AMERICAN HEALTH CONSULTANTS®

EMERGENCY MEDICINE ALERT®

An essential monthly update of developments in emergency medicine

From the Publishers of Emergency Medicine Reports™

American Health Consultants Home Page—http://www.ahcpub.com

CME for Physicians—http://www.cmeweb.com

Hyperbaric Oxygen for Carbon Monoxide Poisoning

ABSTRACT & COMMENTARY

Source: Weaver LK, et al. Hyperbaric oxygen for acute carbon monoxide poisoning. *N Engl J Med* 2002;347:1057-1067.

It is a well-known fact among emergency physicians that acute carbon monoxide (CO) poisoning can result in cognitive sequelae. These cognitive sequelae occur in 25-50% of patients with loss of consciousness or with CO levels above 25%. The value of hyperbaric oxygen therapy for the treatment of CO-poisoned patients, and its ability to prevent long-term cognitive sequelae, has been debated for years among toxicology and hyperbaric experts. This study from the University of Utah addressed this difficult issue.

Patients were eligible for enrollment if they had a documented CO exposure, or an "obvious exposure," with either loss of consciousness (LOC), confusion, headache, malaise, fatigue, forgetfulness, dizziness, visual disturbances, nausea, vomiting, cardiac ischemia, or metabolic acidosis. Patients were excluded if more than 24 hours had elapsed from the time of exposure, they were younger than 16 years of age, they were moribund, or were pregnant. Patients were then randomized to either three hyperbaric chamber sessions (at 3, 2, and 2 atmospheres) or one treatment with normobaric oxygen and two treatments with normobaric room air over the next 24 hours. All patients underwent a battery of neuropsychological tests after the first and third chamber sessions and at two weeks, six weeks, six months, and 12 months. The primary endpoint of the study was the occurrence of cognitive sequelae at six weeks post-treatment.

The trial was designed to include 100 patients in each group, with interim data analyses scheduled after each 25 patients per group. A total of 76 patients actually were enrolled in each group, but the trial was stopped after the third interim analysis, as hyperbaric oxygen was judged to be efficacious. Of the 76 enrolled in each group, complete data were available up to six weeks on 75 patients in the hyperbaric group and 72 patients in the normobaric group. On an intention-to-treat basis, cognitive sequelae occurred in fewer of the patients in the hyperbaric group than the normobaric group (25% vs.

INSIDE

46.1%, p < 0.01). Cognitive sequelae also were fewer on an intention-to-treat basis in the hyperbaric group at 12 months (18.4% vs. 32.9%, p = 0.04), although these data are not as compelling due to more patients lost to follow-up and other confounding factors.

The authors conclude that the treatment of patients with acute, symptomatic CO poisoning with three hyperbaric oxygen treatments within a 24-hour period appears to lower the incidence of cognitive sequelae at six weeks and 12 months.

COMMENTARY BY JACOB W. UFBERG, MD

This is a very well-planned and -executed study that, in my mind, tips the scales heavily in favor of the use of hyperbaric oxygen therapy for patients with acute CO poisoning, specifically the hyperbaric regimen used in this study. This regimen was based on a report that suggested better outcomes after more than two treatments. The authors chose to complete the treatments over 24 hours to limit patient compliance issues.

One very interesting piece of data is that while the two groups had mean CO levels of about 25% initially, both groups had mean CO levels near the normal range by the time of initiation of hyperbaric oxygen therapy. This suggests that the hyperbaric therapy may offer therapeutic mechanisms independent of its ability to hasten carboxyhemoglobin dissociation. ❖

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

Vice President and Group Publisher: Brenda Mooney.
Editorial Group Head: Valerie Loner.
Managing Editor: Allison Mechem.
Marketing Manager: Schandale Kornegay.

GST Registration Number: R128870672.

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

Back issues: $44. One to nine additional copies, $212 each; 10 to 20 additional copies, $159 each.

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail Address: customerservice@ahcpub.com

Editorial E-Mail Address: allison.mechem@ahcpub.com

World-Wide Web: http://www.ahcpub.com

Subscription Prices

United States: $265 per year (Resident rate: $132.50)
Canada: $295 per year plus GST (Resident rate: $147.50)
Elsewhere: $295 per year (Resident rate: $147.50)

Accreditation

American Health Consultants (AHC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. This CME activity was planned and produced in accordance with ACCME Essentials.

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.

Emergency Medicine Alert has been approved by the American Academy of Family Physicians as having educational content acceptable for Prescribed credit hours. This volume has been approved for up to 20 Prescribed credit hours. Term of approval covers issues published within one year from the beginning distribution date of June 2001. Credit may be claimed for one year from the date of this issue. **For CME credit, add $50.**

THOMSON
AMERICAN HEALTH CONSULTANTS

Questions & Comments

Please call **Allison Mechem,** Managing Editor, at (404) 262-5589, between 8:30 a.m. and 5:00 p.m. ET, Monday-Friday.

Conflict of Interest Disclosure

To reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, Drs. Harrigan (editor), Abbuhl, Chan, Felz, Hamilton, Crawford, Shah, and Ufberg have reported no relationships with companies having ties to the field of study covered by this CME program. Dr. Grauer is sole proprietor of KG/EKG Press. Dr. Karras has reported that he is a consultant for Bayer Pharmaceuticals; consultant, speaker and researcher for Aventis Pharma; and a researcher for Bristol-Myers Squibb and Sepracor Inc. Dr. Brady is on the speaker's bureau for Genentech. Dr. Gibbs is a consultant and is involved in research for LMA North America.

Fibrinolysis in Acute MI and Hospital Revascularization Capability

ABSTRACT & COMMENTARY

Source: Mehta RH, et al. Patient outcome after fibrinolytic therapy for acute myocardial infarction at hospitals with and without coronary revascularization capability. *J Am Coll Cardiol* 2002;40:1034-1040.

In this retrospective study of the GUSTO-I database, investigators compared clinical outcomes of more than 25,000 patients with acute myocardial infarction (AMI) who were treated with fibrinolytic therapy in U.S. hospitals with and without coronary revascularization capability for percutaneous transluminal coronary angioplasty (PTCA) and coronary artery bypass grafting (CABG).

Study data included 12,279 patients treated at 286 hospitals with coronary revascularization capability, and 13,236 patients treated at 374 hospitals without such capability. Outcome measures included mortality (in-hospital, 30-day, and one-year), as well as other clinical events following AMI (i.e., recurrent ischemia, reinfarction, congestive heart failure [CHF], shock, and stroke).

Data were adjusted for five baseline variables associated with mortality after myocardial infarction (MI), namely age, systolic blood pressure, heart rate, Killip CHF class, and MI location. Baseline characteristics between the two groups were similar, with the exception of a slightly higher incidence of prior angina, infarct, angioplasty, and CABG in patients seen at hospitals with revascularization capability.

The investigators report no difference between the two groups in rates of recurrent ischemia, reinfarction, CHF, shock, or stroke after fibrinolytic therapy for AMI. There was a slightly lower unadjusted 30-day mortality for patients seen at hospitals with revascularization capability as opposed to those without (6.6% vs 7.2% respectively), but this difference became insignificant after adjustment

for baseline variables. Moreover, adjusted and unadjusted one-year mortality rates showed no significant differences between the two groups (9.6% vs 9.9% respectively).

Approximately 40% of patients who initially received therapy at hospitals without revascularization were transferred to hospitals with such capability. However, fewer than 10% of these patients were transferred within the first six hours for rescue PTCA. Transferred patients, as well as those who initially received fibrinolytic therapy at hospitals with revascularization capability, were more likely to undergo additional procedures such as pulmonary artery catheterization, temporary pacing, PTCA, and CABG.

The authors conclude that there is no difference in outcome for AMI patients treated with fibrinolytic therapy at hospitals without coronary revascularization capability compared to those hospitals with such capability, provided that transfer is available for angiography and revascularization if needed.

COMMENTARY BY THEODORE C. CHAN, MD, FACEP

In the setting of AMI, studies have shown that hospital volume and experience greatly impact outcome and mortality rates for mechanical reperfusion (PTCA and CABG), but not fibrinolytic reperfusion therapy.[1] Similarly, this study reports no outcome difference for AMI patients who receive fibrinolytics at hospitals with and without revascularization capability. The authors suggest that these findings support the concept that as long as access to PTCA or CABG is available, it need not be immediately available to maintain the benefits of fibrinolysis.

There are a number of points to keep in mind regarding this study. First, GUSTO-1 was conducted in the early 1990s. Since that time, new advances have occurred in both pharmacologic reperfusion (the development of newer fibrinolytic agents), and mechanical reperfusion (the use of stents and glycoprotein inhibitors). Second, the two groups were not equivalent at baseline, with a higher percentage of patients with prior diagnosis and treatment of CAD presenting to hospitals with revascularization capability.

Third, this study does not address the ongoing debate regarding the merits of mechanical vs. pharmacologic reperfusion strategies. This study focused on patients who met criteria and received fibrinolytic therapy. The results would not apply to all AMI patients, such as those with cardiogenic shock in whom PCI has been shown to be superior to fibrinolytic therapy.[2] Finally, while this study would appear to argue against the notion of regional heart-care centers for initiating fibrinolytic therapy, such centers still would be necessary to provide access to urgent rescue PCI and revascularization if needed. ❖

References

1. Canto J, et al. The volume of primary angioplasty procedures and survival after acute myocardial infraction. *N Engl J Med* 2000;342:1573-1580.
2. Hochman JS, et al. Early revascularization in acute myocardial infarction complicated by cardiogenic shock. *N Engl J Med* 1999;341:625-634.

Oral Vitamin K Lowers INR Faster than Subcutaneous Vitamin K

ABSTRACT & COMMENTARY

Source: Crowther MA, et al. Oral vitamin K lowers the international normalized ratio more rapidly than subcutaneous vitamin K in the treatment of warfarin-associated coagulopathy. A randomized, controlled trial. *Ann Intern Med* 2002;137:251-254.

THE ANTICOAGULANT EFFECTS OF WARFARIN FREquently need to be reversed for bleeding complications, excessively high international normalized ratio (INR) values, or in preparation for procedures. Although withholding warfarin is eventually effective, occasionally faster reduction in INR is necessary. Crowther and associates tested the hypothesis that oral vitamin K would reduce high INRs faster than subcutaneous vitamin K. Patients with an INR between 4.5 and 10.0 were randomized to receive 1 mg of vitamin K either orally or subcutaneously and warfarin was withheld. The primary outcome was INR on the day after vitamin K. In the 51 patients studied, the mean INR was 6. The INR had decreased to the 1.8-3.2 range the next day in 58% of the oral vitamin K group and 24% of the subcutaneous groups (odds ratio, 4.3; 95% CI, 1.1-17.4; P = .015; number needed to treat = 3). Two patients who received subcutaneous vitamin K had an increased INR the next day; this did not occur in the oral therapy group. Conversely, three patients who received oral vitamin K had INR less than 1.8, whereas none of the subcutaneous group did. Crowther et al concluded that oral vitamin K lowers high INR more rapidly than subcutaneous administration.

COMMENTARY BY MICHAEL H. CRAWFORD, MD

Using vitamin K to reverse a high INR prevents bleeding complications. The risk of major bleeding in patients with an INR greater than 6.0 is reported to be

4%.[1] In this study, no episodes of bleeding were observed, so the incidence actually may be lower. Some have been reluctant to use vitamin K to reverse high INRs for fear of overshooting and precipitating thrombosis, such as in patients with prosthetic valves. Again, in this study no episodes of thrombosis were observed, but three patients did have INRs less than 1.8 after oral vitamin K. The results of this study suggest that in patients with a high risk of bleeding complications and no excessive risk of thrombosis, i.e., prosthetic valve, and an INR greater than 4.5, that low-dose oral vitamin K administration should be considered. Patients with high INRs and a low risk of bleeding, such as many preprocedure patients, should merely have warfarin withheld. ❖

(Dr. Crawford is Professor of Medicine, Mayo Medical School, Consultant in Cardiovascular Diseases and Director of Research, Mayo Clinic, Scottsdale, AZ.)

Reference

1. Hylek EM, et al. Prospective study of the outcomes of ambulatory patients with excessive warfarin anticoagulation. *Arch Intern Med* 2000;160:1612-1617.

Syncope Should Not Be Taken Lightly

ABSTRACT & COMMENTARY

Source: Soteriades ES, et al. Incidence and prognosis of syncope. *N Engl J Med* 2002;347:878-885.

The authors evaluated participants enrolled in the Framingham Heart Study between 1971 and 1998 who reported syncopal events. The patients' reports were confirmed by review of hospital and outpatient records, and the etiology of the syncopal episode was determined by two physicians utilizing all available records and test results. Patients not seeking medical care were classified as having an unknown cause of syncope if there were no features in the history, examination, or electrocardiogram indicative of a cardiac event. The authors excluded patients from analysis if the report of syncope was equivocal or if they were lost to follow-up after the episode.

Of the 7814 study participants followed for an average of 17 years, 822 reported syncope and follow-up data was available for 727. The etiology of syncope could not be determined in 31% of patients. A vasovagal etiology was diagnosed in 24%, followed by orthostatic syncope (10%), medication-related syncope (7%), seizure (7%), cardiac etiology (7%), and stroke/transient ischemic attack (2%). Miscellaneous causes of syncope (micturition, cough) accounted for 13%. The incidence of syncope was the same in men and women.

Survival analysis showed that patients with syncope of any etiology were more likely to have cardiovascular disease and had a risk of death 31% greater than matched controls who did not report syncopal episodes. Among patients with cardiac syncope, the risk of death was twice that of controls. The risk of stroke among patients with neurologic causes of syncope was three times that of controls. Patients with syncope of unknown etiology also had an increased risk of adverse events. Those diagnosed with vasovagal, orthostatic, medication-related, and miscellaneous (but identifiable) causes of syncope had no increased risk of death, stroke, or myocardial infarction compared to controls. The authors conclude that patients with syncope of cardiac, neurologic, or unknown etiologies have an increased risk of death and merit further evaluation, while "benign" etiologies of syncope are not associated with adverse events.

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

While this study does not break new ground, it is by far the largest and most convincing affirmation of the need to take syncope quite seriously. The results confirm guidelines, derived from much smaller study groups, that call for extensive evaluation of patients with syncope deemed likely to be cardiac in etiology.[1,2] It strengthens the argument for intensive investigation—probably in an inpatient setting—for those likely to have syncope related to a neurologic event. Equally important, this study identifies syncope of unknown etiology as a marker of adverse outcome. While the good prognosis of patients with "benign" causes of syncope is reassuring, it emphasizes the need to perform a careful history and examination to distinguish benign syncope from idiopathic syncope.

The authors performed a number of secondary analyses that effectively excluded a number of potentially confounding factors. Unfortunately for the algorithmically inclined physician, they did not attempt to develop management guidelines. ❖

References

1. Martin TP, et al. Risk stratification of patients with syncope. *Ann Emerg Med* 1997;29:459-466.
2. Linzer M. Diagnosing syncope. Part 1: Value of history, physical examination, and electrocardiography. Clinical Efficacy Assessment Project of the American College of Physicians. *Ann Intern Med* 1997;126:989-996.

Special Feature

Metabolic Alkalosis

By Sachin J. Shah, MD

Metabolic alkalosis is the most common acid-base disorder found in hospitalized patients. In a study performed examining 13,430 arterial blood samples, 51% of the acid-base disorders were categorized as metabolic alkalosis.[1] Its prevalence is related to the fact that vomiting, nasogastric suctioning and diuretic use are so common. The mortality with severe alkalosis is very high. In patients with pH greater than 7.55, the mortality rate is close to 45%. In patients with pH greater than 7.65 the mortality increases to 80%.[2] The diagnosis and treatment of metabolic alkalosis are important for all emergency physicians to understand.

Metabolic alkalosis occurs with either a net excess of base or a net loss of acid. In a pure disorder, this leads to an increase in the pH as well as the plasma bicarbonate concentration. The body's normal response is hypoventilation to drive the pCO_2 higher to compensate for the increased bicarbonate. Hypoxia secondary to hypoventilation is a dangerous sequela.[3] The $PaCO_2$ increases from 0.5 to 0.7 mmHg for every 1 mmol increase in the plasma HCO_3 concentration.[4] Alkalemia results in decreased oxygen delivery as well due to the Bohr effect. The shift in the oxygen (O_2) dissociation curve decreases the ability of hemoglobin to release O_2.[5]

Classifications

Metabolic alkalosis can be classified by the response to therapy, the underlying pathophysiology or the primary organ system involved. The most straightforward grouping is by response to therapy and will be discussed here. The two categories are the chloride-responsive and chloride-resistant varieties. The chloride-responsive causes are more common. *(See Table.)*

Chloride-Responsive. Chloride is lost by many mechanisms and through many organs. It can be lost through the gut, kidney, or skin. Gastric fluid contains 60-140 mmol of hydrocholoric acid (HCl).[6] Vomiting or nasogastric suctioning results in alkalosis because the bicarbonate generated during the production of gastric acid is released into the circulatory system.[3]

Diuretics such as furosemide or hydrochlorothiazide may cause metabolic alkalosis as well. They produce a direct loss of chloride, sodium, and fluid in the urine.[7] There are several proposed mechanisms as to why this leads to metabolic alkalosis: 1) increased sodium delivery to the distal nephron accelerates potassium and proton secretion;[8] 2) renin and aldosterone are secreted in response to decreased intravascular volume, which in turn causes less sodium loss but greater secretion of potassium and protons;[3] and 3) potassium secretion will increase bicarbonate reabsorption and ammonia production which will increase net acid excretion by the kidney.[9,10]

A less common presentation of metabolic alkalosis is in patients with chronic respiratory acidosis who have rapid correction of their hypercapnea. These patients are chloride depleted and as the acidosis is corrected, the kidney will inappropriately reabsorb bicarbonate if sufficient chloride is not available resulting in a persistent metabolic alkalosis.[11]

Usually, the kidney filters out excess bicarbonate to return to baseline levels after the inciting events are resolved. Impaired kidney function is responsible for persistent metabolic alkalosis. The kidney either reabsorbs more bicarbonate than it should, filters out less due to a decreased glomerular filtration rate, or both.[3]

Early hypotheses held that decreased intravascular volume was responsible for maintaining metabolic alkalosis. One widely accepted hypothesis for the maintenance of alkalosis went as follows: Volume contraction stimulates fluid resabsorption along with bicarbonate in the proximal tubule, maintaining alkalosis. To correct this, volume expansion is needed. As volume expansion takes place with fluids, bicarbonate and chloride are delivered to the distal tubule, which preferentially reabsorbs chloride. This results in resolution of the alkalosis.[3]

Newer hypotheses suggest that chloride depletion plays a much greater role than fluid status. Studies show that chloride administration by many means, despite persistently low glomerular filtration rate (GFR), decreased plasma volume, or continued bicarbonate loading helped reverse the alkalosis.[12]

Chloride depletion seems to maintain alkalosis via certain renal mechanisms. GFR appears to be decreased due to a tubuloglomerular feedback mechanism. Bicarbonate secretion does not occur because insufficient chloride is available for exchange. Chloride depletion

Table. Chloride-responsive and -resistant causes of metabolic alkalosis

CHLORIDE-RESPONSIVE	CHLORIDE-RESISTANT
Vomiting	Cushing's disease
Aspiration	Renal artery stenosis
Diuretics	Diuretics
Zollinger-Ellison syndrome	Magnesium deficiency
Bicarbonate therapy	Renal failure and bicarbonate therapy
Potassium deficiency	

also increases renin secretion that results in increased aldosterone, which results in potassium wasting.[3]

Chloride-Resistant. The chloride-resistant alkaloses usually result from dietary potassium deficiency or mineralocorticoid excess. Multiple factors contribute to the net gain in bicarbonate. Potassium depletion results in an intracellular shift of protons. It also is associated with enhanced renal ammonia production.[13] Whether primary (adenoma, hyperplasia) or secondary, mineralocorticoid excess acts to promote sodium retention by secreting potassium. Aldosterone stimulates proton secretion and bicarbonate reabsorption in the collecting tubule.[14] The kidney also engages in potassium conservation by reabsorbing bicarbonate.

Another cause of metabolic alkalosis that may be seen in the emergency department (ED) is the milk-alkali syndrome, in which both bicarbonate and calcium are ingested. This may lead to vomiting and hypercalcemia, which increases bicarbonate reabsorption, and a reduced GFR.[3]

ED Presentation

Patients may present to the ED with apathy, confusion, cardiac dysrhythmias, and hypoventilation. History of recent vomiting, medication use (both prescription and over-the-counter), as well as other substances used (i.e., licorice, chewing tobacco) can be very helpful. Also, history of other medical conditions such as Cushing's disease or renal artery stenosis, if known, can help in diagnosing the cause.[3]

After initial assessment of the ABCs and resuscitation, blood work is necessary in the diagnosis and treatment of metabolic alkalosis. The two key laboratory tests are the arterial blood gas and the basic chemistry panel. Urine electrolytes also may help differentiate a chloride-responsive from a chloride-resistant alkalosis. In patients not taking diuretics, urine chloride levels less than 10 mEq/L indicate a chloride depletion state. Urine chloride levels greater than 30 mEq/L are more indicative of chloride-resistant alkalosis.[15]

Treatment

In addition to treating the underlying cause to prevent further alkalosis, existing deficits must be addressed to improve the patient's condition. In patients with chloride-responsive alkalosis, chloride therapy is essential. The selection of the type of fluid, however, depends on the patient's volume status as well as other deficits. A patient with a volume deficit would benefit from 0.9% normal saline solution. Potassium levels should be monitored closely, as well. If necessary, potassium can be replaced by adding potassium chloride to the other intravenous fluids. If fluid overload is an issue, then potassium chloride can be used alone, provided that the patient's potassium level is low or normal.[3]

In settings where the patient's pH is greater than 7.55 and they display signs of severe toxicity such as dysrhythmias, change in mental status, or encephalopathy, immediate correction is the rule. In this scenario, HCl can be used. It can be given as a 100 mmol/L solution. The correction can be estimated by the formula: amount of HCl needed = 0.5 × weight (kg) × desired decrement of bicarbonate. The HCl should be run in no faster than 0.2 mmol/kg/h, and it should be infused in a central vein.[3]

In patients with renal failure, modified dialysis can be pursued. The dialysate must be changed for the process to work effectively. This exchanges bicarbonate for chloride, helping to correct the patient's alkalosis. In patients who are vomiting, proton pump inhibitors may be used to help blunt the production of gastric acid.[3]

In patients whose primary derangement is mineralocorticoid excess, the source of the excess must by found and treated. Patients can be given a potassium sparing diuretic to help reverse the potassium deficit and the sodium overload. Dietary changes will help the patient correct the alkalosis as well. Sodium restriction and potassium supplement also may help reverse the hypertension.[3]

When potassium depletion alone is associated with the alkalosis, repletion is the rule. Depending on the severity of depletion, oral or intravenous repletion may be chosen. Glucose solutions should be avoided initially because this may stimulate the body to release insulin and push potassium into the cell. Frequent chemistry determinations should be undertaken to monitor sodium and potassium levels.[3]

Conclusion

In summary, a systematic approach with an understanding of the pathophysiology can help the clinician correctly diagnose and treat metabolic alkalosis, a condition that carries a high morbidity and mortality rate. Beware the elderly patient with chronic renal insufficiency who presents with a few days of a "sour stomach" and taking large amounts of bicarbonate for relief. This is the perfect setup for metabolic alkalosis. ❖

(Dr. Shah is an Assistant Professor of Emergency Medicine at Temple University Hospital and School of Medicine, Philadelphia, PA.)

References

1. Hodgkin JE, et al. Incidence of metabolic alkalemia in hospitalized patients. *Crit Care Med* 1980;8:725-732.

2. Anderson LE, Henrich WL. Alkalemia associated morbidity and mortality in medical and surgical patients. *South Med J* 1987;80:729-733.
3. Galla JH. Metabolic alkalosis. *J Am Soc Nephrol* 2000; 11:369-375.
4. Javaheri S, Kazemi H. Metabolic alkalosis and hypoventilation in humans. *Am Rev Respir Dis* 1987;136: 1101-1016.
5. Lee GR. *Wintrobe's Clinical Hematology*, 10th ed. Baltimore, MD: Lippincott Williams & Wilkins; 1999: 212-214.
6. Johnson LR. *Physiology of the Gastrointestinal Tract.* New York: Raven;1987.
7. Ellison DH. The physiologic basis of diuretic synergism: Its role in treating diuretic resistance. *Ann Intern Med* 1991;114:886-894.
8. Hropot M, et al. Tubular action of diuretics: Distal effects on electrolyte transport and acidification. *Kidney Int* 1985;28:477-489.
9. Chan YL, et al. Control mechanism of bicarbonate transport across the rat proximal convoluted tubule. *Am J Physiol* 1982;242:F532-F543.
10. Rosen RA, et al. On the mechanism by which chloride corrects metabolic alkalosis in man. *Am J Med* 1988; 84:449-458.
11. Levitan H, et al. The pathogenesis of hypochloremia in respiratory acidosis. *J Clin Invest* 1958;37:1667-1675.
12. Galla JH, et al. Adaptations to chloride-depletion alkalosis. *Am J Physiol* 1991;261:R771-R781.
13. Nakamura S, et al. $NH4^{+}$ secretion in inner medullary collecting duct in potassium deprivation: Role of colonoic H^{+}-K^{+-} ATPase. *Kidney Int* 1999;56:2160-2167.
14. Sabatini S. The cellular basis of metabolic alkalosis. *Kidney Int* 1996;49:906-917.
15. Williamson JC. Acid-base disorder: Classification and management strategies. *Am Fam Phys* 1995;52:584-591.

Physician CME Questions

1. **Hyperbaric oxygen therapy as delivered in the study by Weaver and colleagues:**
   a. increased morbidity but reduced mortality compared with normobaric treatment.
   b. reduced cognitive sequelae at 6 weeks and 1 year after acute carbon monoxide poisoning.
   c. precluded assessment of cognitive function due to study confounders.
   d. reduced 30-day mortality compared with normobaric treatment.

2. **In a retrospective review of AMI patients enrolled in GUSTO-1, Mehta et al reported that:**
   a. fibrinolytic therapy was more successful in high-volume rather than low-volume hospitals.
   b. one-year mortality rates were similar following fibrinolytic therapy at hospitals with and without revascularization capability.
   c. PTCA was equally successful in high- and low-volume hospitals.
   d. recurrent ischemia occurred more frequently followed fibrinolytic therapy at hospitals without revascularization capability.

3. **According to the study by Crowther et al, oral vitamin K appears to reduce excessively elevated INR values:**
   a. faster than equal doses of subcutaneous vitamin K.
   b. faster than equal doses of intravenous vitamin K.
   c. faster than equal doses of intramuscular vitamin K.
   d. All of the above

4. **Compared to patients who have never had syncope, patients with vasovagal syncope:**
   a. have a greater risk of stroke.
   b. have a greater risk of myocardial infarction.
   c. have a greater risk of arrhythmia.
   d. have no increased risk of adverse events.

5. **Syncope of unknown cause:**
   a. carries a higher risk of adverse event occurrence than vasovagal syncope.
   b. usually does not recur.
   c. is more common in pregnant patients.
   d. is associated with age but not gender.

6. **All of the following are causally linked with metabolic alkalosis *except:***
   a. Addison's disease.
   b. diuretic use.
   c. renal artery stenosis.
   d. vomiting.

7. **What is the most common acid-base disorder found in hospitalized patients?**
   a. Metabolic acidosis
   b. Metabolic alkalosis
   c. Respiratory acidosis
   d. Respiratory alkalosis

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.

Chest Pain and Lots of P Waves

By Ken Grauer, MD

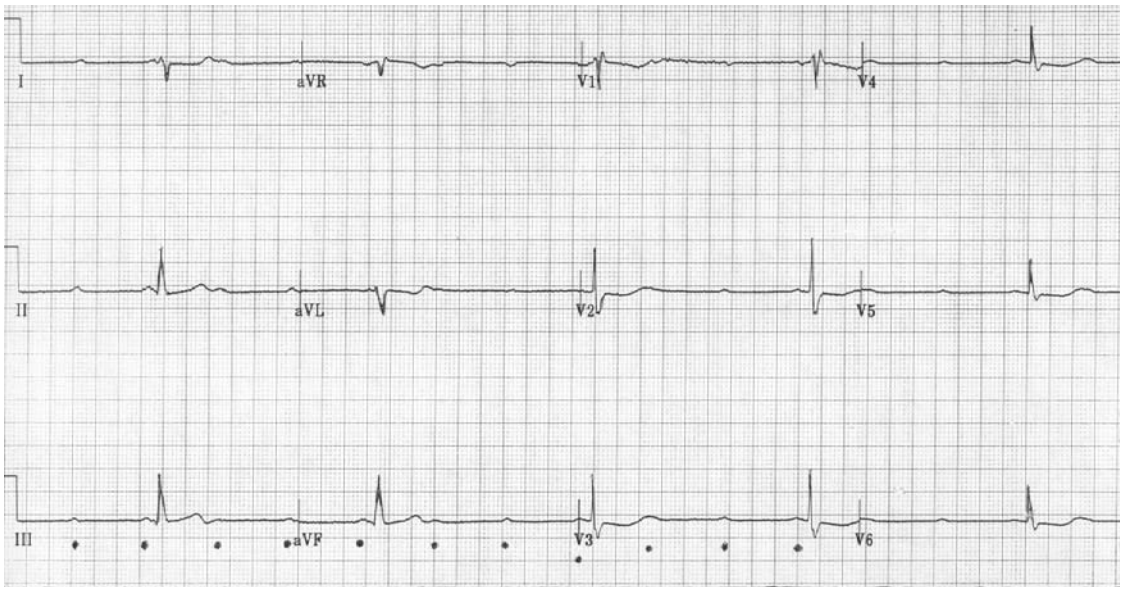

Figure. 12-lead ECG obtained from a 55-year-old woman with chest pain and lots of P waves.

Clinical Scenario: The 12-lead ECG shown in the Figure was obtained from a 55-year-old woman with new-onset chest pain. Many more P waves than QRS complexes are seen on the tracing (*see dots under P waves in leads III, aVF, and V_3*). How would you interpret this ECG?

Interpretation: Although a single lead rhythm strip is lacking, the 12-lead ECG in the Figure can still be interpreted. A narrow-complex marked bradycardia is present that is fairly regular at a rate just over 30 beats/minute. As noted above, many more P waves than QRS complexes are present. The atrial rhythm (marked by the dots) is regular at a rate of between 90-95/minute. Despite the fact that many more P waves than QRS complexes are present, P waves appear to conduct, as evidenced by the presence of a *fixed* PR interval preceding each QRS complex. This finding rules out the possibility of 3° (complete) AV block, in which there is no relationship between P waves and QRS complexes (P waves "march through" the QRS complex when there is 3° AV block). The rhythm must therefore be some type of *high-grade* 2° AV block, in this case with 3:1 AV conduction (3 P waves are present for each QRS complex). Although high-grade AV block (in which many if not most P waves *fail* to conduct) is most often the result of Mobitz II 2° AV block, the *lack* of consecutively conducted P waves anywhere on this tracing precludes definitive diagnosis. It is important to appreciate that on occasion, the usually less severe Mobitz I (Wenckbeach) form of 2° AV block also may be "high grade," with failure of consecutively occurring P waves to conduct. In such situations, the characteristic picture of progressive PR interval lengthening prior to dropping a beat may not be seen.

Analysis of the remainder of the ECG in the Figure reveals marked right axis deviation (RAD) consistent with a left posterior hemiblock (LPHB) pattern, incomplete right bundle-branch block (IRBBB) evidenced by an rsr′ pattern in lead V_1, early transition (a relatively tall R wave is present in lead V_2), and worrisome ST segment depression in leads I, aVL, and V_2-V_6. An ECG obtained one hour earlier showed ST segment elevation in the inferior leads (which has now resolved). The overall picture in this 55-year-old woman with new-onset chest pain is most consistent with acute evolving infero-posterior infarction. Telemetry tracings over the previous hour revealed clear evidence of Mobitz I (Wenckbach) 2° AV block—which in conjunction with the findings of normal QRS duration and acute inferior infarction strongly suggest that the 2° AV block with 3:1 AV conduction seen here is probably also a manifestation of the Mobitz I (Wenckbach) form of 2° AV block. That said—the bifascicular conduction defect and acute infarction with marked bradycardia are clear indication for emergency pacemaker placement. ❖

In Future Issues: **Wolff-Parkinson-White Syndrome**