Anxiety Independently Predicts Infarction in Men

BY DAMIAN McNAMARA
Miami Bureau

MIAMI — Anxiety in men may be a robust and independent predictor of the 10-year incidence of myocardial infarction, according to a study presented at the annual conference of the Anxiety Disorders Association of America.

"This is kind of exciting because most work has been done with psychosocial factors like depression and hostility," Yael E. Avivi said in an interview during the meeting.

Depression and negative affect have been the focus of most of the literature addressing a possible association between psychosocial factors and heart disease. Other researchers have reported evidence suggesting that anxiety contributes to coronary heart disease (Ann. Behav. Med. 1998;20:47-58) and to an elevated risk of fatal coronary heart disease (Circulation 1994;90:2225-9). But these investigators used relatively short assessment scales to measure anxiety, noted Ms. Avivi, a doctoral student in the department of psychology at the University of Miami.

Her associates, including lead author Biing-Jiun Shen, Ph.D., analyzed data that included a more comprehensive assessment to look for a possible association between anxiety and subsequent MI. The study assessed follow-up data for 740 healthy men who entered the Veterans Administration Normative Aging Study in 1986. Initial assessments included the Minnesota Multiphasic Personality Inventory, a comprehensive physical examination, and a cardiovascular disease risk profile. The participants did not have diabetes or a history of MI. The mean age at study entry was 60 years.

The researchers calculated an overall anxiety factor for each participant based on a combined score from four anxiety scales used in the Minnesota Multiphasic Personality Inventory. Those included measures for psychasthenia and social introversion, as well as scores from the Wiggins phobia scale and the Taylor Manifest Anxiety Scale.

During the 10 years of follow-up, there were 60 new-onset myocardial infarctions, including two fatal heart attacks. The researchers used hierarchical logistic regression to predict the likelihood of an MI using the composite score and each of the four anxiety constructs.

"We looked at the odds ratios for predicting new MI incidence when controlling for age, education, marital status, weight, blood pressure, glucose, cholesterol, drinking, smoking, and caloric intake," Ms. Avivi said. "We could control for those and still see a significant effect."

The overall anxiety factor was an independent and significant predictor of subsequent MI in the sample population (odds ratio, 1.46). Also, each of the four anxiety components independently and significantly predicted MI: psychasthenia (odds ratio, 1.42), social inhibition (odds ratio, 1.36), phobia (odds ratio, 1.44), and Taylor Manifest Anxiety (odds ratio, 1.50). In addition, being single and having lower HDL cholesterol levels predicted onset of MI in a multivariate analysis.

"The next question we had was, can depression and other psychosocial factors explain this association?" she said. Interestingly, these other psychosocial factors could not explain the link between anxiety and the new cases of MI that emerged in this study. After controlling for depression, anger, hostility, type A personality, and perceived stress, anxiety remained an independent predictor of a subsequent MI.

The researchers divided participants into quartiles based on their anxiety scores. "We also saw a dose-response effect," Ms. Avivi said. "People with the highest anxiety scores had the highest incidence of MI."

The association between anxiety and acute myocardial infarction was not surprising to Dr. James J. Ferguson, chairman of the Research Committee at the Texas Heart Institute, Houston. Still, he commented, the results could have important implications for any physician treating patients with anxiety.

"Yes, these people are at risk, but what can we do about it?" he asked. He described the findings as an important first step, but added that "there is a long ways to go before we understand how changing anxiety or stress can affect outcomes" and noted that the study did not address that point.

Mechanisms that would explain the relationship between anxiety and subsequent MI remain unknown and require further study, Ms. Avivi said. Anxiety might adversely affect health behaviors, promote atherogenesis, or trigger fatal coronary events through arrythmia, plaque rupture, coronary vasospasm, or thrombosis (Ann. Behavior. Med. 1998;20;47-58).

General distress across a range of negative emotions might play an important role in the relationship between psychosocial factors and coronary heart disease, according to a recently published study that also was based on follow-up data from the Veterans Administration Normative Aging Study (Ann. Behavior. Med. 2006;31:21-9).

Those researchers also concluded that aspects of anxiety may independently increase the risk for coronary heart disease. However, they also assessed anger and depression in their cohort, and they urged future researchers to consider a shared component of these features as a possible explanation for the elevated coronary disease risk. ■

HR Variability Tied to MI Mortality

BY DAMIAN McNAMARA
Miami Bureau

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.

Researchers 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. "Whether any treatment can reduce risk in CHD patients is unclear," he said.

Depression is associated with autonomic nervous system dysfunction, and heart rate variability is a noninvasive method for studying cardiac autonomic nervous system modification, Dr. Carney explained. Heart rate variability reflects the intrinsic firing rate of sinoatrial pacemaker cells, an action that the autonomic nervous system modulates. ■

Gender, Race Gaps Shown in MI Care

ATLANTA — Women with acute MI are roughly one-third less likely than are men to undergo cardiac catheterization and coronary revascularization procedures, a trend that has persisted since the mid-1980s, William J. Kostis, Ph.D., said at the annual meeting of the American College of Cardiology.

Similarly, African Americans with an MI—men as well as women—are roughly one-quarter less likely than are whites to get a revascularization procedure. And again, that trend has held constant since the mid-1980s, said Dr. Kostis of Robert Wood Johnson Medical School, Piscataway, N.J.

These were among the findings of an analysis of nearly 248,000 first MIs in New Jersey during 1986-2002. The data were collected through the state's Myocardial Infarction Data Acquisition System (MIDAS), which includes information on all patients admitted with acute MI to nonfederal hospitals.

The rate of revascularization by percutaneous coronary intervention or coronary artery bypass surgery was less than 10% in both men and women with acute MI in 1986. In men, the rate climbed steadily to 54% by 2002, but in women it reached 34% in the mid-1990s and then leveled off.

Coronary revascularization was less than 10% among African Americans and whites in 1986, but by 2002 it rose to 46% among whites, and to 38% in African Americans. Adjusted 30-day mortality following first MI was significantly greater in women than in men, and in African Americans than in whites. But after further adjustment for rates of procedures, the increased mortality risk in women and African Americans was diminished; it became statistically nonsignificant in African Americans. This implies the lesser use of procedures in these groups is causally related to their higher mortality.

—Bruce Jancin