

# Clinical Briefs in Primary Care<sup>™</sup>

The essential monthly primary care update

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## The Metabolic Syndrome and Total and Cardiovascular Disease Mortality in Middle-Aged Men

**Source:** Lakka HM, et al. *JAMA*. 2002;288:2709-2716.

THE METABOLIC SYNDROME (MBS) HAS 2 currently popular definitions. According to the National Cholesterol Education Program, MBS exists when a patient has at least 3 of the following characteristics: fasting glucose (FPG) > 110 mg/dL, abdominal obesity, triglycerides > 150, HDL < 40 mg/dL, and elevated blood pressure (> 130/85). The World Health Organization (WHO) definition stratifies things just a bit differently, defining MBS as either hyperinsulinemia (upper quartile of the adult, nondiabetic population) or FPG, and any 2 or more of abdominal obesity, dyslipidemia (triglycerides > 150 mg/dL or HDL < 35), and BP > 140/90. Despite these modest differences, the criteria basically define the same group of individuals. Lakka and associates prospectively studied for a mean of 11.6 years a random, age-stratified sample of men in Finland (n = 2682) aged 42 and older, to examine cardiovascular and overall mortality in relation to MBS.

MBS patients had reduced (79%) Kaplan-Meier estimates of overall survival when compared with patients without MBS. Similarly, CHD mortality was 2.4-3.4 times higher in persons with MBS. The prevalence of MBS at baseline was 9-14%. The public health impact of MBS is substantial. Whether specific treatment of MBS will reduce mortality has not been determined. ■

## Amlodipine Fosinopril Combination on Microalbuminuria in Hypertensive Type 2 Diabetic Patients

**Source:** Fogari R, et al. *Am J Hypertens*. 2002;15:1042-1049.

NUMEROUS STUDIES HAVE CONFIRMED the role of ACE inhibitors in modulation of microalbuminuria. The data on effects of calcium channel blockers (CCB) have been conflicting, especially as concerns dihydropyridine CCB (eg, amlodipine, felodipine, nifedipine). Fogari and associates addressed the effects of fosinopril (FOS) and amlodipine (AML), alone or in combination (COM), in an open-labeled, randomized, prospective, parallel group study for 4 years (n = 309).

By 3 months' time, the FOS group had demonstrated a decline in urinary albumin excretion (UAE), which decreased slightly further in the first year, and then stabilized. The AML group also demonstrated a decline in UAE, but not until 18 months into the study, after which point the UAE stabilized. COM therapy produced an impact at 3 months, which increased at 12 months and again at 36 months, and was statistically significantly greater than either monotherapy.

The mechanism by which COM therapy is superior to either monotherapy is uncertain, but the greater reduction in BP achieved (approximately 12/5 greater reduction by the former) is thought to have figured prominently. ■

## Relation Between Alcohol Consumption and C-Reactive Protein Levels in the Adult United States Population

**Source:** Stewart SH, et al. *J Am Board Fam Pract*. 2002;15:437-442.

EPIDEMIOLOGIC DATA CONSISTENTLY indicate that moderate intake of alcohol (ETOH) is associated with reductions in cardiovascular mortality. Though the mechanism by which this effect is achieved is uncertain, increases in HDL by alcohol may explain as much as 50% of the protective effect.

C-reactive protein (CRP) is increasingly recognized as an independent risk factor for cardiovascular endpoints, suggesting an important role of inflammation in promoting atherosclerotic events. To evaluate the relationship between CRP and ETOH, Mainous and associates analyzed data from the National Health and Nutrition Evaluation Survey (NHANES III), which included complete information on 11,572 US adults.

Almost half of the NHANES population were alcohol abstainers; CRP levels in abstainers were significantly greater than in those who drink alcohol, regardless of level of alcohol ingestion. The mechanism by which ETOH might reduce CRP (or inflammation) remains unknown. A small trial of ETOH in healthy volunteers has shown a reduction in CRP and is stimulus for follow-up evaluation in larger studies. ■

# Prostate Cancer Screening

**Source:** Ransohoff DF, et al. *Am J Med.* 2002;113:663-667.

**I**N CONTRAST TO SCREENING FOR breast and colon cancer, both of which have been demonstrated to reduce mortality, prostate cancer screening (PCS) has not yet been proven to favorably affect overall mortality, although some trials have found that PCS screening reduces prostate cancer-related mortality. Hence PCS has not met the same standard as other commonly used screening tools. Because of the discordance between the relative lack of supportive data to provide justification for PCS and the very high frequency of PCS testing, Ransohoff and colleagues sought to evaluate what factors promote PCS. That PCS can result in harm (eg, postsurgical impotence, incontinence) is clear; whether PCS can provide benefit (ie, reduction in mortality) remains to be demonstrated.

Ransohoff et al describe the PCS model as “lacking negative feedback:” a patient who undergoes PCS and has no cancer-suggestive findings feels reassured by these findings and is happy to have partici-

pated; a patient who has an elevated PSA often undergoes medical or surgical intervention. Even in the face of postintervention sequelae, the screened patient may feel that, ultimately, the intervention has spared his life, and he too may be grateful for the PCS.

Currently, whether PCS is mortality-effective is uncertain. Nonetheless, public satisfaction and enthusiasm for PCS remains high. It is conceivable that, in the long run, harm from PCS-stimulated intervention may outweigh benefit. Until the relative risks and benefits of PCS are more clearly defined, clinicians are well advised to review the decision path of PCS with patients before the process is embarked upon, in order that fully informed consent, dispassionately, may be attained. ■

## Can We Trust Home BP Measurement?

**Source:** Bachmann LM, et al. *J Clin Hypertens.* 2002;4:405-407,412.

**T**HE WINDOW OF OBSERVATION OF blood pressure as obtained in the typical office setting has important limitations, with both exaggerations (ie, “white-coat” hypertension), and underestimates (ie, “masked hypertension”) of hypertension burden being well documented. Abnormal circadian BP patterns, such as failure to experience the normal nocturnal decline in blood pressure, predict higher cardiovascular risk yet are not discerned by simple office measurement. Twenty-four-hour Ambulatory Blood Pressure Monitoring (ABPM) can resolve all 3 of these issues but is not without significant expense, and despite the endorsement of ABPM by the JNC VI report and the WHO guidelines, this technique remains only rarely used. Whether home blood pressure measurement, perhaps an intermediate step between office measurement and ABPM, is reliable is the subject of this report.

Bachmann and colleagues included 48 hypertensive patients from a single practice, who had been referred for 24-Hour ABPM. Subjects were randomly assigned to either a group which was asked to keep a personal log of the BP measurements recorded by the ABPM, and advised that their log would be checked for accuracy

against that registered by the ABPM device, or a group who were also advised to periodically record BP measurements as registered by the ABPM device, but who were unaware that the ABPM automatically records and stores BP measurements. Discrepant results occurred when patient-recorded records either had an incorrect time, an incorrect BP value, or a BP was entered as recorded when the ABPM device had not performed such a measurement. Although patients unaware of the ABPM recording capacity were found to have more “fictional” registrations than the “informed” group (10/728 vs 29/616), ultimately these discrepant recordings did not confound the overall mean accuracy of averaged home blood pressure readings. ■

## Systolic and Diastolic Dysfunction

**Source:** Redfield MM, et al. *JAMA.* 2003;289:194-202.

**C**ONGESTIVE HEART FAILURE (CHF) IS typically classified as systolic (ie, reduced ejection fraction), diastolic (normal ejection fraction, with impaired ventricular filling), or both. Indeed, though CHF may have been generally conceptualized solely as “inadequate pumping,” some degree of diastolic dysfunction accompanies almost all patients suffering systolic dysfunction. Additionally, isolated diastolic dysfunction, which may present with identical clinical symptoms as systolic dysfunction, has recently been recognized to be approximately as common as systolic dysfunction in patients with manifest CHF. Redfield and associates evaluated with doppler echocardiography adults older than 45 years of age participating in the Rochester (Minnesota) Epidemiology Project (n = 2042), none of whom entered the study with a diagnosis of CHF.

In this asymptomatic (for CHF) group, validated CHF prevalence was 2.2%, approximately equally divided between systolic and diastolic dysfunction. Diastolic dysfunction, whether mild, moderate, or severe, was found by multivariate analysis to be predictive of all-cause mortality. This trial indicates that diastolic dysfunction, previously regarded as more “benign” than systolic dysfunction, portends significant adverse health outcomes. ■

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