Chronic Heart Failure: Applying New Findings to Optimize Care and Improve Survival

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[Rev Cardiovasc Med. 2004;5(suppl 1):S1-S2]

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¬ he incidence and prevalence of chronic heart failure (CHF) in the United States continues to increase, and associated morbidity and mortality remain unacceptably high. Over 5 million patients in the United States have CHF and they will make 12 million outpatient office visits for CHF this year. The economic burden associated with heart failure is large, with as much as \$28 billion dollars spent annually on the care of heart failure patients.1

There is compelling clinical trial evidence that medical therapies, including angiotensin-converting enzyme (ACE) inhibitors, aldosterone antagonists, and ß-blockers reduce the risk of hospitalization and substantially improve survival rates in patients with heart failure. Recent clinical trials have further defined the role of these agents, as well as that of angiotensin-receptor antagonist therapy. The single most effective medical therapy for CHF has proven to be the use of ß-blockers. In the course of their development, varying ß-blockers have emerged and proved a diverse class of agents in terms of their pharmacology, effect on important clinical outcomes, and tolerability. Whereas some \(\mathbb{G} \)-blockers have shown efficacy in heart failure trials, others were shown to be neutral or harmful—a clear signal that differences among ß-blocker have important clinical implications. The American College of Cardiology/American Heart Association Guidelines on the Diagnosis and Treatment of Heart Failure recommend the use of those \(\mathbb{G}\)-blocking agents and doses that have proven benefit compared to placebo. The recent Carvedilol Or Metoprolol European Trial (COMET) highlights the concept that drugs in the same class cannot necessarily be judged as having the same beneficial effects.

Despite national and international clinical guidelines recommending ACE inhibitor, aldosterone antagonist, and \(\mathbb{G}\)-blocker treatment in stabilized patients with CHF due to systolic dysfunction, a number of studies have documented low

treatment rates in this patient population. The underuse of ß-blocker and other protective therapies in patients with heart failure represents a major clinical practice and public health issue. Recently, systems for in-hospital initiation of evidence-based medical therapies in patients hospitalized with cardiovascular disease have been shown to improve treatment rates, long-term patient compliance, and clinical outcomes. Widespread application of hospital-based ß-blocker treatment initiation programs for heart failure could dramatically increase rates with this proven, cost-effective therapy and thus substantially reduce the risk of recurrent hospitalizations and death in the large number of patients hospitalized with heart failure each year.

The goals of this supplement are to describe the morbidity and mortality risk of CHF, review the results of recent clinical trials, and discuss opportunities to improve the quality of care for this patient population. The new standard of care for patients with heart failure will be discussed and the evidence for supporting the use of ß-blockade in severe heart failure and special populations will be provided along with the results of the COMET trial, which will be reviewed and its important clinical implications discussed. The unique pharmacologic properties of carvedilol that may account for the vascular, metabolic, and antiarrhythmic protective effects observed in clinical trials will be highlighted and the value of hospital-based systems to improve treatment rates and

clinical outcomes will also be described. Strategies that may be useful in bridging the gap between evidence-based medicine and clinical practice will be provided, including a discussion on effective methods to switch ß-blockers in heart failure patients. It is our hope that this information will prove useful in improving the quality of CHF patient care and, through improved use of evidence-based, life-saving therapies, to ultimately reduce death and disability due to this disease state.

References

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