# Heart Rate Variability Tied to Post-MI Mortality

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SAN JUAN, P.R. — Low heart rate variability is significantly associated with an increased risk of death in depressed versus nondepressed patients after an acute myocardial infarction, Robert M. Carney, Ph.D., said at the annual meeting of the American College of Psychiatrists.

Depression is common among patients with a recent, acute myocardial infarction (MI)—incidence of major depression ranges from 15% to 23% in the literature. Other researchers found that low 24-hour heart rate variability is a strong predictor of cardiac mortality in patients with a recent MI (Ann. Noninvasive Electrocardiol. 2005;10:88-101). Heart rate variability was as robust a predictor as ventricular dysfunction or the size of the infarction in this review article.

The aim of the current study was to determine whether 24-hour heart rate variability is lower in depressed patients, and if so, whether this finding explains why depression reduces cardiovascular mortality after an MI, said Dr. Carney, professor of psychiatry and director of the behavioral medicine center at Washington University, St. Louis.

**The study sought to determine whether 24-hour heart rate variability is lower in depressed patients vs. patients who were not depressed.**

He and his associates assessed 305 depressed patients (135 with major depression and 170 with minor depression) with 24-hour ambulatory ECG readings 1-3 weeks post MI. Another group of 366 nondepressed, post-MI patients was included for comparison.

The investigators measured frequency domain heart rate variability using very-low-frequency (VLF) power spectral analysis. "VLF reflects parasympathetic modulation and is one of the best predictors of post-MI mortality," Dr. Carney said.

Dr. Carney and his associates found a difference in log of VLF power (LnVLF) measurements: 6.32 in the depressed group, compared with 6.59 in the nondepressed patients.

"This was statistically significant, but is it clinically significant?" Dr. Carney asked.

In the study, 16% of depressed patients and 7% of nondepressed controls had a VLF below 180 squared milliseconds. The estimated probability of survival over 30 months of follow-up was statistically lower among depressed patients.

"So low heart rate variability is a significant and important factor post MI," Dr. Carney said.

After adjustment for other risk factors, including left ventricular ejection fraction, smoking, older age, and diabetes, the low heart rate variability hazard ratio "goes from 3.1 to 2.8—a tiny difference," he said.

"About 27% of the mortality risk in these patients can be accounted for by low heart rate variability," Dr. Carney said. "So there are other things that are important here—including platelet function and inflammation."

The literature is conflicting about whether treatment of depression provides a beneficial increase in heart rate variability. For example, 10 studies with tricyclic antidepressants yielded mixed results, Dr. Carney said, "and the six SSRI studies are more confusing." Three SSRI studies reported increased heart rate variability, and three reported no change. Studies with other antidepressants offer no clear answer, either. No change in heart rate variability was seen in a nefazodone study, while lower heart rate variability was observed in a bupropion study and a venlafaxine trial.

Dr. Carney assessed the effect of psychotherapy among depressed congestive heart disease (CHD) patients. After 12 sessions of cognitive-behavioral therapy, mean heart rate decreased 5 beats/min and root mean squared successive difference increased. There were no changes in other heart rate variability indices.

"Heart rate variability may be improved through medication, exercise, and cardiac risk factor modification," Dr. Carney emphasized.

"Regardless, depression in cardiac patients should be treated to improve quality of life, because we know we can do that," he said. ■