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INSIDE

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Is Hershey's Heart Healthy?

ABSTRACT & COMMENTARY

By Allan J. Wilke, MD

Professor, Department of Introduction to Clinical Medicine, Ross University School of Medicine, Commonwealth of Dominica

Dr. Wilke reports no financial relationships relevant to this field of study.

Synopsis: *Eating chocolate may be part of a healthy lifestyle.*

Source: Buitrago-Lopez A, et al. Chocolate consumption and cardiometabolic disorders: Systematic review and meta-analysis. *BMJ* 2011 Aug 26;343:d4488. doi: 10.1136/bmj.d4488.

THIS IS A SYSTEMATIC REVIEW AND META-ANALYSIS THAT BUITRAGO-Lopez and colleagues designed to test the association between dietary chocolate and the risk of developing certain cardiovascular disorders (coronary heart disease, stroke, congestive heart failure, and myocardial infarction), diabetes, and the metabolic syndrome. They searched the literature, including Medline, Embase, the Cochrane Library, Scopus, Scielo, Web of Knowledge, AMED, and CINAHL, in the summer of 2010, looking for randomized control trials or cohort, case-control, or cross-sectional studies. Reports were included if they studied non-pregnant adults only and included the disorders of interest. They went back to the authors of the papers that met their criteria for any additional information. Beginning with 4576 references, they eliminated all but seven. These studies include one cross-sectional study from United States and 6 cohort studies from Europe, Asia, and North America. The seven studies included 114,000 mostly white subjects, ranging in age from 25 to 93 years. The studies did not distinguish between dark and white chocolate consumption, but one did report cocoa consumption. Chocolate was consumed in a variety of forms, including candy bars, drinks, snacks, and nutritional supplements. There was no uniformity among the seven studies in how chocolate consumption was measured. Follow-up in the cohort studies ranged from 8 to 16 years. The studies were scored for quality. None of the studies were at the highest level of quality (6 points), but

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all of them scored in the adequate range (4 or 5 points). For the purpose of this meta-analysis, the authors chose to look at only the lowest and the highest levels of chocolate intake for each study. Five of the studies showed an inverse relationship between chocolate intake at the highest level and the development of the diseases of interest. Chocolate intake was associated with a reduction in risk of 29% (for stroke) to 37% (for cardiovascular disease), all statistically significant. The only disorder that did not show a significant beneficial association was heart failure, but it trended that way.

■ COMMENTARY

I love studies that validate what I'm already doing.

This is not the last word on the subject. None of the studies included in this meta-analysis were randomized controlled studies, so the best that can be concluded is that there is an association (but not causation) between chocolate intake and reduction in risk of some cardiovascular diseases, diabetes, and the metabolic syndrome. This meta-analysis does raise several questions. Why isn't heart failure in the list? Since heart failure is a late sequela of heart disease, it may be that it was too late for chocolate to have any effect. How much chocolate needs to be consumed? Is there a dose response? Are there differences between types of chocolate? I'm pulling for dark.

Assuming for the moment that chocolate consumption does reduce risk, how does it do it? A component of chocolate is flavonol — also present in kale, broccoli, tomato, apple, grape, tea, and red wine — which is associated with a reduction in risk of coronary heart disease mortality.[1] Chocolate has antioxidant,[2] antihypertensive,[3] and anti-inflammatory effects.[4] It increases insulin sensitivity, improves vascular endothelial function, and combats atherogenesis and thrombosis.[5]

Chocolate dates back to the Mayan and Aztec peoples who used it in religious ceremonies. They did not add sugar to it. At the turn of this century, a major U.S. chocolate manufacturing company (not Hershey) tried to capitalize on the burgeoning research into the positive health effects of cocoa flavonols and introduced a line of snacks with "real chocolate pleasure, real heart health benefits."[6,7] Chocolate, as it is usually consumed today, cannot be considered a health food. Typically, it is accompanied by large amounts of fat and sugar to make it more palatable. Encouraging chocolate consumption could have the unintended consequence of increasing cardiovascular risk.

The authors conclude, "Corroboration is now required from further studies, especially experimental studies to test causation rather than just association." I want to be part of the intervention group! With the ubiquity of chocolate in our environment, however, I suspect it would be very difficult to do a double-blind, randomized, controlled study. Can you imagine a "chocolate-like" placebo? I think the best advice we can give our patients is that chocolate is not associated with any harmful cardiovascular or metabolic effects, and as with so many other pleasurable things in life, is best enjoyed in moderation. ■

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Questions & Comments
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Predicting Sudden Cardiac Death in Women with CAD: Are We There Yet?

ABSTRACT & COMMENTARY

By Joseph Varon, MD, FACP, FCCP, FCCM

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Dr. Varon receives grant/research support from Baxter and EKR, is a retained consultant for Baxter, and serves on the speakers bureaus for Baxter, EKR, and The Medicines Company.

Synopsis: *Sudden cardiac death (SCD) among postmenopausal women with coronary artery disease is common. The presence of congestive heart failure, reduced kidney function, atrial fibrillation, physical inactivity, and diabetes are independent risk factors for SCD. Some of these factors can be modified and SCD can be prevented.*

Source: Deo R, et al. Risk factor and prediction modeling for sudden cardiac death in women with coronary artery disease. *Arch Intern Med* 2011;171:1703-1709.

This study was aimed at evaluating postmenopausal women with established coronary artery disease (CAD) in an attempt to quantify the risk for SCD in this population, and to compare it with risks factors for mortality from other cardiac and noncardiac clinical conditions. This multicenter trial is a subgroup analysis of the Heart and Estrogen/progestin Replacement Study (HERS), a randomized, double-blinded, placebo-controlled trial of the effect of treatment with 0.25 mg of conjugated estrogens plus 2.50 mg of medroxyprogesterone acetate daily vs placebo on the CAD event rate among 2763 postmenopausal women with documented CAD.[1] All study participants were postmenopausal women younger than 80 years of age with no prior hysterectomy, and a history of at least one of the following: acute myocardial infarction, coronary artery bypass graft surgery, percutaneous coronary angioplasty, or angiographic narrowing of a coronary artery of more than 50%.

Of the 2763 postmenopausal women with CAD in the HERS study, the average age was 67 years, and the median follow-up was 4.1 years. There were no significant differences in the rates of primary CAD events or secondary cardiovascular events, including SCD, among women assigned to the hormone group as compared with the placebo group. SCD was defined by the investigators as death that occurred within 1 hour of the onset of symptoms.

In this trial, there were 254 cardiac deaths and 246 noncardiac deaths during the follow-up period. SCD made up 54% (136 events) of the cardiac-related deaths, with an annual event rate of 0.79% per year (95% confidence interval, 0.67-0.94). Of note, there were no significant differences in most of the baseline characteristics across the different groups.

Those patients who died of cardiac causes (either SCD or others) had a higher prevalence of congestive heart failure and diabetes, a higher body mass index, diabetes, physical inactivity, and a lower serum low-density lipoprotein level than those who died of noncardiac causes. Myocardial infarction, congestive heart failure, and a low glomerular filtration rate were associated with a 2-fold or higher risk for SCD. The participants with no risk factors had an annualized SCD risk of 0.34% compared with 2.90% for those with at least three risk factors.

■ COMMENTARY

Sudden cardiac death remains a significant issue across the world, claiming between 350,000-440,000 lives per year in the United States alone.[2] Moreover, cardiopulmonary arrest in the context of cardiovascular disease occurs suddenly and in the most cases without signs or symptoms. Therefore, studies aimed to identify predictors for SCD are extremely important.

Prior studies have shown that women have a 10-fold lower risk of SCD.[3] Indeed, the annual rate of SCD among women in the HERS study is lower than SCD rates observed in populations with an established cardiomyopathy. Despite these lower rates, a significant group of well-functioning women with CAD remain at risk of SCD.

This well-conducted study is interesting because it shows that congestive heart failure, reduced kidney function, atrial fibrillation, physical inactivity, and diabetes were independent risk factors for SCD in postmenopausal women with CAD. Many of these risk factors can be modified and, hopefully, fatal outcomes prevented. ■

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Which Is Better: Open, Laparoscopic, or Robotic? How Meaningful Is This Question?

ABSTRACT & COMMENTARY

By Frank W. Ling, MD

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Dr. Ling reports no financial relationships relevant to this field of study.

This article originally appeared in the December issue of OB/GYN Clinical Alert. *At that time it was peer reviewed by Catherine Leclair, MD, Associate Professor, Department of OB/GYN, Oregon Health & Science University Portland, OR. Dr. Leclair reports no financial relationships relevant to this field of study.*

Synopsis: *This commentary challenges the urologic medical community to get past its "collective obsession with technology" and try to figure out why some surgeons have better outcomes, irrespective of the surgical approach taken.*

Source: Vickers AJ. Great meaningless questions in urology: Which is better, open, laparoscopic, or robotic radical prostatectomy? *Urology* 2011;77:1025-1026.

THE AUTHOR INFORMS US THAT THE WINNER OF THE 2010 Tour de France was Alberto Contador, riding a Specialized SL3 racing bike. The U.S. rider Chris Horner finished 12 minutes behind riding a Trek, Madone. The best rookie finisher, Daniel Loyd, rode a Cervelo S3, and finished more than 4 hours behind the leaders. The author opines that "no self-respecting urologist" would use this information to claim that the Trek is a faster bike than the Cervelo or that Loyd would have won the race had he ridden a Specialized.

The reader also is told that surgical complication rates among high-volume surgeons who perform radical prostatectomies range from < 5% to > 50%. He also cites in one study that functional outcomes differ by up to 40% with regard to erectile and urinary function. The author points out that the difference between surgeons and their performance dwarfs the inherent differences of the surgical approach. As in the Tour de France, where the focus should not be the bicycle, in urology, the focus should not be on the surgical approach when performing radical prostatectomies.

Comparative publications analyzing complications and success rates are unable to control for pathologists' skills, patient population characteristics, and/or definitions of "success" or "complication." He compares this to asking the three cyclists to go on a 100-mile ride, with the best bike being the one ridden by the first person to get to the finish line, irrespective of the route taken, weather, etc.

The analogy is carried further: The cycle judged to be the best cannot be the one that finishes first because of variables such as the experience of the rider. As with cycling, a skilled, experienced surgeon is different from a novice surgeon, and both are different from the "average" surgeon. In fact, the term "average" raises the statistical issue of results. Vickers points out that depending on how the results are collected and reported, surgical outcomes numerically may look similar, but, as far as patient outcome is concerned, may be very different.

The author concludes that Lance Armstrong said, "It's not about the bike." He asserts that some urologists seem to be saying, "No, but it is all about the robot." Studying how to get the best results should be the goal, but this will require "…far greater investment of time, resources, and scientific ingenuity than retrospective analyses of surgical databases."

■ COMMENTARY

Admittedly, you probably don't get this journal. Even if you did, you probably wouldn't think of this article (it's an opinion piece, not a research study) as something worth reading or reviewing since it's about radical prostatectomy. Forget the fact that none of us perform the procedure. We're being challenged by the author, and he doesn't even know he's doing it! He suggests that urologists as a group get past its "obsession with technology" and try to identify factors that lead to some surgeons getting better results than others, irrespective of the surgical approach used.

First, I like that the author is a PhD in the Department of Epidemiology and Biostatistics at Memorial Sloan-Kettering Cancer Center. He isn't someone who does any of these procedures. In essence, there is less chance that he has a bias regarding one procedure or another. He also is unlikely to have a conflict of interest relating to an instrument company or another product being used during the surgery. We should be watchful for who is writing the articles that we read, particularly when it's involving surgical approaches and technique. Does someone have an axe to grind?

Second, I love the analogy. It makes sense and gives us a fresh perspective on how we look at surgical literature.

Third, what defines "success?" In the case of radical prostatectomy, the urologists are trying to avoid recurrence and maintain intact sexual and urinary function. What about a 5-year success rate? What would the world class cyclists define as "successful?"

Fourth, aren't there factors in the equation beyond just the surgeon's decision-making and skills? What about the patient who has done his "research" and knows the surgical approach that he feels is appropriate for his case? What

about the role of the hospital and its administration who may be publicly extolling the virtues of a newly acquired (and very expensive) piece of equipment? How long a learning curve for new technology is acceptable? Is it the same for everyone? In order for a surgeon to gain needed experience, how long is it reasonable for patients who might not need the new technology to be treated with it?

Finally, you might notice that the cyclist wears a jersey boldly displaying the sponsor's name. I don't think any of us has seen any surgeons with similar commercialized garb entering the operating room (I know I haven't…have you?). Whether the influence of industry on us is subtle or overt, each of us is responsible for being as candid as possible with our patients regarding all surgical approach options … including no surgery at all. Until definitive information is available (and, honestly, we may never get it), overzealous rushing to a new technology is probably no worse than an unswerving aversion to it.

Let's get past our fascination with the latest technology and try to determine how to best serve our patients by getting the best outcomes. Sometimes newer is, indeed, better. Sometimes, it isn't. ■

Pharmacology Update

Rivaroxaban Tablets (Xarelto®)

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

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

RIVAROXABAN, AN ORAL FACTOR XA INHIBITOR, HAS BEEN approved for reducing the risk of stroke in patients with atrial fibrillation (AF). The drug joins dabigatran (Pradaxa) as the second "nonwarfarin" oral anticoagulant approved for this indication. Rivaroxaban was previously approved for the prophylaxis of deep vein thrombosis (DVT) in patients undergoing hip or knee replacement (see Pharmacology Update, August 15, 2011, page 117). The drug is marketed by Janssen Pharmaceuticals as Xarelto.

Indications

Rivaroxaban is indicated for reducing the risk of stroke and systemic embolism in patients with nonvalvular AF and for the prophylaxis of DVT in patients undergoing knee or hip replacement.[1]

Dosage

The recommended dose for patients with AF is 20 mg once daily with the evening meal, as food increases the bioavailability of rivaroxaban. For patients with creatinine clearance between 15 and 50 mL/min, the recommended dose is 15 mg. For the prophylaxis of DVT, the dose is 10 mg daily. The duration of therapy is 12 days for knee replacement and 35 days for hip replacement.[1]

Potential Advantages

Rivaroxaban is dosed once daily compared to twice daily for dabigatran. Similar to dabigatran, laboratory monitoring is not required.

Potential Disadvantages

The drug should not be co-administered with combined P-gp and strong CYP3A4 inhibitors (e.g., ritonavir, intraconazole, ketoconazole).[1] P-gp and strong CYP3A4 inducers may reduce the effectiveness of rivaroxaban while combined P-gp and weak or moderate inhibitor may increase systemic exposure. Discontinuation of rivaroxaban may increase the risk of thrombotic events.[1] Patients on rivaroxaban may develop epidural or spinal hematomas if they receive neuraxial anesthesia or undergo spinal puncture.[1] Currently there is no antidote for rivaroxaban (see Clinical Implications).

Comments

Rivaroxaban is an orally active factor Xa inhibitor. Its efficacy for stroke prevention in AF was based on a multinational, double-blind, randomized, noninferiority comparative trial to warfarin (ROCKET AF),[1,2] which enrolled 14,264 patients with nonvalvular AF with moderate-to-high risk for stroke. The median age was 73 years, 60% were male, 90% had hypertension, and the median $CHADS_2$ score was 3.0 (87% equal to or higher than 3). Patients were randomized to rivaroxaban 20 mg (15 mg with reduced renal function) or dose-adjusted warfarin. The median duration of treatment exposure was 707 days. The primary efficacy outcome was stroke or systemic embolism and the primary safety outcome was a composite of major and nonmajor bleeding events. The rate of stroke or systemic embolism based on intention-to-treat was 2.1% per year for rivaroxaban and 2.4% per year for warfarin (hazard ratio, 0.88; 95% confidence interval 0.75 to 1.03). Noninferiority was demonstrated but not superiority. There were no differences in composite major and nonmajor bleeding events (14.9% vs 14.5% per year) or major bleeding events (3.6% vs 3.4% per year). However, intracranial hemorrhage was lower for rivaroxaban (0.5% vs 0.7% per year, $P = 0.02$).

Major gastrointestinal (GI) bleeding events were higher with rivaroxaban (3.2% vs 2.2%, $P < 0.001$). A higher percent of patients on rivaroxaban discontinued participation in the clinical trial due to bleeding events (4.3% vs 3.1%).

Clinical Implications

This is the second nonwarfarin drug approved for stroke prevention in nonvalvular AF. The first was the thrombin inhibitor dabigatran. A third drug, also a factor Xa inhibitor, apixaban, is awaiting FDA approval. While the new agents were all compared to dose-adjusted warfarin, it is difficult to compare across studies by applying transitive logic due to study differences. In RE-LY (dabigatran), 32.7% had a $CHADS_2$ score above 3 while in ARISTOTLE (apixaban), those above $CHADS_2$ score of 3 were 30.2%, compared to 87% for (ROCKET AF).[2-4] Other differences included the effective dosing of warfarin. For the dabigatran study, the median percent time in therapeutic range (%TTR) for warfarin was about 65% compared to 55% for rivaroxaban and 66% for apixaban. Other observations among these studies: dabigatran and apixaban were superior to warfarin, apixaban showed a lower risk of major bleeds, all three drugs showed a lower risk of intracranial hemorrhage, dabigatran and rivorxaban showed a higher risk of GI bleeds, and dabigatran showed a higher risk of myocardial infarction. Currently there is no antidote for these agents, although a recent report suggests that prothrombin complex concentrate may be able to reverse rivaroxaban but not dabigatran.[5] Reversal of these drugs may be an important clinical issue, as a total of 256 fatal bleedings have been reported with dabigatran worldwide as of November 6, 2011.[6] Boehringer Ingelheim indicated that the number of deaths was within expectations. More clinical experience in practice will determine if one or more of these agents emerges as the preferred agent. ■

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CME Objectives

Upon completion of this educational activity, participants should be able to:

- describe new findings in the differential diagnosis and treatment of various diseases;
- describe the advantages, disadvantages and controversies surrounding the latest advances in the diagnosis and treatment of disease;
- identify cost-effective treatment regimens;
- explain the advantages and disadvantages of new disease screening procedures.

CME Instructions

To earn credit for this activity, follow these instructions:

1. Read and study the activity, using the provided references for further research.
2. Log on to www.cmecity.com to take a post-test; tests can be taken after each issue or collectively at the end of the semester. First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice or renewal notice.
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.
5. Once the completed evaluation is received, a credit letter will be e-mailed to you instantly. You will no longer have to wait to receive your credit letter! ■

CME Questions

1. **The chocolate meta-analysis showed all of the following associations *except*:**
   a. a reduction in risk for stroke.
   b. a reduction in risk for heart failure.
   c. a reduction in risk for myocardial infarction.
   d. a reduction in risk for diabetes.
   e. a reduction in the metabolic syndrome.

2. **In the study by Deo and colleagues, risk factors that were identified in postmenopausal women that were predictive of sudden cardiac death included:**
   a. lower serum estrogen levels.
   b. a prior history of pulmonary embolism, deep vein thrombosis, and coagulopathy.
   c. congestive heart failure, renal dysfunction, atrial fibrillation, physical inactivity, and diabetes.
   d. prior use of thrombolytics.
   e. age older than 80 years.

Clinical Briefs

By Louis Kuritzky, MD, Clinical Assistant Professor, University of Florida, Gainesville

Dr. Kuritzky is an advisor for Endo, Kowa, Pricara, and Takeda.

Barrett's Esophagus: What's the Risk?

Source: Hvid-Jensen F, et al. Incidence of adenocarcinoma among patients with Barrett's esophagus. *N Engl J Med* 2011;365:1375-1383.

For unknown reasons, adenocarcinoma of the esophagus (E-ca) is experiencing the most rapid increase of any known cancer in the United States. Although the absolute incidence of E-ca pales next to lung, prostate, or breast cancer, the inexplicable proliferation of this cancer has spurred increased scrutiny of at-risk individuals. Barrett's esophagus, which is felt to represent an attempt at protective epithelial remodeling in response to the trauma of acid exposure, occurs in as many as 10% of individuals undergoing endoscopy for symptoms of GERD. Once Barrett's is identified, consensus group guidelines suggest ongoing surveillance, despite the absence of outcomes trials indicating that such surveillance improves survival.

The Danish Pathology Registry and Cancer Registry provide an opportunity to review data accrued for the entire population of Denmark. Follow-up of persons with Barrett's esophagus (n = 11,029) over a median of 5.2 years of observation identified 197 cases of E-ca, for an annual incidence of 0.12%. Likelihood of developing E-ca was increased in persons with higher degrees of dysplasia. Based on these data, the authors suggest that ongoing surveillance of Barrett's esophagus might wisely be limited to those with demonstrated dysplasia, since the incidence of E-ca in persons without dysplasia was so very low. ■

The Calcium/Cardiovascular Disease Link

Source: Sabanayagam C, Shankar A. Serum calcium levels and hypertension among U.S. adults. *J Clin Hypertens* 2011;13:716-721.

The relationship between calcium intake — through diet and/or supplements — and vascular health is complex. Some recent epidemiologic surveys have found a positive association between calcium supplements and adverse cardiovascular (CV) outcomes, as well as vascular calcification (i.e., more CV disease and calcification with calcium supplementation than without). Because hypertension (HTN) is the most common vascular antecedent to adverse CV events, investigation of the relationship of calcium to blood pressure (BP) is pertinent.

The National Health and Nutrition Examination Survey (NHANES) has published cross-sectional data from diverse populations throughout the United States for more than 30 years. The authors obtained data from the NHANES population (n = 12,403) of adults over age 20 seeking to examine the relationship between serum calcium levels and BP.

Persons in the highest quartile of serum calcium were 1½ times more likely to have HTN than those in the lowest quartile. Even when adjusted for age, race, alcohol, body mass index, cholesterol, C-reactive protein, glomerular filtration rate, serum albumin, vitamin D, and phosphorus, the relationship between calcium and BP remained.

Several mechanisms through which calcium might incur increased risk of HTN have been offered, including a direct vascular effect, parathyroid activity, and renal vasoconstriction. Before concluding that calcium is simply a "bad guy," it is important to recognize that several reports have shown that *dietary* calcium is *inversely* related to HTN. ■

Life Expectancy: The Japanese Are #1

Source: Murray CJ. Why is Japanese life expectancy so high? *Lancet* 2011; 378:1124-1125.

Even though Japan spends essentially half of what the United States spends on health care (8.5% of their gross domestic product vs 16.4% of ours), they have ranked No. 1 in life expectancy for 30 years. To what might we attribute their success?

That Japan provides universal health coverage can certainly be responsible for a portion of their favorable outcomes, but other factors are also at play. For instance, Japanese public health programs to reduce salt intake and become more aggressive about blood pressure control are credited with substantial reductions in stroke. Indeed, such Japanese hypertension programs have evoked a substantial decline in blood pressure among the population as a whole, especially in women (the gender in which successful blood pressure control is conspicuously less prominent in the United States).

Although it is difficult to measure the direct impact of one additional factor — educational attainment — on health, epidemiologic surveys do consistently indicate a linear relationship between education and positive health outcomes. The generally high educational attainment among Japanese may be a critically important factor.

Current health trends suggest that Japan may not stay in the No. 1 slot: Inadequate tobacco control and a rising BMI among the population, unless counteracted, may result in similar adverse health effects as have been seen in other nations. ■

The Regular SVT Differential

By Ken Grauer, MD, *Professor Emeritus in Family Medicine, College of Medicine, University of Florida*

Dr. Grauer is the sole proprietor of KG-EKG Press, and publisher of an ECG pocket brain book.

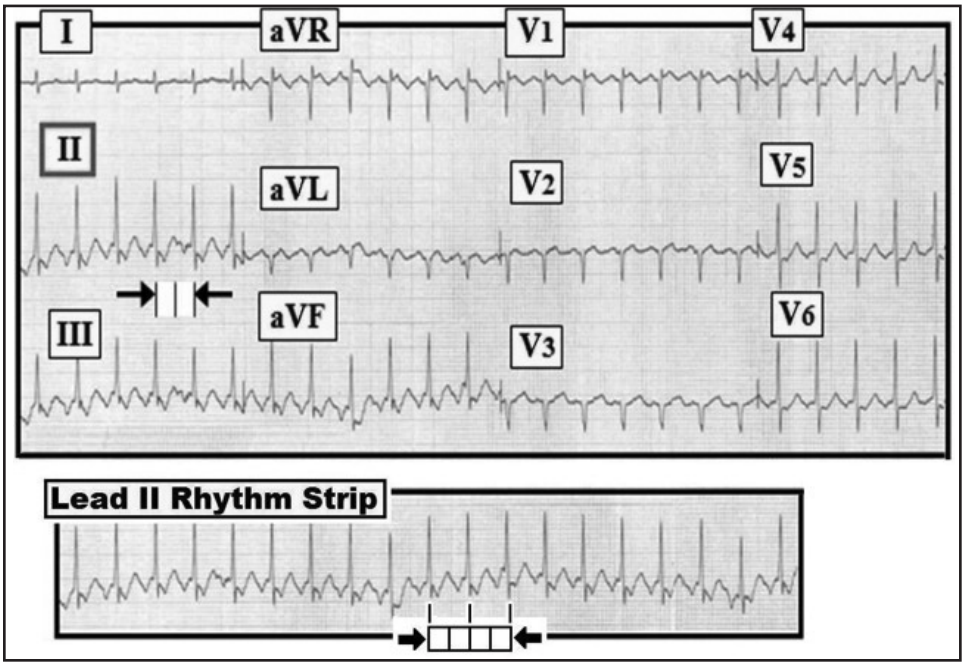

Figure — 12-lead ECG and lead II rhythm strip from a patient with "rapid heart rate." What is your differential diagnosis?

Scenario: Interpret the rhythm for the 12-lead ECG and lead II rhythm strip shown above. The patient was aware of "rapid heart beat" — but was hemodynamically stable at the time the tracing was recorded. What is your differential diagnosis? What diagnostic maneuver might help to determine what the rhythm is?

Interpretation: The rhythm is regular at a ventricular rate that is close to 150/minute (the R-R interval is approximately two large boxes in duration). The QRS complex is narrow (not more than half a large box in duration in any of the 12 leads of the tracing). Normal atrial activity is absent, since upright P waves are *not* seen in lead II. Instead, there is suggestion of atrial activity having a *negative* deflection in lead II (as well as in other inferior leads). There also appears to be a negative *notching* in the ST segment in each of the inferior leads. *Could this all represent atrial activity?*

The best description of the tachycardia defined by the above ECG and rhythm strip is that this represents a *regular* SVT (supraventricular tachycardia) *without* sign of normal atrial activity. This description should prompt consideration of three entities as the most likely cause: 1) sinus tachycardia; 2) atrial flutter; and 3) PSVT (paroxysmal supraventricular tachycardia). Although many other entities might be included in the differential of a regular SVT, the overwhelming majority of cases encountered by primary care clinicians in or out of the hospital will be due to one of these three causes. In this particular case, sinus tachycardia is unlikely because there is no indication of an upright P wave in lead II. We suspect atrial flutter because this is the most common cause of a regular SVT at a rate between 140-160/minute when normal atrial activity is absent. This is especially true when there is hint of a "sawtooth" activity in one or more leads on the tracing (*see leads II, III, aVF; and leads aVR, aVL, V1*). Application of a vagal maneuver in this patient temporarily slowed the rate of AV conduction, allowing "telltale" flutter waves at ~300/minute to appear. ■

In Future Issues:

Yoga for Chronic Low Back Pain