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Method of Participation. This educational activity consists of two lessons with a posttest and should take approximately 5 hours to complete. To complete this educational activity as designed, the participant should, in order, review the learning objectives, read the lessons, and complete the online posttest. Release date July 1, 2010. Expiration date June 30, 2013.

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Target Audience. This educational activity has been developed for emergency physicians.

American College of Emergency Physicians®
ADVANCING EMERGENCY CARE

Lesson 21

Snakebites

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Objectives

On completion of this lesson, you should be able to:

1. Describe the venomous snakes indigenous to the United States.
2. Explain the difference between a dry snakebite and an envenomation.
3. Discuss the local and systemic effects of snake venom.
4. Describe the optimal treatment for a snake envenomation.
5. List treatment resources for snake envenomations available in your community.

From the EM Model

6.0 Environmental Disorders

6.1 Bites and Envenomation

Snakes have long been feared and their bites sensationalized as deadly. Although remedies for snakebites have been recorded in the texts of several ancient civilizations, it has only been in the past century with the development of antivenins that the treatment has progressed beyond those ancient remedies. In reality, snakebites are not very common, nor are they very deadly. In the United States, it is estimated that 45,000 snakebites occur each year of which 8,000 are from venomous snakes.[1] In the past decade, fewer than 10 deaths per year have been attributed to snake envenomations. Worldwide there are an estimated 4.5 to 5 million snakebites each year, causing 125,000 deaths.[2] Of the 120 known indigenous snake species in North America, about 20 are venomous to humans. Although venomous snakebites are not common, they can inflict significant tissue damage to extremities and cause systemic abnormalities. Emergency physicians can effectively reduce the dangers of venomous snakebites with rapid recognition and treatment of any suspicious-appearing bite.

Case Presentations

Case One

A 25-year-old man is brought in by his two friends for a "bleeding arm." His friends report that they were on a hunting trip in southern California, drinking and "having a good time," when the patient went into the woods to urinate. He returned yelling that he scratched his arm on a bush. However, they are unsure what happened, and the patient says he can't remember. The patient denies any past medical or surgical history. He smokes and drinks on occasion and is an avid hunter.

Physical examination reveals a well-nourished man lying on a bed, clutching his arm. He appears intoxicated but is alert and awake. Vital signs are blood pressure 140/90, pulse rate 115, respiratory rate 18, and oral temperature 36.7°C (98.1°F). He has no visible signs of trauma to his head, abdomen, or chest. Breath sounds are clear. Heart sounds are tachycardic but normal. There are two puncture wounds with swelling and erythema but no active bleeding on the lower part of his left arm. When asked specifically if he was bitten by a snake, he admits to sticking his hand into a bush to see if the animal he saw was indeed a snake. On further questioning, the patient states that it was dark at the time and he did not see the snake well but did hear a rattling sound.

Case Two

A 28-year-old woman presents to a Florida hospital complaining that she "got bit." According to the nurse, the wound is small with good hemostasis. The patient states she was bitten on the foot while walking through a dark swampy area. She felt that whatever bit her held on for several seconds. When she kicked her leg violently to shake off the animal, she saw a red, black, and yellow snake slither away. Currently, she complains of intense

Critical Decisions

- What is the optimal prehospital treatment of snakebites?
- How should a venomous snakebite be assessed in the emergency department?
- When should antivenin be given to treat a pit viper bite?
- What are the potential complications of FabAV administration?
- Is there a role for surgical intervention in venomous snakebites?
- Is antivenin safe for use in children and pregnant women?
- What features of an envenomation suggest a coral snake or nonnative snake envenomation?
- What is the appropriate disposition for patients with snakebites?

paresthesias in her foot and of feeling nauseated and restless.

On physical examination, you see a moderately agitated young woman staring at the bite on her left foot. Her vital signs are blood pressure 110/80, pulse rate 110, respiratory rate 18, and temperature 37.2°C (98.9°F). On her lower leg are two small puncture wounds about 1 cm apart, with no active bleeding. When the wound is squeezed, blood can be expressed. There is minimal surrounding edema or erythema. Her pulses are strong in the extremities, and she has full strength to flexion and extension. She has decreased sensation to light touch and pin prick in the left foot extending to her ankle.

■ Case Three

An Arizona hospital is notified that a helicopter transporting a man in critical condition with a snakebite is on route from a nearby national park. The patient had been hiking with friends when he was bitten on the thigh by a large greenish-brown snake with a triangular head. The group was 1 hour from the trailhead, so they wrapped and immobilized the leg and waited for EMS. When EMS arrived, the patient was awake and oriented but was complaining of nausea, a metallic taste in his mouth, and peri-oral anesthesia. Swelling and erythema of the limb had progressed 10 cm from the original site of the bite.

The emergency physician prepares the trauma bay, crash cart, and airway cart and calls the hospital pharmacist and instructs her to begin preparation of the Crotalidae polyvalent immune Fab (ovine) (FabAV). When the patient arrives, he is ill appearing. An assessment of his ABCs reveals that he is hypotensive with thready pulses. Intravenous fluids are started, and blood is drawn for a CBC, coagulation profiles, and chemistries. The trauma survey reveals no injuries except for two puncture wounds 3 cm apart on his right thigh. More noticeable is the progression of the surrounding edema and inflammation, which now extend up to the groin and down to mid-calf.

Snakebite victims often present to emergency departments, and it is the job of an emergency physician to recognize the severity of the bite and provide appropriate and timely treatment. Although snakes are mostly native to the Southern and Southwestern United States, snakebites have been reported throughout the United States. With the increase in travel and the adoption of exotic pets, nonnative snake envenomations are a growing concern.[1,3] With the advent of antivenins, emergency physicians are equipped with treatments to effectively reduce the morbidity and mortality of these patients. Rapid recognition of a snakebite, accurate differentiation between a venomous bite and a nonvenomous bite, and prompt initiation of antivenin are the goals of the emergency physician.

Epidemiology

Approximately 8,000 people a year are reportedly bitten by venomous snakes, and fewer than 10 envenomations result in death. Thirty to forty percent of the snakebites are deemed "dry," with no venom injected into the victim even though the snake has bitten through the skin. The Poison Control Center estimates that 1,700 people received antivenin in 2007.[3]

The vast majority of victims are men in their second to fourth decades of life, and many cases involve alcohol use. This same group also experiences the most snakebite fatalities.[4] Most envenomations occur in the summer months, when snakes are more active and people are often outdoors.

Venomous Snakes

There are five families of venomous snakes: Colubridae, Hydrophiidae, Viperidae, Elapidae, and Crotalidae. The last two are indigenous to the United States. The Crotalidae include rattlesnakes, cottonmouths, and copperheads (Figure 1). At least one species has been identified in every state except Maine, Alaska, and Hawaii. The Elapidae family includes coral snakes and other, nonnative snakes such as mambas, cobras, and kraits.[1,3,5]

The Crotalidae, or pit vipers, are named for the unique heat-sensing pit organs located between the eye and the nostril on either side of the head. These snakes characteristically have fangs, elliptical pupils, and triangular-shaped heads. Nonpoisonous snakes usually have round heads and pupils. Rattlesnake and cottonmouth bites are more serious than copperhead bites because of their highly toxic venom and more aggressive behavior. Envenomations by pit vipers account for 99% of all venomous snakebites in the United States.[1,5]

Figure 1.
Three venomous snakes of the Crotalidae family indigenous to the United States. Images courtesy of Lynn Tunmer, Art Director at the Philadelphia Zoo, Philadelphia, Pennsylvania. (For full-color images, see the online version of this month's issue.)
A. Western diamondback rattlesnake *(Crotalus atrox)*
B. Eastern cottonmouth *(Agkistrodon piscivorus piscivorus)*
C. Northern copperhead *(Agkistrodon contortrix mokasen)*

Rattlesnake venom has two primary components that inflict tissue injury. Specific amino acids in the venom damage the endothelial cells of blood vessels causing increased vascular permeability, hemoconcentration, and third spacing, and in smaller prey leading to pulmonary edema and hypovolemic shock. Secondly, digestive enzymes in the venom cause muscle necrosis and consumption of platelets and fibrinogen. This is demonstrated by an abnormal coagulation panel on laboratory work similar to that of disseminated intravascular coagulation.[1]

Coral snakes are found primarily in the Southern United States and are easily identified by their colored bands. Both coral snakes and the nonvenomous kingsnakes (*Lampropeltis* genus) have the same colored bands. A rhyme can be used to remember whether the snake is venomous; it goes as follows: "red next to yellow, kill a fellow; red next to black, venom lack." Unlike the pit vipers, which strike with a single bite, coral snakes inject venom using a chewing mechanism. It is often reported that coral snakes will hang onto their victims for many seconds until shaken or kicked off.[5] The coral snakes endemic to the United States are the Texas coral snake, the Arizona coral snake, and the Eastern coral snake. The Eastern coral snake is found in the southeastern part of the United States and is considered the most venomous of the three.[6]

Coral snake venom is primarily neurotoxic, blocking acetylcholine receptor sites and inhibiting normal function of skeletal and cardiac muscle. Numbness and paresthesias are also typical effects. Ptosis is commonly the first sign of envenomation and can progress to multiple cranial nerve palsies, respiratory paralysis, and death.[5,6]

CRITICAL DECISION

What is the optimal prehospital treatment of snakebites?

There has been a long history of treating snakebites in the prehospital setting with tourniquets, suction, and incision and drainage. Currently, none of these methods is considered effective, and furthermore, they could cause additional harm. Several case reports recount how victims lost function or required amputation of their affected extremity after a tight tourniquet had been placed. Incision and drainage as well as suction techniques are typically not performed under sterile procedures in the prehospital setting and increase the risk of infection and worsen tissue destruction. The current recommendations are to immobilize the area of the snakebite and to outline the area of erythema and swelling in order to facilitate the assessment of symptom progression. There have also been reports on the benefits, especially in coral snake envenomations, of a wide compression band used to compress lymphatic and venous blood flow while maintaining arterial flow.[7]

It is important to stress to all patients (by EMS or if on the phone) that they should not attempt to kill or capture the snake. There are numerous reports of patients receiving a second bite while trying to capture the snake for identification. Even a snake that is beheaded can have an intact bite reflex. A detailed description of the size, coloring, and type of snake can be sufficient to aid in decisions for treatment. A survey by Corbett et al found that a group of lay people interviewed in Southern California were able to correctly differentiate a venomous from a nonvenomous snake 81% of the time and could identify a rattlesnake 95% of the time.[8] Although it might be helpful to identify the snake, in reality, most snakebites are treated based on clinical presentation and progression of symptoms.

When any patient comes in with a possible snakebite, it is imperative to make an initial assessment followed by a detailed history and physical examination. Frequent reassessment is key to guiding appropriate therapy. Snakebite presentations can range from a stable patient with a "dry" bite to an unstable patient in need of active cardiovascular and respiratory resuscitation. It is important for all emergency physicians to realize that the initially stable-appearing patient can quickly turn unstable. Even patients with the most benign-appearing envenomations should be closely monitored for several hours to watch for progression of the envenomation.

CRITICAL DECISION

How should a venomous snakebite be assessed in the emergency department?

Airway, breathing, and circulation should be evaluated in all patients, and a full set of vital signs should be recorded. Then a detailed history should be elicited, including time of the bite, how the bite occurred, and a comprehensive description of the snake. The presence of fangs, pits, or a rattle, the coloring, and the size of the animal can help guide treatment. It has been suggested that larger snakes deliver more venom with each strike.[9] The patient should be asked about any systemic symptoms (nausea, difficulty breathing, weakness, vision changes, etc.) and also about any local symptoms from the bite such as pain, swelling, and paresthesias. The area of the bite should be closely examined for fang marks, edema, erythema, ecchymosis, and teeth or debris left in the wound. Some authors have recommended gently squeezing the bite to express blood as an indication that the bite penetrated the dermis.[10] At this time, an outline should be drawn around the surrounding edema and time stamped to aid in assessing progression of local symptoms. Note any allergies to medications or to sheep or horse serum products.

Laboratory work for these patients should include a CBC, chemistries, and coagulation profile. An ECG and radiographs should be considered based on the patient's medical history. Tetanus immunization should be provided. Local wound care should be initiated. Intravenous fluids should be infused, and medications for symptom relief may be given. Other treatment considerations are based on the severity of the envenomation and whether the patient will need antivenin.

CRITICAL DECISION

When should antivenin be given to treat a pit viper bite?

Some experts advise the use of a grading scale, although none has been validated, to assist in the decision to use antivenin. One such commonly used grading scale is as follows:

- Grade 0 (minimal): Suspected snakebite with no evidence of envenomation. Very minimal local symptoms and no systemic manifestations or laboratory abnormalities in the first 12 hours.
- Grade I (minimal): Fang wound is present, and local wound inflammation is 1 to 5 inches. No evidence of systemic involvement in 12 hours.
- Grade II (moderate): Widely distributed pain, spreading edema toward the trunk, petechiae and ecchymoses limited to the area of bite. Laboratory abnormalities may be present.
- Grade III (severe): Within 12 hours there are systemic manifestations, cardiovascular abnormalities, elevated fibrin degradation, increased bleeding time, and renal or hepatic abnormalities. Significant and progressing local wound inflammation.
- Grade IV (severe): Same symptoms as Grade III but more rapidly progressive on the order of minutes to hours. There are numerous physical and laboratory abnormalities, including muscle fasciculations and necrosis, convulsions, cardiovascular collapse, and even coma.[3,5]

All Grade II and higher (moderate to severe) envenomations should be

treated with antivenin. Currently there is only one type of antivenin for pit viper envenomations available in the United States. FabAV is made with purified antibody fragments obtained from sheep immunized with four crotaline venoms. Recent studies have demonstrated its greater effectiveness for neutralizing pit viper envenomations while maintaining a better safety and side effect profile than its predecessor, Antivenin polyvalent, a product derived from horse serum.[11,12]

According to the manufacturer's instructions, each vial of FabAV should be reconstituted in 10 mL of sterile water and swirled until a solution is formed. Contents of the reconstituted vials are then further diluted into one 250-mL bag of 0.9% normal saline. Reconstitution times can take 30 to 60 minutes. Each dose must be used within 4 hours.[13] Quan et al report improved reconstitution times by using a technique of continuous and gentle hand rolling of the vial. This resulted in less foaming and in reconstitution times of less than 5 minutes using 10 mL of sterile water and less than 2 minutes using 25 mL of sterile water (a departure from the manufacturer's instructions).[14]

Dosing for FabAV depends on the patient's clinical picture and progression of the disease; however, based on clinical experience, the recommended initial dose is 4 to 6 vials. The infusion should proceed very slowly over the first 10 minutes at rates of 25 to 50 mL/hour with close monitoring for any adverse reaction. The rate should then be increased to 250 mL/hour until completion. If complete arrest of local manifestations and systemic symptoms is not achieved, a repeat dose of 4 to 6 vials should be administered until control is established. Additional 2-vial doses can be given as needed based on the patient's clinical course (PRN regimen) or by scheduled dosing every 6 hours for 18 hours (3 additional doses). In a study by Dart et al comparing the PRN regimen versus schedule dosing, there were no differences in outcome, but the authors caution that half of the patients in the PRN group required additional doses.[11]

CRITICAL DECISION

What are the potential complications of FabAV administration?

Patients can develop an immediate hypersensitivity reaction during infusion of FabAV. These symptoms can range from urticaria and pruritus to airway compromise, hypotension, and frank anaphylaxis. The incidence of anaphylaxis with FabAV is rare with only case reports of this occurrence.[15,16] Mild (rash only) to moderate reactions (rash and wheezing) were reported in 6 of 42 patients (14.3%) in the initial postmarketing trials.[11,17] More recently, studies have reported that the incidence of acute hypersensitivity reactions is between 0 and 5%.[18-20] Even so, it is prudent to have medications such as antihistamines and epinephrine readily available prior to administering the FabAV.

Serum sickness, a delayed type III hypersensitivity reaction, can emerge several days after antivenin administration. Typical symptoms are fever, rash, arthralgia, myalgia, and constitutional symptoms. Up to 23% of patients experience serum sickness.[17] Patients should be educated about these symptoms prior to discharge so they can obtain medications if symptoms appear. Antihistamines and corticosteroids are effective treatments.

During the initial studies of FabAV and in postmarketing clinical experience, some patients receiving antivenin have experienced recurrent coagulopathy. These abnormalities are demonstrated by thrombocytopenia, hypofibrinogenemia, prolongation of prothrombin time, and elevated levels of fibrin split products. This recurrence of coagulopathy was noted on blood work 2 to 5 days following FabAV use, and the abnormalities can persist as long as 2 weeks before normalization. The pathophysiology underlying this phenomenon is unknown, and some experts even question its clinical significance because only rarely do patients have any bleeding complications.[21] The coagulopathy also seems to be resistant to additional doses of FabAV, and additional dosing is not currently recommended.[11,18]

CRITICAL DECISION

Is there a role for surgical intervention in venomous snakebites?

Although there are no randomized controlled clinical trials of surgical treatment versus antivenin use for the treatment of snake envenomations, case series and animal studies have demonstrated an extremely limited surgical role. Incision therapy and excision therapy have not been shown to improve outcome, and studies have shown increased rates of local complications with these surgical techniques. Fasciotomy for envenomations has also become a rare occurrence in the past decade. In a series of seven studies involving 1,257 patients, only two patients were treated with fasciotomy.[22] The available animal and patient data suggest that fasciotomy is unlikely to improve patient survival and functional outcomes. It is postulated that the clinical signs and symptoms of compartment syndrome are mimicked by the toxic effects of snake venom. Experts recommend treatment with more FabAV for the venom-induced myonecrosis instead of surgical fasciotomy.[23] Furthermore, when compartment pressures are actually measured in patients with snake envenomations, they are rarely elevated despite a clinical picture suggesting compartment syndrome. Because compartment pressures cannot be measured in fingers, one study recommends surgical dermotomy (a longitudinal incision through the skin on the medial or lateral aspect of the digit) when a patient presents with an

envenomated, tense, pale, or cyanotic digit.[22]

CRITICAL DECISION

Is antivenin safe for use in children and pregnant women?

There are several small descriptive studies on the use of FabAV in the pediatric population envenomated by pit vipers. FabAV appears to be an effective and safe treatment for children in halting the local effects and improving the coagulopathic effects of venom. In the two largest case series, there were no cases of anaphylaxis or of surgical intervention.[19,24] FabAV was administered in the standard adult doses in both series with good outcomes. Although there are no randomized controlled trials studying the optimal pediatric dosing regimen, some experts caution against using a weight-based (mg/kg) dosing regimen for children. They argue that because the mechanism of FabAV is to bind and neutralize the venom proteins, the effective dose of FabAV is determined by the molar dose of venom protein injected, not the size of the victim.[25] Thus, the standard dosing regimen should apply to both adults and children.

The FDA assignment of FabAV to pregnancy category C means that animal reproduction studies have shown an adverse effect on the fetus and there are no adequate and well-controlled studies in humans, but potential benefits may warrant use of the drug in pregnant women despite potential risks. One such potential risk is that of neuro-developmental problems from the mercury contained in the thimerosal preservative of the FabAV. Each vial contains no more than 104.5 mcg of mercury. Although there are legitimate concerns for fetal mercury toxicity, experts have maintained that FabAV should be administered when clinically indicated for treatment of the mother, as the best chance to ensure fetal survival is to ensure the health and survival of the mother.[26] Furthermore, poor fetal outcome and increased rates of miscarriage have been associated with severity of the envenomation. A detailed discussion of the potential risks and benefits should be initiated with the patient and family to obtain full informed consent prior to antivenin administration.

CRITICAL DECISION

What features of an envenomation suggest a coral snake or nonnative snake envenomation?

The Texas coral snake, Arizona coral snake, and Eastern coral snake are indigenous to the southern parts of the United States. Fewer than 25 envenomations by coral snakes are reported each year. Coral snakebites have minimal local findings and thus the envenomation is less obvious than those of pit vipers; but they can cause significant morbidity and mortality (approximately 10% to 20%) if untreated.[27] For this reason, there should be a high clinical suspicion for coral snakebite in patients with an unidentified bite in areas where these snakes are endemic.

Because coral snake fangs are short and fixed, coral snakes inject their venom by a repeated chewing action and must hang on to their victims for a period of time for a significant envenomation. Bites typically occur on fingers and toes due to the relatively small gape of the snake. Coral snake venom is primarily neurotoxic, causing a nondepolarizing blockade at the neuromuscular junction by binding competitively to the acetylcholine receptor. Symptoms are often delayed in onset (up to 12 hours) and prolonged in effect, lasting weeks to months in severe envenomations. The earliest symptoms can be euphoria, drowsiness, nausea, and paresthesias. Bulbar paralysis typically occurs before peripheral muscle weakness and respiratory failure. Ventilatory support can be necessary in some cases.

North American coral snake antivenin (Wyeth) is the mainstay of treatment.[28] Because it is derived from horse serum, cautionary and preventive measures should be taken for acute hypersensitivity reactions. For a presumed coral snake envenomation, antivenin should be administered early for best effect, even prior to the development of symptoms.[10]

With the increasing popularity of herpetoculture, there are now more than 50 reported snakebites from nonnative snakes per year, and these numbers are increasing. Most of these bites occur at private residences, and presentation is delayed. Because of the barriers in antivenin determination and acquisition, these victims have worse clinical outcomes and a higher case-fatality rate than victims of native snakebites.[29]

For all snake envenomations (especially for coral snakebites and nonnative snakebites), seek expert consultation from the regional poison control center (American Association of Poison Control Centers [AAPCC] 1-800-222-1222) to help guide therapy and to locate antivenins. The Online Antivenin Index, a recent joint venture by the AAPCC and the American Zoo and Aquarium Association (301-562-0777), can help to locate antivenin for nonnative snakebites. If an appropriate antivenin is available at a United States zoo, a request for compassionate release can be made, and the antivenin will be transported to the patient's location.[5,29]

CRITICAL DECISION

What is the appropriate disposition for patients with snakebites?

Grade 0 or I (Mild Envenomations)

Patients with minimal local edema and no systemic or laboratory manifestations of envenomations can usually be safely discharged after several hours (experts recommend 8 to 12 hours) of monitoring and reassessment in the emergency department. All patients should be discharged with analgesics, instructions on local wound care, and return precautions for worsening

Pearls

- In cases of snakebite, patients and EMS should be educated not to try to kill or capture the snake; the danger of being bitten is significant, and identification can often be made from a description of the snake.
- If a patient is showing local signs of envenomation, unless there is specific information to the contrary, it is prudent to assume a pit viper (Crotalid) envenomation; 99% of venomous snakebites in the United States are from pit vipers.
- Mark the leading edge of edema and erythema to track the progression of the envenomation.
- Use gentle continuous hand rolling of the FabAV vials for fastest reconstitution.
- Start the FabAV infusion at a slower rate and monitor for acute hypersensitivity reactions. Have diphenhydramine and epinephrine readily available at the bedside.
- The local poison control center should be contacted to help guide treatment decisions for snake envenomations (American Association of Poison Control Centers: 1-800-222-1222).

Pitfalls

- Failing to recognize that puncture wounds on the extremities could represent a snakebite.
- Using a tight tourniquet, sucking out the venom, or incising and draining a snakebite in the prehospital setting.
- Handling a "dead" snake that the patient brought in for identification—the snake might not be dead and could bite someone else.
- Assuming a snake envenomation is mild because the patient shows minimal symptoms on presentation.
- Under-dosing a child with a venomous snakebite because of weight-based dosing; experts recommend standard adult dosing for children with snake envenomations.

appearance of the wound, which could indicate progressive toxicity or superinfection. Wound infection rates following envenomation are less than 3%, and antibiotic prophylaxis is usually not recommended.[30]

Grade II to IV (Moderate or Severe Envenomations)

Any patient who receives antivenin should be admitted to the hospital. Consideration for ICU admission should be given to patients in whom the local effects of venom are progressing and who require frequent reassessments. Some experts recommend that any patient receiving antivenin should be watched in the ICU. Prior to discharge, all patients should be counseled on avoiding snakebites. Patients can require physical therapy to recover full function of the affected limb.

Case Resolutions

■ Case One

In the case of the young man who was bitten on the arm during a hunting trip, given that this incident occurred in southern California and in light of the patient's description of the snake, it is most likely that the patient was bitten by a rattlesnake. The patient was placed on a cardiac monitor and given an intravenous bolus of normal saline. Blood was drawn for laboratory evaluation, and the emergency physician told the patient that he needed to stay in the department for further evaluation.

An hour later, the patient's area of edema and erythema had spread 10 cm past the initial outline. He also continued to complain of increasing pain and nausea. Although his laboratory studies showed no coagulopathy, the progression of local tissue toxicity signified a moderate envenomation. FabAV was ordered from the hospital pharmacy, and the nurse started mixing the solution. The patient was informed of his clinical situation and admitted to the hospital. Two days later, his primary care doctor informed you that the patient responded very well to the initial bolus dose and required two additional doses during the hospitalization. He recovered fully and had no adverse outcomes.

■ Case Two

In consultation with the local poison control center, the emergency physician determined that the Florida woman with the bite to her foot most likely had been bitten by a coral snake, which is indigenous to the area. The toxicologist advised treating with coral snake antivenin despite the absence of any hard neurologic findings. The antivenin was obtained from the hospital pharmacy, mixed to the manufacturer's specifications, and administered to the patient. She was admitted to the ICU for frequent neurologic checks.

In the ICU, the patient developed cranial nerve palsies and respiratory distress and was intubated. She was successfully extubated on day 5 and eventually discharged to a short-term rehabilitation center.

■ Case Three

In the case of the Arizona hiker brought to the emergency department by EMS, the emergency physician thought it likely that the patient had been bitten by either a large Western diamondback rattlesnake or a large Mojave rattlesnake. Six vials of FabAV were infused immediately, and the patient was watched closely for any signs of improvement. Instead, the patient appeared to be deteriorating and became short of breath and reported increasing pain in the extremity. On reexamination, he had significant wheezing and use of accessory muscles. No oropharyngeal swelling or rash was appreciated. His systolic blood pressure remained in the 90s. Because of the respiratory findings, the patient was given diphenhydramine, 50 mg IV, and epinephrine, 0.3 mg SQ, and the FabAV infusion was stopped for 15 minutes. He responded to both treatments, and the FabAV infusion was restarted at a slower infusion rate. Over the next hour, his airway remained tenuous, so he was

electively intubated and admitted to the ICU.

In the ICU, the patient had decreasing platelets, fibrinogen, and clotting factors on laboratory evaluation, and the local effects of the venom were still progressing. He received a second infusion of 6 vials of FabAV. The thigh continued to swell despite the first two infusions of FabAV. Compartment pressures in the leg were measured and were found to be only mildly elevated. Additional vials of FabAV were infused. The local symptoms improved on day 3 of his ICU stay, and over the course of the next few days the coagulopathy also improved. He was discharged into the care of his family after 10 days in the hospital.

Summary

Venomous snakebites, although relatively uncommon, require rapid recognition and aggressive treatment in the emergency department. All patients need frequent reassessment for progression of the venomous effects. Pit vipers account for 99% of venomous snakebites, and coral snakes and nonnative snakes account for the remaining 1%. Antivenin is the mainstay of treatment and should be given for all but the mildest envenomations. FabAV for pit viper envenomations is safe and effective for adults and children. The local poison control center is a valuable resource and can help to guide therapy.

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The LLSA Literature Review

"The LLSA Literature Review" summarizes articles from ABEM's "2011 Lifelong Learning and Self-Assessment Reading List." These articles are available online in the ACEP LLSA Resource Center (www.acep.org/llsa) and on the ABEM Web site.

Article 3

Metabolic Emergencies

Reviewed by Alison Ridpath, DMD, MPH, MD, and J. Stephen Bohan, MD, MS, FACEP; Harvard Affiliated Emergency Medicine Residency; Brigham and Women's Hospital

Kwon KT, Tsai VW. Metabolic emergencies. *Emerg Med Clin North Am.* 2007;25:1041-1060.

Hypoglycemia is a common metabolic problem, especially in neonates. Frequent causes include infection, adrenal insufficiency, inborn errors, and medication. Symptoms with rapid decline in blood glucose include tachycardia, tachypnea, vomiting, and diaphoresis; poor feeding, altered mental status, lethargy, and seizures are usually associated with slower or prolonged hypoglycemia. Bedside glucose testing should be performed on every pediatric patient who is seriously ill or altered. Early detection is important because permanent brain damage can begin shortly after symptoms develop. Glucagon can be given for refractory hypoglycemia.

Hyperglycemia is often seen in critically ill, non-diabetic patients and can signify an increased mortality risk. The greatest risk of hyperglycemia is dehydration from the urinary loss of glucose and osmotic diuresis. Treatment includes an isotonic solution bolus of 10 to 20 mL/kg given over 1 to 2 hours. The remaining fluid deficit can be restored in the next 24 to 48 hours. More than 50 mL/kg over the first 4 hours of treatment should not be given because of an increased risk of cerebral edema. Insulin therapy, without a bolus in pediatric patients, should begin after the initial fluid bolus. Close potassium monitoring and replacement is important. Bicarbonate is generally not recommended.

Clinical symptoms of hyponatremia, including altered mental status, lethargy, vomiting, diarrhea, seizures, and circulatory collapse, are usually not seen until the level falls below 120 mEq/L. Treatment with 3% hypertonic saline should only be initiated if significant symptoms (seizure and coma) are present. The goal is to raise the sodium level slowly at a rate of 0.5 mEq/L per hour.

Neonates and infants are more susceptible than older children to acidosis. Metabolic acidosis is classified as normal anion gap acidosis (gastroenteritis/diarrhea, renal tubular acidosis, adrenal insufficiency) or increased anion gap acidosis (MUDPILES, inborn errors of metabolism, starvation, chronic renal insufficiency).

Acute adrenal insufficiency is associated with hyponatremia, hyperkalemia, and hypoglycemia and with hypotension unresponsive to fluids. Treatment is aggressive fluid resuscitation and rapid stress doses of corticosteroids. Blood should be collected before treatment for nonemergent testing, if possible.

Highlights

- Check a bedside fingerstick glucose on all critically ill pediatric patients.
- Vomiting, change in mental status, and feeding difficulties are the most common features of metabolic diseases.
- Do not give more than 50 mL/kg of isotonic solution in the first 4 hours of treatment of diabetic ketoacidosis because of the risk of cerebral edema.
- Only treat hyponatremia with 3% hypertonic saline if significant symptoms are present.

Lesson 22

Upper Gastrointestinal Bleeding

Lauren Heath, MD, and Rahul Sharma, MD, MBA, FACEP

Objectives

On completion of this lesson, you should be able to:

1. Describe resuscitation and treatment strategies for patients presenting with acute upper gastrointestinal (GI) tract bleeding and hemorrhage.
2. Discuss the most common causes of upper GI bleeding and the respective latest therapeutic approaches.
3. List factors associated with increased rebleeding and mortality.
4. Discuss which patients should be admitted to the inpatient service and which patients can safely be discharged from the emergency department.

From the EM Model

1.0 Signs, Symptoms, and Presentations
1.2 Abdominal

Upper gastrointestinal (GI) bleeding has an overall incidence nearing 100 per 100,000 population, resulting in more than 300,000 hospitalizations.[1] Upper GI bleeding is bleeding that originates proximal to the ligament of Treitz (suspensory muscle of the duodenum). Traditionally, it is categorized as being either variceal or nonvariceal. Nonvariceal causes of bleeding include peptic ulcer disease (50%), erosive gastritis and esophagitis (25%), Mallory-Weiss syndrome (5% to 15%), vascular ectasias (5% to 10%), Dieulafoy lesions (<5%), and malignancies (<5%). Less common, although important to consider, are ear, nose, and throat sources of bleeding that can appear to originate from the GI tract.[1]

Upper GI bleeding is more common in older patients, with one study finding a mean age of 66 years; it is more prevalent in men.[2]

Upper GI bleeding can vary in presentation, but most cases present in one or more of four ways as follows: melena, hematemesis, coffee-ground emesis, and hematochezia (Table 1). Although variceal bleeding most commonly presents as massive upper GI bleeding, the Canadian Registry on Nonvariceal Upper Gastrointestinal Bleeding and Endoscopy found that 69% of nonvariceal upper GI bleeding presents with melena, 30% with hematemesis, 28% with coffee-ground emesis, and 15% with hematochezia.[3]

Case Presentations

Case One

A 55-year-old homeless man with a history of alcohol dependence presents because he has been vomiting blood. At triage, the patient's chief complaint is mild epigastric pain with nausea. He says that he has been drinking heavily for the past 2 days and started vomiting and having epigastric pain about an hour ago. He denies chest pain, shortness of breath, fevers or chills, bright red blood per rectum, and dark-colored stools.

Physical examination reveals a man who smells of alcohol. His vital signs are blood pressure 80/50, pulse rate 120, respiratory rate 22, and temperature 38°C (100.4°F). Examination of his head and neck reveal some dry blood at the oropharynx and icteric sclera. The patient is able to speak full sentences and has a patent airway. His abdomen is distended; there is epigastric tenderness and caput medusa (distended and engorged umbilical veins across the surface of the abdomen). His extremities reveal 2+ pitting edema, and the neurologic examination is nonfocal.

The patient is placed on a cardiac monitor, and two large-bore intravenous lines are placed. An ECG is unremarkable, and an upright chest radiograph reveals no free air under the diaphragm and clear lungs. The patient is given an intravenous normal saline bolus and is typed and cross-matched for blood. Laboratory abnormalities include an elevated

Critical Decisions

- Which patients with upper gastrointestinal (GI) bleeding require placement of a nasogastric tube and gastric lavage?
- How important is the initial hemoglobin or hematocrit in the management of patients with upper GI bleeding?
- Which patients with upper GI bleeding would benefit from early proton pump inhibitor therapy?
- Which patients with upper GI bleeding require hospitalization?

WBC count of 18,000 cells/mm^3, hemoglobin 7 g/dL, ALT 200 units/L, AST 500 units/L, and an international normalized ratio (INR) of 4.2. After an initial 1-liter bolus of normal saline the patient's blood pressure is 90/55. The patient has another episode of bright red vomiting in the emergency department, and his blood pressure falls to 70/40.

■ Case Two

A 21-year-old college football player presents with a chief complaint of epigastric pain and one episode of "vomiting blood." The patient states that he has had a shoulder injury over the past year and has been taking aspirin and celecoxib. The patient states that 2 days prior to this presentation he was seen in the emergency department for an injury to his ankle and was given an intravenous injection of ketorolac and discharged with a prescription for naproxen. The patient denies any alcohol use. He denies any fevers, chills, lightheadedness, chest pain, or shortness of breath. The remainder of the review of systems is unremarkable.

Physical examination reveals a comfortable young man who does not appear toxic. Vital signs are blood pressure 105/70, pulse rate 100, respiratory rate 18, and temperature 36°C (98°F). He has a normal heart examination, and the lungs are clear. Abdominal examination shows some tenderness in the epigastric area with no rebound or guarding. Rectal examination reveals brown stool that is guaiac negative. The patient is placed on a monitor, and 2 large-bore intravenous lines are placed. His hemoglobin is 12 g/dL, and the remainder of his laboratory work is unremarkable.

■ Case Three

An 86-year-old woman with a history of atrial fibrillation and coronary heart disease presents because she has been feeling weak and she passed out earlier in the day. The patient states that she is on warfarin and had taken a few extra doses because she believed her INR was not therapeutic. The patient also reports having some dark stools over the past 5 days. She reports that she has been feeling weak and "passed out" when she got up this morning. The patient denies any other associated symptoms with this episode of "passing out." She reports some shortness of breath with exertion over the past few days but denies any chest pain, abdominal pain, fevers, chills, or any focal neurologic complaints.

Physical examination reveals a pleasant elderly woman who appears lethargic, with pale skin and conjunctiva. Her pulse is irregularly irregular, and there is a systolic murmur. Lungs are clear. Her abdomen is nontender, and there is no rebound or guarding. Rectal examination shows dark, melanotic stool that is guaiac positive. Neurologic examination is nonfocal. When the patient is asked to sit up she states that she feels extremely lightheaded.

Vital signs are blood pressure 100/60, pulse rate 105, respiratory rate 20, and temperature 37°C (98.6°F). On orthostatic evaluation, the patient's blood pressure drops to 80/55 and her pulse rate increases to 130. Laboratory studies reveal a hemoglobin of 6 g/dL and an INR of 4.2. The patient is placed on a cardiac monitor. The ECG reveals atrial fibrillation with no ischemic changes. Her chest radiograph is unremarkable. Two large intravenous lines are placed, and the patient is typed and cross-matched for blood products.

Causes of Upper Gastrointestinal Bleeding

Peptic Ulcer Disease and Gastritis

Gastritis is inflammation of the gastric mucosa and can be a precursor to peptic ulcer disease. Peptic ulcer disease includes gastric and duodenal ulcers, which have two main etiologies—*Helicobacter pylori* infection and nonsteroidal anti-inflammatory drug (NSAID) use. Additionally, gastrin-secreting tumors or Zollinger-Ellison syndrome are responsible for 1% of cases of peptic ulcer disease.

The stomach is made up of different types of cells, which have different functions and secretions. Mucous cells secrete acidic mucus, parietal cells secrete hydrochloric

Table 1.
Definitions

Melena: the passage of dark and pitchy stools stained with blood pigments or with altered blood
Hematochezia: the passage of bloody feces
Hematemesis: the vomiting of bright red blood
Coffee-ground emesis: emesis consisting of dark, altered blood mixed with stomach contents

acid and intrinsic factor, zymogenic (chief) cells secrete pepsinogens, and enterochromaffin-like cells secrete histamine and gastrin. Several mechanisms protect the gastric mucosa from autodigestion. Disruption to these mechanisms makes the gastric mucosa vulnerable to damage by allowing hydrogen and digestive enzymes access to the tissue. This can lead to inflammation, bleeding, and potential ulceration.

One of the major pathophysiologic causes of this barrier breakdown is *H. pylori* infection. This gram-negative rod grows between the epithelial cell surface and the overlying mucus in the stomach and duodenal gastric mucosa. It causes mucosal inflammation that disrupts the normal defensive barrier of the gastric mucosa and leads to ulceration. It also increases the risk of gastric carcinoma and lymphoma. It is estimated that 70% to 80% of patients with duodenal ulcer and 60% to 70% of patients with gastric ulcers are infected with *H. pylori*. It is more common in lower socio-economic populations and is probably spread by the feco-oral route. Although not yet relevant to emergency department care, diagnosis usually involves urea breath tests, blood tests, stool antigen testing, and mucosal biopsy. Eradication of *H. pylori* with antibiotic treatment results in a shorter recovery and often in a cure.[3]

The second most common cause of peptic ulcer disease is the use of NSAIDs. NSAIDs have both a direct and an indirect effect on the gastric mucosa. They are weak acids that remain non-ionized in the acidic environment of the stomach lumen. This allows diffusion into the mucosal cells, where they ionize and can no longer leave the cell, causing the NSAID molecule to be trapped inside the cell, thereby damaging the cell. This damage is most likely produced as the NSAID decreases prostaglandin secretion, leading to decreased mucus production and hence a vulnerable gastric mucosa. This effect places the gastric mucosa at risk for breakdown and ulceration. It is important to note that a history of ulcers, age older than 60 years, using higher doses of NSAIDs, and concurrent use of glucocorticoids put patients at even higher risk for gastritis and peptic ulcer disease than does NSAID use alone.[3] Furthermore, concomitant use of anticoagulants puts patients at a higher risk of bleeding[4] and makes treatment and cessation of bleeding more challenging.

Cyclooxygenase-2 (COX-2) inhibitors such as celecoxib can also cause peptic ulcer disease. COX-2 inhibitors became popular because they were associated with decreased endoscopic lesions and episodes of upper GI bleeding when compared with the nonselective NSAIDs. This is due to the decrease in prostaglandin suppression as compared to their nonselective counterparts.[1] Their use has been recently challenged in the medical community because of their adverse cardiovascular effects.

Other substances or conditions that can damage the gastric mucosal barrier and result in an increased risk for inflammation and ulcer formation include bile, cigarette smoking, ethanol, pancreatic secretions, shock (septic and hypovolemic), and any condition resulting in stress to the organ systems.

Esophageal Varices

Upper GI bleeding from variceal hemorrhage is a major complication of portal hypertension. Variceal hemorrhage can occur in 25% to 35% of patients with cirrhosis, and 80% to 90% of upper GI bleeding in cirrhotic patients is caused by variceal bleeding. Variceal bleeding is the most dangerous type of upper GI bleeding, resulting in higher morbidity and mortality rates and in higher hospital costs than other causes. Up to 30% of upper GI bleeding caused by varices is fatal, and as many as 70% of patients with nonfatal cases rebleed.

Cirrhosis is the most common cause of portal hypertension, which in turn leads to esophageal varices. These portosystemic collaterals are formed as resistance in the normal venous pathways within the liver is increased. The superficial veins in the distal 5 cm of the esophagus lack support from surrounding tissues and are particularly susceptible to the increased pressure. This increased pressure results in variceal formation.

Not all varices bleed. However, continued alcohol use by alcoholics with poor liver function, as evidenced by hepatic encephalopathy, hypoalbuminemia, ascites, hyperbilirubinemia, and prolonged prothrombin time, puts these patients at high risk for variceal bleeding. Endoscopically, large varices and red signs (eg, red whale markings) on the varices also indicate increased risk. Although these factors rarely affect emergency resuscitative care, they can affect further medical management aimed at preventing bleeding or re-bleeding.[5]

Mallory-Weiss Syndrome

Mallory-Weiss syndrome is bleeding secondary to a longitudinal mucosal tear in the esophagus, classically caused by retching or vomiting. Most cases of Mallory-Weiss tears are self-limiting, and endoscopic hemostasis is not needed.

Neoplasm

Neoplasms are not a major cause of upper GI bleeding but should always remain in the differential. Upper GI bleeding can be a presenting sign of any upper GI neoplasm. Primary malignancies include esophageal, gastric, or duodenal adenocarcinomas; esophageal squamous cell carcinoma; gastric or duodenal lymphomas; and GI stromal cell tumors. Metastases from such primary tumors as colon, lung, or breast cancer can also cause upper GI bleeding. Lastly, in HIV-positive patients, Kaposi sarcoma is one of the top two causes of upper GI bleeding.[6]

Initial Assessment

As with any patient presenting acutely, patients with upper GI bleeding should be resuscitated and then receive further evaluation and

treatment. The patient should initially be assessed for airway compromise and unstable vital signs. Large-bore peripheral intravenous lines should be placed with crystalloid at hand. The patient should be placed on a monitor. Laboratory tests, including a CBC, coagulation profile, and type and screen or cross-match, should be obtained. Type O negative blood should be immediately available and infused if needed. An ECG should also be obtained, especially in patients with a history of cardiovascular disease. Because bleeding can result in a decrease in oxygen-carrying capacity leading to cardiac ischemia, the patient should receive supplemental oxygen. Lastly, insertion of a Foley catheter should be considered to monitor urine output for continued assessment of organ perfusion.

On physical examination, it is important to note tachycardia; cool, clammy skin; and other signs of hypoperfusion and hemorrhage. Orthostatic blood pressures can be helpful. Check mucous membranes and neck veins for additional clues to volume status. Physical findings that suggest the patient could have baseline liver disease include spider angiomata, palmar erythema, jaundice, and gynecomastia. These findings, in the setting of upper GI bleeding, suggest a variceal source. A thorough ear, nose, and throat examination could turn the physician's attention to an occult nasopharyngeal bleeding source as the cause of hematemesis or melena. Assess for any abdominal tenderness, distention, masses, and organomegaly, and perform a rectal examination to detect blood, whether occult or overt in the form of melena or hematochezia. Hematochezia originating from an upper GI source has been associated with higher mortality rates and transfusion requirements.[7]

Following resuscitation, an emergency physician may consider inserting a nasogastric (NG) tube to help determine whether there is active bleeding. This can also confirm an upper GI etiology when the source of the bleeding is in question; however, a negative gastric aspirate does not conclusively exclude an upper GI source. Further, overtly bloody aspirates are generally correlated with high-risk lesions; these patients could quickly require aggressive resuscitation and could require more immediate gastroenterology consultation. An NG tube can also reduce the risk of aspiration by removing contents of the stomach, and this can aid visualization during an endoscopic procedure.

Concomitant with stabilization and resuscitation of the patient, a thorough history should be obtained. It will be important to know if the patient is taking any NSAIDs, antiplatelet agents, or anticoagulation therapies. The physician should ask about previous episodes of upper GI bleeding or documented peptic ulcer disease, *H. pylori* infection, and compliance with prescribed proton pump inhibitor medications. Age is important; older patients are more likely to have a neoplasm as the cause. Constitutional symptoms such as weight loss can also indicate a neoplastic process. Finally, any history of known liver disease or symptoms related to liver disease suggests a variceal hemorrhage.

Diagnostic Strategies

Nasogastric Aspirate and Lavage

CRITICAL DECISION

Which patients with upper GI bleeding require placement of an NG tube and gastric lavage?

An NG tube is an important part of the workup of a patient with upper GI bleeding. Although it might neither definitively confirm nor exclude an upper GI source of bleeding or even whether bleeding has ceased or is ongoing, it is helpful in risk stratification and can even be therapeutic. A retrospective study of 1,190 patients found that 93% of patients with a positive NG tube aspirate indeed had a bleeding site proximal to the ligament of Treitz, as confirmed later by endoscopic examination. Further, none of the same group had lower GI bleeding.[8] Other studies have shown that patients with a positive aspirate are more likely to have active bleeding on endoscopic examination, that those patients with a clear or negative aspirate had a mortality rate of 6%, and that those with a positive aspirate had a mortality rate of 17.6%.[9] There is no exact answer as to which patients need an NG tube and which patients do not. However, it would be good practice to place an NG tube in any patient with severe bleeding, a patient requiring aggressive resuscitation, and any patient that will need definitive airway management. Placement of an NG tube is considered by many as one of the most painful procedures that patients experience in the emergency department. If an NG tube is indicated, the emergency physician should use anesthetics such as nebulized or viscous lidocaine prior to insertion to decrease patient discomfort. If emergent emergency department endoscopy is planned, insertion of an NG tube may be deferred.

Endoscopy

Since the early 1980s, upper endoscopy has become both a diagnostic and a therapeutic tool and has been central in directing further patient care and management. Early endoscopy (within 24 hours of patient presentation) is, therefore, becoming standard in the evaluation of patients with upper GI bleeding. Because it can reveal the exact cause of bleeding, as well as the severity of bleeding, it can greatly reduce morbidity and mortality. In one retrospective study, the mortality rate from upper GI bleeding for patients without endoscopic evaluation was found to be more than twice the mortality rate of patients who had endoscopy (11.1% versus 5.2%).[10] Whether emergency department endoscopy further decreases morbidity, mortality, and

cost remains to be determined. However, it seems that as systems and protocols are put into place, emergency department endoscopy can become not only beneficial but a standard in the management of upper GI bleeding.

Angiography and Technetium Scanning

Angiography and technetium scanning can be useful alternatives when endoscopy has not yielded a definitive diagnosis. This will rarely be considered by an emergency physician, and the decision should be deferred to the GI or admitting medical service. Both of these modalities require a brisk bleeding rate in order to be effective. Angiography requires a bleeding rate of approximately 1 mL/minute, and technetium scanning requires a bleeding rate of approximately 0.5 mL/minute.

Risk Stratification

One of the major challenges of managing upper GI bleeding involves identifying which patients are at high risk for major morbidity or mortality and which patients are at low risk, so that their care in the emergency department can be efficiently managed. Several clinical scoring systems have been developed to address both of these issues. Most include a combination of clinical, laboratory, and endoscopic variables that are weighted to produce a score that can help predict risk of mortality, rebleeding, need for intervention (including surgery), and suitability for early discharge. The scoring systems that include clinical information without endoscopy results are often most helpful to emergency physicians, because early emergency department endoscopy is not always available.

The most commonly used scoring systems are the Rockall score, the Baylor bleeding score, the Cedars-Sinai Medical Center Predictive Index, and the Blatchford score. The differences between these systems can be reviewed in a paper by Peter and Dougherty and a paper by Das and Wong.[7,11] Several factors that have been consistently associated with poor outcome are shock; melena; anemia at presentation; significant fresh blood in vomit, NG aspirate, or rectum; concurrent sepsis; general poor health; liver, renal, and cardiac disease; large ulcer size; persistent bleeding despite endoscopic therapy; and recurrent bleeding.[12] Variceal bleeding is always high risk, and these patients require admission to an ICU setting.

Emergency physicians should immediately assess risk for all patients with upper GI bleeding, and all those at high risk should be admitted to the hospital. Some patients in the low-risk categories, on a case-by-case basis, may be discharged home with close followup by a gastroenterologist (Table 2).[6]

CRITICAL DECISION

How important is the initial hemoglobin or hematocrit in the management of patients with upper GI bleeding?

The Blatchford score is the only published scoring system that includes a hemoglobin or hematocrit as part of its total score. In a maximum score of 23 for highest risk, an initial hemoglobin below 10 receives 6 points; values of 12 or higher for women and 13 or higher for men receive 0 points, with other point levels in between. This internally validated study found utility in initial measurements of hemoglobin, although others have not used it.[11] Arguments can be made both for and against use of the initial CBC in risk stratification, but anemia in the setting of cardiac disease or severe anemia in any patient clearly cannot be ignored. Blood transfusion should be initiated. Often the initial hemoglobin or hematocrit does not reflect the severity of the bleeding. Therefore, frequent monitoring and serial hemoglobin or hematocrit levels are necessary. Emergency physicians should not be misled by a stable initial hemoglobin or hematocrit and should obtain urgent gastroenterology consultation as clinically indicated.

Treatment

As discussed above, initial treatment of upper GI bleeding involves beginning any resuscitative measures needed. Any compromise of airway requires definitive management. Patients should be placed on a cardiac monitor, and volume replacement with crystalloids should begin with two large-bore intravenous lines. If the patient is in hemorrhagic shock, blood transfusion should begin immediately, using type O negative blood if necessary. Because these patients often have comorbid conditions such as coronary artery disease, there is little room for worsening anemia and lowered oxygen-carrying capacity of the blood. These patients should be

Table 2.
Criteria for assessing risk in patients with upper gastrointestinal bleeding

Low-Risk Patients	High-Risk Patients
Age <60 years	Age >60 years
Stable vital signs	Unstable vitals
Systolic blood pressure >100 mm Hg	History of congestive heart failure, diabetes mellitus, cardiac disease
Pulse <100	On anticoagulants
No orthostatic vital sign changes	History of alcoholism
No comorbid conditions (congestive heart failure, chronic obstructive pulmonary disease, etc.)	Elevated BUN, partial thromboplastin time
No active hematemesis or melena	History of peptic ulcer disease
No anticoagulation	Altered mental status
	Actively vomiting and melena

transfused with blood early on in their resuscitation. An ECG should also be obtained in all GI bleeding patients to identify possible cardiac ischemia.

After or during hemodynamic stabilization, pharmacologic therapy should begin. Although most pharmacologic agents have not been shown to improve mortality rates, they have been shown to decrease rebleeding and improve endoscopic treatment outcomes.[6]

CRITICAL DECISION

Which patients with upper GI bleeding would benefit from early proton pump inhibitor therapy?

Proton pump inhibitors are currently the first-line treatment to decrease morbidity in patients with upper GI bleeding. After endoscopy, upper GI bleeding caused by peptic ulcer disease recurs in 15% to 20% of patients. Lau et al showed that high-dose omeprazole decreased rebleeding in patients with endoscopically treated peptic ulcer disease. Moreover, the duration of hospitalization was significantly shortened, and the need for blood transfusion was reduced in patients treated with a proton pump inhibitor.[13]

These findings were confirmed in a metaanalysis by Zed et al. In vitro studies have found that platelet aggregation, disaggregation, coagulation, and fibrinolysis are strongly dependent on intragastric pH. Therefore, any increase in gastric pH, as accomplished by a proton pump inhibitor, will aid in hemostasis of any bleeding lesion. In the analysis, there was a 50% reduction in rebleeding and a 53% reduction in need for surgery, but no significant reduction in overall mortality. It is important to note that this analysis included comparison of proton pump inhibitors not only to placebo but also to H_2-blockers such as cimetidine and famotidine. Earlier studies showed some benefit of H_2-blockers when compared to placebo, and others found no benefit. Hence, H_2-blockers have fallen out of favor, and proton pump inhibitor administration is the new standard.[14,15] Nearly all patients presenting to the emergency department with upper GI bleeding should receive proton pump inhibitor therapy.

Vasopressin causes contraction of the smooth muscle of the GI tract and parts of the vascular bed, hence its use in attempting to achieve hemostasis in the setting of upper GI bleeding. However, because vasopressin has not been shown to decrease mortality, the risks of systemic vasoconstriction, including myocardial and mesenteric ischemia, outweigh the benefits, and it is generally no longer used for upper GI bleeding.[6]

Somatostatin analogs, including octreotide, are pharmaceuticals that decrease splanchnic blood flow and inhibit the release of various GI "messengers." Multiple studies have proved that these analogs are more efficacious in controlling upper GI bleeding and are safer than vasopressin. Although not approved by the FDA for the control of upper GI bleeding, somatostatin seems to be as effective or more effective than sclerotherapy during endoscopy in the treatment of bleeding in cirrhotic patients.[6,12]

Endoscopy is perhaps the most important modality in the management of upper GI bleeding. It aids in diagnosis, treatment, and risk stratification and is useful both for ulcer lesions and for variceal bleeding. An endoscopist can evaluate the mucosa for any lesions, active bleeding, or lack thereof. If a lesion is found and is actively bleeding, epinephrine injection, band ligation, sclerotherapy, or fibrin glue can be employed. Also, a narrowed risk assessment can be made based on whether there is active bleeding,

Pearls

- In patients with a history of cirrhosis of the liver, upper gastrointestinal (GI) bleeding most likely is caused by esophageal varices.
- Involve a gastroenterologist early in the management of upper GI bleeding.
- If the patient is severely hemorrhaging, promptly consult both gastroenterology and surgery, as the patient may need surgical resection of the bleeding area.
- Instruct patients to discontinue drugs that can increase peptic ulcer disease and bleeding such as NSAIDs.
- Begin proton pump inhibitor therapy in every patient, including low-risk patients that are being discharged from the emergency department.
- Do not rely solely on the initial hemoglobin or hematocrit to measure the severity of upper GI bleeding.
- Begin somatostatin with any suspected variceal bleed.
- Severe abdominal pain with hematemesis may be a perforated ulcer; image and consult surgery immediately.

Pitfalls

- Mistaking nosebleeds, dental bleeding, tonsillar bleeding, and red foods and drinks for hematemesis.
- Mistaking bismuth-containing medications, activated charcoal, and iron supplements for melena.
- Forgetting that red meat, turnips, horseradish, and vitamin C can cause a false-positive in an occult blood test.
- Failing to recognize the very high morbidity and mortality rates associated with upper GI bleeding in patients with alcoholism and/or cirrhosis; treat accordingly and admit to the ICU.
- Overlooking cardiac ischemia in a patient with upper GI bleeding and new relative anemia. Obtain an ECG and have a low threshold for giving blood to patients with known cardiac disease.

adherent clots, nonbleeding vessels, or a clean base.

Balloon tamponade is an important tool to control variceal bleeding that is not responsive to somatostatin or endoscopic therapy. The inflated balloon of this specialized nasogastric tube applies direct pressure to the bleeding varix and establishes hemostasis in most cases. There is a significant risk of aspiration and perforation, so many physicians are uncomfortable with its use. It is important to consider definitive airway protection before attempting balloon tamponade. Remember that it is a rescue procedure only. Most varices will rebleed after the balloon is removed, so the balloon should only be removed in a controlled setting and with further therapy options at the bedside.[5]

Angiography, although largely replaced by endoscopy in upper GI bleeding, can be used to locate an elusive bleeding source and then embolize that bleeding vessel.

Surgery in upper GI bleeding has been mostly replaced by pharmacologic and endoscopic management options; however devascularization or surgical resections are still options in the most stubborn cases. A general surgeon should be available for consultation in case the bleeding is not controlled by conventional techniques.

Disposition

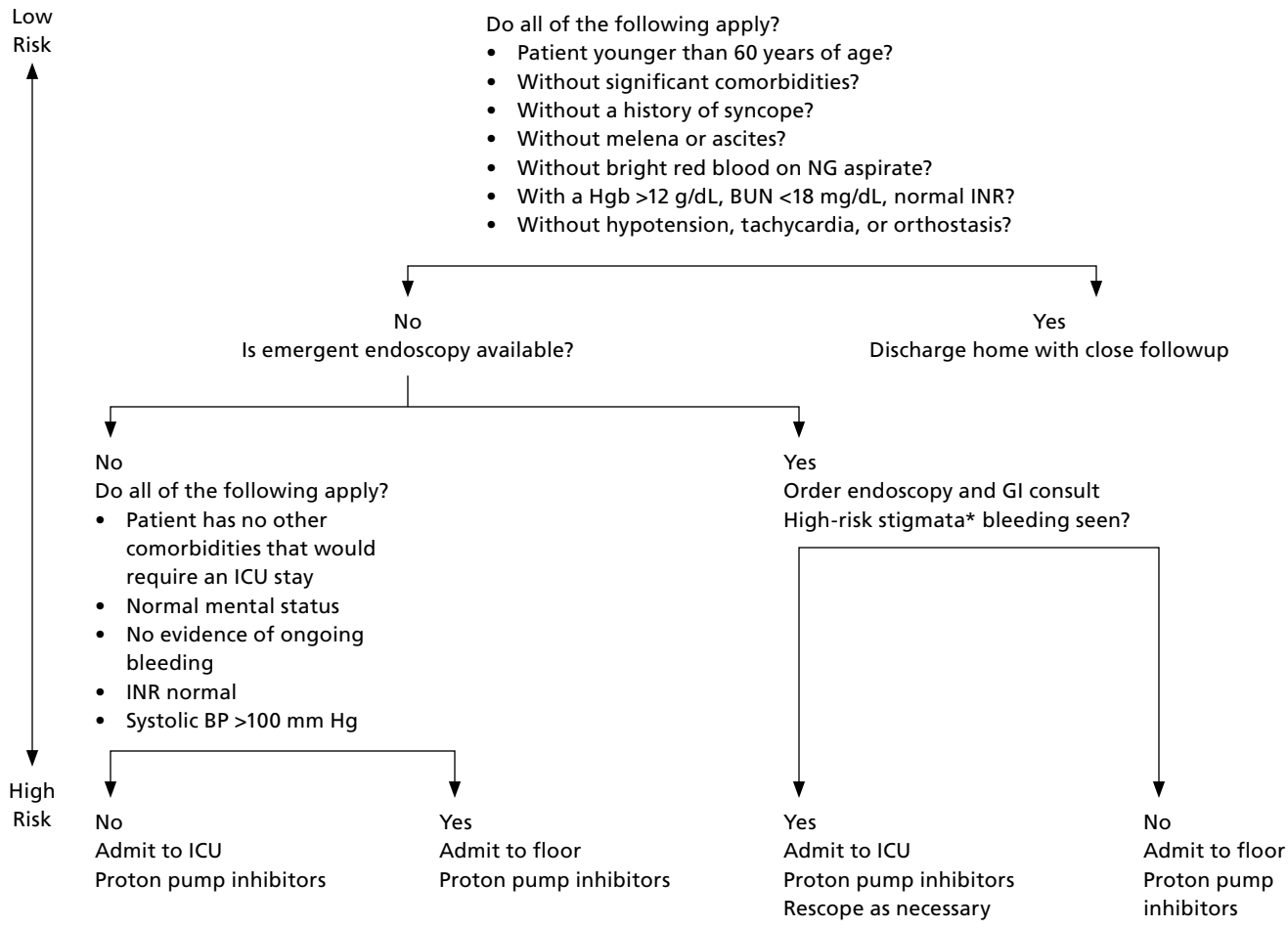
CRITICAL DECISION

Which patients with upper GI bleeding require hospitalization?

Patients who are deemed moderate or high risk for complications or rebleeding should be admitted to the hospital. The setting (ICU or medical floor) should be determined on a case-by-case basis (Figure 1).

It is possible to identify a group of low-risk patients who can be safely

Figure 1.
Disposition algorithm for patients with upper gastrointestinal bleeding. Adapted from: Westhoff JL, Holt KR. Gastrointestinal bleeding: an evidence-based ED approach to risk stratification. *Emerg Med Practice.* 2004;6(3):1. Copyright 2004. Used with permission.



*High-risk stigmata include bleeding vessels or ulcers, adherent clots, and varices.

discharged home after a period of observation.[16-18] Many patients with upper GI bleeding from a nonvariceal source may be managed initially with observation for up to 24 hours while receiving hydration and proton pump inhibitors. Their evaluation should include serial hemoglobin/hematocrit measurements, serial orthostatic vital signs, and gastroenterology consultation for early endoscopy. If the patient is considered to be low risk, with stable vital signs, no evidence of active bleeding, no symptoms, and no significant comorbidities, discharge home with a proton pump inhibitor and close GI followup may be considered.[16-18]

Case Resolutions

■ Case One

In the case of the homeless man who had been vomiting blood, his mental status deteriorated and he was intubated for airway protection. He received O negative blood, and an emergent gastroenterology consultation was obtained. The patient was started on a somatostatin infusion, and an emergent endoscopy was performed. The endoscopy revealed bleeding esophageal varices. Band ligation of the varices was performed, and the patient's bleeding resolved. The patient was transferred to the ICU for further management.

■ Case Two

In the case of the young man who had one episode of vomiting blood, his nasogastric lavage revealed no active bleeding. The patient was judged to be at a very low risk of rebleeding and was observed in the emergency department for 6 hours. Repeat hemoglobin testing showed no changes. The patient's symptoms were judged likely to be the result of his use of NSAIDS in the preceding week and, thus, benign. He was discharged home with a proton pump inhibitor, told to refrain from NSAID use, and scheduled to follow up with his gastroenterologist in the morning for further evaluation and endoscopy.

■ Case Three

In the case of the elderly woman on warfarin, the patient received a transfusion with packed RBCs and fresh frozen plasma. She also received vitamin K. She was transferred to the ICU, where she was evaluated by a gastroenterologist. Endoscopy revealed a bleeding gastric ulcer, which was injected with epinephrine and other sclerosing agents. The patient remained hemodynamically stable, and repeat laboratory values 4 hours later showed an INR of 4 with stable hemoglobin. She was discharged from the hospital on day 4 with no further complications.

Summary

Upper GI bleeding remains a significant cause of morbidity and mortality and involves costly management. It is important for emergency physicians to quickly assess, resuscitate, and provide the most appropriate disposition for these patients. Consider consultation with gastroenterology and surgical services early on in the management of these patients. Not all patients require admission; some low-risk patients may be discharged home with close followup. These decisions should be made in conjunction with a gastroenterologist to ensure close followup. Patients should be educated to refrain from tobacco smoking, alcohol use, and NSAID use in order to limit the complications from upper GI bleeding.

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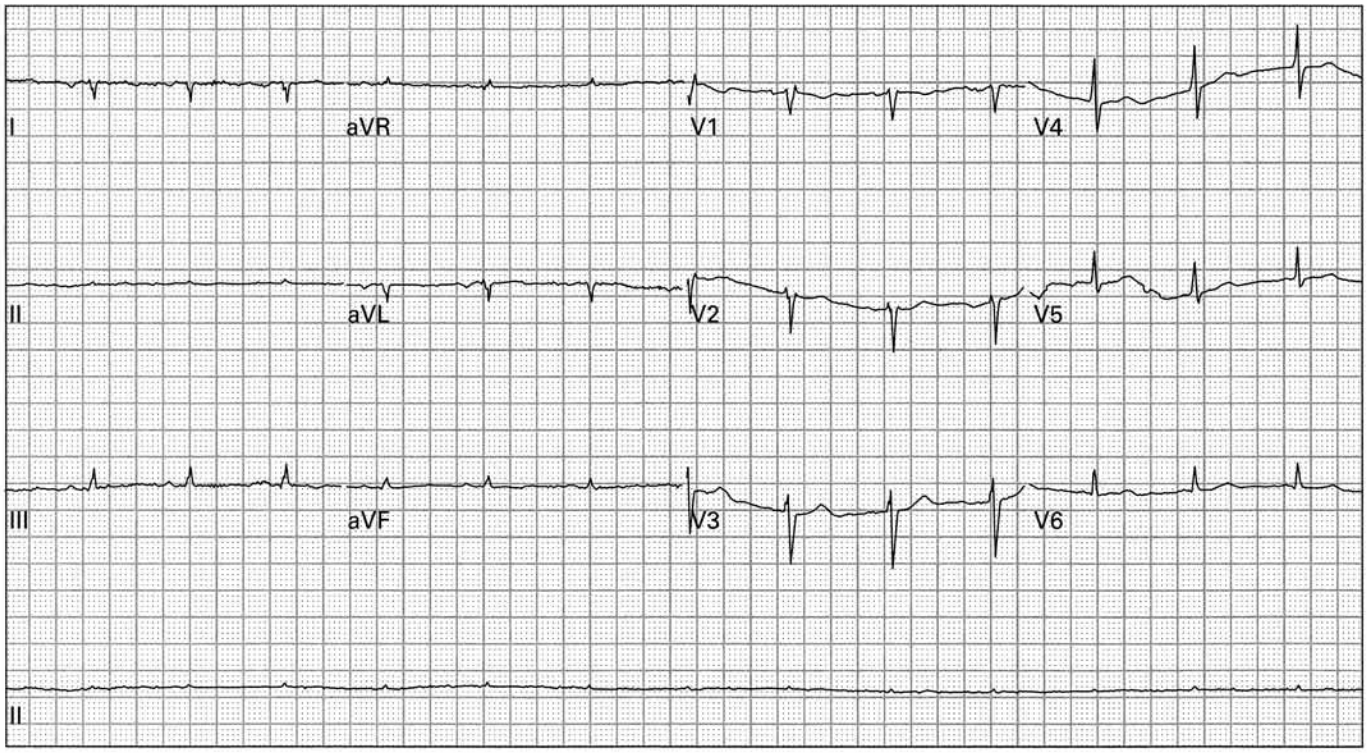
The Critical ECG

A 52-year-old man with lung cancer presenting with worsening dyspnea and orthopnea.



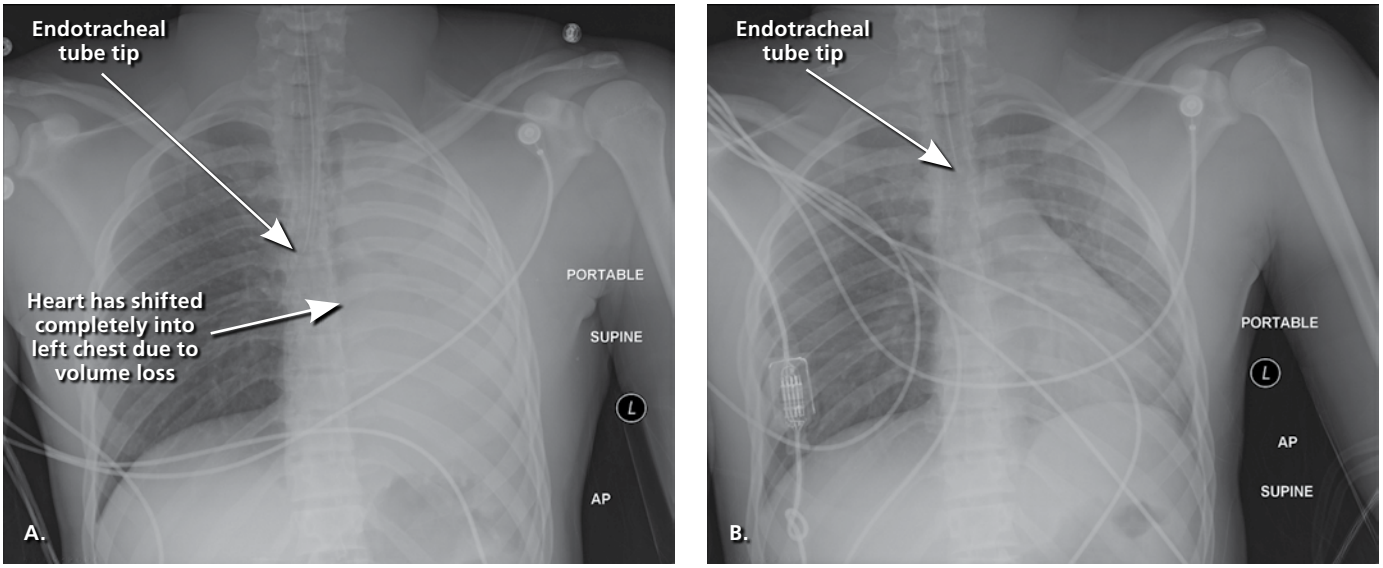
Sinus rhythm, rate 81, low voltage, left posterior fascicular block, nonspecific T-wave flattening in limb leads. The most notable finding on this ECG is the presence of low voltage, which was new in comparison to a previous ECG. Low voltage is generally defined as QRS amplitudes of less than 5 mm in all of the limb leads or QRS amplitudes of less than 10 mm in all of the precordial leads. The differential diagnosis for low voltage includes myxedema, large pericardial effusion, large pleural effusion, end-stage cardiomyopathy, severe chronic obstructive pulmonary disease, severe obesity, infiltrative myocardial diseases, constrictive pericarditis, and prior massive MI. In this case, the patient's low voltage was caused by a very large left-sided pleural effusion caused by lung cancer. The QRS amplitudes increased after a thoracentesis.

Feature Editor: Amal Mattu, MD, FACEP

From: Mattu A, Brady W. *ECGs for the Emergency Physician.* London: BMJ Publishing; 2003:106,142. Available at www.acep.org/bookstore. Reprinted with permission.

The Critical Image

A 17-year-old man presenting with generalized tonic-clonic seizure, continuing on emergency department arrival. He is intubated, and a chest x-ray is obtained.



The initial chest x-ray (A) shows complete collapse of the left lung with mediastinal shift to the left. A second chest x-ray obtained 10 minutes after repositioning the endotracheal tube (B) is nearly normal, showing the remarkably rapid changes in volume that can occur. The mediastinum remains shifted to the left, although this too resolved on a subsequent x-ray.

Chest x-ray A shows signs of volume loss, including a dramatic shift of the mediastinum completely hiding the heart and superior mediastinum within the dense-appearing left hemithorax. This chest x-ray appearance can occur with right main bronchus intubation, collapse of the left lung from obstruction of the left main bronchus (eg, endobronchial carcinoma, mucus plugging, or aspiration of a foreign body into the left main bronchus), or a previous left pneumonectomy. In all of these conditions, the loss of volume of the left lung allows the mediastinum to shift to the left.

This chest x-ray appearance should not be confused with a large pleural effusion or very dense pneumonia. In a pleural effusion, the volume of the left hemithorax contents would be preserved, and the mediastinum would not shift to the left. With pneumonia, similarly, the mediastinum might not shift to the left, although volume loss can occur with pneumonia.

In an intubated patient, malpositioning of the endotracheal tube is the most likely cause of this appearance. When doubt exists, and if the abnormality does not resolve with endotracheal tube repositioning, ultrasound can be used at the bedside to rule out pleural effusion.

Recognize this appearance to avoid unnecessary procedures such as chest tube (for misdiagnosis of pleural effusion or hemothorax) or chest computed tomography.

Feature Editor: Joshua S. Broder, MD, FACEP

CME Questions

Qualified, paid subscribers to *Critical Decisions in Emergency Medicine* may receive CME certificates for up to 5 ACEP Category I credits, 5 *AMA PRA Category I Credits*™, and 5 AOA Category 2-B credits for answering the following questions. To receive your certificate, go to www.acep.org/criticaldecisionstesting and submit your answers online. You will immediately receive your score and printable CME certificate. You may submit the answers to these questions at any time within 3 years of the publication date. You will be given appropriate credit for all tests you complete and submit within this time. Answers to this month's questions will be published in next month's issue.

1. Which of the following snakebites does not present with significant swelling and edema?
   A. bushmaster (crotalid)
   B. copperhead
   C. coral snake
   D. cottonmouth
   E. rattlesnake

2. What percentage of snakebites in the United States are from venomous snakes?
   A. 5%
   B. 10%
   C. 20%
   D. 50%
   E. 75%

3. Which therapies are typically used to treat serum sickness secondary to antivenin administration?
   A. acetaminophen
   B. antibiotics
   C. antihistamines and corticosteroids
   D. antivenin
   E. epinephrine

4. What percentage of venomous snakebites in the United States are from crotalids (pit vipers)?
   A. 20%
   B. 40%
   C. 60%
   D. 80%
   E. 99%

5. Most snakebite victims in the United States are from which demographic group?
   A. children younger than 5 years of age
   B. men 20 to 40 years old
   C. men 50 to 70 years old
   D. women 20 to 40 years old
   E. women 40 to 60 years old

6. Which set of features characterizes pit vipers?
   A. elliptical pupils, red/black/yellow bands
   B. elliptical pupils, round heads, and heat sensing pits
   C. elliptical pupils, triangular heads, and heat sensing pits
   D. round pupils, round heads, and heat sensing pits
   E. round pupils, triangular heads, and heat sensing pits

7. What is the primary action of coral snake venom?
   A. antihistamine release
   B. coagulopathy
   C. myonecrosis
   D. neuromuscular blockade
   E. none, it is a nonvenomous snake

8. What technique has been shown to reduce reconstitution times of FabAV?
   A. hand-rolling
   B. heating the solution
   C. leaving it still
   D. shaking vigorously
   E. using a bicarbonate solution instead of sterile water to reconstitute

9. What is the optimal prehospital treatment for a snake envenomation?
   A. ice and elevation
   B. immobilization
   C. incision and drainage
   D. suction
   E. tight tourniquet

10. What percentage of snakebites are dry, with no venom injected during the bite?
    A. 0 to 10%
    B. 10% to 20%
    C. 25% to 40%
    D. 50% to 60%
    E. 80% to 99%

11. Which of the following is the most common cause of upper gastrointestinal (GI) bleeding?
    A. alcohol-induced gastritis
    B. esophagitis
    C. malignancy
    D. Mallory-Weiss syndrome
    E. peptic ulcer disease

12. Which of the following statements is correct regarding peptic ulcer disease?
    A. approximately 50% of duodenal ulcers are infected with *Helicobacter pylori*
    B. gastric ulcers are the most common type of peptic ulcer disease
    C. gastrin-secreting tumors account for more than 20% of peptic ulcer disease
    D. gastritis can be a precursor to development of peptic ulcer disease
    E. the two main categories of peptic ulcer disease are *H. pylori* and cocaine use

13. Which of the following regarding Mallory-Weiss syndrome is correct?
    A. it is the result of a complete rupture of the esophagus
    B. it is usually self-limiting and does not require endoscopic hemostasis
    C. patients are commonly infected with *H. pylori*
    D. use of octreotide is clearly indicated
    E. use of upright chest radiography will show free air under the diaphragm

14. Hematochezia is defined as:
    A. the passage of bloody feces
    B. the passage of dark and pitchy stools stained with blood pigments
    C. the passing of red-colored stool from digested foods or drinks
    D. the vomiting of bright red blood
    E. the vomiting of dark-colored blood with stomach contents

15. Which of the following is a concerning (high-risk) factor in patients with upper GI bleeding?
    A. age younger than 60 years
    B. history of alcoholism
    C. no anticoagulant use
    D. no active hematemesis or melena
    E. stable vital signs

16. Which of the following is correct regarding proton pump inhibitor therapy for upper GI bleeding?
    A. decreases duration of hospitalization from upper GI bleeding
    B. directly affects platelets
    C. has been associated with increased rates of rebleeding and mortality
    D. has no effect on rebleeding
    E. is less desirable than H_2-blocker therapy

17. Which of the following is correct regarding patients with liver cirrhosis and upper GI bleeding?
    A. massive and difficult-to-control bleeding is a concern
    B. their mortality rate is lower than that of patients with peptic ulcer disease and upper GI bleeding
    C. they are rarely at risk for recurrent bleeding
    D. they are rarely at risk for variceal bleeding
    E. urgent endoscopy is rarely indicated

18. Which of the following is correct regarding vasopressin therapy in patients with upper GI bleeding?
    A. causes smooth muscle relaxation
    B. controls bleeding with few side effects
    C. is associated with myocardial or mesenteric ischemia
    D. is contraindicated in alcoholic patients
    E. slows bleeding by direct effect on platelets

19. In managing massive upper GI bleeding, the physician's first priority is:
    A. call the endoscopist
    B. call the patient's primary care physician
    C. ensure a protected and patent airway
    D. get an ECG
    E. start two large-bore intravenous lines

20. Balloon tamponade:
    A. is commonly used in duodenal bleeds
    B. is easy to use and can be placed by nursing staff
    C. is a first-choice treatment option for severe bleeding
    D. permanently stops bleeding in a short time
    E. should be removed in an ICU setting

Answer key for June 2010, Volume 24, Number 10

1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
B	C	C	A	D	D	C	B	C	A	E	C	B	C	A	C	A	A	D	C

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The Drug Box

Crotalidae Polyvalent Immune Fab (FabAV)

By Joseph Kearney IV, MD; Summa Health System Emergency Medicine Residency

Victims of pit viper envenomation who show progressive signs or symptoms, including unstable vital signs, changes in mental status, declining platelet count or fibrinogen, and worsening of the local injury, should immediately receive antivenin. Crotalidae polyvalent immune Fab, the most commonly available antivenin, is an antibody fragment that works by binding and neutralizing toxins within venom. It should be used within 6 hours of envenomation; but earlier is better. Doses are repeated until the patient's condition stabilizes. Although allergic or serum reactions are possible, most symptoms are mild—rash, urticaria, and pruritus. Because it is expensive, only a few hospitals stock antivenin. Early communication with the hospital pharmacy will facilitate earlier intervention.

Crotalidae Polyvalent Immune Fab (FabAV)	
Mechanism of Action	Venom-specific Fab fragment of immunoglobulin G (IgG) that binds and neutralizes toxins, facilitating their redistribution away from target tissues and elimination from the body.
Indications	For worsening signs or symptoms following crotalid envenomation.
Dosing	Initial dose: 4 to 6 vials, each mixed with 10 mL of sterile water then added to a 250 mL bag of saline, infused over 1 hour. Repeat until symptoms stabilize or improve. After stabilization: 2 vials every 6 hours for 3 doses (ie, at 6, 12, and 18 hours after the symptoms stabilize).
Side Effects	Rash, urticaria, pruritus, nausea, coagulation disorders.
Estimated Cost to Hospital and Patient	Price and availability vary by state.
Contraindication/ Precautions	Patients allergic to papaya or papain should only receive FabAV if the benefits outweigh possible anaphylactic risks. FabAV contains mercury in the form of thimerosal in very small amounts. Patients must be closely monitored for signs of anaphylaxis and serum sickness. Repeat courses of FabAV in subsequent envenomations may be subject to sensitization.

Feature Editors: Michael S. Beeson, MD, MBA, FACEP, and Amy K. Niertit, MD

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