Emergency Stroke Care: Advances & Controversies

Edited by
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Andy Jagoda, MD, FACEP

8 Stroke CME Credits

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Emergency Stroke Care: Advances And Controversies, Volume II

Product Preview Information

The information contained herein is a representative sample of the complete product, and it is intended to provide a sense of the quality and comprehensive nature of the product.

This book, published in March 2017, provides an update on advances and controversies in acute stroke care and builds on the foundation laid in Volume I. Six topics were selected based on their relevance to acute management of stroke and on their impact on outcomes. This book is designed to enhance best practices in acute stroke care by providing an evidence-based analysis of the current literature. Advances and controversies in care are presented with the hope that readers will be engaged to proactively discuss practice with their stroke teams and provide the best possible care available to their patients. Volumes I and II pair perfectly together.

Included In This Book:
1. 70 pages of evidence-based content, covering 6 critical topics
2. 8 AMA PRA Category 1 Credits™ that are stroke specific
3. Summarized information to help you keep up with current guidelines and best practices
4. Treatment recommendations to help you determine the critical actions required when caring for these patients
5. And much more!

The 6 topics covered in this volume address some of the most pressing concerns for emergency clinicians:
1. Stroke Systems Of Care
2. Aneurysmal Subarachnoid Hemorrhage
3. Cerebral Venous Sinus Thrombosis
4. Ischemic Stroke Of The Posterior Circulation
5. Intracranial Hemorrhage
6. Shared Decision-Making In Acute Stroke Care

This product is available in print and online. Each order includes access to the pdf version of the book.
Target Audience: This activity is intended for board-certified emergency medicine clinicians.

Course Director: Andy Jagoda, MD, FACEP, Professor and Chair, Department of Emergency Medicine, Icahn School of Medicine at Mount Sinai, Medical Director, Mount Sinai Hospital, New York, NY

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Acute ischemic stroke continues to be a leading cause of death and disability worldwide. From a population health perspective, prevention strategies are of primary importance. However, when a stroke does occur, health systems must be prepared to maximize access to resources that have been shown to promote good outcomes. Since the National Institute of Neurological Disorders and Stroke trial was published in 1995 demonstrating the benefit of intravenous recombinant tissue-type plasminogen activator (tPA), there has been continued innovation in improving our ability to diagnose and treat stroke, and as a result there has been a decrease in the related morbidity and mortality. Emergency Stroke Care: Volume II provides an update on advances and controversies in acute stroke care and builds on the foundation laid in Volume I. Six topics were selected based on their relevance to acute management of stroke and on their impact on outcomes.

- **Chapter 1** presents recent changes in how stroke centers are designated, with a focus on two critical issues: the new designation of Acute-Stroke-Ready Hospitals (ASRHs), and the impact of endovascular interventions on field transport decision-making. ASRHs are hospitals that have defined stroke care protocols and resources, and are often augmented with “telemedicine” capabilities and linked to primary and comprehensive stroke centers, thus expanding access to advanced consultation and care. Regarding endovascular therapy, five trials have confirmed the benefit of this therapy in the appropriate patient, thus pushing the envelope of who should be transported, when, and where, and placing tremendous new responsibility on the emergency medical services community.

- **Chapters 2 and 3** provide updates on diseases that generally present with the common complaint of headache and that are associated with misdiagnoses when not considered or properly evaluated. Chapter 2 addresses subarachnoid hemorrhage and Chapter 3 addresses cerebral venous thrombosis. Both disease states require an understanding of the clinical progression in order to risk stratify the timing and choice of diagnostic testing. Both are associated with a significant mortality if not diagnosed and appropriately treated. Advances in imaging have changed the diagnostic paradigm in both of these disease states.

- **Chapter 4** addresses posterior circulation strokes and explores why there often is a delay in diagnosis and thus a missed opportunity for therapeutic intervention. Posterior circulation strokes are missed more often than anterior circulation events primarily because of their often-subtle presentation. This chapter provides a systematic approach designed to promote diagnostic accuracy. Likewise, **Chapter 5** explores intracranial hemorrhage and advances in management that lead to improved outcomes in these unstable patients.

- **Chapter 6** explores the topic of shared decision-making and its applications in acute stroke care. Shared decision-making differs from informed consent and, when used correctly, it promotes communication and patient satisfaction. The risk for intracranial hemorrhage from IV tPA in some patients with acute ischemic stroke is as low as 1%, while in others it is more than 15%. This important chapter introduces tools that are useful in facilitating the discussion with patients and families related to risk and benefit of acute interventions; these tools also help clinicians in their decision-making.

Emergency Stroke Care: Volume II is designed to enhance best practices in acute stroke care by providing an evidence-based analysis of the current literature. Advances and controversies in care are presented with the hope that readers will be engaged to proactively discuss practice with their stroke teams and provide the best possible care available to their patients. Excellent outcomes in acute stroke, as in most all emergent conditions, are dependent on fully integrated and engaged prehospital and emergency department providers. Without a strong front line, there is little hope for achieving best possible outcomes for our patients. We hope you enjoy reading this volume and we welcome your feedback and input for future volumes.

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Introduction

Over the past 20 years, stroke systems of care have been developed to maximize outcomes for patients affected by acute stroke by improving the integration and alignment of the component elements. In this context, the concept of the “stroke center” emerged in order to establish standard elements of care that include acute stroke teams, written protocols, emergency department (ED) and stroke unit provisions, connections with emergency medical services (EMS), and quality improvement. In general, stroke systems encompass the entire spectrum of care from prevention to long-term rehabilitation; however, this chapter will discuss the emergent care of stroke patients, including EMS activation and treatment, rapid transfer to facilities capable of rapid diagnostic testing, and thrombolytic treatment to eligible patients.1,2

In 2015, 5 randomized controlled trials were published that demonstrated the benefit of interventional thrombectomy for large-vessel occlusion (LVO) strokes.3-7 This advance in stroke treatment has the potential to dramatically improve outcomes for patients; however, it also created new challenges for stroke systems of care, specifically EMS and hospital-based stroke teams. The challenges raised included the following questions:

• How should patients with LVO strokes be identified in the field?
• How should the benefit of rapid administration of intravenous tissue-type plasminogen activator (IV tPA) at a local hospital be weighed against rapid endovascular care at a more distant hospital?
• Should imaging and treatment be performed in the field?
• Is there a role for telemedicine in the prehospital environment?

This chapter provides an overview of current trends and future directions in acute stroke care for the EMS and the emergency medicine communities.

Selected Abbreviations

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<th>Description</th>
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<tr>
<td>ASRH</td>
<td>Acute-Stroke-Ready Hospital</td>
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<td>BEST-MSU</td>
<td>Benefits of Stroke Treatment Delivered Using a Mobile Stroke Unit trial</td>
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<td>CPSS</td>
<td>Cincinnati Prehospital Stroke Scale</td>
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<td>CSC</td>
<td>Comprehensive Stroke Center</td>
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<tr>
<td>CVA</td>
<td>Cerebrovascular accident</td>
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<td>EMD</td>
<td>Emergency Medical Dispatch</td>
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<td>EMS</td>
<td>Emergency Medical Services</td>
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<td>Extending the Time for Thrombolysis in Emergency Neurological Deficits — Intra-Arterial trial</td>
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<td>HIPAA</td>
<td>Health Insurance Portability and Accountability Act</td>
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<tr>
<td>IV tPA</td>
<td>Intravenous tissue-type plasminogen activator</td>
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<td>LAMS</td>
<td>Los Angeles Motor Scale</td>
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<tr>
<td>LVO</td>
<td>Large-vessel occlusion</td>
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<td>MPDS</td>
<td>Medical Priority Dispatch System</td>
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<tr>
<td>MR CLEAN</td>
<td>Multicenter Randomized Clinical Trial of Endovascular Treatment for Acute Ischemic Stroke in the Netherlands</td>
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<td>mRS</td>
<td>Modified Rankin Scale</td>
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<td>MSU</td>
<td>Mobile stroke unit</td>
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<td>NIHSS</td>
<td>National Institutes of Health Stroke Scale</td>
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<td>PHANTOM-S</td>
<td>Prehospital Acute Neurological Treatment and Optimization of Medical Care in Stroke Study</td>
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# Stroke Systems Of Care

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Aneurysmal Subarachnoid Hemorrhage

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CME OBJECTIVES:  
Upon completion of this article, you should be able to:  
1. Describe the characteristics of acute nontraumatic headache that raise suspicion for aneurysmal subarachnoid hemorrhage.  
2. Identify the most important factors that contribute to early aneurysmal rebleeding and how these can be treated in the emergency department.

Prior to beginning this activity, see the CME information on page 3.

Introduction

In the United States, it is estimated that there are 5.4 million annual emergency department (ED) visits for atraumatic headache, representing roughly 4.5% of all ED visits.1 Among presentations for acute atraumatic headache, no diagnosis has as great a potential for devastating consequences, if missed, as aneurysmal subarachnoid hemorrhage (SAH). The clinical presentation of ruptured “berry” aneurysms occurs along a broad spectrum that ranges from moderate headache at its most subtle, to a more-severe headache with neurologic deficits, to coma and sudden death. Generally speaking, the emergency clinician faces 2 areas of concern with regard to aneurysmal SAH: (1) when to suspect it and how to make the diagnosis, and (2) how to manage the patient once SAH has been confirmed. Over the last several years, advances have been made in both of these areas that the emergency clinician should be aware of.

Clinical Presentation

The classic presentation of aneurysmal SAH is the sudden onset of severe headache, usually described as “the worst headache of my life.”2 However, the degree of the presenting headache is much less important than the suddenness of the onset. Headache may or may not be accompanied by nausea or vomiting, meningismus, and loss of consciousness. In a case series of 109 patients with SAH, headache occurred in 74%, while nausea/vomiting occurred in 77%, loss of consciousness in 53%, and meningismus in 35%, making these signs insensitive for the diagnosis.3 While aneurysm rupture may occur during physical stress or exercise, about half of ruptures occur during nonstrenuous activities, rest, or sleep.4

Misdiagnosis of SAH occurs in 6% to 12% of patients and is associated with devastating outcomes.5 The largest proportion of these misdiagnoses have been documented to occur in ED settings. Patients who present with Hunt and Hess scores of 1 or 2 (mild or severe headache without focal neurologic symptoms; calculation tool available at: https://www.mdcalc.com/hunt-hess-classification-subarachnoid-hemorrhage and whose SAHs were missed were roughly 3 times more likely to be severely disabled and 4 times as likely to die as patients who were diagnosed correctly.6 Patients are most commonly misdiagnosed with tension or migraine headache or the common cold. In the United States, SAH patients who are misdiagnosed are less likely to be fluent in English, less likely to have graduated from high school, and are more likely to have mild initial clinical presentations.6 These factors suggest that clear communication about the precise nature of the headache is important in generating suspicion of the diagnosis on the part of the emergency clinician.

Another important predictor of misdiagnosis has been clinical presentation 1 or more days after the onset of symptoms. Emergency clinicians tend to expect that SAH patients will have severe headache and present to the ED immediately. This finding suggests that clinician bias exists, resulting in the dismissal of patients who present several days after their headaches started, likely because there is a belief that patients with SAH would not wait several days to be evaluated.

The most important question that should be asked when screening patients with atraumatic headache is how the headache started. Even in patients with headache that seems mild to the examiner, report of time to peak intensity of 30 to 60 minutes from onset should raise the possibility of aneurysm rupture.

In a group of patients who reported the rapid progression of headache to peak intensity within 60 minutes, Perry et al validated a clinical decision rule (Ottawa SAH Rule table) to identify patients who should receive imaging and lumbar puncture to rule out SAH, with a sensitivity of 100%.7 In this study,
Aneurysmal Subarachnoid Hemorrhage

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Introduction

Cerebral venous sinus thrombosis (CVT) is a rare but underdiagnosed condition, accounting for approximately 1% of all strokes. The diversity of symptoms in CVT, coupled with often-nonspecific neurologic findings, can lead to a significant delay in diagnosis for many patients. While most cases have a favorable outcome, mortality can be as high as 5% to 10%. Early diagnosis is facilitated by identifying risk factors, maintaining a high clinical suspicion, and ordering appropriate imaging. Initial treatment involves anticoagulation and treatment of any precipitating conditions. Some controversy exists regarding the use of anticoagulation in the presence of hemorrhage, though it remains a recommendation in most guidelines. Thrombolysis or mechanical endovascular interventions may have a role in severe cases.

Venous Anatomy Of The Brain

In the same way that the vascular territories involved in arterial ischemic strokes correspond to specific clinical syndromes, the location of thrombosis within different venous channels helps to explain the diversity of clinical presentations with CVT. Seventy percent of the intracranial blood volume is located in the venous system.1 In the context of CVT, this system can be simplified into 3 categories: (1) the superficial penetrating cerebral veins, (2) the deep venous system, and (3) the sinuses. (See Figure 1.)

Superficial penetrating cortical veins drain into either sinuses or the deep venous system. Occlusion of these veins leads to dysfunction in the cerebral cortex that manifests as neurologic deficits or seizures. Due to variability in collateral venous circulation, patients with similar patterns of venous occlusion may differ substantially in their clinical presentations.2

The Sinuses

The sinuses are venous channels located between reflections of the dura mater. They include the superior and inferior sagittal sinuses, the straight sinus, the transverse or lateral sinuses, the sigmoid sinuses, and the cavernous sinuses. The sinuses receive blood from the deep venous system as well as penetrating cortical veins, and they channel the blood to the internal jugular veins. The superior sagittal sinus drains most of the blood from the cerebral hemispheres, in addition to resorbing cerebrospinal fluid through the arachnoid granulations. It is an unpaired sinus located in the sagittal plane running over the superior aspect of the midline, draining posteriorly into the confluence of sinuses.

The confluence of sinuses is located posteriorly and receives blood from the superior sagittal sinus as well as the straight sinus, draining into the transverse sinuses bilaterally. The transverse sinuses...
# Cerebral Venous Sinus Thrombosis

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Introduction

One-fifth of all ischemic strokes occur in the territory supplied by the posterior circulation, also known as the vertebrobasilar system.\textsuperscript{1,2,3} The evaluation of posterior circulation stroke is often not systematically performed by healthcare providers, and the diagnosis is more commonly missed when compared to anterior circulation stroke.\textsuperscript{4,5} Posterior circulation stroke is also associated with delayed assessment by a neurologist and increased door-to-needle time.\textsuperscript{6}

Neuroanatomy

The posterior circulation supplies blood to the brainstem, thalamus, cerebellum, occipital lobe, and inferomedial temporal lobe. (\textit{See Figure 1.}) The posterior circulation begins as 2 vertebral arteries, each originating from a subclavian artery. Starting at C6, the vertebral arteries ascend in the transverse foramina of the cervical vertebrae, and then enter the foramen magnum to pierce the dura. Intracranially, the vertebral arteries each give rise to a posterior inferior cerebellar artery and subsequently merge to form the basilar artery at the pontomedullary junction. The branches of the basilar artery include the anterior inferior cerebellar artery, the pontine perforators, and the superior cerebellar artery. At the top of the pons, the basilar artery bifurcates into 2 posterior cerebral arteries. The vertebrobasilar system joins the anterior circulation at the circle of Willis, a vascular arrangement that facilitates collateral blood supply when a main branch is occluded.

A common anatomical variant is the fetal origin of the posterior cerebral artery, which is present in 20% to 30% of adult humans.\textsuperscript{7-9} This occurs when the proximal segment of the posterior cerebral artery (P1) is either hypoplastic or absent. Thus, most of the blood to the posterior cerebral artery arrives from the posterior communicating artery, rendering the posterior cerebral artery part of the anterior circulation. In such a circumstance, occlusion of the ipsilateral internal carotid artery can lead to occipital lobe infarction, among other possibilities.
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Ischemic Stroke Of The Posterior Circulation
Intracranial Hemorrhage

Kaitlin Reilly, MD
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CME OBJECTIVES:
Upon completion of this article, you should be able to:
1. Compare the different etiologies for intracerebral hemorrhage and their patient characteristics and imaging finding.
2. Describe the indications for active blood pressure control in the setting of intracranial hemorrhage.
3. List indications and strategies for reversal of anticoagulant and antiplatelet drugs in the setting of intracranial hemorrhage.

Prior to beginning this activity, see the CME information on page 3.

Introduction

Despite advances in therapy, intracranial hemorrhage (ICH) remains the most lethal form of stroke, with only 38% of patients surviving the first year. In the United States, ICH represents 10% to 15% of all stroke cases, with 37,000 to 52,400 patients affected per year. Worldwide, over 1 million patients suffer ICH per year. ICH can be divided into 2 categories: (1) primary ICH, which is caused by disease of the blood vessels in the brain due mainly to hypertension (80%) or amyloid angiopathy (20%); and (2) secondary ICH, which is due to an underlying structural brain lesion, such as a brain tumor or an arteriovenous malformation. Many patients with ICH worsen due to expansion of the hematoma and the development of cerebral edema and herniation, so prompt identification of the signs and symptoms of ICH within the emergency department (ED) setting is critical. This chapter discusses best practices in the identification and initial management of ICH.

Selected Abbreviations

ANNEXA-4 A Study in Patients With Acute Major Bleeding to Evaluate the Ability of Andexanet Alfa to Reverse the Anticoagulation Effect of Direct and Indirect Oral Anticoagulants
ATACH-II Antihypertensive Treatment of Acute Cerebral Hemorrhage-II Trial
DDAVP Desmopressin
ICH Intracranial hemorrhage
INTERACT2 Intensive Blood Pressure Reduction in Acute Cerebral Hemorrhage Trial 2
PATCH Platelet Transfusion Versus Standard Care After Acute Stroke Due To Spontaneous Cerebral Haemorrhage Associated With Antiplatelet Therapy

RE-VERSE AD Reversal of Dabigatran Anticoagulant Effect with Idarucizumab
STICH, STICHII International Surgical Trial in Intracerebral Haemorrhage

Etiology And Differential Diagnosis

Primary Intracranial Hemorrhage

The most common cause of primary ICH, both in the United States and around the world, is hypertension. Chronic hypertension causes pathological changes to the arterioles that feed the brain’s deep structures, leading to lipohyalinosis and Charcot-Bouchard aneurysms that weaken the vessel wall and lead to spontaneous rupture. Because these arterioles are more prone to disease, the locations of hypertensive ICHs follow the distribution of the small feeding vessels and are predictable: putamen, caudate, thalamus, pons, and cerebellar vermis, all so-called “deep” structures. In contrast, lobar hemorrhages (spontaneous hemorrhages involving the frontal, temporal, parietal, and occipital lobes) arise from the superficial vessels feeding the cerebral cortex. These hemorrhages are less likely to be caused by hypertension and are most likely, particularly in the elderly, to be due to cerebral amyloid angiopathy, which is caused by deposition of beta-amyloid protein into the vessel walls, making them weak and prone to spontaneous rupture. These 2 pathologic processes account for the overwhelming majority of ICHs.

Secondary Intracranial Hemorrhage

The causes of secondary ICH, or ICH due to an underlying structural lesion, are much more diverse. They can be divided into 5 categories. (See Table 1, page 52.) These causes should be considered in any patient presenting with an ICH, but it is particularly important to identify a venous sinus thrombosis early because, unique among ICHs, venous sinus thrombosis is treated with anticoagulation. Additionally, patients with lobar ICH or primary intraventricular hemorrhage should undergo vascular imaging to rule out an underlying vascular lesion.
Intracranial Hemorrhage

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Shared Decision-Making In Acute Stroke Care

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CME OBJECTIVES
Upon completion of this article, you should be able to:
1. Describe how the criteria for shared decision-making apply to discussions regarding the use of intravenous tissue-type plasminogen activator (IV tPA) in acute ischemic stroke.
2. Summarize how risk estimates can be translated into decision aids for patients and clinicians.

Prior to beginning this activity, see the CME information on page 3.

Introduction

Whether or not to administer intravenous recombinant tissue-type plasminogen activator (IV tPA) for patients presenting with acute ischemic stroke is the most important and challenging decision in acute stroke care. This therapy has well-documented potential benefits (improved functional outcome) and potential harms (symptomatic intracranial hemorrhage). Depending on the balance between the benefits and the risks, patients and families may have opinions that are different from their clinicians regarding their preferred management. Under certain circumstances, comprehensive stroke care without IV tPA may be a reasonable option. Under other circumstances, administration of IV tPA may result in clear, substantial benefit. Thus, this clinical scenario creates an opportunity for engaging patients and families using an approach known as shared decision-making (SDM). In this chapter, we present the current literature on the topic of SDM in acute ischemic stroke, the potential barriers, and the tools that can facilitate the process.

Shared Decision-Making In The Emergency Department

The care of patients who present to the emergency department (ED) with acute ischemic stroke can be challenging for the emergency clinician. If patients present within 4.5 hours of symptom onset, those who meet treatment criteria are eligible to receive IV tPA. This therapy—the only United States Food and Drug Administration (FDA)-approved treatment for acute ischemic stroke—is associated with the potential benefits of improved functional outcome as well as potential harm, primarily symptomatic intracranial hemorrhage (ICH) and other bleeding complications. The benefit-to-harm ratio will differ as a function of individual patient clinical variables. When the likelihood of harm exceeds the likelihood of benefit, it would be appropriate to recommend against thrombolytic therapy; however, in many cases, the balance between the benefit versus the risk favors treatment. In these cases, tPA should be offered. Since the trade-off can be perceived differently by different patients and their families, SDM can play a critical role in clinical management. This point is underscored in the recent American College of Emergency Physicians Clinical Policy on the management of acute ischemic stroke.

SDM has been proposed as a method to actively engage patients in their healthcare decisions. SDM is defined as a “collaborative process in which patients and providers make healthcare decisions together, taking into account the best evidence available, as well as the patient’s values and preferences.” In SDM, both clinician and patient share information: the clinician offers options and describes their risks and benefits, while the patient expresses what he or she values most. Through an open dialogue, collaborative deliberation is performed, and a decision is made about how to proceed.

For SDM to be appropriate in emergency medicine, 3 criteria must be present: (1) clinical equipoise, (2) patient decision-making ability, and (3) time. (See Figure 1.) When these criteria are not met, other decision-making approaches will be employed. If only 1 medically reasonable option exists (eg, antibiotics for bacterial pneumonia), the

Figure 1. When Is Shared Decision-Making Appropriate In The Emergency Department?

- IS THERE MORE THAN ONE REASONABLE OPTION?
  - No: Compassionate Persuasion, Informed Consent or Refusal
  - Definitively or potentially yes: Extensive Discussion

- IS PATIENT ABLE TO PARTICIPATE IN DECISION?
  - No: Paternalism
  - Definitively or potentially yes: Extensive Discussion

- IS THERE TIME TO ENGAGE PATIENT IN DISCUSSION?
  - No: Unilateral Action
  - Definitively or potentially yes: Extensive Discussion

 Courtesy of Marc A. Probst, MD; and Maggie Breslin, MDes.
Chapter 6

Shared Decision-Making In Acute Stroke Care

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