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For Women with Acute MI, Atypical Presentations Are Typical

ABSTRACT & COMMENTARY

Source: McSweeney JC, et al. Women's early warning symptoms of acute myocardial infarction. *Circulation* 2003;108:2619-2623.

THE AUTHORS OF THIS STUDY SOUGHT TO CHARACTERIZE SYMPtoms women experience in association with an acute myocardial infarction (MI). Patients were identified using hospital discharge codes and were contacted by phone 4-6 months after their cardiac events. All patients underwent a cognitive screening questionnaire, followed by administration of a survey of MI-associated symptoms using a well-validated survey instrument. The patients were questioned about both acute symptoms at the time of the MI and prodromal symptoms, defined as new or different symptoms occurring intermittently before the MI and resolving after the event.

The authors screened 712 women during a three-year period and obtained complete data on 515 subjects. The mean age was 66 years and the vast majority of patients were white. When asked about acute symptoms, only 57% of women experienced any type of chest discomfort at the time of their MI. Other common acute symptoms were shortness of breath (58%), weakness (55%), unusual fatigue (43%), cold sweat (39%), and dizziness (39%). Seventy-eight percent reported prodromal symptoms, but only 30% reported any type of prodromal chest discomfort. The most frequent prodromal symptom was unusual fatigue (71%), while sleep disturbances, shortness of breath, indigestion, and anxiety each were more common than chest discomfort. Any prodromal fatigue or sleep disturbance usually was described as severe. The authors conclude that prodromal symptoms of MI are very common in women and may be important, yet easily overlooked, predictors of MI.

COMMENTARY BY DAVID J. KARRAS, MD, FAAEM, FACEP

Any physician can recite the "typical" symptoms of MI. What many fail to realize, however, is these textbook scenarios were derived from studies of white, middle-aged males. MI presentations of women and ethnic groups remain poorly described. There is

mounting evidence that among women with MI, atypical presentations are more characteristic than are typical presentations. The authors' prior studies found that women surveyed immediately after an MI were likely to miss important prodromal symptoms. This recall bias actually was minimized by delaying the interview until the patient had time to reflect on the symptoms she experienced before the event and determine which symptoms were, in retrospect, transient.

There are a few take-home messages from this study. Foremost is the excellent description of MI-associated symptoms in women, and the finding that only a little more than half of women recall any sort of chest discomfort with their acute MI. Physicians, therefore, should set a low threshold for obtaining an electrocardiogram (ECG) in a woman reporting acute diffuse weakness, profound fatigue, or dizziness. The second point is that prodromal symptoms—notably unusual fatigue, insomnia, indigestion, and anxiety—are common in women during the month prior to their MI. These symptoms may be analogous to "anginal equivalents," and women should be questioned about these nonspecific findings during an emergency department evaluation for potential acute coronary syndrome. ❖

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Do Steroids Provide the Best Treatment for Otitis Externa?

ABSTRACT & COMMENTARY

Source: Van Balen FAM, et al. Clinical efficacy of 3 common treatments in acute otitis externa in primary care: Randomized controlled trial. *BMJ* 2003;327:1.

In this European study, researchers compared three different topical treatment regimens for patients with acute otitis externa: acetic acid alone, acetic acid with steroids (triamcinolone 0.1%), or antibiotic with steroids (neomycin/polymixin with dexamethasone). The investigators compared recovery days, cure, and recurrence rates during the 42 days following therapy.

Overall, 213 patients meeting explicit criteria (external auditory canal redness or swelling or debris associated with typical symptoms including pain, itch, otorrhea, hearing loss, or stuffy feeling fewer than three weeks in duration) were randomized to one of the three treatment regimens in a double-blind fashion. Patients were treated with each agent on a three-times-per-day basis from seven to 21 days based on physician evaluation for cure at seven, 14, and 21 days. Patients maintained daily logs of their symptoms and also were followed up at 42 days by telephone to assess for any recurrences.

Median duration of symptoms was shorter with steroids (7.0 days for acetic acid plus steroid and 6.0 days for antibiotic plus steroid) compared to acetic acid alone (8.0 days). More important, cure rates were markedly improved with the addition of steroids. Cure rates for acetic acid only, acetic acid plus steroid, and antibiotic plus steroid were 29.2%, 47.5%, and 42.5%, respectively, at seven days; 56.9%, 75.4%, and 82.2% at 14 days; and 61.5%, 88.5%, and 86.3% at 21 days. Recurrence rates at 42 days also were higher with acetic acid only (44.7%) compared to acetic acid plus steroid (26.3%) and antibiotic plus steroid (20.6%). In addition, there were no significant differences in adverse side effects between the three different regimens.

Based on their findings, the authors conclude that ear drops containing corticosteroids are more effective in the

treatment of acute otitis externa, particularly when compared with acetic acid alone. In addition, the authors conclude that corticosteroids with either acetic acid or antibiotics are equally effective in terms of patient recovery, symptom resolution, cure rates, and recurrences.

COMMENTARY BY THEODORE C. CHAN, MD, FACEP

Patients with acute otitis externa commonly present to the emergency department. Standard treatment options have focused on rebuilding the natural acid-lipid layer protecting the external auditory canal, reducing soft tissue inflammation, and eradicating the infection, most commonly caused by pseudomonas species and staphylococcal bacteria. A myriad of treatments have included home remedies such as acetic acid (vinegar or commercially available VoSol) and isopropyl alcohol (rubbing alcohol), as well as more sophisticated antibiotic and steroid combinations similar to the antibiotic-steroid regimen arm in this study (such as Cortisporin Otic).

This study is a well-conducted, double-blind, randomized large clinical trial demonstrating that topical acetic acid with corticosteroids or antibiotics with steroids are superior to acetic acid alone in the treatment of acute otitis externa. However, while the investigators found the acetic acid plus steroid combination had similar efficacy to the antibiotic plus steroid combination, they did not compare these regimens against topical antibiotics alone.

Moreover, it is impressive to note that recurrence of symptoms within 42 days of therapy was common in all three treatment regimens. It is not clear what evaluation or treatment occurred for patients with recurrent symptoms. ❖

Valsartan — No Better Than Captopril in MI Patients Complicated by Heart Failure

ABSTRACT & COMMENTARY

Source: Pfeffer MA, et al. Valsartan, captopril, or both in myocardial infarction complicated by heart failure, left ventricular dysfunction, or both. *N Engl J Med* 2003;349: 1893-1906.

Studies demonstrate that angiotensin-converting-enzyme (ACE) inhibitors such as captopril reduce mortality and cardiovascular morbidity for patients with myocardial infarction (MI) complicated by left ventricular systolic dysfunction, heart failure, or both. The authors surmised that since angiotensin II can be generated despite ACE inhibition, further efficacy can be obtained if receptor antagonists are combined with ACE inhibitors. The study was a randomized, double-blind trial conducted at 931 centers in 24 countries. Patients were considered for the study if they had an MI and either radiologic signs of heart failure or evidence of left ventricular systolic dysfunction (an ejection fraction ≤ 0.35 on echocardiography or contrast angiography and ≤ 0.40 on radionuclide ventriculography). Patients were excluded if they had a systolic blood pressure lower than 100 mmHg or a serum creatinine concentration of more than 2.5 mg/dL. Patients randomly were assigned, from 12 hours to 10 days after acute MI, to valsartan (4909 patients), valsartan plus captopril (4885 patients), or captopril (4909 patients). Therapy was begun with either 20 mg of valsartan, 20 mg of valsartan plus 6.25 mg of captopril, or 6.25 mg of captopril (increased gradually). The primary end point was death from any cause. During a median follow-up of two years, slightly fewer than 1000 patients died in each arm of the trial. Furthermore, no difference was seen in death from cardiovascular events, recurrent MI, or hospitalization for heart failure. The valsartan-and-captopril group had the most drug-related adverse events. Hypotension and renal dysfunction were more common in the valsartan only group, and cough, rash, and taste disturbance were more common in the captopril group. The authors conclude that valsartan is as effective as captopril in preventing death after myocardial infarction. Combining valsartan with captopril increased the rate of adverse events without improving survival.

COMMENTARY BY RICHARD J. HAMILTON, MD, FAAEM, ABMT

This study, designed to determine if valsartan offered an advantage over captopril in patients with heart failure after MI, found no difference. In addition, combination therapy only added to the adverse effect profile. Interestingly, that adverse effect profile may have been one of the more useful findings in this study. Patients who received captopril had less hypotension and renal dysfunction, although the annoying cough, rash, and taste disturbance side effects were slightly more common. Angioedema occurred with equal frequency in both groups. Couple this information with the knowledge that the retail price of 90 days of therapy with valsartan (320 mg QD) is $240, while 90 days of therapy with generic captopril (50 mg TID) is $40, according to drugstore.com. Perhaps this study ought

to make us suspect that valsartan has little to offer most MI/heart failure patients over an ACE inhibitor. The exception may be for the 5% or fewer with the side effect of cough. ❖

Glucose, Insulin, and Potassium Infusion in Acute MI: Back to the Future?

ABSTRACT & COMMENTARY

Source: vanderHorst ICC, et al. The Zwolle Infarct Study Group. Glucose-insulin-potassium infusion in patients treated with primary angioplasty for acute myocardial infarction: The glucose-insulin-potassium study: A randomized trial. *J Am Coll Cardiol* 2003;42:784.

HIGH-DOSE GLUCOSE-INSULIN-POTASSIUM (GIK) INFUsion for patients presenting with acute myocardial infarction (AMI) has been advocated since the 1960s. Suggested benefits from GIK include improving cardiac energy metabolism by increased glucose utilization and decreased free fatty acid uptake by myocytes, as well as stabilization of cell membranes in ischemic myocardial tissue.

In this controlled trial from Europe, investigators randomized 940 AMI patients (symptom duration greater than 30 minutes, onset within 24 hours of presentation, and electrocardiographic findings of AMI including ST segment elevation or new left bundle-branch block) to either GIK (476 patients) or no infusion (464 patients) prior to undergoing reperfusion via percutaneous transluminal coronary angioplasty. All patients received standard therapy including nitroglycerin, heparin, and aspirin. The GIK group received a continuous infusion of 80 mmol of potassium chloride in 500 mL of 20% glucose solution at a rate of 3 mL/kg/hr, as well as 50 U of insulin over 8-12 hours (to maintain serum glucose level of 70-110 mEq/dL), initiated as soon as possible after presentation.

Overall, the investigators report that there was no difference between the GIK and no infusion groups in terms of 30-day mortality (4.8% vs 5.8% respectively, $p = 0.50$), as well as no difference in composite 30-day endpoint (death, reinfarction, or revascularization) (8.0% vs 9.9%). However, when patients were stratified by evidence of heart failure on presentation, patients with no signs of heart failure (90.1%, or 856 patients who were Killip Class I) had significantly improved 30-day mortality with GIK (1.2% vs. 4.2%, respectively, $p = 0.01$), as well as an improved composite endpoint (4.2% vs 8.4%, respectively). In patients who presented with signs of heart failure (8.9% or 84 patients who had Killip Class of at least 2), more patients died from heart failure in the GIK group compared to no infusion (30.4% vs 20.6%, respectively).

The authors conclude that while their study found no overall mortality benefit to GIK infusion in AMI, there was a significant mortality benefit in the large subgroup of AMI patients who did not have evidence of heart failure on presentation. These findings may indicate that GIK does have beneficial effects on cellular metabolism in ischemic cardiac cells, but this benefit may be outweighed by the large GIK volume infusion in patients already presenting with evidence of volume overload, cardiac decompensation, and heart failure.

COMMENTARY BY THEODORE C. CHAN, MD, FACEP

This is the largest prospective, randomized trial studying the utility of GIK infusion in the setting of AMI, as well one of the first studies to investigate the role of GIK in combination with emergent angioplasty. The absolute 3% improvement in mortality in patients without evidence of heart failure on presentation is quite remarkable given how little this therapy would cost. Most AMI patients present without heart failure (90%), and this mortality benefit would translate to 30,000 lives saved per year in this country (based on an annual rate of AMI of 1 million).

A few points are of note regarding this study. First, the overall mortality rate (5.3%) for both the GIK and no infusion groups is remarkably low, likely a result of improvements in emergent care and reperfusion for AMI patients in general. Second, this study was performed at a single site which demonstrated excellent door-to-balloon times (fewer than 50 minutes) which undoubtedly led to improved outcomes in both groups. Moreover, it suggests the benefit of the 8-12 hour GIK may occur in preventing reperfusion injury after patency is established, as well as improved cellular metabolism during the ischemic period. Third, a relative large volume of fluid was infused with this particular protocol (2 liters over 8 hours for an 80 kg patient). Similar benefit may be seen with smaller infusions with higher concentrations of GIK that pose less risk for patients with volume overload. Finally, while the results of this study are promising, a larger, multi-center study is needed to determine if GIK truly has particular benefit for subgroups of AMI patients. ❖

Special Feature

Sports Concussions: Grading Severity and Advising on Return to Play

By Andrew D. Perron, MD, FACEP, and Clifford R. Peck, MD

Concussion, or mild traumatic brain injury (MTBI), is a common occurrence in contact and collision sports. The medical literature estimates between 200,000 and 300,000 concussions occur per year in the United States. However, the true incidence is almost certainly higher, since many concussions likely go unnoticed by coaches, trainers, families, and players. Further, the concussion may be recognized only by the individual player, who potentially is motivated to not report the injury out of fear that he or she may be removed from play. For the clinician, the area of sports concussions and MTBI can be confusing due to the relative paucity of scientific evidence to support the clinical decision-making process in the emergency department (ED) and beyond. Good scientific research in this area has been hampered by an inconsistent definition of concussion, widely divergent injury mechanisms, poor means of measuring cognitive deficits, and inconsistent return to play guidelines.

Separating Myth from Fact

A common myth surrounding concussion is that diagnosis requires a transient loss of consciousness (LOC) or amnesia.[1] This is unsettling when recent studies argue that greater than 75% of concussions do not involve LOC.[2] Experts in the field have broadened the definition to include any posttraumatic alteration in mental status that may or may not involve LOC. Some have even called for the term MTBI to be used in place of concussion, as the definition of concussion has become so muddied in the medical literature.

Concussion can occur in any sport, but those with high-velocity collisions between players (e.g., football, hockey, and boxing) have a higher incidence. Many concussion studies have focused on high school, college, and professional football players, which carries an estimated annual incidence of concussion of 10%. A recent study has confirmed what many experts in the field have postulated in the past: namely, that concussion begets concussion. This study prospectively followed a cohort of NCAA football players and reports a strong association between previous concussions and the likelihood of subsequent concussion. For example, a collegiate football player with a history of three or more concussions (lifetime) was three times more likely to have a subsequent concussion, as compared to players without previous concussion.[2] With experts in the field unsure about the cumulative effect of repetitive concussion and the possibility of "the second impact syndrome" (where the individual not yet recovered from and initial mild traumatic brain injury sustains a blow to the head that results in swift, uncontrollable increase in intracranial pressure due to diffuse brain swelling, resulting in death or a permanent vegetative state), researchers have focused on strategies to diagnosis and limit exposure to concussion.[3,4]

Although confusion and amnesia are the cardinal features of concussion, they can present in many ways. Athletes may lose consciousness, appear ataxic, or show memory deficits, but more commonly they will exhibit subtle changes such as a vacant stare, slowed speech, or emotional lability. The process for evaluating the athlete for concussion should be systematic. Any worrisome signs or symptoms should be identified immediately, and evidence of increased intracranial pressure should be treated immediately.[5] In 1997, McCrea et al established a Standardized Assessment of Concussion (SAC) in a group of varsity high school football players. Trainers on the sideline administered a series of questions that tested orientation, immediate memory, concentration, and delayed recall. Compared to baseline values recorded in a control group, the total score (out of 30) obtained by concussive players immediately following injury was significantly lower than that of the total nonconcussive sample.[6]

Grading Concussion Severity

Over the years, multiple attempts have been made to develop criteria to grade concussion in terms of severity and then offer return to play (RTP) guidelines. Currently, there are 23 separate concussion-grading schemes, which has largely served to confuse coaches, trainers, and physicians. The American Academy of Neurology (AAN) published recommendations in 1997 after a thorough literature search and input from a multi-disciplinary panel.[7] Acccording to the AAN recommendations, a Grade 1 concussion involves transient confusion, but no LOC. The concussion symptoms or mental status abnormalities resolve in fewer than 15 minutes. Grade 1 concussion is the most common, yet the most difficult to recognize. Grade 2 concussion does not involve LOC, but symptoms or mental status abnormalities last more than 15 minutes. Any persistent Grade 2 symptoms (greater than one hour) warrant medical observation. Grade 3 concussion involves LOC, either brief (seconds) or prolonged (minutes). Athletes with Grade 3 concussion should be evaluated by a physician.

The AAN also offers recommendations for return to play, but stresses that these are options. No coach, trainer, or physician will be faulted for more conservative measures. After a Grade 1 concussion, the player should be assessed immediately and at five-minute intervals for mental status abnormalities and post-concussive symptoms both at rest and with exertion. The athlete may return to play (RTP) if mental status abnormalities and post-concussive symptoms clear within 15 minutes. If the player experiences a second Grade 1 concussion that day, he should be removed from play and may RTP if asymptomatic for one week both at rest and with exertion. After a Grade 2 concussion, the athlete should be removed from play and disallowed from returning to the contest. He should be re-examined frequently for signs of intracranial pathology. The athlete may RTP if asymptomatic for one week both at rest and with exertion. Following a second Grade 2 concussion that season, the athlete may RTP if asymptomatic for two weeks both at rest and with exertion.

Grade 3 concussion with continued LOC warrants immediate medical attention. In this case, the athlete should be transported by ambulance to the nearest ED for neurological evaluation and possible neuroimaging. If findings are normal at the time of the initial evaluation, the patient may be sent home with family and explicit instructions for care. After a brief LOC (seconds) Grade 3 concussion, the athlete should be removed from play and may RTP if asymptomatic for one week both at rest and with exertion. After prolonged LOC (minutes) Grade 3 concussion, the athlete should be removed from play and may RTP if asymptomatic for two weeks both at rest and with exertion. Following a second Grade 3 concussion, the athlete should be withheld from play for a minimum of one month. Termination of the season should be considered.

The injured athlete with prolonged LOC or deteriorating symptoms should be transported by ambulance for ED evaluation. These patients should undergo appropriate trauma evaluation, including the ABCs (airway, breathing, circulation) of life support and cervical spine immobilization, if appropriate. After complete physical evaluation with particular attention to the neurological examination, the emergency physician must decide whether the patient requires neuroimaging or specialty consultation. Not all patients who suffer a concussion require computed tomography (CT) of the head.[5] However, since data from prospective clinical trials do not exist, the emergency physician must decide on a patient-to-patient basis. Support for head CT includes LOC of more than five minutes, persistent amnesia, focal neurological signs, and concern for a depressed skull fracture.

Summary

There is no single way to manage the concussed athlete, but trends are evident in the literature. The concussed athlete should be removed from play and immediately examined. If prolonged LOC or deteriorating status is evident, the athlete should be examined immediately by a physician. The post-concussed athlete should not RTP until completely asymptomatic at rest and with exertion. Multiple concussions may have a cumulative effect on the athlete and warrant further medical evaluation. Every sport carries a risk of injury. The goal is to foster solid competition while ensuring safety to its participants. In this respect, it is the physician's role to provide objective assessment of the injured athlete and guidance about the advisability of safe return to competition.[8,9] ❖

(Dr. Peck is an emergency medicine resident at Maine Medical Center, Portland.)

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Physician CME Questions

14. The European study comparing treatment regimens for acute otitis externa concluded that:

a. steroids improved the efficacy of treatment when combined with antibiotics or acetic acid.
b. antibiotics are unnecessary and potentially harmful to the ear.
c. steroids improved the efficacy of treatment when combined with boric acid.
d. antibiotics improved the efficacy of treatment when combined

with isopropyl alcohol.

e. steroids improved the efficacy of treatment when combined with gentamycin.

15. Which of the following statements is true regarding women who develop myocardial infarction?

a. Fewer than 10% report having chest pain during the acute event.
b. Severe fatigue during the preceding month commonly is reported.
c. Chest heaviness in the preceding month is a common symptom.
d. Dyspnea during the acute event is an uncommon symptom.

16. Concussion:

a. cannot occur without loss of consciousness.
b. usually occurs only with repetitive impact to the head.
c. begets concussion.
d. usually is associated with nausea and nystagmus.

17. The cardinal features of concussion are:

a. confusion and amnesia.
b. amnesia and nausea.
c. gaze deviation and confusion.
d. nausea and vomiting in the absence of headache.

18. Which regimen proved to be best in terms of long-term mortality reduction for patients with MI complicated by heart failure?

a. Valsartan alone
b. Captopril alone
c. Valsartan alone and captopril alone were equivalently efficacious.

19. The study by the European Zwolle Acute MI Group on GIK infusions in acute MI patients reported that:

a. GIK infusion improved mortality in acute MI patients with heart failure.
b. GIK infusion worsened mortality in acute MI patients overall.
c. GIK infusion improved mortality in acute MI patients without heart failure.
d. low-volume GIK was no different than high-volume GIK in terms of outcome for acute MI patients.

Answer Key

14. a **16. c** **18. c**
15. b **17. a** **19. c**

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, *you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

CME Objectives

To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

Mobitz II in a Patient on Digoxin

By Ken Grauer, MD

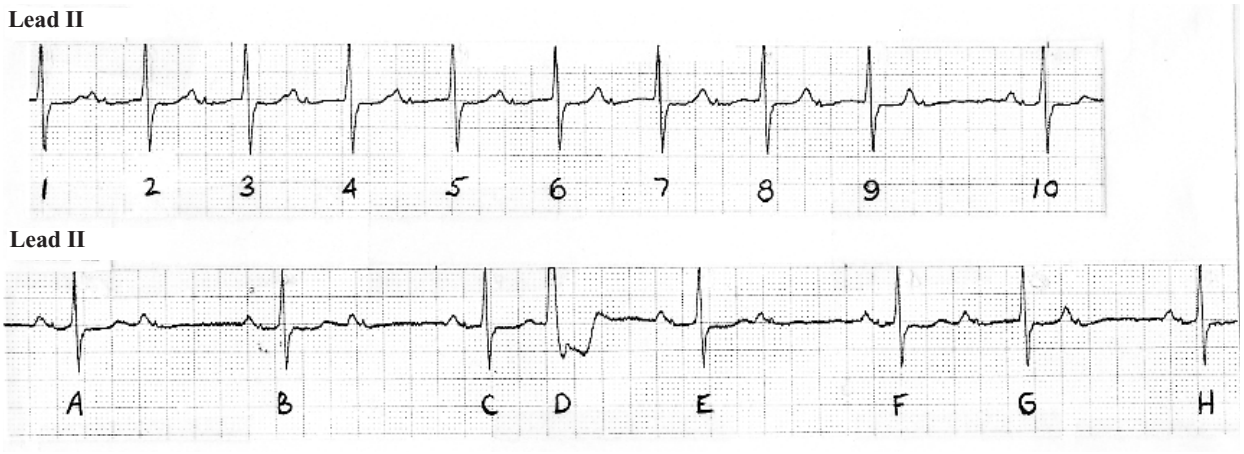

Figure. Nonsequential lead II rhythm strips obtained from a 71-year-old man with heart failure.

Clinical Scenario: The nonsequential rhythm strips shown in the Figure were obtained from a 71-year-old man with a history of congestive cardiomyopathy and renal insufficiency. The patient was admitted for an exacerbation of heart failure. Digoxin was among the many medications he was taking. Assessment of the bottom rhythm strip was 2:1 AV block, Mobitz Type II. Do you agree?

Interpretation: Use of calipers will greatly facilitate interpretation of the two tracings shown in the Figure. Except for the very first T wave in the top tracing, virtual superposition of P waves on top of T waves masks atrial activity until the last beat (beat #10) in the top tracing. Inspection of the T wave of this beat #10 reveals the true shape of what T waves would look like without a superimposed P wave. Knowing this allows us to "walk out" with calipers—an essentially regular atrial rhythm (at a rate of 75-80/minute) throughout both rhythm strips.

With the exception of premature beat D in the lower tracing (which is a premature ventricular contraction [PVC]), the QRS complex is narrow in both rhythm strips. Upright P waves are present in these lead II rhythm strips and appear to be conducting, albeit with a PR interval that is not always constant. Second degree (2°) AV block is present because a number of P waves on these tracings do not conduct. Concern about the presence of 2° AV block, Mobitz Type II is raised because of the short run of 2:1 AV block with a constant PR interval (beats A through F in the bottom tracing). However, Mobitz II 2° AV block is *not* present. Instead, one can definitively diagnose Mobitz I (2° AV block of the Wenckebach type). Distinction between these two forms of AV block is important clinically because of the generally much more serious prognostic implications of Mobitz II, which in the acute setting is usually indication for immediate pacemaker placement.

Statistically, Mobitz Type I 2° AV block is a much more common conduction disturbance than Mobitz Type II. Because of the more proximal level of this conduction defect (which is usually at the level of the AV node), the QRS complex is usually narrow with Mobitz I AV block (as it is here). In contrast, the QRS complex with Mobitz II is usually (though not always) wide. Definitive diagnosis of Mobitz II AV block requires evidence of failed conduction (dropped beats) that occurs in association with the presence of *consecutively* conducted complexes that manifest a constant PR interval. This is why the short run of 2° AV block with 2:1 AV conduction seen in the lower tracing could represent *either* Mobitz I or Mobitz II (since you never see two conducted beats in a row, you cannot tell if the PR interval is increasing or not). That said, there is other clear evidence of Mobitz I 2° AV block on these two tracings. This virtually confirms Mobitz I as the true diagnosis because of the rarity of seeing rapid alternation between the Mobitz I and Mobitz II types of 2° AV block. Digoxin toxicity should be strongly suspected in this older patient with renal insufficiency who manifests Wenckebach type of 2° AV block. ❖

In Future Issues:

Differential Diagnosis of ST Segment Depression on the ECG