
BACKGROUND: We tested the hypothesis that carvedilol inhibits clinical progression in patients with mildly symptomatic heart failure due to left ventricular (LV) systolic dysfunction.

METHODS AND RESULTS: Patients (n = 366) who had mildly symptomatic heart failure with an LV ejection fraction (LVEF) < or = 0.35, had minimal functional impairment (defined as the ability to walk 450 to 550 m on a 6-minute walk test), and were receiving optimal standard therapy, including ACE inhibitors, were randomized double-blind to carvedilol (n = 232) or placebo (n = 134) and followed up for 12 months. The primary end point was clinical progression, defined as death due to heart failure, hospitalization for heart failure, or a sustained increase in heart failure medications. Clinical progression of heart failure occurred in 21% of placebo patients and 11% of carvedilol patients, reflecting a 48% (P = .008) reduction in the primary end point of heart failure progression (relative risk, 0.52; CI, 0.32 to 0.85). This effect of carvedilol was not influenced by sex, age, race, cause of heart failure, or baseline LVEF. Carvedilol also significantly improved several secondary end points, including LVEF, heart failure score, NYHA functional class, and the physician and patient global assessments. Carvedilol reduced all-cause mortality but had no effects on the Minnesota Living With Heart Failure scale, the distance walked in 9 minutes on a self-powered treadmill, or cardiothoracic index. The drug was well tolerated.

CONCLUSIONS: Carvedilol, when added to standard therapy, including an ACE inhibitor, reduces clinical progression in patients who are only mildly symptomatic with well-compensated heart failure.