EMERGENCY MEDICINE ALERT

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Balancing Capacity, Occupancy Key to Solving ED Overcrowding

ABSTRACT & COMMENTARY

Source: Forster AJ, et al. The effect of hospital occupancy on emergency department length of stay and patient disposition. *Acad Emerg Med* 2003; 10:127-133.

IF YOU ARE DEALING WITH A PACKED EMERGENCY DEPARTMENT (ED) and waiting room, and wondering why all the changes in the function of your department have not produced better throughput, this article is a must-read for you. This was an observational study, using administrative data at a 500-bed acute care teaching hospital. All patients presenting to the ED between April 1993 and June 1999 were included in the study. The predictor variable was daily hospital occupancy. Outcome measures included daily ED length of stay for admitted patients, daily request for admissions by the ED service (or "consultation rate" in this Canadian study), and daily admission rate. The authors employed a technique known as autoregressive, integrated, moving average (ARIMA) modeling to control for covariates. This model controlled for the average daily age of ED patients and the average daily "arrival density" index, which adjusts for patient volume and clustering of patient arrivals. In short, the authors tracked the ED length of stay (LOS) during the seven years that the number of hospital beds were purposely reduced from an average of 610 to 432. The reduction in these beds was as a result of cost-saving measures. During this overall trend, daily and seasonal variations in occupancy also were seen and factored into the study.

The results are fascinating and clearly show that as occupancy (as a percentage of available beds) increases, the LOS in the ED increased. Daily ED LOS for admitted patients increased 18 minutes when there was an absolute increase in occupancy of 10%. The ED LOS appeared to markedly increase when hospital occupancy exceeded a threshold of 90%. The number of requests for admissions and admission rates were not influenced by hospital occupancy. The authors conclude that efforts to increase hospital bed availability may be an important strategy to reducing ED overcrowding. Furthermore, they suggest that temporarily increasing the number of beds once occupancy reaches a threshold of 90% could result in shorter ED LOS.

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INSIDE

■ **COMMENTARY BY RICHARD J. HAMILTON, MD, ABMT, FAAEM**

I have a computer game called "Roller Coaster Tycoon" that allows you to set-up and manage an amusement park. When you play this game in earnest, you immediately acquire a practical understanding of queuing theory. You learn that when a ride has too few seats and the line gets too long, you had better increase capacity or people will walk out. If you build too much capacity into a ride, then it takes a long time to recoup your investment, although people are quite pleased with their experience. This study shows that overall occupancy affects ED LOS. However, according to queuing theory, waiting time may even increase when there is insufficient capacity in only some units — such as telemetry or intensive care. I told a hospital administrator that the ED LOS would improve only when we got more telemetry beds and he told me he wouldn't build those beds because I would "only fill them up with more patients." After I got up from the floor, I decided I had better understand how queuing theory was affecting my practice. My simple-minded analysis produced the following insights: 1) the cost of care largely is based on the number of nurses staffing beds (capacity); 2) the greatest profit is obtained when the maximum number of patients that those nurses can care for are in the hospital (highest occupancy); and 3) the greater the occupancy rate, the greater the LOS in the ED (longer queuing for care). In order for a hospital to become more profitable, it must either increase capacity (with the same occupancy rate) or increase occupancy (by decreasing capacity). It is infinitely easier, especially in the short term, to cut back on staffed beds than it is to attract new patients and build services. Thus, the net result is higher occupancy, greater profit margin, and prolonged ED LOS. No wonder we're in trouble!

Why is this important to study? I think that the future of emergency medicine will come into focus when we realize that the ED is overcrowded because the hospital has reached a state of dysfunction, not because we're not working hard enough or we don't have enough point-of-care tests to achieve rapid dispositions and turnaround times. Solutions only will come when we change the way we handle capacity and occupancy of beds in-house (e.g., example, by using a flexible unit for short stay admissions such as an observation unit, or by increasing the staffing when occupancy rises — as the authors suggest). ❖

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Assessing the Need for Pain Medication in the ED

Source: Blumstein HA, et al. Visual analogue pain scores do not define desire for analgesia in patients with acute pain. *Acad Emerg Med* 2003;10:211-214.

THE OBJECTIVE OF THIS STUDY WAS TO INVESTIGATE the ability of the visual analog scale (VAS) to differentiate between patients with acute painful conditions desiring pain medication and those not desiring medication. In this prospective, observational study of a convenience sample of adult patients who presented to an academic emergency department (ED) with acute pain, patients with acute exacerbations of chronic pain were excluded along with other reasonable exclusion criteria. Subjects were asked by research assistants to complete a VAS. "No pain at all," on the far left of the VAS, was separated by a 100 mm line (without markings) from "Severe, uncontrolled pain" on the far right. Patients then were asked to respond "yes" or "no" to the question: "Do you need pain medication?"

A total of 104 subjects participated in this study. Patients requesting pain medication (n = 64) had a mean VAS score of 66 (SD ± 23.1). The mean score for those not requesting medication (n = 40) was 45 (SD ± 29.4). The difference between the means was 21 (95% CI, 10.3-31.3). The area under the receiving operator characteristic curve for the VAS was 0.72 (95% CI, 0.61-0.82).

■ COMMENTARY BY STEPHANIE B. ABBUHL, MD FACEP

This deceptively simple study delivers an important concept to the clinical world of pain management. Despite some methodologic flaws, the results indicate that the VAS cannot adequately discriminate between patients who do and do not want analgesia for their pain. Although there is a statistical difference in the VAS scores of the two patient groups, there is considerable overlap of the scores and no single cutoff point can adequately predict a given patient's desire for medication. Therefore, the routine clinical role of pain measurement tools like the VAS is called into question.

This does not mean that the VAS is not a reliable and effective tool for measuring pain, especially in research settings where measuring a minimum clinically significant difference in pain, regardless of need for analgesia, is important. In the ED, however, the major goal of pain assessment is to determine the desire for pain medication, given that analgesia is our single key intervention. It is true that other interventions, such as reassurance, music, comfort measures, and acupressure, significantly may affect acute pain. However, until we have more data on how these kinds of interventions might be of practical use, we are left needing a tool that identifies pain and primarily measures the need for pain medication. Until further research clarifies the clinical role of the VAS, it may be that the most practical and efficient pain assessment tool in the ED is simply to ask patients initially if they want pain medication and, when reassessing the patients, inquire about the need for more pain medication. ❖

Bispectral Index Monitoring in the ED: Is there a Role?

Source: Gill M, et al. Can the bispectral index monitor quantify altered level of consciousness in emergency department patients? *Acad Emerg Med* 2003;10:175-179.

Bispectral index (BIS) monitoring makes use of a noninvasive device to measure electroencephalographic (EEG) data. Two sensor patches placed on the forehead transmit EEG data to a small computer. The data is converted to a BIS score, a dimensionless number ranging from zero (absence of brain activity) to 100 (wide awake). The data is updated continuously, and appears as a digital display and via a printout. Its use is well established in the anesthesia arena.

In this prospective, observational, ED study, a convenience sample of patients 8 years of age or older with an altered level of consciousness (ALOC) — defined as a Glasgow Coma Scale (GCS) score of 14 or lower — were included. Excluded were patients with known abnormal baseline mental status, deafness, inability to tolerated the BIS sensors, or those with neuromuscular blockade prior to calculation of the GCS. Patients receiving analgesics, sedative/hypnotics, or anticonvulsants were not excluded.

The GCS was calculated by an emergency physician at the earliest possible time, and was followed by application of the BIS sensors within five minutes. BIS scores were correlated with GCS scores. A target population of 100 patients was truncated to 38 subjects, due to an obvious high discordance in BIS/GCS values.

The correlation between BIS and GCS was "moderate" (Spearman's rho = 0.387), and displayed wide variability. For example, patients with a GCS of 3-5 had BIS scores ranging from 47 to 98. Similarly, those with relatively high-end GCS scores (12-14) featured BIS values between 56 and 98. The authors concluded that BIS does not reliably correlate with GCS in ED patients with ALOC.

■ COMMENTARY BY RICHARD A. HARRIGAN, MD

BIS monitoring has been through a development phase using healthy volunteers, and has been validated using other, well-described, objective measures of sedation. In general, a BIS of 83-89 is consistent with lack of recall, and a BIS of 64-72 is consistent with loss of consciousness.[1] Recent studies of the BIS in sedated intensive care patients have shown wide variability in sedation, with 54% of patients over-sedated and 15% under-sedated. Use of the BIS in this setting improved titration of sedation and decreased cost.[2]

Can BIS find a home in the ED? Probably, although it seems to me that the logical focus of study should be on monitoring level of sedation in the ED — mirroring applications that have been explored in the intensive care unit and the operating room. The authors have attempted to correlate BIS to an old saw in emergency medicine — the GCS score — and it did not work out. That is okay, because we don't necessarily need to have an apple when an orange works quite well. So many things impact BIS (including cerebral ischemia and motor activity, as well as medication effects — none of which were controlled for in this study) that it is not surprising that the varied population of all comers with ALOC had

varied BIS scores. It will be interesting to watch the literature as BIS searches for a niche in the ED. ❖

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Severe Malaria — Nail It Before It Nails You

Source: Bruneel F, et al. The clinical spectrum of severe imported falciparum malaria in the intensive care unit. *Am J Resp Crit Care Med* 2003;167:684-689.

Infection with *Plasmodium falciparum* is responsible for more than 5000 deaths every day, predominantly among African children. Yet with modern air transportation, severe malaria easily is transported worldwide during its two-week incubation period and can present in any American emergency department (ED). Up to 1300 cases of imported malaria are documented each year in the United States, an average of 25 per state. Physicians evaluating febrile, severely ill travelers must be vigilant for this potentially fatal infectious syndrome.

To better define the clinical spectrum of severe malaria, Bruneel and colleagues studied 188 adults admitted to a 1200-bed teaching hospital in Paris between 1988 and 1999. All had *P. falciparum* parasites in blood smears and were admitted to intensive care (ICU). Using World Health Organization criteria, 93 of the 188 patients were categorized as having "severe malaria" by the presence of one or more of the following features: 1) unarousable coma with Glasgow Coma Scale score less than 10; 2) hemoglobin less than 5 gm/dL; 3) acute renal failure; 4) pulmonary edema with ARDS; 5) hypoglycemia less than 40 mg/dL; 6) shock with systolic BP less than 80 mmHg; 7) disseminated intravascular coagulation; 8) seizures; 9) acidosis, with pH less than 7.25 or serum HCO_3 less than 15 meq/L.

"Less severe malaria" occurred in 95 of 188 cases and was defined as ICU patients lacking these nine major criteria but with minor criteria, including having impaired consciousness (but arousable), prostration, fever greater than 40°C, jaundice, greater than 5% parasitemia, or high risk conditions. All 188 cases were treated with intravenous quinine and intensive monitoring, with titration of pCO_2 to 35-40, elevated head of bed, normalization of glucose, sedation, and mannitol as appropriate.

Average age of cases was 38 years (14-74); 119 were male. Ninety-four percent of infections were acquired in sub-Saharan Africa (mainly Ivory Coast, Cameroon, Benin, Mali, Congo, Kenya, Zaire, and Gabon). Ninety-six percent took no or inappropriate malaria chemoprophylaxis. Of 93 severe cases, there were 10 fatalities (11%), all of whom took no malaria prophylaxis. Thirty required airlift to Paris for treatment. Average falciparum parasitemia was 4.1% overall vs. 18.2% among fatalities. Temperatures ranged from 39.5°C to 40.5°C. Mean time to malaria diagnosis was four days. The mean ICU stay was 7.5 days (4-13). Forty patients needed mechanical ventilation; 12/93 developed adult respiratory distress syndrome (ARDS). Shock occurred in 24. Fifty-four percent had acute renal failure, necessitating dialysis or continuous venovenous hemofiltration in 29. Fifty-one needed transfusion. Of 28 cases undergoing neuroimaging, three had deep white matter lesions, two had massive cerebral edema, two had ischemia, and one each had herniation, frontal hematoma, and meningeal enhancement. Pneumonia and bacteremia were documented in 14% of cases. Four criteria were associated with mortality: unarousable coma, ARDS, shock, and acidosis ($p < 0.001$ for all four). Among the less severe cases, 94% took inadequate chemoprophylaxis. The mean ICU stay was two days (1-4). There were no deaths among this group.

■ **COMMENTARY BY MICHAEL FELZ, MD**

The authors, and an accompanying editorial, state that this is the largest study ever published of severe imported malaria in a single center. We must, therefore, take heed of these findings. As a medical missionary to Papua New Guinea, I witnessed firsthand the ravages of severe malaria among villagers and expatriates. Bruneel and colleagues' description of the clinical spectrum of severe falciparum malaria is equally vivid to me and provides useful parameters for early diagnosis by ED physicians seeing ill travelers in the United States. The history of recent (< two weeks) sub-Saharan itineraries, so apparent in this study, serves as a persuasive clue for early detection. The four predictors of ICU mortality must be recognized and managed just as aggressively as the parasitemia itself. A 20% overall rate of concomitant bacterial infection warrants broad spectrum antibiotic coverage, as well.

Most glaring of all is the abject lack of adherence to strongly recommended, easily available chemoprophylaxis medications. Over 90% of these infected travelers took no, or inadequate, preventive regimens during

exposure in highly endemic African nations. Why so neglectful? What motivation is required? The factors are many, but the message is clear. *Falciparum* malaria is a killer. Any febrile traveler (to endemic regions) has malaria until proven otherwise. And an ounce of (chemo) prevention is worth pounds of (ICU) cure. ❖

Special Feature

Pharmacology Update: Atypical Antipsychotics

By Richard Harrigan, MD

ANTIPSYCHOTICS AGENTS ARE A DIVERSE CLASS OF drugs used to treat both psychiatric and nonpsychiatric conditions. The latter include control of nausea and emesis; pain and nausea reduction in various headache syndromes; chemical restraint of violent or agitated patients; hiccup suppression; and treatment of various movement disorders (e.g., Tourette's syndrome, Huntington's chorea). Once termed neuroleptics due to a propensity of the older agents to affect neurologic function, this term largely has been discarded. Antipsychotics are currently stratified into two categories: the older, typical agents, and the newer, atypical antipsychotics, which offer improved efficacy and a somewhat different side effect profile. Atypical antipsychotics were developed to address not only the positive symptoms of psychosis (e.g., hallucinations, delusions, and disordered thought) but also the negative symptomatology (e.g., withdrawal, flat affect, and loss of drive). This review will focus on the basic pharmacology, adverse effects, and toxicologic manifestations of the atypical antipsychotics. *(See Table 1.)*

Table 1. Atypical Antipsychotics Available in the United States

GENERIC	TRADE	ROUTE
Clozapine	Clozaril	Oral
Risperidone	Risperdal	Oral
Olanzapine	Zyprexa	Oral/parenteral
Quetiapine	Seroquel	Oral
Ziprasidone	Geodon	Oral/parenteral
Aripiprazole	Abilify	Oral

Pharmacology

Typical, or conventional, antipsychotics (which include a variety of drugs and drug classes, such as: phenothiazines [e.g., chlorpromazine]; butyrophenones [e.g., haloperidol]; and thioxanthenes [e.g., thiothixene]) are well documented as controlling the positive manifestations of psychosis as well as agitation due to other disorders (e.g., substance abuse, dementia). To differing degrees they block dopaminergic, alpha-adrenergic (principally α_1), muscarinic cholinergic, and histaminic receptors, yielding a varied side effect profile linked to their site of antagonism: extrapyramidal symptoms and tardive dyskinesia, orthostatic hypotension, anticholinergic effects, and sedation, respectively.[1]

The atypical antipsychotics are all from differing structural classes, and display different neurochemical profiles. Generally speaking, they preferentially antagonize dopamine-2 (D_2) receptors in the mesolimbic region of the brain over D_2 receptors in the mesocortical and nigrostriatal pathways; therefore, they are less likely to cause cognitive blunting and extrapyramidal symptoms, respectively, than the conventional antipsychotics. They also demonstrate minimal effects at tubuloinfundibular dopaminergic sites, lessening the effect on serum prolactin levels and avoiding the galactorrhea, amenorrhea, and gynecomastia associated with some typical agents.[2] In addition to mesolimbic D_2 selectivity, these atypical agents feature high binding affinity for certain serotonergic sites (e.g., 5-HT_{2A}); antagonism at these receptors in the nigrostriatal and mesocortical regions serves to disinhibit dopaminergic transmission at these sites, decreasing extrapyramidal manifestations and cognitive blunting effects, respectively. The antiserotonergic effects in the limbic neurons also seem to have a direct antipsychotic effect.[2] However, the atypical antipsychotics are similar to their typical predecessors in that they competitively block a number of other neurochemical sites; affinity for these other sites varies from drug-to-drug, and is dose-dependent. *(See "Adverse Effects," below.)*

All atypical antipsychotics are rapidly absorbed, with peak concentrations occurring in 1-6 hours — an important feature when confronted with the overdose patient. These drugs are highly protein bound, lipophilic, and feature a large volume of distribution, thus rendering dialysis an ineffective means of toxicity management. All undergo hepatic metabolism. Clinically important drug-drug interactions do exist. *(See Table 2 for several key interactions.)* A general rule is to use these drugs with caution when other drugs with similar side effect profiles (e.g., central nervous system or respiratory depression, orthostasis, etc.) are co-administered. Cytochrome P450 interactions occur to varying degrees with these agents and should be considered. All atypical antipsychotics are pregnancy category C, with the exception of clozapine, which is category B.

Adverse Effects

Most adverse effects can be anticipated by reviewing the pharmacologic properties of these drugs. Generally speaking, extrapyramidal disorders, tardive dyskinesia, and neuroleptic malignant syndrome appear less frequent-

Table 2. Selected Drug-Drug Interactions

DRUG INTERACTION	EFFECT ON ANTIPSYCHOTIC	NOTES
Aripiprazole/ketoconazole	↑ levels	↓ aripiprazole dose by ½
Aripiprazole/itraconazole	↑ levels	↓ aripiprazole dose by ½
Aripiprazole/quinidine	↑ levels	↓ aripiprazole dose by ½
Aripiprazole/fluoxetine, paroxetine	↑ levels	↓ aripiprazole dose by ½
Aripiprazole/carbamazepine	↓ levels	Double aripiprazole dose
Clozapine/benzodiazapines	synergistic	Respiratory arrest
Olanzapine/carbamazepine	↓ levels	
Ziprasidone/drugs that ↑ QT on ECG*	↑ QTc	Avoid co-administration

* Also avoid using ziprasidone with congenital long QT, hypokalemic, and hypomagnesemic patients.[3]

ly with the atypical antipsychotics than with the typical agents; among the atypicals, risperidone (especially at increased dosages) seems to carry the highest risk for these dopamine receptor-related effects, as it does for prolactin elevation.[2] All can cause a reversible elevation in hepatic transaminases.[2] Blockade at α_1-adrenergic sites (especially clozapine, risperidone, olanzapine, ziprasidone, quetiapine, and aripiprazole) may cause orthostatic hypotension, miosis, and reflex tachycardia. Antihistaminic effects (especially clozapine, olanzapine, and quetiapine) principally include sedation. Muscarinic antagonism, causing both peripheral and central anticholinergic effects, is seen more with clozapine and olanzapine.[2] There has been a link to weight gain, hyperlipidemia, and the development or exacerbation of diabetes mellitus.[2-4] Agranulocytosis is seen in approximately 1% of clozapine-treated patients; even rarer with this agent is an idiosyncratic eosinophilic myocarditis; both are more likely to occur during the first weeks of therapy.[2] Ziprasidone may cause QT prolongation on the electrocardiogram — although apparently not to the extent that thioridazine does — and its use should be avoided with drugs or syndromes that prolong the QT interval. Ziprasidone has not been linked to torsade de pointes thus far.[3,5] Priapism has been associated with clozapine, risperidone, olanzapine, and quetiapine, and probably is mediated by α_1-adrenergic antagonism.[6-8] Priapism traditionally has been linked with the antidepressant trazadone, and several phenothiazine (as well as other typical) antipsychotics, in addition to several antihypertensives; mechanistically speaking, it could be encountered with any of the atypical antipsychotic agents.[8]

Acute Overdose

The high therapeutic index of atypical antipsychotics renders lethal ingestion a rare occurrence. However, toddlers ingesting a single tablet of clozapine, olanzapine, and risperidone have been significantly poisoned;[2] pediatric ingestion of any of these agents, even in small amounts, should be taken seriously. Symptomatic overdose generally features an exaggerated spectrum of the adverse effects described above, particularly CNS depressant and cardiovascular effects. Clozapine has been reported to cause CNS depression and seizures.[9] Risperidone poisoning is usually well-tolerated; however, it has been associated with neurologic (sedation, dystonia, and spasms), cardiovascular (sinus tachycardia, prolongation of the QTc, and wide-complex tachycardia), and electrolyte disturbances.[10,11] Toxicity from olanzapine manifests principally with CNS sedation; miotic pupils have also been described, thus imitating opioid and clonidine overdose.[12] Quetiapine poisoning may cause depressed mental status — as with any of these agents, at times profound — hypotension, and tachycardia; it has been shown to cause significant QT prolongation (710 msec) after massive ingestion.[13,14] Ziprasidone has been reported to cause mild sedation and some QTc prolongation after overdose.[15,16] Experience with aripiprazole is limited, and product information thus far reports only somnolence and vomiting after overdose.[17]

Treatment is largely supportive, and routine toxicologic standards should be employed. There are no specific antidotes known to be effective for these agents when taken in overdose. The emergency physician should be prepared to protect the airway if CNS sedation is profound. Telemetric monitoring should be used, and an electrocardiogram obtained to assess the QTc interval. Hypotension should be treated with intravenous fluids initially; norepinephrine or phenylephrine appear to be good choices for refractory hypotension considering the propensity of atypical antipsychotics to cause α_1-adrenergic blockade. Seizures should be treated with benzodiazepines, and barbiturates if necessary.[2] ❖

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Physician CME Questions

36. All of the following statements regarding the study about the visual analog scale (VAS) for pain are true *except*:
- a. The VAS consists of a 100-mm line without markings and has "No pain at all" on the far left and "Severe, uncontrolled pain" on the far right.
- b. Patients requesting pain medication had a mean VAS score of 66.
- c. Patients refusing pain medication had a mean VAS score of 45.
- d. The receiver operating characteristic curve for the VAS shows that a cutoff of 55 can be used to reliably predict which patients will desire pain medication.

37. In patients at risk from recent travel, severe falciparum malaria is strongly suggested by the presence of:
- a. High fever, impaired consciousness, respiratory distress, and hypotension
- b. High fever, stiff neck, petechial rash, and thrombocytopenia
- c. High fever, anorexia, and radiographic abdominal obstruction
- d. High fever, vomiting, flank tenderness, and urinary leukocyte casts

38. Overcrowding in the emergency department appears to be:
- a. linked mainly to attending physician experience level.
- b. dependent above all else upon seasonal variation.
- c. closely related to hospital occupancy.
- d. directly attributable to point-of-care testing.

39. Bispectral index (BIS) monitoring in the ED:
- a. correlates well with Glasgow Coma Scale scores in alcohol-intoxicated patients.
- b. does not correlate well with Glasgow Coma Scale scores in undifferentiated patients with altered level of consciousness.
- c. is useful in assessing degree of brain injury in the trauma patient.
- d. correlates well with Glasgow Coma Scale scores when the latter scores are less than 7.

40. Atypical antipsychotics can be expected to exhibit, to differing degrees:
- a. anticholinergic effects.
- b. antihistaminic effects.
- c. α_1-adrenergic blockade effects.
- d. All of the above

41. Which of the following drugs is most likely to prolong the QTc interval on the electrocardiogram?
- a. Trazadone
- b. Olanzapine
- c. Ziprasidone
- d. Becrazadine

42. Which of the following side effects frequently has been linked to atypical antipsychotics?
- a. Weight gain
- b. Hirsuitism
- c. Diuretic effect
- d. Vertigo

Answer key:

36. d	38. c	40. d	42. a
37. a	39. b	41. c	

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

Pulmonary Artifact

By Ken Grauer, MD

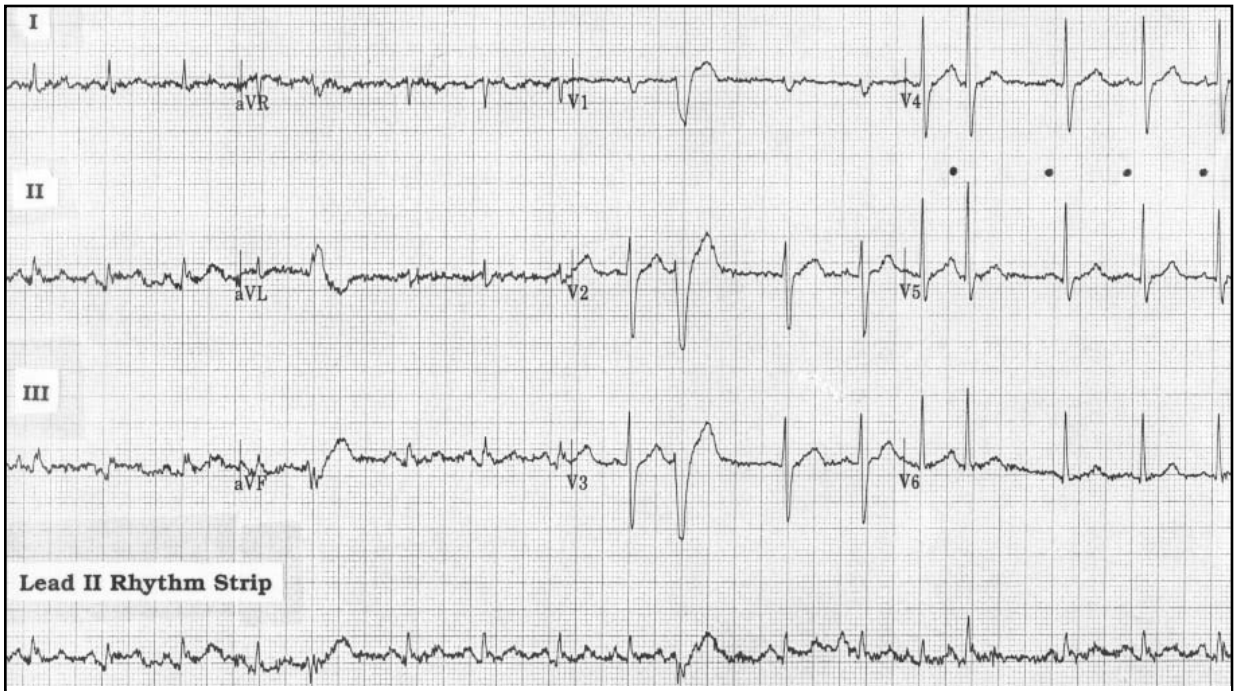

Figure. 12-lead ECG and lead II rhythm strip obtained from a 67-year-old man with pulmonary disease.

Clinical Scenario: The 12-lead ECG and rhythm strip shown in the Figure was obtained from a 67-year-old man with longstanding pulmonary disease who was admitted to the hospital for an exacerbation of this underlying condition. Computerized interpretation of the ECG seen here described the rhythm as "atrial fibrillation with a rapid ventricular response with a number of aberrantly conducted beats." Do you agree with this computerized interpretation?

Interpretation: Computerized ECG interpretations must always be overread by a physician. The purpose of such overreading is to ensure accuracy of the ECG interpretation. As a rule, computerized ECG interpretation systems are exceedingly accurate in measuring intervals, calculating heart rate and axis, and assessment of normal tracings. They are much less accurate in interpretation of the cardiac rhythm when the mechanism is not sinus.

The tracing in the Figure is replete with baseline artifact. This most likely is a result of altered respiration from acute exacerbation of the patient's underlying pulmonary disease. Unfortunately, little definitive information can be derived from the Lead II rhythm strip other than recognition that the rhythm is not regular, especially toward the latter part of the tracing. Nevertheless, near regularity of the rhythm at the beginning of this rhythm strip (at a rate just over 100/minute) and consistent presence of an upright deflection preceding most QRS complexes suggests that the computerized interpretation is wrong, and that the underlying rhythm is not atrial fibrillation. The most helpful clues leading to the etiology of the rhythm lie with the history (longstanding pulmonary disease) and with focusing attention to other parts of this 12-lead tracing, particularly to simultaneously recorded leads V_4, V_5, and V_6. Baseline artifact is much less apparent in these 3 leads. This allows more accurate assessment of the rhythm, and suggests that the underlying irregularity is due either to the presence of multiple premature atrial contractions (PACs), or more likely multifocal atrial tachycardia (MAT) in view of this patient's longstanding pulmonary disease (dots in the Figure between leads V_4-V_5 highlight the timing of several different looking P waves). Occasional QRS widening is most probably the result of aberrantly conducted PACs (the T waves are peaked immediately preceding the wide QRS in leads V_3 and V_6), although occasional ventricular ectopy cannot be excluded. A repeat ECG on this patient was of much better quality and confirmed MAT as the rhythm diagnosis. ❖

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From Stingers to Fangs: Evaluating and Managing Bites and Envenomations

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Significant numbers of bites and envenomations occur annually,[1] with bites and stings by arthropods accounting for about one-third of these. Surprisingly, the order Hymenoptera, which includes bees, wasps, and ants, is the source of most deaths from envenomations in the United States. Whether the creature inflicts a bite or sting that results in an anaphylactic reaction, impressive local effects, or a life-threatening systemic reaction, the emergency physician must be able to institute appropriate and effective treatment. Emergency physicians also must be able to recognize clinical envenomation patterns, since some critically ill patients may not be able to convey the details of the "attack." Since all areas of the country are represented in the envenomation statistics, all emergency physicians should be familiar with identification and stabilization of envenomated patients and know what resources are available locally for further management of these often complicated patients.

—The Editor

Snakes

Approximately 8000 bites from poisonous snakes occur each year in the United States, resulting in 5-15 deaths annually.[1-6] Venomous snakes are found in virtually every state. Two snake families, Elapidae (coral snakes) and Viperidae (pit vipers), are found in the United States. Snakes in the Viperidae family, subfamily Crotalidae, include the rattlesnakes, copperheads, and moccasin snakes. This group accounts for about 90-95% of poisonous snakebites, with coral snakes accounting for 2-3% of bites and exotic snakes accounting for 3-5% of bites.[1,2] This report will concentrate on management of the crotalid snakebites.

Pit vipers have a number of characteristic features that distinguish them from other snakes. *(See Figure 1 a-b.)* A pit is located on each side of the head between the eye and nostril and contains heat-sensitive organs that assist in localizing prey. The pupils generally are elliptical and vertical in nature, as opposed to the round pupil of a harmless snake. The head of a pit viper usually is triangular in shape, not round or narrow. A pair of long-hinged fangs are folded against the palate and move forward when the snake strikes.

Epidemiology. Snakes are poikilothermic and are most active during warm weather and in the daylight. Most bites occur between April and October. Males are bitten more frequently than females (9:1).[1,2,5] Bites occur most often on the extremities, with upper extremities more common in adults (85% of bites) and lower extremities affected more frequently

in children (70%).[7-9] More than half of bites occur when a person is purposely handling a known venomous snake.[10]

The venom is a complex mixture of enzymes that function to immobilize, digest, and kill the snake's prey. Proteolytic enzymes, hyaluronidase, phospholipase, and thrombin-like enzymes contribute to the local and systemic effects seen following an envenomation.

Clinical Manifestations. The clinical presentation of envenomation is variable, depending on the type of snake, site of bite, host factors, and amount of venom injected. Up to 20-25% of bites may be described as "dry" with no envenomation taking place.[3] Pit viper envenomation may cause both local and systemic symptoms. Intense, burning pain is seen at the bite site in most cases of envenomation. One or more puncture marks from fangs frequently are noted. *(See Figure 2.)* Edema occurs at the bite site and progresses at various rates, depending on the amount of venom injected. Erythema and ecchymosis often are noted. Over time, fluid-filled or hemorrhagic bullae may form and eventually lead to necrosis. Extremities, especially digits, may become extremely swollen and tense, leading to possible vascular compromise.[3,5,11-14]

Tender regional lymph nodes may be the first systemic sign of envenomation. Other signs include nausea, vomiting, perioral numbness, metallic taste in the mouth, muscle fasciculations, weakness, bleeding, hypotension, and shock. A Snakebite Severity Score has been developed and validated based on symptoms and signs in six areas: local wound, pulmonary, cardiovascular, gastrointestinal, central nervous system, and hematologic system.[15] *(See Table 1.)* Death is an infrequent occurrence, and the patients frequently present with signs of severe massive systemic effects, suggesting direct venous injection of the venom. Anaphylaxis to the venom also may occur, especially in victims of previous bites, because of prior sensitization and development of IgE antibodies to venom.[4,5,11,16,17] Laboratory evaluation frequently reveals a consumptive coagulopathy and thrombocytopenia. Fibrin degradation products often are elevated and PT and PTT are prolonged. Fibrinolysis is caused by snake venom activation of plasminogen and direct fibrinolysis.

Management. *Pre-hospital Care.* Pre-hospital treatment of snakebite victims, whether by medical personnel or lay bystanders, is the subject of much folklore and controversy. Many traditional first-aid measures actually have been proven to be of little benefit or even harmful to the patient.[11,18] Pre-hospital care of snakebite victims should include assessment and maintenance of the "ABCs" (airway, breathing, and circulation); minimization of systemic venom effects without increasing the risk of local tissue damage; and rapid transport to a facility where definitive treatment can take place.

Several first aid measures recommended in the past that no longer are advised include cryotherapy, incision and suction, and electric shock. Cryotherapy involved packing or immersing the bitten extremity in ice or ice water. It was believed that this lowered enzyme activity and slowed absorption. However, no significant benefit has been noted in studies, and harmful effects (such as tissue loss or amputation) have been seen.[18,19]

Incision and suction of the bite is controversial. Incision potentially can damage deeper structures, especially in the hand or neck.[8,20] Oral suction increases the risk of infection. One device made for mechanical suction of snakebites is the Sawyer Extractor. Both animal and human studies have shown a minimal amount of venom recovered using this device with skin necrosis noted in one study.[20-24] This therapy only could be recommended if applied within minutes of envenomation in a victim who is more than 30-40 minutes away from definitive care.

Electric shock therapy use has been reported since 1986, when high-voltage, low-amperage electric shock was used to treat a variety of bites and stings in native South Americans.[25] No animal models support the use of electric shock.[18,24,25] Significant complications, such as burns, seizures, and myocardial infarction, have been reported.[27]

Currently, the most controversial first aid measure is the use of a constricting band to slow systemic absorption of venom. Arterial tourniquets no longer are used because of the potential

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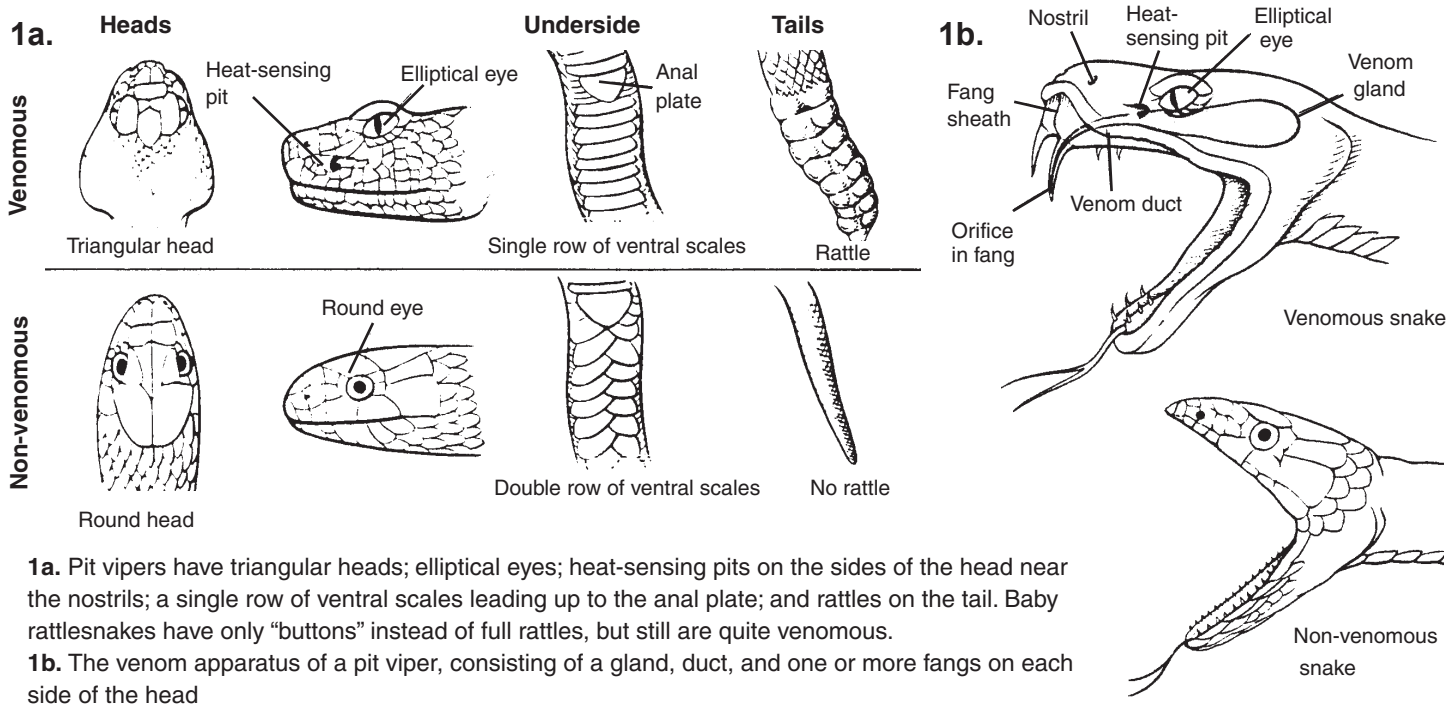
Figure 1a-b. Identification of Poisonous Pit Vipers



1a. Pit vipers have triangular heads; elliptical eyes; heat-sensing pits on the sides of the head near the nostrils; a single row of ventral scales leading up to the anal plate; and rattles on the tail. Baby rattlesnakes have only "buttons" instead of full rattles, but still are quite venomous.
1b. The venom apparatus of a pit viper, consisting of a gland, duct, and one or more fangs on each side of the head

Adapted from: Sullivan JB Jr., Wingert WA, Norris RL Jr. North American venomous reptile bites. *Wilderness Medicine.* 3rd ed. St. Louis:Mosby;1995:684,685.

for limb ischemia.[8] A constricting band is a wide (2-4 cm), flat band that is applied tight enough to occlude superficial veins and lymphatics but loose enough to admit one or two fingers between the band and the extremity to permit deep venous and arterial flow. It has the advantage of delaying the onset of systemic toxicity until antivenin can be given.[28] However, some animal studies have shown that keeping the venom at the bite site may worsen the local necrosis.[28,29] They also show the potential for "bolus effect" of venom into the systemic circulation, leading to rapid deterioration of the patient.[28,30] For now, recommendations for the use of a constricting band would include severe or progressive envenomation in a patient with a prolonged transport time.

While patients are being transported, all nonessential movement should be minimized.[18] The extremity should be splinted in a position of comfort, and constrictive clothing and jewelry should be removed.

Intravenous access should be obtained, vital signs monitored, and parenteral analgesia given if needed. The patient should not be given anything to eat or drink, supplemental oxygen therapy should be administered. Hypotension during transport should be treated vigorously with crystalloid fluid therapy.[11,14,18]

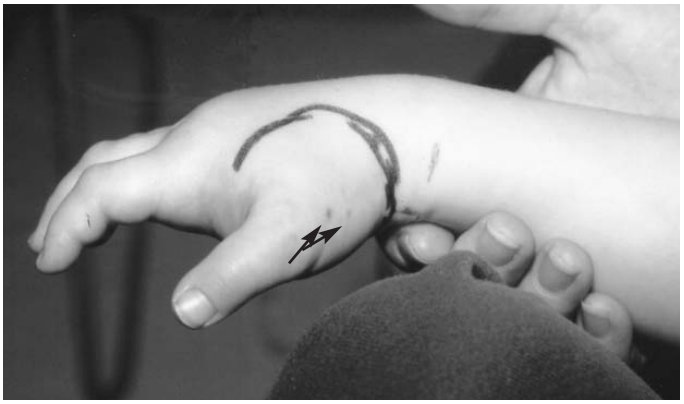
Emergency Department Management. Once again, assessment and maintenance of ABCs should take place on patient arrival in the emergency department (ED). At least one large-bore intravenous line should be started. Recommended laboratory studies are listed in Table 2.[7,12,14] Tetanus status should be determined and updated if needed. Progression of the swelling should be monitored and documented. Antivenin therapy is the mainstay of medical management for moderate to severe envenomations. Surgery may be required in carefully selected patients. Prophylactic antibiotics currently are not recommended.[7,11,31-34] Patients with minimal envenomation may be observed for six hours and sent home if there is no progression of symptoms. All others should be admitted to the hospital, and most should be admitted to an intensive care setting.[11,14,35,36]

Antivenin Therapy. Antivenin administration is the mainstay of medical management of venomous snakebites.[5,8,14] Antivenin (Crotalidae) Polyvalent (ACP) was introduced in 1954 and was the only antivenin available until October 2000. At that time, Crotalidae Polyvalent Immune Fab antivenin (FabAV) was approved for use.[37,38]

ACP is derived from horse serum and carries with it the risk of immediate hypersensitivity reactions, estimated to occur in up to 33% of patients.[8,39,40,41] Doses greater than 10 vials are associated with an almost 100% incidence of serum sickness.[39,40,41] Although ACP has been in use for decades for treating crotalid snakebites, no prospective, randomized trials have been performed.

Indications for administration of antivenin include a progressive venom injury (worsening local injury), coagulation abnormalities, or systemic effects of the envenomation.[11,14,40] Antivenin administration is expected to reverse the coagulopathy and systemic effects and prevent further local injury.[5,7,8,37,42] It currently is recommended that bites from copperheads, the least toxic species of crotalids, not be treated with ACP.[11,43-45] With the increased use and availability of FabAV, this restric-

Figure 2. Copperhead Snakebite



Hand of an 18-month-old child bitten near the thumb *(see arrows)* by a small copperhead snake. Note two fang marks and large amount of swelling.

tion may be reconsidered, as local swelling may be severe.

Dosage of ACP has been based on severity of envenomation. In general, 0-5 vials are used for minimal envenomation, 10-15 vials for moderate envenomation, and 15-20 vials for severe envenomation.[14,46] The patient then is reassessed and additional vials administered when indicated.[14,40,47]

ACP is diluted in a crystalloid solution, depending on the desired amount of volume to be infused. After skin-testing, a small amount of the antivenin is infused slowly, monitoring for anaphylactic reactions. If no adverse reactions occur, the rate of the infusion is increased to deliver the initial antivenin dose during approximately a two-hour period. Anaphylactic reactions are treated in the standard manner.[14,47]

FabAV is produced by immunizing sheep with crotalid snake venom. The serum then is digested, using papain to produce antibody fragments (Fab and Fc). Fc is more immunogenic and is eliminated during the purification process.[37] When initially tested in animals, FabAV was found to be 5.2 times more potent than ACP against crotalid snake venoms.[46] Acute hypersensitivity rates are reported at 20%, with a 23% incidence of serum sickness.[37,40,42,48]

Prospective trials of FabAV have been done and continue to be performed. An initial study of 11 patients who received 4-8 vials of FabAV showed resolution of symptoms with no allergic reactions.[37] However, recurrence of limb swelling and coagulation defects was noted in 27% of these patients. A study done shortly thereafter used a different dosing schedule.[42] Patients initially were treated with six vials of FabAV, and a repeat dose given if needed. They then were randomized to a scheduled group that received two-vial treatments at 6, 12, and 18 hours after initial dose, or to an "as-needed" group. Total dosages were similar in the two groups, with no symptoms recurring in the scheduled group.

Recurrence is described as local or coagulopathy recurrence. Patients with an initial coagulopathy are more likely to experience a recurrence.[49] Recurrence is thought to be due to a number of factors, including failure to neutralize all venom initially and the more rapid clearance of unbound Fab in relation to venom components.[49,50]

Newer recommendations for FabAV administration include an initial dose of 4-6 vials, repeated once for initial control.[11,42,49] Additional two-vial dosages should be scheduled at 6, 12, and 18 hours. All patients should be re-evaluated at least once during the first five days post-treatment. Those with initial coagulation abnormalities should be reassessed approximately every 48 hours until parameters are stable. Retreatment with FabAV may be indicated for recurrence of the coagulopathy.[11,49]

Surgical Management. Several surgical techniques have been used in the management of snakebite victims. The most common ones are incision therapy, excision of the bite site, fasciotomy, and digit dermatomy. Many of these techniques were used in the early management of snakebites to address the issues of tissue necrosis, decreased function, and limb loss,[51-54] in attempts to avoid the risk of antivenin-caused anaphylactic reactions.[39] No randomized, controlled, clinical trials exist comparing surgical procedures to appropriate use of antivenin.[55] A number of studies have shown good functional outcome with minimal or no surgical intervention in patients treated with early, adequate, intravenous antivenin.[5,56-58] With the development and availability of better antivenin products, surgical therapy now is used only in a few carefully selected patients.[55]

Excision of the bite site was used in the hope that significant amounts of venom could be removed.[51] Excisional techniques range from local excision of a subcutaneous "plug" of tissue, to opening an entire extremity and removing all hemorrhagic tissue.[38,51] There is no experimental data to support excision used in this way. It is recommended that debridement of hemorrhagic blebs or frankly necrotic skin take place 3-5 days after the bite occurs.[8,55]

Fasciotomy often has been advocated as primary treatment of crotalid snakebites in the assumption that compartment syndrome is a common complication of the envenomation. The local and systemic toxic effects of crotalid venom mimic the signs and symptoms of compartment syndrome.[55] Massive local edema often is present after a snakebite. This edema, however, generally is confined to the subcutaneous tissue, and rarely is associated with elevated compartment pressures.[59] The only way to determine whether a compartment syndrome exists is to measure intracompartmental pressure.[53,55,56] In cases in which compartment syndrome has developed, use of antivenin has been shown to resolve the majority of them.[11,60,61] It would seem appropriate to proceed to fasciotomy only in those patients with persistently elevated compartment pressure even after antivenin administration.

The finger is an area with limited capacity for edema. Currently, there is no accurate method for measuring compartment pressure in the finger, so a clinical diagnosis is used. A finger that is tense, blue, or pale with absent or poor capillary refill time is a candidate for a digit dermatomy.[53,55] This technique consists of a longitudinal incision through the skin only on the medial or lateral aspect of the digit, extending from the web to the mid-portion of the distal phalanx.[53] This is done using local anesthesia, and

Table 1. Snakebite Severity Score

CRITERION	POINTS
PULMONARY SYSTEM	
No symptoms/signs	0
Dyspnea, minimal chest tightness, mild or vague discomfort, or respirations of 20-25 breaths/min	1
Moderate respiratory distress (tachypnea, 26-40 breaths/min; accessory muscle use)	2
Cyanosis, air hunger, extreme tachypnea, or respiratory insufficiency/failure	3
CARDIOVASCULAR SYSTEM	
No symptoms/signs	0
Tachycardia (100-125 beats/min), palpitations, generalized weakness, benign dysrhythmia, or hypertension	1
Tachycardia (126-175 beats/min), or hypotension, with systolic blood pressure > 100 mmHg	2
Extreme tachycardia (> 175 beats/min), hypotension with systolic blood pressure < 100 mmHg, malignant dysrhythmia, or cardiac arrest	3
LOCAL WOUND	
No symptoms/signs	0
Pain, swelling, or ecchymosis within 5-7.5 cm of bite site	1
Pain, swelling, or ecchymosis involving less than half the extremity (7.5-50 cm from bite site)	2
Pain, swelling, or ecchymosis involving half to all of extremity (50-100 cm from bite site)	3
Pain, swelling, or ecchymosis extending beyond affected extremity (> 100 cm from bite site)	4
GASTROINTESTINAL SYSTEM	
No symptoms/signs	0
Pain, tenesmus, or nausea	1
Vomiting or diarrhea	2
Repeated vomiting, diarrhea, hematemesis, or hematochezia	3
HEMATOLOGIC SYMPTOMS	
No symptoms/signs	0
Coagulation parameters slightly abnormal: PT 20 sec; PTT 50 sec; plts 100-150,000/mL; or fibrinogen 100-150 mcg/mL	1
Coagulation parameters abnormal: PT 20-50 sec; PTT 50-75 sec; plts 50-100,000/mL; or fibrinogen 50-100 mcg/mL	2
Coagulation parameters abnormal: PT 50-100 sec; PTT 75-100 sec; plts 20-50,000/mL; or fibrinogen < 50 mcg/mL	3
Coagulation parameters markedly abnormal, with serious bleeding or the threat of spontaneous bleeding; unmeasurable PT or PTT; plts < 20,000/mL; or undetectable fibrinogen; severe abnormalities of other laboratory values also fall into this category	4
CENTRAL NERVOUS SYSTEM	
No symptoms/signs	0
Minimal apprehension, headache, weakness, dizziness, chills, or paresthesia	1
Moderate apprehension, headache, weakness, dizziness, chills, paresthesia, confusion, or fasciculation in area of bite site	2
Severe confusion, lethargy, seizures, coma, psychosis, or generalized fasciculation	3

Note: Points are assessed on the basis of manifestations caused by the venom itself (antivenin reactions not included). Total score ranges from 1 to 20. A higher score indicates more severe effects.

Key: **PT** = prothrombin time; **PTT** = partial thromboplastin time; **plts** = platelet count

Adapted from Dart RC, Hurlbut KM, Garcia R, et al. Validation of a severity score for the assessment of crotalid snakebite. *Ann Emerg Med* 1996;27:321-326.

the wound heals by secondary intention. This technique should not be used routinely in all finger bites, nor should it be used prophylactically to prevent a digital compartment syndrome.[55]

Spiders

Spiders are in the class Arachnida, part of the phylum Arthropoda. All spiders are carnivores and have fangs that they use to deliver venom to their prey. Most fangs are not strong enough to penetrate human skin. Only two clinically important spiders are found in the United States: the brown recluse and the black widow. These two spiders accounted for about 6% of reported bites and envenomations in 2000.[1]

Brown Recluse. Brown recluse spiders (Loxosceles) are found in most of the United States, but are most common in the

Table 2. Recommended Laboratory Studies in Crotalid Envenomation

Complete blood count
• Platelet count
PT/PTT
• Fibrinogen and fibrin split products
Electrolytes
• Blood urea nitrogen, creatinine
Creatine phosphokinase
• Blood type and crossmatching
Urinalysis

Table 3. Systemic Effects of Brown Recluse Spider Bite

- Fever
- Nausea
- Vomiting
- Arthralgias
- Myalgias
- Rashes
- Hemolysis
- Thrombocytopenia
- Hemorrhage
- Intravascular renal failure

Table 4. Unproven Treatments for Brown Recluse Spider Bites

- Corticosteroids
- Antibiotics
- Dapsone
- Early excision of the bite
- Hyperbaric oxygen therapy
- Cyproheptadine
- Topical nitroglycerin
- Anti-Loxosceles Fab fragments
- Debridement and skin grafting

midwestern and southern states.[62] They are found in woodpiles, sheds, garages, and closets. They also can hide in bedding and piles of clothing. The brown recluse generally is nonaggressive except when threatened or trapped against the skin of a victim.

The spider averages about a centimeter in length and often is light brown to tan in color. There is a distinctive, dark, violin-shaped mark on the dorsal aspect of the cephalothorax. In contrast to most spiders, the brown recluse has six eyes rather than eight.

The venom contains several different enzymes and proteins. Sphingomyelinase D is the enzyme that is most active and the cause for the majority of toxic effects.[62] It is cytotoxic to both endothelial cells and red blood cells.[63] The tissue necrosis is thought to be due to the induction of endothelial disruption, intravascular hemolysis, platelet aggregation, and thrombus formation by sphingomyelinase D. Polymorphonuclear leukocyte-induced vasculitis also contributes to the tissue necrosis.

Clinical Presentation. The bite initially may go unnoticed, but often is accompanied by a mild burning sensation that worsens over several hours.[12,64,65] Pruritus, pain, and erythema occur, as well as a central blister at the bite site. The initial skin lesion then increases in size and develops a purplish discoloration over the next several hours to days.[65] As necrosis continues, the lesion develops into an ulcer of variable size. Extremities are the sites most often affected.[65,66]

Systemic signs and symptoms of envenomation occur in up to 40% of patients, and may include fever, nausea, vomiting, arthralgias, myalgias, and rashes.[62,64,65] Children are more likely to develop hemolysis, thrombocytopenia, hemorrhage, and renal failure.[12,67] Death is rare. *(See Table 3.)*

Management. There currently are no specific tests to diagnose brown recluse spider envenomation, making the definitive diagnosis difficult. Laboratory tests that should be assessed, especially if systemic signs are present, include complete blood count, platelet count, coagulation studies, electrolytes, blood urea nitrogen, creatinine, and urinalysis.[12,64]

All wounds should be cleaned thoroughly and tetanus administered if needed. Other measures should include elevation of the bitten extremity and the judicious use of analgesics.

A variety of treatments have been tried, including corticosteroids, antibiotics, dapsone, early excision of the bite, and hyperbaric oxygen therapy.[68-78] No randomized, controlled studies exist supporting any of these therapies. Animal studies evaluating additional therapies, such as cyproheptadine and topical nitroglycerin, have shown no benefit.[71,79,80] Dapsone, in particular, should be avoided in children as it can lead to methemoglobinemia and hemolysis. *(See Table 4.)*

Animal studies using intradermally administered anti-Loxosceles Fab fragments have shown promise in inhibiting venom-induced inflammation.[81-83] Further research, including human studies, is needed.

If hemolysis occurs, it is important to maintain good urine output. The urine should be alkalinized with the intravenous administration of sodium bicarbonate to keep the urine pH greater than 7. Close monitoring of renal function and hematocrit is important.

Debridement and skin grafting may be necessary if large areas of necrosis are present, but should be delayed until the area clearly has been demarcated.[64,78] In general, skin lesions heal in weeks to months, depending on the size.

Patients with systemic symptoms should be admitted for further monitoring.

Black Widow. Black widow spiders (Latrodectus) are found throughout North America, with the exception of Alaska.[64] They can be found in attics, barns, storage sheds, garages, firewood, hay bales, and outhouses. They also may hide in clothing and shoes, with about 15% of bites occurring while the victim dresses.[84] There are fewer bites from the black widow than the brown recluse.

In the United States, the spider is shiny black, with a red hourglass marking on the abdomen. The female is about 3-4 cm in diameter, and the male is about one-quarter this size. Only the female has fangs large enough to penetrate human skin. The black widow spider has eight eyes and legs.

The venom produced by this spider is one of the most potent venoms known.[85] Although it lacks a tissue toxin, minimizing local effects, it has a potent neurotoxin, alpha-latrotoxin. Its primary site of action is the neuromuscular junction. The venom causes release and inhibits the reuptake of acetylcholine and norepinephrine, resulting in overstimulation of the motor endplate.[86-88]

Clinical Presentation. The bite of the black widow may be painless or present as a pinprick sensation. The majority of bites occur on the extremities.[84] Regional lymph nodes become tender during the next 30 minutes to two hours. Within 1-2 hours, a target lesion may appear at the bite site with some surrounding erythema.[84,89] The hallmark of envenomation is muscle cramping, usually involving the abdomen, chest, and back. This cramping has its onset 30-90 minutes after the bite and peaks in 3-12 hours, with a waxing and waning quality.[84] Autonomic symptoms often include nausea, vomiting, diaphoresis, hypertension, and tachycardia. Death is rare, but hypertension can be life-threatening.[90]

In children, abdominal pain and rigidity are the most common symptoms. There are, however, no peritoneal signs. Marked hypertension is common, as well. Anxiety, agitation, and irritability may be the initial presenting signs, especially in younger children. Grunting and respiratory distress are due to chest and abdominal pain. Weakness, headache, and periorbital edema may be present and persist for days or weeks.[12,84,89-91]

Management. There are no specific tests to diagnose black widow spider envenomation. Laboratory abnormalities rarely occur and are non-specific.[84,89]

As always, the initial priorities should focus on stabilization and maintenance of ABCs, especially in children. All wounds should be cleansed thoroughly and tetanus administered, if indicated. The bitten extremity should be elevated, and a cold compress should be applied.

Treatment is directed at the relief of symptoms. Analgesia is an important component of this treatment. Mild cases may be treated with oral analgesics or narcotics such as codeine or hydrocodone. More severe cases usually require intravenous morphine (0.1-0.2 mg/kg every 2-4 hours). Benzodiazepines such as diazepam, lorazepam, or midazolam can be beneficial in relieving anxiety and providing muscle relaxation through centrally mediated responses. The combination of a narcotic and a benzodiazepine often will alleviate symptoms without further treatment required.[64,89-92]

Calcium gluconate (10%) previously has been used as first-line treatment. Most controlled studies, however, have not shown a benefit to this treatment, and its use has fallen out of favor.[84,90,93]

An antivenin is available for black widow spider envenomations. It generally is indicated only for the severe envenomations that are unresponsive to other treatments.[64,84,89,90,94-96] Indications would include life-threatening hypertension and tachycardia, respiratory difficulty, refractory pain, and high-risk groups such as pediatric patients, pregnant women, and the elderly.[93] One vial of antivenin is diluted in 50-100 mL of normal saline and infused slowly over 30-60 minutes. Rapid, complete resolution of symptoms without relapses is the norm. Administration of the antivenin should be carried out as soon after envenomation as possible, although effective use up to three days after a bite has been reported.[94-96] Immediate hypersensitivity reactions and serum sickness may occur, as the antivenin is derived from horse serum.

Patients with mild symptoms controlled by oral analgesics may be sent home with close follow-up; pain may recur or worsen. Most pediatric patients should be admitted, as well as those patients requiring intravenous analgesics and those exhibiting evidence of hypertension or autonomic symptoms.[64,90,92]

Scorpions

Worldwide, scorpions account for many deaths annually, but in the United States, one death reported in 2000 was the only death reported in the last 30 years.[1] Only one species in the United States, *Centruroides exilicauda*, produces serious toxicity. Otherwise known as the "bark scorpion" because it resides in the bark of trees, this scorpion is found primarily in Arizona and the neighboring southwestern states. It has two pinching claws anteriorly and a tail that ends in a telson. The telson contains a pair of poisonous glands and a stinger. A scorpion grasps its prey with the pincers and stings its victim by arching its tail over its head. Scorpions also may be found in woodpiles, crevices, shoes, and clothing. Most envenomations occur at night.

The venom contains a neurotoxin that is excitatory and affects both autonomic and skeletal neuromuscular systems. Both sympathetic and parasympathetic systems are stimulated.

Clinical Presentation. Most patients will present with only local pain, tenderness, and tingling. Systemic symptoms rarely occur and are more likely to be severe in children.[91,97,98] Sympathetic stimulation may cause tachycardia, hypertension, hyperthermia, diaphoresis, and agitation. Parasympathetic symptoms include hypotension, bradycardia, and SLUDGE (salivation, lacrimation, urination, defecation, and gastric emptying). Young children often present with disconjugate, roving eye movements, jerking of the extremities, and opisthotonus.[64,91,97,99] Complications can include pancreatitis, upper airway obstruction causing respiratory failure, and rhabdomyolysis.[97]

Management. The treatment of scorpion stings is supportive. Cold compresses and over-the-counter analgesics are used for local pain. All wounds should be cleaned thoroughly, and tetanus administered if indicated.[64,91,97]

Assessment and management of ABCs is critical, especially in children. In severe cases, intubation and ventilation may be necessary.[98] Parenteral analgesics and benzodiazepines may be required for severe pain and agitation. Midazolam is preferred by many authors, often as a continuous infusion.[100] Severe tachycardia generally responds to beta-blockers, and hypertension can be treated with intravenous hydralazine.[101]

An antivenin is available for use in Arizona. It is derived from goat serum, and is not approved by the United States Food and Drug Administration. It effectively treats about 70% of cases within 1-3 hours of administration.[98] There is a risk of both immediate hypersensitivity reactions and serum sickness.[102] It is indicated for patients with severe cardiorespiratory or central nervous system dysfunction. The dose is 1 vial mixed in 50 mL of normal saline and infused over 30-60 minutes. Antivenin use may allow discharge from the ED in a select group of patients.[98] Most pediatric patients and others with severe systemic symptoms should be admitted to the hospital.

Hymenoptera

Hymenoptera is an order of arthropods that includes bees, wasps, and ants. They are the leading cause of death from envenomation in the United States, with 40-50 fatalities per year.[1,2] Apids (i.e., honeybees, bumblebees) possess a barbed stinger that remains in the victim after a sting. The vespids (i.e., wasps, hornets, yellow jackets) can sting multiple times and rarely leave the stinger behind.

Yellow jackets cause the majority of allergic reactions from insect stings. They nest in the ground or in walls, and are disturbed by lawn mowing, gardening, and other outdoor activities. Yellow jackets are attracted to food and garbage, as they feed on sugar-containing substances.

Vespids, in general, are more aggressive than bees. The exception would be the Africanized ("killer") honeybee. African bees were brought into Brazil in 1956 to help increase honey production. A few bees escaped and began mating with the established bees. These bees, which have aggressive tendencies, began migrating north, reaching Texas in 1990. They are now found in Texas, Arizona, and southern California.[103] The venom of the Africanized bee is no more toxic than that of other bees.[104,105] However, these bees often attack in swarms, so a large dose of venom is delivered to the victim.

The venom apparatus is located in the posterior end of the abdomen. It consists of the venom glands, a reservoir, and a stinging structure. The venom contains a number of enzymes, including phospholipase A and hyaluronidase. Phospholipase A is thought to be one of the major allergens in the venom.[106] Melittin is a principal component of honeybee venom, which damages cell membranes through detergent-like action.[107]

Clinical Presentation. Hymenoptera stings most often result in swelling, erythema, and pain at the site of the sting. This reaction generally subsides within several hours. Larger local reactions also are common. The swelling extends over a large area, usually peaks within 48 hours, and may last as long as seven days.[108] These probably represent a cell-mediated (type IV) immunologic reaction, although it may be mediated by IgE antibodies. Large local reactions may be confused with cellulitis, although this rarely occurs after a sting.

Serum sickness may occur within 7-10 days after a sting.[109] This is characterized by fever, arthralgias, and urticaria and appears to be immunologically mediated. Other unusual reactions include nephritic syndrome, seizures, Guillain-Barré syndrome, and progressive demyelinating neurologic disease.[110-112]

Anaphylaxis is the most serious complication of a hymenoptera sting. It is estimated that up to 4% of the U.S. population is sensitized to bee stings.[113] Common symptoms include flushing, angioedema, generalized urticaria, pruritus, and nausea. Life-threatening manifestations may include bronchospasm, upper airway edema, hypotension, and shock. Symptoms generally begin within 10-20 minutes after the sting; however, reactions up to 72 hours later have occurred.[108] Most deaths occur within the first hour. This often is due to upper airway obstruction, hypotension, or both.[114] Children suffering anaphylaxis who subsequently are stung tend to have reactions that are similar to or less severe than the initial episode.[115-118]

Management. Mild local reactions can be treated by removing the stinger and cleaning the wound with soap and water. Application of an ice pack or cold compress often provides relief. Oral antihistamines, such as diphenhydramine or hydroxyzine, also are effective.

Traditionally, it has been taught that the stinger should be removed by flicking it or scraping it off, to avoid releasing more venom into the wound. Newer evidence shows that the venom sac continues to contract and inject venom for up to 20 seconds after the sting, so removal of the stinger should be expedited.[119] The method of removal does not seem to affect the amount of venom delivered.

Corticosteroids may be used for the management of large local reactions, as they seem to hasten resolution of the symptoms. Serum sickness should be treated with a course of both oral corticosteroids and oral antihistamines.[64]

Treatment of anaphylaxis starts with the administration of subcutaneous or intramuscular epinephrine. The dose is 0.01 mL/kg of the 1:1000 solution (up to 0.3 mL) and should be administered as quickly as possible.[120] If hypotension or shock is present, an intravenous line should be started and 20 mL/kg boluses of normal saline or lactated Ringer solution given. Intravenous epinephrine (1:10,000 solution) should be given. Multiple doses may be needed.[121]

Attention to airway and breathing occurs simultaneously. Oxygen, intubation, and ventilation may be needed. Inhaled beta-agonists, such as albuterol, may help alleviate bronchospasm. Diphenhydramine (1 mg/kg intravenous) and methylprednisolone (2 mg/kg intravenous) should be given to help block the delayed hypersensitivity reaction. An H_2 blocker such as cimetidine often is used, as well. Patients with life-threatening symptoms should be admitted to the hospital for at least 24 hours.

Patients experiencing allergic symptoms should be discharged with at least two epinephrine autoinjectors (EpiPen, Dey Inc., Napa, CA) and instructions on how to use them. Those patients having anaphylactic reactions should wear a medical alert bracelet and also be referred to an allergist for possible venom immunotherapy.[64]

Conclusion

Envenomations can be frequent occurrences, depending on the area in which you practice. A high index of suspicion is needed to diagnose these conditions, as most do not have specific confirmatory tests. Numerous treatments have been advocated in the past, but supportive care is all that is needed in most instances.

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CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing 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 provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

CE/CME Questions

Please review the text, answer the following questions, check your answers against the key that appears following the questions, and then review the materials again regarding any questions answered incorrectly. ***To receive credit for this activity, you must return the enclosed CE/CME evaluation in the enclosed envelope.*** *For further information, refer to the "CE/CME Instructions" on the previous page.*

This testing procedure has proven to be an effective learning tool for adults. If you have any questions about the new testing method, please contact Customer Service at 1-800-688-2421.

1. Pit vipers have all of the following characteristics *except*:
   A. vertical elliptical pupils.
   B. round head.
   C. heat-sensing pit.
   D. hinged fangs.
   E. single row of ventral scales.

2. Approximately what percentage of snakebites are "dry"?
   A. Less than 5%
   B. 5-10%
   C. Up to 20-25%
   D. 40-50%
   E. 70-75%

3. Cryotherapy is indicated as first-line treatment for victims of snakebites.
   A. True
   B. False

4. Which of the following first-aid measures for snakebites is definitely of value?
   A. Cryotherapy
   B. Incision and suction
   C. Electric shock
   D. Tourniquet
   E. Transport to hospital

5. When should fasciotomy be done on snakebite victims?
   A. As routine treatment
   B. As prophylaxis for extremity bites
   C. When measured intracompartmental pressures remain elevated despite the use of antivenin
   D. When there is massive local edema

6. Which of the following is true?
   A. FabAV appears less potent than ACP.
   B. Recurrence of coagulopathy is more common with FabAV.
   C. Serum sickness is more common with FabAV.
   D. Scheduled does of FabAV are probably not needed for the first 18 hours.
   E. ACP is sheep-serum derived.

7. Which enzyme is thought to be responsible for most of the toxic effects of the brown recluse spider bite?
   A. Sphingomyelinase D
   B. Hyaluronidase
   C. Thrombin-like enzymes
   D. Phospholipase A
   E. Alpha-latrotoxin

8. What is the hallmark of black widow spider envenomation?
   A. Fever
   B. Necrosis at bite site
   C. Tachycardia
   D. Headache
   E. Muscle cramping

9. Which treatment for black widow spider envenomation is no longer recommended as first-line therapy?
   A. Morphine
   B. Valium
   C. Lorazepam
   D. Calcium gluconate
   E. Codeine

10. Which is *not* a sign or symptom of scorpion envenomation?
    A. Tachycardia
    B. Local pain at bite site
    C. Necrotic lesion
    D. Roving eye movements
    E. Hypertension

CE/CME Objectives

Upon completing this program, the participants will be able to:

a.) Quickly recognize or increase index of suspicion for envenomations;

b.) Be educated about how to correctly and quickly stabilize, and then to manage, envenomations;

c.) Understand various diagnostic modalities for envenomations; and

d.) Understand both likely and rare complications that may occur.

Answer Key

1. B	5. C	9. D
2. C	6. B	10. C
3. B	7. A	
4. E	8. E	

In Future Issues: **Pediatric C-Spine**