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Cerebral Edema With Pediatric Diabetic Ketoacidosis: Who Is at Risk?

ABSTRACT & COMMENTARY

Source: Glaser N, et al. Risk factors for cerebral edema in children with diabetic ketoacidosis. *N Engl J Med* 2001;344:264-269.

THIS RETROSPECTIVE STUDY FROM THE PEDIATRIC EMERGENCY MEDICINE Collaborative Research Committee of the American Academy of Pediatrics examines the risk factors for cerebral edema (CE), an uncommon yet devastating complication in children with diabetic ketoacidosis (DKA). The authors identified 61 children with DKA complicated by CE who presented to 10 pediatric centers over 15 years. DKA with CE was defined as a serum glucose less than 300 mg/dL, venous pH less than 7.25 or serum bicarbonate less than 15, ketonuria, alteration in mental status, and either radiographically or pathologically confirmed CE or clinical improvement following specific treatment for CE (hyperosmolar therapy or controlled hyperventilation). For each child with CE, the authors identified six children as controls, three in each of two control groups. The first control group ("random controls") consisted of children with DKA who were selected randomly from all children with DKA at each center during the study period. The second group ("matched controls") consisted of children with DKA who were matched with the children with CE according to age (within 2 years), onset of diabetes mellitus (new vs established), venous pH (within 0.1), and serum glucose (within 200 mg/dL).

Data from each child's medical record were collected including demographics, serum chemistry results, treatment regimen, and changes in laboratory values during treatment. Comparison of the children with CE to the children in the random control group demonstrated that CE was significantly associated with lower initial pCO_2 (relative risk for each decrease of 7.8 mmHg was 3.4; 95% CI 1.9-6.3) and higher initial blood urea nitrogen (BUN) (relative risk for each increase of 9 mg/dL was 1.7; 95% CI 1.2-2.5). Comparison of the children with CE to the matched control group also showed that CE was associated with lower initial pCO_2 and higher initial BUN.

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INSIDE

Examination of therapeutic variables revealed that after adjustment for other covariates, treatment with bicarbonate was associated with an increased risk of CE (relative risk, 4.2; 95% CI 1.5-12.1). Smaller increases in the serum sodium concentration during therapy also were associated with CE. Interestingly, the rates of fluid or sodium administration were not associated with an increased risk of CE after adjustment for covariates.

COMMENT BY JACOB W. UFBERG, MD

The results of this excellent study agree with several previous studies showing that the initial pCO_2 and smaller increases in serum sodium concentration during therapy predict increased risk of cerebral edema. This study also refutes the belief that the rate of fluid administration and the change in serum glucose during therapy are associated with increased risk of cerebral edema. Therefore, cerebral edema in children with DKA may not be caused by a rapid decrease in extracellular osmolarity during treatment, as has been theorized previously.

Despite this study's findings, we should not jump to the conclusion that bicarbonate therapy causes cerebral edema. Cerebral edema occurs in some children prior to the initiation of any therapy. Additionally, there is the possibility that bicarbonate therapy was instituted in response to a change in the clinical status of some of the children who eventually were diagnosed with cerebral edema, even though the authors attempted to control for this. The authors note that although this is a relatively large study, it is still limited in its ability to detect some associations of smaller magnitude. They also note, "confounding factors that were not analyzed may have influenced the detected associations, particularly related to the use of bicarbonate treatment." Therefore, it remains unclear whether bicarbonate therapy, or any therapy for that matter, necessarily causes cerebral edema. What seems crystal clear is that this study, in conjunction with the prior literature, should put to rest the routine use of bicarbonate therapy in children with DKA. ❖

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Dexamethasone for Moderate Croup: Oral vs. Intramuscular

ABSTRACT & COMMENTARY

Source: Rittichier KK, Ledwith CA. Outpatient treatment of moderate croup with dexamethasone: Intramuscular versus oral dosing. *Pediatrics* 2000;106:1344-1348.

CROUP (ACUTE LARYNGOTRACHEOBRONCHITIS), commonly caused by viral infection of the upper respiratory tract, results in airway narrowing and a syndrome of barky cough, stridor, and respiratory distress in children. Current management includes cool mist therapy, and nebulized racemic epinephrine in severe cases. There has been a growing body of literature supporting the use of steroids for croup, and demonstrating benefit in terms of severity scores, need for further therapy, and hospitalization. However, the preferred route and dosing of steroids remains controversial. While most studies used intramuscular (IM) dosing, other studies found benefit with oral (PO) as well as nebulized steroids.

This single-blind, randomized study compared PO and IM dosing of dexamethasone (0.6 mg/kg up to a maximum of 8 mg) for children presenting to the emergency department (ED) with moderate croup. Moderate croup was defined as a clinical syndrome of hoarseness and barky cough associated with a history or presence of stridor or retractions. Those with mild croup (no history of stridor or retractions) and severe croup (severe retractions, cyanosis, or altered mental status), as well as those hospitalized on the initial visit were excluded.

Caregivers were contacted 48-72 hours after the initial ED visit to determine subsequent unscheduled return visits (visits to a health care facility or clinic for the same illness) and unscheduled return failures (visits in which the child needed additional treatment, including steroids, racemic epinephrine, and hospitalization), as well as the caregiver's impression of the child's symptoms (worse, same, better, or resolved).

During the 33-month study period, 1298 patients were diagnosed with croup, of whom 277 were eligible and randomized to the study (139 children to IM and 138 to PO dosing). There was no statistical difference between the two groups either in terms of unscheduled return visits (32% for the IM group; 25% for the PO group) or unscheduled return failures requiring additional treatment (8% IM vs 9% PO).

In addition, the authors conducted a subgroup analysis of those children with more severe croup (higher croup scores, illness duration < 24 hours, and racemic epinephrine administered on initial ED visit) and still found no difference between the IM and PO groups. Finally, caregivers reported their children's symptoms improved or resolved in 133 of the IM and 131 of the PO patients. Based on their findings, the authors conclude that oral dosing of dexamethasone in the setting of croup is equally efficacious to IM dosing.

COMMENT BY THEODORE C. CHAN, MD, FACEP

While the benefit of steroids in the treatment of croup is well-established, the optimal route and dosing remain to be determined. Studies have suggested benefit from parenteral, oral, and even nebulized steroid administration, as well as efficacy at varying doses (such as 0.15-0.6 mg/kg of dexamethasone).[1-3]

Because of its excellent bioavailability and long half-life, dexamethasone has been studied widely as the steroid of choice for croup. This study is one of the first investigations to compare PO and IM dosing of dexamethasone directly, and it found no difference in terms of clinical efficacy between the two routes.

Practitioners often are reluctant to administer oral medications to sick young children because of tolerability and the potential for vomiting. However, an IM injection causes pain and anxiety, and can worsen stridor in an already croupy, crying child. Injections also are not without risk for both patient and staff. In this study, the investigators crushed dexamethasone tablets into syrup and jelly, rather than using the liquid formulation. The authors reported only one episode of vomiting in a child who later tolerated a repeat PO dose. Thus, not only was the PO medication equally efficacious, it also was well-tolerated by the pediatric population.

It should be noted, however, that this study focused only on those children with moderate croup, and that the large majority of ED patients diagnosed with croup (nearly 80%) did not participate in the study. In addition, future studies are needed to examine the optimal dosing of dexamethasone given recent work suggesting good benefit with lower dosages. ❖

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Prehospital Airway Management in Head Injury—Friend or Foe?

ABSTRACT & COMMENTARY

Source: Murray JA. Prehospital intubation in patients with severe head injury. *J Trauma* 2000;49:1065-1070.

THE AUTHORS OF THIS RETROSPECTIVE, REGISTRY-BASED review of 13 Los Angeles trauma centers assessed the outcome of patients with severe head injury to determine whether prehospital intubation was associated with an improved outcome. The Los Angeles County Department of Health Services Trauma Registry was used to identify all patients with a Glasgow Coma Scale (GCS) score of 8 or less over a three-year period. Multiple demographic variables were included in the analysis. Patients were stratified into three groups by type of airway management: intubated; attempted intubation, unsuccessful; and non-intubated. Mortality was determined for each group.

A total of 894 patients met the criteria; 714 (84%) were in the non-intubated group, 81 (10%) were in the intubated group, and 57 (6%) were in the unsuccessfully intubated group. Patients requiring prehospital intubation or in whom intubation was attempted had an increased mortality (81% and 77%, respectively) compared with non-intubated patients (43%). The mortality for patients who had prehospital intubation performed

did not demonstrate an improved survival using a matched cohort of patients. The authors concluded that prehospital intubation did not demonstrate an improvement in survival, and recommended "further prospective randomized trials."

COMMENT BY MICHAEL A. GIBBS, MD, FACEP

While at first glance these results may call into question the proven benefit of early airway control and optimized oxygenation in severe head injury,[1] a closer, more critical look is revealing. First, the three treatment groups were dissimilar. The injury severity score, GCS score, and maximal oxygen intake were significantly less severe in the non-intubated group. Using a "matched" group unfortunately does not overcome this fundamental difference, and comparing the outcome of these very different groups of patients is hazardous. Second, other than "being trained in oral endotracheal intubation," there is no information on the training and skill of the paramedics providing care. The 61% overall success rate for intubation is telling. Third, intubation was performed without the aid of neuromuscular blockade. While this is the reality of most prehospital systems, there are abundant data demonstrating the poor success rate of this mode of airway control, and theoretical data suggesting that this technique may worsen outcome in head injury. Fourth, there were no data describing the number of intubation attempts, the reason for failed endotracheal intubation, or the incidence of airway-related complications.

Despite the best effort of the authors, the data do not answer the research question put forth. In fact, all that can be said about the results of this study is that patients with "really bad" head injuries die more often than do patients with injuries that are not as severe. Period. ❖

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The Magnitude of ED Overcrowding

ABSTRACT & COMMENTARY

Source: Derlet RW, et al. Frequent overcrowding in U.S. emergency departments. *Acad Emerg Med* 2001;8:151-155.

DERLET AND COLLEAGUES RECENTLY PUBLISHED THE results of a national survey of overcrowding in the emergency department (ED). Surveys were sent by mail to 836 randomly selected ED directors in all 50 states. The directors were asked to rate the severity of overcrowding in their ED, the frequency of overcrowding episodes, and the perceived causes of the problem. The respondents were asked to characterize the term "overcrowding" by wait time, bed availability, or physician workload. The directors also were asked to characterize the demographics of their population base and the characteristics of their ED.

Sixty-nine percent of the ED directors surveyed returned the questionnaire. Ninety-one percent reported overcrowding to be a problem. While overcrowding was more prevalent in EDs serving communities of 250,000 or more, it was still reported to be extremely common (87% prevalence) in EDs serving smaller populations. Overcrowding was reported with equal frequency by directors of academic, county, and private EDs. It was related to be a daily problem by 39% of directors. As related to overcrowding, two-thirds of respondents reported concern for risk of poor patient outcome and one-third reported actual instances of adverse events.

The most commonly perceived causes of overcrowding were (in order of mean importance): increased patient acuity, hospital bed shortages, increased ED volume, laboratory and radiology delays, and inadequate ED space. Interestingly, more than one-half of respondents reported that ED overcrowding had developed only within the preceding three years. The authors conclude that frequent, episodic overcrowding is a significant problem in all types of EDs.

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

Although ED overcrowding seemed to disappear from the national radar in the mid-1990s, the issue once again has gained considerable media attention. Unfortunately, most of our information about this issue is based on anecdotal evidence, and there have been no recent studies of the scope or magnitude of the problem. Derlet and colleagues recognize that ED overcrowding cannot be addressed until it is quantified, and this survey is the first recent, large-scale effort to amass objective data regarding the problem.

Although this study was carefully executed and the response rate is excellent for a mail-in questionnaire, the authors acknowledge a number of limitations inherent to this type of research. There is, foremost, no uniform definition of "overcrowding." Directors were asked to define the problem themselves and to relate their perception of its magnitude. In addition, there was no attempt to verify the accuracy of the information provided. Despite these shortcomings, Derlet's study clearly

documents the perception of a national health care crisis and puts the issue clearly on the table for future research and efforts at reform. ❖

Special Feature

Emerging Infectious Diseases

By David J. Karras, MD, FAAEM, FACEP

MANY NOTABLE PHYSICIANS PRACTICING IN THE MID-20th century confidently predicted that infectious diseases would soon be eliminated as significant factors in the landscape of modern life. New antibiotics enabled people to survive previously fatal illnesses and few bacteria demonstrated any resistance to our arsenal of antimicrobials. The advent of effective public sanitation, organization of public health departments, modern food processing techniques, and development of vaccines led to proclamations of victory in the war against microbes.

By the end of the 20th century, however, it was obvious that the victory dance was premature and highly naïve. Mortality rates related to infectious diseases actually rose 58% between 1980 and 1992, and infectious diseases remained the third leading cause of death in the United States. On a global scale, the 1990s were proclaimed the "new era of plagues" and the World Health Organization stated that "the threat of serious global pandemic with profound world-wide human devastation is more significant than at any time in history."[1]

Many of the factors implicated in the resurgence of infectious disease as a paramount threat to public health are, in fact, related to technological advances. Global movement of both people and food has become widely available and essentially instantaneous, allowing for rapid transmission of devastating illnesses.

Sexual promiscuity is common, despite the HIV epidemic. Americans still tend to have sex at younger ages and with more partners than in previous generations (as evidenced by the dramatic rise in serologic evidence of exposure to herpes).[1]

The greatest threat to our complacency regarding infectious disease, however, is the rapid emergence of antibiotic resistance. While bacteria always have had the ability to develop mechanisms allowing evasion of antimicrobial effects, there is overwhelming evidence that abuse of antibiotics by both physicians and patients has greatly accelerated this natural evolutionary process. The crisis of antibiotic resistance that we now face is to a great degree a crisis of our own making.

Of course, new infectious diseases have always cropped up from time to time. Whereas some diseases are predictable, others make dramatic and unforeseeable entrances. To detect and counter these threats, we rely almost entirely on our public health departments, which have been strongly criticized by the Institute of Medicine for lacking comprehensive surveillance systems.[2] In the remainder of this article, a sample of some of the emerging infections that have the potential to profoundly affect the practice of medicine will be reviewed.

Foodborne Illnesses

At least 13 new foodborne illnesses have been identified over the past 20 years.[3] A number of factors conspire to make these illnesses extremely difficult to control: Most of the organisms have reservoirs in healthy animals, they often survive traditional food preparation techniques, and contamination often does not alter the appearance or taste of the food. Outbreaks usually can be identified only after hundreds (or thousands) of individuals have ingested an organism. Contamination in a single processing plant can pose a national or global threat, and multiple government agencies (mostly understaffed) have jurisdiction over the food chain. The antibiotics routinely fed to livestock appear to have enhanced the development of resistant bacterial strains that routinely contaminate food products.

While *Salmonella* has historically been the most common foodborne illness, data from the 1998 FoodNet surveillance program reveal *Campylobacter* to be the greatest offender, accounting for 40% of identifiable enteropathogens compared to 28% for *Salmonella* and 15% for *Shigella*.[4] *Camplylobacter*, which was not even identified as a stool pathogen until 1972, now is believed to be responsible for almost 2.5 million cases of bacterial enteritis per year. The organism has reservoirs in the intestines of poultry and cattle, as well as in fresh water (which often is contaminated by animal waste). Although treatment for campylobacteriosis is primarily supportive, there is good evidence that patients with moderate-to-severe symptoms (high fever, bloody stool, and/or frequent bowel movements) benefit significantly from antibiotic therapy. Quinolones are the drugs of choice for treatment of this and most other causes of bacterial enteritis; azithromycin or trimethoprim/sulfamethoxazole are good alternatives.[5]

Escherichia coli O157:H7 is a toxin-producing enteropathogen that has been responsible for a number of serious outbreaks of hemorrhagic colitis. The primary reservoir of the organism is in cattle intestines, and from there it can go on to contaminate not only meat, cheese,

and unprocessed milk, but also well water and fresh fruit that comes in contact with contaminated soil. In the typical *E. coli* O157:H7 infection, a patient develops cramps and diarrhea 3-4 days following ingestion of contaminated food or water. Stool becomes frankly bloody within 1-2 days. The duration of illness is typically a week, after which most patients will have a complete recovery. Between 5% and 10% of patients (usually children or older individuals) develop hemolytic-uremic syndrome (HUS), which results in renal insufficiency, chronic renal failure, stroke, or death in about one-half of affected individuals.[6] The risk of HUS is markedly greater in patients treated with antibiotics (specifically trimethoprim/sulfamethoxazole).[7] Antimicrobial agents, therefore, are not recommended for patients with hemorrhagic enteritis deemed likely to be associated with an *E. coli* epidemic.

Rodent-Borne Illness: Hantavirus

In 1994, a new disease was reported in New Mexico that caused 17 deaths in mostly healthy young couples within a matter of weeks. The etiologic agent was found to be the hantavirus, which was known to have been the cause of hemorrhagic fever during the Korean War but had not been previously known to cause human illness in this hemisphere. The virus, it was discovered, was ubiquitous in mice in the southwestern United States. Because of a few particularly warm and wet El Niño seasons, the local mouse population had exploded during the year of the initial outbreak.[8] The resulting disease, termed hantavirus pulmonary syndrome (HPS), continues to occur sporadically and carries a 43% fatality rate.

HPS presents dramatically with a flu-like prodrome that is abruptly followed by pulmonary edema and often cardiogenic shock. No radiographic or laboratory abnormalities are pathognomonic of the disease, and diagnosis requires serologic testing.[9] Antimicrobial therapy is not effective, although extracorporeal membrane oxygenation appears to improve the prognosis of patients with severe disease. The only effective mechanism of controlling hantavirus infections appears to be eradication of mouse infestations, which obviously is quite difficult. As of last year, 234 cases of HPS had been confirmed, and the disease had been reported throughout the United States, with the notable exception of the southeastern states.[10] Awareness of this syndrome, therefore, is now essential for physicians throughout the country, particularly those working in rural areas.

Tick-Borne Illness: Ehrlichiosis

Ehrlichia is an obligate intracellular bacterium that was not known to cause disease in humans until 1987. Since that time, more than 2000 cases of human disease have been reported in this country. *E. chaffeensis* is the cause of human monocytic ehrlichiosis (HME), which is transmitted by the Lone Star tick and American dog tick. Human granulocytic ehrlichiosis (HGE) is associated with *E. ewingii* and is spread by deer ticks.[11] The disease appears to be even more common than previously recognized, as one recent study found that 18% of patients with flu-like symptoms living in a tick-endemic region had serologic evidence of acute ehrlichiosis.[12]

The typical ehrlichiosis patient is a middle-age male living in the south-central United States who recently has been exposed to ticks. The disease presents with flu-like symptoms—fever, myalgias, and malaise. Later, abdominal pain and vomiting may be seen and a nonspecific, indistinct rash may be detected. About 20% of patients develop mental status changes and/or meningismus; lumbar puncture reveals pleocytosis and elevated protein in the cerebrospinal fluid. Other laboratory abnormalities include leukopenia with a left shift, thrombocytopenia, and hepatic transaminitis. The disease may have a fulminant course with multisystem organ involvement. Although a 2-10% mortality rate has been reported, it is likely that many milder cases go undetected.[13]

Presently, ehrlichiosis is diagnosed by a compatible history, a peripheral blood smear showing morulae (microcolonies of organisms) within neutrophils or monocytes, and characteristic rises in serum antibodies. At least one-half of patients may be managed as outpatients. Tetracycline is highly effective therapy, as are chloramphenicol (in children), trovafloxacin, and rifampin.

Future Directions

As stated in the report from the Institute of Medicine, when pitted against microbes physicians have mainly their wits to rely upon.[2] Pharmaceutical science clearly will not eradicate infections in the near future as microorganisms always seem to find a way to dodge whatever ammunition is fired. New infectious diseases inevitably will emerge and existing diseases will evolve. Our best bets are to support regional and national surveillance programs. As evidenced by the response of the Centers for Disease Control and Prevention and local agencies to the hantavirus outbreak, such programs have the ability to promptly recognize and respond to serious public health threats, if appropriately funded. Individual physicians need to keep abreast of emerging infections. The *Annals of Emergency Medicine* regularly reviews articles from *Morbidity and Mortality Weekly Report* (*MMWR*) that are relevant to emergency physicians; *MMWR* itself is available on-line free of charge

(http://www.cdc.gov/mmwr). Finally, it is critical that the health care community recognize its contribution to the problem of widespread antibiotic resistance and develop a healthy reluctance to prescribe antibiotics unless they clearly are indicated. ❖

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CME Questions

32. Development of cerebral edema in children with DKA was associated with which of the following treatments?
- a. Bicarbonate
- b. Hyperventilation
- c. Rate of administration of normal saline
- d. Rate of correction of serum glucose

33. In the recent study on dexamethasone for croup, Rittichier and Ledwith found:
- a. Oral dexamethasone was less efficacious than intramuscular steroids for cases of mild croup (children with no history or presence of stridor or retractions).
- b. Nebulized and oral dexamethasone both were *not* effective in the treatment of croup.
- c. Oral dosing was *not* well-tolerated by children and resulted in emesis for the majority of patients.
- d. Oral dexamethasone was as efficacious as intramuscular dexamethasone in cases of moderate croup (children with a history or presence of stridor or retractions).
- e. Dexamethasone should be reserved for those children with severe croup.

34. Regarding the study by Murray examining prehospital airway management, which of the following can be concluded?
- a. Prehospital intubation should *not* be attempted in head trauma patients.
- b. Paramedic training level predicted successful intubation.
- c. Nasotracheal intubation was the best route for head-injured patients.
- d. Data on the number of intubation attempts and reasons for failed intubation were not provided, and would have been helpful when trying to interpret the results.

35. Which of the following is the most common bacterial cause of foodborne illness?
- a. *Salmonella*
- b. *Shigella*
- c. *Campylobacter*
- d. *Yersinia*

36. The most appropriate empiric management of suspected *E. coli* O157:H7 enteritis is:
- a. trimethoprim/sulfamethoxazole therapy.
- b. fluoroquinolone therapy.
- c. azithromycin therapy.
- d. withholding antibiotic therapy.

37. Hantavirus pulmonary syndrome (HPS) has been described in all parts of the United States except the:
- a. Northeast.
- b. Southeast.
- c. Midwest.
- d. Southwest.

38. Ehrlichiosis is characterized by:
- a. a highly distinctive rash.
- b. very high fatality rates.
- c. nonspecific flu-like illness.
- d. recent exposure to infected rodents.

Lateral Infarction? No *and* Yes!

By Ken Grauer, MD

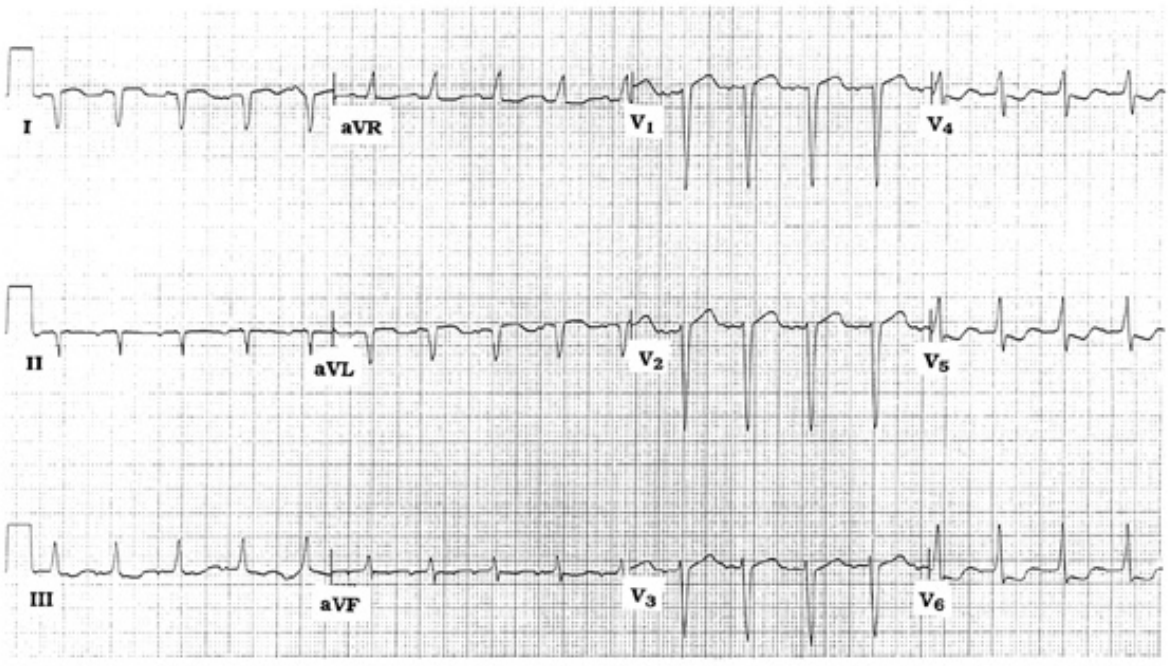

Figure. ECG obtained from a 63-year-old woman with atypical chest pain.

Clinical Scenario: The ECG shown in the Figure was obtained from a previously healthy 63-year-old woman with atypical chest pain. The answer to the question we raise in the title of this ECG Review (*Is there lateral infarction?*) is no *and* possibly yes! Can you explain?

Interpretation: There are several unusual findings on this tracing. The first of these becomes evident when assessing the rhythm: P waves are *not* upright in lead II. Although atrial activity is not readily discernible in lead I, the negative QRS complex and T wave in this lead—in association with the upright QRS complex in right-sided lead aVR (a lead which should normally show complete negativity)—strongly suggest either dextrocardia or lead misplacement as the cause of the unusual pattern. Normal R wave progression in the precordial leads rules out the former (since dextrocardia would result in *reverse* R wave progression). Confirmation of lead misplacement as the cause of this pattern is easily forthcoming by repeating the ECG after verifying that all limb leads are correctly placed. The deep Q waves (QS complexes) in leads I, II, and aVL disappeared, and a normal upright P wave was seen in lead II on repeat ECG.

Precordial leads are *unaffected* by limb lead misplacement. Thus, the worrisome ST segment sagging depression that is present in leads V_4 through V_6 of the Figure was unchanged on repeat ECG, suggesting a possible acute coronary syndrome. There is, therefore, *no* evidence of lateral infarction from inspection of leads I and aVL (because lead misplacement negates the meaning of the findings in these leads)—but ST depression consistent with possible acute infarction is present in the lateral precordial leads. ❖

In Future Issues:

Causes of ST Segment Elevation