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INSIDE

Code Stroke Protocol

ABSTRACT & COMMENTARY

Source: Asimos AW, et al. Therapeutic yield and outcomes of a community teaching hospital code stroke protocol. *Acad Emerg Med* 2004;11:361-370.

With the publication of the National Institute of Neurological Disorders and Stroke (NINDS) trial in 1995,[1] and the subsequent adoption of its findings by the American Heart Association starting in 1996,[2] the use of intravenous tissue plasminogen activator (tPA) has been recommended for the treatment of acute stroke in a subset of patients who meet predefined criteria. Almost from its inception, however, there has been controversy surrounding its safety, efficacy, and applicability to a general emergency department (ED) population. A number of subsequent studies have called into question the findings of the original trial that documented a 6% rate of symptomatic intracranial hemorrhage (ICH) rate, a 17% three-month mortality, and a 12% absolute improvement in the Barthel score, testing functionality at three months. A chief concern of subsequent studies is whether these results can be duplicated in community EDs, where there are less specialized resources for the treatment of stroke.

This retrospective review, performed at Carolinas Medical Center, a large, community teaching hospital with an annual ED census of more than 100,000, looked at its experience with intravenous tPA for stroke during a 56-month time period (1997-2001) and compared its experience with that of the NINDS trial. At this institution, when a patient is identified as a potential code stroke patient, an emergency physician (EP) screens the patient for eligibility criteria. If found eligible, the EP works closely with a private practice neurology group, which will respond to see the patient. The decision to proceed with thrombolysis is made jointly by the EP and the neurologist.

During the specified time period, 255 patients were deemed to meet code stroke criteria. After evaluation, 60 were treated with intravenous tPA (24%). The 36-hour symptomatic ICH rate was 10% (vs NINDs = 6%). At three months, 60% of treated patients achieved greater than 95 on the Barthel Index (vs NINDS = 52%). Mortality at three months in this study was 12% (vs NINDS = 17%). Protocol violations, usually cited as a key fea-

ture in increasing risk of hemorrhage, occurred in 32% of patients in this study, but were equally common in those with and without symptomatic ICH.

The study concludes that intravenous tPA can be given safely in the community hospital setting for stroke, with outcome measures that closely parallel those found in the NINDS trial. While protocol violtions were relatively common at this community teaching hospital, the three-month functional outcomes and mortality rates were equal to or better than those found in the original NINDS study.

COMMENTARY BY ANDREW D. PERRON, MD, FACEP, FACSM

To thrombolyse or not to thrombolyse…

To say that the evidence is conflicting on this topic is a significant understatement. There is now a substantial body of literature on both sides of this topic, and the clinician can find support for either view with little difficulty. So where does this study leave me in terms of making a decision regarding thrombolysis in my community teaching hospital?

While handicapped by its retrospective nature, this study has direct applicability to my ED, and I suspect a large number of other EDs in the United States. Perhaps it is only because I practice in the same environment as described, but this study clearly lays out a logical pathway to developing a code stroke protocol at my institution. Asimos and colleagues are clear in their discussion of the mechanics, algorithms, and care protocols they use to get these results. If I were charged with creating a similar protocol at my institution, I would use this as a blueprint.

That being said, this study does not help me answer the bigger question of whether we should be offering thrombolysis as the standard of care in all EDs across the land. I am continually amazed at the reported rate of protocol violations in these studies, where we go to such extremes to specifically avoid them. It is to the point that any study that reports a rate of less than 30% is immediately suspect. I am somewhat heartened by the fact that in this study the rate of symptomatic ICH was the same in both groups; this has not been shown to be true in other studies. In fact, the one lesson I have learned from previous studies is that if you cannot do this well (i.e., you have significant protocol violations), you unquestionably will hurt patients. Whether this can be reproduced in other community settings remains to be seen.❖

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Septic Joint Difficult to Exclude by Laboratory Tests

ABSTRACT & COMMENTARY

Source: Li SF, et al. Laboratory tests in adults with monoarticular arthritis: Can they rule out a septic joint? *Acad Emerg Med* 2004; 11:276-280.

THE AIM OF THIS RETROSPECTIVE CASE SERIES WAS TO determine the sensitivities of three commonly

obtained laboratory tests often used to determine the likelihood that a septic joint is the cause of an acute monoarticular arthritis. The three tests studied were the peripheral white blood cell count (WBC), the erythrocyte sedimentation rate (ESR), and the white blood cell count of the joint fluid (jWBC). The main goal was to determine whether a septic joint could be ruled out by any of these tests.

Adult patients were included in this study if the diagnosis of septic arthritis was confirmed by a positive arthrocentesis culture or operative findings. Demographic, clinical, and laboratory data were obtained from the computerized records of 73 patients. The sensitivities of elevations in WBC, ESR and jWBC were 48% (95% CI 36-60%), 96% (95% CI 88%-100%), and 64% (95% CI 51-76%), respectively. More than one third (36%) of the patients had jWBCs less than 50,000 cells/mm^3, and 10% (6 of 61 pts) had counts of less than 10,000 cells/mm^3. *Staphylococcus aureus* was the causative organism in 42 patients (58%), and 36% of patients were either HIV–positive or diabetic.

COMMENTARY BY STEPHANIE ABBUHL, MD, FACEP

It would be wonderful if a low jWBC count could rule out septic arthritis but, unfortunately, this is not the case. In this cohort of patients with septic arthritis, there was a wide range of values for each of the laboratory tests. While the mean jWBC was 127,000 cells/mm^3, the range was 168 to 1 million cells/mm^3, and 10% had counts of less than 10,000 cells/mm^3. Even the ESR, which was found to have a sensitivity of 96%, was low (less than 40mm/hr) in 7% of patients and normal (less than 30 mm/hr) in 3% of patients.

It is clear that a high degree of suspicion for septic arthritis must be maintained in the approach to the patient with monoarticular arthritis, regardless of a low jWBC. In cases where suspicion is high enough, this will mean admission and treatment with antibiotics until culture results return. In cases where suspicion is low, the prudent approach will be very close follow-up for improvement without antibiotics until the culture returns. Finally, if there is only a single drop of fluid obtained from a joint tap, the most important test to obtain is a culture and gram stain, not a cell count.

This study is limited by small numbers with broad confidence intervals. However, even if a large study could push the sensitivities significantly up, the fact that some cases of septic arthritis occur with low jWBCs, ESRs, or WBCs limits our ability to use these tests to make absolute decisions about ruling out septic arthritis. In addition, as a retrospective chart review of septic arthritis cases, specificities could not be determined, but common practice tells us that all three of these lab tests are nonspecific. Even jWBCs can be remarkably elevated in crystal disease and other inflammatory causes of monoarticular arthritis. This study did not look at the differential of the jWBC, and it would be interesting to know if this would add any sensitivity to the jWBC in ruling out septic arthritis. It also would be helpful to know if the low jWBCs were found more often in patients who were HIV-positive. ❖

Fluoroquinolone-Resistant Gonorrhea on the Rise: Exposure History is Critical

ABSTRACT & COMMENTARY

Source: Centers for Disease Control. Increases in Fluoroquinolone-Resistant *Neisseria gonorrhoeae* Among Men Who Have Sex with Men — United States, 2003, and Revised Recommendations for Gonorrhea Treatment, 2004. *Morb Mort Wkly Rep MMWR* 2004; 53;335-336.

The Centers for Disease Control and Prevention (CDC) support ongoing surveillance of antibiotic susceptibilities of *Neisseria gonorrhea* isolates obtained from individuals presenting to sexually transmitted disease clinics in multiple U.S. locations. Preliminary data from the first nine months of 2003 suggest that the prevalence of fluoroquinolone (FQ) resistance among all gonorrhea isolates was 4.2%, compared with 2.2% in 2002 and 0.7% in 2001. Excluding Hawaii and California, the prevalence of FQ-resistant gonorrhea in men was just under 1% in 2003. However, among men who have sex with men (MSM), the prevalence of FQ-resistance gonorrhea was 4.9% in 2003, compared with 1.8% for the prior year.

Because the prevalence of FQ-resistant gonorrhea isolates now approaches 5% among MSM, the CDC no longer recommends FQ therapy for treating gonorrhea in this population. This parallels the CDC's recommendations for treating gonorrhea in both MSM and heterosexuals when infections were acquired in California, Hawaii, and Asia.

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

Oral agents largely have replaced the use of intramuscular antibiotics in treating gonococcal urethritis and cervicitis. Because cefixime (an oral, third-generation cephalosporin) has been unavailable for the past few years, FQs have been the drugs of choice. In 2002, the CDC reported that the prevalence of FQ-resistant gonor-

rhea had reached 5% among patients with infections acquired in Asia, the Pacific Islands (including Hawaii), and California. This level of resistance generally is used as the threshold for a change in therapy. The CDC, therefore, recommended that FQs no longer be used to treat infections acquired in these regions.

It was inevitable that FQ-resistant gonorrhea would extend beyond these specific geographic areas. The CDC now recommends ceftriaxone 125 mg intramuscularly (IM) as the preferred therapy for gonococcal urethritis acquired in these endemic regions and in all MSM; spectinomycin 2 g IM is the alternative. Unless co-infection with chlamydia has been ruled out, concomitant therapy with doxycycline or azithromycin also is necessary.

FQ therapy still may be appropriate for heterosexual patients outside the endemic regions. The CDC urges that practitioners obtain a careful travel and exposure history in patients with urethritis, and that they obtain gonorrhea cultures in cases of treatment failure to exclude the possibility of a drug-resistant infection. Fortunately, cefixime soon may again become available, and will be an acceptable oral therapeutic option for infections in MSM and in areas with high rates of FQ-resistant gonorrhea. ❖

BNP: A Hearty Diagnostic Complement to the Chest Film in Heart Failure

ABSTRACT & COMMENTARY

Source: Knudsen CW, et al. Diagnostic value of B-type natriuretic peptide and chest radiographic findings in patients with acute dyspnea. *Am J Med* 2004;16:363-368.

Recently, enthusiasm has emerged for a new investigative assay for beta-type natriuretic peptide (BNP), a cardiac neurohormone released by atrial and ventricular myocytes undergoing volume overload or stretch. BNP has been demonstrated to be of value in the diagnosis of congestive heart failure (CHF); it rises to higher levels in more severe CHF conditions and correlates with pulmonary capillary wedge pressure. To evaluate the complementary roles of BNP and chest x-ray (CXR) in the evaluation of CHF, Knudsen and colleagues studied an international cohort of emergency department (ED) patients with acute dyspnea.

A total of 880 patients was analyzed as a subset of the heralded Breathing Not Properly Multinational Study, conducted at five U.S. teaching hospitals and two academic centers in Paris and Oslo in 1999-2000. Patients were seen in these EDs with acute or suddenly worsening dyspnea. The average age was 64; 53% were male; and 56% were black. Those with acute myocardial infarction, renal failure, trauma, or pneumothorax were excluded. ED physicians performed clinical assessment and electrocardiogram interpretation. Radiologists read all CXRs. BNP levels were determined by point-of-care testing in triplicate, but investigators were blinded as to the results.

Thirty days later, the clinical, laboratory, and radiologic data for each patient were reviewed by two independent cardiologists using the Framingham scoring criteria to adjudicate each case as either dyspnea due to CHF or non-cardiac dyspnea. Based on these analyses, the most frequent final diagnosis was acute CHF in 447/880 cases (51%). Mean BNP levels were significantly higher in CHF patients than in non-cardiac dyspnea cases (689 pg/mL, vs 121 pg/mL; $p < 0.0001$). In acute CHF patients, 401/447 (90%) had BNP levels greater than 100 pg/mL, vs 108/433 cases (25%) without CHF. Presence of cardiomegaly, cephalization, interstitial edema, and alveolar edema on CXR had specificities for CHF of 80, 96, 98, and 99%, respectively. Logistic regression analysis demonstrated that BNP levels greater than 100 pg/ml enhanced the diagnostic accuracy for CHF beyond the CXR findings alone (OR 21.4, 95% CI 14.6-31.3). Further analysis revealed that a BNP level exceeding 100 pg/ml, (OR 12.3), coupled with CXR evidence of cardiomegaly (OR 2.3), cephalization (OR 6.4), and interstitial edema (OR 7.0), greatly enhanced the accuracy of CHF diagnosis beyond that attained by clinical assessment alone ($p < 0.001$ *for all ORs)*. The authors conclude that BNP measurements are complementary to CXR in the detection of CHF in acutely dyspneic patients.

COMMENTARY BY MICHAEL FELZ, MD

BNP levels generated helpful diagnostic information not available from clinical assessment alone. In fact, BNP level was the single strongest statistical predictor of acute CHF, prompting the authors to suggest that BNP assays should become routine measurements in ED patients with acute shortness of breath.

I find this data quite encouraging. It now is possible, and may be wise, to determine BNP values at the very outset of evaluation of the dyspneic ED patient, even while awaiting results from the usual clinical, laboratory, and radiologic investigations. The predictive value of elevated BNP levels greater than 100 pg/mL, and certainly higher values—such as the average of 689 pg/ml demonstrated in the 447 patients proven to have CHF—

would provide persuasive, early diagnostic data that could allow therapeutic interventions (and blessed relief from air hunger) to happen more quickly and efficiently, in the ED. Conversely, a low BNP level (less than 100 pg/ml) would steer me toward non-CHF causes of dyspnea, such as asthma or pulmonary embolus. My impression is that BNP is here to stay as a hearty complement to CXR in the diagnosis of CHF. ❖

Special Feature

Radiologic Hazards and Acute Radiation Exposure

By Theodore C. Chan, MD, FACEP

Growing concern over terrorism and the possible use of weapons of mass destruction, including nuclear and radiologic devices, has drawn attention to the need to prepare for potential, large-scale radiation incidents. Such events may cause mass injuries or have very little clinical impact, but are likely to induce widespread fear and panic due to public misinformation and preconceived notions regarding nuclear and radiologic incidents.

Widespread, uncontrolled radiation exposure has occurred in the past as a result of atomic bomb detonations and fall-out, nuclear reactor accidents, and other intentional and non-intentional mishaps with the use of radioactive material and devices. In fact, radioactive material is used widely in society at university and research laboratories, government agencies, military units, and hospitals and medical centers, where radioactive isotopes are used for various diagnostic and therapeutic purposes. Despite its use in this setting, most metropolitan-area hospitals still are not prepared adequately for a radiologic event.[1]

Radiation Physics

There are several forms of ionizing radiation that vary in their physical properties, body tissue absorption, and biologic effects. Alpha particles are a particulate emission that travel short distances in air and penetrate less than 0.1 mm in tissue. As a result, alpha particles are hazardous primarily when inhaled, ingested, or deposited on open wounds. Beta particles are also a particulate emission, but travel farther distances in air and penetrate more deeply into tissue. As a result, these particles can be hazardous with skin and eye exposure, as well as inhalation or ingestion. Similarly, neutron particulate emission can travel long distances in air and can cause injury both with external and internal contamination. Gamma rays are an electromagnetic wave energy that can travel long distances through air and penetrate most materials with the exception of lead, concrete, or steel. Gamma rays penetrate easily through tissue and can cause internal and external injury. Similarly, x-ray radiation easily penetrates tissues and deposits its energy deep in the body.

The radiation dose absorbed by specific tissues is measured in rads (or the international Gray [Gy] unit equivalent to 100 rads). For example, a fetal x-ray exposure dose from a maternal, two-view chest x-ray is 0.00007 rads.[2] Because different forms of radiation have different biologic effects at the same absorbed dose, the effective dose often is measured in rems (or the international unit sievert [sv], which is equivalent to 100 rems). When dealing with beta particles and gamma rays, the rem essentially is equivalent to the rad.[1]

Important factors minimizing the effects of radiation are distance, time, and shielding. The absorbed dose decreases with the square of the distance from the source, so that doubling the distance diminishes the dose rate to one-fourth. Radioactivity also decays with time depending on the source material and its half-life. As a result, time management of exposures remains a key component of minimizing absorption. Appropriate shielding can minimize exposure significantly. Clothing or paper can provide shielding from alpha particles. Beta particles are stopped by materials such as glass, aluminum, plastic, and other metals. Dense materials such as lead can shield tissues from gamma ray radiation.

Biologic Effects

Radiation can have both acute and long-term effects. High doses of radiation cause cellular death, resulting in acute injury. Generally, rapidly dividing cells, such as intestinal mucosa or bone marrow cells, are most vulnerable to radiation. Lower doses of radiation may not cause cell death, but can induce cellular damage that can lead to malignant transformation years later.[3]

Acute radiation exposure can result in a number of clinical syndromes, including a gastrointestinal syndrome associated with nausea, vomiting, and diarrhea from death of intestinal mucosal stem cells; hematopoietic syndrome associated with bone marrow suppression, leukopenia, and thrombocytopenia; and a cutaneous syndrome in which extensive skin injuries occur such as radiation burns, ulceration, and necrosis.[4] The table lists the clinical effects based on acute whole-body radiation exposure.

As a result of bone marrow suppression, a reduction in lymphocyte count can be seen in exposed individuals. In fact, the absolute lymphocyte count at 48 hours after exposure can be a marker of exposure, injury,

and prognosis.[6] Counts at 48 hours above 1500 cells/mm^3 indicate no significant acute injuries with an overall excellent prognosis for the patient. Lymphocyte counts from 1000-1500 cells/mm^3 indicate an acute absorbed whole-body dose of 0.5-1.9 Gy resulting in clinical symptoms but an overall good prognosis. Counts of 500-999 cells/mm^3 indicate a 2.0-3.9 Gy exposure, severe injury and fair prognosis, whereas counts of 100-499 cells/mm^3 suggest an overall poor prognosis. Counts of less than 100 cells/mm^3 indicate a radiation exposure greater than 8 Gy resulting in a high incidence of death.

Table. Clinical Effects of Radiation Exposure[5]

ACUTE WHOLE-BODY ABSORBED DOSE (Gy)	CLINICAL EFFECTS
0.05 Gy	No symptoms
0.15 Gy	No symptoms, but possible chromosomal/genetic abnormalities
0.5 Gy	Minor decreases in white blood cell and platelet counts
1 Gy	Nausea/vomiting within 48 hours
2 Gy	Nausea/vomiting within 24 hours, marked decreases in white blood cell and platelet counts
4 Gy	Nausea/vomiting/diarrhea within 12 hours; 50% mortality without medical treatment
6 Gy	100% mortality within 30 days without medical treatment due to bone marrow failure
10 Gy	Approximate survival dose with best medical therapy
10-30 Gy	Nausea/vomiting within 5 hours; 1-week latency, then manifestations of severe gastrointestinal damage, sepsis, electrolyte derangement with death likely in 2-3 weeks without medical treatment
>30 Gy	Cardiovascular collapse and central nervous system damage with death within 24-72 hours

Decontamination and Treatment

External contamination occurs with radioactive exposure to skin and clothing. Management should focus on removing and controlling the spread of radioactive materials. Removal of clothing usually results in elimination of nearly 90% of contamination in most cases.[1] Skin decontamination can be performed effectively with the use of water and detergents. Open wounds can be rinsed with saline. All contaminated materials should be placed in large, labeled plastic bags that undergo proper disposal.

Management of internal contamination focuses on a number of approaches: reduction of absorption, dilution, blockage, displacement (by non-radioactive substances), elimination from tissues, and chelation. A number of medications and chelating agents are under investigation for these uses, but many require administration either just before or within hours of exposure. Potassium iodide (KI) or iodate has been recommended after high-dose radiation exposure (i.e., detonation of a nuclear device) to prevent radioiodine from accumulating in the thyroid gland.[7] Recommended daily doses are 130 mg for adults, 65 mg for ages 3-18 years, 32 mg for ages 1 month to 3 years, and 16 mg for infants younger than one month of age. As with other agents, KI must be administered within hours of exposure.

Management of radiologic incidents requires knowledge of the radiation source, dispersal pattern, numbers of victims and associated injuries (i.e., traumatic or blast injuries in the event of a nuclear or "dirty" bomb device), and local health care facilities and resources. Patients should be decontaminated and assessed for dosage exposure, associated injuries, and clinical signs and symptoms of radiation injury. For example, patients who report nausea, vomiting, or diarrhea must be evaluated for acute whole-body exposure. Supportive measures and treatment should be initiated as dictated by the patient's presentation and available resources.

Provider safety remains of paramount importance during such events. For responding field personnel, the use of appropriate personal protective equipment, and just as importantly, time management on the scene, should focus on minimizing exposure and dosage absorbed. Recommended exposure limits for responders to radiation disaster events vary from 5-25 rem per event to exposures of no more than 0.1 Gy per hour during the event (in comparison, recommendations for total occupation exposure are 5 rem per year).[8]

After appropriate decontamination of victims, there is little risk of radiation transfer to health care facilities. In fact, medical personnel at the site of the Chernobyl disaster dealing with decontaminated victims received less than 10 milli-Gy of radiation.[1] Medical personnel still should wear protective clothing and gloves in conformity with universal precautions, and respiratory equipment and respirators generally are not required at the hospital.

Resources

Additional resources and information on radiation exposure and injuries can be obtained 24 hours a day from the U.S. Department of Energy Radiation Emergency Assistance Center/Training Site (865-576-1005). Information also is available on its website at

www.orau.gov/reacts. In addition, a handbook on medical management of radiologic casualties is available at *www.afrri.usuhs.mil*. ❖

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Physician CME Questions

8. In the recent study at Carolinas Medical Center that looked at thrombolytic treatment of acute stroke, the 36-hour rate of intracranial hemorrhage was _____ than that found in the NINDS study.
- a. 10% and lower
- b. 6% and higher
- c. 10% and higher
- d. 6% and lower

9. Which of the following statements is true regarding the treatment of gonorrhea urethritis?
- a. Fluoroquinolones are recommended for infections in men who have sex with men.
- b. Ceftriaxone is acceptable for all populations.
- c. Concomitant therapy for chlamydia is not routinely necessary.
- d. Cefixime is currently the treatment of choice for men who have sex with men.

10. In a male who has sex with other males, a correct treatment choice for gonococcal urethritis in a patient allergic to cephalosporins would be:
- a. spectinomycin.
- b. ciprofloxacin.
- c. levofloxacin.
- d. doxycycline.

11. Regarding the study by Li et al, which of the following statements is true regarding the white blood cell count of joint fluid (jWBC) obtained by arthrocentesis in patients with septic arthritis?
- a. The sensitivity of an elevated jWBC (greater than 50,000 cells/mm^3) was 46% for septic arthritis.
- b. More than one-third of patients with septic arthritis had jWBCs less than 50,000 cells/mm^3.
- c. No patients with jWBCs less than 10,000 cells/mm^3 had septic arthritis.
- d. The mean jWBC in patients with septic arthritis was 127,000 cells/mm^3.

12. In evaluation of acute dyspnea in the ED, the diagnosis of CHF is most strongly supported by:
- a. a BNP level of less than 100 pg/mL and pleural effusion on CXR.
- b. a BNP level of greater than 100 pg/mL and normal CXR.
- c. a BNP level of greater than 100 pg/mL and interstitial edema on CXR.
- d. a BNP level of greater than 100 pg/mL and cardiomegaly on CXR.

13. Alpha particle radiation:
- a. penetrates tissues most deeply.
- b. can be transmitted miles over air.
- c. can be shielded by paper or clothing.
- d. is a form of electromagnetic radiation.

Answer Key: 8. c; 9. b; 10. a; 11. b; 12. c; 13.c

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.

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.

Chest Pain in a 21-Year-Old

By Ken Grauer, MD

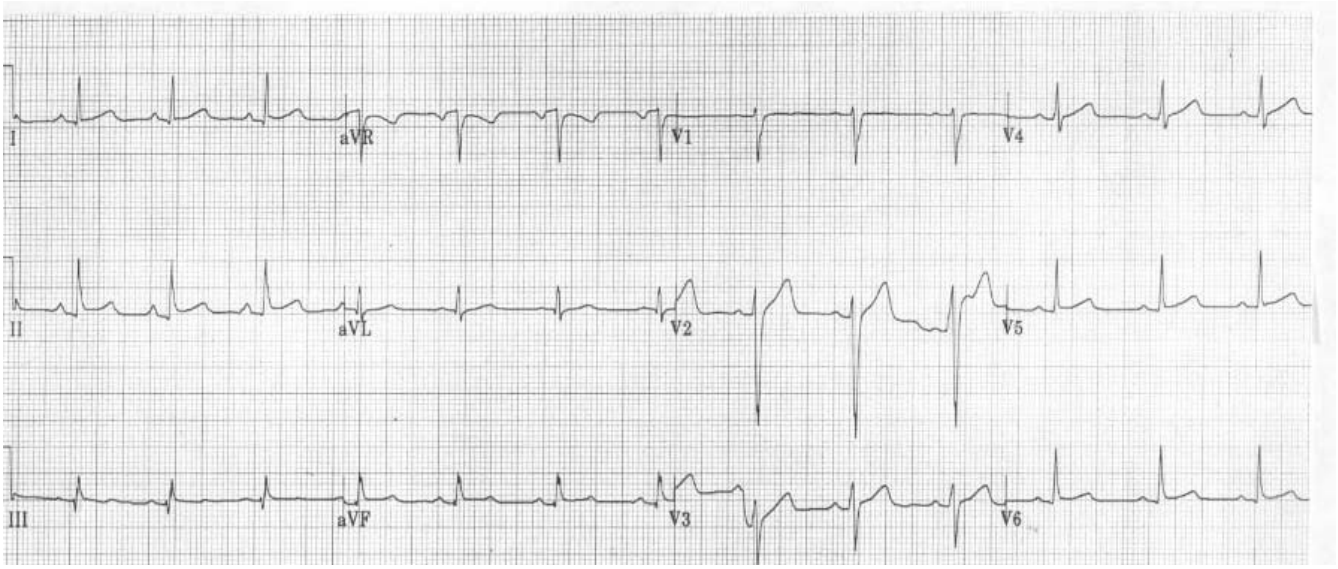

Figure. 12-lead ECG obtained from a 21-year-old male with chest pain.

Clinical Scenario: The electrocardiogram (ECG) in the Figure shows normal sinus rhythm at a rate of 80 beats/minute. The PR, QRS, and QT intervals are normal. There is an rSR' pattern in lead III. The axis is +40°. There is no sign of chamber enlargement. The most remarkable finding on this tracing is the presence of subtle but real ST segment elevation in multiple leads including I, II, aVF, and V2 through V6. The differential diagnosis includes acute infarction, a normal variant (i.e., early repolarization), and acute pericarditis.

Interpretation: The age of the patient in this case makes acute infarction much less likely, especially if there is no history of cocaine ingestion. Also, against a diagnosis of acute infarction is the surprisingly diffuse nature of ST segment elevation in the absence of Q waves, T-wave inversion, and reciprocal ST depression.

The normal variant pattern of early repolarization certainly could be the cause of ST elevation in this case. Not known from the history is whether this ST segment elevation is a new or an old finding. In view of the patient's age, the chances are good that no prior ECG had been recorded in the past. The J point ST segment elevation with an upward concavity is most likely to represent early repolarization when this ECG pattern is long standing and occurs in an asymptomatic individual in the absence of other signs of acute infarction. However, the characteristic J point "notching" that usually is seen clearly in one or more leads with early repolarization is not present in this tracing, and the history suggests that the patient has symptoms (i.e., chest pain). As a result, rather than the normal variant pattern of early repolarization, the ECG in the Figure well may represent the ECG pattern of acute pericarditis.

The initial stage of acute pericarditis (Stage I) manifests diffuse ST segment elevation, which typically may be seen in many (if not all) leads except III, aVR, and V1. Electrocardiographic pearls that support an ECG diagnosis of acute pericarditis are the fact that leads I and II tend to look similar (as they do here), as compared with the case of acute myocardial infarction in which lead III (rather than lead I) looks similar to lead II. PR segment depression (not seen here) is sometimes another subtle clue to acute pericarditis. Further support usually is forthcoming from the history (most typically revealing a preceding upper respiratory infection in an otherwise healthy young adult leading to development of pleuritic chest pain) and physical exam (the finding of a pericardial friction rub is diagnostic). ST segment elevation seen in the Figure here was new, and the patient in this case did turn out to have acute pericarditis. ❖

In Future Issues: **Fluoroquinolones**