INTERNAL MEDICINE ALERT

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INSIDE

Financial Disclosure:
Internal Medicine Alert's editor, Stephen Brunton, MD, serves on the advisory board for Amylin, Boehringer Ingelheim, Novo Nordisk, and Symbiotix; he serves on the speakers bureau of Boehringer Ingelheim, Novo Nordisk, and Teva. Peer reviewer Gerald Roberts, MD, reports no financial relationship to this field of study.

Association of Vitamin D Deficiency and Myocardial Infarction

ABSTRACT & COMMENTARY

By Harold L. Karpman, MD, FACC, FACP

Clinical Professor of Medicine, UCLA School of Medicine

Dr. Karpman serves on the speakers bureau for Forest Laboratories.

Synopsis: *Vitamin D deficiency was found to be present in almost all patients entering with acute myocardial infarctions in a 20-hospital multicenter study.*

Source: Lee JH, et al. Prevalence of vitamin D deficiency in patients with acute myocardial infarction. *Am J Cardiol* 2011;107:1636-1638.

Numerous reports have clearly outlined the association between cardiovascular disease (CVD) and 25-hydroxyvitamin D (vitamin D) deficiency.[1-5] However, the prevalence of vitamin D deficiency in patients presenting with an acute myocardial infarction (AMI) has not been previously reported. Obviously, this information would be most important to know because vitamin D deficiency is readily treatable and, if it is present with significant frequency in patients with AMI, vitamin D administration might be an important and easy way to reduce the incidence of AMIs.

Lee and colleagues performed a substudy analyzing the Translational Research Investigating Underlying Disparities in Acute Myocardial Infarction Patients' Health Status (TRIUMPH) registry in order to investigate the prevalence of vitamin D deficiency at the time of AMI care.[6] Vitamin D deficiency was assessed in 239 subjects enrolled in a 20-hospital prospective myocardial infarction registry. Of the 239 patients, 179 (75%) were considered to be vitamin D deficient (vitamin D level below 20 ng/mL) and 50 (21%) were vitamin D insufficient (vitamin D level between 20-30 ng/dL). Vitamin D deficiency was found to be more common in non-Caucasian patients and in those with lower social support, with lower activity levels, in diabetics, and in subjects who had no medical insurance. They concluded that vitamin

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D deficiency was present in almost all patients with AMI since 96% of the patients in the study were found to have abnormally low vitamin D levels.

■ COMMENTARY

Lee et al's finding of an extraordinarily high prevalence of vitamin D deficiency or insufficiency in patients admitted with an AMI is consistent with published data associating CVD and many of its risk factors with vitamin D deficiency.[4] The authors also confirmed previously described associations between demographic traits and vitamin D deficiency in those subjects with darker skin (i.e., lighter skinned individuals can produce larger amounts of vitamin D with a fixed amount of ultraviolet B radiation),[7,8] diabetes mellitus,[9] and higher body mass indices.[5] It has been suggested that vitamin D deficiency is significantly reduced in obese individuals because the vitamin is sequestered in adipose tissue.[10] One of the major limitations of the Lee study is that because of the overwhelming proportion of patients who were vitamin D deficient or insufficient, it was not surprising to find that the study lacked an adequate control group for comparison.

The results of the current study leave little question that vitamin D deficiency or insufficiency is present in almost all patients with AMI. Obviously, one cannot conclude that vitamin D deficiency or insufficiency is a contributing cause of AMI. However, there seems to be little question that clinicians should measure vitamin D levels regularly in all of their patients — especially in those who are at risk for AMI — and should treat any detected deficiency of vitamin D with oral vitamin D supplementation. ■

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What Works for Painful Diabetic Neuropathy?

ABSTRACT & COMMENTARY

By Joseph E. Scherger, MD, MPH

Clinical Professor, University of California, San Diego, CA

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

Synopsis: *A systematic review covering 48 years gives pregabalin (Lyrica) Level A evidence for the treatment of diabetic neuropathy. Gabapentin, sodium valpro-*

ate, amitriptyline, venlafaxine, and duloxetine may be as effective but only receive a Level B recommendation based on the quality of the studies.

Source: Bril V, et al. Evidence-based guideline: Treatment of painful diabetic neuropathy. *Neurology* 2011;76:1758-1765.

A GROUP REPRESENTING THE AMERICAN ACADEMY OF NEUrology, the American Association of Neuromuscular and Electrodiagnostic Medicine, and the American Academy of Physical Medicine and Rehabilitation conducted a systematic review of the literature from 1960 to 2008 on studies of therapies for the treatment of painful diabetic neuropathy (PDN). The research question asked was: "What is the efficacy of a given treatment (pharmacologic: anticonvulsants, antidepressants, opioids, others; and nonpharmacologic: electrical stimulation, magnetic field treatment, low-intensity laser treatment, Reiki massage, others) to reduce pain and improve physical function and quality of life (QOL) in patients with painful diabetic neuropathy?" The search identified 2234 citations: 463 were considered potentially relevant and 79 articles were selected for the systematic review.

All studies found that pregabalin relieved pain, but the effect was small relative to placebo, reducing pain by 11%-13% on the 11-point Likert scale in the Class I studies. Other studies showed modest effectiveness for gabapentin, sodium valproate, amitriptyline, venlafaxine, and duloxetine. Pain relievers such as dextromethorphan, morphine sulfate, tramadol, and oxycodone also have evidence of effectiveness. Electrical stimulation, topical capsaicin, and isosorbide dinitrate spray were also effective. Treatments not recommended were oxcarbazepine, lamotrigine, lacosamide, clonidine, pentoxifylline, mexiletine, magnetic field treatment, low-intensity laser therapy, and Reiki therapy.

Recommendations from the study group for anticonvulsants were:

1. If clinically appropriate, pregabalin should be offered for the treatment of PDN (Level A).
2. Gabapentin and sodium valproate should be considered for the treatment of PDN (Level B).
3. There is insufficient evidence to support or refute the use of topiramate for the treatment of PDN (Level U).
4. Oxcarbazepine, lamotrigine, and lacosamide should probably not be considered for the treatment of PDN (Level B).

Recommendations for antidepressants were:

1. Amitriptyline, venlafaxine, and duloxetine should be considered for the treatment of PDN (Level B). Data were insufficient to recommend one of these agents over the others.
2. Venlafaxine may be added to gabapentin for a better response (Level C).
3. There is insufficient evidence to support or refute the use of desipramine, imipramine, fluoxetine, or the combination of nortriptyline and fluphenazine in the treatment of PDN (Level U).

Recommendations for other therapies were:

1. Capsaicin and isosorbide dinitrate spray should be considered for the treatment of PDN (Level B).
2. Clonidine, pentoxifylline, and mexiletine should probably not be considered for the treatment of PDN (Level B).
3. The lidoderm patch may be considered for the treatment of PDN (Level C).
4. There is insufficient evidence to support or refute the usefulness of vitamins and alpha-lipoic acid in the treatment of PDN (Level U).

■ **COMMENTARY**

There is still no magic bullet for treating PDN. Even with the neuropathic pain anticonvulsant pregabalin receiving a Level A recommendation in this systematic review, the effect was modest. The authors of this report were quick to point out that pregabalin may not be superior to other drugs, such as the much less expensive gabapentin, but pregabalin benefitted from superior research studies.

I've been using amitriptyline for PDN for most of my 30-plus year career with some effectiveness. It is good to see that it stands up to Level B evidence. Since PDN is a long-term problem, effective inexpensive therapies are important. I see this systematic review confirming that.

I congratulate the neurology organizations for conducting this robust review of so many therapies. Because of this, we in primary care will not be shooting in the dark as much or making unnecessary referrals for this common and often frustrating condition. ■

Family History and Cancer — The Need to Update

ABSTRACT & COMMENTARY

By Rahul Gupta, MD, MPH, FACP

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

Synopsis: *Family history of cancer changes significantly between the ages of 30 and 50 years. Therefore, it is recommended that family history should be updated*

at least every 5 to 10 years to appropriately inform recommendations for cancer screening.

Source: Ziogas A, et al. Clinically relevant changes in family history of cancer over time. *JAMA* 2011;306:172-178.

Documenting the family history of a patient has been the core element of clinical care long before the practice of evidence-based medicine was even proposed. This is primarily based on the fact that many common diseases have genetic, environmental, and lifestyle predispositions that members of a family may share.[1] Family history has such an important role in the practice of medicine that it may motivate positive lifestyle changes, enhance individual empowerment, and influence clinical interventions. Routine family history taking may also help in determining individuals who might benefit from genetic screening. While the meaning of family history may be interpreted broadly and variably depending on the specialty of the provider obtaining such, the intent remains the same — to identify diseases at the earliest possible stage in order to intervene.

Family history of diseases such as cancer is a traditional and commonly accepted risk assessment tool. However, much more attention is paid to new patients than established patients in a primary care practice. Often, family history in established patients is left untouched until patients themselves provide updates or request preventive screenings. Interestingly, not much literature currently exists addressing how often the family history of an established patient should be updated so as to be able to capture changes pertinent to recommend a change in cancer screenings for the individual patient.

In their study, Ziogas et al attempted to quantify how often clinically significant changes occur in the cancer family history of patients that would result in recommendations for earlier or more intense screening throughout adulthood. The authors examined family history longitudinal data in patients enrolled in the population-based Cancer Genetics Network (CGN) registry. Between 1999 and 2009, they examined baseline and follow-up family history data (self-reported) from 26,933 participants enrolled at 14 academic research centers across the United States. Changes in participants' self-reported family history over two periods were assessed: retrospectively (from birth until enrollment into the CGN) and prospectively (from enrollment to time of last completed follow-up survey). Changes in family history that would render individuals for earlier and more intense screening for colorectal, breast, or prostate cancer based on current guidelines from the American Cancer Society were specifically examined.

Retrospective analysis revealed that at age 30, 2.1% of participants would have met criteria for early colonoscopy screening whereas at age 50, this percentage increased to 7.1% and by age 70, it was at about 11%. Similarly, for breast cancer screening, the percentage of participants who would have met criteria for MRI screening were 7.2% of women at age 30 years, 11.4% at age 50 years, and after age 60 years, it leveled off at about 13%. The retrospective prostate cancer analysis demonstrated similar findings of increasing family history until age 60 years. However, the overall percentage of men who would have met criteria for early PSA screening was much lower at only 0.9%. In the prospective analysis, the numbers of participants who newly met criteria for high-risk screening based on family history per 100 persons followed up for 20 years were two for colorectal cancer, six for breast cancer, and eight for prostate cancer. The rate of change in cancer family history was similar for colorectal and breast cancer between both of the analyses. In this study, utilizing two different types of analyses (retrospective and prospective), both analyses demonstrated that clinically relevant family history changes substantially during early and middle adulthood, particularly for colorectal and breast cancer, in a way that the percentage recommended for high-risk screening increases 1.5- to 3-fold between ages 30 and 50 years. Limited data may have contributed to incongruent results of the prostate cancer analyses. In essence, the researchers found that family history of breast and colorectal cancer becomes increasingly relevant in early adulthood, highlighting the need to obtain a comprehensive family history at this time. Therefore, the authors recommend updating the family history at least every 5-10 years to appropriately inform recommendations for cancer screening.

■ COMMENTARY

Evidence-based guidelines often recommend that persons at elevated risk for certain cancers begin screening at a younger age than the general population and consider more sensitive screening tests. However, such guidelines are often written under the assumption that every individual provides to their physician the most updated and accurate family history at each visit. Clearly, this does not occur. So one asks the question — with so many clinical decision tools and assistance available through as many electronic health records (EHR) systems, who will maintain accurate and current history information that is the basis for screening recommendations? In the current study, the authors found a 5% chance that an individual's colorectal cancer screening recommendation would change between the ages of 30 and 50 years based on new family history and a 4% chance that women would be newly identified candidates for breast MRI. We must be reminded that these are potentially curable cancers if diagnosed early. While considerable research has demonstrated the accuracy of self-reported family history, effective "best practice" tools for collecting and utilizing the family history in a primary care practice have yet

to be validated.[2] However, until we reach a stage where EHR systems cannot only talk with each other but also across patients to capture the most accurate and relevant family history automatically, I agree with the authors that for those between 30-50 years of age, we need to update family history every 5-10 years. ■

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Pharmacology Update

Rivaroxaban Tablets (Xarelto™)

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

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

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

THE FIRST ORALLY ADMINISTERED FACTOR XA INHIBITOR HAS been approved by the FDA for the prevention of deep vein thrombosis (DVT). Rivaroxaban was developed by Bayer and is marketed in this country by Janssen Pharmaceuticals as Xarelto.

Indications

Rivaroxaban is indicated for the prophylaxis of DVT in patients undergoing knee or hip replacement surgery (arthroplasty).[1]

Dosage

The recommended dose is 10 mg taken orally once daily starting at least 6 to 10 hours after surgery once hemostasis has been established.[1] The duration of therapy is 35 days for patients undergoing hip replacement surgery and 12 days for knee replacement surgery. Rivaroxaban may be taken without regard to meals.

Rivaroxaban is supplied as 10 mg film-coated tablets.

Potential Advantages

Rivaroxaban, a once-a-day oral medication, is more effective than subcutaneous enoxaparin at reducing the risk of venous thromboembolic events (VTE).

Potential Disadvantages

Concomitant use of drugs that are both p-glycoprotein and strong CYP3A4 inducers should be avoided.[1] Examples are carbamazepine, phenytoin, rifampin, and St. John's wort. Rivaroxaban should be avoided in patients with moderate or severe hepatic impairment, hepatic disease associated with coagulopathy, or severe renal impairment.[1]

Comments

Rivaroxaban is the first factor Xa inhibitor to be approved. Its efficacy and safety were compared to enoxaparin in 9011 subjects in three comparative trials (RECORD 1, 2, and 3).[1-4] Subjects undergoing total hip or knee arthroplasty were randomized to rivaroxaban 10 mg once daily or enoxparin 40 mg subcutaneously once daily. Rivaroxaban was started 6-10 hours after wound closure and enoxaparin was started 12 hours preoperatively. In RECORD 1, subjects who were scheduled to undergo elective hip arthroplasty received prophylaxis (rivaroxaban or enoxaparin) for 33-34 days.[2] In RECORD 2, subjects undergoing total hip arthroplasty received rivaroxaban for an extended duration (31-39 day) compared to short-term enoxaparin (10-14 days).[3] In RECORD 3, subjects undergoing total knee arthroplasty received rivaroxaban or enoxaparin for 10-14 days.[4] The efficacy endpoint was total VTE events. This composite endpoint includes proximal or distal DVT, non-fatal PE, or all-cause mortality. The main secondary efficacy outcome was major VTE (proximal DVT, nonfatal PE, or VTE-related death). The rates for total VTE events were 1.1% for rivaroxaban vs 3.9% for enoxaparin in RECORD 1, and 2% vs 8.4% for RECORD 2. These represent a significant relative-risk reduction of 71% (95% confidence interval [CI]: 50, 83,) and 76% (95% CI: 59, 86), respectively. A larger relative-risk reduction was observed with major VTE (91% and 87%, respectively). In patients with total knee arthroplasty (n = 2432), the VTE rates were 9.7% for rivaroxaban compared to 18.8% for enoxaparin. This represented a significant relative-risk reduction of 48% (95% CI: 34, 60). Overall, bleeding events were similar between rivaroxaban and enoxaparin and were not statistically different (5.8% vs 5.6%). The rate of severe bleeding events was also similar (0.3% vs 0.2%). A systematic review that included the above studies and three other studies showed a risk ratio of 0.38 (95% CI, 0.25, 0.59).[5] Rivaroxaban (10 mg daily for 10 -14 days) was also found to be more effective than enoxaparin (30 mg twice daily for 10-14 days) for the prophylaxis of VTE after total knee arthroplasty (event rate of 6.9% vs 10.1%).[6] The results of a phase III trial of rivaroxaban compared warfarin in the prevention

of stroke and non-CNS embolism in patients with atrial fibrillation were presented last November.[7] This could lead to FDA approval for this indication in the near future.

Clinical Implications

Rivaroxaban, an Xa inhibitor, appears to be more effective than enoxaparin for the prophylaxis of VTE in patients undergoing total hip or knee arthroplasty. Oral administration in postopertative patients represents a major advantage over previous therapies. Rivaroxaban represents an important advance and may eventually be approved for other indications as well, including stroke and non-CNS embolism prophylaxis for patients with atrial fibrillation. ■

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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

9. In a multicenter study, vitamin D deficiency or insufficiency was present in patients suffering an acute myocardial infarction:
   a. in only a small percentage of the patients.
   b. in a moderate number of the patients.
   c. in virtually all of the patients.

10. Which therapeutic agent received Level A evidence based on a systemic review?
   a. Gabapentin
   b. Pregabalin
   c. Amitriptyline
   d. Tramadol

11. By how much would an individual's colorectal cancer screening recommendation change between the ages of 30 and 50 years based on new family history?
   a. 0%
   b. 5%
   c. 10%
   d. 20%
   e. 50%

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

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

Antihypertensive Medication Nonadherence and Blood Pressure

Source: Rose AJ, et al. Effects of daily adherence to antihypertensive medication on blood pressure control. *J Clin Hypertens* 2011;6:416-421.

It comes as no surprise that when patients do not take their blood pressure (BP) medication, a lapse in BP control is anticipated. On the other hand, when a patient presents with an elevated BP and acknowledges omitted doses, it is difficult to be sure whether the observed elevation in BP is solely due to recent omissions, an underlying worsening of BP (requiring an augmentation rather than just simple restoration of treatment), rebound BP elevation, or some combination of these elements. To gain a more concrete insight into the anticipated impact of omitted BP medication in a typical patient population, Rose et al reviewed data from a population (n = 869) enrolled in a trial investigating the effects of physician communication on BP control. A component of the study design was utilization of medication bottles with memory caps that recorded timing and frequency of opening, providing a detailed view of medication administration.

When comparing BP after a 7-day period of poor adherence (< 60% of prescribed medication administered) to a prior period of excellent adherence, BP was 12/7 mmHg higher immediately following the week of poor adherence.

Clinical inertia — failure to intensify treatment despite suboptimal goal attainment — is sometimes innocently propagated by clinician uncertainty about whether uncontrolled BP should simply be attributed to missed doses or needs treatment augmentation. The authors suggest that clinicians consider a maximum BP excursion of 15/8 mmHg as potentially likely due to poor medication adherence, and that when BP elevation is greater than this amount, consider augmentation of antihypertensive treatment rather than simply encouraging better adherence to the existing regimen. ■

PDE5 Inhibition and Cognitive Function

Source: Shim YS, et al. Effects of repeated dosing with Udenafil (Zydena) on cognition, somatization and erection in patients with erectile dysfunction: A pilot study. *Int J Impot Res* 2011;23:109-114.

The therapeutic realm of PDE5 inhibitors has expanded to include not only erectile dysfunction (ED) but also pulmonary hypertension. Animal studies have identified PDE5 activity in the brain, which can be impacted by currently available PDE5 inhibitors since they readily cross the blood-brain barrier. In the animal CNS, increased cyclic GMP (a pharmacodynamic effect of PDE5 inhibition) is seen in pathways associated with memory; studies have confirmed enhanced cognition in animals with impaired cognition related to diabetes, anticholinergic medications, and hyperammonemia who are treated with PDE5 inhibitors.

Udenafil is a PDE5 inhibitor not available in the United States but already in use in other countries (e.g., Korea, Russia) for treatment of ED. Shim et al undertook a trial of udenafil in men with ED but without known cognitive dysfunction (n = 30). Subjects underwent a battery of tests of cognitive function at baseline and 8 weeks later. Testing metrics included measures of general cognitive function, verbal learning for episodic memory, and frontal executive function.

Several tests of cognitive function showed statistically significant improvement. Cognitive function improvement was greater in men whose sexual function scores improved the most. The authors suggest further exploration of the effects of PDE5 inhibition on cerebral flow to gain greater understanding of the favorable cognitive effects they have demonstrated. ■

Can Appendicitis be Cured with Antibiotics Alone?

Source: Vons C, et al. Amoxicillin plus clavulanic acid versus appendicectomy for treatment of acute uncomplicated appendicitis: An open-label, non-inferiority, randomised controlled trial. *Lancet* 2011; 377:1573-1579.

Sometimes, acute appendicitis (AAP) just goes away. We know this because of abdominal explorations that disclose evidence of chronic appendicitis, indicative of one or more prior episodes. Four randomized trials support the relevance of antibiotic treatment for AAP, but definitive conclusions about the appropriate role of antibiotics in AAP treatment have been limited by aspects of previous study design.

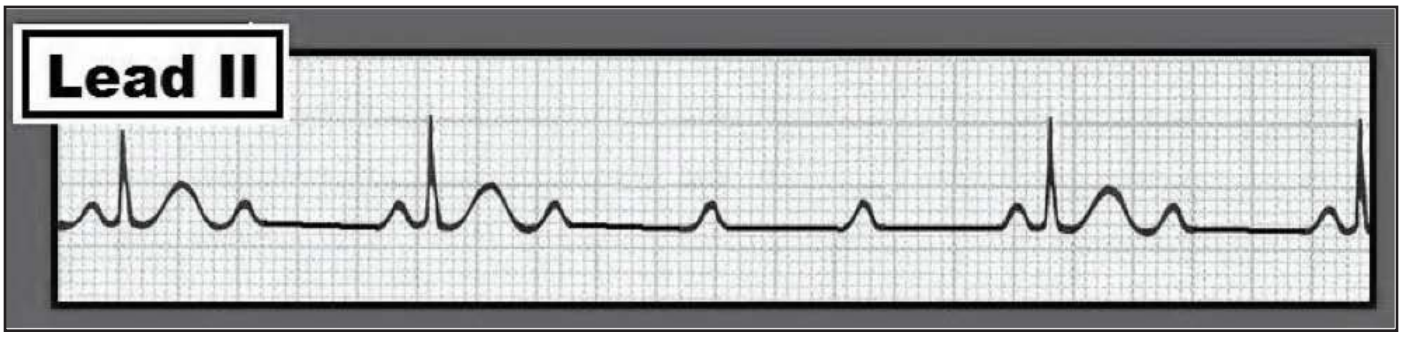
Vons et al performed a controlled trial of adult patients with CT-confirmed uncomplicated AAP who were randomized to antibiotics (amoxicillin/clavulanic acid 3-4 g/d) or surgery. Although one group was assigned to surgery alone, the surgical group also actually received a single parenteral 2 g dose of amoxicillin/clavulanic acid at induction of anesthesia; additionally, if complicated appendicitis was discovered at surgery (i.e., the appendicitis had progressed or was misdiagnosed by CT), antibiotics were subsequently administered even in the surgery group.

Peritonitis within 30 days of intervention — the primary endpoint of the trial — occurred more often in the antibiotic group (8% vs 2%), hence the noninferiority of antibiotic treatment was NOT confirmed. If future tools can do a better job of identifying those who truly have uncomplicated appendicitis, antibiotics may prove to be a more valuable first-line treatment. ■

Not Just 2:1 AV Block — Mobitz II?

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.



Scenario: Interpret the rhythm strip shown above. Does it represent 2nd degree AV block, Mobitz Type II? Can you be sure?

Interpretation: The ventricular rhythm in the Figure is slow and irregular. Nevertheless — the QRS complex is narrow, and the atrial rate is regular at ~115/minute. The P waves immediately preceding each QRS complex manifest a fixed (and normal) PR interval. Thus, these P waves *are* conducting. This means that the rhythm is *not* complete AV block. Since there are many non-conducted P waves on the tracing — the rhythm must represent some form of high-grade 2nd degree AV block.

As opposed to last month's ECG Review (which showed the regular 2-to-1 AV conduction form of 2nd degree AV block) — the tracing here manifests an irregular and slower ventricular rate. There are features of *both* Mobitz I (AV Wenckebach) and Mobitz II on this tracing. In favor of Mobitz II is the low conduction ratio and high grade of AV block. No less than three P waves in a row are non-conducted in the middle of the tracing. However, the QRS complex is narrow — which is highly unusual for Mobitz II.

Clinically — the importance of distinguishing AV Wenckebach (Mobitz I) from Mobitz II relates to the much better prognosis of Mobitz I, a generally better response to treatment with atropine, and a much lower likelihood of needing a pacemaker. In this particular case it is impossible to be certain which form of 2nd degree AV block is present from this tracing alone, since one *never* sees two consecutively conducted P waves. Thus, one *cannot* tell if the PR interval is progressively increasing until the point of non-conduction. Although unusual for Mobitz I — more than one P wave in a row may be blocked on occasion with this conduction disturbance. That said — from a practical treatment perspective — distinguishing between Mobitz I and Mobitz II appears to be less important since a pacemaker may be needed in either case if the high-grade degree of AV block does not improve. ■

In Future Issues:

Measuring Blood Pressure for Decision Making and Quality Reporting: Where and How Many Measures?

Constipation and Risk of Cardiovascular Disease among Postmenopausal Women

Clinical Characteristics and Cardiovascular Magnetic Resonance Findings in Stress Cardiomyopathy