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Does Emergency Department Trauma Care Affect Evaluation of Chest Pain Patients?

ABSTRACT & COMMENTARY

Source: Boutros F, Redelmeier DA. Effects of trauma cases on the care of patients who have chest pain in an emergency department. *J Trauma* 2000;48:649-653.

THIS RETROSPECTIVE STUDY FROM TORONTO EXAMINED WHETHER trauma cases alter the length of stay and quality of care of emergency department (ED) patients with chest pain. The authors identified all trauma cases (Injury Severity Score > 16) presenting over a two-year period. The ED logs were screened to identify a patient entering the ED immediately after a trauma patient had arrived. If the chief complaint was chest pain, the patient was included as a case patient. For each case patient, a control patient was selected whose chief complaint also was chest pain, and who arrived in the ED during the same shift on a preceding day when no concurrent trauma case was in the ED. Seventy case and an equal number of control patients were identified. This sample had an 80% power to detect a one-third or greater difference in the average length of stay for case patients relative to controls.

Patient characteristics of the two groups were similar, including mean age and gender. Almost all of the trauma patients and their matched controls arrived within two hours of each other. The number of patients registered in the hour preceding arrival and the number of total patients in the ED at the time of arrival did not differ between the two groups. There was no significant difference between the two groups in the number of cardiac risk factors or in the number of patients ultimately diagnosed with a cardiac cause of chest pain.

Case patients spent an average of 81 minutes longer in the ED than controls (297 vs 216, $P < 0.01$). No significant interactions were noted during sub-analysis based on age, gender, or final disposition. There were no statistically significant differences in door-to-nurse, door-to-ECG, door-to-IV, or door-to-first medication times.

Using the American College of Emergency Physicians (ACEP) Chest Pain Guidelines, a score between zero and 100 can be calculated to yield a summary quality assurance index for each patient (higher numbers equal greater adherence to guidelines). A score was calculated for each case and control patient by reviewing the physician chart and nursing notes. On average, case patients had lower scores than control patients (75.6 vs 84.4, P = 0.027). Analyzing only those patients who ultimately were diagnosed with cardiac chest pain yielded a greater difference (60.3 vs 85.1, P = 0.002). There were non-significant trends toward an increased number of failures to administer aspirin, treat ongoing pain, and provide adequate instructions regarding treatment and need to return.

COMMENT BY JACOB W. UFBERG, MD

This is a well-done study with several valid conclusions. The authors show conclusively that major trauma influences the length of stay and the quality of care (as per the ACEP guidelines) of ED patients with chest pain. However, we must be careful not to jump to any conclusions that lack supporting data. The study shows an increased overall length of stay for case patients. However, our major concerns in the initial management of the patient with chest pain are the door-to-ECG and door-to-medication times, which did not differ significantly between the two groups. Thrombolytics were used only once, and no patients required urgent percutaneous transluminal coronary angioplasty (PTCA) during this study, so we cannot make any conclusions about possible delays for these treatment modalities.

According to the ACEP guidelines, quality of care suffered for case patients. What is unclear, however, is how this statistically significant difference in scores applies to clinical outcomes. It is still unclear whether the ACEP guidelines are a clinically significant marker of quality of care. As with most studies of patients with chest pain, the majority of patients did not have acute coronary ischemia. The low percentage of patients with actual coronary syndromes, in addition to a relatively small sample size, renders this study incapable of assessing the clinical difference (i.e., difference in short- and long-term morbidity and mortality) between the two groups.

Despite these limitations, this study makes an important point. With chest pain patients spending an average of 81 minutes longer in the ED due to concurrent trauma cases, it is likely that other patients in the ED with less priority are experiencing even longer delays. This study took place in a major trauma center with a team of physicians dedicated only to trauma. Low-volume EDs may find greater delays in the care of other patients, as the smaller number of physicians and nurses may become "stuck" with the trauma patient. Emergency physicians working with trauma patients must "multi-task" in order to maintain patient flow and quality of care. ❖

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More on the ECG Diagnosis of Acute MI in the Setting of Left Bundle-Branch Block

ABSTRACT & COMMENTARY

Sources: Li SF, et al. Electrocardiographic diagnosis of acute myocardial infarction in the presence of left bundle branch block. *Ann Emerg Med* 2000;36:561-565; and Sokolove PE, et al. Interobserver agreement in electrocardiographic diagnosis of acute myocardial infarction in patients with left bundle branch block. *Ann Emerg Med* 2000;36:566-571.

IN THE STUDY BY LI AND ASSOCIATES, THE AUTHORS PERformed a retrospective cohort trial investigating the

use of the Sgarbossa criteria[1] in emergency department (ED) patients with acute myocardial infarction (AMI) and electrocardiographic left bundle-branch block (LBBB) pattern. All patients admitted with suspected acute coronary ischemia and LBBB were eligible. Traditional definitions of AMI (using CPK-MB isoenzyme elevation) and LBBB (electrocardiographic criteria) were employed; the chronicity of the LBBB also was determined (new, old, or unknown). Three physicians, blinded to the clinical diagnosis, interpreted the LBBB ECGs using the Sgarbossa criteria as follows: 1) concordant ST segment elevation (STE) = 1 mm; 2) discordant STE = 5 mm; and 3) concordant ST segment depression (STD) in leads V_1-V_3. Of the 306 patients eligible for study entry, 116 were excluded because of immediate need for therapeutic intervention prior to diagnosis (22); incomplete cardiac enzyme set or medical records (25 and 8, respectively); or presentation not consistent with acute coronary ischemia (61). Data analysis was performed on the remaining 190 patients; 25 (13%) individuals ultimately experienced AMI.

All three criteria demonstrated extremely low sensitivity and extremely high specificity for AMI. Only two indices—concordant STE or having any of the three criteria—had positive likelihood ratios (LR^+) greater than 1; concordant STE had a LR^+ of 16, and having any of the three criteria demonstrated a LR^+ of 3. Eleven patients (6%) had new LBBB, of which six had AMI. Inter-observer agreement for application of this rule among the study physicians was 91% or higher for each of the criteria among all subgroups. The authors concluded that the Sgarbossa criteria lacked sensitivity to exclude AMI in the ED.

In the Sokolove and colleagues study, inter- and intra-observer agreement were tested between emergency physicians (EPs) and cardiologists with regard to the Sgarbossa criteria for the diagnosis of AMI in the LBBB patient. Four EPs and four cardiologists interpreted 224 LBBB ECGs—100 with AMI confirmed by enzyme analysis and 124 without either electrocardiographic or clinical evidence of AMI; a subset of 25 ECGs was re-evaluated by each physician to determine intra-observer reliability. Inter-rater agreement using the Sgarbossa criteria was high, with kappa (?) coefficients of 0.81 for cardiologists, EPs, and all readers combined. The intra-observer reliability was stronger for cardiologists (? = 0.81) than for EPs (? = 0.71), but this difference was not statistically significant. The median sensitivity for EP diagnosis of AMI was 67% compared to a value of 73% for cardiologists. The authors concluded that the study physicians were able to use the criteria with excellent inter- and intra-observer reliability, and speculated that EPs should be able to use the criteria reliably in the evaluation of the chest pain patient with LBBB.

COMMENT BY WILLIAM J. BRADY, MD

LBBB markedly reduces the diagnostic power of the ECG. In fact, until recently, common medical opinion stated that the electrocardiographic diagnosis of AMI was impossible in the presence of LBBB. The introduction of the Sgarbossa criteria in 1996 has led to a re-evaluation of this position. Li and associates concluded that the Sgarbossa criteria are not useful in excluding AMI in the ED. Other literature support this conclusion. The first investigation applied the criteria to patients with chest pain and LBBB, reporting a very low sensitivity for electrocardiographic diagnosis of AMI coupled with poor interobserver reliability.[2] A second study investigated the diagnostic and therapeutic impact of these criteria, and found that none effectively distinguished patients with AMI from patients with noncoronary diagnoses; the authors concluded that electrocardiographic criteria are poor predictors of AMI in the presence of LBBB and suggested that all patients suspected of AMI with LBBB should be considered for thrombolysis.[3] A third investigation followed just this recommendation.[4] A thrombolytic agent was used in all patients with LBBB presumed to have AMI, and they reported an alarmingly high rate of inappropriate thrombolysis. The authors also retrospectively investigated the impact of the criteria on diagnosis and management.[4] These investigators, in contrast to the previously noted reports, found significant accuracy using the Sgarbossa criteria—approximately an 80% correct diagnosis rate using the prediction rule. The authors suggested that, had the prediction rule been employed, inappropriate thrombolysis would have been avoided in many instances.

These ECGs are complicated and the patient care scenarios are challenging; thus, no easy answer is available to the clinician. A LBBB pattern does not entirely invalidate ECG use, it merely reduces the utility of the ECG in arriving at the correct diagnosis. As long as the EP recognizes this limitation, the ECG can be used in the ED evaluation of the potential AMI patient with LBBB. The study by Li et al only reinforces this statement. The clinician must realize that when considering patients with chest pain, LBBB pattern, and clinically suspected AMI, only a minority will manifest obvious electrocardiographic abnormalities as reported by the Sgarbossa group. As in all patients with suspected coronary ischemia, a nondiagnostic ECG in no way excludes the diagnosis of AMI. The Sokolove et al study suggests that these criteria can be reliably used by EPs to evaluate patients with LBBB on ECG for coronary ischemia. ❖

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Over-the-Counter Diet Agents: Dramatic Evidence for Stroke and Health Risk

ABSTRACT & COMMENTARY

Sources: Kernan WN, et al. Phenylpropanolamine and the risk of hemorrhagic stroke. *N Engl J Med* 2000;343:1826-1832; and Haller CA, et al. Adverse cardiovascular and central nervous system events associated with dietary supplements containing ephedra alkaloids. *N Engl J Med* 2000; 343:1833-1838.

PHENYLPROPANOLAMINE (PPA) IS COMMONLY FOUND IN appetite suppressants and cold remedies. Experience with overdoses and case reports have linked the use of products containing PPA to hemorrhagic stroke, often after the first use of these products. Could routine use of this drug result in hemorrhagic stroke as well?

In a case control study by Kernan and colleagues, the authors recruited men and women ages 18 to 49 from 43 U.S. hospitals. Eligibility criteria included the occurrence of a subarachnoid or intracerebral hemorrhage within 30 days before enrollment, and the absence of a previously diagnosed brain lesion. For each case patient, two control subjects were matched for age, race, and sex; nearly 2000 subjects were enrolled. For women, the adjusted odds ratio was 16.58 (95 % CI, 1.51-182.21; P = 0.02) for the association between the use of appetite suppressants containing PPA and hemorrhagic stroke risk. The association was weaker and not statistically significant for women's first use of cough and cold remedies containing PPA, and for men and women combined for cough and cold remedies. An analysis in men showed no increased hemorrhagic stroke risk in association with the use of cough or cold remedies containing PPA. No men reported the use of appetite suppressants. The results suggest that PPA in appetite suppressants, and possibly in cough and cold remedies, is an independent risk factor for hemorrhagic stroke in women.

Dietary supplements that contain ephedra alkaloids (sometimes called ma huang) are widely promoted as "all-natural" means of losing weight and increasing energy. In light of recently reported adverse events related to use of these products, the Food and Drug Administration (FDA) has proposed limits on the dose and duration of the use of such supplements. They have requested an independent review of adverse events reports related to the use of supplements containing ephedra alkaloids in order to assess causation and estimate the level of risk to consumers.

In the second study, by Haller and associates, a standardized rating system was applied to 140 reports of adverse events related to the use of dietary supplements containing ephedra alkaloids that were submitted to the FDA between June 1, 1997, and March 31, 1999. Sixty-two percent of the cases were considered to be definitely, probably, or possibly related to the use of supplements containing ephedra alkaloids. Hypertension was the single most frequent adverse effect (17 reports), followed by palpitations, tachycardia, or both (13); stroke (10); and seizure (7). Ten events resulted in death, and 13 events produced permanent disability. The authors conclude that the use of dietary supplements containing ephedra alkaloids may pose a health risk to some persons.

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

When the first study was submitted to the *New England Journal of Medicine*, the editors considered it so important that they released it as an early report on their Web site (http://www.nejm.org/content/2000/0343/0025/1826.asp). In addition, this study prompted the recall of PPA products. My own experience with PPA supports this study. I have seen overdoses that result in severe headaches, hypertension, seizures, and hemorrhagic stroke. As a result, I stopped prescribing these products a few years ago. Pathophysiologically, the morbidity appears to be related to the property of PPA that induces cerebral hypertension—so much so, in fact, that a reflex bradycardia often is induced in overdose. I suspect the reason that the association between women and

PPA-containing appetite suppressants is so strong is because women are, on average, smaller than men, and because the highest amount of PPA (75 mg) is found in extended-release appetite suppressants, resulting in a higher mg/kg dose than that found in cough and cold preparations.

This study is very important to emergency physicians for two reasons: 1) they should immediately stop prescribing PPA-containing products, and 2) they should ask patients who complain of headache about the use of these products, as they represent a new and important risk for hemorrhagic stroke.

The second report also was published as an early Web site release (http://www.nejm.org/content/2000/0343/0025/1833.asp). This study confirms concerns that these "natural" products are indeed potent pharmaceuticals. In addition, physicians who query patients about the use of these products may find that the sympathomimetic effects of these agents are the cause of clinically relevant morbidity and mortality. ❖

Special Feature

Carbon Monoxide Poisoning: 'Tis the Season

By Richard J. Hamilton, MD, FAAEM, ABMT

EVERYONE ENJOYS THE WINTER FOR THE FUN INDOOR and outdoor activities that are part of the season. However, the season also brings an increased risk of carbon monoxide (CO) poisoning from the combination of cold weather, closed spaces, and fossil fuel heating. When seeing patients in the emergency department (ED), try to remember to put CO poisoning in the differential diagnosis of the many presenting complaints during the winter. While it is easy to set that as a goal during cold weather clinical practice, the protean nature of the symptoms of CO poisoning and the relatively small numbers of patients who suffer from it may make emergency physicians feel that they are chasing at shadows. Rather than review the complex pathophysiology of CO or delve into the controversies regarding treatment, this discussion will focus on the epidemiology and diagnosis of this disease to help emergency physicians identify an easily overlooked problem.

Presentation

In emergency medicine, all things start with the chief complaint. When the chief complaint is "I smelled gas and I have a headache," clinicians need little convincing to consider CO. Although CO in and of itself is odorless and colorless, odors are associated with CO exposure. Natural gas is naturally odorless, but sulfur-containing compounds known as mercaptans are added to the gas line as an olfactory warning. Improperly maintained or blocked heaters may develop a leak or a faulty pilot light and allow natural gas into the environment. Although this does not cause CO toxicity, it is a condition associated with the partial combustion of fossil fuels that could signal CO exposure.

What if the chief complaint is simply, "I have a headache," or "I feel tired?" While this is certainly a clinical presentation for CO poisoning, it is a relatively common complaint. One strategy could be to measure carboxyhemoglobin (COHb) levels of all patients in the ED in the wintertime, but research indicates this would only identify an elevated COHb in 1% of patients.[1] This strategy can be selectively applied to patients complaining of more typical CO complaints—headache, nausea, and dizziness—to increase the yield to about 10%.[2]

More importantly, asking specific questions can have a high predictive value. For example, two important studies examined the predictive value of asking patients whether they used gas ovens to heat their homes or whether they had cohabitants with similar symptoms. The authors noted an association for each of these two issues with CO toxicity in a series of patients.[3] Then they validated this model by asking everyone who came to the ED with headache or dizziness whether they used gas stoves for heating purposes and whether there were similarly affected cohabitants.

To test the validity of this retrospectively derived rule, 65 patients were studied who were unaware of any CO exposure and who presented during the winter of 1986-1987 with headache or dizziness. The algorithm correctly identified three of four patients with COHb levels greater than 10% (75% sensitivity) and correctly excluded 45 of 61 patients with lower levels (74% specificity). The presence of symptomatic cohabitants alone was an equally sensitive (75%) but more specific (90%) marker for elevated COHb levels.[4] Although not perfect, it helps the clinician more confidently sort through the nonspecific complaints that could be related to CO toxicity, and be a bit more parsimonious with testing.

Sources of Poisoning

Ultimately, there is no substitute for an inquisitive clinician with an awareness of the literature. Asking questions about the home or work environment, the health of co-workers or other family members, use of alternative heating methods, or activities at the onset of illness can be very illustrative.

Motor vehicles are a particular concern, and patients have been poisoned from a variety of activities associated with them—repairs, sleeping in vehicles, or occupying rooms adjacent to garages. Simply reading through *Morbidity and Mortality Weekly Reports* from the Centers for Disease Control and Prevention reveals a sampling of CO sources to consider other than heating systems or fires. Sources of CO poisoning reported within the last 10 years (fatal and nonfatal) include propane-powered forklifts, saws, gas grills, ice skating, driving an ice resurfacing machine, attending indoor tractor pulls, camping, playing bingo above an ice rink, cleaning with power washers, and shoveling snow from around idling motor vehicles. These sorts of activities generally are not part of the standard social history; thus, it is paramount to have an appropriate threshold of suspicion when it comes to CO.

Lest emergency physicians presume that an enclosed environment is a prerequisite for CO poisoning, there are a number of case reports of patients developing CO toxicity despite apparent ventilation. Pick-up trucks with campers can create a suction effect when the back window is opened and no front intake of air is present. This relative vacuum in the passenger compartment pulls exhaust into the passenger compartment.[5] One case report of the operator and occupants of an open-air tractor who developed CO toxicity while operating the vehicle at a slow rate of speed in relatively still air highlights the insidious nature of this poison.[6]

In addition, methylene chloride is an overlooked source of CO toxicity. It is a one carbon molecule that is used as a paint stripper and solvent. Dermal or inhalational exposure, followed by absorption and metabolism, can lead to elevated CO levels. These types of exposures have caused the entire spectrum of neurologic and cardiovascular complications seen with CO poisoning.[7,8]

Making the Diagnosis

Once suspicion for acute CO poisoning has been triggered, making the diagnosis is as simple as knowing which test to order and understanding possible confounders. The standard of care is to obtain a venous blood sample for co-oximetry to determine the COHb level. This is reported as a percentage, along with oxyhemoglobin, deoxyhemoglobin, and methemoglobin, when a four wavelength co-oximeter is used. There is no need to perform arterial blood sampling, as CO is so avidly bound to hemoglobin that little CO exchange occurs in the lungs relative to the venous circulation. Remember that pulse oximeters generally mistake COHb for oxyhemoglobin and report falsely elevated saturations.

If the prevalence of CO poisoning is increased due to occupational or household exposures (e.g., firefighters who are seen for smoke inhalation or people who are from communities with older housing and outdated heating systems), investing in and maintaining any of the portable devices that measure exhaled CO may be an efficient approach, especially for identifying COHb levels above 10%.

COHb elevations are considered diagnostic for exposure with certain caveats. Cigarette smoking will elevate COHb levels to a range of 2-10%. Pregnant women and patients who have hemolytic anemia will demonstrate COHb elevations from the breakdown of heme proteins and endogenous CO production, but these are usually less than 10%. Fetal hemoglobin also causes false

COHb elevations as a result of the partial absorption of fetal hemoglobin at light wavelengths that are used to detect COHb, but these generally are less than 5%.

CO toxicity, while often occult, is not always subtle in presentation. Patients with syncope, seizures, coma, acute myocardial infarction, arrhythmias, or cerebrovascular accident also could be suffering from CO poisoning. A specific associated sign, such as cutaneous bullae, should prompt consideration of CO toxicity in the differential diagnosis.[8] Another telling finding is bilateral hypodensities in the globus pallidus on head CT. Although not pathognomonic, these appear to correlate with neurologic injury and have a poor prognosis.[9]

Summary

Clinicians who have a heightened suspicion for this disease and who are vigilant for an occult presentation as a simple viral syndrome or complete coma will be rewarded with a diagnostic save when they make the diagnosis of CO poisoning. Besides, it gives you something to explore the next time you go to the chart rack and see that the next three patients are from the same house with fatigue, nausea, and headache . . . sure it could be influenza, but with CO you just never know. ❖

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CME Questions

17. Concurrent ED trauma cases had what effect on the care of ED patients with chest pain in the study by Boutros and Redelmeier?
a. Lengthened their ED stay
b. Led to higher mortality
c. Shortened their ED stay
d. Led to higher morbidity

18. Patients with LBBB have the greatest likelihood of AMI if the:
a. LBBB pattern is known to be pre-existing.
b. chronicity of the LBBB pattern is indeterminate.
c. LBBB pattern is new.
d. LBBB pattern resolves.

19. According to the study by Li and colleagues, the Sgarbossa criteria for AMI in patients with LBBB is:
a. highly sensitive.
b. highly specific.
c. highly accurate.
d. All of the above

20. The study by Sokolove et al suggests that inter-rater reliability with regard to ECG interpretation for AMI in patients with LBBB is:
a. high for emergency physicians and cardiologists.
b. low for emergency physicians and cardiologists.
c. high for cardiologists only.
d. high for emergency physicians only.

21. New data support the association of hemorrhagic stroke with which agent in women?
a. Chlorpheniramine
b. Pseudoephedrine
c. Dextromethorphan
d. Phenylpropanolamine

22. Which of the following dietary supplement preparations has been linked to adverse effects including hypertension, tachycardia, and stroke?
a. Zinc
b. Calcium
c. Ephedra alkaloids
d. Gingko

23. Poisoning with carbon monoxide has been associated with:
a. carbon tetrachloride.
b. mercaptans.
c. methylene chloride.
d. diethylene fluoride.

24. Information found to be helpful in screening for carbon monoxide poisoning include which of the following?
a. Use of gas ovens to heat the home and history of syncope
b. Use of gas ovens to heat the home and cohabitants with similar symptoms
c. History of syncope and cohabitants with similar symptoms
d. Fatigue, malaise, and myalgia
e. Fatigue, cough, and myalgia

Irregular VT?

By Ken Grauer, MD

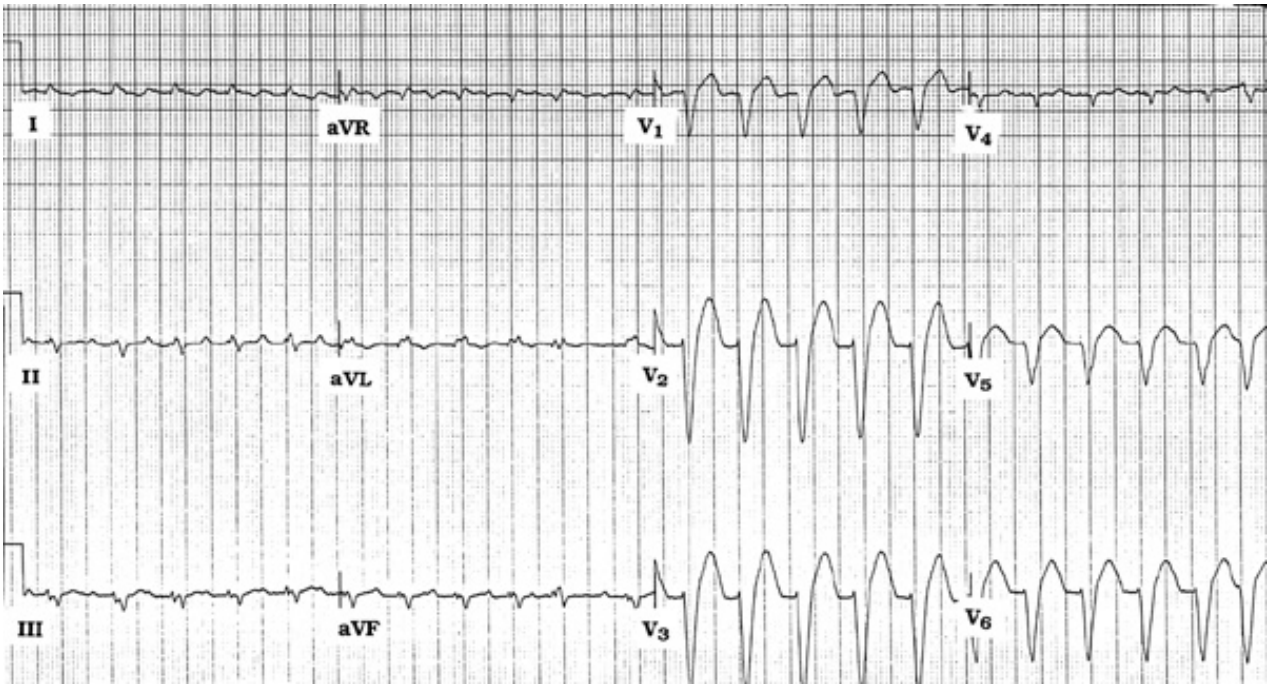

Figure. 12-lead ECG obtained from a woman with coronary disease. Is this VT?

Clinical Scenario: The ECG shown in the Figure was obtained from a woman in her 60s with known coronary disease. In view of the fact that ventricular tachycardia (VT) *may* sometimes be slightly irregular, would you interpret the rhythm in this Figure as probable VT?

Interpretation: The rhythm in the Figure is a fairly regular, wide-complex tachycardia. Whenever this is seen in an older adult with known coronary disease—VT *always* must be assumed until proven otherwise, especially when QRS morphology looks as bizarre as it does in this tracing. That said, the cause of the wide complex tachycardia in the Figure is *not* VT. As is often the case, the key clue for interpreting this rhythm lies with "looking for a pause!"

Whereas on initial inspection one might not think any P waves are present on this tracing, the brief pause preceding the last beat in lead aVF strongly suggests otherwise. A P wave clearly precedes this last beat in lead aVF with a normal PR interval. Looking again at lead II, it can now be seen that the second QRS complex in this lead also is preceded by a P wave with a PR interval similar to the last beat in lead aVF. The small upright deflection at the midpoint of the R-R interval for the other beats in lead II therefore is *not* a T wave, but instead represents sinus node activity. Therefore, the rhythm in this tracing is sinus tachycardia with several premature supraventricular beats (PACs or PJCs)—and QRS widening as the result of an unusual form of IVCD (intraventricular conduction delay). We emphasize that without the pauses in the rhythm described above, one would have to assume VT for this tracing. ❖

In Future Issues:

Octreotide for Refractory Hypoglycemia After Sulfonylurea Overdose