Right-Sided Congestive Heart Failure in Dogs Produced by Controlled Progressive Constriction of the Pulmonary Artery

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With the surgical assistance of Alfred Casper.

Right heart failure was produced in dogs by controlled progressive stenosis of the pulmonary artery. Cardiac enlargement, systemic venous engorgement, tachycardia, hepatomegaly and ascites were consistent findings. The course of cardiac failure was characterized by continual changes in cardiovascular hemodynamics; either cardiac decompensation became progressively severe or circulatory function improved and diuresis resulted. Sodium (Na) retention was always associated with high central venous pressure. Cardiac output was reduced during Na retention except during spontaneous anemia superimposed on cardiac failure. Glomerular filtration rate was frequently normal at the onset of cardiac failure.

Many attempts have been made to produce chronic cardiac failure in experimental animals. Most of the early studies were unsuccessful but within recent years considerable progress has been made. Chronic right heart failure in dogs with experimental stenosis of the pulmonary artery has been reported, but the incidence of the syndrome was too low for the method to be of use.

The present study was undertaken to develop a method by which right-sided congestive failure in dogs could be produced consistently and to define the state of the circulation, kidney function and electrolyte metabolism in these animals. The results have been compared with data on the effect of acute constriction of the pulmonary artery in anesthetized animals and with observations on right heart failure secondary to pure congenital pulmonic stenosis in man.

METHODS

The main trunk of the pulmonary artery was constricted in 13 female mongrel dogs by a ligature which encircled the vessel and extended through a silver cannula to the subcutaneous region. A steel wire encased in nylon or a woven nylon tennis string was used as a ligature. To increase the surface area for contact of the ligature with the vessel and to minimize erosion of the ligature into the vessel, four nylon balls (9 mm in diameter) were placed on the ligature; in addition, the nylon balls and ligature were wrapped with surgical sponge (Ivalon) and a ½ inch layer of the sponge was placed between the wrapped ligature and the pulmonary artery. After the ligature and cannula had been in place for two to three weeks, the ligature was tightened. Using local anesthesia, a short incision was made through the skin to expose the subcutaneous end of the ligature.

Pressures were measured in the right ventricle, right atrium and femoral artery before and after each tightening. The ligature was tightened until either (1) mean right atrial pressure (RAP) reached 150 mm. water, or (2) femoral arterial pressure dropped sufficiently to suggest that further constriction would lead to an irreversible fall in pressure and death. If ascites failed to appear, the pulmonary artery was constricted further at three to seven day intervals on as many occasions as necessary until fluid retention occurred.

The design of the study consisted of control observations made before the initial constriction (except in dogs 8-10) and studies from the time of the appearance of ascites until death. The dogs were kept in metabolic balance cages except during experimental procedures. They were fed a constant synthetic diet containing 80 Cal./Kg. per day, 0.2 Gm./Kg. per day of nitrogen, 60 mEq. per day of Na and 17.6 mEq./day of potassium (K).

Measurements included cardiac output by the direct Fick procedure, pressures from the right ventricle, right atrium and femoral artery, roentgenograms for heart size, postabsorptive glomerular
RIGHT-SIDED HEART FAILURE IN DOGS

TABLE 1.—Cardiovascular Hemodynamic Function at the Onset of Cardiac Failure

<table>
<thead>
<tr>
<th>Dog</th>
<th>Body Weight (Kg.)</th>
<th>Number of Tightenings of Pulmonary Artery Ligature</th>
<th>Cardiac Output (L/min.)</th>
<th>A-V Oxygen Difference (vols%)</th>
<th>Oxygen Consumption (ml./min.)</th>
<th>Heart Rate (beats/min.)</th>
<th>Stroke Volume (cc.)</th>
<th>Mean Femoral Arterial Pressure (mm. Hg.)</th>
<th>Mean Total Peripheral Resistance (dyne/cm.² sec.)</th>
<th>Total Pulmonary Hemodynamic Resistances (mm. Hg.)</th>
<th>Mean Right Atrial Pressure (mm. Hg.)</th>
<th>Right Ventricular Systolic Pressure (mm. Hg.)</th>
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* C and F are abbreviations for control and experimental (cardiac failure) periods respectively.
† Measurements after second tightening of the pulmonary artery ligature; sodium formed after first constriction but a spontaneous diuresis resulted.
‡ The duplicate control values for cardiac output and femoral arterial pressure were obtained several days apart.
§ Control determinations of right ventricular systolic pressure were obtained after the ligature had been inserted around the pulmonary artery; the slightly elevated control values suggest slight constriction of the pulmonary artery although the ligature had not been tightened.

filtration rate (creatinine clearance (Ccr)), effective renal plasma flow (clearance of para-aminohippurate (CPAH)), Na and K balances, T-1824 dye* spaces in plasma and in ascitic fluid and total protein concentrations in plasma and ascitic fluid. Mean femoral arterial pressure and mean RAP were determined from pressure curves by integration; no attempt was made to calculate right ventricular end diastolic pressure. The increase in heart size was estimated from roentgenograms by weighing paper tracings of the contour of the heart. All experimental procedures and chemical methods not described have been reported elsewhere.10, 11

RESULTS

Production of Cardiac Failure.—After one to four tightenings of the pulmonary artery ligature, clinical signs of right-sided congestive heart failure developed in 10 dogs; systemic venous engorgement, cardiac enlargement, tachycardia, hepatomegaly and ascites were consistent findings. Right heart failure resulted after the first tightening of the pulmonary artery ligature in only 2 of the 10 dogs (table 1). In the other 8 animals, arterial pressure fell so low during the first constriction of the pulmonary artery that tightening of the ligature was stopped before RAP reached 150 mm. water, and ascites failed to appear. On several occasions the ligature was loosened because of cardiac arrhythmias, severe dyspnea followed occasionally by respiratory arrest, or a progressive decline in arterial pressure. After the second constriction of the pulmonary artery, 5 of the remaining 8 animals developed ascites whereas 2 dogs required three and 1 animal required four tightenings of the ligature before chronic fluid retention ensued (table 1). Three animals that died within 24 hours following pulmonary artery constriction are not included.

The diameter of the pulmonary artery was reduced from a mean value of 20 mm. for the 10 dogs to 5 to 7 mm. This degree of constriction resulted in slight erosion of the wall of the pulmonary artery in most dogs; however,
TABLE 2.—Cardiovascular and Renal Hemodynamic Function and Electrolyte Excretion at the Onset of Cardiac Failure.

Studies were conducted within 1–2 days after the appearance of ascitic fluid to determine the primary changes associated with Na retention. These initial observations were made to exclude the influence of secondary factors which might obscure the level of functional alterations associated with the appearance of ascites. The results are presented in tables 1 and 2.

Cardiac output was reduced 42 per cent in dogs 4 to 7 and 10; the reduction resulted from a 60 per cent drop in stroke volume. Mean femoral arterial pressure declined less than cardiac output and, consequently, calculated total peripheral resistance increased. RAP increased from 51 to 196 mm. of water (10 dogs); in dogs 1, 2, 5, 6 and 7 the pressure tracings suggested tricuspid regurgitation. Right ventricular systolic pressure increased in all except dog 5; the average increase for 7 dogs was from 38 to 77 mm. Hg. Cardiac size increased 20 per cent (7 animals).

The postabsorptive rate of glomerular filtration (GFR) was unaltered in 5 of 7 dogs but a slight reduction occurred in dogs 6 and 9 (table 2). Renal plasma flow (RPF) was reduced consistently (31 per cent for 7 dogs). Renal Na excretion was reduced markedly whereas urinary K output remained unchanged or increased only slightly. T-1824 dye space in plasma was increased 47 per cent (6 dogs). The total plasma protein concentration decreased from 6.9 to 5.6 gm. per cent (6 dogs). The ascitic fluid protein concentration which was measured simultaneously with

<table>
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<th>Dog</th>
<th>Body Wt. (Kg.)</th>
<th>CcR (cc/min.)</th>
<th>CcR-AH (cc/min.)</th>
<th>Na Excretion (mEq/day)</th>
<th>K Excretion (mEq/day)</th>
<th>T-1824 Dye Space in Plasma (L)</th>
<th>Ascitic Fluid Volume (L)</th>
<th>Plasma Protein (Gm. %)</th>
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* C and F are abbreviations for control and experimental (cardiac failure) periods respectively.
† Na intake = 60 m.Eq./day; K intake = 17.6 m.Eq./day. The control values for electrolyte excretion represent the averages for 8–12 days whereas the values obtained during heart failure are for the first 2–5 days while excretion was relatively constant.
‡ The two control values for CcR and CcR-AH were obtained several days apart and each value represents the average of 3 renal clearance periods.
plasma protein ranged from 4.0 to 5.0 gm. per cent. Plasma electrolytes were unchanged.

**Clinical Course of Cardiac Failure.**—A stable state of cardiovascular hemodynamics was rarely observed; either cardiac failure was progressive and terminated in death within 3–15 days (dogs 1, 2 and 8) or cardiovascular function improved within 6–10 days and ascites ceased to form or disappeared (dogs 3, 4, 6, 7 and 10). Subsequently, the pulmonary artery was reconstituted in dogs 4 and 7 and ascites reappeared; in dogs 6 and 10 ascites reaccumulated spontaneously. Dogs 5, 7 (after second constriction of pulmonary artery) and 9 were digitalized within 3–8 days after the final constriction of the pulmonary artery because of severe cardiac failure; the results of digoxin administration are reported elsewhere.12

During progressive cardiac failure, femoral arterial pressure dropped gradually while RAP showed a further elevation (fig. 1). Right ventricular systolic pressure remained at essentially the same high level until a preterminal state was reached when a slight fall occurred (fig. 1). Measurements of cardiac output were too infrequent during progressive cardiac failure to establish a definite trend. GFR fell as cardiac decompensation progressed and a further fall in RPF occurred. Na retention was almost complete but K balance was either unchanged or slightly negative (fig. 1). Fecal Na and K excretion was not detectably altered.

**Relation of Cardiovascular and Renal Hemodynamic Function to Na Retention by the Kidney.**—Cardiac output was always low at the onset of cardiac failure (table 1) but a spontaneous diuresis resulted in dogs 3, 4 and 10 without an elevation in cardiac output (fig. 2). In dogs 6 and 10, an anemia developed spontaneously toward the end of the course of cardiac failure; cardiac output returned to within normal limits (fig. 2) but RAP remained high or increased further and Na retention continued. GFR was unchanged at the onset of heart failure in 5 of 7 dogs (table

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**Fig. 1.** Right ventricular systolic pressure (RVSP) in mm. Hg, mean right atrial pressure (RAP) in mm. water, femoral arterial systolic (—■—), mean (—●—) and diastolic (—○—) pressures (BP) in mm. Hg, Na excretion [ENa] in mEq/day and K excretion [EK] in mEq/day during the control period and during cardiac failure. Values for RVSP and RAP are plotted as solid columns from lines representing control levels.

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**Fig. 2.** Relation of urinary Na excretion in mEq/day (ordinate) to cardiac index in L/min./M² (abscissa) during the control period, during cardiac failure and at the onset of spontaneous diuresis. The solid symbols for a Na excretion of 50 mEq/day or more and a cardiac index above 3.5 L./min./M² represent control values. The open symbols for dogs 3, 4 and 10 indicate data obtained at the onset of spontaneous diuresis. The remaining symbols are for values obtained during cardiac failure; Na excretion was 30 mEq/day or less. The two high values for cardiac output which are indicated by an A beside the symbol were the result of a spontaneous anemia superimposed upon cardiac failure. Body surface area was calculated from the Meeh-Rubner formula.
Fig. 3. Relation of mean right atrial pressure in mm. water on the ordinate to the accumulation of ascites. Solid symbols represent pressures obtained during Na retention whereas open symbols indicate RAP before ascites formation or at the onset of spontaneous diuresis. The arrows show the change in RAP recorded at the onset of diuresis. Attention is called to the elevated pressures (above the highest normal pressure of 70 mm. water) in the absence of Na retention; these data were obtained in the 8 animals requiring more than one tightening of the pulmonary artery ligature before cardiac failure developed and in the 5 dogs diuresing spontaneously.

2) and failed to increase with the appearance of a spontaneous diuresis. RPF was consistently reduced when ascites appeared (table 2) but Na retention was observed occasionally at a high level of RPF and spontaneous diuresis occurred in the presence of a very low RPF.

Central venous pressure was high throughout the clinical course of cardiac failure. In no instance did ascites develop or continue to accumulate with an RAP below 145 mm. water (fig. 3); the mean control value (10 dogs) for RAP was 51 mm. water (table 1). In all but dogs 5 and 7 more than one tightening of the pulmonary artery ligature was required before a pressure of 145 mm. water or above was reached and ascites developed. When the mean pressure in the right atrium was above 175 mm. water, 60 per cent or more of ingested Na was retained except in dog 7 in which Na retention occurred only with an RAP above 220 mm. water. At the onset of spontaneous diuresis, a drop in RAP of 35 and 50 mm. water occurred in dogs 3 and 6 respectively (fig. 3) but in dogs 7 and 10 the decline in central venous pressure was negligible and in dog 4 a fall in pressure was not recorded until after Na excretion had increased. Failure of a fall in RAP to be recorded consistently and failure of an elevation in cardiac output and GFR to be observed at the onset of increased Na excretion may be a reflection of the limitations of the methods for measuring cardiovascular and renal hemodynamic function.

**DISCUSSION**

The circulatory changes resulting from acute constriction of the pulmonary artery have been studied intensively in experimental animals. From observations in open-chest anesthetized dogs, Fineberg and Wiggers suggested that right ventricular failure occurred when the fatigued myocardium failed to maintain a high right ventricular systolic pressure. At this stage in their preparation, the amount of blood delivered to the left ventricle was decreased, arterial pressure was reduced and initial tension in the right ventricle was high.

The present data show essentially the same qualitative alterations in the circulation as reported by Fineberg and Wiggers and others from studies of acute preparations. However, by slow progressive constriction of the pulmonary artery, a 65-75 per cent reduction in the diameter of the vessel was achieved with less fall in blood pressure than occurred following acute constriction of lesser degree in anesthetized animals. Also, the higher arterial pressure in the present chronic preparation suggests that the reduction in coronary blood flow with its depressant effect on myocardial function was less than in the acute preparation.

The onset of cardiac failure was characterized by low cardiac output, high RAP and, presumably, an elevated right ventricular end diastolic pressure, and appearance of ascitic fluid. At the onset of ascites formation, the state of the circulation varied considerably among the animals and it appears that the degree of myocardial depression was much greater in some dogs than in others. These differences are a reflection of different degrees of constriction (65 to 75 per cent of the original diameter).
and of variation in individual responses. In dog 5, cardiac output was very low and right ventricular systolic pressure was the lowest observed for the entire group during cardiac failure. In this animal, the state of the circulation appeared to be comparable to that suggested by Fineberg and Wiggers for right ventricular failure. In dog 1, right ventricular systolic pressure remained at essentially the same high level until a preterminal state was reached, although RAP increased progressively. These data suggest that the right ventricular myocardium was sufficiently depressed so that further lengthening of the fibers failed to effect a more forceful contraction. On the day before death in dog 1, right ventricular systolic pressure was slightly lower and RAP was elevated more than previously; the findings indicate that dog 1 achieved only preterminally a degree of circulatory failure similar to that at the onset of ascites in dog 5. In contrast to the progressive decline in circulatory function in dogs 1, 2, 5, 8 and 9, cardiovascular function improved in the other five animals and a diuresis resulted. In these animals (dogs 3, 4, 6, 7 and 10), failure of the right ventricle to maintain a normal level of cardiac output at the onset of ascites formation was indicative of a failing myocardium. Additional evidence of impaired myocardial function was provided by the cardiovascular response to digoxin.

Studies of right heart failure secondary to pure congenital pulmonic stenosis in man have provided data for comparison with the present observations. From 1826 to 1948, 27 cases of right heart failure in patients with pure congenital pulmonic stenosis were reported. From 10 individual case reports referred to by Greene and associates the following pertinent findings were obtained: (1) ascites was present in 9 of the 10 cases, (2) systemic venous engorgement was always present; in the patients in whom measurements were made, venous pressure was 25 to 30 cm. of water, (3) hepatomegaly was a consistent finding and hepatic cirrhosis was not infrequent, and (4) enlargement of the right atrium and ventricle was observed in every instance. These findings are similar to the data reported here.

In patients with cardiac decompensation there are reports that venous pressure is not always elevated at the onset of Na retention but in the present study ascites appeared only after a high level of RAP was reached. Failure of this relationship to be found consistently in man may be a reflection of interference by secondary factors so that a clearly defined association is obscured, inadequate measurements of central venous pressure and Na excretion, or a species difference. The present data on Na balance and RAP indicate the need for similar studies in patients with chronic congestive heart failure.

Although Na retention was always associated with a high RAP in the present study, evidence for a causal relationship of elevated venous pressure to reduced Na excretion is lacking. Cardiac output was low during Na retention except in the presence of anemia which appeared toward the end of the course of cardiac failure in two animals. When GFR was decreased during progressive cardiac failure, an affect on Na excretion may have occurred. More clearly defined relationships of cardiovascular and renal hemodynamic functions to Na excretion have been described in dogs with constriction of the thoracic inferior vena cava.

Sodium retention and ascites formation were not dependent upon a low cardiac output but resulted from venous hypertension in dogs with thoracic inferior caval constriction. These animals developed the same type of high protein ascites as observed here and the only consistent alteration in cardiovascular function was a high venous pressure below the constricting ligature; cardiac output was frequently normal. Evidence suggesting that Na retention resulted from an excess of circulating adrenocortical salt hormones was reported for dogs with thoracic inferior caval constriction and ascites.

Summary and Conclusions

Right heart failure has been produced in 10 mongrel dogs by controlled progressive constriction of the pulmonary artery. After 1 to 4 tightenings of the pulmonary artery ligature, clinical signs of congestive heart
failure developed. Cardiac enlargement, systemic venous engorgement, tachycardia, hepatomegaly and ascites were consistent findings. A stable state of circulatory function rarely occurred; either cardiovascular function improved and Na excretion increased or cardiac failure became progressively severe.

Evidence of a failing right ventricular myocardium includes (a) failure of the right ventricle to maintain a normal level of cardiac output, (b) failure of a progressive rise in filling pressure of the right heart to effect a further elevation in right ventricular systolic pressure, and (c) a preterminal fall in right ventricular systolic pressure.

Analysis of the changes in cardiovascular and renal hemodynamic function which might influence Na excretion revealed that Na retention was always associated with a high central venous pressure (above 145 mm. water). Cardiac output was reduced during Na retention except in the presence of a spontaneous anemia superimposed upon cardiac failure. GFR was frequently normal at the onset of cardiac failure.

Acknowledgments

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