measurements in 24 dogs either the anterior descending or circumflex branch of the left coronary artery was obstructed by means of a thrombus-producing electrode catheter advanced fluoroscopically 1-2 cm beyond the vessel's origin (Weisse, Lehan, Ettinger, Moschos, and Regan, 1969). After obstruction, indicated by ST segment elevation in appropriate leads, the dogs were observed for 1 to 4 hr until definite LVEDP elevation occurred before measurements were repeated and transfusions carried out as described above. Since heparin would interfere with thrombus formation, this was not given in these dogs until after myocardial infarction was established. Included in this group were six dogs from the shock group (see below) in whom normotensive blood pressure spontaneously returned after initial hypotension. Pulmonary artery pressures were obtained in 19 of the 30 dogs and right atrial pressures in all.

**Myocardial infarction – shock (28 dogs)**

Cardiogenic shock was produced by a modification of the method of Agress (Agress, Rosenberg, Jacobs, Binder, Schneiderman, and Clark, 1952) whereby one to three 40 mg boli of styrene-divinyl benzene microspheres (300 μm diameter) were suspended in heparinized blood and injected through a single end-hole Sones catheter into either main left coronary artery branch. Cardiogenic shock herein defined included both (1) at least a 33% reduction in aortic systolic pressure, and (2) a systolic aortic pressure below 90 mm Hg persisting at least 30 min after injection. Therefore, appreciable systolic hypotension was present in this group (mean 70 ± SEM 2 mm Hg) In six dogs normotensive levels spontaneously returned 30 min to 2 hr thereafter and pressure measurements in them subsequently considered with the non-shock group. Blood transfusions were performed in four shock dogs, after which arterial pressure rose above shock levels in three; these data were included in the shock group.

**LVEDP-left atrial pressure relationships in myocardial infarction (8 dogs)**

In this group of dogs pressures only were measured with 100 cm No. 7 or 8 Goodale-Lubin catheters advanced to the left ventricle via the femoral artery and left atrium in a retrograde manner through the left ventricle via the right carotid artery. The left carotid artery was used for the electrode catheter and thrombotic obstruction of a main left coronary branch. After myocardial infarction was induced blood volume expansion was carried out with blood or 10% Rheomacrodex® in 10% dextrose, in increments similar to those in the previous groups.
Acute myocardial infarction in previously scarred ventricle (7 dogs)
In these dogs thoracotomy was initially performed and at the apex of the left ventricle a series of 2 mm deep interrupted sutures were placed encompassing a circular area 2 cm in diameter in order to interrupt the blood supply to this area. Three to six weeks later, after recovery from surgery, intracardiac pressures were measured before and after induction of acute myocardial infarction by the thrombus method.

Correlation coefficients ($r$) and regression equations were calculated for LVEDP versus right atrial, pulmonary artery end-diastolic, and left atrial pressures. T tests on $r$s were performed and P values thereof were found from Student's $t$ table.

**Results**
Right atrial and left ventricular end-diastolic (LVEDP) pressures
Throughout the range of LVEDP under normal and abnormal conditions, right atrial pressure tended to rise as LVEDP rose (Fig. 1, Table 1). Although these correlations were highly significant statistically, especially among the normal and non-shock infarction groups, it is evident

![Diagram showing correlation between right atrial and left ventricular end-diastolic pressures](image_url)
Weisse, Narang, Haider, and Regan

TABLE I
Right atrial and LV end-diastolic pressures

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>r</th>
<th>P value</th>
<th>Regression equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normals</td>
<td>31</td>
<td>0.78</td>
<td>&lt;0.001</td>
<td>RAP = 0.80 + 0.35 LVEDP</td>
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<tr>
<td>MI – no shock</td>
<td>134</td>
<td>0.78</td>
<td>&lt;0.001</td>
<td>RAP = 0.19 + 0.23 LVEDP</td>
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<td>MI – shock</td>
<td>56</td>
<td>0.37</td>
<td>&lt;0.01</td>
<td>RAP = 0.29 + 0.09 LVEDP</td>
</tr>
</tbody>
</table>

n = number of measurements; r = correlation coefficient.

FIG. 2 Relationship of pulmonary artery end-diastolic pressure to LVEDP before and after blood transfusions in normal dogs and those with acute myocardial infarction without and with shock. (Top graph: x = normal controls, △ = normals after transfusions; middle and bottom graphs: x = controls, ○ = acute myocardial infarction, ● = myocardial infarction plus transfusions). For infarct groups regression lines drawn for LVEDP values below and above 12 mm Hg. When LVEDP is elevated there is better correlation with PAEDP and less scatter. (See Table 2.) (* highest shock PAEDP value = 33 mm Hg)
from inspection of Fig. 1 that, in the presence of myocardial infarction, LVEDP could be frequently markedly elevated, even to pulmonary oedema levels, while right atrial pressure was still normal or minimally elevated. Thus right atrial pressure was a poor predictor of LVEDP.

**Pulmonary artery end-diastolic pressure (PAEDP) and LVEDP**

This relationship was evaluated within the normal range of LVEDP and when this pressure was distinctly elevated (equal or greater than 12 mm Hg) (Fig. 2, Table 2).

Mean of the combined control PAEDP values obtained in 47 dogs in the first three groups studied was significantly higher than LVEDP (PAEDP = 9 ± SEM 0.4 mm Hg; LVEDP = 4 ± 0.3 mm Hg; P < 0.001). The higher control heart rates in the 38 dogs where PAEDP exceeded LVEDP by more than 2 mm Hg (mean 144 beats/min, range 95–215) and the lower rates (mean 109, range 75–140) in the nine dogs in which the PAEDP was within 2 mm Hg of the LVEDP suggest that inadequate diastolic time for equilibration of pressures may have contributed to this discrepancy (P < 0.001).

Another possibility was that hypoventilation resulting from the lateral decubitus position and causing pulmonary hypertension might have, in part, accounted for the PAEDP-LVEDP differences in the controls. However, the normal arterial blood gases and the persistence of these differences in instances where dogs were slightly hyperventilated and/or studied in the supine position tended to rule this out.

After acute myocardial infarction without shock (group II; middle graph, Fig. 2) with the LVEDP pressure below 12 mm Hg, correlation between this and PAEDP continued to be poor (r = 0.20) but when LVEDP rose to 12 mm Hg or above the correlation improved considerably (r = 0.84). Among the 30 instances in which LVEDP equalled or exceeded 12 mm Hg the PAEDP was within 3 mm Hg in all but eight instances and within 6 mm Hg in all but two. A similar but less striking improvement in correlation (r from 0.42 to 0.75) occurred in the shock dogs (group III, bottom graph) when LVEDP similarly increased. With the exception of one dog, PAEDP was within 3 mm Hg of LVEDP in 11 of 17 measurements and within 6 mm Hg in 16 of 17.

From the data obtained, it was possible to derive an equation using PAEDP as an independent variable to estimate LVEDP within a 95% confidence interval of ± 3 mm Hg in the abnormal ranges studied. For the non-shock dogs the equation was: LVEDP = 3.4 ± 0.89 PAEDP (when LVEDP = 12–34 mm Hg). For the shock dogs LVEDP = 9.0 ± 0.73 PAEDP (LVEDP = 12–36 mm Hg).

Since heart rate after acute myocardial infarction did not decrease, the improved PAEDP-LVEDP relationship could not be accounted for by this. Another factor considered was a change in total pulmonary and pulmonary arteriolar resistances. Since neither left atrial pressure nor pulmonary capillary wedge pressures were measured in the first two infarct groups, group IV was studied to evaluate whether, in acute myocardial infarction, LVEDP could be sub-

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**TABLE 2**

**Pulmonary artery end-diastolic and LV end-diastolic pressures**

<table>
<thead>
<tr>
<th>n</th>
<th>r</th>
<th>P value</th>
<th>Regression equation</th>
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<tr>
<td>Normals</td>
<td>30</td>
<td>0.52</td>
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<tr>
<td>MI - no shock</td>
<td></td>
<td>0.20</td>
<td>&gt;0.2</td>
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<td>LVEDP&lt;12 mm Hg</td>
<td>29</td>
<td>0.84</td>
<td>&lt;0.001</td>
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<td>MI - shock</td>
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<td>0.42</td>
<td>&lt;0.05</td>
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<tr>
<td>LVEDP&lt;12 mm Hg</td>
<td>32</td>
<td>0.75</td>
<td>&lt;0.001</td>
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<tr>
<td>MI - no shock</td>
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<td>0.27</td>
<td>PAEDP = 2.79 + 0.79 LVEDP</td>
</tr>
<tr>
<td>MI - shock</td>
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<td>0.79</td>
<td>PAEDP = 0.29 + 0.77 LVEDP</td>
</tr>
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</table>

n = number of measurements; r = correlation coefficient.
FIG. 3 The close relationship of left atrial and left ventricular end diastolic pressure persists after acute myocardial infarction.

### TABLE 3

<table>
<thead>
<tr>
<th>Dog no.</th>
<th>HR (beats/min)</th>
<th>PAEDP (mm Hg)</th>
<th>LAP (mm Hg)</th>
<th>'PCW' (mm Hg)</th>
<th>LVEDP (mm Hg)</th>
<th>Comment</th>
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<td>1</td>
<td>C 204</td>
<td>11</td>
<td>6</td>
<td>—</td>
<td>7</td>
<td>Note tachycardia</td>
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<td></td>
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<tr>
<td>6</td>
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<td>I 82</td>
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<tr>
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<td>C 160</td>
<td>46</td>
<td>5</td>
<td>—</td>
<td>5</td>
<td>Pulmonary hypertension as a result of severe heart worm infestation</td>
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<tr>
<td></td>
<td>I 96</td>
<td>39</td>
<td>17</td>
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HR = heart rate, PAEDP = pulmonary artery end-diastolic pressure, LAP = mean left atrial pressure, 'PCW' = pulmonary capillary wedge pressure, LVEDP = left ventricular end-diastolic pressure, C = control with scar before acute ischaemia, I = acute ischaemia.
Pressures in acute myocardial infarction

FIG. 4. After acute myocardial infarction (MI) with appreciable elevation of left-ventricular end-diastolic pressure (LVEDP) (and left atrial pressure), although total pulmonary resistance (TPR) rises, pulmonary arteriolar resistance (PAR) falls, so that elevation of left atrial pressure comes to account for the major resistance between the pulmonary artery across the lungs to the left heart.

The effect of scar before and after superimposed acute myocardial infarction is indicated in Table 3. In all dogs, after death, a posterolateral transmural scar was found as a result of the previous surgery. Except for dog 1, where a marked tachycardia was present in the control state, and dog 7, where marked heartworm infestation was later found to be present, PAEDP, left atrial (LAP), and pulmonary capillary wedge pressures correlated well with one another. Before acute ischaemia, in the presence of these small scars (less than 10% of LV by weight) and generally low LVEDP levels, close correlation of PAEDP-LAP-LVEDP was found. After acute ischaemia, however, with marked elevations in LVEDP in four dogs, large atrial impact waves were observed and LVEDP was markedly greater than LAP or PAEDP in three (dogs 4, 6, and 7). Shock did not occur in these dogs.

Discussion
Since knowledge of left atrial pressure (LAP) and left ventricular end-diastolic pressure...
(LVEDP) would be of help in evaluating patients with acute myocardial infarction (AMI) and since, with or without shock, blood volume expansion may have beneficial effects on cardiac performance and left ventricular function (Cohn, Luria, Daddario, and Tristani, 1967; Langsjoen, Sanchez, Lynch, and Inmon, 1968; Loeb, Pietras, Tobin, and Gunnar, 1969; Russell, Rackley, Pombo, Hunt, Potanin, and Dodge, 1970; Ratshin, Rackley, and Russell, 1972), simple right heart pressure measurements predictive of LAP and LVEDP would be of great value.

In normal man mean LAP and LVEDP are nearly identical (Braunwald and Frahm, 1961) and our limited data in dogs suggests a similar relationship. However, in the presence of a chronically diseased left ventricle, Braunwald and Frahm also demonstrated that, due to the presence of a prominent atrial impact wave, the LVEDP was considerably higher than mean LAP. However, in only two of these 26 patients was the underlying left ventricular abnormality related to coronary artery disease. Using the pulmonary capillary wedge pressure to estimate LAP Falicov and Resnekov (1970) demonstrated similar findings in normals versus patients with chronic left ventricular disease, including 14 patients with coronary heart disease in the latter group. No such comparisons have been performed in man with AMI to our knowledge but our experimental results indicate that, in a previously normal left ventricle, AMI does not disrupt the normal LAP-LVEDP relationship. The small scars we produced experimentally did not result in striking elevations in LVEDP or appreciable LAP-LVEDP differences, although with reinfarction and higher LVEDP such was the case. Larger scars which may be present in man may, however, commonly cause such differences (Falicov and Resnekov, 1970). Similarly, although in our study the LAP-LVEDP relationship was not disrupted in cardiogenic shock, the frequent presence of large areas of scar in the clinical condition (Page, Caulfield, Kastor, DeSanctis, and Sanders, 1971) would caution against direct translation of these results to man.

In the absence of pulmonary vascular obstruction or mitral stenosis Kaltman et al. (1966) found that pulmonary artery end-diastolic pressure (PAEDP), LAP and LVEDP tended to reach a common level. Similar correlations were found by Jenkins and associates (Jenkins, Bradley, and Branthwaite, 1970) who compared PAEDP and LAP. Although both groups considered the possibility that, with tachycardia, sufficient time might not be allowed for the PAEDP to fall to the LAP level, the heart rates found in their patients were well below those in our dog studies where this probably was a factor accounting for the PAEDP-LVEDP discrepancies in the control state. After elevation of LVEDP and LAP to clearly abnormal levels, however, this tendency was overcome by a fall in pulmonary arteriolar resistance, apparently allowing for greater ease in transmission of left atrial pressures through the lungs with PAEDP-LAP-LVEDP equivalence despite tachycardia. This effect of acutely increased LAP resulting in decreased pulmonary arteriolar resistance has been previously described in dogs (Borst, McGregor, Whittenberger, and Berglund, 1956; Forsberg, 1971) and cats (Carlill and Duke, 1956).

Our study indicates that the tendency of these three pressures to reach a common level persists after AMI in a previously normal left ventricle. The PAEDP and LAP relationship (as derived from pulmonary capillary wedge pressures) apparently also persists in human AMI whether or not an infarct has occurred previously (Lassers, George, Anderton, Higgins, and Philp, 1970). Our experimental data suggest, however, that in the presence of a previous scar the LVEDP-LAP relationship may be altered. This hypothesis is supported by discrepancies reported between PAEDP and LVEDP in acute human myocardial infarction (Lal et al., 1971) and is not surprising in view of the previously reported LVEDP-LAP discrepancies (Braunwald and Frahm, 1961; Falicov and Resnekov, 1970) and LVEDP-PAEDP discrepancies (Bouchard, Gault, and Ross, 1971) in patients with chronic left ventricular disease.

In summary, the picture that emerges from this and other investigations is illustrated diagrammatically in Fig. 5. In the normal heart PAEDP, LAP and LVEDP tend to reach a common level. This is altered in the presence of pulmonary vascular obstruction when PAEDP will be greater than the other two, and in the presence of mitral stenosis when LAP will
Under normal conditions, pulmonary artery end-diastolic (PAEDP) left atrial (LA), and left-ventricular end-diastolic (LVEDP) pressures tend to reach a common level. This relationship persists after acute myocardial infarction of a normal left ventricle but not necessarily with a previously diseased one wherein prominent atrial impact waves may raise LVEDP appreciably above LAP and PAEDP both before and after acute infarction. The right atrial (RA) pressure in all instances is a poor predictor of left heart pressures.

In the presence of a chronically diseased left ventricle LVEDP will tend to be higher than both LAP and PAEDP.

The practical aspects of interpreting right heart pressures in AMI would appear to be as follows:

Right atrial pressure is a poor predictor of either LAP or LVEDP and may even be within the normal range while the latter are at pulmonary oedema levels.

In initial acute myocardial infarction PAEDP will probably accurately indicate the level of both LAP and LVEDP.

With pulmonary vascular obstruction PAEDP will be greater than LAP and LVEDP. Therefore, because of its ease in performance, determination of ‘pc wedge’ pressure by inflating a Swan-Ganz balloon catheter in a pulmonary artery branch should be done to detect PAEDP/ LAP (pc wedge) discrepancies.

When acute myocardial infarction occurs in the presence of a previously diseased left ventricle, LVEDP may be appreciably greater than LAP and PAEDP. In such cases, monitoring of PAEDP or ‘pc wedge’ pressures may be useful in anticipating the threat of pulmonary oedema and judging effects of therapy in lowering LAP, but will be of doubtful assistance in judging LVEDP. When left ventricular function is to be evaluated, actual LVEDP pressures, or preferably volume, should be measured.

In the presence of severe tachycardia or grossly irregular heart rhythms the ability to use PAEDP effectively as a predictor of left sided pressures may be impaired.

The authors gratefully acknowledge the technical assistance of Miss Katherine Kearney, the statistical assistance of Mrs. Jean Chou, and the secretarial services of Miss Evelyn Vantuono. Cardio-green used in this study was generously supplied by Hynson, Westcott, and Dunning, Inc.

References


