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Prognostic impact of heart rate recovery in chronic heart disease

Abstract: P1334

Prognostic impact of heart rate recovery in chronic heart disease

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Background: Abnormal HR recovery on exercise stress testing (defined as a decrease in the heart rate <13 bpm in the first minute of active recovery) is a powerful predictor of mortality in various patient cohorts; its prognostic impact in patients with chronic heart disease either coronary artery disease (CAD) or heart failure (HF) compared with controls without cardiovascular disease (CVD) has not been reported.

Purpose: We analyzed a large cohort of patients undergoing exercise testing to compare the prognostic impact of HR recovery in patients with CAD or HF versus patients without known CVD.

Methods: 101,455 non-imaging exercise tests performed on patients ages 30–89 years from 1993 to 2010 were analyzed. The final study cohort was limited to the first stress test on Minnesota residents for whom we had complete mortality ascertainment by Mayo Clinic patient records and the Minnesota Death Index. Patients were divided into 3 groups: no CVD, CAD without documented HF, and chronic HF (including both ischemic and non-ischemic). The effect of abnormal HR recovery on mortality was assessed using Cox regression analyses with adjustment for age, sex, and cardiorespiratory fitness (CRF).

Results: A total of 28,502 patients (67% men; age 54±12 years) were included: no CVD (n=22,806), CAD (n=5071) and HF (n=625). CAD and HF patients were older and more likely male than no CVD patients. The prevalence of abnormal HR recovery was 24% in no CVD, 50% in CAD, and 76% in HF. There were 4034 deaths (14%) over a mean follow-up of 12.2±5.2 years; mortality was 8%, 37%, and 54% in no CVD versus CAD versus HF. Adjusted hazard ratios for death according to abnormal HR recovery were very similar across the 3 groups: no CVD = 1.52 [1.37–1.68] versus CAD = 1.45 [1.30–1.61] versus HF = 1.45 [1.06–1.99]. Among the HF patients, only 24% have normal HR recovery, but it is was strongly protective. Abnormal HR recovery predicted mortality equally well in men versus women, and further adjustment of the Cox model for BMI, diabetes, hypertension, current smoking, and beta blockade had essentially no effect on the hazard ratio for abnormal HR recovery. We note that CRF (expressed % predicted) was significantly better in patients with normal versus abnormal HR recovery in all groups (97±21 versus 82±24 in non CVD; 93±22 versus 75±22 in CAD; 75±24 versus 57±23 in HF), which suggests that normalization of HR recovery may be one of the mechanisms by which improved CRF reduces mortality risk.

Conclusions: The prevalence of an abnormal HR recovery doubles in patients with CAD and triples in CHF patients compared to patients with no CVD, and is a strong predictor of mortality with the same risk adjustment in all 3 groups. Abnormal HR recovery predicts mortality independently across sex and after adjustment for multiple risk factors known to affect mortality. Improving CRF may be a potential strategy to improve HR recovery and reduce mortality risk.