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Prophylactic Doxycycline for Lyme Disease

ABSTRACT & COMMENTARY

Source: Nadelman RB, et al. Prophylaxis with single-dose doxycycline for the prevention of Lyme disease after an *Ixodes scapularis* tick bite. *N Engl J Med* 2001:345:79-84.

This study was conducted in westchester, ny, an area hyperendemic for Lyme disease, and spanned a decade. The authors recruited adult patients who had removed attached *Ixodes scapularis*, or deer ticks, within the previous 72 hours. The subjects were randomized in a double-blind fashion to receive either a single dose of doxycycline 200 mg or placebo. The authors followed the subjects for six weeks to detect erythema migrans (the rash pathognomonic of Lyme disease) and seroconversion.

The authors randomized 482 subjects with confirmed *I. scapularis* bites, some of whom had multiple tick bites. Erythema migrans was noted in one of 235 doxycyline-treated patients (0.4%) vs. eight of 247 controls (3.2%), a statistically significant difference ($P < 0.04$). The authors estimate the efficacy of treatment to be 87% (95% confidence interval; 25%—98%). Side effects, primarily nausea and vomiting, were noted in one-third of treated subjects. No patient went on to develop later manifestations of Lyme disease. The authors conclude that a single 200 mg dose of doxycycline, given within 72 hours of an *I. scapularis* tick bite, is effective for prevention of Lyme disease.

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

Because this article received considerable attention even prior to its publication in one of the country's most prominent medical journals, emergency physicians likely are to be confronted by patients with recent tick bites who request prophylactic antibiotic therapy. Indeed, the authors provide compelling evidence for complying with such requests, given certain caveats. First, not all tick bites are from *I. scapularis*, and physicians should make reasonable attempts to have the patient describe the offending tick. A large, easily grasped tick is very unlikely a deer tick, which is, at most, a few millimeters across. Second, while Lyme disease is endemic in most mid-Atlantic states, New England, and Wis-

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consin, it is rare in the Rocky Mountain states and many other areas. Therapy should be tailored to the risk of infection from exposure based on local epidemiologic data.[1] Treatment with this dose of doxycycline was not entirely benign, and gastrointestinal side effects frequently were reported. Finally, bear in mind that this was a small study, with a correspondingly wide efficacy estimate (as low as 25%). Although the Infectious Disease Society of America recently has recommended that patients bitten by deer ticks should not receive prophylactic antibiotics, this landmark study changes everything.[2] ❖

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Rate Control of Rapid Atrial Fibrillation

ABSTRACT & COMMENTARY

Source: Wattanasuwan N, et al. Acute ventricular rate control in atrial fibrillation: IV combination of diltiazem and digoxin vs. IV diltiazem alone. *Chest* 2001;119:502-506.

The mainstays of treating atrial fibrillation (AF) include ventricular rate control, conversion to and maintenance of sinus rhythm, and prevention of embolic events. The primary goal of emergency department (ED) management is rate control, as it is the main determinant of the patient's symptoms. Digoxin, which produces a rapid ventricular response, was the first-line treatment for AF for many years until the early 1990s, when it was supplanted by calcium-channel blockers and beta-adrenergic blockers. The calcium-channel blocker diltiazem currently is among the most popular choices for acute rate control.

This prospective, randomized, open-label study from Long Island College Hospital examined the efficacy of combination diltiazem and digoxin vs. diltiazem alone (all given intravenously) for acute ventricular rate control in patients presenting with AF and rapid ventricular rates. Fifty-two patients met the inclusion criteria, defined as a ventricular rate of greater than 100 bpm. Patients with systolic blood pressure less than 90 mmHg, acute congestive heart failure (CHF), acute coronary syndromes, ventricular rate greater than 200 bpm, coexisting unstable medical conditions, preexcitation syndrome, history of allergy to the study drugs, and those who had taken any antiarrhythmic medications within one week prior to presentation were excluded.

Both groups received diltiazem 0.25 mg/kg intravenously (IV) over two minutes followed by a maintenance continuous infusion of 10 mg/hr. At 15 minutes, a second bolus of 0.35 mg/kg of IV diltiazem was given if the ventricular rate remained greater than 100 bpm. Patients in the combination group also received 1 mg of intravenous digoxin, given as 0.5 mg initially, followed by two doses of 0.25 mg at two and four hours. Doses of digoxin were withheld if the ventricular rate was less than 55 bpm at the scheduled dose time. Patients achieving successful rate control were switched from intravenous diltiazem to 60 mg given orally every six hours, with the first dose given 30 minutes prior to stopping the infusion. All patients had heart rate and cardiac rhythm monitored continuously in the cardiac care unit

(CCU) for 12 hours, and had an echocardiogram within 24 hours of ventricular rate control.

Successful rate control was defined as a rate of less than 100 bpm persisting for one hour or conversion to sinus rhythm. Loss of rate control in patients who had achieved a successful rate control was defined as increased rate to greater than 100 bpm persisting for longer than than 30 minutes or conversion from sinus rhythm back to AF. Study end points included the number of patients with successful rate control, time to successful rate control, and number of episodes of loss of rate control.

The study groups were not significantly different in age, gender, the presence of certain co-morbidities, chronicity of AF, initial heart rate, initial blood pressure, left atrial size, ejection fraction, or the presence of left ventricular (LV) hypertrophy. All patients achieved successful ventricular rate control at 12 hours. The time to ventricular rate control was shorter in the combination group (15 ± 16 min vs 22 ± 22 min), but this was not statistically significant. The number of episodes of loss of rate control was significantly fewer in the combination group, with six patients experiencing 14 episodes of loss of rate control vs. 11 patients with 39 episodes in the diltiazem-alone group. Twelve patients in the combination group and 14 in the diltiazem-alone group converted to sinus rhythm, most of whom remained in sinus rhythm throughout the study period. Seven patients in the combination group and 11 in the diltiazem-alone group required the second bolus of diltiazem. Blood pressures remained comparable between groups throughout the study.

COMMENT BY JACOB W. UFBERG, MD

Previous studies have shown that digoxin used alone for acute ventricular rate control in AF results in a delayed rate control effect and probably lower success rates. Diltiazem results in rapid and effective rate control, but is associated with frequent loss of rate control that necessitates repeated dosages. These additional dosages of a negative inotrope may be undesirable in patients with poor left ventricular systolic function. Previous studies have shown that oral diltiazem in combination with oral digoxin controls rate more effectively than oral diltiazem alone, both at rest and during exertion.

This study demonstrates that diltiazem and digoxin used in combination intravenously result in similar times to rate control, but significantly fewer episodes of loss of rate control when compared with diltiazem alone. The patients in the combination group required fewer repeat boluses of diltiazem, an effect that is desirable for patients with poor LV function. It has been reported previously that episodes of loss of rate control may be associated with a longer median length of hospitalization. Although the authors did not study length of stay, it is conceivable that combination therapy may lead to a decrease in length of stay (CCU and/or hospital) when compared to diltiazem alone. In summary, intravenous combination therapy with diltiazem and digoxin for rate control of rapid AF appears to offer an advantage over diltiazem alone in patients meeting the criteria set forth by the authors. ❖

Imaging of Suspected Mandibular Fracture: What is the Best Modality?

ABSTRACT & COMMENTARY

Source: Nair MK, et al. Imaging of mandibular trauma: ROC analysis. *Acad Emerg Med* 2001;8:689-695.

This paper sought to address the continuing debate as to which approach is superior when imaging the mandible for suspected fracture. In a novel approach, cadaveric specimens were used after fracture had been induced by blunt trauma with a steel pipe in various regions in approximately 50% of mandibles. Fracture existence was verified by examination of defleshed mandibles by an oral and maxillofacial (OMF) radiologist. After being informed of the approximate fracture rate, six trained observers (3 general surgeons and 3 OMF surgeons) interpreted four imaging modalities of the specimens: 1) panoramic view; 2) mandibular series comprised of right and left lateral oblique views, reverse Towne's views, and an anteroposterior (AP) view; 3) digitized images of the mandibular series; and 4) panoramic view together with an AP view. Plain film images were interpreted with 2X magnification and hot light made available; brightness and contrast manipulation was permitted for the digitized images. Sensitivity, specificity, and receiver operating characteristic (ROC) analysis were calculated for the various imaging modalities.

ROC curve analysis demonstrated the panoramic plus AP view combination to be superior to other modalities, with panoramic view alone being next best, and digitized mandibular series performing worst *(see Table)*. Further statistical analysis revealed that there was no significant difference between panoramic and panoramic plus AP view. Not surprisingly, condylar and coronoid fractures were the most difficult to detect ($P < 0.03$). Inter- and intra-observer agreement was high (κ_w 0.76 and 0.81, respectively). The authors conclude that panoramic imaging is adequate for the detection of uncomplicated mandibular fractures, and that this modality affords greater diagnostic accuracy than does the mandibular series.

Table

Sensitivity and Specificity of Various Modalities

View	Sensitivity (%)	Specificity (%)
Panoramic	88	94
Panoramic + AP view	92	97
Mandibular series	74	79
Digitized mandibular series	73	82

COMMENT BY RICHARD A. HARRIGAN, MD, FAAEM

Score one for panoramic imaging. Most recently, an emergency department (ED)-based clinical study by Guss and colleagues found panoramic imaging to be statistically equivalent to the mandibular series,[1] although the number of fractures detected by the latter was higher. The study by Guss et al was superior to those done previously[2-4] in that the methodology was greatly improved, employing prospective analysis for the first time, among other things. Yet it still suffered from a seemingly unavoidable design flaw to which each of these clinical studies fell victim: the lack of a truly satisfactory criterion gold standard. Guss et al used the reading by a neuroradiologist (provided with clinical history) as their gold standard; to be sure, this was an improvement on prior work. Operative evidence of the presence or absence of fracture might be the ideal clinical gold standard, but that benchmark either has been employed inconsistently or not used at all.[1-4] Thus, a study, in this case cadaveric, that utilizes verifiable fractures is highly desirous and an important addition to the literature.

The enhancement of radiographic diagnostic accuracy by the addition of an AP view to the panoramic image, although not demonstrating a statistically significant advantage, highlights a clinical take-home point. When using the panoramic view to screen for fracture, if your suspicion is high and yet the panoramic image does not demonstrate a fracture, perhaps an additional AP view will help rule in or rule out a fracture. ❖

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Fibrinolytic Therapy after Unsuccessful CPR

ABSTRACT & COMMENTARY

Source: Bottiger BW, et al. Efficacy and safety of thrombolytic therapy after initially unsuccessful cardiopulmonary resuscitation: A prospective clinical trial. *Lancet* 2001;357: 1583-1585.

Despite recent advances in emergency cardiac care, survival from out-of-hospital cardiac arrest remains dismally low. Studies suggest many sudden arrests in the field are attributable to associated vascular thrombosis, primarily acute myocardial infarction (AMI), or massive pulmonary embolism (PE).[1] While cardiopulmonary resuscitation (CPR) efforts once were a contraindication to fibrinolytic (i.e., thrombolytic) therapy, there has been renewed interest in such treatment for cardiac arrest.

This European study evaluated the safety and utility of fibrinolytic therapy for out-of-hospital cardiac arrest. The authors postulate that fibrinolytics may be beneficial in this setting by treating the vascular thrombosis (AMI or PE) precipitating cardiac arrest, improving microcirculatory cardiac and cerebral blood flow, and addressing the coagulation abnormalities (such as disseminated intravascular coagulation) associated with cardiac arrest and its aftermath.

The investigators performed a prospective interventional trial on 40 cardiac arrest patients with no return of spontaneous circulation (ROSC) after 15 minutes of resuscitation. Patients received both heparin (5000 U) and recombinant tissue-plasminogen activator (rt-PA, 50 mg) intravenously (with a repeat dose if there was no ROSC within the following 30 minutes). Investigators studied resuscitation-related bleeding complications, ROSC, admission to a cardiac care unit, survival at 24 hours, and hospital discharge. Subjects were compared to 50 historical controls: patients who had suffered field cardiac arrest during the prior year and received only standard resuscitation measures.

Arrest victims who received fibrinolytics had a higher rate of ROSC (68% vs 44%, P = 0.0026) and admission to a cardiac care unit (58% vs 30%, P = 0.0009) compared to historical controls. Compared with standard resuscitation, the odds ratio for ROSC was 2.65 (95% CI; 1.11-6.25) and for admission 3.15 (1.32-7.69). There was a trend toward improved survival at 24 hours (35% vs 22%) and hospital discharge (15% vs 8%), but these findings were not statistically significant. There were no resuscitation-related bleeding complications in either group. The authors did report upper GI bleeding that required transfusion in two patients with fibrinolysis.

Based on these findings, the authors conclude that fibrinolytic therapy for out-of-hospital cardiac arrest is both safe and feasible. The authors believe a larger, randomized study is now warranted on the utility of fibrinolytics for cardiac arrest.

COMMENT BY THEODORE C. CHAN, MD, FACEP

This investigation adds to other small studies, primarily case series, suggesting that fibrinolytic therapy is both safe and efficacious in the setting of cardiac arrest.[2,3] This particular study was restricted to those victims likely to have a poor outcome; that is, those in whom 15 minutes of resuscitation was unsuccessful. In addition, patients in whom asystole was the initial cardiac rhythm (i.e., those with the lowest chance of survival) were included and comprised more than one-half the study population. Accordingly, the improvements in ROSC and admission are notable. Moreover, there was a trend toward improved survival (at 24 hours and discharge), though this finding was not statistically significant, possibly due to the small number of patients.

It is important to note that this investigation was conducted in Europe, where physicians often staff ambulances. In this study, the authors note that the "emergency doctor was aware of inclusion and exclusion criteria and enrolled appropriate patients" in the field. Thus, this study may not be directly applicable to areas in which physicians do not serve as field personnel. The investigators used one-half the standard AMI rt-PA dose with heparin and repeated the dose if there was no ROSC after 30 minutes. As work on fibrinolytics in this setting has just begun, the safest and most efficacious regimen has yet to be determined. In addition, the role of other agents, such as glycoprotein IIbIIIa inhibitors, has yet to be explored.

The investigators acknowledge that their non-randomized study is too small to make definitive conclusions and suggest their study lays the groundwork for a larger, randomized study. Despite the difficulty these days with performing randomized, clinical studies in emergency care and resuscitation (particularly with respect to informed consent), the need for such a study investigating the role of fibrinolytics in cardiac arrest is clear. ❖

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Special Feature

Digoxin Effect on the Electrocardiogram

By Richard A. Harrigan, MD, FAAEM

Much has been written about the therapeutic and toxicologic effects of digoxin (and other digitalis preparations) regarding both the clinical and electrocardiographic manifestations. The electrocardiographic effects associated with digoxin toxicity recently were reviewed in the emergency medicine literature.[1] Less attention has been given to the effect of digoxin—whether in therapeutic or non-toxic situations—on the electrocardiogram (ECG), even though these manifestations are likely to be seen more often by the practicing emergency physician. After a brief review of the pharmacologic effects of digoxin on the heart, the ECG manifestations known as "digoxin effect" or "digitalis effect" will be described. Differential diagnostic possibilities will be presented for these varied electrocardiographic effects.

Pharmacology

Digitalis preparations act by inhibiting the sodium-potassium ATPase pump, thereby increasing intracellular calcium levels.[1] The effect of the drug is largely mediated by the autonomic nervous system, enhancing vagal tone both centrally and peripherally. This leads to a diminished sinus node discharge rate, shortened atrial refractoriness, and prolonged refractoriness of the atrioventricular (AV) node.[2] Parasympathetic mediation of effect is evidenced by the fact that digoxin effects on rate control in atrial fibrillation (a common usage) are not uniform, even in the same person. For instance, when at rest, in a state of vagal tone predominance, the ventricular rate usually is maintained in the desired range (60-100 bpm). However, when the patient with atrial fibrillation exercises—thereby decreasing vagal tone and increasing adrenergic tone—digoxin is less suc-

Table 1

Summary of Principal Digoxin Effects on the ECG

- **ST segment depression**
  - "Coving" or "scooped" morphology
  - Concave upward
- **T wave changes**
  - Decreased amplitude; flattening
  - Inversion
  - Biphasic
- **QT interval shortening**
- **Prominent U waves**
- **PR segment lengthening (may be minor)**

Table 2

Differential Diagnosis of ST Segment Depression on the ECG[3]

- Subendocardial ischemia
- Ventricular hypertrophy
- Ventricular paced rhythm
- Hyperventilation
- Digoxin effect
- Bundle branch block
- Hypokalemia

cessful for maintaining rate control due to a diminished effect on the AV node. Thus, digoxin rarely is used as a single agent to control the ventricular rate in atrial fibrillation.[2]

Digoxin Effect on the ECG

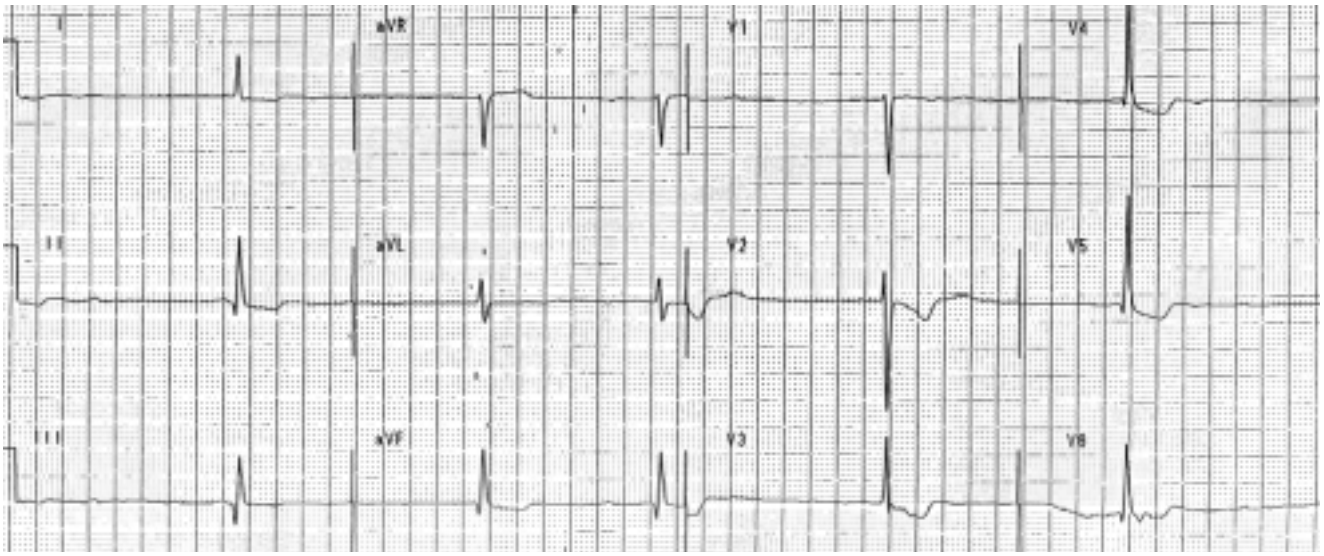
"Drug effects" on the ECG are distinguished from "drug toxicity" in that the former term encompasses all of the effects seen during therapeutic use of the agent. Of course, this is not to say that these drug effects disappear in poisoning situations; rather, they may be joined by the various manifestations of the toxic effect of the drug. *(See Figure.)* Clinical digoxin toxicity, manifested by gastrointestinal, central nervous system, and electrophysiologic changes, may occur in the absence of the drug effect ("digoxin effect") on the ECG. Conversely, prominent changes consistent with digoxin effect may occur with therapeutic or subtherapeutic levels of the drug, in the absence of clinical toxicity.

Morphologic effects. The principal effect classically associated with therapeutic digoxin use is a "coving" or "scooped" depression of the ST segment; this "sagging" typically demonstrates an upward concavity. *(See Table 1.)* This finding is more prominently seen in leads featuring a tall R wave. *(See Figure.)* At times, J-point depression is seen, and it may be difficult to distinguish this from the ST segment depression associated with myocardial ischemia.[3-5] The depressed ST segment may seem to pull the T wave downward, resulting in a biphasic T wave, with the first portion being negative. *(See Figure, especially leads V_3-V_5.)* In some instances, coving ST segment depression is not seen, and ST segment changes may mimic those seen with the strain pattern of ventricular hypertrophy, with a slight upward convexity of the ST segment.[5] Normal subjects orally loaded with digoxin over six weeks were found to have a dose-dependent depression of the ST segment on ECG; this finding was most accentuated at mildly tachycardic rates during exercise (110-130 bpm); higher rates did not increase the amount of ST depression. This finding is in contradistinction to the ST segment depression typically seen in exercise-induced ischemia, wherein higher rates correspond to further depression of the ST segment.[6] Nonetheless, digoxin effect may result in false-positive stress test results.[3]

Perhaps the earliest, but most variable, features of digoxin effect on the ECG are the changes in T wave morphology. The T waves may be flattened, inverted, biphasic, or of course, may remain normal; biphasic T waves may demonstrate peaking of the terminal portion.[7] T wave inversion associated with digoxin effect is characteristically asym-

Figure

Digoxin Effect . . . and More



On this ECG, the characteristic "coving" ST-segment depression of digoxin effect is seen, especially in leads V_3-V_5. The QT interval does appear short, especially for the relatively slow ventricular rate. Coincidentally, this patient also suffered from digoxin toxicity, as evidenced by third-degree AV block (complete heart block). The third QRS complex is a junctional extrasystole, and accounts for the seeming irregularity of the rhythm (the RR intervals between beats 1 and 2, and between beats 4 and 5, are actually the same). Once again, digoxin effect is a drug effect seen at therapeutic levels, and is independent of digoxin toxicity.

metric, with symmetric inversions of the T wave being more typical of such entities as myocardial ischemia or central nervous system disorder.[8] Prominence of the U wave also may be seen with therapeutic levels of digoxin; this is usually best seen in the mid-precordial leads, and is not as profound as that typically associated with hypokalemia.[5]

ECG interval effects. The QT interval is generally known to vary with rate (inversely) and age (directly), and is usually slightly longer in women than in men.[4,5] Whereas there are many diseases and medications that cause prolongation of the QT interval, digoxin is on the short list of things that cause a shortened QT interval. (Hypercalcemia is another recognized cause, as well as early phase hyperkalemia—when T waves are narrowing and peaking.)[3-5,8] Two major ECG texts[3,4] do not describe a lower limit of normal for the QT interval, whereas another[5] states that the lower limit of normal (for rates of 45-115 bpm) is 0.30 seconds. Vagally-mediated slowing of AV nodal conduction manifests as a slight increase of the PR interval for those individuals in sinus rhythm.[4,5]

Summary

Digoxin effect on the ECG is independent of the ECG changes seen in toxicity with this agent. With digoxin effect, changes in wave morphology as well as interval duration may be seen. Effects of the drug on repolarization (an enhanced recovery phase) are responsible for the ST segment, QT interval, and T wave changes associated with digoxin in therapeutic situations.[5] Vagal effects of the drug primarily are responsible for the slight slowing of the sinus rate and increase in AV conduction time (i.e., prolongation of the PR interval) seen in therapeutic dosing of digoxin, but direct effects of the drug also seem to play a role.[5] ❖

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CME Questions

16. Successful rate control of rapid atrial fibrillation was found to:
- a. occur significantly faster when diltiazem and digoxin were given simultaneously.
- b. occur significantly faster with diltiazem alone.
- c. be lost significantly less often when both diltiazem and digoxin were given.
- d. be lost significantly less often when diltiazem was given alone.

17. Postulated benefits of fibrinolytics in the setting of cardiac arrest include all of the following *except:*
- a. reducing the risk of intracerebral hemorrhage.
- b. addressing the vascular thrombosis associated with AMI or PE.
- c. improving microcirculatory flow.
- d. treating disseminated intravascular coagulation and other coagulopathies associated with cardiac arrest.

18. Which of the following is correct regarding prophylactic therapy for Lyme disease?
- a. A single, double-strength dose of doxycycline appears to be effective.
- b. Patients with bites from any type of tick should be treated.
- c. Untreated individuals frequently develop secondary manifestations of Lyme disease.
- d. Gastrointestinal side effects are uncommon.

19. In cadaveric specimens with induced fracture of the mandible, what imaging modality appears to be superior for fracture detection?
- a. Computed tomography
- b. Mandibular series
- c. Digitized mandibular series
- d. Panoramic imaging

20. In cadaveric specimens with induced fracture of the mandible, which fractures were the most difficult to detect radiographically?
- a. Body and angle
- b. Coronoid and condylar
- c. Mental symphysis and ramus
- d. Mental symphysis and body

21. All of the following are ECG manifestations associated with the effect of digoxin at therapeutic levels *except*:
- a. concave upward ST segment depression.
- b. coving or sagging of the depressed ST segment.
- c. QT interval prolongation.
- d. T wave flattening.

22. Patients with clinical evidence of digoxin toxicity such as nausea and arrhythmia will always manifest changes consistent with digoxin effect on their ECG.
- a. True
- b. False

An Overly "Busy" 12-Lead

By Ken Grauer, MD

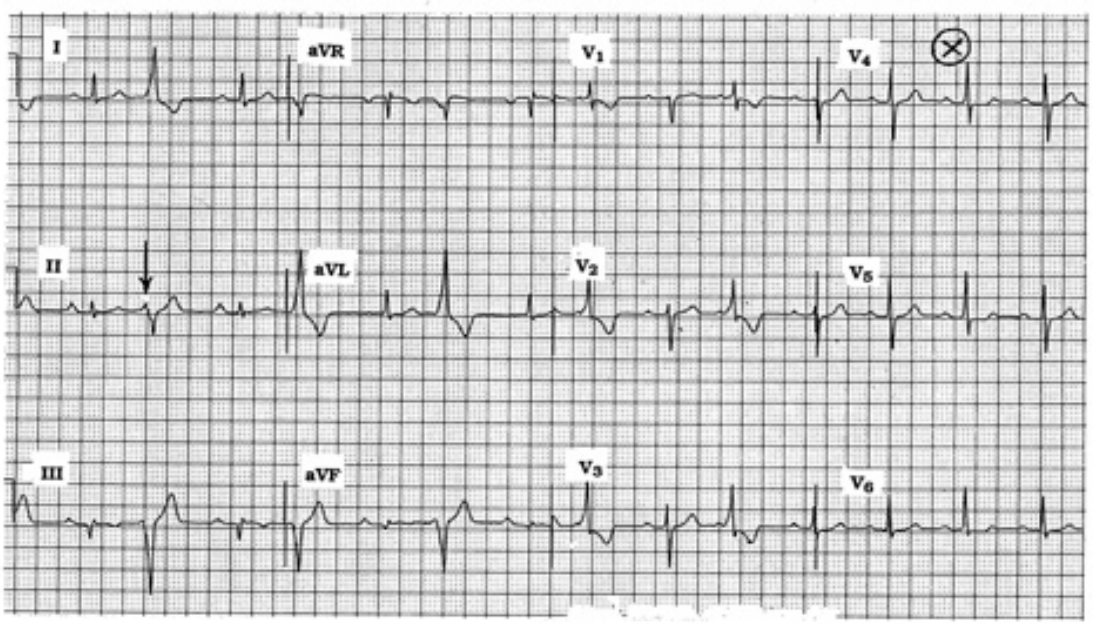

Figure. 12-Lead ECG obtained from a 74-year-old woman with chest pain.

Clinical Scenario: The ECG shown in the Figure was obtained from a 74-year-old woman with chest discomfort. Given this history, how would you interpret this tracing? What is the rhythm? Can you explain why QRS morphology changes slightly in simultaneously recorded leads V_4-V_6 (beat X)?

Interpretation: As suggested by the title of this ECG review—*a lot* is happening on this tracing. Appreciation of several key points may greatly facilitate interpretation. First, remember that despite the change in leads after every three or four beats—time is *continuously* recorded along each longitudinal channel (i.e., a total of 14 consecutive beats are displayed on the middle channel involving successively leads II, aVL, V_2, and V_5). Second, look to see if an underlying sinus rhythm is present. That this is the case in the Figure is confirmed by inspection of the 1st and 3rd beats in lead II, both of which show narrow, normal-appearing QRS complexes preceded by an upright P wave with a constant and normal PR interval. Third, identify the abnormal (non-sinus conducted) beats—and *ignore* these abnormal beats when interpreting the ECG for axis, hypertrophy, and changes of infarction.

Applying these suggestions we note (by assessing the middle recording channel as a multilead rhythm strip) that the rhythm is ventricular bigeminy (every other beat is a PVC). Use of calipers reveals that P waves continue at a regular rate of 85/minute throughout the tracing (the arrow in lead II points to the second P wave in this lead, which is partially hidden by the initial part of the PVC). We suspect the reason for the different but still narrow QRS complex near the end of the tracing (beat X in lead V_4) is that this is a fusion beat.

Interpretation of the rest of the 12-lead ECG is based on morphology of sinus-conducted beats in each lead. Q waves in leads III and aVF, in association with subtle ST elevation in these leads and T wave inversion in lead III, suggest inferior infarction that could be acute may account for this patient's chest pain and frequent ventricular ectopy. ❖

In Future Issues:

Comprehensive Review of Cervical Spine Imaging

Emergency Medicine Alert®

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