Dynamic Responses of the Right Ventricle following Extensive Damage by Cauterization

By Abraham Kagan, M.D.

Previous observations that major destruction of the right ventricular walls causes but slight changes in venous and arterial pressures were confirmed. Furthermore, additional observations indicated that the compensatory reaction of the damaged ventricle to stress is diminished surprisingly little. The results are considered evidence that the cauterization procedure and others of a similar nature do not produce right heart failure and, therefore, would not be expected to cause venous congestion. The subsidiary muscular mechanism which maintains the right ventricular pump remains enigmatic; but evidence is submitted that the damaged right ventricle is regulated independently of the left.

Reinvestigations of apparently well established functions occasionally reveal that generally accepted concepts require amendment or drastic revision. This has recently been the case following studies on right ventricular function. In 1943, Starr and his associates, who were interested in problems of right heart failure, discovered that almost complete thermal destruction of the right ventricle in dogs eventuated in no appreciable elevation of venous or fall of arterial pressures. The complementary studies of Rodbard and Wagner, who primarily stressed low pulmonary arterial resistance, showed that the driving force of right ventricular contraction is not necessary for maintenance of an adequate blood flow to the left atrium. However, the studies of Bakos clearly indicated that the pulmonary arterial pressures and, by inference, the stroke volumes of the right ventricle are not significantly reduced after almost complete inactivation of the right ventricular walls by cauterization. Two inferences might be allowable:

1. Under resting conditions, extensive destruction of the entire right ventricular walls does not lead to right heart failure, a fact of considerable clinical importance.
2. The force expelling blood into the pulmonary circuit can be derived from sources other than contraction of the external walls of the right ventricle, a fact of great physiologic interest. The corollaries do not necessarily follow that contractions of the right ventricular walls are unimportant normally, or that increased vigor of their contractions does not contribute to augmented stroke volumes.

The great importance of the problems presented warranted further experimental studies, first of all, to substantiate the conclusion of previous workers that the right ventricle can maintain essentially normal propulsive force after nearly complete destruction of the external walls, second, to elucidate the subsidiary mechanisms by which essentially normal stroke volumes are possible and, finally, to test how the reaction of such a damaged right ventricle to stress differs from that of a normal one. The present investigation concerned itself with these problems.

Methods
Mongrel dogs weighing between 10 and 20 Kg. were anesthetized with morphine sulfate, 5 mg. per kilogram subcutaneously, and sodium barbital, about 200 mg. per kilogram intravenously. The chest was opened by a midsternal incision, and mild pulmonary ventilation was maintained by an inter-
mittent positive-pressure respirator. The pericar-dium was slit longitudinally and the heart was supported in a pericardial cradle without impeding right atrial filling.

Aortic blood pressure was recorded optically by means of a rigid cannula inserted via the left common carotid artery until its tip just reached the arch of the aorta. The cannula was connected through a short length of lead tubing to a modified Gregg manometer. Mean femoral venous pressure was measured by periodic reading of a saline manometer connected to a femoral vein. Phasic changes in right atrial and right ventricular pressures were recorded optically by rigid cannulas connected by short lengths of lead tubing to Gregg manometers of appropriate frequency and sensitivity. All measurements were made using the level of entrance of the superior vena cava into the right atrium as the zero reference level. In a few experiments movements of the right ventricular wall were recorded optically by a Frank segment capsule connected to a myocardiograph which was sutured to the right ventricular wall. (For technic see Tennant and Wiggers.)

In all experiments the entire free right ventricular wall was damaged as extensively as seemed consistent with its continued integrity by repeated applications of the tip of a Geiger electrocautery unit. The desired degree of damage was achieved in approximately one hour. Arrhythmia could generally be avoided by increasing the temperature of the cautery tip gradually from one just enough to blanch the epicardium to a red heat which caused charring. When, as frequently happened, a coronary vessel was severed by the cautery tip, hemostasis was achieved either by further cauteryization or, in the case of larger vessels, by ligation. When cauteryization was complete the appearance of the heart was identical with that illustrated in a previous article by Bakos. Postmortem examinations revealed destruction of the entire right ventricular wall with the exception of variable patches on the endocardial layers. It was conservatively estimated that between 70 and 85 per cent of the entire wall was certainly destroyed in all successful experiments. In order to maintain an adequate venous return during this traumatic procedure a constant drip of normal saline was maintained through a femoral venous cannula at a rate of approximately 2 cc. per minute for the duration of the experiment.

In studying the response of the right ventricle to stress it was necessary to compare in the same animal the reactions of the damaged ventricle with those of the normal ventricle previous to cauteryization. Initial experiences indicated that, for this purpose, the reactions to increasing venous input were difficult to quantitate. Consequently, the response to equivalent stresses of increasing pulmonary resistance was used. A screw clamp, so calibrated that it required 20 complete turns of a small wheel to go from a wide open to a closed position, was placed around the pulmonary artery just distal to the pulmonary valves. This clamp began to compress the artery at about eight turns and caused complete obstruction at about 18 turns of the wheel, depending on the size of the pulmonary artery. Progressive graded pulmonary artery occlusion to the point of frank decompensation was imposed in rigidly similar fashion both before and after traumatization of the right ventricle.

**Results**

Hemodynamic Effects. Fourteen technically successful experiments furnish the basis of this report. The effects of progressive cauteryization on aortic and right ventricular pressure pulses are shown by segments of tracings in figure 1. Data of hemodynamic changes in another experiment are shown graphically in figure 2. The results of previous investigators were essentially substantiated. Progressive cauteryization does not significantly alter the development of pressure in the right ventricle; the small changes observed are within the range of variation seen over several hours in normal hearts. Aortic pressures are reduced moderately at first, but recover even while cauteryization is in progress. Femoral venous and right atrial pressure show very insignificant changes.

In table 1 are recorded the data from 12 experiments which furnished adequate data before and after cauteryization. Thermal destruction of the right ventricle resulted in very small changes in aortic pressure. The average fall in systolic pressure was 9 mm. Hg, the changes ranging from -34 to +15 mm. Hg. Diastolic pressure fell an average of 10 mm. Hg, the changes ranging from -36 to +20 mm. Hg. Mean femoral venous pressure rose an average of 12 mm. saline with a range of -8 to +40 mm. The average rise in mean right atrial pressure was 16 mm. saline, with a range of 0 to +45 mm. Right ventricular diastolic pressure rose an average of 1.9 mm. Hg (27.1 mm. saline) with a range of 0 to +4 mm. Hg; while right ventricular systolic pressure increased an average of 2 mm. Hg with a range of -3 to +6 mm. Hg. These results on femoral venous and aortic pressures are also in substantial agreement with those reported by Starr, although the range was somewhat wider in this larger series.
Myographic Changes in the Right Ventricle. 
In order to determine whether the right ventricular wall was contracting normally or, as it appeared visually, was expanding during systole after cauterization, simultaneous records of aortic pressure and of movements of the right ventricular wall were taken in five experiments.

Figure A-B. The curve showing the pressure and tension in the right ventricle, as indicated by the drop of the curve at A-B. The lengthening is maintained until the end of systole, C, after which the fibers shorten, as indicated by the rise of the curve C-D. The typical shortening of the right ventricular wall seen in record A is converted to expansion after cauterization (B).

Reactions to Stress of Increased Pulmonary Resistance. The responses of the right ventricle to equivalent increases of pulmonary resistance were studied in seven dogs. The curves reproduced in figure 4 show the nature of the responses before and after right ventricular damage. The control aortic pressure pulse (A) is of good contour with a diastolic pressure of 110, systolic pressure 138, and incisural pressure 125 mm. Hg. In a cycle length of .43 second the duration of systole is .24 second, giving an S/C ratio of .558. The corresponding right ventricular curve is of normal contour. The initial tension is 3 mm. Hg; the maximum pressure is 27 mm. Hg; and the S/C ratio is .535.

At 14 turns of the clamp wheel (B) there is no significant change in the aortic curve, diastolic pressure measuring 112, systolic 140, and incisural pressure 127 mm. Hg; the S/C ratio is .55. Initial tension in the right ventricle has now risen to 5 mm. Hg, and the maximum pressure generated is 48 mm. Hg. The S/C
ratio is .55. At 15 turns (C) aortic pressure is still well maintained, the respective pressures aortic pressures measuring 108 mm. diastolic, 132 systolic, and 121 mm. Hg incisural pres-

Table 1. Pressure Changes following Right Ventricular Cauterization

<table>
<thead>
<tr>
<th>Dog</th>
<th>Estimated per cent Burn</th>
<th>Heart Rate</th>
<th>Femoral Venous Pressure (mean) (mm. saline)</th>
<th>Right Atrial Pressure (mean) (mm. saline)</th>
<th>Right Ventricular Pressure (mm. Hg)</th>
<th>Aortic Pressure (mm. Hg)</th>
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<tbody>
<tr>
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<td>Before/After Before/After Before/After</td>
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<tr>
<td>6</td>
<td>60</td>
<td>139/132</td>
<td>20/30 10/28</td>
<td>50/70</td>
<td>60/52</td>
<td>26/35</td>
</tr>
<tr>
<td>9</td>
<td>60-70</td>
<td>165/161</td>
<td>70/110 50/95</td>
<td>48/48</td>
<td>50/60</td>
<td>25/35</td>
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<tr>
<td>10</td>
<td>90</td>
<td>226/214</td>
<td>60/52 48/48</td>
<td>130/106</td>
<td>100/70</td>
<td>105/70</td>
</tr>
<tr>
<td>11</td>
<td>75</td>
<td>221/183</td>
<td>60/60 50/52</td>
<td>100/82</td>
<td>108/90</td>
<td>116/97</td>
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<tr>
<td>16</td>
<td>75-80</td>
<td>174/174</td>
<td>60/60 50/52</td>
<td>132/127</td>
<td>139/113</td>
<td>132/108</td>
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<tr>
<td>18</td>
<td>75-80</td>
<td>183/175</td>
<td>75/100 50/65</td>
<td>26/35</td>
<td>20/24</td>
<td>143/114</td>
</tr>
<tr>
<td>23</td>
<td>80-90</td>
<td>196/181</td>
<td>36/2 34/2</td>
<td>133/104</td>
<td>120/90</td>
<td>139/115</td>
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<tr>
<td>24</td>
<td>80-90</td>
<td>150/173</td>
<td>36/2 34/2</td>
<td>133/104</td>
<td>120/90</td>
<td>139/115</td>
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<tr>
<td>25</td>
<td>75-85</td>
<td>204/214</td>
<td>36/2 34/2</td>
<td>133/104</td>
<td>120/90</td>
<td>139/115</td>
</tr>
</tbody>
</table>

Average.............. | 50 to 90 | 181/182 | 57/69 39/55 | 24/1.7 29/0 | 100/101 | 119/91 |

Average change after burn.............. | -1 | +12 | +16 | 2.19 | -9/10 |

Range...............| -38 to +49 | -18 to +40 | 0 to +40 | -3/0 to 6/0 | -34/20 to 15/36 |

measuring diastolic, 107; systolic 128, and incisural 116 mm. Hg. The S/C ratio is now .538. In the right ventricle, although initial tension has remained at 5 mm. Hg, the maximum pressure has now reached 60 mm. Hg. The S/C ratio here, too, is now .538, which indicates an abbreviation of the contraction process. At 16 turns (D) pulsat alternans is noted in both curves. Aortic pressure has fallen to 82/68 mm. Hg, with incisural pressure 70 mm. Hg; and the S/C ratio is .516. The right ventricle shows its peak compensation with a pressure maximum of 78 after an initial tension of 7 mm. Hg: S/C ratio is .516, indicating further abridgement of systole. At 16½ turns (E) aortic pressures are diastolic 18, systolic 32, and incisural 20 mm. Hg; the S/C ratio is .485. Right ventricular initial tension is 14, and the maximum pressure is 34 mm. Hg; S/C ratio is .457; i.e., systole continues to shorten. Fifteen minutes after the release of the pulmonary constriction (F), the pressure pulse curves have returned to normal configurations, sure. The S/C ratio is now .55. The right ventricular curve, too, has returned to normal with
a pressure of 23/3 mm. Hg, and the S/C ratio is raised to .60.

the curves of figure 1. Curves G to L in figure 4 show the responses to the second applica-

Fig. 4. Records showing effects of progressive increases of pulmonary arterial resistance on aortic pressure (upper curves) and right ventricular pressure (lower curves). Segments A–F, reactions of normal heart; segments G–L, reactions after cauterization. Discussion in text.

Following this test the right ventricle was cauterized. The detailed changes in the pressure pulses during cauterization are shown in

tion of progressive pulmonary constriction, 15 minutes after the completion of cauterization. The control aortic pressure curve (G) is relatively unchanged in contour, the pressures measuring diastolic 102, systolic 130, and incisural 116 mm. Hg; the S/C ratio is .53. The right ventricular curve shows the earlier systolic peak at 28 after an initial tension of 6 mm. Hg; the S/C ratio is .53. At 14 turns of the wheel (H), the aortic pulse contour remains normal, the pressures, however, having fallen to diastolic 78, systolic 102, and incisural 87 mm. Hg; the S/C ratio is .50. Right ventricular initial tension is 10 mm. Hg; systolic pressure is 30 mm. Hg; and the S/C ratio is .555. At 15 turns (I), aortic pressures have fallen to diastolic 38, systolic 60, and incisural 47 mm. Hg; the right ventricle has reached its peak response at a pressure of 55/15 mm. Hg; and the S/C ratio for both chambers is reduced to

Fig. 5. Plot of data showing effects of equivalent progressive increase in pulmonary constriction in a normal heart and after cauterization of the right ventricle. AS, aortic systolic pressure; AD, aortic diastolic pressure; VS, right ventricular systolic pressure; DS, right ventricular diastolic pressure (initial tension). Discussion in text.
At 16 turns (J) the aortic pressure readings are diastolic 18, systolic 34, and incisural 25 mm. Hg; the S/C ratio is .364. Right ventricular initial tension is 16, maximal tension 40 mm. Hg; its S/C ratio declines to .318. At 164 turns (K) at the height of decompensation the aortic pressures are diastolic 11, systolic 30, and incisural 21 mm. Hg; aortic S/C ratio has now fallen to .155. The right ventricular pressure is 33/19 mm. Hg; its S/C ratio is .136. The greater reduction in relative duration of systole after injury is obvious. 

Fifteen minutes after the release of pulmonary constriction (L), the pulse pressure curves have returned to preconstriction contours and measurements, the aortic pressures measuring diastolic 102, systolic 133, and incisural 120 mm. Hg; the right ventricular pressures measuring 30/5 mm. Hg. The S/C ratios are .487, that is, materially less than before the application of stress.

Comparison of aortic and right ventricular pressure pulses and S/C ratios at equivalent degrees of pulmonary resistance show that in this experiment the ability of the right ventricle to compensate was significantly reduced. Thus, in the normal ventricle aortic pressure was maintained in segment B through increase in left ventricular pressure, but fell considerably after cauterization, as shown in segment H. Again in segment C, the significant elevation of right ventricular pressure sufficed to reduce aortic pressure only to a slight degree, whereas in segment I, no further elevation of right ventricular pressure took place despite a marked elevation of diastolic pressure. The differential effects become very striking in comparison of segments D and J. Also, in the normal condition right ventricular pressure continued to rise until 16 turns of the wheel in segment D, whereas after cauterization no further increase took place with 15 turns of the wheel in segment I.

However, in several experiments the compensatory response of the damaged ventricle corresponded more nearly to the preceding response of the normal ventricle. Thus, in the experiment illustrated graphically in figure 5, aortic pressures both before and after cauterization began to decline after 12 turns of the clamp wheel, and the maximal elevation of right ventricular pressure took place with 14 turns of the wheel. The possibility that more viable muscle was present in the outer walls in these instances cannot be entirely excluded, despite careful postmortem checks of the damage. Nevertheless, the fact that nearly complete compensation was still possible after very extensive damage does raise the question as to the extent that muscular contractions of the free wall contribute to compensatory states in the normal heart.

**Discussion**

The results of previous investigators and their extension in this study, in a way, raise more questions than they answer. However, they give a clue as to possible faults in our methods of studying cardiac failure and may serve as a directive to future approaches.

The original observations of Starr and his associates that almost complete destruction of the right ventricular walls has little effect on arterial and peripheral venous pressures have again been completely confirmed. In addition, the increase in right atrial pressure and initial tension in the right ventricle, though somewhat more definite, is still of a low order of magnitude.

Starr and his associates used these observations as evidence that right heart failure, per se, cannot cause marked venous congestion. It becomes apparent from the studies of Bakos and the present observations that the procedure does not produce right heart failure, because destruction of the walls of the right ventricle spares a hidden subsidiary mechanism by which effective stroke volumes can be maintained through periodic elevation of right ventricular pressure. Consequently, it is highly doubtful whether the problem of right heart failure can be solved by any experimental procedure which inactivates or destroys only the outer walls of the right ventricle. The reason that experimental myocardial depression produced by chemical agents is generally associated with considerable elevation of central and peripheral venous pressures, whereas destruction of the right ventricular walls is not, may conceivably be found in the fact that the former also de-
presses a subsidiary contractile mechanism, while the latter procedure spares it.

Such considerations emphasize the importance of localizing the subsidiary contractile mechanism which is still able to elevate pressures in the right ventricle and pulmonary artery after destruction of the major portion of the right ventricular walls. Bakos' made the reasonable suggestion that contractile energy may be produced through a pull of ventricular muscle bundles upon inactive bands in the right ventricle, with which they are continuous. The idea was based on the architecture of the sinospiral and bulbospiral fibers which envelope the two ventricles. According to such a view, the pull exerted upon inactive right ventricular fibers during left ventricular systole should cause a shortening of the outer wall. However, myographic studies have shown that this does not occur; the right ventricular wall elongates during systole as a result of the elevation of intraventricular pressure created elsewhere. Furthermore, the concept implies that the systolic elevations of right ventricular pressure accord with the force of left ventricular contractions. This, however, is not the case; on the contrary, inverse relations occur when the right ventricle contracts against increased resistance. For example, in segments $H$ and $I$ of figure 4, the decline of aortic pressure and reduction of pulse pressure denotes diminished force of left ventricular contraction, while the right ventricular pressure reaches higher systolic levels. In other words, the activity of the damaged right ventricle is increased, while that of the normal left ventricle is reduced. Obviously, the activity of the two ventricles was maintained by separate mechanisms.

As an alternate mechanism, it could be suggested that the elevation of right ventricular pressure is produced through contraction of the interventricular septum, by shortening of the apex-base axis, by bulging into the right ventricle, or by a combination of these two effects. The results of the experiments presented are consistent with such an explanation only if we assume that septal contraction affects pressure in the right ventricular cavity, but not that in the left. Such a result is not impossible on an anatomic basis, but speculations are unwise. Experimental study of the behavior of the septum and its dynamic effects remains a challenge for future investigators.

**Summary**

1. The entire outer wall of the right ventricle of dogs was damaged as severely as possible by means of an electrothermocautery. This procedure caused a temporary slight fall in aortic pressure and a slight rise in peripheral and central venous pressures, and in right ventricular diastolic pressure during cauterization. The changes following 70 to 85 per cent destruction of the outer walls were of so low an order of magnitude as to lead to the conclusion that a normal, contractile right ventricular wall is not necessary for the maintenance of a normal circulation.

2. The reserve capacity of the damaged right ventricle was tested by the imposition of progressive, graded constriction of the pulmonary artery before and after thermal destruction of the outer right ventricular wall. The response to such imposed stress was impaired less than would be anticipated, indicating that even the reserve capacity of the right ventricle is not always reduced significantly by destruction of the external ventricular wall.

3. The results of this and other investigations indicate that right ventricular failure and venous congestion cannot be produced by extensive destruction of the outer ventricular walls, because a hidden mechanism is spared which is able to maintain approximately normal stroke volumes. The corollary follows that the dynamic effects of right heart failure cannot be studied by experimental procedures which inactivate or destroy only the outer walls of the right ventricle.

4. The subsidiary contractile mechanism which maintains efficient pumping action of the right ventricle after destruction of its outer walls has not been localized. The suggestion that the energy is derived from a pull of left ventricular muscle bands upon continuous but inactive bands of the right ventricle was not supported by experimental evidence. The sug-
gestion that contraction of the interventricular septum may be concerned still requires experimental validation.

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References


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