

# Silent PAD Was Revealed by Exercise ABI Tests

BY MITCHEL L. ZOLER

FROM THE EUROPEAN SOCIETY OF  
CARDIOLOGY CONGRESS 2010

STOCKHOLM – People with a normal ankle:brachial index at rest may still have clinically significant peripheral artery disease that only appears after they walk for a few minutes, according to an analysis of more than 2,100 patients.

An abnormally low ankle:brachial index (ABI) following nonstrenuous exercise proved as prognostic for subsequent mortality as a low ABI at rest, Dr. Inge I. de Liefde said at the European Society of Cardiology Congress 2010. In both cases, an ABI of less than 0.9 identified patients with a greater than twofold risk of dying during 5 years of follow-up, compared with those in the study who had an exercise ABI of at least 1.1. Her analysis also showed that 35% of the people in the study with a resting ABI of 0.9 or greater had an exercise ABI below 0.9, showing that this type of patient is common, said Dr. de Liefde, an anesthesiologist working in the department of vascular medicine at Erasmus University in Rotterdam, the Netherlands.

“Treadmill exercise ABI adds additional, important prognostic information on long-term mortality in patients

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**Major Finding:** Among patients with known or suspected PAD and a normal resting ankle:brachial index (ABI), those with an exercise ABI of less than 0.9 had a significant, 2.56-fold increased risk for death during 5 years of follow-up, compared with patients whose exercise ABI measured at least 1.1.

**Data Source:** Review of 2,164 patients referred to Erasmus University Medical Center during 1993-2005.

**Disclosures:** Dr. de Liefde had no disclosures.

with a normal resting ABI. Based on our results, we recommend that at least patients suspected for PAD [peripheral artery disease], with a resting ABI of 0.9



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DR. DE LIEFDE

or greater who are at least 50 years old and have hypertension undergo treadmill exercise [ABI] testing,” she said.

The people in her study went to Erasmus for evaluation of known or suspected PAD and were all 50 or older with hypertension. But Dr. de Liefde added that

exercise ABI provided a simple and inexpensive diagnostic tool that could also help evaluate a wider adult population.

“Many people have silent PAD that could be diagnosed with the exercise test,” she said in an interview. “It’s a really important and easy test to perform. People with PAD are not always identified by their resting ABI. If exercise ABIs are not measured, you can miss 30%-40% of patients with PAD.”

Although not widely used elsewhere, exercise ABI tests have been done at Erasmus for at least 20 years, she said. Their protocol does not specify a target heart rate. After evaluation for resting ABI, patients walked on a level surface for 5-6 min at 4 km/hour, followed by a repeat ABI measure. The Erasmus staff recorded ankle pressure as the highest systolic pressure measured at either the anterior or posterior tibular arteries,

matched against the highest systolic pressure recorded at the arm.

The 2,164 patients referred to Erasmus during 1993-2005 averaged 63 years old, two-thirds were men and follow-up averaged 5 years. The all-cause mortality rate during follow-up ranged from a low of 5% in patients with a resting ABI of 0.9 or greater and an exercise ABI of at least 1.1, to a high of 26% in patients with a resting ABI of at least 0.9 but an exercise ABI of less than 0.9. The highest follow-up mortality rate, 35%, occurred in patients diagnosed with PAD by a resting ABI below 0.9.

In an analysis that adjusted for cardiovascular risk factors and medication use at the time of ABI measurement, patients with an exercise ABI below 0.9 had a 2.56-fold increased rate of death during follow-up, compared with patients with an exercise ABI of at least 1.1. This increased risk closely matched the 2.73-fold higher rate of death in patients diagnosed with PAD by a resting ABI of less than 0.9. In both cases, the increased risk was statistically significant, Dr. de Liefde said.

In contrast, patients without PAD at rest and an exercise ABI of 0.9-1.09 had no significantly increased mortality rate compared with the reference group. ■

## Heart Failure, MI Risks Rose

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ty, Minn., and participating in the Rochester Epidemiology Project, 1,288 (63%) had one or more clinical risk factors or an echocardiographic abnormality.

The new analysis began with the 2,042 Olmsted County residents and excluded 45 who had symptomatic heart failure, and 6 with a plasma creatinine of more than 2.0 mg/dL.

The researchers measured plasma levels of BNP (specifically amino-terminal pro-BNP) with an immunoassay. These healthy, normal subjects from the community without symptomatic heart failure or elevated creatinine had BNP levels well below the levels found in patients with heart failure, Dr. McKie said.

The analysis examined the risk for death, incident heart failure, or incident MI during an average 9 years of follow-up. People in the highest BNP tertile con-

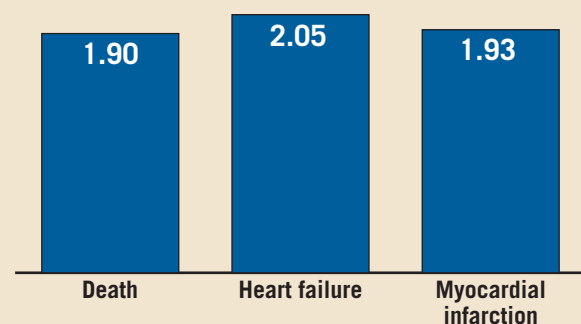
sistently had double the rates of all three outcomes during follow-up as did people in the lowest tertile after adjustment for age, sex, body mass index, clinical risk factors, or echocardiographic abnormalities; differences that were statistically significant (see box below).

The analysis also showed that the link between high BNP levels and adverse outcomes remained significant after adjustment for plasma levels of atrial natriuretic peptide.

“BNP is a more robust prognostic marker than atrial natriuretic peptide in the general population without heart failure,” Dr. McKie said.

Dr. McKie said that he had no disclosures. ■

**Hazard Ratios for Top Tertile of Plasma BNP, Compared With Lowest Tertile**



Notes: Based on 9-year follow-up data from 1,991 people. All hazard ratios were statistically significant.  
Source: Dr. McKie

ELSEVIER GLOBAL MEDICAL NEWS

## High Serotonin Pointed to Decompensated Heart Failure

BY DOUG BRUNK

FROM THE ANNUAL MEETING OF THE  
HEART FAILURE SOCIETY OF AMERICA

SAN DIEGO – Plasma levels of serotonin were significantly elevated in patients with decompensated systolic heart failure, compared with patients in the compensated state and with normal controls, according to a single-center study.

The finding suggests that serotonin has an active role in the progression of heart failure, researchers led by Dr. Ahmed M. Selim reported during a poster session at the annual meeting of the Heart Failure Society of America.

“More studies should be done to test the sensitivity, specificity, and prognostic value of serotonin as a marker for congestive heart failure and also to investigate the therapeutic benefits of the medications affecting this pathway,” wrote the researchers from the department of cardiology at Albert Einstein College of Medicine, New York.

They noted that, while the relationship between heart failure and the serotonergic system has been established in previous research, fluctuations in serotonin levels during the course of the disease and its correlation with exacerbation of heart failure have never been tested.

Dr. Selim, a heart failure research fellow, and his associates collected plasma serotonin levels from 29 patients who were admitted with decompensated heart failure, 61 patients with stable heart failure, and 22

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**Major Finding:** The mean level of plasma serotonin was 2.4 ng/mL in normal patients, 4.1 ng/mL in patients with compensated systolic heart failure, and 11.8 ng/mL in patients with decompensated systolic heart failure.

**Data Source:** A study of 112 patients at the Albert Einstein College of Medicine, New York.

**Disclosures:** The researchers said that they had no relevant financial disclosures to make.

normal controls. They excluded patients receiving medications affecting serotonin receptors and those with pulmonary hypertension. All heart failure patients were on stable doses of heart failure medications and had left-ventricular ejections fractions of 40% or less, while normal controls had a mean ejection fraction of 59%.

Overall, the mean age of patients was 55 years, and 62% were male.

The researchers reported that the mean serotonin level in the control group was 2.4 ng/mL, vs. 4.1 ng/mL in the compensated group and 11.8 ng/mL in the decompensated group. This was independent of age, race, renal function, diabetes, and hypertension. “All results were highly significant,” the researchers wrote.

Dr. Selim and his associates stated that they had no relevant financial disclosures to make. ■