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REPLY

The question of clinical utility of measurement of plasma brain natriuretic peptide (BNP) after an acute myocardial infarction (AMI), raised by Drs. Rashidi and Adler, is most pertinent and relevant. Indeed, a simple measurement of BNP from the blood sample might have clinical importance in estimation of candidacy for implantable cardioverter-defibrillator (ICD) therapy. We would like to add a few more comments to the issue of risk stratification of patients after an AMI and of potential clinical utility of BNP measurement.

The post hoc analysis of the Multicenter Automatic Defibrillator Implantation Trial II (MADIT II) investigators showed that patients with a recent AMI (<18 months) did not have a mortality benefit from the ICD therapy (1). Similar preliminary data were presented at the American College of Cardiology meeting in March 2004 (unpublished) from the Defibrillator In Acute Myocardial Infarction Trial (DINAMIT), where ICD therapy did not reduce all-cause mortality among the patients with a recent AMI (ejection fraction [EF] <0.35) and reduced heart rate variability. Therefore, evaluation of the candidacy for ICD therapy shortly after an AMI is still a challenge and an obvious field for further research.

Our study showed that patients with a low plasma BNP level, measured at the convalescent phase after an AMI, have an extremely low incidence of sudden cardiac death (SCD) (2). Our main implication is that the measurement of BNP can be used to identify the "low-risk" patients who may not need further risk stratification and may not benefit from prophylactic ICD despite depressed left ventricular function. For example, there were no SCDs among the patients with an EF <0.35 and low BPN (<23.0 pmol/l) during the follow-up. Also, BNP did not reach statistical significance in prediction of SCD among patients with an EF between 0.30 and 0.40. However, elevated BNP had a better predictive power among those with preserved left ventricular function (EF >0.40) (relative risk 3.9; 95% confidence interval 1.0 to 16.5; p = 0.05). Patients with an EF >0.40 also constituted the highest cumulative number of SCD events. Prediction and prevention of SCD among the large number of survivors of AMI who have a preserved left ventricular function will be important in future efforts aimed at reducing the overall burden of premature SCD. Measurement of BNP may have clinical value in this respect.

In conclusion, we believe measurement of BNP at the convalescent phase after an AMI is most suitable for excluding patients at risk for future SCD. Evaluation of the candidacy for ICD therapy shortly after an AMI still remains a challenge both for scientists and clinicians.

Heikki V. Huikuri, MD, FACC
Jari M. Tapanainen, MD
Timo H. Mäkikallio, MD
Division of Cardiology
Department of Internal Medicine
University of Oulu
P.O. Box 5000
Kajaanintie 50
Oulu, FIN-90014
Finland
E-mail: heikki.huikuri@oulu.fi

doi:10.1016/j.jacc.2004.07.007

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Does Nondipping Blood Pressure Profile Contribute to Vascular Inflammation During Sleep Deprivation?

We read with great interest the study by Meier-Ewert et al. (1) describing elevated high sensitivity C-reactive protein (hs-CRP) in healthy subjects after both acute total and partial sleep deprivation. The investigators hypothesized that such low-grade inflammation associated with sleep loss might contribute to the increased cardiovascular risk described in sleep complaints.

As far as the mechanisms responsible for such increment of circulating hs-CRP levels after sleep deprivation, the researchers suggest a role for vascular shear stress exacerbated by increased blood pressure (BP) levels (1). In this context, a trend toward an increment of systolic and diastolic BP was observed during both total and partial sleep deprivation. However, only changes of systolic BP across total sleep deprivation achieved statistical significance. In addition, circulating hs-CRP levels significantly increased, starting with the first day of total sleep deprivation, despite irrelevant changes of systolic and diastolic BP. Thus, sleep deprivation-related increments of BP do not completely explain the interesting findings by Meier-Ewert et al. (1).

In this regard, sleep disturbances strongly affect both nighttime blood pressure levels and the nocturnal BP drop in healthy subjects as well as in hypertensive patients (2). The blunted nocturnal decline in BP, also known as "nondipping profile," has been reported to be associated with increased vascular damage, at least in hypertensive patients (3). In keeping with this, a nondipping profile has