AMERICAN HEALTH CONSULTANTS®

EMERGENCY MEDICINE ALERT

An essential monthly update of developments in emergency medicine

From the Publishers of Emergency Medicine Reports™

CME Test and Reader Survey included in this issue

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

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

Celecoxib: Good News but No Cause to CELEBrate

ABSTRACT & COMMENTARY

Source: Silverstein FE, et al. Gastrointestinal toxicity with celecoxib vs nonsteroidal anti-inflammatory drugs for osteoarthritis and rheumatoid arthritis: The CLASS Study: A randomized controlled trial. *JAMA* 2000;284:1247-1255.

NONSTEROIDAL ANTI-INFLAMMATORY DRUG (NSAID) THERAPY CARries a risk of developing significant injury to the upper gastrointestinal (GI) tract. The annualized incidence rate of symptomatic GI ulcers and ulcer complications in NSAID users ranges from 2% to 4% (1%-2% for ulcer complications alone). These side effects generally are considered a consequence of cyclooxygenase (COX)-1 inhibition. This study attempts to determine whether celecoxib, a COX-2-specific inhibitor, is associated with a lower incidence of significant upper GI toxic effects and other adverse effects compared with conventional NSAIDs.

The Celecoxib Long-term Arthritis Safety Study (CLASS) was a double-blind, randomized, controlled trial conducted from September 1998 to March 2000 at 386 clinical sites in the United States and Canada. A total of 8059 patients with osteoarthritis (OA) or rheumatoid arthritis (RA) were enrolled. A total of 4573 patients (57%) received treatment for six months. Patients were randomly assigned to receive 400 mg celecoxib bid (two and four times the maximum RA and OA dosages, respectively; n = 3987); 800 mg ibuprofen tid (n = 1985); or 75 mg diclofenac bid (n = 1996). Aspirin use for cardiovascular prophylaxis (≤ 325 mg/d) was permitted. The incidence of prospectively defined symptomatic upper GI ulcers and ulcer complications (perforation, obstruction, and bleeding [POBs]) and other adverse effects was measured during the six-month treatment period. Patients were excluded if they took NSAIDs (except for stable aspirin use ≤ 325 mg/d); anti-ulcer drugs (except for occasional antacid use); antibiotics alone or in combination with omeprazole, lansoprazole, and ranitidine for treatment of *Helicobacter pylori* infection; or antineoplastics (except methotrexate or azathioprine for RA). Use of oral, intramuscular, and intra-articular glucocorticoids and disease-modifying antirheumatic drugs was permitted.

For all patients, the annualized incidence rates of upper GI ulcer complications (POBs) were 0.76% for celecoxib and 1.45% for NSAIDs (P = 0.09). The incidence of POBs and symptomatic ulcers for celecoxib vs. NSAIDs was 2.08% vs. 3.54%, respectively (P = 0.02). For patients not taking aspirin, the annualized incidence rates of POBs alone and POBs combined with symptomatic ulcers for celecoxib vs. NSAIDs were 0.44% vs. 1.27% (P = 0.04) and 1.40% vs. 2.91% (P = 0.02). For patients taking aspirin, the annualized incidence rates of POBs alone and POBs combined with symptomatic ulcers for celecoxib vs. NSAIDs were 2.01% vs. 2.12% (P = 0.92) and 4.70% vs. 6.00% (P = 0.49). Fewer celecoxib patients experienced chronic GI blood loss, GI intolerance, hepatotoxicity, or renal toxicity. No difference was noted in the incidence of cardiovascular events, irrespective of aspirin use.

Celecoxib, at doses greater than those indicated clinically, was associated with a lower incidence of symptomatic ulcers and POBs—as well as other clinically important toxic effects—compared with NSAIDs at standard dosages. The decrease in upper GI toxicity was strongest in patients not taking aspirin concomitantly.

***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: Suzanne Zunic.
Associate Managing Editor: Paula L. Cousins.
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: $38. One to nine additional copies, $183 each; 10 to 20 additional copies, $137 each.

AMERICAN HEALTH CONSULTANTS™
THOMSON HEALTHCARE

Subscriber Information

Customer Service: 1-800-688-2421
Customer Service E-Mail Address: customerservice@ahcpub.com
Editorial E-Mail Address: paula.cousins@ahcpub.com
World-Wide Web: http://www.ahcpub.com

Subscription Prices

United States: $229 per year (Resident rate: $114)
Canada: $259 per year plus GST (Resident rate: $130)
Elsewhere: $259 per year (Resident rate: $130)

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. This CME activity was planned and produced in accordance with the ACCME Essentials. ***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 2000. Credit may be claimed for one year from the date of this issue. **For CME credit, add $50.**

Questions & Comments

Please call **Paula Cousins,** Associate Managing Editor, at (816) 960-3730 between 8:30 a.m. and 4:30 p.m. ET, Monday-Friday.

Statement of Financial Disclosure

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. Gibbs is a consultant and advisory board member and is involved in research for LMA North America; and Dr. Karras is involved with research for Bayer Pharmaceuticals. Drs. Abbuhl, Chan, Felz, Grauer, Harrigan, Hamilton, and Ufberg report no financial relationships with companies having ties to this field of study.

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

COX-1 is a ubiquitous constitutive isozyme producing prostaglandins responsible for homeostatic functions such as maintenance of GI mucosal integrity. COX-2 is largely a cytokine-induced isozyme producing prostaglandins that mediate pain and inflammation. The search for a drug that would inhibit COX-2 without causing all the COX-1 inhibition side effects has been a pharmaceutical holy grail. Celecoxib is the first of this series of drugs with anti-inflammatory properties and reduced side effects.

Does it live up to the hype? The POB incidence in celecoxib users not taking aspirin is similar to the POB incidence in the general population. In addition, the authors would argue that the high therapeutic dosages used in this study were responsible for the higher than expected rate of POBs and symptomatic ulcers. Indeed, celecoxib appears to reduce the rate of symptomatic ulcers and complications over NSAIDs, but it does not eliminate them. Prudent use of the drug for patients with severe RA and OA is appropriate, especially if the patients are taking aspirin at cardioprotective doses. In my opinion, celecoxib's use purely as an analgesic in routine settings is of limited value when compared to NSAIDs or the mild narcotic combination therapies currently available. ❖

Repositioning for the Patient with Benign Positional Vertigo

ABSTRACT & COMMENTARY

Source: Froehling DA, et al. The canalith repositioning procedure for the treatment of benign paroxysmal positional vertigo: A randomized controlled trial. *Mayo Clinic Proc* 2000; 75:695-700.

IN THIS PROSPECTIVE, RANDOMIZED, DOUBLE-BLIND, CONtrolled trial, 50 patients with a history suggestive of benign positional vertigo (BPV) and unilateral positional nystagmus on physical exam were treated with the canalith repositioning procedure (CRP) or a sham maneuver. Major outcomes included resolution of vertigo and positional nystagmus at a follow-up visit.

Patients were recruited from an outpatient Urgent Care Center and internal medicine practices at the Mayo Clinic. Inclusion criteria were simply positional vertigo and positional nystagmus in either the right or left Dix-Hallpike head-hanging position. Exclusion criteria included gaze-evoked nystagmus, positive result of the

Dix-Hallpike maneuver in both right and left head hanging positions, evidence of ongoing central nervous system disease, otitis media, otosclerosis, and inability to tolerate a diagnostic Dix-Hallpike head-hanging maneuver because of restricted head movement or severe positional vertigo with nausea or vomiting.

In the CRP, each patient was put through a series of five brief maneuvers that are a modification of the Dix-Hallpike maneuver itself. The procedure was repeated until no positional nystagmus was elicited or until five cycles had been performed. All patients were sent home with instructions to sleep the first two nights in a sitting position wearing a cervical collar.

A nurse, blinded to the type of maneuver, performed follow-up evaluations at a mean of 10 days. Twelve of the 24 patients in the CRP group had resolution of symptoms (50%; 95% CI, 30-70%) compared to five of the 26 patients in the sham group (19%; 95% CI, 4-57%; P = 0.02). The results of the Dix-Hallpike maneuver were negative for positional nystagmus in 16 of the CRP group (67%; 95% CI, 48-86%) and in 10 of the sham group (38%; 95% CI, 20-57%; P = 0.046). A number of clinical characteristics obtained at the time of the initial examinations were assessed, and none were found to be associated with the resolution of vertigo.

COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

Although a larger sample size and narrower confidence intervals would be even more convincing, this study is fairly persuasive in showing the benefit of the CRP in many BPV patients. In a recent review, at least three other trials noted benefit from the CRP in BPV patients.[1] This study is the most relevant to the emergency department (ED) setting in that diagnosis was based entirely on history and physical exam without any laboratory tests or vestibular/audiologic testing. In addition, the study was conducted by general internists rather than otorhinolaryngologists.

The rationale for the CRP is anatomic and straightforward. The theory is that free-moving particles in the endolymph of the posterior semicircular canals cause positional vertigo with head movement. The five-step CRP maneuver is thought to transfer the debris from the posterior semicircular canal into the utricle, where it presumably adheres and no longer causes symptoms. The maneuver is not thought to be of any benefit to patients with other causes of vertigo. It is appealing that a simple bedside procedure might treat some of the BPV we see in the ED. It has been noted that the procedure can be difficult in the elderly and in very obese patients, which may limit its use. It also would be interesting to see if there is measurable improvement immediately after the maneuver (as some literature suggests), and if this predicts resolution of symptoms at a later follow-up. In summary, BPV is common, CRP seems simple and brief enough to be practical, and the literature is suggestive of its utility—now we need someone to assess its feasibility and outcome in the ED. ❖

References

1. Furman JM, Cass SP. Benign paraoxysmal positional vertigo. *N Engl J Med* 1999;341:1590-1596.

Another Rapid Sedation Option for Pediatric CT Scanning

ABSTRACT & COMMENTARY

Source: Pomeranz ES, et al. Rectal methohexital sedation for computed tomography imaging of stable pediatric emergency department patients. *Pediatrics* 2000;105:1110-1114.

WHEN CHILDREN IN THE EMERGENCY DEPARTMENT (ED) require immobilization for imaging procedures, it is notoriously difficult for them to remain motionless. Numerous pharmacologic agents have been employed for sedation, but most require intravenous (IV) or intramuscular (IM) injection, display delayed onset of action by oral or nasal routes, or achieve inadequate immobilization. To evaluate a more acceptable modality for pediatric sedation, Pomeranz and colleagues at the University of Michigan studied 100 children, ages 3 to 60 months, who were seen at three academic pediatric EDs in Ann Arbor. Indications for scanning were closed head injury (63%), mental status change, ventriculoperitoneal shunt malfunction, acute ataxia, sinusitis, or inappropriate head circumference.

Methohexital (MXT) 25 mg/kg was administered per rectum with a syringe and 8-Fr feeding tube 15 minutes prior to scanning. In addition to continuous pulse oximetry and ECG monitoring, a nurse observed the procedure and took vital signs every five minutes. Onset, adequacy, and duration of sedation were assessed by the nurse and physician in attendance in all 100 cases.

Average age was 24 months, with an average time to full sedation of 8.2 minutes (range: 6.98-9.42 min). Average time between drug administration and full recovery was 79.3 minutes (range: 71.00-85.74 min). Ninety-two of 99 patients (95%) were adequately sedated for scanning, while three required mild restraint and four (5%) were judged failures. CT images were adequate in 98%. Forty-five of 49 parents (90%) surveyed reported satisfaction with MTX and would accept it

again if required for their child. Ten children (10%) had side effects—hiccoughs, hypersalivation, cough, and brief oxygen desaturation (which responded to chin repositioning). None required intubation.

Pomeranz et al conclude that rectal MXT, an ultra-short-acting barbiturate, is a rapid, effective, and safe agent for pediatric sedation in stable patients too young or anxious to satisfy CT scanning requirements. The authors state, however, that they are not the first group to study rectal MXT in children. Thirty-five years ago, Orallo evaluated this agent in 316 children undergoing general anesthesia, achieving 85% satisfactory sedation.[1] In 1987, another group demonstrated an 82.5% success rate for rectal MXT among 40 children requiring CT or MRI imaging.[2] Manulli and Davies found rectal MXT provided adequate sedation in 87% of cases compared to 83% with chloral hydrate, in 190 pediatric outpatients (average age 25 months) under-going CT or MRI procedures.[3]

COMMENT BY MICHAEL FELZ, MD

I researched rectal MXT via our Drug Information Center and colleagues in Pediatric Anesthesia and the ED. Our institution, like large academic centers elsewhere, tends to employ oral chloral hydrate (80-100 mg/kg) for outpatient pediatric sedation. Onset of action averages 20 minutes or longer, duration of sedation is usually 60-90 minutes, and adequacy of immobility for scanning is 70-90%, although sometimes immobility is unpredictable. Two other agents—midazolam, by oral, nasal, or IV routes, and ketamine IM—do not display MXT's spectrum of simplicity, rapidity, immobilization, and painless administration. Given the noteworthy onset of action—a mere eight minutes for rectal MXT—depth and duration of sedation comparable or superior to other agents, and safety profile documented in this paper and in previous sizeable studies, I consider rectal MXT a viable alternative to chloral hydrate for children undergoing nonpainful procedures. This seems especially applicable when rapid sedation is desirable, IV access is avoidable, and parental satisfaction is a goal. ❖

References

1. Orallo MO, Eather KF. Sodium methohexital as a rectal agent in pediatric anesthesia. *Anesth Analg* 1965;1:97-103.
2. Griswold JD, Liu LMP. Rectal methohexital in children undergoing computerized cranial tomography and magnetic resonance imaging scans. [abstr] *Anesthesiolog* 1987;67:3a.
3. Manulli MA, Davies L. Rectal methohexital for sedation of children during imaging procedures. *AJR Am J Roentgenol* 1993;160:577-580.

Treatment of Chest Pain in Acute Coronary Syndromes

ABSTRACT & COMMENTARY

Source: Baumann BM, et al. Randomized, double-blind, placebo-controlled trial of diazepam, nitroglycerin, or both for treatment of patients with potential cocaine-associated acute coronary syndromes. *Acad Emerg Med* 2000;7:878-885.

THIS TRIAL FROM THE UNIVERSITY OF PENNSYLVANIA IS the first study to compare different treatment modalities in symptomatic patients with potential cocaine-associated acute coronary syndromes. Patients with cocaine-induced chest pain were randomized to treatment with 5 mg diazepam intravenously, 0.4 mg nitroglycerin sublingually, or both every 5 minutes for 15 minutes, or until resolution of symptoms. Inclusion criteria included: age between 18 and 60 years, cocaine use within 24 hours, history suggestive of ischemic chest pain, and the ability to provide informed consent. Major outcomes were reduction in chest pain (measured by visual analog scale), changes in blood pressure and heart rate, and change in stroke index (measured with a transthoracic cardiac output monitor). Forty patients were enrolled (diazepam 12; nitroglycerin 13; both 15). Baseline demographics, cocaine use, chest pain characteristics, initial ECGs, and discharge diagnoses were similar among the three groups.

Chest pain reduction was similar among the three groups (diazepam, -33.3 ± 8.0 mm; nitroglycerin, -30.7 ± 7.1 mm; both, -33.0 ± 7.9 mm; P = 0.6). There were no significant differences in heart rate and blood pressure changes among the treatment groups. Stroke index decreased during treatment for all groups (diazepam, -8.7 ± 3.3; nitroglycerin, -3.1 ± 2.8; both, -1.8 ± 3.1; P = 0.03), but was not statistically significant after adjustment for multiple comparisons. The authors conclude that chest pain resolution and changes in cardiac performance do not differ for patients with potential cocaine-associated acute coronary syndromes when treated with diazepam or nitroglycerin. They also conclude that use of both agents did not offer any advantage over use of either agent alone.

COMMENT BY JACOB W. UFBERG, MD

Cocaine may cause myocardial ischemia through several mechanisms. It causes tachycardia and hypertension leading to increased myocardial oxygen demand, and causes alpha-adrenergic mediated coronary artery vasoconstriction, among other effects. It is believed that central nervous system (CNS) stimulation may exacerbate

the peripheral effects of cocaine, and that blockade of CNS stimulation with benzodiazepines may prevent some of the peripheral sympathomimetic effects of the drug, thus decreasing myocardial oxygen demand. Nitroglycerin has been shown to reverse cocaine-induced coronary artery vasoconstriction and relieve cocaine-induced chest pain. Its effects on mortality and infarct size have been proven in traditional patients with acute myocardial infarction, although they have not been studied in a cocaine-induced chest pain population.

This well-executed study is the first randomized, prospective clinical trial comparing these two treatment modalities. The only apparent limitations of the study lie in the small sample sizes. The authors also acknowledge a larger-than-expected standard deviation, which further reduces the study's ability to detect a difference between groups. Even so, the authors' conclusions appear valid. Diazepam and nitroglycerin performed equivalently in the treatment of potential cocaine-induced acute coronary syndromes, while combination therapy offered no additional benefit. Physicians may wish to choose their treatment based on the presence or absence of hypertension, tachycardia, and CNS excitation. ❖

Special Feature

Acute Myocardial Infarction Diagnosis: Atypical Presentations

By William J. Brady, MD

THE EMERGENCY PHYSICIAN (EP) WILL ENCOUNTER atypical features of acute myocardial infarction (AMI) in approximately 10-30% of myocardial infarction cases.[1-6] Factors contributing to this diagnostic challenge include patient age, alternate chief complaints, atypical discomfort, and certain comorbid states.

A Broad Spectrum of Atypical Complaints

Anginal equivalent complaints, which occur in the "painless" AMI setting, classically include dyspnea, diaphoresis, nausea, and emesis. Other anginal equivalent symptoms to consider are cough, palpitations, and anxiety. Among chief complaints, the most frequently encountered anginal equivalent is dyspnea, which is found in 10-30% of AMI patients, often because of pulmonary edema.[1-3] Isolated emesis and diaphoresis are quite rare, with a 1-3% prevalence.[2,3] The geriatric patient also may present atypically with acute weakness (3-8%) and syncope (3-5%).[4] Unexplained sinus tachycardia, bronchospasm resulting from cardiogenic asthma, and new-onset lower extremity edema all have been reported as anginal equivalent presentations for AMI. Anginal equivalent syndromes often involve neurologic presentations with acute mental status abnormalities and cerebrovascular attacks (CVA), frequently among the elderly. From the perspective of acute delirium, less than 1% of such patients with altered mentation in an emergency department (ED) population will have AMI. Less pronounced mental status abnormalities, including confusion and lethargy, are classically reported findings in the elderly AMI patient and are described in approximately 5% of cases. Myocardial infarction associated with acute stroke is noted in approximately 5-9% of patients, usually older than 60 years.[4]

Atypical Locations

When discomfort or another painful sensation is the complaint associated with AMI, it may be atypical from a number of different perspectives. The patient may complain of pain in an atypical location. The discomfort may be located in non-chest portions of the body (e.g., the epigastrium, anterior neck or jaw, or left upper extremity). While not the classic presentation of an AMI, these symptoms usually do not challenge the EP's diagnostic ability; in the appropriate patient, such pain distributions are suggestive of a coronary ischemic source. The discomfort may be located in the back, posterior neck, and right arm in the rare patient, suggesting another explanation for the unpleasant sensation. Other highly unusual presentations include cephalgia, as well as hip and lower back pain.

Atypical Characteristics

The actual chest discomfort description may range from the classic "crushing, pressure-like, heaviness" to the atypical "sharp, knife-like" or "burning." Taking into account all ED patients presenting with typical terms such as "crushing" or "pressure-like," only slightly more than one-half experienced an acute coronary syndrome (ACS) (approximately 50% AMI and 50% unstable angina).[7] The remaining patients had gastrointestinal, pulmonary, and musculoskeletal diagnoses. Chest discomfort is described as "burning" or "indigestion-like" in an adult chest pain population of which 23% have AMI and 21% have unstable angina.[7] Interestingly, a minority of AMI cases presenting with burning chest discomfort may relate a favorable response to the infamous "GI cocktail." In 19-23% of instances, patients note "sharp," "knife-like," or pleuritic pain because of an ACS.[8] Lastly, the physical examination may mislead the EP with reproducible chest pain being present in as many as 15% of confirmed AMI patients.

The Elderly: Atypical May Be Typical

A patient's advanced age almost always effects the EP's ability to evaluate the patient and assess the potential of AMI. In fact, several studies have demonstrated that the evaluation of acute chest pain with confirmation of the correct diagnosis and initiation of appropriate therapies is very difficult in geriatric patients. Numerous disease states, syndromes, and medications contribute to making the elderly AMI presentation a significant diagnostic challenge. This increased rate of atypical presentation in the elderly translates into the possibility of clinically unrecognized AMI. For example, in one large autopsy series of elderly patients, correct AMI diagnosis was made in less than one-half the patients antemortem; this was especially true in the very old.[4] As a patient ages, a multitude of factors—including autonomic neuropathy, injury to cardiac sensory afferent nerves due to past ischemic heart disease, cortical failure resulting from cerebrovascular or other central nervous system disease, extensive comorbidity, higher pain thresholds, and pre-existing mental status abnormalities—contribute to a higher rate of atypical presentation and unrecognized AMI. Among the geriatric populace, these factors result in an increased prevalence of anginal equivalent chief complaints, "silent" myocardial infarction, and a preponderance of neurologic syndromes.

Atypical presentations are encountered with increasing frequency in sequentially older populations.[4] In elderly patients younger than 85 years, chest pain becomes less frequent while equivalent complaints are noted more often; however, chest pain still is found in the majority of cases. Stroke, weakness, and altered mentation become more common with increasing age and frequently are not accompanied by typical chest discomfort. Atypical presentations occur, yet still are in the minority in this relatively younger geriatric population.

In the patient older than 85 years, atypical presentations are the norm and should be anticipated. The incidence of "painless" AMI increases dramatically with age; 60-70% of elderly AMI patients older than 85 years will present with an anginal equivalent complaint or syndrome—most often with a change in mental status. If one considers all elderly patients with altered mental status in an ED population, however, the AMI diagnosis is found only in 1% of cases. In most acutely ill patients older than 85 years, the clinician should consider not only the potential for AMI but also should actively exclude the diagnosis with appropriate investigations.

The elderly also frequently present with complications of AMI rather than the actual symptoms of the acute ischemic event. For example, very elderly patients presenting with new-onset, unexplained congestive heart failure should be screened for acute ischemia. Similarly, the elderly patient presenting with malignant bradycardia, atrioventricular block, or ventricular arrhythmia should have AMI excluded while appropriate therapies and other evaluations are performed.[4]

Atypical Presentations Common in Diabetics

Patients with diabetes mellitus (DM) suffer AMI more often than the general public; they also experience AMI at an earlier age, present more frequently with atypical manifestations, and suffer more commonly an unrecognized myocardial infarction. Medically unrecognized AMI is felt to occur in approximately 40% of DM patients compared to 25% of the non-DM population.[5] Autopsy studies have demonstrated that myocardial scar without an antemortem diagnosis of myocardial infarction—indicative of the medically unattended, past infarct—is three times more frequent in the diabetic than the nondiabetic patient.[5] The medical and legal literature discussing the missed AMI frequently cite DM history

as a risk factor for an unrecognized event. In fact, DM often is implicated in legal cases as a medical factor leading to the diagnosis error. As with the elderly AMI patient, numerous factors contribute to atypical manifestations of acute ischemic heart disease in the DM patient, including polyneuropathy, an altered perception of cardiac pain, and extensive comorbidity.

The diabetic patient's abnormal perception of myocardial infarction may lead to atypical or less impressive symptoms of AMI. Accurate diagnosis based on historical grounds—the primary tool available to the EP—is made much more difficult. In the diabetic patient, atypical symptoms such as dyspnea, confusion, fatigue, and emesis may be the presenting complaint in as many as 40% of AMIs.[5] The diabetic also may experience more frequently the less-than-classic pain syndromes, including discomfort in unusual locations and with abnormal characteristics. The atypical presentations reduce the rate at which the patient is able to receive adequate medical care. The patient may not feel the pain or may attribute the abnormal sensation to some other malady, and therefore, may not consult a physician at all or may wait until later in the course of the event. If the patient does seek medical attention, the presentation may be such that the physician misses the actual diagnosis, leading to an inappropriate disposition and therapy. ❖

References

1. Canto JG, et al. Prevalence, clinical characteristics, and mortality among patients with acute myocardial infarction presenting with chest pain. *JAMA* 2000; 283:3223-3229.
2. Lusiani L, et al. Prevalence, clinical features, and acute course of atypical myocardial infarction. *Angiology* 1994;45:49-55.
3. Uretski B, et al. Symptomatic myocardial infarction without chest pain: Prevalence and clinical course. *Am J Cardiol* 1977;40:498.
4. Bayer AJ, et al. Changing presentation of myocardial infarction with increasing old age. *J Am Geriatr Soc* 1986;34:263-266.
5. Jacoby RM, Nesto RW. Acute myocardial infarction in the diabetic patient: Pathophysiology, clinical course, and prognosis. *J Am Coll Cardiol* 1992;20:736-744.
6. Bertolet BD, Hill JA. Unrecognized myocardial infarction. *Cardiovasc Clin* 1989;20:173-182.
7. Lee T, et al. Acute chest pain in the emergency room: Identification and examination of low risk patients. *Arch Intern Med* 1985;145:65-69.
8. Tierney WM, et al. Physicians' estimates of the probability of myocardial infarction in emergency room patients with chest pain. *Med Decis Making* 1986; 6:12-17.

CME Questions

33. Celecoxib:
- a. is associated with a lower risk of upper GI ulcers and related complications when compared with ibuprofen.
- b. prevents ulcers when used with ranitidine.
- c. is associated with less colonic ulceration than is ibuprofen.
- d. is contraindicated in patients taking aspirin.

34. All of the following statements are true about benign positional vertigo (BPV) *except*:
- a. BPV is thought to be caused by free-moving particles in the posterior semicircular canal.
- b. Patients with BPV usually experience vertigo but have no nystagmus in either the right or left Dix-Hallpike head-hanging position.
- c. The canalith repositioning procedure is a five-step maneuver that is a modification of the Dix-Hallpike maneuver.
- d. When compared to a sham procedure, the canalith repositioning maneuver has been shown to improve outcomes in BPV patients at 10 days.

35. The most attractive feature of rectal methohexital for pediatric sedation is:
- a. absence of cytochrome p450 interaction.
- b. prompt reversibility with narcotic antagonists.
- c. rapid and predictable immobilization for x-ray.
- d. potentiation of oral or nasal benzodiazepene effect.

36. Options for pediatric sedation for neuroimaging include:
- a. methohexital.
- b. chloral hydrate.
- c. midazolam.
- d. All of the above are correct.

37. In the recent study comparing diazepam and nitroglycerin in the treatment of chest pain in suspected cocaine-associated acute coronary syndromes:
- a. Diazepam outperformed nitroglycerin.
- b. Diazepam and nitroglycerin appeared to be equivalent.
- c. Nitroglycerin outperformed diazepam.
- d. The combination of diazepam and nitroglycerin outperformed either medication given by itself.

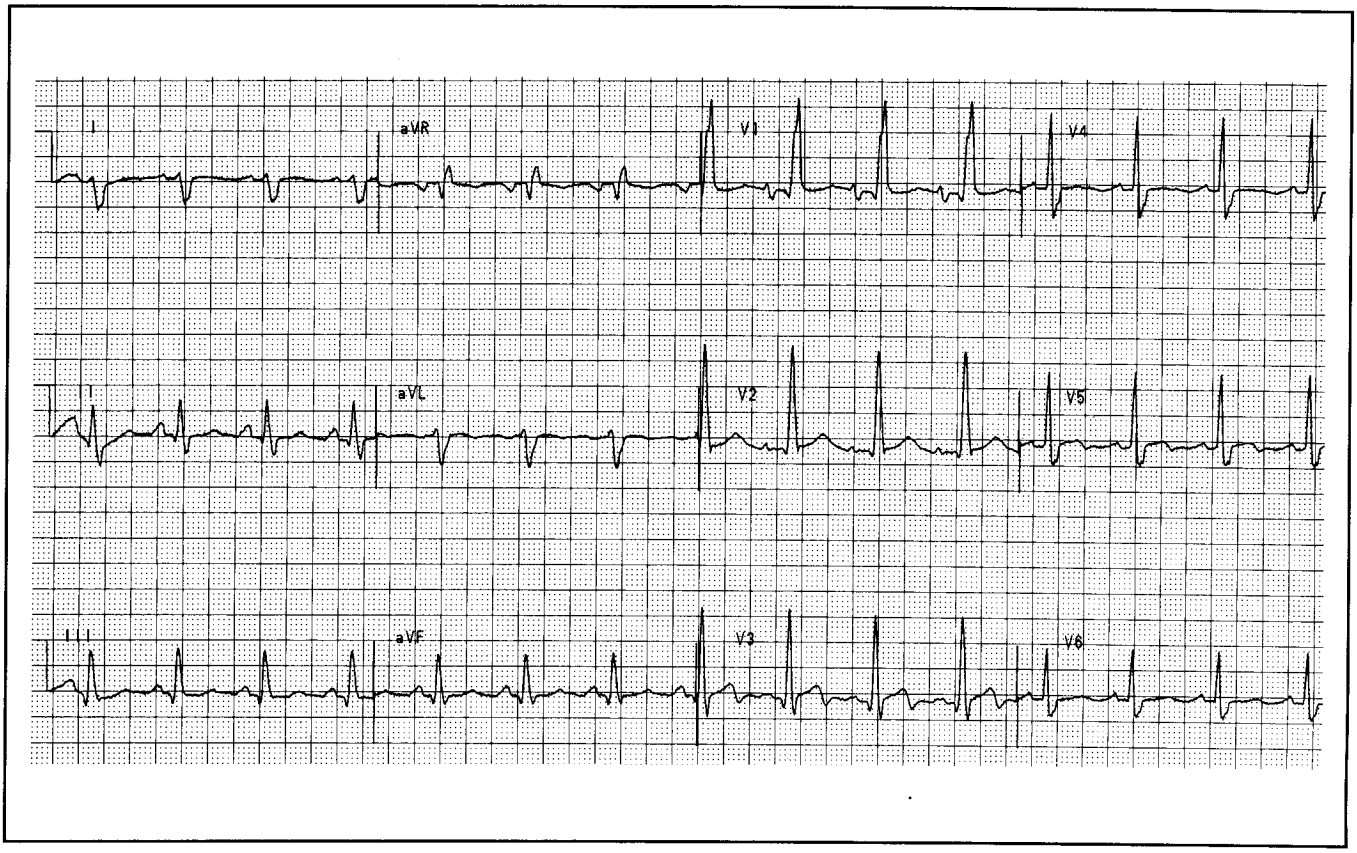
38. Which of the following groups is most likely to present with atypical complaints signaling myocardial infarction?
- a. Paraplegics
- b. The elderly
- c. Men
- d. Hypercholesterolemics

39. The most common anginal equivalent atypical complaint associated with acute myocardial infarction is:
- a. isolated emesis.
- b. syncope.
- c. new-onset, lower extremity edema.
- d. dyspnea.

40. Acute myocardial infarction may present with reproducible chest pain in approximately what percentage of cases?
- a. 15%
- b. 30%
- c. 50%
- d. 75%

An Abnormal ECG in an Asymptomatic Man

By Ken Grauer, MD



Clinical Scenario: The ECG shown in the Figure was obtained from a completely asymptomatic 56-year-old man. How would you interpret this ECG? What would you suspect the patient to have (have had)?

Interpretation: There is a normal sinus rhythm at a rate of 85 beats/minute. The PR interval is normal. However, everything else on this tracing is distinctly abnormal. The QRS complex is clearly prolonged in a pattern consistent with bifascicular block. Specifically, the qR complex in lead V_1 with tall R wave and the wide terminal S waves in leads I and V_6 are consistent with right bundle branch block (RBBB). In addition, the markedly deepened S wave component of the QRS complex in lead I, together with relatively positive complexes in the inferior leads, is consistent with the rightward axis of left posterior hemiblock (LPHB). The relatively tall and peaked P waves in lead II, and biphasic P wave in lead V_1 with peaked initial component and deep negative terminal component are consistent with biatrial enlargement. Small but definite Q waves are seen both in inferior and anterior precordial leads (the latter most likely responsible for loss of the rsR' pattern in lead V_1). Finally, ST segment and T wave morphology is abnormal: The upright T wave in lead V_1 suggests a primary T wave change (the T wave in lead V_1 with RBBB is usually directed *opposite* to the tall terminal R wave)—and beginning T wave inversion in leads V_3 and V_5 suggests an ischemic process.

In view of the fact that this patient is completely asymptomatic, the changes on this ECG are probably not acute. However, biatrial enlargement, bifascicular block, inferior and anterior Q waves, and abnormal ST-T wave changes all strongly suggest a cardiomyopathy that is most likely ischemic in etiology from prior silent infarction(s). At the least, further evaluation with an echocardiogram would seem warranted. ❖

In Future Issues:

Non-operative Management of Adult Blunt Splenic Trauma: Choosing the Right Candidates