

HeartFacts

SOUTHERN CALIFORNIA
CARDIOVASCULAR CONSULTANTS



visit us at www.socalcardiology.com

Congestive Heart Failure

Congestive heart failure (CHF) has become a major public health problem. This disease affects nearly 5 million Americans and results in nearly one million hospitalizations annually. It is the single largest expense for the Medicare system -- the hospital piece alone costing 15 billion a year. Newly diagnosed patients are often shocked by the ominous descriptor "failure" and well they should be. The one year mortality approaches 33 percent and climbs to 50 percent at five years.

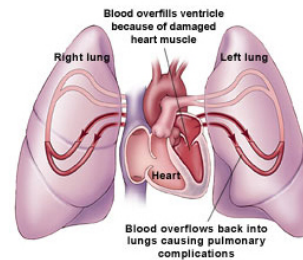
One searches the textbooks in vain for a useful definition of CHF. Herewith, a streamlined effort: chronic/acute injury to the left ventricle resulting in loss of diastolic compliance, elevation of diastolic filling pressure, and alveolar flooding.

Those factors which most commonly injure the left ventricle include in order of descending frequency: hypertension, myocardial infarction/ischemia, valvular heart disease, and cardiomyopathy. These act singly

or in combination to impair left ventricular performance. Seventy-five percent of CHF patient have pre-existing hypertension. In HTN, ventricular myocardium hypertrophies. Hypertrophy causes stiffening of the muscle and loss of compliance. Diastolic relaxation is impaired. **Diastolic dysfunction** results. If the patient goes untreated ventricular dilatation ensues. In the case of MI, the ventricle dilates early and surviving myocardium hypertrophies in compensation. **Systolic dysfunction** is roughly proportional to the amount of myocardium lost.

At a cellular level and regardless of the root cause, myocytes are lost and collagen deposited. Survivor myocytes hypertrophy in compensation.

As dilatation progresses, the limit of elastic compliance is reached and there is a steep rise in LV filling pressure. Since there are no valves in the pulmonary veins, this pressure is passed backward into the pulmonary capillary bed. Capillary



A heart in left-sided failure. The left ventricle pumps inadequately, causing blood to build up in the lungs behind the overfilled ventricle.

integrity is breached and an ultrafiltrate of serum inundates the alveoli. Lung bases are flooded first because of gravity and later the apices. Systemic oxygenation falls. Though the Starling mechanism constitutes the first line of defense against CHF, a second line of compensatory mechanisms is activated once this transudation begins. Neurosympathetic activation attempts to rally the myocardium via adrenergic stimulation but ultimately overwhelms the failing ventricle by producing both venous and arterial vasoconstriction. This shifts blood volume from the venous capacitance bed to the thorax and at

"CHF is best defined as chronic/acute injury to the left ventricle resulting in loss of diastolic compliance, elevation of diastolic filling pressure, and alveolar flooding".

the same time increases after-load to the failing ventricle. More transudation ensues. As cardiac output falls, renal flow is reduced and the juxtaglomerular apparatus activates the renin-angiotensin-aldosterone mechanism mistaking the reduction in renal flow for a fall in blood volume. Salt and water are retained by the kidney, further compounding the problem for the left ventricle.

In my experience, the most useful symptoms and signs of heart failure are orthopnea, tachypnea, jugular venous distention, end-inspiratory rales and vascular congestion on the chest x-ray. Who would have thought a mere five years ago that a blood test confirming the diagnosis

"A useful acronym for the treatment of ADHF is DONNA. This stands for: diuretic, oxygen, nitroglycerine, Natreacor and ACE inhibitor." •

Congestive Heart Failure (continued)

and stratifying the severity of CHF would appear on the scene? That test is here and it is called BNP. It has revolutionized our care of these patients. **B-type natriuretic peptide** is secreted primarily from the ventricles in response to overfilling and stretch. Its serum level correlates with the severity of heart failure. Its actions include venous and arterial vasodilation, renin inhibition, natriuresis, diuresis and diastolic relaxation. In short, it relaxes arteries, veins, and the left ventricle and stimulates the kidneys to dump salt and water. Like the other compensatory mechanisms it can be overwhelmed. Unlike adrenergic and RAA activation BNP does not overwhelm the ventricle. Supraphysiologic doses are indeed therapeutic and form the rationale for treatment of CHF with hBNP (neseritide/Natreacor).

A recent nomenclature subdivides CHF into acutely decompensated heart failure (ADHF) and recompensated heart failure (RCHF). Acute pulmonary edema (APE) is the most dramatic expression of ADHF.

CHF therapy may be divided into three phases: prehospital, hospital and outpatient. Bussman showed some years ago that the administration of sublingual NTG -- one tablet or spray every few minutes to a mean dose of 1600 micrograms -- is an extremely

effective way of reversing acute pulmonary edema. This may be done in the field or office with near instantaneous and gratifying results. It constitutes an excellent first response and is readily available.

In the emergency department, Lasix, oxygen and nitrates are usually given first due to ease of administration. Natreacor should be started early especially if heart failure is moderate to severe or if serum BNP is > 500. (Care must be exercised and the patient monitored as hypotension occurs in a small fraction of patients). Natreacor is the only currently available agent that deals with both the hemodynamic and neurohumoral alterations in CHF.

A useful acronym for the treatment of ADHF is **DONNA**. This stands for: diuretic, oxygen, nitroglycerine, Natreacor and ACE inhibitor.

Modern treatment of recompensated CHF focuses on maintenance of the "re-normalized" hemodynamics and on neurohumoral blockade. The combination of Coreg, ACE inhibitor, and aldosterone antagonist emulates the multiple effects of Natreacor. This combination may reduce one year mortality by as much as 50 percent.

the discharge regimen in patients with RCHF. It stands for beta-blocker, ACE inhibitor, aldosterone antagonist, diuretic and digoxin.

While CHF remains a major threat to the public health, we have major new weapons in the armamentarium. To invert a current phrase -- war is the answer. Treating early and treating aggressively saves lives • L.R. O'Connor, M.D.



Lawrence R. O'Connor, M.D.



Y. Joseph Wen, M.D.

"BAADD is a helpful acronym for the discharge regimen in patients with RCHF. It stands for beta-blocker, ACE inhibitor, aldosterone antagonist, diuretic and digoxin." •

Save Lives, Restore Health, Instill Hope....

visit us at www.socalcardiology.com