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Which Smokers Get Lung Cancer?

ABSTRACT & COMMENTARY

By Barbara A. Phillips, MD, MSPH

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Director, Sleep Disorders Center, Samaritan Hospital, Lexington

Dr. Phillips reports no financial relationship to this field of study.

Synopsis: *The presence of emphysema by CT scan or of airflow obstruction by spirometry predicts an increased risk of lung cancer.*

Source: Wilson DO, et al. Association of radiographic emphysema and airflow obstruction with lung cancer. *Am J Respir Crit Care Med* 2008;178:738-744.

THIS REPORT COMES FROM THE PITTSBURGH LUNG SCREENING study, an ongoing study of individuals at high risk for development of cancer. At baseline, recruited individuals underwent low-dose CT scanning and spirometry, and completed an extensive questionnaire. In addition to questions about occupation, smoking, and general medical history, the questionnaire asked participants to report if they had been told by a physician that they had chronic bronchitis, emphysema, or asthma. They were also asked if they ever had hemoptysis, a dry or productive cough, wheezing, or shortness of breath. CT scanning was repeated at approximately 1 year, and a subset of participants also had a third CT at about 3 years after baseline. Subjects were followed annually for about 3.5 years, and investigators obtained medical records and pathology reports, if applicable.

The final cohort was 3538 people (49% women, 7% minority). About 60% were still smoking at enrollment. One-fourth of them had a history of bronchitis, emphysema, or bronchitis, and two-thirds had symptoms of cough, sputum, or wheezing. Pulmonary function testing demonstrated mild airflow obstruction in 13.6%, moderate obstruction in 22.8%, and severe obstruction in 6.4% of the participants. Nearly half (42%) of the cohort had CT evidence for emphysema; CT scan revealed trace emphysema in 18.8%, mild emphysema in 14.6%, and moderate-to-severe emphysema in 9.1%. Risk factors for emphysema were age, years and numbers of

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cigarettes smoked, a prior diagnosis of emphysema, bronchitis, or asthma, and pulmonary symptoms. Airflow obstruction on spirometry correlated well with evidence of emphysema on CT.

Over an average follow-up period of 3.5 years, there were 99 incident lung cancers (86 non-small cell and 13 small cell). Risk factors for lung cancer were age, years and numbers of cigarettes smoked, and respiratory symptoms. Gender, race, current smoking status, or history of emphysema, bronchitis, or asthma did not predict incident lung cancer. Both airflow obstruction by spirometry and emphysema by CT were strong determinants of development of lung cancer; lung cancer was diagnosed most frequently among people who had both emphysema and moderate-to-severe airflow obstruction.

■ COMMENTARY

We have known for a long time that airflow obstruction (by spirometry) predicts an increased risk of lung cancer.[1-4] What is new about this study is that emphysema detected by CT scanning also strongly predicted an increased risk of lung cancer, and the highest frequency of lung cancer was observed in subjects with both emphysema and moderate-to-severe airflow obstruction. This is important new information, since people who are at risk for lung cancer are increasingly seeking low-dose screening CT scans in attempts to pick up cancer "early." The authors note: "Using low-dose helical CT scanning to screen for lung cancer and emphysema at the same time can be efficient because the screened population is at risk for both diseases."

The accompanying editorial notes that the visual semiquantitative assessment score of emphysema severity used in this study likely provides a more accurate and reproducible assessment of emphysema.[5] The editorial also notes that, while there were no sex differences in lung cancer risk in this study, there appears to be a sex-based differential phenotype of COPD, with men showing more CT evidence of emphysema at all spirometric stages than women do.

This paper also amplifies what we know about symptoms, clinical diagnoses, and cancer risk. In this study, having been told by a physician that you had emphysema, bronchitis, or asthma did not predict increased lung cancer risk, but respiratory symptom of cough, sputum, or wheeze did.

What practical use can we make of these new data? If one of your high-risk patients plans to have a "screening CT" for lung cancer (not currently recommended or covered by insurance, but frequently done nevertheless), suggest that a semiquantitative emphysema score also be included. You can also take the "low-tech" approach, and point out that symptoms alone (cough, sputum, or wheeze) predict an increased cancer risk. ■

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Role of Long-term Secondary Prevention after MI

ABSTRACT & COMMENTARY

By Rahul Gupta, MD, MPH, FACP

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Questions & Comments
Please call **Paula Cousins**, Senior Managing Editor, at (404) 262-5468

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Dr. Gupta reports no financial relationship to this field of study.

Synopsis: *This first known study of the effects of a 3-year cardiac rehabilitation program following a myocardial infarction (MI) included a continuous reinforced educational and behavioral intervention and proved effective in decreasing the risk of several important secondary cardiovascular (CV) outcomes.*

Source: Giannuzzi P, et al. Global secondary prevention strategies to limit event recurrence after myocardial infarction. *Arch Intern Med* 2008;168:2194-2204.

THE MOST SIGNIFICANT QUESTION THAT ARISES IMMEDIately after survival from a myocardial infarction (MI) is the same whether you are the treating physician or the suffering patient: What can I do to prevent the next one? Current guidelines by the American Heart Association/American College of Cardiology recommend a host of actions in risk factor modification for secondary prevention in patients with coronary and other atherosclerotic disease.[1] These include stopping smoking (complete cessation), blood pressure control (< 130/80 mm Hg if diabetes or chronic kidney disease, < 140/90 mm Hg otherwise), lipid management (LDL < 100 mg/dL, but most likely should be < 70 mg/dL), physical activity (30 min, 7 days/wk), weight management (BMI 18.5-24.9 kg/m^2), diabetes management (A1c < 7%), aspirin, ACE inhibitors, beta-blockers, and influenza vaccination. Current cardiac rehabilitation programs (CRPs) contain these specific core components that aim to optimize cardiovascular risk reduction, foster healthy behaviors and adherence to these behaviors, reduce disability, and promote an active lifestyle for patients with cardiovascular (CV) disease.[2] This includes a baseline patient assessment, nutritional counseling, risk factor management, psychosocial interventions, and physical activity counseling with exercise training. An important consideration of these recommendations is the understanding that successful risk factor modification and the maintenance of a physically active lifestyle is a lifelong process. However, most of the existing CRPs are short-term and therefore remain short-sighted in achieving their benefits. The short-term use of these benefits would not be expected to lead to long-term benefits.[3] Medications like aspirin, statins, beta-blockers, and ACE inhibitors used for secondary prevention are often stopped shortly after hospital discharge.[4,5]

The current study (The Global Secondary Prevention Strategies to Limit Event Recurrence After Myocardial Infarction or GOSPEL) was undertaken to assess the effect on quality of care and prognosis of a long-term, relatively intensive rehabilitation strategy after MI. Giannuzzi et al conducted a multicenter, prospective, randomized, open-label, blinded controlled trial at 78 Italian cardiac rehabilitation centers. Following a standard post-MI cardiac rehabilitation program, a total of 3241 patients were randomized in a 1:1 fashion to either an intensive, 3-year-long, multifactorial intervention and behavioral program (intervention group; n = 1620) or usual care (control group; n = 1621). The combination of CV mortality, nonfatal MI, nonfatal stroke, and hospitalization for angina pectoris, heart failure, or urgent revascularization procedure was the primary endpoint. Other endpoints were major CV events, major cardiac and cerebrovascular events, lifestyle habits, and drug prescriptions.

Endpoint events occurred in 556 patients (17.2%). Compared with usual care, the intensive intervention did not decrease the primary endpoint significantly (16.1% vs 18.2%; hazard ratio [HR], 0.88; 95% confidence interval [CI], 0.74-1.04). However, the intensive intervention decreased several secondary endpoints: CV mortality plus nonfatal MI and stroke (3.2% vs 4.8%; HR, 0.67; 95% CI, 0.47-0.95), cardiac death plus nonfatal myocardial infarction (2.5% vs 4.0%; HR, 0.64; 95% CI, 0.43-0.94), and nonfatal MI (1.4% vs 2.7%; HR, 0.52; 95% CI, 0.31-0.86). In addition, a marked improvement in lifestyle habits and in prescription of drugs for secondary prevention was seen in the intervention group. For example, the overall Mediterranean-like dietary habits increased from 26.1% at baseline to 59.6% at 6 months with the intervention group doing better; the 6-month score for physical activity was 6.1% ($P < 0.01$) higher in the intervention group; and the 6-month score for self/stress management adjusted for baseline was 3.8% lower (or better results) in the intervention group ($P < 0.001$). All results were maintained throughout the study.

■ COMMENTARY

In an era where we allow researchers to develop a new paradigm for defining terms like prevention, it would be an immense disappointment if we prematurely discarded the current meaning of prevention. I will illustrate my point as follows: Results of two significant prevention trials were reported in the same week of November 2008. The GOSPEL study as discussed above was the largest one of its kind evaluating the role of a 3-year multifactorial comprehensive cardiac rehabilitation

program in secondary prevention of coronary heart disease. Results from a separate primary prevention study (Justification for the Use of Statins in Primary Prevention: An Intervention Trial Evaluating Rosuvastatin or JUPITER) was also reported in the same time period.[6] The JUPITER trial enrolled healthy subjects who did not have high cholesterol levels or coronary heart disease but elevated high-sensitivity C-reactive protein. This trial of nearly 18,000 patients was stopped early, with only 1.9 of its proposed 4 years of follow-up concluded, when the data and safety monitoring board noted a significant reduction in the primary endpoint among participants assigned to receive rosuvastatin. This trial obviously received a lot of media coverage, largely because the authors were able to demonstrate that a pill could prevent a heart attack or stroke, even if you were healthy with good cholesterol. This is despite the traditional definition of primary prevention, which involves the use of health promotion activities including immunization to prevent specific diseases. Wouldn't a prudent lifestyle modification program achieve similar results without the unknown long-term risks of rosuvastatin? In this regard, even though the GOSPEL trial is a secondary prevention study, it highlights the point that we must continue to put emphasis on a comprehensive lifestyle modification program for our post-MI patients, not just in the short term but as a long-term strategy. ■

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The Significance of Fatigue in the Elderly

ABSTRACT & COMMENTARY

By Mary Elina Ferris, MD

Clinical Associate Professor, University of Southern California

Dr. Ferris reports no financial relationship to this field of study.

Synopsis: *Mortality rates for community-living older adults followed over 10 years showed higher risk for those who felt tired most of the time.*

Source: Hardy SE, Studenski SA. Fatigue predicts mortality in older adults. *J Am Geriatr Soc* 2008;56:1910-1914.

PERSISTENT UNEXPLAINED TIREDNESS (ALSO CALLED fatigue) can be a perplexing complaint in the elderly. This study followed 492 patients aged 65 and older from a Medicare HMO and a Veterans' clinic. At the baseline visit they were asked, "Do you feel tired most of the time?" The 43% who answered "yes" were more likely to be female and white, have worse functional and physical status, and have more concurrent conditions.

Over 10 years of follow-up, almost half the participants died, and these were more likely to be those who reported tiredness at their baseline visit. Mortality rates were 59% for those with fatigue and 38% for those without. The difference in survival persisted even after adjustment for chronic conditions, depressive symptoms, and physical performance. The association did not change with age, sex, or race. One subgroup of those who reported no functional limitations did not demonstrate any association between fatigue and survival.

■ COMMENTARY

The authors speculate that fatigue may be an independent predictor of what they call "subclinical disease, inflammation, or increased work at maintaining homeostasis." The same authors have also found a relationship between a 1-year improvement in gait speed and 8-year survival.[1] They suggest that these simple observations

could be used as a "vital sign" in the care of the elderly. However, the use of fatigue as a reliable marker is hard to imagine, since the perception of fatigue and how it is described may vary widely among patients of different backgrounds. Fatigue severity was not assessed, nor was the influence of different therapies that may have been utilized over the course of the study. A small group of patients who had no functional impairments was free of the association between fatigue and decreased survival.

While I can criticize this study for the many other variables that were not measured, it does confirm a frequent clinical impression that fatigue is associated with poorer outcomes for our patients. Could it be that the gradual process of reaching the end of life includes a diminution in energy and strength? We may not be able to characterize this observation scientifically, but it remains one of the intuitive measures that physicians may use to assess our patients' chances of ongoing survival. ■

Reference

1. Hardy SE, et al. Improvement in usual gait speed predicts better survival in older adults. *J Am Geriatr Soc* 2007;55:1727-1734.

Beating the Blues

ABSTRACT & COMMENTARY

By Eileen C. West, MD

Director of Internal Medicine Women's Health, Clinical Associate Professor of Internal Medicine, University of Oklahoma College of Medicine, Oklahoma City

Dr. West reports no financial relationship to this field of study.

Synopsis: *The American College of Physicians has just published a new clinical practice guideline on the use of second-generation antidepressants in treating depressive disorders.*

Source: Qaseem A, et al. Using second-generation antidepressants to treat depressive disorders: A clinical practice guideline from the American College of Physicians. *Ann Intern Med* 2008;149:725-733.

EVER WONDER WHICH DRUG TO REACH FOR FIRST IN treating a clearly depressed clinic patient? The choices abound and selection can seem ambiguous for many providers. It turns out there is good reason for it. The Clinical Efficacy Assessment Subcommittee of the American College of Physicians addresses the topic of antidepressant medications in the November 18 issue of *Annals of Internal Medicine*. They review 203 studies from 1980 to 2007 and reach a few helpful conclusions.

Their summary focuses on second-generation antidepressants (SSRIs, SNRIs, and SSNRIs), as these are more commonly used and have been shown to have similar efficacy and lower toxicity in overdose than the first-generation tricyclic antidepressants and monoamine oxidase inhibitors. Psychotherapy, cognitive behavioral therapy, and other nonpharmacologic treatments are not addressed.

■ COMMENTARY

Depressive disorders affect 16% of adults in the United States during their lifetime, with an economic burden estimated at $83.1 billion. Depressive disorders include major depressive disorder (MDD), dysthymia, and subsyndromal depression including minor depression. Typically the course of depression is characterized by three phases: acute (6-12 weeks), continuation (4-9 months), and maintenance (1+ year). Relapse is the term used for the return of depressive symptoms during the acute or continuation phases. Recurrence refers to a return of depressive symptoms during the maintenance phase, which is considered a new, distinct episode.

So, which medications work best to treat acute depression, which most effectively treat symptom clusters such as anxiety, insomnia, and pain, and which had the worst side effects? The answers may be surprising. Of the twelve drugs reviewed, all work with about the same effectiveness, and most have similar side effects! (Shhh! Don't let the drug companies hear us say that.) OK, there are a few minor differences. Mirtazapine works faster than an SSRI in the acute phase. Paroxetine is associated with increased rates of sexual dysfunction while bupropion is associated with a lower rate of sexual adverse events. SSRIs result in an increased risk for nonfatal suicide attempts compared to placebo. There is weak evidence suggesting bupropion may be associated with increased risk for seizures and venlafaxine may be linked to increased risk for cardiovascular events. Nefazodone may be associated with increased risk for hepatotoxicity (again, weak evidence).

Unfortunately, 38% of the patients did not achieve a treatment response during 6-12 weeks of treatment and only 46% of patients achieved full remission with any of the second-generation antidepressants. The committee sounded an urgent plea for further research.

Ultimately four recommendations on treatment of

depression with a second-generation antidepressant were made:

1. When choosing pharmacologic therapy to treat patients with acute major depression, select second-generation antidepressants on the basis of adverse effect profiles, cost, and patient preferences.
2. Assess patient status, response to drug therapy, and adverse effects of antidepressant therapy on a regular basis beginning within 1-2 weeks of starting medication.
3. Modify treatment if the patient does not have an adequate response within 6-8 weeks of initiation of therapy for major depressive disorder.
4. Continue treatment for 4-9 months after a satisfactory response in patients with a first episode of major depressive disorder. For patients who have had 2 or more episodes of depression, a longer duration of therapy may be beneficial. ■

Pharmacology Update

Silodosin Capsules (Rapaflo™)

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

Dr. Elliot is Chair, Formulary Committee, Northern California Kaiser Permanente; and Assistant Clinical Professor of Medicine, University of California, San Francisco. Dr. Chan is Pharmacy Quality and Outcomes Manager, Kaiser Permanente, Oakland, CA.

Drs. Chan and Elliott report no financial relationship to this field of study.

THE FDA HAS APPROVED A NEW, HIGHLY SELECTIVE alpha-1 adrenergic receptor antagonist for the treatment of benign prostatic hyperplasia (BPH). Silodosin was developed by Kissei Pharmaceuticals Co., Ltd., in Japan, and will be marketed by Watson Pharmaceuticals Inc, as Rapaflo™.

Indication

Silodosin is indicated for the treatment of symptoms due to BPH.[1]

Dosage

The recommended starting dose is 8 mg once daily. The dose should be reduced to 4 mg in patients with moderate renal impairment. The drug is not recommended for patients with severe renal or hepatic impairment.[1]

Potential Advantages

Concomitant use of silodosin and sildenofil (Viagra®) or tadalafil (Cialis®) in healthy subjects did not cause any symptomatic changes in blood pressure, heart rate, or orthostasis.[2]

Potential Disadvantages

Ejaculatory dysfunction has been associated with silodosin and appears to be more common than with tamsulosin.[3] Intraoperative Floppy Iris Syndrome is a complication associated with cataract surgery in patients on alpha-1 adrenergic receptor blockers.[1]

Comments

Silodosin is a highly selective alpha-1 adrenergic receptor antagonist. It has a higher selectivity for the lower urinary tract than prazosin or tamsulosin.[4] Its safety and efficacy were shown in pooled data from two phase 3, 12-week, randomized, double-blind, placebo-controlled studies. Subjects were randomized to silodosin 8 mg/daily (n = 466) or placebo (n = 457). The mean age was 64.6 years, 89.3% were Caucasian, and 3.9% were African American. Mean peak urine flow rates (Q_{max}) were 8.7-8.9 mL/sec, and the mean International Prostate Symptom Score (IPSS) was 21.3. The primary endpoint was the reduction of IPSS and secondary endpoint was improved peak urine flow rates. At 12 weeks, mean reduction in IPSS was -6.4 for silodosin vs -3.5 for placebo ($P < 0.0001$). Mean improvements in Q_{max} were 2.6 mL/sec and 1.5 mL/sec, respectively ($P = 0.0007$). In addition, the irritative and obstructive subscores of IPSS showed statistically significant reduction relative to placebo. Silodosin (4 mg twice daily) was

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non-inferior to tamsulosin (0.2 mg daily) and actually showed a greater reduction in IPSS at week 2 in Japanese men.[3,4] In an open-label 52-week study, silodosin showed long-lasting benefit as demonstrated by continual reduction in IPSS for 52 weeks.[4] Retrograde ejaculation was the most common adverse event. In a comparative trial with tamsulosin, abnormal ejaculation occurred in 22.3% of silodosin patients compared to 1.6% for tamsulosin.[3] However, the dose of tamsulosin used in the study was lower than the dose typically used in the United States (0.4 mg). Other adverse events include dizziness, light-headedness, diarrhea, orthostatic hypotension, headache, nasopharyngitis, and nasal congestion.[1]

Clinical Implications

Silodosin is the latest addition to the alpha adrenergic antagonist market for the treatment of symptoms due to BPH. Commonly used agents include terazosin, tamsulosin, doxazosin, and alfuzosin. No one agent has shown superiority in terms of improving symptoms or urinary flow, but they can differ in terms of adverse events profile.[5,6] The greater alpha-1a selectivity of silodosin is associated with less blood pressure effect but a higher incidence of abnormal ejaculation including retrograde ejaculation. Current therapies for moderate-to-severe symptoms of BPH include alpha adrenergic antagonists and 5-alpha reductase inhibitors (e.g., finasteride, dutasteride). Recent evidence suggests that a combination of both is more effective than either agent alone in patients with larger prostates and higher PSA levels.[6-8] ■

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CME Objectives

The objectives of *Internal Medicine Alert* are:

- to describe new findings in differential diagnosis and treatment of various diseases;
- to describe controversies, advantages, and disadvantages of those advances;
- to describe cost-effective treatment regimens;
- to describe the pros and cons of new screening procedures.

CME Questions

52. Which of the following predicted increased incidence of lung cancer in the Pittsburgh Lung Screening Study?

a. African-American ethnicity
b. Age younger than 50 years
c. Emphysema on CT scan
d. Self-reported emphysema

53. After a myocardial infarction, a comprehensive cardiac rehabilitation program may continue to provide benefits in secondary prevention for how long after the primary event?

a. 6 months
b. 1 year
c. 2 years
d. 3 years

54. Recent research has linked which of the following with increased years of survival in older adults?

a. Increasing gait speed
b. Lack of depression
c. Lack of fatigue
d. Both a and c
e. None of the above

Answers: 52. c, 53. d, 54. d.

By Louis Kuritzky, MD, Clinical Assistant Professor, University of Florida, Gainesville
Dr. Kuritzky is a consultant for GlaxoSmithKline and is on the speaker's bureau of GlaxoSmithKline, 3M, Wyeth-Ayerst, Pfizer, Novartis, Bristol-Myers Squibb, AstraZeneca, Jones Pharma, and Boehringer Ingelheim.

Uric Acid and CV Risk: Not Ready Yet

Source: Feig DI, et al. Uric acid and cardiovascular risk. *N Engl J Med* 2008;359:1811-1821.

The relationship between uric acid (URA) and cardiovascular (CV) risk has been the subject of controversy for decades. Even if one were to fully accept that the various associations between URA and unfavorable CV outcomes have a causal relationship, the hurdle of prospectively proving that reduction of URA will improve outcomes has yet to be addressed.

URA elevation has been identified as a harbinger of hypertension, as well as obesity, diabetes, and kidney disease, but that may only be part of the story. For instance, that renal disease develops disproportionately in gouty patients is certainly true, but so does hypertension, which is more often the cause of renal disease than uric acid stones or urate nephropathy. The Framingham Heart Study suggests that the relationship of URA and CV risk is not independent of hypertension, hence, it does not qualify as an independent risk factor.

URA may play an etiologic role in development of metabolic syndrome. Even though insulin resistance is regarded by some as a *sine qua non* of metabolic syndrome, URA often precedes the insulin resistance; indeed, animal studies suggest that decreasing URA forestalls metabolic syndrome.

URA is also associated with vascular disease in the carotids and peripheral circulation. Some even attribute favorable CV effects seen in the LIFE study (utilizing losartan) to its favorable effects on lowering uric acid (losartan is the only antihypertensive agent known to lower uric acid).

The pathophysiologic underpinnings linking URA to CV misadventure have some plausibility. All-in-all, the relationship between URA and CV risk is tantalizingly close to compelling. However, even though we have expanded the old aphorism that "close only counts in horse shoes and hand grenades" to include archery, darts, lawn bowling, etc., close still doesn't count in science. ■

CPAP for OSA in Metabolic Syndrome

Source: Dorkova Z, et al. Effects of continuous positive airway pressure on cardiovascular risk profile in patients with severe obstructive sleep apnea and metabolic syndrome. *Chest* 2008;134: 686-692.

The association of obstructive sleep apnea (OSA) with adverse cardiovascular (CV) outcomes is strong. Encouraging data from persons with OSA who have been treated with continuous positive airway pressure (CPAP) have demonstrated that effective CPAP treatment reduces blood pressure. Whether CPAP might have favorable effects on other CV risk factors such as glucose, lipids, and markers of inflammation is less clear. Individuals with metabolic syndrome, who are at increased risk for CV events, provide an appropriate population to study the impact of CPAP treatment for OSA upon CV risk factors.

Dorkova et al studied 32 metabolic syndrome patients with OSA. At baseline and after 8 weeks of CPAP for OSA, risk factors associated with CVD were measured and compared: BP, cholesterol, triglycerides, HDL, fibrinogen, CRP, insulin resistance, and others. Subjects who used their CPAP for at least 4 hours nightly were considered compliant.

Compliant CPAP users enjoyed reduced BP, cholesterol, and insulin resistance. Non-compliant CPAP subjects (i.e., < 4 hrs/night) did not demonstrate these favorable changes. CPAP has been shown, in the high-risk group of patients with metabolic syndrome, to improve the CV risk factor profile. ■

Documentation of Coronary Ischemia Prior to PCI

Source: Lin GA, et al. Frequency of stress testing to document ischemia prior to elective percutaneous coronary intervention. *JAMA* 2008;300: 1765-1773.

For the sake of discussion, let's momentarily agree that percutaneous coronary intervention (PCI) is an appropriate step for persons suffering symptomatic coronary artery disease. The reason to establish this premise is that whether PCI actually offers any meaningful advantage over simple medical management is a matter of great debate. Indeed, the majority of PCIs in the United States are performed for patients with stable coronary disease, despite lack of evidence that outcomes with PCI in this setting are superior to intensive risk factor modification combined with medical therapy. Guidelines for PCI in stable CAD patients suggest that documentation of ischemia (most commonly by treadmill testing) should be obtained prior to PCI.

This study looked at Medicare beneficiaries undergoing elective PCI to see how often PCI had been preceded by stress testing. Of 23,887 Medicare recipients who underwent PCI in 2004, slightly less than half had any record of stress testing within the 90 days immediately preceding their PCI. The authors comment that in the absence of corroboration of ischemia, PCI may have been performed in patients who may not have benefited. ■