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Effect of Lipid-Lowering Therapy on Coronary Artery Calcification

ABSTRACT & COMMENTARY

Synopsis: *Cerivastatin therapy resulted in decreased progression of coronary calcifications.*

Source: Achenbach S, et al. *Circulation.* 2002;106:1077-1081.

Several studies have clearly demonstrated that the quantity of coronary artery calcification measured by electronic beam computed tomography (EBCT) normally increases at a variable rate over time[1-6] reaching as high as 24% annually.[2] Retrospective analyses have revealed that lipid-lowering therapy slows the rate of coronary artery calcium deposition[3,4] but thus far, no prospective investigation that demonstrates the effects of lipidlowering drug treatment on the rate of progression or regression of coronary calcification has been published.

Achenbach and colleagues conducted a cohort study that prospectively compared the rate of change in the amount of coronary calcification before and after lipid-lowering therapy with the cholesterol synthesis enzyme inhibitor cerivastatin. EBCT studies were performed in 66 patients before treatment with cerivastatin and repeated 14 months later after which treatment with cerivastatin (0.3 mg/d) was initiated. A third EBCT study was completed 12 months after treatment initiation and all coronary calcifications were quantified using a volumetric score. Cerivastatin therapy lowered the mean LDL cholesterol level from 164 mg/dL to 107 mg/dL. The median annual relative increase in coronary calcium score was 25% during the untreated period vs. 8.8% 1 year after the lipid-lowering therapy was administered. Equally important, in 32 patients who demonstrated LDL cholesterol levels < 100 mg/dL during drug treatment, the median relative increase in the coronary calcium score was 27% during the control period vs. -3.4% during the treatment period.

COMMENT BY HAROLD L. KARPMAN, MD, FACC, FACP

The measurement of coronary artery calcification with EBCT

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is rapidly earning respect as a powerful early marker of the atherosclerotic burden in the coronary arteries. Although there has been some variability previously reported in coronary calcification scores obtained by EBCT studies, Achenbach et al attempted to control interscan variability by using a standard ECG triggering mechanism and by measuring progression over 2 consecutive time periods in the same individual rather than comparing 2 simultaneous groups over the same time period. Although there were no significant changes in objective measurements (ie, body weight) in the subjects, one must recognize that some of the patients may have modified their lifestyle after being made aware of an abnormality in their coronary artery calcium score early in the study. Of note is the interesting fact that regression of the coronary calcium score was observed in only 7 patients during the control period compared with 24 patients who were receiving the lipid-lowering therapy.

Although this prospective study has many limitations (ie, lack of medication control, interscan variability, etc), it would appear that quantification of coronary artery calcification by EBCT or ultrafast CT will prove to be a promising tool for the assessment of coronary artery calcification progression and/or regression. How this translates into prediction of outcomes or in the evaluation of changes in the coronary atherosclerotic plaque burden and composition remains to be determined. Although many physicians are disturbed by the intense commercialization of CT and coronary artery calcification scoring, it would appear to be important to remember that one should be cautious about throwing out the baby with the bathwater—properly used, the value of these techniques in contemporary clinical practice may prove to be invaluable. For the time being, even though there are many limitations to the Achenbach study, there seems to be little question that lipid-lowering agents may have significant value in diminishing the progression or even possibly contributing to the regression of the coronary atherosclerotic burden. ■

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Menopausal Hormone Replacement Therapy and Risk of Ovarian Cancer

ABSTRACT & COMMENTARY

Synopsis: *There appears to be an increase in ovarian cancer with long-term estrogen-only hormone replacement therapy.*

Source: Lacey J, et al. *JAMA.* 2002;288:334-341.

BETWEEN 1973 AND 1980, THE AMERICAN CANCER Society and the National Cancer Institute conducted the Breast Cancer Detection Demonstration Project

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(BCDDP). More than one quarter of a million women were involved in this study. Follow-up of these participants began in 1979, and 4 phases have been completed. Questions concerning hormone therapy were asked. Initially no distinction was made between estrogen-only replacement therapy (ERT) and estrogen-progestin replacement therapy (EPRT). A distinction was made beginning in 1987.

Using appropriate exclusions, 44,247 women were available for inclusion in this study. Diagnoses of ovarian cancer were verified through medical record review, cancer registry information and the National Death Index. Three hundred twenty-nine cases of ovarian cancer were diagnosed, virtually all of the epithelial type. The mean length of follow-up of the women in the study was 13½ years. The mean age at the start of follow-up was slightly less than 57 years. Parity, use of oral contraceptives, and hysterectomy were inversely associated with ovarian cancer. Estrogen-only hormone replacement therapy was significantly associated with ovarian cancer. Longer use of estrogen resulted in higher risk ratios. For those women who used ERT for 20 years or more, there was a greater than 3-fold increase in ovarian cancer. The increase was approximately 7% per year of ERT usage. There was no significant increase in risk for those women who took only EPRT.

COMMENT BY KENNETH L. NOLLER, MD

This important study was greatly overlooked because of the tremendous publicity surrounded by the decision of the Women's Health Initiative not to continue the estrogen plus progestin arm of their study due to various increased risks. That is unfortunate as this well-done study suggests that there also are risks associated with estrogen-only therapy.

Over the past several years, I have frequently commented on study design, *P*-values, and confidence intervals. Certainly the most powerful type of study uses a prospective, randomized study (experimental) design. Cohort studies, such as this report, are less powerful. Nonetheless, if performed properly and if the risk ratio is significantly elevated, they are quite believable. While an increase of risk of 20% or 30% is usually believable in a prospective randomized trial, a similar increase in a cohort study is questionable.

In this study by Lacey et al, there was a 3-fold (300%) increase in ovarian cancer among estrogen-only hormone replacement users. There was a smaller increase in women who took the medication for only 10 years, but there was a definite dose response association. That is, the risk of ovarian cancer increased with increasing length of usage.

This is quite a good study and it could stand alone. However, other articles have found a similar association, and thus I think it is reasonable for us to believe that the use of estrogen replacement therapy may actually increase a woman's risk of developing epithelial ovarian cancer. What is harder to understand is why this association would be true. So far I am not aware that anyone has developed a believable pathophysiological explanation. ■

Dr. Noller is Professor and Chairman, Department of OB/GYN, Tufts University School of Medicine, Boston, Mass.

Does Treating Inflammation Improve Lipid levels?

ABSTRACT & COMMENTARY

Synopsis: *Antirheumatic therapy lowers lipid levels in patients with rheumatoid arthritis.*

Source: Park YB, et al. *Am J Med.* 2002;113:188-193.

Park and colleagues have previously noted adverse lipid profiles in patients with rheumatoid arthritis (RA). Since inflammation may be associated with atherosclerosis, Park et al sought to determine the effects of treating RA with antirheumatic drugs on the abnormal lipid levels.

They selected 42 patients with newly diagnosed RA who had not been treated with corticosteroiods or disease modifying antirheumatic drugs. Serum lipid levels were measured at baseline and at one year. They then determined whether there were differences in the changes in lipid levels between patients who met the American College of Rheumatology criteria for a 20% improvement in RA and those who did not.

Of the 42 patients, 27 (64%) met the criteria for a 20% improvement in RA activity criteria during the 12-month study. In these patients, mean high-density lipoprotein (HDL) cholesterol levels increased by 21% ($P < 0.001$), and apolipoprotein A-1 levels increased by 23%, ($P < 0.001$). Among the 13 nonresponders, mean HDL levels and mean LDL to HDL ratio did not change significantly. The mean apolipoprotein A-1 level increased by 10%; $P = 0.02$. Among the responders, the number of HDL levels < 35 mg/dL decreased significantly (from 9 to 2

patients; $P = 0.04$). Prednisone and methotrexate were not associated with significant differences in any of the lipid levels. The benefits were achieved without using lipid-lowering agents.

Park et al concluded that active RA is associated with an adverse lipid profile that improves substantially following effective treatment of the RA. This improvement may reduce the risk of cardiovascular disease in this group of patients.

■ **COMMENT BY RALPH R. HALL, MD, FACP**

These studies are important not only because there is an increase in cardiovascular morbidity and mortality in RA but because there is increasing evidence supporting the hypothesis that atherosclerotic vascular disease shares many similarities with other inflammatory/autoimmune diseases.[1]

It is interesting to note that the C-reactive protein in the patients in this study was 10 times higher than in patients without inflammatory disease. Park et al have previously reported that there was a strong correlation between lipid levels and C-reactive protein and that as lipid levels improved in RA patients the C-reactive protein levels decrease.[2]

Park et al point out in their discussion that HDL and apolipoprotein A-1 are negatively correlated with disease activity in several rheumatic disorders, including Kawasaki's disease, systemic lupus erythematosus, Behcet's disease, and gout. In animal models, inflammation decreases the levels of HDL and apolipoprotein A-1 during the acute stage response.

It is interesting to note that the improvement in HDL and apolipoprotein A-1 in this study is greater than one would expect from statins and equal to the response to nicotinic acid.[3,4]

Since there are animal models for RA and tissue is readily available for study there may be avenues for understanding the inflammatory and immunologic responses of atherosclerosis. There are no such tools to investigate and follow the inflammatory response in arteriosclerotic plaques at the present time. As Pasceri and Yeh have stated "the study of the molecular mechanisms of RA may give valuable hints for research on the inflammatory/immunological mechanisms for atherosclerosis and acute coronary syndromes."[1] ■

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The Ambulatory BP Monitoring Effect

ABSTRACT & COMMENTARY

Synopsis: *The presser effect of ABPM for the first time could influence the diagnosis of hypertension.*

Source: Hermida RC, et al. *J Am Coll Cardiol.* 2002;40: 710-717.

AMBULATORY BLOOD PRESSURE MONITORING (ABPM) is a useful adjunct for evaluating white coat hypertension, but little is known about its effect on BP. Thus, Hermida and associates from Spain studied 538 patients with mild-to-moderate office hypertension using ABPM for 48 hours. Approximately one third were on no therapy and one third had more than 1 study. In both treated and untreated patients, significant reductions in BP were noted on the second day ($P < .001$), but not in heart rate. This presser effect was statistically significant for the first 6-9 hours of monitoring. Mean nocturnal BP was the same on both days. In those with repeated tests 3 months apart, this presser effect was absent. Hermida et al concluded that this presser effect of ABPM for the first time could influence the diagnosis of hypertension.

■ **COMMENT BY MICHAEL H. CRAWFORD, MD**

This study makes a strong case for extending initial ABPM beyond 24 hours in order to accurately diagnose white coat hypertension. Apparently the novelty of the first several hours of experience with the device elevates BP significantly compared to the second day whether it is contiguous (48 hours of monitoring) or 3 months later. The magnitude of the pressure drop on day 2 averaged 6-7 mm Hg systolic and 4-5 mm Hg diastolic. Also, about three quarters of the subjects experienced a significant decline in BP. Interestingly, the results were not related to heart rate or levels of physical activity. In addition, the findings were independent of sex and BP treatment. This study is robust because of a large number of subjects with a range of blood pressure and other characteristics. The application of a 2-day monitoring rule will help more accurately define hypertensive subjects and lead to better clinical and research outcomes, albeit at a higher cost. ■

Dr. Crawford is Professor of Medicine, Mayo Medical School; Consultant in Cardiovascular Diseases, and Director of Research, Mayo Clinic, Scottsdale, Ariz.

IV Magnesium Sulfate in the Treatment of Acute Severe Asthma

ABSTRACT & COMMENTARY

Synopsis: *The end point of FEV_1 at 240 minutes favored the use of MgSO4 for the most severe group.*

Source: Silverman RA, et al. *Chest.* 2002;122:489-497.

Silverman and colleagues used the emergency departments in 8 hospitals to assess the role of MgSO4 in acute asthma in a sizeable number of patients (n = 248). They segregated their findings by severity of asthma. Those with less than 25% of predicted FEV_1 on admission had a statistically significant favorable outcome of predicted FEV_1, and had results similar to placebo. They did not include pediatric patients, and continued the nebulized albuterol and IV methylprednisolone, in addition to the 2 g of magnesium that was administered 30 minutes after arrival in the emergency department. However, the end point of FEV_1 at 240 minutes favored the use of MgSO4 for the most severe group.

COMMENT BY SHELDON L. SPECTOR, MD, FACP, FAAA, FACA

Variables that have contributed to the controversy regarding MgSO4 in the literature include: 1) the age of the patients who were studied; 2) initial pulmonary function at the time of arrival in the emergency room; 3) dose of MgSO4 used; 4) route of administration; 5) end point results; 6) the number of patients included in the study; and 7) type of blinding and randomization. Although the majority of trials included adult patients, a few were conducted in the pediatric age group. In fact, 4 trials using pediatric patients support the use of MgSO4 in asthma,[1-4] and one does not.[5] In pediatric studies, a weight-based dosing regimen was usually done and this regimen varied from 25-100 mg/kg.

Some published case reports included asthmatic patients who failed to respond to traditional treatments such as albuterol, aminophylline, and corticosteroids. The mechanism of action of magnesium in alleviating an asthmatic attack is unknown. However, the ability of magnesium to impede movement of calcium and decrease the uptake of calcium by the bronchial smooth muscle is associated with bronchodilatation and inhibition of degranulization of mast cells. Since calcium also has a role in triggering the release of thromboxane and leukotrienes, it may antagonize these mediators, as well. The possibility that magnesium deletion may actually occur with adrenergic excess is another hypothesis. Side effects reported with MgSO4 included flushing or facial warmth; dry mouth; and fatigue. With rapid infusions, bradycardia and hypotension have occurred.

This study contributes to our knowledge regarding which subgroup of severe asthmatic patients might best respond to MgSO4, ie, with an FEV_1 of less than 25% when first seen. The literature might also favor its use in the pediatric population, although more studies are needed. ■

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Increased Caloric Intake Increases the Risk of Alzheimer's Disease

ABSTRACTS & COMMENTARY

Synopsis: *Compared with individuals in the lowest quartile of caloric intake, those in the highest quartile had an increased risk of AD, which showed a hazard ratio of 1.5.*

Sources: Luchsinger JA, et al. *Arch Neurol.* 2002;59: 1258-1263; Roth GS, et al. *Science.* 2002;297:811.

There is evidence that diet could play a role in Alzheimer disease (AD) risk. Caloric intake has been shown to affect aging in animals and, possibly, in humans. Caloric restriction in mice and rats increases average and maximum life span possibly by decreasing oxidative damage. The balance of macronutrients in diet may also affect oxidative stress unrelated to total caloric intake. There is increasing evidence that oxidative damage may increase β-amyloid deposition as well as the intracellular accumulation β-amyloid 1-42.

In the present epidemiologic study, 980 individuals free of dementia at baseline were followed for a mean of 4 years. The daily intake of calories, carbohydrates, fats, and proteins were recalled using a semi-quantitative

food frequency questionnaire that was administered between baseline and the first follow-up visits. Luchsinger and colleagues subsequently examined the risk of AD between quartiles of intake after adjusting for confounders. They found 242 incident cases of AD during the 4023 years of follow-up. Compared with individuals in the lowest quartile of caloric intake, those in the highest quartile had an increased risk of AD, which showed a hazard ratio of 1.5. In individuals with apolipoprotein E ∈4 allele, the hazard ratios of AD for the highest quartile of caloric or food intake were 2.3 as compared to the lowest quartiles. In individuals who did not have the apoliprotein E ∈4 allele, there was no significant effect of caloric intake or fat intake on AD risk.

COMMENT BY M. FLINT BEAL, MD

The main limitation of this study pertains to the measure of nutrient intake. The food frequency questionnaire measures habitual intake during 1 year and does not account for day-to-day variation or longer-term periods of intake. In addition, the measures obtained from the semi-quantitative food frequency questionnaire may not have sufficient precision to make inferences about absolute levels of nutrient intake. Nevertheless, the findings are of interest. This is due to the abundant literature which shows that reduced caloric intake has neuroprotective effects in animal models of neurotoxicity. Reduced caloric intake may reduce oxidative stress and increase both average and maximum lifespan. The relevance of this for normal human aging has recently been shown in a report in *Science* that biomarkers of caloric restriction may predict longevity in humans. In the NIA primate aging study of male rhesus monkeys, caloric restriction resulted in a slightly lower body temperature, reduced insulin levels, and slowed the rate of decline in serum dehydroepiandrosterone sulfate. In the Baltimore longitudinal study of aging in male humans, it was demonstrated that all 3 of these markers showed significant effects on survival. Consistent with beneficial effects of caloric restriction on aging and life span in other animals, individuals with lower temperature and insulin, and those maintaining higher dehydroepiandrosterone sulfate levels had longer survival than their respective counterparts.

These findings both in AD as well as in normal humans suggest that a number of markers associated with caloric restriction may predict increased longevity and reduce incidence of AD. Further studies of these interactions are warranted. ■

Dr. Beal is Professor and Chairman; Department of Neurology; Cornell University Medical College, New York, NY

Pharmacology Update

Eplerenone Tablets (Inspra Searle)—A New Aldosterone Antagonist

By William T. Elliott, MD, FACP, and James Chan, PharmD, PhD

A NEW ALDOSTERONE RECEPTOR ANTAGONIST HAS been approved by the FDA. Eplereone, a selective aldosterone receptor antagonist (SARA), is the first approval in this class since the introduction of spironolactone close to 3 decades earlier. It is chemically similar to canrnone, an active metabolite of spironolactone. Eplerenone will be marketed as Inspra by Searle.

Indications

Eplerenone is indicated for the treatment of hypertension and may be used as monotherapy or in combination with other antihypertensive drugs.[1]

Dosage

The recommended starting dose is 50 mg once daily and may be increased to 50 mg twice daily if adequate blood pressure reduction is not achieved within 4 weeks. No dose adjustment is recommended for patients with mild-to-moderate hepatic impairment.[1]

The drug is contraindicated in patients with serum potassium > 5.5 meq/L, serum creatinine > 2.0 mg/dL in males or > 1.8 mg/dL in females, creatinine clearance < 50 mg/min, and type 2 diabetes with microalbuminuria. Patients should not use potassium supplements or potassium containing salt substitutes.

Eplerenone is available as 50 mg and 100 mg tablets.

Potential Advantages

Eplerenone has greater selectivity for the aldosterone receptor and minimal affinity for other steroid receptors. Potential progestational and antiandrogenic side effects appeared to be less common than with spironolactone.[2] Eplerenone appears to be more effective in African-American patients and patients with low-renin hypertension than losartan.[4,5]

Potential Disadvantages

Eplerenone is less effective than spironolactone in the reduction of blood pressure. A 100 mg daily dose (50 mg twice daily) of eplerenone produces about 75% of the blood pressure reduction compared to 100 mg of

spironolactone.[3] Eplerenone is metabolized by the cytochrome P450 3A4 isoenzyme. Concomitant use with strong inhibitors of this isoenzyme (ie, itraconazole, ketoconazole) is contraindicated.[1]

Comments

Eplerenone is the first competitive aldosterone receptor antagonist to be approved since spironolactone was introduced in the 1970s. Comparative trials suggest that eplerenone is somewhat less effective than spironolactone at equal milligram doses. In an 8-week study in patients with mild-to-moderate hypertension, the reduction in systolic and diastolic blood pressure for 50 mg of eplerenone twice daily (n = 54) and 100 mg daily (n = 49) was approximately 50-75% of that achieved with spironolactone 50 mg twice daily (n = 48).[3] Changes in SBP and DBP from baseline were -16.7 mm Hg and -9.5 mm Hg, respectively, for spironolactone. For eplerenone it was -11.9 mm Hg and -7.8 mm Hg with twice daily dosing and -7.9 mm Hg and -4.4 mm Hg with once daily dosing. A similar trend was reported for 24-hour ambulatory blood pressure monitoring. Eplerenone appears to be equally effective in African-American patients as well as patients with low-renin hypertension.[7] The antihypertensive effect of eplerenone was assessed when added to existing angiotensin converting enzyme inhibitors or angiotensin II receptor antagonist.[6] The frequency of gynecomastia or abnormal vaginal bleeding was 1% and 2.1% in the long-term study.[1]

Eplerenone is currently being evaluated in patients with heart failure as a complication of acute myocardial infarction. The 6200-patient randomized EPlerenone's neurolHormonal Efficacy and Suvival Study (EPHESUS) is expected soon. The primary end points are all-cause mortality and cardiovascular mortality or cardiovascular hospitalization.[9] Secondary end points include time to first event, health status assessment, and economic outcomes. Cost for eplerenone was not available at the time of this review.

Clinical Implications

Nepherenone provides an alternative to spironolactone with possibly a lower frequency of progestational and antiandrogenic side effect. Aldosterone has been reported to have an important role in the cardiovascular pathophysiology as demonstrated by the benefit of spironolactone in severe heart failure.[8] The findings of EPHESUS and other studies involving nepherenone, such as diabetic nephropathy, will define the future role of this new selective aldosterone antagonist. ■

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CME Questions

22. Which one of the following statements is *false*?

a. The increase in the level of HDL cholesterol with treatment with antirheumatic drugs is greater than one would expect with statins.
b. The increase in HDL cholesterol with treatment with antirheumatic drugs is less than one would expect with nicotinic acid.
c. HDL cholesterol levels fall during acute phase infections.
d. Studying the molecular mechanisms of rheumatoid arthritis may be helpful in understanding atherosclerosis.

23. According to the article by Lacey et al, all of the following were found to decrease the risk of developing epithelial ovarian cancer *except*:

a. pregnancy.
b. oral contraceptive use.
c. hysterectomy.
d. estrogen replacement therapy.

24. Repeated ambulatory BP monitoring shows:

a. lower daytime BP on day 2.
b. Higher daytime BP on day 2.
c. Lower nocturnal BP on day 2.
d. Higher nocturnal BP on day 2.

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By Louis Kuritzky, MD

Daily Vitamin E and Multivitamin-Mineral Supplementation and Acute RTI in Elderly Persons

VITAMIN SUPPLEMENTATION HAS been shown to improve cellular immune parameters, but whether vitamin E or multivitamins/minerals (MVIM) have an effect on clinical events has not been clearly elucidated. Since respiratory tract infections (RTI) may become especially consequential for senior citizens, the question of whether vitamin E or MVIM alter the frequency, severity, or duration or such infections is of great clinical relevance.

Graat and associates studied the effect of MVIM, containing traditional RDA levels of multiple vitamins and minerals, including zinc, selenium, iron, magnesium, copper, iodine, calcium, manganese, chromium, molybdenum, and silicium, as well as a separate vitamin E supplement of 200 mg. Study subjects (n = 652) were comprised of noninstitutionalized persons older than age 60 who were followed for 15 months. At baseline, a very small proportion of individuals had suboptimal serum levels of either ascorbic acid (6%) or alpha-tocopherol (1.3%).

MVIM supplementation demonstrated no clinically or statistically significant effect upon RTI incidence, severity, duration, number of symp toms, or restriction of activity. Vitamin E supplementation demonstrated worse outcomes than placebo in reference to illness severity, duration, symptoms, fever, and restriction of activity. Graat et al caution that not only do their data discourage employment of MVIM due to lack of efficacy, but also due to a deleterious effect of vitamin E. ■

Graat JM, et al. JAMA. 2002;288: 715-721.

B-Type Natriuretic Peptide Levels and Outcome in Patients with Heart Failure

BRAIN NATRIURETIC PEPTIDE (BNP) levels reflect the degree of cardiac ventricular wall stress and are useful to diagnose chronic heart failure (CHF), as well as differentiate other dyspnea syndromes (in which BNP levels are not elevated) from CHF. BNP levels correlate with severity of CHF, hence, in any one episode of CHF, their degree of elevation might provide prognostic information. Bettencourt and colleagues examined the relationship between hospital BNP levels (on admission and discharge) in persons with acute decompensation of CHF, and subsequent hospital CHF readmission or death.

All subjects (n = 43) received "standard" CHF treatment, including diuretics (furosemide, and in some cases, spironolactone) and ACE inhibitors. Subjects were followed for 6 months.

When patients were hospitalized for CHF, BNP levels typically decreased with treatment. After hospital discharge, in the group that remained event free during follow-up, the decline in BNP during hospitalization (47%) was much more substantial than the decline in persons who required readmission (17%). Patients whose BNP increased during the index admission were more than 3 times more likely to require readmission or die during follow-up. BNP, and its response to treatment, provides important prognostic information in persons with CHF. ■

Bettencourt P, et al. Am J Med. 2002; 113:215-219.

Influence of Companions During Primary Care Medical Encounters

IT IS COMMONPLACE IN PRIMARY CARE settings for patients to be accompanied by family, friends, or caretakers in the examining room during some portion or all of the clinician-patient interaction. The effect of the "third person" (3P) has received little literature scrutiny. Schilling and colleagues studied 226 adult medical encounters, approximately half of which included another accompanying adult who spent any portion of the visit in the examining room. Patients, companions, and clinicians rated the influence of the companion upon the visit. Aspects of the clinical encounter that were monitored included physician understanding, patient understanding, counseling time, length of visit, treatment, referrals, and number of tests ordered.

Physicians reported that having a companion present generally was either neutral to or increased physician and patient understanding. Almost universally, physicians perceived no effect upon treatments, referrals, or number of tests ordered whether a companion was present. On the other hand, 25-32% of physicians felt that the 3P caused an increase in the length of visit or time spent counseling. Although overall the presence of an adult companion may enhance physician and patient understanding, it appears to be potentially at the expense of greater time required for counseling and the visit itself.

Schilling LM, et al. J Fam Pract. 2002;51:685-690.

In Future Issues: For Better or Worse