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Nephrolithiasis and the Risk of Hypertension in Women

ABSTRACT & COMMENTARY

Synopsis: *While there is an increase in hypertension in women with nephrolithiasis, there was no increase in the risk of incident stones in those with pre-existing hypertension.*

Source: Madore F, et al. *Am J Kidney Dis* 1998;32:802-807.

Hypertension and kidney stones are both common important public health problems. About 20% of the U.S. population has hypertension, defined as a systolic blood pressure of 140 mmHg and/or diastolic blood pressure of 90 mmHg. The prevalence of hypertension increases up to 67% in those 65 years of age or older. About 12% of adults in the United States will form a kidney stone sometime in their life. A positive association between nephrolithiasis and hypertension has been observed in cross-sectional and prospective studies of men but the association has been controversial in women. Madore and associates conducted a prospective study to further evaluate the relationship between nephrolithiasis and hypertension in a cohort of 89,376 female registered nurses aged 34-59 years in 1980, who were enrolled in the Nurses Health Study. Data on nephrolithiasis, hypertension, dietary intake, and related factors was gathered by a biennial mail questionnaire. The reliability of the reported data was confirmed by a random sample comparison of self-reported data and physician office medical records.

On cross sectional analysis, 2.86% women reported a history of nephrolithiasis before 1980 and 13.3% reported a diagnosis of hypertension before 1980. The age- adjusted prevalence odds ratio for hypertension for women with a history of nephrolithiasis was 1.49, compared to those without a history of nephrolithiasis. On prospective analysis, 12,540 women reported a new diagnosis of hypertension between 1980 and 1992, and the age-adjusted relative risk for hypertension in women with a history of nephrolithiasis was 1.36 compared to those without a history of kidney stones. After adjustment for body mass index, and dietary intake of calcium, sodium, potassium, magnesium, caffeine and alcohol, the relative risk for hypertension in those with a history of hypertension was slightly reduced to 1.24.

Volume 2 • Number 12 • April 1999 • Pages 89-96

INSIDE

In contrast, there was no increase in the risk of incident stones in those with pre-existing hypertension, compared to those without hypertension.

COMMENT BY KAMALJIT SETHI, MD, FACP

Men with a history of nephrolithiasis are 29% more likely to develop hypertension compared to those with no history of kidney stone.[1] This prospective analysis reveals a similar risk in women: female subjects with a history of nephrolithiasis are 24% more likely (all variables included) to develop hypertension, compared with those who do not historically have kidney stones. Thus, both sexes are at an equally high risk of developing hypertension if there is a history of nephrolithiasis, even though renal stones occur predominantly in males (male:female ratio, 2-4:1).

What accounts for this association between renal stone disease and hypertension? Hypercalciuria is a frequent abnormality in stone formers and hypertensives have higher urinary calcium excretion compared to normotensives. Furthermore, dietary calcium intake is inversely related to stone risk, and high dietary calcium intake has been reported to be associated with a lower blood pressure. Certainly, it would seem that perturbations in calcium metabolism may be linked to both hypertension and nephrolithiasis, even though much work needs to be done to establish cause and effect.

Given the fact that nephrolithiasis increases the risk for incident hypertension, are there any opportunities for intervention? In those with recurrent stone disease or single stone formers with a strong family history of kidney stones, the following steps should be considered to prevent kidney stones and attenuate metabolic abnormalities:

1. Metabolic evaluation to include measurement of urinary saturation for calcium, oxalate and uric acid, and urinary inhibitor concentration of citrate. Medical therapy with thiazides or citrate may be necessary.
2. Encourage increased fluid intake so that urinary output is about 2 L/d. This ensures the lowest supersaturation for calcium oxalate and uric acid. Water is the fluid of choice.
3. Continue normal dietary calcium and potassium intake, while reducing excessive salt intake. With regard to calcium and potassium, natural dietary sources are recommended rather than supplements.

These are reasonable interventions that are easy and inexpensive. It may be that the risk for future hypertension can be reduced if we can prevent kidney stones. (*Dr. Sethi is Professor of Medicine, Georgetown University and Director, Georgetown Nephrology Section, DC General Hospital, Washington, DC.*) ❖

Reference

1. Madore F, et al. *Am J Hypertens* 1998;11:46-53.

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Role of Streptococcal Infection in Tourette Syndrome and Other Neuropsychiatric Disease

ABSTRACT & COMMENTARY

Synopsis: *Several investigators have presented converging lines of evidence for a role of streptococcal infection in triggering Tourette syndrome (TS), obsessive-compulsive disorder (OCD), and other neuropsychiatric disease.*

Sources: Singer HS, et al. *Neurology* 1998;50:1618-1624; Kurlan R, et al. *Neurology* 1998;50:1530-1534; Garvey MA, et al. *J Child Neurol* 1998;13:413-423; Hall MC, et al. *J Child Neurol* 1998;13:354-356; DiFazio MP, et al. *J Child Neurol* 1998;13:516-518; Sanberg PR, et al. *Lancet* 1998;352:705-706.

Since the original description of sydenham's chorea (SC) many decades ago, a growing appreciation has emerged of the complexity of neurologic disease

that may be related to streptococcal infection. The acronym PANDAS (pediatric autoimmune neuropsychiatric disorders associated with streptococcal infection) is a relatively new diagnostic construct to describe this spectrum of disease.

Several investigators have presented converging lines of evidence for a role of streptococcal infection in triggering Tourette syndrome (TS), obsessive-compulsive disorder (OCD) and other neuropsychiatric disease. Using standard ELISA and Western blot techniques, Singer and colleagues tested serum from 41 patients with TS (33 boys, 8 girls; mean age 11.3 years) and 39 controls (22 boys, 17 girls; mean age 12.1 years) for immune reactivity against human caudate, putamen, and globus pallidus. TS patients generated a significant increase in antineuronal antibodies, largely against putamen and caudate, compared to controls. Markers for streptococcal infection such as antistreptolysin O (ASO) titers were often equivocal.

DiFazio and colleagues reported three male patients, ages 5, 10, and 12 years, who developed a variety of myoclonic movement disorders associated with occult streptococcal infection. Only one patient was culture positive, but all had high ASO and anti-DNAase B titers. The myoclonus was effectively treated with erythromycin or penicillin, and recurred with subsequent reinfection with streptococcus. Similarly, Hall et al reported a case of an 11-year-old boy who, one week following group A beta-hemolytic streptococcal pharyngitis, developed paraparesis with a post-infectious encephalomyelitis. The patient responded well to antibiotics and corticosteroids.

Sanberg et al reported a retrospective case series of 13 Tourette's patients treated with the nicotinic antagonist mecamylamine 2.5-5 mg/d, alone or in combination with haloperidol or sertraline. Four were adults (one female; mean age 34 years) and nine were children (one female; mean age 14 years). Eleven of the 13 improved significantly in motor and vocal tics, as well as behavioral complaints such as irritability and aggression.

COMMENT BY BRIAN R. APATOFF, MD, PhD

Garvey et al and Kurlan provide excellent reviews of TS, tic disorders, and associated behavioral disturbance such as OCD that appear to arise from post-infectious autoimmune mechanisms. Thus, in addition to genetic factors that may determine a predisposition to TS, there appear to be important environmental triggers. An immune response generated against streptococcal antigens may crossreact with neuronal epitopes in the basal ganglia to cause neurological dysfunction. Trifiletti and colleagues at Cornell University Medical College have preliminarily identified a 83-kd brain protein by immunoblot analysis that seems to be recognized by antibodies in the serum of TS and OCD patients.[1] The clinical therapeutic significance of these findings is important. Physicians now recognize the importance of penicillin prophylaxis for group A beta-hemolytic streptococcal infections in avoiding neurologic as well as cardiac complications. Undiagnosed streptococcal infection should always be considered in young patients presenting with new TS, OCD, SC, or other unusual movement disorders. Rather than using harsh neuroleptics for symptomatic management of TS or OCD, investigators are using immunomodulatory therapies such as corticosteroids, IVIG, and plasmapheresis with suggestion of benefit in small uncontrolled trials. Larger numbers of patients in carefully conducted studies will be required to assess the efficacy of these immune approaches that carry significant risks. Until then, pharmacologic agents such as the nicotinic drug mecamylamine may provide better symptomatic control (*Dr. Apatoff is Assistant Professor of Neurology, New York Presbyterian Hospital-Cornell Campus.*) ❖

Reference

1. *Ann Neurol* 1998;44:561.

Calcium for PMS

ABSTRACT & COMMENTARY

Synopsis: *To evaluate the effect of calcium carbonate on the luteal and menstrual phases of the menstrual cycle in PMS, a prospective, double-blind, placebo-controlled, parallel group, multicenter randomized national clinical trial was conducted. This New York St. Luke's-Roosevelt Hospital Center study found that irritability, depression, food craving, aches and pains, and water retention all improved with calcium carbonate supplementation. With the exception of aches and pains, however, the placebo group improved nearly as much in each category.*

Source: Thys-Jacobs S, et al. *Am J Obstet Gynecol* 1988;179:444-452.

Previous reports have suggested that disturbances in calcium regulation may underlie the pathophysiologic characteristics of premenstrual syndrome (PMS) and that calcium supplementation may be an effective therapeutic approach. To evaluate the effect of calcium carbonate on the luteal and menstrual phases of the menstrual cycle in PMS, a prospective, double-blind,

placebo-controlled, parallel group, mulitcenter randomized national clinical trial was conducted.

The study screened 720 healthy premenopausal women (ages 18-45) for moderate to severe, cyclically recurring premenstrual symptoms, prospectively documented over two menstrual cycles. Women were randomly assigned to either calcium supplements (1200 mg) or placebo for three menstrual cycles. Daily documentation of symptoms, adverse effects, and compliance with medications were monitored, with a resulting 17 parameter score.

Data were reported for 466 of the 497 women enrolled. The calcium treated group had a significantly lower premenstrual (luteal phase) symptom score for the second ($P = 0.007$) and third ($P < 0.001$) treatment cycles. By the third treatment cycle, the calcium group was associated with a 48% reduction in total symptom scores from baseline, compared with a 30% reduction in the placebo group.

COMMENT BY JOHN La PUMA, MD, FACP

This New York St. Luke's-Roosevelt Hospital Center study found that irritability, depression, food craving, aches and pains, and water retention all improved with calcium carbonate supplementation. With the exception of aches and pains, however, the placebo group improved nearly as much in each category. The salutary effects were not apparent until the second month.

Why should calcium work in PMS? Evidence of secondary hyperparathyroidism in women with PMS has been demonstrated by the same principal investigator, who postulates serotonergic dysregulation in PMS.

Partially funded by SmithKline Beech-am, makers of TUMS®, questions of blinding (TUMS®' texture and flavor are difficult to emulate) and adequacy of pain relief (analgesics were allowed but not tracked) mar this study's methods. The strong placebo effect is comparable to that observed in trials of fluoxitene for premenstrual dysphoria and alprazolam for PMS.

Calcium carbonate is the least expensive form of supplemental calcium, and if not compounded from oyster shells, is unlikely to contain lead, as do some "natural" calcium supplements. Calcium supplements should be taken with food. Some of the best food sources of calcium include a cup of plain nonfat yogurt (450 mg), 3 ounces of sardines with bones (370 mg), a cup of calcium fortified orange juice (300-350 mg), and a cup of cooked turnip greens (200 mg).

A three-month therapeutic trial of 1200 mg of calcium daily for women with moderate or severe symptoms of premenstrual syndrome should be investigated more carefully. It also will, with weight-bearing exercise, reduce the chance of osteoporosis, especially in Caucasian women. Whether calcium acts as a placebo or changes biochemistry, it is an inexpensive and safe approach. (*Dr. La Puma is Adjunct Professor of Nutrition, Kendall College, and Director, C.H.E.F. Clinic, Alexian Brothers Medical Center, Elk Grove Village, IL.*) ❖

Safety of EMLA Cream in Newborn Infants

ABSTRACT & COMMENTARY

Synopsis: *One-hour application of 1.0 g of EMLA cream is safe when used on the intact skin of term neonates younger than 3 months of age.*

Source: Brisman M, et al. *Acta Paediatr* 1998;87:1191-1194.

Increasing concerns about pain-inducing medical or diagnostic procedures has led to increasing use of topical analgesics in infants and children. The most widely used topical analgesic is EMLA cream, a eutectic mixture of lidocaine and prilocaine. When applied to the skin approximately one hour before a painful procedure such as venipuncture, objective assessments have shown a reduction of signs of discomfort and pain in treated infants and children compared to controls.[1] EMLA cream, because of prilocaine, has a possible side effect of the formation of methomeglobin in a patient's blood. Newborn infants, especially premature infants, have low levels of cytochrome reductase, which is necessary for the reduction of methemoglobin. Therefore, infants are considered to be more susceptible to methemoglobin-inducing agents than older children.[2] Accordingly, EMLA has not been generally recommended for use in the newborn.

Brisman and associates conducted a controlled study of the safety of EMLA cream in term neonates. Forty-seven neonates between 0 and 3 months of age were randomized to have either 1.0 g of EMLA cream or a placebo cream applied to their intact skin for 60-70 minutes. Venous levels of methemoglobin were measured at baseline and at three randomly assigned times, 0.5-18 hours after application. Following application of the creams, the mean methemoglobin level of the EMLA group was 1.17% (range, 0.05-2.53%) compared to a mean of 0.96% (range, 0.50-1.53%) in the placebo group. Blood methemoglobin concentrations of the EMLA group were significantly higher than the controls from 3.5 to 13 hours after application. All of the levels were well below poten-

tially harmful levels. Brisman et al conclude that a local one-hour application of 1.0 g of EMLA cream is safe when used on the intact skin of term neonates younger than 3 months of age. Since premature infants were not studied, no recommendations can be made concerning the use of EMLA cream in low-birth-weight babies. ❖

References

1. Kennedy T, et al. *Pediatr Adolesc Med Rep* 1997;2:46.
2. Ross JD, et al. *Blood* 1972;23:419-423.

Pharmacology Update

Modafinil for the Treatment of Narcolepsy

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

Cephalon inc. has received approval to market modafinil (Provigil), the first nonamphetamine drug approved by the FDA for the treatment of excessive daytime sleepiness associated with narcolepsy. Narcolepsy is a disorder that afflicts about 125,000 Americans and is characterized by inability to stay awake or alert in the daytime, sleep attacks, disrupted nocturnal sleep, and cataplexy. Prior treatment for this disorder has consisted primarily of amphetamine type drugs—agents that are commonly associated with side effects and eventual development of tolerance.

While it is known that modafinil is not an amphetamine, the exact mechanism of action of the drug is not known. It apparently does not appear to bind to receptors associated with sleep/wake regulation, such as norepinephrine, serotonin, dopamine, GABA, melatonin, or benzodiazepine.[1]

Indications

Modafinil is indicated to improve wakefulness in patients with excessive daytime sleepiness associated with narcolepsy.

Dosage

The recommended dose of modafinil is 200 mg as a single dose in the morning. There is no consistent evidence that doses greater than 200 mg confer any additional benefit.[1] Patients with severe hepatic impairment should reduce the dose by half.[1] In the elderly population, consideration should be given to use the lowest effective dose.

Modafinil is supplied as 100 mg and 200 mg tablets. The drug is placed into DEA Schedule IV.

Potential Advantages

The major advantage of modafinil over other drugs, such as amphetamines and methylphenidate, used for narcolepsy is its apparent lower abuse potential. Modafinil is Schedule IV while amphetamine and methylphenidate are Schedule II. In clinical trials (9 weeks with open label up to 40 weeks), modafinil reduced daytime sleepiness, was generally well tolerated, did not affect sleep, and tolerance did not appear to be problematic.[1,2,3] The improvement in average sleep latencies was about 58% based on Maintenance of Wakefulness Test (MWT).

Potential Disadvantages

While modafinil does not appear to have the same abuse potential as amphetamine, it may produce effects similar to other CNS stimulants such as euphoric effects and alteration in mood and/or perception. In addition, monkey studies suggest that modafinil is reinforcing in a manner similar to cocaine.[1] Cocaine is one of the most strongly reinforcing self-administered drugs. A clinical study suggested that modafinil produced psychoactive and euphoric effects and feelings consistent with methylphenidate. Patients should be observed for signs of misuse or abuse.[1]

Albeit rare, chest pain, palpitations, dyspnea, and transient ischemic T-wave changes have been observed in association with mitral valve prolapse or left ventricular hypertrophy. Modafinil is not recommended in patients with a history of left ventricular or ischemic ECG changes, chest pain, arrhythmia, or significant manifestations of mitral valve prolapse in association with CNS stimulants.[1]

In vitro studies suggest that modafinil has the potential to inhibit cytochrome P450 2C19, suppress the expression of 2C9, and slightly induce 1A2, 2B6, and 3A4. If coadministration of modafinil and drugs that are substrates for one or more of these isoenzymes is clinically indicated, the patient should be monitored for potential toxicity or reduced effectiveness.

In clinical trials, common side effects of modafinil relative to placebo include headache (50% vs 40%), nausea (13% vs 4%), and diarrhea (8% vs 4%).[1] Five percent of patients discontinue therapy in these trials.

Comments

Modafinil is the first nonamphetamine or nonmethylphenidate drug approved for the treatment of excessive daytime sleepiness associated with narcolepsy. Effectiveness was established in two U.S. multicenter, placebo-controlled, double-blind, nine-week trials in more than 550 patients. The primary measures of efficacy were sleep latency as assessed by the MWT and the

change in the patient's overall disease status, determined by evaluators, as measured by the Clinical Global Impression of Change (CGI-C). MWT assesses the ability of the subject to remain awake without using extraordinary measures.

It measures latency (in minutes) to sleep onset averaged over four test sessions at two-hour intervals. Modafinil improved average sleep latency from 5.07 to 5.35 for placebo to 8.18 to 8.28 for the 200 mg dose. For CGI-C, 58% to 64% of patients improved compared to 37% to 38% for placebo. There are currently no comparative trials between modafinil and current agents such as amphetamine or methylphenidate, and, therefore, comparative efficacy cannot be assessed. A survey of several agents used to treat narcolepsy suggests that modafinil may be less effective than dextroamphetamine or methylphenidate based on MWT.[4]

The wholesale cost of modafinil is about $7 per day for a 200 mg dose.

Clinical Implications

Narcolepsy is a neurologic disorder of unknown cause characterized by excessive somnolence, cataplexy, sleep paralysis, disrupted nocturnal sleep, and hypnagogic hallucinations.[5] It affects 2-10 individuals per 10,000 and has a gradual onset between the ages of 15 and 35. Sleep paralysis is a paralysis of voluntary muscles that occurs at the entry into or emergence from sleep.[6] Hypnagogic hallucinations are visual hallucinations with auditory and tactile components that occur during onset and emergence from sleep.

Cataplexy is a sudden loss of muscle tone (often dropping of the jaw) triggered by strong emotions such as laughter.[6] The symptoms of this condition have serious personal, social, and economic implications as the ability of the individual to function in normal daily activity can be significantly compromised. Excessive daytime sleepiness is generally the most prominent symptom of narcolepsy. Current pharmacologic treatment includes dextroamphetamine, methylphenidate, and pemoline. These drugs have potential for the development of tolerance and unwanted side effects. Modafinil offers an alternative with milder side effects and may have a lower abuse potential. Long-term safety and efficacy remains to be established. As with other stimulants, it does not affect cataplexy, which is generally managed with tricyclic antidepressants.[5,6] Modafinil is only FDA-approved for use in narcolepsy. Efficacy and safety in improving vigilance in healthy sleep- derived individuals has not been established. Results from a trial of modafinil in sleep apnea patients are expected early next year. ❖

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3. Broughton RJ, et al. *Neurology* 1997;49(2):441-451.
4. Mitler MM, et al. *Sleep* 1991;14(3):218-220.
5. Adams RD, et al. *Principles of Neurology.* 6th ed. McGraw-Hill; 1997:380-402.
6. Fry JM. Sleep Disorders. In: *Merritt's Textbook of Neurology.* 9th ed. William & Wilkens; 1995:875-881.

CME Questions

20. Which of the following statements is true about the association of hypertension and kidney stones?

a. Nephrolithiasis increases the risk for hypertension.
b. Hypertension increases the risk for nephrolithiasis.
c. There is no association between hypertension and nephrolithiasis.
d. None of the above

21. Recent investigations have demonstrated a relationship between streptococcal infection and auto-immune-precipitated neurological disease in children. Which of the following disorders has *not* been verified in this respect?

a. Chorea
b. Recurrent myoclonus
c. Tourette syndrome
d. Generalized epilepsy
e. Myelitis

By Louis Kuritzky, MD

Prevention of Falls in the Elderly Trial (PROFET)

More than one-third of the 8% of persons older than 70 who seek care for fall-related injuries are admitted to the hospital. Improved management tools to prevent fall-related injury would be desirable, but limited, and sometimes, conflicting previous studies fail to fulfill this need. The current randomized, controlled trial evaluated a structured assessment of elderly persons (≥ 65 years), who obtained emergency care because of a fall, to see if such an approach could improve future outcome and reduce future falls.

Evaluation (n = 1031) included general physical status plus details on visual acuity, balance, cognition, effect, prescription medications, and postural hypotension. Each patient was also visited on a single occasion by an occupational therapist, with environmental hazards identified and corrected when possible.

Only one out of six patients had evidence of a cardiovascular or circulatory disorder likely to have contributed to a fall. More than half of the patients had visual impairment, almost two-thirds had poor stereoscopic vision, and more than one-third had cataracts in one or both eyes. Almost three-fourths of the patients were unable to stand on one leg with their eyes open, and cognitive impairments or depression were present in half of patients.

Over the 12-month follow-up period, there were significantly fewer falls in the intervention group. A reduction of 50% in fractures was also noted. Close and colleagues conclude that incorporation of falls and injury prevention strategies provides substantial clinical benefit and should be more widely used. ❖

Close J, et al. Lancet 1999;353:93-97.

Primary Care Physicians' Perceptions of Diabetes Management

Evidence continues to accumulate that better control of diabetes results in better patient outcomes. Unfortunately, more than half of diabetic adults have a glycosylated hemoglobin greater than 9.5%, despite the suggested goal of less than 7%. To gain insight into how primary care physicians view and manage diabetes, a trained research interviewer performed in-depth personal interviews with primary care physicians (FPs and internists), specifically directed toward learning the clinicians' approach to diabetes, feelings about the seriousness of the disorder, observations about patient attitudes toward diabetes, and changes in clinician views about diabetes.

The most consistent emerging theme from clinicians was that diabetes management is a balancing act between ideal medical goals, and realities of patient adherence, preferences, and personal circumstances. Physicians acknowledge that most diabetic patients do not follow management recommendations. Physicians were in agreement with the overall established goals of good glycemic control and complication prevention but did not possess readily accessible tools with which to attain these goals. Helseth and associates suggest that groups that develop guidelines spend additional energies to enhance tools or strategies with which clinicians might better achieve the biochemical and behavioral goals of diabetes management. ❖

Helseth LD, et al. J Fam Pract 1999; 48:37-42.

Sildenafil for Treatment of Erectile Dysfunction in Men with Diabetes

Sildenafil has an established role in the approximately 50% of 40- to 70-year-old men who suffer erectile dysfunction (ED). ED is substantially more common and occurs at a younger age in diabetic men than in the general population. The current randomized, double-blind study specifically examined the role of sildenafil in middle-aged diabetic men (n = 268) with ED.

Patients received 25 mg, 50 mg, or 100 mg of sildenafil or placebo, depending upon efficacy and adverse effect profile, for 85 days. Maintenance dose for 93% of patients receiving an active drug was 100 mg; no patient in this study responded adequately to 25 mg.

Use of sildenafil almost doubled the frequency of adequate erections and intercourse experiences, as well as substantially improving overall satisfaction with sex life. There was no change in frequency of sexual desire. Of these measured factors, placebo only affected overall satisfaction with sex life, though erection rigidity and frequency of satisfactory intercourse did not change. Anticipated side effects of sildenafil (headache and dyspepsia) were proportionally frequent in this population as in previous study populations, but no patient discontinued treatment due to an adverse drug effect. Rendell and colleagues conclude that sildenafil is an efficacious, well-tolerated treatment for ED in diabetic patients. ❖

Rendell MS, et al. JAMA 1999; 281:421-426.

Confirming Dextrocardia: Technician Error

By Ken Grauer, MD

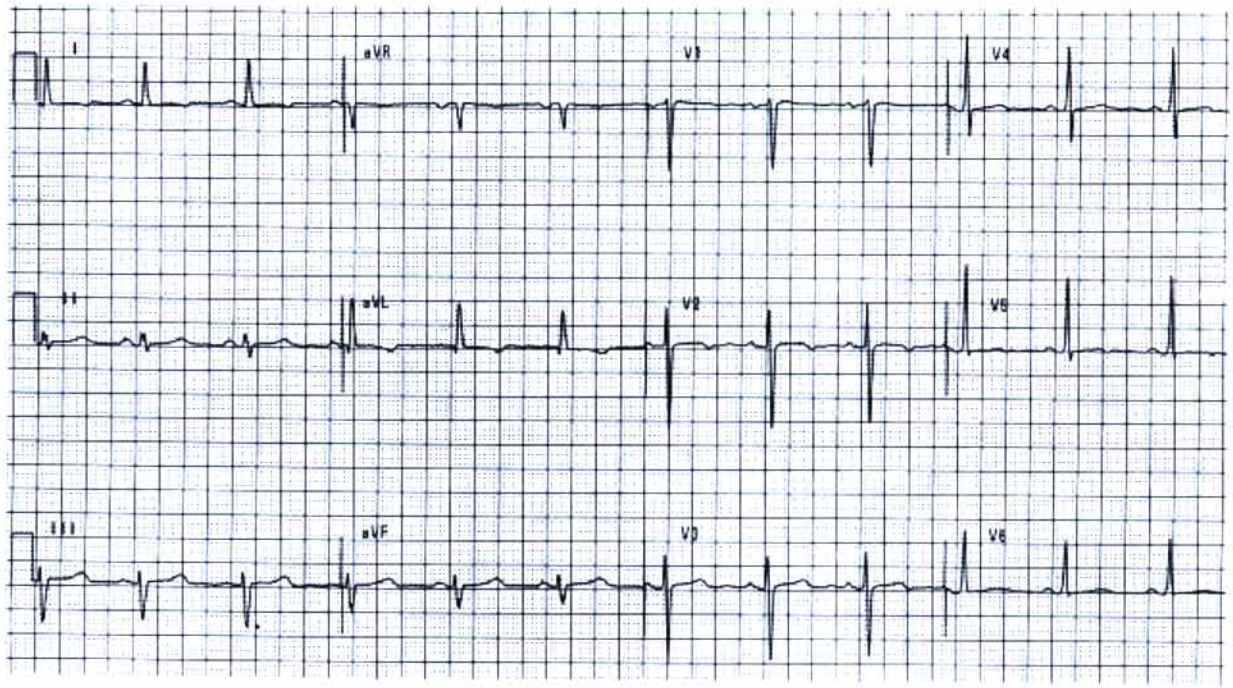

Figure. ECG obtained from a patient with dextrocardia after "reversing the leads." What did the tech do wrong?

Clinical Scenario: The ECG shown in the Figure was obtained from a 60-year-old woman with dextrocardia (it is the follow-up tracing to the ECG shown in last month's ECG Review). In an attempt to cofirm the diagnosis of dextrocardia, the tech had been asked to "repeat the ECG with the leads reversed." How should the tech have been instructed to repeat this ECG? What simpler approach could have been used to confirm dextrocardia?

Interpretation: The finding of complete (or almost complete) negativity of the QRS complex in lead I in association with an upright QRS complex in lead aVR is distinctly abnormal and should always prompt consideration of two clinical entities: 1) dextrocardia; and 2) limb lead misplacement. Practically speaking, the latter is much more common. Assessment of R wave progression in the precordial leads will usually distinguish between these two entities: R wave progression should be normal when there is limb lead reversal, whereas R wave progression is *reversed* when there is dextrocardia (as it was in last month's ECG Review). Verifying correct placement of limb lead electrodes and then repeating the ECG with precordial leads reversed should confirm what the true diagnosis is—in that R wave progression will normalize for a patient with dextrocardia when precordial leads are placed on the right side of the chest (as they do in the Figure). The error the tech made in this case was to also reverse the limb lead electrodes—which is why the QRS complex is now upright in lead I. The simplest way to confirm dextrocardia is to listen for heart sounds on the right side of the chest. ❖

In Future Issues: Selective Serotonin Reuptake Inhibitor Discontinuation Syndrome