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INSIDE

Does the BNP Level Have Prognostic Value in Acute MI?

ABSTRACT & COMMENTARY

Source: Mega JL, et al. B-type natriuretic peptide at presentation and prognosis in patients with ST-segment elevation myocardial infarction: An ENTIRE-TIMI–23 substudy. *J Am Coll Cardiol* 2004;44:335-339.

B-TYPE NATRIURETIC PEPTIDE (BNP) IS RELEASED FROM CARDIAC tissue in response to increased wall stress, and measurement of its level recently has been shown to aid in the diagnosis of congestive heart failure (CHF) in the emergency department (ED). As a result, BNP has been proposed as an additional marker for cardiac injury in the settling of acute coronary syndromes. However, less is known about the utility of this biomarker in this setting, and particularly with ST-segment elevation myocardial infarction (STEMI).

In this substudy of the ENTIRE-TIMI 23 trial (enoxaparin tenecteplase-tPA with or without glycoprotein IIb/IIIa inhibitor as reperfusion strategy in ST-segment elevation MI), 483 patients with STEMI were randomized to receive various treatment arms of fibrinolysis, glycoprotein inhibitor, and heparins, followed by immediate angiography to assess reperfusion. As part of this study, patients also had BNP, troponin I, and C-reactive protein (CRP) levels measured on arrival. Outcome measures were 30-day mortality and evidence of reperfusion success angiographically or electrocardiographically.

The median concentration of BNP in 438 patients who had results available (91%) was 15.6 pg/mL. That level was significantly higher in the 15 patients who died within 30 days of STEMI compared with those who did not (89 vs 15 pg/mL, $P<0.0001$). Patients with B-type natriuretic peptide (BNP) levels in the highest quartile (>32 pg/mL) had an 11-fold higher risk of death compared with all others ($P<0.001$). Using a pre-specified cut-off value of BNP > 80 pg/mL, patients with an elevated BNP level had a significantly higher rate of mortality (17.4% vs 1.8%, $P<0.0001$).

When compared with troponin I and CRP, BNP was a substantially more robust marker of 30-day mortality. Statistically, CRP was not associated with mortality in this study, and troponin levels, in fact, did not offer prognostic information independent of BNP. BNP

levels remained associated independently with mortality, even when adjusted for major clinical predictors of mortality such as age, anterior myocardial infarction location, time of onset, heart rate, blood pressure, and CHF at presentation.

As for reperfusion, BNP levels > 80 pg/mL were associated with increased rates of incomplete reperfusion of the infarct-related artery on angiography and incomplete resolution of ST-segment elevation electrocardiographically. The authors speculate that elevated BNP levels on presentation may be associated with left ventricular hypertrophy, unrecognized ventricular dysfunction, or a large territory of infarct and ischemia that may be associated with impaired reperfusion and increased mortality. They conclude that this study supports the approach of combining BNP, a marker of hemodynamic stress, with more traditional markers of myocardial necrosis, for risk assessment of STEMI patients at the time of presentation. ❖

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COMMENTARY BY THEODORE C. CHAN, MD, FACEP

BNP has emerged as an important diagnostic biomarker for acute CHF exacerbations in patients presenting to the ED.[1] Increased levels of BNP also have been associated with acute myocardial infarction, but the time course and utility of this finding is unclear. This is one of the first studies to assess the prognostic value of BNP on arrival to the ED in patients with STEMI. The authors report that an elevated BNP level was associated with decreaed rates of successful reperfusion after fibrinolysis and increased rates of mortality at 30 days. In fact, in this study, BNP was a more robust prognostic marker when compared with CRP or even troponin.

A few points, however, need to be kept in mind regarding this study. First, as a substudy of a larger, industry-sponsored trial looking at pharmacologic treatment of STEMI, patients received multiple different treatment regimens. It is unclear what, if any, effect those differing pharmacologic treatment arms may have had on the relationship between BNP levels and mortality rates. Second, the overall STEMI mortality rate was quite low in this study (3.5%), with only 15 patients dying within 30 days. A larger study is needed to fully assess the prognostic value of BNP. Third, the cut-off level for BNP of 80 pg/mL is remarkably lower than the standard cut-off level for the diagnosis of CHF. No data are provided regarding the actual range of BNP levels, although quartile levels are provided and the 75th percentile level was still quite low — 192 pg/mL—in those who died at 30 days. It is unclear if this level represents an acute rise in BNP due to the STEMI event or a chronic elevation in patients who are at risk for poor outcome from the event. Indeed, the authors did report that BNP levels greater than 80 pg/mL were associated with patients who were older, had a history of hypertension, angina, and CHF, or were already receiving beta-blockers or angiotensin-converting enzyme inhibitors.

Despite these limitations, the findings of this study are still provocative. They suggest that even modest elevations in BNP levels in patients with STEMI are a marker for increased mortality. Just as important, BNP levels may identify patients who are less likely to respond with complete reperfusion following pharmacologic fibrinolysis and who might benefit from more interventional approaches.

Reference

1. Maisel AS, et al. Rapid measurement of B-type natriuretic peptide in the emergency diagnosis of heart failure. *N Engl J Med* 2002;347:161-167.

Is Low-Dose Succinylcholine a Good Option in RSI?

ABSTRACT & COMMENTARY

Source: El-Orbany MI, et al. The neuromuscular effects and tracheal intubation conditions after small doses of succinylcholine. *Anesth Analg* 2004;98:1680-1685.

ALL EMERGENCY PHYSICIANS SHOULD BE QUITE familiar with the use of succinylcholine for neuromuscular blockade to facilitate endotracheal intubation. Given for this purpose, succinylcholine generally provides excellent intubation conditions within 60 seconds. When given in full intubation doses, succinylcholine is not metabolized fast enough to allow recovery of spontaneous respiration before oxyhemoglobin desaturation occurs.

The authors point out that the effective dose for 95% of people (ED_{95}) of succinylcholine is 0.3 mg/kg, less than one-third the typical dose used in rapid sequence intubation (RSI). This study examined whether succinylcholine given in doses smaller than 1.0 mg/kg can produce satisfactory intubation conditions quickly enough to be used for RSI, but with a shorter recovery time in the event that a cannot-intubate-cannot ventilate situation occurs.

The authors conducted this study in the operating suite. The subjects were American-Society- of-Anesthesiology (ASA) physical status I and II (essentially healthy) patients between the ages of 18 and 65 years scheduled for elective surgical procedures that require general anesthesia and endotracheal intubation. Exclusion criteria included patients with hepatic, renal, cardiac, pulmonary, or neuromuscular disease, and patients taking medications known or suspected to interfere with neuromuscular transmission. Pregnant women, obese patients (body mass index > 28 kg/m^2), patients with a history of abnormal response to succinylcholine, patients with abnormal airway exams, and patients with a history of difficult intubation also were excluded.

One hundred-fifteen patients were assigned randomly to one of five groups (23 patients each) according to the dose of succinylcholine to be given. The dosages tested were 0.3 mg/kg, 0.4 mg/kg, 0.5 mg/kg, 0.6 mg/kg, and 1.0 mg/kg. Induction was performed using fentanyl and propofol, and maintained by propofol and N_2O in oxygen.

After the induction, patients received the assigned dose of succinylcholine. Intubation conditions were graded at 60 seconds using a standardized scale. Time of onset of paralysis, maximal twitch depression, time to initial twitch detection after paralysis, and time to 10%, 25%, 50%, and 90% twitch height recovery were recorded. Time to initial diaphragmatic movement and time to resumption of spontaneous respiratory movement were calculated.

Onset times were decreased with increasing doses of succinylcholine, up to a dose of 0.6 mg/kg (from 82 seconds to 55 seconds), but the onset time using 0.6 mg/kg did not differ significantly from the onset time using 1.0 mg/kg (55 vs 52 seconds). The maximum twitch depression was similar after 0.5 mg/kg, 0.6 mg/kg, and 1.0 mg/kg doses. Intubation conditions often were graded as unacceptable after the 0.3 mg/kg and 0.4 mg/kg doses. Intubation conditions were acceptable in all patients receiving 0.5 mg/kg or more of succinylcholine, with the 0.6 mg/kg and 1.0 mg/kg doses receiving identical marks.

Time to 50% and 90% twitch height recovery was significantly shorter in the 0.6 mg/kg group than in the 1.0 mg/kg group, and the time to resumption of regular spontaneous respiratory movements was significantly shorter in the 0.6 mg/kg group than in the 1.0 mg/kg group (4.0 $\pm$ 0.5 minutes vs 6.2 $\pm$ 0.8 minutes). The authors conclude that 0.5 to 0.6 mg/kg of succinylcholine produces clinically satisfactory intubation conditions one minute after administration. They stated that the latter dose is similar to the 1.0 mg/kg dose in onset time, neuromuscular block intensity, and intubation conditions. The authors recommend using the smaller doses because the shorter apnea time of the smaller doses may avoid critical oxyhemoglobin desaturation in healthy adult patients in whom ventilation cannot be assisted. ❖

COMMENTARY BY JACOB W. UFBERG, MD

Although this study is interesting food for thought, and might be a reasonable change in operating suite practice habits, it does nothing to suggest that we should be changing our emergency department dose of succinylcholine for RSI (which, by the way, is 1.5 mg/kg and not the max dose of 1.0 mg/kg used in this study).

Several limitations make this study impossible to translate to emergency patients requiring emergent intubation. First, this study had very limited inclusion criteria, including only healthy, non-obese patients without any history of major medical problems. Thus, they basically studied patients having little to nothing in common with ED patients requiring RSI. Second, patients had prior induction of general anesthesia prior to the administration of succinylcholine. When performed in the ED, the induction and paralytic agents are given essentially simultaneously, likely putting a greater onus on the para-

lytic agent in establishing favorable intubation conditions in the ED rather than in the operating suite. Finally, patients receiving succinylcholine in the ED for RSI are usually in a condition where there is little alternative to endotracheal intubation. There is no such option in the ED as there was in this study: to ventilate the patient, allow him to wake up, and postpone the elective operation. Thus, the likely next step for emergency physicians is to attempt to secure the airway using difficult airway adjuncts or cricothyrotomy.

EPs are able to intubate more than 95% of patients successfully. Most of the remaining 5% can be ventilated. I, for one, am not ready to let potentially unfavorable intubation conditions jeopardize the majority for the remote possibility that I may encounter a cannot-intubate, cannot-ventilate situation in the very small minority. This study is interesting fodder for future research, however, it is not applicable currently to the ED patient requiring RSI.

Antibiotic Timing for CAP: The 4-hour Rule is Coming

ABSTRACT & COMMENTARY

Source: Houck PM, et al. Timing of antibiotic administration and outcomes for Medicare patients hospitalized with community-acquired pneumonia. *Arch Intern Med* 2004;164:637-644.

FEDERAL AGENCIES AND PROFESSIONAL SOCIETIES currently recommend that patients with community-acquired pneumonia (CAP) receive their initial dose of antibiotics within 8 hours of hospital arrival. The Centers for Medicare and Medicaid Services (CMS) conducted this study to explore further associations between timing of initial antibiotic doses and clinical outcomes. Data were collected retrospectively from more than 18,000 patients with CAP who were at least 65 years of age, not immunocompromised, and had not recently received antibiotics or been hospitalized. The investigators studied in-hospital and 30-day mortality, length of stay, and readmission within 30 days.

Study patients were elderly (generally 75-84 years of age), predominantly white, usually had comorbidities, and were most likely to have class IV pneumonia severity index scores. Compared with those who received antibiotics after 4 hours, those receiving antibiotics within 4 hours of hospital arrival had lower in-hospital mortality (6.8% vs 7.4%, odds ratio [OR] 0.85), lower 30-day mortality (11.6% vs 12.7%, OR 0.85), and shorter length of stay (0.4 days). The authors conclude that antibiotic administration within 4 hours of hospital presentation is associated with decreased mortality and length of stay in older patients with CAP. They recommend a 4-hour antibiotic administration goal to improve outcomes in those patients. ❖

COMMENTARY BY DAVID J. KARRAS, MD

Although this study can be criticized on a number of fronts, the immense size of the database and the impact of the sponsor (CMS) render this a powerful study with far-reaching consequences. The study methodology mitigates many of the limitations inherent to the retrospective design. It is debatable, however, whether there is clinical relevance to the improvements seen in patients who received antibiotics earlier. Reductions of mortality by 0.6% and length of stay by 0.4 days, and mortality odds ratios of 0.85 may be statistically meaningful, but arguably the benefit to antibiotic administration within 4 hours is negligible for practical purposes. Obviously, the present condition of many busy EDs makes it impossible to administer antibiotics within 4 hours of hospital presentation due to crowding and other sources of delay.

Nevertheless, CMS has chosen to use CAP outcomes as one of its measures of hospital quality, and it is likely that federal agencies will point to this study as justification for revision of current standards for timing of antibiotic administration. The not-so-hidden agenda behind CMS studies like this one is to drive hospitals to adopt new systems that improve health care delivery, minimize errors, and improve overall patient outcomes. Despite their flaws, these studies and the resulting clinical guidelines do appear to improve the quality of hospital care. It is incumbent upon EDs to develop mechanisms that permit patients with CAP to be evaluated quickly and receive antibiotics within a very short period. It is likely that the quality of our CAP management now will be judged by a much stricter standard, and major changes in ED patient flow will be necessary to meet that goal.

Special Feature

Interpreting CSF Results

By Esther Chen, MD and Stephanie Abbuhl, MD, FACEP

ACCURATELY MEASURED IN THE SUPINE PATIENT, normal cerebrospinal fluid (CSF) opening pressure is typically between 150-200 mmH_20.[1,2] Readings may

be elevated falsely in patients who are extremely flexed in the fetal position or who are sitting up, and by Valsalva maneuver. Spuriously low pressures can be seen in hyperventilated patients, if there is CSF leakage around the spinal needle, or rarely, if there is blockage of CSF by a herniating mass.[1] High opening pressure may be associated with infections (bacterial, viral, and fungal meningitis; abscesses; and meningoencephalitis), tumors, intracerebral and subarachnoid hemorrhage, and pseudotumor cerebri.

Gross Examination of CSF

Normal CSF is clear and colorless. Turbid CSF results from the presence of white blood cell (WBC) counts ≥ 200 WBC/μL or red blood cell (RBC) counts ≥400 RBC/μL[3] and suggests a bacterial meningitis until proven otherwise. Grossly bloody CSF is apparent with ≥6000 RBC/μL and may be seen after a traumatic LP or in patients with subarachnoid hemorrhage.[3] Xanthochromia, a pinkish or yellowish tinge to the CSF, may be apparent with RBC lysis (e.g. subarachnoid hemorrhage or delayed analysis of a traumatic LP), elevated CSF protein (≥150mg/dL), and systemic hyperbilirubinemia.[3]

CSF Cell Count and Differential

The CSF_{wbc} count and differential are a critical source of information, but not as simple to interpret as many charts in textbooks would imply. Cell counts must be performed promptly to prevent loss of cells from cell lysis or cell absorption to the walls of the collecting tube.[1] The normal adult CSF_{wbc} count is less than 5 cells/μL, with a lymphocytic and monocytic predominance and ≤ 1 polymorphonuclear cell (PMN),[3, 4] although PMNs > 6 have been seen in patients without meningitis, especially in traumatic LPs, where peripheral PMNs are introduced by blood contamination at the time of the procedure.[5]

Blood contamination during a traumatic LP requires careful analysis. CSF pleocytosis may be estimated by subtracting the contaminant WBC count (estimated using the normal peripheral WBC:RBC ratio of 1:700)[3] from the actual CSF_{wbc} count. A pre-*Haemophilus influenzae* vaccine study showed that this adjusted CSF_{wbc} count accurately predicted 90% of patients with culture-positive meningitis.[6] In addition, the CSF_{wbc} count was often more than 100 times the number of WBCs attributed to the trauma. Of the patients with missed diagnoses, four were 6-week-old infants, and one was an elderly woman with overwhelming sepsis. However, because 10% of patients with meningitis still were missed, this formula must be used in conjunction with all the other available clinical and laboratory data.

Cell counts between 5 and 20 cells/μL are indeterminate and may indicate early or partially treated meningitis. A pleocytosis occasionally can be seen after generalized seizures, but this is a diagnosis of exclusion.[7] Patients with bacterial meningitis tend to have a higher CSF pleocytosis (75% with >1,000 cells/μL in one study[8] and a median of 1,380 cells/μL in a smaller study)[9] than viral meningitis (median of 114 cells/μL)[10], although there is significant overlap in these numbers. Therefore, the cell count always should be interpreted with the differential to help distinguish between those two processes.

A lymphocytic predominance typically suggests aseptic meningitis, although it does not eliminate the possibility of a bacterial etiology. Up to 14% of patients with bacterial meningitis may have more than 50% lymphocytes, especially with lower CSF_{wbc} counts (<1,000 cells/μL) and with *Listeria monocytogenes*.[11] Two additional studies also reported CSF lymphocytosis in 6-10% of patients with well-documented bacterial meningitis.[12,13] To complicate the picture more, a lymphocytic pleocytosis also is seen in herpes simplex viral encephalitis (typically with elevated $CSF_{protein}$ levels and a normal or mildly decreased $CSF_{glucose}$ levels), as well as in varicella-zoster, enteroviral, arthropod-borne, and other encephalitides.

PMN predominance, although classically associated with bacterial meningitis, also can be found frequently in aseptic meningitis. In a study of 158 children (30 days-18 years) with acute meningitis during the peak months of enteroviral season, more than 50% PMNs were found in 56% of aseptic and 90% of bacterial causes. In addition, PMN pleocytosis was not limited to the first 24 hours of illness in patients with aseptic meningitis. The sensitivity and specificity of PMN predominance to identify a bacterial etiology was only 90% and 43%, respectively.[9]

$CSF_{glucose}$ Level

$CSF_{glucose}$ is derived from facilitated diffusion of plasma glucose, so normal values range from 45-80mg/dL in patients with normal fasting glucose, or approximately 60% of the serum glucose. Hypoglycorrachia (20-40mg/dL) characteristically is seen in meningitis caused by bacteria, mycobacteria, and fungi. Furthermore, low $CSF_{glucose}$ levels may be found in carcinomatous meningitis, granulomatous infiltration of the meninges (e.g., sarcoidosis, cysticercosis), viral infections (e.g., herpes encephalitis, mumps meningoencephalitis), and rarely, neurosyphilis.[3] $CSF_{glucose}$ levels may be spuriously high in hyperglycemic patients and potentially mask a serious infection. In these patients, the CSF to blood glucose ratio may be more

useful. One author recommends using a ratio of <0.31 to help diagnose a bacterial etiology in patients with diabetes mellitus.[14] However, the sensitivity of the combination of this ratio (<0.31) with a low absolute $CSF_{glucose}$ level (<40mg/dL) for identifying bacterial meningitis was only 73%. It is clear that abnormally low ratios may be seen in the absence of central nervous system (CNS) disease when the serum glucose has risen rapidly. Alternatively, normal ratios can be seen in bacterial meningitis, especially after a diabetic patient has had a sudden decrease in serum glucose level, as might occur after a dose of insulin. Fluctuations in serum glucose, along with the time lag required for CSF equilibrium to occur (1-2 hours), cause a wide variation in the CSF to blood glucose ratios for simultaneously collected samples.

$CSF_{protein}$ Level

The $CSF_{protein}$ level alone has limited value in determining the cause of fever and headache, because it fluctuates in both infectious and noninfectious neurologic diseases. Normal values range from 20 45mg/dL, depending on the laboratory used.[4] Low $CSF_{protein}$ levels typically are seen in processes that either decrease protein entry or increase protein removal (e.g., CSF leaks post-LP, pseudotumor cerebri).[3] Elevated $CSF_{protein}$ levels may be seen in many neurologic diseases and usually reflect an abnormality in the blood brain barrier. Causes of increased $CSF_{protein}$ include not only viral and bacterial meningitis (usually higher in the latter) [3, 4, 9], but also diabetes mellitus (especially when significant peripheral neuropathy is present), brain and spinal cord tumors, multiple sclerosis, CNS syphilis, myxedema, uremia, and connective tissue diseases.[2] When differentiating viral from bacterial meningitis, a $CSF_{protein}$ level above 100mg/dL suggests a non-viral etiology.[4] Unfortunately, however, there is considerable overlap in the protein levels that can be seen in both viral and bacterial meningitis.

Microbiological Analysis of CSF

The Gram stain of CSF fluid is one of the most useful and immediately available tests used to differentiate bacterial from viral meningitis, but again, there are caveats. In one study, organisms were detected by Gram stain in 88% of bacterial or fungal meningitis and 81% of shunt-associated meningitis, including patients on antimicrobial therapy.[15] However, in other studies, the Gram stain has performed more poorly with a sensitivity of only 40-60% in patients with bacterial meningitis.[16] Diagnostic accuracy increases with higher WBC counts[15], number of organisms, and type of organism.[4] Common bacterial causes of meningitis (e.g., *Streptococcus pneumoniae, Neisseria meningitidis, Haemophilus influenzae*) can be detected in more than 75% of cases.[4] Lower rates are found in meningitis caused by gram-negative organisms, anaerobes, and *Listeria monocytogenes*.[4]

New CSF Markers and Tests

The most exciting new tool in CSF evaluation is PCR analysis, which has revolutionized the diagnosis of specific viral CNS infections and also can be used to detect bacterial organisms. Now more widely available, the PCR technique can detect minute quantities of DNA or RNA in as few as 2-5 hours, and does not depend upon the presence of viable organisms (especially helpful in patients who have received out-patient antibiotics or when fastidious organisms are involved). PCR analysis has a sensitivity and specificity exceeding 95% for detecting herpes simplex encephalitis and has replaced brain biopsy as the gold standard.[13] In a recent study of patients with meningococcal meningitis, PCR had a sensitivity and specificity of more than 97%.[17] At our institution, the sample is sent to an outside laboratory. PCR results may be available in 24-48 hours, often before culture results, which makes it extremely helpful in the subsequent management of the patient. PCR testing for common viral causes of meningitis (herpesvirus, enterovirus, varicella-zoster, and others) and for specific bacterial organisms may become routine as they are more readily available.

Other diagnostic tests include India ink stain and cryptococcal antigen detection (serum and CSF) for cryptococcal meningitis. Patients with Lyme disease can present with aseptic meningitis and should have detectable serum and CSF antibodies. Latex agglutination and counterimmune electrophoresis for bacterial antigens can be extremely helpful, especially in partially treated cases. Other new markers and diagnostic tests to detect bacterial meningitis are currently under investigation, such as CSF procalcitonin,[18] CSF C-reactive protein, and specific immunologic tests for bacterial or mycobacterial antigens and antibodies.[1] ❖

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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

26. In the sub-study of the ENTIRE-TIMI 23 trial, the initial BNP level was associated with:

a. an increased 30-day mortality when it was more than 80 pg/mL in STEMI patients.
b. an increased successful reperfusion following angioplasty in STEMI patients.
c. acute pulmonary edema requiring intubation in non-STEMI patients.
d. a decrease in troponin and CRP levels in ACS patients.

27. CSF Gram stain has a reported sensitivity of ____ for the detection of patients with bacterial meningitis.

a. 13-28%
b. 40-88%
c. 79-99%
d. 95-99%

28. Which of the following bacterial organisms is more likely than the other three choices to present with a CSF_{wbc} count less than 1000 cells/mL and a negative CSF Gram stain?

a. *Neisseria meningitidis*
b. *Streptococcus pneumoniae*
c. *Listeria monocytogenes*
d. *Haemophilus influenzae*

29. When compared with a dose of 1 mg/kg, similar intubation conditions were encountered using succinylcholine in a dose of:

a. 0.1 mg/kg.
b. 0.3 mg/kg.
c. 0.4 mg/kg.
d. 0.6 mg/kg.

30. For elderly patients with community-acquired pneumonia, administration of antibiotics within 4 hours:

a. improves overall quality of life.
b. decreases mortality by about half.
c. modestly reduces hospital length of stay.
d. has been shown to be impossible to achieve consistently.

Answers:

26. a; 27. b.; 28. c.; 29. d.;30. c.

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.

RBBB—and Something Else?

By Ken Grauer, MD

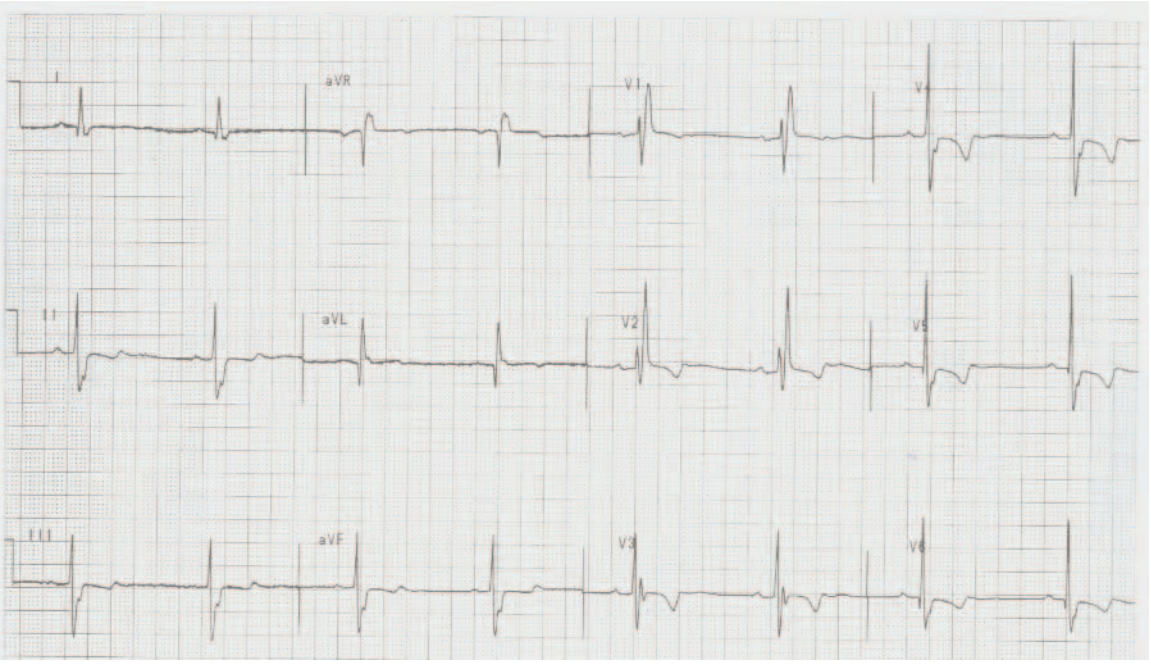

Figure. ECG obtained from a 41-year-old woman with chest pain.

Clinical Scenario: The electrocardiogram (ECG) in the Figure was obtained from a 41-year-old woman with chest pain of suspected cardiac etiology. The tracing shows complete right bundle-branch block (RBBB). How many additional ECG findings of potential concern can you identify that may be relevant in view of this patient's clinical history?

Interpretation: As stated, the ECG in the Figure shows complete RBBB, as determined by the presence of an rSR' pattern in lead V_1 and wide terminal S waves in lateral leads I and V_6. Findings of potential concern in view of the history of chest pain of suspected cardiac etiology include the following: 1) sinus bradycardia and arrhythmia; 2) a deeper than anticipated Q wave in lead aVL; 3) slight ST-segment coving in lead aVL; iv) flattening of the ST segment in lead V_1 (instead of ST depression), and persistence of deep, symmetric T-wave inversion throughout the precordial leads.

Typical bundle-branch block produces a pattern of secondary ST-segment and T-wave changes as a direct consequence of the conduction system defect. Specifically with isolated complete RBBB, the ST segment and T wave are directed opposite to the last QRS deflection in the three key leads (leads I, V_1, and V_6). This normally results in an upright T wave in leads I and V_6, and ST segment depression in lead V_1. Alteration of this typical pattern is said to reflect a primary ST-T wave change, and may be indicative of ischemia and/or infarction superimposed on the underlying bundle-branch block. Thus, the usual pattern of ST-segment depression in lead V_1 has been replaced in this case by a flat ST segment in this lead. Although this change is admittedly subtle and non-specific, of much greater concern is the fairly deep and symmetric (ischemic looking) T-wave inversion in leads V_3 through V_6. This is clearly much more extensive T-wave inversion than should be anticipated with complete RBBB. Although small, septal q waves may be seen in the presence of uncomplicated RBBB, the deep Q wave in lead aVL is not anticipated. Slight coving of the ST segment in this lead could reflect a recent acute change.

In summary, the combination of findings described above in this 41-year-old woman with chest pain should prompt evaluation to ensure that these changes are not acute. ❖

In Future Issues: **Rational Antibiotic Use in Upper Respiratory Tract Infections**