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## TPA in Stroke: The Community Experience

ABSTRACT & COMMENTARY

**Source:** Katzan IL, et al. Use of tissue-type plasminogen activator for acute ischemic stroke: The Cleveland area experience. *JAMA* 2000;283:1151-1158.

The ninds study, published in 1995, demonstrated a beneficial effect of intravenous (IV) tissue plasminogen activator (TPA) for the treatment of ischemic stroke patients presenting within three hours of symptom onset.[1] This led to a dramatic alteration in how ischemic stroke is approached. In order to minimize the risks of TPA (which include a tenfold increased risk of intracerebral hemorrhage [ICH]), several authorities developed guidelines regarding the use of thrombolysis in ischemic stroke. Few data exist regarding the outcomes of patients treated with IV TPA for ischemic stroke outside of the investigational setting. This cooperative study conducted in 29 Cleveland-area hospitals examined patients admitted with a primary diagnosis of ischemic stroke. Primary outcome measures included rate of TPA use, occurrence of symptomatic ICH, proportion of patients receiving TPA not in conjunction with published guidelines, and in-hospital mortality.

Over one year, 3948 patients with acute ischemic stroke were identified. Of these, 70 (1.8%) received IV TPA. Eleven of these patients (15.7%) had a symptomatic ICH. Half of the patients receiving IV TPA displayed deviations from criteria for TPA administration as specified in national guidelines. There was a significantly higher rate of in-hospital mortality among patients receiving TPA compared with patients not receiving TPA (15.7% vs 5.1%). The authors concluded that the Cleveland area experience with IV TPA for ischemic stroke differed from that reported in clinical trials.

### COMMENT BY JACOB W. UFBERG, MD

Several studies have shown good outcomes and low symptomatic ICH rates among patients treated with IV TPA for acute ischemic stroke within the three-hour window. These include not only the NINDS trial, but also the STARS trial, which is found in the same issue of *JAMA* as this study.[2] As Katzen et al point out, however,

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## INSIDE

many of these trials involved researchers experienced with the use of TPA in stroke, and several were performed in centers that participated in the NINDS or ATLANTIS trials.

This article is among the first to examine the use of IV TPA for stroke outside of a trial setting. The Cleveland area's high rate of symptomatic ICH is alarming, as is the threefold risk of in-hospital mortality in TPA-treated patients. Equally concerning is the 50% incidence of guideline violations among these 29 hospitals. The STARS study, also a prospective multicenter trial, showed a 35% rate of guideline violations in TPA-treated patients. This highlights one of the major problems in the use of TPA for acute ischemic stroke: If academic neurologists have such great difficulty adhering to the inclusion and exclusion criteria for the use of TPA, how can the rest of us be expected to use TPA properly? ❖

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2. Albers GW, et al. Intravenous tissue-type plasminogen activator for treatment of acute stroke: The Standard Treatment with Alteplase to Reverse Stroke (STARS) study. *JAMA* 2000;283:1145-1150.



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# Driving and Antihistamines

ABSTRACT & COMMENTARY

**Source:** Weiler JM, et al. Effects of fexofenadine, diphenhydramine, and alcohol on driving performance. *Ann Intern Med* 2000;132:354-363.

The objective of this trial was to compare the effects of fexofenadine (60 mg), diphenhydramine (50 mg), alcohol (approximately 0.1% blood concentration), and placebo on various measures of driving performance. A randomized, double-blind, double-dummy, crossover design was used so that each participant received all four treatments on four successive sessions at weekly intervals in a driving simulator. The participants were 40 licensed drivers with seasonal allergic rhinitis, ages 25 to 44 years, including 15 men and 25 women.

The primary outcome measure was coherence, a measure of a participant's ability to maintain a constant distance from a lead car that varied its speed randomly. Secondary outcome measures were self-reported drowsiness and other driving end points, including lane-keeping, minimum following distance, steering instability, and response to an unexpected blocking vehicle.

Pairwise comparisons revealed that the diphenhydramine group performed car-following with significantly less coherence (0.877 ± 0.019 CI, 0.837-0.911) than the alcohol group (0.920 ± 0.014 CI, 0.891-0.945), fexofenadine group (0.915 ± 0.014 CI, 0.884-0.940), or the placebo group (0.906 ± 0.015 CI, 0.875-0.933). Lane-keeping was impaired after alcohol and diphenhydramine use compared with fexofenadine and placebo. After consuming alcohol, participants performed car-following at significantly smaller minimum following distances (15.1 m) than they did after taking fexofenadine (17.1 m) or placebo (17.4 m). Self-reported drowsiness did not predict lack of coherence and was weakly associated with the other secondary end points.

### COMMENT BY STEPHANIE B. ABBUHL, MD, FACEP

One could argue that the statistically significant differences in the outcome measures of this study do not necessarily translate into clinically significant differences. It is possible that impairment of coherence in a

driving simulator does not predict poor driving performance in real life. In fact, it is not entirely clear why coherence was chosen as the primary outcome measure; an accompanying editorial points out that coherence has not been validated as a true indicator of risk for motor vehicle crashes (MVC).[1]

On the other hand, coherence and the other secondary measures may actually predict a real risk of MVC. Given that we do not have good studies to answer the real-life question, and considering that multiple other studies have shown that sedating antihistamines impair psychomotor performance, it behooves us to prescribe a nonsedating antihistamine for patients who must drive or operate machinery/equipment. If cost prohibits prescribing the nonsedating antihistamines, then strict warnings must accompany discharge instructions so that patients avoid driving or any other task that involves sharp psychomotor performance. It is disturbing that most of the 39 million persons in the United States with allergic rhinitis take over-the-counter medications, which generally contain a first-generation antihistamine.

**Reference**

1. Hennessy S, Strom BL. Nonsedating antihistamines should be preferred over sedating antihistamines in patients who drive. *Ann Intern Med* 2000;132:405-407 (editorial).

# Emergency Cricothyrotomy: Standard or Seldinger?

ABSTRACT & COMMENTARY

**Source:** Eisenburger P, et al. Comparison of conventional surgical versus Seldinger technique in emergency cricothyrotomy performed by inexperienced clinicians. *Anesthesiology* 2000;92:687-690.

The authors compared the first-time performance of surgical (group 1) vs. Seldinger technique (group 2) for cricothyrotomy. Twenty critical care physicians with significant prior intubating experience were randomized to perform each procedure on two adult human cadavers. None had ever performed a cricothyrotomy. Participants completed a 60-minute training session, reviewing the pertinent anatomy and the tools/steps required to complete each procedure. Both procedures were performed using standard methods previously described in the literature. Times to location of the cricothyroid membrane (CTM), tracheal puncture, and first ventilation were recorded. Each participant was allowed only one attempt per procedure.

Tracheal placement was successful for 70% of group 1 vs. 60% of group 2 (P = NS). Failure in group 1 resulted from unsuccessful attempts to locate the CTM (n = 1), abortion of the procedure due to incorrectly presumed esophageal placement (n = 1), and cannula misplacement (paratracheal = 1, esophageal = 1, subcutaneous = 2). Failure in group 2 resulted from kinking of the guide wire (n = 5) and cannula misplacement (paratracheal = 1, esophageal = 1, subcutaneous = 1). Time intervals (mean ± SD) were as follows: 7 ± 9 sec (group 1) vs. 8 ± 7 sec (group 2) for location of the CTM; 46 ± 37 sec (group 1) vs. 30 ± 28 sec (group 2) for tracheal puncture; and 102 ± 42 sec (group 1) vs. 100 ± 46 sec (group 2) for first ventilation. There was no statistical difference between groups for any of the time points measured.

### COMMENT BY MICHAEL A. GIBBS, MD, FACEP

Rapid sequence intubation (RSI) is now the standard for emergency airway management. Success rates are high (97-99%), and surgical airway rescue is rarely required (0.5-2%).[1-3] An inevitable consequence of successful RSI is that most emergency physicians will have very limited (if any) experience with surgical airway management. The introduction of other airway rescue devices (e.g., Combitube, laryngeal mask airway, retrograde intubation, lighted stylet) make the need for cricothyrotomy even less likely. Yet, the final pathway for all failed airway algorithms remains the surgical airway.

The results of this study bring out several important points. First, it is difficult to perform cricothyrotomy after didactic training alone. It is unrealistic to perform a life-saving procedure on a dying patient having only read about it in a textbook! Emergency physicians must seek out opportunities to perfect this crucial skill on realistic models (e.g., animal, human cadaver, post-mortem). Second, the success rate of surgical and Seldinger cricothyrotomy is similarly unsatisfactory in inexperienced hands. Selecting one of the techniques and learning to perform it well is probably more important than which technique is chosen. Third, there is a significant risk of cannula misplacement with both techniques, and guide wire kinking is a common problem with the Seldinger technique.

The "cannot ventilate/cannot intubate" patient is the most feared clinical scenario in emergency medicine. All emergency physicians using neuromuscular blockade must become familiar with alternative airway rescue techniques, as well as cricothyrotomy, be it surgical or Seldinger. ❖

**References**

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3. Walls RM, et al. 2,392 emergency department intubations: First report of the ongoing National Emergency Airway Registry Study (NEAR 97). *Ann Emerg Med* 1999;34:814 (abstract).

# Aseptic vs. Bacterial Meningitis: What's the Diff?

ABSTRACT & COMMENTARY

**Source:** Negrini B, et al. Cerebrospinal fluid findings in aseptic versus bacterial meningitis. *Pediatrics* 2000;105:316-319.

When a patient who is being evaluated for meningitis has a minimally elevated cerebrospinal fluid (CSF) count without a predominance of polymorphonuclear (PMN) cells, the diagnosis of aseptic meningitis is often considered. What of the same patient with predominantly PMN cells seen on the differential 24 hours after the onset of illness—could this still be aseptic meningitis? The answer, in contrast to typical teaching, may be yes. Standard textbooks describe the typical CSF findings in aseptic meningitis as a pleocytosis of between 20 and 1000 white blood cells (WBCs), composed mainly of lymphocytes. Although some patients with aseptic meningitis do have PMN predominance in the CSF, most studies have explained this as an early phenomenon, which is followed by a shift to mononuclear/lymphocyte cells within 24 hours. This often leads to uncertainty in the diagnosis and treatment of meningitis and is a source of confusion and controversy.

The authors performed a retrospective chart review of 158 cases of meningitis in children 30 days to 18 years of age hospitalized during the peak months for enteroviral meningitis (April to October) between 1992 and 1997. There were 138 cases of aseptic meningitis (defined as having at least 20 WBCs/mm$^3$ and the absence of bacterial growth on culture). The remaining 20 cases were diagnosed with bacterial meningitis (positive CSF culture or the presence of a CSF pleocytosis with positive cultures of the blood). CSF variables, including WBC differential and time from the onset of symptoms to the performance of a lumbar puncture, were analyzed. PMNs were considered to be predominant when the percentage of neutrophils added to juvenile forms was greater than 50%. Patients were excluded if they had received antibiotic therapy within the previous five days.

The results demonstrated that the percentage of CSF PMNs in aseptic meningitis was not statistically different for patients who had a lumbar puncture performed either within or beyond 24 hours of the onset of symptoms. Fifty-one percent of the 53 patients with aseptic meningitis and duration of illness greater than 24 hours had a PMN predominance. The mean CSF WBC and PMN percentage for aseptic meningitis was 391 ± 568 and 52% ± 32%, respectively. For the bacterial meningitis patients, the mean CSF WBC was 3461 ± 5841 with 78% ± 18% neutrophils. The sensitivity of a PMN predominance for aseptic meningitis was 57%, whereas the specificity was 10%. The positive predictive value of a PMN predominance for aseptic disease was 81%, but the negative predictive value was 3%. Alternative definitions of PMN predominance from 60% to 90% were not useful as a clinical indicator of bacterial disease. In conclusion, the majority of children in this study with aseptic meningitis had a PMN predominance in the CSF. The PMN predominance was not limited to the first 24 hours of illness. A review of the literature by the authors identified other investigations that report similar findings.

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

In the ED, the diagnosis of aseptic meningitis must be made carefully. For the conservative physician, the patient must be clinically stable and must have a normal mental status, a modest cell count (less than 50 cells/mm$^3$), no PMNs, and a normal glucose, protein, Gram stain, and opening pressure. The use of less conservative cutoffs increases the risk of misdiagnosis. To make the diagnosis even more risk-free, a test for bacterial antigens such as countercurrent immunoelectrophoresis or latex agglutination test should be negative. This paper is interesting because it demonstrates that during enteroviral season, most patients actually have aseptic meningitis, even though they may have more than 50% PMNs and pleocytosis on CSF fluid analysis. It also reminds ED physicians that we admit patients for meningitis not because the source is often bacterial—often it is not—but because it is a serious illness with the potential for deterioration, even if the source is not bacterial. ❖

Special Feature

# Adverse Cardiovascular Consequences of Atropine Administration

*By William J. Brady, MD*

Hemodynamic instability due to bradyarrhythmia is a common event in patients with acute coronary ischemia (ACI). The use of atropine has been widely recommended in these circumstances.[1-3] In fact, the American Heart Association's Advanced Cardiac Life Support guidelines for the treatment of hemodynamically compromising bradycardia and atrioventricular (AV) block include the early use of atropine.[1] Atropine is a parasympatholytic drug that enhances both sinus node automaticity and AV conduction via its direct vagolytic action on AV junctional tissue and subjunctional components of the cardiac conduction system. This mechanism is worth remembering when treating the patient with a transplanted heart, because it renders atropine useless in the denervated heart.

The medical literature contains numerous descriptions evaluating the use of atropine in the prehospital and hospitalized ACI patient. Atropine has been shown to be most effective in patients experiencing ACI compared to patients with nonischemic bradyarrhythmia.[2,3] In the vast majority of cases, atropine administration was associated with either no alteration in the patient's condition or an improvement in the clinical situation.[2,3] While atropine is the drug of choice for compromising bradyarrhythmia in the setting of ACI, it has rarely been associated with the development of adverse consequences.[1-5] These adverse effects are uncommon, despite rather pronounced warning statements in the American Heart Association's Advanced Cardiac Life Support guidelines and other references.[1]

### Adverse effects of atropine

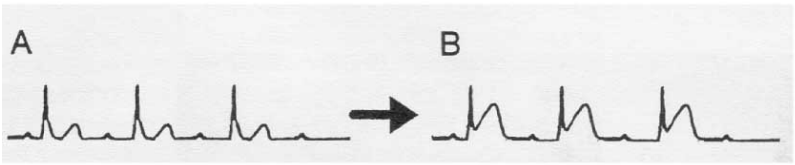
Adverse sequelae of atropine include the potentiation of ACI, a pro-arrhythmic effect, and the worsening of high-grade atrioventricular block.[1-5] Atropine may worsen the ischemia during ACI, such as in the patient noted in Figure 1, an elderly male with second-degree, type I AV block who received atropine and soon after developed chest pain and ST segment elevation.[1] *(See Figure 1.)* Such a complication has not been reported in a prehospital population.[2,3] This case patient demonstrates a possible association of atropine administration with ACI potentiation—the "conversion" of acute ischemia to acute myocardial infarction (AMI). The use of atropine in this instance remains a reasonable option and should be strongly considered. Undoubtedly, acute ischemia is intensified in some cases by hypoperfusion due to vagally mediated bradyarrhythmia; atropine is the antidote for such situations. Once again, an awareness of this potential adverse reaction coupled with a prudent selection of candidates for atropine therapy will demonstrate the risk/benefit ratio in each individual patient and will guide the clinician accordingly.

Regarding arrhythmogenicity, atropine actually has a low rate of such complications. Warren and associates noted a 4% adverse reaction rate in prehospital patients with bradyarrhythmia treated with atropine; one patient developed ventricular fibrillation (VF), while another experienced symptomatic ventricular ectopy requiring therapy.[4] VF has been an infrequent arrhythmic complication of atropine use in patients with symptomatic bradyarrhythmia, particularly involving an ischemic pathophysiology.[5] Recent reports investigating the out-of-hospital patient with unstable bradyarrhythmia noted a similarly low rate of proarrhythmia.[2,3] Four patients (2.3%) in this study had an adverse response to atropine administration. Three ACI patients developed frequent premature ventricular contractions; one experienced ventricular tachycardia with a second episode of ventricular tachycardia in the ED.[2,3] Authorities make the observation that such ventricular arrhythmic adverse reactions are quite rare and recommend an awareness of this complication together with judicious selection of patients for atropine therapy.[5]

Figure 2 depicts such a complication *(see Figure 2)*. An elderly male with sinus bradycardia and hypotension

**Figure 1**

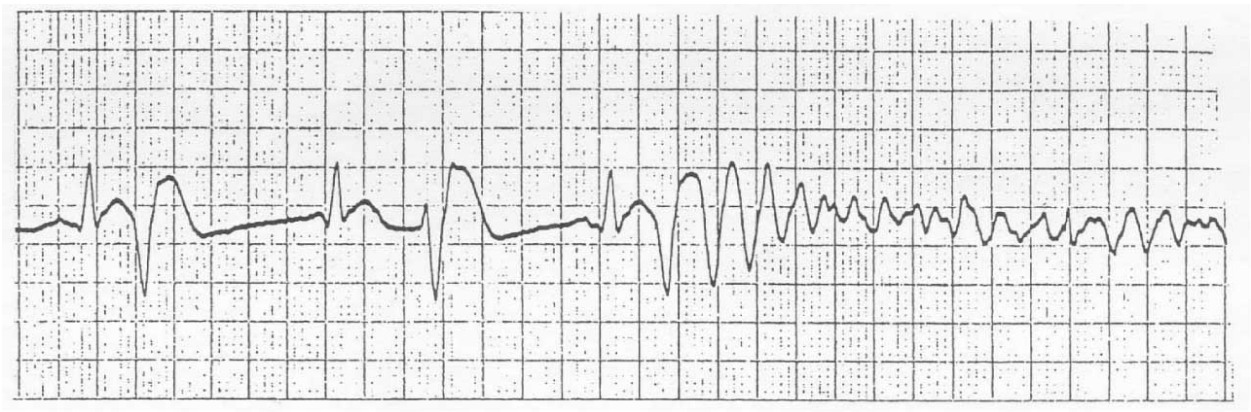
**Potentiation of Ischemia**




An elderly male called 911 because of chest pain. EMS found the patient lethargic and hypotensive. **A:** Prehospital rhythm strip demonstrating third-degree AV block—pre-atropine administration. Note the normal ST segments. **B:** Prehospital rhythm strip demonstrating third-degree AV block at a similar rate with ST segment elevation, suggesting development of AMI. This rhythm strip was obtained soon after atropine administration. Subsequent ECG revealed inferior AMI.

**Figure 1**

**Proarrhythmic Effect**



An elderly male presented to the ED with chest pain and syncope. The initial rhythm strip revealed sinus bradycardia at a rate of approximately 40 bpm. The examination was significant for diaphoresis and hypotension. The patient received atropine 1 mg intravenously. Soon after administration, the patient became unresponsive; the monitor revealed coarse ventricular fibrillation, which was successfully defibrillated to sinus tachycardia. Subsequent evaluation revealed an inferior AMI that was managed with a thrombolytic agent.

received atropine; approximately 90 seconds later, ventricular ectopy was noted with an R-on-T PVC resulting in VF.

A marked reduction in heart rate—a paradoxical worsening of the block—has also been observed in patients with third-degree AV block after atropine treatment in hospital-based scenarios. Interestingly, such paradoxical cardiac slowing has not been reported in the prehospital literature,[2-4] although it is described in hospital-based reports as well as major textbooks and the Advanced Cardiac Life Support guidelines.[1] This paradoxical slowing has been reported rarely in patients with infranodal block, i.e., Mobitz type II second-degree AV block and third-degree AV block with a wide QRS complex. Fortunately, the majority of patients with these rhythms do not manifest this paradoxical reaction.[1] Again, as with the proarrhythmic effects and potentiation of ischemia, careful selection of patients and preparedness for such an event are musts for the emergency physician.

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# CME Questions

**30. In the study comparing the effects of fexofenadine, diphenhydramine, alcohol, and placebo on driving performance, all of the following are true *except*:**
a. Study participants had significantly better coherence (a measure of driving performance) after taking fexofenadine than after diphenhydramine.
b. Self-reported drowsiness predicted lack of coherence.
c. Participants under the influence of alcohol did surprisingly well on the coherence outcome measure, but they kept a shorter distance to the car they were following and had less control over lane-keeping.
d. Coherence is one of many measures of driving performance in a driving simulator and has not been validated as a true indicator of risk for motor vehicle crashes.

**31. According to currently accepted guidelines, what is the time limit (i.e., time after symptom onset) for treating ischemic stroke with intravenous tPA?**
a. One hour
b. Three hours
c. Six hours
d. 12 hours

**32. The CSF shows a predominance of polymorphonuclear cells after lumbar puncture is performed 36 hours after the onset of fever and severe headache in a 5-year-old. The diagnosis is:**
a. bacterial meningitis.
b. aseptic meningitis.
c. bacterial or aseptic meningitis.
d. not consistent with meningitis.

**33. The most common reason for failed Seldinger cricothyrotomy is:**
a. inability to locate the cricothyroid membrane.
b. placement of the cannula in the esophagus.
c. kinking of the guide wire.
d. excessive bleeding.

**34. In the recent Cleveland-area study examining the use of IV tPA for acute ischemic stroke, all of the following were reported *except*:**
a. the intracranial hemorrhage rate for the IV TPA group was 15.7%.
b. 50% of the patients receiving IV TPA were in violation of TPA administration guidelines.
c. the in-hospital mortality rate was higher for IV TPA patients than for those who did not receive IV TPA.
d. the rate of acute myocardial infarction was higher for the IV TPA group.

**35. Possible adverse effects of atropine adminstration for bradyarrhythmia include:**
a. potentiation of ischemia.
b. worsening of theophylline toxicity.
c. blockade of the binding site for isoproterenol.
d. worsening of digoxin toxicity.

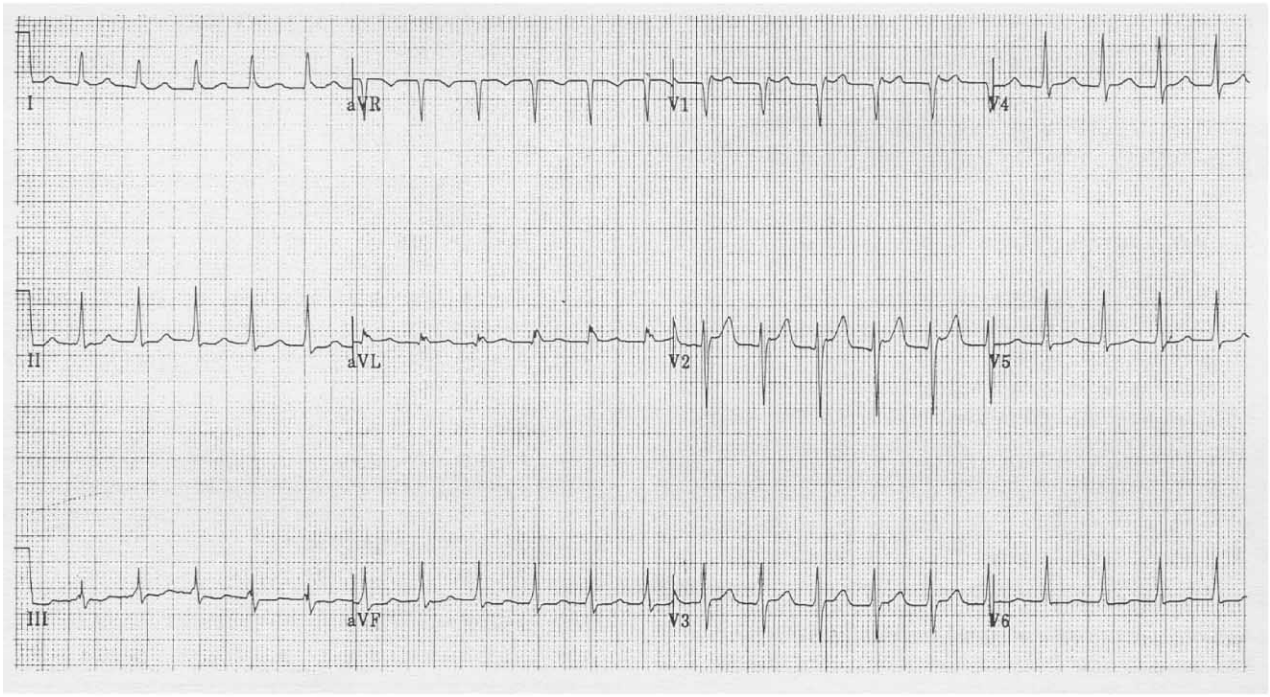
**36. Paradoxical bradycardia after atropine, although unusual, is more likely to occur in which of the following arrhythmic scenarios?**
a. Bradycardia with first degree AV block
b. Multifocal atrial tachycardia
c. Bradycardia with third-degree AV block with a wide QRS complex
d. Ventricular parasystole

**37. In the study by Eisenburger, et al on simulated cricothyrotomy, which of the following conclusions was reached?**
a. Seldinger technique was clearly superior to surgical.
b. Surgical technique was clearly superior to Seldinger.
c. Surgical technique was superior to Seldinger only if performed by a surgeon.
d. Success rates for either technique were similarly unsatisfactory in inexperienced hands.

**38. Atropine is:**
a. a sympatholytic drug.
b. frequently proarrhythmic in the prehospital setting.
c. more effective in ischemic bradyarrhythmias than in those of nonischemic origin.
d. the drug of choice in heart transplant patients with symptomatic bradycardia.

# Regular Tachycardia in a 42-Year-Old Man

*By Ken Grauer, MD*




12-lead ECG obtained from a 42-year-old man complaining of chest discomfort.

**Clinical Scenario:** The ECG shown in the Figure was obtained from a 42-year-old man complaining of atypical chest discomfort intermittently over the past few weeks. The patient was previously healthy. He was symptomatic at the time this tracing was recorded. What entities should be considered in your differential diagnosis? Is there evidence of atrial activity in the Figure?

**Interpretation:** There is a regular supraventricular tachycardia (SVT) at a rate of just under 150 beats/minute. Practically speaking, the differential diagnosis of a regular SVT at this rate consists of three entities: sinus tachycardia, atrial flutter, and paroxysmal supraventricular tachycardia (PSVT). Definitive diagnosis is unfortunately not possible from this single tracing. The rhythm could be sinus tachycardia, with an upright P wave concealed within the T wave seen in lead II. Atrial flutter always should be considered in the differential diagnosis of a regular SVT at a ventricular rate that is close to 150/minute, but the absence of any semblance of flutter activity in all 12 leads on this tracing makes this possibility less likely. Consequently, the most probable diagnosis is PSVT, which we strongly suspect because of the suggestion of subtle retrograde (negative) atrial activity that appears to be notching the terminal portion of the QRS complex in each of the inferior leads and which produces a terminal positive deflection (simulating an r') in lead $V_1$. ❖

## In Future Issues:

New treatment duration for pyelonephritis?