AMERICAN HEALTH CONSULTANTS® A Medical Economics Company

EMERGENCY MEDICINE ALERT

An essential monthly update of developments in emergency medicine

Emergency Medicine Reports

CO: The COntroversy and COnfusion COntinues

ABSTRACT & COMMENTARY

Source: Scheinkstel CD, et al. Hyperbaric or normobaric oxygen for acute carbon monoxide poisoning: A randomized controlled clinical trial. *Med J Aust* 1999;170:203-210.

Using blinded cluster randomization, 191 carbon monoxide (CO) poisoned patients were assigned to hyperbaric oxygen (HBO) or normobaric oxygen (NBO) treatment schemes. All patients were included regardless of CO level (average CO level: HBO 20.5%, NBO 22%). Cluster randomization is used to assign all patients that present in a group to one of the randomized treatments. NBO patients received 100% oxygen at 1.0 atmosphere for 100 minutes. This was achieved using an occlusive non-rebreather face mask and a special oxygen reservoir. These treatments were administered in the hyperbaric chamber, which was flushed with air to simulate pressurization as a sham hyperbaric treatment. HBO patients received 60 minutes of 100% oxygen by occlusive mask at 2.8 atmospheres. Each treatment was administered daily for three days. In between, all patients received oxygen by non-occlusive non-rebreather face mask with high-flow oxygen at 14 L/min. Intubated patients received 100% oxygen. Neuropsychological testing was performed at day three; if abnormal, three more days of treatment were administered. The neuropsychological testing was performed at completion of treatment and at one month.

Randomization resulted in no significant differences between the groups. However, patients in the NBO group were treated one hour sooner than patients in the HBO group on average. Only 46% of patients were available for follow-up. Results showed that HBO patients required more additional treatments than NBO patients (28% vs 15%, P = 0.01). None of the 87 NBO-treated patients had delayed neurological sequelae (DNS); however, five of the 104 HBO patients had DNS. Because many practitioners believe HBO must be delivered within six hours, a post hoc analysis stratifying patients by time to treatment demonstrated equal outcomes for NBO or HBO. The data for that analysis are not reported. Scheinkstel and colleagues conclude that HBO cannot be recommended for treatment of CO poisoning.

Volume 6 • Number 8 • January 2000 • Pages 57-64

INSIDE

COMMENT BY RICHARD J. HAMILTON, MD, FAAEM, ABMT

This report from Australia has been touted as the final word on the ongoing controversy of whether HBO improves outcomes in CO poisoning. On first analysis, Scheinkstel et al have used some clever mechanisms to overcome the typical problems associated with this sort of study — cluster randomization, sham chamber treatments, etc. There are a few small problems with this study: the inclusion of patients with levels lower than 25% without analysis of symptoms, the delays in treatment, etc. However, there is one glaring problem in that the NBO wing of treatment hardly received "normal" amounts of oxygen. These patients received continuous high-flow oxygen via a non-rebreather mask for 3-6 days, as well as 100% oxygen by "occlusive mask." The former is what most hospitals provide as "100% oxygen," and the latter is provided by a special aviator style or inflated-seal mask with rubber retainer straps. This is certainly not what most physicians would consider the treatment currently touted as the alternative to HBO, which is just high-flow oxygen by non-rebreather mask until the CO level drops to zero. The only legitimate interpretation of this study is that continuous high-flow oxygen with intermittent 100% NBO appears to be equivalent to continuous high-flow oxygen with intermittent 100% HBO.

My practice is to refer all patients with syncope, neurologic abnormalities, and cardiac abnormalities with elevated CO levels for HBO. In addition, patients with levels greater than 25%, patients who have been "soaked" in CO (long exposures but with lower levels), and pregnant patients also should be referred. My preference is to transfer them so that the HBO treatment can be initiated within six hours of their exposure. In general, they receive one 2.8 atmosphere dive with two 20-minute exposures to 100% oxygen by tight-fitting non-rebreather. They are subsequently discharged. The outcomes from my referral center are similar to those published here. I would remind those who believe that HBO treatment is of questionable value that the only demonstrably similar treatment protocol requires a stay in the hospital for 3-6 days on high-flow oxygen. In this day and age, that length of stay will be difficult to justify. ❖

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

Group Publisher: Brenda Mooney.
Executive Editor: Park Morgan.
Managing Editor: Valerie Loner.
Associate Managing Editor: Suzanne Zunic.
Senior Production Editor: Brent Winter.
Marketing Manager: Schandale Kornegay.

GST Registration Number: R128870672.

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

Back issues: $37. One to nine additional copies, $175 each; 10 or more additional copies, $131 each.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail Address: customerservice@ahcpub.com
Editorial E-Mail Address: suzanne.zunic@medec.com
World-Wide Web: http://www.ahcpub.com

Subscription Prices

United States: $219 per year (Resident rate: $110)
Canada: $239 per year plus GST (Resident rate: $120)
Elsewhere: $239 per year (Resident rate: $120)

Accreditation

American Health Consultants (AHC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. AHC takes responsibility for the content, quality, and scientific integrity of this CME activity.

American Health Consultants designates this continuing medical education activity for up to 20 hours in category 1 credit towards the Physician's Recognition Award. Each physician should only claim those hours of credit that he/she actually spent in the educational activity. ***Emergency Medicine Alert*** is also approved by the American College of Emergency Physicians for 20 hours of ACEP category 1 credit. This CME activity was planned and produced in accordance with the ACCME Essentials. This program has been reviewed and is acceptable for up to 20 Prescribed hours by the American Academy of Family Physicians. Term of approval is for one year from beginning distribution date of 6/98 with option to request yearly renewal. **For CME credit, add $50.**

Questions & Comments

Please call **Suzanne Zunic,** Associate Managing Editor, at (404) 262-5444 between 8:30 a.m. and 4:30 p.m. ET, Monday-Friday.

Statement of Financial Disclosure

American Health Consultants does not receive material commercial support for any of its continuing medical education publications. In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Brady is on the speaker's bureau for Genentech; Dr. Runge is a consultant with and is involved with research for Baxter Healthcare. Drs. Abbuhl, Grauer, Harrigan, and Hamilton report no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study.

Posterior Fat Pad Sign as an Indicator of Occult Elbow Fracture

ABSTRACT & COMMENTARY

Source: Skaggs DL, Mirzayan R. The posterior fat pad sign in association with occult fracture of the elbow in children. *J Bone Joint Surg* 1999;81-A:1429-1433.

Consecutive pediatric subjects were prospectively enrolled in this study if they had an elevated posterior fat pad noted on initial radiographic evaluation of the post-traumatic elbow, together with the absence of radiographic bony evidence of fracture. All subjects initially received anteroposterior, lateral, and two oblique views; if the attending pediatric radiologist, attending pediatric emergency medicine physician, or orthopedic resident noted a fracture, the subject was excluded. All patients were casted above the elbow. The main outcome measure was the incidence of fracture healing (periosteal elevation, callus formation, or both) seen at follow-up, which was designed to be at three weeks after the initial visit.

Forty-five children were included in the study, with a mean age of 4.5 years. Thirty-four (76%) had evidence of fracture healing, and were thus judged to have had an occult fracture on initial evaluation. Anatomic breakdown

was as follows: supracondylar fracture in 18 patients (53%); proximal ulna in 9 patients (26%); lateral condyle in 4 patients (12%); and radial neck in 3 patients (9%). Skaggs and associates conclude that presence of a posterior fat pad sign on initial radiographic evaluation of the post-traumatic pediatric elbow occurring in the absence of detectable bony fracture should lead the practitioner to treat such patients as though they have a non-displaced fracture of the elbow. Although this is not a new recommendation, the researchers' results were inconsistent with earlier studies that found lower rates of fracture at follow-up. The authors speculate that those studies suffered from design flaws (all were retrospective studies), including follow-up radiography that may have been too early (14 days or earlier). The average time to follow-up imaging in the present study was 20 days (range, 8-37 days); more importantly, the average follow-up x-ray that was negative for fracture was 21.5 days (range, 18-35 days).

COMMENT BY RICHARD A. HARRIGAN, MD, FAAEM, FACEP

Skaggs et al are to be congratulated on performing a prospective study. The high incidence of radiographic evidence of fracture healing seen in follow-up of these elbows that initially had only posterior fat pads as evidence of fracture should remind us to be vigilant for this radiographic sign. There were a few study design flaws worth discussing, however. Whereas both the pediatric radiology and pediatric emergency medicine attending were blinded to the study, the orthopedic resident was not; a positive finding by any of the three led to exclusion from the study population. Thus, all initial evaluators of the data were not on equal footing. It would have been more consistent to make all three aware of the study. The main outcome measure was evidence of new bone formation (signifying fracture healing) seen on follow-up x-ray. No mention is made of who was reading these films, although it is alluded that the attending orthopedists followed these patients, during a discussion of why they were not involved in the initial radiograph evaluation (fear of introducing bias into the study). It is unclear how inconsistency between the attending orthopedist and the attending pediatric radiologist in the interpretation of follow-up films would be reconciled in determining the main outcome measure. Inter-rater reliability data would have been important to report for both initial and follow-up films. Moreover, were those who read the follow-up films blinded to the study? Significant bias would exist if those charged with reading follow-up films were aware of the hypothesis and design of the study. ❖

D-dimer and DVT

ABSTRACT & COMMENTARY

Source: Lee AYY, et al. Clinical utility of a rapid whole-blood D-dimer assay in patients with cancer who present with suspected acute deep venous thrombosis. *Ann Intern Med* 1999; 131:417-423.

There have been several studies suggesting that certain D-dimer assays have high negative predictive values (NPV) for the diagnosis of deep venous thrombosis (DVT) in a general population of outpatients with suspected DVT.[1,2] However, there are also data to suggest that the accuracy of this test in patients with illnesses such as infection, liver disease, and cancer is reduced. This study by the McMaster group compares the clinical utility of a whole blood D-dimer assay (SimpliRed assay) for the diagnosis of DVT in patients with and without cancer.

This was a retrospective analysis of data collected in three prospective studies between 1992 and 1997. In all three studies, D-dimer testing was performed at presentation as part of a diagnostic algorithm that included other tests such as impedance plethysmography, compression ultrasonography, venography, and the assessment of pretest probability. In all three studies, cancer status was recorded at enrollment and before diagnostic testing. To obtain more information on cancer history, charts were reviewed independently by two reviewers who were blinded to D-dimer test results and final DVT status. The sensitivity, specificity, positive and negative predictive values, and likelihood ratios were calculated for the D-dimer assay in patients with and without cancer.

A total of 1068 patients were included in the analysis. The prevalence of DVT was 49% in 121 patients with cancer and 15% in 947 patients without cancer ($P < 0.001$). The NPV was significantly lower in patients with cancer (79% [CI 62.7-90.4]) than in patients without cancer (97% [CI 94.9-97.8]). This significant difference in NPV occurred despite similar sensitivities in the two patient groups (86% [CI 75.0-94.0] in patients with cancer and 83% [CI 75.2-88.5] in patients without cancer). Of the patients with cancer and a negative D-dimer result, 21% had DVT.

COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

This study clearly shows how the prevalence of a disease can impact the clinical utility of a test by influencing the positive and negative predictive values. Despite its

high NPV in patients without cancer, the SimpliRed D-dimer test does not have the same high NPV in patients with cancer, and a negative test cannot be used to reliably exclude DVT in these patients. There may still be a role for D-dimer testing in certain patient groups or in combination with other tests, but additional prospective studies are needed to clarify who, how, and when to use this test in a DVT or pulmonary embolism work-up. This study illustrates the importance of evaluating the performance of a diagnostic test in different patient populations before it is adopted for widespread use. ❖

References

1. Ginsberg JS, et al. Sensitivity and specificity of a rapid whole blood assay for D-dimer in the diagnosis of pulmonary embolism. *Ann Intern Med* 1998;129:1006-1011.
2. Perrier A, et al. Non-invasive diagnosis of venous thromboembolism in outpatients. *Lancet* 1999; 353:190-195.

Predicting Acute MI Despite LBBB: The Controversy Continues

ABSTRACT & COMMENTARY

Source: Edhouse JA, et al. Suspected myocardial infarction and left bundle branch block: electrocardiographic indicators of acute ischemia. *J Accident Emerg Med* 1999;16:331-335.

Edhouse and associates retrospectively investigated the diagnosis and management of the chest pain patient with suspected acute myocardial infarction (AMI) and electrocardiographic left bundle branch block (LBBB). In this study, they explored the use of thrombolytic agents in these patients, as well as the ability to diagnose AMI electrocardiographically using the clinical decision rule developed by Sgarbossa and coworkers.[1]

Seven hundred ninety-seven patients with suspected AMI were analyzed; of these patients, 50 (6%) instances of LBBB were noted. Using either serum marker elevations or postmortem examination findings as the standard for diagnosis of AMI, myocardial infarction was noted in only 26 (52%) patients in this initial group. Thirty-three patients with LBBB were thrombolysed for suspected AMI; ultimately, AMI was diagnosed in only 17 (51%). In retrospective fashion, the clinical decision rule developed by Sgarbossa et al was applied. All patients without AMI had clinical prediction rule scores not suggestive of AMI; approximately 80% of those patients with biochemically proven AMI had scores supporting the diagnosis of AMI. Edhouse et al concluded that the diagnosis of AMI in the chest pain patient with LBBB is difficult; they noted that the clinical decision rule was quite useful in the suspected AMI patient with LBBB.

COMMENT BY WILLIAM J. BRADY, MD

Recent literature has addressed this issue,[2,3] suggesting that the Sgarbossa et al clinical prediction rule is less useful than reported. The first such investigation,[2] which applied the Sgarbossa et al criteria to patients with chest pain and LBBB, found much less promising results — a very low sensitivity coupled with poor interobserver reliability. A second study[3] investigated the diagnostic and therapeutic impact of these criteria, finding them ineffective in distinguishing patients with AMI from those with noncoronary diagnoses. The authors concluded that electrocardiographic criteria are poor predictors of AMI in LBBB situations and suggested that all patients suspected of AMI with LBBB should be considered for thrombolysis. This report, in contradistinction, suggests that the Sgarbossa et al clinical decision rule has value and should be employed in the patient with suspected AMI and LBBB.

Traditional criteria for administration of thrombolytic agents in the AMI patient most often involve electrocardiographic ST segment elevation in an anatomic distribution; the presence of a new LBBB pattern represents another electrocardiographic criterion for such therapy. Many authorities suggest that all patients with LBBB pattern — presumably regardless of its chronicity — and a history suggestive of AMI receive a thrombolytic agent. Such an approach is perhaps reasonable if the physician has a high suspicion of AMI and is comfortable initiating thrombolysis based solely on information from the history and physical examination. Physicians, however, may be uncomfortable administering a thrombolytic agent under such circumstances; in fact, patients with electrocardiographic LBBB and AMI receive thrombolysis less often despite an increased risk of poor outcome[1,4] and the potential for significant benefit.[5] The clinician must realize that of all patients with chest pain, electrocardiographic LBBB pattern without obvious infarction, and clinically presumed AMI, only a minority will actually be experiencing acute myocardial infarction.[1] Treating all patients with LBBB and presumed AMI will subject a number of non-infarction patients to the risks and expense of thrombolysis.

The chest pain patient with LBBB represents a significant challenge to the emergency practitioner. Currently, no single or combination diagnostic approach

exists that will reliably reveal AMI in timely fashion. Even if the Sgarbossa et al clinical prediction rule is found to be less useful for interpreting the ECG in the patient with LBBB, this initial report has merit. It has forced the clinician to review the ECG in detail and casts some degree of doubt on the widely taught belief that the ECG is invalidated in the search for AMI in the LBBB patient. ❖

References

1. Sgarbossa EB, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle branch block. *N Engl J Med* 1996;334: 481-487.
2. Shapiro NI, et al. Validation of electrocardiographic criteria for diagnosing acute myocardial infarction in the presence of left bundle branch block. *Acad Emerg Med* 1998;5:508 (abstract).
3. Shlipak MG, et al. Should the electrocardiogram be used to guide therapy for patients with left bundle branch block and suspected acute myocardial infarction? *JAMA* 1999;281:714-719.
4. Rogers WJ, et al. Treatment of myocardial infarction in the United States (1990 to 1993): Observations from the National Registry of Myocardial Infarction. *Circulation* 1994;90:2103-2114
5. ISIS-2 (Second International Study of Infarct Survival) Collaborative Group. Randomized trial of intravenous streptokinase, oral aspirin, both, or neither among 17, 187 cases of suspected acute myocardial infarction: ISIS-2. *Lancet* 1988;2:349-360.

Special Feature

Motor Vehicle Crash Biomechanics: Interpreting the Polaroid

By Jeffrey W. Runge, MD, FACEP

Over the last decade, emergency medical services (EMS) personnel have become increasingly aware of the need to convey the circumstances of the motor vehicle crash to physicians caring for injured patients. First advocated by Hunt et al,[1] the physician's index of suspicion increases when significant automobile damage is shown in a photograph. Many EMS services carry Polaroid cameras on their units with the notion that a picture is worth a thousand words. The premise behind this is very sound, in that it is as important to stratify the risk of significant injury based on the history as it is to stratify for risk of coronary artery disease in patients with chest pain. Unfortunately for the car crash victim, the relationship between automobile damage and risk of injury is not as widely known among physicians as are the epidemiologic risk factors for coronary artery disease. A few simple historical features can be used by physicians to discriminate between patients on backboards with cervical collars, even when all present similarly.

There are approximately 40,000 people killed on the roads of the United States every year, nearly equal to the death toll for the entire Vietnam War. Yet our culture continues to perpetuate the myth that motor vehicle crashes are accidents and that death by vehicle is somehow an act of God. Otherwise, the nation would make a top priority of the three factors that most affect motor vehicle crash death rates: seat belt use, speed, and alcohol use. Instead, 20-40% of people from state to state do not buckle their seat belts. Alcohol use is responsible for more than 15,000 fatalities per year and a far higher number of injuries. Nevertheless, due to the efforts of those who advocate stricter laws on drunk driving, the use of seat belts, and vehicle design enhancements, there was a 3.8% annual decline in fatalities between 1996 and 1997 in spite of a large increase in vehicle miles traveled over that same period.[2]

Understanding the Characteristics of Impact

The kinematics of crash injury are extremely complex. There are four questions that should be asked every time a patient presents with an injury from a motor vehicle crash to allow an estimate of the forces at play: What did the patient's vehicle interact with (e.g., tree, guard rail, Yugo, or 18-wheeler)? What was the principal direction of impact — frontal, rear, near side, opposite side, or rollover? What was the estimated speed at impact (which is often difficult to precisely discern)? What protective devices were employed (air bag, 3-point seat belt, 2-point seat belt, or a child safety seat)?

About 40% of all fatalities are the result of single-vehicle crashes, one-half of which are rollovers. Of all crashes, fewer than 20% are single-vehicle, and only 6% are single-vehicle rollovers. Thus, single-vehicle crashes are more lethal, with rollovers being disproportionately high. Single-vehicle crashes more often involve occupants who are chemically impaired, traveling at high rates of speed, and unrestrained. Epidemiologists who study motor vehicle crash injury often use an index called "harm" that takes into account not only crash counts but also the economic impact of the crash and the resulting injuries. Although 21% of all crashes are rear, multi-vehicle crashes, they are responsible for only 3%

of harm. Side-impact crashes, especially near-side impacts, produce disproportionately more harm.

Crash engineers refer to the severity of impact, related to the speed of impact, as △v. This is the change in velocity experienced by the vehicle and its occupants at impact until the velocity is zero. The average △v for a tow-away crash in the United States is around 19 mph. For all crashes, △v is around 5 mph, and for those where the police are involved, the average △v is around 10 mph. While more than one-half of tow-away crashes occur at v less than 19 mph, more than half of all harm is caused from crashes with △v = 16-30 mph, and 23% from crashes with △v > 30 mph. The clinical significance is that most of the injuries seen in a typical emergency department are caused by relatively low-velocity crashes and cause a small proportion of overall societal harm.

Understanding Energy Transfer During a Crash

Whereas most injury biomechanics discussions begin with the formula $e = mv^2$, the energy forces during a motor vehicle crash are better understood by considering stopping distance and stopping time as a determinant of G-forces delivered to the body. In order to calculate or estimate G-forces, one must have some knowledge of the △v and the stopping distance (the distance traveled from the beginning of impact until the velocity reaches zero). The formula is:

$$G = \frac{\triangle v\ (ft/sec)^2}{\text{stopping distance (ft)} \times 30}$$

For a healthy adult younger than 60 years of age, average maximum human tolerance to quick-pulse velocity change is approximately 30 G-forces. Using this relationship, it is apparent that in order to reduce G-forces, one may reduce the △v, as in the enforcement of speed laws or effective braking. Equally effective are methods to increase the duration of the impact and increase the distance the body travels throughout the duration of the crash, as in the depth of an air bag, the deformation of the vehicle, padded surfaces within vehicles, and energy-absorbing barriers in the environment. Even small increases in stopping distance decrease the G-forces passed to the body by a factor of 30. It is also easy to understand why near-side impact crashes are more dangerous. Near-side impact crashes allow far less stopping distance than do frontal or rear crashes, and the chances for passenger department incursion are much higher.

The Four Collisions in a Crash and "Ride Down"

Understanding the four collisions during crash makes it apparent why seat belts work, and why the lack of a seat restraint invariably leads to increased tissue damage. The first collision occurs when the vehicle strikes another object. The second collision occurs when the occupant contacts fixed objects in the vehicle (e.g., the seat belt in the case of a restrained person, or the steering wheel, windshield, roof rail, or other objects when occupants are unrestrained). The third collision occurs when the occupants' organs continue to move relative to the body surface, and are damaged either through contact with fixed tissue such as the skull, or by tethering as in the duodenum or aortic arch. A fourth collision may occur if there are loose objects in the vehicle, such as backseat passengers who are unrestrained, or other loose objects, making contact with the occupant.

Under conditions ideal for injury prevention, the first collision occurs in a vehicle that has a large capacity for deformation, creating a longer stopping distance and absorption of a larger amount of energy. The second collision would ideally occur with a seat belt

early in the impact phase allowing the occupant to "ride down" the crash, decelerating over the entire duration of the crash. This sustained duration reduces the damage from the third collision, as the person's organs experience less deformation, as opposed to a quick-pulse deceleration. Further stopping distance can be added to the restrained occupant by a fully deployed air bag. An unrestrained occupant keeps moving during the first part of the impact and stops suddenly on contact with a vehicle surface. In cars equipped with air bags, unrestrained occupants may contact the air bag prior to or during deployment, which may add to the deleterious effects of the second collision as the speed and energy of the air bag deployment is added to the $\triangle v$.

Part of the history that should be obtained from the patient or EMS personnel is the type of vehicles involved and the surface contacted. Clearly, a three-cable guard rail in the middle of an interstate, designed to give 12 to 15 feet of stopping distance at 60 mph, or a water-filled barrier at an exit ramp, afford greater stopping distance and time duration than does a large tree. Moreover, a modern vehicle with crumple zones and a collapsible steering column will afford greater stopping distance than a 1957 solid chrome collector's special. Where multi-vehicle crashes occur, size matters. Occupants of large vehicles that hit small vehicles fare better than do occupants of small vehicles interacting with large vehicles. Thus, an estimation of energy forces using the available parameters of mass and velocity and stopping distance should be ascertained when taking the history.

While biomechanics is not a simple subject that can be summarized in a page or two, these basic concepts can help the emergency physician treat motor vehicle crash patients less blindly. An estimation of velocity at impact, principal direction of impact, seat belt use and air bag presence, and external vehicle and environmental interactions, when taken together, serve as a useful guide to the likelihood of severe injury. Risk stratification and a more studious approach to test ordering is good for patients and results in a better use of resources. ❖

References

1. Hunt RC, et al. Comparison of motor vehicle damage documentation in EMS run reports compared with photo documentation. *Ann Emerg Med* 1993;22:651-656.
2. U.S. Department of Transportation. National Highway Traffic Safety Administration. "Traffic Safety Facts." http://www.nhtsa.dot.gov.
3. Peterson TD, et al. Motor vehicle safety: Current concepts and challenges for emergency physicians. *Ann Emerg Med* 1999;34:384-393.

CME Questions

8. In the recent study by Lee et al comparing the clinical utility of a whole blood D-dimer assay (SimpliRed assay) in patients with and without cancer, the authors found:

a. the prevalence of DVT to be almost 50% in patients with cancer undergoing a DVT evaluation compared to 15% in patients without cancer.
b. the negative predictive value of the SimpliRed D-dimer assay in patients with and without cancer was about the same.
c. the SimpliRed D-dimer assay can be used to exclude DVT in patients with cancer.
d. that of the patients with cancer and a negative D-dimer test, only 2% had DVT.

9. In the pediatric patient with elbow trauma:

a. the posterior fat pad sign is always indicative of fracture.
b. occult fracture may be implied by the presence of the anterior fat pad.
c. evidence of occult fracture has been found at follow-up in 76% of patients with only a posterior fat pad on initial radiographs.
d. occult fat pad signs have been found at follow-up in 76% of patients with bony evidence of fracture on initial plain films.

10. Which of the following is *not* one of the four collisions in a crash?

a. Vehicle strikes object.
b. Occupant strikes fixed interior object.
c. Organs move within occupant till stopped.
d. Cell-cell deceleration.

11. G forces delivered to the body can be estimated by dividing $\triangle v$ by:

a. stopping distance (feet) $\times$ 30.
b. mv^2.
c. incursion distance, cubed.
d. $^4/_3\ \pi r^3$.

12. In the study by Edhouse et al on left bundle branch block (LBBB) and suspected acute myocardial infarction (AMI):

a. LBBB occurred in 52% of patients with AMI.
b. 51% of patients thrombolysed who had LBBB ultimately had AMI.
c. LBBB occurred in 52% of thrombolysed patients.
d. 33% of patients with AMI had LBBB.

13. Methodological problems in the study by Scheinkstel et al on the treatment of carbon monoxide (CO) toxicity include all of the following *except*:

a. treatment delays.
b. the inclusion of patients with CO levels < 25% without symptom analysis.
c. lack of cluster randomization.
d. lack of generalizability of their normobaric oxygen delivery strategy to the general medical community.

Does LBBB Prevent Diagnosis?

By Ken Grauer, MD

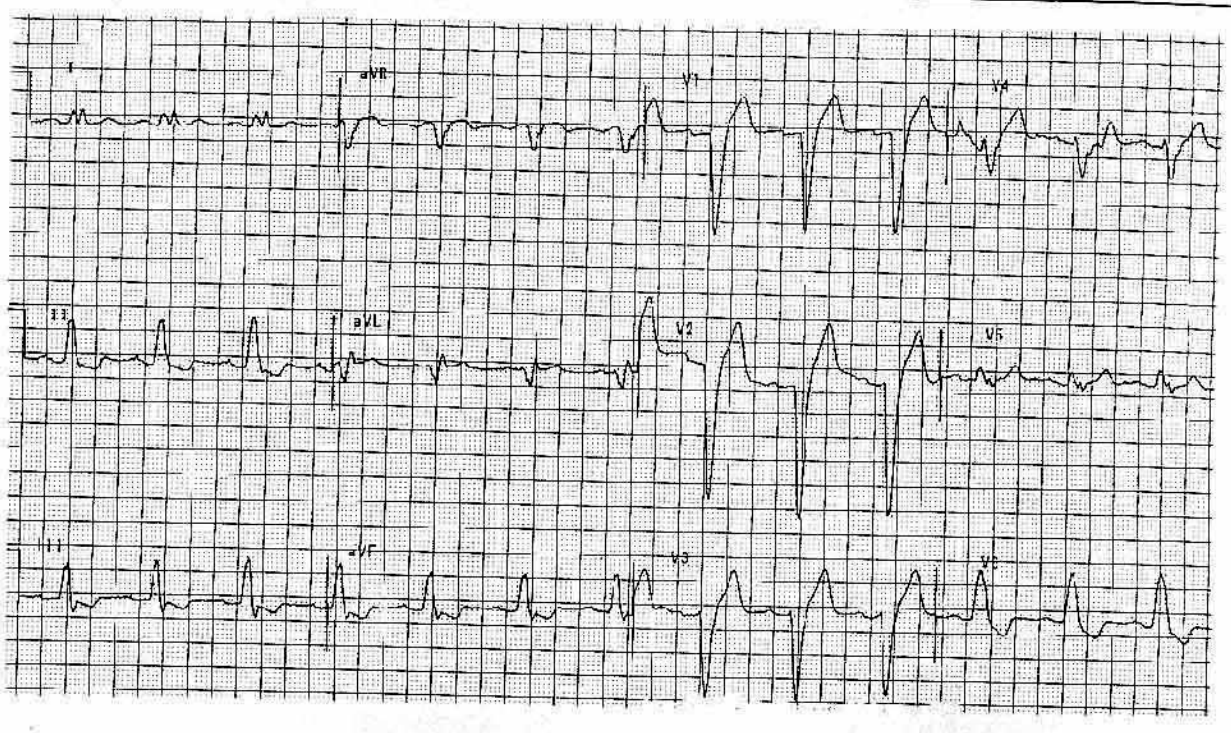

Figure. ECG obtained from a 69-year-old man with known LBBB. Can anything else be said about this tracing?

Clinical Scenario: The ECG shown in the figure was obtained from a 69-year-old man who was known to have complete left bundle branch block (LBBB). Is it possible to draw any other conclusions from evaluation of his ECG?

Interpretation: The rhythm is sinus at a rate of 80 beats/min. As noted above, the patient has complete LBBB. Despite opinion to the contrary, myocardial infarction can sometimes be diagnosed despite the presence of complete LBBB. Prior infarction is suggested in the above tracing by the presence of the wide and deep Q wave in lead aVL, late notching of the upslope of the S wave in two or more mid-precordial leads (in this case, leads V_4 and V_5), and primary ST-T wave changes (unexpected ST segment elevation in lead aVL, and the presence of an upright T wave in leads I and aVL). Q waves should not normally be present in lateral leads with typical LBBB. ST segments and T waves are normally directed opposite to the last QRS deflection in the three key leads (I, V_1, V_6) with typical bundle branch block. Thus, although one often will not be able to comment on the likelihood of past or present infarction in the setting of LBBB, the tracing shown here illustrates an example in which acute infarction should nevertheless be strongly suspected. ❖

Suggested Reading

1. Hands ME, Cook EF, Stone PH. ECG diagnosis of myocardial infarction in the presence of complete LBBB. *Am Heart J* 1988;116:23-31.
2. Sgarbossa EB, et al for the GUSTO Investigators. ECG diagnosis of evolving acute myocardial infarction in the presence of LBBB. *N Engl J Med* 1996;334:481-487.
3. Grauer K. *12-Lead ECGs: A 'Pocket Brain' for Easy Interpretation.* Gainesville, FL: KG/EKG Press; 1998:23, 26.

In Future Issues: Managing Ethanol Withdrawal