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INSIDE

Unwelcome Import: SARS Arrives in U.S. Emergency Departments

ABSTRACT & COMMENTARY

Source: Centers for Disease Control and Prevention. Update: Severe acute respiratory syndrome—United States, 2003. *Morb Mortal Wkly Rep MMWR* 2003;52:357-360.

As of April 23, 2003, a total of 4288 cases of severe acute respiratory syndrome (SARS) in 25 countries had been reported to the World Health Organization. Internationally, there had been 251 deaths, yielding a case-fatality rate of 6%. In the United States, there were 245 cases of SARS in 37 states. Thirty-nine cases were classified as "probable," and 206 as "suspect." Tests for a SARS-associated novel coronavirus (SARS-CoV) were positive in about half of probable cases and in none of the suspect cases in the United States, but test results were available for fewer than one-fifth of cases as of the end of April 2003.

All SARS cases reported in the United States involved patients who had traveled to areas endemic for SARS or had come in close contact with a SARS case. Ninety-one percent of patients had traveled to mainland China, Hong Kong, Singapore, Hanoi, or Toronto. A few cases were health care workers who provided care to a SARS patient or household contacts of a SARS patient. Thirty-seven percent of all U.S. SARS cases, and 69% of probable cases, required hospitalization. Two patients required mechanical intubation.

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

The Centers for Disease Control and Prevention updated its interim case definition for SARS on April 20, 2003. A *suspect* case is a patient with a respiratory illness of unknown etiology who has a temperature greater than 100.4°F, one or more signs of respiratory illness (cough, dyspnea, or hypoxia), and either traveled to an endemic region within 10 days of symptom onset or had close contact with a suspected SARS case. A *probable* case of SARS is defined as one meeting all these criteria, and having either radiographic evidence of pneumonia or respiratory distress syndrome, or autopsy findings consistent with respiratory distress syndrome for which no other cause is identified. As of late April 2003, areas con-

sidered at risk for transmission of SARS were mainland China, Hong Kong, Hanoi, Singapore, and Toronto.[1]

While the media have focused on the fact that there have been more than 245 reported cases of SARS in the United States, it is very important to note that the SARS-CoV test has so far been negative in each of the suspect cases and in about half of the three dozen probable cases. The large number of pending tests makes it dangerous to extrapolate this data, but it appears that fewer than 10% of all reported SARS cases in the United States ultimately will be diagnosed with the illness. The current case definition is appropriately lax at this time, and any febrile patient with respiratory complaints having traveled to a SARS-endemic region is presently considered to be a suspect case.

Patients meeting SARS criteria who present to the ED should be identified by the triage nurse and immediately segregated in respiratory isolation rooms. Health care workers should employ airborne infection precautions (i.e., N-95 respirators), contact precautions (i.e., gowns and gloves), as well as standard precautions (i.e., hand washing). Eye protection also is recommended.[2] ❖

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Emergency Medicine Alert, ISSN 1075-6914, is published monthly by Thomson American Health Consultants, 3525 Piedmont Rd., NE, Bldg. 6, Suite 400, Atlanta, GA 30305.

Vice President and Group Publisher: Brenda Mooney.
Editorial Group Head: Valerie Loner.
Managing Editor: Allison Mechem.
Marketing Manager: Schandale Kornegay.

GST Registration Number: R128870672.

Periodicals postage paid at Atlanta GA 30304. **POSTMASTER:** Send address changes to ***Emergency Medicine Alert***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $44. One to nine additional copies, $212 each; 10 to 20 additional copies, $159 each.

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Subscription Prices

United States: $265 per year (Resident rate: $132.50)
Canada: $295 per year plus GST (Resident rate: $147.50)
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EMS Response Time May Dictate Choice of CPR or Defibrillation in the Field

ABSTRACT & COMMENTARY

Source: Wik L, et al. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation—A randomized trial. *JAMA* 2003;289: 1389-1395.

EARLY DEFIBRILLATION HAS BEEN SHOWN TO MARKEDly improve survival rates for patients who suffer sudden cardiac arrest from ventricular fibrillation (VF) and pulseless ventricular tachycardia (VT). As a result, immediate defibrillation has been recommended as a part of ACLS guidelines for VF and VT.[1] However, the chance for successful defibrillation and conversion to a perfusing cardiac rhythm drops precipitously with time delay. Animal studies have suggested that, in cases of prolonged VF/VT, a short period of cardiopulmonary resuscitation (CPR) prior to defibrillation may "prepare" the myocardium and improve conversion rates for defibrillation.

In this European study, investigators randomized 200 out-of-hospital VF/VT cardiac arrest victims to immediate defibrillation and standard cardiac arrest care (96 patients in the standard group) vs. three minutes of CPR prior to defibrillation (104 patients in the CPR-first group) performed by responding emergency medical services (EMS) personnel. The authors report no differences between the two groups in terms of the primary outcome measure (survival to hospital discharge), or secondary outcome measures (hospital admission after return of spontaneous circulation [ROSC], neurologic outcome, and one-year survival).

The investigators then compared the standard and CPR-first groups by EMS response time. With response times of five minutes or less (81 patients), there again was no difference found between the standard and CPR-first groups in terms of survival to hospital discharge, ROSC or one-year survival. However, with response times greater than five minutes (119 patients), there was a significant improvement in the CPR-first group in terms of survival to discharge (22% vs 4%), ROSC (58% vs 38%), and one-year survival (20% vs 4%) when compared with standard care.

In the data analysis, the authors also determined that the benefit of three minutes of CPR prior to defibrillation improved with longer EMS response times. Calculated odds ratios for survival favoring CPR-first were 0.4 for a less than one minute response time, three for a seven-minute response time, and 6.1 for a nine-minute response time.

Based on their findings, the authors conclude that, compared with standard care for VF/VT, CPR first offered no advantage. However, for VF/VT patients with EMS response times greater than five minutes, CPR first did confer a significant improvement in outcome and survival.

COMMENTARY BY THEODORE C. CHAN, MD, FACEP

In this randomized study, the authors found that three minutes of CPR for VF/VT out-of-hospital victims improved outcomes in those with EMS response times greater than five minutes. Just as important, the authors demonstrated that the CPR-first approach had no detrimental impact on those with response times less than five minutes or on the entire group of VF/VT victims.

The idea that CPR may "prepare" the myocardium or "coarsen" VF and improve countershock success has been suggested in animal studies, as well as before-after studies in human victims.[2,3] However, this is one of the earliest randomized studies comparing immediate countershock to CPR-first prior to defibrillation in out-of-hospital VF/VT victims. Not only did the authors find this method to benefit survival in cases of longer response times, but also found excellent neurologic outcomes for the survivors (89% with minimal or no neurologic deficits) with no difference in either group. This finding suggests that CPR prior to defibrillation did not result in more survivors with significant neurologic impairment; that is, more "saves" did not come at the expense of worse neurologic status.

It is worth noting that this study was performed in Europe, where physicians staff many of the EMS crews (nearly one-quarter of patients in this study were attended by a physician in the field). In addition, EMS response times do not necessarily reflect overall victim downtime, which is likely the more critical time element for overall survival. More than half the victims in each group received bystander CPR, and it is unclear what impact such efforts may have had on the differences between the CPR-first and standard care groups. Just as important, while use of lidocaine and epinephrine were no different between the two groups, it is unclear what impact newer agents, such as amiodarone, or efforts such as public access defibrillation, may have on the findings of this study. ❖

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Continued Complications Plague tPA in Stroke

ABSTRACT & COMMENTARY

Source: Bravata DM, et al. Thrombolysis for acute stroke in routine clinical practice. *Arch Intern Med* 2002;162: 1994-2001.

The National Institute of Neurological Disorders and Stroke rt-PA (NINDS) study demonstrated that tissue plasminogen activator (tPA) therapy for acute ischemic stroke can be given safely and effectively.[1] Over the last several years, only a small percentage of patients with acute strokes were treated with tPA, and many stroke experts are pushing for increased tPA use in the United States. However, only a small number of studies using tPA for stroke have been completed since the NINDS trial, with mixed results.

Studies performed in stroke centers and by NINDS investigators have shown favorable outcomes and low rates of major complications. Studies performed in other settings have shown much higher rates of intracranial hemorrhage (ICH) and other complications, possibly related to large numbers of protocol violations. The

purpose of this retrospective cohort study was to compare adverse outcomes among patients given intravenous tPA in routine clinical practice to those given tPA in the NINDS cohort, and to examine the relationship of protocol deviations to adverse outcomes.

Sixty-three patients in 16 Connecticut hospitals were identified for study inclusion. The authors collected rates of in-hospital mortality, symptomatic and asymptomatic ICH, and major or minor extracranial hemorrhage (ECH). A protocol deviation was defined as deviation from the American Heart Association (AHA) Guidelines for Thrombolytic Therapy for Acute Stroke, and was classified as either major (presence of a contraindication to use indicated on the tPA package insert) or minor (deviation from AHA guidelines which was not classified as major).

In-hospital mortality was higher in the Connecticut cohort than in the NINDS cohort (25% vs 13%), as was the rate of ICH (17% vs 11%). The rate of symptomatic ICH was similar in the two cohorts (6% vs 6%), but the rate of asymptomatic ICH was higher in the Connecticut cohort (11% vs 4%, p = not significant). The rate of major ECH was higher in the Connecticut group (13% vs 2%), but the rate of minor ECH was similar.

In the Connecticut group, 67% of patients treated with tPA had at least one major protocol deviation, and 90% had at least one minor protocol deviation. Over-all, 97% had at least one protocol deviation. The most common major protocol deviations were dosing errors, initiation of therapy after the three-hour window, known bleeding diathesis, and evidence of active internal bleeding.

Not surprisingly, in-hospital mortality increased as the number of major protocol deviations increased. The in-hospital mortality rate of patients with one major protocol deviation was 29%, and was 36% for patients with two or more major protocol deviations. The in-hospital mortality rate was 23% for patients with one minor protocol deviation, and 32% for those with at least two minor protocol deviations. Among patients without any major protocol deviations, the mortality rate of the Connecticut cohort was similar to that of the NINDS cohort (14% vs 13%).

In 30% of the 63 cases, the clinicians documented that they were aware of the protocol deviations. Most of these included physician documentation by the clinician that tPA was being given outside of recommended guidelines.

COMMENTARY BY JACOB W UFBERG, MD

This study looked at only a small cohort of patients receiving tPA, but the results mimic those of the Cleveland study.[2] When the physicians do not adhere tightly to the NINDS/AHA protocol, the number of complications greatly increases, probably enough to wipe out the advantages demonstrated by the NINDS investigators.

No matter where you stand on the tPA for ischemic stroke controversy, one fact is perfectly clear: If you are using tPA, do it properly! Have a protocol in place that easily can be followed, and make sure it adheres strictly to the guidelines proposed by the NINDS investigators and the AHA. If a patient does not meet the strict criteria for receiving tPA, or if you are not sure whether the patient meets criteria, go with conservative therapy. ❖

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Special Feature

The Brugada Syndrome

By William J. Brady, MD and Amal Mattu, MD

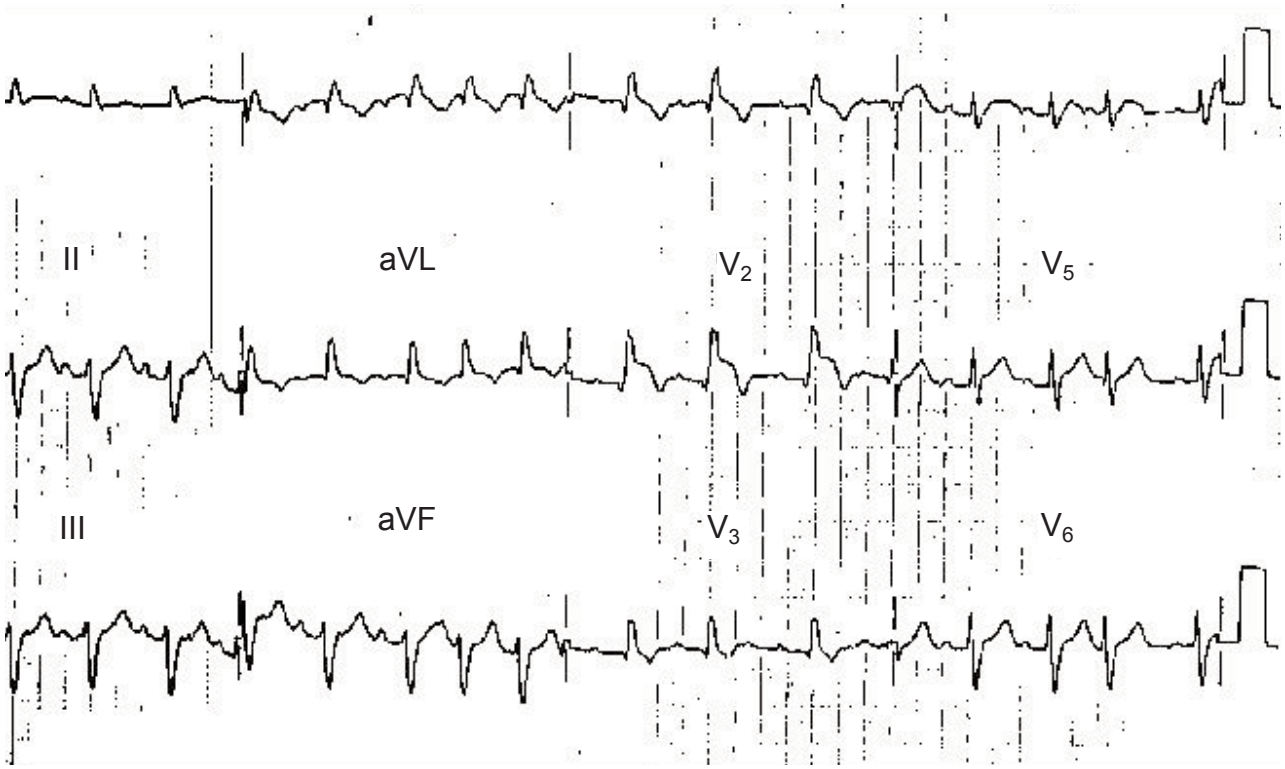
Introduction

The Brugada syndrome is a recently described cardiac disorder involving patients with structurally normal hearts who lack coronary artery disease and experience ventricular dysrhythmias.[1-3] These patients present with sudden cardiac death, as well as characteristic electrocardiographic abnormalities when in sinus rhythm, including right bundle-branch block (RBBB) pattern and right precordial ST segment elevation (STE). In fact, this syndrome likely accounts for approximately 50% of patients with sudden death who lack organic heart disease.[3] Brugada and Brugada initially described this presentation in 1992; they characterized the syndrome in a series of patients with the classic electrocardiographic findings who survived recurrent episodes of sudden cardiac death.[1] Contrary to earlier reports focusing entirely on young Asian males, the Brugada syndrome is encountered in patients of multiple ethnic groups, in both genders, and in all age groups.[2,4-6] It is, however, found to a significantly greater extent in Asian males, likely due to increased genetic transmission in these ethnic groupings.[7]

Clinical Spectrum

Patients with the Brugada syndrome experience symptomatic ventricular dysrhythmias, most often

Figure 1. 12-lead ECG after successful defibrillation to normal sinus rhythm



Note the widened QRS complex with RBBB configuration and ST segment elevation in the right precordial leads.

polymorphic ventricular tachycardia which degenerates into ventricular fibrillation.[2] Patients present across a spectrum of initial severity, ranging from a history of syncope in the patient with normal sinus rhythm, to incessant, malignant ventricular dysrhythmia in the individual with active cardiac arrest. If the dysrhythmia is self-terminating, the patient will note dizziness or complain of syncope; in such instances, the clinician must be familiar with the "resting" electrocardiographic findings of RBBB and right precordial STE when the patient is in normal sinus rhythm. Alternatively, if the dysrhythmia continues, it ultimately will degenerate into ventricular fibrillation.

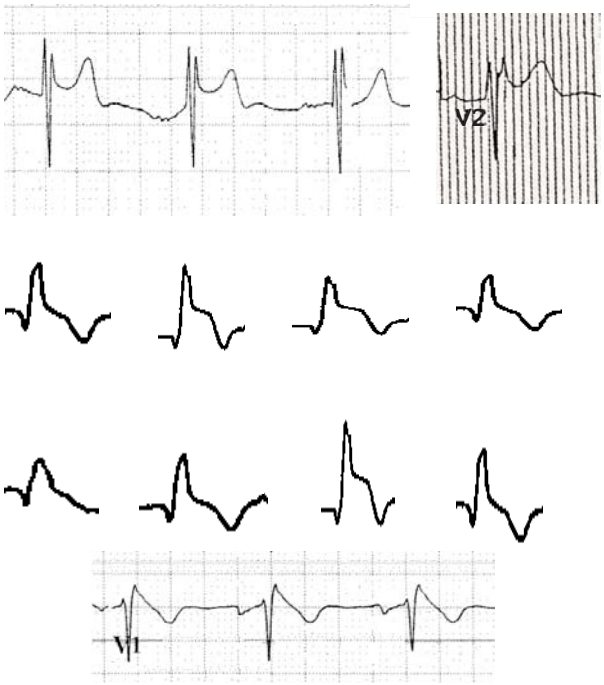
A familial occurrence has been observed in a segment of patients with the Brugada syndrome, suggesting a genetic component to the disease. Recent studies have confirmed a genetic predisposition; mutations in the gene SCN5A (which encodes for the cardiac sodium channel) have been identified in patients with the Brugada syndrome.[2,8-10] The mutation produces an improperly functioning sodium channel. This sodium channel malfunction is intensified by increasing body temperature, is precipitated by certain drugs (particularly agents which effect the cardiac sodium channels), and is enhanced by various autonomic disturbances.[11,12]

Individuals with a family history of sudden cardiac death who demonstrate RBBB with right precordial ST segment elevation should be considered as possible Brugada syndrome patients. The clinician must realize that malignant ventricular dysrhythmias occur in up to one-third of initially asymptomatic patients within two years of the initial discovery of the syndrome features.[7]

ECG Findings in the Brugada Syndrome

The classic electrocardiographic findings include RBBB and STE in the right precordial leads (V_1 to V_3). *(See Figures 1-3.)* With increased physician awareness of the syndrome, variants in the original electrocardiographic presentations have been described. These variations, in fact, reflect minimal alteration from the original presentations. Patients may demonstrate an incomplete RBBB, STE in a more limited distribution (leads V_1 to V_2), and various forms of STE. Two types of STE morphologies have been described in the right precordial leads: convex ("coved") and concave ("saddle-type").[3,4,13] *(See Figures 2 and 3.)* The electrocardiographic changes in the Brugada syndrome are not constant; in fact, these abnormalities have been known to change over time—ranging from alterations in the previously noted pattern to complete resolution.[3,13] Dysrhythmias in the Brugada syndrome include ventricular

Figure 2. ST segment elevation in Brugada syndrome



tachycardia (monomorphic and polymorphic) and ventricular fibrillation; polymorphic ventricular tachycardia is the predominant form of ventricular tachycardia seen in this patient group. No electrocardiographic features are diagnostic of the Brugada syndrome when the patient is in a ventricular dysrhythmia.

Treatment and Disposition

Urgent therapy involves electrical defibrillation and other cardiorespiratory support; antiarrhythmic agents have proven to be of little benefit in the resuscitation phase of management. Furthermore, antiarrhythmic agents are of minimal value in chronic therapy.[2,7] Admission to the hospital or same-day cardiology consultation are recommended. Electrophysiologic testing will confirm the diagnosis with induction of ventricular dysrhythmia. Further confirmation of the diagnosis is made by accentuation of the ST segment elevation with type I antiarrhythmic agents in the electrophysiology laboratory. Once the diagnosis is confirmed, patients must be treated with placement of an automatic internal cardioverter-defibrillator (AICD). With placement of an AICD, mortality has been avoided in all cases thus far.[7] The reported mortality without appropriate therapy is approximately 30% within two years after the index presentation.[2] ❖

(Dr. Mattu is assistant professor, Department of Surgery/Division of Emergency Medicine, University of Maryland, Baltimore.)

Figures 3A-B. Details of ST segment elevation

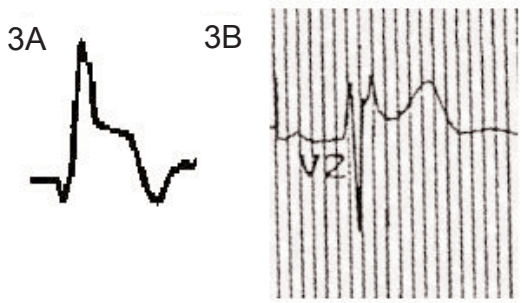


Figure 3A: The "coved" type ST segment elevation. Note the convex morphology. Figure 3B: The "saddle" type ST segment elevation. Note the concave morphology.

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Physician CME Questions

1. **A patient who has traveled to a SARS-endemic area would be suspected of having SARS if which of the following were present?**
   a. Pulmonary infiltrates without a fever
   b. Cough and fever
   c. Severe pharyngitis and fever
   d. Diarrhea or vomiting and fever

2. **As of April 2003, which of the following is *not* considered a SARS-endemic area?**
   a. Hong Kong
   b. Singapore
   c. Hanoi
   d. Jakarta

3. **Giving tPA for acute ischemic stroke outside of the AHA-recommended guidelines appears not to lead to increased complications.**
   a. True
   b. False

4. **The study by Wik et al on the care of out-of-hospital VF/VT cardiac arrest victims found that CPR prior to defibrillation:**
   a. improved survival to hospital discharge, but not one-year survival.
   b. was unnecessary for those victims who received bystander CPR.
   c. increased rates of ROSC, but resulted in worse neurologic outcomes for survivors.
   d. worsened survival rates for patients who had unwitnessed cardiac arrests.
   e. improved survival to hospital discharge in cases with longer response times.

5. **Which of the following issues is *not* considered appropriate and correct in the management of the Brugada syndrome?**
   a. Urgent cardiology referral
   b. Administration of amiodarone
   c. Defibrillation in the patient with ventricular tachycardia
   d. Admission to a monitored inpatient unit
   e. Electrophysiologic testing

6. **Which of the following best characterizes the electrocardiographic findings associated with Brugada syndrome—right bundle-branch block morphology and:**
   a. coved or saddle-shaped ST segment elevation in the left precordial leads.
   b. delta wave in the right precordial leads.
   c. coved or saddle-shaped ST segment elevation in the right precordial leads.
   d. delta wave in the left precordial leads.

7. **Brugada syndrome has been associated principally with which dysrhythmia?**
   a. Polymorphic ventricular tachycardia
   b. Atrial fibrillation with preexcitation and a rapid ventricular response
   c. Sinus arrest
   d. Complete heart block

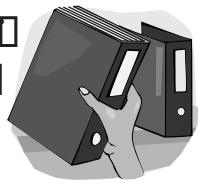
CME Objectives

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.

Answer Key

1. b
2. d
3. b
4. e
5. b
6. c
7. a



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'Scoopies' on ECG

By Ken Grauer, MD

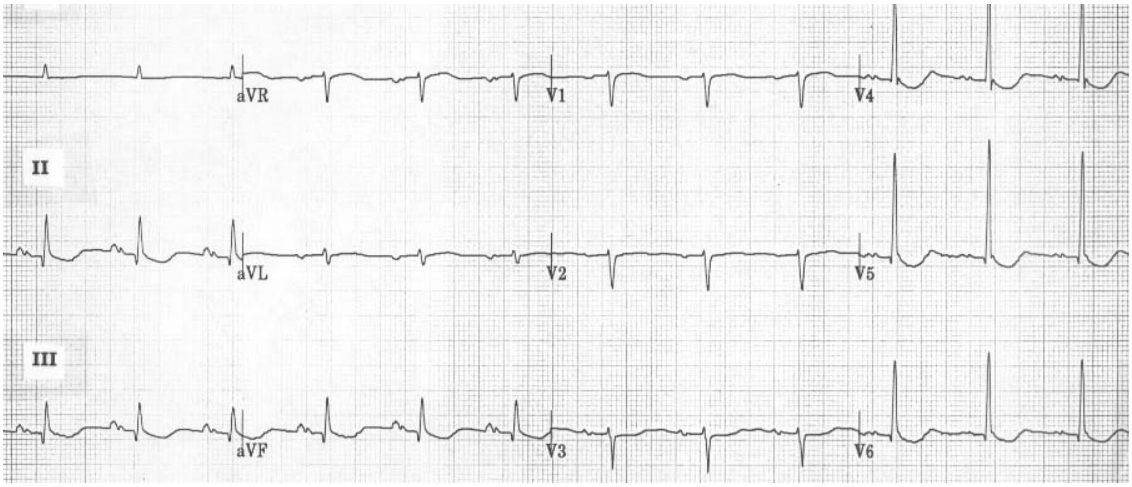

Figure. 12-lead ECG obtained from an older man on multiple medications.

Clinical Scenario: The ECG in the Figure was obtained from an older man on multiple medications. The patient was known to have longstanding cardiopulmonary disease and chronic renal failure, and he presented to the emergency department for an acute exacerbation of his underlying condition. He has chronic angina, but reports no recent change in his chest pain pattern. In view of this history, how would you interpret his 12-lead ECG? Which cardiac drug do you most strongly suspect he is taking?

Interpretation: The rhythm is sinus at a rate of just over 75 beats/minute. The PR, QRS, and QT intervals are all normal. Mean QRS axis is +70°. Left atrial enlargement (LAE) is suggested by the P-mitrale (notching) pattern seen in the inferior leads. QRS amplitude falls shy of criteria for left ventricular hypertrophy (LVH). Perhaps the most remarkable finding on this ECG is the *scooped* appearance (without QT prolongation) of the ST segment in multiple leads.

There are many potential causes of ST segment depression on a 12-lead ECG. Among the most important causes of ST depression to routinely consider whenever one notes this finding are: 1) "strain" (from ventricular hypertrophy); 2) ischemia (which could be chronic or acute); 3) rate-related (if tachycardia is present); 4) digoxin effect; and 5) hypokalemia/hypomagnesemia. In many cases, more than one of these factors will be operative, creating a combination effect on ST-T wave appearance. Clues to the specific etiology of the ST-T wave abnormality are sometimes forthcoming from morphologic appearance. Thus, *symmetric* T wave inversion is more suggestive of ischemia, whereas *asymmetric* ST segment depression is more suggestive of "strain." Morphologic appearance is subject to variation, however, and overlap of patterns is not uncommon. As a result, either symmetric or asymmetric ST-T wave depression may at times be due to the *simultaneous* presence of *both* ischemia and strain in the same patient. Clinical correlation and comparison with prior tracings are essential for accurate interpretation.

Patients who are taking the drug digoxin are likely to manifest one of three ECG patterns: 1) asymmetric ST segment depression that is very similar in appearance to that seen with "strain" from LVH; 2) a *scooped* appearance to the ST segment in multiple leads; or 3) no effect at all on ST-T wave appearance. The interesting clinical point is that the presence or absence of "dig effect" on the ST-T wave (patterns 1 or 2) is *unrelated* to the serum digoxin level, and that as many as one third of patients who are taking this drug manifest no ECG changes at all (pattern 3). Diffuse ST segment "scooping" of the type shown in the Figure strongly suggests that digoxin was among the medications that this patient was taking. ❖

In Future Issues:

Etomidate in Children Undergoing RSI