AMERICAN HEALTH CONSULTANTS® A Medical Economics Company

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

Noninvasive Positive-Pressure Ventilation for Community-Acquired Pneumonia

ABSTRACT & COMMENTARY

Source: Confalonieri M, et al. Acute respiratory failure in patients with severe community-acquired pneumonia: A prospective randomized evaluation of noninvasive ventilation. *Am J Respir Crit Care Med* 1999; 160:1585-1591.

This multicenter, prospective, randomized trial compared standard treatment plus noninvasive positive pressure ventilation (NPPV) delivered through a face mask to standard treatment alone in patients with severe community-acquired pneumonia and acute respiratory failure. Exclusion criteria were: severe hemodynamic instability, requirement for emergent cardiopulmonary resuscitation, home mechanical ventilation or long-term oxygen supplementation, concomitant severe disease with a low expectation of life, inability to expectorate, or contraindications to the use of the mask.

There were 56 consecutive patients (28 in each arm) enrolled, and the two groups were similar at study entry. The need for endotracheal intubation was 21% in the NPPV groups and 50% in the standard treatment group (P = 0.03). The mean duration of intensive care unit stay was significantly lower in the NPPV group (1.8 days vs 6.0 days, P = 0.04). The two groups had a similar intensity of nursing care workload, time interval from study entry to endotracheal intubation, duration of hospitalization, and hospital mortality. Among patients with chronic obstructive pulmonary disease (COPD), those randomized to NPPV had a lower intensity of nursing care workload (P = 0.04) and improved two-month survival (88.9% vs 37.5%; P = 0.05).

COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

This study is important because it is the first randomized, controlled trial to evaluate NPPV in patients with acute respiratory failure caused by community-acquired pneumonia. The authors convincingly showed that NPPV was associated with a significant reduction in the rate of endotracheal intubation and duration of ICU stay. A post-hoc analysis was done (with significant limitations) that

Volume 6 • Number 10 • March 2000 • Pages 73-80

INSIDE

suggested a survival advantage at two months in patients with COPD.

Most of the evidence to date has suggested that the benefits of NPPV may be seen primarily in patients with a COPD exacerbation. A meta-analysis of controlled trails with NPPV concluded that in patients with COPD, there is strong evidence that NPPV decreases the need for intubation, decreases mortality, and shortens ICU stay.[1] Other potential advantages of NPPV include avoiding the complications of intubation such as ventilation-associated pneumonia and sinusitis, preserving airway defense mechanisms, improving patient comfort, preserving speech and swallowing, and improving clearance of respiratory secretions. The data are more limited in patients with acute respiratory failure not related to COPD. Patients with cardiogenic pulmonary edema appear to benefit from CPAP for certain outcomes, but further studies are needed to clarify the effects of NPPV on hemodynamics and infarction rates in these patients. A multicenter randomized trial in asthma patients is currently under way.

It is encouraging that we may be able to expand our use of NPPV to include carefully selected pneumonia patients. Even though the actual number of ED patients that fit these criteria may be quite small, this study provides us with additional rationale to consider using NPPV in patients (especially with COPD) who have a reasonable chance of needing intubation in the next few hours. ❖

References

1. Keenan SP, et al. Effect of noninvasive positive pressure ventilation on mortality in patients admitted with acute respiratory failure: A meta-analysis. *Crit Care Med* 1997;27:1685-1692.

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: Valerie Loner.
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.

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 6/99 with option to request yearly renewal. **For CME credit, add $50.**

Questions & Comments

Please call **Suzanne Zunic,** 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. Drs. Abbuhl, Chan, Elliot, 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.

Oligoanalgesia: What is the Correct Dose of Morphine?

ABSTRACT & COMMENTARY

Source: Todd KH, et al. Ethnicity and analgesic practice. *Ann Emerg Med* 2000;35:11-16.

One of the most essential and humane skills emergency physicians should possess is the ability to alleviate pain. Yet, in our daily practice, statements such as "you shouldn't give so much pain medication because the patient might get addicted," "please don't medicate the patient until I've had a chance to examine their abdomen," or "that patient doesn't look like they're in so much pain" are routinely echoed throughout the emergency department. Although these ancient, dogmatic and frankly barbaric concepts have been scientifically disproved, the tendency toward undermedication persists. While the phenomenon, known as oligoanalgesia, has been recognized for more than 10 years, Todd and colleagues have recently shed new light on this dark subject.

Using a retrospective design, the charts of 217 patients with isolated long-bone fractures were analyzed to identify trends in analgesic use. Shockingly, white patients were statistically more likely to receive analgesics than African-American patients (74% vs 57%). These results were unfortunately comparable to a previous study by the same authors, which demonstrated a similar relationship between analgesic use for hip fractures in Hispanic white patients and non-Hispanic white patients.[1]

COMMENT BY ROBERT HOFFMAN, MD

Two immediate problems warrant discussion. First, we should all be vocally appalled that the charts of only somewhere between 57% and 74% of patients with long-

bone fractures documented the need for and administration of analgesics. It is incumbent upon us all to begin quality assurance projects that expect no less than 100% compliance with the standard to assess, medicate, and reassess pain. Second, we should strive to increase our awareness of our own biases with regard to how we assess the need for and the choice of analgesics in all patients. Specifically, we must learn and then teach that how patients "look" may have little bearing on their need for analgesia. Routinely giving patients culturally meaningful scales to help them express and quantify their pain for us and allowing patients to have a more active role in the choice and amounts of their medication seem like easy first steps.

This week, I asked 10-15 emergency medicine residents a simple question: "What is the analgesic dose of morphine?" Some said nothing, some admitted they did not know, and a few said 0.1 mg/kg. Not a single one responded correctly that the analgesic dose of morphine is as much as is required to relieve the patient's pain. While I was glad for my patients that there would always be an attending physician at their bedside, I was concerned that such a fundamental concept was never learned on day one of our education. ❖

Reference

1. Todd KH. Ethnicity as a risk factor for inadequate emergency department analgesia. *JAMA* 1993;269: 1537-1539.

Minor CK-MB Elevations Not so Minor After All

ABSTRACT & COMMENTARY

Source: Alexander JH, et al. Association between minor elevations of creatine kinase-MB level and mortality in patients with acute coronary syndromes without ST-segment elevation. *JAMA* 2000;283:347-353.

The criteria used to diagnose myocardial infarction (MI) have remained the same for 20 years. These criteria require the presence of at least two of the following three elements to diagnose MI: a history of ischemic-type chest discomfort, evolutionary changes on serial electrocardiograms, and a rise and fall in serum cardiac enzymes. The authors used the 9461 patients enrolled in the PURSUIT trial to evaluate the relationship between peak CK-MB level (as an estimate of infarct size) and outcome, to determine whether a threshold CK-MB elevation exists below which there is no increased risk for mortality. The PURSUIT trial compared placebo to the glycoprotein IIb/IIIa inhibitor eptifibatide (Integrilin) for patients with cardiac chest pain without ST-elevation. Mortality at 30 days and six months was assessed according to peak CK-MB level (0-1, > 1-2, > 2-3, > 3-5, > 5-10, or >10 times the upper limit of normal). Mortality at 30 days and six months increased from 1.8% and 4.0% respectively in patients with normal peak CK-MB levels to 3.3% and 6.2% at peak CK-MB levels 1-2 times normal; to 5.1% and 7.5% at peak CK-MB levels 3-5 times normal; and to 8.3% and 11.0% at peak CK-MB levels greater than 10 times normal. In conclusion, this study demonstrates that in patients with cardiac chest pain without ST-segment elevation, a strong relationship exists between the magnitude of CK-MB elevation and mortality, and the risk begins just above the upper limit of normal. Alexander and associates go on to state that small CK-MB elevations represent clinically important evidence of myocardial necrosis and should be considered sufficient cardiac-marker criteria for a diagnosis of MI in patients with cardiac chest pain. Elevation of CK-MB above the upper limit of normal identifies a group of patients at higher risk of death.

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

The number of laboratory tools for finding the sick cardiac patients in the emergency center has grown in recent years. For example, since adding troponin I to our cardiac enzyme panel, I have identified a significant number of patients with myocardial infarction that I may have overlooked with simple isoenzymes and ECG findings. This article provides insight into another laboratory tool—the total CK-MB. Alexander et al provide solid evidence that one elevated total CK-MB seems to have some utility as a predictor of short-term mortality and probably should be regarded as evidence of acute MI. In my practice, that means I should be more aggressive when a patient with cardiac chest pain has a single elevated CK-MB, even with an ECG without acute injury pattern. Furthermore, I might be able to gauge the level of intervention on the degree to which the CK-MB is elevated. No data are available to show that a particular intervention (such as thrombolysis) may be beneficial, but this will guide the other difficult decisions we make everyday about the use of heparin, beta blockers, or intravenous nitrates in cardiac chest pain. Perhaps the 20-year-old World Health Organization guidelines need reconsideration, but I'll accept these data as a reason to lower my threshold for action now. ❖

Special Feature

The Additional Lead Electrocardiogram in Acute Myocardial Infarction

By William J. Brady, MD

The 12-lead electrocardiogram (ecg) is a less-than-perfect indicator of acute myocardial infarction (AMI). The sensitivity of a single 12-lead ECG for the diagnosis of AMI is relatively poor. It has been suggested that the sensitivity of the 12-lead ECG may be improved if three additional body surface leads are employed in selected individuals.[1,2] Acute posterior myocardial infarctions (PMI) and right ventricular myocardial infarctions (RVMI) are likely to be underdiagnosed, as the standard lead placement of the 12-lead ECG does not allow these areas to be assessed directly.[3] Additional leads frequently used include leads V_8 and V_9, which image the posterior wall of the left ventricle, and lead V4R (alternatively labeled RV4), which reflects the status of the right ventricle. *(See Figure 1 for correct placement of the additional ECG leads.)* The standard ECG, coupled with these additional leads, constitutes the 15-lead electrocardiogram, the most frequently employed extra-lead ECG in clinical practice. A more detailed description of the extent of the myocardial injury may be obtained if additional leads are used to augment the standard 12-lead ECG in selected patients.

Right Ventricular Infarction

The standard 12-lead ECG findings for RVMI include ST-segment elevation in the inferior distribution as well as in the right precordial chest leads, particularly lead V_1—perhaps the only lead on the standard ECG that reflects changes in the right ventricle. *(See Figure 2.)* At times, coexisting acute PMI may obscure the ST-segment elevation resulting from RVMI in lead V_1 as seen in the patient with the acute inferoposterior myocardial infarction with right ventricular involvement. Recordings from leads placed on the right side of the chest are much more sensitive and specific in detecting the changes of RVMI. The right-sided precordial electrodes are placed across the right side of the chest in a mirror image of the standard left-sided leads. They are labeled V_1R to V_6R (or RV_1 to RV_6). The clinician may use either these six right-sided leads or the single lead V_4R. Lead V_4R (right fifth intercostal space mid-clavicular line) is the most useful lead for detecting ST-segment elevation associated with RVMI and may be used solely in the evaluation of possible RVMI. *(See Figure 2.)* The ST-segment elevation that occurs in association with RVMI is frequently quite subtle *(see Figure 2)*, reflecting the relatively small muscle mass of the right ventricle. At other times, the ST-segment elevation is quite prominent and similar in appearance to the ST-segment changes seen in the standard 12-lead ECG. These changes are often transient, frequently resolving within 10 hours of the onset of symptoms.

Posterior Wall Infarction

On the standard 12-lead ECG, changes associated with necrosis of the posterior wall of the left ventricle are reflected in the anterior chest leads.[4] These electrodes are opposite rather than adjacent to the site of damage, and the changes seen are the reverse of what one would normally expect. From the perspective of the standard 12-lead ECG, the "typical" findings indicative of transmural AMI will be reversed. This reversal results from the fact that the endocardial surface of the posterior wall faces the anterior precordial leads (V_1 through V_3)

Figure 1

Lead Placement for Additional Lead ECGs

Correct placement of the additional leads V_4R, V_7, V_8, and V_9. Placement of lead V_4R (large arrow, alternatively termed RV_4) is depicted. The posterior leads, V_8 and V_9 (large arrows), are located on the patient's left posterior thorax in the following fashion: lead V_8 at the tip of the scapula and V_9 halfway between lead V_8 and the left border of the vertebral bodies. Lead V_7 (small arrow) may also be added; if used, it is placed equidistant from lead V_8 to V_9, along the posterior axillary line, the same distance from V_8 as is V_9.

Figure 2

ECG Changes: Acute Inferior MI with Right Ventricular Involvement

Electrocardiographic complexes in a patient with inferior AMI with right ventricular involvement. The typical ST segment elevation is seen here in the inferior leads II, III, and aVf. This patient developed marked hypotension after nitroglycerin administration, suggestive of right ventricular infarct. Analysis of lead RV_4 revealed ST segment elevation (arrow) of approximately 1.5 mm, diagnostic of an acute right ventricular myocardial infarction.

in the standard 12-lead ECG. ST-segment depression, prominent R waves, and upright T waves in leads V_1 through V_3—"when reversed"—may represent the ST-segment elevation, Q waves, and T-wave inversions, respectively, of acute PMI. If one considers the "reverse nature" of these ECG abnormalities when applied to the posterior wall, the findings assume a more recognizable, ominous meaning.

Abnormalities noted on the standard 12-lead ECG suggestive of acute PMI include the following (in leads V_1, V_2, and/or V_3): horizontal ST-segment depression; a tall, upright T wave; a tall, wide R wave; and an R/S wave ratio > 1.0. *(See Figure 3.)* Further, the combination of horizontal ST-segment depression with an upright T wave increases the diagnostic accuracy of these two separate electrocardiographic findings. It must be remembered that a dominant R wave—which is equivalent to an evolving Q wave—takes a number of hours to develop and therefore is not frequently seen on the initial ECG.[5,6]

Summary

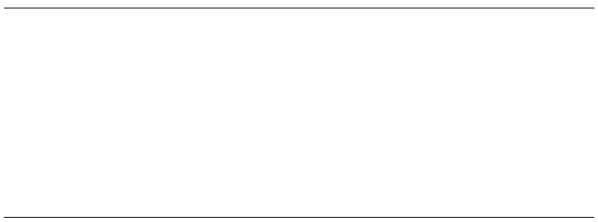
The use of the 15-lead ECG may help define the full extent of myocardial injury in patients with AMI. The use of additional leads may confirm the diagnosis of RVI in hypotensive patients presenting with an inferior AMI. Alternatively, in patients with inferior AMI, the detection of RVMI prior to the development of hypotension will assist the clinician in the proper, "gentle" use of vasodilating agents. In patients with ST-segment depression in leads V_1-V_3, the use of the additional leads V_8 and V_9 may help to distinguish between a PMI, anterior wall ischemia, and reciprocal changes. The discovery of an isolated PMI—i.e., the distinction from anterior wall ischemia—will enable the physician to offer appropriate therapy in the most expeditious fashion. The 15-lead ECG is recommended in any patient presenting with an inferior AMI, any infarction involving the lateral wall of the left ventricle, or any patient with ST segment depression in leads V_1 - V_3. ❖

References

1. Pollack M, et al. Emergency department diagnosis of acute posterior-wall myocardial infarction using left posterior chest leads. *Acad Emerg Med* 1997;34:399 (abstract).
2. Melendez LJ, et al. Usefulness of three additional electrocardiographic chest leads (V_7, V_8, and V_9) in the

Figure 3

ECG Changes: Acute Posterior MI



Electrocardiographic abnormalities suggestive of acute posterior infarction include the following (in leads V_1, V_2, or V_3): [1] horizontal ST segment depression; [2] a tall, upright T wave; [3] a tall, wide R wave; and [4] an R/S wave ratio > 1.0 (in lead V_2 only). In this example, the right precordial leads V_1 and V_2 demonstrate the classic findings of acute posterior wall myocardial infarction. "A" refers to the prominent R wave, which is unusual in the right precordial leads. "B" refers to the ST-segment depression; here, lead V_1 demonstrates horizontal depression. "C" indicates the upright T wave, which, when encountered in the setting of ST-segment depression, is strongly suggestive of acute posterior wall infarction. Posterior leads V_8 and V_9 demonstrate ST-segment elevation. Note that the degree of elevation is less pronounced than expected with acute infarction.

diagnosis of acute myocardial infarction. *CMAJ* 1978; 119:745-748.
3. Rich MW, et al. Electrocardiographic diagnosis of remote posterior wall myocardial infarction using unipolar posterior lead V_9. *Chest* 1989;96:489-493.
4. Conover MB. *Understanding Electrocardiography: Arrhythmias and the 12-lead ECG.* 6th ed. St. Louis: Mosby-Year Book; 1992.
5. Goldberger AL. *Myocardial Infarction: Electrocardiographic Differential Diagnosis.* 4th ed. St. Louis; Mosby; 1991.
6. Aufderheide TP, Brady WJ. Electrocardiography in the Patient with Myocardial Ischemia or Infarction. In: Gibler WB, Aufderheide TP, eds. *Emergency Cardiac Care.* St. Louis: Mosby-Year Book; 1994.

Pharmacology Update

Aggrenox

By William T. Elliott, MD, FACP, and James Chan, PharmD, PhD

Boehringer ingelheim pharmaceuticals has teamed two old staples—aspirin and dipyridamole—to create a new agent for the prevention of stroke in patients who have had a previous stroke or a transient ischemic attack (TIA). The company received FDA approval in November for aspirin and extended release dipyridamole which will be marketed under the trade name Aggrenox. It joins aspirin, ticlopidine, and clopidogrel on the list of drugs that are used for stroke prevention.

Indications

Aggrenox is approved to reduce the risk of stroke in patients who have had transient ischemia of the brain or complete ischemic stroke due to thrombosis.

Dosage

The recommended dose is aspirin 25 mg and dipyridamole 200 mg (1 Aggrenox capsule) twice daily. Drug-food interaction has not been studied.[1]

Potential Advantages

The fixed combination product offers two different mechanisms of antiplatelet action. Aspirin is a cyclooxygenase inhibitor, while dipyridamole is believed to affect platelet aggregation by inhibiting phosphodiesterase.[2] Results from the two-year European Stroke Prevention Study (ESPS-2) of 6602 patients indicated that this fixed combination reduced the risk of stroke (fatal or nonfatal) by 37% compared to placebo. There was also a 16.3% reduction in stroke end points compared to dipyridamole alone and an 18.1% reduction over aspirin alone (50 mg/day).[6]

Potential Disadvantages

The frequency of common side effects include headache (38.2% vs 33.1% for aspirin only) and diarrhea (12.1% vs 6.6% for aspirin alone). These tend to diminish over time.[2] Aggrenox has been associated with a decline in hemoglobin of 0.25 g/dL, hematocrit of 0.75%, and erythrocyte count of $0.13 \times 106/mm^3$.[1]

Comments

The ESPS-2 trial is the first trial to demonstrate that a fixed combination of aspirin and dipyridamole is more effective than aspirin alone. However, the 50 mg daily aspirin dose used in the trial was at the low end of

the recommended dose range (50-325 mg). The results from previous trials of this combination have generally been unimpressive. After a review of randomized trials involving combinations of aspirin and dipyridamole, the Antiplatelet Trialists concluded that the difference between aspirin and dipyridamole and aspirin alone is likely to be smaller than the difference between antiplatelet and no antiplatelet treatment.[3,4] The ESPS-2 results, based on an intent-to-treat analysis, indicated that the fixed combination, compared to aspirin alone, reduced the risk of all strokes (22%, P = 0.008) and frequency of TIAs (24.4%, P < 0.001). However, no statistical difference was observed in combined end points of stroke or death, death from any cause, or myocardial infarction. The rates of myocardial infarction were, however, low in the study groups.[1] While therapy may lengthen the time to a subsequent stroke, it does not appear to affect the severity of the recurrent stroke.[5]

Aggrenox costs $2.95 per day, which is significantly more than aspirin and generic dipyridamole (< $1.00). The manufacturer states in Aggrenox labeling that this product is not interchangeable with the individual components of aspirin and dipyridamole, although this claim does not seem to be backed up by scientific evidence.

Clinical Implications

Stroke is the third leading cause of death after heart disease and cancer, and is the leading cause of serious, long-term disability. About 730,000 people have a stroke each year and, of these, more than 80% are first attacks.[8] Risk factors for stroke include increasing age, male gender, hypertension, hyperlipidemia, diabetes, carotid artery disease, heart disease, tobacco, previous stroke, and transient ischemic attacks. Unless contraindicated, antiplatelet therapy is recommended for stroke prevention in persons with a history of transient ischemic attack or a previous thromboembolic stroke.

Aspirin has been the most frequently prescribed drug and is considered the standard. To balance effectiveness and tolerability, a lower dose of aspirin (50-325 mg) has been recommended.[7] Several newer products have been approved by the FDA on the basis of studies compared to aspirin. These include ticlopidine, clopidogrel, and the fixed combination of aspirin/dipyridamole, all of which have shown varying degrees of benefit over aspirin. It is not clear if one of these products would replace aspirin as the standard. There are currently no comparative trials among the newer products. ❖

References

1. Aggrenox Product Information. November 1999. Boehringer Ingelheim Pharmaceuticals.
2. Hervey PS, et al. *Drugs* 1999;58:469-475.
3. Wilterdin JL, et al. *Arch Neurol* 1999;56:1087-1092.
4. Antiplatelet Trialists' Collaboration. *BMJ* 1994;308: 81-106.
5. Sivenius J, et al. *Neurology* 1999;53:825-829.
6. ESPS Group. European Stroke Prevention Study. *Stroke* 1990;21:1122-1130.
7. Albers GW, et al. *Neurology* 1999;53(7 Suppl 4): S25-31.
8. Statistics from the National Stroke Association and American Heart Association.

CME Questions

20. Electrocardiographic findings associated with acute posterior wall myocardial infarction include all of the following *except*:

a. ST-segment depression in leads V_1 and V_2.
b. Upright T waves in leads V_1 and V_2.
c. ST-segment elevation in leads V_1 and V_2.
d. ST-segment elevation in leads V_8 and V_9.

21. All of the following are true about noninvasive positive pressure ventilation (NPPV) *except*:

a. Several studies have shown that NPPV decreases the need for intubation in many chronic obstructive pulmonary disease (COPD) patients.
b. NPPV has been shown to decrease mortality in COPD patients in some studies.
c. In patients with acute respiratory failure and severe community-acquired pneumonia, NPPV has been shown to significantly decrease the need for intubation when compared to a control group.
d. In the randomized, controlled trial of NPPV in patients with respiratory failure and community-acquired pneumonia, there was no difference in the length of ICU stay in the two groups.

22. Electrocardiographic findings associated with acute right ventricular myocardial infarction include:

a. T wave inversion in V_8.
b. ST-segment depression in V_4R.
c. ST-segment elevation in V_4.
d. ST-segment elevation in V_4R.

23. Elevations of CK-MB:

a. are meaningless unless accompanied by EKG changes suggestive of acute myocardial infarction.
b. are meaningless unless present on consecutive serum samples.
c. may represent clinically significant myocardial necrosis.
d. may represent clinically significant myocardial necrosis only if they are elevated 10 times normal.

24. A recent study of analgesia practices in ED patients with long-bone fractures:

a. demonstrated analgesia was not given equally to black and white patients.
b. found analgesia to be adequate in all patients.
c. found analgesia to be adequate in all white patients.
d. demonstrated age differences in analgesia.

Clearance for Surgery?

By Ken Grauer, MD

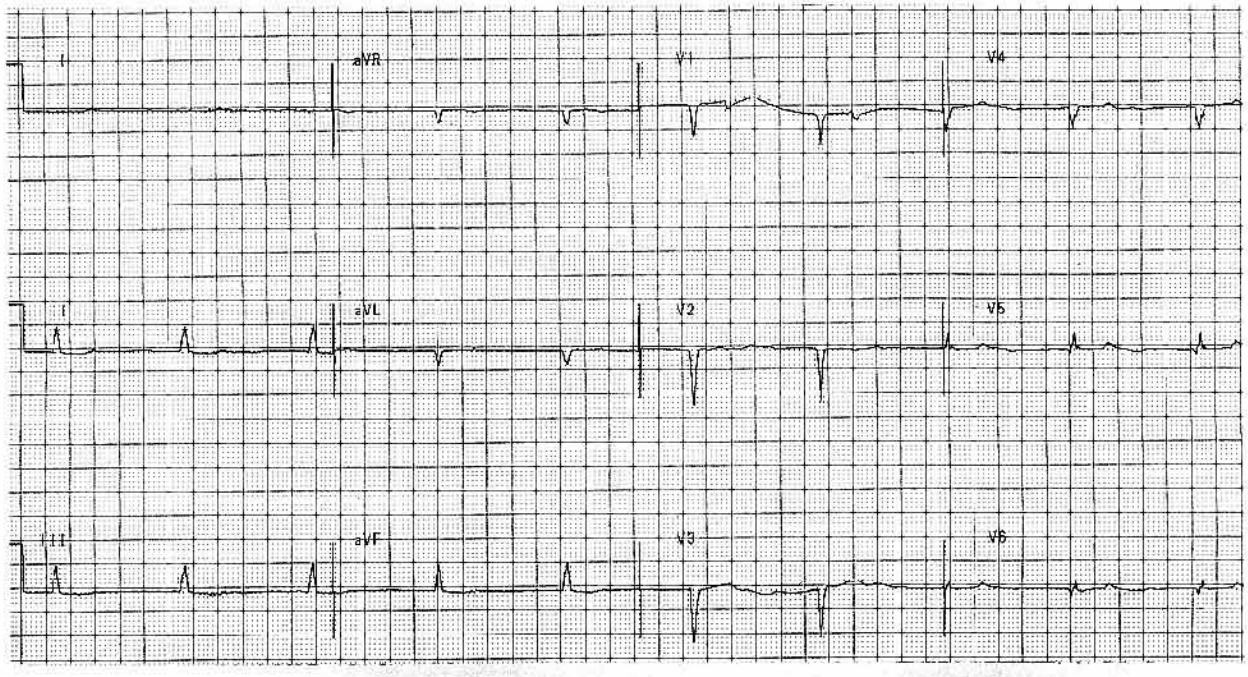


Figure. Preoperative 12-lead tracing obtained from a 78-year-old man on multiple medications.

Clinical Scenario: The ECG shown in the figure was obtained from a 78-year-old man as part of his routine preoperative clearance evaluation. The patient had a history of ischemic heart disease and heart failure. He was on multiple medications. He was completely asymptomatic at the time this ECG was recorded. Would you clear him for surgery?

Interpretation: The rhythm is regular at a rate of between 55 and 60 beats/min. Notable for its absence is the lack of an upright P wave in lead II. Given that the QRS complex is narrow, this defines the rhythm as junctional. The small amplitude upright deflection in lead V_1 may represent a retrograde P wave, albeit with a prolonged R-P interval. Otherwise, the axis is vertical, there is evidence of prior anterolateral infarction, and diffuse ST segment and T wave flattening is seen. However, there are no acute changes.

In view of the fact that the patient has underlying heart disease, one has to inquire if digoxin is among the multiple medications he is taking. In point of fact, this patient's serum digoxin level was in the toxic range.

Digoxin toxicity is notorious for producing a variety of cardiac arrhythmias, especially accelerated junctional rhythms. The presence of an underlying conduction disorder in this patient may have accounted for the relatively slow rate of this digitalis-induced arrhythmia. The patient's operation should be delayed until his digoxin level returns to the therapeutic range. ❖

In Future Issues: Plain Film Imaging of Mandibular Fractures