Enclosed in this issue: Trauma Reports

EMERGENCY MEDICINE ALERT

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Ottawa Ankle Rules Applied to Children with Mixed Results

ABSTRACT & COMMENTARY

Source: Clark KD, et al. Evaluation of the Ottawa ankle rules in children. *Pediatr Emerg Care* 2003;19:73-78.

THE OTTAWA ANKLE RULES (OAR) FIRST WERE INTRODUCED BY IAN Stiell, et al in 1993.[1] Stiell's goal was a rule with 100% sensitivity for clinically significant fractures (> 3 mm fracture fragment), while not missing any clinically important fractures. The OAR met this goal with an associated specificity of 40% (i.e., the clinician will still obtain many negative radiographs, but no clinically significant fractures will be missed). The OAR, as originally tested, excluded patients younger than 18 years of age. Because there are significant developmental and physiologic differences between children and adults, including age-related variations in fracture epidemiology, the potential for physeal growth plate injury, and the inability of young children to localize pain, it has remained a question whether these rules can be applied to a pediatric patient population. Salter-Harris injuries went unaddressed in Stiell's study, as all patients were older than 18 and thus not at risk for such injuries. Finally, while fractures smaller than 3 mm were considered clinically insignificant in adults, it is unclear whether this same criterion can be applied to children.

The purpose of this prospective study was to evaluate the OAR in children younger than 18 years presenting to a pediatric ED. All fractures were considered clinically significant (specifically including Salter-Harris I [SHI] injuries and avulsion fractures < 3 mm). Data were collected on 195 patients enrolled in the study from April 1995-June 1997. Mean patient age was 12.6 years. Forty fractures (21%) were ultimately identified, with SHI fractures of the distal fibula being the most common fracture (15/40 [38%]). Thirty-five patients were excluded from sensitivity and specificity calculations (the majority due to incomplete data collection forms). Overall, the OAR yielded a sensitivity of 83% (95% CI, 65-94%) and a specificity of 50% (positive predictive value = 28%, negative predictive value = 93%). When patients with avulsion fractures were excluded, the sensitivity and specificity remained essentially unchanged. Utilizing the OAR in this group would have decreased the number of radiographs by 44%, but five fractures (17%) would have been missed. Using a logistic regression method, the authors found three characteristics in this age group that could have increased sensitivity to 93% (with a specificity of 25%). These alternative three characteristics were: inability to walk immediately after the event, tender deltoid ligament, and swelling at the distal tibia. Using these revised OAR, the authors conclude that they could reduce unnecessary radiographs by 22%, but admit that they would have missed two fractures in doing so.

COMMENTARY BY ANDREW D. PERRON, MD, FACEP

There is no question that the OAR have become widely disseminated and adopted throughout the field of emergency medicine. Besides reducing the number of unnecessary radiographs, they can speed a patient's course through the ED, ultimately resulting in improved ED throughput. The applicability of these rules to pediatric patients, as well as what constitutes a clinically significant fracture in this population, remains subject to debate. Two prior prospective studies have been published regarding the applicability of the OAR in children.[2,3] Both studies found 100% sensitivity in predicting clinically significant fractures. One small study[2] included all fractures as clinically significant, while another, large series[3] excluded specifically the fractures included in this study (SHI and avulsion < 3 mm). The authors of this current study feel that these reported sensitivities are overly optimistic, given the question of whether all fractures are clinically significant, and whether the OAR successfully can be applied in children.

The orthopedic literature supports the belief that most SHI injuries resolve without any growth disturbance. Further, the radiographic identification of these injuries frequently is difficult to make acutely, and often becomes apparent only on subsequent radiographs. In clinical practice, avulsion fractures smaller than 3 mm are not intervened upon, even in the pediatric patient population. Thus, the two injuries that potentially can be missed remain clinically insignificant in the pediatric patient population. In regard to the revised OAR that Clark has suggested based on logistic regression, they have intuitive merit. (When was the last time you could get a 6-year-old to decide if he or she had posterior tibial tenderness limited to the distal 6 cm of the bone?) A prospective evaluation of these three criteria would be a welcome addition to the clinician's armamentarium in evaluating ankle injuries in children. ❖

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Burst Steroid Therapy for COPD Exacerbations

ABSTRACT & COMMENTARY

Source: Aaron SD, et al. Outpatient oral prednisone for emergency treatment of chronic obstructive pulmonary disease. *N Engl J Med* 2003;348:2618-2625.

THIS PROSPECTIVE RANDOMIZED, DOUBLE-BLIND, placebo-controlled trial from Ian Stiell's group looked at the use of prednisone 40 mg daily for 10 days vs. placebo in 147 patients presenting to any of 10 emergency departments (EDs) who were well enough to be discharged after treatment for exacerbation of their chronic obstructive pulmonary disease (COPD). Exacerbation was defined as a recent increase in at least two of the following three criteria: breathlessness, sputum volume, or sputum purulence. Definitions of COPD were reasonably stringent, and an effort was made to exclude asthmatics. Other exclusion criteria centered on coexistent disease (active pneumonia or congestive heart failure; uncontrolled diabetes mellitus; or liver/kidney failure) or recent use of oral/intravenous steroids (prior 30 days). Patients were randomized at discharge from the ED, and also were given a 10-day course of oral antibiotics, as well as 30 days of therapy with inhaled albuterol and ipratropium bromide with spacers. Inhaled steroids were continued at discharge if they were part of the patient's medical regimen at the time of enrollment; both groups were equivalent in the use of these agents.

The main outcome measure was the relapse rate at 30 days, and this was rigorously verified. The prednisone group had a significantly lower relapse rate than did the placebo group (27% vs 43% for placebo, P = 0.05; relative risk of 30-day relapse after prednisone therapy 0.63 [95%CI: 0.40-1.01]). Time to relapse for 25% of the patients also was delayed significantly in the prednisone group (23 days vs seven days in the placebo group, P = 0.04). At 10 days, the prednisone group showed significant improvement in lung function (forced expiratory volume in one second) and subjective dyspnea scores. There was no difference between groups, however, in health-related quality-of-life scores; the authors linked this to significant increases in appetite (46% vs 22%, P = 0.003), weight gain (13% vs 1%, P = 0.01) and insomnia (48% vs 21%, P = 0.001), as well as nonsignificant trends toward a higher rate of depression and anxiety, in the prednisone group. The authors concluded that 10 days of prednisone therapy in patients discharged from the ED with exacerbations of COPD reduces the risk of relapse at 30 days, and offers a small advantage over placebo.

■ **COMMENTARY BY RICHARD A. HARRIGAN, MD**

This well-designed trial offers guidance as to how to use oral steroids in patients we are discharging from the ED after treatment for their COPD exacerbation. A 10-day "burst" of prednisone without taper offered improvement in subjective and objective indices of lung function, and a decreased relapse rate. Other studies have shown improvement in disease with prolonged (two weeks as good as eight weeks) steroid therapy with taper vs. placebo, when begun in the inpatient setting.[1] Hyperglycemia requiring treatment was found to be a significant downside of steroid therapy in one study, with a 15% rate in the steroid group vs. a 4% rate in the placebo (p = 0.02).[1] This study by Aaron and colleagues did not specifically look at hyperglycemia; two patients with diabetes in each group reported high blood sugars, but the incidence of unreported hyperglycemia was unknown. The lack of improvement in health-related quality of life suggests we should warn our patients discharged on prednisone about not only the benefits, but the risks[2]; specifically those identified in this study—that they may experience an increase in appetite, weight gain, and difficulty sleeping. It also would be prudent to warn patients—especially diabetics—about the possibility of hyperglycemia. ❖

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Imaging in Low Back Pain: Is X-ray or Rapid MRI Better?

ABSTRACT & COMMENTARY

Source: Jarvik JG, et al. Rapid magnetic resonance imaging vs radiographs for patients with low back pain: A randomized controlled trial. *JAMA* 2003;289:2810-2818.

THIS RANDOMIZED, CONTROLLED TRIAL COMPARED rapid magnetic resonance imaging (MRI) with plain radiographs as the initial diagnostic imaging test in patients with low back pain. Patients 18 years and older were recruited from four different imaging centers where the patient's physician had ordered radiographs to evaluate their low back pain. The primary outcome measure was the modified Roland back pain disability scale, done 12 months after randomization, with higher scores signifying worse function. Secondary outcome measures included back pain scores; the Medical Outcomes Study 36-Item Short Form Health Survey; days of reduced or lost work; patient satisfaction with care; reassurance and preference scores; and costs.

At 12 months, primary outcomes of functional disability were obtained from 89% of the 380 patients enrolled. The baseline characteristics of the radiograph and rapid MRI groups were similar. The 12-month modified Roland score in the radiograph group was 8.75 vs.

9.34 in rapid MRI group (mean difference, -0.59; 95% CI -1.69 to 0.87). The mean differences in the secondary outcomes were not statistically significant. Ten patients in the rapid MRI group vs. four in the radiograph group had lumbar spine operations (risk difference, 0.34; 95% CI, -0.06 to 0.73). The rapid MRI strategy had a mean cost of $2,380 vs. $2,059 for the radiograph strategy (mean difference, $321; 95% CI, -1100 to 458).

COMMENTARY BY STEPHANIE ABBUHL, MD

This well-done study showed that substituting lumbar spine radiographs with a rapid MRI scan in an outpatient population resulted in no long-term difference in disability, pain, or general health status. On the other hand, it does not appear that rapid MRI causes harm or greatly increases costs, and it provides more reassuring information for both patients and physicians (but does not increase overall satisfaction). Not surprising, there was a trend toward more lumbar spine operations in the rapid MRI group and these patients did not have improved outcome scores when compared to those who did not have surgery.

From the ED vantage point, one obvious question is whether the same results would be found in our patients. Interestingly, the patients were referred not only from primary care physicians, but half were referred from rheumatologists, physical medicine specialists, and other provider types. While there is limited clinical information about the patients in this study, the results appear to lend more evidence for our practice of avoiding time-consuming and expensive imaging for low back pain when there are no "red flags" on either history or physical exam. We already knew that plain radiographs are infrequently necessary for non-trauma related back pain, and now we know that more is probably not better. ❖

Special Feature

Hyperkalemia: Electrocardiographic Recognition and Initial Therapeutic Considerations

By William J. Brady, MD

Hyperkalemia is an electrolyte disorder with life-threatening potential. The spectrum of clinical presentation is wide, ranging from asymptomatic laboratory discovery to cardiac arrest. The most common cause is red blood cell hemolysis, which occurs after the patient's blood sample has been obtained. Hyperkalemia also frequently is encountered in patients with renal failure (both acute and chronic); diabetic ketoacidosis and other acidotic states; digoxin toxicity; type IV renal tubular acidosis; and medication-related issues (agents that affect kidney function or the renal reclamation of potassium).

The electrocardiographic manifestations of hyperkalemia may involve all phases of the cardiac impulse. The expected progression of electrocardiographic changes associated with hyperkalemia is well described.[1] *(See Table 1.)* Significant variation may be found among patients at any particular serum potassium level, however. Furthermore, the electrocardiogram (ECG) may not demonstrate classic abnormality in all patients;[2-5] in fact, the ECG may appear normal, nonspecifically abnormal, or may reveal unusual abnormalities such as a heart block and bundle branch block.

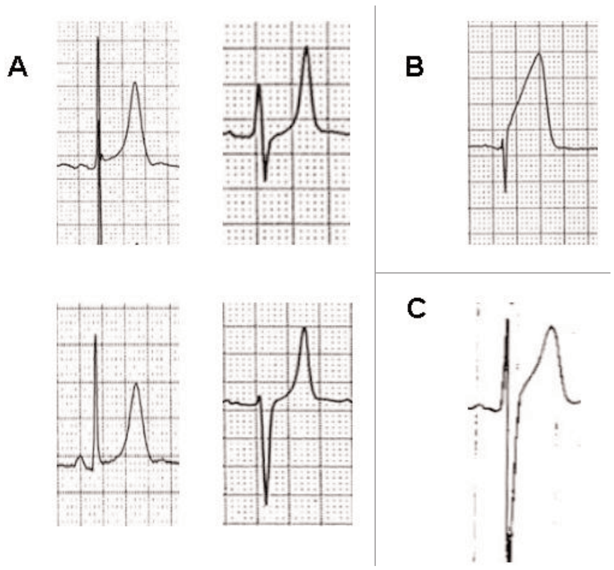
Table 1. Electrocardiographic Manifestations of Hyperkalemia Relative to Serum Potassium Level

SERUM POTASSIUM (MEQ/L)	ELECTROCARDIOGRAPHIC MANIFESTATIONS
5.5 - 6.5	Prominent T waves • Tall, narrow, symmetric • Most prominent in precordial leads
6.5 - 8.0	Decreased P wave amplitude Prolonged PR interval QRS complex widening • Minimal-to-sine wave configuration Prominent T wave Dysrhythmia • Atrioventricular block • Intraventricular block • Bradycardia • Ventricular ectopy
8.0 mEq/L and higher	Sinoventricular rhythm • Absence of P wave • QRS complex widening • Progressing to sine wave QRS complex widening Ventricular tachycardia Ventricular fibrillation Asystole

Characteristic Changes in the ECG

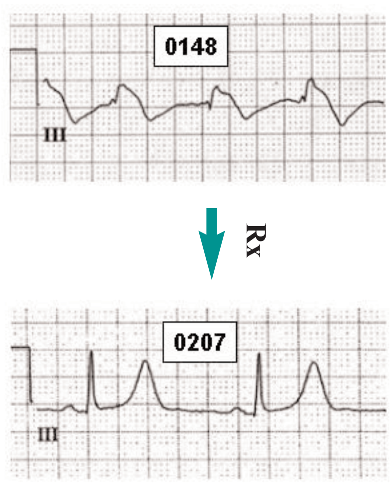
Peaked or prominent T waves in the precordial leads are among the earliest and most common findings on the ECG. Modest increases in the serum potassium enhance or accentuate repolarization of the myocyte, which are manifested electrocardiographically by alterations in the T wave.[2] *(See Figure 1.)* The T wave becomes prominent and is described as tall and narrow with a symmet-

Figure 1. Prominent T Waves of Varying Causes



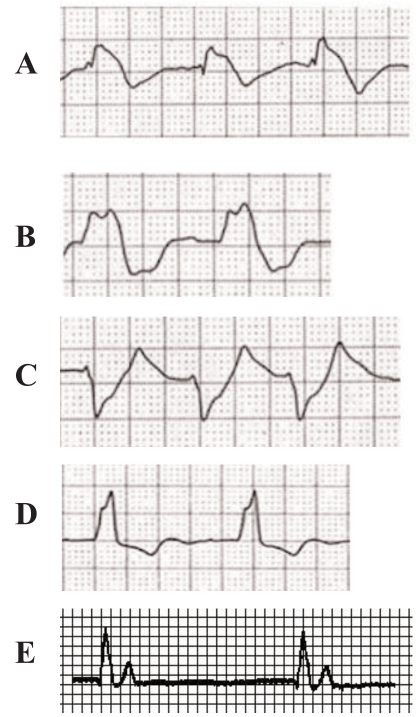
A—Hyperkalemia. Note the tall, narrow, symmetric structure. **B**—Acute myocardial infarction. Note the tall, broad, asymmetric structure. **C**—Benign early repolarization. Note the associated ST segment elevation at the J point with concave morphology.

Figure 3. Improvement of Hyperkalemic Changes with Therapy



Initial ECG (lead III shown) of a patient with hyperkalemia. On presentation at 0148, the rhythm is slow with a markedly widened QRS complex. With therapy, the QRS has markedly narrowed at 0207; prominent T waves are seen.

Figure 2. Wide QRS Complexes of Varying Causes



Hyperkalemia seen in **A, B**, and **C.** The QRS complex is markedly widened with a partial sinusoidal configuration, consistent with the sinoventricular rhythm of pronounced hyperkalemia. **D**—Wide QRS complex of bundle branch block. **E**—Wide QRS complex of idioventricular rhythm.

ric structure. The polarity of the T wave also may change, particularly in patients with left ventricular hypertrophy, with the normally inverted lateral T waves becoming upright or "pseudonormalized."[4]

Continued increase in the serum potassium will produce a slowing or prolongation of cardiac conduction. All cardiac myocytes are sensitive to elevated potassium levels, but atrial tissue is significantly more sensitive. The P wave will lessen in amplitude early in the process, with complete disappearance as the potassium rises. Despite the electrocardiographic loss of the P wave, sinus rhythm continues with maintained sinus node activity.[6] In fact, sinus impulses (bypassing the atria) are conducted along internodal tracts directly to the atrioventricular node, creating the "sinoventricular" rhythm of hyperkalemia.[2,6] At progressively higher serum levels, the QRS complex widens *(See Figure 2),* at times resembling a bundle branch block. Eventually, the QRS complex blends with the T wave, forming a "sine-wave" appearance on the ECG. Progressive increases in the potassium level eventually result in ventricular fibrillation and asystole.[4]

Treatment of Hyperkalemia

The resuscitative management of hyperkalemia *(See Table 2)* is guided in large part by the patient's clinical situation, including the electrocardiographic findings; in fact, the ECG should guide both the urgency as well as the magnitude of therapy. The goals of therapy are a reduction of the serum potassium level coupled with a

Table 2. Treatment Options in the Setting of Hyperkalemia

AGENT	MECHANISM	DOSE	RESPONSE	CAUTION
Calcium	Membrane stabilization	10 mL IV	Transient Onset—within 10 min Duration—30 min	Cautious use in digoxin toxicity
Insulin/glucose	Intracellular shift Cellular glucose pump	Regular insulin 10 units IV plus Glucose 50 g IV	Transient Onset—5-20 min Duration—60 min Magnitude—1.0 mEq	Monitor for hypoglycemia
Magnesium	Intracellular shift Antidysrhythmic	1 to 2 grams IV	Transient Onset—5 to 20 min Duration—variable Magnitude—0.5 mEq	Monitor for respiratory depression
Albuterol	Intracellular shift cAMP pump	0.5 mg in 100 mL NS/15-30 min IV 10-20 mg in 4 mL NS over 10 min nebulized	Transient Onset—5-10 min Duration—30 min Magnitude—0.5-1.0 mEq	Monitor for tachycardia
Other sympatho-mimetic agents (e.g. epinephrine)	Intracellular shift	1 mg (1:10,000) IV	Transient Onset—immediate Duration—unknown Magnitude—unknown	Epinephrine only in cardiac arrest scenarios
Sodium bicarbonate	Intracellular shift	1mEQ/kg IV Onset—5-10 min Duration —variable Magnitude—0.5 mEq reduction/ 0.1 pH increase	Transient	Significantly more effective in acidotic states Beware volume overload
Saline	Intracellular shift and removal (via urinary flow)	Adequate infusion to establish urinary flow	Permanent	Monitor for pulmonary edema; only useful with intact renal function and urine production
Polystyrene binding resin	Removal (via gut)	30-60 g PO/PR	Permanent Magnitude—0.5-1.0 mEq	Potential for aspiration in altered mentation patients
Hemodialysis	Removal		Permanent Magnitude—50 mEq	Requires sub-specialty consultation
Peritoneal dialysis	Removal		Permanent Magnitude—10 mEq	Requires presence of peritoneal catheter

stabilization of the myocardial cell membrane. The serum potassium temporarily is reduced with a transient shift of the electrolyte intracellularly, and permanently lowered with potassium removal from the body.

Stabilizing the Cardiac Membrane. Response to therapy is often prompt with real-time changes noted on the electrocardiographic monitor. *(See Figure 3.)* The most appropriate initial medication is calcium, which works by restoring a more appropriate electrical gradient across the cell membrane. In essence, calcium "fools" the cell into thinking that a more "normal" electrical difference exists between the intracellular and extracellular compartments. Intravenous administration of calcium will result in a transient narrowing (lasting no longer than 30 minutes) of the QRS complex, and thus is most appropriately given to the patient with a widened QRS complex. Note that calcium does not cause intracellular shift of potassium. Calcium chloride (13.6 mEq/10 mL) contains roughly three times the elemental calcium of that in the gluconate preparation (4.6 mEq/10 mL); calcium chloride should be administered through a large peripheral vein, if possible, in that it is highly sclerosing. The dose is 10 mL IV over one minute in the patient with spontaneous circulation; with cardiac arrest, a similar dose is given via rapid IV push. The maximum dose is 20 mL within any given 30-minute period; repeat administrations may be required. Extreme caution is advised in the setting of hyperkalemia related to digoxin toxicity; anecdotal reports suggest an increased tendency towards asystole in this clinical setting.

Transcellular Potassium Shift. Several agents are capable of transiently moving potassium from the extracellular to intracellular space. *(See Table 2.)* This intracellular shift is short-lived, yet temporarily will reduce cardiac irritability and stabilize the patient while more definitive therapies are arranged. These medications include glucose/insulin, beta-adrenergic agonists, magnesium, sodium bicarbonate, and intravenous saline. Note that the potassium-lowering effect of these various therapies is transient, with repeat administration necessary if hemodialysis has not been initiated.

Potassium Removal. Complete and permanent removal of potassium from the body is accomplished via furosemide-hastened saline diuresis, binding resins, and dialysis. *(See Table 2.)* Hemodialysis is the treatment of choice in such situations and should be employed in the vast majority of patients who presented with a sine-wave QRS complex or who have experienced cardiac arrest related to hyperkalemia. Peritoneal dialysis may be used, yet it removes less potassium over a much longer period of time compared with hemodialysis. ❖

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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.

Physician CME Questions

19. The Ottawa ankle rules for radiography:

a. are not applicable in sports-related injuries.
b. are specific but not sensitive.
c. are not meant for use in the emergency department.
d. have been generalized to children with variable success.

20. Patients discharged from the ED with 10 days of prednisone for treatment of acute exacerbations of their chronic obstructive pulmonary disease (COPD) should expect:

a. weight loss.
b. a higher incidence of pneumothorax.
c. increased appetite.
d. fewer ED visits for COPD over the next 12 months.

21. In the study comparing rapid MRI to radiographs in patients with low back pain, the following results are true *except*:

a. Back-related disability was similar in both groups at 12 months.
b. Measures of pain were similar in both groups at 12 months.
c. Despite a higher rate of surgery in the rapid MRI group, the outcomes were not better in this group.
d. The mean cost of health care services in patients randomized to rapid MRI was significantly higher than the radiograph group.

22. Chose the *incorrect* pairing of medical therapy and clinical issue in the management of hyperkalemia:

a. Sodium bicarbonate/best in acidotic states
b. Calcium/only in digoxin toxicity
c. Magnesium/in all treatment scenarios
d. Polystyrene binding resin/slow therapeutic response

23. Prominent T waves as seen in hyperkalemia typically are:

a. symmetric.
b. wide.
c. evident only after loss of P waves.
d. not seen in patients with renal failure.

Answer Key: 19. d; 20. c; 21. d; 22. b; 23. a.

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.

ECG Review

Pulmonary Pattern and What Else?

By Ken Grauer, MD

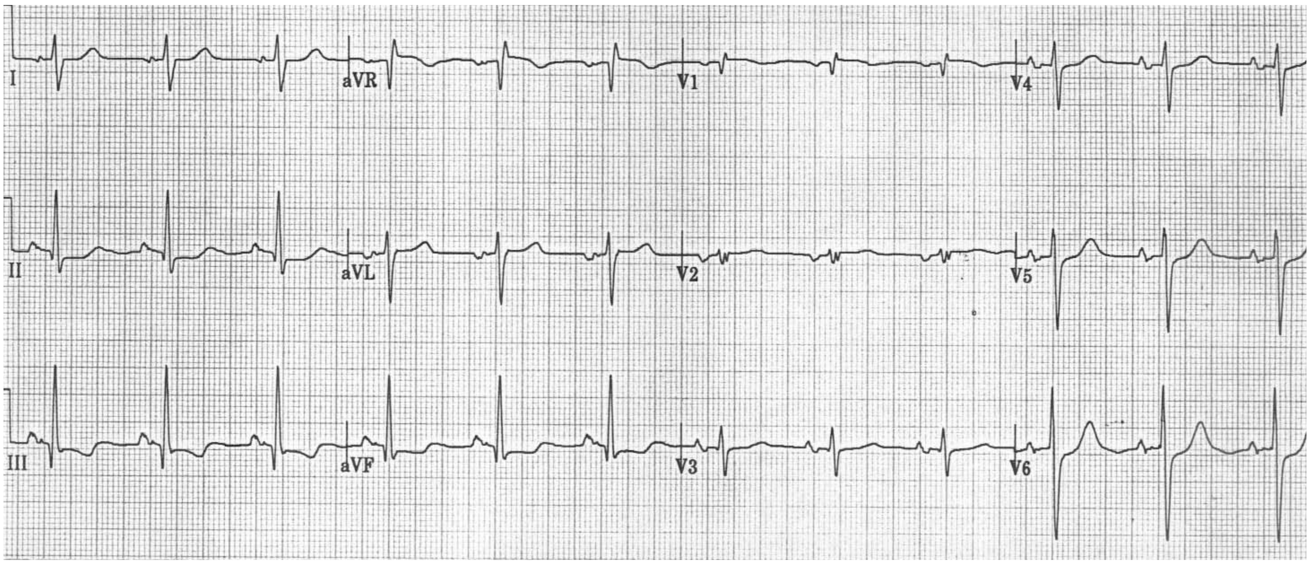

Figure. 12-lead ECG obtained from a 78-year-old man with long-standing pulmonary disease and new-onset heart failure.

Clinical Scenario: The ECG in the Figure was obtained from a 78-year-old man with long-standing pulmonary disease and new-onset heart failure. Based on the low voltage in leads V_1, V_2, V_3, the rightward frontal plane axis, incomplete right bundle-branch block (RBBB), and persistent precordial S waves, the computer interpreted the overall pattern as consistent with pulmonary disease. What else should be added to your interpretation?

Interpretation: The ECG diagnosis of right ventricular hypertrophy (RVH) in adults is often quite difficult to make. This is because the electrocardiogram represents a balance of electrical forces between the left and right ventricles at any given instant in time. The much larger and thicker left ventricle usually accounts for a predominance of these electrical forces, even when there is clinical evidence of mild-to-moderate pulmonary disease. In contrast to what occurs in children, in whom much lesser degrees of RVH are needed to produce a predominance of right-sided forces (seen on ECG as a dominant R wave in lead V_1), it is only with more severe degrees of RVH and/or pulmonary hypertension that definite ECG criteria for this diagnosis usually are seen. Suspicion for long-standing pulmonary disease (with possible RVH/pulmonary hypertension) should, therefore, be raised by the combined ECG findings of rightward axis, incomplete RBBB, low voltage in several precordial leads, and persistent precordial S waves in leads V_4, V_5, V_6—even in the absence of a tall R wave in lead V_1 and ECG criteria for right atrial enlargement. Although the ST-T wave changes in the inferior leads of the tracing seen here may indeed reflect right-sided "strain" (from RVH), it is important to emphasize that these changes also could reflect ischemia. This point is especially relevant in this patient with new-onset heart failure. However, an even more worrisome finding on this tracing is the subtle but definitely present coved ST segment elevation in lead V_1. The patient in this case died from acute myocardial infarction. ECG changes from long-standing pulmonary disease were felt to "mask" ECG evidence of the large acute infarction that was evolving with the exception of the above noted subtle ST segment changes in the inferior leads and in lead V_1.

In Future Issues:

Retropharyngeal Abscess in Children