CLINICAL PROGRESS

Congestive Heart Failure

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THE physician, when consulted by a patient whose symptoms simulate a cardiovascular disorder, proceeds to diagnosis in systematic fashion. He determines first whether such symptoms as fatigue, dyspnea, palpitation, pain, or edema represent exaggeration of physiologic responses or whether they result from pathologic alterations of the circulation. When clinical decision favors the latter, differentiation is then made between symptoms resulting from ischemic or inflammatory changes in heart muscle, which initiate pain or disturbances in rhythm and conduction, and those related to failure of the heart as a pump, which produce the clinical picture of congestive heart failure. If symptoms point to disturbance in the ability of the heart to pump blood, he searches for objective evidence of organ dysfunction such as weakness, decreased vital capacity, pulmonary rales, increased venous pressure, engorgement of the liver, and edema. If both the history and physical findings support the diagnosis of congestive heart failure, the next step involves a clinical estimate of cardiac output. If there is considerable precordial activity, loud heart sounds, warm extremities, capillary pulse, pistol-shot sounds over the large arteries, and a low diastolic blood pressure, the cardiac output is elevated and heart failure has occurred in a setting of unusual demand for blood. Thyrotoxicosis, nutritional deficiency, anemia, patent ductus arteriosus, arteriovenous fistula, cirrhosis of the liver, and extensive Paget’s disease of bone must be evaluated.

If the resting circulation is decreased, he considers first the common and then the rare types of heart disease that depress myocardial function. He attempts to establish the etiology of the heart disease and the morphologic changes produced by disease because these factors will vary his choice of therapy. He looks for secondary factors, infection or infarction, that may make the primary disease more severe. He reviews the course of treatment to determine if any present difficulties are manifestations of too vigorous therapy.

This paper reviews the more striking disturbances in physiology produced by heart failure, points out the importance of an accurate etiologic and morphologic diagnosis in relation to therapy, and differentiates between disturbances resulting from heart failure and those produced by treatment.

DISTURBANCES IN PHYSIOLOGY

The physiologic disturbances in congestive heart failure have always fascinated students of clinical medicine. Knowing that the heart supplies the peripheral tissues with blood, one might expect heart failure to produce a clinical picture of dry rot comparable to that seen in arterial occlusive disease. Instead, one finds no evidence of clear-cut nutritional deficiency in the organs but a complicated picture of weakness, dyspnea, blood and water logging of various organs, and generalized edema. The clinical picture produced by the decreased pumping action of the heart is one of organ dysfunction and congestion rather than of organ death. This is possible because organs receive blood for 2 purposes: to sustain life of the organ, and to perform the special functions that are important to the economy of the entire organism. When blood supply is reduced, the life of the tissue may not be threatened, but its capacity to perform the various specialized functions whose coordination results in smooth body function may be greatly reduced. For example, the kidney will not suffer from ischemia sufficient to produce structural damage if its blood flow is reduced to one fifth of the normal level; however, there will be

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many functions that the normal kidney can perform that the kidney with the reduced blood supply cannot.

The striking effect of the decrease in blood supply on specialized organ function has caused many persons to emphasize the peripheral effects of heart failure. We have lung failure secondary to heart failure, likewise liver failure and kidney failure. The heart, the organ directly involved and producing these secondary effects, may be completely silent. When localized injury to the heart occurs from coronary artery disease, the heart hurts and the clinical picture of angina pectoris and myocardial infarction is produced; when the pumping action of the heart is interfered with, we have congestive heart failure with no dramatic symptoms pointing directly to the heart.

Regardless of the cause, all congestive heart failure is characterized by a decrease in pumping ability of one or both ventricles. Organ dysfunction results from (a) acute congestion of organs, particularly the lungs, because one ventricle delivers blood faster than the other ventricle can remove it, and (b) decreased blood supply because of the sick heart. In acute heart failure produced either by a rapid decrease in muscle strength from myocardial infarction or by a sudden increase in work load from heavy exercise, acute pulmonary congestion resulting from left ventricular failure dominates the picture. In generalized chronic heart failure diminished organ blood flow greatly influences the clinical picture. In many patients a combination of decreased organ blood flow and of ventricular imbalance is seen.

No absolute level of cardiac output can be correlated with the presence or absence of congestive failure. The heart must pump enough blood to supply properly all of the tissues of the body and this amount of blood will vary depending on the level of organ activity. In myxedema the cardiac output may fall to low levels without the development of congestive failure. Organ blood flow will be reduced but in proportion to the degree of inactivity produced by the myxedema. In hyperthyroidism skin and muscle flow are elevated and the cardiac output is above the normal level. When the output falls to a level below that required by the hyperthyroid patient, signs of heart failure develop even though the output may be higher than that of a resting subject with a normal heart. Likewise in anemia, the heart must pump more of the thin blood and its output is increased. A decrease in output below the level needed to properly supply the organs with the thin blood will result in heart failure, though the output may still exceed that of a normal subject of similar size. The administration of packed red blood cells to an anemic subject will cause the cardiac output to fall. Heart failure may disappear as the cardiac output falls because a smaller amount of normal blood can supply the needs of the body.

In the natural course of slowly progressing heart failure, symptoms develop with a cardiac output above the resting level. Dyspnea and edema commonly appear during periods of work activity and disappear when the patient can rest from Friday night to Monday morning. During the week his heart is stimulated by his increased physical activity and responds by an increase in output. The rise is less than the needs of the body and symptoms of congestive failure develop. During the weekend he is able to rest. The heart works less but manages to supply the organs with all of the blood they need and the symptoms of congestive failure disappear.

When failure develops in patients who have illnesses that cause an increased cardiac output at rest, they will have a somewhat elevated output with failure. Their outputs are high but not high enough because of heart disease. When no complicating illnesses are present, heart failure persisting at rest is associated with a fall in cardiac output. Even though there is no reason on purely physiologic grounds for distinguishing between those patients with failure who have a high cardiac output and those who have a low output, there is a definite therapeutic reason. If the condition causing the hyperactive circulation can be corrected, the heart may prove entirely adequate for a normal load.

Fatigue is one of the dominant symptoms of congestive failure. Muscular exercise causes a large increase in blood flow to muscles and an
increase in cardiac output. If the heart is unable to supply the muscles with the extra needed blood, metabolism is altered and fatigue results.

Patients with congestive heart failure become short of wind. In more advanced failure they are dyspneic at rest and have an increased ventilatory volume. In a broad sense the dyspnea of heart disease is analogous to that caused by any diffuse disease of the lung. Failure of the heart has caused lung disease. Study of the respiration shows a decreased vital capacity and a decreased volume of residual air. It takes more effort to move air in and out of the stiffened lungs, and the muscles of respiration with an impaired blood supply become fatigued.

The lungs are engorged in congestive failure for 2 reasons. 1. Sudden and dramatic failure of the left ventricle causes the lungs to be flooded with blood. The blood accumulating in the lungs is that which is passed on to the right ventricle by venomotor mechanisms capable of propelling blood to the right side of the heart. There is less blood in the systemic circulation and more in the lungs. Because of the high capillary pressure the blood leaving the lung has lost water. As it circulates through the systemic capillaries, it picks up extracellular fluid, which it loses as it circulates through the lungs. In its pure form this type of pulmonary edema occurs without weight gain. The pulmonary edema fluid is that which is normally present in the body and not an excess accumulated because of changes in renal function caused by heart failure. 2. Selective accumulation in the lungs of the water and salt retained by the kidney occurs because of increased pulmonary capillary pressure. In more chronic left ventricular failure, the pulmonary capillary pressure remains constantly elevated and the degree of pulmonary edema will vary greatly with the volume of extracellular fluid. When salt and water are ingested and not promptly excreted by the kidney, weight gain occurs. The retained fluid accumulates preferentially in areas of high capillary pressure. Under the conditions of a chronic elevation of pulmonary capillary pressure, the amount of pulmonary edema is increased by the ingestion of salt and decreased by the use of diuretics and salt restriction without changing the quantity of circulating blood in the lungs. The only requirement in the presence of an increasing extracellular volume is the persistently elevated pulmonary capillary pressure.

As pulmonary congestion becomes more severe, the resting ventilation increases. The oxygen tension of the blood is slightly lowered but not enough to affect appreciably the degree of oxygen saturation. The overbreathing causes a fall in carbon dioxide tension and a tendency to respiratory alkalosis. The increased ventilation masks the degree of lung dysfunction that is present. If the ventilatory volume is decreased by morphine or by breath-holding, oxygen saturation of arterial blood immediately falls. The lung has lost its reservoir function, which allows the maintenance of relatively normal blood gases during periods of apnea. We see this same loss of reservoir function in persons who have a reduced lung capacity because of marked obesity. Again, minor changes in breathing rhythm are associated with marked arterial oxygen unsaturation.

We have usually looked on the level of arterial oxygen saturation as a rather fixed value. This view is relatively true in subjects with a normal vital capacity, normal pulmonary mixing, and normal alveolar capillary membranes. It becomes much less true in diseased states. In cardiac patients who have replaced air space with fluid and blood, arterial oxygen saturation will vary greatly when anything decreases the increased pulmonary ventilation. In the obese patient with decreased lung capacity, a second factor is also present. Because of the extreme degree of obesity, oxygen utilization of the body at rest is high and the oxygen left in the lung during periods of decreased ventilation is removed at a fast rate.

Edema in chronic congestive heart failure is generalized and causes a gain in weight. Sodium excretion is impaired but there is relatively little difficulty in excreting water, potassium, and nonelectrolytes. The ability of the kidney to excrete hydrogen ions and ammonia remains good. No one factor accounts for the phenomenon of edema in heart failure. Both mechanical
and hormonal factors seem to be of importance. The renal blood flow is greatly reduced and the glomerular filtrate moderately reduced in chronic heart failure, regardless of the cause of the failure. When less sodium is presented to the tubules, sodium reabsorption tends to be more complete and less is excreted. Heart failure causes an increase in the excretion and presumably the production of aldosterone, which decreases the excretion of sodium. The facts that adrenalectomy may decrease the edema of heart failure, presumably without increasing glomerular filtrate, and that aldosterone-producing tumors do not cause edema in the absence of heart failure suggest that these 2 mechanisms may operate synergistically.

The increase in central venous pressure caused by heart failure seems to be dependent primarily on increased venous tone. There is not enough blood in the lungs that can be mobilized by pulmonary venous constriction to flood the peripheral venous system and cause a marked rise in venous pressure. The central venous pressure of congestive heart failure is lowered by ganglionic-blocking agents, which interrupt sympathetic vasoconstrictor impulses to the venous system.

Hydrostatic increases in venous pressure are of major importance in determining the distribution of the salt and water retained by the kidney. During the day the high hydrostatic pressure in the dependent portions of the body may compete successfully with the lungs for retained salt and water. When the patient lies down and the hydrostatic venous pressure is lowered in the extremities, fluid re-enters the blood stream and tends to be deposited in the lungs because of the high pulmonary capillary pressure; acute nocturnal attacks of pulmonary edema may then occur.

**Importance of Etiologic Factors in Heart Disease**

In former years, more attention was paid to the diagnosis of heart failure and less attention to the cause of the heart disease underlying the heart failure. The therapeutic schedule was the same regardless of the cause of the failure because there were fewer specific measures available to combat the principal causes of heart disease, for example, rheumatic fever, coronary arteriosclerosis, hypertension, congenital deformities, and bacterial endocarditis. A notable exception was syphilis. During the past 30 years, additions to our knowledge of basic disease mechanisms and the introduction of effective drugs (sulfonamides, antibiotics, antihypertensives, steroids), new dietary principles (restricted sodium and low fat diets), and spectacular surgical procedures for prevention and treatment of these diseases have placed a mandatory responsibility upon the physician to recognize and treat properly such disease states in order to forestall heart injury and prevent heart failure. Classic examples would include (1) prevention of acute rheumatic fever by adequate treatment of the preceding streptococcal infection and prophylaxis of recurrent rheumatic fever by penicillin; (2) control of hypertension by drugs, diet, and sympathectomy; and (3) surgical ligation of a patent ductus arteriosus. Bacterial endocarditis, uniformly fatal in past years, now may be cured in at least 70 per cent of cases by antibiotic therapy. Although no specific therapy for coronary arteriosclerosis is presently available, the utility of prolonged anticoagulant therapy and of low-fat diets may hold promise for the future.

In addition to these common etiologic types of heart disease, it is equally important to recognize some less frequent causative factors, since they may produce congestive heart failure alone, or precipitate and maintain heart failure in previously diseased hearts. Some represent reversible forms of heart disease for which specific therapy is available; for example, hyperthyroidism, myxedema, anemia, obesity, arteriovenous fistula, constrictive pericarditis, and beriberi. Of these, hyperthyroidism is most important and easily overlooked unless clinical suspicion embraces every older patient exhibiting congestive failure and atrial fibrillation, particularly when fibrillation fails to respond to adequate digitalis dosage. The presence of normal sinus rhythm during congestive failure or an essentially normal heart rate after digitalization does not exclude the diagnosis of hyperthyroidism. Prominent staring eyes,
silky moist skin, weight loss, which may be hidden by edema, enlarged thyroid, forceful heart sounds, wide pulse pressure, absence of constipation when confined to bed, and systolic sounds over great vessels represent clues to diagnosis and the need for determinations of blood cholesterol, basal metabolic rate, protein-bound iodine, or radioactive iodine uptake for confirmation. Myxedema is missed when one fails to appreciate the significance of hair loss, dry pallid skin, husky voice, pugdy facial appearance, slow mentation, macroglossia, distant heart sounds, slow pulse, and "slow return-phase tendon reflexes," particularly in older women. The presence of an active circulation, forceful heart sounds, tachycardia, increased pulse pressure and blood velocity, and hemoglobin below 8.0 Gm. per 100 ml. of blood, strongly suggest anemia as an etiologic factor. Obesity, already mentioned as a cause of heart failure, must be considered in the massively obese patient who slumbers easily and exhibits cyanosis and Cheyne-Stokes respiration at rest. Arteriovenous fistula must be carefully excluded in those patients presenting cardiac enlargement, accentuated heart sounds, wide pulse pressure, and a history of trauma, stab, gun shot or bullet wound, or even operation for ruptured intervertebral lumbar disk. When an extremity contains a fistula, the part is larger than its counterpart, the veins are prominent, and edema will be disproportionately greater. The continuous murmur, with systolic accentuation, which can be obliterated by local pressure and thus effect cardiac slowing, is diagnostic. Excision of the fistula can restore heart size to normal or near normal after years of circulatory embarrassment and marked cardiac enlargement. Constrictive pericarditis, often confused with cirrhosis of the liver because of hepatomegaly, ascites, and edema, can be easily differentiated by the diminished heart sounds, decreased pulse pressure, "paradoxical" pulse and blood pressure, and distended neck veins secondary to elevated venous pressure. In the United States beriberi heart disease is rare and most commonly associated with chronic alcoholism and overt deficiency states, such as pellagra and polyneuritis. It is characterized by an active circulation, tachycardia, wide pulse pressure, high cardiac output, and specific therapeutic response to thiamine chloride but not to digitalis. Except for thiamine chloride there are no known states of vitamin deficiency that produce heart failure, although vitamin deficiency has long been suspected as a background for heart disease of cryptogenic etiology. Heart failure in association with cirrhosis of the liver is of uncertain etiology because of the multiplicity of factors involved, including poor nutrition, interference with protein and carbohydrate metabolism, vitamin deficiency, and anemia. It must be watched for, however, in the cirrhotic individual who develops cardiac dilatation, and must be treated before pulmonary congestion and distended neck veins appear.

Heart failure resulting from acute nephritis also should be recognized as an etiologic entity because the failure reverses spontaneously as the acute process subsides. Examination of the urinary sediment for red cell casts and fat-laden renal epithelial cells will distinguish acute nephritis from other causes of heart failure in which albuminuria, cells, and casts are commonly found. Hypertension usually accompanies nephritic heart failure but the relationship is not mandatory. Toxemia of pregnancy can be appropriately treated by caloric and sodium restriction, antihypertensive drugs, and interruption of pregnancy.

Chronic cor pulmonale with right heart failure usually results from parenchymatous disease of the lung, obstruction of pulmonary blood flow, and secondary pulmonary hypertension. Dyspnea, cyanosis, polycythemia, and clubbing of the fingers and toes are common and, when failure intervenes, venous pressure elevation, liver enlargement, and edema ensue. Primary pulmonary hypertension leading to heart failure, although rare, is a distinct entity. Often forgotten is the fact that repeated small emboli can produce cor pulmonale without cough, hemoptyis, or painful pleuritic episodes. Such emboli arise from pelvic and prostatic plexuses but more frequently from leg veins, particularly when the legs are edematous during congestive failure itself. These emboli can induce, increase, or maintain heart failure.
unless their existence is suspected and appropriate treatment, either anticoagulation or venous ligation, is instituted. Suspicion should be aroused when the patient already in heart failure manifests restlessness, bouts of fever, cyanosis, dyspnea, tachycardia, unresponsive-ness to the anticoagulant program, and disproportionate swelling of one extremity even in the absence of painful calf muscle, Homans' sign, cough, hemoptysis, or pleural pain.

Other rare and exotic forms of heart disease causing heart failure exist but the present state of medical knowledge does not permit curative treatment. Among these may be included endocardial fibroelastosis, disseminated lupus erythematosus, scleroderma, amyloidosis, and hemochromatosis. Although steroid therapy may be of benefit in lupus disease, no specific therapy remains for the other etiologic causes. The future, however, may bring new effective methods of therapy so that continued recognition may hold interest for more than medical curiosity.

The above discussion has concerned the primary and major causes of heart disease. Often unappreciated are the common secondary and precipitating factors for heart failure when decompensation is borderline or imminent. Physical activity, not strenuous but that ordinarily pursued, may induce overt failure in the patient exhibiting cardiac enlargement and gallop rhythm or mitral stenosis. Physical exertion increases the metabolic demands of the tissues upon the heart for blood and, when cardiac output becomes inadequate for tissue needs, congestive failure results. Pulmonary edema may appear abruptly during any strenuous physical exercise when left ventricular output of blood falls below the amount delivered by the pulmonary veins, or in the presence of valvular obstruction offered by mitral stenosis.

This is also true of minor respiratory infection, particularly when associated with cough. High fever, pneumonitis, or myocarditis need not be present. That active carditis is the principal underlying cause of heart failure in rheumatic children and young adults deserves continued emphasis.

A careful history often elicits the fact that patients who suffer from rheumatic or degenerative heart disease carry on well until the onset of atrial fibrillation. Heart failure slowly appears as a result of this inefficient rhythm unless the heart rate is adequately controlled by digitalis. Rapid ectopic rhythms, particularly paroxysmal atrial and ventricular tachy-cardia, usually cause more abrupt appearance of heart failure by compromise of the diastolic pause of heart muscle. Although the degree of mental stress and its effects upon the body cannot be accurately measured, the importance of its existence is frequently ignored. Emotional tension and anxiety, whether acute or chronic, can convert compensation to decompensation and maintain heart failure resistant to the usual forms of therapy until such stress is ameliorated or eliminated.

Following mitral valve surgery, acute metabolic changes are often observed, which have been ascribed to the trauma and stress of operation. Urinary excretion immediately falls, water and salt are retained, blood and extracellular fluid volume rise, and the hematocrit declines. Blood sodium and chloride concentrations fall to hyponatremic and hypochloremic levels, while blood potassium concentration remains normal or even rises. Marked weakness, lethargy, dyspnea, cyanosis, and edema appear and heart failure again becomes clinically evident. One feature of the syndrome would appear to be dilution in the presence of a maintained but moderate postoperative intake of fluid in the face of a low urinary output of fluid. Blood sodium concentrations, however, frequently fall below that which can be fully accounted for by the amount of fluid retention. This suggests a transfer of sodium from the extracellular to the intracellular space where it replaces potassium. Sharp restriction of fluid intake without sodium administration in the first 96 hours is the most effective method of preventing this syndrome, which, once developed, may prove refractory to treatment and may even be fatal unless water diuresis can be achieved. Mercurial diuretics are commonly ineffective, but alcohol as an inhibitor of antidiuretic hormone holds definite promise as a therapeutic agent. The underlying cause of the metabolic disturbance is thought to be
the release of antidiuretic hormone and adrenal steroid secondary to operative stress. The possible antidiuretic effect of reduction of pressure or stretch in the left atrium by commissurotomy and relief of valve obstruction must be considered, since the syndrome is rarely as severe after other forms of cardiac or thoracic surgery. However, the syndrome has been observed in typical form after exploration of the mitral valve is undertaken without performing commissurotomy.

Classic recurrent congestive failure, occurring physiologically once monthly, is presented by a few young menstruating women suffering from mitral stenosis. During the premenstrual week, fluid retention, amounting to 5 to 10 pounds of weight gain, may culminate in increasing dyspnea, edema, and cardiac asthma unless fluids and salt are sharply restricted and diuretics administered. Estrogen liberation in the premenstrual period is the presumed cause of the salt and water retention in these patients.

**Symptoms of Congestive Heart Failure and Those Resulting from Treatment**

Difficulties frequently confront the physician in the differentiation of those symptoms resulting from heart failure per se, and those resulting from the therapeutic program. The problem basically is whether the patient has received too little or too much treatment. As a generalization it may be stated that those symptoms present before therapy can safely be attributed to heart failure itself; those symptoms appearing during treatment must be assumed to result from one or more phases of the treatment and must be carefully assessed, particularly when the manifestations of failure are receding. Refractoriness to treatment with appearance of new symptoms indicates a careful review of the entire clinical picture and the therapeutic program.

Cerebral symptoms comprising restlessness, irritability, poor concentration, depression, and varying degrees of mild delirium are common in advanced congestive heart failure as a result of reduced cardiac output, reduced cerebral blood flow, cerebral anoxia, and the resultant disturbance of cerebral metabolism. As heart function deteriorates, stupor and coma appear as terminal events. Restoration of compensation by proper treatment usually reverses most of these symptoms unless cerebral thrombosis is a complicating feature. In older patients the mere institution of a strict low-sodium diet has produced similar symptoms of irritability, personality change, and depression, which are reversed only by restoration of normal diet. Over-digitization likewise can induce headache, depression, irritability, and personality change but these are usually accompanied by visual disturbance, gastrointestinal symptoms, or cardiac arrhythmias in various combinations. Electrolyte disturbances such as hyponatremia, hypochloremia, alkalosis, hypokalemia, acidosis, and azotemia are common underlying causes of cerebral symptoms. They result from too vigorous treatment of congestive failure, particularly in the presence of renal disease. During heart failure itself or during the recovery phase, acute psychoses may appear for the first time and prove resistant to treatment. Psychoses usually afflict the older, but the young and middle-aged groups are not immune.

Gastrointestinal symptoms represent an integral part of the symptomatology of heart failure. The reflex pathways existing between heart and stomach through the medulla allow anorexia, nausea, and vomiting to be produced by stimulation of the reflex arc at various points. Passive congestion of the abdominal viscera is the usual mechanism for gastric dysfunction in heart failure, and symptoms clear as compensation is restored and edema disappears. Central stimulation of the pathway in the medulla also results in nausea and vomiting. Acute myocardial infarction or the excessive deposition of digitalis glycoside in heart muscle stimulates the cardiac end of the reflex arc and often produces identical gastrointestinal symptoms. The recognition of digitalis-induced gastrointestinal symptoms is not usually difficult when digitalization is accomplished evenly over periods of 2 to 4 days, for a characteristic sequence of anorexia followed by nausea, then vomiting and, occasionally, diarrhea occurs. Large-dose therapy causes nausea and vomiting to appear more abruptly.
Additional clues to the existence of intoxication are the appearance of visual symptoms, scotomata or yellow vision, rhythm disturbances, and alterations in the electrocardiogram. In older patients, rigid dietary restriction of sodium often leads to anorexia and nausea and eventually to vomiting unless normal foods are restored or some compromise effected. Thirst, anorexia, and nausea also result from electrolyte abnormalities.

Because of varied underlying types and degrees of myocardial disease, patients suffering from congestive heart failure may exhibit A-V block, bundle-branch block, or any rhythm disturbance from premature beats to ventricular tachycardia. Rhythm disorders also may be produced by excessive doses of digitalis or may follow the potassium loss through excessive diuresis, which increases the heart's sensitivity to digitalis glycosides. When the total dosage of digitalis administered is known, the problem of differentiating digitalis toxicity from symptoms of heart disease is usually easy but may be difficult when the amount is unknown. When digitalis is first prescribed, the physician should give the optimum dose that produces the desired result—that of strengthening and slowing of the heart beat with production of diuresis and weight loss. These simple therapeutic effects are often disregarded and digitalis administration is carried to the point of intoxication. Premature ventricular beats generally appear first and are followed by coupling as toxicity from digitalis increases. Atrial fibrillation may result from the rapid administration of digitalis but rarely is ventricular tachycardia observed. When rhythm alterations are of digitalis origin, visual disturbances and gastrointestinal symptoms are often present in mild form and easily overlooked unless special inquiry is made. The electrocardiogram gives important diagnostic aid by exhibiting S-T depression, flattening or inversion of the T waves, shortening of the corrected Q-T interval (Q-Tc) below 0.40 second, and delay in A-V conduction time beyond 0.20 second. In most instances, full digitalization will shorten the Q-Tc interval in both normal and abnormal electrocardiograms. The reduction in the mean Q-Tc interval for a large group will average 0.04 second. The longer the Q-T interval initially, the greater is this effect of digitalis. Only rarely will a Q-Tc remain beyond 0.40 second after digitalization is complete. Thus, when the Q-Tc interval lies beyond 0.40 second, it is unlikely that full digitalization is present. The converse is not true; a Q-Tc interval of 0.40 second or below does not necessarily mean that digitalization is present because the Q-Tc interval in the normal or abnormal electrocardiogram may fall in this range before digitalis. Paroxysmal atrial tachycardia with block must always be suspected as a digitalis-induced rhythm. During normal rhythm the heart rate may rise and congestive failure increase from digitalis poisoning. Potassium chloride may abolish the premature beats or bigeminal rhythm produced by digitalis. When final differentiation between too much or too little digitalis cannot be made with certainty, it is best to discontinue digitalis and observe the patient as digitalis excretion proceeds. Premature beats that arise from heart failure per se generally are abolished as myocardial function improves under digitalis therapy. The use of intravenous acetylstrophanthidin to produce cardiac irritability as an indication of the residual amount of digitalis glycoside deposited in heart muscle is dangerous and impractical for general use.

One of the frequent and important problems in congestive heart failure centers upon the early recognition of alterations in electrolyte metabolism. Disturbances in the blood constituents—sodium, potassium, chloride, calcium, carbon dioxide, and nitrogen—can produce a variety of cerebral, gastrointestinal, and genitourinary symptoms including weakness, drowsiness, apathy, restlessness, confusion, irritability, thirst, anorexia, nausea, muscular cramps, decrease in urinary output, and refractoriness to diuretic treatment. Such alterations are more likely to occur when the course of heart failure is complicated by too vigorous treatment including rigid low-sodium diet, mercurial diuretics, nausea, vomiting, or diarrhea, the use of ammonium chloride, ion exchangers, or carbonic anhydrase inhibitors in the presence of renal disease or renal failure. Disturbances rarely occur in heart failure from
salt restriction alone unless mercurial diuretics are administered or renal disease is associated with excessive salt loss. Under these circumstances, particularly in older subjects, the blood electrolyte picture must be followed with special care.

The most common electrolyte alteration is a hypochloremic alkalosis resulting from loss of chloride in excess of sodium during vigorous mercurial diuresis. Elevation of blood nitrogen and refractoriness to diuretic therapy commonly accompany the syndrome. Serum sodium and potassium are normal. The syndrome is reversed by the administration of ammonium chloride and diuretic responsiveness is restored.

It is possible to distinguish 2 general forms of hyponatremia: one related primarily to sodium depletion, in which serum sodium and chlorides fall to low levels, urinary chlorides decrease, and blood nitrogen and hematocrit rise; and the second, a dilution form of hyponatremia, in which sodium and chloride are reduced in the presence of increased extracellular fluid volume and lowered hematocrit. The sequence of events in the edematous cardiac under treatment may be very helpful in clinical differentiation. If hyponatremia appears after good mobilization of edema fluid and copious diuresis, and is accompanied by cerebral and gastrointestinal symptoms or peripheral collapse, depletion of sodium by mercury has very likely occurred. Repetitive large parencenteses may also induce sodium depletion when sodium intake is restricted. The dilution type of hyponatremia must be suspected when the cardiac remains edematous, becomes refractory to treatment in spite of continued mercurial injections, and exhibits symptoms of weakness, anorexia, thirst, and drowsiness. Salt depletion is less common than dilution hyponatremia and tends to respond to hypertonic sodium chloride infusion. The dilution form is a serious disturbance and when fully developed is rarely reversed. Hyponatremia occurring in a patient who has not been treated vigorously is a sign of serious illness. Salt administration under these circumstances aggravates thirst and fails to overcome the hyponatremia. Water diuresis through improved myocardial and renal function is needed but difficult to achieve, and most patients succumb. Potassium depletion also may occur as a result of vigorous diuretic therapy, particularly if food intake is poor. This may cause an increased sensitivity of the myocardium and the appearance of ectopic rhythm disturbances as manifestations of digitalis intoxication produced by potassium deficiency. Cation-exchange resin will bind sodium, potassium, and calcium and when administered over prolonged periods can produce hyponatremia, hypokalemia, hypocalcemia, and acidosis. Ammonium chloride and carbonic anhydrase inhibitors (Diamox) produce acidosis, hyperchloremia and potassium loss. Such disturbances are more likely when both compounds are administered simultaneously in large doses for long periods, particularly in the presence of renal disease when the kidney can no longer compensate for changes in acid base equilibrium. The development of drowsiness, weakness, stupor, coma, hyperpnea, low plasma carbon dioxide content, and elevated serum chloride and nonprotein nitrogen should suggest the clinical picture.
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