High-Risk Chief Complaints II: Disorders of the Head and Neck

Lauren Nentwich, MD[a,*], Andrew S. Ulrich, MD[b]

KEYWORDS

• Headache • Seizure • Acute neurologic deficits • Throat pain
• Neck pain • Ocular emergencies • Difficult airway

Of the many different complaints of patients presenting to the emergency department (ED), some of the most difficult to diagnose and manage involve pathology of the head and neck. Often diagnoses of conditions affecting this part of the body are elusive, and occasionally, even once the diagnosis has been made, the management of these disorders remains challenging. This article addresses some of the high-risk chief complaints of the head and neck regarding diagnosis and management. The high-risk chief complaints that are discussed in this review include headache, seizure, acute focal neurologic deficits, throat and neck pain, ocular emergencies, and the difficult airway.

HEADACHE

Headache is a common complaint of patients presenting to the ED. A query of the National Hospital Ambulatory Medical Care Survey for 1999 to 2001 found that headache accounts for 2.1 million ED visits per year, or 2.2% of total ED visits.[1] Most patients with headache suffer from a benign primary headache, such as migraine, tension-type, cluster, or other primary headaches.[2] However, a smaller but notable percentage of patients (up to 19%) have headaches due to secondary causes including those that are life-threatening and caused by serious intracranial pathology, such as vascular disorders, infections, or tumors.[3] A study of malpractice claims against emergency physicians in Massachusetts found that 3.6% of all claims were in regard to patients with central nervous system (CNS) bleeding, many of whom present with a chief complaint of headache.[4] As such, when treating patients who present with acute headache, it is the primary role of the emergency physician to accurately differentiate between benign headaches and those secondary to intracranial pathology. This section

[a] Department of Neurology, Stroke Research Center, Massachusetts General Hospital, 175 Cambridge Street, Suite 300, Boston, MA 02114, USA
[b] Department of Emergency Medicine, Boston Medical Center, Boston University School of Medicine, 1 Boston Medical Center Place, Boston, MA 02118, USA
* Corresponding author.
E-mail address: lnentwich@partners.org (L. Nentwich).

Emerg Med Clin N Am 27 (2009) 713–746
doi:10.1016/j.emc.2009.08.002 **emed.theclinics.com**
0733-8627/09/$ – see front matter

discusses the important high-risk features to consider when evaluating patients who present to the ED with headache.

Age

New-onset headache in older patients is worrisome, as age 50 years and older has been shown to be an independent predictor of intracranial pathology.[1,5,6] An analysis of the data from the National Hospital Ambulatory Medical Care Survey from 1999 to 2001 showed that patients with headache who were age 50 years and older had a 6% rate of pathologic findings and an 11% admission rate compared with 1% and 4%, respectively, for younger patients.[1] As such, emergency physicians must be vigilant in the evaluation of older patients presenting with headache, and should maintain a lower threshold for obtaining additional testing. Current recommendations advise that patients older than 50 years presenting with new type of headache but normal neurologic examination should be referred for an urgent neuroimaging study.[7]

History

Onset

Patients who present with a history of acute, sudden-onset severe headache are at increased risk for intracranial pathology. The differential diagnosis for sudden-onset, severe headache is broad and includes many serious causes, such as subarachnoid hemorrhage, intracerebral hemorrhage, cerebral venous sinus thrombosis, arterial dissection, or pituitary apoplexy.[6–8]

The most common serious pathology associated with sudden-onset, severe headache is spontaneous subarachnoid hemorrhage (SAH), and patients classically present with a thunderclap ''worst headache of life.'' In addition to the headache, signs and symptoms of SAH may include nausea or vomiting, photophobia, neck pain or nuchal rigidity, loss of consciousness, or focal neurologic deficits. SAH is a medical emergency that is frequently misdiagnosed, and patients presenting with sudden-onset, severe headache should evoke a high level of suspicion for SAH. Noncontrast cranial computed tomography (CT) is the initial test of choice in making the diagnosis, but the sensitivity of CT in the diagnosis of SAH declines with time from onset of symptoms. In the first 12 hours after SAH, the sensitivity of CT is 98% to 100%, declining to 83% at 24 hours, and to 57% to 85% 6 days after SAH onset. Therefore, lumbar puncture to look for red blood cells (RBCs) or xanthochromia must be performed in all patients with a high suspicion of SAH and a negative cranial CT.[9] Given the high morbidity and mortality of SAH, patients who present with new sudden-onset severe headache should undergo emergent cranial CT and then lumbar puncture for analysis of the cerebrospinal fluid if the cranial CT is normal.[7–9] If both tests are normal then SAH is adequately excluded, and patients may be discharged or undergo further evaluation for other causes of sudden-onset severe headache as clinically indicated.[7]

Response to analgesics

Some clinicians have tried anecdotally to use a favorable response to analgesics as an indication of a benign cause of headache, but this is not a reliable indicator to rule out intracranial pathology. The current understanding of headache suggests a single mechanism for all head pain regardless of the etiology. In addition, the placebo effect in combination with the possible nonspecific sedating effects of the analgesia may produce an altered response. As such, a positive response to therapy cannot be used as a diagnostic indicator of the underlying cause of acute headache, and further evaluation should be pursued as clinically indicated.[7,10]

Physical Examination

Abnormal neurologic examination

A careful neurologic examination is mandatory in all patients who present for evaluation of headache, and an abnormal finding on neurologic examination is the strongest clinical predictor of intracranial pathology.[5,6,11] Ramirez-Lassepas and colleagues[5] reviewed 468 records and found an abnormal finding on neurologic examination to have a highly significant association with intracranial pathology, with a positive predictive value of 39%. Abnormalities can be focal, such as a motor or sensory deficit, or nonfocal, such as altered mental status or papilledema.[5,6,11] Abnormal focal neurologic signs and symptoms usually indicate a stroke or mass lesion. Whereas an abnormal nonfocal neurologic examination can also be seen with these pathologies, it is generally observed in CNS infectious processes, idiopathic intracranial hypertension, or a toxic-metabolic process.[2] Given the strong correlation between abnormal neurologic examination and severe intracranial pathology, a new headache combined with an abnormal neurologic finding is an indication for emergent neuroimaging[7] as well as further testing as clinically indicated.

Associated symptoms

The presence of associated symptoms with headache has been shown in certain studies to be predictive of intracranial pathology. Ramirez-Lassepas and colleagues[5] showed an independent association between the presence of associated symptoms and intracranial pathology but did not describe the associated symptoms. In a prospective observational study of 589 consecutive patients presenting with headache by Locker and colleagues,[6] patients with headache who presented because of associated symptoms were found to have a higher likelihood of intracranial pathology. Of these patients presenting due to associated features, 52% complained of neurologic symptoms other than headache whereas 48% complained of nonneurologic symptoms such as collapse, nausea and vomiting, and nonneurologic symptoms related to otolaryngology, respiratory, or gastrointestinal systems.[6] The American College of Emergency Physicians clinical policy suggests that associated symptoms that should prompt consideration of neuroimaging to evaluate for intracranial pathology include worsening of headache with Valsalva, headache waking patient from sleep, and headache associated with syncope, nausea, or sensory distortion.[7] In patients presenting with headache with other evidence of systemic illness (such as fever, signs of meningeal irritation, or cutaneous rash), a CNS infectious process should be considered.[2]

Medical History

Human immunodeficiency virus infection

Human immunodeficiency virus (HIV) is a neurotrophic virus that easily crosses the blood-brain barrier, accounting for a variety of neurologic complications. CNS complications of HIV infection include HIV encephalopathy, aseptic meningitis, opportunistic infections of the CNS, and neoplasms of the CNS. Many of these entities produce headache, and need to be ruled out when caring for the HIV-infected patients complaining of headache.[12] A prospective descriptive study performed by Rothman and colleagues[13] studied 110 HIV-infected patients to determine predictors of new focal CNS lesions. In their study, 17% of patients were found to have a new focal lesion. The 4 clinical findings that were independently associated with focal findings on cranial CT were new seizure, decreased or altered orientation, headache that was different in quality from prior headaches, and prolonged headache of duration 3 days or longer. In addition, focal motor deficit had a high positive predictive value for a new focal lesion. Current

recommendations advise that HIV-infected patients with a new or prolonged headache should be considered for emergent neuroimaging study.[7]

Malignancy

Headache is a common neurologic complaint in patients with cancer. Cancer patients can have multiple serious causes of their headaches, including intracranial metastases and hemorrhage into intracerebral tumor or metastases. Prospective observational studies have found intracranial metastases in 32% to 54% of cancer patients referred for evaluation of new or changed headache.[14,15] When evaluating patients for possible intracranial metastases, magnetic resonance imaging with contrast is the most sensitive and specific test to evaluate for brain metastases. Noncontrast cranial CT may be use as an emergency imaging modality in patients presenting with symptoms suggestive of stroke or CSF flow obstruction. However, noncontrast or contrast CT does not adequately exclude all brain metastases,[16] and contrast-enhanced magnetic resonance imaging (MRI) should be obtained in all cancer patients with new or changed headache.

Pregnancy

The majority of pregnant women with headaches have benign causes.[7] However, women do have an increased risk of both ischemic and hemorrhagic strokes associated with pregnancy, and the 3 days surrounding delivery up to 6 weeks postpartum is the time of highest risk.[17] In addition, pregnancy is considered a risk factor for developing cerebral venous thrombosis,[18] and the initial presentation of eclampsia may be a chief complaint of headache. No firm recommendations for the evaluation of headache in pregnancy exist,[7] but given the increased risk, intracranial pathology as well as preeclampsia should be considered in all pregnant and early postpartum women presenting with headache.

Pediatrics

Headache is a common complaint in children and adolescents. Although most headaches in children are benign due to either primary headache or benign secondary causes, it has been shown that secondary life-threatening headaches occur in 4.1% to 6.9% of children presenting to the ED with a chief complaint of headache. The neurologic etiology of the headaches attributed to dangerous intracranial disease includes subdural hematoma, epidural hematoma, ventriculoperitoneal shunt malformation, brain abscess, brain tumor, pseudotumor cerebri, aseptic meningitis, and brain malformation (such as Chiari type I).[19,20] A retrospective chart review of 277 children presenting to the ED with headache where 6% of the children had a life-threatening headache found several factors associated with life-threatening headache compared with benign headaches. The factors included preschool age, onset of headache attacks less than 2 months before presentation, pain located in the occipital region or patients unable to describe location of pain, patients unable to describe quality of pain, very intense pain, and associated neurologic signs. All children in this study with serious underlying neurologic conditions had objective neurologic signs, such as: papilledema, ataxia, hemiparesis, and abnormal eye movements.[19]

SEIZURE

Seizures are a common presenting complaint to EDs, and by some estimates, seizures account for 1% to 2% of ED visits.[21,22] Epilepsy, a condition resulting in recurrent, unprovoked seizures, effects as many as 4 million people.[23,24] Seizures that occur as a result of insults to the brain or neurologic system, such as anatomic, toxic, or metabolic abnormalities, are referred to as secondary or reactive seizures. One of

the primary goals of ED management of seizures is the identification and treatment of reversible causes of secondary seizures. Rapid identification and initiation of treatment is critical to limit or prevent end organ damage or permanent neuronal injury, which can result from prolonged uncontrolled seizure activity. In this section the high-risk features in the evaluation and management of patients who present to the ED with seizures is addressed.

Differentiating Between Primary and Secondary Seizures

Estimated incidence of first-onset seizure accounts for approximately 0.3% of all ED visits. Patients who present with active seizures are often easily identifiable; however, some patients may present after events that are more difficult to categorize as seizures, such as sudden loss of consciousness, myoclonic movements, and incontinence. In these circumstances, patients need to have other conditions ruled out before being diagnosed with first-time seizure.

The most common serious condition that can be misinterpreted as a seizure is syncope. [25] It is essential to appropriately identify syncope, as it carries a high risk for significant morbidity and mortality if unrecognized and misdiagnosed as seizure activity. There can be important clinical signs or preceding events that help differentiate between seizure and syncope. Patients with seizure activity may have prodromal symptoms, including stereotyped smells or taste, lip smacking, or a sensation of ''déjà vu.'' Periseizure activity may include oral trauma such as tongue biting. Postictal confusion or agitation may also be present. To differentiate, syncope is often preceded by lightheadedness, diaphoresis, chest pain, or palpitations. Syncopal episodes may have clear precipitants such as prolonged standing, painful stimuli, sudden neck turning, or micturition. Postsyncopal return to baseline tends to be more rapid than recovery from seizures. In many circumstances patients will be unable to provide critical information, thus it is important to obtain an accurate description from anyone who witnessed the event.

Aside from syncope, several other medical conditions should be included in the differential diagnosis of seizures (**Box 1**).

Evaluation

Laboratory testing

It is recommended that serum electrolytes be checked in patients with new onset seizures, as 2.4% to 8% of patients who present with seizures will have electrolyte abnormalities.

Hypoglycemia is a common cause of seizures. There is no definitive plasma glucose threshold regarding seizures; however, levels approaching 45 mg/dL are considered potential for inducing seizures. Some patients may become ictal at higher levels. Patients with possible seizure activity should have rapid bedside/field finger stick test performed to evaluate for low serum glucose. Rapid infusion of glucose should be initiated and should be repeated if patient remains hypoglycemic. Glucagon, 1 mg intramuscularly or subcutaneously, can be given to hypoglycemic patients without intravenous access as a temporizing measure.

Although rare, various electrolyte disturbances may precipitate seizures. Hypernatremia, usually a result of dehydration, is associated with seizure activity, particularly when serum sodium levels exceed 160 mEq/L. Hyponatremia with levels less than 120 mEq/L is often complicated by seizures. The rate at which sodium rises or falls, and not the actual level, is the best determination of the risk for neurologic sequela. Sodium correction should be performed cautiously.

Box 1
Differential diagnosis of conditions with seizure like symptoms

Breath-holding spells
Episodic dyscontrol syndrome/rage attacks
Fugue states
Hyperventilation
Hypoglycemia
Migraine narcolepsy/cataplexy
Movement disorders
Night terrors
Nonepileptic seizures
Panic attacks
Paroxysmal vertigo
Psychogenic seizures
Syncope
Transient global amnesia
Transient ischemic attack/stroke

From Tarabar AF, Ulrich AS, D'Onofrio G. Seizures. In: Adams JG, editor. Emergency medicine. Philadelphia: Elsevier; 2008. p. 1051–62; with permission.

Hypercalcemia reduces neuronal excitability and is rarely a cause of seizures. However, hypocalcemia, at levels less than 7 mEq/L, is associated with seizure activity. Low calcium levels are found frequently in conjunction with hypomagnesemia, which is also epileptogenic.

Electrocardiogram

An electrocardiogram (ECG) should be obtained in every patient with the first onset of seizures, or with the suspicion of cardiac cause of decreased CNS perfusion. In addition to ischemia, the most important disorders that have to be excluded are related to conduction abnormalities and resultant dysrhythmias, including complete heart block. Widening of QRS complex can occur after an overdose from certain medications, particularly cyclic antidepressants. More specific ECG changes, terminal 40 ms R wave in lead aVR can also assist in identifying cyclic antidepressant toxicity. Prolonged QTc interval can be found in numerous overdoses. Tachyarrhythmias are often seen in the setting of cocaine and methylxanthine toxicity.

Neuroimaging

Although no definitive study demonstrates that it is essential, the standard of care seems to be to obtain a CT scan of the brain in every patient with first onset of seizures. CT scan of the brain should also be obtained in any patient with persistent change of mental status, focal neurologic deficit, or suspicion of organic intracerebral lesion.[26] Early CT scan is essential for identifying surgically correctable causes. If there is a concern for trauma, CT scan should also be used to rule-out cervical spine and intracranial injury.

Electroencephalography

Electroencephalography (EEG) records brain electrical activity and is used for definitive diagnosis of epilepsy and related conditions. The need for EEG in the emergent setting is limited to patients whose seizures are uncontrollable despite aggressive treatment or whose seizure activity is more difficult to diagnose.

Intubated patients, who are paralyzed or have had induction of phenobarbital coma and general anesthesia, should be continuously monitored with EEG to exclude seizure activity, because in these situations they may not able to manifest obvious seizure activity due to neuromuscular paralysis.

Treatment

Airway

One of the primary goals during the evaluation and treatment of seizures is to preserve patent airway and oxygenation, as well as to prevent aspiration in patients who are postictal. This goal can be achieved with simple maneuvers involving administration of supplemental oxygen, and perhaps providing a jaw trust/chin lift, cautiously inserting an oropharyngeal airway, or repositioning the head. Despite the dramatic presentation, including cyanosis, few patients who are actively seizing will require endotracheal intubation.

Pharmacologic treatment

Benzodiazepines should be administered immediately as they have been shown to control the majority of seizures regardless of cause, through increase of γ-aminobutyric acid (GABA) activity. Studies have shown that lorazepam is more effective than diazepam for the initial control of seizures, although both agents are acceptable.[22,24] When intravenous access is difficult, intramuscular or rectal administration of valium or lorazepam are alternative treatment options. Continued seizure activity should be treated with a second dose of benzodiazepines, along with the addition of a second agent. Phenytoin is the second drug of choice, but requires patient monitoring when used intravenously. Early initiation of phenytoin helps achieve a therapeutic level in a timely manner, but delivery is rate limited (no faster than 50 mg/min) to avoid hypotension.

Drug- or Toxin-Induced Seizures

GABA is the chief inhibitory transmitter in the brain. Every drug that can decrease in GABA activity in the CNS can cause seizure. Treatment is generally directed at providing enough GABA stimulus with benzodiazepines. However, certain drugs are associated with particular types of toxicities and they may require specific treatment or antidote (**Table 1**).

Removal of the toxin or drug and secondary decontamination is the hallmark of the treatment of drug- or toxin-induced seizures. Decontamination in the ED typically involves administration of charcoal; however, the clinician should be cautious with those patients who are postictal or who may develop recurrent seizures, as aspiration and need for intubation must be considered.

Cocaine

The most commonly abused substance that causes seizures is cocaine. Benzodiazepines will help to control seizures, agitation, and hyperthermia. It is important to maintain adequate fluid resuscitation to protect against renal damage from potential rhabdomyolysis. If the patient is hyperthermic, cooling measures involving intravenous fluids, ice packs, and even ice baths in the case of refractory hyperthermia should be applied to protect against multi-organ failure and death.

Table 1
Most common drugs associated with seizures

Medication/Drug	Comment
Camphor	Brief, tonic-clonic seizures, usually self limited
Cocaine/amphetamines	Tachycardia, hypertension
Phencyclidine	Rotary nystagmus
Cyclic antidepressants	Can be excluded with ECG Severe toxicity can cause cardiac dysrhythmias Treatment with bicarbonate will control ECG changes, but not the seizures Benzodiazepines are drug of choice
Isoniazid (INH)	Treatment B6
Lindane	Usually ingestion of topical preparation
MDMA (Ecstasy)	Usually associated with hyponatremia Morning after "rave" party Fluid restriction is usually sufficient therapy
Strychnine	Painful muscle spasm.
Theophylline	Wide pulse pressure Tachycardia Hypokalemia Hyperglycemia

From Tarabar AF, Ulrich AS, D'Onofrio G. Seizures. In: Adams JG, editor. Emergency medicine. Philadelphia: Elsevier; 2008. p. 1051–62; with permission.

Alcohol

Alcohol-related seizures (ARS) are defined as adult-onset seizures, generally after the age of 25 years, which occur in the setting of chronic alcohol dependence. ARS are often caused by alcohol withdrawal. However, risk factors such as preexisting epilepsy, structural brain lesions, the use of illicit drugs, or metabolic disorders may also contribute to seizures in patients who drink heavily. ARS are typically brief, have generalized tonic-clonic activity, and occur 6 to 48 hours following the last drink. Sixty percent of patients have multiple seizures without treatment, and the interval between the first and the last seizure is typically less than 6 hours.

The diagnosis of ARS is made only after exclusion of other potential causes. New-onset seizures in an alcohol-dependent patient should prompt a thorough evaluation similar to that described for any person presenting with a new-onset seizure. CT scan of the brain should be performed with a new onset seizure, partial seizure, status epilepticus, and a prolonged postictal state, or if there is evidence of any head trauma. A finger stick for serum glucose determination is important.

In the majority of patients, lorazepam has been shown to prevent subsequent seizures after the first presenting seizure. In a randomized control trial of patients presenting with an ARS, only 3% had a subsequent seizure during a 6-hour observation period, compared with 24% in the placebo group ($P<.001$).[27] Following an observation period of 4 to 6 hours, in the absence of further seizure activity, patients may be safely discharged. These patients ideally should be offered alcohol detoxification programs.

Opioids

Although opioids are not generally associated with the seizures, it is worth mentioning several exceptions including meperidine, propoxyphene, and tramadol. Patients may present with an opioid toxidrome and seizure activity. Of note, administration of naloxone can actually precipitate and worsen meperidine-associated seizures.

3,4-Methylenedioxymethamphetamine

3,4-Methylenedioxymethamphetamine (MDMA; Ecstasy) is an amphetamine-related drug that is associated with seizure activity. Patients who are consuming MDMA typically present with brief tonic-clonic seizure secondary to hyponatremia. MDMA can also lead to transient syndrome of inappropriate antidiuretic hormone secretion (SIADH). Most of these patients can be treated with fluid restriction and observation.

Other medications

Other drugs that can be associated with seizure activity are worthy of mention. Cyclic antidepressants are notorious for their propensity to cause seizures due to GABA inhibition. Seizures are a manifestation of severe toxicity, so it is important to rapidly assess and protect the airway, complete gastrointestinal decontamination, and administer intravenous benzodiazepines, as tricyclic antidepressant overdoses can lead to rapid deterioration. Bicarbonate is the primary treatment of cyclic antidepressant overdose and is effective in treating cardiac conduction abnormalities, but has no role in controlling seizure activity. Isoniazid (INH) overdose should be suspected in the setting of status epilepticus that is not responsive to conventional treatment. Treatment is vitamin B6 (pyridoxine). Methylxanthines (caffeine, theophylline) in overdose are notorious for causing seizures through the antagonism of adenosine. Usually the seizures are short acting and can be controlled with benzodiazepines. However, in severely toxic patients hemodialysis is necessary.

Special Considerations

Status epilepticus

Status epilepticus is defined as seizures that persist more than 20 minutes, as this is the duration seen in animal studies to cause damage to the CNS neurons. However, given the need for earlier intervention to prevent permanent neuronal injury, the operational definition of status epilepticus is continuous seizures lasting at least 5 minutes or 2 or more discrete seizures between which there is incomplete recovery of consciousness.[28]

Status epilepticus has a mortality rate of 20% in adults.[28] Early aggressive administration of intravenous anticonvulsants is the keystone of successful treatment of status epilepticus. **Table 2** summarizes the medications that can be used in the treatment of status epilepticus. Neurology should be consulted early for assistance in management of patients with persistent seizures in possible status epilepticus.

Pregnancy

Pregnancy can precipitate seizure episodes in patients with underlying seizure disorders, and prolonged seizure activity results in increased morbidity and mortality to the fetus. Some women may require a higher dose of antiepileptic medications to maintain therapeutic levels while pregnant.

Eclampsia should be considered in all women of child-bearing age who present with seizures. Eclamptic seizures can occur from the 20th week of gestation up to 6 weeks postpartum, and during this period every new-onset seizure should be initially treated as eclampsia until proven otherwise. The ‘‘classic symptoms’’ of eclampsia are hypertension, proteinuria, and edema, but up to 30% of eclamptic women do not present classically. Pregnant women who have hypertension, proteinuria, headache, visual disturbances, abdominal pain with nausea, or edema should be presumed to have eclampsia until proven otherwise. The treatment of eclampsia is intravenous magnesium, blood pressure control and concomitant delivery of the fetus.

Table 2
Summary of medications used for the treatment of status epilepticus

Medication	Dose (Load)	Dose (Maintenance)	Comments
Diazepam	10 mg over 2 min	Repeat every 5–10 min	Respiratory depression, hypotension
Lorazepam	2–8 mg IV	Repeat once in 10–15 min if seizure persists	Respiratory depression, hypotension
Midazolam	0.1–0.2 mg/kg or 2.25–15 mg IV		Respiratory depression, hypotension
Phenytoin	15–20 mg/kg @ max. rate 50 mg/min	100 mg IV/PO every 6–8 h	Hypotension, ataxia
Fosphenytoin	15–20 mg/kg @ max. rate 150 mg/min		Faster loading time, needs to be metabolized to be effective
Pentobarbital	5–20 mg/kg IV @ 25 mg/min 5 mg/kg is effective for induction anesthesia for most	2.5 mg/kg/h	Severe respiratory depression Faster effect than phenobarbital
Phenobarbital	15–20 mg/kg @ max. 50 mg/min	120–240 mg every 20 min	Severe respiratory depression, hypotension
Propofol	1–2 mg/kg IV	2–4 mg/kg/h	Severe respiratory depression, acidosis (children)
Valproic acid	20 mg/kg at 20 mg/min	Repeat if needed	Only if everything else fails; may be beneficial for the patients who are already on valproic acid but subtherapeutic

IV, intravenous; PO, by mouth
From Tarabar AF, Ulrich AS, D'Onofrio G. Seizures. In: Adams JG, editor. Emergency medicine. Philadelphia: Elsevier; 2008. p. 1051–62; with permission.

Human immunodeficiency virus

People with HIV infection may have a CNS mass or infection as the cause of seizure, which can be the first manifestation of AIDS. HIV-positive patients who present with new-onset seizures, or those that do not have the diagnosis of epilepsy, require CT scan and, if negative, a lumbar puncture. Patients who are HIV positive, as well as those patients who are immunosuppressed, are at risk for numerous infectious entities that may induce seizure activity (**Table 3**).

Central nervous system infection

Patients at high risk of meningitis or encephalitis should be treated with appropriate intravenous antibiotics even before lumbar puncture. Timely administration of

Table 3
Causes of seizures in HIV population

Focal CNS lesions
Cerebral toxoplasmosis
Primary CNS lymphoma
Progressive multifocal leukoencephalopathy
Focal viral encephalitis
Cytomegalovirus
Varicella zoster virus
Herpes simplex virus
Bacterial abscess
Cryptococcoma
Tuberculosis abscess
Mass lesion
Toxoplasmosis
Lymphoma
Meningitis/encephalitis
Cryptococcal
Bacterial/aseptic
Herpes zoster
Cytomegalovirus
HIV encephalopathy/AIDS dementia complex
Progressive multifocal leukoencephalopathy
CNS tuberculosis
Neurosyphilis

From Tarabar AF, Ulrich AS, D'Onofrio G. Seizures. In: Adams JG, editor. Emergency medicine. Philadelphia: Elsevier; 2008. p. 1051–62; with permission.

antibiotics and antiviral medication in patients with CNS infections can improve survival and reduce mortality.

Neurocysticercosis (NCC) is the most common parasitic CNS infection in the world, and has been increasing in the United States since 1980.[29] In endemic areas, namely Latin America, Asia, and Africa, NCC is a frequent cause of late-onset epilepsy. Most of these patients will respond to treatment with phenytoin or carbamazepine. Albendazol is the mainstay antiparasitic treatment for NCC. Patients diagnosed with NCC may require treatment with steroids to control cerebral inflammation and the appropriate medication regimen for meningitis, cysticercal encephalitis, and angiitis.

Intracranial pathology

Patients with intracranial hemorrhage who are anticoagulated or have elevated international normalized ratio may require rapid administration of fresh frozen plasma and vitamin K to prevent further bleeding. It is essential not to delay treatment pending laboratory confirmation of anticoagulated status. Patients with brain tumors, evidence of increased intracranial pressure, or hydrocephalus need immediate attention of neurosurgical service. Administration of steroids may be prudent to reduce the mass effect of intracranial tumors.

Disposition

Every patient with persistent seizures, change of mental status, or significant underlying medical condition should be admitted to the hospital. Patients in status epilepticus should be admitted to the intensive care setting. Patients with chronic seizure disorder can be discharged if they return to their baseline neurologic status. Patients who present with first-onset seizures should have follow-up arranged with a neurologist for further workup and eventual treatment if warranted.

Patients who are discharged should be advised to avoid precipitating factors and informed of the importance of adherence to anticonvulsant regimens. Patients should be given written instructions to avoid driving motor vehicles and not to engage in any activity whereby unexpected seizures with transient loss of consciousness can lead to injury or death. It is important for emergency medicine physicians to be familiar with state and local rules regarding mandatory reporting of seizure patients, as some states require reporting of these patients to motor vehicle departments.

ACUTE FOCAL NEUROLOGIC DEFICITS

An acute neurologic deficit is one that develops in 24 hours or less, with many deficits developing much more rapidly over a period of seconds to minutes. Focal deficits occur when there is an injury or dysfunction in a localized area of the nervous system. Acute focal neurologic deficits may consist of multiple signs and symptoms, including altered level of consciousness or other mental status changes, visual disturbances, cranial nerve abnormalities, weakness or paralysis, sensory abnormalities, speech difficulties, ataxia, neglect, and vertigo.

Patients with acute focal neurologic deficits often present emergently for evaluation, and can pose challenges to the emergency physician in the diagnosis and management of their complaints. The most common cause of acute focal neurologic deficits affecting the head and neck is stroke or transient ischemic attack (TIA). Proper management of patients with stroke requires a rapid and accurate diagnosis by the emergency physician. However, there are many conditions other than acute stroke that cause focal neurologic deficits, some of which are dangerous, and require rapid diagnosis and intervention as well. This section focuses on the high-risk aspects of the diagnosis and management of acute stroke, as well as the differential diagnosis of stroke and other critical conditions to consider in patients presenting with acute focal neurologic deficits.

Stroke

Stroke is defined as the sudden loss of neurologic function caused by an interruption in the blood supply to the brain. Stroke is the third leading cause of death and the leading cause of long-term disability in the United States. Stroke is a common disease affecting approximately 795,000 people in the United States annually.[17] The rapid diagnosis and management of patients who present to the ED with stroke is essential to improving long-term outcomes and lessening the impact of the disease.

The cause of acute stroke is either ischemia or hemorrhage. Diagnosis and management differs, depending on the etiology of the stroke and the stroke type. This section addresses the high-risk features of caring for patients with acute stroke in the ED.

Ischemic stroke

Ischemic stroke is defined as a permanent cerebral injury secondary to prolonged disruption of cerebral flow. Ischemic stroke has 3 different causes: large artery atherosclerosis, embolic events, or small-vessel disease.[30] Approximately 87% of all strokes

are ischemic in nature.[17] In patients presenting to the ED with acute-onset neurologic deficits, difficult management decisions occur in both the approach to diagnosis and the treatment of patients with ischemic stroke.

Patients with an anterior circulation strokes often have acute onset of motor and sensory deficits in the contralateral arm, leg, and face, accompanied by aphasia or neglect. As such, the diagnosis in this subset of patients is usually straightforward. However, diagnostic efforts can be complicated by stroke mimics. A variety of conditions mimic ischemic stroke and can be the cause of a patient's acute focal neurologic deficits.[31–37] It is important that the clinician consider and exclude the various stroke mimics in patients presenting with acute focal neurologic deficits. The different causes that can mimic an acute stroke will be discussed in further detail later in this section.

Posterior circulation strokes occur in about 20% of ischemic events, and many remain undiagnosed or incorrectly diagnosed. Dizziness, vertigo, headache, vomiting, double vision, ataxia, numbness, and weakness involving structures on both sides of the body are frequent symptoms in patients with posterior circulation strokes, and should evoke a high degree of suspicion from the physician evaluating the patient. Any patient with suspected posterior circulation stroke should undergo neuroimaging, preferably with MRI, as CT often provides limited visualization of the posterior fossa structures due to artifacts related to the skull.[38] However, it is important to remember that CT is useful in ruling out intracranial hemorrhage in the brainstem and cerebellum, and should be used when clinically indicated.

The treatment of acute ischemic stroke is another potential area of high risk for the emergency physician. The only current Food and Drug Administration approved therapy for the treatment of acute ischemic stroke is intravenous administration of recombinant tissue plasminogen activator (r-tPA) within 3 hours of symptom onset.[39] A recent study has shown that the drug may be safe and effective if given up to 4.5 hours from symptom onset if more stringent patient selection criteria are used.[40] Although the treatment time has been expanded, earlier treatment continues to be associated with more favorable outcomes.[41] However, treatment with intravenous rt-PA can be complicated by symptomatic intracranial hemorrhage, which may be fatal. Given these factors, it is important that all patients presenting with acute ischemic stroke have neuroimaging to exclude intracranial hemorrhage and have an accurately documented time of symptom onset. Urgent neurologic consultation is also recommended for assistance in making the decision on which patients should receive treatment with intravenous r-tPA. Although written consent is not necessary before the administration of intravenous rt-PA for the treatment of ischemic stroke, a full discussion of the potential risks and benefits of treatment with the patient and family members is recommended.[42]

Transient ischemic attack

Although conventional clinical definitions defined TIA as a focal neurologic deficit lasting less than 24 hours, this definition has recently been updated.[43] The new definition for TIA is a "brief episode of neurological 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."[44] TIAs are an important predictor of stroke, with reports of 90-day risk of stroke in patients who have suffered a TIA as high as 10.5% with 50% of those occurring in the first 2 days.[45] As such, all patients with transient focal neurologic deficits consistent with a TIA should have neurology consultation for further evaluation and ischemic stroke risk reduction. In addition, although there are no established guidelines as to when to admit patients with TIA, it is prudent for the emergency physician to have a low threshold for admitting patients with TIA given the substantial risk of stroke within the first 48 hours.

Hemorrhagic stroke

Hemorrhagic strokes account for approximately 13% of all strokes, with 10% caused by intracerebral hemorrhage (ICH), and the other 3% caused by SAH.[17] The high-risk features of hemorrhagic stroke relate to the diagnosis and early clinical management of patients with hemorrhagic stroke.

ICH is a medical emergency requiring rapid recognition and diagnosis. Frequent early ongoing bleeding often causes progressive clinical deterioration, and patients who suffer a stroke due to ICH have a high mortality rate, approximately 44% to 48% at 1 month.[46] Patients with ICH classically present with sudden neurologic deficits characterized by smooth symptomatic progression over minutes to hours. Patients may have focal neurologic deficits or decreased level of consciousness, depending on the location and size of the hematoma. In addition, patients may only present with nonspecific signs and symptoms, such as headache, vomiting, increased blood pressure, or meningismus, thereby making the diagnosis more elusive. CT and MRI are both first-choice initial imaging options, and either is the essential diagnostic test in the evaluation of patients with potential ICH.[47] Failure to consider ICH and obtain diagnostic neuroimaging in patients with the aforementioned neurologic findings is a high-risk area of emergency medicine. Once diagnosis is reached, urgent consultation with a neurologist or neurosurgeon is essential. Patients with ICH due to iatrogenic anticoagulation must be rapidly reversed.[47] Due to frequent ongoing bleeding, the risk of neurologic and cardiovascular instability in patients with ICH is highest during the first 24 hours, and patients should be admitted to an intensive care unit for frequent monitoring.[48]

The most common cause of SAH is trauma, but spontaneous SAH can by caused by a ruptured aneurysm or arteriovenous malformation. SAH is discussed in detail in the headache section of this article. Patients with SAH classically present with a severe, thunderclap headache with or without focal neurologic deficits. Approximately 20% of patients describe a sentinel headache that occurs within 2 to 8 weeks of overt SAH. SAH is a difficult diagnosis, and is frequently missed by physicians. Patients who present with acute onset of severe headache should evoke a high level of suspicion for SAH. Noncontrast cranial CT is the initial test of choice for making the diagnosis, but its sensitivity declines with the time from onset of symptoms. Lumbar puncture must be performed to look for RBCs and xanthochromia in all patients with a high suspicion of SAH and a negative cranial CT. Patients found to have a SAH should undergo further vascular imaging to look for aneurysms, as well as emergent evaluation by a neurologist or neurosurgeon. Potential for acute neurologic deterioration in patients with SAH is significant. All patients should be emergently evaluated and stabilized, and ultimately monitored in a neurosurgical unit, as failure to detect herniation or worsening neurologic status is another high-risk feature of the management of patients with SAH.[9]

Cerebral venous thrombosis

Cerebral venous thrombosis (CVT) is a rare cerebrovascular disorder and an uncommon cause of stroke. Unlike arterial stroke, CVT most often affects young adults and children. CVT is a challenging diagnosis, as patients' presentations are highly variable and include headache, focal neurologic deficits, seizures, altered mental status, altered level of consciousness, and papilledema.[18] Risk factors for developing a cerebral venous sinus thrombosis include hypercoagulable conditions, pregnancy/puerperium, oral contraceptives, trauma, and infections located near the cerebral sinuses. Routine neuroimaging such as CT or MRI may produce subtle findings that may be missed, but MR venography confirms the diagnosis.[43] It is important

to consider the diagnosis in young and middle-aged patients with unusual headache or neurologic deficits in the absence of vascular risk factors, in patients with intracranial hypertension, and in patients with CT evidence of hemorrhagic infarcts, especially if the infarcts are in multiple territories.[22] Once the diagnosis is made, treatment with unfractionated heparin and low molecular weight heparin may be recommended and should be guided by neurology consultation.[43]

Stroke Mimics

A stroke mimic is a nonvascular condition that can simulate the signs and symptoms of a stroke. There are a variety of conditions that can mimic TIA or stroke (**Box 2**). Not only can these conditions complicate diagnostic efforts in the rapid assessment of patients with potential acute stroke, but many stroke mimics are critical diagnoses unto themselves and must be recognized early for proper intervention.

Various studies evaluating the frequency of misdiagnosis of patients presenting with signs and symptoms of acute stroke have found the incidence of stroke mimics to be around 3% to 19%.[32–38] A study by Kothari and colleagues[34] reviewed 446 patients evaluated in the ED with a hospital admission or discharge diagnosis of acute stroke, to determine the ability of emergency physicians to accurately identify patients with stroke. In this study, all of the patients with ICH or SAH were correctly diagnosed by the emergency physician, and 5% (19 of 365) of the patients diagnosed with ischemic stroke or TIA by the emergency physician were discharged with a different final diagnosis. An observational study by Scott and colleagues[36] evaluated 151 consecutive patients with an initial diagnosis of acute ischemic stroke treated with intravenous tPA. Six of 151 (4%) patients had a final diagnosis other that acute ischemic stroke or TIA. These stroke mimics suffered no intracranial hemorrhage, had less disability at discharge, and were younger than patients with acute ischemic stroke or TIA. Another study by Winkler and colleagues[37] evaluated 250 consecutive patients treated with intravenous tPA, and found that 7 (2.8%) had a final diagnosis other than stroke or TIA. All of these patients had a favorable outcome, with none suffering from intracranial hemorrhage.

Given the relative frequency of stroke mimics, it is important that all patients who present with acute focal neurologic deficits should have a thorough evaluation. The first step should be to check blood glucose on arrival. Vital signs should be monitored frequently. Basic laboratory work should include a complete blood cell count, electrolyte levels, coagulation studies, urinalysis, and toxicology screens. Brain imaging should be ordered as indicated. In patients with difficult presentations, neurology consultation is indicated.

OCULAR EMERGENCIES

Ophthalmologic complaints are one of the common presentations to EDs, with estimates as high as 3% of all emergency visits.[49] In the vast majority of these cases, emergency physicians can safely manage these patients. However, prompt and appropriate recognition, and initiation of treatment of entities that are at high risk for vision loss, is essential to mitigate the possibility of serious and permanent visual deficits. The challenge facing emergency physicians is to quickly identify these high-risk injuries and ensure timely ophthalmologic consultation. In most cases, visual or eye complaints should be seen in follow-up by an ophthalmologist, the timing of which can be determined after discussion with the consulting service. This section reviews those injuries that, if missed or have a significant delay in diagnosis, have a high likelihood of resulting in permanent ophthalmologic sequelae.

Box 2
Conditions that can mimic acute ischemic stroke

- Seizure
  - Postictal paralysis
  - Nonconvulsive seizures
- Space-occupying lesions
  - CNS tumor (primary, metastatic)
  - Subdural hematoma
  - Epidural hematoma
  - Cerebral abscess
- Infectious
  - Systemic infection (ie, sepsis)
  - Meningitis
  - Encephalitis
- Toxic-metabolic disturbances
  - Hypoglycemia
  - Hyperglycemia with hyperosmotic coma or DKA
  - Hyponatremia
  - Hepatic encephalopathy
  - Alcohol withdrawal
  - Wernicke encephalopathy
  - Drug toxicity
- Vestibular dysfunction
  - Labyrinthitis
  - Ménière disease
  - Benign positional vertigo
- Demyelinating disease (ie, multiple sclerosis)
- Trauma
- Complicated migraine
- Peripheral nerve neuropathy
- Psychiatric disorders
  - Acute confusional state
  - Somatoform disorders
  - Malingering
  - Dementia
- Hypertensive encephalopathy

Initial Evaluation

A simple but essential part of every ophthalmologic examination is a visual acuity test. In conjunction with vision, pupillary activity, extraocular movements, and fundoscopy should be evaluated. Both the affected and nonaffected eyes should be tested

individually, then together to evaluate binocular vision. Pinhole testing corrects most refractive errors, and when vision improves there is likely a problem with the lens. However, when pinhole testing fails to improve vision, the pathology is more likely to be located in the retina or CNS. Fluorescein examination is another critical component of a complete ophthalmologic examination. Fluorescein examination should be performed in all evaluations of ocular trauma, potential foreign bodies, or suspected infections. A variation of the standard fluorescein examination is the Seidel test, screening for globe rupture. Another important component of the ophthalmologic examination is the slit lamp, which allows for a magnified, stereoscopic evaluation of the eye and surrounding structures. The slit lamp is especially useful in the examination of the anterior chamber.[50] Tonometry is essential to diagnose acute angle glaucoma and to establish a baseline intraocular pressure following blunt eye trauma.[51] Lid eversion is useful in the inspection of the tarsal conjunctiva and fornices, as well as being essential for localizing foreign bodies. Ocular ultrasonography is rapidly gaining popularity in the ED evaluation of ophthalmologic complaints. Ocular ultrasonography can be especially useful in the trauma patient, in whom significant periorbital swelling can limit direct visualization of the eye.[52]

High-Risk Diagnoses

When there is suspicion for high-risk ophthalmologic conditions or injuries, emergency ophthalmologic consultation is warranted (**Box 3**). However, there are numerous additional conditions for which ophthalmologic consultation is reasonable and appropriate. In these circumstances, discussion with an ophthalmologist can help determine the subsequent need and urgency of an ophthalmologist's evaluation.

Trauma

Patients with evidence of significant globe trauma, either blunt or penetrating, should be evaluated emergently by ophthalmology. Retrobulbar hemorrhage may be vision threatening, and often requires immediate decompression to ensure adequate blood supply to the optic nerve. In this situation, a lateral canthotomy should be performed as soon as possible. Lacerations involving the nasolacrimal system, the lid margin, or

Box 3
Conditions that warrant emergent ophthalmologic consultation

- Trauma
  - Ruptured globe
  - Lid laceration through margin, nasolacrimal system or canaliculus
- Endophthalmitis
- Angle closure glaucoma
- Acute vision loss
  - Central artery occlusion
  - Optic neuritis
  - Retinal detachment

Data from Magauran B. Conditions requiring emergency ophthalmologic consultation. In: Kahn J, Magauran B, Mattu A, editors. Ophthalmologic emergencies. Emerg Med Clin North Am 2008;(26):233–8.

tarsal plate require specialized repair, and should be performed by plastic surgery or ophthalmology, depending on institutional preference.[53]

Endophthalmitis

Endophthalmitis is the inflammation of the aqueous or vitreous humors. Endophthalmitis can occur as the result of infection, trauma, or postsurgical complications. Suspicion mandates emergent ophthalmologic consultation for treatment options, including surgical intervention.

Acute-angle closure glaucoma

Patients with acute-angle closure glaucoma typically present with general eye pain, often associated with nausea, vomiting, headache, or abdominal pain. Elevated intraocular pressure (IOP) greater than 50 mm Hg may induce optic nerve atrophy if left untreated.[54] Treatment can and should be initiated by emergency physicians, but an ophthalmology consultation is important for monitoring of IOP. Patients refractory to treatment may require urgent surgical intervention.

Acute vision loss

Whether monocular or binocular, acute vision loss should be evaluated by ophthalmology as soon as possible. There are multiple causes, some of which are not directly related to the eye. Pain can help differentiate and direct appropriate treatment. Central artery occlusion results in sudden, painless vision loss. This condition is primarily the result of an embolic event, and immediate ophthalmologic consultation is essential, as irreversible vision loss can occur within 4 hours.[53]

Optic neuritis

In the setting of optic neuritis, patients present with painful eye movements, usually associated with monocular vision loss, visual field deficits, and change in color perception. Vision loss may progress rapidly over hours. Treatment with steroids should be initiated quickly, after consultation with ophthalmology or neurology.

Retinal detachment

Acute retinal detachment typically presents as new onset of floaters, squiggly lines, or ''cobwebs'' that are associated with visual field deficits. Examination with a standard ophthalmoscope is generally insufficient, as the detachment is likely to be located at the retinal periphery.[54] Eventual visual outcome is associated with the presence of macular involvement and time until repair. Therefore, early ophthalmology consultation is paramount.

Definitive Treatment

Eye irrigation is the immediate and primary treatment for any exposure. In the setting of chemical burns, copious irrigation has shown to have the greatest effect on visual prognosis.[55] Regardless of alkali versus acid exposure, the treatment is the same, and immediately on presentation, irrigation should be initiated. Physical examination should be limited to, at most, a rapid visual acuity and determination of pH. Irrigation should continue for up to 30 minutes after return to normal pH. On discontinuation, the pH should be rechecked 5 to 15 minutes later, to ensure that further chemical has not remained; this is especially important in the setting of alkali burns, where liquefactive necrosis leads to absorption into the anterior chamber.[56]

Removal of ocular foreign bodies is a frequent, but critical task for emergency medicine. It is essential to differentiate those that are superficial, on the surface of the eye, from those that are imbedded within the globe. In the case of the latter, the priority of the emergency physician is to prevent further damage until removal can be performed

by an Ophthalmologist. Care should be taken to protect the eye with a shield, elevate the head of the bed, and minimize patient movement. Removal of uncomplicated superficial foreign bodies can be performed on the cooperative patient with the use of local anesthetic and a slit lamp. Removal can often be successful with gentle irrigation. Other techniques include a cotton-tipped applicator or 25-gauge needle held tangentially to the globe.

Paracentesis of the anterior chamber is a technique rarely performed by emergency physicians, but one that can be vision saving in the setting of retinal artery occlusion. In this condition, painless, sudden, monocular vision loss is accompanied by a pale optic disc or cherry-red spot on fundoscopy. Vision loss can be permanent without proper treatment within 48 hours of onset of symptoms.[57] The primary goal of treatment is to increase retinal blood flow or to dislodge the occluding clot. Other therapeutic interventions include digital orbital massage and intravenous acetazolamide, or topical β-blockers to decrease IOP.

Lateral canthotomy is another rarely used but important vision-sparing procedure. Lateral canthotomy is used to emergently relieve retroorbital pressure following blunt trauma, because in the setting of significant retrobulbar hemorrhage the optic nerve can become ischemic in as little as 90 minutes.[58]

In summary, ophthalmologic complaints result in relatively frequent visits to EDs. Familiarity with those entities that are high risk for permanent visual damage is essential for all emergency physicians. Rapid diagnosis, treatment, and ophthalmologic consultation may help increase the chance to avoid serious, permanent vision deficits.

THROAT AND NECK PAIN

The anatomy of the neck is highly complex, with many important structures. Patients' complaints of throat or neck pain can result in diagnoses ranging from benign, self-resolving problems to much more severe, life-threatening conditions with high morbidity and mortality if not diagnosed and treated correctly. This section focuses on some of the high-risk conditions for emergency physicians to consider when evaluating and treating patients presenting with complaints of throat or neck pain.

Throat Pain

Sore throat or throat pain is one of the most common chief complaints of patients treated in an outpatient setting.[59] The differential diagnosis for patients presenting with throat pain is extensive, ranging from benign viral pharyngitis to life-threatening emergent conditions with acute airway obstruction. The majority of patients presenting with sore throat have infectious pharyngitis, and 1.4% of all patients presenting to United States EDs ultimately receive this diagnosis.[49] However, it is the responsibility of the emergency physician to quickly recognize the less common but far more serious causes of throat pain that may cause airway obstruction, as precise and rapid intervention may be life saving. This section discusses the diagnosis and management of high-risk causes of throat pain, including deep neck infections and epiglottitis.

Deep neck infections

Deep neck infections, such as cellulitis or abscesses, are infections that occur in the potential spaces of the facial planes of the neck.[60] These infections are difficult to diagnose due to the complex anatomy of the neck, with the multiple fascial planes forming a framework of at least 11 deep neck spaces. The fascial planes create important barriers and limitations to the spread of infection, but can also serve to direct infectious spread once their natural resistance is overcome.[61] A detailed discussion of the anatomic planes and spaces in the neck is beyond the scope of this review,

but a listing of the fascial spaces where deep neck infections can occur is listed in **Box 4**.[62]

This section reviews the signs and symptoms, treatment, and complications of deep neck infections. These infections are high-risk complaints, due to the rapid onset and potential fatal complications if not properly diagnosed and treated.

Signs and symptoms There are numerous signs and symptoms that may be present in patients presenting with deep neck infections. Presenting symptoms may include fever and chills, sore throat, neck pain, odynophagia, dysphagia, neck stiffness, voice changes, tongue base pain, dyspnea, otalgia, and sialorrhea.[62–65] In a retrospective review of 169 cases by Wang and colleagues,[65] the most common presenting symptoms were sore throat (72%) and odynophagia (63%). When peritonsillar abscess was excluded from their analysis, the most common presenting symptoms were neck swelling (70%) and neck pain (63%). Physical signs of patients with deep neck infections include neck swelling, trismus, elevated temperature, tachycardia, drooling, elevation of the floor of the mouth, and bulging of the pharyngeal wall.[62–65] Pain out of proportion to physical findings may suggest the presence of a deep neck infection.

Box 4
Cervical fascial spaces and their synonyms

- Spaces formed by splitting anterior layer
  - Space of parotid gland
  - Space of submaxillary gland
  - Space of body of mandible
  - Masticator space—masseteric, mandibulopterygoid, temporal pouch
- Space deep to the anterior layer of the deep cervical fascia
  - Retropharyngeal space—retrovisceral space, retroesophageal visceral compartment (posterior)
  - Lateral pharyngeal space—parapharyngeal, peripharyngeal, pharyngomaxillary, pterygopharyngeal, pterygomandibular, pharyngomasticatory
    - Anterior—prestyloid
    - Posterior—poststyloid
  - Submandibular space
    - Sublingual—floor of mouth
    - Sumylohyoid—submaxillary, submandibular
- Other spaces
  - Danger space
  - Prevertebral space—paravertebral space
  - Carotid sheath
  - Pretracheal space—visceral compartment (anterior)
  - Peritonsillar space

From Marra S, Hotaling AJ. Deep neck infections. Am J Otolaryngol 1996;17(5):287–98; with permission.

Emergency physicians must be observant for advanced airway signs in patients presenting with possible deep neck infections. Signs such as voice changes, stridor, dyspnea, shortness of breath, and use of accessory muscles signify impending airway obstruction or respiratory arrest, and require immediate intervention.

Treatment The maintenance of a safe and secure airway is the most important therapeutic goal in the management of patients with deep neck infections,[66] as death from loss of an airway can rapidly occur in patients with advanced disease.[67] In many cases of patients with deep neck infection, airway observation in a closely monitored setting may be a reasonable management option, but there is no consensus on the optimal timing of when to observe versus when to intervene.[66] When in doubt, taking control of the airway is the most conservative method, as patients with deep neck infection may progress quickly and airway compromise can occur with little warning.[68]

Indications for immediate aggressive airway management include patients in respiratory distress or impending airway compromise as noted by physical examination or diagnostic imaging.[61] Conventional endotracheal intubation is often difficult in patients with deep neck infections due to distorted airway anatomy, immobility of soft tissues, or trismus. In addition, direct laryngoscopy can be dangerous, as it may precipitate acute airway collapse or can cause rupture of the abscess with subsequent aspiration of pus.[61,66] General anesthesia is also dangerous in advanced cases as it may precipitate complete airway closure, thereby necessitating emergency tracheostomy.[67]

Tracheostomy under local anesthesia has long been considered the gold standard for airway management of patients with deep neck infections, but can be unpractical and risky in certain patients. Blind nasal intubation should never be performed in patients with deep neck infection as it carries risk of damage to the inflamed pharyngeal mucosa, with possible resulting bleeding, abscess perforation, or complete airway obstruction. Awake fiberoptic intubation using topical anesthesia is gaining popularity as the initial choice for airway management, and should be the first approach when it can be performed by an experienced operator. Tracheostomy under local anesthesia should be performed in situations where a fiberoptic bronchoscope is not available, the clinician is not skilled with awake fiberoptic intubation, or attempts at intubation have failed.[61,66,67] Cricothyrotomy or emergency tracheostomy may be performed for emergent airway control when sudden, complete loss of airway necessitates immediate surgical intervention.[61]

Once the airway is determined to be secure, broad systemic antibiotics should be given to patients with presumed or known deep space infection. If treatment is started early when the infection is at the stage of cellulitis, the condition may resolve by fibrosis without abscess formation. However, if the infection is not treated until pus has formed or if antibiotics fail, the abscess must be drained surgically.[69]

Cultures of deep neck abscesses are commonly polymicrobial, reflecting the oropharyngeal and odontogenic nature of these infections. The most frequently isolated aerobic microorganisms are *Streptococcus viridians*, *Klebsiella pneumoniae*, β-hemolytic streptococci, *Staphylococcus aureus*, and *Streptococcus pneumoniae*. The most frequent isolated anaerobic microorganisms include *Prevotella* spp, *Peptostreptococcus* spp, and *Bacteroides* spp.[60,61,63–65,70] *K pneumoniae* is the most common infection in diabetics.[60,61,70]

Early empirical antibiotic coverage should be started immediately. Penicillin with a β-lactamase inhibitor or a β-lactamase-resistant antibiotic combined with an antibiotic that is effective against anaerobes are good initial choices for empirical therapy. If a patient is at risk for methicillin-resistant *S aureus*, adding empirical vancomycin should be considered. The addition of gentamycin for effective gram-negative

coverage against *K pneumoniae* is highly recommended for diabetic patients,[61] but special attention should be paid to renal function in these patients.

Evaluation of deep neck infections may be done by ultrasound, plain film radiography, contrast-enhanced CT, or MRI.[61] Contrast-enhanced CT or MRI are useful in characterizing the nature of the deep neck infection and aiding in early recognition of complications. As CT is more available, less expensive, and less time consuming than MRI, contrast-enhanced CT is the standard of care in evaluating deep neck infections.[71]

For many years, open surgical drainage has been the mainstay in the treatment of deep neck infections. However, some centers propose attempting treatment with broad-spectrum intravenous antibiotics alone in certain patients with small abscess who are clinically stable and have no evidence of abscess in "danger spaces" (prevertebral, anterior visceral, and vascular visceral spaces) or in more than 2 places, or have evidence of descending infection.[71] For the emergency physician, it is prudent to consult a surgeon early for assistance in the management of all cases of suspected deep neck infection.

Complications Life-threatening complications of deep neck infection included upper airway obstruction, descending mediastinitis, pleural empyema, pericarditis, pneumonia, jugular vein thrombosis, sepsis, and carotid pseudoaneurysm or rupture.[65] Studies have shown that complicated deep neck infections are associated with patients presenting with neck swelling or respiratory difficulty as well as those found to have an extended space abscess with more than 2 involved spaces. Factors that were found to be less likely associated with a complicated abscess were male gender, presenting complaint of neck pain, and odontogenic causes of infection.[60,65,70]

Epiglottitis

Acute epiglottitis, or supraglottitis, is an inflammatory process of the epiglottis and the adjacent structures that can lead to life-threatening acute respiratory obstruction. In the past, epiglottitis occurred most frequently in children and was associated with *Haemophilus influenzae* type b (Hib) infection.[72] However, since the introduction of the Hib vaccine in the United States in 1985, the incidence of acute epiglottitis in children has decreased while the incidence in adults has remained stable or increased.[73,74]

Acute epiglottitis can be difficult to diagnose, and can lead to rapid and unpredictable airway obstruction. Due to the difficulty of its diagnosis and management, epiglottitis is listed as 1 of 8 high-risk diagnostic categories in a review of malpractice claims filed by emergency physicians in Massachusetts from 1975 to 1993.[4] The emergency physician must maintain a high index of suspicion to make the diagnosis, and then intervene quickly and appropriately. This section reviews the diagnosis and management of acute epiglottitis as well as the high-risk features to be aware of when treating a patient with epiglottitis.

Signs and symptoms Children typically present with fever, irritability, sore throat, and difficulty breathing, with rapidly progressive stridor and respiratory distress.[72,74] Adults generally present with milder disease, and their main complaints are usually sore throat or odynophagia.[74,75] The presence of a severe sore throat in an adult, especially when accompanied by anterior neck tenderness, should prompt the emergency physician to consider epiglottitis in the differential diagnosis.[76] Other signs and symptoms of acute epiglottitis include muffled voice, fever, pharyngitis, tenderness in the anterior neck, drooling, cervical adenopathy, and cough.[74,75] Pain out of proportion to physical findings may suggest epiglottitis.

Diagnosis Diagnosis of epiglottitis is made by history, clinical examination, radiography, and laryngoscopy. Lateral neck soft tissue radiographs may demonstrate obliteration of the vallecula, swelling of the aryepiglottic folds, edema of the prevertebral and retropharyngeal soft tissues, ballooning of the hypopharynx, or an enlarged, thumb-shaped epiglottis in patients with acute epiglottitis.[77] However, these soft tissue neck radiographs are not reliable in the diagnosis of acute epiglottitis due to low sensitivity and specificity. A retrospective chart review by Stankiewicz and colleagues[78] showed that of 30 adults diagnosed with acute epiglottitis, only 1 lateral neck radiograph was read as positive and of 48 children with documented epiglottitis, only 26 lateral neck radiographs were read as abnormal. A retrospective review of hospital records of cases of acute epiglottitis in Rhode Island showed that of 287 soft tissue neck radiographs performed, 247 had findings diagnostic or suggestive of acute epiglottitis, yielding a sensitivity of only 86%.[74]

Diagnosis of acute epiglottitis requires direct visualization of an erythematous and swollen epiglottis and adjacent structures by laryngoscopy. Because of the risk of airway obstruction with direct laryngoscopy, the procedure should be performed in children only when skilled personnel and equipment are available to secure the airway.[72] The procedure should also be performed with extreme caution in adults to avoid sudden airway obstruction.[77] However, in a study of 129 cases of acute epiglottitis in adults by Frantz and colleagues,[75] indirect laryngoscopy did not precipitate airway compromise in any patient. In addition, Mayo-Smith and colleagues[74] reviewed hospital records of 407 cases of epiglottitis in children and adults, and found that direct and indirect laryngoscopic examinations did not precipitate an acute airway obstruction in a single patient.

Management Patients with suspected acute epiglottitis require immediate otolaryngologic consultation, close monitoring of the airway with intubation if necessary, and treatment with intravenous antibiotics.[72,77]

Acute epiglottitis can result in sudden, unpredictable airway obstruction in previously healthy individuals. Younger children (younger than 5 years) are predisposed to sudden airway collapse, and should be immediately intubated in the operating room with an otolaryngologist or surgeon present. In addition, patients with acute airway obstruction or severe respiratory distress at time of presentation should have an airway established immediately, with surgical backup for immediate cricothyrotomy or tracheostomy if intubation fails. Individual with signs and symptoms of impending respiratory distress, including respiratory discomfort, stridor, or drooling, should have an artificial airway established as part of their initial care. In addition, patients who present with symptoms of short durations (<12–24 hours), rapid progression, or with significant enlargement of the epiglottis on radiography or laryngoscopy should strongly be considered for establishment of an urgent artificial airway. Patients with mild symptoms and without respiratory difficulty, stridor, or drooling, and who have only mild swelling on laryngoscopy, may be considered for observation in the intensive care unit without an artificial airway but with high vigilance to the patient's airway patency.[74] The decision to observe the patient without an artificial airway should be made in consultation with an otolaryngologist.

In addition to careful airway management, patients with acute epiglottitis should be started on intravenous antibiotics urgently. Although the Hib vaccination has greatly reduced the Hib infection in children, there is occasional failure of the vaccine, and cases of Hib epiglottitis can still present in both vaccinated and unvaccinated children. Bacteria more commonly associated with epiglottitis include *S pneumoniae*, *S aureus*, and β-hemolytic streptococci. Empirical urgent treatment with cefotaxime, ceftriaxone, or ampicillin/sulbactam is recommended.[72]

Neck Pain

Occult cervical spine injury

The incidence of cervical spine injury following blunt trauma has been estimated to be around 4.3%,[79] and diagnosis of cervical spine injury is a high-risk area, as unrecognized injury can result in catastrophic neurologic disability. The management of potential cervical spine injuries in patients who have sustained major trauma or are comatose involves liberal use of imaging, and maintaining cervical immobilization until consultation with a trauma surgeon. However, in less severe trauma the decision of which stable, alert trauma patients require cervical spine imaging can be a difficult one. Two clinical decision rules have been validated and are sensitive for determining the need for cervical spine imaging in patients with blunt trauma: these are the NEXUS Low-Risk Criteria and the Canadian C-Spine Rule.[80,81]

The NEXUS Low-Risk Criteria requires that cervical spine radiography is indicated for patients with blunt trauma unless they meet the following criteria: no posterior midline cervical spine tenderness, no evidence of intoxication, a normal level of alertness, no focal neurologic deficit, and no painful distracting injuries. This decision instrument was validated in a prospective observational study at 21 centers across the United States, and was found to have a sensitivity of 99% and specificity of 12.9%. The decision instrument identified all 8 of 818 patients who had cervical spine injury, and only 2 of those 8 patients were classified as having a clinically significant injury.[80]

The Canadian C-Spine Rule comprises 3 main questions: (1) is there any high-risk factor present that mandates radiography (ie, age $\geq$65 years, dangerous mechanism, or paresthesias in the extremities)? (2) is there any low-risk factor present that allows safe assessment of range of motion (ie, simple rear-end motor vehicle collision, sitting position in ED, ambulatory at any time since injury, delayed onset of neck pain, or absence of midline C-spine tenderness)? and (3) is the patient able to actively rotate neck 45° to the left and right? In this study, patients had to be alert (GCS = 15) and stable (normal vital signs). Patients were excluded if they did not fulfill the first 2 criteria, were younger than 16 years, were injured more than 48 hours previously, had penetrating trauma, presented with acute paralysis, had known vertebral disease (ankylosing spondylitis, rheumatoid arthritis, spinal stenosis, or previous cervical surgery), had returned for reassessment of the same injury, or were pregnant. In the derivation study, the Canadian C-Spine Rule demonstrated a sensitivity of 100% and specificity of 42.5% for identifying clinically important cervical spine injuries.[81]

These 2 studies were compared in a prospective cohort study conducted in the 9 Canadian EDs where the initial Canadian C-Spine Rule was derived. In this study, the sensitivity of the Canadian C-Spine Rule versus NEXUS was 99.4% versus 90.7%, and the specificity was 45.1% versus 36.8%.[82] Either of these 2 clinical decision rules may be used in evaluating the awake, alert patient who has recently sustained blunt trauma.

Cervicocranial artery dissection

Dissections of the carotid and vertebral arteries usually arise from an intimal tear in the vessel, allowing arterial blood to enter the wall of the artery and form a false lumen, which then results in either stenosis or aneurysmal dilatation of the vessel.[83] Such dissections are relatively common causes of stroke, especially in young patients, via artery-to-artery embolism or stenosis with occlusion of the proximal vessel causing ischemic strokes, or vessel rupture resulting in a hemorrhagic stroke.[43] Although trauma is commonly associated with cervicocranial artery dissections, at least 50% of people with dissections and stroke have no clear history of antecedent neck

trauma.[43,84] The high-risk features of cervicocranial artery dissections are related to the subtle clinical presentations of this elusive diagnosis.

Signs and symptoms In carotid artery dissection, the classical clinical presentation is a patient presenting with pain on one side of the head, face, or neck accompanied by a partial Horner syndrome (oculosympathetic palsy, consisting of miosis and ptosis), and followed hours to days later by cerebral or retinal ischemia. This triad is found in less than one-third of patients, but the presence of any 2 elements should strongly suggest the diagnosis. Other local manifestations may include headache (present in approximately 2-thirds of patients), cranial nerve palsies, pulsatile tinnitus, or an objective bruit noted on physical examination.[83]

The initial manifestations of vertebral-artery dissection are less distinct and often present as pain in the back of the neck or head, which is frequently assumed to be musculoskeletal initially, followed by ischemia of the posterior circulation. A headache occurs in about two-thirds of patients and is almost always in the occipital area.[83]

Diagnosis and treatment Diagnosis of cervicocranial dissection can be made via multiple imaging modalities, including angiography, MRI and MR angiography, Doppler sonography, and CT angiography.[85] Once dissection is diagnosed, urgent consultation with a neurologist is required. If intracerebral hemorrhage is excluded, emergent anticoagulation with intravenous heparin may be recommended to prevent thrombotic complications. Most dissections will heal spontaneously, but surgical or endovascular treatment is an option in patients who have failed conservative medical management.[43,83]

DIFFICULT AIRWAY

Airway management is a critical skill for the emergency physician. When intubation is indicated, the most important question the physician can ask is "Is this airway difficult?"[86] A difficult airway is one in which the preintubation examination identifies attributes that may make bag-mask ventilation, laryngoscopy, intubation, the use of an extraglottic device, or surgical airway management more difficult than if those features were not present. A difficult airway can become a failed airway when the initial method chosen for airway management is not successful and an alternative method must be undertaken.[86,87]

Given the high level of acuity and necessity for rapid intervention of many emergency patients requiring airway management, difficult airways are common in the ED, with some estimates as high as 20% of all airways managed in the ED.[86] Despite this fact, the incidence of failed airway is relatively low, around 2% to 3%. An observational study by Sakles and colleagues[88] found that of 610 patients requiring airway control in the ED, only 13 (2.1%) were complicated by a failed airway, with 7 (1%) of those patients requiring rescue by cricothyrotomy. Similar results were seen in a prospective observational study by Bair and colleagues[87] of 7712 intubations in 30 different EDs that were enrolled in the NEAR (National Emergency Airway Registry) project. In this study, a total of 207 (2.7%) of emergency intubations were failed airways requiring rescue technique. Of the 207 failed airways, the majority occurred when rapid sequence intubation (RSI) was not used initially, and consequently RSI was the most common rescue technique accounting for half of all rescued airways. In addition, 44 (0.6%) airways required rescue by surgical intervention (cricothyrotomy or tracheostomy).

Although the failed airway is relatively uncommon in the ED, the morbidity and mortality associated with a failed airway is high. It is essential for emergency

physicians to recognize the difficult airway early and thoughtfully plan for its management, so as to minimize the number of failed airways in the ED. This section focuses on the high-risk feature of airway management by discussing predictors of a possible difficult airway as well as alternative airway management techniques that may be used to rescue a failed airway.

Recognizing the Difficult Airway

The failed airway is a dreaded complication of emergency airway management. To minimize failure in airway management, it is important to recognize the difficult airway in advance in order to execute the appropriate plan to maximize airway success.[86] This section discusses how to recognize patients who may be difficult to mask ventilate, perform laryngoscopy on, or intubate, so that the airway management in these patients can be planned to minimize the likelihood of a ''can't intubate, can't oxygenate'' complication.

Difficult mask ventilation

Mask ventilation is an essential component of airway management,[89] and is of utmost importance in ventilating and oxygenating the patient who is impossible to intubate. The technique for effective mask ventilation is not reviewed here, but should be mastered by all health care providers involved in airway management. This section reviews the predictors of difficult mask ventilation, so that providers can quickly recognize patients who may be difficult to mask ventilate and proceed accordingly.

Difficult mask ventilation may be caused by inadequate mask seal, excessive gas leak, or excessive resistance to the ingress or egress of gas. Signs of inadequate mask ventilation include absent or inadequate chest movement, absent or inadequate breath sounds, auscultatory signs of severe obstruction, cyanosis, gastric air entry or dilatation, decreasing or inadequate oxygen saturation, absent or inadequate exhaled carbon dioxide, absent or inadequate spirometric measures of exhaled gas flow, and hemodynamic changes associated with hypoxemia or hypercarbia.[89]

Several studies have looked at the predictors of difficult or impossible mask ventilation in the anesthesiology setting. A prospective observational study by Langeron and colleagues[90] reported difficult mask ventilation in 75 of 1502 (5%) patients undergoing general anesthesia, with 1 case of impossible ventilation. In this study, the independent factors for difficult mask ventilation were age older than 55 years, body mass index (BMI; calculated as the weight in kilograms divided by height in meters squared) greater than 26 kg/m^2, presence of a beard, lack of teeth, and history of snoring. Similar results were found in a prospective study by Kherterpal and colleagues,[91] which reported 313 of 22,660 (1.4%) patients as difficult to mask ventilate. Independent predictors of difficult mask ventilation were BMI 30 kg/m^2 or greater, presence of a beard, Mallampati score of III or IV, age 57 years or older, severely limited mandibular protrusion, or a history of snoring. Kherterpal and colleagues[92] then evaluated factors associated with impossible mask ventilation in an observational study that reported 77 of 53,041 patients (0.15%) who were impossible to mask ventilate. The 5 independent predictors of impossible mask ventilation were neck radiation changes, male sex, sleep apnea, Mallampati classification of III or IV, and the presence of a beard.

In managing the emergent airway, the validated predictors of difficult mask ventilation are best summarized by Murphy and Walls in the *Manual of Emergency Airway Management* using the mnemonic MOANS (**Box 5**). This mnemonic allows for rapid recall in the ED and can be easily used by any clinician.[86] Although patients who are difficult or impossible to mask ventilate are rare, it is important to recognize

Box 5
Predictors of difficult mask ventilation: MOANS

M—Mask seal

May be complicated by beards, blood on face, or disruption of the lower face

O—Obesity/obstruction

BMI greater than 26 kg/m^2 (including pregnant patients in their third trimester) makes mask ventilation more difficult

Obstruction caused by airway edema, hematomas, tumors, or foreign bodies may complicate mask ventilation

A—Age

Age older than 55 years is associated with a higher risk of difficult mask ventilation due to loss of muscle and tissue tone in the upper airway

N—No teeth

Difficult to obtain an adequate mask seal as the edentulous patient's face tends to cave in

S—Stiff/history of sleep apnea or snoring

Conditions causing stiff lungs resistant to ventilation (ie, reactive airway disease, pulmonary edema, acute respiratory distress syndrome, advanced pneumonia)

History of sleep apnea or snoring

Data from Murphy MF, Walls RM. Identification of the difficult and failed airway. In: Walls RM, Murphy MF. Manual of emergency airway management, 3rd edition. Philadelphia: Lippincott Williams and Wilkins; 2008. p. 81–93.

patients who may be difficult to mask ventilate early, so as to be prepared to use alternative methods to oxygenate and ventilate these patients.

Difficult laryngoscopy and intubation

Unanticipated difficult laryngoscopy and intubation is one of the major challenges of airway management. A landmark article in the *British Medical Journal* by Cass and colleagues[93] in 1956 was the first attempt to identify the anatomic features that may predict difficult intubation. In this article, Cass and colleagues looked at 5 cases of difficult intubation and noted the associated physical features, such as short muscular necks with a full set of teeth, receding lower jaws with obtuse mandibular angles, protruding upper incisors, and poor mobility of the mandible. Since this initial article, numerous investigators have tried to determine with high precision which physical attributes apparent on bedside physical examination could reliably predict successful versus failed laryngoscopy and intubation. Unfortunately, many of these investigations have met with limited or no success,[94–97] and a rapid screening test that is 100% sensitive with a high positive predictive value remains elusive.

In the absence of a proven and validated rule with high sensitivity and specificity in predicting difficult intubation, it is important to have a way to quickly identify those patients who might be difficult to intubate in an emergency setting so that an appropriate plan may be made. The LEMON method (**Box 6**) devised by the US National Emergency Airway Management Course is a simple assessment tool assembled from an analysis of the difficult airway prediction instruments in the anesthesia literature, and is currently undergoing a validation study by the multicenter National Emergency Airway Registry Project (NEAR III).[86] This tool has been favorably assessed in

Box 6
Predictors of difficult laryngoscopy and intubation: LEMON method

L—Look externally

Look at the patient for any external characteristics or a gestalt "feeling" that may indicate difficult laryngoscopy, intubation, or ventilation

E—Evaluate the 3-3-2 Rule

3: Mouth opening—the interincisor distance should be at least 3 finger breadths

3: Length of the mandibular space—the distance between the tip of the mentum and the chin-neck junction (hyoid bone) should be at least 3 finger breadths

2: Position of the glottis in relation to the tongue—the distance between the chin-neck junction (hyoid bone) and the thyroid notch should be at least 2 finger breadths

M—Mallampati score

Reflects the relationships among mouth opening, the size of the tongue, and the size of the oral pharynx

Classes relate to degree of difficulty intubating:

I: Soft palate, uvula, fauces, pillars visible—No difficulty

II: Soft palate, uvula, fauces visible—No difficulty

III: Soft palate, base of uvula visible—Moderate difficulty

IV: Hard palate only visible—Severe difficulty

O—Obstruction/obesity

Upper airway obstruction is always a marker for a difficult airway

Obese patients tend to have poor glottic views by direct laryngoscopy

N—Neck mobility

Cervical spine immobilization makes intubation more difficult. Intrinsic cervical spine immobility (ie, ankylosing spondylitis or rheumatoid arthritis) can make direct laryngoscopy extremely difficult or impossible

Data from Murphy MF, Walls RM. Identification of the difficult and failed airway. In: Walls RM, Murphy MF. Manual of emergency airway management, 3rd edition. Philadelphia: Lippincott Williams and Wilkins; 2008. p. 81–93.

a prospective observational study by Reed and colleagues.[98] In this study, a rapid airway assessment score based on the LEMON method successfully stratified the risk of difficult intubation in the ED, and verified an association between patients with large incisors, a reduced interincisor distance, or a reduced thyroid to floor mouth distance and poor laryngoscopic view (Cormack and Lehane laryngoscopy grades 2, 3, or 4). The LEMON method is a useful tool for the rapid assessment and risk stratification of emergency patients requiring intubation, so that the care of patients who may be at risk of difficult intubation can be managed carefully and a failed airway avoided.

Alternative Techniques for Rescue or Difficult Airways

The most important aspect of emergency airway management is to anticipate and prepare for the difficult airway or the failed airway requiring rescue. There are numerous alternative airway techniques, both invasive and noninvasive, to use in difficult or failed airway situations (**Box 7**).[99] The exact technique by which to perform

Box 7
Alternative techniques for rescue or difficult airways

- Endotracheal tube introducers
- Blind intubation
  - –Blind nasotracheal intubation
  - –Digital tracheal intubation
- Fiberoptic intubation
  - –Flexible
  - –Rigid and semirigid stylets
- Lighted stylet intubation
- Video laryngoscopy
- Extraglottic devices
  - –Supraglottic
    - Laryngeal mask airway
  - –Retroglottic
    - Esophageal tracheal Combitube
    - King LT Airway
    - Rush EasyTube
- Surgical airway
  - –Cricothyrotomy
  - –Needle cricothyrotomy with percutaneous tracheal ventilation
  - –Tracheostomy
  - –Translaryngeal guided ("retrograde") intubation

Data from Walls RM, Murphy MF, editors. Manual of emergency airway management, 3rd edition. Philadelphia: Lippincott Williams and Wilkins; 2008.

these alternative techniques is beyond the scope of this review. However, it is the responsibility of every emergency physician to be as well trained as possible in these alternative techniques so as to provide the best possible care to the emergency patient and properly manage the emergency airway.

SUMMARY

Disorders of the head and neck are high-risk areas for the emergency physician. The anatomy is complex and the pathology can be diverse. A high degree of suspicion must be maintained for all potentially life-threatening conditions when patients present with complaints of the head and neck.

REFERENCES

1. Goldstein JN, Camargo CA Jr, Pelletier AJ, et al. Headache in United States emergency departments: demographics, work-up and frequency of pathological diagnoses. Cephalalgia 2006;26(6):684–90.

2. Lipton RB, Bigal ME, Steiner TJ, et al. Classification of primary headaches. Neurology 2004;63(3):427–35.
3. Locker T, Mason S, Rigby A. Headache management—are we doing enough? An observational study of patients presenting with headache to the emergency department. Emerg Med J 2004;21(3):327–32.
4. Karcz A, Korn R, Burke MC, et al. Malpractice claims against emergency physicians in Massachusetts: 1975–1993. Am J Emerg Med 1996;14(4):341–5.
5. Ramirez-Lassepas M, Espinosa CE, Cicero JJ, et al. Predictors of intracranial pathologic findings in patients who seek emergency care because of headache. Arch Neurol 1997;54(12):1506–9.
6. Locker TE, Thompson C, Rylance J, et al. The utility of clinical features in patients presenting with nontraumatic headache: an investigation of adult patients attending an emergency department. Headache 2006;46(6):954–61.
7. Edlow JA, Panagos PD, Godwin SA, et al. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with acute headache. Ann Emerg Med 2008;52(4):407–36.
8. Cortelli P, Cevoli S, Nonino F, et al. Evidence-based diagnosis of nontraumatic headache in the emergency department: a consensus statement on four clinical scenarios. Headache 2004;44(6):587–95.
9. Bederson JB, Connolly ES Jr, Batjer HH, et al. Guidelines for the management of aneurysmal subarachnoid hemorrhage: a statement for healthcare professionals from a special writing group of the Stroke Council, American Heart Association. Stroke 2009;40(3):994–1025.
10. Pope JV, Edlow JA. Favorable response to analgesics does not predict a benign etiology of headache. Headache 2008;48(6):944–50.
11. M S, Lamont AC, Alias NA, et al. Red flags in patients presenting with headache: clinical indications for neuroimaging. Br J Radiol 2003;76(908):532–5.
12. Chaisson R, Volberding P. Clinical manifestations of HIV infection. In: Mandell GL, Bennett JE, Dolin R, editors. Mandell, Douglas and Bennett's principles and practice of infectious diseases. 4th edition. New York: Churchill Livingstone; 1995. p. 1217–53.
13. Rothman RE, Keyl PM, McArthur JC, et al. A decision guideline for emergency department utilization of noncontrast head computed tomography in HIV-infected patients. Acad Emerg Med 1999;6(10):1010–9.
14. Christiaans MH, Kelder JC, Arnoldus EP, et al. Prediction of intracranial metastases in cancer patients with headache. Cancer 2002;94(7):2063–8.
15. Argyriou AA, Chroni E, Polychronopoulos P, et al. Headache characteristics and brain metastases prediction in cancer patients. Eur J Cancer Care (Engl) 2006; 15(1):90–5.
16. Baehring J, Quant E, Hochber F. Metastatic neoplasms and paraneoplastic syndromes. In: Goetz CG, editor. Textbook of clinical neurology. 3rd edition. Philadelphia: Saunders Elsevier; 2007. p. 1081–93.
17. Lloyd-Jones D, Adams R, Carnethon M, et al. Heart disease and stroke statistics—2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2009;119(3):e21–181.
18. Stam J. Thrombosis of the cerebral veins and sinuses. N Engl J Med 2005; 352(17):1791–8.
19. Conicella E, Raucci U, Vanacore N, et al. The child with headache in a pediatric emergency department. Headache 2008;48(7):1005–11.
20. Kan L, Nagelberg J, Maytal J. Headaches in a pediatric emergency department: etiology, imaging, and treatment. Headache 2000;40(1):25–9.

21. ACE Clinical Policies Committee; Clinical Policies Subcommittee on Seizures. Clinical policy: critical issues in the evaluation and management of adult patients presenting to the emergency department with seizures. Ann Emerg Med 2004; 43(5):605–25.
22. Engel J Jr, Starkman S. Overview of seizures. Emerg Med Clin North Am 1994; 12(4):895–923.
23. Walker MC. The epidemiology and management of status epilepticus. Curr Opin Neurol 1998;11(2):149–54.
24. Dunn MJ, Breen DP, Davenport RJ, et al. Early management of adults with an uncomplicated first generalised seizure. Emerg Med J 2005;22(4):237–42.
25. McKeon A, Vaughan C, Delanty N. Seizure versus syncope. Lancet Neurol 2006; 5(2):171–80.
26. Tarabar A, Ulrich A, D'Onofrio G. Seizures. In: Adams J, Barton E, editors. Emergency medicine. Philadelphia: Saunders/Elsevier; 2008. p. 1051–62.
27. Adams J. Emergency medicine. Philadelphia: Saunders/Elsevier; 2008.
28. Lowenstein DH, Alldredge BK. Status epilepticus. N Engl J Med 1998;338(14): 970–6.
29. Takayanagui OM, Odashima NS. Clinical aspects of neurocysticercosis. Parasitol Int 2006;55(Suppl):S111–5.
30. Jolley S, Allen T. Transient ischemic attack and acute ischemic stroke. In: Adams J, Barton E, editors. Emergency medicine. Philadelphia: Saunders/Elsevier; 2008. p. 1072–82.
31. Goldstein LB, Simel DL. Is this patient having a stroke? JAMA 2005;293(19): 2391–402.
32. Norris JW, Hachinski VC. Misdiagnosis of stroke. Lancet 1982;1(8267):328–31.
33. Allder SJ, Moody AR, Martel AL, et al. Limitations of clinical diagnosis in acute stroke. Lancet 1999;354(9189):1523.
34. Kothari RU, Brott T, Broderick JP, et al. Emergency physicians. Accuracy in the diagnosis of stroke. Stroke 1995;26(12):2238–41.
35. Libman RB, Wirkowski E, Alvir J, et al. Conditions that mimic stroke in the emergency department. Implications for acute stroke trials. Arch Neurol 1995;52(11): 1119–22.
36. Scott PA, Silbergleit R. Misdiagnosis of stroke in tissue plasminogen activator-treated patients: characteristics and outcomes. Ann Emerg Med 2003;42(5): 611–8.
37. Winkler DT, Fluri F, Fuhr P, et al. Thrombolysis in stroke mimics: frequency, clinical characteristics, and outcome. Stroke 2009;40(4):1522–5.
38. Savitz SI, Caplan LR. Vertebrobasilar disease. N Engl J Med 2005;352(25): 2618–26.
39. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med 1995; 333(24):1581–7.
40. Hacke W, Kaste M, Bluhmki E, et al. Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. N Engl J Med 2008;359(13):1317–29.
41. Marler JR, Tilley BC, Lu M, et al. Early stroke treatment associated with better outcome: the NINDS rt-PA stroke study. Neurology 2000;55(11):1649–55.
42. Adams HP Jr, del Zoppo G, Alberts MJ, et al. Guidelines for the early management of adults with ischemic stroke: a guideline from the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research

Interdisciplinary Working Groups: the American Academy of Neurology affirms the value of this guideline as an educational tool for neurologists. Stroke 2007; 38(5):1655–711.
43. Sacco RL, Adams R, Albers G, et al. Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack: a statement for healthcare professionals from the American Heart Association/American Stroke Association Council on Stroke: co-sponsored by the Council on Cardiovascular Radiology and Intervention: the American Academy of Neurology affirms the value of this guideline. Circulation 2006;113(10):e409–49.
44. Albers GW, Caplan LR, Easton JD, et al. Transient ischemic attack—proposal for a new definition. N Engl J Med 2002;347(21):1713–6.
45. Johnston SC, Gress DR, Browner WS, et al. Short-term prognosis after emergency department diagnosis of TIA. JAMA 2000;284(22):2901–6.
46. Flaherty ML, Haverbusch M, Sekar P, et al. Long-term mortality after intracerebral hemorrhage. Neurology 2006;66(8):1182–6.
47. Broderick J, Connolly S, Feldmann E, et al. Guidelines for the management of spontaneous intracerebral hemorrhage in adults: 2007 update: a guideline from the American Heart Association/American Stroke Association Stroke Council, High Blood Pressure Research Council, and the Quality of Care and Outcomes in Research Interdisciplinary Working Group. Stroke 2007;38(6):2001–23.
48. Qureshi AI, Tuhrim S, Broderick JP, et al. Spontaneous intracerebral hemorrhage. N Engl J Med 2001;344(19):1450–60.
49. Nawar EW, Niska RW, Xu J. National Hospital Ambulatory Medical Care Survey: 2005 emergency department summary. Adv Data 2007;(386):1–32.
50. Broocker G. The ophthalmic examination. In: Wolfson A, Hendey G, Hendry P, et al, editors. Harwood-Nuss' clinical practice of emergency medicine. 4th edition. Philadelphia: Lippincott, Williams & Wilkins; 2005. p. 112–7.
51. Knoop K, Dennis W, Hedges J. Ophthalmologic procedures. In: Roberts J, Hedges J, Chanmugam A, et al, editors. Clinical procedures in emergency medicine. 4th edition. Philadelphia: Saunders/Elsevier; 2004. p.1241–79.
52. Blaivas M, Theodoro D, Sierzenski PR. A study of bedside ocular ultrasonography in the emergency department. Acad Emerg Med 2002;9(8):791–9.
53. Hayreh SS, Zimmerman MB, Kimura A, et al. Central retinal artery occlusion. Retinal survival time. Exp Eye Res 2004;78(3):723–36.
54. Vortmann M, Schneider JI. Acute monocular visual loss. Emerg Med Clin North Am 2008;26(1):73–96, vi.
55. Kuckelkorn R, Kottek A, Schrage N, et al. Poor prognosis of severe chemical and thermal eye burns: the need for adequate emergency care and primary prevention. Int Arch Occup Environ Health 1995;67(4):281–4.
56. Magauran B. Conditions requiring emergency ophthalmologic consultation. Emerg Med Clin North Am 2008;26(1):233–8, viii.
57. Babineau MR, Sanchez LD. Ophthalmologic procedures in the emergency department. Emerg Med Clin North Am 2008;26(1):17–34, v-vi.
58. Colby K. Approach to the ophthalmologic patient. In: Beers M, Porter R, Jones T, et al, editors. The Merck manual of diagnosis and therapy. 18th edition. Whitehouse Station (NJ): Merck Research Laboratories, Division of Merck & Co., Inc.; 2006. p. 867–928.
59. Cooper RJ, Hoffman JR, Bartlett JG, et al. Principles of appropriate antibiotic use for acute pharyngitis in adults: background. Ann Intern Med 2001;134(6):509–17.
60. Lee JK, Kim HD, Lim SC. Predisposing factors of complicated deep neck infection: an analysis of 158 cases. Yonsei Med J 2007;48(1):55–62.

61. Vieira F, Allen SM, Stocks RM, et al. Deep neck infection. Otolaryngol Clin North Am 2008;41(3):459–83, vii.
62. Marra S, Hotaling AJ. Deep neck infections. Am J Otolaryngol 1996;17(5): 287–98.
63. Bottin R, Marioni G, Rinaldi R, et al. Deep neck infection: a present-day complication. A retrospective review of 83 cases (1998–2001). Eur Arch Otorhinolaryngol 2003;260(10):576–9.
64. Eftekharian A, Roozbahany NA, Vaezeafshar R, et al. Deep neck infections: a retrospective review of 112 cases. Eur Arch Otorhinolaryngol 2009;266(2):273–7.
65. Wang LF, Kuo WR, Tsai SM, et al. Characterizations of life-threatening deep cervical space infections: a review of one hundred ninety-six cases. Am J Otolaryngol 2003;24(2):111–7.
66. Karkos PD, Leong SC, Beer H, et al. Challenging airways in deep neck space infections. Am J Otolaryngol 2007;28(6):415–8.
67. Ovassapian A, Tuncbilek M, Weitzel EK, et al. Airway management in adult patients with deep neck infections: a case series and review of the literature. Anesth Analg 2005;100(2):585–9.
68. Shockley WW. Ludwig angina: a review of current airway management. Arch Otolaryngol Head Neck Surg 1999;125(5):600.
69. Brook I. Microbiology and management of peritonsillar, retropharyngeal, and parapharyngeal abscesses. J Oral Maxillofac Surg 2004;62(12):1545–50.
70. Huang TT, Liu TC, Chen PR, et al. Deep neck infection: analysis of 185 cases. Head Neck 2004;26(10):854–60.
71. Boscolo-Rizzo P, Marchiori C, Zanetti F, et al. Conservative management of deep neck abscesses in adults: the importance of CECT findings. Otolaryngol Head Neck Surg 2006;135(6):894–9.
72. Alcaide ML, Bisno AL. Pharyngitis and epiglottitis. Infect Dis Clin North Am 2007; 21(2):449–69, vii.
73. Frantz TD, Rasgon BM. Acute epiglottitis: changing epidemiologic patterns. Otolaryngol Head Neck Surg 1993;109(3 Pt 1):457–60.
74. Mayo-Smith MF, Spinale JW, Donskey CJ, et al. Acute epiglottitis. An 18-year experience in Rhode Island. Chest 1995;108(6):1640–7.
75. Frantz TD, Rasgon BM, Quesenberry CP Jr. Acute epiglottitis in adults. Analysis of 129 cases. JAMA 1994;272(17):1358–60.
76. Carey MJ. Epiglottitis in adults. Am J Emerg Med 1996;14(4):421–4.
77. Shores C. Infections and disorders of the neck and upper airway. In: Tintinalli JE, Kelen GD, Stapczynski JS, editors. Emergency medicine: a comprehensive study guide. 6th edition. New York: McGraw-Hill; 2004. p. 1494–501.
78. Stankiewicz JA, Bowes AK. Croup and epiglottitis: a radiologic study. Laryngoscope 1985;95(10):1159–60.
79. Grossman MD, Reilly PM, Gillett T, et al. National survey of the incidence of cervical spine injury and approach to cervical spine clearance in U.S. trauma centers. J Trauma 1999;47(4):684–90.
80. Hoffman JR, Mower WR, Wolfson AB, et al. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. N Engl J Med 2000; 343(2):94–9.
81. Stiell IG, Wells GA, Vandemheen KL, et al. The Canadian C-spine rule for radiography in alert and stable trauma patients. JAMA 2001;286(15):1841–8.
82. Stiell IG, Clement CM, McKnight RD, et al. The Canadian C-spine rule versus the NEXUS low-risk criteria in patients with trauma. N Engl J Med 2003;349(26):2510–8.

83. Schievink WI. Spontaneous dissection of the carotid and vertebral arteries. N Engl J Med 2001;344(12):898–906.
84. Bassi P, Lattuada P, Gomitoni A. Cervical cerebral artery dissection: a multicenter prospective study (preliminary report). Neurol Sci 2003;24(Suppl 1):S4–7.
85. Provenzale JM. MRI and MRA for evaluation of dissection of craniocerebral arteries: lessons from the medical literature. Emerg Radiol 2009;16(3):185–93.
86. Murphy M, Walls R. Identification of the difficult and failed airway. In: Walls R, Murphy M, editors. Manual of emergency airway management. 3rd edition. Philadelphia: Lippincott Williams & Wilkins; 2008. p. 81–93.
87. Bair AE, Filbin MR, Kulkarni RG, et al. The failed intubation attempt in the emergency department: analysis of prevalence, rescue techniques, and personnel. J Emerg Med 2002;23(2):131–40.
88. Sakles JC, Laurin EG, Rantapaa AA, et al. Airway management in the emergency department: a one-year study of 610 tracheal intubations. Ann Emerg Med 1998; 31(3):325–32.
89. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Anesthesiology 2003;98(5):1269–77.
90. Langeron O, Masso E, Huraux C, et al. Prediction of difficult mask ventilation. Anesthesiology 2000;92(5):1229–36.
91. Kheterpal S, Han R, Tremper KK, et al. Incidence and predictors of difficult and impossible mask ventilation. Anesthesiology 2006;105(5):885–91.
92. Kheterpal S, Martin L, Shanks AM, et al. Prediction and outcomes of impossible mask ventilation: a review of 50,000 anesthetics. Anesthesiology 2009;110(4): 891–7.
93. Cass NM, James NR, Lines V. Difficult direct laryngoscopy complicating intubation for anaesthesia. Br Med J 1956;1(4965):488–9.
94. Savva D. Prediction of difficult tracheal intubation. Br J Anaesth 1994;73(2): 149–53.
95. Tse JC, Rimm EB, Hussain A. Predicting difficult endotracheal intubation in surgical patients scheduled for general anesthesia: a prospective blind study. Anesth Analg 1995;81(2):254–8.
96. Iohom G, Ronayne M, Cunningham AJ. Prediction of difficult tracheal intubation. Eur J Anaesthesiol 2003;20(1):31–6.
97. Shiga T, Wajima Z, Inoue T, et al. Predicting difficult intubation in apparently normal patients: a meta-analysis of bedside screening test performance. Anesthesiology 2005;103(2):429–37.
98. Reed MJ, Dunn MJ, McKeown DW. Can an airway assessment score predict difficulty at intubation in the emergency department? Emerg Med J 2005;22(2): 99–102.
99. Walls RM, Murphy MF. Manual of emergency airway management. 3rd edition. Philadelphia: Lippincott Williams & Wilkins; 2008.