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HEART FAILURE IN SEARCH OF A CURE

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Over the last fifteen years, the treatment of congestive heart failure has radically changed. Based on large randomized clinical trials, five different drug classes that incrementally improve survival have been introduced in the treatment of symptomatic patients with congestive heart failure: ACE inhibitors; beta blockers; aldosterone antagonist; the combination of nitrates and hydralazine; and angiotensin II receptor blockers. Also, it became clear that strategies thought to be useful might in fact be harmful, for example, the use of inotropic agents and some anti-arrhythmic drugs. Today, the one-year mortality of patients with severe systolic dysfunction and New York Heart Association function class II-III is in the order of 10%, a significant improvement for the symptomatic patient. However, the mortality rate for patients with severe symptomatic heart failure remains high - up to 90% in one year for those in need of chronic inotropic therapy, for example.

Another frontier of great progress has been the aggressive use of conventional high-risk surgical interventions like coronary revascularization with off-pump techniques and minimally invasive strategies. Recognition that mitral valve repair rather than mitral valve replacement preserves cardiac function permits surgical treatment of patients who were not otherwise candidates for conventional surgery.

In addition to improved pharmacologic treatment and surgical techniques, a new era in the treatment of chronic heart failure emerged with the introduction of implantable defibrillators and cardiac resynchronization therapy that incrementally improve survival by decreasing the risk of sudden cardiac death and by preventing the progression of heart failure.

Considering the above observations, most therapeutic strategies for treating chronic heart failure prolong life by decreasing the rate of progression or the risk of arrhythmic death. However, there are no curative strategies; the treatment of heart failure induces remission of the illness rather than a cure. Clearly, if we accept this premise, there are ample opportunities to search for a cure. The articles in this issue of the *Methodist DeBakey Cardiovascular Journal* present a review of new mechanisms for cardiac injury, the role of the mitochondria in cell injury, the role of inflammation in the progression of heart failure, and strategies aimed to decrease inflammation. New strategies for diagnosing and treating heart failure in the setting of diabetes are also presented. Finally, of great importance, Dr. Youker presents a strategy at the MDHVC for bringing new discoveries to early clinical application - our ultimate goal as clinician investigators. Collectively, we present an overview of ongoing strategies in search for a "cure" for the syndrome of heart failure.