Dynamics of Complete Right Ventricular Failure in Dogs Maintained with an Extracorporeal Left Ventricle

By John C. Rose, M.D., Eric J. Lazaro, M.D. and Herbert P. Broida, Ph.D.

By replacing the left ventricle of a dog with an extracorporeal pump, complete failure of the right ventricle (e.g., fibrillation) caused marked elevation of central venous pressure, the degree of elevation being a linear function of left ventricular output. In addition, expansion of the intravascular volume was required in order to maintain circulation by the left ventricle alone. Edema formation was accentuated by the hypervolemia and venous hypertension.

According to the classical “backward failure” theory of heart failure, elevation of central venous pressure is due to “back pressure” behind an incompetent right ventricle. This concept was supported by Starling’s experiments with the heart-lung preparation. Edema was thought to result from increased transudation of fluid into the tissues secondary to a rise in capillary blood pressure.

Warren and Stead pointed out some inconsistencies between these concepts and clinical observations made in certain patients with congestive heart failure—chiefly that weight gain, signifying an accumulation of extracellular fluid, may precede an elevation of venous pressure during the development of the syndrome. Starr, Jeffers and Meade cauterized the myocardium of the right ventricle in dogs, and found that no significant rise in venous pressure occurred despite evident impairment of the pumping action of the ventricle. Despite evidence that destruction of right ventricular function by this method was incomplete, this result leads to the conclusion that incompetence of the right ventricle does not produce venous hypertension. These studies favored the concept of “forward failure.”

Study of the right ventricular pump is difficult in the intact circulation; the anatomic union of the ventricles prevents the experimental production of failure of either one. Replacement of the left ventricle with an extracorporeal mechanical pump has provided a new experimental approach to the study of right ventricular function. If the output of the mechanical left ventricle remains constant under varying conditions, changes in the total circulation of the dog resulting from alterations in right ventricular function cannot be ascribed in any part to alteration in the function of the unchanging left ventricle. In this experimental preparation, the peripheral circulation and its environment are intact. It is thus a less simplified scheme than the heart-lung preparation, in which an artificial resistance was substituted for the peripheral circulation.

Despite the recognized limitations of this experiment, certain analogies can be drawn between failure of the right ventricle in patients and in this experimental preparation. Observations have been made of the immediate effects of total failure of the right ventricle with constant left ventricular output; hemodynamic responses to changes in left ventricular output in dogs without functioning right ventricles; and the relationship of hypervolemia and edema formation to right ventricular failure.

Materials and Methods

Dogs weighing between 12 and 25 Kg. were anesthetized with sodium pentobarbital, 25 mg./Kg. given intravenously. Each dog was maintained with 100 percent oxygen by intermittent endotracheal positive pressure. The mechanical left ventricular pump...
pump, and the operative procedure by which the extracorporeal connections were made have been described in detail in a previous communication.\(^3\)

The rate of the pump could be varied at will through a range of from 35 to 280 strokes per minute, independently of stroke volume. Stroke volume could be varied from zero to 180 cc. per stroke. The operating output range of the pump was between 200 and 8,000 cc per minute. Output remained perfectly constant when high resistances were applied to the output tubing.

Blood was drained passively, aided by gravity, from the left atrial appendage via \(\frac{3}{4}\) inch I.D. Tygon tubing. It was conducted to a plastic reservoir, the volume of which varied between 250 and 1,500 cc. The reservoir level was constantly recorded. From the reservoir, blood was pumped, via an air-trap, to a T-tube placed in the descending thoracic aorta which permitted flow both proximally and distally. The right ventricle continued to function effectively, keeping pace with the output of the pump.\(^6\) Knowing the pump output, alterations in the right ventricular output could be measured by the rate of rise or fall of the reservoir level. Methods used for recording intravascular pressures in these experiments have also been described.\(^3\)

The pump and reservoir were primed prior to each experiment with mixtures of 5 percent gelatin in normal saline (Plasmoid\(^*\)) and blood. In experiments in which red blood cell mass and plasma volume were measured, the priming fluid was whole blood. Dog blood was obtained within the week prior to each experiment and stored cold in standard vacuum bottles containing ACD solution. Splenectomy was seen to dissect among the muscle bundles of the heart and the easiest and most efficient method was to inject 5 to 6 cc of air directly into the myocardium. Air was seen to dissect among the muscle bundles of the heart and ventricular fibrillation usually ensued within one minute.

Red blood cell mass and plasma volume were determined in the following manner: 15 cc of blood from the animal to be used in the experiment were refrigerated overnight in a bottle containing 5 cc ACD solution and approximately 300 microcuries of Cr\(^{51}\) (Rachromate). The bottle was then centrifuged and the superantum plasma removed. Red blood cell washing with normal saline was repeated at least three times. Saline solution and about 40 microcuries of radioiodinated (I\(^{125}\)) albumin (RISA) were then added to make a total mixture of about 20 cc. An aliquot of the suspension was saved for determination of red blood cell and plasma radioactivity and for hematocrit.

Measurements were made by injecting 2 cc aliquots of the well-mixed suspension intravenously in the intact dog, and into the reservoir in the pump preparation. Blood samples were collected directly from the left atrium at 10, 15 and 20 minute intervals after injection. Samples were collected at 10, 5 and zero minutes prior to injection when a previous volume measurement had been made, in order to correct for disappearance of red cell and plasma radioactivity.

Hematocrit readings were obtained for each sample. One cc plasma and the washed red blood cells from 2 cc whole blood were counted in a scintillation well-type counter. Emission counts per minute per cc plasma and per cc red blood cells were plotted on semilogarithmic paper against time for volume calculations.

The reservoir level reading for the time of each sample collection made possible the calculation of the total intravascular volume exclusive of the extracorporeal blood. For each experiment, the total volume and hematocrit reading of the primer solution was carefully measured, as well as the volume of gelatin or saline solution added when reservoir depletion occurred.

**RESULTS**

*Plasmoid was generously supplied by Joseph P. Webb, M.D., Medical Director, The Upjohn Company, Kalamazoo, Mich.*

**The Immediate Hemodynamic Effects of Complete Right Ventricular Failure.** Figure 1 shows an example of the consistently observed response to the induction of acute ventricular fibrillation in dogs maintained on the mechanical left ventricle. Ventricular fibrillation did not occur immediately with the injection of air into the myocardium but the heart beat became ineffective at that moment. There was little or no change in systemic arterial pressure. Arterial pressure appeared to be almost exclusively a function of left ventricular (pump) output. (In a few instances, transient rises or falls in systemic arterial pressure occurred that were evidently reflex in origin.)

Pulmonary arterial pressure fell precipitously when the beat became ineffective. Falling to nearly zero mm. Hg, it began to rise as the central venous pressure rose.

Simultaneous with the loss of effective con-
traction of the right ventricle, the pressure in the central veins rose sharply, and the pump reservoir level fell. Blood continued to pour into the venous system where it was not emptied by the right ventricle. The right ventricle became tense and distended, and the pattern of tricuspid insufficiency appeared in the venous pressure contour as the heart dilated. This promptly disappeared with the onset of ventricular fibrillation.

In a variable period of time (from 1 to 5 minutes), as equilibrium was reached; the venous pressure was stable at a high level and the animal no longer took up fluid at that particular constant left ventricular output. In 10 experiments, the central venous pressure rose from a mean pre-fibrillation level of 4.6 mm. Hg to a mean post-fibrillation equilibrium level of 23.5 mm. Hg (Mean rise 18.9 mm. Hg, standard deviation ±5.4, range 10 to 26 mm. Hg).

The mean fluid uptake in this same group of experiments was 1,606 cc. (range 700 to 2,000 cc.). The “left ventricular” pump output was maintained at a level between 1,400 and 2,200 cc. per minute, constant in each individual experiment.

After the equilibrium was reached, at which the total circulation was maintained by the mechanical left ventricle, the venous pressure exceeded the pulmonary arterial pressure. Data on this veins-to-lungs pressure gradient are given below.

The same alterations were noted when the heart failed spontaneously without attempts to produce ventricular fibrillation. In experiments in which the right ventricle failed suddenly and spontaneously, the electrocardiogram remained essentially unchanged as the right ventricular end-diastolic and venous pressures rose. The heart dilated and the reservoir level fell. Twenty to 30 minutes later, ventricular fibrillation was induced and no significant alterations in venous or right ventricular pressure were noted except for cessation of a weak pulse which had persisted and which was due to ineffective ventricular contractions.

The Hemodynamic Effects of Altering “Left Ventricular” Pump Output in Dogs with Ventricular Fibrillation. When equilibrium was established with a constant venous pressure and reservoir level, the output of the mechanical pump was increased and decreased in a stepwise fashion in a range between 800 and 4,000 cc. per minute. The resulting aortic and pulmonary arterial pressures obtained in six such experiments are shown in figure 2. Pressure in all parts of the vascular system were found to be functions of pump output.

Figure 3 shows the central venous pressure...
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INCREASING PUMP OUTPUTS

DECREASING PUMP OUTPUTS

Fig. 2. Chart showing alterations in mean aortic and mean pulmonary arterial pressures in response to increasing then decreasing pump outputs in dogs with ventricular fibrillation. (Systemic arterial pressures for decreasing outputs in dog 50 are not charted because of technical failure in the recording apparatus.) Semilogarithmic plot.

Fig. 3. Chart showing fluid uptake and central venous pressures in the same experiments charted in figure 2. For comparison, central venous pressures for three animals with adequately functioning right ventricles, taken from a previous communication, are also charted. See text for discussion.

alterations in these six experiments. The venous pressures in the fibrillating animals are compared to venous pressures obtained in similar experiments on dogs with intact right ven-

Fig. 4. Chart showing fluid compartments measured in dog 59. Edema volumes are calculated by subtracting volumes measured by isotope dilution from volumes expected by known additions of fluid to the experimental system. See text for further discussion.
### TABLE 1.—Data Obtained in Seven Pump Experiments in which Plasma Volume and Red Blood Cell Mass were Determined by Isotope Dilution

<table>
<thead>
<tr>
<th>Dog</th>
<th>Weight Kg</th>
<th>Experimental Condition</th>
<th>Intravascular Plasma Volume cc</th>
<th>Intravascular RBC mass cc</th>
<th>T.B. Hct.*</th>
<th>L.A. Hct.</th>
<th>Edema Volume cc</th>
<th>Venous Pressure mm. Hg</th>
<th>Fluid Added cc</th>
<th>E.V./F.A. %</th>
</tr>
</thead>
<tbody>
<tr>
<td>50</td>
<td>22</td>
<td>Intact</td>
<td>429</td>
<td>474</td>
<td>53/50</td>
<td>4</td>
<td>265</td>
<td>2650</td>
<td>(29)</td>
<td>10.1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>1422</td>
<td>507</td>
<td>27/33</td>
<td>25</td>
<td>19/19</td>
<td>32</td>
<td>G. 950</td>
<td>33.8</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>1471</td>
<td>458</td>
<td>24/25</td>
<td>32</td>
<td>19/19</td>
<td>1035</td>
<td>S. 1000</td>
<td>42.4</td>
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<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>1410</td>
<td>390</td>
<td>19/22</td>
<td>20</td>
<td>19/19</td>
<td>773</td>
<td>S. 1000</td>
<td>36.5</td>
</tr>
<tr>
<td>58</td>
<td>22</td>
<td>Intact</td>
<td>717</td>
<td>1013</td>
<td>59/56</td>
<td>2</td>
<td>19/19</td>
<td>1301</td>
<td>S. 1000</td>
<td>27.5</td>
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<tr>
<td></td>
<td></td>
<td>Pump On</td>
<td>727</td>
<td>181</td>
<td>19/19</td>
<td>6</td>
<td>19/19</td>
<td>135</td>
<td>G. 1850</td>
<td>5.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>840</td>
<td>298</td>
<td>20/20</td>
<td>16</td>
<td>19/19</td>
<td>773</td>
<td>S. 1000</td>
<td>27.5</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>1375</td>
<td>333</td>
<td>16/20</td>
<td>19</td>
<td>19/19</td>
<td>1301</td>
<td>S. 1000</td>
<td>36.5</td>
</tr>
<tr>
<td>59</td>
<td>20</td>
<td>Intact</td>
<td>755</td>
<td>580</td>
<td>43/49</td>
<td>4</td>
<td>19/19</td>
<td>293</td>
<td>6</td>
<td>11.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pump On</td>
<td>580</td>
<td>308</td>
<td>26/25</td>
<td>6</td>
<td>19/19</td>
<td>293</td>
<td>6</td>
<td>11.0</td>
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<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>1169</td>
<td>270</td>
<td>21/23</td>
<td>22</td>
<td>19/19</td>
<td>1171</td>
<td>G. 800</td>
<td>33.9</td>
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<tr>
<td>60</td>
<td>12</td>
<td>Intact</td>
<td>504</td>
<td>436</td>
<td>42/38</td>
<td>5</td>
<td>19/19</td>
<td>362</td>
<td>14</td>
<td>23.8</td>
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<tr>
<td></td>
<td></td>
<td>Pump On</td>
<td>570</td>
<td>496</td>
<td>46/41</td>
<td>14</td>
<td>19/19</td>
<td>362</td>
<td>14</td>
<td>23.8</td>
</tr>
<tr>
<td>61</td>
<td>24</td>
<td>Intact</td>
<td>630</td>
<td>711</td>
<td>53/58</td>
<td>6</td>
<td>19/19</td>
<td>362</td>
<td>14</td>
<td>23.8</td>
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<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>886</td>
<td>648</td>
<td>42/46</td>
<td>13</td>
<td>19/19</td>
<td>528</td>
<td>14</td>
<td>23.8</td>
</tr>
<tr>
<td>64</td>
<td>14</td>
<td>Intact</td>
<td>1278</td>
<td>666</td>
<td>35/35</td>
<td>13</td>
<td>19/19</td>
<td>1189</td>
<td>25</td>
<td>38.1</td>
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<tr>
<td></td>
<td></td>
<td>Pump On</td>
<td>540</td>
<td>276</td>
<td>34/39</td>
<td>6</td>
<td>19/19</td>
<td>464</td>
<td>12</td>
<td>36.0</td>
</tr>
<tr>
<td>65</td>
<td>14</td>
<td>Intact</td>
<td>460</td>
<td>317</td>
<td>40/41</td>
<td>12</td>
<td>19/19</td>
<td>1301</td>
<td>19</td>
<td>36.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>1156</td>
<td>478</td>
<td>29/34</td>
<td>22</td>
<td>19/19</td>
<td>485</td>
<td>22</td>
<td>21.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pump On</td>
<td>520</td>
<td>707</td>
<td>58/61</td>
<td>7</td>
<td>19/19</td>
<td>809</td>
<td>14</td>
<td>41.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Fibrillating</td>
<td>520</td>
<td>519</td>
<td>49/41</td>
<td>14</td>
<td>19/19</td>
<td>809</td>
<td>14</td>
<td>41.6</td>
</tr>
</tbody>
</table>


† P. = Volume of primer blood (Hematocrit of primer in parentheses); G. = gelatin; S. = saline.

‡ E. V./F. A. = Edema volume divided by total fluids added (primer plasma, gelatin or saline) up to the time of measurement.

In the six experiments charted in figure 3 normal saline was used as the replacement fluid as the reservoir level fell with flow increases. Fluid uptake and losses are plotted against pump output changes in the same figure. As outputs increased, the mean increase in the animals' fluid volumes was about 500 cc. per 1,000 cc./min. increase in pump output. As the outputs were decreased, despite return of all pressures to previous levels (figs. 2 and 3), the fluid taken into the experimental animal was not returned to the reservoir. This observation led to the study of plasma volume and the red blood cell mass, in order to determine how much of this fluid remained in the intravascular compartment.

Hematocrit Readings, Plasma Volume and Red Blood Cell Mass in Animals with Ventricular...
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![Chart showing alterations in plasma volume in seven dogs maintained on the mechanical left ventricle and after induction of ventricular fibrillation.](image)

**Fig. 5.** Chart showing alterations in plasma volume in seven dogs maintained on the mechanical left ventricle and after induction of ventricular fibrillation. Ordinates = cc.

**Fibrillation Maintained with the Mechanical Left Ventricle.** The data obtained in seven blood volume experiments are shown in table 1. Not shown in table 1 is the volume of blood in the extracorporeal system (the pump and the reservoir). This volume, read at the time of each measurement, was converted into volume of plasma and red blood cells, using simultaneous hematocrit values. Since the isotopes measured total circulating volumes, the extracorporeal plasma and red blood cell volumes were subtracted from these total volumes to arrive at values for intravascular plasma and red cell volumes.

The percentage of fluid added to the experimental system which was lost as edema fluid was calculated by adding the plasma volume of the intact dog to the plasma volume of the primer blood and all fluids added to the reservoir after the pump was turned on. This known volume was divided into the difference between it and the measured volume (intravascular plus extracorporeal) as determined by isotope dilution. A typical experiment to clarify these compartments and their measurement is charted in figure 4.

In several experiments a marked loss of circulating red blood cells was noted. There was no visible hemolysis in blood samples taken in any of these experiments. However, autopsy revealed marked splenomegaly. Since it is recognized that the dog spleen is capable of altering its volume markedly, splenectomy was done prior to the experiment in dogs 60, 61 and 65.

The total body hematocrit, based on the volume measurements, was compared to the hematocrit of left atrial blood as a check on the accuracy of volume measurements. This ratio ranged between 0.82 and 1.19 (mean 0.98).

Plasma volumes did not increase in the pump preparation before ventricular fibrillation. However, edema occurred in all of these animals, ranging between 135 and 809 cc, or from 5.3 to 41.6 percent of the total fluid added to the system. When ventricular fibrillation was induced, all plasma volumes showed great increases, simultaneous with marked increases in venous pressure (fig. 5). Edema volumes then increased to between 21.1 and 53.0 percent of the total added fluid (with the exception of the first measurement made in dog 56).

In relation to the total fluid added to the system, it is evident that the edema volume exceeded the plasma volume increase. Edema occurred in the pump preparation prior to an increase in plasma volume, in the absence of right ventricular failure. When the right ventricle was inactivated, the intravascular plasma volume rose and edema formation was accelerated. These later phenomena were associated with marked venous hypertension.

No pattern could be observed in these experiments. Proportions of fluid leaving and remaining within the vascular compartment were extremely variable. However, the reliability of the volume measurements is supported by the correlation between actual and calculated hematocrit readings, and by experiments in progress which indicate that no significant amount of radioiodinated albumin leaves the intravascular space in these preparations.

**DISCUSSION**

The most significant difference between the intact circulation and this experimental preparation was the extracorporeal reservoir into
which the left atrium passively drained. Acute discrepancies between right and left ventricular outputs were adjusted by adding to the reservoir when right ventricular output failed, so that the reservoir continued to supply blood for the constant left ventricular output. Thus, the effective blood volume was not reduced by the increased capacity of the venous system. Such a rapid compensation cannot occur acutely in the intact circulation.

The anatomic association of the right and left ventricles implies that when the force of contraction of one is diminished, the other ventricle may either supply energy for contraction to the damaged ventricle or may itself lose force of contraction. If this is true, the ventricles must be anatomically dissociated for the study of isolated ventricular failure.

Despite the observation of several authors 2-4 that the venous pressure of resting animals did not rise following destruction of the right ventricular myocardium, the experiments of Landis and co-workers 8 indicated that in similar animals, exercise produces a sustained elevation of venous pressure. In resting animals studied acutely, the competence of the heart must be reduced to nearly lethal levels before the central venous pressure rises significantly. The experiments presented here support the views of Landis and associates. The pump animal does not represent a resting animal since the left ventricular output is controlled and constant. Therefore, a constant load may be imposed on the right ventricle regardless of its ability to maintain an output equal to that of the left ventricle.

In pump animals with adequately functioning right ventricles, the venous pressure rises only slightly while the right ventricle may increase its output greatly. 6 On the other hand, maintenance of the output of a damaged or functionless right ventricle requires great increases in venous pressure as shown in the present experiments in which the output of the pump was increased and decreased.

The idea of the unessential nature of right ventricular function was supported by Rodbard and Wagner. 10 They created anastomoses between the right atrium and main pulmonary artery in dogs, then occluding the pulmonary artery proximal to the shunt. Despite the fact that these animals survived up to only one hour, they concluded that the venous pressure of the intact animal was sufficient to perfuse the low pressure pulmonary circulation with blood without benefit of the right ventricular pump. Venae cavae-to-pulmonary artery shunts were created in our laboratory 11 and pressures measured in the systemic arterial and venous, and pulmonary arterial systems. Even when elevated with infusions to the levels higher than the normal pulmonary arterial pressure, the central venous pressure was insufficient to perfuse the lungs adequately without the energy supplied by right ventricular contraction.

A pump was used to replace the left ventricle by Jamison, Gemeinhardt, Alai and Bailey. 11 The output of their pump was varied according to the volume of the venous return. The range of venous pressures was noted as between 125 and 200 mm Hg. These authors concluded that given enough power, the left ventricle could take over the work of the right heart. This is indeed the case, as shown in the present experiments, but the resulting circulation is extremely unphysiologic as evidenced by marked venous hypertension, hypervolemia and edema formation.

The analogy between the fluid dynamics of this experiment and that of naturally occurring heart failure is not clear because of the compensating pump reservoir, although it was evident that the plasma volume did not expand until the right ventricle failed (fig. 5). Since plasma volume expansion was accompanied by inadequate return to the left heart pump, and was simultaneous with the development of marked venous hypertension, the venous system was engorged with blood. Venous engorgement and venous hypertension were necessary for an adequate right ventricular output when only the left ventricle was functioning. These were of a degree that we have found impossible to attain in intact animals. 11

Since edema occurred before the heart fibrillated in several instances, the mechanism of its formation in this experiment is still obscure. However, edema formation was accelerated by right heart failure, hypervolemia and venous...
hypertension. The animal’s fluid volume was increased as pump flow rate was augmented, but failed to decrease as pump flow was lowered (fig. 3). Thus, fluid entered the extravascular space at increasing rates as total blood flow increased. Since the venous pressure returned to previous levels as pump output was decreased, it would seem that the edema did not influence the venous pressure through changes in tissue pressure.

**Summary and Conclusions**

Acute isolated right ventricular failure was produced in dogs maintained with a mechanical left ventricle of controlled output. The following observations were made: (1) An immediate and marked elevation of central venous pressure occurred which was simultaneous with the uptake of large quantities of fluid from the pump reservoir. (2) In the absence of right ventricular contractions, the venous pressure became a linear function of left heart output. (In dogs with normally functioning right ventricles, venous pressure was only slightly elevated by marked increases in pump output). (3) The intravascular volume became passively expanded when right heart failure was induced. Together with venous hypertension, hypervolemia appeared necessary for maintenance of the total circulation by the left ventricle alone. (4) Edema formation was accelerated by right heart failure, hypervolemia and venous hypertension.

These observations emphasize the essential nature of normal right ventricular function in maintenance of the central venous pressure within physiologic ranges. They illustrate the unfavorable phenomena resulting from maintenance of the total circulation by one pump, regardless of its capacity. They lend support to the theory that venous hypertension directly results from right ventricular failure. Finally, they suggest that hypervolemia is a necessary accompaniment of cardiac incompetence.

**Acknowledgments**

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**Summario in Interlingua**

Acute isolate disfallimento dextero-ventricular esseva producite in canes mantenite per medio de un mechanic ventriculo sinistre a rendimento controlate. Le sequente constataiones esseva facite. (1) Un immediate e marcate elevation del pression venose central occurreva simultaneamente con le introcezione de grande quantitates de fluido ab le reservoir del pumpa. (2) In le absentia de contractiones dextero-ventricular, le pression venose deveniva un function linear del rendimento sinistro-cardiac. (In canes con ventriculos a functionamento normal, le pression venose eseva solo levemente elevate per marcate augmentos del rendimento del pumpa.) (3) Le volumine intravascular esseva passivemente expandite quando disfallimento dextero-cardiac esseva induite. Insimul con hypertension venose, hypervolemia pareva necessari pro le mantenentia del circulation total via le ventriculo sinistre sol. (4) Le formation de edema esseva accelerate per disfallimento dextero-cardiac, hypervolemia, e hypertension venose.

Iste observationes sublinea le character essential del normal functionamento dextero-ventricular in le mantenentia del pression venose central intra le limites physiologic. Illos illustra le disfavorabile phenomenos que resulta del mantenentia del circulation total per un sol pumpa, sin reguardo a su capacitate. Illos supporta le theoria que hypertension venose resulta directamente ab disfallimento dextero-ventricular. Finalmente, illos indica que hypervolemia es un accompaniamento necessari de incompetentia cardiac.

**References**

Hyperstereoscopy—an Improved Method for Visualizing Intercoronal Channels

Physiologic optics can play many tricks. The old adage “Seeing is believing” is not acceptable in science. For instance, the evidence for coronary intercommunications supplied by stereoradiography has been criticized by Mahain and Schlesinger on the ground that separate overlapping vessels in different planes can give the appearance of communications.

It has been calculated that vessels in planes separated less than 5 mm. appear in the same plane. A hyperstereoscopic method has been described by which vessels in planes separated by only 1.4 mm. can be distinguished. Such a procedure may prove useful in distinguishing real from apparent connections in microscopic preparations.

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