The George E. Brown Memorial Lecture
Circulatory Congestion and Heart Failure

By Ludwig W. Eichna, M.D.

I N ACCEPTING the honor, and the responsibility, of the George E. Brown Lecture-ship for 1959 I do so, not for myself, but for a fine group of stimulating colleagues. It is their work and their thinking that will constitute the substance of this lecture. This presentation will, therefore, have a personal and restricted focus, a desirable feature in that one area and one viewpoint is examined in depth, an undesirable feature in that many pertinent investigations by others are not given the attention that they certainly merit.

Man, almost alone, develops spontaneously the syndrome of congestive heart failure. Accordingly, the observations to be presented have all been derived from man. They will examine the interrelationships between the two major manifestations of the circulation in congestive heart failure, namely, circulatory congestion and reduced cardiac output. Three principal areas will be considered: (1) the nature of circulatory congestion and its significance as an index of heart failure, (2) the effect of the circulatory congestion itself on the function of the heart and on the function of one peripheral organ, the kidney, and (3) the relation of lowered cardiac output to circulatory congestion and to activity and survival.

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Supported by grants-in-aid from the New York Heart Association, Inc., the Life Insurance Medical Research Fund, and the National Heart Institute, U. S. Public Health Service.


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Circulatory Congestion as an Index of Heart Failure

A cardinal feature of congestive heart failure is the presence of circulatory congestion. This congestion involves the venous beds behind either the right side of the heart, the left side, or both. Venous congestion behind the right ventricle produces the characteristic distended veins, the enlarged liver, and the edema; congestion behind the left ventricle causes the dyspnea, orthopnea, and pulmonary rales. It is these manifestations of circulatory congestion that the clinician recognizes at the bedside; their cause he must infer. Clinico-pathologic correlation long ago established that these congestive changes occur in patients with heart disease and, more recently, hemodynamic determinations have demonstrated that in such cardiac patients the output of the heart is decreased below normal.1,2,3 It is characteristic that the presence of circulatory congestion, regardless of the circumstances under which it appears, has come to be accepted as diagnostic of heart failure and specifically of failure of the myocardium to circulate adequate amounts of blood.

The first point to be examined is whether this traditional concept is correct.4 Consider the situation that results from the administration of excessive intravenous infusions5-13 to a patient with a normal heart, particularly if there is oliguria, as in lower nephron nephrosis. The normal heart is important to the argument. Blood volume is artificially increased, circulatory distention develops and with it appear the characteristic manifestations of circulatory congestion, already detailed. The clinical picture is similar to, indeed indistinguishable from, congestive heart failure. But has the heart failed? It is difficult to understand how the heart by improv-
ing any of its functions, including increasing the cardiac output, can relieve the circulatory congestion. This situation illustrates the hemodynamic disturbances here designated noncardiac circulatory congestion. Its clinical manifestations are essentially similar to those of congestive heart failure; its course, however, is not failure of the heart muscle.

A series of correlated hemodynamic observations by Farber, Berger, Rader, Smith, and Albert indicated that both systemic and pulmonic vascular congestion may rise on a noncardiac basis to produce a clinical syndrome that closely simulates congestive heart failure. The data suggested 3 categories (table 1) of such noncardiac circulatory congestion, and the patients in each category have been traditionally considered to have congestive heart failure.

Category I is characterized by mechanical obstruction to flow in or about the heart, and includes patients with constrictive pericarditis, tricuspid stenosis, and most typically the severe, “pinch-cock” mitral stenosis without significant myocardial involvement, as indicated by the absence of right ventricular failure and by a normal-sized or slightly enlarged heart. The roentgenographic silhouette does not always give a true impression of the nature of cardiac enlargement. For example, in a patient (T. R.) with severe mitral stenosis the huge cardiac silhouette extending from chest wall to chest wall, was due to a giant left atrium. The ventricles, the propelling component of the heart, were of normal size and weighed 300 Gm.

Category II is characterized by the accumulation of excessive amounts of water and salt in patients with normal hearts and includes the edematous states associated with the following conditions: the administration of large amounts of salt-retaining steroids, anuria, or oliguria as in lower nephron nephrosis and acute diffuse glomerulonephritis. Albert and Smith observed that noncardiac subjects who became edematous when receiving large doses of corticotropin (ACTH) or salt-retaining steroids developed not only symptoms of dyspnea and orthopnea but also enlarged cardiac silhouettes and evidences of pulmonary congestion. The associated hemodynamic changes were increased vascular and intracardiac pressures of a degree similar to congestive heart failure, but cardiac output remained normal. Following cessation of medication hemodynamic alterations and heart size returned to normal. The edema and circulatory congestion of acute nephritis is also associated with cardiac enlargement but the heart again returns quickly to normal size following diuresis (fig. 1A, A').

Category III is characterized by circulatory congestion in the hyperkinetic circulatory states, the so-called “high output heart failure,” and includes the edematous states occurring in beriberi, arteriovenous fistula, and severe anemia, again in patients with normal hearts. The cardiac enlargement and circulatory congestion in these patients subside with specific but noncardiac medication and the heart returns to normal size (fig. 1B, B' and 1C, C').

The characteristically normal heart size after recovery from circulatory congestion is a significant differentiating feature that separates these noncardiac patients from typical congestive heart failure, in which the heart remains enlarged even after recovery of compensation.

If these 3 types of circulatory congestion are truly noncardiac, that is, nonmyocardial in origin, they should differ from typical con-
gestive heart failure, occurring in patients with heart disease, in significant hemodynamic functions. Accordingly, differences were sought and indeed were observed, in 3 hemodynamic parameters: the dynamics of cardiac function; the circulation and function of peripheral organs—the kidney was chosen for study because of its key role in edema formation; and the hemodynamic and clinical response to specific medication.

Consider first the parameter of circulation and cardiac function. With respect to intracardiac pressures (fig. 2), the pressures in the pulmonary artery, right ventricle, and right atrium were elevated above normal in patients in each of the 3 categories of noncardiac circulatory congestion, and these elevations were generally comparable to the values in congestive heart failure. The normal right atrial pressure in “pinch-cock” mitral stenosis is expectedly not elevated, since the right ventricle does not fail in this situation and right-sided congestion is absent. The similarity in the pressure values indicates simply that the location and degree of circulatory congestion in the 3 categories of noncardiac circulatory congestion are the same as in congestive heart failure and verifies quantitatively the clinical finding of congestion in each. Subsequent data will examine

Figure 1

Heart size in circulatory congestion due to excess water and salt retention (A) and hyperkinetic states (B, C). For each vertical panel the upper x-ray was taken during circulatory congestion and the corresponding lower x-ray after recovery from congestion following noncardiac medication. A, A', M.K., male, 70 years, acute glomerulonephritis. B, B', M.M., female, 38 years, beriberi. C, C', W.S., male, 48 years, severe anemia.
cardiac pressures, may occur with no, or little, increase in total blood volume. Accordingly, blood volume is not the sole determinant of circulatory congestion. Vasoconstriction of all components of the vascular tree, specifically including the veins\textsuperscript{30–33} and cardiac chambers, appears to be a very significant additional factor. Vasoconstriction is considered to contribute to the venous congestion in 2 ways: (1) by re-distributing blood from the minute vessels, essentially the venules, to the more distensible central collecting compartments, the large veins and atria and (2) by acting upon this increased local blood volume by an increase in venous tone. The result is an elevation in venous pressure and in intracardiac residual pressures.

The differences between congestive heart failure and noncardiac circulatory congestion become more fundamental when the more primary cardiac function of cardiac output is considered (fig. 4). Cardiac output is typically below normal in congestive heart failure\textsuperscript{1–7, 34, 35} and, in contrast, essentially normal in the circulatory congestions associated with intracardiac obstruction and excessive

**Figure 2**
Comparison of intracardiac pressures in congestive heart failure and noncardiac circulatory congestion. At the top of each vertical panel is listed the category of circulatory congestion, at the bottom the individual disease states for each category. For pulmonary artery pressure (PA) and right ventricular pressure (RV) systolic pressure is plotted by the top and diastolic pressure by the bottom of the black columns. The short horizontal line through the pulmonary artery pressure column is mean pressure. Right atrial pressure (RA) is mean pressure plotted by the top of the black column. The tops of the horizontal, vertically hatched bands indicate maximum normal systolic pressure and the bottom of the bands maximum normal diastolic pressure. The numbers immediately below the abscissa indicate the number of subjects whose data have been averaged for each corresponding set of vertically arranged black columns. When a different number of subjects entered into a particular average, the number of subjects is now indicated by the number above the corresponding column.

whether these similar circulatory congestions are associated with, and due to, the same or different hemodynamic causes.

With respect to blood volume in congestive heart failure, the total blood volume is usually,\textsuperscript{6, 25–27} but not always,\textsuperscript{28, 29} increased well above normal, and in the patients here studied averaged 30 per cent above predicted values (fig. 3). In the noncardiac circulatory congestions the blood volume was quite often not increased, and when it was increased the increments were frequently less marked than in congestive heart failure (fig. 3).

These data indicate that circulatory (venous) congestion, with elevated residual intracardial pressures, may occur with no, or little, increase in total blood volume. Accordingly, blood volume is not the sole determinant of circulatory congestion. Vasoconstriction of all components of the vascular tree, specifically including the veins\textsuperscript{30–33} and cardiac chambers, appears to be a very significant additional factor. Vasoconstriction is considered to contribute to the venous congestion in 2 ways: (1) by re-distributing blood from the minute vessels, essentially the venules, to the more distensible central collecting compartments, the large veins and atria and (2) by acting upon this increased local blood volume by an increase in venous tone. The result is an elevation in venous pressure and in intracardiac residual pressures.

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**Figure 3**
Comparison of blood volume in congestive heart failure and noncardiac circulatory congestion. (See legend figure 2 for manner and details of plotting.) Total blood volume is indicated by the height of the entire column, red cell volume by the solid portion, and plasma volume by the open portion of the column. The predicted blood volume (total) for each group of subjects is indicated by the corresponding horizontal line. The numbers above each column give the number of subjects whose data have been averaged for the value charted.
Comparison of cardiac output and its relation to oxygen consumption in congestive heart failure and noncirculatory congestion. (See legend figure 2 for manner and details of plotting.) Total oxygen consumption ($O_2$ Cons.), cardiac output as cardiac index (C.I.), and arterial-mixed venous oxygen difference (A-V $O_2$ Diff.) are indicated by the tops of the respective columns. The horizontal vertically hatched bands indicate, in this and subsequent charts, the range of normal values for the corresponding functions.

Figure 4

Comparison of renal hemodynamic functions and water and electrolyte excretions in congestive heart failure and noncardiac circulatory congestions. (See legends figures 2 and 4 for manner and details of plotting.) Renal plasma flow (RPF), glomerular filtration rate (GFR), filtration fraction (FF), sodium excretion (Na), and water excretion (Vol) are indicated by the heights of the respective columns. Since the patients were not receiving a fixed salt and water intake, no normal range of water and electrolyte excretion is indicated.

Renal plasma flow and the only moderately reduced glomerular filtration rate result in the strikingly increased filtration fraction, so characteristic of congestive heart failure. In contrast, in the three categories of noncardiac circulatory congestion renal plasma flow and glomerular filtration rate were both more nearly normal and, of particular significance, the filtration fraction was not increased, or only very slightly so. Renal clearance measurements were not possible on the anuric patient. The low glomerular filtration rate and filtration fraction in glomerulonephritis results from the specific glomerular lesion in this disease. No data were obtained in patients with congested states due to salt-retaining hormones; the data following ACTH administration are for subjects without circulatory congestion and are included to indicate the type of effect produced on renal function by this hormone. Urinary excretions of water and electrolyte were variable and tended to be equally low in both types of congestion. Their significance is

Circulation, Volume XXII, November 1960
Comparison of the effect of intravenous digoxin on the arterial-mixed venous oxygen difference in congestive heart failure and noncardiac circulatory congestion. (See legend figure 6 for details of plotting.) (From Transactions of the Association of American Physicians 67: 72, 1954.)

1.5 mg.) of digoxin were determined. The digitalis glycoside produced no, or minimal, hemodynamic effects in the 3 types of noncardiac circulatory congestion, in contrast to prompt and decided improvement in hemodynamic functions in congestive heart failure (figs. 6-9).1, 2, 4, 40, 41

The cardiac output in congestive heart failure (CHF) increased from typically low values to normal levels within 1 to 2 hours after the intravenous administration of digoxin (fig. 6). In contrast, the normal cardiac output of "pinch-cock" mitral stenosis (MS) and glomerulonephritis (GN), the slightly lowered output of the anuric congestion (LN), and the high outputs of beriberi (BB) and anemia (A) all remained unchanged.

The response of the arterial-mixed venous oxygen difference following intravenously administered digoxin was likewise different in the 2 types of circulatory congestion and, as expected, reflected the changes in cardiac output (fig. 7). The abnormally high values in congestive heart failure fell to normal limits within 2 hours of administration of digoxin, whereas no changes occurred in the normal (MS, GN, LN) or low (A and BB) A-V oxygen differences of the noncardiac circulatory congestions.

In congestive heart failure the typically
Comparison of the effect of intravenous digoxin on the right ventricular end-diastolic pressure in congestive heart failure and noncardiac circulatory congestion. (See legend figure 6 for details of plotting.) (From Transactions of the Association of American Physicians 67: 72, 1954.)

elevated intracardiac residual pressure, represented by the right ventricular end-diastolic pressure, was lowered markedly and characteristically, again within 1 to 2 hours, by intravenously administered digoxin.\textsuperscript{42, 43} In the noncardiac circulatory congested states intravenous digoxin either did not affect this intracardiac pressure (GN, LN, A) or induced falls of smaller magnitude (BB) (fig. 8).

A prompt, considerable, and continuing diuresis of water and sodium was induced by the intravenously administered digoxin in congestive heart failure.\textsuperscript{41, 44} Again in contrast, no increase in urine volume and a much smaller increase in sodium excretion followed the intravenous digoxin in 3 patients (LN, GN, and BB) with noncardiac circulatory congestion (fig. 9).

It is possible that single, intravenous doses of digitalis glycosides are not so optimally therapeutic as they are considered to be and that prolonged digitalis medication is required for full therapeutic effect. Accordingly, full digitalization, with digitalis leaf given orally, was carried out in some patients over several days. The results paralleled and substantiated the acute effects of therapeutic single doses of digoxin given intravenously: decided improvement in congestive heart failure, no, or equivocal, benefit in the 3 noncardiac congested states. For example, digitalis leaf in full therapeutic doses induced neither significant diuresis, weight loss, nor reduction of peripheral venous pressure in patient E. P. with severe hypochromic microcytic anemia and circulatory congestion (fig. 10). Ventricular premature contractions occurred after 3 days of fairly intensive digitalis medication, indicating adequate dosage of the drug, but the circulatory congestion remained largely unchanged and the patient desperately ill. Two transfusions were given. Diuresis, weight loss, and relief of circulatory congestion occurred as the erythrocyte concentration, following transfusion and iron medication, rose to normal values (fig. 10). Oral digitalization has also failed to induce a diuresis or relieve the venous congestion in acute glomerulonephritis, events which then followed spontaneous improvement in the disease process.

Similarly, beriberi heart disease failed to respond to digitalization, whereas the subsequent administration of thiamine produced an excellent diuresis.\textsuperscript{45}

The differences in response to intravenous and oral digitalis appear to satisfy the proposed third criterion for separating noncardiac circulatory congestion from congestive heart failure.

The differences in general and local hemodynamic functions between congestive heart failure and the 3 categories of noncardiac circulatory congestion make it difficult to accept for all states of circulatory congestion a common etiology of heart, that is myocardial failure. Similar to both types of congested states are the clinical and hemodynamic evidences of circulatory congestion. But the congestion itself appears to be a secondary and nonspecific manifestation. Differences in the more primary hemodynamic functions separate the 2 types of circulatory congestion and offer the more reliable parameters of hemodynamic function to differentiate congestive heart failure, occurring in patients with diseased hearts, from noncardiac circulatory congestion, occurring in patients without intrinsic heart disease. Such a differentiation seems preferable to the common practice of grouping all circulatory congestions under the
single category of congestive heart failure, simply because of the presence of similar but nonspecific, clinical manifestations.

The following classification (table 2) is proposed:

Circulatory congestion, whether systemic or pulmonie, is itself a nonspecific hemodynamic manifestation and results from an increased central blood volume plus an increased vascular tonus. The increased central blood volume may be part of an increased total blood volume or the result of a redistribution of a more normal blood volume. The increased vascular tonus probably results from neurogenic and humoral responses. Circulatory congestion occurs when the heart is diseased, in which case the myocardium fails and the cardiac output fails. This is heart failure and results from failure of the heart’s contracting force. This state should be differentiated from clinically similar circulatory congestions which occur under 3 types of circumstances: (1) when there is obstruction to blood flow in and about the heart, (2) when there is excess retention of water and salt, in both of which states the cardiac output is usually normal, and (3) in the hyperkinetic congested states, in which cardiac output is increased. In these states the heart appears not to fail as a pump and the circulatory congestion appears not to be of cardiac origin.

Such a differentiation of circulatory congestions is not a matter of semantics. An understanding of the different physiologic disturbances in the 2 types of circulatory congestion not only helps clarify the basic mechanisms of disease processes, but also finds practical application in proper therapy; digitalis benefits one, congestive heart failure, but is questionably effective in the other, the noncardiac circulatory congestions, in which states diuretic therapy is likely to be more effective. A practical consideration: the 2 types of circulatory congestion can often be differentiated by the simple bedside test of the circulation time. When properly performed, the circulation time correlates quite well with cardiac output: a decreased or normal circulation time indicating a high or normal cardiac output and hence a noncardiac congested state, an increased circulation time indicating a low cardiac output and congestive heart failure. Also, it should be recognized that noncardiac circulatory congestion is much less frequently encountered than congestive heart failure and that combinations of the 2 types of congestion may occur in the same patient. The data presented have dealt with carefully chosen instances of typical, uncomplicated, noncardiac and cardiac circulatory congestions. Excluded from consideration were combinations of the 2 states in which the physiologic disturbances partake, in varying degrees, of each; for example, occluding mitral stenosis associated with left ventricular failure and a large heart, or exacerbation of acute nephritis in a patient with chronic nephritis and hypertension, which has caused some left ventricular failure as well.

**Effect of Circulatory Congestion on Cardiac Function and Renal Function**

Let us turn now to the second area for consideration, the effect of circulatory (venous) congestion itself on cardiac, hemodynamic, and renal functions. Sobol, Kessler, and Rader measured the changes in these functions when circulatory congestion was decreased by means not involving the heart. A steady state of primary, peripheral vasodilation was produced for 1 hour by the controlled, continuous, intravenous infusion of the ganglion-blocking agent, Arfonad. Arfonad was chosen because it has a periph-

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**Table 2**

*Circulatory (Venous) Congestion (Increased central venous blood volume plus increased vascular tone)*

<table>
<thead>
<tr>
<th>Category</th>
<th>Description</th>
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<tbody>
<tr>
<td>I. Cardiac</td>
<td>Congestive heart failure—low cardiac output</td>
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</table>
| II. Noncardiac (normocardial) | Cardiac mechanical obstruction  

Excessive water  
and salt retention  

Hyperkinetic congested states—high cardiac output

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*Source: Circulation, Volume XXII, November 1969*
Comparison of effect of intravenous digoxin on water and sodium excretions in congestive heart failure and noncardiac circulatory congestion. (See legend figure 6 for details of plotting.) No band of range of normal values is indicated since the patients were not receiving a fixed salt and water intake. (From Transactions of the Association of American Physicians 67: 72, 1954.)

Figure 9

Ineffectiveness of oral digitalis therapy in circulatory congestion associated with severe anemia. Each vertical arrow in the horizontal panel HGB indicates transfusion of 500 ml. of blood. The height of the horizontal panels Veins, Rales, Liver, Edema represent 4+. VP, venous pressure; HGB, hemoglobin.

posed of noncardiac subjects and hypertensive and normotensive cardiac subjects compensated from congested heart failure. The decrease in cardiac filling pressures could not be achieved with this agent without a concomitant fall in systemic arterial pressure. The degree of reduction of systemic pressure was so controlled, however, that the lowered pressure did not fall outside the normal range (fig. 11).

Associated with the fall in intracardiac residual pressures, and reduction in the congested state, cardiac output increased in the patients with congestive heart failure, presumably as a result of the decrease in ventricular filling pressure (fig. 12). After the dilatation, as intracardiac residual pressure rose, cardiac output fell to the initial level. The rise in cardiac output was small, averaged 15 per cent, and did not carry the output into the normal range. This small increase becomes more significant, however, when contrasted with the fall in cardiac output, to below the normal range, which occurred during the period of decreased ventricular filling pressure in the control noncardiac and compensated cardiac subjects (fig. 12).

The significance of the rise in cardiac out-
put in congestive heart failure during the period of reduction of the elevated ventricular filling pressure, was substantiated by a concomitant and sizable fall (average 22 per cent) in arterial-mixed venous oxygen difference and by its rise to control levels in the postdilatation period after stopping the Arfonad infusion (fig. 12). Furthermore, an increase (small) in oxygen content of mixed venous blood paralleled the fall in A-V oxygen difference and thus further indicated that cardiac function had improved. Witness also, the contrasting behavior of the A-V oxygen difference in the noncardiac and compensated control subjects, a small increase or no change.

These experiments suggest that circulatory congestion of itself may have an undesirable effect upon cardiac function, increased congestion acting to reduce the cardiac output, which then rises as the congestion is removed. The observations, however, are complex, for systemic arterial pressure also fell. The increased cardiac output may, therefore, be merely a manifestation of an increase in output against a lessened external resistance, and not a function of improved myocardial contraction resulting from decreased central circulatory congestion.

The increase in cardiac output during lowering of the elevated ventricular filling pressure in congestive heart failure and the contrasting decrease in cardiac output during lowering of the normal filling pressure in noncongested states suggests that Starling's law\(^{56-58}\) of the heart applied in intact man. Unfortunately, the falls in systemic and pulmonary arterial pressures make such a conclusion debatable. Since cardiac work is the product of cardiac output and the pressure against which the output is ejected, the increase in cardiac output, which occurred when ventricular filling pressure was lowered in congestive heart failure, is offset by the associated lowering of the arterial pressures.

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**Figure 11**

*Effect of vasodilatation, produced by intravenous infusion of ganglion-blocking agent (Arfonad), on intracardiac and vascular pressures in congestive heart failure (CHF) and in nonfailing circulation (Comp. & Normal).* The data are averages for the following groups: Congestive heart failure: normotensive, 5 patients; hypertensive, 5 patients. Nonfailing circulation: normotensive, 2 noncardiac subjects and 1 patient recovered from congestive heart failure; hypertensive, 2 patients recovered from congestive heart failure. For each line the first point gives the average of the data of 2 or 3 control periods, the second point gives the maximum effect during vasodilatation, the third point gives the average of the data of 2 or 3 observations during 1 hour of vasodilatation, the fourth point gives the data of the post-vasodilatation or recovery period. The 2 horizontal lines in each panel indicate the range of normal values. RA, right atrium; RV, right ventricle.
Accordingly, minute work and stroke work
of the heart, both of the left and right
ventricle, and in both hypertensive and normo-
tensive subjects, did not change as the central
filling pressure fell (fig. 13). In the non-
congested states minute and stroke work de-
creased as filling pressures fell. Such an
analysis would indicate that Starling’s law
of the heart held for normal states but did
not maintain in the failing heart with a
congested circulation. Nevertheless, an
interesting hemodynamic state was produced,
wherein a heart, perhaps incapable of greater
work, was able to deliver more blood to the
circulation and thereby meet more fully the
needs of the peripheral organs and of the
body as a whole. It is not intended here to
suggest that these acute short time events are
applicable to long-term maintenance medica-
tion with vasodilating agents. Reports in the
literature indicate beneficial results from such
an approach\textsuperscript{59} but further observations and
analysis are required, particularly in view of
the reduced water and electrolyte excre-
tions that occurred during the period of
reduced pressures.\textsuperscript{46, 60, 61}

Because Arfonad produced a hemodynamic
state that did not lend itself to a rigid test
of Starling’s law of the heart, Ziffer and
Rader\textsuperscript{62} repeated studies of this type using
sodium nitrite to obtain reduction of circu-
laratory congesting pressure. The advantage of
sodium nitrite is that it acts chiefly upon
the postarteriolar, that is the venous, seg-
ment of the circulation and hence is less likely
to lower arterial pressure in the supine sub-
ject.\textsuperscript{63, 64} This drug also has no direct cardiac
effect. Accordingly, 0.1 to 0.2 Gm. of
the drug was given by mouth and within 15
minutes the right atrial and right ventricular
end-diastolic pressures were decreased, with-
out a lowering of the arterial pressures by
more than 5 to 10 mm. Hg. The pressure
changes persisted for about 1 hour and were
gone by 2 hours after ingestion of the drug.

Under these conditions of decreased circu-
laratory congestion and lowering of the elevated
ventricular filling pressures of congestive
heart failure the cardiac output again in-
creased (fig. 14). Since the changes in arte-
rial pressure and heart rate were very small,
minute work and stroke work of the ventricles
obviously increased. Again in contrast, in
subjects without circulatory congestion, car-

\textbf{Figure 12}

\textit{Effect of vasodilatation, produced by intravenous infusion of ganglion-blocking agent (Arfonad), on cardiac output (left) and arterial mixed venous oxygen difference (right) in congestive heart failure (CHF), and in nonfailing circulation (Comp. & Normal). Details of plotting are the same as in figure 11.}
Effect of vasodilatation, produced by intravenous infusion of ganglion-blocking agent (Arfonad), on cardiac work in congestive heart failure (CHF), and in nonfailing circulation (Comp. & Normal). Details of plotting are the same as in figure 11, except that the horizontal line separates the data for the 2 ventricles and not the range of normal values. The numbers indicate subjects averaged for the corresponding data charted. (From Journal of Clinical Investigation 38: 557, 1959.)

Figure 13

Effect of vasodilatation, produced by oral sodium nitrite, on cardiovascular dynamics in congestive heart failure. The data are arranged vertically for each of 4 subjects represented by initials along the abscissa (subject F.Z. was studied in 2 bouts of congestive failure). For each subject the first point indicates the average of control observations (C at top), the second point gives the data during maximum vasodilatation occurring at a time after ingestion of NaNO₂ indicated by the first “plus number” at the top, the third point (when present) gives the data after return to the control state at the time after ingestion of NaNO₂ indicated by the second “plus number” at the top. Artery = systemic arterial pressure, vertical line connects systolic and diastolic pressure, horizontal dash gives mean pressure; V₁ = ventricular rate; RV₂ = right ventricular end diastolic pressure, solid dot; RA = right atrial mean pressure, open circle; O₂ Cons. = total oxygen consumption; A-V = arterial-mixed venous oxygen difference; C.I. = cardiac index.

Figure 14

stinate the force governing the arrangement of the ultrastructural components of the contractile protein of the myocardial cell and this arrangement could well be the ultimate factor determining the subsequent contraction of the fiber. This line of reasoning lends support to the many observations that have related cardiac function to filling pressure, and Starling’s observations, as well as Frank's, dealt with this parameter.

At any rate, the observations do indicate that circulatory congestion, with its associated increased intracardiac residual pressures, has
was maintained for 30 to 50 minutes, without knowledge or discomfort to the subject, without change in arterial pressure and, in the few instances measured, without change in cardiac output. Renal functions were then determined before, during, and after inflation of the balloon.

In these noncardiac subjects, congestion of the inferior vena caval drainage area, including the kidneys, produced prompt decreases in urine output and in the excretion of sodium and potassium (fig. 16). Arterial pressure remained essentially unchanged. Renal plasma flow and glomerular filtration rate were reduced immediately upon inflation of the balloon but returned well toward control levels as the congestion was maintained. Nevertheless, decreased water and electrolyte excretion continued throughout the period of congestion. Following deflation of the balloon and relief of venous congestion all functions returned to the control level.69-73

Congestion of the kidney was not essential to the decreases in water and electrolyte excretion. Congestion of the inferior vena cava below the renal veins induced similar decreases in urine flow and in sodium excretion, with no, or minimal, change in renal blood flow and glomerular filtration (fig. 17).68, 74, 75 Furthermore, congestion of the superior vena caval drainage area also produced decreases in water, sodium, and potassium excretion, again without significant change in blood pressure, renal blood flow, or glomerular filtration (fig. 18).68, 76 Unlike the response to inferior vena caval congestion, the reduced water and electrolyte excretions occurring during congestion of the superior vena cava appeared to endure beyond the period of venous congestion and persisted after deflation of the balloon.

These observations indicate that venous congestion of itself, at least apart from systemic arterial pressure and renal blood flow, acts to decrease the excretion of water and electrolytes. Furthermore, this effect does not require congestion of the kidney but appears to be related to the size of the vascular bed that is congested. When the kidney is in-

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**Table 1**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>D.L.</th>
<th>G.K.</th>
<th>A.B.</th>
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**Figure 15**

Effect of vasodilatation, produced by oral sodium nitrite, on cardiovascular dynamics in nonfailing circulation. Details of plotting are the same as in figure 14.

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the deleterious effect of decreasing the cardiac output and work of the diseased heart, and that relief of the congestion may be expected of itself to improve the function of such hearts.

Consider now the effect of venous congestion itself on the circulation and function of peripheral organs and again the kidney was chosen for study.

Farber and Becker produced, in noncardiac subjects, congestion of any desired segment of the vena caval vascular bed, both including and excluding the kidney. A catheter, equipped with a balloon, which could be inflated with Diodrast solution, was placed in the superior vena cava or in the inferior vena cava above or below the renal veins. By gradual inflation of the balloon, distal venous pressure was raised to 150 mm. to 250 mm. of water, levels similar to those in congestive heart failure. The venous congestion.
volved in the congested area, the effect is more pronounced.

The question obviously arises, whether these changes in circulation, cardiac function, and renal function, resulting acutely as they did

during relatively short periods of alteration in circulatory congestion, have any significance with respect to long-term effects in clinical congestive heart failure. Rader, Berger, and Smith have determined the hemodynamic effects resulting from long-term relief of venous congestion in patients with congestive heart failure. The aim was to relieve the venous congestion by noncardiac means. Control hemodynamic measurements were made on patients who had not received digitalis or diuretic therapy for at least 1 month and who were in typical, low-output congestive heart failure. Mercaptopurin, a mercurial diuretic considered to have no intrinsic cardiac effect, since the mercury is stabilized
In all instances mercurial diuresis relieved the circulatory congestion, lowered the venous pressure to normal, and often returned right ventricular end-diastolic pressure and right atrial pressure to normal levels. All patients were improved, usually to the extent that they could be considered clinically compensated. In half of the subjects, generally patients in their first or second episode of congestive failure, cardiac output rose, often considerably, as the congested state was relieved, and digitalization produced no further change in subjective improvement, vascular and cardiac pressures, or cardiac output (fig. 19). Relief of the venous congestion itself had effectively returned cardiac function toward normal. In a sense this result may be interpreted as an indication of the Starling law effect in a chronic situation.

In the remaining half of the subjects, usually in a repeated episode of congestive failure due to neglect to take medication, mercurial diuresis removed equally well the circulatory congestion, reduced the residual cardiac pressures, removed the edema, and produced equal subjective improvement in the patient, but cardiac output did not rise above that determined during congestive heart failure. Full doses of digitalis leaf now produced an increase in cardiac output, indicating in these subjects a cardiotonic effect improving cardiac function (fig. 20). Finally, in a third group of subjects, usually in chronic congestive heart failure with repeated decompensation developing quickly after lapse of therapy, cardiac output did not rise when venous congestion was removed by either diuretic or digitalis therapy, or both. Subjective improvement followed removal of the circulatory congestion, however, regardless of lack of improvement in cardiac function and cardiac output.

The observations thus far discussed clarify the relationship of symptoms to the congested state on the one hand and to cardiac output on the other. It is apparent that the classical manifestations of congestive heart failure, the dyspnea, orthopnea, tachypnea, tachycardia, venous distention, and edema are man-

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**Figure 18**

Effect of superior vena caval congestion on renal hemodynamic functions and water and electrolyte excretions; representative response. Details of plotting are the same as in figure 16. Vena caval congestion was induced by inflating a balloon in the superior vena cava just proximal to its entrance into the right atrium. (From Journal of Clinical Investigation 32: 1143, 1953.)

with sulfhydryl and hence avoids the possible cardiotonic effect of the xanthines used in stabilizing mercuriuride, was then administered repeatedly until the circulatory (venous) congestion had disappeared and the patient was edema-free and subjectively essentially recovered. Hemodynamic measurements were then repeated, usually 2 to 3 weeks after the initial measurements, to determine the effects of removal of the circulatory congestion. Digitalis leaf was next given orally until full digitalization was achieved, when a third set of measurements was made, now to determine whether the cardiotonic effect of this drug produced any hemodynamic effect beyond that obtained by relief of venous congestion by diuretic medication.

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*EICHHNA*
ifestations of circulatory congestion and not of the cardiac output itself. Circulatory congestion produces the symptoms regardless of whether the cardiac output is low, or high or normal. Once the congestion is removed, the patient is relieved of these manifestations, again regardless of whether the cardiac output remains high, low, or returns to normal. An interesting example of this relationship is indicated by patient C. McC. (fig. 21). Severe mitral stenosis had markedly curtailed her activity to the point that she suffered from incipient and overt pulmonary edema and required oxygen inhalation equipment for home use to secure relief. Her high pulmonary artery pressure (61/32 mm. Hg) indicated the severity of the pulmonary congestion. A mitral commissurotomy relieved the pulmonary congestion, as evidenced by the fall of pulmonary artery pressure to normal. With relief of the circulatory congestion respiratory distress disappeared and the patient discarded her now useless oxygen inhalation equipment. Two years after operation the cardiac output was found to be unchanged and just as low after operation as before it, both at rest and on exercise (fig. 21). And yet the patient had become normally active without symptoms and without developing congestive failure. She did, however, continue digitalis therapy.

Relief of symptoms, with persistence of a low cardiac output, is a common occurrence today in cardiac patients when judicious use of digitalis and diuretics prevents the development of circulatory congestion. Increased extraction of oxygen, and perhaps other substances, from the blood appears to constitute an effective mechanism compensating for poor myocardial activity and decreased blood flow. The lowered cardiac output, however, accounts for a very important symptom in heart failure, namely, fatigue, particularly on exertion. Too often a patient is considered "compensated" from heart failure because the objective and subjective manifestations of circulatory congestion have been removed, often by noncardiac medication, while the actual state of heart failure remains unrecognized in the fatigue and dyspnea that exertion induces. It is necessary to recognize and to give greater significance to the manifestations of fatigue as evidences of heart failure, and not to rely solely upon the nonspecific manifestations of circulatory congestion.

Lowered Cardiac Output and Survival

The third area to be considered concerns the relationship of cardiac output to activity and survival, after the onset of congestive heart failure. A continuing study is correlating long-term, follow-up hemodynamic measurements with clinical observations on patients recovered from acute congestive heart failure and maintained on digitalis and, when necessary, diuretic medication. The still insufficient data suggest that the duration of survival seems less dependent on the level of cardiac output than on elevation of intracardiac pressures. Patients recovered from their first episode of congestive heart failure and receiving

Figure 19

Comparison of effect of mercurial diuretic therapy and digitalis therapy on cardiac output in congestive heart failure. Plotted are subjects in whom cardiac output increased following diuretic therapy, without (generally) further increase following digitalis therapy. Each vertical panel represents 1 patient in congestive heart failure for the time represented by number below abscissa. The 2 horizontal lines encompass the normal range of values. Solid lines indicate values obtained during resting state, dotted lines during exercise. For each patient the first point indicates values during untreated congestive heart failure, the middle point indicates values after mercurial diuresis to "dry weight," the third point gives values following full digitalization carried out after the diuretic therapy. Panels with only 2 points give data after mercurial diuresis and digitalis therapy respectively, data during untreated congestive failure were not obtained in these patients.
Figure 20
Comparison of effect of mercurial diuretic therapy and digitalis therapy on cardiac output in congestive heart failure. Plotted are subjects in whom cardiac output failed to change after diuretic therapy, with increase in output following subsequent digitalis therapy (except for subjects in chronic heart failure with fixed low cardiac output). Details of plotting are the same as in figure 17.

maintenance digitalis therapy undergo hemodynamic determinations and are grouped by calendar years. The initial hemodynamic data and follow-up data of subjects surviving 5 years or longer are compared with similar data derived from patients dying in less than 5 years. There is little to choose between the 2 groups with respect to age or a series of hemodynamic factors, including total oxygen consumption, blood volume, arterial oxygen content and saturation, systemic arterial pressure, right atrial pressure, right ventricular end-diastolic pressure, cardiac output, and A-V oxygen difference. Certainly long survival has been consistent with low cardiac outputs and high A-V oxygen differences, attesting again the satisfactory homeostasis of increased oxygen extraction from blood in compensating for a lowered cardiac output. The one hemodynamic measurement that seems to separate the 2 groups is the level of right ventricular systolic, and pulmonary arterial, pressure: subjects in whom right ventricular pressure was elevated (systolic pressure in excess of 45 mm. Hg) usually died within 5 years of the onset of congestive failure, whereas subjects with lowest right ventricular pressures (normal range) have led fairly asymptomatic lives for 5 to 9 years. Why elevation of right ventricular pressure should have an adverse effect is not clear. It is established, however, that oxygen uptake by the myocardium is not so much a factor of the external work performed as it is a function of the tension that the myocardium is required to sustain.81-86 Obviously, then, the load on the myocardium is greater when hypertension is present. Furthermore, patients may sustain low, and even very low, cardiac outputs for a number of years without signs or symptoms of circulatory congestion or fatigue on effort. The progressively decreasing cardiac output with aging is well known.87, 88 In the aged, cardiac output is often reduced to levels as low as those in congestive heart failure, yet circulatory congestion does not develop. Serial hemodynamic data on a representative cardiac subject, now followed for 10 years, illustrate the paradox of low cardiac output associated with excellent activity (fig. 22). The patient initially presented with congestive heart failure which responded to digitalis and he became asymptomatic and edema free. He remains so and still takes digitalis. The unusual feature is the exertional activity that this man undertakes without symptoms. Over the 10 years he has continued to walk a great deal and prides himself on walking 30 to 50 city blocks and up 6 to 8 flights of stairs, all without stopping, dyspnea, or fatigue. During this period his cardiac output has remained in the range of severe congestive heart failure, 1.70 to 2.0 L./M.²/min. Even though circulatory congestion may have been removed by the medication taken, the level of cardiac output is so low that fatigue and dyspnea would be expected. It is important that the right ventricular pressure has remained essentially normal over 9 years. Here, then, is the paradox, why do circulatory congestion and exertional fatigue develop in one group of subjects but are absent in another group—both with apparently equally low cardiac outputs? No explanation is apparent for this discrepancy and since this discussion has been presented from the standpoint of the primary significance of cardiac output in congestive heart failure, the paradox is not a satisfying
Figure 21
Effect of mitral commissurotomy on cardiovascular dynamics in patient (C.M.C., a 40-year-old woman) with severe mitral stenosis. The postoperative data were determined 2 years after operation.

<table>
<thead>
<tr>
<th>Before Operation</th>
<th>After Operation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PA mmHg</strong></td>
<td><strong>RV mmHg</strong></td>
</tr>
<tr>
<td>6/5/31</td>
<td>6/0/5</td>
</tr>
<tr>
<td><strong>RA mmHg</strong></td>
<td><strong>BA mmHg</strong></td>
</tr>
<tr>
<td>4/6/40</td>
<td>139/71</td>
</tr>
<tr>
<td><strong>O₂ CONS. ML/A²/min</strong></td>
<td><strong>O₂ VOLS %</strong></td>
</tr>
<tr>
<td>120</td>
<td>16.03</td>
</tr>
<tr>
<td><strong>PA O₂ VOLS %</strong></td>
<td><strong>P A V O₂ VOLS %</strong></td>
</tr>
<tr>
<td>9.90</td>
<td>6.13</td>
</tr>
<tr>
<td><strong>CI L/min</strong></td>
<td><strong>Exercise</strong></td>
</tr>
<tr>
<td>1.95</td>
<td>2.47</td>
</tr>
</tbody>
</table>

Figure 22
Cardiovascular dynamics over course of 9 years in patient (W.G., a 71-year-old man) with arteriosclerotic heart disease. Initial congestive heart failure in 1949 responded to digitalis. Maintenance digitalis therapy continued thereafter.

<table>
<thead>
<tr>
<th>Date</th>
<th>1949</th>
<th>1950</th>
<th>1952</th>
<th>1955</th>
<th>1958</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHF</td>
<td>2+</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Medication</td>
<td>NONE</td>
<td>DIGITALS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>O₂ CONS. ML/A² MIN</td>
<td>124</td>
<td>117</td>
<td>104</td>
<td>111</td>
<td>115</td>
</tr>
<tr>
<td>FA O₂ VOLS %</td>
<td>1.26</td>
<td>1.58</td>
<td>1.20</td>
<td>1.66</td>
<td>1.69</td>
</tr>
<tr>
<td>PA O₂ VOLS %</td>
<td>5.04</td>
<td>8.73</td>
<td>10.90</td>
<td>10.34</td>
<td>10.96</td>
</tr>
<tr>
<td>A-V O₂ VOLS %</td>
<td>7.57</td>
<td>6.46</td>
<td>6.10</td>
<td>6.34</td>
<td>5.74</td>
</tr>
<tr>
<td>C.I. L/min</td>
<td>1.64</td>
<td>1.61</td>
<td>1.70</td>
<td>1.75</td>
<td>2.06</td>
</tr>
<tr>
<td>PA mmHg</td>
<td>35.6</td>
<td>35.6</td>
<td>34.10</td>
<td>32.64</td>
<td>—</td>
</tr>
<tr>
<td>RV mmHg</td>
<td>37.13</td>
<td>34.8</td>
<td>20.5</td>
<td>33.1</td>
<td>—</td>
</tr>
<tr>
<td>RA mmHg</td>
<td>12.6</td>
<td>6.4</td>
<td>6</td>
<td>9.10</td>
<td>10</td>
</tr>
<tr>
<td>FA mmHg</td>
<td>132/88</td>
<td>137/77</td>
<td>128/65</td>
<td>153/63</td>
<td>113/65</td>
</tr>
</tbody>
</table>

one at which to arrive. It is, nonetheless, a paradox at which our present information brings us.

Speculation into the future may not be amiss at this point. In consideration of heart failure the clinician has traditionally equated heart failure with circulatory, that is venous, congestion. The observations here presented indicate that the two are not synonymous. More recently, hemodynamic measurements have tended to equate heart failure with either low cardiac output or low cardiac external work, parameters that are concerned with what the heart does for the total circulation, for the body as a whole. This function of the heart is obviously important, for upon it depends the survival of the total organism and from its inadequacy follow such abnormal circulatory phenomena as circulatory congestion. Focus upon the external work of the heart, however, loses sight of myocardial cellular events. It is apparent that for a given external work accomplished, myocardial cellular metabolism may be relatively normal or very abnormal, depending upon different situations of cardiac output and the pressure against which the output is delivered.81–86

After all, the myocardium is a muscle and it is an anatomic happenstance that its contraction expresses blood, physiologically vital though that blood may be for the organism. But with respect to the heart muscle itself, it is the intrinsic cellular metabolism, and that metabolism alone, which determines whether the heart is contracting normally or is failing.

The relationship between myocardial tension and oxygen utilization, suggested by both animal experiments and clinical experience in man, indicates a fruitful area of investigation in the problem of heart failure. It is trite to say that cellular biochemical relationships will resolve the problems of the failing heart, for they will. Until that day, it is well for the clinician and the physiologist to begin considering the patient with heart disease not from the standpoint of circulatory congestion nor cardiac output and cardiac external work, but from the standpoint of the myocardial cell and its metabolism. Clues to that metabolism should be sought, not only in experimental determinations, but also at the clinical level. Such clues may give a better indication, than congestion or output, of cardiac function and indicate whether the heart muscle is normal or failing, and failing badly or mildly. Certainly new parameters are required to indicate the true status of the heart in many clinical situations, for example, the noncongested cardiac patient vigorously treated with digitalis and diuretics, severe cardiac failure without congestive changes, the tachycardias, sudden heart failure, and retention of activity in spite of low output.

In recapitulation, and in closing, several points seem clear: (1) Circulatory (venous) congestion is the hemodynamic disturbance responsible for the symptoms usually associ-
ated with congestive heart failure; removal of the congestion, regardless of how accomplished, relieves the symptoms. (2) Circulatory congestion is a nonspecific hemodynamic disturbance and may arise when the heart does not fail as a pump. This is noncardiac circulatory congestion. (3) The term congestive heart failure should be reserved for those states of circulatory congestion in which there is myocardial failure. (4) Circulatory congestion of itself has undesirable effects on the heart and impairs cardiac function when the heart muscle is already involved. (5) Circulatory congestion of itself affects the kidney to impair its function in excreting salt and water. (6) A low cardiac output is a preferred indicator of heart failure and is the function responsible for the symptoms of exertional dyspnea and fatigue, often overlooked as symptoms of heart failure. Less clear-cut points are (7) Starling’s law of the heart appears to hold in intact man in both the congested and noncongested circulatory states. (8) A low cardiac output is consistent with survival for years and appears to carry a less dangerous prognostic significance than elevation in ventricular pressures. A totally unclear and paradoxical point relegated for future resolution is, (9) the relationship of low cardiac output to adequate activity on the one hand and congestive heart failure on the other. A speculation (10) would indicate that in the future heart failure will be considered not from the standpoint of resultant gross manifestations (circulatory congestion) or external work performed by the heart (cardiac output and cardiac work) but from the standpoint of intrinsic myocardial metabolism, regardless of what the clinical manifestations or external cardiac work may be.

**Acknowledgment**

I want very much to express my deep appreciation to all my colleagues, whose ideas and work I have here presented, to the investigators, Drs. Roy E. Albert, J. Deaver Alexander, Adolph R. Berger, Saul J. Farber, Arthur C. Fox, Richard H. Kessler, Edmund D. Pellegrino, Bertha Rader, Warren W. Smith, Bruce J. Sobol, Maria F. Stack, Harry Taube, Norman S. Wikler, Sol Youngwirth, and Albert M. Ziffer, and to the technical staff, Flavio Ameiro, Bernice Aparo,


**Summario in Interlingua**

Le conferentiaro recapitulava su presentation in le sequente punctos:

1. Congestion circulatoria (i.e. venose) es le disturbation hemodynamic responsabili pro le symptomas que es usualmente associate con congestive disfallimento cardiale. Le resolution del congestion—sin reguardo al natura del mesure usate—allevia le symptomas.

2. Congestion circulatoria es un nonspecifica disturba
tion hemodynamic que pot occurri sin que le corde manifesta ulle dysfunction in su labor de pumpa. In tal caso le congestion circulatoria es noncardiac.

3. Le termino "congestive disfallimento cardiale" debera esser reserve al statos de congestion circulatori in que il existe un disfallimento myocardial.

4. Le presentia de congestion circulatori per se produce effectos adverse in le corde e resulta in un vitiation del function cardine si le musculo del corde es jam affide.

5. Congestion circulatori per se affiche le reues con le resultato de dysfunction del mehismono renal de exeretion de sal e aqua.

6. Un basse rendimento cardiale es un excellent indicator de disfallimento cardiale. Illo es le causa responsabile pro le symptomas de dyspnea post ef
tortio e de fatiga (que es frequentemente neglige
te como manifestations de disfallimento cardiale.

7. Il pare que le lege del corde, formulate per Starling, es valide in le homine intacte in stato circulatori tanto congestionate como etiam non congestionate.

8. Un basse nivello del rendimento cardiale es compatibile con le superviventa del patiiente durante longe annos. Illo pare esser minus hasardose ab le puncto de vista del prognosis que un nivello elevate del tension ventricular.

9. Un question ancora completely obscur e paradoxe concerne le relation inter basse rendimentos cardiale e le duple possibilitate de activitate adequate o congestive disfallimento del corde. Iste question debe esser resolvite in le futuro.

10. Il es permittite speculare que in tempore veni
cente le disfallimento del corde va esser considerate minus ab le puncto de vista del resultant manifestations grossier (i.e. le congestion circulatori) o ab le puncto de vista del externe travaliu effectuate per le corde (i.e. rendimento cardiale e labor cardiale) se il plus tosto ab le puncto de vista del metabolismo intrinsic del myocardio, sin reguardo al character del manifestaciones clinica o al externe travaliu cardiale in le caso individual.

*Circulation, Volume XXII, November 1960*
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Research is not something divorced from the ordinary activities of the wards or laboratory to be carried on in an exclusive way by some special group. It is a mode of working involving a mental attitude, a lively curiosity, an alertness in face of the unusual, an eagerness to utilize what fortune brings before one in the way of material for supplying gaps in our knowledge.—C. MacFie Campbell, M.D., late Professor of Psychiatry, Harvard University. *Annual Report of the Boston Psychopathic Hospital*, 1936, p. 11.
The George E. Brown Memorial Lecture: Circulatory Congestion and Heart Failure
LUDWIG W. EICHTNA

doi: 10.1161/01.CIR.22.5.864
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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