The 3-Minute Emergency Medicine Medical Student Presentation: A Variation on a Theme

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Abstract

Oral presentations are a critical element in the communication of medical knowledge between students and faculty, but in most locations, the amount of time spent on teaching the oral presentation is minimal. Furthermore, the standard oral presentation does not work well within the emergency medicine (EM) setting, due to time constraints and the different principles that make EM a unique specialty. This article provides a suggested approach on how to educate students on optimal oral presentations in EM, as well as providing a link to an online guide instructing medical students how to give oral presentations.

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As Dr. William Donnelly stated in his article ''The Language of Medical Case Histories,'' ''[oral presentations] are the way in which physicians at every level of training communicate to each other their understanding of particular patients and their medical problems, what has been done about the problems, and what is being done about them.''[1] The expectations for these presentations vary depending on the expertise of the medical student and on the clinical service where the student is learning. As the field of emergency medicine (EM) evolves, there is a growing interaction between medical students and other members of the EM team, including residents and faculty. Medical students from all 4 years of training now come into contact with the emergency department (ED). However, their oral presentation training is primarily provided by other services. Because of the need in EM to provide a rapid assessment in addition to telling the patient's ''story'' effectively, a specific style of presentation is required for EM.

In addition, we believe that the majority of the student and resident educational interactions with attending physicians in EM occur during oral presentations, when the student provides his or her analysis of the patient's story to the other medical team members. Other interactions, such as direct patient contact and chart review, occupy a large amount of the student's interaction time with patients and are often not observed by superiors. Thus, the majority of the resident and attending's impression of a student, and ultimately the student's evaluation, is directly linked to how well the student presents. As a fourth-year medical student wrote from the University of California, San Francisco, ''. . . no matter how much compassion and warmth I may have with patients, my superiors grade me more on how polished I am, how well crafted my presentation is.''[2] In this article, we will summarize traditional presentation methods, elucidate how the EM presentation varies from the standard, and offer our guidelines for a successful presentation. Although these suggestions have not been studied, we have had success teaching this method to our medical students. Our goal is to have a student be able to present all pertinent information under 4 minutes, with the ultimate goal of the ''3-minute presentation.''

HISTORY OF THE ORAL PRESENTATION

The evolution of the oral presentation is not well described in the medical literature. The earliest mention of the patient narrative was in 1846 by Erasmus Fenner (dean of the New Orleans Medical School) who required students to read their patient write-ups to professors on rounds.[3] The patient narrative began prior to the creation of the written medical record; however, we theorize that the format of the oral presentation most likely tracked the evolution of the written medical record. Therefore, the ''standard'' oral presentation follows the same format as the written medical record, but the oral presentation focuses on information related to the chief complaint (CC).

As of 2003, the oral presentation has taken another step in evolution, with the ''SNAPPS'' format, developed

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at Case Western Reserve University School of Medicine. SNAPPS focuses on students keeping their patient summaries brief, narrowing the differential to two or three etiologies, analyzing the information to determine the most likely cause of the CC, probing the attending for knowledge by asking questions, planning the patient's management, and finally, selecting an issue related to the case for self-directed learning.[4] The creators of SNAPPS recognized the limited educational experience that many students undergo during oral presentations. Therefore, SNAPPS was developed to ''engage the learner and create a collaborative learning conversation in the context of patient care.''[4] Even though the SNAPPS format was designed for outpatient oral presentations, the brevity of the patient's history and the limited differential diagnosis are aspects that can be applied to EM.

More recently, a study from Boston University School of Medicine showed that a multifaceted intervention introducing specific guidelines for oral presentations did improve medical students' narrative skills.[5] The guidelines were compiled with input from more than 60 faculty members of the Department of Medicine. Prior to the guideline intervention, 33 of 111 (30%) students received a rating of ''excellent'' during their medicine clerkship. With the integration of the guidelines the following year, 42 of 96 (44%) students received an ''excellent.''[5] The response from the medical students in the study showed appreciation of specific guidelines to explain why data should be included and in which order it should be placed in the oral presentation.

WHY DO STUDENTS STRUGGLE WITH THE ORAL PRESENTATION?

Didactic and on-site training are the two general ways medical students receive education on how to give oral presentations. Didactic training occurs primarily in the first 2 years of medical school, while on-site training occurs during clerkships. Schools may include sessions during their Principles of Clinical Medicine courses in Years 1 and 2 or in the Transition to Clerkship at the end of Year 2. Although the Liaison Committee on Medical Education (the accrediting body for physician programs) states that in a medical school there ''must be specific instruction in communication skills as they relate to physician responsibilities, including communication with patients, families, colleagues, and other health professionals,'' there is no requirement for a specific amount of time to be spent teaching oral presentation skills.[6] Another reason students may have difficulty acquiring proper oral presentation skills may be due to ''no universally accepted or widely used tool to help learners improve oral presentation skills.''[5]

On-site training also has its challenges. As a teaching technique, many students are often asked to duplicate presentations of more senior members of the team. However, an article summarizing student interviews about this issue commented that ''effective presenters alter the structure and organization of their presentations, but could not articulate how, when, or why these alterations were chosen . . . as a result, students were not easily able to understand or mimic those successful presentations that they witness by more experienced team members . . . in fact, experts may not be the ideal models for novices.''[7] This article provides a framework for students and educators to refine oral presentations, whether in the didactic or clinical environment.

IMPORTANT CHARACTERISTICS IN EM

In addition to the rigors of learning ''general'' oral presentation skills, the unique characteristics in EM compound the difficulty of learning presentation skills. Many EM traits often lead students, who are proficient with oral presentations on other services, to have difficulty with oral presentations in EM. Rosen's landmark paper, ''The Biology of Emergency Medicine,''[8] describes the fundamental differences of EM from other services. These differences provide a unique framework to the oral presentation: 1) assume that every patient has a life- or limb-threatening condition, 2) juggle multiple patients simultaneously, 3) prioritize patients according to level of concern, and 4) address patient loyalty and follow-up issues and consequences of incomplete medical records.

These principles mandate presentations to be concise and to the point without sacrificing essential information

Table 1
How the Axioms of Emergency Medicine (EM) Care Translate into an Abbreviated Presentation, with Specific Teaching Points to be Elaborated on by the Instructor

Important EM Traits →	Characteristics of Oral Medical Record due to the Important EM Traits
Assume every patient has a life-/limb-threatening condition	Be concise. The listener expects the presenter to use clinical judgment to edit patient information, with an emphasis on characteristics that apply to the inclusion or exclusion of life threats
Juggle multiple patients simultaneously	Present in less than 5 minutes. State CC first and focus only on CC unless other concerning problems arise
Prioritize patients	Only talk about the most pressing issues; as there are multiple patients with pressing issues, focusing a presentation allows for rapid assessment of the critical nature of their complaint and subsequent triage among other patients
Address patient loyalty issues and consequences of incomplete medical records	Obtain a complete history. As patients are not tied to a specific practitioner, ''hospital hopping'' is more common, meaning a complete picture cannot rely on medical records. Therefore, it is critical to get a detailed interview

CC = chief complaint.

for the listener to easily formulate a plan of diagnosis and therapy. The fourth principle, which initially focused on loyalty to specific physicians and frequency of primary care visits, is now even more applicable as there are rarely ties to specific hospitals or health care systems, resulting in fractured and incomplete medical records (see Table 1). By applying these overarching principles of EM to the oral presentation, the student maintains focus on the key components of EM practice.

EM ORAL PRESENTATIONS

The following sections are the required elements of a ''typical'' EM oral presentation: chief complaint (CC), history of presenting illness (HPI), medications, allergies, physical exam, summary statement, problem assessment, and plan. Detailed instructions to create an EM oral presentation primarily for medical students, *EM Oral Presentation Instruction Manual*, is available as an online data supplement at http://www.blackwell-synergy.com/doi/suppl/10.1111/j.1553-2712.2008.00145.x/suppl_file/acem_145_sm_DataSupplementS1.pdf.

One might notice the minimization of past medical history (PMHx), past surgical history (PSHx), social history (SocHx), and family history (FmHx) in the above list. Their diminished emphasis is necessary for a speedy and efficient oral presentation in EM. By decreasing the number of sections, the student is compelled to include vital information contained in these areas in other parts of the presentation, or not to mention them, as they may not be pertinent to the reason for the patient's visit to the ED. Of note, pertinent PMHx should be included in the first sentence (the one liner) of the HPI.

The ability to determine pertinent information is difficult for student physicians and is directly limited by the student's level of medical knowledge. We therefore suggest that students err on the side of safety and include questionable pertinent information. However, we do encourage educators to specifically identify incorrectly ''labeled'' data and explicitly explain why the data were ''mislabeled.''

WHAT IS PERTINENT INFORMATION?

One way for a student to determine ''pertinence'' is to have a short differential diagnosis list for the specific CC. Then, by using principles of pathophysiology (mechanism, course of the disease, complications), which a second- or third-year student should know, the student can ask clarifying questions about each etiology on the differential list. For example, if the CC is abdominal pain and the potential differential includes gastric ulcer, cholecystitis, and pancreatitis, the student should ask clarifying questions such as ''is the pain worse at night?,'' ''worse before or after meals?,'' ''worse during fatty meals?,'' ''any back pain?,'' or ''any alcohol use?'' The answers to the above questions are pertinent and should therefore be placed in the HPI. The student will have the ability to obtain relevant information during the extensive interview process, and this information can then be narrowed to provide a concise story to the listeners. An absence of these key pieces of information should provide a clue to the educator that these possibilities were not on the medical student's differential and will then provide an opportunity to discuss alternative differential diagnoses that the medical student may have missed.

As students obtain more clinical and presentational experience, they will become more proficient at including only pertinent data. Early in their medical training, students have limited ability in grouping patient information as pertinent and nonpertinent.[9] Lingard and Haber[9] suggest that ''if you give [students] section headings, they'll always put something under them, even if all the information we need is really contained in the first two sections of the presentation.'' If determining information relevance is related to clinical knowledge, then by definition, students will have limited abilities in this area. Therefore, it is vital that the educator not use vague comments such as ''tell me only the stuff I need to know'' or ''give me information that is only relevant to the chief complaint'' for feedback to students. Instead, we recommend giving students specific explanations of why certain information in the presentation should be left out to change the learner's misconceptions about what is really pertinent information. On the other hand, if critical information is not included, the educator should elucidate the knowledge deficit that results in the absence of the critical information from the presentation. Keeping these guidelines in mind, we will discuss each individual section of the oral presentation and how that applies to the EM setting.

HPI

The HPI in EM tends to include more information from other sections like review of systems (ROS), FmHx, and SocHx due to the need for speed and efficiency in EM presentations. All of the pertinent information from the ROS, FmHx, and SocHx should be included in the HPI to save time. This provides students an abbreviated template as a guide to limit details of the patient's medical issues.

PMHX/PSHX/FMHX/SOCHX

As previously mentioned, any pertinent information to the CC should be mentioned in the HPI. If done correctly, there should be no formal mention of titles like PMHx, PSHx, SocHx, or FmHx in the oral presentation. An example would be: ''*This patient is a 40-year-old man with a past history of coronary artery disease, hyperlipidemia, and hypertension who comes to the ED complaining of chest pain.*'' This is also the initial moment for the educator to realize the knowledge base of the medical student. With an inappropriate or incomplete initial statement, the educator will be able to provide teaching points on presentation skills.

ROS

As the student gains more clinical knowledge, the presentation of the ROS should become smaller and smaller until ultimately there is little to no mention of ROS. At first, beginning students should mention all patient

complaints. By obtaining as much information in the ROS during the interview as possible, the student will be assured that he or she has not missed anything. Information the student believes is pertinent to the CC is mentioned in the HPI. Information the student believes is not pertinent or is of uncertain relevance to the CC should be mentioned in the ROS.

There are situations where some nonpertinent complaints are serious enough to be relabeled as a second CC. For example, the patient's CC is a leg injury, but further questioning also reveals the patient to have dysuria, back pain, fever, and chills, which is concerning for pyelonephritis. If the patient is allowed only one CC, then dysuria, back pain, fever, and chills are not pertinent data and by definition should be stated in the ROS. However, at times, complaints in the ROS get forgotten or even ignored. Therefore, dysuria should be moved from ROS and added to the HPI as a second CC: ''*The patient is a 45-year-old female who came to the ED complaining of a traumatic leg injury and dysuria.*'' The student should then divide the patient's history into two HPIs: one telling the pertinent information of the leg injury, and the other telling the pertinent information of the dysuria. Without this ''refocusing'' of a second CC, the educator is at high risk for missing a key element that the medical student may not consider important due to their lack of knowledge base. For example, the dual CCs of arthritis and urethritis will trigger in the educator the concern for Reiter's syndrome, but this association may be lost on the novice learner.

Medications/Allergies

Medical students should be reminded to mention all medications and allergies. Medications have numerous side effects, and even though the medication might not be causing the CC, the concern for future drug reactions with therapeutic medications mandates the knowledge by the educator of all the patient medications. However, students should only mention the drug; the dosing schedule should only be discussed if applicable to the case or in the discussion that follows the presentation.

Physical Exam

The physical exam portion of the EM presentation should be similar to the ''review of systems'' section, focused on the pertinent positives and negatives, with the remainder left out, under the assumption that the other components are not applicable to this patient's case. The same caveat for the ROS also applies. With less medical knowledge, the basic learner may not know what physical exam findings are important based on a specific patient's complaints. As such, it is incumbent on the educator to ask about unmentioned pertinent positives and negatives.

Summary Statement

The summary statement should be one to two sentences that encapsulate the entire clinical picture of the patient's visit to the ED. The first sentence should be approximately the same as the first sentence in the HPI. ''The patient is a {age}-year-old {gender} with a history of {pertinent PMHx} who presents with {CC}.'' The second sentence should include only the most important complaints, physical exam findings, studies, or labs values. We believe that beginning students should not give a diagnosis in the summary statement, which differentiates the summary statement from an impression statement. This is not an area where the student should present the final diagnosis, as it is unlikely for a definitive diagnosis to be possible at this stage in the patient's workup. Instead, this is the summation of the history and physical elements that will assist in formulating the differential diagnosis.

Problem Assessment and Plan

The problem assessment is the first section in the oral presentation where the medical student should give his or her opinion. The patient's problems should be mentioned from the most life-threatening to least life-threatening. There is no ''right'' order, since everyone

Table 2
How to Correct Common Mistakes of the Oral Presentation

Pitfalls in Oral Presentations	Example	Method on How to Change Pitfall
Failure to include relevant PMHx	An elder patient has an acute episode chest pain but student does not mention patient had a CABG 2 years prior	Tell the student that any conditions that can cause the CC should be labeled pertinent and included in the oral presentation
Including nonrelevant ROS in the HPI	Patient has chest pain but the student also mentions in the HPI that the patient has also had a knee replacement in the distant past	Ask the student why this piece of information was included, and then specifically explain why the knee replacement is not relevant to the chest pain
Including PE findings in the HPI or ROS	The patient complains of a swollen knee after a skiing accident, painful to walk but the knee had full range of motion and was not tender	Remind the student that anything they see or do to the patient should only be mentioned in the physical exam section
Poor body language	The student has distracting gestures during presentation	Explain why body movements are distracting and encourage verbal descriptions

CABG = coronary artery bypass graft; CC = chief complaint; HPI = history of presenting illness; PE = physical exam; PMHx = past medical history; ROS = review of systems.

will have different opinions. However, this order is critical for the educator to elucidate; it allows insight into the student's thought processes as to possible life threats. The first mentioned problem does *not* have to be the patient's CC. For example, a patient complains of abdominal pain, but since arriving to the ED has started vomiting large quantities of blood. The first problem mentioned should be hematemesis, not abdominal pain, even though it was the abdominal pain that brought the patient to the ED. Next, the speaker should quickly list life-threatening etiologies of the problem, any labs or studies needed, and recommendations for current treatment.

Additional Training Techniques

It is expected that medical students will not achieve excellence with initial presentations. It is also common for students to substitute additional errors in presentations as initial errors are corrected. We have discussed the most common errors that we have found and correction methods in Table 2. If time permits, students should be allowed to present each case two times. The first time is the way the student believes the case should be presented. After specific feedback from the listener, the student's second presentation of the same case will include corrections to reinforce proper technique.

SUMMARY

With medical students spending increasing time in the ED, there is a greater need for student education on how to deliver patient narratives since ''high-quality oral presentations have the potential to promote coordinated patient care, enhance the efficiency of rounds, and encourage teaching and learning.''[5] The four axioms of EM require a rapid and efficient student presentation. However, a direct result of students' limited clinical knowledge is the inability to determine nonrelevant from pertinent details and can lead students to include extraneous facts causing lengthy presentations. As EM educators, we believe that it is important for all students who rotate through the ED to be able to tell the patients story in a ''3-minute'' format.

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Supplementary Material

The following supplementary material is available for this article:

Data Supplement S1. Oral presentations in emergency medicine (PDF file)

This material is available as part of the online article from: http://www.blackwell-synergy.com/doi/suppl/10.1111/j.1553-2712.2008.00145.x/suppl_file/acem_145_sm_DataSupplementS1.pdf

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(This link will take you to the supplementary material).

Oral Presentations in Emergency Medicine

This guide is designed to help medical students establish and refine their presentation skills, with a focus on the emergency medicine presentation. Please note that there are two key elements to giving presentations: good feedback and flexibility of presenting style. You should make sure to use this guide in concert with feedback you get from your attending and should realize each attending will be slightly different. With the ability to modify your presentation based on feedback, we are certain you will develop the skills needed to communicate the critical information of a medical presentation both concisely and completely.

Objectives of the EM Oral Presentation

1. Tell the patientôs story < 3 min in order to start diagnostic tests and treatment quickly.
2. Only state pertinent information.
3. Presentation must be fluid, flowing from one section to another with no hesitation and done with confidence.

The Oral Presentation Outline

1. Chief Complaint (CC)
2. History of Presenting Illness (HPI)
   a. One liner
   b. 
   c. 
   d. 
   e. 
   f. 
   g. **PERTINENT** Past Medical History (PMH)/ Past Surgical History (PSH)/ Social History (SocHx)/ Family History (FmHx)
3. Review of Systems (ROS)
4. All medications
5. All allergies
6. Physical Exam (PE)
7. Summary Statement
8. Problem Assessment
9. Plan

Figure A

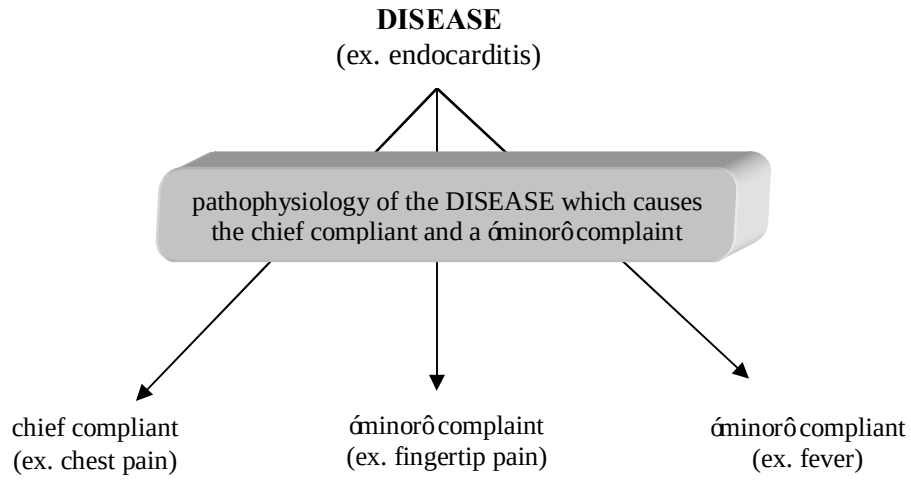


Figure B

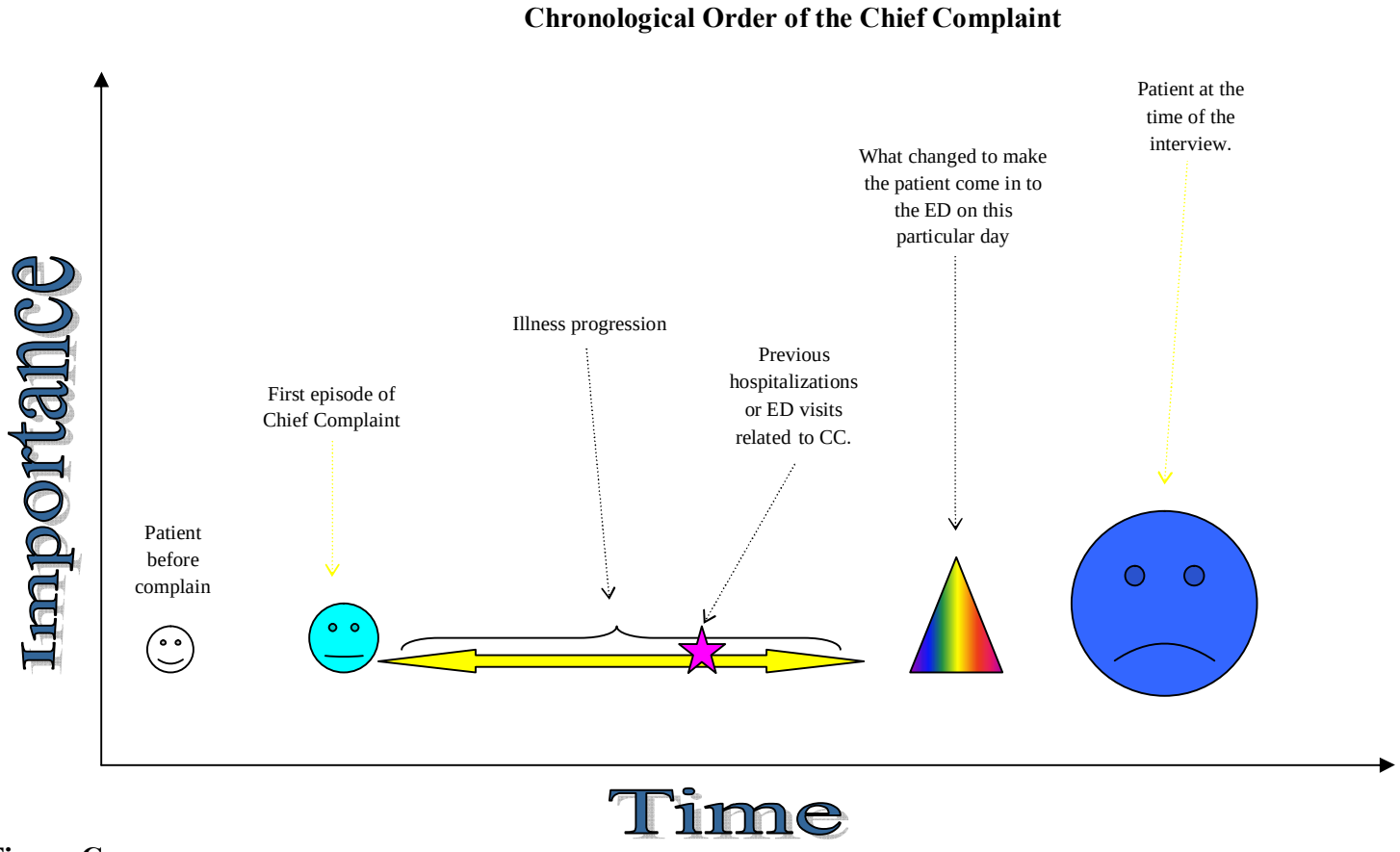
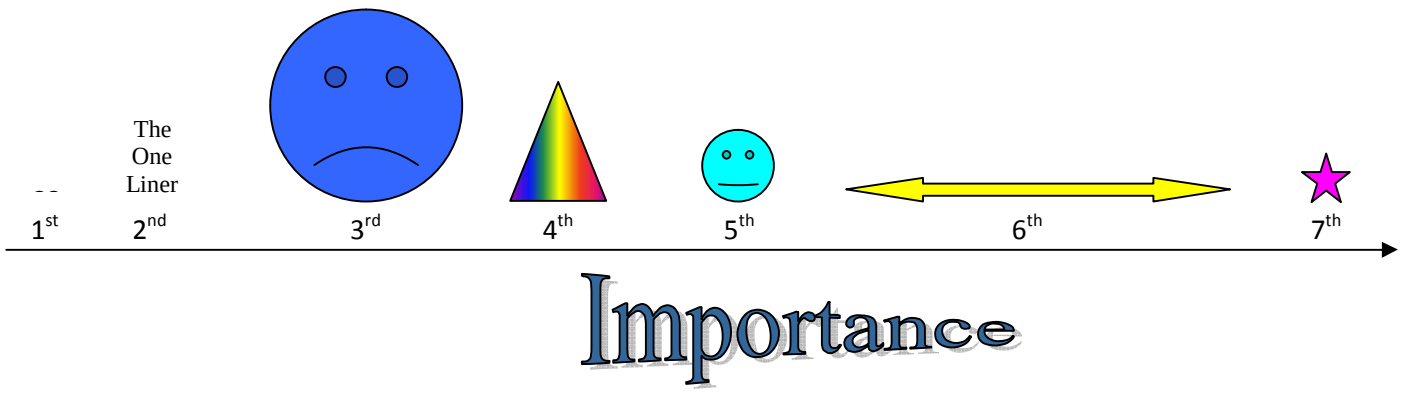


Figure C



Intro

The overall feel for oral presentations in the emergency department is to give concise sentences in a bullet-point like fashionð taking this mentality will hopefully rid you of extra words and phrases. Most importantly is using a format that makes sense, which will increase fluidity and confidence of oral presentations.

What does 'pertinent' really mean?

Before discussing the individual sections of the oral presentation, the vague term of ópertinentô must be clearly defined. Often students are interrupted during their oral presentation by the listener who says ñonly give me the pertinent informationò or ñtell me what I need to know to treat this patientò. These interruptions are likely due to the listenerôs frustration with the medical studentôs regurgitation of too many facts. Therefore, it is critical for medical students to become more proficient dividing all the facts into 2 categories: pertinent and non-pertinent information. The skill of labeling information as pertinent or non-pertinent requires a significant level of clinical knowledge; therefore, students will naturally have limited abilities. When students receive non helpful phrases such as ñonly give me info that is related to the chief complaintò, you should respectively ask the educator for specific explanations as to why a given piece of data is or is not ópertinentô.

Of note, students should generally not duplicate presentations of senior residents or attending, because these instructors have mastered the oral presentation and might not use the same format as required by medical students.

Table 1. Illustrates one way how ópertinentô patient information is determined.

Complaint	Possible etiologies of complaint	Pertinent questions	Example phrases to be stated in the HPI*
Chest Pain	Acute Coronary Syndrome	Have you ever had this type of chest pain before?	*Patient had similar chest pain a year ago.*
		Does the chest pain increase with walk?	*Chest pain increases with ambulation but decreases with rest*
		Is the chest pain sharp, dull, or burning in nature?	*Chest pain is dull and substernal with radiation down left arm.*
	Pulmonary Embolism	Do you feel short of breath?	*Patient does not have shortness of breath*
		Does the chest pain change when you breathe?	*Chest pain is non-pleuritic*
		Have you ever had blood clots before?	*Patient has never had a deep venous thrombosis.*

***The responses to the pertinent question column are pertinent by association and thus should be stated in the HPI section, not the ROS section, of the oral presentation**

Another way to determine ópertinentô information is as follows (depicted in Figure A): if you believe a symptom/complaint could be caused/explained by the same pathophysiology that could be causing the CC, then by definition that information is pertinent. Letôs say the patient has chest pain for a chief complaint. During the review of system questioning, the patient also complains of fingertip pain. Is the fingertip pain important enough to mention in the presentation? If you believe the fingertip pain is related to the chief complaint (therefore pertinent information) then it is stated in the HPI. On the other hand, if you believe the fingertip pain is NOT related to the chief complaint (therefore non pertinent information) then it is mentioned in the ROS. Since you have limited clinical knowledge (for now), **all** complaints should be mentioned because experienced clinicians might be able to connect the pieces together that students can not do yet.

Letôs say the previously mentioned finger pain resulted during a basketball game. Since the mechanism causing the fingertip pain (trauma during a basketball game) could not also cause the patientôs chest painð the fingertip pain is NOT pertinent to the chief complaint. Therefore, fingertip pain should be mentioned in the ROS. However, if the medical student believes there is a way the finger trauma could cause the chief complaint of chest pain, then the fingertip pain is pertinent information and should be mentioned in the HPI. Of note, any information mentioned in the HPI should NOT be repeated in the ROS.

Now letôs say the patient is febrile and an intravenous drug user. Now you believe the patient has endocarditis which is causing the chest and the fingertip pain. Since endocarditis can cause the chief complaint (see Figure C) of chest pain and cause fingertip pain (Oslerôs nodes), fingertip pain is pertinent information and should be mentioned in the HPI and not in the ROS. In other words, if you believe any óminorô complaints (which the patient usually mentions during ROS questioning) are being caused by the underlying process that could also cause the CC, then mention the óminorô (pertinent) complaints in the HPI. If the óminorô complaints are not caused by the underlying process thatôs causing the CC, then the óminorô (non-pertinent) complaints should be mentioned in the ROS. In summary, students should mention all patient complaints in the oral presentation. However, the difficulty is in what section should state the complaints (in the HPI or in the ROS).

Chief Complaint

Quickly stating the CC, prior to stating the one liner of the oral presentation, orientates the listener. If not mentioned, the listener becomes frustrated due to not having a reference point and thus stops paying attention. Itôs like going to lecture and not being told what the lecture is about. Exampleð *"The chief complaint is abdominal pain".*

History of Presenting Illness (HPI)

The HPI can be one of the most difficult sections for students due to the great variability of styles. Therefore, Figure B and C were created to illustrate the difference between the patientôs chronological story (Figure B) and the oral presentation story (Figure C). There are 3 general ways to present the HPI in the oral presentation:

1. In order of importance
2. Chronologically
3. No organization

For most attendings and complaints, method (1) is the best way to deliver the HPI, because there are 2 unwritten but important rules in oral presentations:

i) listeners have limited memory space

ii) listeners have short attention spans

Method (1) takes in account both of the above rules by having the most important information (☹ and ▲) located at the beginning of the oral presentation– illustrated by Figure C.

The second most common and entirely acceptable method is method (2). The reason for method (2)'s less popularity is that (2) does not address the previously mentioned unwritten rules of oral presentations. When presenting in chronological order, by the time the speaker gets to describing the CC (☹) the listeners (with their "short attention spans" and "limited memory space") will not remember as many important facts. On the other hand, in some situations method (2) will work better than method (1) but unfortunately only experience will help the medical student decide when to use method (2).

Method (3) is how many medical students give oral presentations. All the information is in the HPI but in no particular order. No attending wants to hear an unstructured oral presentation. Please avoid method (3) as much as possible!

Most importantly, by keeping Figure C in your head, when you get interrupted with questions you will not lose your place and you will know what section to mention next.

a. The One Liner

The point of the one liner is to state important patient specific stats to help clinicians stratify certain disease risks in the patient. The items always included in the one liner are: the patient's age, sex, pertinent medical history <u>relating</u> to the CC, and the CC. Do NOT use diagnostic terms to describe the chief compliant. If the patient complains of chest pain, the CC = chest pain. The CC does Í angina, which is a diagnosis. In my experience there are two pathologies that should be in the one liner almost all of the time– Diabetes Mellitus (DM) and Hypertension (HTN). Why? Because DM and HTN are very common in the general population and over time they can affect every organ. However, DM and HTN can be left out of the one liner in situations where the CC could not be caused by DM or HTN.

Example of the one liner– ñ*The patient is a {age} year old {sex} with a history of {pertinent PMH} who presents with {CC}"*

b. ☹

The blue sad face represents the patient at the time of the interview. The CC should be fully evaluated (location, radiation, what makes it better or worse…). Also this is where all the positive and negative pertinent information goes. In other words, mention any complaints YOU think are related to the CC. And do not list any complaints if YOU believe they are not related to the CC– they go under the ROS section. For example, ñ*The chest pain is dull, substernal with radiation only to the left arm. Chest pain gets worse with ambulation and improves with rest and sublingual nitrogen. The pain began this morning with three out of ten and now is eight out of ten."*

<u>Examples of common Chief Complaints</u>

PAIN– *"The patient describes the pain as {#} out of ten and is located substernally/hip/big toe… The pain is sharp/dull/pressure/throbbing in nature which is exacerbated by exercises/inspiration… but is alleviated by exercise/rest/medications…"*

DIARRHEA– *"The patient complains of diarrhea for the past {#days} with approximately {# episodes/day}. The stool is {color} with {no} hematochezia/melena. The diarrhea is {not} associated with food. The patient does {not} complain of being ill prior to diarrhea."*

HEADACHE– *"Pt. complains of {unilateral, bilateral} headache which began approximately*

{#days} ago. The headache is {throbbing, continuous} which is {not} associated with {any} facial symptoms such as tears, facial numbness… No vision changes during episodes. Patient can not recall any triggers. Headache is {not} preceded by auras or exacerbated by exercise."

c. 

Why the patient came into the ED is an important piece of information that is often forgotten by medical students. Since the beginning of the patientôs illness, the patient has not sought medical assistance, but what happened to the patient to compel him/her to come to the ED. Patients sometimes volunteer the information right a wayð ñI started having chest pain this morning, which I never had before, so I decided to come inò or ñI have had a headache off and on for the past five years but last night the headache was totally different and woke me upò. It is apparent in the previous two examples what changed to make the patient seek medical adviceð new chest pain and different headache, respectively. But what about the following example: ñI been having diarrhea for the past three daysò. It might be easy to stop here and say she came in because sheôs having diarrhea for 3 days, is tired, and wants treatment. But if no one asked specifically why she came into the ED today and not yesterday or 2 days ago, the medical student would not find out that the patient noticed some blood in her stool this morning but has not had one since and therefore did not mention it until specifically asked. Then the patient gets really emotional and states that colon cancer runs in her family and her father died around her age due to undiagnosed colon cancer. Yes, you might get this info later on in the interview such as FmHx <u>BUT</u> you might not.
Exampleó*"Patient came to the ED today because of* _____ò {pain became more severe, pt could not take it any longer, family persuaded pt to come in, medication stopped workingé }.

d. 

Every listener wants to know how long the patient has had the chief complaint. The CC duration is important because the ranking of the differential diagnosis will change depending if the CC has been going on for 2 days versus 2 years. Keep it short and sweet.
Exampleð ñ*The {chief compliant} started approximately {time} ago."*

e. 

The progression of the chief compliant is useful to relay how rapidly the CC is changing. Keep it big picture, do not give too much detail. The listener whatôs to know if the CC is getting: worse, better, or unchanging. If the CC is getting worse tell how itôs getting worse (it the pain lasting longer, becoming more frequent, does not respond to meds nowé). Like wise if the CC is getter better quickly explain how.
Example—*"Since the first episode, the CC has been getting {worsening/improving/unchanged} due to {reason why CC is worse/improved}."*

f. 

Briefly mention previous hospitalizations or emergency department visits IF the prior encounter is ópertinentô to the present CC. What should be included is: Prior CC related to todayôs CC, date of hospitalization/ED visit, pertinent test results (CT, MRI, stress testé), pertinent lab results (cbc, lipase, LFTs, Alcé), and discharge treatment.
Example—*"The patient was previously hospitalized for a similar chief complaint of chest pain 2 months ago. Patient had ST elevation and elevated troponins. Discharge diagnosis was Acute*

Myocardial Infarction with medical management"

—"The patient had a previous emergency department visit for a similar complaint of right upper quadrant abdominal pain 2 days ago. Right upper quadrant ultrasound then was normal. Patient sent home with the diagnosis of Abdominal Pain of Unknown Etiology with ibuprofen for pain."

What about the PMH, PSH, SocHx, and FmHx

One might notice the lack of Past Medical History (PMH), Past Surgical History (PSH), Social History (SocHx), and Family History (FmHx) from the above list. Their removal is necessary for a speedy and efficient oral presentation in EM. If all sections were included, the speaker would be tempted to add non-relevant information to fill in the ósectionsô. By decreasing the number of sections, the speaker is compelled to discard non-relevant information. If done correctly, there should be no formal mention of titles like PMH, PSH, SocHx, and FmHx. The less medical knowledge one has the less ability to determine what data is pertinent and not. Therefore, students should error on the side of safety and include questionable pertinent information.

ROS

For beginners, all complaints get mentionedð itôs just figuring out if the complaint goes in the HPI or ROS. However, resident training and higher have enough clinical knowledge to leave out mundane complaints. Right now assume you donôt know enough to leave out complaints. There might be a connection between the CC and a lesser complaint that an attending can make but a student might miss.

If there are no complaints that should go in the ROS then use the following phrase: *"Review of Systems is as previously mentioned in the HPI."* If there are complaints in the ROS then use the following phrase: *"Review of Systems is as previously mentioned in the HPI but also includes...{non pertinent complaints}"*

There are situations where some non-pertinent complaints are serious enough to be relabeled as a second chief compliant. For example, the patientôs chief complaint is a leg injury but further questioning also reveals the patient to have dysuria, back pain, fever and chills which is concerning for pyelonephritis. If the patient is allowed only one chief compliant, then dysuria, back pain, fever and chills are not pertinent data and by definition should be stated in the ROS. However, at times, complaints in the ROS get forgotten or even ignored. Therefore, dysuria should be moved from ROS and added to the HPI as a second chief complaintð ñ*The patient is a 45 year old female who come to the ED complaining of a traumatic leg injury and dysuria."* Then you should divide the patientôs history into two HPIôsð one telling the pertinent information of the leg injury, the other telling the pertinent information of the dysuria.

PE

Always mention the Vital Signs first. It doesnôt matter what order they are mentioned, but a common order is Temperature, Blood Pressure, Heart Rate, Respiratory Rate, and Oxygen Saturation. With oxygen saturation always mention modality of the oxygen delivery (room air, nasal cannula, continuous positive airway pressureé). A patient with an O_2 saturation of 91% on room air is much different than a patient with the same O2 saturation but is receiving 100% oxygen via a mask. There are some exceptions where vitals do not have to be recited individually such as minor trauma complaints like laceration, broken toes/fingersé). In these

cases it is usually acceptable to say *"the vitals are within normal limits".* However, make sure you know the specific values if asked.

For clarification, saying ñ*vitals are within normal limits*ò does NOT equal ñ*vitals are stable*ò. Vitals within normal limits mean that the patientôs vitals fall within a range of universally acceptable values. óStableô vitals mean you have been getting serial vital values which are not changing. Furthermore, stable vitals can be ónormalô or óabnormalô. Normal stable vitals signs are unchanging vitals within the normal limits. Abnormal stable vital signs are unchanging but are not within the normal range. After the vitals, only mention the pertinent physical exam findings. It is assumed that you did a complete physical exam from head to toe and that all exams (lung, cardiovascular, GI, Neuroé) are normal unless otherwise specified.

Exampleð {CC = hand laceration} ñ*The vitals are within normal limits. The physical exam is non-contributory except for a 2 inch laceration on the thenar eminence. The laceration was superficial, no foreign bodies identified. The first digit had full range of motion, full strength, and no loss of sensation."*

—{CC = abdominal pain} *"The vitals are: temperature of 38.5, blood pressure of 135 over 87, heart rate of 98, respiratory rate of 16, and oxygen saturation of 98 percent on room air. The physical exam is non-contributory except for the abdominal exam which revealed a distended abdomen, hyperactive bowl sounds, diffuse tenderness to palpation but no guarding and no rebound tenderness"*

Labs/Studies

Usually there are no lab/study results to report prior to the oral presentation. However if there are labs and/or studies to report, do NOT recite all the data. For labs, only mention the abnormal values. Example—*"the complete blood count is within normal limits and the chem 7 is within normal limits except for a sodium of 125."* For studies, only mention the overall impression the radiologist reports or your personal impression of the study.
Exampleð *"the chest x-ray shows a left lower opacity"*

Summary Statement

The purpose of the summary statement is to give an overall clinical picture in 2-4 sentences. There are 3 main components (listed as A, B, and C) which should be included into the summary statement:

A) **<u>The one liner</u>**

Within the one liner include the following components:

a. progression of the chief complaintï getting better, getting worse, or is static
b. chief complaint is chronic or acute

Example ð ñ*The patient is a 50 year old male with a history of Coronary Artery Disease and Coronary Artery Bypass Graft times two who presents with* ***<u>improving</u>*** ***<u>acute</u>*** *chest pain.*ò

ð ñ*The patient is a 45 year old male with no significant past medical history who presents with* ***<u>worsening</u>*** ***<u>acute</u>*** *ankle pain.*ò

B) **<u>1-2 important symptoms and/or physical exam findings.</u>**

Exampleð ñ*The chest pain is similar to a previous myocardial infarction in that pain decreases with rest and sublingual nitrogen and also has a friction rub on exam.*ò

ð *"The right metacarpophalangeal joint is* ***<u>swollen</u>****,* ***<u>erythematous</u>****, and* ***<u>painful</u>***

which is similar to previous episodes occurring after drinking large quantities of alcohol."

C) **1-2 important diagnostic studies or labs if available**.

Example: ñ*The **electrocardiogram** showed ST segment elevation in the inferior leads and the first **troponin** is still pending.*ò

ð *"The ankle **radiographs** shows soft tissue swelling, no fracture and the **joint tap** has needle shaped crystals."*

Do not recite all complaints, abnormal physical exam findings, or lab values in the summary statement because they were already mentioned earlier in the oral presentation. However, do mention the most important pertinent findings to refresh the listenerôs memory.

For clarification, many students are instructed to give an impression statement after the physical exam section instead of a summary statement. However, students often are not given an explanation of how the two are different. To clarify the point, the two statements have been translated into symbolic definitions:

Summary Statement = the one liner + most important symptoms/PE findings + most important studies/labs

Impression = Summary Statement + speakers opinion of the most likely etiology or etiologies explaining the patientôs clinical picture.

Students should save their opinion for the Problem Assessment section in order to prevent the common mistake of only discussing one or two etiologies that can happen with using an Impression statement. As medical students gain experience, many switch to using the Impression instead of the Summary Statement which is acceptable but a more technical method.

Problem Assessment

The problem assessment is where each problem gets mentioned with you giving your analysis. The first problem mentioned does NOT have to be the patientôs chief compliant. For example, the patient complains of abdominal pain; but, since arriving to the ED the patient has started vomiting blood. The first problem mentioned should be hematemesis, not abdominal pain, even though the abdominal pain originally brought the patient to the ED. The general rule is to mention the most life threatening problem to the least life threatening problem.

Example ï 1. hematochezia
2. abdominal pain
3. headache

Within each problem you should give your assessment of the possible etiologies .

a. list the differential diagnosis

Once again there is no correct order. Since the job of the emergency department is to rule out life threatening causes you should list the most harmful etiologies first followed by the most likely etiologies. A general rule is to state 2 ï 4 etiologies from the harmful category and 2 ï 4 etiologies from the likely category. Do not mention every etiology because the presentation must be kept ideally under 3 -5minutes.

Example: ñ*The differential diagnosis includes…"*

b. Give and explain facts that support and negate each etiology mentioned.

Use physical exam findings, labs or studies, patientôs risk factorsé to argue for or against each etiology mentioned.

One way to structure the Problem Assessment is as follows:

ñ*The most harmful etiologies are 1)____ , 2)____ and 3)___. 1)____ is a possibility because of _____. However, _____ and _____ do not support this because of _____. 2)__ is a possibilitybecause of _____ but is less likely due to _____ and _____. 3)___ is …. The most likely causes are i)____, ii)___, and iii)__. I believe the most likely etiology is _____ because of _____ and _____ but ____ and _____ do not support this because of _____…"*

Example —*"The harmful etiologies could be a septic join, fracture, or ligament tear. A septic joint is a possibility due to the ankle being swollen, hot and erythematous. Also the patient is an intravenous drug user which increases the risk of a septic joint. Fracture is another possibility but the patient does not remember any traumatic events, the joint is hot which is unlikely with a fracture, and there is diffuse tenderness—not point tenderness which would be expected with a fracture or ligament tears. The more likely etiology is an acute gouty episode because of a positive family history, recent alcohol use and the involved joint is the 1st metacarpophalangeal joint which is classically the involved joint for gout."*

Plan

The plan should include labs or studies to help confirm your diagnosis or eliminate possible etiologies. Generally, it is assumed the listener knows why the tests are being ordered and thus you should only give a brief explanation to the listener why each test should be ordered. Also, the plan should include how the patient should be taken care of right now. For instance, if the patient is in pain, give an analgesic. If the patient is dehydrated, give fluids.

Exampleð ñ*Therefore the current plan is to:*

1. aspirate joint to check synovial fluid for crystals and send fluid for culture, gram stain, and white blood cell count.

2. for immediate pain relief, give 4 milligrams of morphine and give naproxen for anti-inflammation."

Putting the Summary Statement, Problem Assessment and Plan together

"The patient is a 45 year old male with no significant past medical history who presents with worsening acute ankle pain. The right metacarpophalangeal joint is swollen, erythematous, and painful which is similar to previous episodes occurring after drinking large quantities of alcohol."

'The harmful etiologies could be a septic join, fracture, or ligament tear. A septic joint is a possibility due to the ankle being swollen, hot and erythematous. Also the patient is an intravenous drug user which increases the risk of a septic joint. Fracture is another possibility but the patient does not remember any traumatic events, the joint is hot which is unlikely with a fracture, and there is diffuse tenderness—not point tenderness which would be expected with a fracture or ligament tears. The more likely etiology is an acute gouty episode because of a positive family history, recent alcohol use and the involved joint is the 1st metacarpophalangeal joint which is classically the involved joint for gout

Therefore the current plan is to:

1. aspirate joint to check synovial fluid for crystals and send fluid for culture, gram stain, and white blood cell count.

2. for immediate pain relief, give 4 milligrams of morphine and give naproxen for anti-inflammation."

We hope you have found this guide to be helpful. Remember, be flexible in your structure and rely on your attending or upper level residents to provide appropriate feedback. Sometimes they need encouragement, so don't be afraid to ask what you could have done better in your presentation. Also, remember that you are still a student. Your presentations still matter in terms of medical care, so err on the side of including more as opposed to less. Lastly, practice! Take advantage of every opportunity to present a patient that you can; you won't get better without trying!

CHAPTER 197 Emergency Ultrasound

Vivek S. Tayal and Casey M. Glass

PERSPECTIVE AND DEFINITIONS

Emergency ultrasound (US) is the simultaneous performance and interpretation of sonographic examinations at the bedside of the patient in a focused manner to diagnose, monitor, and treat emergency medical conditions.[1,2]

Emergency US is an emergency clinician-performed study.[1,3,4] Although the operator is typically an emergency physician,[1,3,4] he or she may be an emergency physician assistant, nurse practitioner, emergency medicine (EM) resident, or trained emergency nurse or paramedic—all under the supervision of a trained, credentialed emergency physician.[5,6] Emergency physicians who have advanced training in ultrasound imaging are also known as emergency sonologists. Emergency US is typically performed in hospital emergency departments but may also be performed in other areas of the hospital, emergency stand-alone centers, out-of-hospital mobile transport such as ambulances or helicopters, disaster scenarios, military engagements, international rescue work, and remote settings such as space, sea, or land centers with limited or no medical access.[1,7-14]

Emergency US is a different paradigm in which the same physician or clinician performs and simultaneously interprets the examination in addition to integrating the results in the clinical scenario.[1,15] This is unlike other specialties, such as radiology, cardiology, and obstetrics and gynecology, that often use a trained technologist to perform the examination and a physician in that specialty to interpret or "read" the examination.[16,17]

Emergency US examinations are typically focused examinations of an area of the body, organ system, or physiologic pattern or goal-directed investigations of an emergency symptom or sign. Not all pathology will be detected by such examinations. Additionally, the physics of ultrasound may limit the detection of pathology or create pseudopathology that causes diagnostic difficulty. The primary limitation of such studies is the experience and skill of the performing physician.

HISTORY

A half century of scientific work in many countries produced the initial clinical medical uses of US in the 1950s by physicians interested in its advantages over traditional imaging, including noninvasiveness, lack of ionizing radiation, and superior resolution.[17]

Emergency physicians started using US in the 1980s as a way to rule out emergent "silent" diagnoses, such as ectopic pregnancy, intraperitoneal hemorrhage, hemopericardium, cholelithiasis, renal colic, and aortic aneurysm. A model curriculum was created by a Society for Academic Emergency Medicine (SAEM) task force that initiated formal guidance for emergency US programs.[18,19] Due to resistance within hospitals from traditional imaging specialties such as radiology and cardiology, the American Medical Association House of Delegates, led by the American College of Emergency Physicians (ACEP), carried a resolution suggesting that hospital credentialing committees follow specialty-specific guidelines for US-based credentialing.[20]

Specialty-specific guidelines were created in 2001 by ACEP that specified emergency US scope of practice, primary applications, training pathways, the number of procedures required for training prior to credentialing, quality assurance and documentation guidance, and training course outlines.[1] Today, more than 95% of emergency medicine residencies teach US, and approximately 30% of community hospitals have instituted bedside US performed by emergency physicians.[21,22] New applications are being described, including procedural guidance.

TRAINING, CREDENTIALING, AND ACCREDITATION

Emergency US is one of three competency assessments required of EM residents by the Residency Review Committee for Emergency Medicine.[6,23,24] For emergency physicians in practice, initial training often takes place through continuing medical education courses followed by a period of proctoring or supervision.[1,25]

APPLIED ULTRASOUND PHYSICS AND INSTRUMENTATION

By definition, US is sound greater than 20,000 Hz; US is a longitudinal, mechanical, and directional wave that is transmitted through mediums. US is kinetic energy, so all users of this technology should use the ALARA (as low as reasonably achievable) principle by performing procedures only when needed and limiting the time of sonographic investigation.[26]

US waves are characterized by their amplitude (deflection from a baseline), wavelength (the wave cycle distance, usually measured in microns), and frequency (the number of cycles per second). Modern diagnostic US transducers emit at millions of cycles per second or MHz. The frequency of the transducer can be adjusted to improve image quality; lower

frequencies penetrate deeper tissue at the expense of resolution, whereas higher frequencies improve resolution but cannot penetrate deeper structures.

M-mode or motion mode displays received waves over both time and distance and is used to calculate rates, speeds, and distances by sending a steered one-dimensional line across tissue. The most familiar display is B-mode or brightness mode, which graphs the amplitude of reflected US waves as shades of gray from black to white on a monitor screen. As sound hits reflective interfaces, it returns to the transducer at higher amplitudes than waves that continue through tissue. In addition, sound can be attenuated, refracted, and reverberated away from the transducer. These reflections or lack thereof create a brightness graph that is depicted in two planes creating the two-dimensional image. One can adjust the brightness of the image by changing the overall gain or change the amount of gain at certain depths, which is called time-gain compensation (TGC). This allows for a more discrete amplification of sound at different levels. By increasing overall gain or amplification and TGC at different levels, one can adjust for the loss of returning information.

US resolution can be described as axial, in the long axis of the transducer, or lateral, perpendicular to the long axis of the transducer. Typically, higher probe frequencies produce higher axial resolution. The focus of the transducer is the image area where the US beam produces the best lateral resolution. The area from the probe to the focus is called the near field, and that from the focus to the end of the image is called the far field. The best imaging with maximum reduction in artifacts occurs at the focus.

Focal zones can be increased for more resolution, but frame rate (the speed at which the screen updates the image) will decrease, causing a slow-motion image that is poor for imaging moving tissues. One can also change from a sector transducer (a curved abdominal or phased array transducer) to a linear transducer for better lateral resolution on the edges of the transducer.

Some machines allow the adjustment of dynamic range, which is the ratio of initial intensity and final intensity of returning echoes. Adjusting dynamic range allows for more contrast (e.g., echocardiography) or more gray scale (e.g., soft tissue). Harmonics is another technology that listens for multiples of the frequency sent out and often cleans up images near the screen such as in the gallbladder, apex of the heart, or soft tissue of skin.

Doppler US shows the velocity of moving structures, typically red blood cells, thus representing flow. Color flow Doppler shows direction (by red or blue colors representing flow in opposite directions) and velocity of flow (by degrees of brightness of the color, where lighter is higher velocity). Power Doppler or power angiography is also a representation of velocity of flow, but it describes only the presence of flow and not direction.

Gray scale US has the best resolution when the direction of the probe and sound waves are perpendicular to the object because the sound is reflected back up to the transducer. Doppler US has the best resolution with the probe parallel or less than 60 degrees from the direction of blood flow. This often means realignment by tilting of the probe to create better imaging based on the mode of US.

Other US terms and artifacts commonly used are listed in Table 197-1.

Table 197-1 Common Definitions

Window—soft tissue where transducer is placed to interrogate tissue in the body
Anechoic—without sounds (black)
Echogenic—with sounds (white)
Hyperechoic—with more reflected sounds than adjacent tissue (more echogenic)
Hypoechoic—with less sound than adjacent tissue (less echogenic)
Shadowing—sound blocked by a reflective barrier
Enhancement—more echogenicity due to increased velocity of sound, typically behind a fluid-filled structure such as the bladder or gallbladder
Reverberation—an artifact that results from multiple reflections at a given interface, often in parallel
Mirror image—a propagation speed artifact that repeats images that are beyond a strong reflector (e.g., diaphragm), creating a mirror image
Lateral cystic shadowing—a refraction artifact seen on the edge of cystic structures causing an artificial shadow
Beam width artifact—a volume-averaging artifact causing artifactual echoes in the inferior aspect of anechoic structures or spaces

EQUIPMENT

A variety of US machines are available, including palm size, laptop, cart based, and combination devices. US machines have controls for gain, depth, and freeze and have print or acquire image controls. Additional controls including TGC, frame rate, measurement, dynamic range, M-mode, power Doppler, color directional Doppler, spectral Doppler, patient ID, transducer selection, and image review. Images are produced in digital stills, digital video clips, thermal paper stills, or DVD or video recordings.

Modern multifrequency probes can select among several US frequencies to maximize either tissue penetration or image quality depending on the application. Probe design and footprints (the actual transducer surface area touching the patient) can be divided into three categories: flat linear array probes, curved linear array probes, and phased array probes. Flat linear probes give a square or rectangular picture with excellent lateral resolution at the expense of width of field. Curved linear array probes, which give a wider field of view at the expense of lateral resolution, are usually used for deep tissue penetration applications, such as evaluation for pregnancy or abdominal aortic aneurysm. The endocavitary probe is a highly curved linear array probe that is most commonly used for endovaginal evaluation. Phased array probes offer a smaller footprint than curved linear array probes and were originally developed for cardiac evaluations, but they can also be used for abdominal studies. They offer excellent image quality for the evaluation of moving structures but with worse resolution of static structures.

APPLICATIONS AND CATEGORIZATION

In 1994, SAEM categorized US applications broadly into abdominal, cardiac, obstetric/gynecologic, and special applications.[18] In 2001, ACEP developed specialty-specific guidelines that categorized applications more narrowly to include trauma, pregnancy, abdominal aortic aneurysm (AAA), and cardiac for pericardial effusion and cardiac activity, biliary, renal, and procedural applications.[1] Recent studies have investigated emergency US in diagnosing deep vein thrombosis (DVT) and pneumothorax.[4] New applications include soft tissue, ocular, and musculoskeletal studies.[27-32] Therapeutic applications of transcranial Doppler are being studied by emergency physicians.[33]

The most common uses of US by community emergency departments in decreasing order are trauma, cardiac (cardiac arrest and pericardial effusion), AAA, pelvic, biliary, procedural, renal, and DVT.[22]

Procedural guidance can assist many emergency percutaneous procedures such as vascular access, torso cavities evacuation (e.g., thoracentesis, paracentesis, and pericardiocentesis), arthrocentesis, lumbar puncture, abscess drainage, nerve blocks, and other percutaneous procedures.[4]

Emergency US is used in clinical pathways or algorithms to rapidly identify or exclude emergency medical conditions.[34,35]

Trauma Ultrasound

The trauma US exam is also called the focused assessment with sonography in trauma (FAST) examination.[4,36,37] The trauma examination originally focused on the peritoneal space, as an evaluation for hemoperitoneum, using the right flank, left flank, and pelvic windows for detection of dependent peritoneal fluid and pleural effusion.[38,39] Newer versions include the EFAST (extended FAST) for the evaluation of potential pneumothorax and FASTER for evaluation of the extremities.[40]

The FAST exam is based on the premise that fluid within the peritoneum circulates throughout the abdomen and pelvis and settles in dependent spaces.[41,42] The volume of fluid required for a positive US depends on the site of injury and site of sonographic detection but generally 250 mL or greater is required, and nearly 600 mL of fluid is necessary to cause a positive flank stripe when fluid is from the pelvis.[43] Pericardial fluid is contained within the parietal and visceral pericardium. Once a certain volume is reached, the pressure in the pericardial space increases exponentially, causing pericardial tamponade.[44] Generally, 50 mL is required to cause a hemodynamic compromise in a patient without prior pericardial inflammation. However, tears in the pericardium that communicate with the pleural space or even the peritoneum can cause false-negative exams. Pleural fluid can be detected by US, but this is dependent on the position of the body.[45] Pneumothorax detection is based on the absence of the normal sonographic sliding of the visceral and parietal pleura.

The indications for the torso trauma US examination (often called the EFAST) include the need to detect any of the following in an injured or ill patient: pathologic free intraperitoneal fluid (typically hemoperitoneum, uroperitoneum, bile, or bowel contents), hemopericardium, hemothorax, or pneumothorax.[46] Typically, fluid in the peritoneum, pericardium, and pleural cavity is anechoic, but it can have echogenicity with clotting, depending on the age of the clot. Compared with other fluid-filled structures in the abdomen and pelvis, peritoneal free fluid generally has sharp edges and an irregular shape, whereas most visceral or vascular structures have intrinsically smooth oval contours and less abrupt edge detail.

The FAST technique uses a low- to middle-frequency probe (2–5 MHz) to evaluate dependent peritoneal spaces, pleural spaces, and the pericardium. Within the peritoneum, dependent spaces include the following, grouped by tissue window: right flank—this evaluates the hepatorenal space, also called Morison's pouch (Fig. 197-1), the right subphrenic space, and the right costophrenic angle; left flank—this evaluates the perisplenic space between the diaphragm and spleen, the splenorenal space, the perirenal space, and the left subphrenic space; and suprapubic view—this view is performed by placing the transducer superior to the pubic bone with a full bladder to visualize the pouch of Douglas in females and the retrovesical space in men. Extra views include the paracolic gutters inferior from the flank views and medial from the iliac spine to visualize free fluid surrounding the bowel.[47]

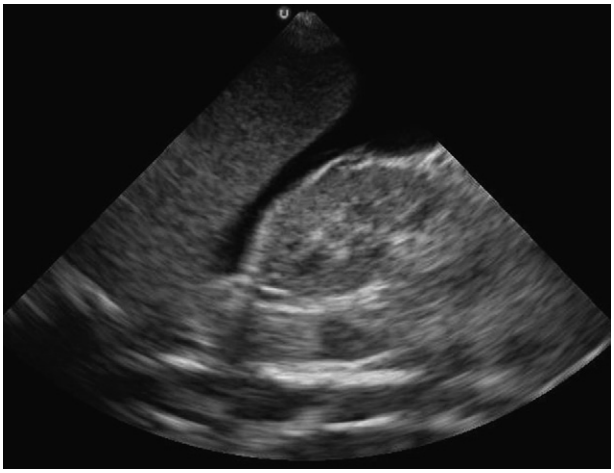


Figure 197-1. Free fluid in the peritoneum in Morison's pouch seen from right flank.

The typical cardiac views include the transverse subcostal images, which visualize the four cardiac chambers and pericardial space, whereas the sagittal subcostal view assesses the pericardial space and the inferior vena cava with regard to its collapsibility and diameter, indicating right heart pressures. A good alternative in patients with long chest walls or obesity is the parasternal view, which assesses the anterior and posterior pericardial spaces.

For pleural fluid, the superiorly angled flank views allow visualization of the costophrenic angles. The evaluation for pneumothorax utilizes either a low-frequency probe at a shallow depth or, more typically, a high-frequency probe for better resolution, placed at the anterior chest in the second intercostal space for anterior pneumothorax and at the axilla for large pneumothoraces and pleural effusion.

The sensitivity of the FAST exam ranges from 60 to 99%, and its specificity ranges from 80 to 99%.[48-50] The large range of sensitivity is related to the comparative gold standard used for each study, with clinical endpoints suggesting higher accuracy. The trauma US does not capture every peritoneal injury, although the need for intervention on undetected injuries has not been studied. Randomized, controlled trials show reduced morbidity, time to operating room, and charges for patients with US use during resuscitation,[51,52] and a positive US increases the odds ratio of laparotomy.[53]

Patients with traumatic pericardial effusions have reduced times to operative management and reduced mortality rates with US,[44] but the mediastinal injuries more common in blunt trauma have not been assessed without transesophageal echocardiography.[54] Pleural effusion has been studied with variable accuracy.[55] US diagnoses pneumothorax with excellent sensitivity and specificity compared to plain chest x-ray and variable accuracy compared to computed tomography scanning.[56]

There are certain patient populations in whom FAST evaluation has limited performance. In patients with penetrating chest injuries, the use of the FAST exam seems to be efficacious for both pericardial and nearby peritoneal injuries,[57] but in patients with purely anterior penetrating trauma to the abdomen, the sensitivity of trauma US is poor because significant bowel injuries can occur without significant hemoperitoneum.[58,59]

The accuracy of the FAST exam in children is very similar to that in adults, but caution is advised because many pediatric

injuries can be observed or treated nonoperatively with angiography.[60] In obstetric patients, the few observational studies that have been performed have shown reasonable sensitivity and specificity, although abruption and fetal viability may necessitate an earlier operative course.[61]

In patients with pelvic fractures, there have been mixed results, with sensitivity from 20 to 80%.[62-64] More important, the detection of free fluid in an unstable patient with a pelvic fracture may be due to uroperitoneum from bladder injury rather than hemoperitoneum from vascular injury, clouding the decision for laparotomy versus pelvic embolization.[64] In addition, retroperitoneal injuries to the genitourinary tract are not assessed with four-quadrant FAST exam.

Pelvic Ultrasound

Pelvic US by emergency physicians was initiated to address one of the epidemics of the modern era—ectopic pregnancy.[65-69] Typically, pelvic US is used to confirm intrauterine pregnancy, thereby indirectly excluding ectopic pregnancy in the vast majority of patients.[70,71] Other uses during pregnancy include detection of fetal viability, incomplete abortion, ectopic pregnancy, and molar pregnancy. Nonpregnancy pelvic US uses include the detection of tubo-ovarian abscesses, masses, and hemoperitoneum in the hemodynamically unstable patient.[72]

Indications for sonographic evaluation of the first-trimester pregnant patient include symptoms or signs that suggest an ectopic pregnancy, molar pregnancy, fetal demise, or for dating the pregnancy. Standard definitions for intrauterine pregnancy describe a gestational sac with a yolk sac or fetal pole within the fundus of the uterus (Fig. 197-2).[70] An embryonic or fetal demise has US findings of a fetal pole greater than 5 mm without fetal heart rate or a gestational sac greater than 20 mm in diameter without a fetal pole. A molar pregnancy appears as an echogenic, cystic uterus with disorganized echoes and is associated with high β-hCG concentrations without the sonographic finding of intrauterine pregnancy. Findings of an ectopic pregnancy include a chorionic ring or gestational sac with evidence of a yolk sac or fetal pole outside the uterus or in an abnormal location in the uterus, such as the cornu or cervix.[73] Excluding the groups of IUP, embryonic demise, molar pregnancy, and ectopic pregnancy, there is a class called "indeterminate," which may account for 20% of pregnant patients presenting to the ED in the first and early second trimesters. Heterotopic pregnancies can occur at a rate of 1/5000 and be detected by the same techniques and definitions.[74]

Pelvic US is performed by either transabdominal or endovaginal techniques. The transabdominal technique utilizes a low-frequency transabdominal transducer placed over the lower abdomen suprapubically. Ideally, the patient has a full bladder, which provides a sonographic window, but this may not be necessary if the uterus is large or the patient is thin. Advantages of the transabdominal technique include wider field of view, detection of large pelvic masses, and greater depth of field. In the endovaginal technique, the transducer is placed in the vagina, optimally with an empty bladder to visualize the same structures. Endovaginal transducers are high frequency, providing excellent axial resolution but poor penetration of distant structures. Early intrauterine yolk sacs and fetal poles within gestational sacs may be detected more clearly, and ectopic pregnancies can be identified with more accuracy. Endovaginal US can detect small (7 mL) amounts of peritoneal fluid. Endovaginal US in the hemodynamically compromised patient has replaced culdocentesis for detection of ruptured ectopic pregnancy.

Emergency physician-performed pelvic US has reduced the morbidity of ectopic pregnancy by shortening the time to diagnosis and operating room treatment, and it has resulted in greater initial detection of abnormal pregnancy, reduced emergency department patient throughput times, and increased patient satisfaction with emergency department care.[66,68,75-78]

Pelvic US in nonpregnancy states has shown good accuracy for tubo-ovarian abscess and improved decision-making for female patients with right lower abdominal pain.[79]

Cardiac Ultrasound

The use of cardiac US by emergency physicians is presentation and symptom specific and focused in nature. Indications include cardiac arrest, possible pericardial effusion, trauma, chest pain, hypotension, and for procedural guidance. Cardiac US is often combined with other applications to form algorithms and protocols for certain symptoms or signs, such as dyspnea or hypotension.

Cardiac US is performed through the transthoracic and transabdominal windows using small curvilinear or phased array transducers. Typical views include the subcostal four-chamber and long-axis views, parasternal long-axis view, parasternal short-axis view, and apical four-chamber view. Although most emergency physicians are very comfortable with the subcostal view from the FAST exam, the parasternal long-axis view is a good alternative and good window for left ventricular assessment.[80,81] In addition, the apical four-chamber view provides excellent comparison of the right and left ventricles in terms of size and function.

Cardiac US performed by emergency physicians shows good accuracy in detection of pericardial effusion (Fig. 197-3),

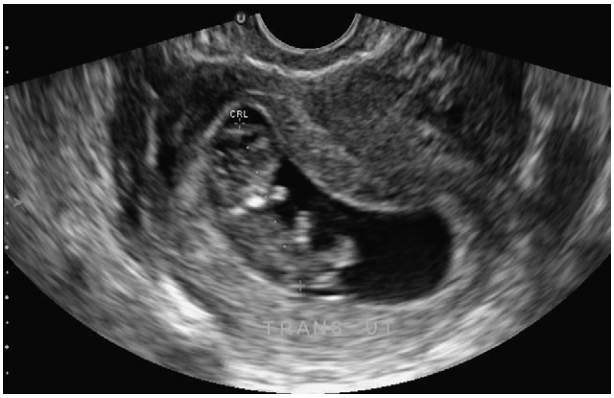


Figure 197-2. Intrauterine pregnancy with fetus on endovaginal scanning.

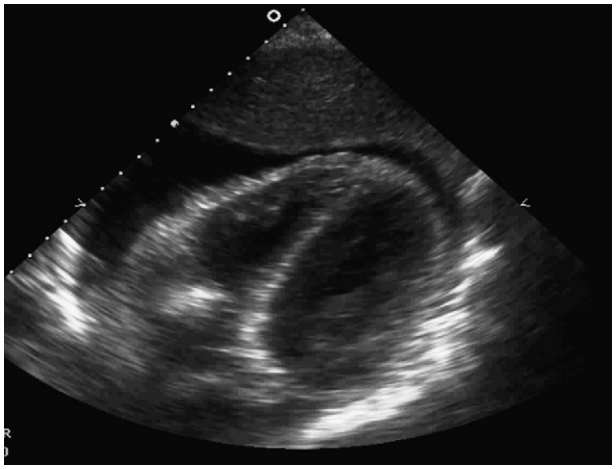


Figure 197-3. Pericardial effusion in subcostal view.

assessment of left ventricular function, and evaluation of patients with undifferentiated shock. In addition, aspects of cardiac US are being used for the assessment of intravascular volume status, cardiac output, and in the evaluation of dyspnea.

In cardiac arrest, US detects ventricular motion in both asystole and pulseless electrical activity (PEA).[82-86] In asystole, two studies have shown poor prognosis in the absence of sonographic ventricular activity.[84,85] Use of cardiac US in PEA and near-PEA states can be diagnostic for pericardial effusion.[82] Cardiac US can detect pseudo-asystole by revealing ventricular fibrillation or coordinated cardiac contractions without a palpable pulse.[86] Cardiac US can detect ventricular capture when the patient is paced, transcutaneously or by a transvenous pacer. It can also identify pneumothorax, another treatable cause of cardiac arrest.[83]

Emergency cardiac US is diagnostic for detection of both medical and traumatic pericardial effusions.[87] In patients with dyspnea, pericardial effusion is detected with excellent accuracy.[88] In trauma, use of cardiac US reduces time to operative intervention and reduces mortality in patients with penetrating cardiac injury.[44] Detection of effusions in blunt trauma has also been described. Patients with cardiac US performed by emergency physicians for pericardial effusion have significantly reduced hospital length of stay and charges.[89]

Cardiac US is used for detection of pericardial effusions, chamber enlargement, and global activity in chest pain syndromes.[90] In chest pain, US has been studied for the evaluation of pericardial tamponade, pulmonary embolus, cardiogenic shock, aortic dissection, pneumothorax, and bony chest wall fracture.[87,91,92]

US protocols have been developed to evaluate patients with undifferentiated hypotension.[93-97] Cardiac US windows with the addition of abdominal views can assess for effusion, global ventricular activity, ventricular chamber size, inferior vena cava (IVC) size and respiratory change, peritoneal fluid, and abdominal aortic aneurysm. Such a combined US protocol rapidly narrows the differential diagnosis. Sepsis is the most common diagnosis for patients with hyperdynamic ventricular activity.[97]

Central pressures can be estimated by examining the IVC size and collapsibility.[98,99] Several studies show that this technique has good accuracy in assessing blood loss and as a marker of hypovolemia. Correlation studies have show moderate agreement between CVP and IVC measurements, with actual cardiac qualitative assessment as good as or better than the IVC measurements.

Cardiac US is also a procedural guide for placement of transvenous pacer wires and for pericardiocentesis. Transvenous pacer wires are often placed through central veins, and placing them into the right ventricular apex can be difficult. US facilitates placement by imaging the wires in real time as they pass through the tricuspid valve and approach the apex of the right ventricle. US can also document ventricular capture. The probe is placed in the subxiphoid space for both applications.

Abdominal Vascular Ultrasound

Abdominal aortic aneurysm is another silent disease for which US has been used by emergency physicians for diagnosis and management of patients with flank, abdominal, or back pain and for evaluating unexplained hypotension in the older patient.[100-103] The use of US to detect aortic dissection in the chest and the abdomen has been described but is not common.[104-106]

The technique involves imaging of the aorta from the subxiphoid space to the umbilicus to evaluate for dilation above a diameter of 3 cm (Fig. 197-4). Due to the increased incidence of infrarenal aneurysm, the technique must visualize the aorta from diaphragm to the aortic bifurcation. Fusiform aneurysms are more common, but saccular aneurysms can also be present, necessitating imaging in both the transverse and longitudinal planes.[107] If an aneurysm is found, a peritoneal view of Morison's pouch is performed to detect intraperitoneal fluid.

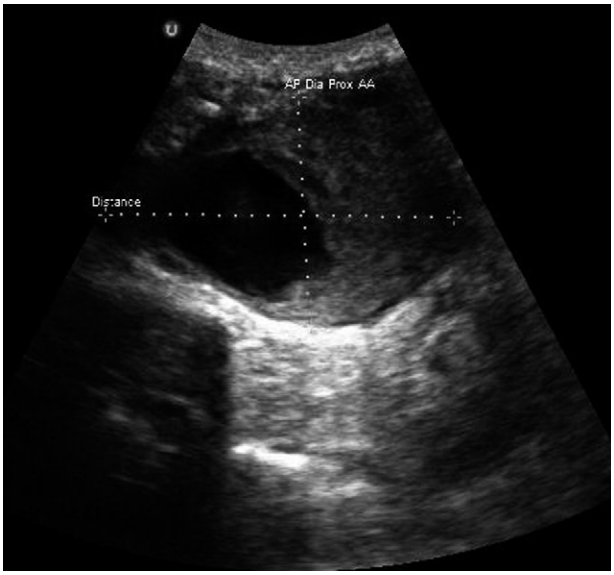


Figure 197-4. Large abdominal aortic aneurysm with echogenic intraluminal clot.

Emergency physician use of US for AAA detection has shown good accuracy compared to other imaging modalities and laparotomy.[100-102,108] It also has management implications in older patients with lower back pain, where elimination of the presence of AAA can lead to other management decisions. Screening for AAA in the emergency department with US has been shown to be effective.[109]

Aortic dissection may be detected by a combination of abdominal and cardiac scanning.[106] Anywhere in the aorta, a linear echogenic flap across the lumen of the aorta is suggestive of dissection, with color Doppler possibly detecting different flows on either side of the flap. The cardiac US signs include unexplained pericardial effusion; a dilated aortic root; aortic insufficiency; and a linear echogenic flap seen in the ascending aorta, aortic arch (via a suprasternal notch), or descending aorta.

Biliary Ultrasound

Biliary US to detect gallstones and associated cholecystitis was one of the early applications in emergency medicine.[110-112] The sonographic technique involves a modified biplanar approach to the right upper quadrant to evaluate the gallbladder and surrounding area. A complete evaluation includes visualizing the common bile duct. The diagnosis of cholelithiasis is made after identification of echogenic foci within the gallbladder lumen with shadowing (Fig. 197-5). Other image patterns include stones that will not shadow, sludge, and the wall echo sign of a gallbladder full of gallstones. Signs of cholecystitis include a dilated gallbladder, increased gallbladder wall thickness, sonographic Murphy's sign, and pericholecystic fluid. A nonmobile stone in the gallbladder neck is highly suggestive of eventual cholecystitis.[113] A common bile duct

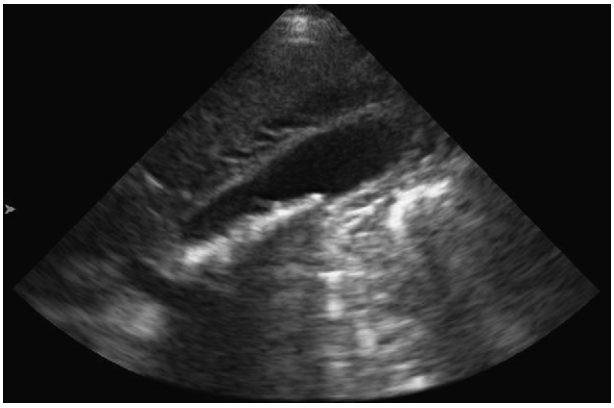


Figure 197-5. Echogenic layering gallstones seen in gallbladder with posterior shadowing.

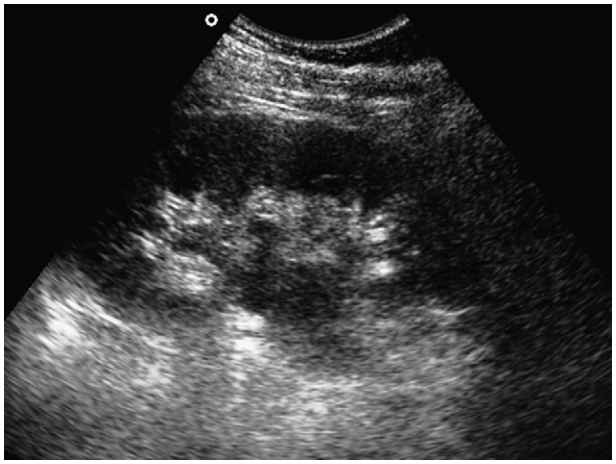


Figure 197-6. Hydronephrosis in renal pelvis and calyces.

greater than 6 mm in people younger than 60 years and less than 10 mm in elders may indicate choledocholithiasis. Biliary US has been shown to be fast and accurate, with a sensitivity of 94% and a specificity of 96% in detecting gallstones.[110,114,115] Biliary US has a primary role compared to nuclear medicine testing.[112]

Renal Ultrasound

Renal or urinary tract US is one of the early applications for the diagnosis of urinary obstruction, resulting in hydronephrosis. The lack of ionizing radiation and the rapidity of renal US make it an attractive option for the investigation of unexplained flank, back, or groin pain.

The performance of renal US includes biplanar views of the kidneys with emphasis on dilation of the calyceal system and pelvis of the respective kidney. In addition, visualization of the bladder can diagnose secondary hydronephrosis from an obstructed bladder stone and may demonstrate nonobstructive bladder jets through the use of Doppler US. The windows for the two kidneys are very similar to the trauma flank series with the exception that the patient may be rolled on the opposite side so that the transducer may be placed more posteriorly on the back if needed. The bladder view is performed suprapubically, and calculations of volume may be made with on-machine calculators or by formulas.

Renal US has a sensitivity of 83% and a specificity of 92% in detecting hydronephrosis (Fig. 197-6).[116,117] Genitourinary tract obstruction protocols have shown great sensitivity in eliminating renal obstruction from a differential diagnosis.[118] Bladder US is useful for detection of a full bladder and the presence of a Foley catheter and also for procedure guidance (superpubic aspiration or Foley placement).[119-124]

Extremity Vascular Ultrasound

The swollen extremity often requires sonographic imaging to assess for DVT. Emergency physicians commonly utilize compression US to rule out DVT.[125-128]

The two-level compression technique involves visualizing the compressibility of the common femoral and popliteal veins, whereas the three-level technique adds visualization of the compressibility of the junction of the superficial femoral and deep femoral veins (Figs. 197-7 and 197-8). Upper extremity veins have been investigated, but this is not a common application because Doppler is needed for sampling of the subclavian vein, which is not compressible under the clavicle.[129]

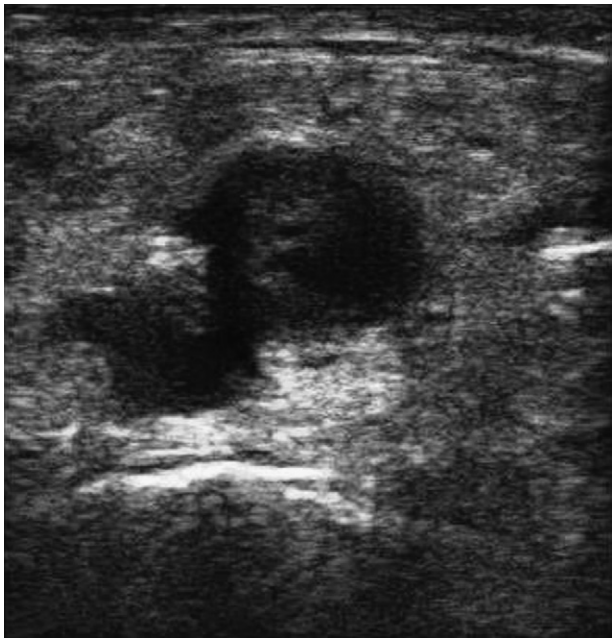


Figure 197-7. Deep vein thrombosis with layering clot in popliteal vein seen on compression ultrasound.

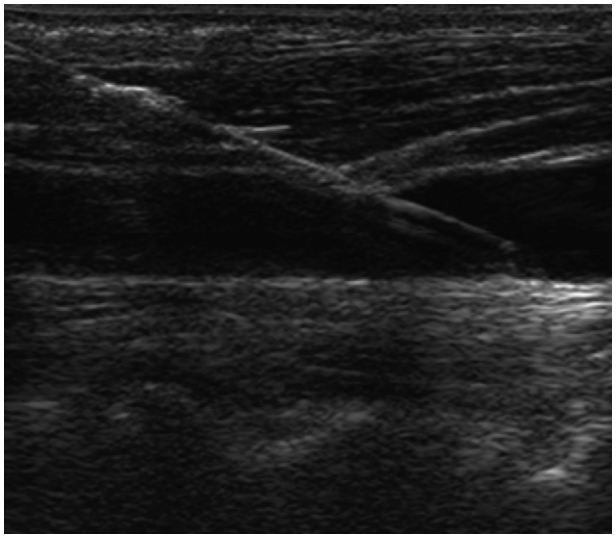


Figure 197-8. A needle in an internal jugular vein for procedural guidance.

The accuracy of this technique ranges from 70 to 99%, depending on operator experience.[130,131] Hospital charges and time in the emergency department are reduced for patients who have venous US performed by emergency physicians to rule out DVT.[132,133]

Thoracic and Tracheal Ultrasound

Thoracic applications include the detection of pleural effusion, pneumothorax, as well as other pathologic lung states. The technique of thoracic US utilizes a low-frequency probe to survey for pleural effusions and a high-frequency probe to detect pleural lines and related artifacts. Pleural fluid appears as an anechoic collection above the diaphragm. In addition, the lung may be collapsed and visualized as an echogenic floating structure. Normally, the parietal and visceral pleural lines slide against each other, and lack of sliding is the finding most consistent with pneumothorax.[134,135] Confounding factors include adhesions of the pleura, chronic obstructive pulmonary disease, and prior pneumothorax. A lung point sign is the edge of the pneumothorax where the lung is still adherent to the parietal pleura, and part of the image shows no sliding until the lung moves into the interspace with respirations.[136] The accuracy of US for detection of pneumothorax is better than that of plain chest x-ray in the acute setting, but there are concerns regarding reduced accuracy after 24 hours.[137-139]

Severe pneumonia is visualized as echogenic "liver-like" echogenicity as the lung accumulates fluid with consolidation. Peripneumonic collections are common and may indicate inflammation.

Pulmonary edema is indicated by the presence of comet tails, which are reverberation artifacts that reflect from the parietal pleural interface into the lung. Normally found in dependent areas of the lung, the widespread distribution of these artifacts in an apical lung may indicate increased lung congestion.[140,141]

Tracheal US has been explored for the confirmation of endotracheal intubation with relatively good sensitivity and specificity.[142,143] The dynamic movement of the endotracheal tube creates a flutter through the recognized shadow of the airway, but static US seems to lack accuracy.[144,145] This technique may have a role in patients in cardiac arrest or with equivocal end-tidal carbon dioxide, and it has been used in pediatric patients with significant decreased time to confirmation compared with chest radiography.[146]

Ocular Ultrasound

Ocular US has been described for intraocular pathology such as retinal detachment, retinal hemorrhage, vitreal hemorrhage, intraocular foreign body, dislocated lens, and retro-orbital hemorrhage.[30] The eye is an excellent acoustic window, and short-duration gray scale US over a closed eyelid can visualize both the anterior and the posterior chamber well. In addition, the optic nerve sheath diameter can be measured behind the eye, reflecting intracranial pressure.[32,147-149]

Soft Tissue Ultrasound

US in soft tissue is facilitated due to the lack of air and bone in the skin and subcutaneous tissue except in the hand and foot. Soft tissue US is used to differentiate cellulitis from abscess, for detection of foreign bodies and hernia, and for the evaluation of other soft tissue pathology.[27,28,150] The technique involves the use of a high-frequency linear transducer to image the soft tissue from normal skin to the abnormal area. Cellulitis or edema will cause an echogenic pattern with cobblestoning between fat lobules. Abscesses are irregular hypoechoic to anechoic collections within the subcutaneous layer but may connect with the surface. US is diagnostic in soft tissue disease states such as cellulitis, abscess, and necrotizing fasciitis.[150-153] In the ABSCESS study, clinical exam had a sensitivity of 86% and specificity of 70%, whereas US had a sensitivity of 98% and specificity of 88%. Management in half of the patients with clinical cellulitis was changed based on results of US.[27]

US can detect peritonsillar abscess with differentiation of cellulitis versus abscess, and it can be used for guidance of peritonsillar aspiration.[154] It can also detect early Ludwig's angina with abscess in the soft tissues and fascia of the face.[155]

Detection of foreign bodies is characterized by variable echogenicity in the tissue with unexpected shadowing beneath the foreign body. Metal foreign bodies may have characteristic highly reflective echogenicity and ring-down artifacts. Bedside US has had variable accuracy for detection of foreign bodies in simulated cases but better accuracy in clinical settings.[156-158] Use of a water bath may aid the detection of superficial foreign bodies.[159]

Musculoskeletal Ultrasound

The use of US for musculoskeletal disorders is focused on joint effusion and fractures. US is excellent for detecting fluid in joints and confirming effusions and guiding drainage procedures.[29,160,161] Joint effusions typically are anechoic or echogenic, depending on type and age. Diagnosis of ligamentous injuries and muscular avulsion and hemorrhage has also been reported.[162-164] Muscular and tendon abnormalities are detected by anechoic and heterogeneous abnormalities. Detection is often assisted by movement of the limb. Tendons and their anistrophic longitudinal fibrils can be visualized near joints, with pathologic tears appearing as a discontinuity of the tendon. Muscles are hypoechoic with echogenic borders. Tears or hemorrhage may be seen as interruption of this normal pattern.[164] Water bath techniques provide a better acoustic window for very shallow tissue such as fingers.

In addition, the characteristic reflection of bone with immediate shadowing can be used to visualize normal bone and its contour. Detection of fractures requires identification of a defect in the bony cortex.[40,164-166] The ability to image the bony cortex can be useful for guidance of fracture reduction.[167] Comparison with the contralateral side may also be helpful.

US is accurate in the detection of fractures, joint effusions, and hematomas.[160,167-169] Prospective evaluation of finger fractures demonstrates good accuracy.[169] Other fractures studied include femur, sternal fracture, rib fracture, and forearm fractures.[40]

US is also used to guide anesthetic blocks and hematoma blocks.[170-172]

Transcranial Doppler Ultrasound

Transcranial Doppler US has been used by some emergency physicians for detection of abnormal flow patterns in the brain and for detection and treatment of middle cerebral artery strokes. An advanced procedure, typically using a phased array transducer over a bone or orbital window in the skull, color Doppler and spectral Doppler scanning is performed at different levels of intracranial vessels for detection of abnormal patterns.[173]

Testicular Ultrasound

Testicular scanning is an advanced US application that requires visualization of the normal and symptomatic testicle using a

high-frequency probe. The normal testicular structures, including the normal homogeneity of the testicle, are evaluated, and Doppler scanning is performed for evaluation of normal venous and arterial flow. Absence or a difference in flow from the unaffected side indicates possible torsion, whereas increased flow indicates epididymitis. Experienced emergency physicians accurately diagnosed testicular pain with a sensitivity of 95% and specificity of 94% in a study of 36 patients, which included 3 patients with torsion.[174]

Abdominal Bowel Ultrasound

The use of bedside US for evaluation of appendicitis, diverticulitis, and other bowel pathology is an area of increasing interest. There has been variable experience with the use of graded compression US to detect appendicitis, with reported sensitivity of 67% and specificity of 92%.[175] The novel use of US to detect diverticular disease and hernias has been described.[176,177]

Ultrasound for Procedural Guidance

In 2001, in response to the Institute of Medicine report "To Err Is Human," the Agency for Healthcare Research and Quality (AHRQ) sanctioned a report on actions that may improve patient safety.[178] The report contains a recommendation for US guidance for internal jugular central line insertion. Since then, the use of US for procedural guidance has expanded for almost every known emergency procedure, but especially vascular access.

There are several key concepts regarding procedural guidance with US. Procedural guidance can be static or dynamic.[179] Static guidance suggests that US has been placed over the anatomic area, and the area is marked while angle and distance information is noted. Dynamic guidance describes procedures performed with real-time US visualization of the needle entering the anatomic area.

There are two approaches to vein cannulation: transverse, where the vein appears as a circular structure on the screen, and longitudinal, where the vein appears as a tubular structure along the width of the screen. In the transverse approach, the probe's long axis is centered transverse (90 degrees) to the long axis of the anatomic area, guiding the needle to bisect the probe at its center gives centering and depth information. In the longitudinal axis, the probe is placed along the long axis of the anatomic area, and the needle is introduced in the long axis of the probe, giving depth and trajectory information.

US guidance for central line insertion has been the most studied application and most advocated use among US-guided procedures.[180-182] Both the AHRQ and the National Institute for Health and Clinical Excellence have recommended US guidance for central line insertion.[183,184] US guidance for internal jugular (IJ) central line insertion can be recommended as a best practice and safe practice.[180,185]

US permits the physician to assess the IJ for overlap with the carotid artery, vessel diameter, and the presence of luminal clot or vessel obliteration.[186] IJ central line insertion has been studied in the emergency department, intensive care unit, and radiology suite. In the emergency department, there is decreased time to flashback and improved success in the difficult stick patient and improved overall success rate, first attempt rate, reduced time to insertion, and reduced complication rate with US guidance for IJ cannulation.

Femoral vein insertion has been studied in cardiac arrest patients, and improved cannulation rate and reduced complications have been reported.[187] Several maneuvers can optimize the success rate of femoral vein cannulation, including reverse Trendelenburg position and placing pressure on the iliac vein proximal to the femoral vein.[188]

Studies of US-guided subclavian insertion suggest that US does not contribute to procedural success because of a lack of a convenient window for the subclavian vein. At the location where the vein is usually cannulated, visualization is obstructed by the clavicle. However, more laterally, toward the junction of the axillary vein and subclavian veins, the vein can be visualized in the chest wall.[189,190] Although it may be cannulated there, the more difficult challenge may be inserting the catheter through the pliable soft tissue of the anterior-superior chest.[191] The supraclavicular fossa is a window for central line insertion, and the subclavian and brachiocephalic veins may be imaged from this site.[192]

Emergency physicians are able to use US to insert peripheral intravenous catheters in difficult patients with high success rates.[193-195] Nurses have also been taught to use US for peripheral venous guidance.[196,197]

Arterial access, including radial artery aspiration and cannulation, is more successful with the use of US guidance.[198,199]

Numerous other procedures using US guidance have been described in emergency practice. They are listed in Table 197-2.

Table 197-2 Ultrasound Guided Emergency Procedures

ULTRASOUND	PROCEDURE
Vascular access	Central venous access[182,210,211] Internal jugular vein Subclavian near axillary vein Subclavian with superclavicular approach Femoral vein Peripheral vein[193,197,212] Basilic Brachial Cephalic Forearm veins Intraosseus needle[213] Arterial cannulation[198,214] Radial artery Arterial sampling[215]
Torso fluid collections	Paracentesis[216,217] Thoracentesis[218] Pericardiocentesis[219]
Cardiac	Pacer placement[220–223]
Musculoskeletal	Arthrocentesis[29,224,225] Hip Knee Other joints Fracture reduction Foreign body removal[226] Tendon sheath injection[227]
Soft tissue	Abscess drainage[228] Hernia reduction[229]
Anesthesia	Interscalene Peripheral nerves Axillary[192] Femoral nerve[230] Hematoma block
Airway	Endotracheal tube placement[231–234]
Urinary bladder	Superpubic aspiration and cystostomy[235–237] Foley guidance[238]
Neurologic	Lumbar puncture[239–243]

Out-of-Hospital Ultrasound: Disasters and Remote Settings

US has been used in various settings, including out-of-hospital, the military battlefield, disaster settings, critical care settings, and international medicine settings. The use of US has been described in European and other settings that utilize physicians in the out-of-hospital arena, including ground, helicopter, air, and space.[9,200-204]

The military was a source of funding for small compact US machines and has been an extensive user of US in combat situations, including the frontline, combat support hospital, and tertiary hospital settings.[8,205,206]

US in disaster and other mass casualty situations has been described, including their use during the Armenian earthquake of 1988. With smaller, more compact machines, US capability may become part of disaster facilities and plans.[207]

INTEGRATION INTO EMERGENCY MEDICINE PRACTICE

US technology has rapidly been integrated into emergency medicine practice due to the need for effective, noninvasive, nonpainful, portable imaging techniques.[208] US, whether introduced in medical school, residency, or clinical practice, is a skill that requires constant attention with hands-on practice and interpretation.[15,209] However, with the integration of US into emergency medicine practice, emergency care has become more rapid, efficient, safe, and accurate.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 21 Abdominal Pain

Rimon N. Bengiamin, Gavin R. Budhram, Kelly E. King, and John M. Wightman

PERSPECTIVE

Abdominal pain is a common emergency department (ED) complaint but, for many reasons, is often diagnostically challenging. The nature and quality of abdominal pain may be difficult for the patient to convey. Physical examination findings with this complaint are variable and can be misleading. The location and severity of the pain may change over time. Benign-appearing symptoms and presentations may evolve into life-threatening conditions. Conversely, patients presenting with severe symptoms may carry a relatively benign diagnosis. All of these factors make evaluation of patients with acute abdominal pain challenging in the ED setting.

Epidemiology

Abdominal pain is a common presenting complaint, accounting for up to 10% of all ED visits. Some of the most common causes of acute abdominal pain are listed in Table 21-1. Many patients present with pain and other symptoms that are not typical of any specific disease process. A specific diagnosis may not be possible in about one in every four individuals presenting with this chief complaint.[1] In addition, several adult groups deserve special consideration: the elderly (older than 65 years of age), the immunocompromised, and women of reproductive age.

Elderly patients with acute abdominal pain are more likely to have a life-threatening process as the cause of their pain. Conditions such as diverticulitis, ruptured abdominal aneurysm, or mesenteric ischemia may manifest atypically and be rapidly progressive. Decreased diagnostic accuracy, coupled with increased probability of severe disease, results in increased mortality in elderly patients with abdominal pain.[2]

Increasingly, emergency physicians are seeing patients in immunocompromised states secondary to HIV/AIDS, chemotherapy, and immunosuppressive drugs. For many reasons, these patients also prove challenging. Their clinical presentation can be misleading owing to atypical physical and laboratory findings, such as lack of fever or elevated white count. In regard to infection, the scope of the differential diagnosis also should be broader than usual.[3–7] Presentations in the immunocompromised patient may be highly variable and subtle and are discussed in Chapter 181.

The evaluation of abdominal pain in women involves a differential diagnosis of considerable extent and often requires a more in-depth physical exam and further diagnostic testing. Pelvic organs may be the source of significant pathology in both the pregnant and the nonpregnant patient. The possibility of ectopic pregnancy in women of reproductive age greatly increases the risk of serious disease with a high potential for misdiagnosis. During pregnancy the uterus becomes an abdominal rather than a pelvic organ and may displace the normal intraperitoneal contents, adding complexity to the evaluation of these patients.[8] Nonpregnant patients require evaluation for various ovarian and uterine pathology states.

Pathophysiology

Pathology in the gastrointestinal and genitourinary tracts remains the most common source of pain perceived in the abdomen. Also, pain can arise from a multitude of other intra-abdominal and extra-abdominal locations (Box 21-1). Abdominal pain is derived from one or more of three distinct pain pathways: visceral, somatic, and referred.

Visceral pain results from stimulating autonomic nerves invested in the visceral peritoneum surrounding internal organs. It is often the earliest manifestation of a particular disease process. Distention of hollow organs by fluid or gas and capsular stretching of solid organs from edema, blood, cysts, or abscesses are the most common stimuli. This discomfort is poorly characterized and difficult to localize. If the involved organ is affected by peristalsis, the pain often is described as intermittent, crampy, or colicky. In general, visceral pain is perceived from the abdominal region that correlates with the embryonic somatic segment:

- *Foregut* structures (stomach, duodenum, liver, and pancreas) are associated with *upper abdominal pain*.
- *Midgut* derivatives (small bowel, proximal colon, and appendix) are associated with *periumbilical pain*.
- *Hindgut* structures (distal colon and genitourinary tract) are associated with *lower abdominal pain*.

Visceral pain can be perceived in a location remote from the actual disease process. Localization occurs with the extension of the disease process beyond the viscera. A classic example is that of the early periumbilical pain of appendicitis (midgut). When the parietal peritoneum becomes involved, the pain localizes to the right lower quadrant of the abdomen, the usual location of the appendix.

Somatic pain occurs with irritation of the parietal peritoneum. This is usually caused by infection, chemical irritation, or another inflammatory process. Sensations are conducted by

Table 21-1 Common Causes of Abdominal Pain

CAUSATIVE DISORDER/CONDITION	EPIDEMIOLOGY	ETIOLOGY	PRESENTATION	PHYSICAL EXAMINATION	USEFUL TEST(S)
Gastric, esophageal, or duodenal inflammation	Occurs in all age groups.	Caused by gastric hypersecretion, breakdown of mucoprotective barriers, infection, or exogenous sources.	Epigastric radiating or localized, associated with certain foods. Pain may be burning. In some cases, exacerbation in supine position.	Epigastric tenderness without rebound or guarding. Perforation or bleeding leads to more severe clinical findings.	Uncomplicated cases are treated with antacids or histamine H_2 blockers before invasive studies are contemplated. Gastroduodenoscopy is valuable in diagnosis and biopsy. Testing for *H. pylori* with blood or biopsy specimens. If perforation is suspected, an upright chest radiograph is obtained early to rule out free air. CT may be beneficial.
Acute appendicitis	Peak age in adolescence and young adulthood; less common in children and elders. Higher perforation rate in women, children, and elders and in pregancy. Mortality rate is 0.1% but increases to 2–6% with perforation.	Appendiceal lumen obstruction leads to swelling, ischemia, infection, and perforation.	Epigastric or periumbilical pain migrates to RLQ over 8–12 hr (50–60%). Later presentations associated with higher perforation rates. Pain, low-grade fever (15%), and anorexia (80%) common; vomiting less common (50–70%).	Mean temperature 38° C (100.5° F). Higher temperature associated with perforation. RLQ tenderness (90–95%) with rebound (40–70%) in majority of cases. Rectal tenderness in 30%.	Leukocyte count usually elevated or may show left shift. Urinalysis may show sterile pyuria. CT is sensitive and specific. US may have use in women, pregnancy, and children with RLQ pain.
Biliary tract disease	Peak age 35–60 yr; rare in patients younger than 20. Female-to-male ratio of 3:1. Risk factors include multiparity, obesity, alcohol intake, and use of birth control pills.	Passage of gallstones causes biliary colic. Impaction of a stone in cystic duct or common duct causes choecystitis or cholangitis.	Crampy RUQ pain radiates to right subscapular area. Prior history of pain is common. May have nausea or postprandial pain. Longer duration of pain favors diagnosis of cholecystitis or cholangitis.	Temperature normal in biliary colic, elevated in cholecystitis and cholangitis. RUQ tenderness, rebound, and jaundice (less common) may be present.	WBC count elevated in cholecystitis and cholangitis. Lipase and liver function tests may help differentiate this from gastritis or ulcer disease. Ultrasound shows wall thickening, pericholecystic fluid, stones, or duct dilatation. Hepatobiliary scintigraphy diagnoses gallbladder function.
Ureteral colic	Average age 30–40 yr, primarily in men. Prior history or family history of stones is common.	Family history, gout, *Proteus* infection. Renal tubular acidosis and cystinuria lead to stone formation.	Acute onset of flank pain radiating to groin. Nausea, vomiting, and pallor are common. Patient usually writhing in pain.	Vital signs usually normal. Tenderness on CVA percussion with benign abdominal examination.	Urinalysis usually shows hematuria. Noncontrast CT is sensitive and specific. US with fluid bolus useful diagnostically.

Diverticulitis	Incidence increases with advancing age, affects males more often than females. Recurrences are common. Often called "left-sided" appendicitis.	Colonic diverticula may become infected or perforated or cause local colitis. Obstruction, peritonitis, abscesses, fistulas result from infection or swelling.	Change in stool frequency or consistency commonly reported. LLQ pain is common. Associated with fever, nausea/vomiting; rectal bleeding may be seen.	Fever usually of low grade. LLQ pain without rebound is common. Stool may be heme-positive.	Results on most tests usually normal. Plain radiographs may show obstruction or mass effect. CT is often diagnostic.
Acute gastroenteritis	Common diagnosis. Seasonal. Most common misdiagnosis of appendicitis. May be seen in multiple family members. History of travel or immune compromise.	Usually viral. Consider invasive bacterial or parasitic in prolonged cases, in travelers, or immune-compromised patients.	Pain usually poorly localized, intermittent, crampy, and diffuse. Diarrhea is key element in diagnosis; usually large-volume, watery. Nausea and vomiting usually begin before pain.	Abdominal examination usually nonspecific without peritoneal signs. Watery diarrhea or no stool noted on rectal examination. Fever is usually present.	Usually symptomatic care with antiemetics and volume repletion. Heme-positive stools may be a clue to invasive pathogens. Key is not using this as a "default" diagnosis and missing more serious disease.
Constipation and obstipation	More common in females, the elderly, the very young, and patients on narcotics.	Idiopathic or hypokinesis secondary to disease states (low motility) or exogenous sources (diet, medications).	Abdominal pain; change in bowel habits.	Variable, nonspecific without peritoneal signs. Rectal exam may reveal hard stool or impaction.	Radiographs may show large amounts of stool. This is a diagnosis of exclusion.
Nonspecific abdominal pain	More common in persons of young and middle age, women of childbearing age or persons of low socioeconomic status, and patients with psychiatric disorders. Up to 10% of patients older than 50 years of age will have intra-abdominal cancer.	Unknown. Early or undiagnosed presentation of pathologic conditions.	Variable but tends to be chronic or recurrent.	Variable but no peritoneal signs. Rectal exam should be done to evaluate for subtle signs of pathology, including heme-positive stool, fistulas, and fissures.	Variable and often can be done on an outpatient basis.

CT, computed tomography; CVA, costovertebral angle; LLQ, left lower quadrant; LUQ, left upper quadrant; RLQ, right lower quadrant; RUQ, right upper quadrant; US, ultrasonography; WBC, white blood cell.

BOX 21-1 IMPORTANT EXTRA-ABDOMINOPELVIC CAUSES OF ABDOMINAL PAIN

Thoracic
Myocardial infarction/unstable angina
Pneumonia
Pulmonary embolism
Herniated thoracic disk (neuralgia)
Pericarditis/myocarditis

Genitourinary
Testicular torsion

Abdominal Wall
Muscle spasm
Muscle hematoma
Herpes zoster

Infectious
Streptococcal pharyngitis (more often in children)
Rocky Mountain spotted fever
Mononucleosis

Systemic
Diabetic ketoacidosis
Alcoholic ketoacidosis
Uremia
Sickle cell disease
Porphyria
Systemic lupus erythematosus
Vasculitis
Glaucoma
Hyperthyroidism

Toxic
Methanol poisoning
Heavy metal toxicity
Scorpion bite
Snake bite
Black widow spider bite

Adapted from Purcell TB: Nonsurgical and extraperitoneal causes of abdominal pain. Emerg Med Clin North Am 7:721, 1989.

the peripheral nerves and are better localized than the visceral pain component. Figure 21-1 illustrates some more typical pain locations corresponding to specific disease entities. Somatic pain is often described as intense and constant. As disease processes evolve to peritoneal irritation with inflammation, better localization of the pain to the area of pathology generally occurs.

Referred pain is defined as pain felt at a distance from its source because peripheral afferent nerve fibers from many internal organs enter the spinal cord through nerve roots that also carry nociceptive fibers from other locations, as illustrated in Figure 21-2. This makes interpretation of the location of noxious stimuli difficult for the brain. Both visceral pain and somatic pain can manifest as referred pain. Two examples of referred pain are the epigastric pain associated with an inferior myocardial infarction and the shoulder pain associated with blood in the peritoneal cavity irritating the diaphragm.

Gynecologic and obstetric presentations are discussed in other chapters. Notably, any abdominal pain in a female may represent referred pain from pelvic structures or an extension of a pelvic process, as in the case of perihepatic inflammation with pelvic inflammatory disease.

DIAGNOSTIC APPROACH

The clinical approach should focus on early stabilization, history, physical examination, and any ancillary tests collectively facilitating appropriate management and disposition plans.

Differential Considerations

Classically, potential diagnoses are divided into intra-*abdominopelvic* (intraperitoneal, retroperitoneal, and pelvic) causes (e.g., appendicitis, cholecystitis, pancreatitis) and extra-*abdominopelvic* processes (e.g., pneumonia, myocardial infarction, ketoacidosis).

Although significant morbidity and mortality can result from many disorders causing abdominal pain, a few processes warrant careful consideration in the ED. Table 21-2 lists important potentially life-threatening nontraumatic causes of abdominal pain. This group represents the major etiologic disorders likely to be associated with hemodynamic compromise and for which early therapeutic intervention is critical.

Rapid Assessment and Stabilization

As with any complaint, triage is the first critical step in management. Most patients presenting with abdominal pain do not have hemodynamic instability, but up to 7% of these patients may have a life-threatening process. This percentage is higher in elders and immunocompromised patients.[1]

Physiologically compromised patients should be brought to a treatment area immediately and resuscitation initiated. Profound shock or protracted emesis can lead to airway compromise necessitating intubation. These patients are often severely volume depleted and require rapid intravenous access and volume resuscitation with an isotonic crystalloid solution, titrated to a physiologic endpoint.

Extreme conditions such as ruptured abdominal aortic aneurysm, massive gastrointestinal hemorrhage, ruptured spleen, and hemorrhagic pancreatitis may require blood or blood product replacement. Bedside ultrasonography can be used to quickly evaluate patients for free intraperitoneal fluid, volume status, and presence of aortic pathology. Ultrasound assessment should be part of the initial physical examination and can be invaluable in guiding treatment and disposition. Because any of the immediately life-threatening entities may necessitate surgical intervention or management, early surgical consultation is indicated.

Pivotal Findings

History

A careful and focused history is central to unlocking the puzzle of abdominal pain. Box 21-2 lists some historical questions with high yields for serious pathology. Language and cultural differences may influence accurate communication and mutual understanding.

Abrupt onset often is indicative of a more serious cause; however, delayed presentations also may represent a surgical condition. Surgical causes of abdominal pain are more likely to manifest with pain first, followed by nausea and vomiting, rather than with nausea and vomiting followed by pain. Localization and pain migration also are helpful components of the pain history. Diffuse pain generally is nonsurgical, but it may represent the early visceral component of a surgical process. Colicky pain is indicative of hollow viscus distention, and

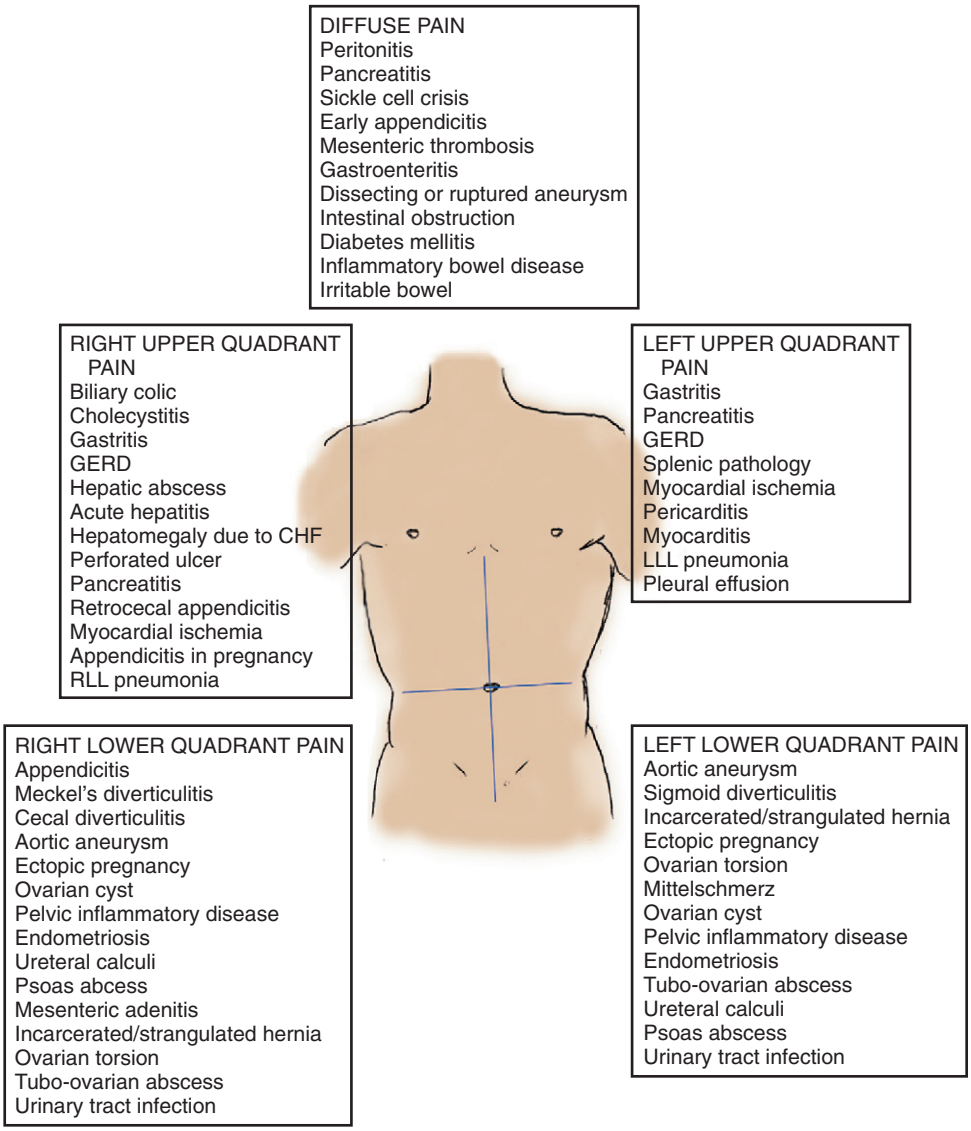


Figure 21-1. Differential diagnosis of acute abdominal pain. CHF, congestive heart failure; GERD, gastroesophageal reflux disease; LLL, left lower lobe; RLL, right lower lobe.

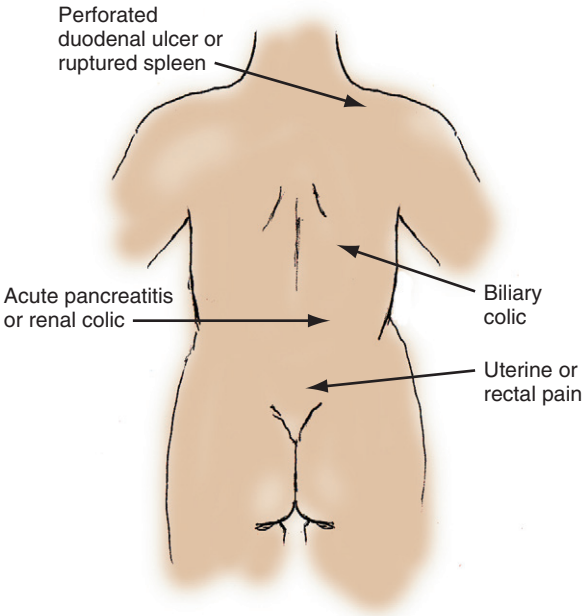


Figure 21-2. Common locations of referred pain from abdominal etiology.

duration and time of colic may give clues to the identity of the culprit organ, as displayed in Figure 21-3.

The severity and descriptive nature of the pain are the most subjective aspects of the pain history, but a few classical descriptions are recognized, such as the following:

- The diffuse, severe, colicky pain of bowel obstruction
- The "pain out of proportion to examination" observed in patients with mesenteric ischemia
- The radiation of pain from the epigastrium straight through to the midback associated with pancreatitis, either related to primary organ inflammation or secondary to a penetrating ulcer
- The radiation of pain to the left shoulder or independent pain in the left shoulder associated with splenic pathology, diaphragmatic irritation, or free intraperitoneal fluid
- The onset of pain associated with syncope seen in perforation of gastric or duodenal ulcer, ruptured aortic aneurysm, or ruptured ectopic pregnancy

Physical Examination

The objective evaluation begins with measurement of the vital signs. Significant tachycardia and hypotension are indicators that hypovolemia or sepsis may be present. Tachypnea

Table 21-2 Potentially Life-threatening Causes of Abdominal Pain

CAUSE	EPIDEMIOLOGY	ETIOLOGY	PRESENTATION	PHYSICAL EXAMINATION	USEFUL TOOL(S)
Ruptured ectopic pregnancy	Occurs in females of childbearing age. No method of contraception prevents ectopic pregnancy. Approximately 1 in every 100 pregnancies.	Risk factors include nonwhite race, older age, history of STD or PID, infertility treatment, intrauterine contraceptive device placed within the past year, tubal sterilization, and previous ectopic pregnancy.	Severe, sharp constant pain localized to the affected side. More diffuse abdominal pain with intraperitoneal hemorrhage. Signs of shock may be present. Midline pain tends not to be ectopic pregnancy.	Shock or evidence of peritonitis may be present. Lateralized abdominal tenderness. Localized adnexal tenderness or cervical motion tenderness increase the likelihood of ectopic pregnancy. Vaginal bleeding does not have to be present.	β-hCG testing necessary in all females of childbearing age (10–55 yr); combined with ultrasonography, preferably transvaginal in early pregnancy, usually is diagnostic. FAST exam is useful in evaluating for free fluid in patients with shock or peritonitis.
Ruptured or leaking abdominal aneurysm	Incidence increases with advancing age. More frequent in men. Risk factors include HTN, DM, smoking, COPD, and CAD.	Exact etiology is undetermined. Contributing factors include atherosclerosis, genetic predisposition, HTN, connective tissue disease, trauma, and infection.	Patient often asymptomatic until rupture. Acute epigastric and back pain often associated with or followed by syncope or signs of shock. Pain may radiate to back, groin, or testes.	Vital signs may be normal (in 70%) to severely hypotensive. Palpation of a pulsatile mass is usually possible in aneurysms 5 cm or greater. The physical examination may be nonspecific. Bruits or inequality of femoral pulses may be evident.	Abdominal plain films abnormal in 80% of cases. Ultrasound can define diameter and length but can be limited by obesity and bowel gas. FAST exam can be helpful in evaluating for leak by looking for free fluid. Spiral CT test of choice in stable patients.
Mesenteric ischemia	Occurs most commonly in elders with CV disease, CHF, cardiac dysrhythmias, DM, sepsis, and dehydration. Responsible for 1 of 1000 hospital admissions. Mortality 70%. Mesenteric venous thrombosis associated with hypercoagulable states, hematologic inflammation, and trauma.	20–30% of lesions are nonocclusive. The causes of ischemia are multifactorial, including transient hypotension in the presence of preexisting atherosclerotic lesion. The arterial occlusive causes (65%) are secondary to emboli (75%) or acute arterial thrombosis (25%).	Severe pain, colicky, that starts in periumbilical region and then becomes diffuse. Often associated with vomiting and diarrhea. Sometimes postprandial. "Mesenteric or abdominal angina."	Early examination results can be remarkably benign in the presence of severe ischemia. Bowel sounds often still present. Rectal examination important because mild bleeding with positive guaiac stools can be present.	Often a pronounced leukocytosis is present. Elevations of amylase and creatine kinase levels are seen. Metabolic acidosis due to lactic acidemia is often seen with infarction. Plain radiographs of limited benefit. CT, MRI, and angiography are accurate to varying degrees.

Intestinal obstruction	Peaks in infancy and older age. More common with history of previous abdominal surgery.	Adhesions, carcinoma, hernias, abscesses, volvulus, and infarction. Obstruction leads to vomiting, "third spacing" of fluid, or strangulation and necrosis of bowel.	Crampy diffuse abdominal pain associated with vomiting.	Vital signs usually normal unless dehydration or bowel strangulation has occurred. Abdominal distention, hyperactive bowel sounds, and diffuse tenderness. Local peritoneal signs indicate strangulation.	Elevated WBC count suggests strangulation. Electrolytes may be abnormal if associated with vomiting or prolonged symptoms. Abdominal radiographs and CT are useful in diagnosis.
Perforated viscus	Incidence increases with advancing age. History of peptic ulcer disease or diverticular disease common.	More often a duodenal ulcer that erodes through the serosa. Colonic diverticula, large bowel, and gallbladder perforations are rare. Spillage of bowel contents causes peritonitis.	Acute onset of epigastric pain is common. Vomiting in 50%. Fever may develop later. Pain may localize with omental walling off of peritonitis. Shock may be present with bleeding or sepsis.	Fever, usually of low grade, is common; worsens over time. Tachycardia is common. Abdominal examination reveals diffuse guarding and rebound. "Boardlike" abdomen in later stages. Bowel sounds are decreased.	WBC count usually elevated due to peritonitis. Amylase may be elevated; LFT results are variable. Upright radiographic view reveals free air in 70–80% of cases with perforated ulcers.
Acute pancreatitis	Peak age in adulthood; rare in children and elders. Male preponderance. Alcohol abuse and biliary tract disease are risk factors.	Alcohol, gallstones, hyperlipidemia, hypercalcemia, or endoscopic retrograde pancreatography causing pancreatic damage, saponification, and necrosis. ARDS, sepsis, hemorrhage, and renal failure are secondary.	Acute onset of epigastric pain radiating to the back. Nausea and vomiting are common. Pain disproportionate to physical findings. Adequate volume repletion is important in the initial therapy.	Low-grade fever common. Patient may be hypotensive or tachypneic. Some epigastric tenderness usually present. Because pancreas is retroperitoneal organ, guarding or rebound not present unless condition is severe. Flank ecchymosis or periumbilical ecchymosis may be seen if process is hemorrhagic.	Lipase determination is test of choice. Ultrasound exam may show edema, pseudocyst, or biliary tract disease. CT scan may show abscesses, necrosis, hemorrhage, or pseudocysts. CT is ordered if severe acture pancreatitis is suspected. Rule out gallstones with ultrasound exam.

ARDS, acute respiratory distress syndrome; β-hCG, β human chorionic gonadotropin; CAD, coronary artery disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; CT, computed tomography; CV, cerebrovascular; DM, diabetes mellitus; FAST, focused assessment with sonography in trauma; HTN, hypertension; LFT, liver function test; MRI, magnetic resonance imaging; PID, pelvic inflammatory disease; STD, sexually transmitted disease; WBC, white blood cell.

BOX 21-2 HIGH-YIELD HISTORICAL QUESTIONS

1. *How old are you?* Advanced age means increased risk.
2. *Which came first—pain or vomiting?* Pain first is worse (i.e., more likely to be caused by surgical disease).
3. *How long have you had the pain?* Pain for less than 48 hours is worse.
4. *Have you ever had abdominal surgery?* Consider obstruction in patients who report previous abdominal surgery.
5. *Is the pain constant or intermittent?* Constant pain is worse.
6. *Have you ever had this before?* A report of no prior episodes is worse.
7. *Do you have a history of cancer, diverticulosis, pancreatitis, kidney failure, gallstones, or inflammatory bowel disease?* All are suggestive of more serious disease.
8. *Do you have human immunodeficiency virus (HIV)?* Consider occult infection or drug-related pancreatitis.
9. *How much alcohol do you drink per day?* Consider pancreatitis, hepatitis, or cirrhosis in patients with history or signs of significant intake.
10. *Are you pregnant?* Test for pregnancy—consider ectopic pregnancy.
11. *Are you taking antibiotics or steroids?* Effects of these drugs may mask infection.
12. *Did the pain start centrally and migrate to the right lower quadrant?* High specificity for appendicitis.
13. *Do you have a history of vascular or heart disease, hypertension, or atrial fibrillation?* Consider mesenteric ischemia and abdominal aneurysm.

From Colucciello SA, Lukens TW, Morgan DL: Abdominal pain: An evidence-based approach. Emerg Med Pract 1:2, 1999.

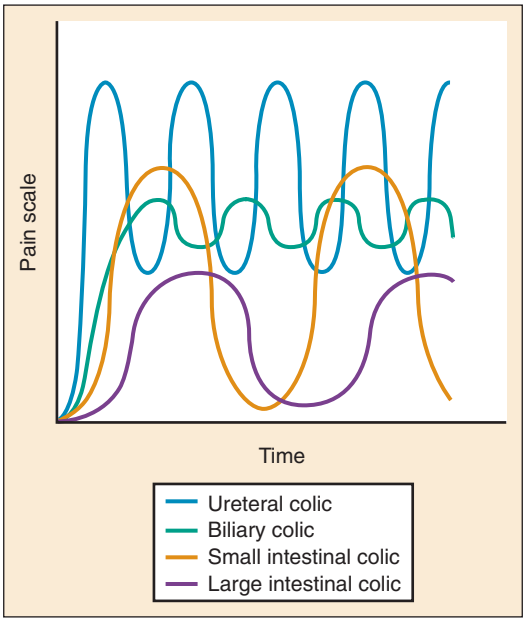


Figure 21-3. The characteristics of colicky abdominal pain.

may be an indication of metabolic acidosis from gangrenous viscera or sepsis, hypoxemia from pneumonia, or simply a catecholamine-induced reaction to pain. Elevated temperature often is associated with intra-abdominal infections. Although important, vital signs may be misleading and should be interpreted in the context of the entire presentation. Tachycardia may develop late for various reasons in hypovolema. Temperature often is normal in elderly patients with laparotomy-proven intraperitoneal infections.[9] Septic elderly patients also may present with hypothermia.

A thorough abdominal examination is an essential part of evaluation of the patient with abdominal pain. This requires properly positioning the patient supine and exposing the abdomen. The examination should begin with inspection for any signs of trauma, bruising, or skin lesions. The patient should be asked to localize the area of maximal tenderness by pointing with one finger. The abdomen can be mentally divided into four quadrants: right upper, right lower, left upper, and left lower; each area is then examined individually. Tenderness in one quadrant often corresponds with the location of the diseased organ, which will direct the workup (see Fig. 21-1). Some disease processes may manifest with pain that is not exclusively within one specific quadrant, such as the suprapubic pain of a urinary tract infection or the midepigastic pain of a gastric ulcer. Although 80% of patients with suspected appendicitis present with right lower quadrant abdominal tenderness, 20% of patients with proven appendicitis do not.[10]

Rectal examination may have limited use in the evaluation of abdominal pain, except that associated with intraluminal gastrointestinal hemorrhage, prostatitis, or perirectal disease. The main utility of the rectal examination is in the detection of heme-positive stool, anal fissures or fistulas, or stool impaction. Rectal examination has not been shown to increase diagnostic accuracy for appendicitis when added to external physical examination of the abdomen.[11]

The abdominal evaluation should include a pelvic examination in female patients with lower abdominal pain or an otherwise uncertain diagnosis. The pelvic exam should be done early in the evaluation of the female patient with abdominal pain to help differentiate an abdominal from a pelvic source. This information is helpful in choosing an imaging modality. Pelvic ultrasound exam is helpful in evaluating uterine and ovarian pathology, whereas computed tomography (CT) is more beneficial in evaluation of suspected intra-abdominal pathology. Although the pelvic exam may guide the initial choice of imaging modality, overlap in exam findings is common. For example a patient with right lower quadrant tenderness may have both right adnexal tenderness and tenderness over McBurney's point—necessitating exclusion of both appendicitis and ovarian torsion. The diagnosis highest on the differential list should be ruled out first using the corresponding imaging modality.

In the male patient with abdominal pain, the urogenital system should be examined. Diseases such as prostatitis, orchitis, and epididymitis commonly cause abdominal pain in males. Furthermore, inguinal hernias are more common in males, with the possibility of strangulation or incarceration in the inguinal canal making a thorough genitourinary examination mandatory.

In view of the evolving nature of abdominal pain, repetitive examinations may be useful. This is common practice with respect to suspected appendicitis and has improved the diagnostic accuracy in patients whose presentations were atypical.[2]

Ancillary Testing

Urinalysis and testing for pregnancy are perhaps the most time- and cost-effective adjunctive laboratory tests available. Results often can be obtained quickly, so the former can lead to an early diagnosis and the latter may significantly affect

further evaluation and management approaches. It is necessary to interpret urinalysis results within the context of the patient's clinical picture. Pyuria, with or without bacteriuria, often is present in a variety of conditions besides a simple urinary tract infection. For example, appendicitis may feature sterile pyuria.[12] Similarly, hematuria usually is present with the relatively benign condition of nephrolithiasis but also may indicate an abdominal aortic aneurysm.

Complete blood counts frequently are ordered for patients with abdominal pain, but findings seldom are contributory to a diagnosis. Despite the association of elevated white blood cell (WBC) counts with many infectious and inflammatory processes, the WBC count is neither sufficiently sensitive nor specific to be considered a discriminatory test to help establish or rule out a serious cause for the pain. Even serial WBC counts have failed to differentiate surgical from nonsurgical conditions. The WBC count is therefore not helpful for diagnosis. Serum electrolytes, even in the presence of protracted emesis or diarrhea, are abnormal in less than 1% of patients. These studies are not indicated for most patients in the absence of another indication. Blood urea nitrogen concentrations can be elevated in gastrointestinal hemorrhage and dehydration, but such conditions are better detected and quantified by history and physical examination. Increased serum creatinine usually is indicative of renal dysfunction. Blood glucose, anion gap, and serum ketone determinations are useful in diabetic ketoacidosis, one cause of acute abdominal pain and tachypnea.

Liver enzymes and coagulation studies are helpful only in a small subset of patients with suspected liver disease.[13] If pancreatitis is suspected, the most useful diagnostic result is serum lipase elevated to at least double the normal value, because it is more specific and more sensitive than serum amylase for this process. Measurement of serum amylase is of no value if a serum lipase level is available.[14] Serum phosphate and serum lactate levels are elevated late in bowel ischemia, and such determinations may be useful if this entity is suspected but cannot be considered either sufficiently sensitive or specific to establish or exclude the diagnosis on their own.

Plain radiography of the abdomen has limited usefulness in the evaluation of acute abdominal pain. Suspected bowel obstruction, foreign body, and perforated viscus are the main indications. CT of the abdomen has become the imaging modality of choice with nonobstetric abdominal pain. It allows visualization of both intraperitoneal and extraperitoneal structures and has a high degree of accuracy, establishing a diagnosis in more than 95% of cases in one study[15] and increasing the confidence in diagnosis in another.[16] Incidental findings are common on CT scans and may lead to a diagnosis. Patients who undergo CT have a change in diagnosis more often than those who do not.[17] The proper execution and interpretation of CT studies will reduce morbidity, mortality, and medical expenses.[18,19]

CT has increased diagnostic utility in elderly patients for several reasons. Older people with abdominal pain may have twice the rate of surgery[20-22] and a six- to eight-fold increase in mortality compared with younger adults.[20,21] Furthermore, evaluation of abdominal pain in the elderly often is more challenging owing to unreliable findings on physical examination including vital signs, difficulties in history taking, physiologic age-related changes, and comorbid conditions. In the elderly population, CT results change management or disposition decisions in a significant proportion of patients.[23] Table 21-3 lists the most common findings on CT scans in elderly patients with abdominal pain.

Some controversy surrounds the use of oral contrast in abdominal CT in the critically ill ED patient. Technologic advances have improved image acquisition and resolution, and preliminary studies have shown that intravenous contrast alone may now be adequate in the evaluation of certain suspected pathologic processes, such as solid organ or bowel wall disease.[24] CT with intravenous contrast alone also has been shown to be sensitive and specific for the confirmation or exclusion of acute appendicitis.[25] The exclusion of oral contrast in these patients significantly decreases ED time to disposition and improves patient satisfaction.

Bedside transabdominal and transvaginal ultrasonography have emerged as extremely useful adjuncts, decreasing time to diagnosis of life-threatening abdominopelvic conditions. Useful indications include the following:

- Identification of an intrauterine pregnancy, effectively lowering the chances of an ectopic pregnancy to less than 1 in 20,000 (In women using fertility aids, however, identification of intrauterine pregnancy does not exclude ectopic pregnancy, in keeping with an increased incidence of heterotopic pregnancy.)
- Measurement of the cross-sectional diameter of the abdominal aorta to determine whether an abdominal aortic aneurysm exists
- Detection of free intraperitoneal fluid indicating hemorrhage, pus, or extrusion of gut contents
- Use as a diagnostic aid for detection of the following non–life-threatening conditions:
  - Gallstones or a dilated common bile duct, which may be a clue to the presence of choledocholithiasis
  - Pericholecystic fluid or gallbladder wall thickening, which may be indicative of cholecystitis
  - Free intraperitoneal fluid indicating ascites
  - Hydronephrosis indicating possible obstructive uropathy
  - Inferior vena cava distention or collapse as an indicator of volume status

Table 21-3 Most Common Diagnostic Computed Tomography (CT) Findings in Older Patients Presenting to the Emergency Department with Acute Abdominal Pain

FINDING	PERCENT OF ABDOMINAL CT SCANS
Small bowel obstruction or ileus	18%
Diverticulitis	18%
Urolithiasis	10%
Cholelithiasis	10%
Abdominal mass/neoplasm	8%
Pyelonephritis	7%
Pancreatitis	6%

From Hustey FM, Meldon SW, et al: The use of abdominal computed tomography in older ED patients with acute abdominal pain. Am J Emerg Med 23:259–265, 2005.

The results of sonographic examinations are operator-dependent, and misdiagnosis can occur because of failure to detect or identify pathology, incorrect identification of normal anatomy as pathologic, or overinterpretation of correctly identified findings (e.g., the mere presence of gallstones does not indicate that cholelithiasis is the cause of the pain). The emergency physician must be properly trained in image acquisition and interpretation, and ultrasound evaluation in the radiology department should be sought if there is ambiguity or uncertainty in findings.

DIFFERENTIAL DIAGNOSIS

The differential considerations with abdominal pain include a significant number of potentially life- or organ-threatening entities, particularly in the setting of a hemodynamically unstable or toxic-appearing patient. Severely ill patients require timely resuscitation and expeditious evaluation for potentially life-threatening conditions. A focused history and exam should be performed, and the patient should be placed in a monitored acute care area well equipped for airway control, quick intravenous access, and fluid administration. Only then should appropriate diagnostics be initiated (bedside focused assessment with sonography in trauma [FAST] and aorta ultrasound assessment and radiographic, electrocardiographic, and laboratory studies). This approach is particularly important in dealing with elderly or potentially pregnant patients (see Tables 21-1 and 21-2).

Women of reproductive age who present with abdominal pain should undergo pregnancy testing early, and a known pregnancy or a positive result on urine or serum pregnancy testing associated with abdominal pain in the ED should be considered to represent an ectopic pregnancy until proved otherwise. If evidence of blood loss is present, early obstetric consultation and diagnostic ultrasonography should be promptly sought. Bedside transabdominal sonography may identify free intraperitoneal fluid during the evaluation of shock, which may be sufficient evidence to justify operative intervention in the context of a positive pregnancy test and appropriate history and physical findings.

Despite the limitations already described, the approach to the differential diagnosis of abdominal pain generally is based on the location of maximum tenderness. Figure 21-1 shows locations of subjective pain and maximal tenderness on palpation related to various underlying causes. In women of childbearing age, a positive result on pregnancy testing may indicate ectopic pregnancy, but the entire spectrum of intra-abdominal conditions remains in the differential diagnosis, as for the nonpregnant patient. When the very broad differential list is compartmentalized by both history and physical examination, ancillary testing should proceed to either confirm or support the clinical suspicion.

Despite the significant variety of tests available, close to one half of the patients presenting to the ED with acute abdominal pain will have no conclusive diagnosis. It is incumbent on the clinician to reconsider the extra-abdominal causes of abdominal pain (see Box 21-1), with special consideration in elderly and immunocompromised patients, before arriving at the diagnosis of "nonspecific abdominal pain."

EMPIRICAL MANAGEMENT

The main therapeutic goals in managing acute abdominal pain are physiologic stabilization, mitigation of symptoms (e.g., emesis control, pain relief), and expeditious diagnosis, with consultation, if required.

There is no evidence to support withholding analgesics from patients with acute abdominal pain to preserve the accuracy of subsequent abdominal exams; in fact, the preponderance of evidence supports the opposite. Pain relief may facilitate the diagnosis in patients ultimately requiring surgery.[26-28] In the acute setting, analgesia usually is accomplished with intravenously titrated opioids. Meperidine (Demerol) has an unfavorable side effect profile and should be avoided. Intravenous ketorolac, the only parenteral nonsteroidal anti-inflammatory drug available in North America, is useful for both ureteral and biliary colic,[29,30] as well as some gynecologic conditions, but is not indicated for general treatment of undifferentiated abdominal pain. Among patients with gastrointestinal hemorrhage and potential surgical candidates, ketorolac has been shown to increase bleeding times in healthy volunteers.[31]

Aside from analgesics, a variety of other medications may be helpful to patients with abdominal pain. The burning pain caused by gastric acid may be relieved by antacids.[32] Intestinal cramping may be diminished with oral anticholinergics such as the combination agent atropine-scopolamine-hyoscyamine-phenobarbital (Donnatal), although evidence for this is scant and highly variable.

Antiemetics such as promethazine, prochlorperazine, ondansetron, granisetron, or inapsine can be useful for nausea and vomiting. Gastric emptying by nasogastric tube with suction is appropriate for suspected small bowel obstruction and intractable pain or vomiting.

If intra-abdominal infection is suspected, broad-spectrum antibiotic therapy should be initiated promptly. Abdominal infections are often polymicrobial and coverage for enteric gram-negative, gram-positive, and anaerobic bacteria must be included. In the choice of antibiotic or combination, the following should be considered:

- Unless local antibiotic resistance surveillance indicates otherwise, second-generation cephalosporins (e.g., cefamandole, cefotetan, cefoxitin) or quinolone (ciprofloxacin, levofloxacin) may be combined with metronidazole for the initial dose of antibiotics in the ED. Other noncephalosporin, β-lactam agents with β-lactamase antagonists (e.g., ampicillin-sulbactam, piperacillin-tazobactam, ticarcillin-clavulanate) are alternatives.
- Many enteric gram-negative bacilli mutate rapidly to produce β-lactamases that are poorly antagonized by specific drug combinations containing clavulanate, sulbactam, or tazobactam. A carbapenem (e.g., imipenem, meropenem) or cefepime is an alternative for patients who may have recently received other antibiotics.[10,33]

Whether to provide coverage for *Enterococcus* species is still a subject of debate, and the decision to treat for these bacteria specifically can be made after consultation. Immunocompromised patients may require antifungal agents.

DISPOSITION

Because up to 40% of patients presenting with acute abdominal pain receive the diagnosis of nonspecific abdominal pain, the disposition can be as difficult as the diagnosis in these patients. Categories for disposition may include surgical versus nonsurgical consultation and management, admission for observation, and discharge to home with follow-up evaluation.[34] The decision to admit a patient to an observation unit or a hospital bed must factor in the following:

- Information gained from the history, physical examination, and test results
- The likelihood of any suspected disease
- Any potential ramifications of progression of a known disease, or of incorrect diagnosis or management
- The likelihood of appropriate (or any) and timely follow-up after hospital discharge

Clinically stable patients may be discharged from the ED with appropriate follow-up care, possibly to include repeated physical exam or additional diagnostic imaging if indicated.

In the case of nonspecific abdominal pain that is considered potentially worrisome, it is prudent to have the patient reevaluated after 8 to 12 hours. This can be done through a return visit to the ED, an appointment with a primary care physician, or an observation unit protocol.

Before discharge of a patient with an undiagnosed cause of nonspecific abdominal pain, several conditions should be met: The abdominal examination findings should be benign overall, with normal vital signs. Pain and nausea should be controlled, and the patient should be able to eat and drink. If a patient is to be discharged home without a specific diagnosis, clear instructions to the patient must include the following information:

- What the patient has to do for relief of symptoms or to maximize chances of resolution of the condition (e.g., avoiding exacerbating food or activities, taking medications as prescribed)
- Under what circumstances, with whom, and in what time frame to seek follow-up evaluation, if all goes as desired on the basis of what is known when the patient is in the ED
- Under what conditions the patient should seek more urgent care because of unexpected changes in his or her condition (such as with natural progression of the process before improvement, incorrect diagnosis made in the ED, or untoward reactions to medications)

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 99 Stroke

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PERSPECTIVE

Background

Stroke is the third leading cause of death in the United States and a leading cause of long-term disability.[1] It afflicts more than 700,000 patients per year,[1] with an in-hospital mortality rate of 5 to10% for ischemic stroke[2,3] and 46% for intracerebral hemorrhage (ICH).[4] Although 50 to 70% of stroke survivors will regain functional independence, 15 to 30% will be permanently disabled, and 20% will require institutional care at 3 months. The estimated cost of stroke for 2007 is $62.7 billion.[1] In terms of emergency care, almost 2% of all 911 calls and 4% of hospital admissions from the emergency department (ED) are for patients with potential strokes.[5,6]

Stroke can be defined as any vascular injury that reduces cerebral blood flow (CBF) to a specific region of the brain, causing neurologic impairment. The onset of symptoms may be sudden or stuttering, often with transient or permanent loss of neurologic function. Approximately 80% of all strokes are ischemic in origin, caused by the occlusion of a cerebral vessel.[3,4] The rest are hemorrhagic strokes due to the rupture of a blood vessel into the parenchyma of the brain (ICH) or into the subarachnoid space (subarachnoid hemorrhage [SAH]). Only ischemic stroke and ICH are discussed in this chapter.

In the past, treatment for stroke consisted of stabilization, observation, and rehabilitation. In recent years, a better understanding of the pathophysiology of neuronal injury and the introduction of new therapies have led to a shift to early evaluation and treatment. Current interventional treatment regimens include blood pressure management, anticoagulation, thrombolytic therapy, catheter-based interventions, and surgery. The key to success is early identification and treatment of patients with stroke before neurologic deficits become irreversible.

Epidemiology

Ischemic Stroke

An estimated 430,000 "first-ever" ischemic strokes occur each year in the United States, of which 10 to 15% are transient ischemic attacks (TIAs). These may result either from in situ thrombosis or embolic obstruction from a more proximal source, usually the heart. In more than one third of these first-ever strokes, no cause is found[7,8] (Table 99-1).

Approximately one third of all ischemic strokes are thrombotic in nature. These can be due to either large- or small-vessel occlusions. The incidence of large-vessel occlusions is higher among men than among women and among whites than black Americans.[7,8] Common areas for large-vessel occlusions are cerebral vessel branch points, especially in the distribution of the internal carotid artery. Thrombosis usually results from clot formation in the area of an ulcerated atherosclerotic plaque (which forms in the area of turbulent blood flow, such as a vessel bifurcation). A marked reduction in flow results when the stenosis occludes more than 90% of the blood vessel diameter. With further ulceration and thrombosis, platelets adhere to the region. A clot then either embolizes or occludes the artery.

Lacunae, or small-vessel strokes, involve small terminal sections of the vasculature and more commonly occur in black Americans and patients with diabetes and hypertension.[7] A history of hypertension is present in 80 to 90% of patients who experience lacunar strokes. The subcortical areas of the cerebrum and brainstem often are involved. The infarcts range in size from a few millimeters to 2 cm and are seen most commonly in the basal ganglia, thalamus, pons, and internal capsule. They may be caused by small emboli or by a process termed *lipohyalinosis*, which occurs in patients with hypertensive cerebral vasculopathy. Although nearly 20 lacunar syndromes have been described, the most common of these lacunar syndromes are pure motor strokes, pure sensory strokes, or ataxic hemiparesis. Because they are subcortical and well localized, lacunar strokes do not cause cognitive impairment, aphasia, or simultaneous sensorimotor findings.

One fourth of all ischemic strokes are cardioembolic in nature.[7,8] Embolization of a mural thrombus in patients with atrial fibrillation is the most common pathomechanism, and patients with atrial fibrillation have a four- to fivefold increased risk for development of a stroke.[9] Almost 20% of stroke patients will have atrial fibrillation on their admission electrocardiogram (ECG).[10] Strokes due to atrial fibrillation are more likely to involve large cerebral vessels, to be more severe, and to carry a higher mortality rate.[9,10] Noncardiac sources of emboli may include diseased portions of extracranial arteries, resulting in an artery-to-artery embolus. One common example is amaurosis fugax, in which emboli from a proximal carotid artery plaque embolizes to the ophthalmic artery, causing transient monocular blindness.

Approximately 12.2 ischemic strokes per 1000 nonfatal myocardial infarctions (MIs) occur within 1 month after the index event. Furthermore, 11.1 ischemic strokes per 1000 nonfatal MIs occur during the patient's hospitalization.[11] Independent predictors of stroke after acute MI are advanced age, diabetes,

Table 99-1 Estimated Number of First-Ever Strokes/TIAs in the United States

STROKE SUBTYPE	ESTIMATED NUMBER
Large-vessel	69,000 (16%)
Small-vessel/lacunae	76,000 (17.5%)
Cardioembolic	113,000 (26%)
Stroke of uncommon mechanisms	15,000 (3.5%)
Infarction of unknown etiology	157,000 (36.5%)
Total strokes/TIAs	430,000 (100%)

TIA, transient ischemic attack.
Data from Woo D, et al: Incidence rates of first-ever ischemic stroke subtypes among blacks: A population-based study. Stroke 30:2517, 1999; and Petty GW, et al: Ischemic stroke subtypes: A population-based study of incidence and risk factors. Stroke 30:2513, 1999.

hypertension, history of previous stroke, anterior location of index MI, previous MI, atrial fibrillation, heart failure, and nonwhite race.[11] The use of aspirin has been shown to reduce the incidence of post-MI stroke by 46%.[12]

Approximately 3 to 4% of all strokes occur in patients between the ages of 15 and 45 years. Although atherosclerosis is the most common cause in older patients, causative disorders and conditions in younger patients often are uncommon or are reversible. Pregnancy, the use of oral contraceptives, antiphospholipid antibodies (such as lupus anticoagulant and anticardiolipin antibodies), protein S and C deficiencies, and polycythemia all predispose patients to sludging or thrombosis, thereby increasing the risk of stroke. Fibromuscular dysplasia of the cerebral vasculature also may lead to stroke, and in rare instances, prolonged vasoconstriction from a migraine syndrome causes stroke. Recreational drugs such as cocaine, phenylpropanolamine, and amphetamines are potent vasoconstrictors that have been associated with both ischemic and hemorrhagic stroke.

Carotid and vertebral dissections often are associated with trauma but may follow such mild events as turning the head sharply. Carotid and vertebral dissections also are seen more frequently in people with underlying pathology of the vessel wall, such as in fibromuscular dysplasia and connective tissue disorders. Alteration in the vessel intima can lead to vessel stenosis, occlusion, or embolism. The patient may report a minor preceding event such as spinal manipulation, yoga, working overhead, coughing, or vomiting.[13] Presenting manifestations may include headache, facial pain, visual changes, cranial nerve palsies, pain over the affected vessel, Horner's syndrome, amaurosis fugax, subarachnoid hemorrhage, or an ischemic stroke. The headache frequently is unilateral and may occur days before onset of the other neurologic symptoms.[13] Although angiography has been the standard diagnostic study, dissections are increasingly being diagnosed by less invasive modalities such as ultrasonography, magnetic resonance imaging (MRI), and computed tomography angiography (CTA).[14] Medical therapy includes early anticoagulation if SAH is not suspected. If symptoms recur despite anticoagulation, the patient may be eligible for endovascular intervention. Carotid or vertebral dissection is not considered a contraindication to use of tissue plasminogen activator (t-PA) in the eligible patient.[15] This entity is considered a major cause of stroke in younger patients.[13,16,17]

A TIA was historically defined as a neurologic deficit that has complete clinical resolution within 24 hours. A newer definition of TIA proposed by Albers[18] is a "brief episode of neurologic dysfunction caused by a focal disturbance of brain or retinal ischemia, with clinical symptoms typically lasting less than 1 hour, and without evidence of infarction." This updated definition evolved as newer imaging techniques demonstrated infarction in up to 81% of patients with neurologic symptoms lasting less than 24 hours.[19,20] TIAs constitute an important warning sign for the future development of cerebral infarction. Approximately 10% of the patients who experience a TIA will experience a stroke within 3 months of the sentinel event, and one half of these occur within the first 2 days.[21] A majority of TIAs last less than 5 minutes, but the course can be variable.[22] Three or more TIAs occurring within 72 hours are termed crescendo TIAs. Almost one half of all patients 65 years of age and older with "first-ever" TIAs have MRI evidence of a previous, clinically silent, cerebral infarction.[20] These lesions usually are deep and less than 1 cm in diameter and often involve the nondominant right hemisphere.

BOX 99-1 Most Common Sites for Hypertensive Intracranial Hemorrhage

Affected Area	Frequency
Putamen	44%
Thalamus	13%
Cerebellum	9%
Pons	9%
Other cortical areas	25%

Hemorrhagic Stroke

Spontaneous ICH causes 8 to 11% of all acute strokes and is twice as common as SAH. It carries a 30-day mortality rate of up to 50%, with one half of patients dying in the first 2 days. Among survivors, only 1 in 5 are living independently at 6 months.[23] The incidence of ICH is higher in men than in women and in Asians than in whites, and it occurs more commonly in young and middle-aged black Americans than in whites of similar age.[24]

ICH may occur in association with long-standing hypertension (hypertensive hemorrhage), amyloid angiopathy in the elderly, or arteriovenous malformations (AVMs). Hypertensive hemorrhage results from degenerative changes in the small penetrating arteries and arterioles leading to the formation of microaneurysms, most commonly in penetrating vessels of the middle cerebral artery. Two thirds of these bleeding episodes occur within the region of the basal ganglia. The most common sites for hypertensive hemorrhage are listed in Box 99-1. The hematoma that forms usually enlarges, causing local tissue injury and a subsequent increase in intracranial pressure (ICP).

ICH due to amyloid angiopathy tends to be lobar in nature and to occur more commonly in the elderly, with a higher incidence in whites than in black Americans. Sudden increases in blood pressure that occur with such drugs as phenylpropanolamine and cocaine can cause ICH. Other causes include the use of anticoagulants, tumors, and AVMs (especially in the young).

Bleeding from an AVM may be subarachnoid or intraparenchymal, or both. In AVMs, the hemorrhage into the subarachnoid space generally is confined to the area of the AVM, and the major clinical presentation is due to the intraparenchymal involvement, with focal neurologic deficits. AVMs are more likely to bleed into the ventricles and subarachnoid space than are hypertensive ICHs and usually have a less disruptive impact on cerebral function. An AVM producing hemorrhage

is more common in younger patients than hypertensive ICH. Such patients may have no history of hypertension.

PRINCIPLES OF DISEASE

Pathophysiology

The cerebral vasculature supplies the brain with a rich flow of blood that contains the critical supply of oxygen and glucose necessary for normal brain function. When a stroke occurs, there are immediate alterations in CBF and extensive changes in cellular homeostasis. A complete interruption of CBF, which is rare, results in loss of consciousness within approximately 10 seconds and death of vulnerable pyramidal cells of the hippocampus within minutes. In stroke, collateral circulation helps maintain some blood flow to the ischemic region. The normal CBF is 40 to 60 mL/100 g of brain per minute. When CBF drops below 15 to 18 mL/100 g of brain per minute, several physiologic changes occur. The brain loses electrical activity, becoming electrically "silent," although neuronal membrane integrity and function remain intact. Clinically, the areas of the brain maintaining electrical silence manifest a neurologic deficit, even though the brain cells are viable. When CBF is below 10 mL/100 g of brain per minute, membrane failure occurs, with a subsequent increase in the extracellular potassium and intracellular calcium and eventual cell death. The ischemic penumbra is the area of the brain surrounding the primary injury, which is preserved by a tenuous supply of blood from collateral vessels. This border zone of neuronal tissue is the area of greatest interest to investigators for possible salvage in both ischemic and hemorrhagic stroke. As defined by CBF, the ischemic penumbra consists of brain tissue with blood flow of 10 to 18 mL/100 g of brain per minute in which electrical silence is present but irreversible damage has not yet occurred. In ischemic stroke, the duration of occlusion plays a critical role in neuronal survival.[25] Increasing the duration of occlusion increases both the irreversibility of deficits and the amount of cerebral infarction. In experimental animals, occlusion of cerebral vessels for longer than 6 hours leads to irreversible neurologic deficits. Thus, ischemic stroke trials using fibrinolytic or antiplatelet agents have attempted to recanalize occluded arteries and reperfuse ischemic areas of the brain within a 2- to 6-hour therapeutic window.[26-30] In patients with ICH, a complicated series of events including red blood cell lysis and increased blood-brain barrier permeability can lead to edema formation and secondary brain injury.[31,32] Using a protocol similar to that in ischemic stroke trials, investigators have begun looking at the feasibility of ultra-early hematoma evacuation in patients with ICH.[33-36]

Anatomy and Physiology

Blood is supplied to the brain by the anterior and posterior circulations. The anterior circulation originates from the carotid system and perfuses 80% of the brain including the optic nerve, retina, and frontoparietal and anterior-temporal lobes. The first branch off the internal carotid artery is the ophthalmic artery, which supplies the optic nerve and retina. As a result, the sudden onset of painless monocular blindness (amaurosis fugax) identifies the stroke as involving the anterior circulation (specifically the ipsilateral carotid artery) at or below the level of the ophthalmic artery. The internal carotid arteries terminate by branching into the anterior and middle cerebral arteries at the circle of Willis.

The anterior cerebral artery supplies the basal and medial aspects of the cerebral hemispheres and extends to the anterior two thirds of the parietal lobe (Fig. 99-1). The middle cerebral artery feeds the lenticulostriate branches that supply the putamen, part of the anterior limb of the internal capsule, the lentiform nucleus, and the external capsule. Main cortical branches of the middle cerebral artery supply the lateral surfaces of the cerebral cortex from the anterior portion of the frontal lobe to the posterolateral occipital lobe.

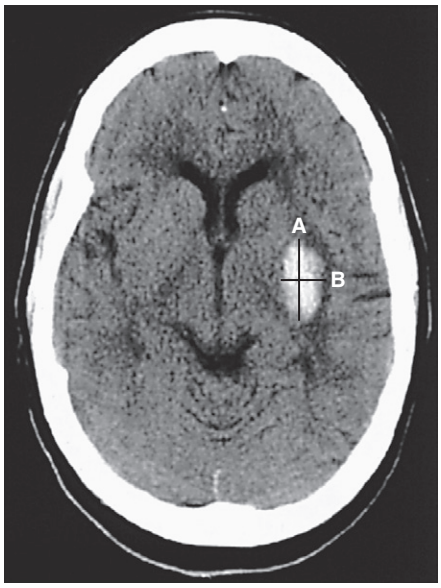

Figure 99-1. The CT slice with the largest area of hemorrhage is identified. The largest diameter of the hemorrhage on this slice is measured in centimeters (A). The largest diameter 90 degrees to A on the same slice is measured (B). C is the approximate number of 10-mm slices on which the intracerebral hemorrhage was seen. The volume of the hemorrhage equals A multiplied by C, divided by 2 (ABC/2).

Although the posterior circulation is smaller and supplies only 20% of the brain, it supplies the brainstem (which is critical for normal consciousness, movement, and sensation), cerebellum, thalamus, auditory and vestibular centers of the ear, medial temporal lobe, and the visual occipital cortex. The posterior circulation is derived from the two vertebral arteries that ascend through the transverse processes of the cervical vertebrae. The vertebral arteries enter the cranium through the foramen magnum and supply the cerebellum by the posterior inferior cerebellar arteries. They join to form the basilar artery, which branches to form the posterior cerebral arteries.

The extent of injury in either an anterior or a posterior stroke depends on both the vessel involved and the presence of collateral blood flow distal to the vessel occlusion. A patient with excellent collateral blood flow from the contralateral hemisphere may have minimal clinical deficits despite a complete carotid occlusion. By contrast, a patient with poor collateral flow may have hemiplegia with the same lesion.

CLINICAL FEATURES

Ischemic Stroke

The signs and symptoms of an ischemic stroke may appear suddenly and without warning or may have a stuttering, insidious onset. Disruption of the flow to one of the major vascular limbs of the cerebral circulation will result in physiologic disruption to the anatomic area of the brain supplied by that blood vessel. Ischemic strokes can be classified as anterior or posterior circulation strokes depending on the vasculature involved. The presence of neurologic deficits is highly dependent on collateral flow. In addition to the vascular supply involved, ischemic strokes can be further described by the

temporal presentation of their neurologic deficits. A "stroke in evolution" is one in which focal neurologic deficits worsen over the course of minutes or hours. Approximately 20% of anterior circulation strokes and 40% of posterior circulation strokes will show evidence of progression. Anterior circulation strokes may progress within the first 24 hours, whereas posterior strokes may progress for up to 3 days. Propagation of thrombus is postulated as a likely mechanism for progression. With anterior circulation strokes (involving variously and primarily the carotid, anterior, and middle cerebral arteries), the clinical presentation rarely includes complete loss of consciousness unless the lesion occurs in the previously unaffected hemisphere of a patient who has experienced a previous contralateral stroke. Occlusions in the anterior cerebral artery mainly affect frontal lobe function. The patient has altered mentation coupled with impaired judgment and insight, as well as the presence of primitive grasp and suck reflexes on physical exam. Bowel and bladder incontinence may be features. Paralysis and hypesthesia of the lower limb opposite the side of the lesion are characteristic. Leg weakness is more pronounced than arm weakness in anterior cerebral distribution stroke. Apraxia or clumsiness in the patient's gait also may be noted.

Marked motor and sensory disturbances are the hallmarks of occlusion of the middle cerebral artery. They occur on the side of the body contralateral to the side of the lesion and usually are worse in the arm and face than the leg. Such disturbances may involve only part of an extremity or the face but will almost always be accompanied by numbness in the same region as that of the motor loss. Hemianopsia, or blindness in one half of the visual field, occurs ipsilateral to the lesion. Agnosia, or the inability to recognize previously known subjects, is common, and aphasia may be present if the lesion occurs in the dominant hemisphere. Patients often have a gaze preference toward the affected hemisphere because of disruption of the cortical lateral gaze centers. The clinical aphorism is that a patient looks *at* a destructive lesion (stroke) but *away from* an irritative lesion (seizure focus).

Aphasia, a disorder of language in which the patient articulates clearly but uses language inappropriately or understands it poorly, also is common in dominant-hemisphere stroke. Aphasia may be expressive, receptive, or a combination of both. *Wernicke's aphasia* occurs when the patient is unable to process sensory input such as speech and thus fails to understand verbal communication (receptive aphasia). *Broca's aphasia* refers to the inability to communicate verbally in an effective way, even though understanding may be intact (expressive aphasia). Aphasia should be distinguished from dysarthria, which is a motor deficit of the mouth and speech muscles; the dysarthric patient articulates poorly but understands words and word choices. Aphasia is important to recognize because it usually localizes a lesion to the dominant (usually left) cerebral cortex in the middle cerebral artery distribution. *Aphasia* and *dysphasia* are terms that are used interchangeably but must be distinguished from *dysphagia*, which is difficulty in swallowing.

Pathology in the vertebrobasilar system (i.e., posterior circulation strokes) can cause the widest variety of symptoms and as a result may be the most difficult to diagnose. The symptoms reflect cranial nerve deficits, cerebellar involvement, and involvement of neurosensory tracts. The brainstem also contains the reticular activating system, which is responsible for mediating consciousness, and the emesis centers. Unlike those with anterior circulation strokes, patients with posterior circulation stroke can present with loss of consciousness and frequently have nausea and vomiting. The posterior cerebral artery supplies portions of the parietal and occipital lobes, so vision and thought processing are impaired. Visual agnosia, the inability to recognize seen objects, may be a feature, as may alexia, the inability to understand the written word. A third nerve palsy may occur, and the patient may experience homonymous hemianopsia. One of the more curious facets of this syndrome is that the patient may be unaware of any visual problem (visual neglect). Vertigo, diplopia, visual field defects, weakness, paralysis, dysarthria, dysphagia, syncope, spasticity, ataxia, or nystagmus may be associated with vertebrobasilar artery insufficiency. Posterior circulation strokes also demonstrate crossed deficits, such as motor deficits on one side of the body and sensory loss on the other. In anterior circulation strokes, by contrast, abnormalities are always limited to one side of the body.

A focused neurologic exam should assess level of consciousness, speech, cranial nerve function, motor and sensory function, and cerebellar function. Level of consciousness and fluency of speech can be rapidly assessed in a dialogue with the patient to determine the presence of dysarthria or aphasia. The head should be evaluated for signs of trauma. Pupillary size and reactivity and extraocular movements provide important information about brainstem function, particularly cranial nerves (CN) III through VI; an abnormal third nerve function may be the first sign of tentorial herniation. Gaze preference suggests brainstem or cortical involvement. Central facial nerve weakness from a stroke should be distinguished from the peripheral causes of CN VII weakness. With a peripheral lesion, the patient is unable to wrinkle the forehead. Assessment of facial sensation, eyebrow elevation and squinting, smiling symmetry, gross auditory acuity, gag reflex, shoulder elevation, sternocleidomastoid strength, and tongue protrusion complete the cranial nerve evaluation.

Motor and sensory testing is performed next. Muscle tone can be assessed by moving a relaxed limb. Proximal and distal muscle group strength should be assessed against resistance. Pronator drift of the arm is a sensitive sign of motor weakness and can be tested simultaneously by having the patient sit with eyes closed and arms outstretched, with palms toward the ceiling, for 10 seconds. Asymmetrical sensation to pain and light touch may be subtle and difficult to detect. Double simultaneous extinction evaluation tests for sensory neglect and can be easily performed by simultaneously touching the right and left limbs. The patient may feel both the right and left sides being touched individually but may not discern touch on one side when both are touched simultaneously. Similarly, the ability to discern a number gently scratched on a forearm, graphesthesia, is another easily tested cortical parietal lobe function. These tests can help differentiate a pure motor deficit of a lacunar stroke from a sensorimotor middle cerebral artery deficit.

Cerebellar testing and the assessment of reflexes and gait complete the examination. Finger-to-nose and heel-to-shin evaluations are important tests of cerebellar functions. Asymmetry of the deep tendon reflexes or a unilateral Babinski's sign may be an early finding of corticospinal tract dysfunction. Gait testing is commonly omitted yet is one of the most informative parts of the neurologic examination. Observing routine ambulation and heel-to-toe walking can assess for subtle ataxia, weakness, or focal cerebellar lesions.

The National Institutes of Health Stroke Scale (NIHSS) is a useful and rapid tool for quantifying neurologic deficit in patients with stroke and can be used in determining treatment options[37] (Box 99-2). NIHSS scores have been shown to be reproducible and valid and to correlate well with the amount of infarcted tissue on CT scan.[38,39] The baseline NIHSS can identify patients who are appropriate candidates for fibrinolytic therapy as well as those at increased risk for hemorrhage.

BOX 99-2 NIH Stroke Scale Scoring Form

Item	Scoring Definitions	Score
1a. *Level of consciousness (LOC)*	0 = alert and responsive 1 = arousable to minor stimulation 2 = arousable only to painful stimulation 3 = reflex reponses or unarousable	
1b. *LOC-related questions*: Ask patient's age and month. Must be exact.	0 = both correct 1 = one correct (or dysarthria, intubated, foreign language) 2 = neither correct	
1c. *Commands*: Open/close eyes, grip and release nonparetic hand. (Other one-step commands or mimic also acceptable.)	0 = both correct (acceptable if impaired by weakness) 1 = one correct 2 = neither correct	
2. *Best gaze*: Horizontal EOM by voluntary or doll's eye maneuver.	0 = normal 1 = partial gaze palsy; abnormal gaze in one or both eyes 2 = forced eye deviation or total paresis that cannot be overcome by doll's eye maneuver	
3. *Visual field*: Use visual threat if nec. If monocular, score field of good eye.	0 = no visual loss 1 = partial hemianopsia, quadrantanopia, extinction 2 = complete hemianopsia 3 = bilateral hemianopsia or blindness	
4. *Facial palsy*: If patient is stuporous, check symmetry of grimace to pain.	0 = normal 1 = minor paralysis, flat NLF, asymmetrical smile 2 = partial paralysis (lower face = UMN lesion) 3 = complete paralysis (upper and lower face)	
5. *Motor arm*: Arms outstretched 90 degrees (sitting) or 45 degrees (supine) for 10 seconds. Encourage best effort. Indicate paretic limb in score box.	0 = no drift for 10 seconds 1 = drift but doesn't hit bed 2 = some antigravity effort, but can't sustain 3 = no antigravity effort, but even miminal mvt counts 4 = no movement at all X = unable to assess due to amputation, fusion, fracture, etc.	L or R
6. *Motor leg*: Raise leg to 30 degrees (from supine) for 5 seconds. Indicate paretic limb in score box.	0 = no drift for 5 seconds 1 = drift but doesn't hit bed 2 = some antigravity effort, but can't sustain 3 = no antigravity effort, but even miminal mvt counts 4 = no movement at all X = unable to assess owing to amputation, fusion, fracture, etc.	L or R
7. *Limb ataxia*: Check finger-nose-finger, heel-shin position sense; and score only if out of proportion to paralysis.	0 = no ataxia (or aphasic, hemiplegic) 1 = ataxia in upper or lower extremity 2 = ataxia in upper *and* lower extremity X = unable to assess owing to amputation, fusion, fracture, etc.	L or R
8. *Sensory*: Use safety pin. Check grimace or withdrawal if patient is stuporous. Score only stroke-related losses.	0 = normal 1 = mild-moderate unilateral loss but patient aware of touch (or aphasic, confused) 2 = total loss, patient unaware of touch; coma, bilateral loss	
9. *Best language*: Describe cookie jar picture, name objects, read sentences. May use repeating, writing, stereognosis.	0 = normal 1 = mild-moderate aphasia (speech difficult to understand but partly comprehensible) 2 = severe aphasia (almost no information exchanged) 3 = mute, global aphasia, coma; no one-step commands	
10. *Dysarthria*: Read list of words.	0 = normal 1 = mild-moderate; slurred but intelligible 2 = severe; unintelligible or mute X = intubation or mech barrier	
11. *Extinction/neglect*: Simultaneously touch patient on both hands, show fingers in both visual fields, ask about deficit, left hand.	0 = normal, none detected (vis loss alone) 1 = neglects or extinguishes to double simultaneous stimulation in any modality (visual, auditory, sensation, spatial, body parts) 2 = profound neglect in more than one modality	

EOM, extraocular movement; NIH, National Institutes of Health; NLF, nasolabial fold; UMN, upper motor neuron.
Modified from online document. Massachusetts General Hospital Stroke Service. Available at: http://www2.massgeneral.org/stopstroke/pdfs/scoring_form.pdf

Table 99-2 Clinical Findings Associated with Ischemic Strokes, ICH, and SAH

FINDING	ISCHEMIC STROKE	ICH	SAH
Headache	11–17%	33–41%	78–87%
Vomiting	8–11%	29–46%	45–48%
Decreased LOC	13–15%	39–57%	48–68%
Seizure	0.3–3%	6–7%	7%

ICH, intracranial hemorrhage; LOC, level of consciousness; SAH, subarachnoid hemorrhage.

Data from Bogousslausky J, Van Melle G, Regli F: The Lausanne Stroke Registry: Analysis of 1000 consecutive patients with first stroke. Stroke 19:1083, 1988; Foulkes MA, et al: The Stroke Data Bank: Design, methods, and baseline characteristics. Stroke 19:547, 1988; and Mohr JP, et al: The Harvard Cooperative Stroke Registry: A prospective registry. Neurology 28:754, 1978.

In addition, it has been used as a prognostic tool to predict outcome and is currently being used by some stroke centers to stratify patients for entry into treatment trials.[37-40]

Hemorrhagic Stroke

The classic presentation of ICH is the sudden onset of headache, vomiting, severely elevated blood pressure, and focal neurologic deficits that progress over minutes. Similar to ischemic stroke, ICH often is associated with a motor and sensory deficit contralateral to the brain lesion. The patient may present with agitation and lethargy but may quickly deteriorate, with progression to stupor or coma. Almost 40% will demonstrate significant growth in hemorrhage volume within the first few hours.[41] Although headache, vomiting, and coma are common, a significant proportion of patients do not have these findings, and the clinical presentation may be similar to that of patients with ischemic stroke (Table 99-2).

Ongoing assessment of airway and mental status is of paramount importance in patients with ICH because precipitous deterioration is always a possibility. The respiratory pattern also may be affected in hemorrhagic stroke. Cheyne-Stokes respirations (increasing and decreasing depth of respirations with periods of apnea) may occur with a large ICH. Putaminal hemorrhages may cause deep, irregular respirations, whereas patients with cerebellar hemorrhage may have a normal respiratory pattern.

The pupillary examination can be extremely helpful in determining the location and extent of the insult. Pontine hemorrhage classically manifests with pinpoint pupils because of the interruption of the descending sympathetic tracts and unopposed parasympathetic stimulation. Dilated pupils may result from bleeding into the putamen, whereas bleeding into the thalamus may manifest with anisocoria, miosis, or a sluggish pupillary response. Cranial nerve abnormalities may result from cerebellar hemorrhages. The parasympathetic fibers course along the outside of CN III. As a result, compression of the nerve results in loss of pupillary reactivity before anisocoria. As noted previously, the physical exam may be insufficient to distinguish an ischemic stroke from an ICH, and radiographic confirmation is required.

As with ischemic stroke, a careful neurologic exam is important in localizing the region and extent of injury. Baseline NIHSS and Glasgow Coma Scale (GCS) scores can be used to assess stroke severity, although the GCS score may be more feasible to follow for neurologic deterioration (Box 99-3).

Poor prognostic indicators for patients with ICH include a decreased level of consciousness on arrival, intraventricular hemorrhage, and an ICH volume of greater than 40 cc, all of which can be assessed in the ED. The ABC/2 technique is a quick and accurate method of measuring ICH volume at the bedside[42] (see Fig. 99-1).

BOX 99-3 GLASGOW COMA SCALE

Eye Opening (E)	Verbal Response (V)	Motor Response (M)
4 = Spontaneous	5 = Normal conversation	6 = Normal
3 = To voice	4 = Disoriented conversation	5 = Localizes to pain
2 = To pain	3 = Words, but not coherent	4 = Withdraws to pain
1 = None	2 = No words; only sounds	3 = Decorticate posture
	1 = None	2 = Decerebrate posture
		1 = None

Scoring
Total score = E + V + M

Available at http://www.ssgfx.com/CP2020/medtech/glossary/glasgow.htm.

DIFFERENTIAL CONSIDERATIONS

Ischemic Stroke

Extra-axial collections of blood secondary to trauma can mimic stroke. An epidural or subdural hematoma can cause an altered mental status, focal neurologic signs, and rapid progression to coma. Elderly patients, who represent the age group at highest risk for stroke, can be victims of recurrent falls that lead to chronic subdural hematomas. Carotid dissection may occur after neck trauma or sudden hyperextension and may be associated with focal neurologic signs and symptoms, as with an aortic dissection that extends into the carotid arteries. The diagnosis is supported by a compatible history or relevant findings on contrast angiography or magnetic resonance angiography (MRA).

Other structural lesions that may cause focal neurologic signs include brain tumors and abscesses. Air embolism should be suspected in the setting of marked atmospheric pressure changes, such as in scuba diving or during medical procedures or injuries that may allow air into the vascular system. Seizures, altered mental status, and focal neurologic findings also may be manifestations of air embolism.

Metabolic abnormalities also can mimic stroke syndromes. Hypoglycemia often is responsible for an altered mental status and is a well-known cause of sustained focal neurologic symptoms that can persist for several days. Wernicke's encephalopathy causes ophthalmoplegia, ataxia, and confusion that can be mistaken for signs of cerebellar infarction.

Migraine may present with focal neurologic findings, with or without headache. A seizure followed by Todd's postictal paralysis may mimic stroke. Bell's palsy, labyrinthitis, peripheral nerve palsy, and demyelinating diseases may all mimic stroke. Meniere's disease may be difficult to distinguish from a posterior circulation stroke or TIA. Dizziness, vertigo, hearing loss, and tinnitus in Meniere's disease are common, whereas difficulties with vision, speech, or other focal symptoms are uncommon.

Like stroke, giant cell arteritis is a disease of the elderly. It may cause severe headache, visual disturbances, and, rarely, aphasia and hemiparesis. Other symptoms include intermittent fever, malaise, jaw claudication, morning stiffness, and

myalgias. The diagnosis should be suspected in patients with a very high erythrocyte sedimentation rate (ESR) and is confirmed by temporal artery biopsy. Collagen vascular diseases such as polyarteritis nodosa, lupus, and other types of vasculitis may cause stroke syndromes.

Venous sinus thrombosis is another cause of focal neurologic symptoms that most commonly affects the superior sagittal sinus and lateral sinuses. The cerebral venous sinuses receive blood from the cortical veins and deep veins. A venous sinus can become occluded through thrombus formation or compression (such as from a tumor or abscess). If collateral circulation is not sufficient, cerebrospinal fluid pressures can become elevated and venous congestion can develop, resulting in petechial or hemorrhagic infarction. Multiple risk factors predisposing affected patients to venous sinus thrombosis are recognized, including trauma, infectious processes, hypercoagulable states, low-flow states, compression of the venous sinus, dehydration, various drugs (such as androgens, "ecstasy," and oral contraceptives), and pregnancy or the postpartum state.[43] In many patients, no predisposing risk factor will be identified until further workup is completed.

The diagnosis of cerebral venous thrombosis can be difficult because of the nonspecific nature of symptoms, as well as the variable time frame of symptom onset (from hours to a few weeks). Patients may present with generalized headaches, nausea, vomiting, paresis, visual disturbances, depressed level of consciousness, seizures or even symptoms generally ascribed to psychiatric disorders (such as depression).[43,44] Depending on the location of the thrombus, physical examination of the patient may reveal papilledema, proptosis, or palsies of cranial nerves III, IV, and VI, as well as other focal neurologic signs and symptoms.[44]

The diagnosis of cerebral venous thrombosis sometimes can be made by CT. The so-called delta sign can be seen on a noncontrast CT scan as a dense triangle in the superior sagittal sinus. On a contrast-enhanced CT scan, this same area will lack demonstration of full contrast (the empty delta sign) owing to presence of thrombus in portions of the vessel's lumen. At many institutions, MRI and magnetic resonance venography (MRV) are the preferred modalities for the noninvasive detection of cerebral venous thrombosis. These studies can diagnose cerebral venous thrombosis by visualizing thrombosis within a vessel and provide evidence of hemorrhagic foci or of dilated venous collateral circulation.[43, 44]

The treatment of venous sinus thrombosis includes the use of heparin even in those patients with demonstrated hemorrhage. There is no evidence that low-molecular-weight heparin is superior to unfractionated heparin. In some cases, thrombolytic agents have been directly infused into the thrombus using endovascular techniques. Neurosurgical intervention has not been proved to be of benefit.[43]

The mortality rate from venous sinus thrombosis is approximately 10%.[45] In view of the nonspecific nature of the symptoms, it is important to keep this diagnosis in mind. In women who are pregnant or have recently given birth, the possibility of venous sinus thrombosis should be a primary consideration in the setting of new-onset neurologic disorders.

Hemorrhagic Stroke

The differential diagnosis for ICH is similar to that for ischemic stroke; considerations include migraine, seizure, tumor, abscess, hypertensive encephalopathy, and trauma. Hypertensive encephalopathy and migraine also can manifest with headache, nausea, and vomiting. Although focal neurologic signs are uncommon, they may occur with these entities. With hypertensive encephalopathy, patients usually exhibit marked elevation in blood pressure and other evidence of end-organ injury, including proteinuria, cardiomegaly, papilledema, and malignant hypertensive retinopathy. These patients usually improve significantly with treatment of their hypertension. Migraines frequently are associated with an aura, and the patient often has a history of similar headaches. The differentiation between ICH and labyrinthitis can be especially difficult in the elderly. The abrupt onset of vertigo, vomiting, and nystagmus can represent a peripheral process such as labyrinthitis or a central process such as cerebellar or brainstem infarct or hemorrhage. Age older than 40 years and a history of hypertension or other risk factors for ICH increase the possibility of a cerebellar hemorrhage. Pathologic features specifically referable to the brainstem must be sought. These include hiccups, diplopia, facial numbness, dysphagia, and ataxia. Vertiginous patients often have a strong desire to remain immobile with the eyes closed, but this must not preclude a thorough cranial nerve and cerebellar examination, including gait. Gross ataxia should be present with cerebellar stroke and absent with labyrinthine disease. A head CT scan should be strongly considered in patients older than 40, to assist in differentiating between labyrinthitis and cerebellar hemorrhage.

DIAGNOSTIC STRATEGIES

Ischemic Stroke

Although clinical data can help establish the diagnosis, cause, and location of the stroke, confirmatory diagnostic tests often are required to establish the final etiology or to eliminate other causes for the deficits. The immediate ED evaluation should include a blood glucose determination, cranial imaging (CT or MRI scan), and an ECG.

An emergent noncontrast cranial CT is the standard initial imaging technique for evaluating a patient with a potential stroke in the ED. It can quickly differentiate an ischemic stroke from ICH and other mass lesions. This information is crucial to subsequent therapeutic decisions. A CT scan can identify almost all parenchymal bleeds larger than 1 cm in diameter. Recent studies have yielded a sensitivity of 92 to 98% for the detection of SAH by CT scan; however, recent small studies suggest that sensitivity may be even higher with fifth-generation CT scanners.[46] More research is needed to confirm the sensitivity of these fifth-generation CT scanners, owing to small sample sizes in studies examining this issue to date. In a majority of ischemic strokes, gross signs of infarction will not appear on routine CT scans for at least 6 to 12 hours, depending on the size of the infarct. However, subtle, early ischemic changes have been noted in up to 60 to 80% of noncontrast CT scans within the first 3 hours in patients with middle cerebral artery occlusions.[47-49] These early ischemic changes include the hyperdense artery sign (acute thrombus in a vessel), sulcal effacement, loss of the insular ribbon, loss of gray-white interface, mass effect, and acute hypodensity (Fig. 99-2). Additionally, CTA can be used to identify the presence of intravascular thrombosis, vasculature dissection, or stenosis. In cases in which arterial dissection is suspected, imaging with MRA or CTA is indicated.[14]

The clinical importance of early ischemic CT findings in regard to fibrinolytic therapy within 3 hours of symptom onset is questionable because the ability of treating physicians to reproducibly identify these findings is poor and their clinical significance is questionable.[50,51] Only acute hypodensity and mass effect have been shown to be associated with an increased risk of ICH after fibrinolysis (over that in treated patients without these findings). However, these findings do not

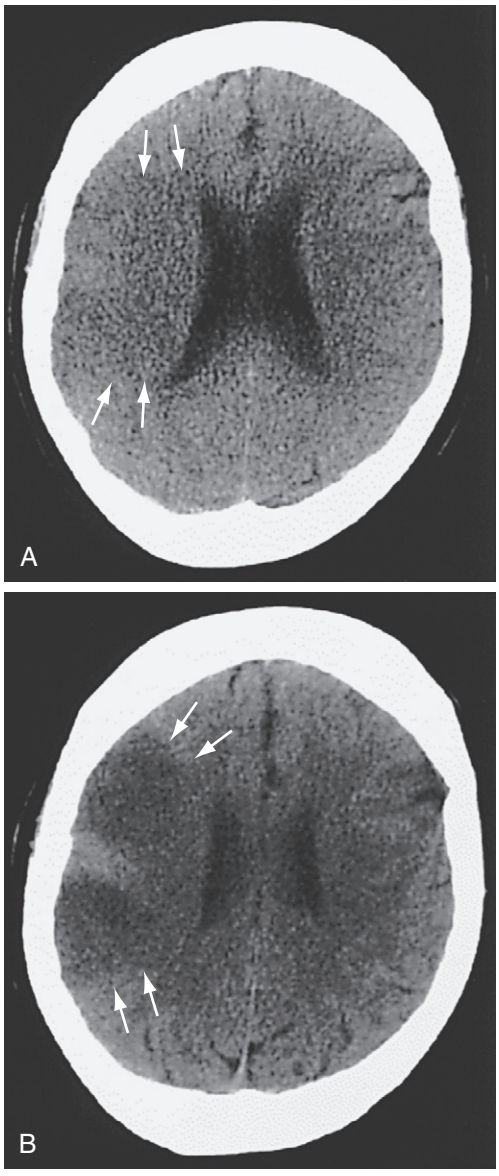

Figure 99-2. **A,** CT scan taken 2 hours and 50 minutes after a large right middle cerebral artery occlusion. There are subtle, ultra-early ischemic changes, including loss of the gray-white interface *(arrows)* and subtle evidence of sulcal effacement. **B,** CT scan of same patient approximately 8 hours after symptom onset shows acute hypodensity *(arrows)* and more prominent sulcal effacement.

exclude appropriate patients from fibrinolytic therapy, because the chance for excellent neurologic outcome at 3 months was better with such therapy than with placebo despite the presence of such abnormalities, and the risk of symptomatic ICH, severe disability, or death at 3 months was no different between treatment groups.[50] Patients with a hyperdense artery sign and acute hypodensity of one third of the middle cerebral artery distribution tend to have a poorer prognosis; however, their outcomes are still better with t-PA treatment than without such treatment.[50]

The role of MRI in the ED evaluation of stroke continues to evolve. MRI can visualize ischemic infarcts earlier and identify acute posterior circulation strokes more accurately than CT. In addition, recent studies suggest that it is as effective as CT in identifying ICH.[52,53] However, availability, difficulty in accessing critically ill patients, and scan time limit its general use. Advances in MRA technology have allowed a noninvasive method of demonstrating large-vessel occlusions of the anterior and posterior circulation, though small intracranial vascular occlusions may not be readily apparent. With the improvements in MRI and MRA speed and resolution, some stroke centers are replacing CT protocols with limited "stroke protocol" MRI or MRA as the initial imaging modality of choice. The choice of initial cranial imaging modality is highly dependent on the speed with which these scans can be done and interpreted at each individual center.

Diffusion-weighted imaging (DWI) and perfusion-weighted imaging (PWI) are MRI techniques that take minutes to perform and may allow differentiation between reversible and irreversible neuronal injury. In one study of patients with DWI-PWI mismatch, 30-day clinical outcomes were better after early reperfusion than without such early reperfusion (56% and 19%, respectively).[54] Of importance, however, early reperfusion in patients without DWI-PWI mismatch did not result in a favorable outcome. Currently, further studies are being performed by clinicians and researchers to determine if t-PA administration in patients with DWI-PWI mismatch can increase reperfusion rates and limit expansion of the DWI lesion.[55]

Other potential imaging modalities include CTA and perfusion scans. In CTA, a computed tomography (CT) scan is enhanced by an intravenous contrast agent to better define the vasculature of the brain. Areas of vascular stenosis and occlusion can be visualized with this technique. This information can then be used by interventionalists to determine whether or not a lesion is amenable to intra-arterial t-PA therapy or mechanical retrieval. Also requiring intravenous contrast, perfusion CT scans can reveal perfusion deficits within different regions of the brain. Additionally, CTA and perfusion CT can differentiate reversible from irreversible ischemic insults.[56,57] Further studies using these newer imaging techniques to determine eligibility for thrombolytic therapy are ongoing.

An ECG should be obtained because atrial fibrillation and acute MI are associated with up to 60% of all cardioembolic strokes. The hematologic evaluation should include a complete blood count with platelet count and coagulation studies. A toxicologic screen and cardiac isoenzyme assay should be considered if appropriate. Elevated blood viscosity even when hematocrit levels are not frankly polycythemic can affect blood flow and prognosis. A platelet count can identify thrombocytosis or thrombocytopenia, which may precipitate a thrombosis or hemorrhage. Coagulation studies are especially helpful to guide management for patients in whom anticoagulation is being considered or for patients with a hemorrhagic stroke. Cocaine or amphetamine ingestion should be considered with either an ischemic or hemorrhagic stroke in patients younger than 40 years of age.

Other ancillary diagnostic tests to consider include an echocardiogram, carotid duplex scan, and angiogram. Some centers are performing these studies as part of an observation unit protocol in the ED. An echocardiogram can identify a mural thrombus, tumor, patent foramen ovale, or valvular vegetation in those patients in whom a cardioembolic stroke is suspected. An echocardiogram also should be considered in patients with no obvious cause for their stroke. Carotid duplex scanning may be helpful in patients with known or suspected high-grade carotid stenosis with worsening neurologic deficit or crescendo TIAs.[58,59] These patients may be candidates for heparinization or emergent carotid endarterectomy. Carotid duplex studies can accurately identify carotid artery stenosis with occlusion of more than 60% vessel diameter, but an angiogram is required to distinguish 95% stenosis from a complete occlusion.

Angiography is the definitive test to demonstrate stenosis or occlusion of both large and small blood vessels of the head

and neck. It can detect subtle abnormalities, such as with dissection, that may not be demonstrated with other imaging techniques.

Hemorrhagic Stroke

The hematologic evaluation for the patient with hemorrhagic stroke should be performed in the same manner as for the patient with ischemic stroke. Particular attention should be directed to uncovering the presence of a coagulopathy. A drug screen should be obtained to evaluate for use of sympathomimetics if substance abuse is suspected. Increased sympathetic outflow secondary to the hemorrhage may lead to an increase in dysrhythmias. Dysrhythmias also may signal impending brainstem compression from an expanding hemorrhage.

As in ischemic stroke, the cranial CT scan is the diagnostic test of choice to evaluate for an ICH.[23] The CT scan will reliably diagnose up to 95% of ICHs, although very small lesions may not be visible. Hemorrhages that are several days old may appear as isodense regions.

STROKE MANAGEMENT

Ischemic Stroke

With the recent focus on rapid recognition, evaluation, and treatment of stroke, EDs have attempted to streamline the care of these patients to meet recommended time goals (Table 99-3). This has led to the development of various stroke protocols, critical pathways, and acute interventional stroke teams that often are deployed in the field before the patient even arrives to the ED.

In the prehospital setting, the focus should be on maintenance of the "ABCs" (airway-breathing-circulation), rapid identification, early hospital notification, and rapid transport.[60] Although it is unusual for patients with ischemic stroke to be unresponsive on presentation, their ability to communicate may be altered secondary to dysphasia. After an ischemic stroke, patients usually can maintain their airway unless the brainstem is affected or unless significant cerebral edema is compressing the opposite hemisphere. Patients with intact protective airway reflexes should receive oxygen if they are hypoxic (oxygen saturation less than 95%),[61] and a monitor and intravenous line established.

Overhydration should be avoided to prevent cerebral edema. By contrast, dehydration may lead to decreased cerebral perfusion, and saline infusion should be given if dehydration is suspected. Dextrose-containing solutions should be avoided in normoglycemic patients suspected of having had a stroke, because elevated blood glucose levels may worsen an ischemic deficit.[60] Out-of-hospital personnel should attempt to rapidly ascertain the patient's blood sugar. If this is not possible, glucose should be given only to diabetic patients in whom hypoglycemia is strongly suspected. Electrocardiographic monitoring is necessary because of the frequency of cardiac causes of ischemic stroke.[60]

The circumstances surrounding the stroke as well as concomitant medical conditions should be ascertained. The initial out-of-hospital responders should document the exact time the patient was last seen to be neurologically normal and the level of neurologic functioning; reversible defects may completely resolve by the time the patient has arrived at the hospital. The level of consciousness, gross focal motor deficits, difficulty with speech, clumsiness, facial asymmetry, and any other focal deficits should be noted. Prehospital stroke scales have been developed to assist in differentiating patients who have had a stroke from those who have not and to identify potential candidates for fibrinolytic therapy.[62-65] Early recognition, notification, and transport by emergency medical service (EMS) and other out-of-hospital personnel have been shown to be valuable in enabling early treatment.[60]

In the ED setting, the ABCs should be reassessed on an ongoing basis, because patients may experience rapid deterioration, even with subacute stroke. They may be found at home 1 or 2 days after the event has occurred and may have concomitant illnesses, such as aspiration pneumonia, dehydration, hypothermia, rhabdomyolysis, or myocardial ischemia. Fever necessitates a thorough evaluation to identify the source of infection, followed by prompt institution of appropriate treatment. As supported by strong evidence, even minor degrees of hyperthermia will result in worsening of the neurologic injury.[66]

Blood Pressure Management. The management of blood pressure in patients with acute ischemic stroke and TIA is controversial because of limited data. Current guidelines for the management of hypertension in patients with acute ischemic stroke recommend that antihypertensive treatment be reserved for those with markedly elevated blood pressures, unless fibrinolytic therapy is planned or specific medical indications are present.[60,67] These medical indications include (1) acute myocardial infarction, (2) aortic dissection, (3) true hypertensive encephalopathy, and (4) severe left ventricular failure.

Oral or parenteral agents should be withheld unless the patient's systolic pressure is greater than 220 mm Hg or diastolic pressure is greater than 120 mm Hg or mean arterial pressure is greater than 130 mm Hg (Table 99-4). If parenteral agents are used, labetalol or enalapril is favored because of ease of titration and limited effect on cerebral blood vessels. Sublingual nifedipine or sublingual nitroglycerin is not recommended because either agent can produce a precipitous drop in blood pressure.

If fibrinolytic therapy is planned, stringent control of blood pressure is indicated to reduce the potential for bleeding after the thrombolytic is administered (see Table 99-4).[60,68] Thrombolytic therapy is not recommended for patients whose systolic pressure is consistently greater than 185 mm Hg or whose diastolic pressure is 110 mm Hg at the time of treatment. Simple measures can be used to try lowering blood pressure below this level. Recommended approaches include the use of nitroglycerin paste and one or two doses of labetalol, 10 to 20 mg given intravenously. If more aggressive measures are required to reduce blood pressure below 185/110 mm Hg, the use of t-PA is not recommended. Once thrombolytic therapy has been initiated, blood pressure must be monitored closely and hypertension treated aggressively.

Just as problematic as high blood pressure can be, low blood pressure can be quite detrimental to patients with ischemic

Table 99-3 NINDS-Recommended Stroke Evaluation Targets for Potential Thrombolytic Candidates

MANAGEMENT COMPONENT	TARGET TIME FRAME
Door to doctor	10 minutes
Door to CT completion	25 minutes
Door to CT scan reading	45 minutes
Door to treatment	60 minutes
Access to neurologic expertise*	15 minutes
Access to neurosurgical expertise*	2 hours

*By phone or in person.
CT, computed tomography; NINDS, National Institute of Neurological Disorders and Stroke.

Table 99-4 Emergency Antihypertensive Therapy for Acute Ischemic Stroke

BLOOD PRESSURE*	TREATMENT
Nonthrombolytic Candidates	
1. DBP >140 mm Hg	Sodium nitroprusside (0.5 µg/kg per minute). Aim for 10 to 20% reduction in DBP.
2. SBP >220, DBP >120, or MAP† >130 mm Hg	10–20 mg labetalol‡ IV push over 1–2 minutes. May repeat or double labetalol every 20 minutes to a maximum dose of 150 mg.
3. SBP <220, DBP <120, or MAP† <130 mm Hg	Emergency antihypertensive therapy is deferred in the absence of aortic dissection, acute myocardial infarction, severe congestive heart failure, or hypertensive encephalopathy.
Thrombolytic Candidates	
Pretreatment	
1. SBP >185 or DBP >110 mm Hg	1–2 inches of nitro paste or one to two doses of 10–20 mg labetalol‡ IV push. If BP is not reduced and maintained at <185/110 mm Hg, the patient should not be treated with TPA.
During and after treatment	
1. Monitor BP	BP is monitored every 15 minutes for 2 hours, then every 30 minutes for 6 hours, and then hourly for 16 hours.
2. DBP >140 mm Hg	Sodium nitroprusside (0.5 µg/kg per minute).
3. SBP >230 or DBP 121–140 mm Hg	*Step 1*: Give 10 mg labetalol‡ IV push over 1 to 2 minutes. May repeat or double labetalol every 10 minutes to a maximum dose of 150 mg, or give the initial labetalol bolus and then start a labetalol drip at 2–8 mg/minute. *Step 2*: If BP is not controlled by labetalol, consider sodium nitroprusside.
4. SBP 180–230 or DBP 105–120 mm Hg	10 mg labetalol‡ IVP. May repeat or double labetalol every 10–20 minutes to a maximum dose of 150 mg or give initial labetalol bolus and then start a labetalol drip at 2–8 mg/minute.

*All initial blood pressures should be verified before treatment by repeating reading in 5 minutes.
†As estimated by one-third the sum of systolic and double diastolic pressures.
‡Labetalol should be avoided in patients with asthma, cardiac failure, or severe abnormalities in cardiac conduction. For refractory hypertension, alternative therapy with sodium nitroprusside or enalapril may be considered.
BP, blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; SBP, systolic blood pressure; TPA, tissue plasminogen activator.

BOX 99-4 FIBRINOLYTIC THERAPY FOR ACUTE ISCHEMIC STROKE: INCLUSION AND EXCLUSION CRITERIA

Inclusion Criteria

1. Age 18 years or older
2. Clinical diagnosis of ischemic stroke causing a measurable neurologic deficit
3. Time of symptom onset well established to be less than 180 minutes before treatment would begin

Exclusion Criteria

1. Evidence of intracranial hemorrhage on noncontrast head CT
2. Only minor or rapidly resolving stroke symptoms
3. High clinical suspicion of subarachnoid hemorrhage even with normal CT findings
4. Active internal bleeding (e.g., gastrointestinal or urinary bleeding within last 21 days)
5. Known bleeding diathesis, including but not limited to:
   - Platelet count <100,000/µL
   - Patient has received heparin within 48 hours and had an elevated activated partial thromboplastin time (greater than upper limit of normal for laboratory)
   - Recent use of anticoagulant (e.g., warfarin sodium) and elevated prothrombin time >15 seconds
6. Within 3 months of intracranial surgery, serious head trauma, or previous stroke
7. Within 14 days of major surgery or serious trauma
8. Recent arterial puncture at noncompressible site
9. Lumbar puncture within 7 days
10. History of intracranial hemorrhage, arteriovenous malformation, or aneurysm
11. Witnessed seizure at stroke onset
12. Recent acute myocardial infarction
13. On repeated measurements, systolic pressure >185 mm Hg or diastolic pressure >110 mm Hg at time of treatment, requiring aggressive treatment to reduce blood pressure to within these limits

CT, computed tomography.

stroke.[69] Normally normotensive stroke patients with low blood pressure or normally hypertensive stroke patients with low or even low-normal blood pressure should be given a fluid bolus to try to increase cerebral perfusion. This is especially important in patients who present in a dehydrated state. If initial fluid challenge is ineffective, the patient may require vasopressor therapy (e.g., with dopamine) to gradually increase MAP and improve cerebral perfusion.[67]

Acute Drug Therapy. To date, only the use of intravenous t-PA has been approved by the U.S. Food and Drug Administration (FDA) for treatment of patients with acute ischemic stroke. These recommendations initially were based on the results of the National Institute of Neurological Disorders and Stroke (NINDS) trial, although subsequent analysis of other studies has supported its use.[70-73] Concern has emerged regarding the safety of the use of t-PA in community practice.[74] However, a meta-analysis of non–trial-related use of t-PA (n = 2639) in community practice demonstrated efficacy and safety for t-PA similar to those reported in the NINDS trial.[75] The current recommendation for recombinant t-PA (rt-PA) is that it be administered intravenously at a dose of 0.9 mg/kg to a maximum of 90 mg (10% of the dose given as a bolus followed by an infusion lasting 60 minutes). Treatment must be initiated within 3 hours of the onset of ischemic symptoms in patients who meet strict inclusion and exclusion criteria (Box 99-4). Intravenous t-PA is not recommended when the time of stroke onset cannot be ascertained reliably, including strokes recognized on awakening. In addition, caution is warranted in treating patients with large strokes (NIHSS score of 20 or higher) or early CT changes from a recent major infarction (e.g., acute hypodensity or mass effect), because they are at increased risk for symptomatic hemorrhage.[76] Earlier studies have demonstrated the importance of adhering to the inclusion-exclusion criteria established by the NINDS trial.[74,77] A recent study suggests that patients with mild or rapidly resolving symptoms may still benefit from the use of intravenous t-PA.[78] The use of intravenous t-PA beyond the 3-hour window

has not been demonstrated to be of clinical benefit, although a meta-analysis of these studies suggests a beneficial effect in a specific subset of patients.[27-29,70]

Intra-arterial thrombolysis is an alternative treatment for eligible patients presenting beyond the 3-hour time window but within 6 hours of symptom onset.[37,79,80] In the Prolyse in Acute Cerebral Thromboembolism II (PROACT II) study, patients with middle cerebral artery strokes were 58% more likely than those who received a placebo to have little or no neurologic disability at 90 days when treated with prourokinase up to 6 hours after stroke onset.[80] In addition, the use of intra-arterial thrombolysis in patients with posterior circulation strokes and those unresponsive to initial treatment with intravenous t-PA also is being evaluated and has shown favorable outcomes.[37,81-84]

A variety of mechanical clot retrieval devices are being investigated. The best studied is the Mechanical Embolus Removal in Cerebral Ischemia (MERCI) retrieval device. This corkscrew-like device has been shown to be successful in recanalizing intracranial lesions of the internal carotid artery and has demonstrated improved clinical outcomes and survival compared with patients without successful recanalization of the artery.[85] Successful recanalization has been achieved when performed within an 8-hour window of symptom onset.[86,87] In 2004, the FDA cleared the MERCI retrieval device for use in the setting of acute ischemic stroke.

Previous studies have focused on the use of antiplatelet agents in acute ischemic stroke. Data from two large trials involving almost 40,000 patients indicate that early use of aspirin in patients with acute ischemic stroke who were not treated with a fibrinolytic agent was associated with a small but significant reduction in rates of stroke recurrence and mortality.[88] These studies in combination suggest a number needed to treat of 77 (i.e., 77 stroke patients would need to be treated with daily aspirin therapy to prevent a poor outcome, such as death, dependency at discharge or at 6 months after stroke, in 1 patient). The need for acute administration of aspirin in the ED is unclear, because patients were given aspirin up to 48 hours after stroke onset. Aspirin should not be given for the first 24 hours in patients who have received a fibrinolytic agent, because this has been associated with an increased risk of ICH and death.[89]

The use of low-molecular-weight or unfractionated heparin is common in patients with acute ischemic stroke or TIAs, but its value is unproved. Some studies suggest that heparin may reduce the risk of subsequent ischemic stroke but increase the risk of hemorrhagic stroke. To date, no studies have definitively established the efficacy of anticoagulants in the management of acute ischemic stroke.[90-92] However, heparin sometimes is considered in patients at high risk for stroke progression, including patients with crescendo TIAs or TIA due to a cardioembolic source, patients with a high-grade carotid artery stenosis, patients with posterior circulation TIA, and patients with evolving strokes. Heparin is recommended for the treatment of carotid and vertebral artery dissection unless a contraindication such as intracranial extension is present. If a dissection is diagnosed and the patient has no symptoms of ischemia, treatment with antiplatelet therapy alone may be an option.[13] Heparin therapy should not be initiated in patients with suspected endocarditis or in any patient until a CT scan has ruled out intracranial bleeding. Owing to the lack of consistent evidence of efficacy, the most prudent course in the ED setting is to determine the need for heparin therapy in conjunction with the patient's neurologist or the admitting physician.

Other innovative approaches to stroke care, including mild to moderate hypothermia and early hemicraniectomy, are currently being investigated.

Management of Intracranial Hemorrhage. The patient with a potential ICH requires rapid assessment and transport to a care center with CT scanning capability and intensive care management facilities. Out-of-hospital management is similar to that for ischemic stroke. The circumstances surrounding the event, as well as other concomitant medical conditions, also should be ascertained. The initial level of consciousness, GCS score, any gross focal deficits, difficulty with speech, clumsiness, gait disturbance, or facial asymmetry should be noted.

Supportive care involving attention to airway management and perfusion is of the highest priority. Patients with hemorrhagic stroke are more likely to have an altered level of consciousness that may rapidly progress to unresponsiveness requiring emergent endotracheal intubation. Intravenous access should be established and cardiac monitoring should be initiated. Evaluation of blood glucose and appropriate dextrose and naloxone administration should be considered in any patient with altered mental status.

Considerable disagreement exists regarding optimal blood pressure management in the patient with ICH. Hypertension may cause deterioration by increasing ICP and potentiating further bleeding from small arteries or arterioles. On the other hand, hypotension may decrease CBF, thereby worsening brain injury. In general, recommendations for treatment of hypertension in patients with ICH are more aggressive than those for patients with ischemic stroke. The current consensus regarding management of ICH is to recommend antihypertensive treatment with parenteral agents for systolic pressures higher than 160 to 180 mm Hg or diastolic pressures higher than 105 mm Hg. Treatment for lower pressures remains controversial.[23,60] Nitroprusside is the agent most commonly recommended because it can provide rapid and consistent lowering of the blood pressure to the desired level, and adjustments can be rapidly made. Nitroprusside provides a rapid onset, is titratable, and has no effect on mental status. Disadvantages include the need for careful monitoring (ideally with an indwelling arterial catheter) and the theoretical risk of worsening the hemorrhage due to the vasodilatory effects of nitroprusside on cerebral vessels. Labetalol is another therapeutic option. More recently, nicardipine has been proposed as an optimal antihypertensive agent in the setting of cerebrovascular emergencies, owing to both its good titration profile, which may create less need for adjunctive antihypertensive drugs, and its favorable cerebral hemodynamic effects.[93]

Hyperventilation and diuretics such as mannitol have been used when ICH is complicated by signs of progressively increasing ICP, clinical deterioration associated with mass effect, or impending uncal herniation.[23] These interventions should not be used prophylactically. Mannitol moves fluid from the intracranial compartment, thereby reducing cerebral edema. Although this effect may be temporarily helpful in the acute setting, the brain tissue will reequilibrate and rebound swelling can occur and worsen the patient's clinical status. The effectiveness of mannitol in the setting of ICH is questionable.[94-96] Hypertonic saline is being investigated as an alternative agent.[97] Use of steroids in cerebral hemorrhage, once a common practice, appears to be harmful and is not recommended. Other experimental modalities include barbiturate coma and hypothermia.

Seizure activity can cause neuronal injury, elevations in ICH, and destabilization of an already critically ill patient. In addition, nonconvulsive seizure may contribute to coma in up to 10% of patients in a neuro–intensive care unit.[23] Seizure prophylaxis (fosphenytoin 18 mg/kg) should be considered for patients with ICH, especially those with lobar hemorrhage.

Surgery is not beneficial in most cases of ICH. Selected patients with sizable lobar hemorrhage and progressive neuro-

logic deterioration may benefit from surgical drainage. Surgery is more efficacious in patients with cerebellar hemorrhage. The clinical course in cerebellar hemorrhage is notoriously unpredictable. Patients with minimal abnormalities may experience sudden deterioration, with progression to coma and death, with little warning. For this reason, most neurosurgeons will consider emergent surgery for patients with cerebellar hemorrhage within 48 hours of onset. In cases of severe intraventricular hemorrhage or hemorrhages in the posterior fossae, the normal circulation of cerebrospinal fluid (CSF) can become interrupted, leading to the development of hydrocephalus. This condition is characterized by an abnormal rise in CSF volume. In such cases a ventricular catheter should be inserted by a neurosurgeon.

DISPOSITION

Ischemic Stroke and Transient Ischemic Attacks

"Stroke center" definitions have been proposed, and a national certification process for primary stroke centers is now under way despite considerable political controversy. In broad terms, institutional certification as a primary stroke center requires the establishment of a stroke infrastructure (such as a stroke team, stroke unit, patient care protocols, and support services including CT scanning and laboratory testing availability), as well as institutional administrative support and strong leadership.[98] Additionally, recommendations also have been established for comprehensive stroke centers (CSCs). CSCs are expected to have the capability to provide the full spectrum of care to patients with stroke and other cerebrovascular diseases. More specifically, CSCs should offer advanced imaging modalities, perform surgical and endovascular interventions, and maintain a core infrastructure such as a stroke unit and stroke registry.[99] The establishment of PSCs and CSCs is intended to improve outcomes for stroke patients by providing them with a high level of coordinated care.

It has been recommended that patients with symptoms consistent with an acute stroke should be transported to emergency facilities capable of initiating fibrinolytic therapy within 1 hour of hospital arrival. At a minimum, this requires emergent CT capabilities, an institutional "acute stroke protocol," and availability of a physician versed in the use of thrombolytic therapy. Intensive care monitoring and neurosurgery capabilities should be available within 2 hours of drug initiation, either at the treating hospital or by helicopter or ground transport to an appropriate health care facility.[100]

In most cases, once the diagnosis of an acute stroke or stroke syndrome is established and the patient is stabilized, hospitalization will be necessary for further evaluation and treatment. Patients may deteriorate over the first 24 hours and require close in-hospital monitoring. Most patients can be managed on a general medical or telemetry unit. Patients with large acute hemispheric strokes (associated with increased risk for herniation) or with significant posterior circulation–related changes and those treated with a fibrinolytic agent should be monitored in a step-down or intensive care unit for at least 24 hours.

Some hospitals have specialized stroke units that provide organized, multidisciplinary care to stroke inpatients. A recent evidence-based medicine review amalgamated the results of 26 trials that compared the outcomes for more than 5500 stroke patients treated in either specialized stroke units or general wards. This review found a 14% reduction in the odds of post-stroke mortality for patients treated in a stroke unit compared with patients managed on a general ward (odds ratio [OR], 0.86; 95% confidence interval [CI]: 0.76 to 0.98). A similar reduction was found for the composite outcomes of death or institutionalized care (OR, 0.82; 95% CI: 0.73 to 0.92) and death or dependency (OR, 0.82; 95% CI: 0.73 to 0.92). All outcomes were independent of age, sex, or stroke severity, and evidence for increase in hospital length of stay by treatment in a stroke unit was lacking.[101]

In select cases, patients with multiple previous strokes who have been thoroughly evaluated or those who experience mild new episodes or have a completed stroke days to weeks after the event may be treated in the ED and discharged home after discussion with their primary care physician or neurologist. Close follow-up should be arranged.

Patients with new-onset TIAs should be hospitalized for evaluation and workup owing to the substantial short-term risk of stroke and other adverse events.[21] Exception can be made for patients with only minimal anterior circulation symptoms, for whom an extensive ED evaluation constitutes appropriate management. This evaluation must include a CT scan, a carotid Doppler study, MRA or CTA of the anterior circulation, and an echocardiogram (if indicated). A medically or surgically treatable cause for TIAs (e.g., high-grade carotid stenosis, mural thrombus) should be sought, which would require in-hospital treatment such as anticoagulation, stenting, or carotid endarterectomy. If the patient's symptoms have completely resolved, results of the workup are negative, and close neurologic follow-up can be arranged, outpatient therapy may be appropriate. The decision to start the patient on an antiplatelet agent should be made in conjunction with the neurologist.

The ABCD (*a*ge, *b*lood pressure, *c*linical features, *d*uration of TIA symptoms) score has been validated as a good predictor of future stroke risk in patients with TIA treated in the ED. ABCD scores range from 0 to 6, with a higher score indicating a higher future stroke risk; this scoring system is based on the following criteria: age 60 years or older = 1 point; systolic blood pressure greater than 140 mm Hg and/or diastolic greater than 90 mm Hg = 1 point; unilateral weakness = 2 points; speech disturbance without weakness = 1 point; symptom duration 10 to 59 minutes = 1 point; symptom duration 60 minutes or longer = 2 points. Patients with an ABCD score of 5 or 6 in the ED have a 30-day risk of stroke eight times that of patients with an ABCD score less than 5 (hazard ratio 8.01; 95% CI: 3.21 to 19.98).[102]

Hemorrhagic Stroke

All patients with an acute hemorrhagic stroke in whom surgical intervention is a consideration should be admitted to an intensive care unit under the care of a neurologist or a neurosurgeon. If this is unavailable at the evaluating institution, the patient should be transported to an appropriate institution.

Acknowledgment

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KEY CONCEPTS

- Patients presenting with the signs and symptoms of an acute ischemic stroke within 3 hours of symptom onset should be evaluated for thrombolytic therapy within the NINDS-recommended time frames (see Table 99-3).
- Carotid Doppler, MRA, or CTA studies are recommended before discharge of a patient with TIA from the ED.
- Overly aggressive blood pressure management should be avoided in patients with acute ischemic stroke.
- Accurate time of symptom onset should be documented in all patients with stroke.
- Assessment of gait is essential to rule out posterior circulation stroke in patients presenting with vertigo.
- The possibility of carotid or vertebral dissection should be considered in young patients with stroke and in patients with headaches and neck pain with acute stroke.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 145 General Approach to the Poisoned Patient

Ken Kulig and Louis J. Ling

PERSPECTIVE

Most poisoned patients seen in the emergency department are adults with acute oral drug overdoses. Other common clinical scenarios include accidental poisoning in children; drug abuse through smoking, snorting, or intravenous routes; chronic poisoning, usually from environmental, industrial, and agricultural chemical exposure; medication reactions or interactions; and envenomation. Management requires both a general approach and specific actions directed at the particular toxin or toxins involved, as outlined in the various chapters in this section. Clinical studies have modified the management of poisoned patients, such as the use of gastric decontamination, but much of the toxicology literature, especially with unusual poisonings, remains case based. Regional poison centers and medical toxicologists have a concentrated experience in managing poisoned patients and can be called upon for advice and assistance, when necessary.

INITIAL APPROACH TO THE POISONED PATIENT

With rare exception, the priorities of care for a poisoned patient are identical to those for all patients coming to the emergency department. Patients who are contaminated with an agent that might injure health care personnel require decontamination before treatment to avoid disabling the hospital staff or the entire health care facility. Except for specific lifesaving antidotes against certain toxins, most poisoned patients require only supportive therapy for recovery. The initial workup should determine whether a specific patient has been exposed to an agent for which an antidote (or other specific treatment) exists.[1] A thorough poisoning history and toxicologic physical examination are followed by the selective use of laboratory tests.

After initial stabilization of a critically ill patient, specific antidote therapy is administered while a detailed history and physical examination are performed. Hypoglycemia must always be considered in a patient with altered mental status or seizures and should be evaluated by bedside glucose testing rather than empiric administration of hypertonic glucose solution.[2] *Naloxone* can be given to patients with respiratory depression while preparations are made to secure the airway because a positive response may obviate the need for intubation. *Flumazenil* is not indicated in an undifferentiated overdose patient, and its use should be limited to confirmed acute benzodiazepine overdose in a patient known to not be a regular benzodiazepine user (e.g., an adolescent who impulsively ingests a parent's benzodiazepine). Indiscriminate use may force a chronic benzodiazepine user into severe benzodiazepine withdrawal. Likewise, the patient may have ingested tricyclic antidepressants or other drugs likely to cause seizures. In either case, the use of flumazenil can carry a substantial risk of seizures. Patients with benzodiazepine overdose respond well to supportive care. *Thiamine* should be administered when dextrose is given to nutritionally compromised, alcoholic patients with altered mental status (100 mg in the maintenance intravenous line is sufficient and safe).[3]

A complete *overdose history* is required, particularly when the ingested agent is unknown or the patient is suicidal (Box 145-1). Valuable clues often come from unexpected sources, such as the patient's previous medical records, the pharmacy where prescriptions were filled, or the prescribing physician as listed on the patient's prescription bottles. Whenever possible, field personnel should bring the patient's medications to the hospital with them. If the ingested agent is a hazardous chemical (e.g., pesticide) that might endanger hospital personnel, it should be brought to the hospital in an airtight container or secured at the scene. Precise product identification information must be ascertained so that a hazardous materials reference system can be consulted. When it is suspected that the contents of the container are not the original product, the substance should be checked against the product label. It is easy to confuse the different types of chemical agents with agents with similar names found in many homes, and some may have specific properties that affect treatment. In rare cases, overdose patients may deliberately attempt to deceive caregivers by hiding the ingested agents.

Vital signs, including pulse oximetry, are important in the diagnosis of poisoning and should be measured accurately and repeated as indicated. At least one measurement of temperature should be included. Respirations should be counted, not estimated. A cardiac monitor or 12-lead electrocardiogram should be evaluated for QRS and QT intervals, morphology, and rhythm. The physical examination in a comatose patient should ensure that concomitant treatable conditions (e.g., intracranial hemorrhage and central nervous system [CNS] infection) are not missed. Focal neurologic findings could be possible indicators of intracranial catastrophe or severe head trauma.

The pupillary examination may give misleading information. Some opioid agonists, especially propoxyphene and pentazocine, may not produce the characteristic miosis of opioid

BOX 145-1 OBTAINING AN OVERDOSE HISTORY

- Obtain all prescription bottles and other containers when possible. Perform a pill count. Be sure that the bottles contain the medications listed. Identify any unknown tablets.
- Contact the prescribing physician(s) or the pharmacy as listed on the bottles to determine previous overdoses or other medications that the patient may have available. Identify underlying medical and psychiatric disorders and medication allergies. Review past medical records.
- Talk to the patient's family and friends in the emergency department. If necessary, call the patient's home to ask questions of others. The persons providing the important elements of the history should be identified in the chart.
- Search the patient's belongings for drugs or drug paraphernalia. A single pill hidden in a pocket, for example, may provide *the* most important clue to the diagnosis.
- Have family members (or the police) search the patient's home, including the medicine cabinet, clothes drawers, closets, and garage: such searches may also provide clues that make the diagnosis. This has the added benefit of involving the family in the patient's care.
- Always look for track marks on the patient. Consider body packing or body stuffing.

intoxication. When multiple drugs are ingested, the expected pupillary findings related to any particular agent may be modified or absent.

Physical stigmata of intravenous drug use (track marks) should be sought in both usual (e.g., antecubital fossa) and unusual (e.g., under the tongue and top of the feet) locations. A critical condition of unknown cause may be a result of "body packing" or "body stuffing," complicated by rupture of packets of cocaine, heroin, or amphetamines (see Chapter 152). Rectal, vaginal, and radiographic examination of the abdomen should be performed in these circumstances.

Other important physical findings are evidence of aspiration or noncardiogenic pulmonary edema on chest auscultation. Bowel sounds may be increased or decreased if agents affecting the cholinergic nervous system have been ingested. A rectal examination to detect melena or hematochezia may also provide evidence of suicidal ingestion of anticoagulant medication.

Unusual odors of the patient's breath, skin, clothing, vomitus, or nasogastric aspirate may also provide useful diagnostic clues (Table 145-1).[4] The absence of such odors, however, should not be taken as evidence that the agents listed are not present.

Table 145-1 Odors in Overdose History

ODOR	POSSIBLE INTOXICANT
Bitter almonds	Cyanide
Carrots	Water hemlock (cicutoxin)
Fishy	Zinc or aluminum phosphide
Fruity	Ethanol, acetone, isopropyl alcohol, chlorinated hydrocarbons (e.g., chloroform)
Garlic	Arsenic, dimethyl sulfoxide (DMSO), organophosphates, yellow phosphorus, selenium, tellurium
Glue	Toluene, other solvents
Pears	Chloral hydrate, paraldehyde
Rotten eggs	Disulfiram, hydrogen sulfide, *N*-acetylcysteine, dimercaptosuccinic acid (DMSA)
Shoe polish	Nitrobenzene
Wintergreen	Methyl salicylate

TOXIC SYNDROMES AND ANTIDOTES

The term *toxidrome* refers to a syndrome or constellation of physical findings that can be attributed to a specific class of toxins and can provide important clues to narrow the differential diagnosis.[1] The general rules outlined here have many exceptions, and polydrug overdoses may result in overlapping and confusing mixed syndromes. Nevertheless, this approach may confirm the history, provide the clinician with a starting point for management, and suggest useful laboratory tests. The most common toxidromes are the anticholinergic syndrome, sympathomimetic syndrome, opioid/sedative/ethanol syndrome, cholinergic syndrome, and serotonin syndrome (Table 145-2).

The *anticholinergic syndrome* occurs frequently because many common medications and plants have anticholinergic properties. Anticholinergic CNS poisoning causes mild temperature elevation and acute delirium with mumbling speech and typical "picking movements" of the fingers. Suppression of cholinergic inhibition of the heart rate leads to tachycardia. Inhibition of the secretory functions of the integument causes dry mouth and skin, and the face is typically flushed. Unopposed sympathetic drive of the ciliary apparatus causes wide papillary dilation. Most patients recover with supportive therapy, but the delirium may last a day or more. *Physostigmine* may be a useful antidote in carefully selected patients and quickly resolves the delirium. It should not be used with a possible cyclic antidepressant overdose where it is associated with asystole.

The *sympathomimetic syndrome* is usually seen after acute or chronic abuse of cocaine, amphetamines, or decongestants (e.g., phenylpropanolamine). Patients may be delusional; amphetamine, in particular, may cause complicated, intricate, and paranoid delusions. Seizures may occur, and the postictal state can contribute to the altered mental status. Blood pressure is usually elevated, the pulse is rapid (except with pure alpha-adrenergic agonists, which can cause reflex bradycardia), the pupils are dilated, and piloerection may be seen. In massive overdoses of sympathomimetic agents, cardiovascular collapse can occur with the development of shock and wide-complex dysrhythmias. This clinical picture can mimic that of overdose of cardioactive drugs or cyclic antidepressants. In contrast to the diaphoresis seen with anticholinergic syndrome, the skin in sympathomimetic syndrome is dry.

An extreme presentation of sympathomimetic excess can be excited delirium (Box 145-2). In this state, patients are agitated, hyperthermic, violent, and possess "superhuman strength." Frequently, many security personnel are required to control these individuals. These individuals may have a severe metabolic acidosis and hyperkalemia, which can cause sudden cardiovascular collapse. It is critical to sedate these patients and control hyperthermia aggressively while treating their acidosis and hyperkalemia simultaneously.

All sedative/hypnotic agents, when taken in sufficient dosage, cause general anesthesia with a complete loss of awareness and reflex activity. The CNS depressant (opioid/sedative/ethanol) syndrome is the most common toxic syn-

Table 145-2 Common Toxic Syndromes (Toxidromes)

Anticholinergic	
Common signs	Delirium with mumbling speech, tachycardia, dry flushed skin, dilated pupils, myoclonus, slightly elevated temperature, urinary retention, decreased bowel sounds. Seizures and dysrhythmias may occur in severe cases.
Common causes	Antihistamines, antiparkinsonians, atropine, scopolamine, amantadine, antipsychotics, antidepressants, antispasmodics, mydriatics, muscle relaxants, many plants (e.g., jimson weed, *Amanita muscaria*)
Sympathomimetic	
Common signs	Delusions, paranoia, tachycardia (or bradycardia with pure alpha-agonists), hypertension, hyperpyrexia, diaphoresis, piloerection, mydriasis, hyper-reflexia. Seizures, hypotension, and dysrhythmias may occur in severe cases.
Common causes	Cocaine, amphetamine, methamphetamine and its derivatives, over-the-counter decongestants (phenylpropanolamine, ephedrine, pseudoephedrine). In caffeine and theophylline overdoses, similar findings, except for the organic psychiatric signs, result from catecholamine release.
Opioid/Sedative/Ethanol	
Common signs	Coma, respiratory depression, miosis, hypotension, bradycardia, hypothermia, pulmonary edema, decreased bowel sounds, hyporeflexia, needle marks. Seizures may occur after overdoses of some narcotics (e.g., propoxyphene).
Common causes	Narcotics, barbiturates, benzodiazepines, ethchlorvynol, glutethimide, methyprylon, methaqualone, meprobamate, ethanol, clonidine, guanabenz
Cholinergic	
Common signs	Confusion, central nervous system depression, weakness, salivation, lacrimation, urinary/fecal incontinence, gastrointestinal cramping, emesis, diaphoresis, muscle fasciculations, pulmonary edema, miosis, bradycardia/tachycardia, seizures
Common causes	Organophosphate and carbamate insecticides, physostigmine, edrophonium, some mushrooms

Modified from Kulig K: Initial management of ingestions of toxic substances, N Engl J Med 326:1677, 1992.

BOX 145-2 TOXINS CAUSING DELIRIUM

Anticholinergics
Cocaine
Lithium
MAOIs
Mushrooms with muscinol/ibotenic acid
Phencyclidine
Salicylates
Sedative withdrawal
Solvents
Steroids
Sympathomimetics (cocaine, amphetamines)

MAOIs, monoamine oxidase inhibitors.

drome seen in the emergency department, and a depressed sensorium is its hallmark. Mixing agents in this class (e.g., ethanol and benzodiazepines) is common. As the drugs are absorbed at higher doses, the patient becomes increasingly obtunded, the deep tendon reflexes diminish, and, finally, the vital signs deteriorate as medullary drive of respiration and cardiovascular function is attenuated.

Respiratory depression is particularly pronounced with opioid overdose, and the respiratory rate is often diminished before decreases in blood pressure or pulse occur. The diagnosis of opioid overdose is confirmed by the use of naloxone (Narcan) or nalmefene (Revex) in adequate doses.[2,5] *Naloxone* has an elimination half-life of 1.1 hours, whereas that of nalmefene is 10.8 hours. *Nalmefene* is especially useful when the offending opioid has a very long elimination half-life (e.g., methadone, with a half-life of 15–40 hours). Close observation, investigation of alternative causes of depressed mental status when suggested by the clinical course, and airway intervention when indicated are the keys to successful management. Comatose patients often present without a history and need to be managed aggressively, securing the airway when needed. These patients may need basic labs and head CT if their presentation or course is suspicious for stroke, infection, or head trauma while considering a drug overdose alone or in combination with one of these medical conditions. A serum ethanol level that is not commensurate with the level of CNS depression raises suspicion of intracranial injury, hemorrhage, or infection.

The *cholinergic syndrome* is uncommon but important to recognize because lifesaving treatment is available. Cholinergic syndrome causes the patient to be "wet," as opposed to the anticholinergic syndrome, which causes the patient to be "dry." The wetness is manifest by profuse sweating and excessive activity of virtually the entire exocrine system, often accompanied by vomiting, diarrhea, and urinary incontinence. The mnemonic SLUDGE is used to recall the specific elements of the syndrome: *s*alivation, *l*acrimation, *u*rination, *d*efecation, *g*astrointestinal cramping, and *e*mesis. The CNS (e.g., confusion, coma, and seizures) and the skeletal muscles (e.g., weakness and fasciculations) can also be involved. The pupils are often miotic. Cholinergic syndrome is most frequently caused by organophosphate or carbamate pesticide exposure, which may be through unsuspected dermal contamination. Anticholinergic agents are also the foundation of "nerve agents" such as sarin, which was used in the Tokyo subway attack. Recognition of the syndrome led to the use of atropine and cholinesterase regenerators, with a subsequent good outcome in many patients.[6]

Serotonin syndrome ensues when there is a drug interaction involving the selective serotonin reuptake inhibitors (SSRIs) or an overdose of an SSRI.[7] Fluoxetine (Prozac), sertraline (Zoloft), paroxetine (Paxil), fluvoxamine (Luvox), and citalopram (Celexa) are commonly used SSRIs. Other drugs that are serotonin reuptake inhibitors (SRIs) also inhibit the reuptake of other neurotransmitters and are therefore not specific. These drugs include venlafaxine (Effexor), nefazodone (Serzone), and mirtazapine (Remeron). Drug interactions between many drugs can cause the serotonin syndrome described in Chapter 159. These drugs include the SSRIs,

SRIs, monoamine oxidase inhibitors (MAOIs), tryptophan, sympathomimetics, tricyclic and other antidepressants, meperidine, dextromethorphan, and lithium. Because of the long-lasting effects of the SSRIs, the syndrome can occur when one of the active agents is ingested even weeks after use of an SSRI has been discontinued.

Serotonin syndrome is characterized by altered mental status, fever, agitation, tremor, myoclonus, hyper-reflexia, ataxia, incoordination, diaphoresis, shivering, and sometimes diarrhea.[7] The diagnosis relies on the drug history, and it is difficult to distinguish serotonin syndrome from an overdose of cocaine, lithium, or MAOIs; the neuroleptic malignant syndrome; or thyroid storm. Patients may deteriorate slowly and become critically ill after an apparently benign manifestation.

As possible toxidromes are investigated, the use of specific antidotes should be considered (Table 145-3).

TOXICOLOGY LABORATORY

A toxicology screen (usually urine, sometimes blood and urine, and occasionally including gastric contents) only rarely results in identification of the ingested agent for three major reasons. First, the laboratory does not attempt to screen for many substances, even commonly ingested agents that are capable of causing critical illness (Box 145-3).[8]

Second, the urine screen is often performed soon after the ingestion, when the drug concentration is too low for a positive result. Even the drug responsible for life-threatening symptoms (e.g., tricyclic antidepressant) may be negative on the urine screen soon after ingestion. Other drugs, such as γ-hydroxybutyrate, are present relatively briefly in blood and urine and may therefore be negative in samples collected even on the same day.

Third, drugs found on screening may not be those responsible for the initial symptoms, especially if the drugs are not quantified (e.g., benzodiazepines and cocaine parent compound). In such cases, a positive screen may not relate to the patient's current findings and symptoms. Drugs with a large volume of distribution or high fat solubility may be detected in urine for a long time after the last dose. Cocaine metabolites may be detected for days and marijuana for weeks after the last exposure. Proper interpretation of urine screens requires consideration of the patient's current clinical condition. In addition, the results of toxicology screens are not usually available until many hours after most of the important treatment decisions are made. Screening results rarely change the clinical management of patients.[8] Toxicology screens are often very expensive, and their use is not warranted in most routine drug overdoses. The full toxicology screen is most useful in patients who (1) present with their first psychotic episode or (2) are critically ill for an unknown reason when identification of an otherwise unsuspected toxin may change management.

Alternatives to a full toxicology screen include (1) obtaining discrete drug levels (e.g., acetaminophen, which should be considered in almost all intentional ingestions), (2) a qualitative urine screen for drugs of abuse, or (3) no toxicology tests.[8,9] Quantitative measurements of suspected drugs may be helpful. In the agitated or seizing patient, elevated salicylate, theophylline, or lithium levels would significantly alter the management. Several commercial urine testing kits have a rapid turnaround time, primarily for drugs of abuse. Electrolyte levels help identify metabolic acidosis by the carbon dioxide content ("bicarbonate level"), which should be repeated if low to ensure that the acidosis is resolving. A persistent, unexplained metabolic acidosis should prompt urine examination for oxalate crystals (suggestive of ethylene glycol poisoning), a serum salicylate level, and methanol and ethylene glycol levels. A normal arterial blood gas or electrolyte measurement does not rule out such ingestion because metabolic acidosis is delayed and does not appear until after metabolism of the acids from ethylene glycol/methanol or until after erratic and slow absorption of salicylate. Arterial blood gas measurement is rarely helpful but may confirm when pulse oximetry is misleadingly low with carboxyhemoglobin and methemoglobinemia.

Rhabdomyolysis should be diagnosed by obtaining a serum CPK level when there is severe agitation or hyperthermia, or if the patient is thought to have been unresponsive for a prolonged period of time (i.e., by history of presence or pressure sores). A urinary dipstick for blood (myoglobin) and a serum creatine kinase should be checked. Rhabdomyolysis and its treatment are discussed in Chapter 125. Noncardiogenic pulmonary edema on a chest radiograph suggests opioid or salicylate overdose. Selective abdominal radiographs can detect smuggled packets. Some drugs are radiopaque (e.g., heavy

BOX 145-3 DRUGS, CHEMICALS, AND GROUPS *NOT DETECTED* BY A COMPREHENSIVE TOXICOLOGY SCREEN

Ammonia
Anesthetic gases
Antibiotics
Anticoagulants
Beta-blockers
Borates
Bromides
Caustics/corrosives
Colchicine
Cyanide
Digitalis glycosides
Disulfiram
Ergot alkuloids
Ethylene glycol
Fentanyl and its derivatives
Fluorides
H_2 antagonists
Hallucinogens (e.g., LSD)
Herbicides
Household products
Hypoglycemics
Insect repellents
Isoniazid
Laxatives
Lithium
Metals
Monoamine oxidase inhibitors
Most antihypertensives
Most cardiac medications
Muscle relaxants
Mushrooms
Newer antidepressants (e.g., fluoxetine, sertraline, paroxetine, bupropion, buspirone)
Nitrates/nitrites
NSAIDs
Paraquat
Pesticides
Phenol
Plants
Solvents
Thyroid hormone
Vitamins

NSAIDs, nonsteroidal anti-inflammatory drugs.

Table 145-3 Antidotes Used in the Emergency Department

TOXIN USED FOR	ANTIDOTE	DOSE AND COMMENTS
Acetaminophen	*N*-acetylcysteine	140 mg/kg PO, then 70 mg/kg q4h for up to 17 doses or 150 mg/kg IV load over 1 hr with 50 mg/kg over 4 hr followed by 100 mg/kg over 16 hr
Anticholinergics	Physostigmine	1–2 mg IV in adults, 0.5 mg in children over 2 min for anticholinergic delirium, seizures, or dysrhythmias
Arsenic, lead, and mercury	BAL	3–5 mg/kg IM only
	D-Penicillamine	20–40 mg/kg/day; 500 mg tid in adults; may cross-react with penicillin in allergic patients
Benzodiazepines	Flumazenil	0.2 mg, then 0.3 mg, then 0.5 mg, up to 5 mg; not to be used if patient has signs of TCA toxicity; not approved for use in children, but probably safe
Black widow spider bite	*Latrodectus* antivenin	One vial by slow IV infusion is usually curative; may cause anaphylaxis
Beta-blockers	Glucagon	5–10 mg in adults, then infusion of same dose per hour
	Insulin and glucose	10 U insulin with dextrose 25 g initially, then 0.1–1.0 U/kg/hr, 10–30 g/hr
Calcium channel blockers	Calcium	1 g calcium chloride IV in adults, 20–30 mg/kg/dose in children, over a few minutes with continuous monitoring. Repeat as needed
	Glucagon	5–10 mg in adults, then infusion of same dose per hour
	Insulin and glucose	10 U insulin with dextrose 25 g initially, then 0.1–1.0 U/kg/hr with 10–30 g/hr
Cyanide	Hydroxycobalamin	5 mg in 100 ml of NS over 15 min. Repeat if necessary
Cyanide, hydrogen sulfide	Sodium thiosulfate	50 mL of 25% (12.5 g; 1 ampule) in adults; 1.65 mL/kg IV in children
	Sodium nitrate	10 mL of 3% (300 mg; 1 ampule) in adults; 0.33 mL/kg slowly IV in children
	Hydroxycobalamin	50 mg in 50 mL given over 15 min
Digitalis glycosides	Digoxin-specific Fab	10–20 vials if patient in ventricular fibrillation; otherwise dose fragments based on serum digoxin concentration or amount ingested
Ethylene glycol	Fomepizole	15 mg/kg × 1, then 10 mg/kg q12h × 4, until ethylene glycol < 20 mg/dL. Adjust dose during dialysis
	Pyridoxine	100 mg IV daily
	Thiamine	100 mg IV
Hydrofluoric acid	Calcium gluconate	3.5 g in 5 oz of KY jelly topical; apply liberally to affected skin
Iron	Deferoxamine	15 mg/kg/hr IV; higher doses reported to be safe
Isoniazid, hydrazine, and monomethylhydrazine	Pyridoxine	5 g in adults, 1 g in children, if ingested dose unknown; antidote may cause neuropathy in very large doses
Lead	DMSA (succimer)	Reported useful for arsenic and lead as well; one 100-mg capsule per 10-kg body weight tid for 1 wk, then bid, with chelation breaks
	EDTA	75 mg/kg/day by continuous infusion; watch for nephrotoxicity, best done in hospital
Methanol	Folate or leucovorin	50 mg IV q4h in adults while patients have serious toxicity
	Ethanol	Loading dose, 10 mL/kg of 10%; maintenance dose, 0.15 mL/kg/hr of 10%; double rate during dialysis
	Fomepizole	15 mg/kg × 1, then 10 mg/kg q12h × 4, until methanol < 20 mg/dL. Adjust dose during dialysis
Methemoglobin-forming agents	Methylene blue	1–2 mg/kg IV, one 10-mL dose of 10% solution (100 mg) is typical for an adult without anemia
Opioids	Nalmefene	2 mg; much longer half-life than naloxone
	Naloxone	2 mg; less to avoid narcotic withdrawal, more if inadequate response; same dose in children
Organophosphates and carbamates	Atropine	Test dose, 1–2 mg IV in adults, 0.03 mg/kg in children; titrate to drying of pulmonary secretions
	Protopam	Loading dose, 1–2 g IV in adults, 25–50 mg/kg in children; adult maintenance, 500 mg/hr or 1–2 g q4–6h
Rattlesnake bite	*CroFab* antivenin	Five vials minimum dose by infusion in normal saline; increases in rate dependent on patient tolerance; may cause anaphylaxis
Serotonin syndrome	Cyproheptadine	4 mg PO as needed; no parenteral form available; antidote may cause anticholinergic effects
Sulfonureas	Octreotide	50 µg SC q12h, 5–10 µg/kg/24 hr IV
Tricyclic antidepressants	Bicarbonate	44–88 mEq in adults, 1–2 mEq/kg in children; best used by IV push and not by slow infusion
Valproic acid	Carnitine	100 mg/kg IV or PO loading dose with 25 mg/kg q6h

BAL, British anti-Lewisite; DMSA, dimercaptosuccinic acid; EDTA, ethylenediaminetetraacetic acid; TCA, tricyclic antidepressant.

metals, phenothiazines, potassium, calcium, and chlorinated hydrocarbons such as chloral hydrate), but radiography is rarely helpful in the evaluation of a poisoned patient except to monitor the decontamination of iron, lead, or body packets.[10]

DECONTAMINATION

Gastric decontamination rarely affects the clinical outcome in the undifferentiated poisoned patient and should not be undertaken routinely.[11,12] Children with accidental ingestion rarely consume enough drug to cause symptoms, and the fatality rate for those cases is less than 0.0025%.[13] Decontamination with activated charcoal (AC) has not been proven to improve outcome either in the undifferentiated overdose patient or in any specific poisoning. It should be considered only in the small number of cases in which it is early enough (<1 hour after ingestion) to make a difference or for medications of a type and in quantities that may be truly toxic beyond the requirement for supportive care. These medications include beta-blockers, calcium channel blockers, and cyclic antidepressants. Because gastric decontamination has not been shown to be of benefit and carries some risk, particularly if a nasogastric tube is required, it should be used selectively and with caution, if at all.[11,12,14-16]

If charcoal is used, 50 g of an oral slurry of AC is usually sufficient. If the patient is obtunded or uncooperative and the benefits of AC administration are thought to outweigh the risks (principally aspiration), intubation should be considered and the AC can be administered through a nasogastric tube. A cathartic, such as sorbitol, was formerly recommended to speed AC transit through the gut, but cathartics have never been shown to be of benefit and, in general, should be avoided. Repeated doses of sorbitol can cause dehydration. Agents that do not adsorb to charcoal include ions (e.g., acids and alkalies, lithium, borates, and bromides), hydrocarbons, metals (e.g., iron), and ethanol, but adsorption to charcoal does not equate to lesser toxicity or improved outcome.

Whole-bowel irrigation with a polyethylene glycol solution, although of no proven outcome benefit, rarely may be used in certain cases of severe, recent ingestion of lithium or metals such as iron or lead or in patients with ingestion of sustained-release formulations of highly toxic drugs. It has also been used to aid in the evacuation of drug packets from body packers.[17] Whole-bowel irrigation is unpleasant for both patient and staff, and it should be considered only after consultation with a toxicologist. It is typically done by placing a nasogastric tube and continuously infusing a bowel preparation solution, such as Go-Lytely, beginning at 1 or 2 L/hour and continued until the rectal effluents are clear.

Gastric lavage is rarely, if ever, indicated and should be considered only when a patient is seen within a few minutes (<1 hour) after the ingestion of a highly toxic substance (e.g., calcium channel blocker and cyclic antidepressant). Gastric lavage has not been shown to improve the clinical course or outcome of undifferentiated poisoned patients, although it has not been studied in selected higher risk populations.[18] In the rare circumstances in which gastric lavage is performed, a large (30-F or greater) orogastric tube is used, and specially designed lavage systems with large-bore tubes are available for this purpose. Syrup of ipecac has not been shown to change the clinical outcome of poisonings and is no longer used in emergency departments and rarely in out-of-hospital settings. Its use has been restricted because of its abuse by patients with bulimia.

Exposure of the eye to caustic chemicals and irritants requires immediate irrigation with large amounts of water or readily available fluid, as outlined in Chapter 151. It is more important to begin the irrigation immediately, preferably before transfer to the emergency department. Exposure to a gas does not require decontamination because the patient and rescuers are not at risk once the patient is removed from the toxic environment. The exception is when the patient's skin or clothing is contaminated with a liquid that is evaporating. The most important intervention to limit dermal exposure is to remove all clothing as soon as possible, ideally at the scene. Skin should be irrigated with warm water and with attention to skin folds and other areas that might be missed. This includes the axilla, beneath nails, behind the knees, the genitalia, and in the scalp. For hydrocarbons or solvents, soap can be added. The skin should not be abraded with overly aggressive scrubbing, which could increase skin absorption. Ideally, skin decontamination occurs as soon as possible after exposure, at an out-of-hospital site before transport.

DISPOSITION AND CONSULTATION

The decision to admit a patient is not difficult when the patient manifests serious toxicity. When the patient is minimally symptomatic but has ingested a potentially dangerous substance, the decision is more difficult. Identification of an agent that causes a particular risk for the patient, especially cardiovascular instability, seizures, or respiratory depression, generally mandates admission to the hospital or to an observation unit in the emergency department. A 6-hour period of observation for a minimally symptomatic patient is usually sufficient, except for some extended-release preparations. Patients with cardiac dysrhythmia, conduction disturbance, altered mental status requiring intubation, or the need for frequently titrated agents (e.g., pressors) should be admitted to the intensive care unit or a monitored inpatient unit. If the patient is acutely suicidal, a sitter or secure environment may be required.

Regional poison centers use a single nationwide toll-free number, 1-800-222-1222, and can provide specific, current advice, especially for more esoteric or unfamiliar poisons. Consultation with a medical toxicologist is particularly helpful when an uncommon agent has been ingested, the patient is not following the anticipated clinical course, or specific interventions such as administration of antibody therapy or dialysis are contemplated.

KEY CONCEPTS

- A thorough history from many sources is the key to toxicologic diagnosis.
- Common toxidromes should guide judicious use of antidotes.
- Minimally symptomatic patients do not benefit from toxicology screening or extensive laboratory investigation.
- Good supportive care is the key to management.
- Activated charcoal is rarely indicated in overdose, and other methods of gut decontamination (gastric lavage and whole-bowel irrigation) are virtually never helpful. Activated charcoal may decrease absorption of many drugs, but it has not been shown to improve outcome, and its use should be carefully weighed against potential complications.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 18 Chest Pain

James E. Brown and Glenn C. Hamilton

PERSPECTIVE

Nearly 6 million patients present to the emergency department (ED) each year with complaints of chest pain, constituting 5% of all patients seen in EDs in the United States.[1] Chest pain is a symptom caused by several life-threatening diseases and has a broad differential diagnosis. It is complicated by a frequent disassociation between intensity of symptoms and signs and seriousness of underlying pathology.

Epidemiology

The epidemiology of the critical diagnoses causing chest pain varies widely. Acute coronary syndromes (ACS), aortic dissection, pulmonary embolism (PE), pneumothorax, pericarditis with tamponade, and esophageal rupture are potentially catastrophic causes of chest pain. Due to its high incidence and potential lethality, ACS is the most significant potential diagnosis in the ED. Of all deaths in the United States, 36% are attributed to cardiovascular diseases; these account for approximately 870,000 deaths per year.[2] Historically, emergency physicians misdiagnose 3 to 5% of myocardial infarctions (MIs), accounting for 25% of malpractice losses in emergency medicine.[3,4] Thoracic aortic dissection has an incidence of 0.5 to 1 per 100,000 population with a mortality rate exceeding 90% if misdiagnosed. The true incidence of PE is unclear, with estimates of 70 per 100,000. This equates to approximately 100,000 PE cases per year in the United States.[5] Although the incidence of tension pneumothorax is also unclear, the incidence of spontaneous pneumothorax ranges from 2.5 to 18 per 100,000 total patients. The total incidence of esophageal rupture is 12.5 cases per 100,000 persons. The true incidence of pericarditis is unknown, but is diagnosed in 1 of every 1000 hospital admissions.[6] Up to 5% of ED chest pain patients without acute ST elevation MI may have pericarditis.[7]

Pathophysiology

Afferent fibers from the heart, lungs, great vessels, and esophagus enter the same thoracic dorsal ganglia. Through these visceral fibers, each organ produces the same indistinct quality and location of pain. The quality of visceral chest pain varies widely and is described as "burning," "aching," "stabbing," or "pressure." Since dorsal segments overlap three segments above and below a level, disease of a thoracic origin can produce pain anywhere from the jaw to the epigastrium. Radiation of pain is caused by somatic afferent fibers synapsing in the same dorsal root ganglia as the thoracic viscera. This stimulation can "confuse" the patient's central nervous system into misperceiving that the pain originates in the arms or shoulders.

DIAGNOSTIC APPROACH

Differential Considerations

Due to the indistinct nature of visceral pain, the differential diagnosis of chest pain is broad and includes many of the most critical diagnoses in medicine and many nonemergent conditions (Table 18-1).

Rapid Stabilization and Assessment

The initial questions are, "Must I intervene immediately?", and "What are the life-threatening possibilities in this patient?" The answers are usually apparent within the first few minutes after assessing the patient's appearance and vital signs. One of the critical diagnoses is tension pneumothorax. If a patient presents with chest pain, respiratory distress, shock, and unilateral reduction or absence of breath sounds, immediate intervention with needle or tube thoracostomy is required. Additionally, patients with severe derangements in vital signs require stabilizing treatment during a search for the precipitating cause. Patients who present with respiratory distress require immediate intervention and lead the emergency physician to consider a more serious cause of the pain (Fig. 18-1; also see Chapter 17).

All patients, except those with obvious benign causes of chest pain, must have an electrocardiogram (ECG) within minutes of reporting their pain. This ECG should be read for acute MI by the emergency physician as soon as it is completed. Patients with positive ECG findings and those considered at high risk are triaged directly to the treatment area and monitored. Symptomatic derangements in vital signs are addressed. If vital signs are stable, a focused history and physical examination are performed. Most patients also require a chest radiograph to evaluate the chest pain. If a cardiac cause is suggested and vital signs are stable, pain relief with nitroglycerin (0.4 mg sublingual every 3–5 minutes) may be appropriate. Aspirin (81–325 mg) is a consideration for patients without hemorrhagic disorders, known allergies, or vascular dissections. Clopidogrel (loading dose 300 mg) or other anti-

Table 18-1 Differential Diagnosis of Chest Pain

ORGAN SYSTEM	CRITICAL DIAGNOSES	EMERGENT DIAGNOSES	NONEMERGENT DIAGNOSES
Cardiovascular	Acute myocardial infarction Acute coronary ischemia Aortic dissection Cardiac tamponade	Unstable angina Coronary spasm Prinzmetal's angina Cocaine-induced pericarditis or myocarditis	Valvular heart disease Aortic stenosis Mitral valve prolapse Hypertrophic cardiomyopathy
Pulmonary	Pulmonary embolus Tension pneumothorax	Pneumothorax Mediastinitis	Pneumonia Pleuritis Tumor Pneumomediastinum
Gastrointestinal	Esophageal rupture (Boerhaave)	Esophageal tear (Mallory-Weiss) Cholecystitis Pancreatitis	Esophageal spasm Esophageal reflux Peptic ulcer Biliary colic
Musculoskeletal			Muscle strain Rib fracture Arthritis Tumor Costochondritis Nonspecific chest wall pain
Neurologic			Spinal root compression Thoracic outlet Herpes zoster Postherpetic neuralgia
Other			Psychologic Hyperventilation

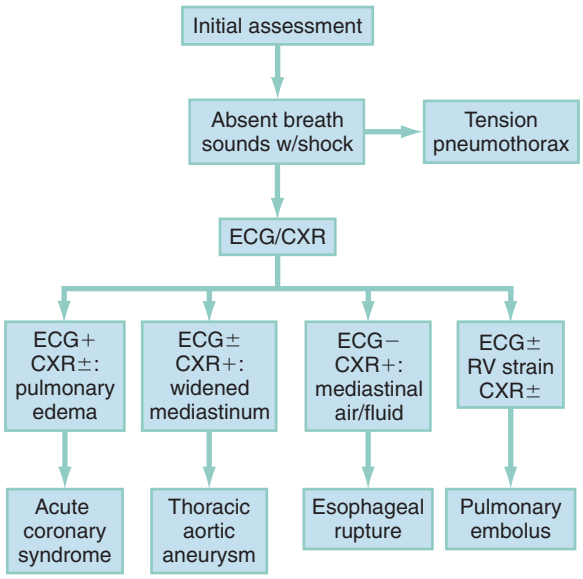

Figure 18-1. Initial assessment of critical diagnoses. CXR, chest x-ray; ECG, electrocardiogram; RV, right ventricular.

platelet agents may also be an alternative. Patients with low voltage on the ECG, diffuse ST segment elevation, elevated jugular venous pressure on examination,[8] and signs of shock should undergo prompt bedside cardiac ultrasound.

Pivotal Findings

The broad and complex nature of chest pain defies application of a simple algorithm. An organized approach to a patient with chest pain is essential, however, to ensure that all causes are evaluated appropriately. The history and physical examination are key to diagnosis. Information pertinent to the differential diagnosis is obtained by the history, physical examination, and ECG in 80 to 90% of patients.

History

1. The patient is asked to describe the character of the pain or discomfort. Descriptions such as "squeezing," "crushing," or "pressure" lead the emergency physician to suspect a cardiac ischemic syndrome, although cardiac ischemia can also be characterized by nonspecific discomfort, such as "bloating" or "indigestion." "Tearing" pain that may migrate from the front to back or back to front is the classic description in aortic dissection. "Sharp" or "stabbing" pain is seen more in pulmonary and musculoskeletal diagnoses. Patients complaining of a "burning" or "indigestion" type of pain may initially be thought to have a gastrointestinal etiology, but due to the visceral nature of chest pain, all causes of pain may present with any of the preceding descriptions. Of note, descriptors may vary among ethnic groups, and, for example, "sharp" may mean "severe."
2. Additional history about the patient's activity at the onset of pain may be helpful. Pain occurring during exertion suggests an ischemic coronary syndrome, whereas progressive onset of pain at rest suggests acute MI. Pain of sudden onset is more typical with aortic dissection, PE, or pneumothorax. Pain after meals is more indicative of a gastrointestinal cause.
3. The severity of pain is commonly quantified using a 1-to-10 pain scale. Alterations in pain severity are documented at times of onset, peak, present, and after intervention.
4. The location of the discomfort is described. Pain that is localized to a small area is more likely to be somatic versus visceral in origin. Pain localized at the periphery of the

chest is more likely with a pulmonary rather than cardiac etiology. Lower chest or upper abdominal pain may be of cardiac or gastrointestinal origin.

5. Any description of radiation of pain should be noted. Transthoracic pain through to the back should suggest aortic dissection or gastrointestinal causes, especially pancreatitis or posterior ulcer. Inferioposterior myocardial ischemia may also present primarily as thoracic back pain. Radiation to the arms, neck, or jaw increases the likelihood of cardiac ischemia.[9,10] Pain located primarily in the back, especially interscapular back pain that migrates to the base of the neck, suggests aortic dissection.[11]
6. Duration of pain is another important historical factor. Pain that lasts a few seconds is rarely of cardiac origin.[12] Pain that is exertional but lasts for only a few minutes after rest may be a manifestation of cardiac ischemia.[9] Pain that is maximal at onset may be due to aortic dissection.[11] Pain that is not severe and persists over the course of days is less likely to be of serious origin than pain that is severe or has a stuttering or fluctuating course.
7. The clinician should consider aggravating or alleviating factors. Pain that worsens with exertion and improves with rest is more likely related to coronary ischemia.[9] Pain related to meals is more suggestive of a gastrointestinal cause. Pain that worsens with respiration is seen more often with pulmonary, pericardial, and musculoskeletal causes.
8. Other associated symptoms may suggest the visceral nature of the pain (Table 18-2). Diaphoresis should lead to an increased clinical suspicion for a serious or visceral cause. Hemoptysis, a classic PE sign, is rarely seen.[13] Near-syncope and syncope lead to higher likelihood of a cardiovascular cause or PE. Dyspnea is seen in cardiovascular and pulmonary disease. Nausea and vomiting may be seen in cardiovascular and gastrointestinal complaints.
9. A history of prior pain and the diagnosis of that episode can facilitate the diagnostic process, but the physician must be wary of prior presumptive diagnoses that may be misleading. A prior history of cardiac testing, such as stress testing, echocardiography, or angiography, may be useful in determining if the current episode is suggestive of cardiac disease. Similarly, patients with previous spontaneous pneumothorax or PE[14] are at increased risk of recurrence.
10. The presence of risk factors for a particular disease is primarily of value as an epidemiologic marker for large population studies (Box 18-1). In the ED, presence of risk factors in an individual patient without established disease has minimal or no effect on the clinical likelihood (pretest probability) of a specific disease process.

Table 18-2 Significant Symptoms of Chest Pain

SYMPTOM	FINDING	DIAGNOSIS
Pain	Severe, crushing, pressure, substernal, exertional, radiation to jaw, neck, shoulder, arm	Acute MI Coronary ischemia Unstable angina Coronary spasm
	Tearing, severe, radiating to or located in back, maximum at onset, may migrate to upper back or neck	Aortic dissection
	Pleuritic	Esophageal rupture Pneumothorax Cholecystitis Pericarditis Myocarditis
	Indigestion or burning	Acute MI Coronary ischemia Esophageal rupture Unstable angina Coronary spasm Esophageal tear Cholecystitis
Associated syncope/ near-syncope		Aortic dissection PE Acute MI Pericarditis Myocarditis
Associated dyspnea (SOB, DOE, PND, orthopnea)		Acute MI Coronary ischemia PE Tension pneumothorax Pneumothorax Unstable angina Pericarditis
Associated hemoptysis		PE
Associated nausea/ vomiting		Esophageal rupture Acute MI Coronary ischemia Unstable angina Coronary spasm Esophageal tear Cholecystitis

DOE, dyspnea on exertion; MI, myocardial infarction; PE, Pulmonary embolism; PND, paroxysmal nocturnal dyspnea; SOB, shortness of breath.

Physical Examination

Specific findings may be found in a variety of causes (Table 18-3).

Ancillary Studies

The two most commonly performed studies in patients with chest pain are the chest radiograph and 12-lead ECG (Table 18-4). An ECG should be performed within 10 minutes of arrival in all patients with chest pain in whom myocardial ischemia is a possibility.[15,16] This generally includes all male patients 33 years old and older and female patients over the age of 39 who complain of pain from the umbilicus to the mandible unless a noncardiac cause is readily apparent. Rapid acquisition of the ECG facilitates the diagnosis of acute MI and expedites the National Heart, Lung, and Blood Institute's recommended "door to treatment" times from arrival to percutaneous coronary intervention (PCI) or thrombolytic therapy in acute MI. Patients with a new injury pattern on ECG (Table 18-5) or new ischemic ECG changes should have appropriate therapy instituted at this point (Fig. 18-2; see also Chapter 77). An ECG showing right ventricular strain pattern, in the appropriate setting, should raise the clinical suspicion for PE. Diffuse ST segment elevation helps make the diagnosis of pericarditis.

A chest radiograph is performed for patients with a possibly serious cause of chest pain. Pneumothorax is definitively diagnosed at this point. A wide mediastinum or ill-defined aortic knob increases the clinical suspicion for acute aortic dissection. Pleural effusion, subcutaneous air, or mediastinal air-fluid

Table 18-3 Pivotal Findings in Physical Examination

SIGN	FINDING	DIAGNOSES
Appearance	Acute respiratory distress	PE Tension pneumothorax Acute MI Pneumothorax
	Diaphoresis	Acute MI Aortic dissection Coronary ischemia PE Esophageal rupture Unstable angina Cholecystitis Perforated peptic ulcer
Vital signs	Hypotension	Tension pneumothorax PE Acute MI Aortic dissection (late) Coronary ischemia Esophageal rupture Pericarditis Myocarditis
	Tachycardia	Acute MI PE Aortic dissection Coronary ischemia Tension pneumothorax Esophageal rupture Coronary spasm Pericarditis Myocarditis Mediastinitis Cholecystitis Esophageal tear (Mallory-Weiss)
	Bradycardia	Acute MI Coronary ischemia Unstable angina
	Hypertension	Acute MI Coronary ischemia Aortic dissection (early)
	Fever	PE Esophageal rupture Pericarditis Myocarditis Mediastinitis Cholecystitis
	Hypoxemia	PE Tension pneumothorax Pneumothorax
Cardiovascular examination	Significant difference in upper extremity blood pressures	Aortic dissection
	Narrow pulse pressure	Pericarditis (with effusion)
	New murmur	Acute MI Aortic dissection Coronary ischemia
	S_3/S_4 gallop	Acute MI Coronary ischemia
	Pericardial rub	Pericarditis
	Audible systolic "crunch" on cardiac auscultation (Hamman's sign)	Esophageal rupture Mediastinitis
	JVD	Acute MI Coronary ischemia Tension pneumothorax PE Pericarditis
Pulmonary examination	Unilateral diminished/absent breath sounds	Tension pneumothorax Pneumothorax
	Pleural rub	PE
	Subcutaneous emphysema	Tension pneumothorax Esophageal rupture Pneumothorax Mediastinitis
	Rales	Acute MI Coronary ischemia Unstable angina
Abdominal examination	Epigastric tenderness	Esophageal rupture Esophageal tear Cholecystitis Pancreatitis
	Left upper quadrant tenderness	Pancreatitis
	Right upper quadrant tenderness	Cholecystitis
Extremity examination	Unilateral leg swelling, warmth, pain, tenderness, or erythema	PE
Neurologic examination	Focal findings Stroke	Aortic dissection Acute MI Coronary ischemia Aortic dissection Coronary spasm

JVD, jugular venous distention; MI, myocardial infarction; PE, pulmonary embolism.

BOX 18-1 RISK FACTORS ASSOCIATED WITH POTENTIALLY CATASTROPHIC CAUSES OF CHEST PAIN

- Acute coronary syndromes
  - Past or family history of coronary artery disease
  - Age
    - Men >33 years
    - Women >40 years
  - Diabetes mellitus
  - Hypertension
  - Cigarette use/possible passive exposure
  - Elevated cholesterol (LDL)/triglycerides
  - Sedentary lifestyle
  - Obesity
  - Postmenopausal
  - Left ventricular hypertrophy
  - Cocaine abuse
- Pulmonary embolism
  - Prolonged immobilization
  - Surgery >30 minutes in last 3 mo
  - Prior deep vein thrombosis or pulmonary embolus
  - Pregnancy or recent pregnancy
  - Pelvic or lower extremity trauma
  - Oral contraceptives with cigarette smoking
  - Congestive heart failure
  - Chronic obstructive pulmonary disease
  - Obesity
  - Past medical or family history of hypercoagulability
- Aortic dissection
  - Hypertension
  - Congenital disease of the aorta or aortic valve
  - Inflammatory aortic disease
  - Connective tissue disease
  - Pregnancy
  - Arteriosclerosis
  - Cigarette use
- Pericarditis or myocarditis
  - Infection
  - Autoimmune disease (e.g., systemic lupus erythematosus)
  - Acute rheumatic fever
  - Recent myocardial infarction or cardiac surgery
  - Malignancy
  - Radiation therapy to mediastinum
  - Uremia
  - Drugs
  - Prior pericarditis
- Pneumothorax
  - Prior pneumothorax
  - Valsalva's maneuver
  - Chronic lung disease
  - Cigarette use

Table 18-4 Ancillary Testing of Patients with Chest Pain

TEST	FINDING	DIAGNOSIS
ECG	New injury	Acute MI Aortic dissection
	New ischemia	Coronary ischemia Coronary spasm
	RV strain	PE
	Diffuse ST segment elevation	Pericarditis
CXR	Pneumothorax with mediastinal shift	Tension pneumothorax
	Wide mediastinum	Aortic dissection
	Pneumothorax	Esophageal rupture Pneumothorax
	Effusion	Esophageal rupture
	Increased cardiac silhouette	Pericarditis
	Pneumomediastinum	Esophageal rupture Mediastinitis
ABG	Hypoxemia, A-a gradient	PE
$\dot{V}/\dot{Q}$ scan or spiral CT	High probability or any positive in patient with high clinical suspicion	PE

ABG, arterial blood gas; CT, computed tomography; ECG, electrocardiogram; MI, myocardial infarction; RV, right ventricular.

Table 18-5 Electrocardiogram Findings in Ischemic Chest Pain

Classic myocardial infarction	ST segment elevation (>1 mm) in contiguous leads; new LBBB; Q waves ≥0.04 sec duration
Subendocardial infarction	T wave inversion or ST segment depression in concordant leads
Unstable angina	Most often normal or nonspecific changes; may see T wave inversion
Pericarditis	Diffuse ST segment elevation; PR segment depression

LBBB, left bundle-branch block.

level may be seen in esophageal rupture. Increased cardiac silhouette may indicate pericarditis or cardiomyopathy.

Pneumomediastinum is seen with esophageal rupture and mediastinitis. A serum D-dimer assay may help discriminate patients with PE from those with a possible gastrointestinal cause. A low serum D-dimer in a patient without a high pretest probability of PE effectively excludes the diagnosis.[13,17,18] (see Chapter 87.)

Patients at high pretest probability for PE should undergo diagnostic imaging (multidetector computed tomography [CT], or, less commonly, pulmonary angiography or a ventilation-perfusion lung scan).[19] High pretest probability warrants initiation of anticoagulation (heparin or low-molecular-weight heparin) therapy in the ED before the imaging study, in the absence of a contraindication.

Patients with suspected thoracic aortic dissection may be evaluated by CT angiography, transesophageal echocardiography, or magnetic resonance imaging. Selection of imaging modality depends on patient status and availability of the testing equipment.[20]

CT with a 64 or higher detector scanner has the potential to rule out all of the life-threatening causes of chest pain. Although the "triple rule out" of ACS, PE, and thoracic dissection are the causes most commonly discussed, pneumothorax, mediastinitis, and pericardial effusions are also diagnosed with CT.[21,22]

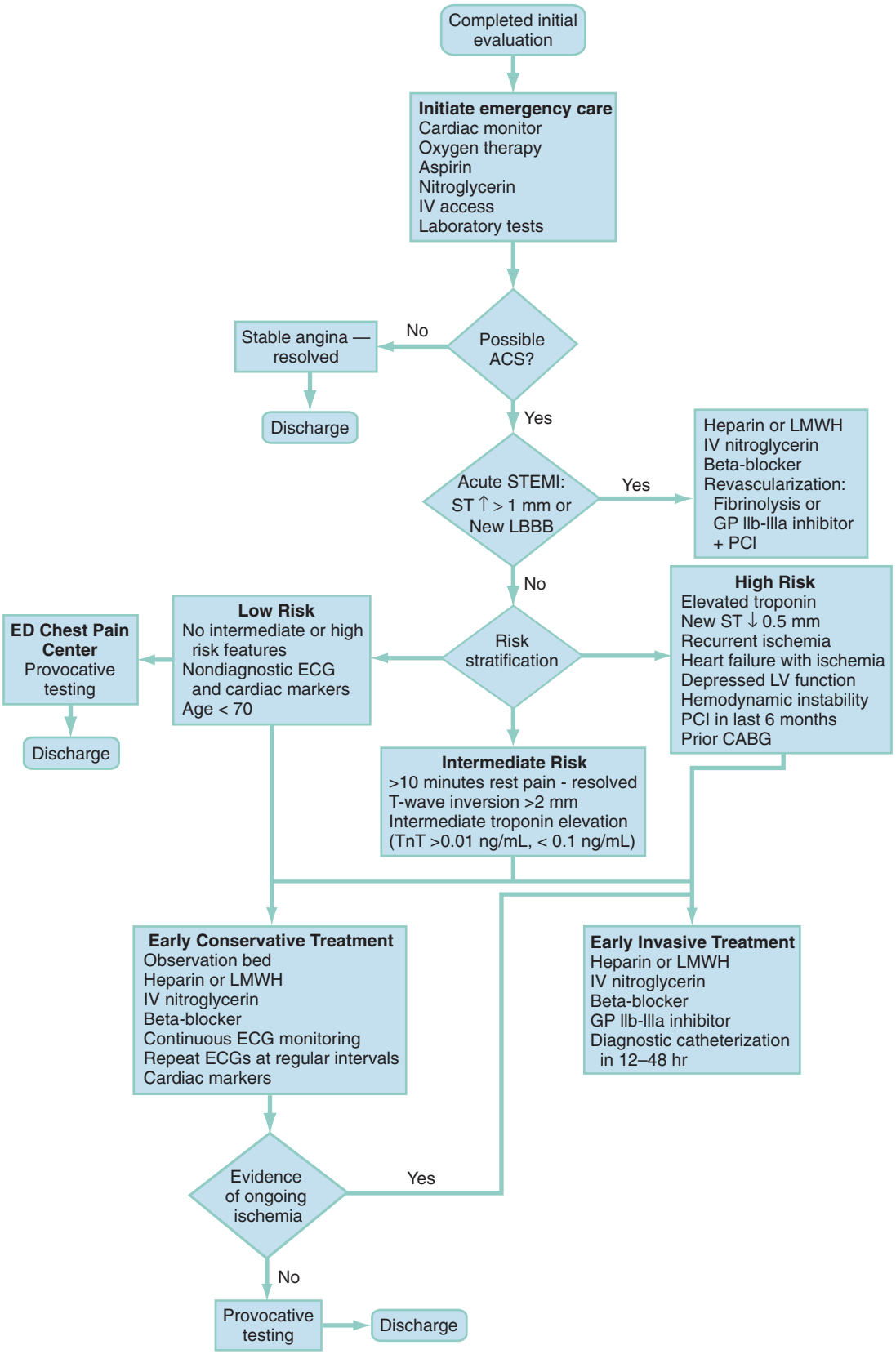

Figure 18-2. Clinical guidelines for emergency department management of chest pain of myocardial ischemic origin. ACS, acute coronary syndrome; CABG, coronary artery bypass graft; ECG, electrocardiogram; GP, glycoprotein; IV, intravenous; LBBB, left bundle-branch block; LMWH, low-molecular-weight heparin; LV, left ventricular; MI, myocardial infarction; PCI, percutaneous coronary intervention; ST, echocardiographic peak; STEMI, ST segment evaluation myocardial infarction; TnT, troponin T. (Adapted from Gibler WB, Cannon CP, Blonikalns AL, et al: Practical implementation of the guidelines for unstable angina/non-ST-segment elevation myocardial infarction in the emergency department: A scientific statement from the American Heart Association Council on Clinical Cardiology (Subcommittee on Acute Cardiac Care), Council on Cardiovascular Nursing, and Quality of Care and Outcomes Research Interdisciplinary Working Group, in Collaboration with the Society of Chest Pain Centers. Circulation 111:2699, 2005.)

Table 18-6 Causes and Differentiation of Potentially Catastrophic Illness Presenting with Central Chest Pain or Discomfort

	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORTING HISTORY	PREVALENCE IN EMERGENCY DEPARTMENT	PHYSICAL EXAMINATION	USEFUL TESTS	ATYPICAL OR ADDITIONAL ASPECTS
Myocardial Infarction	Discomfort is usually moderately severe to severe and rapid in onset. May be more "pressure" than pain. Usually retrosternal, may radiate to neck, jaw, both arms, upper back, epigastrium, and sides of chest (left more than right). Lasts more than 15–30 min and is unrelieved by NTG	Diaphoresis, nausea, vomiting, dyspnea	May be precipitated by emotional stress or exertion. Often comes on at rest. May come on in early awakening period. Prodromal pain pattern often elicited. Previous history of MI or angina. Age >40 years, positive risk factors, and male sex increase possibility	Common	Patients are anxious and uncomfortable. Blood pressure usually is elevated, but normotension and hypotension are seen. The heart rate is usually mildly increased, but bradycardia can be seen. Patients may be diaphoretic and show peripheral poor perfusion. There are no diagnostic examination findings for MI, although S_3 and S_4 heart sounds and new murmur are supportive	ECG changes (new Q waves or ST segment–T wave changes) occur in 80% of patients. CK-MB and troponins are helpful if elevated, but may be normal	Pain may present as "indigestion" or "unable to describe." Other atypical presentations include altered mental status, stroke, angina pattern without extended pain, severe fatigue, syncope. Elderly may present with weakness, congestive heart failure, or chest tightness. 25% of nonfatal MIs are unrecognized by patient. The pain may have resolved by the time of evaluation
Unstable Angina	Changes in pattern of preexisting angina with more severe, prolonged, or frequent pain (crescendo angina). Pain usually lasts >10 min. Angina at rest lasting 15–20 min or new-onset angina (duration <2 mo) with minimal exertion. Pattern of pain change important in gauging risk for AMI. Unpredictable responses to NTG and rest	Often minimal. May have mild diaphoresis, nausea, dyspnea with pain. Increasing pattern of dyspnea on exertion	Not clearly related to precipitating factors. May be a decrease in amount of physical activity that initiates pain. Previous history of MI or angina. Over 40 years old, presence of risk factors, and male sex increase probability	Common	Nonspecific findings of a transient nature, may have similar cardiac findings as in MI, especially intermittent diaphoresis	Often no ECG or enzyme changes. Variant angina (Prinzmetal's) has episodic pain, at rest, often severe, with prominent ST segment elevation	May be pain-free at presentation. Full history is essential. Fewer than 15% of patients hospitalized for unstable angina go on to acute MI. May respond to NTG. May manifest similarly to non–Q wave infarction

Aortic Dissection	90% of patients have rapid-onset severe chest pain that is maximal at beginning. Radiates anteriorly in chest to the back interscapular area or into abdomen. Pain often has a "tearing" sensation, and may migrate	Neurologic complications of stroke, peripheral neuropathy, paresis or paraplegia, abdominal and extremity ischemia possible	Median age 59 years. History of hypertension in 70–90% of patients. 3:1 ratio males to females. Marfan's syndrome and congenital bicuspid aortic valves have increased incidence	Rare	Often poorly perfused peripherally but with elevated BP. In 50–60% of cases, there is asymmetrical decrease or absence of peripheral pulses. 50% of proximal dissections cause aortic insufficiency. Other vascular occlusions: coronary (1–2%), mesentery, renal, spinal cord. New-onset pericardial friction rub or aortic insufficiency murmur supportive of diagnosis	ECG usually shows left ventricular hypertrophy, nonspecific changes. Chest film shows abnormal aortic silhouette (90%). Aortic angiography has diagnostic accuracy of 95–99%. Transesophageal echocardiogram, CT, MRI most useful in screening	Rare for patient to present pain-free. May present with neurologic complications. Physical examination findings may be minimal. Dissection into coronary arteries can mimic MI Ascending aortic aneurysms are more often approached surgically. Descending are generally managed medically
Pulmonary Embolism	Pain is more often lateral-pleuritic. Central pain is more consistent with massive embolus. Abrupt in onset and maximal at beginning. May be episodic or intermittent	Dyspnea and apprehension play a prominent role, often more than pain. Cough accompanies about half the cases Hemoptysis occurs in <20%. Angina-like pain may occur in 5%	Often some period of immobilization has occurred, e.g., postoperative. Pregnancy, oral contraceptives, heart disease, and cancer are all risk factors. Previous DVT or PE is the greatest risk factor	Uncommon in ambulatory patients, but common in departments with high volumes of elderly or medically complex patients	Patients are anxious and often have a respiratory rate >16/min. Tachycardia, inspiratory rales, and an increased pulmonic second sound are common. Fever, phlebitis, and diaphoresis are seen in 30–40% of patients. Wheezes and peripheral cyanosis are less common	Arterial blood gases show Po_2 < 80 mm Hg in 90%. Widened A-a gradient is helpful. Chest film is usually normal, although 40% show some volume loss, oligemia, or signs of consolidation due to pulmonary infarction. Lung perfusion scan rules out, if truly negative	Patients may present with dyspnea with or without bronchospasm. Acute mortality rate is 10%. Emboli usually from lower extremities above knee, prostate/pelvis venous plexus, right heart. May be subtle cause of COPD exacerbation

Table 18-6 Causes and Differentiation of Potentially Catastrophic Illness Presenting with Central Chest Pain or Discomfort—cont'd

	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORTING HISTORY	PREVALENCE IN EMERGENCY DEPARTMENT	PHYSICAL EXAMINATION	USEFUL TESTS	ATYPICAL OR ADDITIONAL ASPECTS
Pneumothorax	Pain is usually acute and maximal at onset. Most often lateral-pleuritic, but central pain can occur in large pneumothorax	Dyspnea has a prominent role. Hypotension and altered mental states occur in tension pneumothorax	Chest trauma, previous episode, or asthenic body type	Infrequent	Decreased breath sounds, increased resonance on percussion. Elevated pressure in neck veins occurs in tension pneumothorax	Chest film definitive. Inspiratory and expiratory films may enhance contrast between air and lung parenchyma. Tension pneumothorax should be diagnosed on physical examination	May be subtle in COPD, asthma, cystic fibrosis. Can be complicated by pneumomediastinum
Esophageal Rupture	Pain usually is preceded by vomiting and is abrupt in onset. Pain is persistent and unrelieved, localized along the esophagus, and increased by swallowing and neck flexion	Diaphoresis, dyspnea (late), shock	Older individual with known gastrointestinal problems. History of violent emesis, foreign body, caustic ingestion, blunt trauma, alcoholism, esophageal disease	Rare	Signs of lung consolidation, subcutaneous emphysema may be present	Chest film usually has mediastinal air, a left-sided pleural effusion, pneumothorax, or a widened mediastinum. pH of pleural effusion is <6.0. Diagnosis supported by water-soluble contrast esophagram or esophagoscopy	Patient may present in shock state. This entity often considered late in differential diagnostic process
Pericarditis	Dull, aching recurrent pain unrelated to exercises or meals. Or it may be a sharp, stabbing, pleuritic-type pain that does not change with chest wall motion. May be severe. Not relieved by NTG	Dyspnea, diaphoresis	Pain is often worse when supine, but improves sitting up. Often preceded by viral illness or underlying disease (SLE or uremia)	Rare Tamponade even more rare complication	Friction rub may be heard, often fleeting, position-dependent (50% of patients).	ECG pattern typical for ST segment elevation across the precordial leads. Erythrocyte sedimentation rate may be elevated	More common in 20- to 50-year-olds. May have associated tachycardias, ventricular dysrhythmias. Idiopathic most common (80%). Treated with aspirin, NSAID

AMI, acute myocardial infarction; CK-MB, an isoform of creatine kinase; COPD, chronic obstructive pulmonary disease; CT, computed tomography; DVT, deep vein thrombosis; ECG, electrocardiogram; MRI, magnetic resonance imaging; NSAID, nonsteroidal anti-inflammatory drug; NTG, nitroglycerin; PE, pulmonary embolus; SLE, systemic lupus erythematosus.

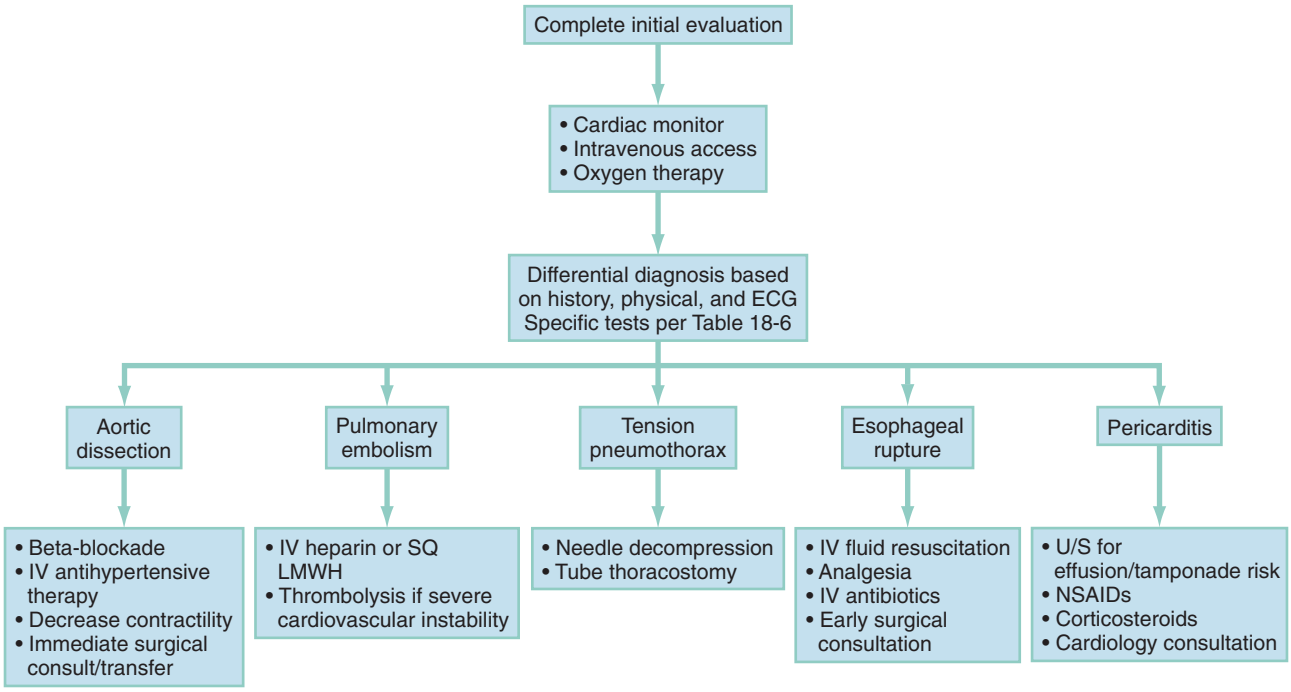

Figure 18-3. Clinical guidelines for emergency department management of chest pain from potentially catastrophic nonmyocardial origins. ECG, electrocardiogram; IV, intravenous; NSAIDs, nonsteroidal anti-inflammatory drugs; SQ, subcutaneous; LMWH, low-molecular-weight heparin; U/S, ultrasound.

Laboratory testing is useful in the evaluation of ACS. Creatine kinase (CK) is associated with multiple false-positive results and has no use in the evaluation of unstable angina. CK-MB, an isoform of CK, is more specific for cardiac ischemia. Evaluating this enzyme produces fewer false-positive results, and peak sensitivity approaches 98%. Sensitivity at 4 hours is, however, only about 60%. CK-MB isoforms improve sensitivity at 4 hours to 80%, approaching 93% at 6 hours. The current universal definition of MI places CK and CK-MB in a secondary role to troponins.[23]

Troponins (I and T), when elevated, identify patients with ACS who have the highest risk for an adverse outcome.[23,24] Sensitivity for acute MI at 4 hours is 60%, rising to nearly 100% by 12 hours.[25,26] Elevated troponin in the correct clinical setting is synonymous with acute MI and is embedded in the universal definition of MI.

DIAGNOSTIC TABLE

After the patient is stabilized and assessment has been completed, the findings are matched to the classic and atypical patterns of the seven potentially critical diseases causing chest pain. This matching process is continual while evaluating the patient and monitoring the response to therapy. Any inconsistency in findings with the primary working diagnoses requires a rapid review of the pivotal findings and the potential diagnoses (Table 18-6).

MANAGEMENT AND DISPOSITION

The management of ACS is discussed in Chapter 76. Figure 18-3 outlines the approach to treatment of critical noncardiac diagnoses. Patients with critical diagnoses generally are admitted to the intensive care unit. Patients with emergent diagnoses typically are admitted to the hospital, most often on telemetry units. Patients with nonemergent diagnoses are most frequently treated as outpatients. Hospitalization is required in certain circumstances, particularly when patients have other comorbid conditions.

Frequently, no definitive diagnosis is established. Any patient with almost any type of chest pain may be having coronary ischemia, PE, or aortic dissection. When a clear pattern does not emerge to allow the emergency physician to make an alternative diagnosis confidently, continued evaluation, hospitalization, or observation admission may be the best course.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 13 Confusion

J. Stephen Huff

PERSPECTIVE

The term *confusion* connotes an alteration in higher cerebral functions, such as memory, attention, or awareness. Confusion is a symptom, not a diagnosis. Clinical jargon includes "altered mental status," "delta MS" (change in mental status), "altered mentation," and "change from baseline." Additionally, the ability to sustain and focus attention is impaired. Symptoms of confusion may fluctuate, as may the level of consciousness. Implicit in the definition is a recent change in behavior. Chronic mental status changes such as dementia typically have a different clinical chronology. Other forms of altered mentation include states of diminished alertness on the coma spectrum; these presentations may result from some of the same pathophysiologic processes causing confusion and are discussed in Chapter 15. Confusion may range in severity from a mild disturbance of short-term memory to a global inability to relate to the environment and process sensory input. This extreme state is termed *delirium*. Delirium has two subtypes: hyperactive and hypoactive.[1] Hyperactive delirium is characterized as an acute confusional state associated with increased alertness, increased psychomotor activity, and disorientation and is often accompanied by hallucinations. In hypoactive delirium (sometimes referred to as quiet delirium), the confusional state is present but the patient has a reduction in alertness and behavior. Confusion has many causes, and an orderly approach is necessary to discover the causative diagnosis.

Epidemiology

Physicians underestimate the incidence of confusion in patients.[2,3] Often, confusion is accepted as an incidental or secondary component of another condition. A patient with injuries from a motor vehicle crash or with dyspnea may be confused, but the primary condition overshadows the underlying abnormal mental status. When confusion exists as an isolated or unexplained finding, it is more likely to receive full and immediate consideration by the clinician. Confusion is estimated to occur in 2% of emergency department (ED) patients, 10% of all hospitalized patients, and 50% of elderly hospitalized patients.[2,4]

Pathophysiology

Conceptually, consciousness may be divided into elements of alertness or arousal and elements constituting content of consciousness. Confusion is largely a problem of the content portion of consciousness. Many different clinical processes may disrupt optimal cortical functioning and result in confusion. The pathophysiology is not straightforward. Widespread cortical dysfunction is thought to result from substrate deficit (hypoglycemia or hypoxemia), neurotransmitter dysfunction, or circulatory dysfunction. Compounding this problem is the idea that the reserve of central nervous system (CNS) function varies from individual to individual; individuals with a preexisting impairment may become confused after even minor changes in their normal state.

DIAGNOSTIC APPROACH

Differential Considerations

The observation of acute confusion prompts a search for an underlying cause. Four groups of disorders encompass most causes of diffuse cortical dysfunction: (1) systemic diseases secondarily affecting the CNS, (2) primary intracranial disease, (3) exogenous toxins, and (4) drug withdrawal states (Box 13-1).[1] Focal cortical dysfunction, such as from tumor or stroke, typically does not cause confusion, although exceptions are encountered. Likewise, subcortical or brainstem dysfunction most frequently results in a diminished level of alertness and consciousness, not confusion.

Rapid Assessment and Stabilization

Most patients with acute confusion do not require immediate interventions. Three crucial exceptions are hypoglycemia, hypoxemia, and shock. A complete set of vital signs, including temperature and oxyhemoglobin saturation, and a bedside blood glucose level should be determined promptly for all confused patients. Oral or intravenous glucose therapy is indicated if low blood glucose is discovered. Supplemental oxygen and intravenous fluid are administered as necessary.

Patients should be protected from harming themselves or others. Close observation may need to be supplemented by medications or physical restraint. Family members may offer valuable assistance in observing and comforting the patient.

In a patient with abnormal or unstable vital signs, initial diagnostic and management efforts are directed toward treatment of the systemic condition. A confused patient with acute pulmonary edema, hypoxia, and confusion obviously requires evaluation and treatment of the pulmonary edema, not a screening test for cognitive functioning.

BOX 13-1 MAJOR CATEGORIES: DIFFERENTIAL CONSIDERATIONS

Primary intracranial disease
Systemic diseases secondarily affecting the central nervous system
Exogenous toxins
Drug withdrawal

BOX 13-2 FINDINGS THAT MAY HELP DIFFERENTIATE BETWEEN ORGANIC AND FUNCTIONAL CAUSES OF CONFUSION

Organic	Functional (psychiatric)
History	History
Acute onset	Onset over weeks to months
Any age	Onset ages 12 to 40 years
Mental status examination	Mental status examination
Fluctuating level of consciousness	Alert
Disoriented	Oriented
Attention disturbances	Agitated, anxious
Poor recent memory	Poor immediate memory
Hallucinations: visual, tactile, auditory	Hallucinations: most commonly auditory
Cognitive changes	Delusions, illusions
Physical examination	Physical examination
Abnormal vital signs	Normal vital signs
Nystagmus	No nystagmus
Focal neurologic signs	Purposeful movement
Signs of trauma	No signs of trauma

Generally, in patients with schizophrenia and other psychiatric disorders, tests of cognition, orientation, and attention are normal unless the condition is severe. The term *psychosis* implies a disorder of reality testing and thought organization severe enough to interfere with normal daily functioning. Psychosis is a nonspecific syndrome, and careful evaluation is required to differentiate between psychiatric and organic origins (e.g., drug intoxication or other systemic process) (Box 13-2).

Pivotal Findings

A patient with an altered state of consciousness including confusion is evaluated by taking a focused history and conducting a pertinent examination, performing rapid bedside screening investigations, and observing the response to certain therapies (e.g., dextrose or naloxone). Additional evaluation may include laboratory testing and diagnostic imaging with various modalities. Useful information that provides the diagnosis or strongly suggests the etiology is found roughly in descending order from the patient's history, the examination including results of rapid bedside testing, and the response to ED therapies; the results of laboratory testing and diagnostic imaging are less often useful.[5]

History

Confusion is often reported by family members or caregivers; frequently the patient is not aware of the confusion and seemingly glosses over problems. Families may articulate the complaint as confusion but also may describe rambling, disorientation, speaking to persons not there, the patient's inability to find his or her way around familiar surroundings, or simply "not being right." An essential goal of the history is to determine when the patient last exhibited "normal" thinking and behavior.

Attention deficit is the common denominator in confusional states. The initial task in evaluating the patient is to define the symptoms and severity of confusion. The specific behaviors that are of concern to the patient or caregivers should be defined. Often, the family is the most valuable source for information; a physician or other caregiver with an established relationship with the patient also may be helpful. The duration of the confusion, any recent changes in medications, and recent illnesses are important points in the clinical history. Hallucinations are not unique to psychiatric illness and can commonly occur in confusion states, especially delirium. Hallucinations in delirium tend to be visual (with or without auditory components), powerful, fleeting, and poorly organized. A history of medication or substance abuse and any recent changes, especially cessation of benzodiazepines or ethanol, should be sought.

ORIENTATION TO TIME
"What is the date?"

REGISTRATION
"Listen carefully. I am going to say three words.
You say them back after I stop.
Ready? Here they are . . .
HOUSE (pause), CAR (pause), LAKE (pause).
Now repeat those words back to me."
[Repeat up to 5 times, but score only the first trial.]

NAMING
"What is this?" [Point to a pencil or pen.]

READING
"Please read this and do what it says."
[Show examinee the words on the stimulus form.]
CLOSE YOUR EYES

Figure 13-1. Mini-mental state examination sample items. (Reproduced by special permission of the Publisher, Psychological Assessment Resources, Inc., 16204 North Florida Avenue, Lutz, Florida 33549, from the Mini Mental State Examination, by Marshal Folstein and Susan Folstein. Copyright © 1975, 1998, 2001 by Mini Mental LLC, Inc. Published 2001 by Psychological Assessment Resources, Inc. Further reproduction is prohibited without permission of PAR, Inc. The MMSE can be purchased from PAR, Inc., by calling (800) 331-8378 or (813) 968-3003.)

Physical Examination

The patient's confusion may be obvious at the bedside. In other cases, confusion may be subtle, and informal assessment of mental status and cognitive abilities may fail to detect it. The mini-mental state examination (MMSE) (Fig. 13-1) commonly is recommended as a screening instrument but is used infrequently in the ED because of the time required to administer it.[6,7] A more rapidly performed screening tool, the Quick Confusion Scale (QCS; Fig. 13-2), has been developed and tested in ED patients.[8-10] This tool objectively measures elements of the patient's mental status in 2 to 3 minutes and correlates well with the MMSE.[9,11] The tasks measured by either the MMSE or the QCS require adequate attention on

ITEM	SCORE (highest number in category indicates correct response; decreased scoring indicates increased number of errors)	WEIGHT	SCORE
What year is it now?	0 or 1 (score 1 if correct; 0 if incorrect)	x2	
What month is it?	0 or 1 (score 1 if correct; 0 if incorrect)	x2	
Repeat phrase and remember it: "John Brown, 42 Market Street, New York"			
About what time is it? (answer correct if within the hour)	0 or 1 (score 1 if correct; 0 if incorrect)	x2	
Count backwards from 20 to 1	0, 1, or 2 (score 2 if correct; 1 if 1 error; score 0 if more than 2 errors)	x1	
Say the months in reverse	0, 1, or 2 (score 2 if correct; 1 if 1 error; score 0 if more than 2 errors)	x1	
Repeat the memory phrase (each underlined portion is worth 1 point)	0, 1, 2, 3, 4, 5 (score 5 if correctly performed; each error drops score by one)	x1	
		TOTAL	_____

Final score is sum of the totals; score less than 15 suggests the presence of altered cognition and need for further assessment.

Figure 13-2. Quick Confusion Scale.

the part of the patient. If the patient's attention span is greatly impaired, detailed testing may be impossible. Digit repetition forward (five or six digits) and backward (four digits) is a brief screen for attention function. Alternatively, spelling a commonly used word backward ("world" is frequently used) measures a patient's ability to concentrate. Screening tests may detect confusion not obvious in casual conversation, identifying the need for further investigations.[12,13]

The physical examination may suggest a cause for confusion such as congestive heart failure or pneumonia. A fever suggests an infection as the cause of altered mental status and should prompt a search for the source, particularly urinary tract infection in the elder patient. Any new focal neurologic findings suggest a possible mass lesion or stroke and should trigger neuroimaging. In this regard, testing of gait and tandem gait, if possible, may be invaluable. Aphasia, fluent or nonfluent, is a focal sign suggesting a lesion in the dominant cerebral hemisphere. In confusional states, speech may be abnormal and is often incoherent, and the rate of speech may be either rapid or slowed. Involuntary movements, such as asterixis or tremor, may be present. The various toxidromes may assist in the identification of an intoxication or drug effect as the cause of confusion.

Laboratory Tests

The results of the history and physical examination frequently guide the clinician in the choice of laboratory tests most likely to yield valuable diagnostic information. Pulse oximetry may reveal hypoxia, or bedside glucose testing may reveal hypoglycemia or hyperglycemia. In the presence of a fever, chest radiography and urinalysis often reveal the source of the infection causing the altered mentation. In elder patients, urinalysis should be performed whether or not fever is present. Other tests commonly available in the ED and useful in the evaluation of a confused patient are serum electrolyte testing (especially sodium) and electrocardiography. Electrocardiography is indicated in elderly patients because myocardial infarction may present as confusion. The complete blood count, although commonly performed, is unlikely to provide useful diagnostic clues. Arterial blood gas testing is rarely indicated or useful, unless pulse oximetry is not reliable.

If common and simple tests do not suggest a solution, more complex testing should be initiated in the ED, observation unit, or inpatient service. The clinical situation and overall condition of the patient determine the speed and direction of evaluation. Additional laboratory work is often of decreasing yield but may reveal the cause of confusion. Serum ammonia, calcium, thyroid function, and selected drug and toxicologic testing may be ordered in this second tier of evaluation. Blood and urine cultures should be obtained in the febrile patient when hospital admission is anticipated and a clear infectious source is not evident. Paracentesis or thoracentesis may be appropriate if ascites or pleural effusion is present. Cranial computed tomography (CT) scanning is usually done to screen for CNS lesions in the absence of another identified source for the confusion. Focal findings on CT increase the yield of this test, but unanticipated abnormalities are often found on neuroimaging. Lumbar puncture may discover or exclude CNS infection if no other source has been identified. Cerebrospinal fluid examination may clarify a diagnosis of bacterial meningitis, encephalitis, aseptic meningitis, or subarachnoid hemorrhage. If the cause of confusion remains unclear or if the patient is unable to function safely in his or her current environment, admission may be necessary for additional ongoing assessment, including diagnostic testing not usually available in the ED, such as magnetic resonance imaging or electroencephalography.[5]

BOX 13-3 CRITICAL AND EMERGENT DIAGNOSES

Critical

Hypoxia/diffuse cerebral ischemia
- Respiratory failure
- Congestive heart failure
- Myocardial infarction
- Shock

Systemic processes
- Hypoglycemia

CNS infections

Hypertensive encephalopathy

Elevated intracranial pressure—medical and surgical origin

Emergent

Hypoxia/diffuse cerebral ischemia
- Severe anemia

Systemic diseases
- Electrolyte and fluid disturbance
- Endocrine disease
  - Thyroid
  - Adrenal
- Hepatic failure
- Nutrition/Wernicke's encephalopathy
- Sepsis, infection

Intoxications and withdrawal
- CNS sedatives
- Ethanol
- Other medication side effects, particularly anticholinergics

CNS disease
- Trauma
- Infections
- Stroke
- Subarachnoid hemorrhage
- Epilepsy/seizures
  - Postictal state
  - Nonconvulsive status epilepticus
  - Complex partial status epilepticus

Neoplasm

Note: These represent a partial diagnosis; causes are myriad. "Critical" in this case means conditions that need immediate assessment and correction within moments, such as oxygenation and ventilation problems or hypoglycemia. Because confusion represents CNS failure, other problems may be considered critical as well and may require intensive care unit admission, depending on severity.
CNS, central nervous system.

DIFFERENTIAL DIAGNOSIS

Certain critical and emergent diagnoses require prompt recognition to prevent morbidity or mortality (Box 13-3). The diagnosis of confusion implies the exclusion of other states of altered mental status, such as coma and decompensated psychiatric syndromes. A new focal neurologic deficit points to a focal defect of the CNS, which is less likely to cause the global cortical dysfunction necessary for confusion. Stroke rarely causes confusion, but resulting disturbances in speech or understanding may mimic a confusional state. The diagnosis of stroke is relatively straightforward if a new motor deficit is present. Occasionally, other focal neurologic abnormalities may mimic a confusional state. A person with a new visual field deficit and visual neglect may have difficulty ambulating in familiar surroundings and be labeled as confused, but this

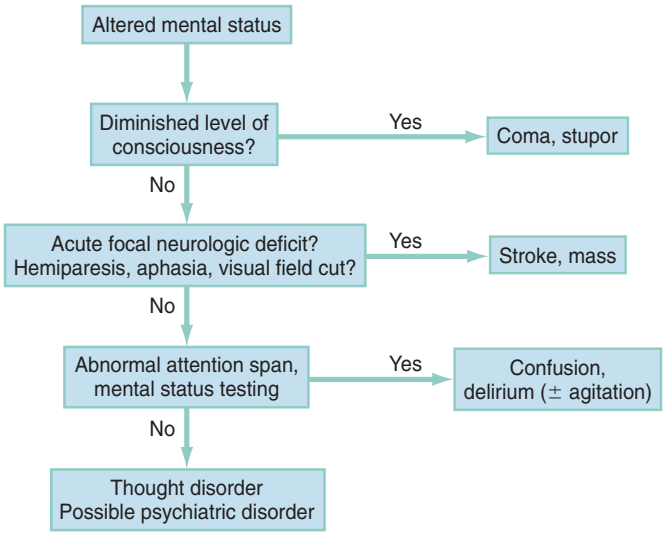

Figure 13-3. Diagnostic algorithm for confusion.

reflects focal neurologic injury and not a confusional state from global CNS dysfunction. Careful assessment of mental status assists in resolving the diagnostic dilemma. Frontal lobe dysfunction from stroke, subdural hematoma, or tumor may result in personality changes and the report of "confusion" by family or friends.

Altered mental status may be divided into three different categories depending on the findings of diminished level of consciousness, acute focal neurologic deficit, or abnormal attention span. Placement into one of these categories may guide the differential assessment and therapy (Fig. 13-3).

EMPIRICAL MANAGEMENT

Ideally, treatment is directed at the underlying cause of the confusion. Investigations continue until a likely diagnosis is discovered or consultation and admission are deemed necessary (Fig. 13-4). Many febrile patients are found to have a systemic infectious cause of the confusion. Urinary tract infections and pneumonia are the more common sources, but soft tissue infections also warrant consideration. CNS infections are encountered less frequently but have potentially devastating consequences if not recognized promptly. Antibiotic treatment for coverage of common causes of meningitis may be considered in ill febrile patients while definitive evaluation is in progress.

Postictal confusion is common in patients with seizures but should improve within 20 to 30 minutes. If the patient remains unconscious or confused after a seizure, the possibility of ongoing or intermittent seizure activity (i.e., nonconvulsive seizures) should be considered. Nonconvulsive status epilepticus, an epileptic twilight state, is unusual but does occur, and may be particularly difficult to recognize in the elderly[14] (see also Chapter 15).

Sometimes it may be necessary to treat confusion or agitation for patient safety. Environmental manipulations, such as dim lighting or psychosocial support, may be helpful. Confinement or physical restraint may be necessary at times for patient safety; institutional guidelines should be followed. Benzodiazepines or butyrophenones may be used if necessary to decrease agitation. These medications may alter mental status further, making evaluation more difficult.

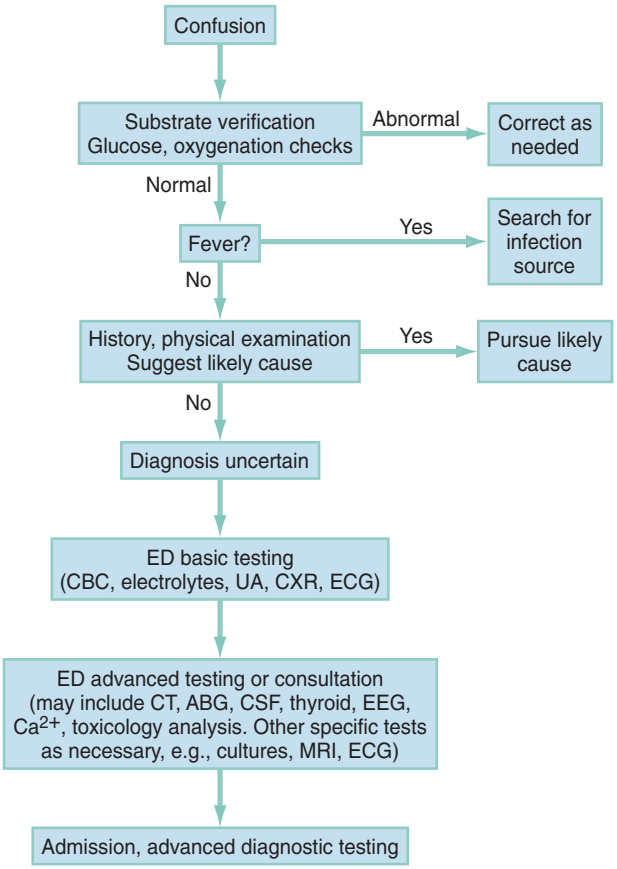

Figure 13-4. Management algorithm for confusion. ABG, arterial blood gas; CBC, complete blood count; CT, computed tomography; CSF, cerebrospinal fluid; CXR, chest x-ray; ECG, electrocardiogram; MRI, magnetic resonance imaging; UA, urinalysis.

DISPOSITION

Most patients presenting with confusion are admitted to the hospital or ED observation unit for additional diagnostic procedures, extended observation, and treatment. Exceptions include patients with rapidly resolved confusional states after treatment for insulin-induced hypoglycemia, after generalized seizures of known origin, or after recovering from self-limiting intoxicants or withdrawal states, such as those related to ethanol or recreational drugs. These patients may be observed and then discharged after successful identification and resolution of acute confusional state. Unresolved confusion or unexplained findings on repeat mental status screen should prompt admission or careful reevaluation before considering discharge.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 17 Dyspnea

Sabina Braithwaite and Debra Perina

PERSPECTIVE

Dyspnea is the term applied to the sensation of breathlessness and the patient's reaction to that sensation. It is an uncomfortable awareness of breathing difficulties that in the extreme manifests as "air hunger." Dyspnea is often ill defined by patients, who may describe the feeling as shortness of breath, chest tightness, or difficulty breathing. Dyspnea results from a variety of conditions, ranging from nonurgent to life-threatening. Neither the clinical severity nor the patient's perception correlates well with the seriousness of underlying pathology and may be affected by emotions, behavioral and cultural influences, and external stimuli.[1,2]

The following terms may be used in the assessment of the dyspneic patient:

Tachypnea: A respiratory rate greater than normal. Normal rates range from 44 cycles/min in a newborn to 14 to 18 cycles/min in adults.

Hyperpnea: Greater than normal minute ventilation to meet metabolic requirements.

Hyperventilation: A minute ventilation (determined by respiratory rate and tidal volume) that exceeds metabolic demand. Arterial blood gases (ABG) characteristically show a normal partial pressure of oxygen (Po_2) with an uncompensated respiratory alkalosis (low partial pressure of carbon dioxide [Pco_2] and elevated pH).

Dyspnea on exertion: Dyspnea provoked by physical effort or exertion. It often is quantified in simple terms, such as the number of stairs or number of blocks a patient can manage before the onset of dyspnea.

Orthopnea: Dyspnea in a recumbent position. It usually is measured in number of pillows the patient must use to lie in bed (e.g., two-pillow orthopnea).

Paroxysmal nocturnal dyspnea: Sudden onset of dyspnea occurring while reclining at night, usually related to the presence of congestive heart failure.

Epidemiology

Dyspnea is a common presenting complaint among emergency department patients of all ages. Causes vary widely and may be due to a benign, self-limited condition or significant pathology that can produce long-term morbidity and premature mortality.

Pathophysiology

The actual mechanisms responsible for dyspnea are unknown. Normal breathing is controlled both centrally by the respiratory control center in the medulla oblongata, as well as peripherally by chemoreceptors located near the carotid bodies, and mechanoreceptors in the diaphragm and skeletal muscles.[3] Any imbalance between these sites is perceived as dyspnea. This imbalance generally results from ventilatory demand being greater than capacity.[4]

The perception and sensation of dyspnea are believed to occur by one or more of the following mechanisms: increased work of breathing, such as the increased lung resistance or decreased compliance that occurs with asthma or chronic obstructive pulmonary disease (COPD), or increased respiratory drive, such as results from severe hypoxemia, acidosis, or centrally acting stimuli (toxins, central nervous system events). Pulmonary stretch receptors also are thought to play a role.

DIAGNOSTIC APPROACH

Differential Considerations

Dyspnea is subjective and has many different potential causes.[5] The differential diagnosis list can be divided into acute and chronic causes, of which many are pulmonary. Other etiologies include cardiac, metabolic, infectious, neuromuscular, traumatic, and hematologic (Table 17-1).

Pivotal Findings

History

Duration of Dyspnea. Chronic or progressive dyspnea usually denotes primary cardiac or pulmonary disease.[6] Acute dyspneic spells may result from asthma exacerbation; infection; pulmonary embolus; intermittent cardiac dysfunction; psychogenic causes; or inhalation of irritants, allergens, or foreign bodies.

Onset of Dyspnea. Sudden onset of dyspnea should lead to consideration of pulmonary embolism (PE) or spontaneous pneumothorax. Dyspnea that builds slowly over hours or days may represent a flare of asthma or COPD; pneumonia; recurrent, small pulmonary emboli; congestive heart failure; or malignancy.

Table 17-1 Differential Diagnoses for Acute Dyspnea

ORGAN SYSTEM	CRITICAL DIAGNOSES	EMERGENT DIAGNOSES	NONEMERGENT DIAGNOSES
Pulmonary	Airway obstruction Pulmonary embolus Noncardiogenic edema Anaphylaxis Ventilatory failure	Spontaneous pneumothorax Asthma Cor pulmonale Aspiration Pneumonia	Pleural effusion Neoplasm Pneumonia (CAP score < = 70) COPD
Cardiac	Pulmonary edema Myocardial infarction Cardiac tamponade	Pericarditis	Congenital heart disease Valvular heart disease Cardiomyopathy
Primarily Associated with Normal or Increased Respiratory Effort			
Abdominal		Mechanical interference Hypotension, sepsis from ruptured viscus, bowel obstruction, inflammatory/infectious process	Pregnancy Ascites Obesity
Psychogenic			Hyperventilation syndrome Somatization disorder Panic attack
Metabolic/endocrine	Toxic ingestion DKA	Renal failure Electrolyte abnormalities Metabolic acidosis	Fever Thyroid disease
Infectious	Epiglottitis	Pneumonia (CAP score < = 70)	Pneumonia (CAP score < = 70)
Traumatic	Tension pneumothorax Cardiac tamponade Flail chest	Simple pneumothorax, hemothorax Diaphragmatic rupture	Rib fractures
Hematologic	Carbon monoxide poisoning Acute chest syndrome	Anemia	
Primarily Associated with Decreased Respiratory Effort			
Neuromuscular	CVA, intracranial insult Organophosphate poisoning	Multiple sclerosis Guillain-Barré syndrome Tick paralysis	ALS Polymyositis Porphyria

ALS, amyotrophic lateral sclerosis; CAP, community-acquired pneumonia; COPD, chronic obstructive pulmonary disease; CVA, cerebrovascular accident; DKA, diabetic ketoacidosis.

Positional Changes. Orthopnea can result from left-sided heart failure, COPD, or neuromuscular disorders. One of the earliest symptoms seen in patients with diaphragmatic weakness from neuromuscular disease is orthopnea.[7] Paroxysmal nocturnal dyspnea is most common in patients with left-sided heart failure,[6] but also can be found in COPD. Exertional dyspnea commonly is associated with COPD, but also can be seen with poor cardiac reserve and abdominal loading. Abdominal loading, caused by ascites, obesity, or pregnancy, leads to elevation of the diaphragm, resulting in less effective ventilation and dyspnea.

Trauma. Dyspnea can result from trauma, causing fractured ribs, flail chest, hemothorax, pneumothorax, diaphragmatic rupture, pericardial effusion, cardiac tamponade, or neurologic injury.

Symptoms

Patient descriptions of dyspnea vary significantly and generally correlate poorly with severity. Fever suggests an infectious cause. Anxiety may point to panic attack or psychogenic dyspnea, if no organic cause can be isolated. PE or myocardial infarction may present with isolated dyspnea or with associated chest pain, particularly if the pain is constant, dull, or visceral.[8,9] If the pain is sharp and worsened by deep breathing but not by movement, pleural effusion and pleurisy or pleural irritation from pneumonia or PE are possible. Spontaneous pneumothorax also may produce sharp pain with deep breathing that is not worsened by movement.

Signs

Physical signs in dyspneic patients may be consistent with specific illnesses (Table 17-2). Physical findings found in specific diseases also can be grouped as presenting patterns (Table 17-3).

Ancillary Studies

Specific findings obtained from the history and physical examination should be used to determine which ancillary studies are needed (Table 17-4). Bedside oxygen saturation determinations, or selective use of ABGs when oximetry is not reliable, are useful in determining the degree of hypoxia and the need for supplemental oxygen or assisted ventilation. An additional resource for quickly assessing ventilatory status is noninvasive waveform capnography. Using both the end-tidal CO_2 value and the shape of the waveform itself can be helpful in assessing the adequacy of ventilations as well as potential causes of the dyspnea (See Chapter 3). An electrocardiogram may be useful if the etiology is cardiac or suggests acute pulmonary hypertension.

Serum electrolytes may suggest less common possible causes, such as hypokalemia, hypophosphatemia, diabetic ketoacidosis, or hypocalcemia. A complete blood count may identify severe anemia or thrombocytopenia associated with sepsis. The white blood cell count is not sufficiently sensitive or specific to be of discriminatory value. Cardiac markers and D-dimer assay may be useful in pursuing etiologies such as

Table 17-2 Pivotal Findings in Physical Examination

SIGN	PHYSICAL FINDING	DIAGNOSES TO CONSIDER
Vital signs	Tachypnea	Pneumonia, pneumothorax
	Hypopnea	Intracranial insult, drug/toxin ingestion
	Tachycardia	PE, traumatic chest injury
	Hypotension	Tension pneumothorax
	Fever	Pneumonia, PE
General appearance	Cachexia, weight loss	Malignancy, acquired immune disorder, mycobacterial infection
	Obesity	Hypoventilation, sleep apnea, PE
	Pregnancy	PE
	Barrel chest	COPD
	"Sniffing" position	Epiglottitis
	"Tripoding" position	COPD/asthma with severe distress
	Traumatic injury	Pneumothorax (simple, tension), rib fractures, flail chest, hemothorax, pulmonary contusion
Skin/nails	Tobacco stains/odor	COPD, malignancy, infection
	Clubbing	Chronic hypoxia, intracardiac shunts or pulmonary vascular anomalies
	Pallid skin/conjunctivae	Anemia
	Muscle wasting	Neuromuscular disease
	Bruising	Chest wall: rib fractures, pneumothorax
		Diffuse: thrombocytopenia, chronic steroid use, anticoagulation
	Subcutaneous emphysema	Rib fractures, pneumothorax, tracheobronchial disruption
	Hives, rash	Allergic reaction, infection, tick-borne illness
Neck	Stridor	Upper airway edema/infection, foreign body, traumatic injury, anaphylaxis
	JVD	Tension pneumothorax, COPD or asthma exacerbation, fluid overload/CHF, PE
Lung examination	Wheezes	CHF, anaphylaxis
		Bronchospasm
	Rales	CHF, pneumonia, PE
	Unilateral decrease	Pneumothorax, pleural effusion, consolidation, rib fractures/ contusion, pulmonary contusion
	Hemoptysis	Malignancy, infection, bleeding disorder, CHF
	Sputum production	Infection (viral, bacterial)
	Friction rub	Pleurisy
	Abnormal respiratory pattern (e.g., Cheyne-Stokes)	Intracranial insult
Chest examination	Crepitance or pain on palpation	Rib or sternal fractures
	Subcutaneous emphysema	Pneumothorax, tracheobronchial rupture
	Thoracoabdominal desynchrony	Diaphragmatic injury with herniation; cervical spinal cord trauma
	Flail segment	Flail chest, pulmonary contusion
Cardiac examination	Murmur	PE
	S_3 or S_4 gallop	PE
	S_2 accentuation	PE
	Muffled heart sounds	Cardiac tamponade
Extremities	Calf tenderness, Homans' sign	PE
	Edema	CHF
Neurologic examination	Focal deficits (motor, sensory, cognitive)	Stroke, intracranial hemorrhage causing central abnormal respiratory drive; if long-standing, risk of aspiration pneumonia
	Symmetrical deficits	Neuromuscular disease
	Diffuse weakness	Metabolic or electrolyte abnormality (hypocalcemia, hypomagnesemia, hypophosphatemia), anemia
	Hyporeflexia	Hypermagnesemia
	Ascending weakness	Guillain-Barré syndrome

CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; JVD, jugular venous distention; PE, pulmonary embolism.

cardiac ischemia or PE. Amino-terminal pro-B-type natriuretic peptide (NT-proBNP) analysis adds both diagnostic and prognostic value for several causes of dyspnea, including heart failure, PE, and ischemic cardiac disease.[9-11] Combinations of specific serum markers can also help define pathology.[12-14] Specialized tests, such as ventilation-perfusion scans, chest computed tomography, pulmonary angiography, or, rarely, conventional pulmonary angiography, may confirm the diagnosis of PE.[15] If dyspnea is believed to be upper airway in origin, direct or fiberoptic laryngoscopy or a soft tissue lateral radiograph of the neck may be useful.

DIFFERENTIAL DIAGNOSIS

The range and diversity of pathophysiologic states that produce dyspnea make a simple algorithmic approach difficult.[16] After initial stabilization and assessment, findings from the history, physical examination, and ancillary testing are collated to match patterns of disease that produce dyspnea. This process is updated periodically as new information becomes available. Table 17-3 presents recognizable patterns of disease for common dyspnea-producing conditions, along with specific associated symptoms.

Table 17-3 Diagnostic Table: Patterns of Diseases Often Resulting in Dyspnea

DISEASE	HISTORY: (DYSPNEA)	ASSOCIATED SYMPTOMS	SIGNS AND PHYSICAL FINDINGS	TESTS
Pulmonary embolism	HPI: abrupt onset, pleuritic pain, immobility (travel, recent surgery) PMH: malignancy, DVT, PE, hypercoagulability, oral contraception, obesity	Diaphoresis, exertional dyspnea	Tachycardia, tachypnea, low-grade fever	ABG (A-a gradient), D-dimer ECG (dysrhythmia, right heart strain) CXR (Westermark sign, Hampton's hump) $\dot{V}/\dot{Q}$, spiral CT, MRV Pulmonary angiogram Ultrasound positive for DVT
Pneumonia	Fever, productive cough, chest pain	Anorexia, chills, nausea, vomiting, exertional dyspnea, cough	Fever, tachycardia, tachypnea, rales or decreased breath sounds	CXR, CBC, sputum and blood cultures
Bacterial	SH: tobacco use			ABG if hypoxia suspected Waveform capnography if altered mental status
Viral	Exposure (e.g., influenza, varicella)			
Opportunistic	Immune disorder, chemotherapy			
Fungal/parasitic	Exposure (e.g., birds), indolent onset	Episodic fever, nonproductive cough		
Pneumothorax	Abrupt onset ± trauma, chest pain, thin males more likely to have spontaneous pneumothorax	Localized chest pain	Decreased breath sounds, subcutaneous emphysema, chest wall wounds or instability	CXR: pneumothorax, rib fractures, hemothorax
Simple				Ultrasound positive for pneumothorax
Tension	Decompensation of simple pneumothorax	Diaphoresis	Above JVD, tracheal deviation, muffled heart sounds, cardiovascular collapse	Clinical diagnosis: requires immediate decompression. May verify using bedside ultrasound
COPD/asthma	Tobacco use, medication noncompliance, URI symptoms, sudden weather change PMH: environmental allergies FH: asthma	Air hunger, diaphoresis	Retractions, accessory muscle use, tripoding, cyanosis	CXR: rule out infiltrate, pneumothorax, atelectasis (mucus plug) Waveform capnography
Malignancy	Weight loss, tobacco or other occupational exposure	Dysphagia	Hemoptysis	CXR, chest CT: mass, hilar adenopathy, focal atelectasis
Fluid overload	Gradual onset, dietary indiscretion or medication noncompliance, chest pain PMH: recent MI, diabetes, CHF	Worsening orthopnea, PND	JVD, peripheral edema, S_3 or S_4 gallop, new cardiac dysrhythmia, hepatojugular reflux	CXR: pleural effusion, interstitial edema, Kerley B lines, cardiomegaly ECG: ischemia, dysrhythmia NT-proBNP
Anaphylaxis	Abrupt onset, exposure to allergen	Dysphagia	Oral swelling, stridor, wheezing, hives	

ABG, arterial blood gas; CBC, complete blood count; CHF, congestive heart failure; CT, computed tomography; CXR, chest x-ray; DVT, deep vein thrombosis; ECG, electrocardiogram; FH, family history; HPI, history of present illness; JVD, jugular venous distention; MI, myocardial infarction; MRV, magnetic resonance venography; NT-proBNP, amino-terminal pro-brain natriuretic peptide; PE, pulmonary embolism; PMH, past medical history; PND, paroxysmal nocturnal dyspnea; SH, social history; URI, upper respiratory infection.

Table 17-4 Ancillary Testing in the Dyspneic Patient

CATEGORY	TEST	FINDINGS/POTENTIAL DIAGNOSES
Laboratory	Pulse oximetry, selective ABG use	Hypoxia, hyperventilation (muscular weakness, intracranial event)
	Waveform capnography	CO_2 retention (COPD, sleep apnea), obstructive or restrictive pulmonary pattern Metabolic versus respiratory acidosis (DKA, ingestions) A-a gradient (PE) Elevated carboxyhemoglobin (inhalation injury or CO poisoning)
	Complete blood count	WBC Increase: infection, stress demargination, hematologic malignancy Decrease: neutropenia, sepsis Hgb/Hct: anemia, polycythemia Smear: abnormal Hgb (i.e., sickling), inclusions Platelets: thrombocytopenia (marrow toxicity)
	Chemistry	BUN/Cr: acute/chronic renal failure K/Mg/Phos: low levels resulting in muscular weakness Glucose: DKA D-dimer: abnormal clotting activity NT-proBNP: heart failure, PE Troponin: cardiac ischemia or infarct
Cardiac	ECG	Ischemia, dysrhythmia, $S_1Q_3T_3$ (PE), right heart strain
	Echocardiogram	Pulmonary hypertension, valvular disorders Wall motion abnormalities related to ischemia, intracardiac shunts
Radiologic	Chest radiograph	Bony structures: fractures, lytic lesions, pectus, kyphoscoliosis Mass: malignancy, cavitary lesion, infiltrate, foreign body Diaphragm: eventration, elevation of hemidiaphragm, bowel herniation Mediastinum: adenopathy (infection, sarcoid), air Cardiac silhouette: enlarged (cardiomyopathy, fluid overload) Soft tissue: subcutaneous air Lung parenchyma: blebs, pneumothorax, effusions (blood, infectious), interstitial edema, local consolidation, air bronchograms, Hampton's hump, Westermark's sign
	$\dot{V}/\dot{Q}$ scan	PE
	Pulmonary angiogram	PE, intervention (thrombolysis)
	CT	Mass lesion, adenopathy, trauma, PE
	MRI	PE, bony and soft tissue lesions, vascular abnormality
	Soft tissue neck radiograph	Epiglottitis, foreign body
	Ultrasound	Pneumothorax, pleural effusion, impaired cardiac function or pericardial effusion
Fiberoptic	Bronchoscopy	Mass lesion, foreign body Intervention (stenting, biopsy)
	Laryngoscopy	Mass lesion, edema, epiglottitis, foreign body

A-a, alveolar-arterial; ABG, arterial blood gas; BUN, blood urea nitrogen; CHF, congestive heart failure; CO, carbon monoxide; COPD, chronic obstructive pulmonary disease; Cr, creatinine; CT, computed tomography; DKA,.diabetic ketoacidosis; ECG, electrocardiogram; MRI, magnetic resonance imaging; NT-proBNP, amino-terminal pro-brain natriuretic peptide; PE, pulmonary embolism; V/Q, ventilation-perfusion; WBC, white blood cell.

Critical Diagnoses

Several critical diagnoses should be promptly considered to determine the best treatment options to stabilize the patient. Tension pneumothorax is such a critical diagnosis. If a dyspneic patient has diminished breath sounds on one side, ipsilateral hyper-resonance, severe respiratory distress, hypotension, and oxygen desaturation, prompt decompression of presumptive tension pneumothorax is necessary. Bedside ultrasonography may assist in confirming pneumothorax. If obstruction of the upper airway is evidenced by dyspnea and stridor, early, definitive assessment and intervention must occur in the emergency department or operating room. Complete obstruction by a foreign body warrants the Heimlich maneuver until the obstruction is relieved or the patient is unconscious, followed rapidly by direct laryngoscopy. Congestive heart failure and pulmonary edema can produce dyspnea and respiratory failure and should be treated as soon as possible if severe.[17] Significant dyspnea and wheezing can be seen in anaphylaxis and must be treated promptly to prevent further deterioration. Severe bronchospastic exacerbations of asthma at any age may lead rapidly to respiratory failure and arrest and should receive vigorous attention, including continuous or frequent administration of a beta-agonist aerosol.[18] As mentioned earlier, waveform capnography is a valuable tool for assessing the severity and determining the cause of respiratory distress.

Emergent Diagnoses

Asthma and COPD exacerbations can result in marked dyspnea with bronchospasm and decreased ventilatory volumes.[19] Sudden onset of dyspnea with a decreased oxygen saturation on room air accompanied by sharp chest pain may represent PE.[15] Dyspnea accompanied by decreased breath sounds and tympany to percussion on one side is seen with spontaneous pneumothorax. Dyspnea associated with decreased respiratory effort may represent a neuromuscular process, such as multiple sclerosis, Guillain-Barré syndrome, or myasthenia gravis.[14] Unilateral rales, cough, fever, and dyspnea usually indicate pneumonia.

Figure 17-1 provides an algorithm for assessment and stabilization of a dyspneic patient. The initial division is based on the degree of breathing effort associated with the symptoms.

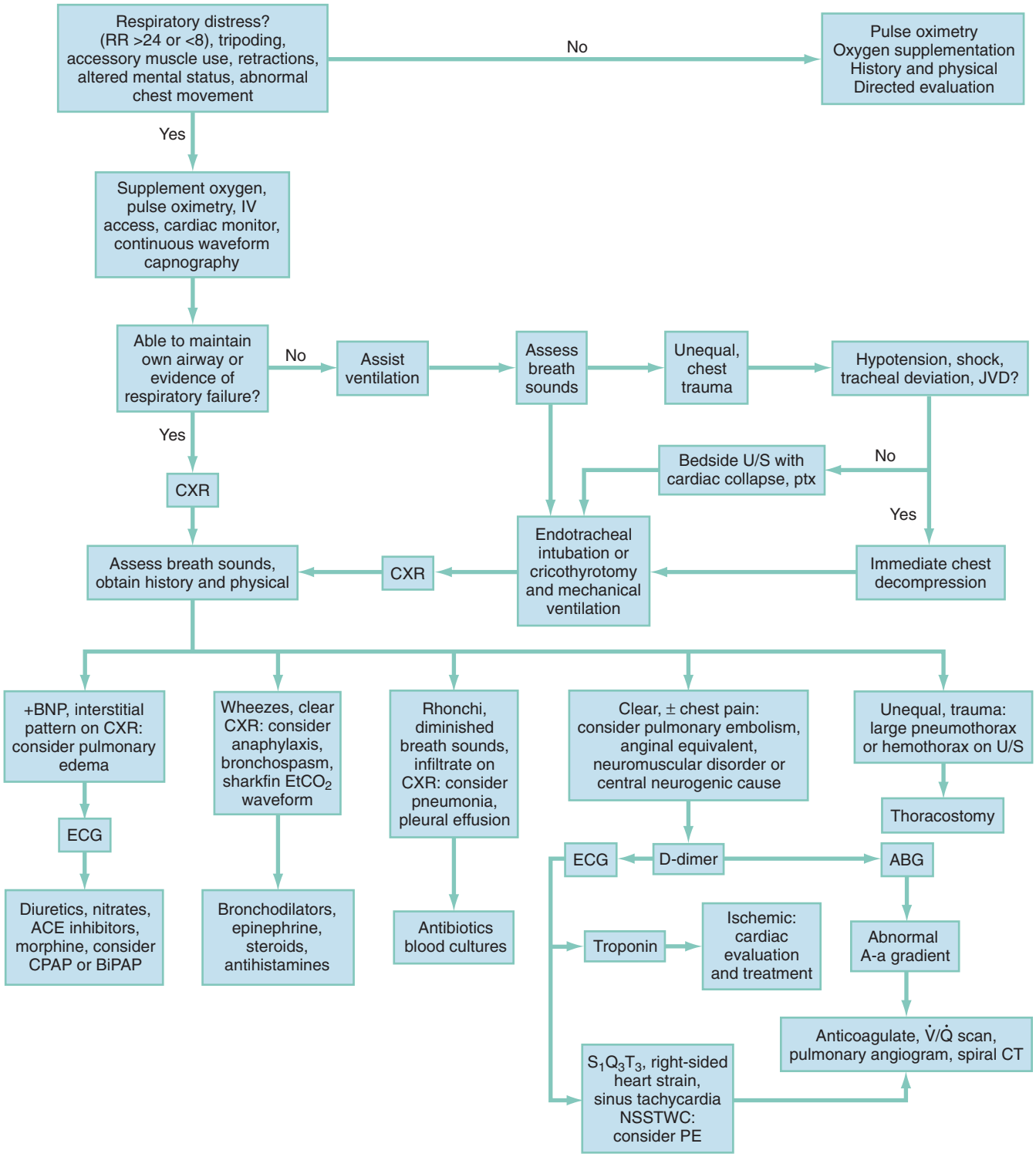

Figure 17-1. Rapid assessment and stabilization of a dyspneic patient. ABG, arterial blood gas; ACE, angiotensin-converting enzyme; BiPAP, biphasic positive airway pressure; BNP, B-type natriuretic peptide; CO, carbon monoxide; CPAP, continuous positive airway pressure; CT, computed tomography; CXR, chest x-ray; ECG, electrocardiogram; $EtCO_2$, end-tidal carbon dioxide; IV, intravenous; JVD, jugular venous distention; NSSTWC, nonspecific ST wave changes (on ECG); PE, pulmonary embolism; RR, respiratory rate; $\dot{V}/\dot{Q}$, ventilation-perfusion ratio; U/S, ultrasound.

The most critical diagnoses must be considered first and appropriate intervention taken as necessary.

All patients experiencing dyspnea, regardless of possible cause, should be promptly transported to the treatment area. Bedside pulse oximetry should be obtained, and the patient should be placed on a cardiac monitor. If the pulse oximetry is less than 98% saturated on room air, the patient should be placed on supplemental oxygen either by nasal cannula or mask depending on the degree of desaturation detected. If necessary, the patient should be intubated, and breathing should be assisted with manual or mechanical ventilation.

When the airway has been secured, rapid assessment of the patient's appearance and vital signs can help determine the need for further stabilization. Decreased mental alertness, inability to speak in more than one-word syllables, or certain types of body positioning, signal the presence of significant

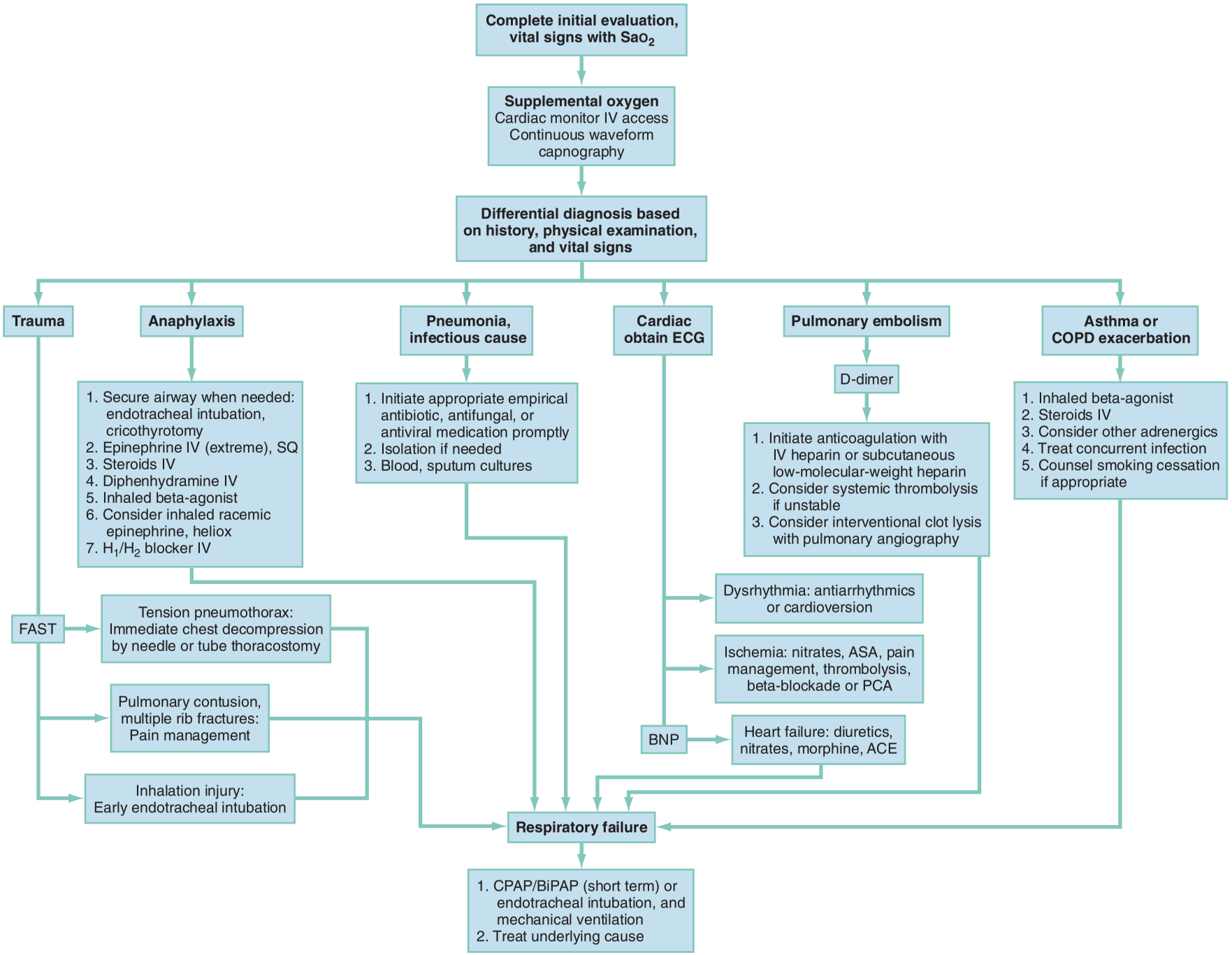

Figure 17-2. Clinical guidelines for emergency department management of dyspnea. ACE, angiotensin-converting enzyme; ASA, acetylsalicylic acid; COPD, chronic obstructive pulmonary disease; CPAP/BiPAP, continuous positive airway pressure/biphasic positive airway pressure; ECG, electrocardiogram; IV, intravenous; PCA, patient-controlled analgesia; SQ, subcutaneous.

respiratory distress and the need for rapid intervention. After stabilization has occurred, the cause of the dyspnea can be further investigated.

EMPIRICAL MANAGEMENT AND DISPOSITION

The management algorithm for dyspnea (Fig. 17-2) outlines the approach to treatment for most identifiable diseases. Unstable patients or patients with critical diagnoses must be stabilized and may require admission to an intensive care unit. Emergent patients who have improved in the emergency department may be admitted to an intermediate care unit. Patients diagnosed with urgent conditions in danger of deterioration without proper treatment or patients with severe comorbidities, such as diabetes, immunosuppression, or cancer, may also require admission for observation and treatment.

Most patients in the nonurgent category can be treated as outpatients if good medical follow-up can be arranged. If dyspnea persists despite therapy and no definitive cause has been delineated, the best course of action is hospitalization for observation and ongoing evaluation. If no definitive diagnosis can be obtained and the symptoms have abated, the patient may be discharged with good medical follow-up and instructions to return if symptoms recur.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

PART VI

Emergency Medical Services

CHAPTER 190

Emergency Medical Services: Overview and Ground Transport

Thomas H. Blackwell

PERSPECTIVE

Development of Emergency Medical Services

Before the advent of civilian ambulance services, the sick and injured were transported by any means available, including passerby motorists, wagons, farm machinery, delivery carts, buses, or taxicabs. Figure 190-1 depicts the early Larrey ambulance used during the Napoleonic Wars, the Rucker Wagon used during the American Civil War, and a modern ambulance used today. In 1865, the Commercial Hospital in Cincinnati established the first hospital-based ambulance service. Four years later, the first city service began at New York's Bellevue Hospital[1].

In 1965, the President's Commission on Highway Safety recommended a National Accident Response Program to decrease death and injury from highway accidents.[2] Results from a second national survey by the National Academy of Sciences—National Research Council were used to draft a white paper entitled "Accidental Death and Disability: The Neglected Disease of Modern Society."[3] Published in 1966, this document described the hazardous conditions of emergency care provision at all levels and outlined the necessary building blocks for future EMS maturation. These national efforts were the impetus for congressional legislation that directed the U.S. Department of Transportation (DOT)—National Highway Traffic Safety Administration (NHTSA) to develop a program for improving emergency medical care.

During the mid-1960s, out-of-hospital cardiac care included field defibrillation programs in Belfast, Northern Ireland, and cardiac arrest research in several U.S. cities.[4,5] In 1969, the first National Conference on EMS convened, resulting in the development of a curriculum, certification process, and national registry for the emergency medical technician-ambulance (EMT-A). By 1972, the U.S. Department of Labor recognized the EMT as an occupational specialty.[6] Interested physicians and nurses later provided advanced educational courses and practical experiences for the EMTs, and thus began the paramedic providers.[7,8]

Additional programs prompted Congressional passage of the EMS Systems Act of 1973 (P.L. 93-154), which authorized funding that dramatically improved the development of comprehensive regional EMS delivery systems. Efforts to improve pediatric emergency care occurred in 1984 when Congress adopted the Emergency Medical Services for Children (EMS-C) initiative through the Health Services, Preventive Health Services, and Home Community Based Services Act of 1984 (P.L. 98-555).[9] An Institute of Medicine (IOM) study, released in 1994, promoted the concept of EMS-C becoming integrated not just into existing EMS systems but into comprehensive systems of care provision, including injury prevention, primary and definitive care, and rehabilitation services.[10]

More than 40 years since publishing the 1966 white paper, the IOM released a report on the status of emergency care entitled "The Future of Emergency Care in United States Health System." The report focused on three separate, yet related, topics: (1) emergency care: at the breaking point, (2) emergency medical services at the crossroads, and (3) emergency care for children: growing pains.[11-13]

A major focus included the need to strengthen the integration of EMS into the entire healthcare system, because lack of such coordination often results in patients being diverted from overcrowded to inappropriate or distant facilities. The recommendation was to ensure that the delivery of emergency medical and trauma care is organized into a coordinated, regional system such that patients receive care at the most appropriate facility based on their injury or illness. Additional recommendations targeting EMS improvement included national accreditation for paramedic educational programs, adopting a national certification system for individual state licensure, and recognizing common levels of EMS certification across the United States.

The concern for inadequate funding for EMS systems operations and disaster response was also addressed. Recommendations included Congress developing regionally funded, multiyear demonstration projects that encourage states to identify and test strategies for creating seamless systems of care, workforce strengthening, evidence-based practices, and disaster preparedness. It was further recommended that an advisory committee be created to work with the Centers for Medicare and Medicaid Services to improve reimbursement and policies related to reimbursement.

Finally, a small, yet significant, proportion of EMS transports involve the pediatric population; thus, it is often difficult for prehospital providers to maintain the knowledge and skills necessary to care for critically ill or injured children. Many plans, including disaster preparedness, often neglect children. As such, the IOM report recommended several items, most importantly that the care of children be integrated into the overall EMS system and not separated from adults, with pediatric emergency care competencies being defined and training enhanced to maintain those competencies. Because it is diffi-

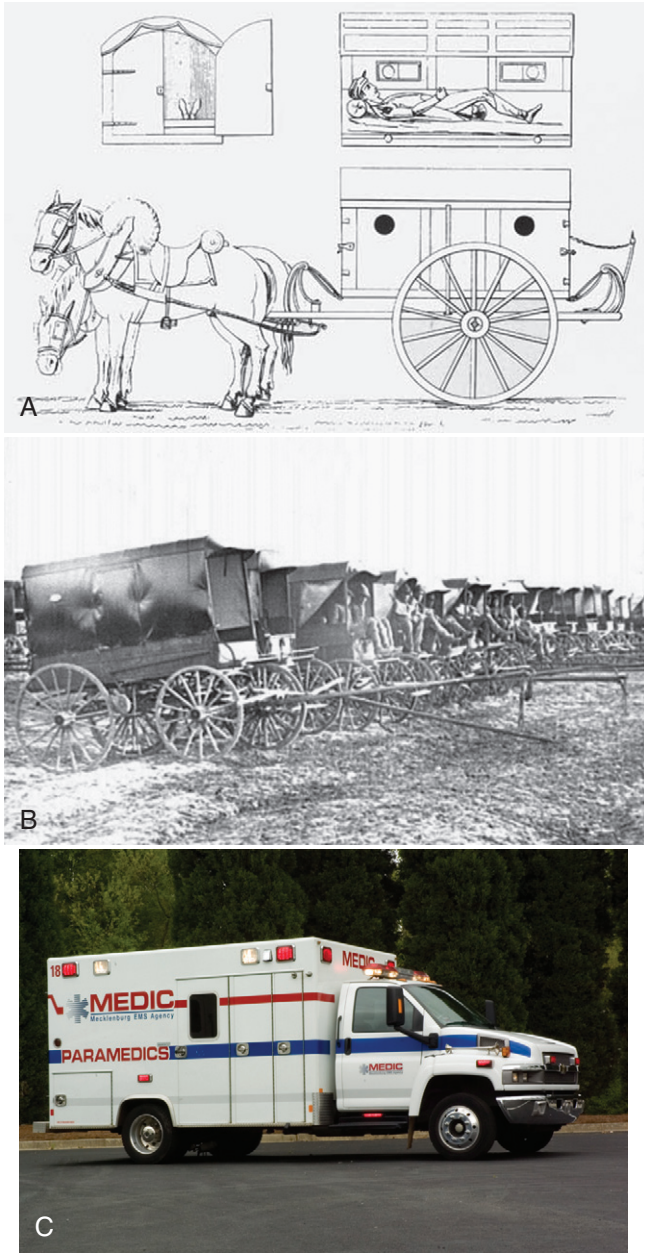

Figure 190-1. Larrey's Flying Ambulance (**A**), Letterman's Rucker Wagon (**B**), and the modern ambulance used today (**C**). (**A**, courtesy of the National Library of Medicine, History of Medicine Division; **B**, courtesy of the Library of Congress, Prints and Photographs Division, LC-88171-2585 DLC; and **C**, courtesy of Monroe Hicks, Mecklenburg EMS Agency, Charlotte, North Carolina.)

cult to ascertain whether systems have targeted the pediatric population, the IOM further recommended that a pediatric coordinator be included in all EMS systems to advocate for ensuring equipment, medications, training, and protocols are appropriate for children.

Each of the three IOM reports supported the concept of the federal government developing national standards for emergency care performance indicators and evaluation and protocols for triage, treatment, and transport of patients. To accomplish this objective, a lead federal agency should be identified. Debate exists whether EMS at a national level should remain under the NHTSA or reside in other applicable agencies (e.g., Health and Human Services, Homeland Security). Regardless, the parent organization must ensure that research is supported to improve the knowledge base and support evidence for the practice of out-of-hospital medical care.

Emergency Medical Service Systems

Multiple EMS system designs exist, all predicated on the type of community served. While this is a local decision, all states incorporate an administrative office that governs or oversees the provision of EMS activities. Typically the role is not to direct any individual service, but to assist in planning, licensing services, and establishing or enforcing the scope and standards for practice. Other functions may include training, examining, certifying and recertifying providers, record keeping, data collection, and auditing or investigating programs. A description of systems for the 200 most populous cities in the United States is periodically published in the *Journal of Emergency Medical Services*.[14] For simplicity, the following categorization of systems will be used: private and public agencies; basic life support (BLS) and advanced life support (ALS) services; and single-tiered, multitiered, and first responder systems.

Private and Public Agencies

Where local government has not assumed primary responsibility for EMS services, communities may depend on private providers. Financial responsibility varies but usually depends on federal reimbursement (Medicare or Medicaid) and user fees. A local government subsidy may or may not supplement the operation. If multiple providers are serving one jurisdiction, calls may be allocated by rotation or specified zone coverage. Dispatching varies depending on the system but may be by the provider or by a central agency. Medical direction is often provided by a contracted physician or physician oversight board.

Hospital-based EMS systems are few in number and may be managed by a single hospital or hospital corporation. Not all hospital-based EMS programs are considered private, in that the hospital may be a division under local or state government or operating under a public authority. Like private models, financial responsibility is usually in the form of user fees, with or without additional subsidy. Dispatching may be provided by a local public safety agency that may also be responsible for police and fire communications. An emergency physician from a sponsor or base hospital typically provides medical direction for these systems.

A public utility model is a hybrid between private and public design that allows local government to contract with a private or public provider. The successful bidder for service becomes a contracted entity that agrees to provide the specified services (ALS, BLS, or both) to the catchment area and, depending on the arrangement, may bill the patient directly or receive uniform reimbursement. Depending on local structure and interagency agreement, dispatching may be performed by an existing public safety organization or by the parent company. Medical direction is usually a specified individual subject to contractual terms.

When government officials were faced with planning and establishing EMS systems during the early maturation periods, many decided that the fire department was the logical choice to incorporate EMS. Fire stations were strategically located throughout the community, and personnel were already used to providing emergency response. Firefighters could be cross-trained as a firefighter-paramedic or dedicated to either fire or EMS function with the opportunity for transfer. Public EMS systems that were not incorporated into fire departments evolved into their own separate entity, referred to as a municipal third-service system. Such agencies are operated by local municipalities and are endorsed and supported by local government. Many cities have been successful in combining

police, fire, and EMS under a global public safety agency, with all department heads or chiefs reporting to one manager or administrator. Financially, public EMS systems may be supported by a tax base, which may or may not be supplemented by user fees. Regardless of design, medical oversight for a municipal EMS system may be provided by a physician appointed and contracted by a local hospital, an advisory council, or medical oversight board.

Basic Life Support and Advanced Life Support Service

BLS describes the provision of emergency care without the use of advanced therapeutic interventions. Skills include airway management (oral and nasal airways, bag-mask ventilation), cardiopulmonary resuscitation (CPR), hemorrhage control, fracture and spine immobilization, and childbirth assistance. Defibrillation using an automated external defibrillator (AED) is often included by many BLS systems. Services are provided by certified or medical first responders or emergency medical technicians (EMTs) certified at the basic level (EMT-B).

BLS systems may be associated with poor survival rates from out-of-hospital cardiac arrest, especially those not incorporating AED technology.[15,16] Alternatively, there is debate on the effectiveness of ALS for medical and traumatic emergencies.[17,18] Despite this evidence, few urban communities across the United States operate solely at the BLS level. Many rural and some suburban EMS services rely on volunteers who may not wish to become advanced-level providers. Because these services may have low call volumes, it becomes more difficult for personnel to maintain advanced skills and a proficient knowledge base.[19] Also, such communities may not have access to medical supervision or hospital sponsorship for ALS care.

Systems categorized as ALS offer a more comprehensive level of service by highly educated providers, usually certified at the intermediate or paramedic level (EMT-I or EMT-P, respectively), or equivalent levels depending on individual state certifications. Provider skills include advanced airway interventions, intravenous (IV) line placement, medication administration, cardiac monitoring and manual defibrillation, and certain invasive procedures. Most EMS systems in urban cities operate at the ALS level of care.

The number of EMT-Ps in any jurisdiction has come under scrutiny, in that cities with more paramedics per capita tend to have lower survival rates.[20] Although this may seem implausible, one explanation might be that the number of patient encounters per paramedic decreases and the sharpness of skills degrades when that community is saturated with paramedics.

Single-Tiered, Multitiered, and First-Responder Systems

In a single-tiered system, every response regardless of the call type receives the same level of personnel expertise and equipment allocation (all BLS or ALS). Multiple-tiered systems use a combination of ALS and BLS levels depending on the nature of the call. Differences in cost and effectiveness between a mixed ALS-BLS service and an all ALS service have been debated. Currently, there is a steady decrease in systems that provide mixed ALS-BLS care.[21,22] A single-tiered ALS response may prove to be cost-effective in specific locales, ensures the capability of providing a consistent advanced level of care to all patients regardless of illness or injury severity, and obviates the potential for undertriage or overtriage by 9-1-1 telecommunicators. Alternatively, a multitiered system may meet the needs of individual communities or agency infrastructure. This design often meets with employee satisfaction and has the potential to preserve ALS resources for higher priority calls, in that BLS transport of nonurgent patients allows for ALS ambulances to be available for potential critical responses.

Regardless of single- or multiple-tier design, EMS systems usually include first-responder (FR) services as part of their structure. The FR, usually a police officer or firefighter, is the nontransport BLS or ALS provider who quickly responds to the scene of an emergency to provide initial care before definitive medical care and transportation assets arrive. The FR quickly assesses the situation and patient(s), determines whether additional resources are required, initiates patient care, and provides advance information to responding personnel.

The design of an EMS system is targeted toward providing quality patient care in the briefest period of time following unexpected injury or illness. A desirable and cost-effective design might include BLS nontransport FRs with short response times (average 2–4 min), having the capability of providing early defibrillation and airway support, coupled with ensuing ALS care and transport services.[23]

Levels of Provider and Scope of Practice

At the federal level, NHTSA is responsible for developing the National Standard Curriculum for the different certification levels. Individual state legislation is responsible for provider levels recognized, initial and continuing medical education requirements at each level, testing, and time intervals for course completion and recertification. The following sections outline the DOT recommendations for the four common levels of provider with suggested hours of training and incorporated skills (Table 190-1).

First Responder

The FR is typically the first to arrive on the scene of an incident. Initial scene and patient assessment, along with limited life-saving interventions, are primary functions. Along with CPR and basic airway management skills, the FR should be able to control hemorrhage and initiate spinal immobilization.

The four elements referred to as the "chain of survival" by the American Heart Association (AHA), which decrease mortality from out-of-hospital cardiac arrest, are early access to care, CPR, defibrillation, and advanced airway management and medications.[24] Because early defibrillation may improve the odds of survival of out-of-hospital cardiac arrest, the use of an AED should be a mandatory procedure for the FR.[25,26]

The DOT recommends 40 hours of didactic instruction for the standard FR course and 16 to 36 hours for refresher training. A clinical rotation is not part of the curriculum.[27]

Emergency Medical Technician—Basic

The EMT-B is the minimum level required to staff a BLS ambulance and is commonly used for nonemergency and convalescent transport services. In addition to the skills of the FR, the EMT-B is also involved with triage, more detailed patient assessment, and transportation. Like FRs, EMT-Bs should have the capability of providing early defibrillation.[28]

In 1995, NHTSA released the revised EMT-B curriculum. The initial course requires approximately 110 hours of instruction and includes 46 lessons, each with cognitive, effective, and psychomotor objectives.[29] Many states have expanded the course to include more skills such as AED use, epinephrine autoinjections, albuterol administration by hand-held nebulizer or metered-dose inhaler, and IV fluid therapy. For recertification, the DOT recommends a 24-hour refresher course,

Table 190-1 EMS Provider Level Training and Skills

EMS PROVIDER LEVEL	DOT RECOMMENDED HOURS OF TRAINING	SKILL SET
First responder	Initial: 40 hr Refresher: 16–36 hr	Initial scene and patient assessment and stabilization Basic airway skills CPR Control hemorrhage Spinal immobilization
EMT—Basic	Initial: 110 hr Refresher: 24-hr refresher course, 48 hr of continuing education, and a BLS course every 2 years	First responder skills plus: Triage and detailed patient assessment AED May assist in some systems: Use of epinephrine autoinjectors for anaphylaxis; albuterol for wheezing
EMT—Intermediate	Initial: 300–400 hr and includes didactic and clinical experience	EMT—basic skills plus: Endotracheal intubation Manual defibrillation IV line placement Limited pharmacologic treatments May assist in some systems: Laryngeal mask airway
EMT—Paramedic	Initial: 1000–1200 hr Refresher: 48-hr refresher course, 24 hr of yearly continuing education, and BLS and ALS courses at the pediatric and adult levels	EMT—intermediate skills plus: Cardiac rhythm recognition Expanded pharmacologic treatments Needle decompression of a tension pneumothorax Needle or surgical cricothyrotomy Transthoracic cardiac pacing

AED, automated external defibrillator; ALS, advanced life support; BLS, basic life support; CPR, cardiopulmonary resuscitation; DOT, Department of Transportation; EMT, emergency medical technician; IV, intravenous.

48 hours of continuing education, and a BLS course every 2 years.

Emergency Medical Technician—Intermediate

The EMT-I was established to allow a more comprehensive approach to care when paramedic services were unavailable or unobtainable. Many states recognize the EMT-I certification, but others designate alternative, but comparable, levels depending on specific skills and procedures. The intermediate level is useful for rural systems because it supplies an ALS for less cost and educational time expended. The scope of practice for the EMT-I varies across the United States. Most systems allow the EMT-I to establish an IV line and to manually defibrillate. Limited administration of medications and use of adjunctive airway devices (e.g., blind insertion airway device or laryngeal mask airway) may be integrated skills.

The DOT recommends 300 to 400 hours of initial education that includes didactic classroom lectures combined with hospital and field experiences.[30]

Emergency Medical Technician—Paramedic

The EMT-P is the most advanced out-of-hospital provider. Paramedics have the capability to address most out-of-hospital emergencies. The scope of practice includes a wide variety of therapeutics and procedures including cardiac rhythm recognition, expanded pharmacologic treatments, and advanced airway interventions. Other important invasive procedures include needle decompression of a tension pneumothorax, needle or surgical cricothyrotomy, and transthoracic cardiac pacing.

A recent revision of the National Standard Curriculum for the EMT-P calls for approximately 1000 to 1200 instructional hours, including didactic, clinical, and field education. All course content focuses on technical and professional competency. Additional modules are included that allow programs to incorporate an expanded scope of practice.[31] With the expansion of EMS technology and management career options, many paramedic educational programs have advanced from 1-year certificate curriculums to 2-year associate or 4-year baccalaureate degrees. Recertification requirements include a 48-hour refresher course, 24 hours of yearly continuing education, and BLS and ALS courses at the pediatric and adult levels.

Material Resources

Prior to the mid-1960s, few if any regulations governed system design, operations, and equipment. As EMS development progressed, guidelines for emergency vehicle specifications were adopted by the DOT and equipment lists were proposed. Today, the American College of Surgeons, the ACEP, and the EMS-C Program continue publishing documents that recommend design, equipment, and medications for ambulances.[32,33]

Medications

During the 1980s, many believed that prehospital drug administration was unjustified and simply delayed hospital transport.[34,35] Moreover, there is a profound paucity of outcomes-based research into the use of various medications in the out-of-hospital environment.[36] There is significant evidence for early defibrillation and certain advanced cardiac life support medications, which are carried by most ALS services.[37] The wide variety of alternative medications is less uniform. This includes respiratory and anaphylaxis medications, preparations for altered mental status, analgesics, and antiemetics. Medications are traditionally administered in the field by the parenteral route, but the intranasal route is becoming popular for certain preparations. The beneficial aspects are that absorption is rapid with an onset of action similar to parenteral administration. Two medications that are commonly administered intranasally are naloxone for narcotic overdose and midazolam for pediatric seizure.[38,39]

Equipment

Basic ambulance equipment should include items necessary for emergency procedures (i.e., airway support, hemorrhage control, fracture and spine immobilization, childbirth), personal protection, patient movement, and basic rescue procedures. Additional patient care equipment is predicated on the level of provision outlined by the system design.

Ambulances

Three basic ambulance vehicle designs are recognized by the DOT: type I, type II, and type III. Both type I and type III ambulances incorporate a modular patient compartment mounted on a conventional truck and van chassis, respectively. The type II vehicle is a standard van. The larger medium-duty vehicle, mounted on a business-class chassis, has become popular in recent years. This configuration requires less periodic maintenance and offers extended service time. Each ambulance manufacturer promotes various interior cabinetry and all include sufficient lighting, outlets for 110-volt equipment, suction, oxygen systems, and external audible and visual warning devices. The six-pointed blue star, or "Star of Life," surrounding the staff of Aesculapius is recognized worldwide as the standard symbol for EMS.[40]

Communications

Integral to out-of-hospital care systems, EMS communications involve multiple components, all interlinked to support expedient patient care. Effective communication systems include public information and education programs regarding general access to care, technology to ensure simplified access, means of call prioritization and management of available resources, protocols for providing emergency patient care instructions prior to EMS arrival, ability to communicate with allied agency and hospital personnel, educational opportunities for telecommunicators, and quality improvement processes.

Access

Since 1973, the 9-1-1 universal emergency access telephone number has been adopted by many communities throughout the United States. Basic 9-1-1 service simply connects a caller to a central communications center or public safety answering point (PSAP). Most primary PSAPs are under the domain of law enforcement. Although many of these handle all public service (police, fire, EMS) calls, many larger cities have secondary PSAPs for fire and EMS. Enhanced 9-1-1 provides additional information by immediately displaying the caller's telephone number and address.

Emergency Medical Dispatch

Dispatching encompasses multiple elements that assist patients in receiving expeditious medical care.[41] It is estimated that 30% of EMS calls are for nonemergent conditions, with only 15 to 20% being considered critical or life-threatening.[42]

The emergency medical dispatcher (EMD) is responsible for ascertaining the primary medical condition and severity. Communication centers that model their dispatch response protocols on priority use a finite list of common chief complaints, each having associated predetermined questions. Answers to these questions ultimately dictate a predefined response mode. Depending on the response assigned and system configuration, an ambulance (BLS or ALS) and possibly an FR resource is dispatched to respond in an emergency or nonemergency mode. When critical conditions are identified, the EMD may proceed in giving specific prearrival instructions to assist the caller in providing critical interventions prior to EMS arrival. These include procedures such as opening and clearing an airway, performing CPR, controlling hemorrhage, and assisting with childbirth. Such assistance dramatically narrows the response time interval for receiving emergency medical care.

Systems Status Management

Depending on system size, population served, and resources available, the use of systems status management has proven beneficial for many services. Based on historical data, high-performance or peak-demand periods of the day coupled to service areas or call location can be identified so that coverage plans or posting assignments may be instituted. Such mechanisms place ambulances at predetermined locations where potential calls are likely to occur. Response vehicles may be equipped with an automatic vehicle locator that functions as a telemetry unit, or global positioning satellite system that provides a site interface with the computer-aided dispatch system. This site information is helpful when staging or redeployment of vehicles is required during periods of high call volume or when resources are limited.

Field Communications

While at the scene or during transport, EMTs usually have the capability of communicating with hospital staff. A consultative patient report may be given to receive medication or intervention orders, or simply for arrival notification. EMS providers should also have the capability of communicating with all allied public safety agencies for mutual aid purposes, mass casualty situations, or disaster responses. If air medical services are available, EMS and fire personnel must have the capability of communicating with the helicopter pilot and crew members. Scene personnel must relay landing zone information and potential hazards to the pilot and should provide a preliminary patient report to the medical crew.

MEDICAL DIRECTION

An EMS medical director is a physician with specialized interest and knowledge of patient care activities unique to the out-of-hospital environment. Medical oversight must extend from the communications center through all components of field care. Typically, a contractual arrangement for services provides the physician with administrative authority to implement patient care protocols, to interact with all aspects of the system, and to remove a provider from practice if medical care or behavior is substandard. Published guidelines describing the activities and performance of an EMS medical director have been prepared by ACEP, National Association of Emergency Medicine Services Physicians (NAEMSP), NHTSA, and Health Resources and Services Administration (HRSA).[43-45]

Medical direction consists of off-line (indirect) and on-line (direct) control. Off-line medical control includes protocol development, personnel education, prospective and retrospective patient care review, and other quality improvement processes. Direct medical control concerns real-time interaction between a physician or designee and the field provider.

Indirect Medical Control

Medical accountability for patient care activities is the basis for indirect medical control and functions either

before a patient is encountered (prospective) or after hospital transport has occurred (retrospective). Patient care guidelines and protocol development for EMTs and EMDs, continuing medical education, medicolegal policies, and quality and performance improvement processes are important elements.

Protocols

Perhaps the most important duty of the medical director is to develop patient care protocols. Protocols serve as preestablished practice guidelines that define the standard of care for most illnesses or injuries encountered in the out-of-hospital setting. Operational issues, such as hospital designation and destination policies, termination of resuscitation, and patient transport refusal, may be included. Depending on state regulations, protocols may include standing orders for particular clinical situations in which EMTs may perform certain procedures or administer medications for predefined patient conditions prior to communication with hospital personnel. Protocol development should be driven by system resources and patient needs and should include guidelines for triage and care of specific patient populations, including trauma patients, newborns, and children.

Regardless of local communication protocols, out-of-hospital providers should always be able to discuss a case with a physician for clarification or guidance when clinical questions or controversial situations arise. Furthermore, hospital notification is always important when critical patients are being transported.

Education

Medical directors should be familiar with and actively involved in local or regional educational programs for initial and continuing education courses for all levels of EMT certification. Course curriculum development and administration, evaluation, and revision processes should be understood. Systems that incorporate their own educational programs allow for modifications that reflect intrinsic needs of the system and the providers.

Field personnel and telecommunicators must be given regularly scheduled courses that improve competency in knowledge and skills. Instructional formats and resources to accomplish educational objectives may include didactic classroom lectures, skill labs, direct field observation, or distance learning models for self-paced opportunities. Standardized core content is important for maintaining consistency and quality of care.

Quality and Performance Improvement

Once patient care protocols are developed and implemented, there must be mechanisms, such as retrospective patient care report review or direct field observation, for evaluating individual and system performance and patient outcome. Deviations from specific protocols may reflect problems with individual EMTs, medical control personnel, or the protocol itself, each requiring education and reevaluation. Deficiencies, both operational and clinical, must be identified for appropriate remediation to occur, which may be in the form of counseling, educational instruction, or revisions of system design or patient care protocols.[46] Competency, knowledge retention, and skill performance are measurable parameters. Time standards (e.g., out-of-chute time [time from ambulance notification to deployment], response time, and scene time) are equally important measures.[47]

Direct Medical Control

Direct medical control is the concurrent direction of EMTs providing patient care. This may be in the form of radio or telephone communications or by direct scene observation and may be considered centralized or decentralized. In a centralized system, a selected hospital is designated as the lead facility (base station hospital, resource hospital, or sponsor hospital) and is responsible for providing all direct medical control orders and notification regardless of the receiving facility. In a decentralized system, each hospital functions as a base station, providing direction to EMTs transporting patients to its facility.

Personnel responsible for direct medical control must be knowledgeable about the entire EMS system, receiving facilities, protocols, medication formulary and equipment, administrative and operational issues, and medicolegal implications for certain presenting situations. Systems whose protocols include standing orders may only require direct communication for specific reasons. Thus, while these medical and administrative protocols may guide EMTs through most circumstances, medical control consultation may assist with medicolegal issues, situational problems at the scene, patient nontransport, or a multitude of potential ethical dilemmas that may be encountered. Nevertheless, direct medical control is usually invaluable for notifying a receiving facility for treatment room and staff preparation when critical or potentially critical patients are being transported.

OUT-OF-HOSPITAL MEDICAL CARE AND CONTROVERSIES IN MANAGEMENT

Airway Support and Respiratory Emergencies

Interventions

Respiratory complaints account for a significant number of EMS responses. Basic measures to control and support a patient's airway include manual maneuvers (e.g., chin lift or jaw thrust), oral and nasopharyngeal devices, and use of bag-mask ventilation. At a more advanced level, interventions may include blind-insertion airway devices (e.g., Combitube or laryngeal mask airway), which have been shown to enable faster placement and provide improved minute ventilation. Studies have shown that basic-level EMTs were able to successfully place laryngeal mask airways in simulated arrest models and also demonstrated an improved minute ventilation with these devices when compared with bag-valve mask ventilations.[48,49] Similar studies have demonstrated that laryngeal mask airways are more successful than endotracheal intubation for paramedics, because they provide a faster technique, require fewer attempts for successful insertion, and improve ventilation.[50,51]

Commonly used by air medical services, drug-assisted intubation (DAI) and rapid sequence intubation (RSI) procedures have recently expanded in ground transport services, despite a lack of supporting evidence. Several long-standing programs have achieved great success using RSI; however, others have not appreciated the benefits and have questioned the usefulness.[52,53] Several studies have challenged the effectiveness of out-of-hospital intubation, particularly in view of an alarming incidence of esophageal intubation in some systems and poor outcomes with the use of RSI for head-injured patients.[54,55] One prospective, randomized study of pediatric out-of-hospital airway management concluded that in the urban setting, bag-mask ventilation may be superior to intubation in

certain patient groups.[56] Although controversy exists and the debate will continue, most would agree that in order to have a successful airway management program, the educational and quality management component must be meaningful and should be as comprehensive and compulsive as possible.[57] For programs using DAI or RSI procedures, the experiential component should include operating room time and simulator sessions. Ideally, training would also occur in an ED setting where patients requiring emergent intubation would potentially have the full complement of confounding variables (e.g., combative status, full stomachs, blood and vomit in the airway).

Traditionally used in the hospital, continuous positive airway pressure (CPAP) is intensifying in the out-of-hospital setting. The effectiveness of out-of-hospital use of CPAP has been demonstrated; however, patient outcome studies have been limited.[58,59] Out-of-hospital use would require strict protocols that would outline such variables as indications and contraindications, clinical applications, mental status assessment, hemodynamic status, and mechanisms for transferring the patient at the hospital.

Medications

Most advanced programs have adopted the use of clinically proven medications for bronchospasm, chronic obstructive pulmonary disease, and anaphylaxis, but no studies have demonstrated benefit to administration of these medications in the out-of-hospital environment. While some studies might be considered unethical (e.g., an out-of-hospital study of epinephrine for anaphylaxis), others (e.g., out-of-hospital use of beta$_2$-agonists or steroids for asthma, or loop diuretics for pulmonary edema) could easily be performed, with the results far from certain. Pending further studies, most systems have adopted the position that these medications do not harm patients in the out-of-hospital setting, may be helpful, and may provide comfort and clinical improvement for most patients experiencing varying degrees of respiratory distress. The overhead related to training and maintenance of knowledge related to these additional, probably unnecessary, medications is rarely considered.

Cardiovascular Emergencies

Interventions

Previous research has demonstrated the effectiveness of early defibrillation for terminating ventricular fibrillation and improving survival rates from sudden cardiac death.[60] Advances in technology have improved such that defibrillators, traditionally used by paramedics, are now used by a variety of public safety responders and bystanders. Public access defibrillation (PAD) programs are being implemented throughout the country, with devices being placed in high-volume, populous, and secluded areas such as airports and airplanes, casinos, and office buildings.[61] The effectiveness of PAD is currently being investigated.[62] The acquisition and transmission of out-of-hospital 12-lead electrocardiograms is becoming more prevalent as well. Although expensive to implement, several studies have revealed minimal delays in scene time while obtaining the ECG, and a shorter time to intervention (thrombolytic administration or catheterization lab admission) by using this technology.[63,64]

Although the statistics for cardiac arrest survival across the United States are dismal, those that do survive may suffer some degree of hypoxic encephalopathy. Recent evidence suggests that cooling patients who achieve a spontaneous return of circulation following cardiac arrest, especially with ventricular fibrillation as the initial rhythm, achieve higher survival rates and level of neurologic functioning.[65,66] The explanation may be due to several mechanisms, including a decrease in neuronal cell oxygen consumption, cell membrane protection, slowing of degradative reactions resulting from reperfusion, and limiting acidosis.[67] International guidelines now call for the institution of hypothermia for patients who are resuscitated from cardiac arrest, and many out-of-hospital systems have implemented protocols that may include administration of chilled saline, sedation, or neuromuscular blockers, in coordination with receiving hospital emergency departments.

Medications

Traditional cardiac medications recommended by advanced cardiac life support are used by most ALS systems. Recent investigations involving amiodarone as an out-of-hospital agent to terminate refractory ventricular fibrillation have resulted in higher survival rates to hospital arrival; however, improvement in survival to discharge is still not significant.[68] Whether amiodarone should replace lidocaine for out-of-hospital ventricular fibrillation requires further investigation, although many systems have already made this expensive change. The use of out-of-hospital fibrinolytic agents for acute ST elevation myocardial infarction has not gained wide acceptance and may only be a useful intervention for systems having prolonged transport times, or if hospitals may not have catheterization or intervention facilities available. Future recommendations for out-of-hospital use of these agents remains speculative.

Traumatic Emergencies

Interventions

Interventions for specific medical emergencies, such as cardiac arrest (i.e., defibrillation, intubation, IV and medication administration), may be effectively performed while on the scene or prior to hospital transport. Alternatively, it is widely accepted that most interventions for traumatic injuries should be performed while en route to the hospital, and all efforts should be extended to reduce on-scene time.

The issue of IV fluid administration has gained controversy over the past several years. High-volume IV fluid for hemodynamic instability resulting from traumatic injury has traditionally been the accepted standard in most out-of-hospital care systems. Recent data, however, support a paradigm shift to restrictive or hypotensive resuscitation for penetrating truncal injuries. Restoration of hemodynamic stability with fluid resuscitation prior to definitive surgical hemostasis may lead to increased morbidity.[69] Likewise, the use of the pneumatic antishock garment has been shown to increase mortality rates in penetrating torso injuries and is no longer recommended.

Similar to medical patients, definitive airway support by endotracheal intubation may be beneficial for severely injured patients although these benefits of intubation in improving patient outcome have not been clearly delineated. To be successful, paramedics must exhibit the technical skills to rapidly place the endotracheal tube correctly, assess the placement, and move the intubated patient appropriately. In addition, providing the correct minute and tidal volumes is equally important. Overzealous personnel subconsciously delivering hyperventilatory rates may impair cardiac output and cause further tissue damage. Patients sustaining blunt head injury pose special problems that must be expeditiously addressed and resolved. Intubation provides a solid means of providing ventilatory assistance and airway protection, but the procedure and postintubation care may negate these potential benefits.

BOX 190-1 EMTALA REQUIREMENTS FOR PATIENT TRANSFERS

Complete certification (risks and benefits) of transfer
Informed consent obtained from the patient or family
Appropriate transportation (equipment and personnel) arranged
Treatment and stabilization performed
Acceptance from receiving facility ensured
Appropriate patient care data sent (fax or with patient)

EMTALA, Emergency Medical Treatment and Active Labor Act.

Attempting to intubate head-injured patients may result in dental or soft tissue damage in those patients with clenched teeth, and intracranial pressure may be exacerbated from an intact gag reflex or from subsequent regurgitation. Recent studies on the use of RSI in head-injured patients reveal that patients experience significant hypoxia and bradycardia during the procedure, and outcome is actually worse.[48] Thus, the role of RSI in prehospital airway management in trauma patients is in question, just as it is for medical patients.

INTERFACILITY AND SPECIALIZED TRANSPORTS

Transportation between health care facilities may occur for several reasons including patient preference, unavailable diagnostic or therapeutic resource availability at the transferring facility, or managed care requirements that patients be cared for in predesignated hospitals following stabilization. Hospital corporations engaged in networks or alliances that share resources and services depend on interhospital transport systems to convey patients to allied institutions for specialized tests or procedures. Likewise, critical patients admitted to less specialized facilities may need to be transferred to tertiary care or designated trauma centers. Whereas long-distance transports may be best accomplished by air medical services, regional or local transports should use ground systems. These may be provided by either local EMS resources or those owned and operated by the hospital.

Interfacility transfer of patients that is medically indicated must fall under a set of requirements referred to as the Emergency Medical Treatment and Active Labor Act (EMTALA).[70] Although the EMS system providing the transport plays a key role, these guidelines primarily involve particular information and obligations that must be satisfied by the transferring and receiving facility prior to transfer. It is important to note that an unstable patient should not be transferred to another facility at the request of a managed care organization unless the transferring hospital is incapable of providing standard care and the receiving hospital does have the capability to manage the condition and foreseeable complications. Box 190-1 lists various requirements that must be completed prior to transferring a patient to another facility.

Depending on patient condition, specialized transport services may function at the BLS or ALS level, providing emergency or nonemergency transportation. Patient transfers considered ALS may include interhospital (either ED or intensive care unit) neonatal or high-risk infant, critical cardiac, or trauma transports. Personnel configuration depends on system design and level of care provided. Many programs use a nurse-paramedic combination. Patients requiring specialized care may need the services of specifically trained individuals, such as respiratory therapists, neonatal nurses, or other specialized critical care personnel. The presence of a physician is not mandatory but may be useful in selected cases.

As with any EMS activity, all interfacility transports should be reviewed for appropriateness of transfer and medical care provided. In 1993, The Practice Management Committee of ACEP updated the 1990 policy statement on interfacility transfers.[71]

Table 190-2 EMS Resource and Contact Information

RESOURCE	URL
Advocates for EMS	www.advocatesforems.org
American Ambulance Association	www.the-aaa.org
American College of Emergency Physicians	www.acep.org
Centers for Disease Control and Prevention	www.cdc.gov
Commission on Accreditation of Ambulance Services	www.caas.org
EMS Division, NHTSA	www.nhtsa.dot.gov/people/injury/ems
Maternal and Child Health Bureau, EMS-C	www.ems-c.org
National Association of EMS Educators	www.naemse.org
National Association of EMS Physicians	www.naemsp.org
National Association of EMTs	www.naemt.org
National Association of State EMS Officials	www.nasemsd.org
National Registry of EMTs	www.nremt.org

EMS, emergency medical services; EMT, emergency medical technician; NHTSA, National Highway Traffic Safety Administration.

THE FUTURE

Providing quality, efficient, and responsible health care to the right patient, in the right setting, at the right time will always be laudible objectives for any system, but there is a need for research to demonstrate which interventions are conducive to better patient outcome. As the call volumes increase, it is imperative that systems focus on those interventions, from both the training and health care delivery perspectives, that are known to be of benefit. Agencies and organizations involved in EMS development and oversight are listed in Table 190-2.

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The references for this chapter can be found online by accessing the accompanying Expert Consult website.

PART IV

Environment and Toxicology

CHAPTER 137 Frostbite

Daniel F. Danzl

PERSPECTIVE

Peripheral cold injuries occur primarily in humans. The highest homeostatic priority is to maintain the body's core temperature. This is accomplished through vasoconstriction and shunting, which prevents adequate heat distribution to the extremities. As a result, failure to achieve adequate protection from the environment results in injuries that are usually preventable.[1–4]

Peripheral cold injuries include both freezing and nonfreezing syndromes, which may occur independently or in conjunction with systemic hypothermia.[5] Frostbite is the most common freezing injury.[6] Trench foot and immersion foot are nonfreezing injuries that result from exposure to wet cold.[7] Nonfreezing injury that usually occurs after exposure to dry cold is termed *chilblains* (pernio).

The incidence and severity of frostbite correlate with the predisposing factors associated with the cold stress. Most cases of civilian frostbite result from routine exposure to cold by individuals who have not given due consideration to risk factors for cold injury.[8–10] Well-equipped treks up the world's highest peaks have been completed without cold injury when appropriate steps are taken to address these factors.[11] Current trends show an increase in outdoor recreational activities that produce exposure to unanticipated drastic climatic changes.[12,13] Unsheltered and homeless people are no longer the most likely group at risk.

Military history is replete with accounts of the effects of cold injury on combat troops.[14,15] Amputations and time lost to local cold injuries in both World Wars and the Korean conflict were extensive. Trench foot was particularly common among the Royal Marines in the Falkland Islands and United States troops in the Vietnam War.

Napoleon's Surgeon General, Baron de Larrey, first recorded the disastrous effects of the freeze-thaw-refreeze cycle.[16] During the 1812 to 1813 Russian invasion and retreat, soldiers would acutely thaw frozen extremities directly over open fires. The subsequent refreeze further increased tissue destruction. Unfortunately, the resultant gangrene was misattributed to this rapid thawing of frostbite and trench foot injuries. Therefore, gradual thawing, often including friction massage with snow, remained the standard treatment regimen until the 1950s.[17,18] In addition to dry radiant heat rewarming and massage, another misdirected rewarming modality was immersion thawing in icy water. In 1961, Mills ultimately popularized rapid warm immersion rewarming after extensive experience with severe Alaskan frostbite cases.[18,19]

PRINCIPLES OF DISEASE

Physiology

Human cold stress should induce adaptive behavioral reactions such as an attempt to find heat or shelter. In addition, complex endocrinologic and cardiovascular physiologic responses are engaged. Peripheral cooling of the blood activates the preoptic anterior hypothalamus. This central thermostat orchestrates temperature regulation. This dynamic process encompasses catecholamine release, thyroid stimulation, shivering thermogenesis, and peripheral vasoconstriction.

Cutaneous circulation is one of the keys to maintaining thermoneutrality. Baseline cutaneous circulation greatly exceeds the nutritional requirements. This reflects the skin's "radiator" function to maintain thermostability. Cutaneous blood flow in the euthermic 70-kg human averages 200 to 250 mL/min. Heat stress causes vasodilation that can increase this amount to 7000 mL/min. In contrast, extreme cold-induced vasoconstriction reduces flow 10-fold to less than 50 mL/min.

During cold stress, peripheral vasoconstriction limits radiant heat loss. Acral skin structures (fingers, toes, ears, nose) contain a plethora of arteriovenous anastomoses. These facilitate shunting and subsequent drastic reductions in blood flow to these areas. This "life-versus-limb" mechanism reflects the homeostatic attempt to prevent systemic hypothermia.

In contrast to heat exposure, humans do not appear to display significant physiologic adaptation to the cold. Exposing extremities to temperatures down to 15° C results in maximal peripheral vasoconstriction with minimal blood flow. Continued exposure to progressively colder temperatures down to 10° C produces the "hunting response," which is termed *cold-induced vasodilation*.[20] These periods of vasodilation, recurring in 5- to 10-minute cycles, interrupt vasoconstriction and serve to protect the extremity. Eskimos, as well as Lapps and others of Nordic extraction, are capable of stronger cold-induced vasodilation than individuals from tropical regions. Measuring the speed of cold-induced vasodilation may help predict an individual's risk for cold injury.[21]

Pathophysiology

The pathologic phases that occur with local cold injury often overlap and vary with the extent and rapidity of the cold response (Box 137-1). Frostbite occurs when the tissue temperature drops to less than 0° C. There are two putative

BOX 137-1 FREEZING INJURY CASCADE

Prefreeze phase
- Superficial tissue "cooling"
- Increased viscosity of vascular contents
- Microvascular constriction
- Endothelial plasma leakage

Freeze-thaw phase
- *Extra*cellular fluid ice crystal formation*
- Water diapedesis across cell membrane
- *Intra*cellular dehydration and hyperosmolality
- Cell membrane denaturation or disruption
- Cell shrinkage and collapse

Vascular stasis and progressive ischemia
- Vasospasticity and stasis coagulation
- Arteriovenous shunting
- Vascular endothelial cell damage or prostanoid release
- Interstitial leakage or tissue hypertension
- Necrosis, demarcation, mummification, or slough

*Extremely rapid cooling produces more initial intracellular than extracellular ice crystallization.

mechanisms of tissue injury: architectural cellular damage from ice crystal formation and microvascular thrombosis and stasis.

In the *prefreeze* phase, tissue temperatures drop below 10° C and cutaneous sensation is lost. Before ice crystal formation, microvascular vasoconstriction occurs along with endothelial leakage of plasma into the interstitium. Radiation and conduction of heat from deeper tissues prevent crystallization until the skin temperature drops well below 0° C.

In the *freeze-thaw* phase, the timing, location, and rate of ice crystal formation depend on the exposure circumstances. In addition to ambient temperatures, wind and moisture increase the freezing rate. Skin must cool below 0° C to freeze because of the underlying radiation of heat.

During usual exposure conditions, ice crystal formation initially occurs extracellularly. This results in diapedesis of water exiting the cell to maintain osmotic equilibrium. Cellular dehydration increases the intracellular osmolarity and electrolyte concentrations. When approximately one third of the cellular volume is lost, cellular collapse and death result. This may occur with or without direct architectural damage from the crystals. Extracellular crystallization also increases the tissue pressure on cell membranes and surrounding vascular structures. Sludging, stasis, and cessation of flow are initiated at the capillary level.

The third phase, progressive microvascular collapse, first affects venules and then arterioles. Red blood cells sludge and form microthrombi during the first few hours after the tissues are thawed. Factors adversely affecting flow include hypoxic vasospasm, hyperviscosity, and direct endothelial cell damage. Anaerobic metabolism subsequently extends the surrounding injury. Tissues are deprived of nutrients and oxygen. Ultimately, plasma leakage and arteriovenous shunting result in thrombosis, increased tissue pressure, ischemia, and necrosis.

Some direct skin injury is reversible. For example, frozen skin grafted to a normal site can survive. Zones immediately surrounding injured areas are potentially salvageable. The histopathology of frostbite suggests that some changes in the epidermis are primary and some reflect damage to the endothelial cells. During initiation of rewarming, these tissues are revitalized.

An additional insult, progressive dermal ischemia, is partially mediated by thromboxane.[22] Fluid analysis of clear vesicles identifies prostaglandins. When subdermal vascular plexi are injured, hemorrhagic blisters develop that also contain these prostanoids. The arachidonic acid breakdown products released from underlying damaged tissue into the blister fluid include both prostaglandins and thromboxane. These mediators produce platelet aggregation, vasoconstriction, and leukocyte immobilization.[23]

The ultimate determinant of progressive tissue damage appears to be injury to the microvasculature. Endothelial cells are the tissue most susceptible to freezing injury. After thawing, the vasculature is patent only temporarily. Platelet and erythrocyte aggregates promptly clog and distort the vasculature. Intense vasoconstriction coupled with arteriovenous shunting occurs at the interface between normal and damaged tissue. The injured viable vasculature remains distorted. Local arteritis, medial degeneration, and intimal proliferative thickening are seen. Nerve and muscle tissues are also more susceptible to cold injury than connective tissue. For example, nonviable hands and feet can be moved after thawing if the tendons are intact.

Edema progresses for 48 to 72 hours after tissue has been thawed. Leukocyte infiltration, thrombosis, and early necrosis become apparent as this edema resolves. The dry gangrene carapace of frostbite is superficial in comparison to arteriosclerotic-induced, full-thickness gangrene. Final clinical demarcation between viable and nonviable tissue can require more than 60 to 90 days, hence the historical surgical aphorism, "Frostbite in January, amputate in July."[19] Advances in imaging modalities can accelerate the identification of demarcation.

Predisposing Factors

The extent of peripheral cold injury is determined by the type and duration of cold contact with the skin[24,25] (Box 137-2).

Any conditions affecting judgment can jeopardize the physiologically tropical human. In urban settings, cold injuries are often attributed to overt or covert risk taking or psychiatric impairment or ingestion of intoxicants. Ethanol also produces peripheral vasodilation, which increases heat loss. Whenever self-protective instincts are blunted, appropriate adaptive maneuvers may not be undertaken.

Direct skin contact with good thermal conductors such as metal, water, and volatile liquids affects the extent and rapidity of tissue destruction. Commercial aerosol spray propellants such as propane and butane are potentially hazardous. One passenger on a commercial airline flight suffered a full-thickness lumbar injury from a "cold pack" provided by the stewardess that contained dry ice.[24–26] Overenthusiastic application of standard ice packs while treating soft tissue injuries can also result in tissue loss.[27] Although air alone is a poor thermal conductor, associated cold and wind (wind chill index) markedly increase heat loss.

■ CLINICAL FEATURES

Symptoms and Signs

"Frostnip" is a superficial cold insult manifested by transient numbness and tingling that resolves after rewarming. This does not represent true frostbite, because no tissue destruction occurs.

The symptoms of frostbite usually reflect the severity of the exposure. The most common presenting symptom is numbness, present in more than 75% of patients. All patients

BOX 137-2 PREDISPOSING FACTORS

Physiologic
Genetic
Core temperature
Previous cold injury
Acclimatization
Dehydration
Overexertion
Trauma: multisystem, extremity
Dermatologic diseases
Physical conditioning
Diaphoresis, hyperhidrosis
Hypoxia

Mechanical
Constricting or wet clothing
Tight boots
Vapor barrier, alveolite liners
Inadequate insulation
Immobility or cramped positioning

Psychological
Mental status
Fear, panic
Attitude
Peer pressure
Fatigue
Intense concentration on tasks
Hunger, malnutrition
Intoxicants

Environmental
Ambient temperature
Humidity
Duration of exposure
Wind chill factor
Altitude and associated conditions
Quantity of exposed surface area
Heat loss; conductive, evaporative
Aerosol propellants

Cardiovascular
Hypotension
Artherosclerosis
Arteritis
Raynaud's syndrome
Cold-induced vasodilation
Anemia
Sickle cell disease
Diabetes
Vasoconstrictors, vasodilators

have some initial sensory deficiency in light touch, pain, or temperature. Anesthesia is produced by intense vasoconstrictive ischemia and neurapraxia. Acral areas and distal extremities are the usual insensate sites. The distal extremities—the fingers, toes, nose, ears, and penis—are specific locations at risk. Patients often complain of clumsiness and report a "chunk of wood" sensation in the extremity. The history of complete acute anesthesia in a painful cold digit suggests a severe injury.

Significant pain usually accompanies reestablishment of perfusion. With partial tissue destruction, intermittent pain may be noticed during ongoing exposure. The dull continuous ache evolves into a throbbing sensation in 48 to 72 hours. This often persists until tissue demarcation several weeks to months later.

Chilblains (pernio) is a mild form of cold injury that often follows repetitive exposure. These "cold sores" appear less than 24 hours after exposure and usually affect facial areas, the dorsa of the hands and feet, and the pretibial areas. Young women with a history of Raynaud's phenomenon or systemic lupus erythematosis or with antiphospholipid antibodies are especially at risk. Persistent vasospasm and vasculitis result in burning, pruritus, erythema, and mild edema. Plaques, blue nodules, and ulcerations can develop and last 1 to 2 weeks.

The other common nonfreezing cold injury is trench foot (immersion foot). This remains a significant threat during recreational activities and expeditions in cold, wet climates. Trench foot is produced by prolonged exposure to wet cold at temperatures above freezing.[7] It usually develops slowly over several days and results in neurovascular damage in the absence of ice crystal formation. Immersion foot commonly develops while a person is wearing sweat-dampened or neoprene socks, vapor-barrier boots, or constrictive gaiters. Patients who soak their feet for hours each night in cool water for pain relief are also at risk.

The clinical presentation varies. Most patients' symptoms include cool, pale feet that are numb or tingle. Later the feet appear cyanotic, cold, and edematous. Often, numbness and leg cramping are present. The clinical hallmark is that after rewarming, the skin remains erythematous, dry, and very painful to touch. Rubor on dependency and pallor on elevation are caused by vasomotor paralysis.

Bullae that are indistinguishable from those seen with frostbite commonly develop. Vesiculation proceeds to ulceration and liquefaction gangrene in severe cases. Protracted symptoms of pain during weightbearing, cold sensitivity, and hyperhidrosis often last for years. Prevention of trench foot often simply requires continual drying of socks.

Classically, the initial presentation of frostbite is deceptively benign. Most patients do not arrive in the emergency department with frozen, insensate tissue. Frozen tissues often appear mottled or violaceous-white, waxy, or pale yellow. In severe cases, the examiner will not be able to roll the dermis over bony prominences. Rapid rewarming results in an initial hyperemia, even in severe cases. After thawing, partial return of sensation should be expected until blebs form.[19]

Favorable initial symptoms include normal sensation, warmth, and color. Soft, pliable subcutaneous tissue suggests a superficial injury. A residual violaceous hue after rewarming is ominous. Early formation of clear large blebs that extend to the tips of the digits are more favorable than delayed appearance of smaller hemorrhagic blebs. These dark vesicles are produced by damage to the subdermal vascular plexi. Vesicles and large bullae eventually form in 6 to 24 hours.

Lack of edema formation suggests significant tissue damage. Post-thaw edema usually develops in less than 3 hours. In severe cases, frostbitten skin forms an early black, dry eschar until mummification and apparent demarcation.

Historically, frostbite, like burns, has been classified into degrees of injury. Anesthesia and erythema are characteristic of first-degree frostbite. Superficial vesiculation surrounded by edema and erythema is considered second-degree. Third-degree frostbite produces deeper hemorrhagic vesicles. Fourth-degree injuries extend into subcuticular, osseous, and muscle tissues.

Classification by degrees is often incorrect in relation to the actual severity of the frostbite and thus therapeutically misleading. Mills suggests two simple retrospective classifications.[18,19] Superficial or mild frostbite does not entail eventual tissue loss, whereas deep or severe frostbite does result in tissue loss. As a result, it is not feasible to predict, on presentation, the eventual tissue loss. Another classification attempts to establish severity based on clinical features coupled with early bone scan results.[28]

DIAGNOSTIC STRATEGIES

Many ancillary diagnostic imaging techniques attempt to diagnose the severity of injury. Unfortunately, none consis-

tently and accurately predict tissue loss at the time of initial examination.

Routine baseline radiographs should be obtained, and at follow-up radiographs will begin to demonstrate specific frostbite abnormalities 4 to 10 weeks after injury. Intravenous isotope studies have mixed success experimentally and clinically.[29,30] In one study, triple-phase bone scans performed 2 days after cold injury demonstrate ischemic tissue at risk.[31,32] Delayed bone scans in 7 to 10 days can image deep tissue and bone infarction. The absence of radionuclide uptake even after 10 days, however, does not reliably predict the eventual need for amputation. The patient should be advised that accurate prediction of eventual tissue loss is difficult. As an ancillary tool, scintigraphy predicts the eventual demarcation line better than thermography.[33,34] Scintigraphy as early as day 2 may predict tissue loss and monitor the efficacy of treatment.[35]

Large vessel angiography does not assess the microvasculature at presentation because of vasospasm. Papaverine may help distinguish vasospasm from frostbite sludging and vascular injury. Transitory vascular instability often lasts 2 to 3 weeks. Angiography does facilitate evaluation of associated traumatic or chronic vascular abnormalities. Doppler ultrasonography and digital plethysmography are insensitive but may help determine the need for sympathetic blockade.

In clinical practice, magnetic resonance imaging and magnetic resonance angiography may be superior to technetium bone scanning. In one study, the clear-cut line of demarcation was noted before clinical demarcation.[36]

MANAGEMENT

Field rewarming of frozen tissue is rarely practical. If possible, constricting or wet clothing should be removed and affected areas insulated and immobilized. Friction massage is not efficacious and increases tissue loss. Frozen parts should be kept away from dry heat sources in the transport vehicle to prevent a gradual partial thaw.

A direct relationship exists between the length of time that tissue is frozen and the ultimate extent of cellular damage. Rewarming should not be initiated in the field, however, if there is any potential for interrupted or incomplete thawing. Tissue refreezing is disastrous, and it is preferable to ambulate to safety on frozen extremities if rescue will be delayed.

When evacuation is not possible, rapid field rewarming, preferably in water at 40° to 42° C, may be the only option. Logistic considerations include risks to the party, the availability of shelter and necessary equipment, and the anticipated mode of eventual transportation.

Emergency Department

Prethaw

Pertinent history regarding the ambient temperature, wind velocity, and duration of exposure should be obtained. The type of apparel worn, the circumstances surrounding rescue, and the presence of preexisting cardiovascular or neurologic diseases that could affect tissue loss should be noted.[37]

After stabilizing the core temperature and addressing associated conditions, rapid thawing should be initiated. Treatment should not be delayed while awaiting the results of laboratory and radiographic studies. Most patients have some degree of dehydration and benefit from crystalloid administration. Poor oral intake and hypothermia-induced cold diuresis further increase blood viscosity and sludging.

Thaw

Frozen or partially thawed tissue should be rapidly and actively rewarmed by immersion in gently circulating water that is carefully maintained at a temperature of 40° to 42° C by thermometer measurement.[18] Marginal tissue can be thermally injured when the water temperature exceeds 42° C. Although a circulating tank is ideal for the extremities, a large container suffices for the hands or feet. In some cases, 35° to 40° C water is better tolerated and less painful.

Incomplete thawing and increased tissue loss are hazards when lower water temperatures are used. Rewarming should be continued until the part feels pliable and distal erythema is noted. This usually requires 10 to 30 minutes of submersion. Active gentle motion of the part by the patient during rewarming should be encouraged, but avoid direct tissue massage.

Parenteral analgesia is often indicated during rewarming of deep frostbite. Reperfusion is intensely painful. It produces throbbing, burning pain, and tenderness. A common error is premature termination of rewarming, which results in a partial thaw. Sensation is often diminished after thawing until it disappears with bleb formation.

Patients with completely frozen extremities are invariably hypothermic and at risk for significant fluid and electrolyte fluxes during rewarming. The acute thawing of large amounts of distal musculature extinguishes peripheral vasoconstriction. This results in the sudden return of cold, hyperkalemic, acidotic blood to the central circulation. This produces "core temperature after-drop," which is dysrhythmogenic. In the most severe cases, extracorporeal rewarming should be considered to manage these massive metabolic and electrolyte derangements (Box 137-3).

Post-thaw

The injured extremities must be kept elevated to minimize edema formation. Sterile dressings should be applied and

BOX 137-3 EMERGENCY DEPARTMENT REWARMING PROTOCOL

Prethaw
- Assess Doppler pulses and appearance
- Protect part—no friction massage
- Stabilize core temperature
- Address medical and surgical conditions
- Rehydrate patient
- Prevent partial thaw and refreeze

Thaw
- Provide parenteral ketorolac and analgesia
- Administer ibuprofen 400–600 mg q 6 hours PO
- Immerse part in circulating water that is thermometer-monitored at 37° to 40° C
- Encourage gentle motion of part

Post-thaw
- Dry and elevate part
- Aspirate or débride clear vesicles
- Débride broken vesicles and apply topical antibiotic or sterile aloe vera ointment every 6 hours
- Leave hemorrhagic vesicles intact
- Consider tetanus and streptococcal prophylaxis
- Provide hydrotherapy at 37° C tid
- Consider phenoxybenzamine in severe cases
- Consider imaging, angiography, and thrombolysis
- Obtain admission and serial photographs

involved areas handled gently. Persistent cyanosis in the extremities after a complete thaw may reflect increased fascial compartment pressure. Because of the cold-induced anesthesia, other occult soft tissue injuries are often not appreciated by the patient or physician. Tissue pressures should be monitored carefully, although decompressing escharotomies are usually not necessary during the initial treatment rendered in the emergency department.

The clinical role of thromboxane inhibition in frostbite is limited. Thromboxane inhibition does not appear to result in additional clinically significant tissue salvage. In one experimental model, methimazole did not improve tissue survival even when therapy was initiated immediately.[38] Progressive secondary dermal ischemia is addressed by attempts to limit the accumulation of the products of arachidonic acid breakdown. Topical aloe vera (Dermaide) every 6 hours is a specific thromboxane inhibitor when applied directly to frostbitten areas[23] but has not definitively been proven to salvage tissue. Other alternatives include topical antibiotic ointment. Systemically, ibuprofen appears preferable to salicylates. Although both agents inhibit this cascade, ibuprofen also produces fibrinolysis. Parenteral ketorolac should also be considered.

Frostbite blister management varies widely. Earlier recommended options for large clear blisters included leaving them intact, débridement, or aspiration (Fig. 137-1). Although all clinicians débride broken blisters, many prefer to aspirate intact clear blisters rather than leaving them intact. In contrast, if hemorrhagic blisters are débrided, secondary desiccation of deep dermal layers appears to extend the injury (Fig. 137-2). In this case, aspiration is preferable to débridement.

In severe cases, parenteral penicillin is indicated for streptococcal prophylaxis. Cultures and Gram stains of areas adjacent to the damaged tissue should be performed. Common organisms include staphylococci, streptococci, and *Pseudomonas* species. Broad-spectrum prophylaxis should be considered if excessive heat was used to thaw tissue, since liquefaction and infection are inevitable. Tetanus can also occur after frostbite.

Management of the chilblains syndrome is usually supportive. Nifedipine (20–60 mg daily) is an effective treatment for refractory perniosis.[39–41] Topical or systemic corticosteroids have also been useful. Other options include oral pentoxifylline or limaprost, a prostaglandin E_1 analogue.

Adjunctive Treatment

Numerous ancillary modalities have been suggested for frostbite.[42] The only treatment consensus is removal from the cold and rapid complete thawing in a 40° to 42° C bath.

Capillary flow ceases early after cold injury, whereas thrombosis proceeds.[11,32] This observation has led to multiple experimental antithrombotic and vasodilation treatment regimens, although most lack adequate controls. Most of these studies were conducted before the elucidation of some of the pathophysiologic consequences of frostbite. As demonstrated in a pilot study on triple-phase bone scans, thrombolytic agents may restore some flow to severely frostbitten limbs. The fibrinolytic agent urokinase can also conserve slowly thawed tissue.

In one study, intravenous tissue plasminogen activator and heparin reduced predicted digit amputations in severe frostbite.[35] Nonresponders had over 24 hours of exposure, over 6 hours of warm ischemia, or multiple freeze-thaw cycles. In another study, intra-arterial tissue plasminogen activator decreased the incidence of amputations when administered within 24 hours.[43]

Low-molecular-weight dextran may inhibit intravascular cellular aggregation. Animal models suggest that low-molecular-weight dextran is not harmful. Pentoxifylline, a phosphodiesterase inhibitor, may decrease blood viscosity and increase tissue oxygenation.[44] Its ability to increase red blood cell flexibility facilitates revascularization and may enhance tissue survival. The suggested dosage is 400 mg three times daily for 2 to 6 weeks.[45]

Various anti-inflammatory drugs and other agents have not been conclusively evaluated. These include steroids, nonsteroidal anti-inflammatory drugs, dipyridamole, dimethyl sulfoxide, nonionic detergents, and calcium channel blockers.[46,47] A long-acting alpha-blocker, phenoxybenzamine, may decrease vasospasm while increasing peripheral blood flow. The dosage starts with 10 mg/day to a maximum of 60 mg/day. With this agent, adequate hydration is necessary to prevent orthostatic hypotension.

Hyperbaric oxygen produces vasoconstriction and subsequently reduces cutaneous blood flow. A small number of patients report a temporary flush and increased limb motion, but this appears to depend on the elapsed time interval after injury. Hyperbaric oxygen could accelerate demarcation. Insufficient data on severe frostbite exist to assess the potential value of hyperbaric oxygen therapy for tissue salvage.[48,49]

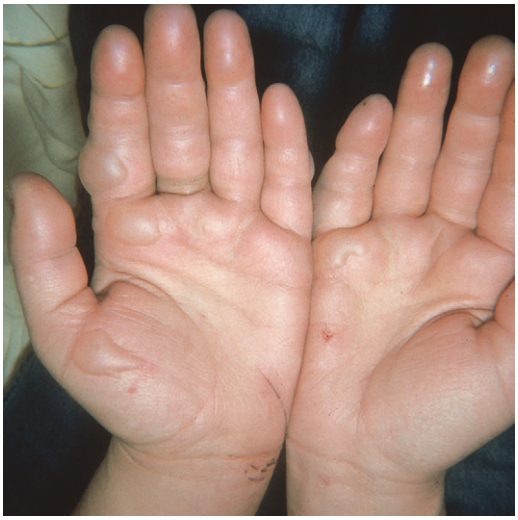


Figure 137-1. Frostbite with clear vesiculations. (Courtesy of Bill Mills, MD.)

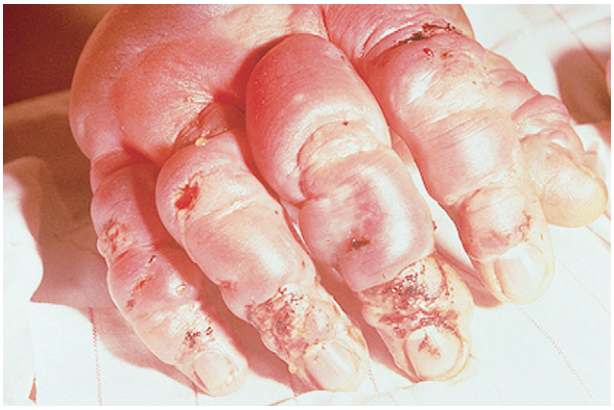


Figure 137-2. Severe frostbite with early hemorrhagic vesicles. (Courtesy of Bill Mills, MD.)

The theoretical benefits of sympathectomy include relief of painful vasospasm, decreased edema, and tissue salvage. Long-term vasodilation could protect against repeated cold injury and some of the degenerative sequelae of frostbite. The value of these benefits is speculative. Epidural spinal cord stimulation combined with conventional treatment may reduce pain and conserve tissue.

A "medical" sympathectomy results from direct injection of an agent such as reserpine into an artery. Common injection sites include the radial, brachial, and femoral arteries. This injection produces local depletion of arterial wall norepinephrine for 2 to 4 weeks. No significant systemic effects are appreciated with the recommended dose of 0.5 mg. This can be repeated in 2 to 3 days. Parenteral reserpine is not commercially available in the United States. Guanethidine is an alternative.

There is angiographic documentation of temporary improvement in perfusion and vasospasm after medical sympathectomy. When tissue is rapidly thawed, experimental attempts to demonstrate further enhancement of tissue salvage have failed. Intra-arterial reserpine may prove most useful in patients with residual pain after gradual thawing.

Early results with surgical sympathectomy were encouraging. Decreased pain, edema, and residual autonomic dysfunction were reported. Tissue demarcation also appeared to accelerate. Bouwman and colleagues[50] performed unilateral surgical sympathectomy on 10 patients with bilateral matched frostbite injuries. Delayed protection against reinjury was one direct benefit. Ultimately, however, there was no increased tissue salvage. Mills observes that surgical sympathectomy produces a smoother initial clinical course but no long-term benefits, with the possible exception of decreased causalgia.[18,19]

DISPOSITION

Except in minor cases, all patients should be hospitalized to determine the extent of injury. Superficial injuries to facial structures may be followed on an outpatient basis. Damaged tissues are best protected with loose sterile sheets and towels rather than compressive dressings. Feet must be kept elevated under a protective cradle. Sterile cotton pledgets should be placed between the toes, and the hands may rest elevated on the chest.

Whirlpool hydrotherapy with an antiseptic should be performed two to three times daily for 20 to 30 minutes. Range-of-motion exercises should be encouraged during immersion. Severe cases may require position-of-function splinting. Hydrotherapy is continued as the eschar sloughs. At this point, sterile precautions may be discontinued. During hospitalization, all vasoconstrictive agents, including nicotine, should be avoided.

SEQUELAE

Direct neuronal damage and residual abnormalities in sympathetic tone are responsible for most of the common symptomatic sequelae of frostbite. In a series of military patients with documented frostbite, 65% had long-term residual symptoms. Vasospasm with secondary cold intolerance is the other major sequela.[51]

Intermittent paresthesias resulting from ischemic neuritis are reported after the first week. The severity of this symptom often reflects the extent of tissue damage. Symptoms may persist for many months. Burning electric shock sensations are worse at night, after heat exposure, and on first returning to ambulation. Thermal perception is also altered. Hyperhidrosis suggests an abnormal sympathetic nervous system response and often serves as both a cause and an effect of frostbite.

Delayed cutaneous findings include nail deformities and pigmentation changes. Squamous and epidermoid cell carcinoma can occur. Osseous reabsorption and subchondral lytic defects develop months after the cold insult. In pediatric patients, concerns include premature fusion, destruction, and fragmentation of epiphyses. Shortening of the distal phalanges is common.[51] Frostbite arthritis also occurs, commonly 3 to 10 years later. Thumb sparing is a characteristic idiosyncrasy. Clenching of the fists can spare both thumbs and metacarpophalangeal joints. Identification of subchondral cysts following bone infarction differentiates frostbite arthritis from osteoarthritis.[52] In severe cases involving extremity muscle compartments, rhabdomyolysis and subsequent renal failure are a concern. Continuous monitoring of serum muscle enzymes and urinalyses is warranted.

BOX 137-4 SEQUELAE OF FROSTBITE

- Neuropathic
  - Pain
    - Phantom
    - Causalgia
    - "Tabes" burning
    - Chronic
  - Sensation
    - Hypesthesia
    - Dysesthesia
    - Paresthesia
    - Anesthesia
  - Thermal sensitivity
    - Heat
    - Cold
  - Autonomic dysfunction
    - Hyperhidrosis
    - Raynaud's syndrome
- Musculoskeletal
  - Atrophy
  - Compartment syndrome
  - Rhabdomyolysis
  - Tenosynovitis
  - Stricture
  - Epiphyseal fusion
  - Osteoarthritis
  - Osteolytic lesions
  - Subchondral cysts
  - Necrosis
  - Amputation
- Dermatologic
  - Edema
  - Lymphedema
  - Chronic or recurrent ulcers
  - Epidermoid or squamous cell carcinoma
  - Hair or nail deformities
- Miscellaneous
  - Core temperature after-drop
  - Acute tubular necrosis
  - Electrolyte fluxes
  - Psychological stress
  - Gangrene
  - Sepsis

Surgical decisions regarding amputation are complex.[51,53] The amount of tissue eventually salvaged often exceeds even optimistic initial estimates. Historically, the natural progression of demarcation, mummification, and eventual sloughing was allowed to occur. Necrotic tissue was classically débrided at least 1 to 2 months after injury when the line of demarcation was clear. Exceptions include refractory pain, sepsis, and supervening gangrene. Advances in radiologic assessment of tissue viability are facilitating earlier surgical intervention. Most grafts and amputations now occur at 3 to 4 weeks after injury. Free flap tissue transfer to salvage function after earlier débridement of soft tissues should be a consideration.[53,54]

Various neuropathic, musculoskeletal, and dermatologic sequelae of frostbite are listed in Box 137-4.[55]

KEY CONCEPTS

- Premature termination of thawing in 40° to 42° C water is a common error. Reperfusion of completely frozen tissue is very painful and will require parenteral analgesia.
- The early formation of clear blebs is more favorable than delayed hemorrhagic blebs, since the latter reflect damage to the subdermal vascular plexi.
- The patient should be advised that accurate prediction of eventual tissue loss is not always possible at presentation, despite imaging.
- Thrombolytic agents may restore some flow to severely frostbitten limbs.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 16 Headache

Christopher S. Russi

PERSPECTIVE

Epidemiology

Up to 85% of the U.S. adult population complains of significant headaches at least occasionally, and 15% does so on a regular basis. Headache as a primary complaint represents between 3 and 5% of all emergency department (ED) visits. The vast majority of patients who have the primary complaint of headache do not have a serious medical cause for the problem. Tension headache accounts for approximately 50% of patients presenting to the ED, another 30% have headache of unidentified origin, 10% have migraine-type pain, and 8% have headache from other potentially serious causes (e.g., tumor, glaucoma). It is estimated that less than 1% of patients who present to the ED with headache have a life-threatening organic disease.[1] The percentages can create a false sense of security, and headache is disproportionately represented in emergency medicine malpractice claims. Although still rare, the most commonly encountered life-threatening cause of severe sudden head pain is subarachnoid hemorrhage (SAH); approximately 20,000 potentially salvageable cases of SAH present to EDs each year. It is estimated that between 25 and 50% of these are missed on the first presentation to a physician.[2] The other significant, potentially life-threatening causes of headache occur even less frequently. Meningitis, carbon monoxide poisoning, temporal arteritis, acute angle-closure glaucoma, intracranial hemorrhage (ICH), cerebral venous sinus thrombosis, and increased intracranial pressure can often be linked with specific historical elements and physical findings that facilitate their diagnosis.

Pathophysiology

The brain parenchyma is insensitive to pain. The pain-sensitive areas of the head include the coverings of the brain—the meninges—and the blood vessels, both arteries and veins supplying the brain, and the various tissues lining the cavities within the skull. The ability of the patient to specifically localize head pain is often poor. Much of the pain associated with headache, particularly with vascular headache and migraines, is mediated through the fifth cranial nerve. Such pain may proceed back to the nucleus and then be radiated through various branches of the fifth cranial nerve to areas not directly involved. A specific inflammation in a specific structure (e.g., periapical abscess, sinusitis, or tic douloureux) is much easier to localize than the relatively diffuse pain that may be generated by tension or traction headaches. Pains in the head and neck may easily overlap. They should be thought of as a unit when considering complaints of headache.

DIAGNOSTIC APPROACH

Differential Considerations

The differential diagnosis of headache is complex because of the large number of potential disease entities and the diffuse nature of many types of pain in the head and neck region (Table 16-1). However, in evaluating the patient with a headache complaint, the top priority is to exclude intracranial hemorrhage (SAH and ICH), meningitis, encephalitis, and mass lesions. Carbon monoxide is an exogenous toxin, the effects of which may be reversible by removing the patient from the source and administering oxygen. Carbon monoxide poisoning is a rare example of a headache in which a simple intervention may quickly improve a critical situation. On the contrary, returning the patient to the poisoned environment without a diagnosis could be lethal.

Rapid Assessment and Stabilization

If the patient presents in a critical or comatose state, initial stabilization, including airway management, is undertaken as indicated, preceded by a neurologic examination if at all possible. For purposes of the initial assessment, headache can be divided into two categories: accompanied by altered mental status and without altered mental status. Whenever a patient's mental status is impaired, brain tissue is initially assumed to be compromised. The principles of care centered on cerebral resuscitation address the seven major causes of evolving brain injury: lack of substrate (glucose, oxygen), cerebral edema, intracranial mass lesion, endogenous or exogenous toxins, metabolic alterations (fever, seizure), ischemia, or elevated intracranial pressure.

Pivotal Findings

History

The history is the pivotal part of the workup for the patient with headache (Table 16-2).

1. The patient should be asked to describe the *pattern and onset of the pain*. Patients often relate frequent and recurrent headaches similar to the one they have

Table 16-1 Differential Diagnosis

ORGAN SYSTEM	CRITICAL DIAGNOSES	EMERGENT DIAGNOSES	NONEMERGENT DIAGNOSES
Neurologic, CNS, vessels	Subarachnoid hemorrhage	Shunt failure Traction headaches Tumor/other masses Subdural hematomas	Migraine, various types Vascular, various types Trigeminal neuralgia Post-traumatic Postlumbar puncture Headaches
Toxic/metabolic Environmental	Carbon monoxide poisoning	Mountain sickness	
Collagen vascular disease	Temporal arteritis		
Eye/ENT		Glaucoma/sinusitis	Dental problems/temporomandibular joint disease
Musculoskeletal			Tension headaches Cervical strain
Allergy			Cluster/histamine headaches
Infectious disease	Bacterial meningitis/encephalitis	Brain abscess	Febrile headaches/nonneurologic source of infection
Pulmonary/O_2		Anoxic headache Anemia	
Cardiovascular		Hypertensive crisis	Hypertension (rare)
Unspecified			Effort-dependent/coital headaches

CNS, central nervous system; ENT, ear, nose, and throat.

Table 16-2 Significant Symptoms

SYMPTOM	FINDING	POSSIBLE DIAGNOSES
Sudden onset of pain	Lightning strike or thunder clap with any decreased mentation, any positive focal finding or intractable pain	Subarachnoid hemorrhage
"Worst headache of their life"	Associated with sudden onset	Subarachnoid hemorrhage
Near syncope or syncope	Associated with sudden onset	Subarachnoid hemorrhage
Increase with jaw movement	Clicking or snapping. Pain with jaw movement	Temporomandibular joint disease
Facial pain	Fulminant pain of the forehead and area of maxillary sinus. Nasal congestion	Sinus pressure or dental infection
Forehead or temporal area pain (or both)	Tender temporal arteries	Temporal arteritis
Periorbital or retro-orbital pain	Sudden onset with tearing	Temporal arteritis or acute angle-closure glaucoma

on this ED visit. A marked variation in headache pattern can signal a new or serious problem. The rate of onset of pain may have significance. Pain with rapid onset of a few seconds to minutes is more likely to be vascular in origin than pain that developed over several hours or days.

Almost all studies dealing with subarachnoid bleeding report that patients moved from the pain-free state to severe pain within seconds to minutes. The "thunder clap" or "lightning strike" headache is a real phenomenon, and this response to questioning may lead to the correct diagnosis of subarachnoid hemorrhage, even if the pain is improving at the time of evaluation.[3]

2. The patient's *activity at the onset of the pain* may be helpful. Certainly, headaches that come on during severe exertion have a relationship to vascular bleeding, but again, there is enough variation to make assignment to any specific cause highly variable. The syndrome of coital or postcoital headache is well known, but coitus is also a common time of onset for SAH. These headaches require the same evaluation on initial presentation as any other exertion-related head pain. If the patient can recall the precise activity in which he or she was engaging at the time of the onset of the headache (e.g., "I was just getting up out of the chair to answer the doorbell"), sudden onset is extremely likely and evaluation for SAH is warranted.
3. If the patient or nonhospital medical personnel can relate a *history of head trauma*, the differential diagnosis and emergent causes have narrowed significantly. The considerations now focus on epidural and subdural hematoma, traumatic SAH, skull fracture, and closed-head injury (i.e., concussion and diffuse axonal injury).
4. Toxoplasmosis, cryptococcal meningitis, and abscess are considered higher in the differential in patients with a *history of HIV or immunocompromised state*. Although such entities are rare, it is important to remember that this subset of patients may have serious disease without typical signs or symptoms of systemic illness (e.g., fever and meningismus).
5. The *intensity of head pain* is difficult to quantify objectively. Almost all patients who present to the ED consider their headache to be "severe." Use of a pain scale of 1 to 10 may help differentiate patients initially but has more value in monitoring their response to therapy.

6. The *character of the pain* (i.e., throbbing, steady), although sometimes helpful, may not be adequate to differentiate one type of headache from another.
7. The *location of head pain* is helpful when the patient can identify a specific area. It is useful to have the patient point or try to indicate the area of pain and the emergency physician to then properly examine that area. Unilateral pain is more suggestive of migraine or a localized inflammatory process in the skull (e.g., sinus) or soft tissue.[4] Occipital headaches are classically associated with hypertension. Certainly, temporal arteritis, temporomandibular joint disease, dental infections, and sinus infections frequently have a highly localized area of discomfort. Meningitis, encephalitis, SAH, and even severe migraine, although intense in nature, are usually more diffuse in their localization.
8. *Exacerbating* or *alleviating factors* may be important. Patients whose headaches rapidly improve when they are removed from their environment may have carbon monoxide poisoning. Most other severe causes of head pain are not rapidly relieved or improved when patients get to the ED. Headaches on awakening are typically described with brain tumors. Intracranial infections, dental infections, and other regional causes of head pain tend not to be improved or alleviated before therapy is given.
9. *Associated symptoms and risk factors* may relate to the severity of headache but rarely point to the specific causes (Box 16-1). Nausea and vomiting are completely nonspecific. Migraine headaches, increased intracranial pressure, temporal arteritis, and glaucoma can all manifest with severe nausea and vomiting, as can some systemic viral infections with headache. Such factors may point toward the intensity of the discomfort but are not specific in establishing the diagnosis.
10. A *prior history of headache*, although helpful, does not rule out current serious problems. It is extremely helpful, however, to know that the patient has had a workup for severe disease. Previous ED visits, computed tomography (CT) magnetic resonance imaging, and other forms of testing should be inquired about. Patients with both migraine and tension headaches tend to have a stereotypical recurrent pattern. Adherence to these patterns is also helpful in deciding the degree to which a patient's symptoms are pursued.

Physical Examination

Physical findings associated with various forms of headache are listed in Table 16-3.

Ancillary Testing

The vast majority of headache patients do not require additional testing (Table 16-4). The single largest consistent mistake made by emergency physicians in the workup of the headache patient is believing a single CT scan clears the patient of the possibility of SAH or other serious intracranial disease. The CT scan can miss 6 to 8% of patients with SAH, especially in patients with minor (grade I) SAH, who are most treatable.[5] The sensitivity of CT for identifying SAH is reduced by nearly 10% for symptom onset greater than 12 hours and by almost 20% at 3 to 5 days. The basic approach to integrating CTs and lumbar puncture in the assessment of headache is outlined in Figure 16-1.[6,8,9]

BOX 16-1 RISK FACTORS ASSOCIATED WITH POTENTIALLY CATASTROPHIC ILLNESS

1. Carbon monoxide poisoning
   a. Breathing in enclosed or confined spaces with engine exhaust or ventilation of heating equipment
   b. Multiple family members with the same symptoms
   c. Pattern of recurrence in one setting (where the exposure is occurring), relief when not in that setting
   d. Wintertime and working around machinery or equipment producing carbon monoxide (furnaces, etc.)
2. Meningitis/encephalitis/abscess
   a. History of sinus or ear infection or recent surgical procedure
   b. Immunocompromised state
   c. General debilitation with decreased immunologic system function
   d. Acute febrile illness—any type
   e. Extremes of age
   f. Impacted living conditions (e.g., military barracks, college dormitories)
   g. Lack of primary immunizations
3. Temporal arteritis
   a. Age > 50
   b. Females > males 4:1
   c. History of other collagen vascular diseases (e.g., systemic lupus)
   d. Previous chronic meningitis
   e. Previous chronic illness such as tuberculosis, parasitic infection, fungi
4. Glaucoma—sudden angle-closure
   a. Not associated with any usual or customary headache pattern
   b. History of previous glaucoma
   c. Age >30
   d. History of pain increasing in a dark environment
5. Increased intracranial pressure
   a. History of previous benign intracranial hypertension
   b. Presence of a cerebrospinal fluid shunt
   c. History of congenital brain or skull abnormalities
6. Cerebral venous sinus thrombosis
7. Intracranial hemorrhage (ICH)
   a. Subarachnoid hemorrhage (SAH)
      i. Sudden severe pain. "Worst headache of life."
      ii. Acute severe pain following sexual intercourse or straining (i.e., heavy lifting)
      iii. History of SAH or cerebral aneurysm
      iv. History of polycystic kidney disease
      v. Family history of subarachnoid hemorrhage
      vi. Hypertension—severe
      vii. Previous vascular lesions in other areas of the body
      viii. Young and middle-aged
   b. Subdural hematoma (SDH)
      i. History of alcohol dependency with or without trauma
      ii. Current use of anticoagulants
   c. Epidural hematoma (EDH)
      i. Traumatic injury
      ii. Lucid mentation followed by acute altered mentation or somnolence
      iii. Anisocoria on physical examination

Table 16-3 Pivotal Findings on Physical Examination

SIGN	FINDING	POSSIBLE DIAGNOSES
General appearance	Alteration of mental status—nonfocal	Meningitis/encephalitis Subarachnoid hemorrhage Anoxia Increased CSF pressure
	Alterations of mental status with focal findings	Intraparenchymal bleed Tentorial herniation Stroke
	Severe nausea/vomiting	Increased CSF pressure Acute angle-closure glaucoma Subarachnoid hemorrhage
Vital signs	Hypertension with normal heart rate or bradycardia	Increased CSF pressure Subarachnoid hemorrhage Tentorial herniation Intraparenchymal bleed
	Tachycardia	Anoxia/anemia Febrile headache Exertional/coital headaches
	Fever	Febrile headaches Meningitis/encephalitis
HEENT	Tender temporal arteries	Temporal arteritis
Fundi—loss of spontaneous venous pulsations or presence of papilledema	Increased CSF pressure	Mass lesions
	Subhyaloid hemorrhage	Subarachnoid hemorrhage
	Acute red eye (severe ciliary flushing) and poorly reactive pupils	Acute angle-closure glaucoma
	Enlarged pupil with third nerve palsy	Tentorial pressure cone Mass effect (i.e., subdural, epidural, tumor, intraparenchymal hemorrhage)
Neurologic	Lateralized motor or sensory deficit	Stroke (rare) Subdural hematoma, epidural hematoma, hemiplegic or anesthetic migraine (rare)
	Acute cerebellar ataxia	Acute cerebellar hemorrhage Acute cerebellitis (mostly children) Chemical intoxication—various types

CSF, cerebrospinal fluid; HEENT, head, eyes, ears, nose, and throat.

Table 16-4 Diagnostic Adjuncts in Headache Assessment

TEST	FINDING	DIAGNOSIS
Erythrocyte sedimentation rate (ESR)	Significant elevation	Temporal arteritis
ECG	Nonspecific ST-T wave changes	Subarachnoid hemorrhage Increased CSF pressure
CBC	Severe anemia	Anoxia
CT—head	Increased ventricular size	Increased CSF pressure
	Blood in subarachnoid space	Subarachnoid hemorrhage
	Blood in epidural or subdural space	Epidural/subdural hematoma
	Bleeding into parenchyma of brain	Intraparenchymal hemorrhage
	Areas of poor vascular flow	Pale infarct
	Structural/mass lesion	Traction headache secondary to mass effect
Lumbar puncture/CSF analysis	Increased pressure	Pseudotumor cerebri Mass lesions Shunt failure
	Increased protein	Tumor/other structural lesions
	Increased RBCs	Subarachnoid hemorrhage
	Increased WBCs	Infection
	Positive Gram's stain	Infection
	Decreased glucose	Infection

CBC, complete blood count; CSF, cerebrospinal fluid; CT, computed tomography; ECG, electrocardiogram; RBC, red blood cell; WBC, white blood cell.

Table 16-5 Causes and Differentiation of Potentially Catastrophic Illness Presenting with Nontraumatic Headache

DISEASE ENTITIES	PAIN HISTORY	ASSOCIATED SYMPTOMS	SUPPORT HISTORY	PREVALENCE	PHYSICAL EXAMINATION	USEFUL TESTS	ATYPICAL OR IMPORTANT ASPECTS
Carbon monoxide poisoning	Usually gradual, subtle, dull, nonfocal throbbing pain	May wax and wane as they leave and enter the involved area of carbon monoxide. Throbbing may vary considerably	Exposure to engine exhaust, old or defective heating systems, most common in winter months	Rare	No focal neurologic findings. May need cognitive testing	Carbon monoxide level, cognitive testing	May improve on the way to the hospital. Occurs in groups, may involve entire families or groups of people exposed to the carbon monoxide
Subarachnoid hemorrhage	Sudden onset, "thunder clap" or "lightning strike," severe throbbing	Whenever altered mental status is present, the outcome is decidedly worse	History of polycystic kidney disease. History of chronic hypertension	Uncommon	Frequently decreased mentation—meningismus, increased blood pressure, decreased pulse, decreased spontaneous venous pulsations, rarely subhyaloid hemorrhage	CT. Lumbar puncture	If CT positive, immediate involvement of neurosurgery. If CT negative, lumbar puncture
Meningitis/ encephalitis/ abscess	Gradual—as general symptoms increase, headache increases—nonfocal	Decreased mentation prominent, irritability prominent. With abscess, focal neurologic findings may be present	Recent infection Recent facial or dental surgery or other ENT surgery	Uncommon	Fever—late in course, decreased spontaneous venous pulsations	CT. Lumbar puncture	When such infection suspected, treat. Do not delay antibiotics and steroids awaiting laboratory results
Temporal arteritis	Often pain developing over a few hours from mild to severe. Virtually always focal in nature	Decreased vision, nausea, vomiting intense—may confuse diagnosis	Age over 50. Other collagen vascular diseases or inflammatory diseases	Uncommon	Tender temporal arteries	Sedimentation rate	Usually unrelated and rapidly progressive
Acute angle-closure glaucoma	Sudden in onset	Nausea, vomiting, decreased vision	History of glaucoma. History of pain going into dark area	Rare	"Steamy" cornea. Midposition pupil poorly reactive. Acute red eye	Measurement of intraocular pressure	Rapid intervention with medications required—if no relief, immediate surgery may be required
Increased intracranial pressure syndromes	Gradual, dull, nonfocal	Vomiting, decreased mentation	History of CSF shunt or other congenital brain or skull abnormality	Uncommon	Papilledema. Loss of spontaneous venous pulsations	CT. Shunt function study. If OK, lumbar puncture	Shunt failure or other cause of significant increased CSF pressure requires involvement of neurosurgery

CSF, cerebrospinal fluid; CT, computed tomography; ENT, ear, nose, and throat.

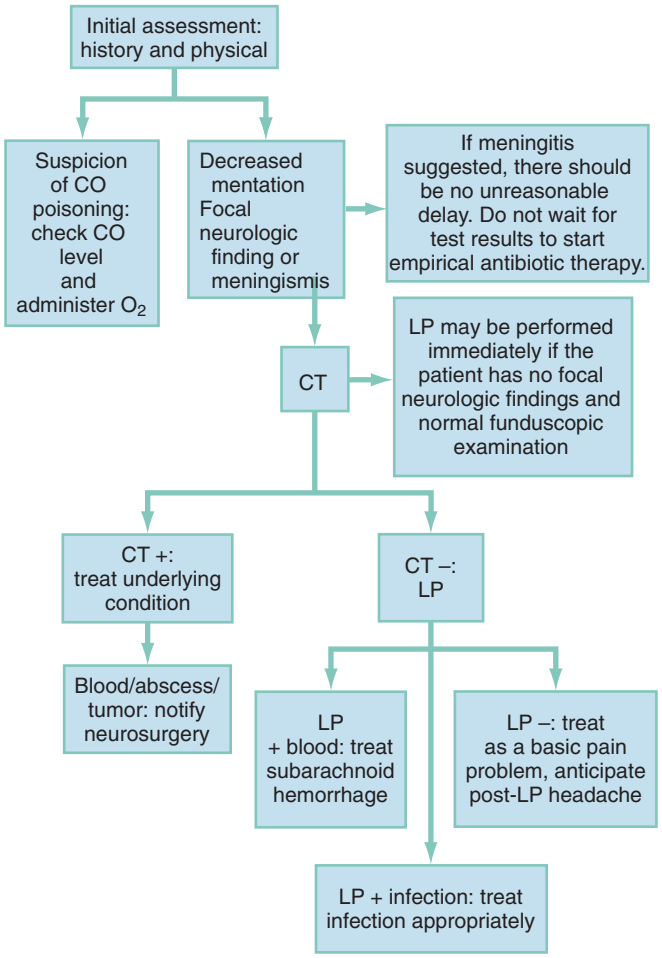


Figure 16-1. Initial assessment and treatment of headache. CO, carbon monoxide; CT, computed tomography; LP, lumbar puncture.

Obtaining cerebrospinal fluid should not delay antimicrobial treatment if intracranial infection is suggested. Intravenous antibiotics should precede lumbar puncture. Abnormal mental status, signs of increased intracranial pressure, papilledema focal findings on the neurologic examination, or any other indication suggestive of focal intracranial lesion requires CT before lumbar puncture.

DIFFERENTIAL CONSIDERATIONS

Certain historical and physical findings can help the emergency physician decide whether the patient falls into an "all clear" or a "warning signal" group. In the warning group, further investigation and testing should be performed on all patients who present with any of the following: (1) sudden onset of headache, (2) "the worst headache ever," (3) decreased or altered mental status, (4) true meningismus, (5) unexplained fever or bradycardia, (6) focal neurologic deficits on examination, (7) symptoms refractory to treatment or worsening under observation, (8) new onset of headache with exertion, or (9) history of HIV. These patients have the highest risk for significant disease.

In addition, a group of reliable "all clear signals" indicates patients who do not require further investigation when *all* are present: (1) previous identical headaches, (2) normal alertness and cognition by both examination and history of the event, (3) normal examination of the neck showing no meningismus, (4) normal vital signs, (5) normal or nonfocal neurologic examination, and (6) improvement under observation or with treatment.

Sequential evaluation and assessment of data are ongoing processes. Patients should be reevaluated while in the ED, and inconsistent findings may require a rapid review of the situation and rethinking of the diagnosis (Table 16-5).[7]

MANAGEMENT

Empirical

Patients with headache represent a spectrum of disease. Patients with headache need to receive triage for evaluation according to their symptoms. Clearly, patients with abnormal vital signs or altered mental status require evaluation before patients with less severe symptoms. If history and physical examination point toward potentially lethal causes, however, effort should be made to establish the diagnosis rapidly with ancillary testing. Pain treatment should be started early. The pain medication of choice depends on the particular patient, underlying vital signs, allergies, and general condition; but relief of pain is still an essential part of the physician's job and should have little effect on the diagnostic workup.

Specific

Specific management for headache is described in Chapter 101. The challenge in emergency medicine, however, is to eliminate life-threatening causes of headache and to treat the patient's pain.

DISPOSITION

Most patients presenting with headache are discharged from the ED with appropriate analgesia and follow-up. These represent patients in the all-clear category or those found to have no serious disease after a careful evaluation and testing. Any patients in whom warning findings are noted require more extensive assessment.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 51

Musculoskeletal Back Pain

Michelle Lin

LOW BACK PAIN

PERSPECTIVE

Background

Approximately 70 to 90% of adults during their lifetime experience *acute low back pain*, defined as pain lasting less than 6 weeks in duration.[1,2] The etiology for the back pain remains unknown in 85% of affected persons after appropriate initial investigation—a situation frustrating for both physician and patient.[3] Frequent diagnoses in such cases include "acute lumbosacral strain," "lumbago," and "mechanical back pain." These nonspecific, catch-all terms reflect the diagnostic challenge and lack of pathognomonic tests for low back pain. More accurately, these patients should be diagnosed with *idiopathic low back pain*. Regardless, most cases resolve spontaneously within 6 weeks. In recent studies on the management of acute low back pain, the most significant discovery—contradictory to traditional teaching from the 1980s—is the need to avoid bedrest for these patients, including those with sciatica symptoms.[4,5] Management recommendations for chronic back pain remain controversial, however, and the condition accounts for a significant proportion of costs to the health care system.

Epidemiology

In the ambulatory care setting, the medical complaint of back pain is the fifth most common reason for a visit to a physician's office, with 15 million annual visits in the United States.[6] Low back pain primarily affects adults 30 to 60 years of age and has a tremendous impact on worker productivity, with significant economic consequences. In people younger than 45 years of age, persistent chronic back pain ranks as the leading cause of disability among chronic ailments. Among people 45 to 64 years of age, it ranks third, behind coronary disease and arthritis.[7] It also is the second leading cause of pain resulting in lost productive time from work, following headache.[8] Overall, patients with back pain account for billions of dollars in total direct and indirect costs in the United States.[1,2,9]

The natural history of most cases of low back pain follows a benign and self-limited course. In a large pooled analysis, back pain decreases by 58% within 1 month. If the pain does not resolve within 3 months, however, it is unlikely to resolve after 12 months. The recurrence rate for pain is 66 to 84% within the first 12 months.[10]

Risk factors for low back pain are continually being investigated. Data obtained so far have been inconclusive and often contradictory.[11,12] It seems that the most consistent risk factor for future back problems is a history of previous back problems. Heavy lifting, pushing and pulling, or vibration at work; poor job satisfaction; smoking; and family history all seem to predispose to future back problems, whether causal or merely linked.[1,9,13-15] Early evidence also points to a genetic predisposition to lumbar disk disease.[16] Multiple other factors have been investigated, including body habitus, various occupations, and psychological profile, with conflicting results.[11,17,18]

PRINCIPLES OF DISEASE

Anatomy and Physiology

The lumbosacral spine consists of five lumbar vertebrae and the sacrum. Moving from anterior to posterior, each vertebra can be divided into the cylindrical vertebral body, two pedicles, two transverse processes, two overarching laminae, and a spinous process. These structures surround the neural canal, which houses the spinal cord and nerve roots and has a midsagittal anteroposterior diameter of 15 to 23 mm. The paired superior and inferior articulating processes join the articulating processes one vertebral level above and below. Each articulation site is called a *facet joint*. An intervertebral disk interposes between each vertebral body, providing elasticity and stability to the vertebral column. Each disk consists of an inner colloidal gelatinous substance, the *nucleus pulposus*, and an outer capsule, the *annulus fibrosus*, which is thinner posteriorly than anteriorly (Fig. 51-1).

Various ligaments and muscles also provide stability to the lumbosacral spine. The anterior and posterior longitudinal ligaments course along the anterior and posterior surfaces of the vertebral bodies. The posterior longitudinal ligament forms a border between the intervertebral disks and the neural canal. As expected, because this ligament thins as it runs inferiorly from L1 to S1, 95% of lumbar disk herniations occur at the L4–5 and L5–S1 levels, causing pain and neurologic deficit in the L5 to S1 distribution. Most herniations extrude posterolaterally to impinge a nerve root asymmetrically.[19] The ligamentum flavum courses just anterior to the laminae within the neural canal. With age, this ligament can thicken, potentially causing spinal stenosis.

The spinal cord ends at the L1–2 interspace, and the lower cauda equina nerve roots extend inferiorly, exiting the sacral

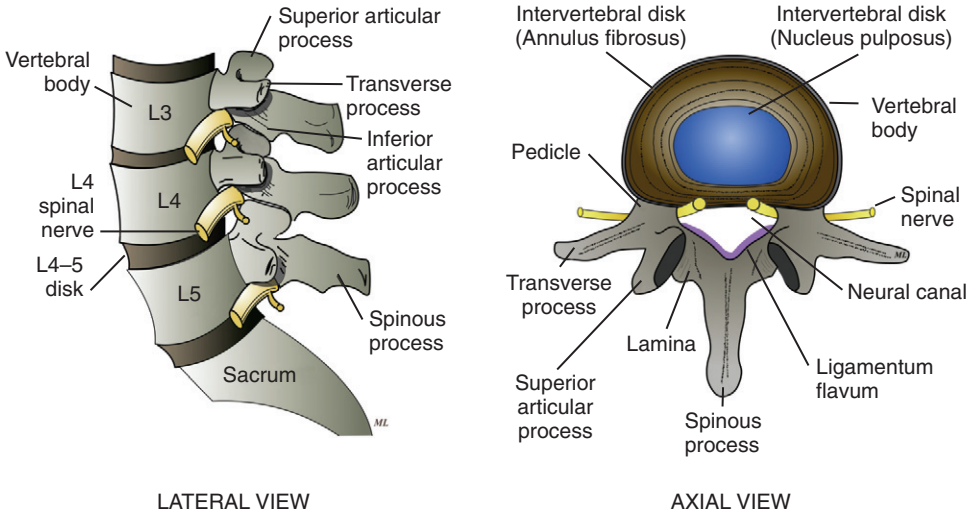


Figure 51-1. Lateral and axial views of lumbar vertebral anatomy.

foramina as peripheral nerves to the lower extremity. Pain fibers supplying structures in the lumbosacral region primarily arise from the posterior rami and sinuvertebral nerves at each lumbar vertebral level. The nucleus pulposus and inner annular fibers of the intervertebral disk are unique in their lack of any pain fibers.[20] This anatomic feature correlates consistently with magnetic resonance imaging (MRI) evidence of disk pathology in 28 to 33% of asymptomatic patients. The significance of these findings is unclear.[9,20,21]

Pathophysiology

Most conditions of low back pain have no proven cause. It is estimated that 85% of patients have no definitive diagnosis and are presumed to have pain originating from the soft tissue, including the muscles and ligaments.[9,19] In the other 15% of patients with a known etiology, the pain may originate in the (1) nerve root, (2) articular facets, or (3) bone itself.

In low back pain of *nerve root origin*, a spinal nerve root can become inflamed and painful with external impingement. Disk herniation, usually at the L4–5 and L5–S1 levels, is the most common cause of *sciatica* (i.e., pain radiating down the posterior leg from sciatic nerve root irritation). As the disk starts to desiccate and degenerate, starting in their 30s, patients are at increased risk for outward herniation of the nucleus pulposus with consequent nerve root impingement. With further aging, these disks progressively shrink in size. In keeping with these findings, disk herniations typically are found in patients 30 to 50 years of age. Local nerve ischemia from physical compression also may contribute to the inflammation and pain. Studies also show that exposure of the nucleus pulposus during disk herniation may result in local neural inflammation, leading to pain.[22] Nerve root impingement also can occur with spinal stenosis, a narrowing of the neural canal from congenital narrowing or, more often, from degenerative or hypertrophic changes of the disks, vertebrae, facet joints, and ligamentum flavum.[23]

The two most critical conditions causing nerve irritation are cauda equina syndrome and a spinal epidural abscess. *Cauda equina syndrome* is most commonly due to a massive central disk herniation usually compressing multiple, bilateral nerve roots, causing back pain radiating to both legs, saddle anesthesia, and impaired bowel and bladder function. Emergent surgical decompression is indicated to preserve neurologic function. An *epidural abscess* similarly results in nerve root impingement, causing significant back pain. These rare infections develop most commonly from the hematogenous spread of *Staphylococcus aureus*.[19]

Congenital and developmental spinal abnormalities also may cause back pain by nerve root inflammation in some cases, but much less frequently than was thought previously. Conditions such as kyphosis and scoliosis usually do not cause pain unless the degree of vertebral column misalignment is pronounced.[24] Similarly, spondylolisthesis (slippage of one vertebral body on another) does not usually cause pain if the slippage is less than 25% of the vertebral body depth. Even in patients with higher grades of anterior slippage (anterolisthesis), the development of severe back pain is rare. Although it is one third as common as anterolisthesis, backward slippage (retrolisthesis) is almost always associated with back pain. Spondylolisthesis occurs most often at the L5–S1 level (82.1%), followed by the L4–5 level (11.3%) and the L3–4 level (0.5%).[25] Multifactorial causes of spondylolisthesis include degenerative changes and trauma. Spondylolisthesis often is associated with bilateral pars interarticularis defects in the affected vertebra (spondylolysis).

In low back pain of *articular facet origin*, as with any other joint in the body, degenerative changes in the synovial articular facets in the lumbosacral spine occur with age. Although the exact role and significance of articular facet joints in back pain are unclear, facet pathology for it has been suggested to contribute to 15 to 45% of chronic back pain cases.[26]

In low back pain of *bone origin*, direct irritation of the vertebral bone and its periosteum can cause back pain. The causes for spondylitis (osteomyelitis of the axial skeleton) can range from a slowly progressing tuberculosis infection (Pott's disease) to a more acute bacterial infection. Typically, bacteria seed the bone from a hematogenous source, such as from a skin wound or urinary tract infection, or directly from intravenous drug use. The most common bacterial culprit is *S. aureus*. Primary and metastatic bone neoplasms can cause back pain from tumor infiltration into the bone. Primary bone tumors, such as multiple myeloma, chordoma, Ewing's sarcoma, and osteosarcoma, are 25 times less frequent than metastatic disease.[27] Of the neoplasms, breast, lung, prostate, thyroid, and kidney cancers and lymphoma are the most likely to metastasize to bone. Inflammatory conditions, such as ankylosing spondylitis and other arthropathies, and osteoporosis also can cause back pain. In osteoporosis, the generalized decrease in bone mineralization can cause pain from microfractures of the vertebral column.

Referred pain, most commonly from intraperitoneal and retroperitoneal abdominal pathology, also must be considered in patients with back pain. Functional processes play a substantial role in back pain, especially for prolonged symptoms lasting more than 4 to 6 weeks. Functional causes range from fear, depression, and personality disorders to financial motivation. In such cases, no anatomic or pathophysiologic correlation with the reported pain can be found.

Chronic back pain is complex and multifactorial. Not only are the structural causes unclear, but the nonphysical factors are variable and difficult to determine. It is likely that many of these patients do have some kind of chronic pain. What is unknown is why chronic pain triggers depression, drug dependence, and malingering in some people but not in others. One difference may be the degree of disruption that the condition causes in the patient's lifestyle. A normally active and athletic person who is incapacitated is more profoundly affected than someone who is habitually sedentary. Psychological factors and potential compensation play a large role in the behavior of many patients with chronic back pain.[28,29]

Pediatric back pain results in a diagnosis more often than adult pain.[30,31] Children complaining of back pain require appropriate investigation. They may turn out to have spondylolysis with a variable degree of spondylolisthesis, Scheuermann's disease (kyphosis and osteochondritis of the vertebral endplates), an infectious disease, or a neoplastic process. Disk herniation in children is comparatively rare, but when it does occur, the presentation is similar to that in adults.[30]

CLINICAL FEATURES

Signs and Symptoms

A thorough history and physical examination are crucial in evaluating all patients with acute low back pain. The classic historical (Table 51-1) and physical findings with various entities associated with low back pain are reviewed in this section. Although most of these etiologic disorders are benign, four such disorders have been identified by the Agency for Health Care Policy and Research as "cannot miss" or "red flag" diagnoses: spinal fracture, cauda equina syndrome, spinal infection, and malignancy.[32] A methodical and focused approach to the history and physical examination can help assess the patient's pretest probability for each of these disease entities and determine whether further tests should be ordered.

Table 51-1 Historical Clues for the Etiology of Low Back Pain

QUESTIONS FOR PATIENT	POTENTIAL DIAGNOSIS
Does the back pain radiate down past the knees?	Radiculopathy and likely a herniated disk
Is the pain worse with walking and better with bending forward and sitting?	Spinal stenosis
Do you have morning back stiffness that improves with exercise?	Ankylosing spondylitis
Are you older than 50 years of age?	Osteoporotic fracture, spinal malignancy
Has there been any recent history of blunt trauma?	Fracture
Do you take long-term corticosteroids?	Fracture, spinal infection
Do you have a history of cancer?	Spinal metastatic malignancy
Does your pain persist at rest?	Spinal malignancy, spinal infection
Has there been persistent pain for longer than 6 weeks?	Spinal malignancy
Has there been unexplained weight loss?	Spinal malignancy
Is the pain worse at night?	Spinal malignancy, spinal infection
Are you immunocompromised (e.g., HIV infection, alcoholism, diabetes)?	Spinal infection
Have you had fevers or chills?	Spinal infection
Do you have pain, weakness, or numbness in both legs?	Cauda equina syndrome
Do you have bladder or bowel control problems?	Cauda equina syndrome

HIV, human immunodeficiency virus.

Uncomplicated Musculoskeletal Back Pain

Most patients with back pain can be classified in the category of those with uncomplicated musculoskeletal back pain, after exclusion of worrisome disease processes. Often, patients are unable to recall an inciting incident. The pain usually is characterized as an "ache" or "spasm" and is localized asymmetrically in the lumbar paraspinous muscle, with radiation to the buttock or posterior thigh proximal to the knee. Movement exacerbates the pain, and rest relieves it. No associated deficit in sensation, strength, or bowel or bladder sphincter tone is identified in the history or on the clinical examination. The sole physical findings may be regional lumbosacral tenderness and a limited range of motion of the lower back. This diagnosis of exclusion is made only after ruling out the more worrisome causes of back pain.

Radiculopathy

Approximately 1% of all patients with low back pain exhibit signs of lumbar radiculopathy (i.e., nerve root irritation).[33] The most common etiologic process is herniation of a lumbar disk; other causes include spinal stenosis, malignancy, and infection. The most common type of lumbar radiculopathy is sciatica—an L5 or S1 radiculopathy. Patients with sciatica describe their pain as radiating from the low back to the legs, distal to the knee. Such pain is characterized as "shooting," "lancing," "sharp," or "burning." Associated symptoms include focal numbness or weakness in one of the lower extremities. Exacerbating triggers include sitting, bending, coughing, and straining; relieving factors include lying supine and still.

On physical examination, the patient frequently exhibits tenderness to palpation in the sciatic notch. The *straight leg raise* (SLR) *test* is a fairly sensitive assessment tool to determine if the patient has sciatica. The SLR test is done with the patient supine and the legs extended. The symptomatic leg is passively raised while keeping the knee straight. The presence of back pain, which radiates past the knee when the leg is elevated 30 to 70 degrees, suggests an L5 or S1 radiculopathy. If the SLR test results in isolated low back pain without radiation symptoms to the legs, however, it is considered to be a *negative* finding. Through pooled analysis, the SLR test has a sensitivity of 91% but a low specificity of 26%, meaning that a negative result is fairly accurate in ruling out sciatica.[34] Corroborative tests for sciatica include the "bowstring sign" (reproduction of pain with deep palpation of the taut "bowstring" posterior tibial nerve in the midline popliteal fossa) and

reproduction of pain with foot dorsiflexion when the leg is elevated just short of the pain threshold during the SLR test. As an alternative to the SLR test, with the patient in a seated position, the knee can be extended ("flip test"), which also should stretch the sciatic nerve. Reproduction of the pain often causes the patient to lean backward reflexively from the pain, almost "flipping" back into a supine position.

A *crossed SLR test* is done by passively raising the patient's asymptomatic leg while keeping the knee straight. The presence of pain radiating from the back to the opposite affected leg has a sensitivity of only 29% but a high specificity of 88% for sciatica, meaning that a positive result on crossed SLR testing is almost pathognomonic for sciatica, whereas a negative result is nondiagnostic.[34]

A *reverse SLR test* is performed to detect L3 or L4 radiculopathy. With the patient prone, each hip is passively extended. If there is irritation of the L3 or L4 nerve root, pain is elicited.

In addition to stressing lumbar nerve roots, a thorough examination of the lower extremities detects subtle abnormalities associated with radiculopathies. This examination includes mapping the distribution of pain and assessing individual nerve root function, specifically strength, sensation, and reflexes. For the sensory examination, the earliest deficit can be detected by examining the most distal aspect of the dermatome. Specifically, light touch and pinprick sensation should be tested on the medial foot (L4), in the area between the great and second toes (L5), and on the lateral foot (S1) (Fig. 51-2).

Herniated Disk

Patients with herniated lumbar disks usually are 30 to 50 years of age and often have a long history of recurrent nonradicular low back pain, theoretically from irritation of the outer annular fibers of the disk. When the nucleus pulposus of the disk prolapses through the annulus fibrosus, local nerve root inflammation and radiculopathy result. Coughing, sitting, and any movement in general exacerbate the patient's pain and radiculopathy symptoms. The severity of leg pain from radiculopathy often overshadows the back pain. Sciatica findings have a sensitivity of 95% for lumbar disk herniation, meaning that herniation is extremely unlikely in the absence of sciatica.

The physical examination should focus on lower extremity neurologic function and signs of radiculopathy. Weakness of ankle dorsiflexion, great toe extension, ankle plantar flexion, and knee extension is associated with respective specificities of 70%, 70%, 95%, and 99% for lumbar disk herniation.[19]

Spinal Stenosis

Patients with spinal stenosis are typically older (mean age of 55 years) and constitute only 3% of all patients with low back pain.[3,35] The classic history, identified in 60 to 75% of patients with spinal stenosis, is one of subacute or chronic pain and lower extremity radiculopathy that occurs with walking and is relieved with rest and, uniquely, bending forward at the waist.[36] Because these symptoms mimic peripheral vascular claudication symptoms, pain from spinal stenosis is termed *pseudoclaudication*. Typically, vascular claudication lasts 5 minutes after resting, whereas pseudoclaudication lasts 10 to 15 minutes. Patients with spinal stenosis obtain symptom relief with spine flexion and bending forward because these maneuvers increase spinal canal diameter and reduce spinal cord tension. Similarly, sitting also helps to relieve the symptoms in these patients, in contrast with patients with herniated disks. A typical history describes walking uphill without pain but experiencing pain on walking downhill, when the back is extended.

On physical examination, most patients are found to have a lumbar radiculopathy at one or multiple levels and increased back pain with extension.[36] Classically, patients with spinal stenosis walk with a slightly bent-forward position. To help differentiate spinal stenosis from vascular claudication, peripheral pedal pulses and ankle-brachial indices should be checked.

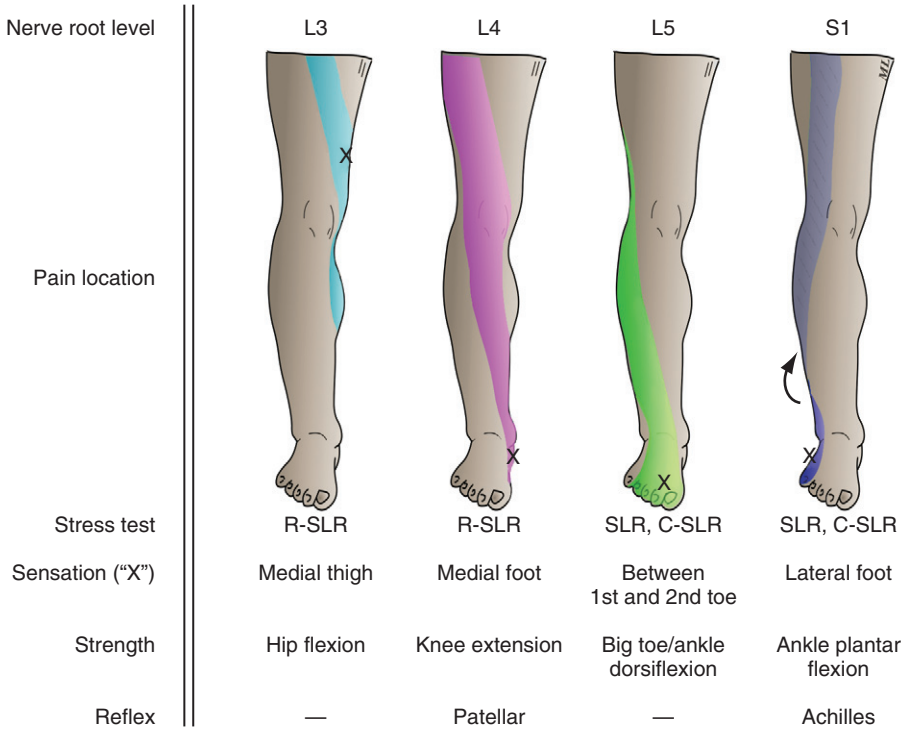


Figure 51-2. Physical examination findings for L3–S1 radiculopathy. The X marks the ideal location to test for sensation for each nerve root. C-SLR, crossed straight leg raise; R-SLR, reverse straight leg raise; SLR, straight leg raise.

Degenerative Spondylolisthesis

Most cases of spondylolisthesis, forward displacement of one vertebral body over another, are caused by degenerative changes. This condition is most prevalent in adults older than age 50 and occurs most commonly at the L4–5 and L5–S1 junctions. Two thirds of older patients with radiographically documented degenerative spondylolisthesis are asymptomatic.[37] For patients with pain, bending, twisting, and lifting activities aggravate the symptoms. Radiculopathies, spinal stenosis symptoms, or both may coexist. On physical examination, clinical findings may include a loss of lumbar lordosis, a step-off along the midline spine if the spondylolisthesis is severe, tight hamstrings, or a radiculopathy.

Arthropathies

Inflammatory arthropathies, such as ankylosing spondylitis, rheumatoid arthritis, and psoriatic arthritis, all are associated with subacute and chronic low back pain. Patients with these conditions exhibit a decreased range of spinal flexibility. Commonly with ankylosing spondylitis, patients complain of morning back stiffness and pain relief with exercise. On physical examination, patients with inflammatory arthropathy (not just AS) may have nonspecific manifestations, such as decreased spinal mobility, sacroiliac joint tenderness, and decreased chest expansion.

Red Flag Diagnosis: Fracture

In all patients who have experienced significant blunt trauma to the back or only minimal trauma in the setting of osteoporosis, fractures of the spinal column must be considered. Among patients taking long-term corticosteroids, who are predisposed to the development of early osteoporosis, back pain had a specificity of 99.5% for a spinal fracture in one series.[19] This subpopulation of patients must be assessed for a fracture despite the absence of trauma. On examination, tenderness along the midline spine and paraspinous muscles from concurrent muscle spasm usually can be elicited.

Red Flag Diagnosis: Cauda Equina Syndrome

Cauda equina syndrome results from a sudden compression of multiple lumbar and sacral nerve roots. Although it is an extremely rare presentation of back pain, it constitutes a neurosurgical emergency. The usual cause is a massive central disk herniation, but other potential etiologic disorders include spinal epidural abscess, hematoma, trauma, and malignancy. Patients with cauda equina syndrome present with back pain and multiple-level radiculopathies, often involving both legs. Difficulty with bladder or bowel function also may be a feature. Diagnostic dilemmas arise because patients can present atypically with equivocal neurologic compromise and only mild to moderate pain.

The most consistent examination finding in cauda equina syndrome is urinary retention. With a high sensitivity for this finding of 90%, this disease process is unlikely if the patient's postvoid residual urine volume is less than 100 to 200 mL. *Saddle anesthesia*, sensory deficit over the buttocks, upper posterior thighs, and perineal area, frequently is an associated finding, with a sensitivity of 75%. In 60 to 80% of cases, the rectal examination reveals decreased sphincter tone.[19]

Red Flag Diagnosis: Spinal Infection

Epidural abscess and spondylitis (osteomyelitis of the vertebral bone) are two types of dangerous spinal infections. Patients at higher risk include injection drug users, alcoholics, immunocompromised patients (e.g., patients with human immunodeficiency virus, diabetes mellitus, chronic renal failure, or long-term corticosteroid use), the elderly, patients who have sustained blunt trauma to the back, patients with an indwelling catheter, and patients with a recent bacterial infection. With epidural abscess, approximately 20% of patients have no comorbid illnesses or risk factors. The most common bacterial culprit is *S. aureus*, spreading hematogenously from a remote site or from direct extension of a local infection, such as spondylitis or disk space infection. Less common culprits are streptococcal strains and enteric gram-negative bacilli. Patient history reveals back pain even at rest and subjective fevers. On physical examination, there is often tenderness to percussion over the spinous process near the abscess location. Spinal epidural abscess remains a diagnostic challenge because approximately 50% of the patients have no neurologic deficits, and 50% may be afebrile on initial presentation.[38] With this subtle and often chronic presentation, many cases are misdiagnosed on initial presentation. Accordingly, an awareness of this entity as a possible cause of low back pain is important for prompt diagnosis of this neurosurgical emergency, which carries a mortality rate as high as 23%.[38,39]

With spondylitis, infection often begins as a subtle hematogenous seeding of the disk space, causing diskitis. Subsequent contiguous spread of the disk space infection causes vertebral endplate erosion, leading to spondylitis. As with epidural abscess, the most common bacterial culprit is *S. aureus*. Less commonly, enteric gram-negative bacilli and *Mycobacterium tuberculosis* (in Pott's disease) are the infecting organisms. Injection drug users also are at risk for *Pseudomonas* spondylitis. The history typically reveals a more indolent course of back pain, with subjective fevers. The physical examination findings can range from nonspecific tenderness of the spine to radiculopathy and cauda equina syndrome. Similarly nondiagnostic, the presence of fever has a sensitivity of only 27 to 50% for spondylitis.[19]

Red Flag Diagnosis: Malignancy

Vertebral infiltration with a tumor can be caused by either a primary or, more commonly, a metastatic malignancy. Affected patients generally are older than 50 years of age and often complain of subacute or chronic back pain that is worse at night. Risk factors include a history of known cancer (98% specificity), unexplained weight loss (94% specificity), persistent pain despite bedrest (90% specificity), and pain lasting more than 1 month (81% specificity).[40] On examination, these patients typically have mild to moderate spinal tenderness. Examination of the organs in which tumors are most likely to metastasize to bone, including breast, prostate, and lung, is indicated.

Referred Back Pain

Referred pain often is difficult to differentiate from pain originating from the lumbosacral structures. It is vital, however, to make the distinction. A sudden onset of severe, "tearing" back pain is classically an aortic dissection. Abdominal pain radiating to the back may be due to a ruptured abdominal aortic aneurysm in an elderly patient with atherosclerotic disease. Alternatively, abdominal pain radiating to the back could be from pancreatitis in a chronic alcoholic. Unilateral paraspinal pain associated with fever and nausea in a young woman could indicate pyelonephritis. In all such cases, a thorough examination of the abdomen, genitourinary system, and cardiovascular system is essential. Pinpointing the primary cause of the pain may radically alter the therapy for the patient.

Functional Back Pain

Distinguishing functional pain from "real" pain often is difficult, but several clues can be elicited from the history. A prolonged history of nonanatomic pain complaints, vague pain descriptions without localization, multiple lawsuits over similar problems, and lack of coordinated care for a problem that otherwise seems to dominate the patient's life all suggest that a search for a physical cause would be fruitless. In such cases, secondary gain for the patient's complaints often is likely.

On physical examination, maneuvers can be performed to try to detect functional back pain, if a psychological overlay is suspected. The first recommended maneuver is performing the SLR test from the sitting instead of the supine position. Seemingly focused on the knee examination, the physician extends the patient's knee; this physiologically reproduces the SLR by stretching the L5 and S1 nerve roots. A positive response includes reproduction of the patient's pain and extension of the back while seated to decrease traction on the sciatic nerve. A positive result on SLR testing in the supine position but a negative result in the seated position, and vice versa, suggests a nonphysiologic cause for the pain.

A second sign is apparent superficial tenderness. Some patients, to impress the physician with their degree of pain, respond dramatically to superficial palpation. This response is atypical for patients with genuine back pain. Nondermatomal sensory loss and widespread nondermatomal pain complaints also are unlikely to be caused by physiologic processes.

Third, back pain should not be elicited by pushing down on the patient's scalp against the cervical spine. This maneuver axially loads only the cervical and not the lumbar spine.

Fourth, a patient who generally overreacts during the examination probably is not giving a true reflection of the actual discomfort. All of these signs are believed to correlate well with psychopathology but have poor prognostic value. They are suggestive of malingering and functional complaints but are neither sensitive nor specific enough to rule out organic pathology.[41,42]

Back Pain in the Elderly

In elderly persons with back pain, musculoskeletal processes and disk herniation are less likely etiologic disorders. Instead, spinal stenosis and degenerative spondylolisthesis should be considered. Also, the incidence of more worrisome diagnoses, such as an osteoporotic fracture, spinal infection, and malignancy, is much greater in this patient population. Consequently, in these cases, the threshold for further investigation should be much lower.

Back Pain in Children

The likelihood of a congenital cause for back pain, such as leg-length discrepancy and spondylolisthesis, is greater for children than for adults. Spondylolisthesis is diagnosed most often in patients older than 10 years of age who are involved heavily in sports and complain of low back pain worsened with activity. In a retrospective study in an urban pediatric emergency department (ED) over a 1-year period, the most common causative disorders in patients with back pain complaints included direct trauma (25%), muscular strain (24%), sickle cell crisis (13%), idiopathic (13%), urinary tract infection (5%), and viral syndrome (4%).[30] A history suggesting infection or malignancy in children is similar to that in adults. Radicular symptoms are relatively rare in children. Functional processes are suggested when the pain is present only with certain undesired activities, such as housework or chores.

■ DIAGNOSTIC STRATEGIES

Laboratory Evaluation

In the absence of historical and physical findings suggesting "red flag" diagnoses for low back pain, laboratory evaluation is unnecessary. When a patient presents with back pain suggestive of a spinal infection or malignancy, however, laboratory studies may help with risk stratification. Specifically, a complete blood cell count, erythrocyte sedimentation rate (ESR), and urinalysis should be performed. Additional laboratory studies should be tailored to the patient's history and physical examination. Liver function testing and determination of amylase or lipase level may be indicated to investigate abdominal complaints.

For a spinal infection, the ESR usually is elevated (above 20 mm/hour), whereas the serum white blood cell (WBC) count may or may not be elevated.[43] In one study, 13 of 40 (32%) patients with a spinal epidural abscess had a falsely reassuring WBC count less than 11,000/μL.[39] When patients are diagnosed with a spinal infection, blood should be drawn for cultures because a single strain, most commonly *S. aureus*, can be isolated in 50 to 90% of the cases.[39,44] Performing a lumbar puncture to evaluate the cerebrospinal fluid is unnecessary and is relatively contraindicated because of the risk of seeding the fluid with bacteria.

For a bony malignancy, the ESR also usually is elevated, whereas the WBC may be equivocal. The hematocrit may be low secondary to anemia of chronic disease. Other additional helpful laboratory tests include measurement of alkaline phosphatase, prostate-specific antigen assay, and serum immunoelectrophoresis and urine testing for light chains (for multiple myeloma).

Radiology

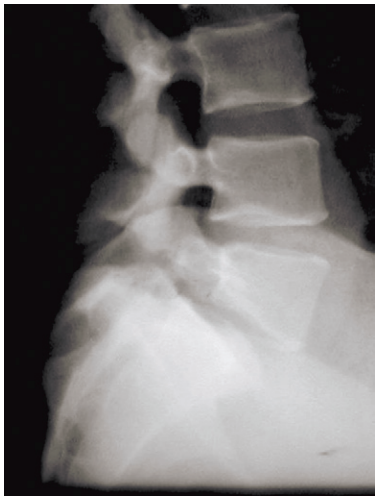
Plain Radiography

The utility of "screening" lumbosacral plain radiographs for all patients with acute low back pain is extremely low. Plain radiographs contribute little to patient management in the absence of concerning "red flag" findings and needlessly expose the patient to radiation.[45] Most patients with back pain do not need radiographs. In those cases in which radiographs have been obtained, normal findings are usual, but incidental findings, which may or may not be the true cause of the patient's pain, also are relatively common and may include spondylolisthesis, abnormal spinal curvature, disk space wedging, or degenerative changes.[9] Currently accepted indications for radiographs in patients with back pain are listed in Box 51-1.

Patients with radicular symptoms suggesting a herniated disk do not require radiographs. In addition to being undetect-

BOX 51-1 INDICATIONS FOR PLAIN LUMBOSACRAL RADIOGRAPHS IN PATIENTS WITH LOW BACK PAIN

- Age younger than 18 or older than 50 years
- Any history of malignancy or unexplained weight loss
- Any history of fever, immunocompromise, or injection drug use
- Recent trauma, other than simple lifting
- Progressive neurologic deficits or other findings consistent with cauda equina syndrome
- Prolonged duration of symptoms beyond 4 to 6 weeks



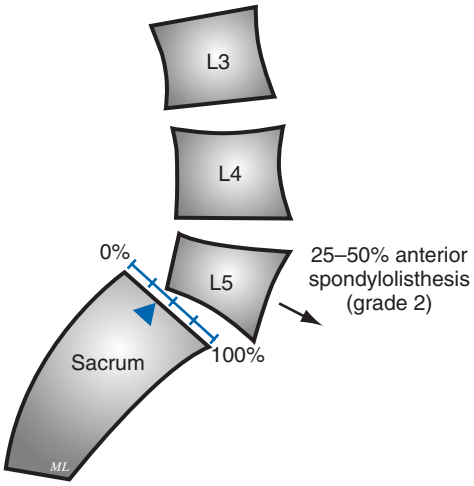


Figure 51-3. Lateral plain radiograph and schematic diagram of grade 2 anterior spondylolisthesis of L5 on S1. Grading is based on the percentage of slippage, with 0 to 25%, 25 to 50%, 50 to 75%, and greater than 75% corresponding to grades 1 to 4, respectively.

able on plain radiographs, disk herniations resolve with conservative management in most cases.

If radiographs are obtained, anteroposterior and lateral views usually are sufficient in the ED, although many centers also prefer to obtain a coned-down lateral sacral view. Oblique views are not necessary except in children, in whom spondylolysis and spondylolisthesis may be more prevalent.[46]

On plain radiographs, spondylolisthesis, vertebral osteomyelitis, and vertebral metastatic disease have classic appearances. Spondylolisthesis (Fig. 51-3) is classified into grade 1 through grade 4 based on the severity of the anterior slippage of one vertebral body over another. Grade 1, which is often asymptomatic, involves less than 25% slippage. Grade 2 through grade 4 involves 25 to 50%, 50 to 75%, and at least 75% slippage, respectively.

Spondylitis (Fig. 51-4) is characterized by erosion of contiguous vertebral endplates and a shortened disk space height, best seen on the lateral view. Because the anterior subchondral vertebral bone and disk space are highly vascular, it follows that spondylitis has a predilection for these areas because of the hematogenous spread of infection. With more advanced disease, vertebral bone erosion and collapse may occur.

Vertebral metastatic disease (Fig. 51-5) can manifest as either a blastic (hyperdense) or a lytic (hypodense) lesion and has a predilection for the vertebral body and pedicles. In contrast with osteomyelitis, the intervertebral disk space is spared.

If a red flag diagnosis is of concern, a plain radiograph may rule out a fracture but may not be adequate to rule out other pathologic processes, such as cauda equina syndrome, spinal infection, and malignancy. For cauda equina syndrome, patients more often have normal or nonspecific plain film findings because the most common etiology is a central disk herniation. For spinal infection and vertebral malignancy, the sensitivity of a plain radiograph is only fair at 82% and 60%, respectively.[45] In these cases, the patient subsequently should undergo MRI if the there is a high clinical suspicion.

Computed Tomography and Magnetic Resonance Imaging

For assessment of fractures of the vertebral column, computed tomography (CT) is superior to MRI. In the case of a fracture, CT helps to elucidate the integrity of the spinal canal and the risk for spinal cord impingement. For all other red flag diagnoses—cauda equina, spinal infection, and malignancy—MRI is the definitive investigative modality. Its superior tissue resolution, especially of the spinal cord and intervertebral disks, and its capability of generating more accurate sagittal reconstructions make MRI the ideal imaging modality. MRI is able to differentiate subtle soft tissue pathology, such as a spinal epidural abscess (Fig. 51-6). No radiation exposure is incurred with MRI, whereas one CT scan of the spine exposes the patient to approximately 4 years' worth of natural background radiation.[47]

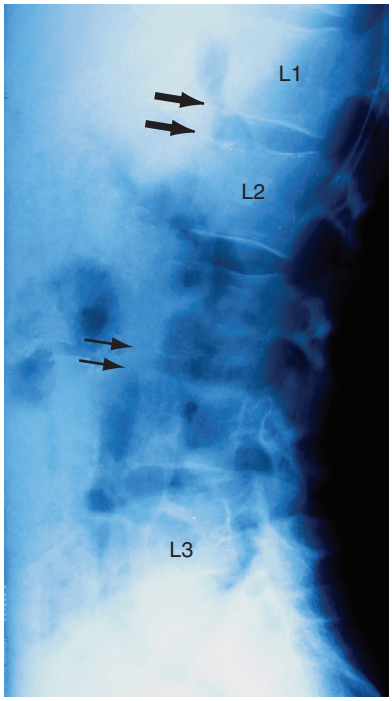


Figure 51-4. Lateral plain radiograph shows *Staphylococcus aureus* spondylitis of L3 and L4. There is narrowing of the L3–4 disk space and erosion of the vertebral endplates of L3 and L4 *(small arrows)*. Notice the distinct vertebral endplate margins in unaffected areas *(large arrows)*.

Although disk herniation is easily visualized on MRI, patients with findings consistent with an uncomplicated disk herniation (i.e., without objective findings of motor or sensory deficit on examination) should not routinely undergo MRI imaging because of the self-limited nature of the disease in most cases. Overimaging in patients with lumbar radiculopathy may lead to an overdiagnosis of disk herniations because

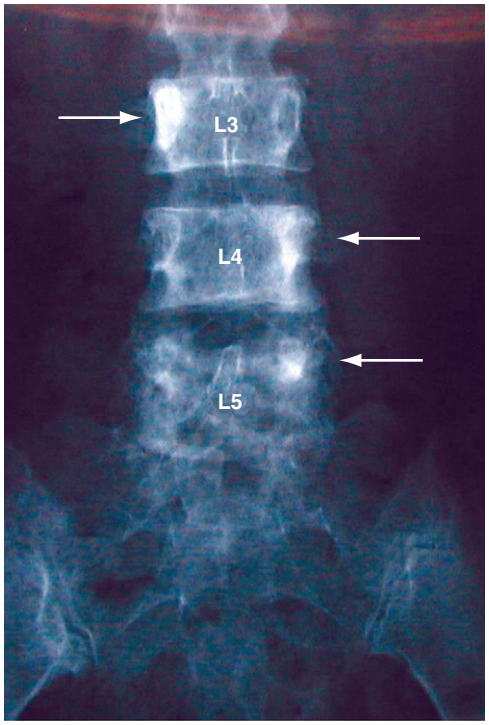


Figure 51-5. Anteroposterior plain radiograph shows blastic infiltration of metastatic breast cancer to the pedicles of L3 to L5 *(arrows)*.

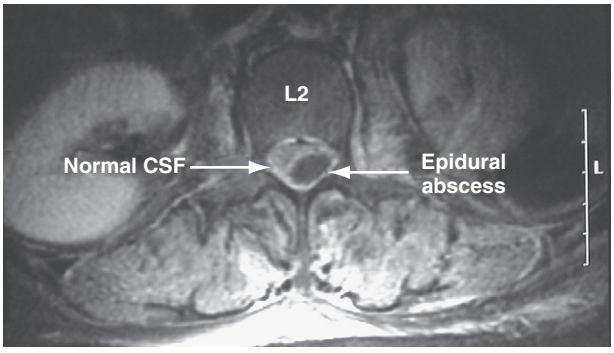


Figure 51-6. Axial T_2-weighted magnetic resonance imaging of *Staphylococcus aureus* L2 epidural abscess impinging the dorsolateral aspect of the spinal canal. CSF, cerebrospinal fluid. (Image contributed by Dr. Stephen Bretz.)

incidental MRI-documented herniations have been shown to occur in 28 to 33% of asymptomatic patients. The result may be unnecessary surgical intervention.[9,48,21]

Special Investigations

Unless a process other than uncomplicated back pain or disk herniation is suspected, other investigations are not required. MRI is the definitive test for most conditions. Radionuclide scans have been used for locating suspected malignancy, infectious foci, and occult fractures as in spondylolysis. Nuclear medicine scans are regarded as sensitive but nonspecific.

DIFFERENTIAL CONSIDERATIONS

Nonspecific low back pain is in many ways a diagnosis of exclusion. In a typical patient, within the 18- to 50-year age range with acute low back pain and with no radiculopathy, previous malignancy, weight loss, or fever, the diagnosis is almost certainly uncomplicated musculoskeletal back pain. When the patient falls outside of the aforementioned parameters, a wide variety of diagnostic possibilities must be entertained.

Almost anything can cause low back pain. Box 51-2 presents an extensive list of diagnostic considerations, but it is useful to look at the most common and most serious causes of low back pain other than musculoskeletal lumbosacral pain. One of the most life-threatening causes of referred back pain is a leaking or ruptured abdominal aortic aneurysm. The reader is referred to appropriate chapters for further discussion of specific problems.

MANAGEMENT

Because most patients with acute low back pain without objective sensory or motor loss on examination experience symptomatic resolution within 4 to 6 weeks, only conservative management is needed. In general, MRI and surgery are reserved for the few patients who have worrisome systemic signs and patients with refractory, debilitating back pain. Over the past few decades, the accepted practice has shifted 180 degrees, from an overaggressive recommendation for invasive surgical intervention to the minimalistic recommendation of symptomatic pain control and early return to activity. The recommended role of the physician in back pain management is to obtain a correct diagnosis, rule out significant pathology, avoid excessive investigation, provide analgesia, and educate the patient.[49] The management for various etiologic categories of disorders that may cause low back pain is summarized in Figure 51-7. For management of fractures and referred pain, the reader is referred to the appropriate chapters.

Uncomplicated Musculoskeletal Back Pain

Besides a thorough history and physical examination, no further investigations are required for uncomplicated low back pain. Only pain control and patient education are indicated. Aside from an initial parenteral opioid or nonsteroidal anti-inflammatory drug, most patients can be managed with oral nonsteroidal medications. Ibuprofen is an ideal choice because it is inexpensive, but various nonsteroidal anti-inflammatory drugs have been shown to have the same efficacy. It is unclear whether ibuprofen is superior to acetaminophen.[50] Short-term opioids also occasionally are needed for breakthrough pain in the acute setting. Various other medications have been advocated, including benzodiazepines and other muscle relaxants. Based on the current conflicting literature, these medications probably do not provide a significant added benefit, but they do increase the incidence of side effects such as drowsiness and drug dependence.[51] Corticosteroids have no role in the treatment of uncomplicated low back pain.

In terms of patient education, one of the outdated practices of back pain management was that physicians convinced patients that they were sick. This was done by overinvestigating, overtreating, putting patients to bed, and taking them off work. It now has been shown convincingly and repeatedly that all of those interventions are excessive. Instead, patients should be educated about why they are not undergoing radiographic studies of their lumbosacral spine or laboratory tests and should be reassured of the likely benign course of the pain. Most patients can be convinced by education and an explanation of radiation dosing. A typical lumbosacral spine series involves as much gonadal irradiation as that incurred with a daily chest radiograph for 5 or 6 years.[52] Patients also are discouraged from the outdated recommendation of strict bedrest. Compared with patients who are prescribed strict

BOX 51-2 Differential Diagnosis for Low Back Pain

Localized/Common
Uncomplicated musculoskeletal back pain
Intervertebral disk herniation
Spinal stenosis
Spondylolisthesis
Osteoarthritis
Fracture

Localized/Uncommon
Infection
Spondylitis
Epidural abscess
Diskitis
Herpes zoster

Malignancy
Metastatic
- Breast
- Lung
- Prostate
- Kidney, thyroid, colon (less common)

Primary
- Multiple myeloma
- Lymphoma
- Leukemia
- Primary cord/extradural tumors
- Osteoid osteoma
- Other primary bone tumors

Pediatric
Spondylolisthesis/spondylolysis
Severe scoliosis
Scheuermann's disease

Rheumatologic
Ankylosing spondylitis
Psoriatic arthritis
Polymyalgia rheumatica
Reiter's syndrome

Vascular
Arteriovenous malformation of spinal cord
Epidural hematoma

Life-Threatening Referred Pain
Abdominal aortic aneurysm

Gastrointestinal System
Biliary pathology
Pancreatitis
Peptic ulcer disease
Diverticulitis

Genitourinary System
Renal colic
Pyelonephritis
Prostatitis
Cystitis

Gynecologic System
Menstrual cramps
Spontaneous abortion
Labor
Ectopic pregnancy
Pelvic inflammatory disease
Endometriosis
Ovarian cyst
Ovarian torsion

Hematologic System
Sickle cell crisis

Functional
Somatization disorder
Depression
Fibrositis
Malingering

bedrest, patients who remain active experience earlier resolution of pain and return to work sooner.[4] Patients should be made aware, however, that the back pain has a 66 to 84% likelihood of recurring within 12 months.[10]

Other supplemental treatment modalities have been shown to be of debatable efficacy in the management of acute and chronic low back pain. These include acupuncture, physiotherapy, chiropractic manipulation, massage, ultrasound, traction, and transcutaneous nerve stimulation.[9,53-56]

Lumbar Disk Herniation

Like patients with uncomplicated low back pain, patients with disk herniations and radiculopathy do not benefit from strict bedrest.[5,49] In the acute setting, these patients should receive analgesics, but further investigation with laboratory tests and radiographs is not necessary. Most of these patients experience symptomatic resolution within 6 weeks with conservative, nonsurgical management.[57,58]

Corticosteroid injections into the epidural space have been advocated for sciatica in the belief that this treatment helps to relieve some of the inflammation associated with disk herniation. Although some reduction of symptoms may be obtained initially, no long-term benefit or reduction in the need for later surgery has been documented.[59] The use of systemic steroids in back pain and disk herniation remains controversial. Although there is no proven benefit, the anti-inflammatory effects make empirical sense in the context of radiculopathy. A large retrospective review showed no definite benefit of systemic steroids in either setting, but the definitive trial is yet to be done.[51]

When the pain from disk herniation persists for longer than 4 to 6 weeks, outpatient MRI is indicated. With a documented herniation, these patients may benefit from surgical diskectomy although this remains controversial compared to conservative management.[60] Other indications for surgery include intractable pain and worsening motor or sensory deficit. Although surgical patients tend to have earlier relief of pain compared with nonsurgical patients, the 4- and 10-year results are the same. Microsurgery techniques and laser therapy have not been shown to confer any advantage over conventional techniques.[61]

Spinal Stenosis

Patients with spinal stenosis should be managed conservatively with pain medications. In the absence of alarming red flag findings, these patients do not require laboratory or radiographic studies in the ED. These patients may be candidates for surgery if they show any of the following conditions: pro-

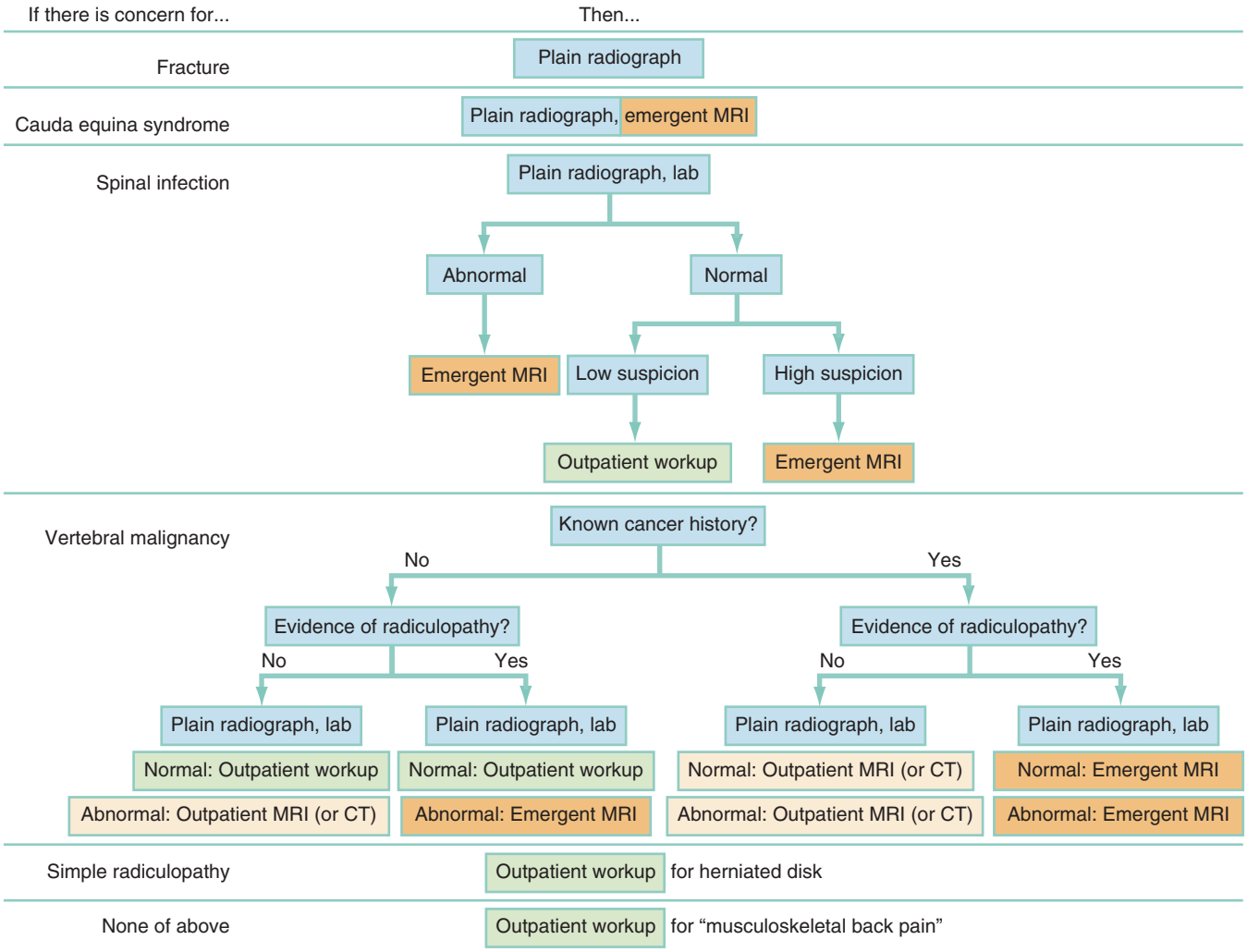


Figure 51-7. Algorithm for management of low back pain. The patient's history may be concerning for more than one red flag diagnosis.

gressive neurologic deficit, progressive reduction in ability to walk secondary to pseudoclaudication, evidence of cauda equina syndrome, or intractable pain. Elective surgical decompression is more controversial. A 10-year longitudinal study showed that no findings predicted which patients would benefit more from surgery than from conservative management.[62] The benefits of surgery also must be weighed against the risks of surgery itself, because these patients usually are elderly.

Degenerative Spondylolisthesis

Patients with symptomatic degenerative spondylolisthesis are managed conservatively with analgesia and lifestyle changes, which include the avoidance of repetitive bending, heavy lifting, and twisting at the waist. For patients with refractory or severe back pain, outpatient MRI and neurosurgical referral for possible operative decompression are recommended.

Red Flag Diagnosis: Fracture

See Chapter 40, Spinal Injuries.

Red Flag Diagnosis: Cauda Equina Syndrome

Cauda equina syndrome is a neurosurgical emergency that requires urgent operative decompression to help preserve distal neurologic function. All patients with concerning findings, such as saddle anesthesia or a large postvoid residual volume, require emergent MRI. On average, outcome is improved if decompression takes place within 48 hours of symptom onset.[63] Early evidence, however, indicates that delayed operative decompression under a more controlled setting may be performed without any adverse effects, particularly in patients who have overflow urinary incontinence.[64]

Red Flag Diagnosis: Spinal Infection

If findings on the history or physical examination are worrisome for a spinal infection, further investigation is of paramount importance. With a low index of suspicion, normal results on serum WBC count, ESR, and lumbosacral plain radiograph can safely rule out infection. The patient's history and examination are worrisome for spinal infection and further investigation is paramount. In patients with moderate to high pretest probability for a spinal infection, the next step is to obtain an emergent MRI.

Pyogenic spinal infections should be treated with neurosurgical drainage and decompression, in addition to broad-spectrum intravenous antibiotics that cover at least for *S. aureus* and gram-negative bacilli until blood culture results return. Vancomycin should be included in the antibiotic regimen, given the increased prevalence of methicillin-resistant *S. aureus*. For injection drug users, antibiotic coverage for

Pseudomonas is necessary. The recent literature suggests that a nonsurgical management approach may be acceptable for patients at lower risk, such as those who are hemodynamically stable and neurologically intact.[65]

Red Flag Diagnosis: Malignancy

An algorithmic guideline to the management of back pain that is worrisome for malignancy involves subdividing patients into two categories: patients with and patients without a history of previous cancer. Development of spinal metastasis has been reported in 20 to 85% of patients with cancer.[66,67] These patients are subdivided further into those with and those without evidence of a radiculopathy.

Most patients fall into the classification of *back pain without a history of cancer and without a radiculopathy*. They have a history that is only suggestive of a malignancy, such as unexplained weight loss or back pain that is worse at night. These patients require further risk stratification with plain radiographs and laboratory tests, including a complete blood cell count and ESR. With normal results, these patients can be referred to their primary care physician for further workup. The physician should not feel completely reassured that malignancy has been ruled out, however, because plain films have a false-negative rate of 10 to 17% for vertebral bone metastasis. This false-negative rate is likely to be due to the fact that a cancer needs to erode at least 50% of the bone before becoming radiographically apparent.[68] With abnormal results, such as a bone lesion or extremely elevated ESR level (above 100 mm/hour), an urgent CT scan or MRI should be performed on an outpatient basis within the next 3 to 7 days.

For patients *without a history of cancer but with signs of radiculopathy*, the workup also includes a plain radiograph, complete blood cell count, and ESR. If the test results are normal, the patient should be referred to his or her primary care physician for further evaluation for malignancy and other potential causes of radiculopathy, including spinal stenosis and disk herniation. If the workup shows a bone lesion on plain radiographs or an extremely elevated ESR level (greater than 100 mm/hour), the patients should undergo emergent MRI because (1) the presence of radiculopathy may be an early harbinger of impending spinal cord compression from a mass effect and (2) it often is difficult on plain radiography to distinguish between a neoplasm from early spondylitis (especially tuberculous osteomyelitis) and an osteoporotic fracture causing a vertebral collapse.[69] If MRI is unavailable, multidetector CT imaging can be performed initially to screen for the presence of malignant bone infiltration, although definitive spinal cord and nerve root imaging will require MRI. The presence of a vertebral mass lesion on CT images suggests spinal cord compression by the mass as a cause for the pain and other symptoms.

For patients *with a history of previous cancer and low back pain*, advanced imaging is indicated, either emergently or urgently within 3 to 7 days. In the absence of clinical manifestations of radiculopathy, these patients should undergo outpatient MRI (or CT) regardless of plain radiography findings and laboratory results. Plain radiography is too insensitive to rule out a vertebral neoplastic process definitively. If radiculopathy is present, however, these patients require emergent MRI regardless of plain radiography findings because of the concern for spinal cord compression. In a study of patients with a confirmed diagnosis of cancer who had back pain and radiculopathy, the risk of epidural spinal cord compression was 25% despite normal radiographic findings, and 88% with radiographic evidence of vertebral metastasis.[70]

In all patients undergoing emergent MRI to evaluate for vertebral malignancy and cauda equina syndrome, dexamethasone should be administered as soon as these conditions are suspected to reduce the potential mass effect. In addition to high-dose corticosteroids, patients with a vertebral neoplasm also may benefit from radiation therapy.

Pediatric Back Pain

Management of back pain in children is similar to management for adults and depends on the underlying causative disorder. Spondylolisthesis is managed by observation, with only 4 to 5% of cases worsening. Progression usually stops as skeletal maturity is achieved in the late teens. Current recommendations are for limited contact sports in children with less than 30 to 50% slippage and surgical stabilization for children with slippage greater than 30 to 50%. Treatment becomes more aggressive if the child is symptomatic.[71]

Chronic Back Pain

Patients with chronic back pain often are regarded as the most challenging of all patients with back pain. The cause of chronic back pain is complex and multifactorial and usually requires a multidisciplinary approach for the greatest chance for success. Psychosocial factors, including depression, drug dependence, and financial gain, undoubtedly play a significant role in the behavior of many of these patients, making proper assessment and treatment impossible in the ED.

After appropriate evaluation has ruled out a red flag cause for the back pain, ED management involves analgesia and referral for follow-up care. The main decision usually centers on the use of narcotics, which should be individualized in accordance with the physician's assessment of the clinical scenario. Patients exhibiting drug-seeking behavior classically are from out of town, have a primary care physician who cannot be contacted, or are reportedly allergic to all nonopioid medications.

DISPOSITION

Almost all patients with uncomplicated back pain can be discharged from the ED with follow-up with their primary care physician. In rare circumstances, severe pain or inadequate support for convalescence at home may preclude discharge. For patients who have a red flag diagnosis of cauda equina syndrome or epidural abscess, immediate neurosurgical consultation is required for emergent surgical decompression. For patients with spondylitis, hospitalization will be necessary for administration of intravenous antibiotics. With intractable pain from vertebral malignancy, the decision to hospitalize the patient for pain control, administration of high-dose corticosteroids, and radiation therapy should be made in conjunction with a neurosurgeon, an oncologist, and a radiation therapist.

One of the most important aspects of management of low back pain is the discharge instructions for the patient. Not only are clear and simple instructions useful to the patient, but they also constitute a medicolegal necessity for the physician. Physicians are advised to avoid using purely medical terms. The discharge instructions should include the following:

1. *Diagnosis*: Distinguish between uncomplicated (musculoskeletal) back pain and diskogenic radiculopathy.
2. *Activity*: Recommend maintaining active mobility as limited only by pain, avoiding heavy lifting until symptoms resolve, and getting back to full activity as soon as possible.

3. *Reassurance*: Educate patients about the likely benign nature of the etiologic condition responsible for the pain.
4. *Warnings:* Instruct patients to return to the ED immediately if they experience any of the following: fever; loss of bladder or bowel control; numbness or tingling around the anus, vagina, or penis; or new pain or weakness down one or both legs.

Pediatric patients with back pain require the same discharge instructions as for adults. In patients with pain secondary to spondylolisthesis, activity restrictions should be made in conjunction with an orthopedic surgeon. ED disposition for a patient with chronic back pain is relatively simple: pain management with nonopioids, when possible, and referral to a primary care physician for follow-up care. The prognosis is guarded because patients who are off work for 6 months usually are still off work after 2 years.[28]

UPPER BACK PAIN

PERSPECTIVE

Background

Thoracic pain is far less common than low back pain. Thoracic pain usually has a musculoskeletal origin, but other, more emergent causes must be considered first, including thoracic aortic dissection, pulmonary embolism, and esophageal disease. Compared with lumbar disk herniation, which is fairly common, thoracic disk disease is extremely rare, difficult to diagnose, and difficult to treat.

Epidemiology

The actual incidence of thoracic pain is unknown. The incidence of symptomatic thoracic disk disease is low, with estimates at 1 in 1 million.[72] The average age is in the 40s with equal gender distribution. Surgery for thoracic disks accounts for less than 4% of all disk operations.[73] Metastases are more common in the thoracic spine than in the lumbar spine, with 60 to 70% of spinal metastases localizing there.[9,74]

PRINCIPLES OF DISEASE

Anatomy and Physiology

The thoracic vertebral column can be regarded as an extension of the cervical column with the addition of ribs. There are 12 thoracic vertebrae, connected by the anterior and posterior longitudinal ligaments and the ligamentum flavum, similar to the lumbar vertebrae. Also similarly, intervertebral disks provide elasticity and stability to the thoracic column. The spinal canal diameter remains unchanged through the thoracic and lumbar levels, but at the thoracic level, the space around the spinal cord is smaller than at the lumbar level. Because lumbar nerve fibers have not yet branched off from the spinal cord, the thoracic cord is thicker than the lumbar cord. Significant neurologic abnormalities may result from minimal spinal canal impingement at the thoracic level.

Pathophysiology

Common thoracic soft tissue pain is likely to be a combination of sprain and muscle inflammation. As in the lower back, innervation of the paravertebral area is provided by the sinuvertebral nerve, and any anatomic disruption of surrounding structures results in nonspecific pain. Thoracic disk herniations, which most commonly occur in the midthoracic to lower thoracic spine, cause pain and neurologic symptoms in much the same way as for lumbar disks. It is not clear why the clinical presentation is so varied, although a possible cause is a higher number of centrally herniated disks, resulting in much more frequent myelopathic symptoms.[73]

CLINICAL FEATURES

Symptoms and Signs

History

Nondiskogenic thoracic back pain usually manifests as paraspinal discomfort. A history of trauma or recent unusual activity may or may not precede the onset of pain. Complaints with thoracic disk herniations are variable but usually are associated with long-standing pain or neurologic symptoms, or both. Pain may be localized to one part of the thoracic vertebrae, it may radiate down to the sacrum, or it may have a radicular component along the ribs. Central disk herniation can manifest as diffuse abdominal and back pain or burning sensation in the lower extremities. Associated findings may include mild weakness, spasticity, gait disturbance, bowel or bladder dysfunction, and paraplegia. These usually progress until the condition is diagnosed. Because of the variable and often subtle signs and symptoms, thoracic disk disease typically is not diagnosed until 20 months after the first clinical presentation. Pain from other causes should be sought in the initial assessment. A history of trauma, fever, previous malignancy, cardiovascular disease, or gastrointestinal problems may indicate problems originating outside the thoracic spine and may warrant further investigation.

Physical Examination

In patients with benign musculoskeletal pain, the physical examination fails to disclose evidence of a pathologic process. These patients may experience mild to moderate paraspinal tenderness to palpation, pain with motion, and even discomfort from chest wall expansion with respirations, but objective findings are minimal.

Physical findings in patients with thoracic disk herniation will vary with the location and degree of herniation. Objective findings may range from a normal-looking spine to loss of posterior column function (position, touch, vibration) or unilateral or bilateral weakness. Gait and sensory abnormalities are common. Hypotonic abdominal reflexes may be present with distal hyperreflexia. A Babinski response may be present. Myelopathy may result in urinary retention. Muscle wasting may be present with chronic symptoms. The possibility of other pathologic conditions should be kept in mind during the physical examination, with appropriate assessment tailored to the presentation and level of clinical suspicion.

DIAGNOSTIC STRATEGIES

Laboratory Evaluation

In the face of an unremarkable history and physical examination, the likelihood that laboratory evaluation will yield useful results is extremely small. In appropriate clinical circumstances, assessment for malignancy, infection, and inflammation should be undertaken.

Radiology

The usefulness of radiologic assessment is dubious in a patient with atraumatic acute thoracic back pain in whom preexisting illness or neurologic abnormalities are absent. As a general

BOX 51-3 DIFFERENTIAL DIAGNOSIS FOR THORACIC BACK PAIN

- Uncomplicated musculoskeletal back pain
- Spinal cord and nerve root pathology (e.g., disk herniation, tumor, hematoma)
- Vertebral column disease (e.g., primary or metastatic malignancy, osteomyelitis)
- Disk infection
- Primary neurologic disease
- Degenerative and autoimmune arthropathies
- Herpes zoster
- Vascular disease (e.g., thoracic aortic dissection, acute coronary syndrome, pulmonary embolism)
- Thoracic cavity pathology (e.g., pleuritis, pericarditis, pneumonia, esophageal pathology)
- Intraperitoneal and retroperitoneal abdominal pathology (e.g., peptic ulcer disease, pancreatitis, hepatobiliary disease)

guide, however, the following factors should prompt basic radiologic studies and appropriate further investigations: clinical suspicion for presence of other conditions; unexplainable symptoms; extremes of age; concern for trauma, tumor, infection, gastrointestinal pathology, or vascular pathology; and prolonged symptoms. Like patients with lower back pain, patients with a history of cancer and upper back pain should undergo plain radiography and possibly CT or MRI to assess for vertebral metastatic disease, especially because metastases have a predilection for the thoracic spine.

MRI has become the modality of choice for evaluating a herniated thoracic disk. The incidence of asymptomatic disk herniations seen on MR images is 37%. Most herniated thoracic disks, whether symptomatic or not, have been reported to recede spontaneously.[72]

DIFFERENTIAL CONSIDERATIONS

Although muscular back pain is extremely common, Box 51-3 summarizes the important considerations in the expansive differential diagnosis for thoracic back pain.

MANAGEMENT

Commonly, thoracic musculoskeletal pain is managed with analgesia. No studies have shown the need for any difference in management from that for musculoskeletal low back pain.

Thoracic disk disease is difficult to diagnose and manage. Symptomatic pain management and outpatient follow-up care are recommended. In view of the limited space in the spinal canal at the thoracic level compared with the lumbar level, spinal cord compression from a herniated disk is more likely at the thoracic level. Any disk herniation that precipitates an acute neurologic deficit warrants MRI and early neurosurgical evaluation.

DISPOSITION

Patients with benign back pain in any part of the thoracic vertebral column can be discharged with referral for follow-up care by the primary care physician. Patients with a suspected thoracic disk herniation require close outpatient follow-up. Most cases of subjective discomfort resulting from thoracic disk disease without objective neurologic findings resolve spontaneously, with one study showing a 77% improvement rate.[72] Although clear guidelines are lacking for when emergent neurosurgical consultation is required for thoracic disk herniation, patients with significant pain or neurologic compromise should be assessed rapidly.

KEY CONCEPTS

- The key to differentiating between benign and serious pathology in low back pain is a focused and thorough history and physical examination.
- The absence of sciatica findings practically rules out a lumbar disk herniation.
- Screening laboratory tests and plain radiographs are not indicated for patients with uncomplicated low back pain or a simple disk herniation.
- The best treatment for uncomplicated back pain and disk herniation is analgesia and resumption of normal activities—not bedrest.
- Age younger than 18 or older than 50 years, fever, injection drug use, immunocompromised status, symptoms lasting more than 4 to 6 weeks, night pain, and previous malignancy all are red flag warnings of potentially serious causes of back pain and should be heeded and evaluated accordingly.
- Although thoracic disk herniation can result in significant upper back pain, other, more dangerous etiologic disorders, such as aortic dissection, pulmonary embolism, and acute coronary syndrome, must be considered first.
- The associated findings on the neurologic examination with thoracic disk herniations can be extremely variable, ranging from nonspecific paresthesias to significant upper motor neuron weakness.
- The incidence of vertebral metastatic malignancy is higher in the thoracic spine than in the lumbar spine.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 167

Pediatric Respiratory Emergencies: Lower Airway Obstruction

Richard J. Scarfone and Jeffrey A. Seiden

ASTHMA

Perspective

Introduction and Epidemiology

Asthma is the most prevalent chronic disease of childhood, affecting almost 7 million children in the United States.[1] In the past 25 years, childhood asthma prevalence rates have more than doubled.[1] The public health burden posed by this disease, as assessed by emergency department (ED) visits, hospitalizations, and deaths, remains at a historically high level. About 3% of all ED visits among children are for asthma, accounting for 750,000 such visits annually.[1] Similarly, about 3% of all hospitalizations for children are due to asthma, totaling about 200,000 per year.[1] In addition, there are astonishing racial disparities among children with this condition. Compared with white children, black children have a 60% higher prevalence rate, a 260% higher ED visit rate, a 250% higher hospitalization rate, and a 500% higher death rate due to asthma.[1]

Thus, asthma is one of the few chronic diseases of childhood for which there have been increases in prevalence, morbidity, and mortality in recent decades. These trends are occurring despite unprecedented investments in terms of money, preclinical and clinical research, and national focus. The reasons for these trends are no doubt multifactorial and are beyond the scope of this discussion. This portion of the chapter will focus on the recognition, evaluation, and clinical management of children in the ED with acute asthma.

Distinguishing Principles of Diseases

Anatomy and Physiology

Asthma, a lower airway disease marked by bronchoconstriction, mucosal edema, and pulmonary secretions, may lead to respiratory failure if not treated in a timely or effective manner. Important anatomic and physiologic differences exist between children and adults that may hasten the development of respiratory failure, mandating that clinicians quickly recognize and take appropriate measures to reverse respiratory distress.

An upper respiratory infection (URI) associated with copious rhinorrhea, a common trigger of an asthma exacerbation, may significantly increase airway resistance. Further, a small decrease in the internal diameter of the upper airway causes a greater increase in resistance for the young child compared to the adult. In fact, just one millimeter of edema can decrease the cross-sectional area of the infant's airway by 75%.

Regarding the thorax, the young child has a compliant chest wall and horizontally located ribs. These factors limit use of the thorax to increase tidal volume; instead, ventilation is dependent on diaphragmatic movement. However, abdominal distention as might occur with crying or swallowing of air will impede diaphragmatic breathing. With the inability to significantly increase tidal volume, minute ventilation becomes rate dependent, quickly leading to fatigue.

An infant under 12 months of age has an oxygen consumption index that is double that of an adult, due to a higher rate of metabolism. Increased airway resistance and chest wall compliance necessitate more rapid breathing and increased energy expenditure. Increased work of breathing may account for as much as 15% of total oxygen consumption, at a time when oxygenation is poor. As a result, the child will develop hypoxemia quite rapidly in response to respiratory disease. The child with significant respiratory distress and inadequate oxygenation may become bradycardic, leading to cardiopulmonary arrest within minutes, if appropriate interventions are not undertaken.

Clinical Features

Clinical Evaluation

All acutely wheezing children arriving for ED care should be attached to a cardiorespiratory monitor and have oxygen saturation determined by pulse oximetry. If needed, supplemental oxygen should be provided. After this, the clinician may begin the clinical assessment.

History

In evaluating the child with acute wheezing, the treating physician should obtain a concise history, perform a brief and focused physical examination, determine the initial degree of illness, and initiate appropriate therapy. After therapy has begun, a more comprehensive history and physical examination can be conducted. The initial history should include questions regarding the child's age, duration and severity of symptoms, possible choking episode (foreign body aspiration), and recent medication use. The parents should be able to relate how the severity of this attack compares to that of previous exacerbations. A history of difficulty sleeping, eating, or speaking as a result of this attack suggests a moderate to severe

exacerbation. The names, doses, and frequency of administration of asthma medications recently received should be noted. A child who has been receiving very aggressive therapy with short-acting beta$_2$-agonists (SABAs) just prior to ED arrival may not respond favorably to that same therapy in the ED. Any comorbidities should be identified early in the clinical course.

A more comprehensive history should include questions regarding asthma triggers such as URIs, cigarette smoke, allergies, or exercise. Inquiries regarding fever and hydration status should be part of a complete review of systems. A past medical history of frequent asthma exacerbations, ED visits for asthma, or hospitalizations to either the general ward or the intensive care unit (ICU) would raise the concern for poorly controlled asthma. The impact that asthma has in the child's life may be gauged by the monthly frequency of daytime or nighttime symptoms such as cough, wheezing, shortness of breath, or chest tightness as well as missed days of school or restricted activity. A child who meets criteria for persistent asthma should be receiving daily anti-inflammatory therapy and those over age 5 years should be monitoring symptoms with a peak flow meter.[2] If the child is wheezing for the first time, then inquiries regarding other possible causes of wheezing (see Differential Considerations) should be made. Family and social histories should focus on asthma, cystic fibrosis, or atopic disease and the adequacy of support systems at home.

Physical Examination

The initial focused physical examination of the wheezing child should include obtaining vital signs and assessing the level of consciousness. A child who is anxious, restless, or lethargic may be hypoxemic. No single asthma score has been universally adopted to assess degree of illness or treatment responses.[3,4] Most asthma scores include key clinical factors such as respiratory rate, degree of wheezing, inspiratory to expiratory ratio, use of accessory muscles, and oxygen saturation in room air.[5] Such scores can assist in assessing the pretreatment degree of illness at ED triage, as well as tracking the child's response to therapy.

For a child with severe disease, wheezing may be audible without a stethoscope or, if aeration is extremely poor, no wheezing may be detected. Asymmetrical wheezing suggests pneumonia, pneumothorax, or the presence of a foreign body. Palpation of the chest and neck may reveal subcutaneous air, associated with a pneumomediastinum or pneumothorax. Following this initial assessment, the remainder of the physical examination may be performed. The most anxiety-provoking aspects of the examination such as otoscopy should be delayed until after treatment is well underway.

Diagnostic Strategies

Pulse Oximetry and Arterial Blood Gas

Adjunctive studies such as arterial oxygen saturation measured by pulse oximetry may assist in determining the initial degree of illness.[6] Pulse oximetry is noninvasive, inexpensive, and provides objective data regarding the degree of illness of a wheezing child. The oxygen saturation of any child with respiratory distress should be determined soon after ED arrival and supplemental oxygen should be provided if the oxygen saturation is 92% or less.

With the widespread use of pulse oximetry, physicians rarely need to obtain an arterial blood gas (ABG), especially if the sole purpose is to determine the partial pressure of oxygen. The acquisition of an ABG should be reserved for the child with severe disease to measure the extent of respiratory acidosis and hypercapnia. The timing of this test is important. For a severely ill child requiring admission to the ICU, it may be helpful to obtain this test after ED therapy has been initiated and after a clinical plateau has been reached. ABG results can then be used as a baseline that may be compared to subsequent results during the hospitalization. An apparently "normal" partial pressure of carbon dioxide ($Paco_2$) or pH may actually reflect severe disease. For example, a "normal" $Paco_2$ of 40 mm Hg in a child with extreme tachypnea and retractions suggests impaired ventilation and impending respiratory failure.

Peak Expiratory Flow Rate

Measuring the peak expiratory flow rate (PEFR) is a means of obtaining an objective assessment of exacerbation severity but it has limited utility in the evaluation of acutely ill children. Young children, in particular, may be unable to properly comply with this testing, and in one study just two thirds of children above age 5 years were able to complete PEFR testing during an asthma exacerbation.[3] Ideally, the PEFR should be determined with the child standing and the best of three attempts recorded. Therefore, moderately to severely ill or younger children may not be able to cooperate with this assessment.

Chest Radiographs

URIs marked by low-grade fever and coughing are common triggers of asthma exacerbations. These signs overlap with those found among children with pneumonia, making it difficult to determine the necessity of obtaining a chest radiograph (CXR) in the evaluation of an acutely wheezing child. No set of predictors has been found that can accurately identify children likely to have abnormalities on CXR.[7] Hyperinflation, interstitial markings, and atelectasis are common radiographic findings that may be seen in a wheezing child, but these should not result in initiating antibiotic therapy or other changes in management. More serious conditions associated with asthma such as pneumonia, pneumomediastinum, or pneumothoraces are much less common. Rarely is an unsuspected diagnosis made on the basis of a CXR in an acutely wheezing child, even if the child has never wheezed before.[8]

It should not be a routine practice to obtain CXRs for all wheezing children, even those who are wheezing for the first time or those who are being hospitalized.[8] CXRs should be considered for those with focal chest findings, fever, extreme distress, or history of choking. Reassessment after treatment to evaluate for the resolution of focal findings may further decrease the need for obtaining a CXR. This selective approach will be more cost-effective and lessen unnecessary radiation exposure and overuse of antibiotics. On the other hand, clinicians may have a lower threshold for obtaining CXRs for infants with first time wheezing because of a slightly greater likelihood of uncovering an anatomic abnormality.

Differential Considerations

Although most children with wheezing have asthma, other conditions must be considered. A differential diagnosis for childhood asthma is listed in Table 167-1. Of these conditions, bronchiolitis, laryngotracheobronchitis (croup), pneumonia, and gastroesophageal reflux are those that clinicians will encounter most often. Bronchiolitis is the one disease that is most commonly confused with asthma. Although the viruses associated with bronchiolitis infect children of all ages, clinical

Table 167-1 Differential Diagnosis of Asthma

CONDITION	DISTINGUISHING CHARACTERISTICS
Infections	
Bronchiolitis	Infant, preceding URI, seasonal, no history of atopy, no family history of asthma
Laryngotracheobronchitis (croup)	Inspiratory stridor, barky cough, fever, response to humidified air
Pneumonia	Focal wheezing, rhonchi, rales, grunting, fever
Tuberculosis	Diffuse adenopathy, weight loss, prolonged fever
Bronchiolitis obliterans	Prolonged cough and/or chest pain, inhalational exposure to toxin
Anatomic/Congenital	
Gastroesophageal reflux	Frequent emesis, weight loss, aspiration
Cystic fibrosis	Diarrhea, weight loss, chronic cough, salty sweat
Congestive heart failure	Rales, murmur, gallop, hepatosplenomegaly, cardiomegaly and/or pulmonary vascular congestion on chest radiograph
Tracheoesophageal fistula	Choking, coughing, cyanosis with feeds
Mediastinal mass	Chest pain, mediastinal density on chest radiograph
Vascular ring	Stridor, cyanosis, apnea, high-pitched brassy cough, dysphagia
Acquired	
Foreign body aspiration	History of choking, toddler, asymmetrical pulmonary examination, unilateral hyperinflation on chest radiograph
Anaphylaxis	Abrupt onset, urticarial rash, angioedema, history of allergies

bronchiolitis marked by wheezing is seen almost exclusively in those less than 12 months of age. Patients with bronchiolitis typically present between November and March. There is much clinical overlap between asthma and bronchiolitis and the two cannot be distinguished on physical examination findings alone. A complete discussion of bronchiolitis is included later in this chapter.

Croup may have a viral or allergic etiology and affects children from infancy through early school age. The hallmark of the disease is inflammation of the upper airway, resulting in a harsh, barky cough and inspiratory stridor. Symptoms are often worse at night. Asthma will not present with stridor alone, but a subset of children with croup may present with both stridor and wheezing and may be misdiagnosed with asthma.

Children with pneumonia may sometimes present with a component of wheezing, although respiratory rales or rhonchi are the usual auscultative findings. Infants and young children may also have high fever, cough, grunting, nasal flaring, or retractions, whereas more classic asymmetrical pulmonary findings are more easily detected in older children.

In addition to asthma, gastroesophageal reflux can account for recurrent wheezing in infants. In these children, poor lower esophageal sphincter function results in regurgitation and aspiration of gastric contents, leading to reflex bronchospasm. Young infants with recurrent wheezing, frequent "spitting up" of feeds, or failure to thrive should be referred for a diagnostic workup.

Management

For purposes of patient management, it is best to stratify children by degree of illness, based on the initial clinical assessment (Fig. 167-1). This will help to ensure the timely initiation of an appropriately aggressive approach for sicker children while minimizing adverse effects from unnecessary therapy among those with milder exacerbations. Of course, during the ED stay, the illness severity may change, making frequent examinations to assess response to therapy essential.

Mild Exacerbation

A mild exacerbation is characterized by alertness, slight tachypnea, expiratory wheezing only, a mildly prolonged expiratory phase, minimal accessory muscle use, and an oxygen saturation of greater than 95%. Children who are able to provide a PEFR should have a value greater than 70% of personal best. Patients with a mild exacerbation, especially those who were not receiving any asthma therapy prior to the ED visit, will usually require SABA therapy only. The Expert Panel of the National Heart, Lung, and Blood Institute (NHLBI) recommends that patients receive therapy every 20 minutes in the first hour of care.[2] Often, children with mild exacerbations improve promptly with just one or two SABA treatments. Many of these patients are managed without systemic corticosteroids. However, systemic corticosteroids may be given to those who are already undergoing a course of treatment with them prior to ED arrival or to those who do not respond promptly to SABA therapy (see later section on Moderate Exacerbation).

Due to its rapid onset of action, relatively long duration of action, and good safety profile, racemic albuterol has become the SABA of choice to treat children with acute asthma. Options for mode of delivery include small-volume nebulizers (NEBs) or metered dose inhalers with spacers (MDI-Ss), and recent studies have assessed the use of levalbuterol in this setting.

Nebulizers versus Metered Dose Inhalers with Spacers. There is considerable debate regarding the optimal method to deliver SABAs to children with acute asthma. About three fourths of pediatric emergency medicine physicians report using NEBs to administer SABAs, regardless of illness severity.[9] NEBs provide a passive means of receiving aerosolized medication. Precise coordination between respiration and aerosol delivery is not needed, and medications such as anticholinergics as well as humidified oxygen may be delivered concurrently with the SABA. However, delivery is inefficient, with only about 10% of the drug in the reservoir delivered to the small airways.[10–12] In addition, administration takes about 10 minutes, increasing respiratory therapy time and costs, and an external power source is needed, limiting portability.

On the other hand, spacers used with MDIs provide a reservoir of medication that is available to be inhaled. Therefore, precise coordination between actuation and inhalation is not needed and there is no need for breath-holding. Drug deposition in the oropharynx and systemic absorption is reduced with the employment of a spacer.[13] The decreased administration time associated with MDI-S use may result in reduced costs.[14–16] The portability of MDI-S allows older children to use them during the school day. Face mask–equipped spacers are available for children too young to use the spacer's mouth-

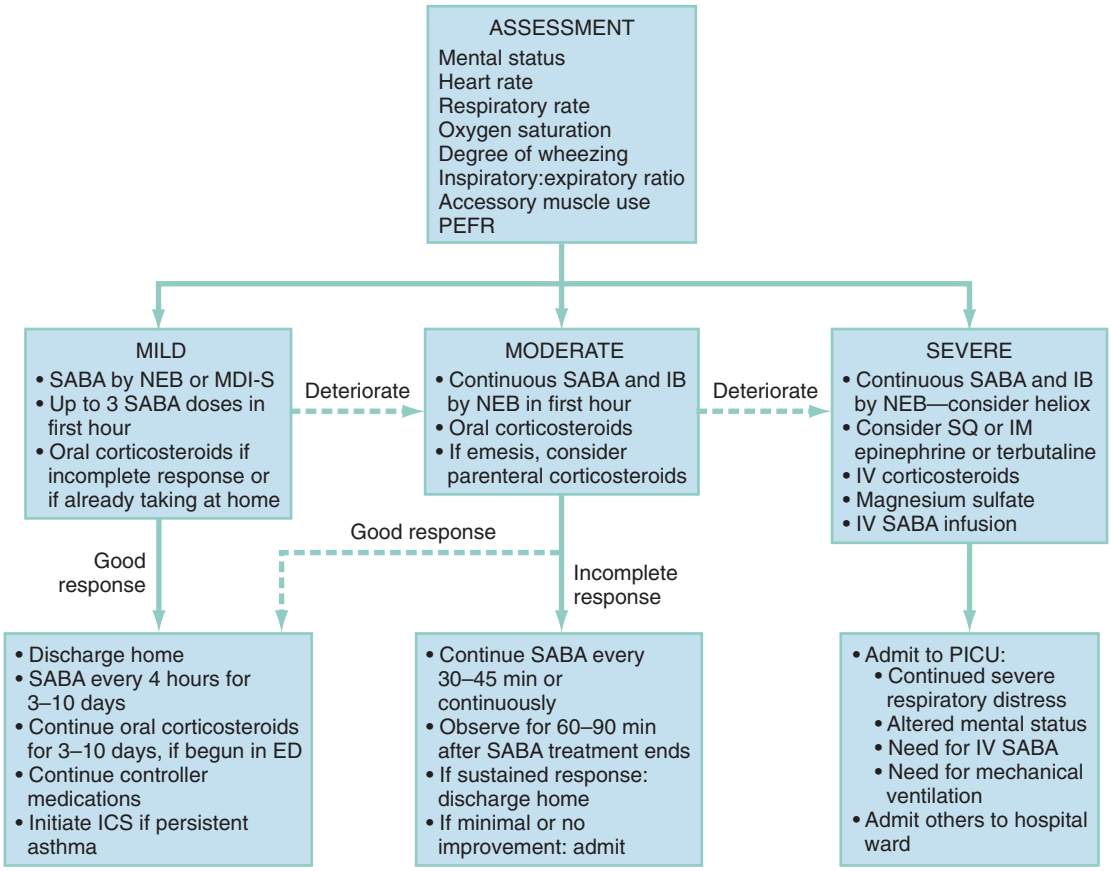


Figure 167-1. Emergency department management of acute asthma.

piece, although mouthpieces are preferable for older children to decrease nasal filtering of drug, which may reduce lung deposition.[17] Following each actuation, children should take five to eight breaths in order to completely empty the spacer.

These NEB disadvantages, along with the development and widespread use of spacers, have led investigators to assess the role of MDI-S to deliver SABAs in the ED. Numerous clinical trials and meta-analyses have consistently demonstrated that delivery by MDI-S is at least as effective as delivery by NEBs.[13,18–32] Among children 1 to 4 years old, MDI-S use was associated with a greater reduction in wheezing and a lower hospitalization rate.[16] One systematic review evaluated trials in which a total of 2066 children with acute asthma were randomized to receive SABAs by one of these two methods.[23] Those children treated with MDI-S had shorter ED length of stay and trended toward a reduced hospitalization rate. The American College of Chest Physicians and American College of Asthma, Allergy, and Immunology concluded that "both the NEB and MDI-S are appropriate for the delivery of SABA in the ED."[33] Thus, compared to NEBs, MDI-S administration has been shown to be equally effective for children of all ages,[16,22] with a wide range of illness severity and by multiple outcome measures.

Typically, when administering racemic albuterol by intermittent nebulization, a dose of 0.15 mg/kg is placed in the reservoir of a NEB. This dose is well established as one that is both effective and safe.[34,35] Optimal dosing for albuterol administered by MDI-S is not as well defined. A recent review assessed 10 randomized controlled trials comparing MDI-S to NEBs to deliver SABAs to children with acute asthma.[13] In some studies up to seven times more drug was placed in the NEB reservoir compared to that released by MDI-S, yet outcomes were similar. This reflects the inefficiency inherent with NEB delivery, with much drug being lost to the environment. Multiple puffs of SABAs delivered by MDI-S seems to be well tolerated, even by young children.[16,22] In one trial, children 1 to 4 years old treated with six puffs of albuterol by MDI-S had less tachycardia than those treated with 2.5 mg of albuterol by NEB.[16] The 2007 NHLBI guidelines state that "equivalent bronchodilation can be achieved either by high doses (4 to 12 puffs) of a short-acting beta agonist by MDI with a valved holding chamber … or by nebulizer"; they suggest a dose of four to eight puffs.[2] Table 167-2 provides recommended SABA doses stratified by the patient's weight.

Racemic Albuterol versus Levalbuterol. Another consideration in the use of SABAs is the potential role of levalbuterol. Racemic albuterol is an equal mix of the active R-albuterol and the inactive S-albuterol. R-albuterol produces bronchodilation as well as side effects such as tachycardia and tremors. S-albuterol was long thought to be inert. However, there is some evidence that S-albuterol may increase reactivity to histamine, have proinflammatory effects, and exhibit "characteristics of a typical contractile agent."[36–42] Further, there seems to be preferential retention of S-albuterol in the lungs of healthy volunteers[43–45]; this may account for diminished effectiveness with frequent dosing. Levalbuterol is pure R-albuterol without the S component. In theory, levalbuterol should be more effective than racemic albuterol at half the dose because there are no competing harmful effects from the S-isomer.

Studies assessing the use of levalbuterol to treat children with acute asthma have not consistently demonstrated this theoretical advantage. In the first of these clinical trials, levalbuterol (1.25 mg) was compared with racemic albuterol (2.5 mg) in the ED treatment of over 500 children with acute asthma.[46] The use of levalbuterol was associated with a

Table 167-2 Recommended Doses of Medications for Acute Asthma

Albuterol	0.15 mg/kg/dose (0.03 mL/kg/dose, max 1.0 mL) So:	
	<10 kg	2.5 mg (0.5 mL)
	11–19 kg	3.75 mg (0.75 mL)
	>20 kg	5 mg (1.0 mL)
Continuous albuterol	1.0 mg/kg/hr by nebulization (max 20 mg/hr)	
	<10 kg	10 mg/hr (2 mL/hr)
	10–20 kg	15 mg/hr (3 mL/hr)
	>20 kg	20 mg/hr (4 mL/hr)
Albuterol by MDI	Dose is not well established	
	<10 kg	2–4 puffs
	11–19 kg	4–6 puffs
	>20 kg	6–8 puffs
Levalbuterol	Half the recommended albuterol doses	
Ipratropium bromide	<20 kg	250 µg/dose
	>20 kg	500 µg/dose
L-epinephrine (1:1000) or terbutaline (1.0 mg/mL)	0.01 mL/kg/dose SC or IM (max 0.4 mL) May be repeated every 10–15 min	
IV terbutaline	10 µg/kg bolus over 10 min, then 0.1–0.3 µg/kg/min infusion Every 30 min, may increase infusion by 0.3 µg/kg/min to a max of 5 µg/kg/min	
Prednisone	2 mg/kg (max 60 mg), in ED 1 mg/kg/dose bid, home therapy	
Dexamethasone	0.6 mg/kg PO, 2 doses 24 hr apart	
IV methylprednisolone	1–2 mg/kg (max 125 mg)	
IV magnesium sulfate	50–75 mg/kg over 20 min (max 2.5 g)	

bid, twice daily; ED, emergency department; IM, intramuscularly; IV, intravenous; max, maximum; MDI, metered-dose inhaler; PO, orally; SC, subcutaneously.

decreased need for hospitalization. However, the baseline hospitalization rate in this study was quite high even though patients with all degrees of illness severity were enrolled. Subsequently, three other randomized trials comparing the ED use of the two drugs have failed to find a levalbuterol benefit.[47–49] These three studies analyzed children with a wide range of illness severities and baseline hospitalization rates and used various outcome measures such as asthma scores, pulmonary function tests, and hospitalization rates. Racemic albuterol was demonstrated to be as effective as levalbuterol under each of these circumstances.

To date, there are no published data assessing the use of continuously nebulized levalbuterol in the ED treatment of children with asthma. The acquisition cost of levalbuterol is over 10 times greater than racemic albuterol.[49] Until there are more compelling data to demonstrate conclusively that the additional costs of levalbuterol are offset by the need for fewer nebulizations, decreased length of ED or hospital stay, or decreased need for hospitalization, racemic albuterol should remain the drug of choice for children with acute asthma exacerbations.

Disposition. Most children with a mild exacerbation will be able to be discharged home. Those sustaining clinical improvement 60 minutes after the most recent SABA treatment may be discharged. SABAs should be continued for the next 3 to 10 days. If systemic corticosteroid therapy was administered in the ED, it should also be continued for 3 to 10 days. Children should continue all other asthma controller medications, including inhaled corticosteroids (ICS).

For those who are not already receiving ICS, it is unclear if prescribing them at ED discharge leads to improved short-term outcomes. Among adult asthmatics discharged from the ED after acute asthma exacerbations, the addition of inhaled flunisolide did not lead to improved outcomes.[50] Of note, though, compliance with the inhaled medication was low and many patients were lost to follow-up. On the other hand, adults randomized to inhaled budesonide following ED discharge had a marked decrease in relapse rates, frequency of SABA use, and asthma symptoms.[51] A review concluded that there is "insufficient evidence that ICS provide additional benefits" when added to systemic corticosteroids at ED discharge.[52] Pediatric emergency medicine physicians rarely prescribe ICS at ED discharge, even to children who have persistent asthma.[53]

Rather than prescribing ICS to prevent ED relapse, emergency physicians should consider longer-term goals for those with persistent disease. National guidelines state that ICS are the medications of choice when initiating long-term controller therapy for children with persistent asthma.[2] Further, these drugs are safe and well tolerated at recommended doses. Longitudinal studies show that daily use of ICS may decrease growth velocity, but these changes are small and reversible.[54,55] Therefore, ED physicians should identify children who, during the preceding month, have had frequent asthma symptoms, nighttime awakenings, and the need for frequent use of SABAs for asthma control. As stated by national asthma guidelines, initiation of ICS therapy at ED discharge "should be considered" for these patients.[2] Those already taking low doses of daily ICS may benefit from an increase in dosing.

In addition to prescribing medications, ED physicians should also provide asthma education at discharge. Some EDs employ the use of a video or DVD to provide standardized information to families while they undergo ED therapy. This education should include how to identify and avoid asthma triggers, a written asthma action plan explaining proper steps to take in response to an asthma flare, review of discharge medications, and proper MDI-S use. Also, follow-up asthma care within 1 to 4 weeks should be arranged.

Summary. For children with mild asthma exacerbations, racemic albuterol should be administered every 20 minutes, as needed (Fig. 167-1). Most children will respond promptly to therapy and be well enough to be discharged home after 1 or 2 treatments. Systemic corticosteroids may be considered for those who exhibit a suboptimal response to SABAs (see later section on Moderate Exacerbation). NEBs or MDI-S are each reasonable options to deliver SABAs intermittently. Table 167-2 lists recommended doses for SABAs and other asthma medications in the ED, and Table 167-3 provides a recommended strategy for SABA administration. Rather than base the method of delivery on the issue of efficacy, clinicians should assess other factors. The answers to questions such as the number of treatments a child is likely to need, the anticipated cooperation with a given delivery method, the need to deliver concurrent medications, and costs will help to guide decision-making.

Moderate Exacerbation

Children who are alert but very tachypneic with wheezing throughout expiration, an inspiratory:expiratory ratio of 1:2, and significant use of accessory muscles are experiencing a moderate asthma exacerbation. Typically, the oxygen saturation will be 92 to 95% and the PEFR will be 41 to 70% of the personal best. As with children experiencing more mild attacks,

Table 167-3 Short-Acting Beta$_2$-Agonists in Acute Asthma

	MILD	MODERATE	SEVERE
Delivery method	Intermittent NEB or MDI-S	Continuous by NEB for 1 hour, then reassess	Consider SC or IM Continuous by NEB
Comments	Most patients will need 1–2 treatments Allows for MDI-S teaching No IB needed	Not superior to MDI-S Easier to adhere to NHLBI guidelines for the first hour of therapy Concurrent IB therapy is more easily delivered	Better outcomes in severe asthma

IB, ipratropium bromide; IM, intramuscularly; MDI-S, metered dose inhaler with spacer; NEB, nebulizer; NHLBI, National Heart, Lung, and Blood Institute; SC, subcutaneously.

the cornerstone of therapy is aggressive SABA therapy. In addition, other medications such as ipratropium bromide (IB) and corticosteroids should be added and consideration should be given to delivering SABAs continuously.

Anticholinergics. Stimulation of airway cholinergic receptors results in reflex bronchoconstriction, which may be blocked with the use of anticholinergic agents such as IB. This medication is available as an MDI as well as a solution for nebulization that may be mixed directly with racemic albuterol. The MDI formulation should not be given to patients with allergy to peanut or soy because it contains soya lecithin; this is not a concern with the solution for nebulization.

Studies have shown that use of SABAs with IB is more effective than SABAs alone.[56,57] In a randomized, double-blind clinical trial, three doses of IB administered concurrently with the first three SABA treatments were shown to be superior to just one dose of IB.[56] In another study, over 400 children were randomized to receive racemic albuterol and prednisone alone or that therapy plus IB.[57] Those judged to be moderately ill did not experience an IB benefit. However, among those with an initial PEFR less than 50% predicted, the use of IB resulted in a significantly lower hospitalization rate. A recent systematic review and meta-analysis compared the use of SABAs plus anticholinergics with SABAs alone among children older than 18 months.[58] In the 16 trials assessed, combination therapy was associated with significantly lower hospitalization rates and improvements in asthma scores and pulmonary function testing. These investigators concluded that multiple doses of IB added to SABAs should be standard treatment for children with moderate to severe asthma exacerbations.

Clinical benefits following IB use may be delayed for up to 60 minutes.[56] However, it is inexpensive and since less than 1% is absorbed systemically, it is virtually free of adverse effects.[58] IB should be administered to children with moderate exacerbations. Three doses may be mixed with racemic albuterol and delivered concurrently and continuously by NEB in the first hour of care (see Table 167-3). This means of administration, although not superior to delivery by MDI-S, will help to ensure compliance with the goal of the equivalent of three NEB treatments in the first hour of care.[2] Alternatively, four to eight puffs of IB may be given every 20 minutes in the first hour of care,[2] but these children will also need to receive a substantial number of puffs of SABAs and, as clinicians care for other patients, there may be delays in receiving appropriately aggressive therapy.

Systemic Corticosteroids. There is compelling data to show that the prompt use of corticosteroids can decrease the need for hospitalization and that they should be used routinely for patients with moderate disease.[5,59–64] Clinicians must decide the optimal agent and route of administration.

Oral versus Parenteral. Two early clinical trials established the efficacy of parenterally administered corticosteroids in the ED. Compared with those treated with placebo, adults treated with intravenous (IV) methylprednisolone had a lower hospitalization rate,[59] as did children treated with intramuscular (IM) methylprednisolone.[60] Scarfone and colleagues were the first to demonstrate the efficacy of orally administered corticosteroids in this setting.[5] Children treated with frequent SABAs and oral prednisone had a reduced need for hospitalization compared to those treated with SABA therapy alone. Further, a meta-analysis determined that compared to placebo, oral corticosteroids were effective in reducing the need for hospitalization among children with acute asthma exacerbations.[63]

There have been few clinical trials directly comparing oral and parenteral therapy. In one small study, there were no differences in any outcome measures for children in the ED with moderate to severe asthma who were treated with equal doses of either IV or oral methylprednisolone.[65] The most recent NHLBI guidelines recommend using oral corticosteroids since this form of administration is less invasive and the benefits seem to be equivalent to parenteral therapy.[2,66,67] Further, oral corticosteroid therapy is inexpensive, the drugs are rapidly and completely absorbed, and this mode of administration provides the potential for out-of-hospital administration either at home or in a physician's office.

Prednisone versus Dexamethasone. As with SABAs, clinicians have a choice in the specific corticosteroid to be used. Oral prednisone has been the drug of choice in this setting. Significant clinical benefits begin 2 hours after administration, are most pronounced among the sickest children, and result in a decreased need for hospitalization.[5] Dexamethasone phosphate may be given orally or parenterally and has a substantially longer (36–72 hours) half-life than prednisone (18–36 hours).[68,69] Investigators recently treated children in the ED with either 0.6 mg/kg of dexamethasone or 2 mg/kg of prednisone in a randomized fashion.[69] Those in the dexamethasone group were provided one additional dose to take the following day, while those in the prednisone group were given a prescription for 4 additional days of prednisone. There were no differences in hospitalization or relapse rates or symptom persistence. Significantly fewer dexamethasone patients vomited the study drug in the ED and reported noncompliance with it after ED discharge. Of note is that Orapred, a more palatable form of oral prednisone, was not used in this study.

These data suggest that either dexamethasone or prednisone may be used in the treatment of moderately ill children with acute asthma. Given that clinical benefits from corticosteroids are delayed and that all moderately ill children will require corticosteroids whether or not they require hospitalization, they should be administered as soon as possible after ED arrival in an attempt to hasten clinical improvement and perhaps prevent the need for hospitalization.[61,64] Since SABAs are administered by inhalation and corticosteroids may be given orally, most children with a moderate asthma exacerbation can be managed without the insertion of an IV line. This avoids unnecessary pain and anxiety, as well as the delays in drug administration associated with IV line insertion. IM therapy is a reasonable option for children who vomit orally administered corticosteroids yet do not require an IV line for other reasons.

Inhaled Corticosteroids. The use of ICS for the ED treatment of acute asthma is an area of ongoing research. In three clinical trials, ICS were compared to oral prednisone in the ED setting.[70–72] Scarfone and colleagues treated children with either nebulized dexamethasone or oral prednisone.[70] The two groups had similar rates of hospitalization, although there was a trend toward greater rate of improvement among the dexamethasone-treated children. A potential limitation to the widespread use of nebulized dexamethasone is that it contains sodium bisulfite, a preservative that may induce wheezing among allergic individuals. Budesonide is an ICS that has a high topical activity and low systemic absorption[73] and is effective in the treatment of children with croup.[74,75] Investigators from India found that three nebulized doses of budesonide was superior to one dose of prednisone.[71] In another trial, fluticasone-treated children were more likely to be hospitalized and experienced a significantly smaller degree of improvement compared with those treated with prednisone.[72] Finally, investigators determined that children treated in the ED with inhaled triamcinolone had lower hospitalization rates and relapse rates compared to those treated with either prednisone or IV corticosteroids.[76]

Rather than replacing systemic corticosteroids, other investigators have assessed whether or not the addition of ICS to systemic therapy results in clinical benefits; however, few such studies have been performed in children and few have assessed the need for hospitalization.[77–79] Thus, the niche for ICS in the ED treatment of acute asthma is still being defined. Additional areas for further investigation include determining the optimal agent, the proper dose, the appropriate mode of delivery, and defining the patient population most likely to benefit from this therapy. At this time, there is no proven role for the routine use of ICS in the ED setting. Due to its greater bioavailability and proven benefits, moderately ill children should receive systemic corticosteroid therapy.

Intermittent versus Continuous. Children requiring very frequent intermittently nebulized albuterol may benefit from receiving albuterol continuously instead. In one clinical trial among asthmatic children, patients were randomized to receive the same total dose of albuterol nebulized either intermittently or continuously, over 2 hours.[80] Those in the continuous group had a greater mean improvement in their asthma scores and significantly less respiratory therapy time, although there were no differences in mean PEFR or admission rates. A systematic review found that those treated with continuously nebulized SABAs had lower rates of hospitalization, greater improvements in pulmonary function tests, and similar rates of adverse events compared with those treated intermittently.[81]

Perhaps the greatest advantage of continuous over intermittent therapy is one of a practical nature: it allows greater compliance with the goal of delivering the equivalent of three intermittent albuterol treatments in the first hour of care.[2] In addition, this method will result in less respiratory therapy time and costs, has been shown to be safe,[82,83] and may benefit the sickest patients the most.[84,85] On the other hand, young children may not tolerate a face mask for prolonged periods.

Many clinicians find it helpful to determine the total of racemic albuterol that would be delivered if three treatments were to be given intermittently over an hour, place that total dose in the NEB reservoir, and administer it continuously over an hour. Alternatively, a dose range may be used based on the child's weight (see Table 167-2).

Summary. A suggested approach to the management of children with moderately acute asthma is as follows. Supplemental oxygen should be provided if the initial oxygen saturation is 92% or less in room air. Albuterol and IB should be administered continuously by nebulization for 1 hour (see Fig. 167-1). This will ensure that an appropriate amount of each is delivered in the first hour of ED care. As soon as possible after ED arrival, a single dose of either oral prednisone or dexamethasone should be given. IM dexamethasone is an option for children who vomit the initial oral corticosteroid dose within 15 minutes of its administration or who vomit repeated doses.

After 1 hour of therapy, a clinical reassessment should be made; evaluation at this time is better than the initial assessment at predicting the need for hospitalization.[86] At this point, patients can generally be grouped into one of three categories: markedly improved, not improved or worse, or slightly improved. Children who are markedly improved may be observed without SABAs to ensure that there is no clinical deterioration. It is wise to delay a disposition decision until at least 60 minutes after the most recent SABA treatment so that a clinical relapse may be noted. In making the disposition decision, the clinician must determine the patient's physical examination findings and also consider the frequency of prior hospitalizations and ED visits and issues such as compliance and support systems. The medications and education to be provided at ED discharge are the same as outlined for those with a mild exacerbation.

Children who remain moderately ill after the first hour must continue to be treated aggressively with SABAs, either continuously or with frequent administration of intermittent therapy. If after 2 hours subjective and objective measures reveal that the degree of respiratory distress is unchanged or worse, then hospitalization is warranted. On the other hand, there will be a subset of patients who demonstrate some degree of clinical improvement at the 2-hour assessment but are not yet well enough to be sent home. One study showed that among prednisone-treated children who would have been hospitalized after 2 hours of ED therapy, less than half were actually hospitalized when aggressive SABA therapy was continued for an additional 2 hours and none returned to the ED within 48 hours of discharge.[5] Presumably, the onset of prednisone's effects allowed these children to avoid hospitalization. Therefore, continuing to treat such children with SABAs for a total of 3 to 4 hours, perhaps in an ED observation area, would be expected to avoid the need for many hospitalizations.

Severe Exacerbation

A severe exacerbation is characterized by restlessness or lethargy, extreme tachypnea and tachycardia, wheezing that is audible without a stethoscope, an inspiratory:expiratory ratio exceeding 1:2, significant use of accessory muscles, and an oxygen saturation less than 92%. Some older children with a severe exacerbation may have bradypnea because of a prolonged expiratory phase and wheezing may not be audible if aeration is markedly decreased. The PEFR will typically be less than 40% predicted, although most children will be too ill to use the peak flow meter.

Figure 167-1 provides an outline for the approach to managing severely ill children. They should be attached to a cardiorespiratory monitor and blood pressure cuff with continuous monitoring of oxygen saturation by pulse oximeter. As with the moderately ill child, supplemental oxygen and continuously nebulized albuterol and IB should be provided soon after arrival. To achieve an oxygen saturation of 95% or greater, it may be necessary to employ a non-rebreathing face mask. Severely ill children may be too sick to tolerate oral medications and will likely need an IV catheter for other indications. Therefore, an IV line should be established as soon as possible and a dose of methylprednisolone given.

Subcutaneous or Intramuscular Therapy. For children with very poor inspiratory flow, nebulized SABAs may not be effectively delivered to the smallest airways. Short inspiratory time, prolonged expiration, and low inspiratory pressures will impair delivery of inhaled medications. Here, subcutaneous (SC) or IM terbutaline or epinephrine should be used, especially if an IV line is yet to be established. Terbutaline may be preferable because it is a more selective agent with fewer side effects such as tremors, vomiting, or palpitations. Very ill anxious young children who are uncooperative with the inhalation treatments may also benefit from this therapy. There are no data to suggest one mode of administration is superior to the other, although IM therapy is recommended for children with bronchospasm due to anaphylaxis.[87] SC or IM therapy may be repeated every 10 to 15 minutes, as needed.

Magnesium Sulfate. There is accumulating evidence that magnesium sulfate may benefit adults and children with severe asthma. Recent meta-analyses determined that use of magnesium resulted in improved outcomes for both adults[88] and children.[89] In two separate trials, children with a suboptimal response to initial SABA therapy who were randomized to receive magnesium had significantly greater improvements in pulmonary function studies compared to those treated with placebo.[90,91] In contrast, Scarfone et al conducted a randomized, controlled trial assessing the use of 75 mg/kg of magnesium in asthmatic children.[92] They sought to determine if magnesium was efficacious as a component of *initial* therapy for children with moderate to severe exacerbation, without waiting to judge response to early albuterol therapy. No magnesium benefits were found for this population.

Magnesium is inexpensive and has minimal adverse effects.[93] The most common adverse effect is hypotension; this may be avoided by infusing the dose over 20 minutes. The most recent NHLBI guidelines recommend the consideration of magnesium for select patients; this represents a key difference from prior reports. At this point, existing literature indicates that magnesium should be considered for moderately ill patients who have a suboptimal response to SABAs, IB, and corticosteroids as well as for all severely ill children.

Intravenous Short-Acting Beta$_2$-Agonists. The NHLBI guidelines conclude that there are insufficient data to make recommendations regarding the use of IV SABAs.[2] Similarly, a recent systematic review of randomized, controlled trials failed to support this practice.[94] However, of the 15 studies included, just 3 were performed in children and just 3 assessed the combination of IV and inhaled SABAs compared to inhaled SABAs alone.[95–97] Rather than definitively demonstrating lack of efficacy, these data highlight the need for more and larger clinical trials.

Potential adverse effects from the use of IV SABAs are substantial and include dysrhythmias, hypertension, and hypokalemia. Due to the concern for toxicities, there is no role for initiating therapy with IV SABAs, even for severely ill children. However, for those who are poorly responsive to the interventions outlined above, with impending respiratory failure, the risk-benefit ratio shifts toward their use.

Heliox. Heliox is a low-density mixture (often in a 70:30 ratio) of helium and oxygen that results in less turbulent flow through narrowed airways. Theoretically, its use is associated with decreased work of breathing, less respiratory muscle fatigue, and a lower likelihood of ventilatory failure. In one trial, children with acute severe asthma treated with heliox had a significantly greater decrease in pulsus paradoxus and dyspnea index, and increase in PEFR, compared to others.[98] A more recent study compared the use of heliox versus oxygen alone to deliver continuously nebulized SABAs.[99] At 240 minutes, children in the heliox group had greater improvement as assessed by decreased asthma scores and need for hospitalization. However, after reviewing 10 clinical trials assessing the use of heliox, investigators concluded that there is insufficient evidence to support the use of heliox for all patients with asthma,[100] although it may be considered for severely ill children who are not responding to more conventional therapy.[2]

Mechanical Ventilation. When making decisions about the need for mechanical ventilation of the severely ill patient, one must assess the entire clinical picture, including duration of wheezing, illness severity, response to therapy, as well as ABG results. Making this decision based on the ABG results alone should be discouraged. For example, the child with a pH of 7.10 and a $Paco_2$ of 55 who shows marked improvement with IV SABA therapy may not require ventilatory assistance, yet the one with a pH of 7.18 and a $Paco_2$ of 50 who appears fatigued and is not responding to therapy probably does. Ketamine is a bronchodilator and is the drug of choice for sedation and analgesia of the asthmatic child who requires intubation.

With mechanical ventilation, air trapping with resultant pneumothorax is a major concern. *Permissive hypercapnia* is a term used to describe a strategy of minimizing tidal volumes and respiratory rate in order to minimize peak pressures. A degree of hypercapnia is accepted and may be treated with sodium bicarbonate. Enough expiratory time must be allowed for air exit from the lungs.

There are many potential therapies that are not recommended for the treatment of acutely ill asthmatic children. These include methylxanthines such as aminophylline, the routine use of antibiotics in the absence of known bacterial infection, aggressive hydration, chest physiotherapy, mucolytics, sedation, and noninvasive ventilation.[2]

Summary

Asthma is one of the few diseases in which there have been increases in prevalence, morbidity, and mortality in the past 2 decades. More than ever, physicians need to be vigilant in the recognition and treatment of children with acute asthma. In all cases, appropriate doses of albuterol should be given promptly and early use of multidose IB and corticosteroids is indicated for those who are moderately or severely ill. Aggressive use of SC or IM terbutaline, continuously nebulized albuterol, and magnesium is warranted for those who are severely ill, while IV SABAs are reserved for those not responding to the above therapy.

■ BRONCHIOLITIS

Perspective

Background

Bronchiolitis is an acute infectious disease that results in inflammation of the small airways in children younger than 2 years. This process manifests clinically as wheezing and increased work of breathing along with the typical signs and symptoms of a URI. Nearly all children are affected by the viruses that cause bronchiolitis at least once during their first 2 years of life, but it is more common for infants younger than 12 months to develop clinical bronchiolitis.

Epidemiology

Bronchiolitis is a seasonal disease, with most cases occurring between November and April in temperate climates. It

accounts for approximately 3% of all ED visits in the United States.[101] Overall, approximately 19 to 27% of children presenting to an ED with bronchiolitis are admitted for inpatient management.[101,102] This disease accounts for more than 20% of acute care hospitalizations for children younger than 1 year, and the total cost for all bronchiolitis-related hospitalizations is more than $500 million per year.[103] Hospitalization rates vary depending on many factors, and these rates have increased dramatically over the past 2 decades. In the United States, Hispanic children and those of Alaskan or American Indian descent are more likely to be admitted to the hospital after presenting with bronchiolitis.[101,104] Males and younger infants are also more likely to need admission.[105] Other factors that seem to be associated with hospitalization are poverty, household crowding, exposure to environmental tobacco smoke, and day care attendance.[106] Clearly, underlying chronic medical conditions, such as congenital heart disease or chronic lung disease, lead to a more severe course in patients with this disease.

Bronchiolitis is rarely fatal, with an average mortality rate of 2.0 per 100,000 live births in the United States. Low birth weight (<2500 g), low 5-minute Apgar score, high birth order, and young maternal age are all associated with an increased risk of death.[107,108] Breast-feeding, on the other hand, appears to be associated with a less severe clinical course.[109]

Distinguishing Principles of Disease

Etiology

Many viruses are implicated as the underlying cause for bronchiolitis. Respiratory syncytial virus (RSV) is the most common agent identified in children diagnosed with this disease, estimated to cause up to 70% of cases in previously healthy children.[110] Other viruses commonly isolated are parainfluenza, human metapneumovirus, influenza, adenovirus, and rhinovirus.[110–115]

Pathophysiology

Most respiratory viruses that cause bronchiolitis in children are transmitted from one host to another via fomites spread from hand to nose or via droplets produced by sneezing or coughing of respiratory secretions. Shedding of the virus often begins prior to the onset of significant clinical symptoms and can continue for 2 to 3 weeks in an immunocompetent infant. The typical incubation period is 2 to 8 days from the time of initial contact.[116]

In an infected patient, viral replication often begins in the epithelial cells of the upper airway before spreading to the mucosal surfaces of the lower respiratory tract. The infected epithelial cells are generally destroyed via lysis or apoptosis, which results in the desquamation of these cells and release of host inflammatory mediators.[117] Affected lungs demonstrate epithelial cell necrosis, monocytic inflammation and edema of the peribronchial tissues, and mucus and fibrin plugging of the distal airways upon histologic examination. These findings translate into the clinical findings of wheezing and lower airway obstruction in an infant with bronchiolitis. Younger infants, whose distal airways are of smaller caliber and whose immune systems lack active immunity to most respiratory viruses, are prone to more severe clinical symptoms.[106] Severe lower airway obstruction leads to air-trapping and atelectasis, resulting in mismatched ventilation and perfusion and hypoxemia. In addition, younger infants are at increased risk for fatigue, leading to hypercarbia and respiratory failure.

Clinical Features

Patient History

Typically, infants with bronchiolitis are less than 12 months of age and present during the winter months. The first symptoms are generally those of an upper respiratory infection, such as nasal congestion and copious rhinorrhea. This is followed within a few days by development of a tight cough, often associated with difficulty feeding. Some parents will report the presence of audible wheezing as well. A history of fever is common, though not universal, with one study reporting fever in approximately one third of patients admitted with bronchiolitis.[118] Very young infants may present with a history of apnea, and this may precede the onset of typical symptoms of respiratory infection. It is essential to ascertain information regarding the infant's hydration status, including the amount and frequency of oral intake, urine output, vomiting, and diarrhea.

A child's past medical history is important as well. Specifically, comorbidities such as congenital heart disease, chronic lung disease, or prematurity can significantly impact the clinical course of bronchiolitis. A past history or family history of wheezing or atopy may provide clues in differentiating bronchiolitis from asthma, particularly in the older infant. Other elements of the patient history that may be helpful are whether the child attends day care or if there are household contacts with respiratory symptoms.

Physical Examination

As in any pediatric patient, an assessment of vital signs and general appearance is crucial to the evaluation of an infant with bronchiolitis. Common vital sign abnormalities include fever, tachycardia, tachypnea, and hypoxia. Pulse oximetry is noninvasive, inexpensive, and provides objective data regarding the degree of illness of a wheezing child. The oxygen saturation (Sao_2) of any moderately or severely ill infant should be obtained soon after ED arrival as an adjunct to the physical examination. With the use of pulse oximetry, an ABG is generally unnecessary to assess a patient's oxygenation. Thus, the acquisition of an ABG should be reserved for those with severe disease to measure the extent of hypercarbia and respiratory acidosis. Irritability or lethargy may be present, especially in those infants with more severe disease. Nasal flaring and retractions are visible signs of respiratory distress that may be present. Lung auscultation often reveals decreased air movement, rales, rhonchi, wheezing, and an increased ratio of expiratory to inspiratory times. Using these physical examination findings, the clinician may stratify patients into mild, moderate, and severe categories of disease (Table 167-4).

The combination of poor feeding and increased insensible fluid losses often impacts an affected infant's hydration status. A careful assessment of the anterior fontanel, mucous membranes, and skin turgor can assist in determining whether an infant is dehydrated.

Complications

Clinicians caring for children with bronchiolitis need to understand the typical course of the disease, both for their ability to accurately diagnose and manage these patients and to advise parents about the recovery phase. The worst phase of the illness that may necessitate hospitalization generally occurs in the first few days, and median length of hospital stay has been reported to be between 2 and 3 days.[119,120] However, the entire course of illness can last much longer, with a median duration of 12 days.[121] Coughing and noisy breathing, in particular, can last for more than 4 weeks.

Table 167-4 Suggested Bronchiolitis Assessment Tool

	MILD	MODERATE	SEVERE
Feeding	Normal	Less	Poor
Sao_2 in room air	≥95%	92–94%	<92%
Respiratory rate (breaths/min)	<60	60–70	>70
Retractions	None/minimal	Intercostal	Substernal
Accessory muscle use	None	None	Neck or abdominal
Wheeze	None/minimal	Moderate expiratory	Severe inspiratory/expiratory, audible without stethoscope
Air exchange	Good, equal BS	Localized, decreased BS	Multiple areas of decreased BS

BS, breath sounds.

Bacterial acute otitis media (AOM) is the most common condition associated with bronchiolitis, with a prevalence of up to 60%.[122] The bacterial pathogens are similar to those recovered in other children with AOM; thus, it should be treated according to standard recommendations. Other concurrent bacterial infections are rare. In one study of more than 2000 children hospitalized with RSV bronchiolitis, approximately 1% also had a urinary tract infection (UTI).[123] Pathologic bacteremia and meningitis were not found in any of these patients. Similar rates of UTI without bacteremia are found in febrile children with clinical bronchiolitis, with or without documented RSV infection.[124] Infants less than 8 weeks old with fever and bronchiolitis present a unique dilemma for ED clinicians. The rate of serious bacterial infections (SBIs), defined as UTI, bacteremia, bacterial meningitis, or bacterial enteritis, among all febrile infants less than 8 weeks of age is reported to be up to 12%.[125,126] However, in infants with documented RSV infection or clinical bronchiolitis at the time of ED presentation, the rate of SBI is substantially lower.[127,128] In a large, prospective, multicenter study, Levine and colleagues reported that 7% of febrile infants less than 61 days of age who were RSV positive had a concurrent SBI, compared with 12.5% of those who were RSV negative. Of the patients with SBIs, most (82%) had a UTI. Bacteremia was rare and only occurred in infants less than 1 month of age. None of the RSV-positive infants had bacterial meningitis. As a result, most would advocate performing a workup for UTI in febrile infants between 1 and 2 months of age who are known to be RSV positive or have clinical bronchiolitis. Additional testing to obtain cultures of cerebrospinal fluid and blood may be done selectively. Similarly, these infants may not require empiric antibiotic therapy for presumed SBIs. On the other hand, in the first month of life, all febrile infants should undergo testing to evaluate for SBIs and be empirically treated with antibiotics regardless of RSV status.

Apnea is commonly reported in young infants with bronchiolitis, especially those who are admitted for inpatient management. Eight percent of admitted patients have a history of apnea, and nearly 3% will develop apnea during the hospital stay.[129,130] Risk factors for developing in-hospital apnea include age less than 1 month in full-term infants, postconceptional age less than 48 weeks in preterm infants, and a history of apnea prior to admission. The absence of all of these risk factors has a high negative predictive value for the development of in-hospital apnea.[130]

The long-term outcomes after bronchiolitis have also been examined extensively. It is clear that those who experience the disease in infancy have a higher prevalence of lower respiratory diseases, including asthma, in adolescence and adulthood.[131–134] Whether there is a causal relationship remains undetermined.

Differential Considerations

Asthma is the condition that has the most clinical overlap with bronchiolitis. Physical examination findings alone cannot distinguish the two. Younger age, presentation during winter months, antecedent URI symptoms, and the absence of prior or family history of atopic disease and wheezing all suggest bronchiolitis as the cause of wheezing in an individual patient. Some infants will have clinical features consistent with both conditions. For example, a 12-month-old may present in July with a URI and wheezing for the first time. For this child, a clinician may choose to initiate therapy for acute asthma. A complete discussion of other conditions that must be differentiated from bronchiolitis is included earlier in this chapter and in Table 167-1.

Diagnostic Strategies

Bronchiolitis should be diagnosed primarily on the basis of history and physical examination. Viral diagnostic testing can be performed on nasopharyngeal secretions using enzyme-linked immunosorbent assays, indirect fluorescent antibody detection, polymerase chain reaction, or viral culture. Generally, this is not warranted for those patients for whom outpatient management is sufficient. However, identifying a viral respiratory pathogen can be useful in certain situations. For example, if test results can be obtained rapidly, identifying a viral etiology may eliminate the need for further laboratory evaluation for infants who present with fever. In addition, having a specific viral diagnosis can allow for the appropriate cohorting of patients admitted for inpatient management, thus decreasing nosocomial transmission among patients and staff. Providers should exercise contact and droplet precautions until the causative viral agent is identified.

There is tremendous variability in the use of diagnostic imaging; in one series, a CXR was obtained for more than 70% of infants hospitalized with bronchiolitis.[135] In children with clinical findings that are typical for bronchiolitis, however, radiographic imaging is rarely necessary. Hyperinflation, atelectasis, and peribronchial cuffing are the findings most commonly associated with this disease. In ambulatory patients with acute lower respiratory infections, obtaining a CXR does not affect clinical outcome,[136] and this practice has been associated with increased usage of unnecessary antibiotics.[135] Further, the likelihood of a CXR revealing findings inconsistent with the clinical diagnosis of bronchiolitis is less than 1%.[137] Diagnostic imaging may be helpful in patients with severe distress, significant hypoxia, or an atypical presentation or clinical course. In summary, as recommended by the American Academy of Pediatrics (AAP) Subcommittee on Diagnosis and Management of Bronchiolitis, "clinicians should diagnose

bronchiolitis and assess disease severity on the basis of history and physical examination. Clinicians should not routinely order laboratory and radiologic studies for diagnosis."[138]

Management

Therapy

While the diagnosis of bronchiolitis is fairly straightforward, the management of children with the disease often presents clinicians with confusing and controversial dilemmas. The literature is often contradictory, making it difficult to reach a consensus. As a result, there exists wide practice variation in the management of bronchiolitis.[139–141] However, it is clear that a consistent, evidence-based approach to this disease can lead to more efficient and effective care.[130,142–145] Supportive care, such as providing hydration and supplemental oxygen, is the cornerstone of therapy for affected children.[138] A management strategy, stratified by the patient's initial degree of illness, is outlined in Figure 167-2.

SABAs are the treatment of choice for children with wheezing due to asthma. However, the evidence supporting their use in wheezing caused by bronchiolitis is less favorable than for asthma. In a recent meta-analysis of 22 clinical trials, a small short-term benefit in clinical score was observed for children with bronchiolitis treated with SABAs. This treatment had no significant effect on rates or duration of hospitalization. While rare, adverse effects such as tachycardia, decreased oxygen saturation, flushing, and hyperactivity occurred more frequently in children treated with SABAs.[146] Thus, the AAP does not recommend the routine use of SABAs for bronchiolitis; instead, clinicians should consider a trial of such medications to determine if a patient has a positive clinical response.[138]

Similar controversy exists with respect to the utility of nebulized epinephrine in treating bronchiolitis. A meta-analysis of 14 studies concluded that there is not enough evidence to support the use of epinephrine for inpatients, but it does provide some clinical benefit over other bronchodilators and placebo for outpatients.[147] Treatment does not decrease the rate of hospitalization, nor the length of hospital stay.[148] One difficulty with the use of epinephrine in the ED is that it is not a treatment that can be continued at home. Thus, nebulized epinephrine should be considered for children with moderate to severe distress in whom beta-agonist therapy was not effective and who will likely require hospitalization. As with SABAs, nebulized epinephrine should be continued only for those patients who demonstrate a clinical benefit.

Studies on the use of nebulized anticholinergic agents (e.g., ipratropium bromide) have not been conclusive. While one study in the ED setting reported a decreased need for additional treatment for patients receiving an anticholinergic medication in addition to SABAs,[149] a similarly designed ED study found no benefit.[150] There is currently not sufficient evidence to recommend the use of anticholinergic agents for young children with wheezing and suspected bronchiolitis.[151]

Many of the symptoms of bronchiolitis are a result of increased and thickened respiratory secretions. A great deal of literature supports the use of nebulized hypertonic saline in the treatment of cystic fibrosis, in which clearance of thickened secretions is vital.[152–154] Although there is not yet enough literature to definitively recommend its use for bronchiolitis, one recent study suggests that nebulized hypertonic saline is a safe medication that reduces the length of stay for hospitalized children.[155] Chest physiotherapy has also been examined as a means for clearing respiratory secretions. A meta-analysis of three randomized, controlled trials revealed no improvement in clinical score, length of stay, or oxygen requirement after chest physiotherapy.[156]

Systemic corticosteroids are well established as being effective treatment for wheezing due to acute asthma. Despite reports that more than half of infants may be prescribed corticosteroids when diagnosed with bronchiolitis,[141] well-designed controlled trials have demonstrated no benefit for their use in terms of rate of admission, clinical score, or any other outcome.[138,157,158] Specifically, Corneli and colleagues conducted a double-blind, randomized trial comparing oral dexamethasone with placebo in 600 children with acute moderate-to-severe bronchiolitis. The investigators concluded that oral dexamethasone had no significant effect on the rate of hospitalization, respiratory status after 4 hours of observation, or later outcomes, such as length of inpatient stay, repeat medical visits, and adverse events. Inhaled corticosteroids also provide no positive effect on clinical course.[159]

While infants with severe bronchiolitis requiring intensive care and mechanical ventilation frequently have concurrent or secondary bacterial infections,[160] this is an uncommon complication for most children. Despite some reports that clarithromycin may hasten recovery in RSV bronchiolitis,[161] there is no evidence for the routine use of antibiotics for bronchiolitis,

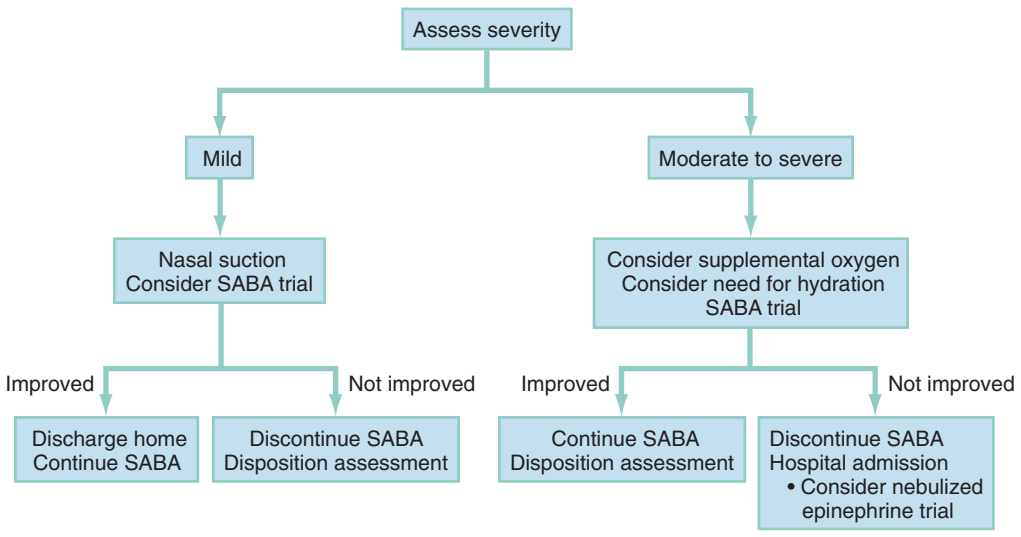


Figure 167-2. Emergency department management of bronchiolitis.

and they should be reserved for patients with true bacterial infections.[138,162]

Ribavirin is a specific antiviral agent directed toward treatment of RSV infections. Several smaller studies suggest a small benefit with respect to duration of mechanical ventilation and length of stay in patients with severe disease.[163] Its high cost and potential risks to caregivers, however, limit its role in routine management. Ribavirin may be indicated in selected cases of documented RSV bronchiolitis with severe respiratory compromise.[138]

Prophylaxis

Though ED clinicians generally do not have a role in administering preventive medications, they should be aware of the options available to their patients. Palivizumab (Synagis) consists of monoclonal antibodies against RSV. While RSV-specific immune globulin is not effective in treating the acute disease process,[164] palivizumab is quite effective in reducing hospitalization rates for RSV in certain high-risk populations.[165–168] It is recommended for most children younger than 24 months with chronic lung disease, congenital heart disease, or prematurity and is administered as a monthly intramuscular injection during the high prevalence months.[169]

Disposition

An essential component in the evaluation and management of bronchiolitis in the ED is the ability to predict the severity of its clinical course. Because it is a dynamic disease, evaluations at a single point in time may not be sufficient to fully estimate its severity; thus, serial examinations are necessary. A number of demographic and clinical features have been associated with a severe clinical course. These factors, which may mandate hospitalization, include age less than 3 months, gestational age less than 34 weeks, ill appearance, hypoxemia (Sao_2 < 95%), tachypnea (>70 breaths/min), and significant atelectasis on CXR (if obtained).[170] Most of the literature, however, focuses on inpatients. In addition to younger age and prematurity, a history of hemodynamically significant congenital heart disease, chronic lung disease, or an immunocompromised state have all been associated with higher morbidity and mortality among inpatients.[138,171,172]

Ultimately, the ED clinician must assess more than just the child's degree of respiratory distress. Patients for whom the respiratory symptoms negatively impact their ability to feed and maintain hydration warrant inpatient admission. Assessing the family's ability to continue supportive measures at home and to seek further medical care if necessary is central to the disposition decision. In children for whom outpatient management is deemed appropriate, expedient follow-up (within 24 hours) with a primary care provider is essential. If SABA therapy provides a sustained clinical improvement in symptoms in the ED, it should be continued as part of home therapy. On the other hand, for the subset of infants who fail to respond to SABA therapy yet meet other criteria for discharge, outpatient treatment with SABAs is not warranted. As in all of pediatric medicine, anticipatory guidance must be provided. Parents should be instructed about the signs of worsening respiratory distress, including poor feeding, retractions, increased tachypnea, lethargy, or irritability; seeking immediate medical care is warranted should these signs occur.

Summary

Bronchiolitis is a common respiratory disease in infancy that accounts for many ED visits and hospitalizations. It is primarily a clinical diagnosis based on key history and physical examination findings. Serious complications, such as apnea, are rare and mostly occur in very young infants or those with underlying medical conditions. Treatment is largely supportive, and the use of nebulized SABAs may be helpful for a subset of patients. Ultimately, ED clinicians must understand the dynamic and variable nature of bronchiolitis and be able to effectively predict the severity of its clinical course.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 4 Shock

Alan E. Jones and Jeffrey A. Kline

PERSPECTIVE

In philosophic terms, shock can be viewed as a transition between life and death. Whether shock results from hemorrhage, sepsis, or cardiac failure, mortality rates exceed 20%.[1-3] In scientific parlance, shock results from the widespread failure of the circulatory system to oxygenate and nourish the body adequately. In the laboratory the scientist defines the metabolic effect of shock quantitatively, by examining the mechanisms by which shock alters mitochondrial energy transfer, evokes the production of toxic chemicals, and reduces their removal. At the bedside, however, the clinician identifies shock by linking the clinical impression, synthesized from the patient's history of present illness, age, underlying health status, and general appearance, with quantitative data, including vital signs, blood chemistry, urine output, and direct measurements of oxygenation. When the clinical impression and the quantitative data suggest widespread organ hypoperfusion, emergent resuscitation must restore normal tissue oxygenation and substrate delivery to prevent deterioration into systemic inflammation, organ dysfunction, and death.

At the subcellular level, shock first affects the mitochondria. Mitochondria function at the lowest oxygen tension in the body, but paradoxically, they consume almost all the oxygen used by the body. More than 95% of aerobic chemical energy comes from mitochondrial combustion of fuel substrates (fats, carbohydrates, ketones) plus oxygen (O_2) into carbon dioxide (CO_2) and water (H_2O). Mitochondria therefore have been referred to as the "canaries in the coal mine" because they are affected first in conditions of inadequate tissue perfusion.[4,5] When mitochondria have inadequate oxygen, the cell catabolizes fuels to lactate, which inexorably accumulates and diffuses into the blood.

Classification

For years, shock has been classified into four broad categories based on Blalock's 1940 description: hematologic, neurologic, vasogenic, and cardiogenic.[6] This basic organization scheme remains useful today. Box 4-1 outlines five categories of shock that generally have specific mechanisms and treatments.

Epidemiology

The epidemiology of shock in the emergency department (ED) context remains speculative because shock is rarely listed as a primary coding diagnosis and depends on defining criteria. Arterial hypotension, defined as a systolic blood pressure less than 100 mm Hg, is measured at least one time in 19% of ED patients[7]; however, diagnosed traumatic, cardiogenic, or septic shock is less common, constituting about 1 to 3% of all ED visits.

This chapter reviews the metabolic, systemic, and inflammatory responses that occur in all types of circulatory shock and discusses specific pathophysiology of the major causes of shock.

Specific Causes

Hemorrhagic Shock

Hemorrhagic shock results from a rapid reduction in blood volume, which causes baroreceptor activation and leads to vasoconstriction, increased strength of cardiac contraction, and increased heart rate (HR). Cardiovascular response to hemorrhage can vary with underlying cardiopulmonary status, age, and presence of ingested drugs. Responses of HR and blood pressure (BP) are notoriously variable in hemorrhage, so no firm conclusion can be made at the bedside about the presence or absence of hemorrhagic shock simply by evaluating HR and BP.[8] In general, hemorrhage first increases pulse and cardiac contraction, then increases vasoconstriction. Blood loss causes an elevated pulse rate with a slight increase in the diastolic BP, causing the pulse pressure (difference between systolic and diastolic BP) to narrow. As blood loss continues ventricular filling decreases, and cardiac output drops, followed by a reduction in systolic BP. Before the total cardiac output begins to decrease, blood flow to noncritical organs and tissues begins to decrease, and their cells produce and release lactic acid.

Consequently, acidemia often precedes any significant decrease in cardiac output with hemorrhage.[9] However, the blood contains bicarbonate ions that buffer the blood pH, keeping it near neutral, even as lactic acid accumulates in blood. The base *deficit*, defined as the amount of strong base that would have to be added to a liter of blood to normalize the pH, represents an index of how far the bloodstream has dipped into its reserve of bicarbonate buffer. A normal base deficit is more positive than −2 mEq/L. Accordingly, the arterial and venous blood base deficit can become more negative early in hemorrhage even while blood pH and BP remain in the normal range. The base deficit, therefore, crudely represents the physiologic endpoint that distinguishes trivial blood loss from clinically significant hemorrhage. In addition to

BOX 4-1 CATEGORIES OF SHOCK ACCORDING TO PRIMARY TREATMENT

Causes That Require Primarily the Infusion of Volume
Hemorrhagic shock
- Traumatic
- Gastrointestinal
- Body cavity

Hypovolemia
- Gastrointestinal losses
- Dehydration from insensible losses
- Third-space sequestration from inflammation

Causes That Require Improvement in Pump Function by Either Infusion of Inotropic Support or Reversal of the Cause of Pump Dysfunction
Myocardial ischemia
- Coronary artery thrombosis
- Arterial hypotension with hypoxemia

Cardiomyopathy
- Acute myocarditits
- Chronic diseases of heart muscle (ischemic, diabetic, infiltrative, endocrinologic, congenital)

Cardiac rhythm disturbances
- Atrial fibrillation with rapid ventricular response
- Ventricular tachycardia
- Supraventricular tachycardia

Hypodynamic septic shock
Overdose of negative inotropic drug
- Beta-blocker
- Calcium channel antagonist overdose

Structural cardiac damage
- Traumatic (e.g., flail mitral valve)
- Ventriculoseptal rupture
- Papillary muscle rupture

Causes That Require Volume Support and Vasopressor Support
Hyperdynamic septic shock
Anaphylactic shock
Central neurogenic shock
Drug overdose

Problems That Require Immediate Relief from Obstruction to Cardiac Output
Pulmonary embolism
Cardiac tamponade
Pneumothorax
Valvular dysfunction
- Acute thrombosis of prosthetic valve
- Critical aortic stenosis

Congenital heart defects in newborn (e.g., closure of patent ductus arteriosus with critical aortic coarctation)
Critical idiopathic subaortic stenosis (hypertrophic obstructive cardiomyopathy)

Cellular Poisons That Require Specific Antidotes
Carbon monoxide
Methemoglobinemia
Hydrogen sulfide
Cyanide

chemical buffering, the body responds to small reductions in arterial pH by activating brainstem chemoreceptors, which increase minute ventilation, leading to reduced partial pressure of carbon dioxide in arterial gas ($Paco_2$).

After approximately one third of the total blood volume is acutely lost, cardiovascular reflexes can no longer sustain adequate filling of the arterial circuit, and frank hypotension supervenes. Arterial hypotension is generally and arbitrarily defined as an arterial BP below 90 to 100 mm Hg. Usually coincident with the development of hypotension, bicarbonate buffers become overwhelmed, and increased alveolar ventilation becomes ineffective, culminating in reduced arterial pH. Hemorrhagic shock causes an activation of the hypothalamic-pituitary-adrenomedullary axis, with release of stress hormones that cause glycogenolysis, lipolysis, and mild hypokalemia. Therefore, in the ED, patients sustaining traumatic hemorrhage generally have an arterial lactate concentration greater than 4.0 mmol/L, a $Paco_2$ less than 35 mm Hg, and mild hyperglycemia (150–170 mg/dL) and hypokalemia (3.5–3.7 mEq/L). Although hemorrhagic hypotension reduces lung perfusion, arterial hypoxemia should not be attributed simply to blood loss, but instead should prompt investigation for aspiration, airway obstruction, alveolar consolidation, or lung injury.

The second phase of organ injury from hemorrhagic shock occurs during resuscitation. It has been said that the acute phase of hemorrhage "cocks the gun," and resuscitation "pulls the trigger" to cause organ injury from hemorrhagic shock. During resuscitation, neutrophils become most aggressive, binding to the lung endothelium and causing capillary leaks that characterize the adult respiratory distress syndrome (ARDS). Inflammatory cytokines are liberated during resuscitation, and membrane injury occurs in many cells. In the liver, damage from inflammation and reactive oxygen species from neutrophils is compounded by persistent microischemia. During resuscitation from hemorrhagic shock, the normal balance of vasodilation by nitric oxide (NO) versus vasoconstriction by endothelins becomes distorted, producing patchy centrilobular ischemic damage in the liver, which may produce an immediate rise in blood transaminase levels. A growing body of evidence suggests that resuscitation from hemorrhage exerts greater injury to the heart than the actual hypotensive insult.[10,11] Depending on the degree of hypotensive insult, the kidney may manifest acute spasm of the preglomerular arterioles, causing acute tubular necrosis. Systemic metabolic changes can impair fuel delivery to the heart and brain, secondary to depressed hepatic glucose output, impaired hepatic ketone production, and inhibited peripheral lipolysis.[12]

Septic Shock

Septic shock can be produced by infection with any microbe, although in as many as half of the cases of septic shock, no organism is identified. One of the most well-studied mediators of sepsis is lipopolysaccharide, contained in the outer cell membrane of gram-negative bacteria. Infusion of lipopolysaccharide into humans or animals produces cardiovascular, immunologic, and inflammatory changes identical to those observed with microbial infection. In recent years, multicenter trials of sepsis have suggested the emergence of gram-positive organisms as the chief cause of sepsis in hospitalized patients.[13] Two lines of reasoning imply that gram-positive sepsis will continue to increase in prevalence:

1. More patients are being treated at home for chronic immunocompromising diseases with indwelling catheters, which serve as excellent portals of entry into the vascular space for *Staphylococcus aureus* and coagulase-negative staphylococci.
2. The frequency of community-acquired infections caused by antibiotic-resistant gram-positive organisms has greatly increased in recent years, including infections caused by *Staphylococcus aureus*, *Streptococcus pneumoniae*, and *Streptococcus pyogenes*.

Septic shock causes three major effects that must be addressed during resuscitation: relative hypovolemia, cardiovascular depression, and induction of systemic inflammation. Septic shock produces relative hypovolemia from increased venous capacitance, which reduces right ventricular filling. Septic shock often causes absolute hypovolemia from gastrointestinal volume losses, tachypnea, sweating, and decreased ability to drink during development of the illness. Sepsis also induces capillary leak, which leads to relative loss of intravascular volume into third spaces. Recent evidence has shown that septic shock causes myocardial depression simultaneously with vasodepression and capillary leak. Direct measurements of cardiac contractility have shown that cardiac mechanical function becomes impaired early in the course of septic shock, even in the hyperdynamic stages.[14] Multiple mechanisms may explain depressed heart function in sepsis, including actions of specific cytokines (most notably tumor necrosis factor alpha [TNF-α] and interleukin 1 beta [IL-1β]),[15] overproduction of NO by nitric oxide synthase (iNOS),[16] and possibly impairment in mitochondrial oxidative phosphorylation[17] coincident with reduced mechanical efficiency.[18] Evidence indicates that circulating mediators, myocardial cellular injury from inflammation, and deranged metabolism interact synergistically to injure the heart during septic shock. Systemic inflammation causes capillary leak in the lung, which may cause alveolar infiltration characteristic of ARDS early in the treatment of septic shock in up to 40% of patients.[13] With the potential for early development of ARDS, more profound ventilation/perfusion $(\dot{V}/\dot{Q})$ mismatching, and pneumonia or pulmonary aspiration, hypoxemia is more severe with septic shock than hemorrhagic shock.

Cardiogenic Shock

Cardiogenic shock (myocardial pump failure) results when more than 40% of the myocardium undergoes necrosis from ischemia, inflammation, toxins, or immune destruction. Otherwise, cardiogenic shock essentially produces the same circulatory and metabolic alterations observed with hemorrhagic shock. Undoubtedly, impaired baseline cardiac function can contribute to the development of circulatory shock secondary to infection, hemorrhage, or vasodilatory drug overdose. However, when shock results from a pure cardiac cause, severe left ventricular dysfunction is evident on echocardiography early in the course. Patients with severe dysfunction are far more likely to have a cardiogenic cause of shock than patients with normal or moderate left ventricular dysfunction.[19]

CLINICAL FEATURES

Patients frequently present to the ED in shock with no obvious cause. Rapid recognition of shock requires the integration of information from immediate history and physical examination, and a diagnosis of shock can be strongly supported by the presence of a worsening base deficit or lactic acidosis. In general, patients with shock exhibit a stress response: they appear ill, pale, often sweating, usually tachypneic or grunting, and often with a weak and rapid pulse (Box 4-2). HR can be normal or low in cases of shock, especially when the patient is taking prescribed drugs that depress HR or the circumstance is complicated by profound hypoxemia. BP initially can be normal because of adrenergic reflexes. Although arterial BP as a sole measurement remains an unreliable marker of circulatory status, the finding of a single systolic BP less than 100 mm Hg in the ED is associated with a threefold increase in in-hospital mortality and a tenfold increase in sudden and unexpected death.[7] The HR/systolic BP ratio may provide a better marker of shock than either measurement alone; a normal ratio is less than 0.8.[20] Urine output provides an excellent indicator of organ perfusion and is readily available with insertion of a Foley catheter. Measuring urine output, however, requires at least 30 minutes to accurately determine if output is normal (>1.0 mL/kg/hr), reduced (0.5–1.0 mL/kg/hr), or severely reduced (<0.5 mL/kg/hr). Point measurements of the arterial lactate concentration and the base deficit can be rapidly performed and provide accurate assessment of global perfusion status. A lactate concentration greater than 4.0 mM or a base deficit more negative than –4 mEq/L predicts the presence of circulatory insufficiency severe enough to cause subsequent multiple organ failure.[21] Once the empirical criteria for circulatory shock are discovered, the next step is to consider the cause of shock. Figure 4-1 is an algorithm of potential decisions to facilitate diagnosis in a patient with undifferentiated shock.

BOX 4-2 EMPIRICAL CRITERIA FOR DIAGNOSIS OF CIRCULATORY SHOCK*

Ill appearance or altered mental status
Heart rate >100 beats/min
Respiratory rate >20 breaths/min or $Paco_2$ <32 mm Hg
Arterial base deficit <–4 mEq/L or lactate >4 mM/L
Urine output <0.5 mL/kg/hr
Arterial hypotension >20 minutes duration

*Regardless of cause. Four criteria should be met.

Use of the history, vital signs and physical examination documented by outside providers represents a valuable insight into a patient's physiologic status prior to any medical intervention and can be useful in ED management. Studies suggest that both medical and trauma patients with hypotension prior to being seen in the ED have a three- to fourfold higher in-hospital mortality rate than patients without hypotension.[22,23]

The primary survey must ensure presence of a patent airway as well as sufficient respiratory effort for adequate oxygenation and ventilation. The physical examination should be performed on an undressed patient and should begin with a quick head-to-toe inspection. Dry mucous membranes suggest dehydration, whereas distended jugular veins suggest cardiac failure or obstruction from pulmonary embolism (PE) or cardiac tamponade. Muffled heart sounds suggest cardiac tamponade, whereas a loud machine-like systolic murmur indicates acute rupture of a papillary muscle or rupture of the interventricular septum. Bilateral pulmonary rales in a patient with a normal rectal temperature help to define the presence of primary left ventricular failure. Wheezing suggests bronchospasm from anaphylaxis or, less likely, cardiac failure or PE. Abdominal tenderness may indicate peritoneal inflammation or occult trauma. Rectal examination may disclose occult gastrointestinal hemorrhage. Rectal temperature should be performed as early as is reasonable on every patient with suspected shock.

The neurologic examination documents responsiveness, cognition, and the presence of any focal deficits. In children, documentation should include level of alertness, response to parents, appropriateness of crying, pupillary function, symmetry of grimace, symmetry of extremity movements, and motor tone in infants.

Laboratory, radiographic, and other ancillary data should be ordered to assess tissue and vital organ perfusion and to diagnose injury from trauma, find the source of infection with sepsis, or identify the cause of cardiac failure. A chest radiograph, electrocardiogram, finger-stick glucose measurement,

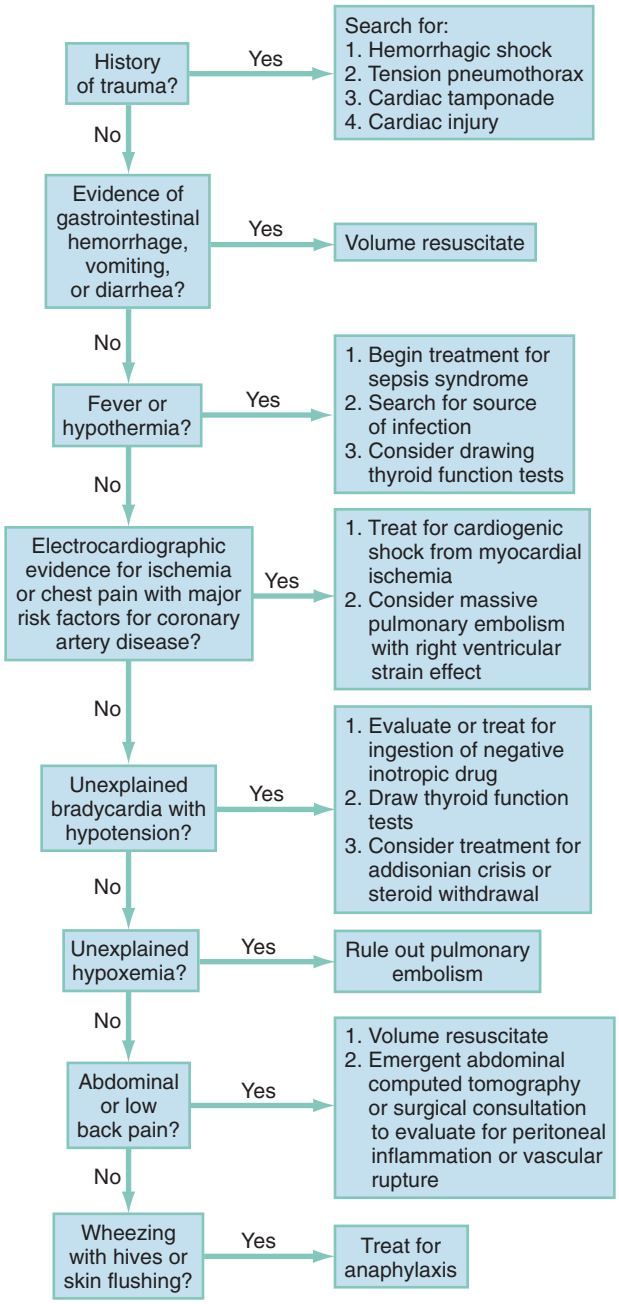


Figure 4-1. Flow diagram to classify undifferentiated shock.

complete blood count (CBC), urinalysis, serum electrolytes, and kidney and liver function tests are all indicated in the ED assessment. Arterial blood gases are ordered for a base deficit calculation and to correlate arterial oxygen partial pressure (Pao_2) with that measured by pulse oximetry, when the latter is deemed unreliable. Serum lactate measurement should be performed as early as possible in patients with suspected shock. Either venous or arterial lactate concentrations can be used.[24-27] If peripheral venous lactate is used, time, storage temperature, and tourniquet use have no significant effect on in vitro lactate production by erythrocytes if the measurement is done within 15 minutes after the sample is obtained.[28] Some EDs have bedside ultrasound capability, and both cardiac and abdominal scanning can be rapidly performed at the bedside to screen for inadequate central venous volume, occult hemoperitoneum, abdominal aortic aneurysm, left ventricular failure, and cardiac tamponade. A systematic ultrasound protocol can significantly improve the physician's ability to accurately diagnose the cause of undifferentiated shock in ED patients,[29] and the finding of hyperdynamic left ventricular function in patients with undifferentiated shock strongly suggests sepsis as the cause.[30]

BOX 4-3 Definitions and Criteria for Septic, Hemorrhagic, and Cardiogenic Shock

Septic Shock

Systemic Inflammatory Response Syndrome (SIRS)

Two or more of the following:

1. Temperature >38°C or <36°C
2. Heart rate >90 beats/min
3. Respiratory rate >20 breaths/min or $Paco_2$ <32 mm Hg
4. While blood cell count >12,000/mm^3, <4000/mm^3, or >10% band neutrophilia

Severe Sepsis

SIRS with suspected or confirmed infection and associated with organ dysfunction or hypotension; organ dysfunction may include presence of lactic acidosis, oliguria, or altered mental status

Septic Shock

SIRS with suspected or confirmed infection with hypotension despite adequate fluid resuscitation; septic shock should still be diagnosed if vasopressor therapy has normalized blood pressure

Hemorrhagic Shock

Simple Hemorrhage

Suspected bleeding with pulse <100 beats/min, normal respiratory rate, normal blood pressure, and normal base deficit

Hemorrhage with Hypoperfusion

Suspected bleeding with base deficit <–4 mEq/L *or* persistent pulse >100 beats/min

Hemorrhagic Shock

Suspected bleeding with at least four criteria listed in Box 4-2

Cardiogenic Shock

Cardiac Failure

Clinical evidence of impaired forward flow of the heart, including presence of dyspnea, tachycardia, pulmonary edema, peripheral edema, or cyanosis

Cardiogenic Shock

Cardiac failure plus four criteria listed in Box 4-2

Consensus definitions of shock show the spectrum of hypoperfusion for the following three common causes of shock (Box 4-3):

1. *Septic shock.* The American College of Chest Physicians, European Society of Intensive Care Medicine, Society for Critical Care Medicine, American Thoracic Society, and the Surgical Infection Society[31] developed international consensus definitions for distinguishing septic shock from its precursor conditions, the systemic inflammatory response syndrome (SIRS), sepsis, and severe sepsis. Although this particular consensus requires persistent hypotension after fluid resuscitation to strictly define septic shock, initiation of treatment for empirically diagnosed severe sepsis or septic shock should not await the onset of hypotension.
2. *Hemorrhagic shock.* The American College of Surgeons has divided hemorrhagic shock into four stages, depending on

the severity of blood loss and the physiologic response to this loss, but such arbitrary divisions are of little value. A more useful approach defines hemorrhagic shock as being present when systemic hypoperfusion manifests as lactic acidosis with organ dysfunction.

3. *Cardiogenic shock*. Cardiogenic shock should be thought to be present whenever cardiac failure (ischemic, toxic, or obstructive) causes systemic hypoperfusion that manifests as lactic acidosis with organ dysfunction.

MANAGEMENT

Monitoring Perfusion Status

In the effort to resuscitate a patient with circulatory shock, the clinician must follow specific indices of systemic perfusion and organ function to know if the resuscitation effort is working. In all patients with shock, circulation must be monitored by continuous electrocardiography and pulse oximetry. Cuff sphygmomanometer measurement of BP should be performed frequently during resuscitation. Because cuff sphygmomanometer measurement may be inaccurate in severe hypotensive states, the use of an arterial pressure monitoring line should be considered, especially if vasoactive medications are being administered. BP and HR correlate poorly with cardiac index (CI) in shock and often underestimate the severity of systemic hypoperfusion.[8] Moreover, children with hypovolemic shock frequently demonstrate a normal BP until they rapidly deteriorate.[32] Urine output should be measured as an index of vital organ perfusion (about 1 mL/kg/hr in persons without renal disease). A downward trend of the serum lactate concentration or upward trend of the base deficit, when observed with improving vital signs and urine output, is a reliable gauge of the adequacy of resuscitation and prognosis in shock from any cause.[21,33] A rising lactate concentration (or refractory hypotension with worsening base deficit) despite ongoing resuscitation is a portent of imminent death, and vigorous resuscitation efforts or specific procedural intervention should be instituted.

Most patients with shock can be fully resuscitated with peripheral venous access established with two catheters of at least a size 18 gauge. Monitoring of central venous pressure (CVP) as part of a goal-directed resuscitation may improve outcome in patients with septic shock.[34] Patients with cardiac failure or renal failure may benefit from closer measurement of the CVP and insertion of a central venous catheter. An 8.5-French catheter (Cordis Sheath) allows for accurate measurement of the CVP and insertion of a pulmonary artery catheter or other monitoring device if needed. In children a 3- or 5-French bilumen catheter can be placed in the femoral vein with few complications.[35] To reduce the potential for limb damage from extravasation from a peripheral IV, vasoactive medications are optimally administered through a central venous catheter. If vasoactive medications are administered, additional peripheral intravenous catheters are required for infusion of crystalloid and other treatments. Many patients with renal disease or cancer have indwelling catheters. In patients with empirical criteria for shock, this catheter should be used for IV access, unless satisfactory access has already been established at other anatomic sites. In EDs where the standard practice is not to use these ports at the request of other physicians, a specific hospital policy and training session should be developed to make an exception in the case of circulatory shock. In general, failure to administer fluids rapidly and in sufficient quantity outweighs considerations about preservation of the line for future therapy.

Quantitative Resuscitation

Quantitative resuscitation (also called goal-directed therapy, goal-oriented resuscitation, or hemodynamic optimization) was first described in 1988 and refers to the practice of resuscitating patients to predefined physiologic endpoints indicating that systemic perfusion and vital organ function have been restored.[36] Since that time many studies have evaluated the efficacy of such a therapeutic approach to shock, and a meta-analysis of these studies confirms its benefit for reducing mortality rates.[37] For many years in the intensive care unit (ICU), physicians have relied on the use of the pulmonary artery catheter (PAC) to help optimize left ventricular filling indices. At present the use of the PAC remains controversial. In the last 5 years, five randomized controlled trials investigating the management of critically ill patients with a PAC have been published.[38-42] None have found a benefit in terms of survival or length of stay. Insufficient data have been published to support the use or avoidance of PACs in ED populations; however, extrapolating from ICU studies, PACs have no role in the management of shock in the ED.

Several alternative methods to the PAC have been proposed as endpoints to resuscitation in the ED. The *lactate clearance* index refers to serial measurements of venous or arterial lactate.[33,43] Lactate clearance involves measuring the blood lactate concentration at two or more times. If the lactate concentration has not decreased by 10% two hours after resuscitation has begun, additional steps must be undertaken to improve systemic perfusion.[43] Resuscitation should continue until the lactate concentration drops below 2 mM/L. Clinical trials are presently investigating the utility of lactate clearance as an endpoint of resuscitation, which will have ramifications for the increasing use of point-of-care lactate testing platforms in the ED.

Mixed venous oxygen saturation (Svo_2) measurements reflect the balance between oxygen delivery and oxygen consumption. Previous studies have suggested that the Svo_2 can be used as a surrogate to CI when targeting normalization of endpoints ($Svo_2 = 65\%$ or CI 2.5–3.5 L/min/m^2) for therapeutic intervention in critically ill patients.[44] Although Svo_2 requires the use of a PAC, the central venous oxygen saturation ($Scvo_2$) drawn from the central circulation has been shown to closely parallel the Svo_2, especially when tracking changes or trends in the values.[45]

Early quantitative resuscitation, which incorporates multiple indices of circulatory and oxygenation status, was shown in one randomized controlled trial to significantly reduce mortality and morbidity rates in ED patients with severe sepsis or septic shock.[34] Patients are resuscitated within the first 6 hours of care to achieve normalization of CVP and mean BP and to maintain a $Scvo_2$ greater than or equal to 70% (Fig. 4-2). The decrease in mortality rate from this new treatment strategy, termed *early goal-directed therapy*, has been found effective in smaller prospective before-and-after studies of patients with sepsis.[46,47] Large multicenter validation of this resuscitation strategy in sepsis is underway. However, it has not been tested in other causes of shock but shows the value of using defined physiologic endpoints to measure systemic perfusion during resuscitation from shock in the ED. This approach also further substantiates the importance of the first 6 hours of resuscitation.

Ventilation

Rapid sequence intubation is the preferred method of airway control in most patients with shock (see Chapter 1). Intubation

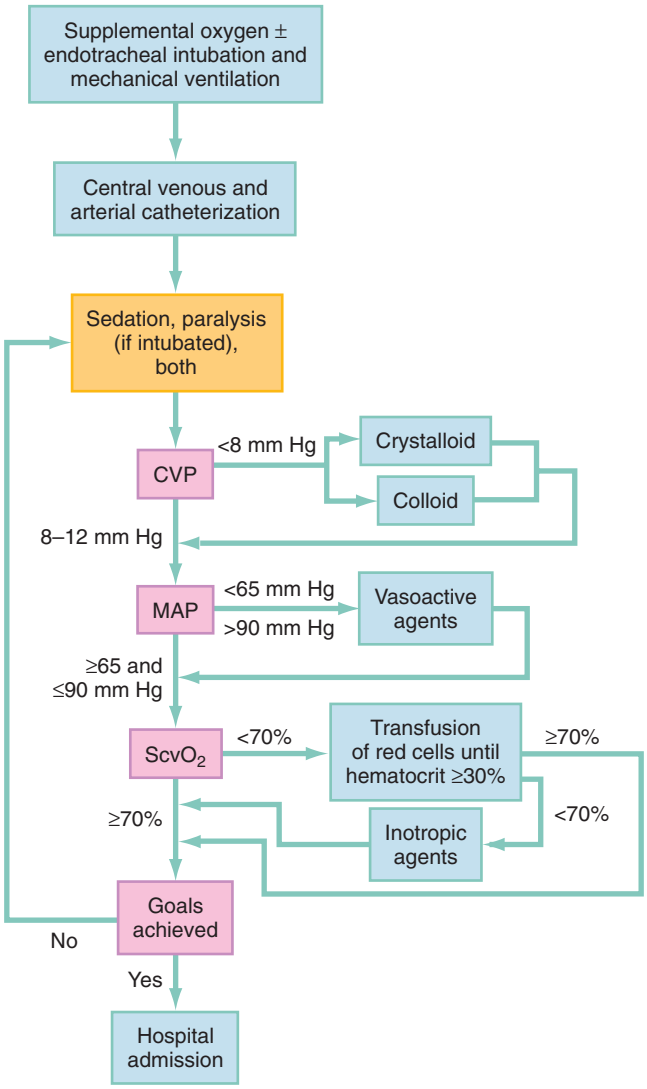


Figure 4-2. Flow diagram outlining the protocol for quantitative resuscitation when treating patients with severe sepsis or septic shock. This protocol outlines specific hemodynamic and physiologic parameters the clinician should seek to achieve within the first 6 hours of care. This protocol is focused on resuscitation and should be used in conjunction with standard clinical care for patients with suspected infection, such as appropriate diagnostic studies to determine the focus of infection and appropriate antimicrobial agents to treat the infection. CVP, central venous pressure; MAP, mean arterial pressure; $ScvO_2$, central venous oxygen saturation. (Redrawn from Rivers E, et al: Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 345:1368, 2001.)

prevents aspiration, increases oxygenation, treats acute respiratory failure, provides initial treatment for metabolic or hypercarbic acidemia, and protects the patient who will be sent to an uncontrolled environment (e.g., for tests). Intubation also reduces the work of breathing, which, in the hypoperfused patient, further exacerbates lactic acidemia. Strenuous use of accessory respiratory muscles can increase oxygen consumption by 50 to 100% and decrease cerebral blood flow by 50%.[48,49] More importantly, if the patient has increased airway resistance (e.g., bronchospasm with anaphylaxis) or a decrease in lung compliance (e.g., pulmonary edema, ARDS), a more negative intrathoracic pressure must be generated to fill the lungs with each inspiration. The greater suction effect is also exerted on the left ventricle, impeding its ability to eject and increasing functional afterload. Positive pressure ventilation removes this impedance and can improve ventricular function and cardiac output up to 30%.[48]

BOX 4-4 CLINICAL MANAGEMENT GUIDELINES FOR FOUR COMMON CAUSES OF SHOCK

Hemorrhagic Shock

Ensure adequate ventilation/oxygenation
Provide immediate control of hemorrhage, when possible (e.g., traction for long bone fractures, direct pressure)
Initiate judicious infusion of isotonic crystalloid solution (10–20 mL/kg)
With evidence of poor organ perfusion and 30-minute anticipated delay to hemorrhage control, begin packed red blood cell (PRBC) infusion (5–10 mL/kg)
With suspected central nervous system trauma or Glasgow Coma Scale score <9, immediate PRBC transfusion may be preferable as initial resuscitation fluid
Treat coincident dysrhythmias (e.g., atrial fibrillation with synchronized cardioversion)

Cardiogenic Shock

Ameliorate increased work of breathing; provide oxygen and positive end-expiratory pressure (PEEP) for pulmonary edema
Begin vasopressor or inotropic support; norepinephine (0.5 µg/min) and dobutamine (5 µg/kg/min) are common empirical agents
Seek to reverse the insult (e.g., initiate thrombolysis, arrange percutaneous transluminal angioplasty)
Consider intra-aortic balloon pump counterpulsation for refractory shock

Septic Shock

Ensure adequate oxygenation; remove work of breathing
Administer 20 mL/kg of crystalloid or 5 mL/kg of colloid, and titrate infusion to adequate central venous pressure and urine output
Begin antimicrobial therapy; attempt surgical drainage or débridement
Begin PRBC infusion for hemoglobin < 8 g/dL
If volume restoration fails to improve organ perfusion, begin vasopressor support; initial choice includes dopamine, infused at 5–15 µg/kg/min, or norepinephrine, infused at 0.5 µg/min

Volume Replacement

The next imperative in shock is to decide when "the tank is full." The goal in volume replacement is slightly elevated left ventricular end-diastolic *volume*, which is a difficult measurement to make in the ED. The CVP is most often used to estimate right ventricular filling pressure and is used in some quantitative resuscitation algorithms. Because both ventricles tend to stiffen during shock, a high CVP (10–15 cm H_2O) is often needed to produce adequate filling volume. It is a long way, however, from the CVP measurement to actual knowledge of left ventricular end-diastolic volume; a presumed adequate CVP must be substantiated by increases in urine output and BP and decreasing lactate concentrations.[50]

Treating Specific Causes

Box 4-4 presents the general treatment approach for the four common causes of shock.

Hemorrhagic Shock

Standard treatment for hemorrhagic shock consists of rapidly infusing several liters of isotonic crystalloid in adults or three successive 20-mL/kg boluses in children. Colloids, including albumin and hydroxyethyl hetastarch (Hespan), can be used as well but at considerable increase in cost and without effect on morbidity or mortality rates.[51] Colloids offer the theoretic advantage of a high osmotic pressure, which should help to maintain a normal intravascular volume after retransfusion from hemorrhage. If criteria for shock persist despite crystalloid infusion (see Box 4-2), packed red blood cells (PRBCs) should be infused (1–2 units in adults or 5–10 mL/kg in children). Type-specific blood should be used when the clinical scenario permits, but uncrossmatched blood should be used at the earliest opportunity for patients with arterial hypotension and uncontrolled hemorrhage. O-negative blood is used in women of childbearing age and O-positive blood in all others (see Chapter 5). Substantial evidence supports the use of leukodepleted blood, which has been filtered to remove donor neutrophils.[52] Leukodepleted blood is used in countries outside the United States because it produces less retransfusion-related organ damage.[53]

The infusion of hemoglobin-based oxygen carriers as alternatives to PRBCs for resuscitation of hemorrhagic shock have been extensively studied. In a large randomized controlled trial, diaspirin cross-linked hemoglobin, a purified and chemically modified human hemoglobin substrate, was compared with crystalloid for initial resuscitation in the critically injured, and its use resulted in a higher mortality rate at interim analysis, resulting in termination of the trial.[54] Other artificial hemoglobin substitutes may be available in the future but at present show no benefit over PRBCs.

Recent studies have endorsed the concept of either delayed resuscitation or hypotensive resuscitation for hemorrhagic shock. This is discussed in Chapters 34, 42, and 43. Controlling hemorrhage remains the cornerstone of treating hemorrhagic shock, and evidence continues to support immediate surgery when direct vascular control cannot otherwise be obtained (see Chapter 34).

Septic Shock

Septic shock begins as an infectious nidus, which triggers a domino effect of cellular, microvascular, hematologic, and cardiovascular dysfunction. Treatment begins by establishing adequate ventilation to correct hypoxia and acidosis and to reduce systemic oxygen consumption and left ventricular work. This often requires endotracheal intubation and sedation for mechanical ventilation. The controversy regarding the use of etomidate in patients with septic shock is discussed in Chapter 1.

The second goal is to achieve adequate ventricular filling. The choice of fluids in treating septic shock is probably less important than scrupulous monitoring for adequate tissue perfusion. However, choices for fluid resuscitation should involve consideration of availability and the cost-benefit ratio. Initial volume replacement should include rapid infusion of 20 to 25 mL/kg of crystalloid. If hypoperfusion is persistent, 5- to 10-mL/kg boluses of a colloid should be considered. Blood should be transfused in the ED to restore hematocrit to at least 30 to 35%.

The third directive is to eradicate the infection with antimicrobial therapy and, where necessary, surgical drainage. A recent study reported that in adult patients with septic shock, effective antimicrobial administration within the first hour of documented hypotension was associated with increased survival to hospital discharge and that each hour delay in antimicrobial administration over the first 6 hours after recognition was associated with an average decrease in survival of 7.6% per hour.[55] The choice of antimicrobial agent can be directed by clinician experience and institutional minimal infective concentration (MIC) data. When no focus can be found in septic shock, a semisynthetic penicillin with a β-lactamase inhibitor, in combination with an aminoglycoside plus vancomycin is a rational empirical choice. When neutropenia is suspected in a patient with sepsis syndrome, the progression to refractory, fatal septic shock can be cataclysmic. Neutropenia is suggested in patients who have recently undergone chemotherapy. Chemotherapy patients with sepsis represent a special challenge because the pathophysiology may be complicated by anemia, thrombocytopenia, dehydration from vomiting, and the effect of adjunctive steroid therapy. Chemotherapy patients often have indwelling catheters, which predisposes them to more unusual causes of sepsis, including gram-positive bacteria and fungi[56] (see Chapter 143).

Septic shock refractory to volume restoration (urine output or BP remains low; lactate increases) requires vasopressor support. The primary goal of vasopressor support is to increase cardiac output and oxygen delivery to vital organs. Norepinephrine (0.5–30 μg/min) or dopamine (5–20 μg/kg/min) are the first-choice vasopressors for correcting hypotension in septic shock. Norepinephrine is more potent than dopamine and thus may be more effective at reversing hypotension; however, dopamine may be preferred in the setting of inadequate systolic heart function.[57] Dobutamine may also be used with norepinephrine to increase cardiac output and maintain adequate oxygen delivery. A recent multicenter randomized controlled trial of 330 subjects reported that, when simultaneous blood pressure and inotropic support were necessary, there is not a difference in safety or efficacy between epinephrine (0.2 μg/kg/min starting dose) alone and norepinephrine plus dobutamine.[58] Many other studies of different vasopressor regimens are ongoing; however, to date there is no definitive evidence to clearly support the use of one vasopressor over another in septic shock.[59]

Drotrecogin alfa activated (or activated protein C), a recombinant human activated protein with anti-inflammatory, antithrombotic, and profibrinolytic properties, has been investigated in large multicenter trials for the treatment of patients with systemic inflammation and organ failure from acute infection.[60] The institution of activated protein C therapy is not part of the routine ED management of sepsis as there is a large window of time for treatment initiation (within 24 hours of meeting criteria). If this therapy is considered, consultation with the ICU physician who will assume care of the patient is recommended because the therapy is continued for 96 hours.

The use of corticosteroids in the treatment of sepsis and septic shock has been investigated with mixed results. The results of two large randomized controlled trials confirm that there is no role for high-dose, short-course corticosteroid therapy in septic shock.[61,62] Recently, two large multicenter randomized trials of low-dose hydrocortisone treatment failed to show survival benefit among all patients with septic shock.[63,64] One of the studies did show a survival benefit to use of low-dose hydrocortisone among patients who did not adequately respond to a corticotropin stimulation test[64]; however, the larger study did not find this survival benefit.[63] Most current guidelines recommend that low-dose hydrocortisone should only be administered to patients receiving chronic steroid replacement and in patients with refractory shock despite adequate fluid and vasopressor support, and corticotropin stimulation testing is no longer considered of value.[57]

Cardiogenic Shock

The immediate treatment of cardiogenic shock focuses on improving myocardial contractility and pump function. Cardiogenic shock is traditionally defined as the combination of systemic signs of hypoperfusion with arterial systolic BP less than 90 mm Hg (or 30% below a known baseline). If the work of breathing is tiring the patient, if severe pulmonary edema is causing significant hypoxemia, or if respiratory failure is imminent, intubation and mechanical ventilation should be initiated, followed by emergent treatment of bradydysrhythymias or tachydysrhythmias and inotropic support. Barbiturates are not recommended for sedation or anxiety in the intubated patient, because they may have exaggerated negative inotropic effects. Cautious use of benzodiazepines, supplemented by fentanyl for analgesia, is the best approach. Improving perfusion often ameliorates the anxiety and restlessness that accompanies shock states. Etomidate and ketamine have the least risk for hemodynamic compromise and should be used (but in reduced doses) for intubation, accompanied by a full dose of succinylcholine. Prior to administration of vasoactive medications, if hypovolemia is present, it should be corrected by infusing crystalloid or blood products. To improve myocardial contractility, vasopressors or inotropic agents should be administered. The choice of which agent to use depends on signs and symptoms and on the systolic blood pressure (SBP). If the SBP is less than 70 mm Hg and the signs and symptoms of shock are present, norepinephrine is the agent of choice. If the SBP is between 70 and 100 mm Hg and the signs and symptoms of shock are present, dopamine should be used. However, if the SBP is 70 to 100 mm Hg and there are no signs or symptoms of shock, dobutamine is the agent of choice.[3] All of these agents should be started at the same doses used for septic shock. For refractory hypotension and shock, amrinone or milrinone may improve cardiac output, although no empirical evidence is available to support their routine use. Amrinone and milrinone are biperidin derivatives that increase cyclic adenosine monophosphate (cAMP) by inhibiting phosphodiesterase (complex F-III). A loading dose of 0.75 mg/kg for amrinone or 50 µg/kg for milrinone is necessary, followed by a titrated constant infusion for either drug (5–10 µg/kg/min for amrinone and 0.5 µg/kg/min for milrinone).

When pharmacologic support fails to improve indices of perfusion, the next step is to initiate intra-aortic balloon pump counterpulsation (IABPC). This requires the facilities and personnel of a high-level ICU or coronary care unit (CCU). Controlled trials have shown IABPC to improve short-term survival, improve post-thrombolytic patency rates, and reduce stroke morbidity. IABPC increases cardiac output by a mean of 30% in refractory cardiogenic shock and can prolong survival until interventional procedures can be performed. IABPC may be contraindicated in patients with aortic insufficiency or severe peripheral vascular disease.

The dismal outcome of cardiogenic shock complicating acute myocardial infarction (MI) has been improved in recent years. Evidence suggests that emergent revascularization is not superior to medical management in reducing mortality rates in the short term; however, significant improvements in general mortality rates are seen at both 6 months and 1 year[65,66] (see Chapter 77). At present the management of acute MI with cardiogenic shock proceeds as follows and constitutes optimal therapy: (1) ensure adequate ventilation and oxygenation, (2) treat emergent dysrhythmias, (3) initiate vasopressor/inotropic support, (4) administer aspirin if the patient is not allergic, and (5), heparin anticoagulation and arrangement for emergent percutaneous coronary intervention.

KEY CONCEPTS

- Circulatory shock can occur with normal arterial blood pressure, and not all patients with arterial hypotension have circulatory shock.
- A base deficit more negative than –4 mEq/L or a serum lactate > 4.0 mmol/L indicates the presence of widespread circulatory insufficiency in suspected shock.
- Urine output is a reliable index of vital organ perfusion in patients with suspected shock.
- Ill patients with tachycardia, a worsening base deficit, and low urine output should be diagnosed with circulatory shock.
- Use of defined physiologic endpoints to measure systemic perfusion during resuscitation (quantitative resuscitation) is a valuable approach to optimal resuscitation in ED patients with shock.

The references for this chapter can be found online by accessing the accompanying Expert Consult website.

CHAPTER 27 Vaginal Bleeding

Hilarie Cranmer and Mark Foran

PERSPECTIVE

Vaginal bleeding is one of the most frequent chief complaints of women presenting for emergency care. Normal vaginal bleeding occurs cyclically in women who have achieved menarche, mean age 12.5 years, until menopause, mean age 51 years, in North America. The normal cycle, defined as the first day of bleeding of one cycle to the first day of bleeding of the next cycle, lasts 28 days, plus or minus 7 days, and average volume of blood loss is 60 mL. Vaginal bleeding is defined temporally as midcycle (ovulatory), premenstrual, menstrual, and postmenstrual. Abnormal vaginal bleeding is classified on the basis of the duration, amount, and frequency of bleeding (Table 27-1). It occurs in women of all ages, and it can result from a number of causes, including anatomic abnormalities, complications of pregnancy, malignancies, infections, systemic diseases, and endocrinologic imbalances. Typically, premenarchal or postmenopausal vaginal bleeding is rarely life-threatening, but bleeding as a complication of pregnancy has a significantly increased risk of morbidity and mortality for the mother and fetus.[1,2]

Epidemiology

Approximately 5% of women aged 30 to 45 years will see a physician for vaginal bleeding. Nonpregnancy causes are classified as ovulatory, anovulatory, and nonuterine. Menorrhagia secondary to anovulation is seen in 10 to 15% of all gynecologic patients. It is common in perimenarchal and perimenopausal women, as well as in patients with endocrine disorders, polycystic ovary syndrome, exogenous hormone use, and liver or renal disease. Nonuterine bleeding must also be considered.[3] Approximately 20% of all pregnant patients have vaginal bleeding before the 20th week of gestation; more than 50% of these women spontaneously abort. Vaginal bleeding is reported in 50 to 80% of ectopic pregnancies. Ectopic pregnancy is the most common cause of maternal death in the first trimester of pregnancy, accounting for 9% of pregnancy-related maternal deaths in the United States, and the second leading cause for maternal mortality overall, after postpartum hemorrhage. Teenagers and women of color have the highest risk of death related to ectopic pregnancy. Vaginal bleeding after the 20th week of gestation occurs in approximately 4% of pregnancies; approximately 30% of cases are due to placental abruption (abruptio placentae), and 20% are due to placenta previa. Postpartum hemorrhage accounts for nearly 30% of pregnancy-related maternal deaths. The most common cause of postpartum hemorrhage in the first 24 hours is uterine atony. After 24 hours, retained products of conception are frequently the etiology.[4]

Pathophysiology

Pregnant Patients

The differential diagnosis of vaginal bleeding in early pregnancy (before the 20th week of gestation) includes ectopic pregnancy; threatened, inevitable, missed, or incomplete abortion; implantation bleeding; cervicitis; cervical conditions such as polyp or ectropion; bleeding from the urinary or gastrointestinal tract; and cervical carcinoma. Risk factors for ectopic pregnancy should increase clinical suspicion but are often absent. These include tubal abnormalities due to past infection or surgical scarring and assisted reproductive techniques. Disruption of the blood supply to the ectopic gestational sac can cause hemorrhage into the fallopian tube, or the size of the developing sac fetus can lead to rupture through the tubal wall.[5]

Spontaneous abortion is the most common complication of pregnancy and is defined as the passing of a pregnancy prior to completion of the 20th gestational week. It implies delivery of all or any part of the products of conception, with or without a fetus weighing less than 500 g. Threatened abortion is bleeding of intrauterine origin occurring before the 20th completed week, with or without uterine contractions, without dilatation of the cervix, and without expulsion of the products of conception. Complete abortion is the expulsion of all of the products of conception before the 20th completed week of gestation, whereas incomplete abortion is the expulsion of some, but not all, of the products of conception. Inevitable abortion refers to bleeding of intrauterine origin before the 20th completed week, with dilatation of the cervix without expulsion of the products of conception. In missed abortion, the embryo or fetus dies, but the products of conception are retained in utero. In septic abortion, infection of the uterus and sometimes surrounding structures occurs.[6]

Placental abruption can occur spontaneously or secondary to abdominal trauma with transmission of forces to the uterus. An increased incidence is seen in association with cocaine use, hypertension, preeclampsia, HELLP (hemolysis, elevated liver enzymes, and low platelets) syndrome, smoking, increased maternal age, and abnormal implantation of the placenta (e.g., placenta previa, accreta, increta, or percreta). Placenta previa

occurs when the implanted placenta overlays the cervical os. Bleeding is due to partial separation of the placenta from the uterine wall. Uterine atony occurs when myometrial dysfunction prevents the uterine corpus from contracting, allowing continued bleeding at the placental site. Atony is more likely to occur with conditions that overdistend the uterus, such as polyhydramnios, multiparity, prolonged labor, induced labor, high pitocin usage during labor, precipitous labor, magnesium therapy, or intrauterine infection (chorioamnionitis).[7]

Nonpregnant Patients

The pathophysiology of nonpregnant vaginal bleeding varies with age group. Children may present with foreign bodies, genital trauma, or severe vulvovaginitis causing mucosal breakdown and hemorrhage. Sexual abuse must always be considered. In adolescent girls and women, anovulatory uterine bleeding occurs when estrogen stimulates endometrium proliferation without the stabilizing effect of progesterone, causing spontaneous sloughing of the endometrium. Submucosal leiomyomas cause hemorrhage by disrupting the endometrial vascular supply and the ability of the uterus to contract to stop bleeding. Cervical and endometrial polyps have vascular pedicles and are prone to bleed.

Table 27-1 Definitions of Vaginal Bleeding

Polymenorrhea	Abnormally shortened cycle, with bleeding occurring every 21 days or sooner
Oligomenorrhea	A cycle duration of 35 days or longer
Menorrhagia	Cycle occurs at regular intervals but lasts for more than 7 days and involves the loss of more than 80 mL of blood
Hypomenorrhea	Cycle occurs at regular intervals but has a decrease in monthly blood loss
Intermenstrual bleeding	Bleeding that occurs between regular periods
Metrorrhagia	Bleeding that is frequent and irregular
Menometrorrhagia	When metrorrhagia becomes prolonged
Dysfunctional uterine bleeding	Abnormal vaginal bleeding due to anovulation
Postcoital bleeding	Bleeding after sexual intercourse, suggesting cervical pathology
Postmenopausal bleeding	Any bleeding that occurs more than 6 months after the cessation of menstruation

DIAGNOSTIC APPROACH

Differential Considerations

The differential diagnosis can be categorized by age of presentation and frequency of cause (Table 27-2). Primary coagulation disorders account for almost 20% of acute menorrhagia in adolescents. Von Willebrand's disease is the most common; however, myeloproliferative disorders and immune thrombocytopenia are also possibilities.[8] After immediate resuscitation and stabilization of unstable patients, pregnancy status is determined. Patients presenting with hemodynamic instability require intravenous access, fluid resuscitation, stabilization with blood components, and consultation with obstetrics/gynecology (or, less often, surgery). Concurrently, steps must be taken to prevent further vaginal bleeding. In hemodynamically unstable patients, surgical intervention is often necessary to control bleeding effectively. Ectopic pregnancy should be considered in all women of childbearing age who present with abdominal or pelvic complaints or with unexplained signs or symptoms of hypovolemia.

Nonuterine causes of vaginal bleeding must be included in the differential diagnosis, systematically addressed during the history taking and physical examination, and pursued with relevant investigations and consultations, if indicated. Potential sources of nonuterine bleeding include the cervix, vagina, lower urinary tract, and lower gastrointestinal tract. Cervical causes include carcinoma, polyps, condylomata, eversion of squamocolumnar junction associated with oral contraceptive use or pregnancy, trauma, and some infections. Vaginal sources of bleeding include carcinoma, sarcoma, adenosis, lacerations, infections, and retained foreign bodies. Lower urinary tract lesions, such as urethral faruncles and infected urethral diverticula, may also mimic vaginal bleeding.

Pivotal Findings (Symptoms, Signs, and Laboratory)

Symptoms

The volume, duration, and timing of bleeding should be ascertained. The average tampon or pad absorbs 20 to 30 mL of

Table 27-2 Causes of Vaginal Bleeding by Age in Descending Order of Frequency

	PREPUBERTAL	ADOLESCENT	REPRODUCTIVE	PERIMENOPAUSAL	POSTMENOPAUSAL
Most common	Vaginitis	Anovulation	Pregnancy	Anovulation	Endometrial lesions, including cancer (30%)
	Anovulation	Pregnancy	Anovulation	Uterine leiomyomas	Exogenous hormone use (30%)
	Genital trauma or foreign bodies	Exogenous hormone use	Exogenous hormone use	Cervical and endometrial polyps	Atrophic vaginitis (30%)
		Coagulopathy (von Willebrand's disease)	Uterine leiomyomas	Thyroid dysfunction	Other tumor: vulvar, vaginal, cervical (10%)
			Cervical and endometrial polyps		
Least common			Thyroid dysfunction		

vaginal effluent, although the number of pads or tampons used is unreliable because personal habits vary greatly among women. Amenorrhea may not indicate pregnancy, and bleeding during approximately the time of the last expected period does not exclude pregnancy. Bleeding during or after intercourse may indicate a cervical lesion and is more common in pregnancy because of increased blood flow to the cervix. Abdominal pain may indicate critical, emergent, or noncritical causes, depending on the severity of pain, bleeding, and hemodynamic state. During active labor, a history of previous cesarean section, cocaine abuse, or high doses of oxytocin or prostaglandins should raise the suspicion of uterine rupture. A history of trauma should be considered in an adolescent with bleeding, and sexual assault should be considered in an adult in whom abuse is present. In the pregnant patient, there is significant increased risk of maternal and fetal morbidity and mortality after blunt trauma, such as motor vehicle accident, interpersonal violence, or falls. Associated symptoms of nausea, breast tenderness, urinary frequency, and fatigue may indicate that the patient is pregnant. In the absence of pregnancy, vaginal discharge, pelvic pain, and fever may suggest pelvic inflammatory disease. Pelvic inflammatory disease is very rare during pregnancy.

Signs

A thorough evaluation includes recording and interpreting vital signs, abdominal and pelvic examinations, and, in the pregnant patient of sufficient gestational age, fetal heart tones and fundal height. Vaginal bleeding associated with hemodynamic shock alerts the clinician to ruptured ectopic pregnancy. Fetal heart tones that are diminished to less than 100 or that are absent in a gravid female may indicate fetal distress. Pelvic examination may reveal the source of bleeding; however, after the 20th week of gestation, ultrasound should precede pelvic examination to avoid disruption of a possible placenta previa. Bedside transabdominal ultrasound imaging may reveal free intraperitoneal fluid in an unstable patient, which should lead to immediate gynecologic or surgical evaluation.

Uterine size, measured from the symphysis pubis to the fundus, is the quickest means of roughly estimating gestational age. This distance in centimeters equals the gestational age in weeks (e.g., 24 cm = 24 weeks), which allows some early indication of fetal viability if delivery is necessary. Usually, 24 or 25 weeks is used as the cutoff point for fetal viability. As a rough guide, the fetus is potentially viable when the dome of the uterus extends beyond the umbilicus. Fetal heart tones can be detected by auscultation at 20 weeks of gestation or by Doppler probe at 10 to 14 weeks. If either the uterus is less than 24 cm in size or fetal heart tones are absent, the pregnancy is probably too early to be viable, and treatment is directed solely at the mother.

Ancillary Testing

In hemodynamically compromised patients, blood is obtained for hematocrit, platelet count, prothrombin time, partial thromboplastin time, ABO and Rh typing, and cross-matching of blood. Ultrasound is the imaging modality of choice for simultaneous assessment of the mother and the fetus. In the pregnant trauma patient, it is useful in the detection of major abdominal injury (sensitivity 80%, specificity 100%) and for establishing fetal well-being or demise, gestational age, and placental location.[9] Computed tomography and magnetic resonance imaging are rarely indicated in the evaluation of vaginal bleeding, except in the case of pregnant trauma patients to diagnose potentially life-threatening injuries in those patients not proceeding directly to surgical intervention.

Qualitative pregnancy tests in clinical use are typically reported as positive when the β-hCG concentration is 20 mIU/mL or higher in urine and 10 mIU/mL or higher in serum. At this level of detection, the false-negative rate for detection of pregnancy will not be more than 1% for urine and 0.5% or less for serum. In clinical use, the performance of urine qualitative testing has been found to be 95 to 100% sensitive and specific compared with serum testing. When a bedside urine test is negative and ectopic pregnancy is still being considered, a quantitative serum test should be performed. The sensitivity of quantitative serum testing for the diagnosis of pregnancy is virtually 100% when an assay capable of detecting 5 mIU/mL or more of β-hCG is used.[10] The discriminatory level of serum β-hCG for ectopic pregnancy is 1500 to 2000 mIU/mL.[11] Below this level, with no evidence of an intrauterine pregnancy (IUP) on transvaginal ultrasound, ectopic pregnancy as well as normal IUP are still possible. Above this level, ectopic pregnancy is diagnosed by the absence of an IUP on transvaginal ultrasound. In stable patients with minimal symptoms who are below the discriminatory level, serial quantitative β-hCG levels every 48 hours may distinguish ectopic pregnancy from IUP and spontaneous abortion in pregnancies less than 5 to 7 weeks of gestation. A system for close follow-up with gynecology is essential to an outpatient strategy for such patients. Additional testing such as progesterone level may help to distinguish normal verses abnormal pregnancy. A progesterone level of less than or equal to 5 ng/mL indicates a nonviable pregnancy, ectopic pregnancy, or IUP and excludes normal pregnancy with 100% sensitivity (Figs. 27-1 and 27-2).[12]

EMPIRICAL MANAGEMENT

All patients who present in shock with a surgical abdomen or evidence of intra-abdominal free fluid should be resuscitated and promptly evaluated with immediate consideration of operative intervention in consultation with obstetrics/gynecology and surgery.

Pregnant Patients

If ectopic pregnancy is suspected and the serum or urine β-hCG is positive, and the patient is hemodynamically unstable, immediate surgical consultation is indicated. If bleeding presents with shock after the 20th week of pregnancy, stabilization is performed while obtaining a transabdominal ultrasound to evaluate the placenta (location in placenta previa and separation and hemorrhage in placentae abruptio). In the presence of vaginal bleeding in these patients, bimanual or speculum vaginal examination or transvaginal ultrasound should not be undertaken until placenta previa is excluded. High-grade third-trimester bleeding should prompt immediate obstetric consultation, even before diagnostic studies elucidate the possible cause. Vaginal delivery is the preferred management of third-trimester vaginal bleeding in the absence of placenta previa, but cesarean section is indicated if (1) fetal distress is present and vaginal delivery is not imminent, (2) there is severe abruption with a viable fetus, (3) life-threatening hemorrhage exists, or (4) the patient has failed a trial of labor.

Uterine rupture may present with excessive vaginal bleeding, uterine pain, and a change in abdominal contour. A soft horizontal lump often appears below a hard fundus, representing expanding hematoma and a retracting uterus, respectively. Emergent surgical delivery is indicated.

Urgent cesarean section is performed if excessive vaginal bleeding accompanies the rupture of membranes and the fetus

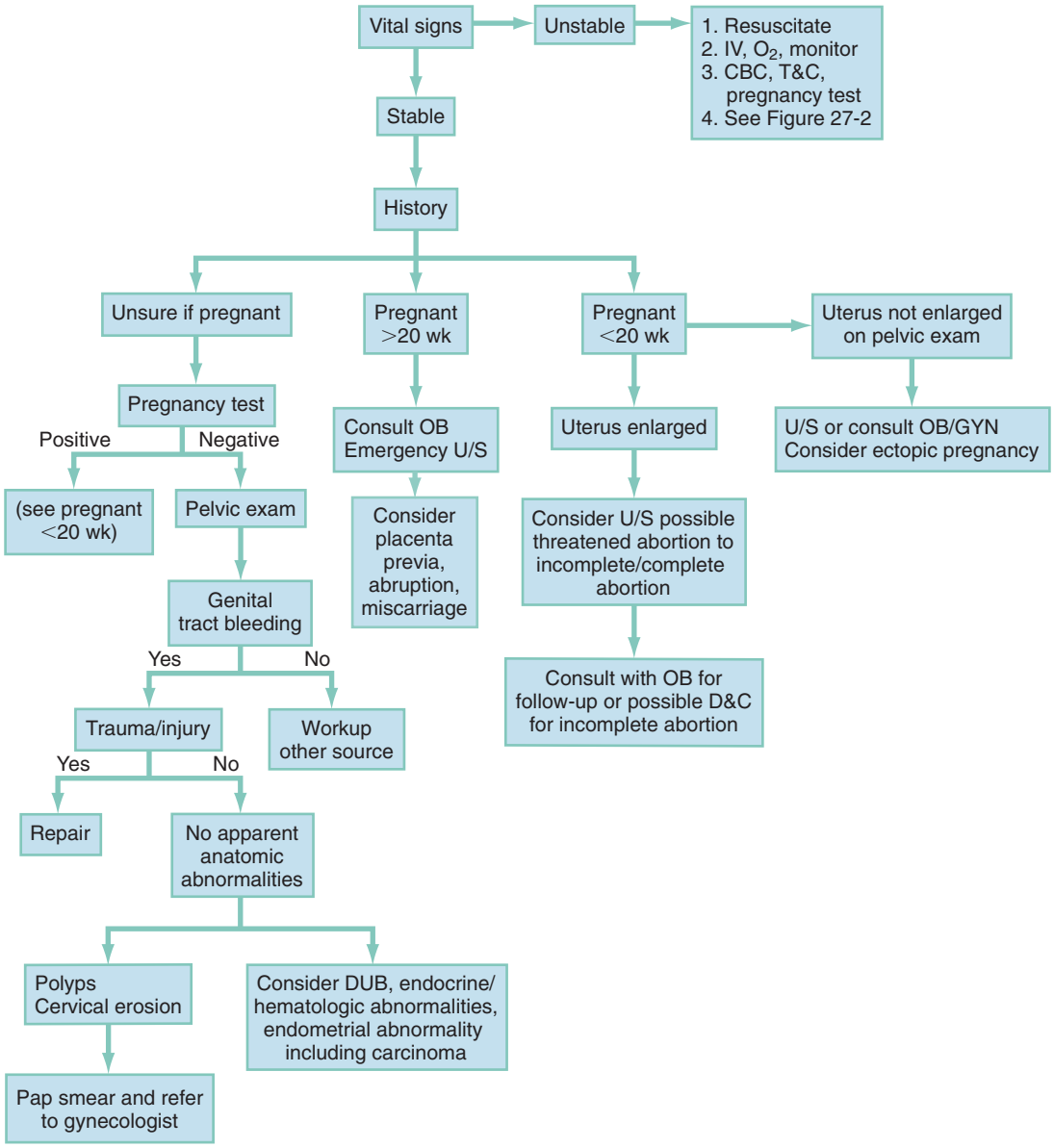


Figure 27-1. Diagnostic approach to patient with vaginal bleeding.

shows signs of distress. Painless vaginal bleeding with rupture of membranes classically suggests vasa previa; it indicates fetal bleeding and requires emergent cesarean section. If after delivery of the fetus the placenta adheres abnormally and has difficulty separating, placenta accreta is likely present and may require urgent hysterectomy to prevent life-threatening hemorrhage. If available, interventional radiology for thromboembolization may be considered. Firm bimanual compression of the uterus or insertion and inflation of a Foley catheter with a 30-mL balloon may limit hemorrhage until surgery is arranged. Uterine atony often responds to vigorous uterine massage and intravenous oxytocin.[13]

Evidence for the administration of anti-D immunoglobulin (Rhogam) for the prevention of Rh seroconversion in pregnant women is limited. Nevertheless, it is recommended to administer anti-D immunoglobulin to Rh-negative women in all cases of documented first-trimester loss of established pregnancy, including threatened abortion, incomplete abortion, and ectopic pregnancy. One may consider administration of anti-D immunoglobulin in cases of minor trauma in Rh-negative pregnant women.[14]

Nonpregnant Patients

In nonpregnant patients, heavy vaginal bleeding may be under ovulatory control or related to anovulatory dysfunctional uterine bleeding. Nonsteroidal anti-inflammatory drugs are the mainstay of treatment for both conditions, although the exact mechanism of action is not clearly understood.[15] In nonpregnant hemodynamically unstable patients, consider administering IV conjugated estrogen (Premarin) 25 mg and repeat doses if necessary until bleeding stops, usually within 1 to 5 hours. If bleeding continues after IV estrogen, insert a pediatric Foley catheter into the cervical os and inflate to tamponade the bleeding. Distend the balloon with saline until the bleeding stops. A larger balloon may be needed and this can be left in place for 12 to 24 hours.[16] Hemodynamically stable patients can be referred for outpatient ultrasound and/or endometrial biopsy. All patients with abnormal uterine bleeding should receive close follow-up from a primary care physician or gynecologist. Outpatient treatment with oral contraceptives can arrest bleeding. Patients older than 35 years or with risk factors for endometrial cancer should have an endometrial

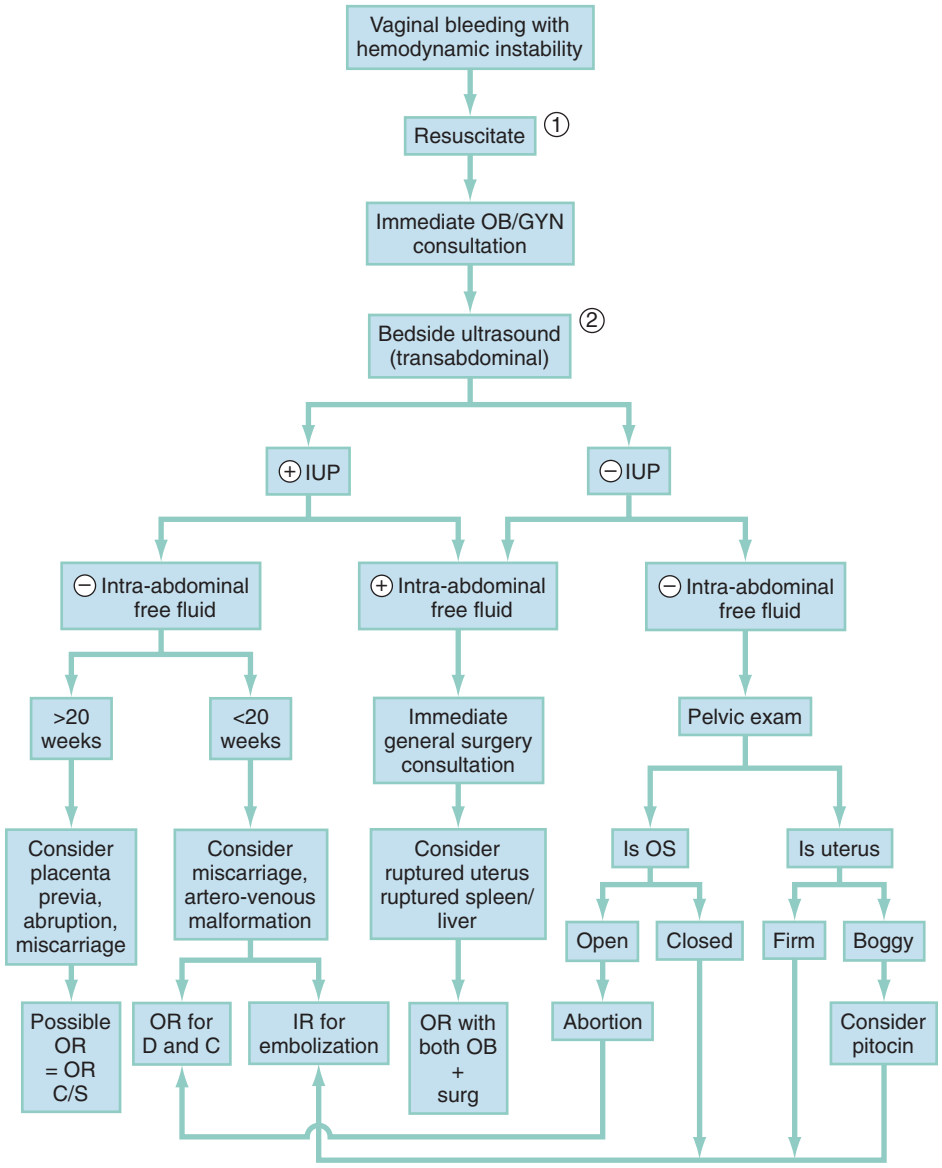


Figure 27-2. Diagnostic approach to unstable patient with vaginal bleeding.

① Consider immediate transfusion with RH negative blood products

② Transvaginal ultrasound is ideal for diagnosis but may not be available at bedside for unstable patients

IR = interventional radiology
D and C = dilitation and curretage
C/S = cesarean section
OR = operating room
IuP = intrauterine pregnancy

biopsy within one week of starting hormonal manipulation. A baseline hemoglobin/hematocrit is recommended. Finally, other medical causes, such as hypothyroidism, hemostasis disorders, or anticoagulant therapy, must be considered and appropriate outpatient consultation obtained.

DISPOSITION

In a patient with postpartum uterine atony or coagulopathy, medical management is often sufficient. Obstetrics consultation is rarely indicated. In a preadolescent patient, abuse must be ruled out before the patient is discharged to her current environment. In a nonpregnant stable patient, malignancy always should be suspected, and additional inpatient or timely outpatient gynecologic workup is indicated. Laboratory studies such as thyroid function and prolactin levels may be helpful to the consultant or in the initial outpatient workup of dysfunctional uterine bleeding, but they are not required in the emergency department setting.[17]

The references for this chapter can be found online by accessing the accompanying Expert Consult website.