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Creatine Supplementation Increases Renal Disease Progression

ABSTRACT & COMMENTARY

Synopsis: *Creatine supplements should be used with caution and supervision, if at all, in individuals with renal insufficiency.*

Source: Edmunds JW, et al. *Am J Kidney Dis.* 2001;37(1):73-78.

Oral creatine supplementation has seen a huge surge in popularity since it was shown to be performance enhancing during short, high-intensity exercise. Other beneficial effects include favorable effects on lipid profiles, and neurodegenerative and musculosketelol disorders. Most of the total-body creatine is stored in muscle.

Controversy exists as to whether creatine supplementation has a detrimental effect on renal function. No controlled long-term studies are available, and most studies are small with a short duration and are comprised of only healthy subjects. Several recent reports raised serious concerns that creatine supplementation could cause serious kidney damage.[1,2]

To study the potential long-term effects of creatine supplementation on renal function, Edmunds and colleagues studied a rat model of renal disease. The Han: Sprague-Dawley (SPRD)-cy rat resembles human autosomal dominant polycystic kidney disease and was chosen for its short lifespan. Four-week-old male and female rats were randomized to receive a standard diet or a creatine-supplemented diet for 6 weeks. To mimic human consumption, the rats received 2.0 g/kg creatine for 1 week followed by one-fifth the dose for the next 5 weeks. Renal function was assessed with serum urea, creatinine, and creatine clearance measurements. After 6 weeks, cystic kidney disease progression was assessed by measurement of kidney size, cyst fluid content, and cyst scores.

Creatine supplementation was associated with detrimental effects after 6 weeks in this rat model. Total kidney weight increased 10%, and there was a 25% increase in cyst scores (P = .017), and a 2.1% increase in renal fluid content (P = .006). Serum urea worsened by 16% (P = .023), and creatinine clearance was reduced by 23% (P = .016) with creatine supplementation.

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INSIDE

COMMENT BY KAMALJIT SETHI, MD, FACP

We are consuming creatine like never before—more than 2.5 million kg were consumed last year in the United States by athletes, teenagers, and others.[3] The primary source of dietary creatine is meat, and 250 g of meat yields 1 g of creatine. Creatine supplementation increases skeletal muscle stores by 20% and increases short-term performance more in female athletes than in men, hence the motivation for its use as an ergogenic agent.

There are a series of questions:

1. Does creatine supplementation have an adverse effect on renal function in normal subjects? Probably not, although only short-term studies have been done.
2. Is there an adverse renal effect if high-protein intake and creatine supplements are combined in those with normal renal function? The answer is unknown and unstudied.
3. Are there deleterious effects of oral creatine supplements in those with a propensity for renal failure? If what is true of mice is true of men, then indeed deleterious renal effects may occur. Creatine supplements should be used with caution and supervision, if at all, in individuals with renal insufficiency. ❖

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Does Pravastatin Prevent Stroke?

ABSTRACT & COMMENTARY

Synopsis: *The pooled results demonstrate a statistically significant reduction in total stroke and nonfatal stroke attributable to pravastatin administration.*

Source: Byington RP, et al. *Circulation*. 2001;103:387-392.

Strokes, which are the second leading cause of death in the Americas and Europe, occur in 600,000 patients each year and are the leading cause of disability and increased health care costs in the United States resulting in 160,000 deaths.[1] Whereas numerous studies have demonstrated that the risk of coronary heart disease events is reduced by lipid-lowering therapy,[2-6] the effects of lipid-lowering on stroke events have not been well established even though studies with older lipid-lowering agents have suggested that modest reduction in cholesterol did not reduce stroke.[7,8] More recently, the proven efficacy of the HMG-CoA reductase inhibitors (or statin drugs) on the ravages of coronary artery disease have heightened the expectation that these agents might also have a beneficial effect on the prevention of stroke.

The Prospective Pravastatin Pooling (PPP) Project was initiated in 1992 before 3 large, placebo-controlled, randomized trials, which included 19,768 patients with 102,559 person-years of follow-up, had been completed. The effect of pravastatin given in a dosage of 40 mg/d on stroke events was investigated, and a prospectively defined pooled analysis of these 3 large trials[4-6,9-11] was performed.[12] When the 13,173 patients from the 2 secondary prevention trials[7,8] were combined, there was a 22% reduction in total strokes and a 25% reduction in nonfatal stroke. The beneficial effect of pravastatin on total stroke incidence was observed across a wide range of

patient characteristics. Pravastatin was associated with a 23% reduction in nonhemorrhagic stroke in the secondary prevention group. The West of Scotland Coronary Prevention Study, which was a primary prevention trial in hypercholesterolemic men, exhibited a similar, although somewhat smaller, reduction in total stroke incidence.

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

The data presented by Byington and colleagues in the Prospective Pravastatin Pooling Project represent a carefully performed systematic overview of 3 extremely large lipid-lowering trials. The pooled results demonstrate a statistically significant reduction in total stroke and nonfatal stroke attributable to pravastatin administration. The analyses also clearly demonstrated that pravastatin was more effective than the older, nonstatin lipid-lowering therapies in reducing stroke rates. The consistent reduction in stroke rate across the trials and subgroups were particularly striking, and the drug appeared to have similar beneficial effects on patients taking aspirin and whether they were taking blood pressure-lowering medications.

It is important to recognize that stroke has many causes; therefore, in evaluating specific forms of drug therapy used for prevention, one must separate thrombotic from hemorrhagic strokes. Pravastatin appears to have strong beneficial effects in preventing atherothrombic strokes and has no effect on the prevention of hemorrhagic stroke. Since numerous papers suggest that plaque rupture is the primary pathogenic mechanism in acute thrombotic events in coronary and cerebral arteries, plaque stabilization appears to be an important process by which statins are effective in reducing the frequency of thrombotic events in these areas. Therefore, the nonlipid effects of statin drugs may be more important in the prevention of cerebrovascular disease than are the lipid-lowering actions of the drug. Also, statins have been shown to have an anti-inflammatory effect independent of their LDL-lowering actions by virtue of their ability to reduce inflammatory markers such as C-reactive protein, and this effect may result in improved endothelial function thereby decreasing the incidence of acute coronary events.

From a clinical point of view, the PPP Project results provide strong evidence supporting the view that patients with a prior history of myocardial infarction should be treated with statin drugs—not only to prevent recurrent myocardial infarctions but also possibly to reduce the incidence of stroke event rates. Since stroke is a devastating event in the lives of patients and their families, clinicians should be able to use the results of the PPP Project to generate greater patient acceptance of prolonged statin therapy. Although the principle use of these drugs will largely remain in the prevention of coronary artery heart disease, the evidence of stroke benefit with the use of statin drugs demonstrated by the PPP Project is an important contribution for both clinicians and patients. As we enter an era in which we may be using statin therapy to treat and prevent all cerebrovascular and peripheral arterial thrombotic events and, in fact, even for the treatment of occult atherosclerosis in all arterial systems, we thereby will be reducing the incidence of acute coronary and cerebrovascular events. ❖

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Does Ranitidine Increase the Incidence of Nosocomial Pneumonia?

ABSTRACT & COMMENTARY

Synopsis: *Ranitidine is ineffective in preventing gastrointestinal bleeding in ICU patients and may increase the risk of pneumonia.*

Source: Messori A, et al. *BMJ*. 2000;321:1-7.

Messori and colleagues in florence, italy, performed a series of meta-analyses of available

randomized controlled trials of the use of ranitidine and sucralfate for the prevention of stress ulcer bleeding in ICU patients. They searched Medline and other databases for English-language studies with placebo controls.

Five separate meta-analyses were performed. The first of these examined the effectiveness of ranitidine vs. placebo in five trials including a total of 398 patients, and found that ranitidine had the same effectiveness as placebo (odds ratio of bleeding, 0.72, 95% CI 0.30-1.70, $P = .46$). The planned second meta-analysis of sucralfate vs. placebo could not be performed, as only one clinical trial met Messori et al's entry criteria. Three studies comprised 311 patients in the third meta-analysis of ranitidine vs. placebo with respect to nosocomial pneumonia. In this and the fourth meta-analysis, of sucralfate vs. placebo in two studies totalling 226 patients, no difference in the incidence of pneumonia with respect to placebo vs. either drug could be found. However, in the fifth meta-analysis, directly comparing ranitidine to sucralfate in a total of 1825 patients in eight studies, there was a significantly higher incidence of nosocomial pneumonia in patients receiving ranitidine (odds ratio, 1.35; 95% CI, 1.07-1.70; $P = .012$).

The mean quality score in the four meta-analyses that could be completed ranged from 5.6-6.6 on a 10-point scale. Messori et al conclude that ranitidine is ineffective in preventing gastrointestinal bleeding in ICU patients and may increase the risk of pneumonia. Because of small numbers of published studies and total reported patients, Messori et al were unable to make any definitive statements about the clinical effects of sucralfate. They recommend that current recommendations on prophylaxis of stress ulcers be revised.

COMMENT BY DAVID J. PIERSON, MD, FACP, FCCP

Most published studies of drugs for prophylaxis against stress ulcer bleeding in the ICU compare one supposedly active agent with another. According to Messori et al, this is the first ever meta-analysis of the effects of ranitidine vs. placebo on the incidence of gastrointestinal bleeding in ICU patients. A previous meta-analysis on H2 blockers and gastrointestinal bleeding[1] included five trials using cimetidine, the use of which in the ICU has now largely been abandoned, which generally favored the therapy, plus three trials with negative results using ranitidine. As Messori et al point out, there is only a single placebo-controlled, randomized clinical trial using sucralfate;[2] they believe that no conclusions as to the efficacy of that drug can be made from that study.

Both ranitidine and sucralfate are widely used to prevent gastrointestinal bleeding in ICU patients. According to the *British Medical Journal*, although a number of groups have recommended the prophylactic use of these agents, the Food and Drug Administration has not approved either drug for this purpose. This study casts considerable doubt on the clinical use of our current practice, and Messori et al emphasize that presently "there are insufficient data on effectiveness to conclude anything one way or another." Once again, further trials are needed. (*Dr. Pierson is Professor of Medicine, University of Washington; Medical Director, Respiratory Care, Harborview Medical Center, Seattle, Wash.*) ❖

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Inhaled Triamcinolone on the Decline in Pulmonary Function in COPD

ABSTRACT & COMMENTARY

Synopsis: *This study of 1116 patients with mild-to-moderate COPD found inhaled triamcinolone did not slow the rate of decline in FEV_1, nor did it have any effect on overall mortality. However, inhaled triamcinolone decreased airway reactivity, the symptom of dyspnea, and decreased physician visits and hospitalizations for respiratory complaints.*

Source: The Lung Health Study Research Group. *N Engl J Med*. 2000;343:1902-1909.

Chronic obstructive pulmonary disease (COPD) is a leading cause of morbidity and mortality in the United States and is characterized by a progressive decline in pulmonary function. Cessation of smoking has been found to slow down the rate of decline, but less than half of patients succeed in quitting. Airway inflammation is felt to be a contributing factor in the pathophysiology of COPD; however, unlike asthma, the data on the use of inhaled steroids has been inconsistent. The authors in this study hypothesized inhaled steroids would decrease the rate of decline of pulmonary function in patients with COPD.

These are the results of the Lung Health Study Research Group. They conducted a multicenter, randomized, placebo-controlled trial using inhaled triamcinolone. The patients had an FEV_1/FVC ratio of less than

0.70 and an FEV_1 30-90% of predicted. All were current smokers or quit no more than 2 years prior. Exclusion criteria included use of bronchodilators or oral or inhaled steroids within the previous year. The patients received either placebo or inhaled triamcinolone dosed at 6 inhalations twice daily for a total daily dose of 1200 mg/d. Spirometry was performed before and after 2 doses of isoproterenol. In addition, the subjects underwent methacholine bronchial provocation.

A total of 1116 patients were enrolled (559 treatment, 557 placebo) whose average age was 56 years old. The average FEV_1 prior to bronchodilator administration was 64.1% of predicted, and the mean duration of follow-up was 40 months. There was no significant difference in the decline of FEV_1 between the treatment group and placebo group either before bronchodilator use (decline of 48.6 mL/y for treatment and 49.9 mL/y for placebo) or after bronchodilator use (decline of 44.2 mL/y for treatment and 47 mL/y for placebo). However, the triamcinolone group had less reactivity to methacholine at 9 and 33 months (P = .02). Using the American Thoracic Society—Division of Lung Disease questionnaire, the authors found the only significant improvement in the treatment group was dyspnea (P = .02). Complaints of cough, phlegm production, and wheezing did not significantly improve in the treatment group. Overall, mortality did not improve in the treatment group (15/559 vs 19/557 for the placebo group; P = .49), nor was there an improvement in the quality-of-life score for the treatment group.

The frequency of visits to the emergency room was no different between the two groups for either respiratory or nonrespiratory conditions (P = .36 for respiratory conditions; P = .17 for nonrespiratory). However, there were less unscheduled physician visits and hospitalizations for respiratory conditions in the treatment group (outpatient visits per 100 person-year: 1.2 for treatment group, 2.1 for placebo; P = .03). In terms of side effects, the triamcinolone group had a significantly higher percentage decrease in bone mineral density at the lumbar spine and femoral neck when compared to placebo.

COMMENT BY DAVID OST, MD & CHARLES SCOTT HALL, MD

The use of inhaled corticosteroids in COPD remains questionable despite their widespread use for this condition.[1] A recent study from Denmark concluded there was no clinical benefit from inhaled budesonide in terms of either FEV_1 or symptoms.[2] A larger European study (the ISOLDE trial) found, although there was no effect on the rate of decline of FEV_1, inhaled steroids produced a small increase in FEV_1 after bronchodilators, less exacerbations, and slower decline in health status.[3] There is also evidence that inhaled steroids produce short-term improvements in FEV_1, but these effects are not sustained beyond 9 months.[4] The Lung Health Study Research Group found that triamcinolone did not affect the rate of decline of FEV_1 when compared to placebo; however, airway reactivity, dyspnea, and health care use did improve. The authors felt that although they did not exclude asthma, by excluding patients who regularly use bronchodilators or corticosteroids, they disqualified patients with clinically significant asthma. The use of triamcinolone in this study was also associated with a significant decrease in bone mineral density from baseline. This emphasizes that clinicians must weigh the risks and benefits of using inhaled steroids in their COPD population. In patients with refractory COPD who require frequent urgent visits, a trial of inhaled corticosteroids may be warranted. For those with more stable disease, fewer urgent visits, and higher risk of osteoporosis, inhaled corticosteroids may not be of benefit. (*Dr. Hall is a Fellow in Pulmonary and Critical Care Medicine, North Shore University Hospital-NYU School of Medicine, Manhasset, NY.*) ❖

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Pharmacology Update

Once-a-Week Fluoxetine, 'Prozac Weekly'

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

With the pending patent expiration of fluoxetine (Prozac), Eli Lilly is introducing another version of their blockbuster selective serotonin reuptake inhibitor (SSRI). Following the introduction of fluoxetine as Sarafem for premenstrual dysphoric disorder, Lilly has received FDA approval to market a once-weekly formulation of the drug for the treatment of depression. This takes advantage of the long elimination half-lives of the fluoxetine and its active metabolite, norfluoxetine, along with formulation of the drug in enteric-coated pellets. The product is marketed as Prozac Weekly.

Indications

Fluoxetine is indicated for maintaining an antidepressant response after initial acute treatment with fluoxetine 20 mg.[1,2]

Dosage

Fluoxetine 90 mg is dosed once-weekly. Weekly dosing should be initiated 7 days after the last daily dose of fluoxetine 20 mg. Fluoxetine Weekly is available as a 90-mg capsule.

Potential Advantages

Once-weekly dosing may improve compliance and may also enhance psychological well being.[1] Findings from a study by Lilly suggest that compliance with once-weekly dosing was higher compared to daily dosing, 85.9% vs. 79.4%.[3]

Potential Disadvantages

It is not clear if once-weekly administered fluoxetine provides the same degree of protection from relapse as once-daily fluoxetine.[2] Average trough plasma concentrations were 76% lower for fluoxetine and 47% lower for the active metabolite, norfluoxetine.[2] A weekly dosing regimen is an uncommon regimen, and some patients may have difficulty remembering their doses.

The effect of variation in GI transit time (eg, diarrhea) on the pharmacokinetics of fluoxetine-weekly is not known. Fluoxetine-weekly has not been studied for other conditions such as obsessive compulsive disorders, bulimia nervosa, or premenstrual dysphoric disorder.

Comments

The long elimination half-lives of fluoxetine (4-6 days) and its active metabolite, norfluoxetine (about 9 days) are conducive to less frequent dosing. The efficacy of a weekly regimen is supported primarily by a multicenter, placebo-controlled, double-blind, randomized study.[1]

Patients with DSM-IV criteria for nonpsychotic major depression and who had a modified 17-item Hamilton Rating Scale for Depression (HAM-D-17) of 18 or greater and a Clinical Global Impression-Severity of Illness scale (CGI-S) score of 4 or greater were treated with fluoxetine 20 mg for 13 weeks. A total of 501 responders (HAM-D-17 of 9 or less and CGI-S of 2 or less) were then randomized to fluoxetine 90 mg weekly, fluoxetine 20 mg, or placebo for 25 weeks. The primary end points were the percent of patients that relapsed and time to relapse. Relapse was defined as meeting criteria for major depression and an increase in CGI-S score of 2 or more relative to the rating before randomization. The percentages of relapse were 26% for fluoxetine 20 mg, 37% for fluoxetine-weekly, and 50% for placebo. While there was no statistical difference between fluoxetine regimens, daily dosing was numerically higher.

The times to relapse were 105 days, 109 days, and 86 days for fluoxetine 90 mg, 20 mg, and placebo, respectively. Nervousness and impaired concentration or thought process were the most frequent side effects for the 90-mg dose compared to the 20-mg dose, 13.7% vs. 6.3% and 8.9% vs. 1.6%, respectively. However, these side effects were similar to the frequencies seen with placebo. Prozac Weekly is priced about 5% less than Prozac daily.

Clinical Implications

Fluoxetine 90 mg weekly may be considered for maintenance therapy for patients who have responded to daily administered fluoxetine. The effectiveness may be less than that of a daily regimen in preventing relapse. If a satisfactory response is not achieved, returning to a daily regimen should be considered. ❖

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CME Questions

24. Creatine supplements in renal insufficiency may cause:

a. progressive azotemia.
b. improvement in anemia.
c. uncontrolled hypertension.

25. Which of the following statements about the use of ranitidine as prophylaxis against gastrointestinal bleeding in ICU patients is true?

a. It is approved by the FDA for this use.
b. Compared to placebo, it decreases the incidence of gastrointestinal bleeding.
c. It decreases the incidence of nosocomial pneumonia.
d. All of the above
e. None of the above

26. Fluoxetine Weekly:

a. is marketed as Prozac Weekly.
b. is available as a 90-mg capsule.
c. may be considered for maintenance therapy for patients who have responded to daily administered fluoxetine.
d. has not been studied for other conditions such as obsessive compulsive disorders, bulimia nervosa, or premenstrual dysphoric disorder.
e. All of the above

By Louis Kuritzky, MD

Estrogen Replacement Therapy and Ovarian Cancer Mortality in U.S. Women

The preponderance of epidemiologic data associates both endometrial and breast cancer with postmenopausal estrogen replacement therapy (ERT). The relationship of ovarian cancer to ERT is less clear. Recent case-control studies have suggested an increased risk with ERT, especially of long duration. Rodriguez and colleagues investigated the association between ERT and ovarian cancer mortality in a large population of female participants in the Cancer Prevention Study II (n = 676,526). Data were accrued over 14 years of observation, and include almost 1000 ovarian cancer deaths.

Even users of ERT had a slightly increased rate of ovarian cancer mortality (rate ratio = 1.23). This positive association increased in strength with duration of ERT use, so that persons using ERT for more than 10 years had an approximately 2-fold increase in relative risk. When coupled with the earlier case-control studies, this current report strengthens the concerns that ERT, especially of long duration, increases the risk of ovarian cancer mortality. Nonetheless, since total lifetime risk of ovarian cancer mortality is relatively small (< 2%), other potential favorable effects of ERT in other tissue compartments must be taken into account in the risk-benefit analysis. Additionally, the effect of concomitant progestational treatment has not been comprehensively addressed. ❖

Rodriguez C, et al. JAMA. 2001;285: 1460-1465.

Effects of A Low-Molecular Weight Heparin on Thrombus Regression and Recurrent Thromboembolism in Patients with Deep-Vein Thrombosis

Low molecular weight heparin (LMWH) has been found to be as useful as traditional unfractionated heparin (HEP) for early management of deep vein thrombosis (DVT) or pulmonary embolism (PE), but offers the advantage that monitoring activated partial thromboplastin. (APPT) is not necessary. Meta-analysis of earlier trials has suggested that LMWH affords a greater likelihood of thrombus regression, as well as a reduced rate of clinical recurrence. The current trial (n = 1137) was developed to explore the relative efficacy of LMWH and HEP in reference to thromboembolic recurrence and thrombus regression, assessed by venography.

LMWH, whether administered once or twice daily, demonstrated statistically significantly greater likelihood of thrombus regression (relative likelihood = 1.3 compared to HEP). LMWH was also significantly less likely to be associated with recurrent thromboembolic events.

Breddin and colleagues conclude that LMWH is more effective than unfractionated heparin in reduction of thrombus size, recurrent DVT, and new pulmonary embolism. ❖

Breddin HK, et al. N Engl J Med. 2001;344:626-631.

Effects of Vitamin E on Lipid Peroxidation in Healthy Persons

There is much debate about the perceived potential benefit of antioxidant therapies, including vitamin E, upon cardiovascular, oncologic, and neurologic end points. Oxidized LDL has been particularly associated with progressive atherosclerotic vascular damage. It has been postulated that vitamin E might reduce the ability of lipids to become oxidized, yet a model for quantification of such an oxidation protective effect has been lacking until recently. Meagher and colleagues used 2 newly developed quantitative markers of lipid peroxidation status: isoprostanes and 4-hydroxynonenenal (4-HNE). Subjects received doses of vitamin E ranging from 200-2000 IU daily for 8 weeks (n = 30).

Irrespective of dose used, there was no demonstrable effect of vitamin E supplementation on markers of lipid peroxidation. Recent large data sets, such as the HOPE trial, also failed to demonstrate a beneficial effect of vitamin E supplementation on cardiovascular end points. Meagher et al question the potential benefit of supplemental vitamin E consumption. ❖

Meagher EA, et al. JAMA. 2001;285: 1178-1182.

Marcapasos in an Older Woman

By Ken Grauer, MD

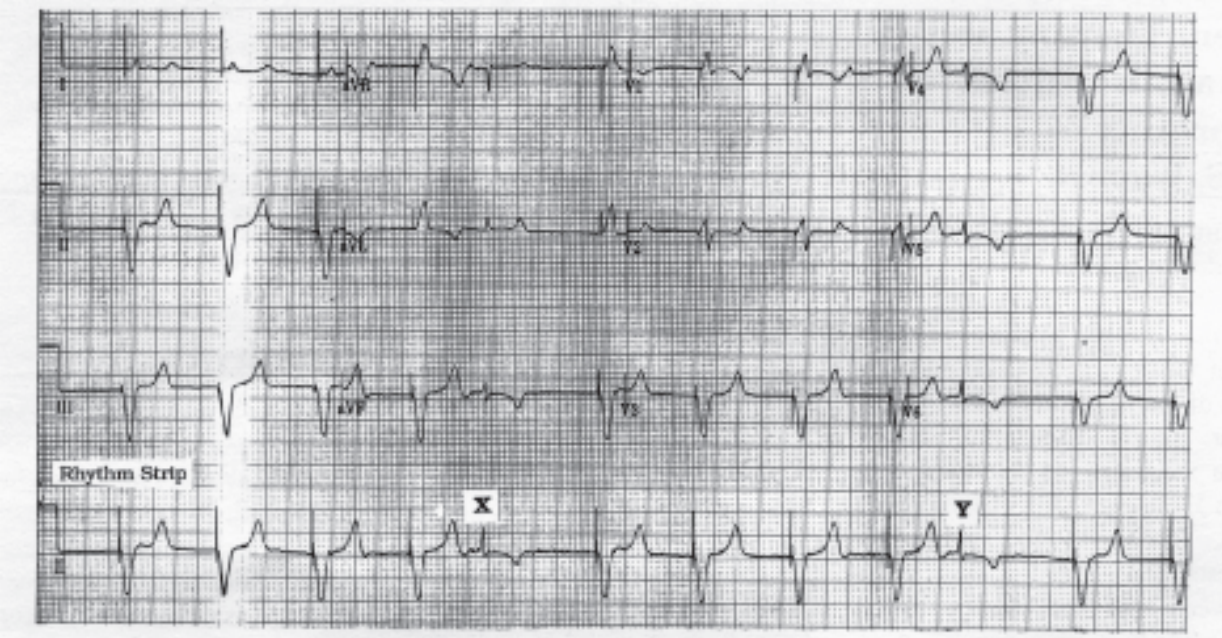

Figure. 12-lead postoperative ECG obtained from an 87-year-old woman with dyspnea.

Clinical Scenario: An 87-year-old Hispanic woman had a postoperative ECG performed as part of her evaluation for dyspnea that developed following abdominal surgery. There was no chest pain. A permanent cardiac pacemaker ("*marcapasos*") had been implanted years earlier for some type of rhythm disorder. Her 12-lead ECG is shown above. Does it appear that the pacemaker is functioning appropriately? Can you identify two *other* findings on this ECG that may be relevant to her clinical situation?

Interpretation: With exception of two spontaneous beats (labeled *X* and *Y*), the lead II rhythm strip at the bottom of the tracing shows regular pacer spikes at a rate of 70 beats/minute. Each pacer spike reliably *captures* the ventricles (evidence by the fact that each pacer spike is followed by both a QRS complex and T wave). Sensing function of the pacer is also appropriate, as judged by the finding of a constant R-to-spike interval (interval from spontaneous beat until the next pacer spike) that is appropriately the same as the inherent pacer rate. The key to detecting the findings of concern on this tracing lies with focusing on the two spontaneous beats (*X* and *Y*)—and in viewing QRS morphology of these spontaneous beats in each of the three simultaneously recorded leads. Despite not being certain of the underlying spontaneous rhythm (impossible to determine if a P wave precedes beats *X* and *Y* in lead II)—what *can* be said is that spontaneous QRS morphology in leads II and aVF, as well as in leads V_4, V_5, and V_6 show ST segment coving, slight ST segment depression, and moderately deep and symmetric T wave inversion. Serial Troponin I values were found to be positive for acute infarction.

The second ECG finding of concern on this tracing is more subtle, and relates to the appearance of the T wave of several paced complexes. Although ST segment/T wave morphology in paced complexes is rarely indicative of specific pathology, one is struck by the peaked appearance of many of the paced beats in this tracing. In further support of our suspicion, the T wave appearance in the spontaneous beat seen in lead aVL is peaked enough to merit checking serum electrolytes, which revealed moderate postoperative hyperkalemia. ❖

In Future Issues:

Alternative Medicine Use in Older Americans

Antibiotics Anonymous Redux*

By Stan Deresinski, MD, FACP, Editor, Infectious Disease Alert

Are *You* Antibiotic Dependent?

- ❑ Do you prescribe antibiotics to relieve tension?
- ❑ Do you prescribe antibiotics more than other physicians but are able to hide it?
- ❑ Do you sometimes feel guilty about the way you prescribe antibiotics?
- ❑ Do you have a strong urge to prescribe antibiotics at a particular time of day?
- ❑ Have you lost ambition since you began prescribing antibiotics in this way?
- ❑ Has another physician advised you to stop or cut down your prescribing?
- ❑ Are you harder to get along with when you are heavily prescribing?
- ❑ Have you ever tried to cut back?
- ❑ Do you have difficulty sleeping a full night?
- ❑ Have you ever been in trouble with the antibiotic police?
- ❑ Have you ever done anything while prescribing that you don't remember (have a blackout)?
- ❑ Have you ever promised yourself you would cut back on your prescribing and then broken that promise?
- ❑ Have you ever tried to convince people that you were not prescribing antibiotics when you were?
- ❑ Do you wish people would mind their own business about your antibiotic prescribing—that they stop telling you what to do?
- ❑ Have you ever switched from one kind of antibiotic to another in the hope that this would keep you from going over the edge?
- ❑ Have you had to have an eye-opener (ie, prescribed an antibiotic immediately upon awakening, in the last year)?
- ❑ Do you envy people who can prescribe antibiotics without getting into trouble?

For those who have answered yes to one or more of these questions, I have begun the development of a 12-step program. Unfortunately, I have only been able to develop half of a 12-step program.

- ❑ You must admit that you are powerless over your antibiotic prescribing.
- ❑ You must believe that a power (an antibiotic guru) greater than yourself can restore you to sanity.
- ❑ You must make a decision to turn your will and life over to the care of that power.
- ❑ You must make a searching and fearless moral inventory of yourself.
- ❑ You must admit to the power and to yourself the exact nature of your misprescribing.
- ❑ You must humbly ask the power to remove your antibiotic shortcomings.

* *Lockwood WR. Letter: Antibiotics anonymous. N Engl J Med 1974;290:465-466.*