The Clinical Recognition of Congestive Heart Failure

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The recognition of early or mild congestive heart failure in the ambulatory patient is a common clinical challenge in everyday practice. Early diagnosis requires attention to symptoms, signs, and radiographic changes which may be minimal. This paper reviews basic pathophysiological principles involved in congestive heart failure and summarizes etiological factors which may cause or precipitate congestive heart failure. The symptoms, signs, and subtle radiological findings of early congestive heart failure are also described in some detail.

Few physicians have difficulty recognizing congestive heart failure in a patient with pulmonary edema or gross peripheral edema. This review stresses some of the salient features of heart failure. Our purpose is to help the clinician recognize cardiac failure in an ambulatory patient with minimal and supposedly nonspecific symptoms.

For the practicing physician, congestive heart failure is best defined in terms of a constellation of symptoms and physical findings. Nevertheless, the conceptualization of certain basic pathophysiologic observations makes the clinical features of the condition more meaningful. Figure 1 describes the classic Starling's law in which some measurement of systolic performance is plotted against some measurement of end-diastolic dimension of the left ventricle. If the dotted line represents the amount of systolic performance required for the human organism in the resting state, one can see that the normal heart can provide this requirement with little or no stretching. The characteristics of the failing heart are considerably different. In order to maintain the same level of systolic performance, the failing ventricle must be markedly stretched.

The first hallmark of a failing heart is an enlarged, stretched ventricle. This allows the ventricle to enhance contraction. Since diastolic pressure increases with diastolic volume in the failing heart, the second hallmark of ventricular failure is elevated pressure transmitted from the left ventricle back into the atrium, pulmonary veins, and capillaries. The pressure in the pulmonary venous and capillary system is normally a low pressure system. (From 5-6 mm Hg up to 10-12 mm Hg is considered normal.) In congestive heart failure the transmitted pressure exceeds the oncotic pressure of the blood and there is a movement of fluid from the pulmonary venous system into the interstitial space of the lungs. With normal plasma proteins and in the absence of pulmonary infection this occurs when the pulmonary capillary pressure exceeds 25 mm Hg. Much of the symptomatology of congestive heart failure is due to pulmonary mechanics that are altered by the presence of this fluid in the interstitial space. This will be expanded below.

When one is entertaining the diagnosis of congestive heart failure, one is obligated to simultaneously ask two additional questions: (1) What is the nature of the underlying heart disease? and (2) Are there factors or conditions that have precipitated the development of congestive heart failure? Table 1 is a clinically useful classification of the etiology of congestive heart failure. The most common underlying cause is arteriosclerotic heart disease. This diagnosis is established by having an associated history of angina pectoris, a history of a myocardial infarction, or definite electrocardiographic evidence of ischemic heart disease at rest or following exercise stress testing.

Table 2 is a list of factors known to precipitate congestive heart failure. Acute myocardial infarction heads this list. When a patient suddenly develops congestive heart failure or acute pulmonary edema, it is sound medical practice to assume that he/she has sustained an acute myocardial infarction until proven otherwise. Iatrogenic causes include the patient's failure to take medication and fluid overload. With a marginally compensated heart as little as 100 or 200 cc of saline given rapidly may precipitate heart failure. The presence of pulmonary emboli is frequently difficult to document. An extensive review of the clinical features of pulmonary emboli has been published recently.[1] Pulmonary infections should always be considered since appropriate therapy can reverse the process. The rupture of a chordae tendineae cordis presents a catastrophic clinical picture which is associated with the sudden appearance of a mitral insufficiency murmur. The less common factors that precipitate congestive heart failure listed in Table 2 are actually rare. Since they are all amenable to appropriate therapy, overlooking these factors would be a great disservice to the patient.

Symptoms

Cough

It was Paul Dudley White[2] who, in 1947, brought to our attention the fact that cough may be an early and predominant symptom of cardiovascular disease. A summary of this paper reads: "The cough may be chronic, spasmodic, or paroxysmal . . . . Congestion of the lungs is undoubtedly the most frequent cause for the cough associated with heart disease, and the pulmonary congestion is usually due

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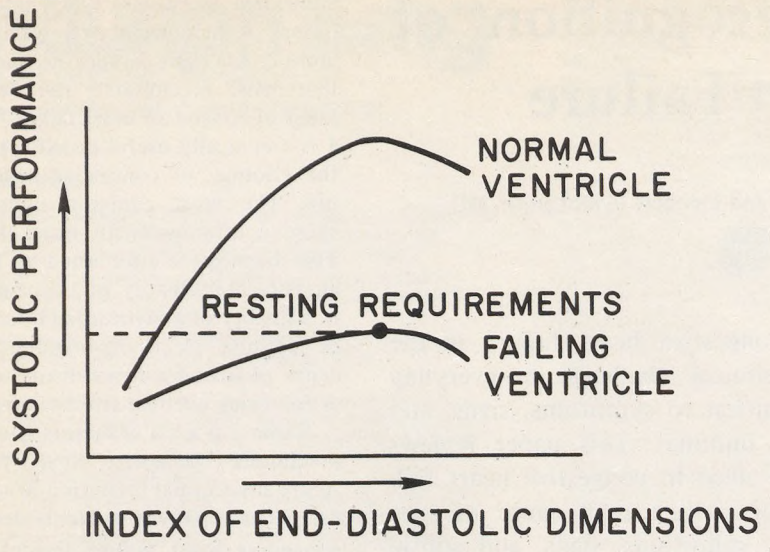

Figure 1. Relationship of systolic performance to end-diastolic dimensions in the normal and failing ventricles. In the failing heart the required systolic performance is accomplished by a marked stretching of the ventricle during end-diastole. This increased diastolic volume is accompanied by an increased diastolic pressure.

Table 1. Etiology of Congestive Heart Failure

Rank Order	Relative Frequency
1. Arteriosclerotic heart disease	60-70%
2. Hypertensive heart disease	15-20%
3. Rheumatic heart disease	5-10%
4. Congenital heart disease	1-5%
5. Cor pulmonale	1-5%

Table 2. Factors that precipitate Congestive Heart Failure

Common

1. Acute myocardial infarction
2. Iatrogenic (including patient noncompliance)
3. Pulmonary emboli
4. Pulmonary infection
5. Ectopic rhythms

Less Common but amenable to therapy

1. Thyrotoxicosis
2. Anemia
3. Bacterial carditis
4. Prostatic hypertrophy
5. Rupture of chordae tendineae cordis

to left ventricular failure . . . . It may be only after brief exertion that the patient notices a cough. At other times the cough is chronic and may be particularly bothersome at night. It frequently is a contributing cause of insomnia . . . . The cough is often a precursor of paroxysmal dyspnea." The true nature of this cough may be obscured by its appearance in the clinical setting of an upper respiratory tract infection. A clue that there is underlying heart disease may be that the respiratory infection is unusually severe or prolonged. If one suspects that a persistent cough is due to underlying congestive heart failure, one should search for the confirmatory physical or x-ray findings that are discussed in detail below.

Nocturia

In a classic review, Eugene Stead[3] noted "certain clinical observations have been made repeatedly . . . . Water drinking and urine making become dissociated. Most of the water is taken during the day and most of the urine made at night. Swelling during the day and nocturia is a usual story." Figure 2 helps explain this phenomena. The renal plasma flow of normal patients is not affected by mild exercise. In contrast, the renal plasma flow of patients with congestive heart failure is markedly decreased with exercise. This suggests that while ambulatory and exercising, there is a decreased renal blood flow, decreased filtration rate, and increased fluid retention in cardiac patients. When the cardiac patient becomes recumbent and rests at night, the renal plasma flow returns toward normal, the filtration rate increases, and a large volume of urine is produced.

Exertional Dyspnea

Harrison[4] proposed that the subjective sensation of dyspnea occurs when the quotient Ventilation ÷ Vital Capacity exceeds a critical value for a given individual. Ventilation is the total movement of air in the lungs over a given period of time. Vital capacity is the amount of air that is expelled with a maximal expiratory effort following a maximal inspiratory effort. In Harrison's study, which has been repeatedly confirmed, the vital capacity is lower than normal in patients with congestive heart failure. In his experimental study of the mechanism responsible for development of dyspnea with exercise, he found that the vital capacity did not undergo further reduction with exercise. The dyspnea with congestive heart failure is produced by total ventilation that increased more during exertion and remained elevated longer after exertion than did normal subjects. He suggested that afferent impulses from the moving muscles play a role in the production of dyspnea because of a reflex increase of ventilation during exertion. Frank[5] showed that for each stage of functional impairment in patients with congestive heart failure there is a progressive decrease in measured vital capacity. Christie[6] suggests that this decreased vital capacity is due to a stiffness of the lungs secondary to the interstitial edema. He showed that a coefficient of distensibility (the force required to distend the lung by 20 percent of the functional residual air) was two or three times greater for patients with congestive heart failure than for normal control patients. This measurement of stiffness decreases in a given patient following successful therapy of heart failure.

Orthopnea

Becoming short of breath in the recumbent position is a symptom suggestive of congestive heart failure. It is also found in patients with chronic

pulmonary disease. As one moves from the upright to the recumbent position, there is a sudden increase in the venous return from the legs and splanchnic viscera. In normal patients it has been demonstrated that the vital capacity can decrease about five percent from the upright to the horizontal position. In patients with congestive heart failure with symptoms of orthopnea, the decrease in vital capacity may be as great as 25 percent.[7] Returning to Harrison's ratio, one can appreciate why this marked decrease in vital capacity would produce the symptom of dyspnea. In addition, there are mechanical factors relating to dyspnea such as the elevation of the diaphragm into the chest.

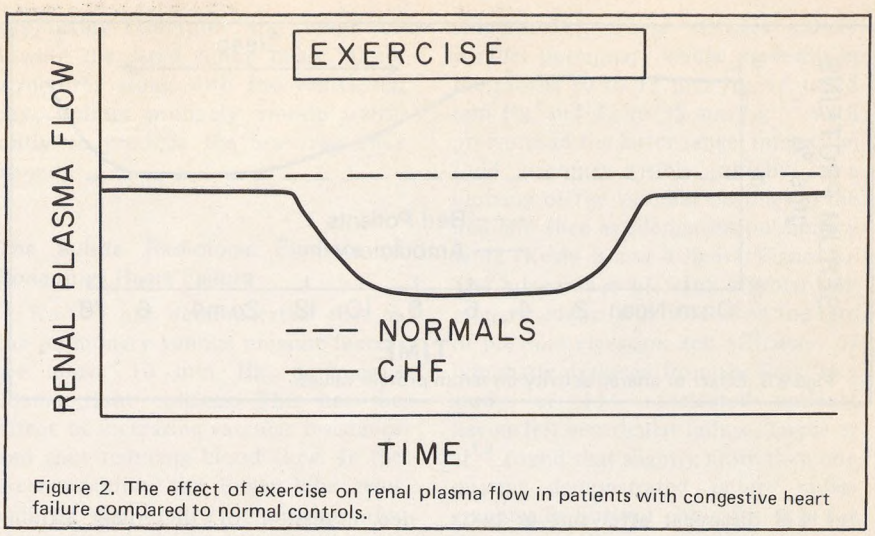

Figure 2. The effect of exercise on renal plasma flow in patients with congestive heart failure compared to normal controls.

Paroxysmal Nocturnal Dyspnea (PND)

PND is one symptom that is relatively specific for cardiac disease. A similar symptom can on occasion be produced in a patient with chronic lung disease as a consequence of pooling secretions during sleep at night. It is important to obtain a thorough history. Classically, the patient has been asleep in the recumbent position for a number of hours. He/she is awakened by a terrifying sensation of suffocation and is forced to sit up or stand up. He/she may try to get additional air by opening a window. This experience lasts for minutes to hours, averaging about one hour. There are a number of theories concerning the mechanisms that are responsible for the production of this symptom. A major contributing factor is increased venous return as peripheral edema fluid slowly reenters the vascular system when the patient assumes the recumbent position. The major support for this theory is found in the work by Perera and Berliner.[8] Figure 3 is a schematic representation of the effect of altered activity on serum protein values. You will note that bed patients have a lower than normal protein value which does not fluctuate. Ambulatory patients with congestive heart failure have a significant drop in serum protein values during the recumbent sleeping hours. This is secondary to hemodilution. Attacks of PND occur in the early morning hours when the serum proteins are at the lowest level. Also, these investigators demonstrated that patients with PND developed a decrease in serum proteins, an increase in venous pressure, and a decrease in vital capacity a few hours after going to sleep. Within a few hours they developed an attack of PND. When these studies were repeated after the acute episode, all three values returned toward control figures. These authors state that "it seems probable, therefore, that in the patient already subject to left ventricular strain by virtue of underlying organic disease, the increase in circulating plasma volume accompanying rest in the horizontal position may prove a significant factor inducing PND and pulmonary edema. The effect of the horizontal position upon the resting individual is comparable to the administration of a small but sustained infusion."

Wheezing or Cardiac Asthma

Wheezing represents a variation of PND. Paul Dudley White[9] carefully followed 250 cases and made the following observations. Cardiac asthma is paroxysmal, coming on during sleep or following exertion, producing both inspiratory and expiratory distress. The patient is forced to sit up or stand and the attacks last an average of one hour. The physical finding of wheezing is produced by a 75 percent or greater narrowing of the larger airways. Cardiac asthma, a true reflex hyperexcitability of the bronchial musculature, is relieved by epinephrine. Although there is no known ill effect from small doses of epinephrine, the conventional treatment for cardiac asthma is morphine sulfate. The prognosis of patients with cardiac asthma is poor, with over 50 percent of patients dying within two years of the initial onset of the symptom.

Why does wheezing occur in some patients with PND and not others? Allergists[10] believe that cardiac asthma occurs in those patients in whom asthma or hay fever were present prior to the onset of heart failure. There are some general clues that the wheezing is of cardiac origin. Those patients who begin to wheeze for the first time after the age of 40 are more likely to have cardiac asthma. Accompanying signs of congestive heart failure, such as elevated neck vein pulsations, a ventricular (S-3) gallop, or x-ray changes suggest the presence of heart disease. In actual practice, the suspicion that repeated bouts of nighttime wheezing represent cardiac asthma may be confirmed if the symptoms disappear after treatment with digitalis.

Physical Findings of Congestive Heart Failure

Rales

The moist rales that are frequently heard in the setting of congestive heart failure are produced by the opening of small airways. The presence of interstitial pulmonary edema with the accompanying stiffness of the lungs account for the closure of small airways. Deep inspiration will open these airways. Rales may be heard only on the dependent side and may shift to the opposite side on turning the patient. Those rales that appear secondary to shallow respiration or perhaps atelec-

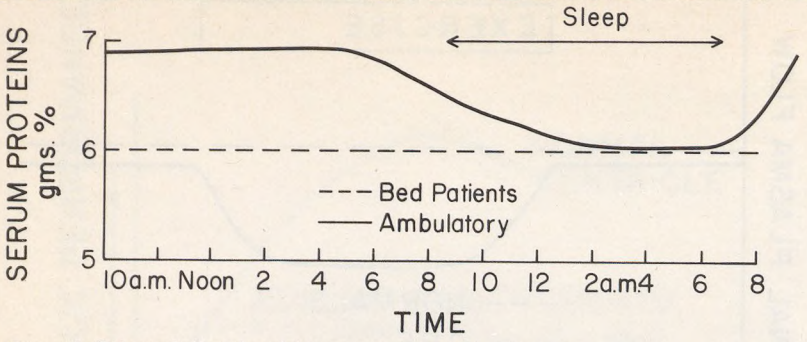

Figure 3. Effect of altered activity on serum protein values.

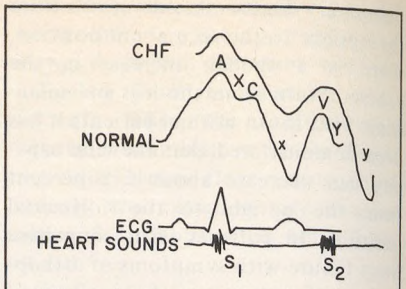

Figure 4. Temporal relationship of venous pulsations and heart sounds.

tasis will disappear after one or two deep breaths. The rales of congestive heart failure will persist. Congestive heart failure may be present without rales and rales may be secondary to noncardiac conditions.

Elevated Neck Vein Pulsations and Hepatojugular Reflux

The careful and proper examination of the pulsations of the internal jugular vein can be a helpful aid in the clinical diagnosis of congestive heart failure. It is important that the patient be properly positioned. The neck vein pulsations are seen best with the head elevated by a 30 to 45° flexion at the waist. A small pillow behind the neck will relax the sternocleidomastoid muscles. For a number of theoretical reasons the right internal jugular vein best reflects central venous properties. The patient's head may be turned slightly to the left. A light shown tangentially over the neck will facilitate visualization of the pulsations. Examination of the neck vein pulsations is most meaningful with the simultaneous auscultation of the heart sounds. The specific wave form can be related to the heart sounds.

Figure 4 demonstrates what is ideally seen. The atrial contraction forms the A-wave which is seen at the same time that the first heart sound is heard. The C-wave is probably produced by transmission of pressure through the closed tricuspid valve at the onset of systole but is difficult to see with the naked eye. The more obvious observation is the X descent. As venous return continues to fill the atrium during systole, the venous pressure increases and produces the V-wave. The Y descent is caused by the decompression of the right atrium with the opening of the tricuspid valve and ventricular filling. The Y descent or collapse is seen after the second heart sound. Clinically, the X and Y collapses or descents are more obvious than the peak of each wave.

The carotid arterial pulse frequently adds confusion. One can feel the carotid in the left neck while simultaneously observing the right. The upstroke of the carotid pulse will coincide with the more undulating fall of the jugular pulse, ie, the X and Y descents. Pressure with a pencil slightly above the clavicle will occlude the internal jugular and, by obliterating the venous pulsation, will leave only the carotid pulsation.

A cardiologist armed with pressure tracings can infer much from the contour of these specific waves. In congestive heart failure, the configuration of the pressure tracing itself is not unlike that seen in normal control patients. In this clinical setting, the physician is looking for an increased height of the neck vein pulsations which is a reliable indication of the central venous pressure. At a 30 to 45° elevation, the height of the A-wave should not be more than 3 to 4 cm above the sternal angle of Louis. The proper way to record this observation is that the height of the pulsations of the internal jugular pulse is ___ cm above the sternal notch, with the patient at ___° flexion. The standard description that there is or is not neck vein distention is not precise or helpful. The only vein that will become obviously distended is the external jugular vein.

When measuring the height of the venous pulsations, it is important to do so during inspiration. Expiration, particularly in patients with chronic obstructive lung disease, will artificially elevate the pulsations because expiration increases the intrathoracic pressure.

Incipient or well-compensated congestive heart failure may not be accompanied by an elevation of the neck vein pulsations in the resting state. Minimal exercise or a sudden increase in venous return may produce demonstrably increased central venous pressure. Looking for hepatojugular reflux is a helpful maneuver. Firm but gentle pressure in the right upper quadrant of the abdomen will increase venous return to the heart as blood is "squeezed" from the splanchnic viscera. In normal individuals, there will be a transient elevation of central venous pressure for a few cardiac cycles. This elevation will persist in patients with congestive heart failure. It is important to sustain the pressure on the abdomen for 30 to 60 seconds and to make the observations at the end of that time. Again, the level of neck vein pulsation is recorded during inspiration. A positive hepatojugular reflux means that there is a significant and sustained rise in the level of neck vein pulsations with pressure over the abdomen. This is a reliable sign for congestive heart failure.

Ventricular Gallop (S-3)

The development of a ventricular gallop in an adult is very suggestive of congestive heart failure. The extra sound can be heard in children and adolescents but is rarely heard in adults over 30 years of age without significant heart disease being present. In fact, when this gallop rhythm develops in a patient with underlying heart

disease, it is an indication for initiating therapy. Figure 5 demonstrates the timing of the S-3 gallop. This occurs early in diastole during the rapid filling stage of the ventricle. This is a low-pitched sound, which is heard best with a bell at the apex or along the left sternal border. The exact cause of this sound is not known; however, a currently popular theory is that during the rapid filling phase of the ventricle, there is a sudden checking of motion on the ventricular wall. The A-V valve supporting structures may tense following the rapid filling phase. These structures along with the ventricular myocardium probably vibrate transiently to produce the low frequency sound.

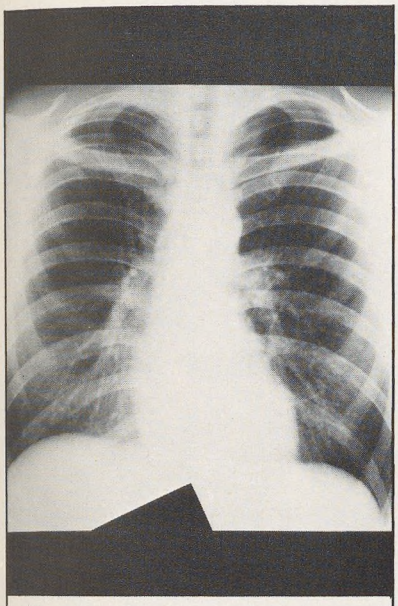

Figure 5a. Normal erect PA shows upper lobe vessels to be half the size of those in the lower lobes.

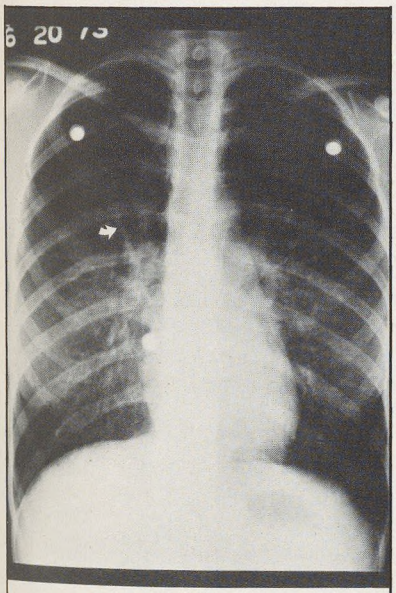

Figure 5b. Patient with pulmonary venous pressure of 25 mm Hg shows enlarged upper lobe pulmonary veins (arrow) and 2:1 upper to lower size ratio.

The Subtle Radiologic Findings of Congestive Heart Failure

West[11] has demonstrated that as the pulmonary venous pressure increases above 10 mm Hg, perivenous edema fluid collects. This has the effect of increasing vascular resistance and thus reducing blood flow. In the erect position, the lower lobe veins will be the first to experience this change because of the hydrostatic component in those veins below the level of the left atrium. Consequently, there will be a selective increase in vascular resistance in the lower lobe and hence, for any given level of cardiac output, increased blood flow through the upper lobes. It is this change that is visible radiographically.

A good quality posteroanterior radiograph exposed in the erect position is desirable for the assessment of early congestive heart failure. Although it may be difficult for the patient, the trouble obtaining this is justified. Most such patients will be more comfortable erect and, if necessary, at least an anteroposterior projection can be obtained without moving from either stretcher or chair.

A normal chest radiograph (Figure 5a) shows upper lobe vessels to be approximately half the size of the lower lobe vessels for any given distance from the hilum. With increasing pulmonary venous pressure, there is a demonstrable change in this ratio from 1:2 to 1:1, 2:1, and finally, 3:1 (Figure 5b). These changes closely parallel pulmonary wedge pressures in the ranges 10 to 15 mm Hg, 15 to 25 mm Hg, and 25 to 35 mm Hg.[12] With pressures in the latter range, interstitial fluid becomes visible initially as a blurring of the vascular outlines in the hila and then as edematous pulmonary septa (Kerly B and A lines) (Figure 6). The appearance of frank alveolar pulmonary edema will depend on the rate of pressure elevation and efficiency of lymphatic drainage from the lung. In a study of 114 unselected patients having left ventricular failure, Logue et al[13] found that slightly more than one quarter demonstrated failure radiographically but not clinically. It is for this reason that a careful analysis of the differential sizes of pulmonary vessels is urged so that the physician may be better prepared for a sudden deterioration in the patient's condition. However, care must be taken not to make a false-positive diagnosis in patients with diminished ventilation of the lung bases. This would be obvious in the presence of pulmonary infiltrates but can be less obvious with basal emphysema in α-1 antitrypsin deficiency or when there is poor diaphragmatic excursion for any reason.

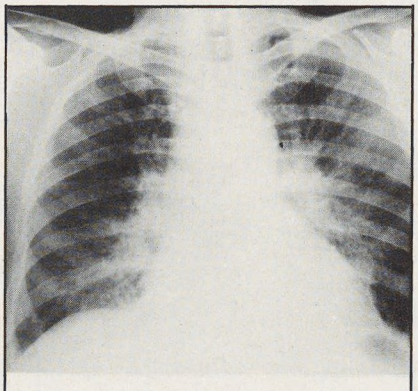

Figure 6. Severe Congestive Heart Failure, a 3:1 ratio, and early interstitial edema.

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