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INSIDE

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Rethinking Antibiotic Therapy for Mild-to-Moderate CAP

By Jacob Ufberg, MD

Assistant Professor of Emergency Medicine, Residency Program Director, Department of Emergency Medicine, Temple University School of Medicine, Philadelphia, PA

Dr. Ufberg discloses that he is a researcher for Pfizer Pharmaceuticals.

Source: Mills GD, et al. Effectiveness of beta-lactam antibiotics compared with antibiotics active against atypical pathogens in non-severe community acquired pneumonia: meta-analysis. *BMJ* 2005;330:456-460.

FOR MORE THAN 100 YEARS, *Streptococcus pneumoniae* HAS been considered the major causative organism in community-acquired pneumonia (CAP); however, more recent microbiologic advances led to the discovery of *Mycoplasma pneumoniae*, *Legionella pneumophilia*, and *Chlamydia pneumoniae*—the so-called atypical pathogens. The distinguishing feature of the atypical pathogens is the lack of in vitro response to beta-lactam and sulfonamide antibiotics, while clinical presentations are quite similar.

Evidence for the need to cover atypical pathogens in CAP is weak, and published guidelines on the initial treatment of CAP vary. While the American Thoracic Society (ATS) supports the treatment of all CAP using agents with activity against atypical pathogens, the British Thoracic Society considers *S pneumoniae* the most important target of initial therapy and does not suggest targeting atypical pathogens in all cases.

This study was a meta-analysis of double-blind, randomized, controlled monotherapy trials comparing beta-lactam antibiotics with antibiotics active against atypical pathogens in adults with CAP. The primary outcome was failure to achieve improvement or clinical cure at the time of outcome assessment in each trial. The 18 trials identified for inclusion were performed in more than 30 countries between 1980 and 2000 and included 6749 participants. The trials used nine different fluoroquinolones, two macrolides, and one ketolide; the beta-lactams used were predominantly amoxicillin, amoxicillin/clavulanic acid, or cefaclor. Common exclusion criteria (although different in each trial) included hospital-acquired

or aspiration pneumonia, immunocompromise, and major hepatic or renal dysfunction. The resulting patient mix in the trials was younger and had a better risk profile than observational pneumonia cohorts.

The overall rate of treatment failure for all trials was 18%. The authors found no significant heterogeneity between studies, and no significant difference between treatments in any study. In the combined analysis, the authors found no evidence that antibiotics active against atypical pathogens were superior to beta-lactam antibiotics (relative risk 0.97, 95% CI, 0.87 to 1.07). Separate analyses of studies using fluoroquinolones (RR 0.99, 95% CI, 0.88 to 1.11) and macrolides and ketolides (0.81, 95% CI, 0.58 to 1.14) similarly showed no superiority of antibiotics active against atypical pathogens.

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

Vice President and Group Publisher: Brenda Mooney.
Editorial Group Head: Glen Harris.
Managing Editor: Martha Jo Dendinger.
Marketing Manager: Nan Reeves.

GST Registration Number: R128870672.

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

Back issues: $48. One to nine additional copies, $234 each; 10 to 20 additional copies, $173 each.

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

Subscription Prices

United States: $299 per year (Resident rate: $144.50)
Canada: $329 per year plus GST (Resident rate: $159.50)
Elsewhere: $329 per year (Resident rate: $159.50)

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This publication does not receive commercial support.

The overall mortality rate was 1.9%; this number is in keeping with the 1.5% mortality rate reported previously for mild-to-moderate pneumonia (i.e., class 1 to 3 by the prognostic index scoring developed by Fine and colleagues).[1] The authors observed no difference in mortality between the study arms (RR 1.20, 95% CI, 0.84 to 1.71). In the 18 studies, 311 patients were diagnosed with *M pneumoniae*, 115 with *C pneumoniae*, and 75 with *Legionella* species. No treatment effect was found in patients with *M pneumoniae* (RR 0.60, 95% CI, 0.31 to 1.17) or *C pneumoniae* (RR 2.32, 95% CI, 0.67 to 8.03). The antibiotic failure rate for agents active against atypical pathogens was, however, statistically lower among patients diagnosed with CAP caused by *Legionella* species.

The authors concluded that their data did not support the need for antibiotics active against atypical pathogens in the initial management of adults with mild-to-moderate CAP. They stated that when antibiotics active against atypical pathogens were used, only Legionella pneumonia showed statistical improvement in outcome. They further stated that Legionella is sufficiently uncommon (<3%) in mild-to-moderate CAP, such that coverage for this possibility is not warranted in the initial management of non-severe CAP. The authors stated that this study provided the best evidence currently available regarding the need for antibiotics in non-severe CAP. ❖

COMMENTARY

This is a fascinating study that directly opposes the ATS guidelines for the outpatient treatment of mild-to-moderate CAP. While the ATS guidelines are based upon available evidence and expert opinion, this study provided contrary evidence. Physicians should feel comfortable in prescribing beta-lactam antibiotics in low-risk patients receiving outpatient treatment for CAP. Several issues will limit the extrapolation of these data to the community at large, however.

First, the patients in this study generally were younger and healthier (with less comorbidity) than the average cohort of CAP patients. It is important to note that these results should not be applied to patients with mild-to-moderate CAP who are being admitted for intravenous antibiotics, as almost all patients received oral therapy in this study. (Approximately half of pneumonia patients admitted to a hospital have mild-to-moderate pneumonia.) The exclusion criteria above also should be noted; these findings may not apply to the excluded patient groups.

Second, this study found a significant treatment difference among patients with Legionella pneumonia.

While this subgroup is small, physicians should be aware of local differences in prevalence of pathogens causing CAP; these differences may affect prescribing patterns. Also, physicians should be aware of some of the common clinical and laboratory findings associated with Legionella, and expand antibiotic coverage on a case-by-case basis if Legionella seems more likely.

Third, physicians must be aware of local antibiotic susceptibility patterns for all relevant organisms, including *S pneumoniae*.

Biphasic Anaphylaxis

By Richard Harrigan, MD
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Source: Smit de V, et al. Anaphylaxis presentations to an emergency department in Hong Kong: Incidence and predictors of biphasic reactions. *J Emerg Med* 2005;28:381-388.

ANAPHYLAXIS IS ONE DISEASE ENTITY THAT HAS THE respect of any emergency physician who has encountered it; multisystem effects and rapid progression make it a true emergency that requires quick diagnosis and treatment. Furthermore, the specter of recurrence after the initial anaphylactic reaction has been terminated successfully – termed a "biphasic reaction" – is well reported; yet, the expected duration of time between cessation and recurrence has been difficult to characterize. To study this entity is challenging; the nature of the disease does not lend itself to a randomized, placebo-controlled trial. Most of the literature on the subject is in the form of case reports and case series, as well as review papers. This retrospective chart review of a four-year experience in a busy Asian emergency department (ED) with an annual census of approximately 200,000 adds to the current understanding of the presentation and clinical course of anaphylaxis.

Inclusion criteria were based upon capturing patients who were triaged by nursing staff to the resuscitation room with suspected anaphylaxis; a variety of synonyms for the disease and it manifestations were used to select patients from resuscitation room log book records. Patients then were excluded if charts were not available or the physician's discharge diagnosis was not anaphylaxis. Biphasic reactions were defined as any reaction occurring after initial treatment and resolution of symptoms – either in the ED or after admission to the ED observation unit or to the inpatient setting. After only nine exclusions, 282 patients were found to meet study criteria. A variety of clinical and demographic data were then reported. The capricious nature of the syndrome was evidenced in that no clinical feature occurred in more than 80% of the patients—the top four were urticaria (79%), flushing/pruritis (74%), dyspnea (66%), and angioedema (61%). More than 90% were treated with steroids and antihistamines—predominantly H_1 blockers—only 4% received H_2 blockade. Apparently, this is a hospital-specific phenomenon. Only 67% received epinephrine. Ninety-six percent were admitted: more than half to the observation unit (planned stay 12-24 hours) and 8% to the intensive care unit. Astonishingly, the median ED length-of-stay was 42 minutes. (That is most assuredly not happening in my ED!)

Fifteen patients (5.3%) experienced a biphasic reaction. The mean time from ED presentation to onset of the biphasic reaction was slightly more than 8 hours (SD 5.46, range 1.4-23 hours), and the mean time from initiation of treatment to biphasic reaction was approximately 7½ hours (SD 5.46, range 1.2-22.5 hours). In those patients who developed a biphasic reaction, cutaneous manifestations were more common and respiratory features less common—but this finding may be only incidental. The majority of biphasic reactions were mild, and the symptoms of recurrence were similar to those on initial presentation. In the 15 patients who had a biphasic reaction, the onset was more than 8 hours after presentation in eight patients. Had all patients been observed for 24 hours after presentation, no one would have been discharged before developing his biphasic reaction. There were no fatalities in this study. ❖

COMMENTARY

This was the largest case series to date reporting on biphasic reactions in anaphylaxis. Previous reports (*See Table*) described similarly low rates of 3-6%—close to that seen in this study (5.3%).[1-3] Other studies have reported higher rates: 18% [4] and 20%.[5] These studies had lower enrollment numbers (34 and 25, respectively); thus, if one fewer case were included in each, the incidence would have dropped to about 15%. Therefore, what can we take away from this observational study about the nature of biphasic reactions? As in previous studies, they seem to be hard to predict, generally well tolerated, and seem to mimic the initial presentation in terms of clinical features. The time to onset of the second phase seen in this study and others includes some fairly late presentations. It appears that observing people for 24 hours is safest, but is not always feasible or necessary. Knowledge of the potential for biphasic reactions emphasizes the point that all patients discharged after

Table. Biphasic Reactions in Anaphylaxis

STUDY	PATIENTS	INCIDENCE OF BIPHASIC	TIME TO BIPHASIC
Douglas	103	6%	1-72 hr
Lee (pediatric)	105	6%	1-28 hr
Brady	67	3%	24-48 hr

treatment for anaphylaxis should be given a prescription for injectable epinephrine, along with instructions on how and when to use the drug.

This study suffered some of the usual limitations of most retrospective studies (e.g., the incidence of the various clinical features depends upon the clinicians' documentation of those features). Follow-up seemed fairly tight in this study. All Hong Kong residents have unique identification numbers that allowed the authors to search for their reappearance at the study hospital as well as other institutions; therefore, it is likely that the reported incidence of biphasic reactions is valid. Perhaps the term anaphylaxis was applied a little too broadly in patient recruitment—only 94% of patients had more than one organ system affected—yet, the definition of anaphylaxis includes the term "multisystem." Nonetheless, this study is a valuable addition to the anaphylaxis literature.

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CT vs Plain Film Radiography for Cervical Trauma

By Michael A. Gibbs, MD, FACEP,
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Source: Holmes AF, et al. Computed tomography versus plain radiography to screen for cervical spine injury: A meta-analysis. *J Trauma* 2005; 58:902-905.

THE AIM OF THIS STUDY WAS TO COMPARE THE ACCURACY of plain film radiography and computed tomography (CT) for the detection of cervical injuries in blunt trauma patients. The authors conducted a MEDLINE review of all relevant studies published between 1995 and 2004. Articles were excluded for any of the following reasons: 1) plain films series failed at a minimum to include three views (e.g., anterior/posterior, lateral, open-mouth/odontoid); 2) CT scan did not extend from the occiput to the superior aspect of the first thoracic vertebrae; and 3) distance between cuts on the CT scan was more than 5 mm. Of 712 articles, seven met inclusion criteria and provided data for both plain films and CT in all study patients. These seven studies provided the substrate for the meta-analysis.

Patient entry criteria were highly variable for each study, and there were no randomized controlled trials. For identifying patients with cervical spine injury, the pooled sensitivity for cervical plain film radiography was 52% [95% CI, 47% to 56%], and for CT imaging was 98% [95% CI, 96% to 99%]. ❖

COMMENTARY

So, is it time to jump on the *CT-everyone* bandwagon? Not so fast! At first glance, the results of this study are compelling. CT imaging provided outstanding anatomic definition and an overall diagnostic accuracy of nearly 100%—almost twice that seen with conventional plain film radiography. The question is: How do we apply this information to clinical practice?

The authors acknowledge that all seven studies in their meta-analysis were conducted at large trauma centers, with a high percentage of enrolled patients sustaining severe multisystem trauma. In this patient population, the incidence of cervical injury is expected to be high. Furthermore, these patients are likely to undergo coincident CT imaging of the head and torso. In this setting, it makes good clinical and logistical sense to forgo plain film imaging and to move directly to cervical tomography as part of a more comprehensive "trauma CT protocol."

However, it is not clear that the same logic can be applied to alert, stable, low-risk patients with a much lower pretest probability of cervical fracture. Mower and colleagues reviewed the NEXUS experience in a broader population of ED trauma patients (n=34,065; fractures in 818 [2.4%]).[1] While CT imaging was not done in all patients, the performance of plain film imaging was reviewed carefully. Based upon the NEXUS data, the following generalities can be applied as a rule of 3s:

1. Inadequate plain films will miss approximately **30%** of injuries. This is a sobering reminder that the emergency physician should never settle for inade-

quate films; if adequate films are unobtainable, cervical CT imaging is the mandatory next step.
2. Adequate plain films will miss roughly **3%** of *stable* cervical fractures
3. Adequate plain films will miss about **0.3%** of *unstable* cervical fractures.

The use of CT imaging as a primary screening test in low-risk patients has not been studied; based upon the NEXUS data, plain films likely will remain the appropriate first step in this population. CT imaging should be used to supplement inadequate or suspicious plain film findings. Like everything else in medicine: clinical judgment is essential.

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Special Feature

Injectable Therapy for Diabetes Mellitus: Insulin and Beyond

By Richard A. Harrigan, MD,
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Introduction

THE PHARMACOLOGIC ARMAMENTARIUM FOR THE treatment of diabetes mellitus (DM) is expanding rapidly. Oral antidiabetic agents, which include five classes of drugs, recently have been reviewed elsewhere.[1] Parenteral therapy for DM, which includes a variety of insulins and insulin analogues, as well as two new injectable non-insulin agents, also is growing. The emergency physician must be knowledgeable of the various drugs, their onsets and durations of action, as well as their adverse effect and toxicity profiles. The following is a pharmacology update on parenteral agents for the treatment of DM.

Insulins and Insulin Analogues

Insulin therapy can be characterized as prandial (bolus) insulin, basal insulin, or an insulin supplement to correct hyperglycemia.[2,3] The first group functions similarly to the physiologic release of insulin when food is ingested. Basal insulin agents mimic the relatively constant, but low-level release of endogenous insulin that affects lipolysis and glucose release from the liver. Supplemental insulin also can be given in response to blood sugar spikes that arise anywhere along the way in the eating cycle. Regular and NPH insulins target both prandial and basal insulin needs, whereas insulin analogues address these two components separately.[2,3]

An awareness of the varying onsets of action, peaks, and durations of these agents is vital when assessing hypoglycemic patients in the ED to not underestimate maximum effect and to anticipate what lies ahead after discharge. All these times are difficult to commit to memory, and they are subject to individual patient variation in absorption and distribution (*Table*). Two recent reviews on the topic—written by the same author—report different durations of action for some of these agents, providing evidence that pharmacokinetic variation exists.[2,3] Some basic rules can be deduced, however.

Rapid-acting agents. The newer rapid-acting insulin analogues (e.g., insulin lispro and insulin aspart) have an onset of action within a few minutes and peak about the same time the traditional short-acting agent (regular insulin) begins its onset of action—approximately 30-60 minutes. The duration of effect for these rapid-acting agents is shorter than that of regular insulin. Remember when contrasting the rapid-acting insulin analogues (lispro and aspart) with standard regular insulin that these rapid-acting analogues reach twice the maximal concentration in half the time of regular insulin. These differing onsets of action make the timing of insulin injection an issue; rapid-acting agents should be injected *at* mealtime, whereas regular insulin should be administered 20-30 minutes *before* the meal.[3]

Pre-mixed agents. The premixed insulins are no longer simply combinations of regular and NPH (*Table*). The familiar Humulin 70/30 is 70% NPH and 30% regular insulin; the less commonly used Humulin 50/50 is half NPH and half regular. They combine the onset times of regular insulin with the duration of the NPH. Note that the 75/25 premixed insulin is actually 75% NPL (functionally indistinguishable from NPH) and 25% insulin lispro. Thus, in parallel to traditional premixed regimens, it combines the onset time of the lispro (5-15 min) with the duration of the basal insulin (10-16 hr). To further complicate this situation, there is another analogue combination available that is also a 70/30 mix—70% NPL and 30% aspart. Its onset and duration are the same as that of the 75/25 agent discussed above. One way to remember which agent is which is to look at the trade names. Humulin 70/30 and Humulin 50/50 are the traditional mixes of NPH and regular. Huma*log* 75/25 and Novo*log* 70/30 contain insulin ana*log*ues; thus, they have a rapid onset of action (*Table*).

Table. Insulins and Insulin Analogues[2,3]

INSULIN	ALIAS	ONSET	PEAK	DURATION
Rapid-acting		5-15 min	30-90min	4-6 hr
Lispro	*Humalog*			
Aspart	*Novolog*			
Short-acting		30-60 min	2-3 hr	5-10 hr
Regular U-100				
Regular U-500				
Buffered reg	Velosulin			
Intermediate-acting				
Isophane	NPH	2-4 hr	4-10 hr	10-18 hr
	Humulin N			
	Novolin N			
Insulin zinc	Lente	2-4 hr	4-12 hr	12-20 hr
	Humulin L			
	Novolin L			
Long-acting				
Insulin zinc extended	Ultralente	6-10 hr	10-16 hr	18-24 hr
Glargine	*Lantus*	*2-4 hr*	*no peak*	*20-24 hr*
Pre-mixed				
70% NPH/30% Regular		30-60 min	dual	10-16 hr
50% NPH/50% Regular		30-60 min	dual	10-16 hr
75% NPL/25% Lispro		5-15 min	*dual*	*10-16 hr*
70% NPL/30% Aspart		5-15 min	*dual*	*10-16 hr*

NPH = neutral protamine Hagedorn

NPL = neutral protamine lispro — functionally idential to NPH.[3]

Insulin analogues are in *italics*.

Long-acting agents. Introduced in the United States in 2001, insulin glargine (Lantus) forms a precipitate at the injection site, enabling it to act as a depot preparation and thereby, releasing the drug slowly during the course of the day (and night) without having a peak (*Table*). Its strength seems to be a lower rate of hypoglycemia than is seen with NPH insulin.[2,3] Insulin glargine is given customarily at bedtime, although there are data that show it can be administered successfully at breakfast or dinner also.[3]

Adverse effects. The principal side effect of insulin and its analogues is hypoglycemia, the timing of which can be anticipated by knowledge of the onset of action, peak effect, and duration of each agent.

Beyond Insulin: Other Injectable Agents

Traditionally, if a diabetes patient says he takes shots for glucose control, this statement has meant that he was taking insulin. That is no longer true. Two agents were approved by the Federal Drug Administration (FDA) this past spring: one for treatment of type I and II DM and the other solely for use in type II DM.

Pramlintide (Symlin). This drug can be used in either type I or type II diabetes patients in whom adequate blood sugar control cannot be achieved with insulin alone. This is the first agent beyond insulin to be approved for type I DM. This drug is a synthetic version of amylin, a natural hormone secreted by the pancreatic beta cell in response to hyperglycemia in parallel to the release of insulin. Its principal mechanism of action is to inhibit gastric emptying and, to a lesser degree, suppress the secretion of glucagon; it also suppresses the appetite.[4] The half-life of pramlintide is about 50 minutes, and it is metabolized primarily by the kidneys. Hepatic dysfunction should not affect this agent, and there is no need for dose reduction in patients with a creatinine clearance of more than 20 mL/min; it has not been studied in dialysis patients.[5]

Pramlintide is administered immediately prior to major meals; type II diabetes patients begin with 60 µg subcutaneous (sc) and may be increased to 120 µg if no significant nausea has occurred in 3-7 days. The dose should be reduced to 60 µg if the higher dose is associated with significant nausea. Importantly, the pre-prandial rapid-acting or short-acting insulin dose should be reduced by 50% when pramlintide is initiated; this caution includes pre-mixed regimens (e.g.,70/30, 75/25 insulin). Once the optimal dose of pramlintide is reached, the insulin regimen should be titrated to fine-tune glucose control. In type I diabetes patients, the initial dose is 15 µg sc, with upward titration in 15 µg-increments until a maximum of 60 µg; similarly, pre-meal insulin should be halved, and dose titration is guided by nausea.[5]

Blood glucose levels should be monitored frequently during initiation of pramlintide. Injections should be into the abdomen or thigh (not the arm), and should be rotated. The agent must not be mixed with insulin preparations in the same syringe, and it should be injected at least two inches away from the insulin injection site. Missed doses should not be given later. Principal side effects include hypoglycemia and nausea. The drug is contraindicated in gastroparesis, and it should be noted that pramlintide administration will delay absorption of drugs taken concomitantly by mouth. It should not be utilized when prokinetic drugs (e.g., metoclopramide) are used also. The drug is not approved currently for pediatric patients and is FDA pregnancy category C. Overdose data are limited, but severe nausea, vomiting, and dizziness are expected.[5]

Exenatide (Byetta). Indicated for type II diabetes only, this drug is an incretin—a drug class (prototype: glucagon-like peptide-1) that enhances glucose-dependent secretion of insulin from the beta cell, impedes inappropriately high glucagon secretion, and also delays gas-

tric emptying. Exenatide augments insulin release only in the presence of elevated blood sugar; as the serum glucose level falls toward normal, insulin secretion ebbs. Although it modulates glucagon, it does not interfere with the appropriate glucagon response to hypoglycemia. Exenatide reaches peak concentrations approximately two hours after sc injection. The drug is eliminated principally by the kidney. Hepatic insufficiency is not expected to affect exenatide, however, the drug is not recommended in severe renal insufficiency (i.e., creatinine clearance < 30 mL/min).

Exenatide is indicated as a pharmacologic adjunct for type II diabetes patients not achieving adequate glycemic control with metformin, a sulfonylurea, or both; notably, it has not been studied in patients taking insulin, thiazolidinediones (e.g., pioglitazone), meglitinides (e.g., repaglinide), or alpha-glucosidase inhibitors (e.g., acarbose). Adding this agent to sulfonylurea therapy may require dose reduction of the latter. Initial treatment includes administration of a 5-µg dose sc in the proximal arm, thigh, or abdomen at any time within 60 minutes before the morning and evening meal; it should not be given after the meal, and missed doses should not be made up. The dose may be increased to 10 µg twice daily if clinically indicated. It can indirectly cause hypoglycemia if a sulfonylurea is being used also. As with pramlintide, exenatide's effect on gastric emptying may affect co-administration of other drugs; agents that depend upon achieving a certain threshold for efficacy (e.g., oral contraceptives, antibiotics) should be taken more than one hour prior to exenatide. Principal adverse effects are hypoglycemia (expected only with sulfonylurea therapy), nausea, and decreased appetite. Exenatide is not for use in children or patients with type I diabetes and is FDA pregnancy category C. Limited experimental overdose data demonstrated profound nausea and vomiting, as well as hypoglycemia. ❖

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Physician CME Questions

13. Beta-lactam antibiotics appear sufficiently efficacious in the treatment of patients with:

a. nosocomial pneumonia.
b. aspiration pneumonia.
c. severe community-acquired pneumonia.
d. mild-to-moderate community-acquired pneumonia.
e. *Pneumocystis carinii* pneumonia.

14. Adequate cervical spine plain films will miss approximately _____ % of unstable cervical spine fractures.

a. 30%
b. 3%
c. 0.3%
d. 0.03%

15. Biphasic anaphylactic reactions appear to be:

a. uniformly fatal.
b. similar in clinical features to the initial reaction.
c. seen only after treatment with epinephrine.
d. preventable only through the use of antihistamine patches.

16. Insulin glargine:

a. is known also as Lantus.
b. is a long-acting insulin analogue.
c. does not peak, but rather maintains a steady serum level over time.
d. all of the above

Answers: 13. d; 14. c; 15. b; 16. d.

CME Objectives

To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, *you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

ECG Review

Right Answer/Wrong Reason

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Dr. Grauer is the sole proprieter of KG/EKG Press.

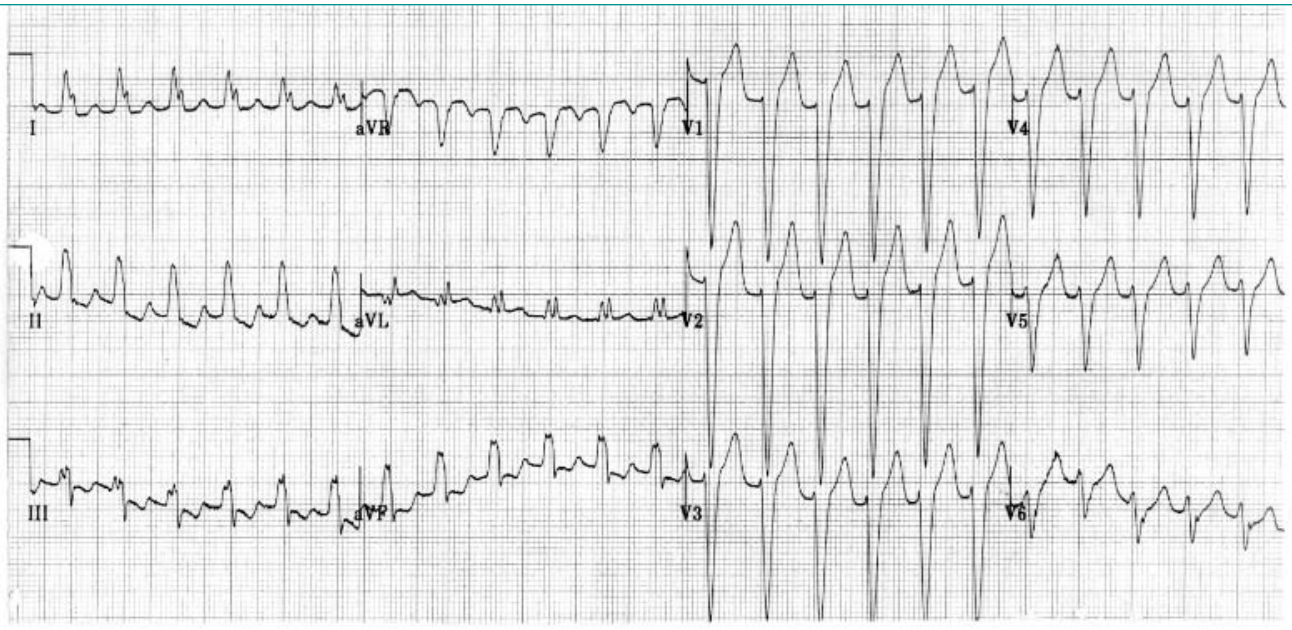

Figure. 12-lead ECG recorded from a 61-year-old woman with a history of hypertension and chest pain.

The 12-lead electrocardiogram (ECG) shown in the Figure was obtained in the emergency department (ED) from a 61-year-old woman with a history of significant hypertension. She was alert, oriented, and not in acute distress at the time this tracing was recorded, although she was markedly hypertensive and experiencing some chest pain. No prior ECG was available. The patient was treated in the ED with several doses of adenosine and eventually converted to sinus rhythm. Your thoughts on the rhythm and the management?

Interpretation: The fact that adenosine was selected and repeated as treatment suggests that the ED physician thought the rhythm was likely supraventricular. Because the patient converted to normal sinus rhythm following this treatment, it would be difficult to argue that with success. That said, assessment of this ECG and the clinical situation should strongly favor ventricular tachycardia (VT) as the diagnosis with a statistical likelihood of between 80-90%. Therefore, in similar situations, a different approach might be preferable.

The rhythm in the Figure is a regular wide complex tachycardia (WCT) at a rate of about 150 beats/minute. A small upright deflection is seen in lead II midway between QRS complexes, however there is no way to know if this upright deflection represents a P wave or a T wave (or fusion of the two). Although this WCT rhythm could represent a supraventricular tachycardia with either preexisting bundle-branch block or aberrant conduction, one should remember that the statistical likelihood that a regular WCT without definite atrial activity will be VT exceeds 80%. In view of this patient's age (older adult), history of severe hypertension (underlying heart disease likely), presenting complaint (i.e., chest pain), and unusual QRS morphology for left bundle-branch block (rS complexes across the precordium), the odds that this rhythm represents VT approach 90-95%. In such cases, specific antiarrhythmic therapy aimed at treating VT (e.g., IV amiodarone) or electrical cardioversion may be preferable to a multiple dose trial of adenosine. ❖

In Future Issues:

How late is too late for PCI in AMI?