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Children often present to the emergency department (ED) complaining of headaches. While most headaches in children are not signs of serious, underlying disorders, some headaches may be the first presentation of a migraine headache. Migraine is the most common cause of recurrent, intermittent headaches in children.[1-3] However, migraine headaches often are underdiagnosed and undertreated in children.

Migraine symptoms, in general, and especially in children, can vary dramatically in terms of character and severity. The diagnosis of migraine headache is based primarily on the history of symptoms and supported by a positive family history and a normal physical examination. Neurodiagnostic tests often are unnecessary and serve only to exclude nonmigraine disorders.

Recent advances in the understanding of the pathophysiology and genetics of migraine have led to new therapeutic interventions for the treatment of migraine headaches.

This review presents a comprehensive approach to the child with migraine headaches. An overview of the current understanding of the pathophysiology of migraine headaches is presented, followed by a description of common and classic migraine headaches, migraine variants, migraine equivalents, and treatment options. The author concludes with a summary of the therapeutic approach to the child who presents acutely with a migraine headache.

—The Editor

Pediatric Migraine: Recognizing and Managing Big Headaches in Small Patients

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Introduction

Headaches commonly occur in children. Interestingly, many children who present with headaches are suffering from migraines. However, many parents, and even clinicians, are unaware that migraine headaches commonly occur in pediatric patients.

Migraine headaches are considered to be a hereditary disorder, characterized by autosomal dominant inheritance with incomplete penetrance, and are the most common cause of recurrent intermittent headaches in children.[1-3] Migraine headaches occur in 3-5% of all children and in as many as 18% of adolescents.[4] Prevalence rates in preschool children probably are underestimated, as migraine symptoms tend to be atypical in the younger age group and rarely are identified as migraines.

Prior to puberty, prevalence rates in children are equal among both sexes, but post-pubertal girls are more than twice as likely as boys to suffer from migraine headaches, most likely because of the triggering effect of the menstrual cycle on migraine headaches.[1] A 1999 population based survey found that in individuals 12 years of age and older, 18.2% of females were found to have migraine headaches compared to 6.5% of males.[5]

It is important to recognize that many children begin to have migraine headaches at a young age. A prospective, longitudinal, 10-year follow-up study of pediatric patients with migraine found that 24.3% of patients had their first headache before 6 years of age and 57% at 6-10 years of age.[6] Among adults with headaches, nearly half say that their headaches began before they were 10 years of age.[7]

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Most investigations into the diagnosis and management of migraine headaches have occurred in the adult population. However, our current understanding of migraine headache suggests that the diagnosis, presentation, natural history, prognosis, and response to medications are different for children than for adults. Unfortunately, only a few controlled trials have investigated the acute and prophylactic use of medications in the treatment of migraine in children.

The Pathophysiology of Migraine

Understanding the mechanisms by which migraines occur in children has led to new therapeutic options and more effective treatments for migraine headaches and symptoms.

Previously, three major mechanisms for the pathogenesis of migraine had been proposed: vascular, humoral, and neural. The vascular theory suggested that during the initial phase of a migraine headache, a wave of cortical excitation followed by cortical depression spread over both hemispheres from back to front, in association with decreased regional blood flow and transitory neurologic disturbances.[8] In subsequent phases of migraine progression, increased blood flow was thought to occur in the internal and external carotid circulation, distending pain-sensitive extra-cranial arteries and resulting in a sterile inflammation.

The humoral theory proposed that increased serotonin levels resulted in vasoconstriction of the innervated vascular system and decreased cerebral blood flow. As hypoperfusion progressed, aura and headache developed.

Finally, the neural mechanism proposed that migraine headaches occur due to an inherited sensitivity of the trigeminal vascular system.[8-12] This mechanism suggested that cortical, thalamic, or hypothalamic mechanisms initiated an attack due to some internal or external stimuli. The locus ceruleus and nucleus raphe dorsalis were stimulated, which in turn stimulated the cortex via serotoninergic and noradrenergic pathways and produced a spreading wave of neuronal depression. The cranial vasculature also was stimulated, resulting in neurogenic inflammation and secondary vascular reactivity. Released vasoactive peptides stimulated endothelial cells, mast cells, and platelets, creating a cascade that resulted in sterile inflammation of dural and pial blood vessels from which nociceptive afferents transmitted pain signals centrally via the trigeminal nerve.

Currently, most investigators believe that migraine is a disorder that combines aspects of both the neural and vascular theories. In fact, serotonin is believed to play a key role in the pathophysiology of migraine headaches and has been the focus of some of the newer antimigraine medications.

The Diagnosis of Migraine

While children often present to the ED complaining of a headache, diagnosing a child with a migraine headache can be difficult. While there are many reasons why making the diagnosis of migraine is difficult, uncertainty about the definition of a migraine headache, its varied presentations, and lack of a clear understand-

Table 1. IHS Criteria for the Diagnosis of MIgraine with and without Aura in Children

MIGRAINE WITH AURA

1. Have at least two attacks fulfilling Criteria No. 2.
2. Each attack has at least three of the following four characteristics:
   a. Fully reversible aura symptoms indicating focal cerebral cortical and/or brainstem dysfunction
   b. More than one aura symptom that develops gradually in more than four minutes or has more than two symptoms that occur in succession
   c. No aura symptom lasting more than 60 minutes. If more than one aura symptom is present, accepted duration is proportionally increased.
   d. The headache follows aura symptoms with a free interval of fewer than 60 minutes, but may begin before or simultaneously with the aura.

MIGRAINE WITHOUT AURA

1. Have at least five attacks fulfilling Criteria Nos. 2-4
2. Headache attacks lasting 4-72 hours (2-48 hours in patients younger than15 years)
3. The headache has at least two of the following characteristics:
   a. Unilateral location
   b. Pulsing quality
   c. Moderate to severe intensity
   d. Aggravation by routine physical activities
4. During an attack, the headache is accompanied by one of the following symptoms:
   a. Nausea and/or vomiting
   b. Photophobia and/or phonophobia

Table 2. Proposed Modified IHS Criteria for the Diagnosis of Migraine without Aura in Children

MIGRAINE WITHOUT AURA

1. Have at least five attacks fulfilling Criteria Nos. 2-4.
2. Headache attacks lasting 1-48 hours
3. During an attack, the headache has at least two of the following characteristics:
   a. Bilateral or unilateral location
   b. Pulsating quality
   c. Moderate to severe intensity
   d. Aggravation by routine physical activities
4. During an attack, the headache is accompanied by one of the following symptoms:
   a. Nausea and/or vomiting
   b. Photophobia and/or phonophobia

ing of its pathophysiology are of particular concern. The International Headache Society (IHS) has developed criteria for the diagnosis of migraine headaches and classifies migraines as either migraine without aura or migraine with aura.[8] *(See Table 1.)* In essence, the diagnosis of migraine headache in children is based on an evaluation of the presenting clinical signs and symptoms. However, many investigators believe that the IHS criteria for migraine without aura are relatively insensitive for the diagnosis of migraines in children and have suggested several revisions to the criteria. Two modifications to the original criteria, a reduction in the minimal time required for the duration of the headache and the inclusion of a bifrontal location for the headache, increase the sensitivity of the criteria for diagnosing migraine headaches in children from 66% to 88%.[13] Table 2 lists the proposed revisions to the IHS criteria for the diagnosis of migraine without aura in children.

History and Physical Examination. The evaluation of the child who presents with a migraine headache begins with the history and physical examination. In particular, the history often will provide clues to the diagnosis, with the physical examination excluding other causes of headaches.

A history of migraine headaches often is documented in at least one parent in 90% of children with migraine. In addition, a family history of motion sickness also is common, as well as a history of the child suffering from motion sickness, bruxism, fainting spells, sleep talking, and sleepwalking.[14,15] Children with a history of migraine headaches also are more likely to suffer from ice cream headaches than children without such a history.[16]

Interestingly, infantile colic also may be a migraine-related phenomenon. Recent studies have suggested that compared to control subjects, children with migraine headaches have a higher prevalence of sleep disturbances and infantile colic.[17,18] Children with a history of infantile colic also are more likely to have a family history of migraine.[17] In a recent case study of an infant with colic who also was thought to have migraine headaches, complete resolution of the symptoms of colic occurred following the institution of anti-migraine therapy (cyproheptidine).[19]

While migraine attacks can begin at any time of the day, they more commonly occur during the late afternoon in children and even earlier in the day for adolescents. Most children who suffer from migraine headaches will have 1-4 attacks per month. However, children may be asymptomatic for long intervals and then suffer a cluster of attacks. While migraine typically is one-sided in adult patients, a bilateral headache is more common in children. In addition, the headache more often is described as throbbing.

Many triggers have been identified for the initiation of migraine headaches in both children and adults. Common triggers include stress, fatigue, hunger, head injury, exercise, menses, foods, and weather changes. Many of the foods that have been identified as triggers of migraine headaches contain tyramine, phenylethylamine, caffeine, or artificial sweeteners and colorings. Often, a headache diary can help identify migraine triggers in children.

A child who presents with a headache should have a thorough medical history obtained and a complete physical examination performed, including the measurement of vital signs. The physical examination should include a thorough neurological examination and an examination of the optic fundus to exclude the diagnosis of a more serious neurological condition, such as a brain tumor.

Parents should be informed that most children with brain tumors will present with other concerning clinical signs and symp-

toms in addition to that of a headache. In a review of children with known brain tumors, investigators found that 85% of children had an abnormal physical examination within two months of the onset of headache.[21] Common findings included ataxia, head tilt, and papilledema. Historically, the following factors were found to be more commonly associated with children with a brain tumor headache: 1) recurrent morning headaches; 2) headaches that repeatedly awaken the child from sleep; 3) intense, prolonged, incapacitating headaches; and 4) changes in the quality, frequency, and pattern of headaches. Seventy-eight percent of patients with a brain tumor headache presented with repeated episodes of emesis.

Diagnostic Testing. Currently, no consensus exists among investigators and clinicians as to the role of routine diagnostic testing in the evaluation of children with headache. This includes the use of routine laboratory testing, cerebrospinal fluid examination, electroencephalographs (EEG), and neuroimaging. It is important to remember, however, that while the child who presents with a classic presentation of migraine headache may not require routine diagnostic testing, the patient who presents acutely with a severe headache that is not classic for migraine still may require testing to determine whether an underlying disease process is present. This review will concentrate on the evaluation of the child with a headache thought to be migraine in origin. As such, there is no evidence to suggest a role for routine laboratory testing, lumbar puncture, and EEG testing in the evaluation of the child with migraine headache.[20]

More problematic is the routine use of neuroimaging, either computed tomography (CT) or magnetic resonance imaging (MRI), in the evaluation of children with migraine headaches. Often, parents, and even children, may be concerned that the headache is a symptom of a more serious underlying problem, such as a brain tumor. In fact, parents may be insistent on radiographic imaging of the head. In general, however, CT and MRI are not required for the standard evaluation of a child presenting with a migraine headache.

Recently, Lewis and Dorbad conducted a retrospective review of the use of neuroimaging in the evaluation of children presenting with a migraine headache who had a normal neurological examination.[22] Of 107 patients with migraine, 40% had CT scans, only two of which were considered abnormal. Twelve patients had MRIs, of which two were considered abnormal. Abnormalities included a Chiari type 1 malformation (N = 2), an arachnoid cyst (N = 1), and a dilated Virchow-Robin space (N = 1). None of the findings changed the diagnosis, management, or outcome of patients. The authors concluded that neuroimaging is not warranted in the routine evaluation of children presenting with a migraine headache and a normal neurological examination.

Similarly, Maytal and colleagues studied 133 patients presenting with headache and reported that of 78 patients who had either an MRI or CT scan, only four patients were found to have abnormalities,[23] and none of the abnormalities were considered clinically relevant or related to the presenting complaint.

Classic Migraine—Migraine with Aura. While classic migraine is the best-recognized presentation of migraine, it occurs in fewer than one-third of children with migraine headaches.[24] Historically, classic migraine has been described as a biphasic event. Typically, symptoms of aura occur during the first phase, and headache, nausea, and vomiting occur during the second phase. The phrase "classic migraine" largely has been replaced by the phrase "migraine with aura."

During a migraine with aura, symptoms may occur only during the first phase, resulting in what is termed a migraine equivalent; only during the second phase, resulting in headache and vomiting; or during both. An aura is defined as any abrupt neurological change that is fully reversible and precedes a migraine headache. The most common symptoms to occur during the first phase of a migraine headache include visual aberrations, such as the perception of dancing lights, blind spots, blurred vision, visual hallucinations, or transitory blindness (the aura). Visual symptoms tend to be specific for each child suffering from migraine headaches.

Migraine headaches may terminate after the first phase without headache or proceed to the second phase with symptoms of headache and nausea. Headaches tend to be dull at first, becoming throbbing, pulsating, or pounding. Headache is unilateral in two-thirds of older children and adult patients and most intense in the region of the eye, forehead, or temple. However, younger patients with migraine headaches often will complain of bilateral temporal pain. Eventually, the headache becomes constant and diffuse, lasting a variable length of time. Anorexia and photophobia often are present.

Common Migraine. Common migraine is the most common form of migraine in children. Common migraines differ from classic migraine in that the symptoms do not regularly develop in a biphasic mode of visual aura followed by headache. Most children with common migraine often will present with malaise, dizziness, nausea, and vomiting that is followed by either a unilateral or bilateral, pounding headache. The child will appear sick, often will want to lie down, and will be extremely sensitive to light and sound. The headache often will end when the child falls asleep. The phrase "common migraine" largely has been replaced by the phrase "migraine without aura."

Migraine Equivalents. Migraine equivalents or complicated migraines are migraine headaches that are associated with transient neurological deficits or alterations in states of consciousness. Migraine equivalents are thought to occur due to prolonged vasoconstriction and ischemia of specific cerebral areas. The type of neurological deficit that occurs, and thus the type of complicated migraine, is dependent on the vascular territory affected. In migraine equivalents, neurological deficits often will precede the symptom of headache, but can occur following it instead. In some instances, a headache may not even occur. Migraine equivalents usually are benign, and many children will go on to develop more typical migraine headaches later in life. Types of migraine equivalents include hemiplegic or hemisensory migraines, ophthalmo-

plegic migraines, basilar artery migraines, acute confusional migraines, and the Alice in Wonderland syndrome. (*See below.*)

Hemiplegic or hemisensory migraines, more common in younger children than in adults, are characterized by the sudden onset of hemiparesis or hemisensory loss followed by a contralateral headache. Symptoms are thought to occur due to vasoconstriction and neuronal dysfunction resulting in ischemia in the distribution of the middle cerebral artery.[25] Symptoms can last hours to days, though the hemiparesis often outlasts the headache.[25] Interestingly, the hemiparesis may alternate sides during subsequent attacks.[25]

Ophthalmoplegic migraines are characterized by unilateral eye pain, headache, and transient ipsilateral third nerve palsy and is thought to occur either because of edema involving the internal carotid artery within the cavernous sinus or because of edema involving the distal basilar artery.[25] Third nerve involvement occurs in 80% of cases.[26] The eye pain in ophthalmoplegic migraines often is severe and located behind the eye.[25] On examination, the affected eye will be noted to move laterally due to the unopposed action of the sixth nerve. In addition, ptosis, diplopia, and mydriasis may occur. Ophthalmoplegic migraines have been known to occur in babies younger than 1 year of age. During an attack, headache symptoms can last for hours, but the ophthalmoplegia can last days to weeks. Early, high-dose corticosteroid treatment is recommended to rapidly resolve symptoms of an acute episode and potentially to prevent permanent oculomotor nerve injury.[27]

Basilar artery (or Bickerstaff) migraines often present with a constellation of visual symptoms, including blindness, vertigo, ataxia, loss of consciousness, and drop attacks. Basilar artery migraines are the most common form of migraine equivalent to occur in children (3-19%) and occur more frequently in girls.[25,28] The initial symptoms of basilar artery migraine usually are visual and the episode usually is associated with an occipital headache.

Acute confusional migraines rarely occur in children. Difficult to diagnose, acute confusional migraines are characterized by restlessness, combative or hyperactive behavior, and occasional loss of consciousness. A headache usually precedes the symptoms, and the attacks can be triggered by mild head trauma.[25] Attacks usually begin when the child is 5-15 years of age. Males more often are affected than females. Symptoms of acute confusional migraine typically last 4-6 hours but can last for days.

Micropsia, metamorphosia, olfactory, auditory, or gustatory hallucinations and distortion of body image, spatial relations, and time sense characterize the Alice in Wonderland syndrome. The Alice in Wonderland syndrome sometimes is described as a migraine variant rather than as a complicated migraine. A headache may or may not occur with this syndrome, and most attacks are not associated with a headache.

Migraine Variants. Migraine variants are defined as the occurrence of episodic neurological complaints not associated with headache, in children who develop typical migraine headaches later on in life. Migraine variants are felt to be related to migraine by their periodicity, paroxysmal nature and the fact that children who have these symptoms often develop true migraine headaches. However, a family history of migraine may or may not be present. Difficult to diagnose, the relationship of migraine variants to migraine headache often is made after more typical migraine symptoms develop later in the child's life.

A number of distinct migraine variants have been recognized. These include benign paroxysmal vertigo, cyclic vomiting, abdominal migraine, and benign torticollis. A recent investigation into the occurrence of migraine variants in a pediatric neurology practice found that of 5848 patients seen during an eight-year period, 1106 had migraines and 108 (1.8% of total patients, 9.8% of patients with migraines) had a history of a migraine variant.[29] The most common migraine variant reported was benign paroxysmal vertigo, occurring in 38% of all patients with migraine variants.

Children with benign paroxysmal vertigo usually will present with monthly attacks of vertigo that decrease in frequency as the child grows older. Attacks often begin when the child is 2-6 years of age.[25] Symptoms generally last several minutes and the child will appear frightened, often grabbing a stationary object for stability. Nystagmus and ataxia commonly occur, and the child will complain of nausea and vomiting. Specific triggers, such as fatigue, often are identified. If attacks are frequent and disabling, diphenhydramine may be of benefit, though acute therapy usually is not needed due to the brief nature of the attacks.[30] Benign paroxysmal vertigo is the most common cause of vertigo in young children.[31] Attacks typically will resolve in 1-2 years and be supplanted by more typical migraine headaches.[32]

Cyclic vomiting usually occurs in children between the ages of 4-8 years. Episodes often occur monthly and are characterized by episodes of abdominal pain, nausea, and vomiting. Autonomic symptoms, including lethargy and pallor, also can occur.[33] Episodes will occur frequently and regularly. A headache may or may not be present during an attack. Between episodes, the child appears well.[33] In most children, the disorder will last several years and tends to resolve by late childhood or early adolescence.[33]

Similarly, abdominal migraine is characterized by episodes of crampy or colicky abdominal pain located in the periumbilical or epigastric areas. Attacks can last minutes to hours and can be accompanied by nausea and vomiting. In between attacks, the child appears well. Abdominal migraine often develops in children 4-10 years of age and typically resolves in 1-2 years, only to be replaced by more typical migraine headaches.

Because both cyclic vomiting and abdominal migraines share similar historical and clinical features and respond similarly to the blockade of serotonin receptors, they probably represent related conditions. Evaluation should focus on eliminating possible gastrointestinal or metabolic causes. Affected children can be treated with intravenous hydration and antiemetic medications such as ondansetron. Sumatriptan has been used to treat acute episodes of cyclical vomiting.[34] Effective prophylactic medications used in the prevention of abdominal migraine include propanolol and cyproheptadine.[35]

Table 3. Commonly Used Medications for the Treatment of Migraine Headaches in Children

MEDICATION	PEDIATRIC DOSE	ADULT DOSE
Dihydroergotamine	Not established	1-2 mg/dose or 6 mg/day IM or IV
Metoclopramide	0.1-0.2 mg/kg/dose IV	10-20 mg/dose IV
Chlorpromazine	> 6 months: 0.5-1 mg/g/dose PO 10 mg/m^2/dose IM, IV	 10-25 mg/dose PO 25-50 mg/dose IM, IV
Prochlorperazine (>2 y/o or 10 kg)	0.1 mg/kg/dose PO or PR	5-10 mg/dose IM 5-10 mg/dose PO 25 mg/dose PR
Promethazine	0.25-0.5 mg/kg/dose PO, IV, IM, or PR	12.5-25 mg/dose PO, IV, IM
Sumatriptan	Not established	5 mg/dose IN 6 mg/dose SQ 100 mg/dose PO
Acetaminophen	10-15 mg/kg/dose PO	325-650 mg/dose PO
Ibuprofen	10 mg/kg/dose PO	400-800 mg/dose PO
Naproxen	5-7 mg/kg/dose PO	250-500 mg/dose PO
Ketorolac	0.5 mg/kg/dose IM, IV Max 120 mg/day	60-90 mg/dose IV or IM 10 mg/dose PO

Benign torticollis is defined as recurring episodes of head tilt (torticollis) in a child younger than 1 year of age. Torticollis may be accompanied by pallor, headache, nausea, and vomiting, irritability, and drowsiness. The side of the torticollis often will vary during attacks. Extra-ocular movements are normal, and attacks are not related to feeding or changes in positioning. The condition is short-lived and resolves spontaneously, though rarely symptoms can persist for days. The child appears well between episodes. A family history of migraines or motion sickness often is found, and many infants with torticollis develop benign paroxysmal vertigo and more typical migraine headaches later in life.

Treatment

Many children who present to the ED with a headache or symptomatology consistent with migraine will require emergent treatment for their symptoms. Two treatment strategies should predominate during the ED visit—aborting the acute attack and preventing future attacks. While the physician often will concentrate on providing acute relief for the headache, preventing future attacks and recurrences also should be considered. Importantly, many patients who receive abortive treatment for a migraine headache will have recurrence of symptoms within 24-72 hours.

Acute Therapy. Effective treatment of migraine headaches often begins with reassuring the child and his or her family that the headaches are treatable and do not represent an underlying problem such as a brain tumor, and in appropriate situations ordering diagnostic testing to exclude more serious pathology. In addition, the clinician should explain to the parents that the headaches are not a psychological illness or a ploy on the part of the child to avoid school.

The symptoms that patients present with directly affect the types of therapies used to treat the migraine. Patients who are in severe pain should receive parenteral medications as therapeutic drug levels can be reached much more quickly intravenously compared to the oral route.

In general, prior to the use of any medication, the child should be placed in a dark, quiet environment. Ice packs can be applied to the head to help alleviate pain. Following a complete history and physical examination that includes an assessment of the patient's severity of pain and hydration status, a decision on the most appropriate treatment option can be made. Table 3 lists the medications most commonly used in the treatment of migraine headaches in children. A description of commonly used treatment modalities follows.

Sleep. Sleep often is effective in relieving most migraine attacks.[36] Investigators reported in a recent study of 133 children younger than 8 years of age who had presented to an outpatient setting with a migraine attack that 62% of episodes resolved following a period of sleep. However, having a child sleep in the ED as the sole treatment option may not be practical. Instead, sleep can be used as an adjunctive treatment to other treatment options.

Alternative Therapies. Relaxation techniques, self-hypnosis, and biofeedback all have been evaluated as treatment options for pediatric migraine.[36-42] All three treatment modalities have shown some success in relieving or preventing the symptoms of pediatric migraine. However, these therapies may not be practical as a treatment option in the ED setting.

Intravenous Fluids. Since gastric stasis often accompanies migraine headaches, and patients often are dehydrated due to the vomiting associated with their migraines, intravenous fluids should be given when parenteral medications are administered. Rehydration may help alleviate the nausea and vomiting associated with migraine headaches and may provide some pain relief.[43]

Acetaminophen. Acetaminophen has been shown to be highly effective for treating the pain, functional disability, photophobia

and phonophobia in adult patients with migraine.[43] In comparison to ibuprofen and placebo, acetaminophen has been shown to be superior to placebo and slightly less effective than ibuprofen in the treatment of migraine headaches in children.[44] A child who presents to the ED with a migraine headache, who has not been treated with over-the-counter medications prior to arrival, and who has milder symptoms can be treated with acetaminophen early during the course of his or her evaluation.

Non-steroidal Anti-inflammatory Drugs (NSAIDs). NSAIDs often are used in the initial treatment of children with migraine headaches. Many theories have been proposed as to the role of NSAIDs in this setting, but most investigators believe that they act via inhibition of prostaglandins and of platelet aggregation.

During the initial phase of migraine attacks, plasma serotonin levels increase, and as the attack progresses, decrease. Serotonin is carried almost exclusively by platelets. NSAIDs may treat or prevent migraine headaches by inhibiting platelet aggregability and thus decrease serotonin levels.[45] Many NSAIDs currently are available for use and no one oral NSAID has been shown to be superior to any other in the treatment of migraine headaches.[8]

Ibuprofen, at a dose of 7.5-10 mg/kg, has been shown to be effective in the treatment of migraine headaches in children.[44,46] Ketorolac has been found to be highly effective in the treatment of adult patients with migraine headaches.[47,48] However, ketorolac has not been well studied in the treatment of children with migraine headaches. Naproxen sodium has been found to reduce the severity and duration of headache and photophobia in adult patients with migraine headache without aura.[49] However, naproxen sodium also has not been extensively evaluated in the treatment of children with migraine headaches.

A child who presents to the ED with a migraine headache, who has not been treated with medications prior to arrival, and who has milder symptoms can be treated with an oral NSAID early in his or her evaluation. Intravenously or intramuscularly administered ketorolac can be used for patients with more severe symptoms or for those who cannot tolerate an oral medication.

Opiates. Opiates have not been found to be consistently effective in the treatment of migraine headaches. In addition, opioids are believed to be less effective as a pain reliever in a serotonin depleted state such as that found in migraine.[50] Therefore, opiates should be prescribed rarely, if at all, for the treatment of migraine headaches in children and should be considered a treatment of last resort.

Triptans. Increasingly, triptans are being used in the acute treatment of migraine headaches in children. Triptans act as 5HT-receptor agonists and their use results in vasoconstriction of intracranial arteries.

Sumatriptan is the most common triptan agent used and has been used in the treatment of pediatric migraine.[51] Sumatriptan is available in three forms; subcutaneous injectable, tablet, and nasal spray. Intranasally administered sumatriptan appears to have a more rapid onset of effect than the oral formulation and results in fewer adverse reactions than the subcutaneous form.[52]

Intranasal sumatriptan has been found to be efficacious in the treatment of migraine headaches in adolescents.[53-55] Nasal sumatriptan also has been shown to be effective in children 5-12 years of age.[56]

Subcutaneous sumatriptan has been shown to be effective in the treatment of migraine headaches in adolescents.[57,58] Oral sumatriptan, however, has not been found to be efficacious in the treatment of migraine headaches in children.

Side effects related to the use of sumatriptan include flushing, chest pain, and scalp burning. In addition, sumatriptan is relatively expensive and is contraindicated in those who are intolerant of its smooth muscle stimulating properties, such as those with a history of reactive airway disease.[59]

A number of new formulations of triptans recently have been approved for the treatment of migraine headaches. Several have been evaluated in the management of acute attacks. Eletriptan, zolmitriptan, rizatriptan, and almotriptan have been shown to be effective in the treatment of migraine in adults.[60-62] While few data pertaining to their use in children exist, both rizatriptan and zolmitriptan have been shown to be effective in the treatment of migraine headaches in adolescents.[63,64]

Triptans as a class of medication are considered contraindicated for the treatment of complicated migraines because of their vasoconstrictive properties. The neurological symptoms associated with these migraine subtypes are thought to occur due to vasoconstriction, and that by increasing vasoconstriction, triptans could increase the risk of brain infarction. However, investigators have not found that the use of triptans results in an increase in blood flow velocity in the basilar artery or middle cerebral artery, as would be seen if vasoconstriction of one of the arteries was occurring.[65,66] In addition, the safe use of triptans in the treatment of basilar migraine and migraine with prolonged aura has been reported.[67] Along these same lines, triptans should not be given concomitantly with ergotamines.

Dihydroergotamine/Ergotamines. Ergot derivatives, potent vasoconstrictors, commonly are used in the treatment of migraine headaches. Dihydroergotamine (DHE) is an ergot derivative that results in vasoconstriction of the external carotid arteries by directly affecting serotonin receptors and through alpha-adrenergic blockade.[68] Given parenterally in combination with an antiemetic, DHE has been found to be safe and effective in treating children with migraine headache.[69] The addition of an antiemetic such as metoclopramide, in combination with DHE, can help reduce the symptoms of nausea and vomiting associated with migraine headaches and can enhance the effect of DHE. A common side effect noted with DHE in children is a sense of terror on the part of the child and extreme agitation. These side effects usually resolve within five minutes following administration of the medication. DHE can be given intravenously, intramuscularly, subcutaneously, or intranasally. A sublingual preparation is available. DHE has been shown to be better tolerated than ergotamine.

Ergot derivatives are contraindicated in patients with peripheral vascular disease, hypertension, coronary artery disease, and

pregnancy. Ergot derivatives should not be used with the triptan class of medications, potentially compounding each medication's vasoconstrictive effect. In addition, ergot derivatives may exacerbate the gastrointestinal complaints associated with migraine. However, dihydroergotamine has less peripheral vasoconstrictive effects than most other ergot derivatives, and thus causes fewer side effects.

Phenothiazines. Phenothiazines are centrally acting dopamine antagonists that produce an antiemetic effect at the chemoreceptor trigger zone. Phenothiazines, such as chlorpromazine and prochlorperazine, have been used to treat migraine headaches. Both exert neuroleptic effects. Chlorpromazine has been shown to significantly improve symptoms in adults with migraine headache.[70] Chlorpromazine may act either by altering serotonin levels through inhibition of monoamine reuptake or by a direct effect at serotonergic receptors, or through its neuroleptic effects.[71]

Prochlorperazine also has been used in the treatment of children with migraine headaches. A prospective, randomized, double-blind, placebo controlled trial compared prochlorperazine to metoclopramide and placebo in the ED treatment of migraine headaches in adults.[59] Intravenous prochlorperazine was shown to be superior to both metoclopramide and placebo in relieving headache and decreasing symptoms of nausea. Recently, the use of prochlorperazine buccal tablets was compared to ergotamine tartarate and placebo in the acute treatment of adults with migraine headaches.[72] The buccal preparation produced faster improvement and greater efficacy than either placebo or ergotamine. Promethazine hydrochloride suppositories also have been shown to be effective in relieving the symptoms of migraine headache in children. Finally, prochlorperazine has been shown to be highly effective in the treatment of severe, intractable migraine in children.[73]

Metoclopramide. Metoclopramide is a dopamine antagonist that directly acts at the chemoreceptor trigger zone in the brain.[74] In addition, investigators have found that it may interact with the serotonergic system as well, specifically as an antagonist at the 5-HT3 receptor located in the trigeminovascular system.[68] Interestingly, metoclopramide is pharmacologically similar to phenothiazines and has been shown to increase the absorption of analgesics by promoting gastric motility.[75] Because of its actions, metoclopramide has been found to be an effective treatment in the management of migraine headaches.

Metoclopramide recently has been studied as a single agent in the treatment of adults with migraine headaches.[74] Ellis and colleagues evaluated metoclopramide alone and in combination with ibuprofen in a randomized, prospective, double-blinded, placebo-controlled study of the treatment of migraine headaches.[74] Metoclopramide was found to be highly effective when used alone in the treatment of migraine headache. Ibuprofen was not found to act additively or synergistically with metoclopramide.

More often, however, metoclopramide is used in conjunction with other pharmacological agents in the treatment of migraine headaches.[75] Metoclopramide can be given as an oral preparation but also can be given parenterally or rectally. As an intravenous preparation, metoclopramide can be paired with ketorolac in the acute treatment of migraine headaches in children. Metoclopramide also often is given with DHE to treat the nausea that frequently accompanies its use. The IM and PR preparations of metoclopramide also have been used for the treatment of migraine headaches.

Valproate. Antiepileptic medications have been used in the acute management of migraine headaches in both children and adults. Intravenous valproate has been shown to be highly effective in the acute management of migraine headaches in adolescents and has been shown to be highly effective in adult patients as well.[76,77] In an open-label, randomized trial, intravenous valproate compared favorably to intramuscular dihydroergotmine, used in combination with metoclopramide, for the acute treatment of migraine headache in adults.[78] However, headache relief was not as likely to be sustained at 24 hours in those patients treated with valproate.

Lidocaine. Lidocaine long has been known for its anesthetic properties and has been used most commonly for local and regional anesthesia. Recently, lidocaine has been used in the acute management of migraine headaches. In a recently conducted randomized, double-blind, controlled trial of intranasal lidocaine used in the management of adult patients with migraine headache, 55% of patients treated with intranasal lidocaine had relief of their headaches following its use.[79] Relapse of headache, however, occurred early and often following treatment. One possible mechanism by which intranasal lidocaine is thought to relieve the headache of migraine is through its effects on the sphenopalatine ganglion. However, the exact mechanism is unclear. More recently, a similarly conducted study has reported that intranasal lidocaine may not nearly be as effective.[80] In a randomized, double-blind, placebo-controlled clinical trial, no difference was found in the number of patients who experienced pain relief when treated with intranasal lidocaine or when treated with placebo. Intranasal lidocaine has not been evaluated in the treatment of children with migraine headaches and its use cannot be advocated at this time.

Corticosteroids. Many patients who present with an acute migraine headache successfully can be treated with one of the many available therapeutic options. However, some patients may be much more difficult to treat and may in fact present with severe, intractable headaches (status migrainosus). Such patients may benefit from treatment with parenterally administered corticosteroids. In addition, corticosteroid treatment has been shown to decrease the duration of ophthalmoplegia and decrease pain in patients with ophthalmoplegic migraine.[25] Corticosteroids also have been used in the treatment of more typical migraine headaches. However, in general, steroid therapy should be reserved for those patients with more severe and intractable headaches.

Preventive Therapy. While most clinicians caring for a child with a migraine headache will focus on the immediate relief of symptoms, the clinician also should consider the use of preventive therapy for the child with frequent and severe headaches once the

acute episode has been managed. In addition, many patients who receive treatment in the ED for an acute headache, and have relief of symptoms, will return to the ED within the next 48 hours with recurrence of symptoms. Therefore, preventive therapy, including not only the use of pharmacologic agents but also a complete assessment of the patient's lifestyle and possible headache triggers, can help prevent patients from having recurrence of symptoms and can lead to an improvement in their daily lives.

Preventive therapy should begin with an assessment of the patient's headache frequency and severity. While this may not be practical in an ED, headache calendars can be provided to families to document the frequency, severity, and associated symptoms of their child's headaches and to identify potential triggers, environmental factors, and life stressors. The elimination of potential triggers should be the primary goal of migraine prophylaxis.

Once the physician has begun an investigation into the potential triggers of the child's migraine headaches, attention can be turned to preventing future episodes. While avoiding potential triggers should be the ultimate goal of preventive therapy, this may not be practical, especially after an initial attack.

Both pharmacologic and nonpharmacologic preventive measures have been described in migraine prophylaxis in children. Nonpharmacologic preventive measures have been shown to reduce the frequency of migraine headaches in adults and may play a role in the management of migraine headaches in children. However, these measures have not been well studied in children.

Pharmacological agents used in migraine prophylaxis should be started only in collaboration with the child's primary care physician. Migraine prophylactic medications should be considered when children are having more than two headaches per month that are treated inadequately with standard migraine medications, or if children are suffering from attacks that are so severe that their quality of life is impacted. Medications that have been used in migraine prophylaxis include beta blockers such as propranolol, calcium channel blockers, serotonin receptor antagonists, antidepressants such as tricyclic antidepressants, antiepileptics, NSAID agents, cyproheptadine, and sodium valproate.

When a decision has been made to begin prophylactic medications, medications should be started at a low dose and increased slowly. New medications should be tried for 1-2 months and tapered and discontinued once headaches are well controlled.

Beta-blockers are the most common medications used in migraine prophylaxis. Beta-blockers are thought to prevent migraine headaches by antagonism of serotonin receptors or through modulation of adrenoreceptors.[81] Investigations that have evaluated the use of beta-blockers in the prevention of migraine headaches in children, however, have not shown consistent results, with some studies even suggesting that beta-blockers were no more effective than placebo in the prevention of migraine headaches.[12,82] Therefore, it is not clear if beta-blockers should be routinely used for migraine prophylaxis in children. In addition, beta-blockers should not be used when patients have a history of reactive airway disease, diabetes mellitus, orthostatic hypotension, or cardiac disorders associated with bradyarrhythmias.

Both tricyclic antidepressants and selective serotonin reuptake inhibitors have been used for migraine prophylaxis in children. The antimigraine effects of tricyclic antidepressants seem to be independent of their antidepressant effects. No controlled studies of the use of these agents in children or adolescents have been reported.

Cyproheptadine is an antihistamine with both antiserotonergic and calcium channel blocking properties. Cyproheptadine has been used widely for migraine prophylaxis in children. While safe and efficacious in young children, the medication is not well tolerated in adolescents. Cyproheptadine has been compared to placebo, propanolol, and a combination of cyproheptadine and propanolol in the prophylactic treatment of migraine headaches in children.[83] While cyproheptadine reduced the frequency, duration and severity of migraine headaches as compared to placebo, the combination of cyproheptadine and propanolol provided the greatest relief. Cyproheptadine has a sedative effect in children and is also an appetite stimulant.

Sodium valproate long has been used as a prophylactic agent for adults with migraine headaches. However, pediatric studies have been limited. A recent study in children with migraines, however, suggests that sodium valproate may be highly effective and safe as a preventative medication.[84] Topiramate, a broad-spectrum antiepileptic drug used for the treatment of multiple seizure types, also has been shown to be highly effective when used as a prophylactic agent for migraine headaches.[85]

Therapeutic Approach to the Child with a Migraine Headache

When a child presents to the ED with a headache, the first objective for the clinician is to determine if the headache is a symptom of a serious underlying disorder. Once the clinician determines that the headache does not herald a serious problem and decides that the headache is in fact a migraine, attention should be turned to treatment strategies.

The management of migraine headache in children should be individualized to the patient, taking into account the frequency and severity of attacks, the presence and degree of disability and the presence of associated symptoms such as nausea and vomiting. The patient's history of, response to, and tolerance for specific medications also should be considered.

A reasonable first-line approach in a child who presents with a migraine headache, who is not vomiting and is able to tolerate oral medications, will be to treat first with ibuprofen. If treatment is unsuccessful, parenteral medications often will be given, in particular a combination of metoclopramide and ketorolac. If the child with a migraine headache presents with significant vomiting or cannot tolerate oral medications, parenteral medications will be given first. Again, first-line treatment consists of a combination of intravenous metoclopramide and ketorolac along with intravenous fluids.

If first-line parenteral therapy fails, and the child does not have a complex migraine, DHE would be the next agent of choice and the child would be admitted to the hospital for further treatment and observation. It is at this time that the neurology subspecialist often is consulted.

A recent trend is to use intravenous valproate when first-line therapy fails. Again, if a decision is made to use valproate, the patient will be admitted to the hospital, and the neurology subspecialists will be consulted.

Regardless of the treatment used to alleviate the migraine headache attack in the ED, patients who are discharged from the hospital will often be started on naproxen sodium as both prophylactic and acute therapy. In addition, parents will be encouraged to keep a headache diary to identify potential triggers of migraine attacks.

Summary

Migraine headaches are a common problem of childhood and are associated with significant morbidity, school absence, and lifestyle disruption. However, migraine headaches often are under recognized in the pediatric population and often are undertreated.

Migraine symptoms in children can vary dramatically in their character and severity. Diagnosis primarily is based on the patient's history and a thorough physical examination. Laboratory and radiographic testing rarely, if ever, are required. Effective medications are available for the acute and prophylactic treatment of migraine headaches in children. However, most medications effectively have been studied only in adult patients. Currently, there are very few well controlled trials of migraine medications in children.

References

1. Forsyth R, Farrell K. Headache in childhood. *Pediatr Rev* 1999;20:39.
2. Winner PK. Headaches in children. When is a complete diagnostic workup indicated? *Postgrad Med* 1997;101:81.
3. May A, Ophoff RA, Terwindt GM, et al. Familial hemiplegic migraine locus on 19p13 is involved in the common forms of migraine with and without aura. *Hum Genet* 1995;96:604-608.
4. Wober-Bingol C, Wober C, Wagner-Ennsgraber C, et al. HIS criteria for migraine and tension-type headache in children and adolescents. *Headache* 1996;36:231-238.
5. Lipton RB, Stewart WF, Diamond S, et al. Prevalence and burden of migraine in the United States: Data from the American Migraine Study II. *Headache* 2001;41:646-657.
6. Hernandez-Latorre MA, Roig M. Natural history of migraine in childhood. *Cephalalgia* 2000;20:573-579.
7. Raskin NH. Migraine: Clinical aspects. In: *Headache.* 2nd ed. New York: Churchill Livingstone; 1988:41-98.
8. Bulloch B, Tenenbein M. Emergency department management of pediatric migraine. *Pediatr Emerg Care* 2000;16:196-204.
9. Moskowitz M. The visceral organ brain: Implications for the pathophysiology of vascular head pain. *Neurology* 1991;41:182.
10. Welch KMA, Barkley GL, Tepley N, et al. Central neurogenic mechanisms of migraine. *Neurology* 1993;43:S21.
11. Solomon GD. The pharmacology of medicines used in treating headaches. *Semin Pediatr Neurol* 1995;2:165.
12. Lewis DW, Scott D, Rendin V. Treatment of pediatric headaches. *Expert Opin Pharmacother* 2002;3:1433-1442.
13. Cano A, Palomeras E, Alfonso S, et al. Migraine without aura and migrainous disorder in children: International Headache Society (IHS) and revised IHS criteria. *Cephalalgia* 2000;20:617-620.
14. Barabas G, Ferrari M, Matthews WS. Childhood migraine and motion sickness. *Pediatrics* 1983;33:188.
15. Barabas G, Ferrari M, Matthews WS. Childhood migraine and somnambulism. *Neurology* 1983;33:948.
16. Raskin NH, Knittle SC. Ice cream headache and orthostatic symptoms in patients with migraine. *Headache* 1976;16:222-225.
17. Jan MMS and Al-Buhairi AR. Is infantile colic a migraine-related phenomenon? *Clin Pediatr* 2001;40:295-297.
18. Bruni O, Fabrizi P, Ottaviano S, et al. Prevalence of sleep disorders in childhood and adolescence with headache: A case-control study. *Cephalalgia* 1997;17:492-498.
19. Katerji MA, Painter MJ. Infantile migraine presenting as colic. *J Child Neurol* 1994;9:336-337.
20. Lewis DW, Ashwal S, Dahl G, et al. Practice parameter: Evaluation of children and adolescents with recurrent headaches. Report of the Quality Standards Subcomittee of the American Academy of Neurology and the Practice Committee of the Child Neurology Society. *Neurology* 2002;59:490-498.
21. Honig PJ, Charney EB. Children with brain tumor headaches: Distinguishing features. *Am J Dis Child* 1982;136:121-124.
22. Lewis DW, Dorbad D. The utility of neuroimaging in the evaluation of children with migraine or chronic daily headache who have normal neurological examinations. *Headache* 2000;40:629-632.
23. Maytal J, Bienkowski RS, Patel M, et al. The value of brain imaging in children with headaches. *Pediatrics* 1995;96:413-416.
24. Chu ML, Shinnar S. Headaches in children under 7 years of age. *Ann Neurol* 1990;28:433.
25. Rothner AD. Complicated migraine and migraine variants. *Semin Pediatr Neurol* 2001;8:7-12.
26. Lewis DW. Migraine and migraine variants in children and adolescents. *Semin Pediatr Neurol* 1995;2:127.
27. Carlow TJ. Oculomotor ophthalmoplegic migraine: Is it really migraine? *J Neuro-Ophthalmol* 2002;22:215-221.
28. Singer HS. Migraine headaches in children. *Pediatr Rev* 1994;15:94.
29. Al-Twaijri W, Shevell MI. Pediatric migraine equivalents: Occurrence and clinical features in practice. *Pediatr Neurol* 2002;26:365-368.
30. Fenichel GM. Migraine as a cause of benign paroxysmal vertigo of childhood. *J Pediatr* 1967;71:114-115.
31. Shevell M. A guide to migraine equivalents. *Contemp Pediatr* 1998;15:71.
32. Lanzi G, Balottin U, Fazzi E, et al. Benign paroxysmal vertigo childhood: A long-term follow-up. *Cephalgia* 1994;6:458.
33. Li BUK. Cyclic vomiting syndrome: Age-old syndrome and new insights. *Semin Pediatr Neurol* 2001;8:13-21.
34. Benson J, Zorn S, Book L. Sumatriptan (Imitrex) in the treatment of cyclic vomiting. *Ann Pharmacol* 1995;29:997-998.
35. Russell G, Abu-Arafeh I, Symon DN. Abdominal migraine: Evidence for existence and treatment options. *Paediatric Drugs* 2002;4:1-8.
36. Aaltonen K, Hmalainen ML, Hoppu K. Migraine attacks and sleep in children. *Cephalalgia* 2000;20:580-584.
37. Holden EW, Levy JD, Deichmann MM, et al. Recurrent pediatric headaches: Assessment and intervention. *J Develop Behavior Pediatr* 1998;19:109.
38. McGrath PJ, Humphreyes P, Goodman JT, et al. Relaxation prophylaxis for childhood migraine: A randomized placebo-controlled trial. *Dev Med Child Neurol* 1988;30:626.
39. McGrath PJ, Humphreys P, Keene D, et al. The efficacy and efficiency of a self-administered treatment for adolescent migraine. *Pain* 1992;49:321.
40. Hermann C, Blanchard EB, Flor H. Biofeedback treatment for pediatric migraine: Prediction of treatment outcome. *J Consult Clin Psychol* 1997; 65:611.
41. Allen KD, McKeen LR. Home-based multicomponent treatment of pediatric migraine. *Headache* 1991;31:467.
42. Baumann RJ. Behavioral treatment of migraine in children and adolescents. *Paediatric Drugs* 2002;4:555-561.
43. Lipton RB, Baggish JS, Stewart WF, et al. Efficacy and safety of acetaminophen in the treatment of migraine: Results of a randomized, double-blind, placebo-controlled, population-based study. *Arch Intern Med* 2000;160: 3486-3492.
44. Hamalainen ML, Hoppu K, Valkeila E, et al. Ibuprofen or acetaminophen for the acute treatment of migraine in children: A double-blind, randomized, placebo-controlled, crossover study. *Neurol* 1997;48:102-107.
45. Diamond S, Freitag FG. Do non-steroidal anti-inflammatory agents have a

role in the treatment of migraine headaches? *Drugs* 1989;37:755.
46. Lewis DW, Kellstein D, Burke B, et al. Children's ibuprofen suspension for the acute treatment of pediatric migraine. *Headache* 2002;42:780-786.
47. Shrestha M, Singh R, Moreden J, et al. Ketorolac vs chlorpromazine in the treatment of acute migraine without aura. A prospective, randomized, double-blind trial. *Arch Int Med* 1996;156:1725.
48. Davis CP, Torre P, Schafer N, et al. Ketorolac as a rapid and effective treatment of migraine headache: Evaluation by patients. *Am J Emerg Med* 1993;11:573.
49. Johnson ES, Ratcliffe DM, Wilkinson M. Naproxen sodium in the treatment of migraine. *Cephalgia* 1985;5:5.
50. Edmeads JG. Migraine. *CMAJ* 1988;138:107-113.
51. Ferrari MD. Sumatriptan in the treatment of migraine. *Neurology* 1993;43:S43.
52. Fuseau E, Petricoul O, Moore KH, et al. Clinical pharmacokinetics of intranasal sumatriptan. *Clin Pharmacokinetics* 2002;41:801-811.
53. Ueberall M. Sumatriptan in paediatric and adolescent migraine. *Cephalalgia* 2001;21(Suppl 1):21-24.
54. Ueberall MA. Intranasal sumatriptan for the acute treatment of migraine in children. *Neurology* 1999;52:1507-1510.
55. Winner PW, Rothner AD, Saper J, et al. A randomized, double-blind, placebo-controlled study of sumatriptan nasal spray in the treatment of acute migraine in adolescents. *Pediatrics* 2000;106:989-997.
56. Hershey AD, Powers SW, LeCates S, et al. Effectiveness of nasal sumatriptan in 5- to 12-year-old children. *Headache* 2001;41:693-697.
57. Macdonald JT. Treatment of juvenile migraine with subcutaneous sumatriptan. *Headache* 1994;34:581-582.
58. Linder S. Subcutaneous sumatriptan in the clinical setting: The first 50 consecutive patients with acute migraine in a pediatric neurology office practice. *Headache* 1996;36:419.
59. Coppola M, Yealy DM, Leibold RA. Randomized, placebo-controlled evaluation of prochlorperazine versus metoclopramide for emergency department treatment of migraine headache. *Ann Emerg Med* 1995;26:541.
60. Diener HC, Jansen JP, Reches A, et al. Efficacy, tolerability and safety of oral eletriptan and ergotamine plus caffeine (Caffergot) in the acute treatment of migraine: A multicentre, randomized, double-blind, placebo-controlled comparison. *Euro Neurol* 2002;47:99-107.
61. Geraud G, Compagnon A, Rossi A. The COZAM Study Group. Zolmitriptan versus a combination of acetylsalicylic acid and metoclopramide in the acute oral treatment of migraine: A double-blind, randomized, three attack study. *Euro Neurol* 2002;47:88-98.
62. Mathew NT. Use of rescue medication in trials of almotriptan versus placebo in the treatment of acute migraine. *Clin Therapeutics* 2002;24:520-529.
63. Winner P, Lewis D, Visser WH, et al. Rizatriptan 5 mg for the acute treatment of migraine in adolescents: A randomized, double-blind placebo-controlled study. *Headache* 2002;42:49-55.
64. Linder SL, Dowson AJ. Zolmitriptan provides effective migraine relief in adolescents. *Int J Clin Pract* 2000;54:466-469.
65. Totaro R, De Mattheis G, Marini C, et al. Sumatriptan and cerebral blood flow velocity changes during migraine attacks. *Headache* 1997;37:635-639.
66. Limmroth V, May A, Auerbach P, et al. Changes in cerebral blood flow velocity after treatment with sumatriptan or placebo and implications for the pathophysiology of migraine. *J Neurol Sci* 1996;138:60-65.
67. Klapper J, Mathew N, Nett R. Triptans in the treatment of basilar migraine and migraine with prolonged aura. *Headache* 2001;41:981-984.
68. Graf WD, Phillip SR. Pharmacologic treatment of recurrent pediatric headache. *Pediatr Ann* 1995;24:477.
69. Linder S. Treatment of childhood headache with Dihydroergotamine mesylate. *Headache* 1994;34:578.
70. Bell R, Montoya D, Shuaib A, et al. A comparative trial of three agents in the treatment of acute migraine headache. *Ann Emerg Med* 1990;19:1079.
71. Lane PL, McClellan BY, Baggoley CJ. Comparative efficacy of chlorpromazine and meperidine with dimenhydrinate in migraine headache. *Ann Emerg Med* 1989;18:360.
72. Sharma S, Prasad A, Nehru R, et al. Efficacy and tolerability of prochlorperazine buccal tablets in treatment of acute migraine. *Headache* 2002;42: 896-902.
73. Kabbouche MA, Vockell AL, LeCates SL, et al. Tolerability and effectiveness of prochlorperazine for intractable migraine in children. *Pediatrics* 2001; 107:E62.
74. Ellis GL, Delaney J, DeHart DA, et al. The efficacy of metoclopramide in the treatment of migraine headache. *Ann Emerg Med* 1993;22:191.
75. Volans GN. Research review migraine and drug absorption. *Clin Pharmacokinet* 1978;3:313.
76. Stillman MJ, Zajac D, Rybicki LA. The acute treatment of headaches with intravenous valproate. *Neurology* 2001;56(suppl. 3):A392.
77. Mathew NT, Kailasam J, Meadors L, et al. Intravenous valproate sodium (depacon) aborts migraine rapidly: A preliminary report. *Headache* 2000; 40:720-723.
78. Edwards KR, Norton J, Behnke M. Comparison of intravenous valproate versus intramuscular Dihydroergotamine and metoclopramide for acute treatment of migraine headache. *Headache* 2001;41:976-980.
79. Maizels M. Scott B, Cohen W, et al. Intranasal lidocaine for treatment of migraine. A randomized, double-blind, controlled trial. *JAMA* 1996;276:319.
80. Blanda M, Rench T, Gerson LW, et al. Intranasal lidocaine for the treatment of migraine headache: A randomized, controlled trial. *Acad Emerg Med* 2001; 8:337-342.
81. Wasiewski WW. Preventive therapy in pediatric migraine. *J Child Neurol* 2001;16:71-78.
82. Solomon GD. Pharmacology and use of headache medications. *Cleve Clin J Med* 1990;57:627-635.
83. Rao BS, Das DG, Taraknath VR, et al. A double-blind controlled study of propranolol and cyproheptadine in migraine prophylaxis. *Neurol India* 2000;48: 223-226.
84. Serdaroglu G, Erhan E, Tekgul H, et al. Sodium valproate prophylaxis in childhood migraine. *Headache* 2002;42:819-822.
85. Storey JR, Calder CS, Hart DE, et al. Topiramate in migraine prevention: A double-blind, placebo-controlled study. *Headache* 2001;41:968-975.

Physician CME Questions

101. Migraine without aura is more common in children than migraine with aura.
    A. True
    B. False

102. Corticosteroids can be an effective treatment for which migraine equivalent?
    A. Hemiplegic migraine
    B. Basilar artery migraine
    C. Ophthalmoplegic migraine
    D. Alice in Wonderland syndrome

103. Which of the following behaviors has/have been associated with migraine headaches in children?
    A. Sleepwalking
    B. Sleep talking

CME Objectives

The CME objectives for *Pediatric Emergency Medicine Reports* are to help physicians:

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

b.) Understand the epidemiology, etiology, pathophysiology, historical and physical examination findings associated with the entity discussed;

c.) Be educated about how to correctly formulate a differential diagnosis and perform necessary diagnostic tests;

d.) Apply state-of-the-art therapeutic techniques (including the implications of pharmacologic therapy discussed) to patients with the particular medical problems discussed;

e.) Provide patients with any necessary discharge instructions.

C. Motion sickness
D. All of the above

104. What is the most common migraine variant to occur in children?
A. Benign paroxysmal vertigo
B. Cyclic vomiting
C. Abdominal migraine
D. Benign torticollis

105. Infantile colic has been associated with migraine headache in children.
A. True
B. False

106. Metoclopramide, while effective as single agent in the management of migraine headaches, has been used effectively in combination with what other medication?
A. Naprosyn
B. Sumatriptan
C. Ketorolac
D. Acetaminophen

107. Ergotamines and triptans safely can be used as combination therapy in the treatment of children with migraine headaches.
A. True
B. False

108. Migraine headache is considered to be a hereditary disorder.
A. True
B. False

109. A history of migraine headaches often is documented in at least one parent less than 20% of the time.
A. True
B. False

110. Which of the following may be a trigger of a migraine headache?
A. Caffeine
B. Stress
C. Fatigue
D. Hunger
E. All of the above

Answer Key: 101. **A**; 102. **C**; 103. **D**; 104. **A**; 105. **A**; 106. **C**; 107. **B**; 108. **A**; 109. **B**; 110. **E**

Note to Readers

The October 2003 issue of *Pediatric Emergency Medicine Reports* incorrectly stated the issue date as November 2003. The volume and issue number were correct; only the date on the front cover masthead was incorrect. We apologize for the error.

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PEDIATRIC Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Pediatric Migraines

IHS Criteria for the Diagnosis of Migraine with and without Aura in Children

MIGRAINE WITH AURA

1. Have at least two attacks fulfilling Criteria No. 2.
2. Each attack has at least three of the following four characteristics:
   a. Fully reversible aura symptoms indicating focal cerebral cortical and/or brainstem dysfunction
   b. More than one aura symptom that develops gradually in more than four minutes or has more than two symptoms that occur in succession
   c. No aura symptom lasting more than 60 minutes. If more than one aura symptom is present, accepted duration is proportionally increased.
   d. The headache follows aura symptoms with a free interval of fewer than 60 minutes, but may begin before or simultaneously with the aura.

MIGRAINE WITHOUT AURA

1. Have at least five attacks fulfilling criteria #2-4
2. Headache attacks lasting 4-72 hours (2-48 hours in patients younger than15 years)
3. The headache has at least two of the following characteristics:
   a. Unilateral location
   b. Pulsing quality
   c. Moderate to severe intensity
   d. Aggravation by routine physical activities
4. During an attack, the headache is accompanied by one of the following symptoms:
   a. Nausea and/or vomiting
   b. Photophobia and/or phonophobia

Proposed Modified IHS Criteria for the Diagnosis of Migraine without Aura in Children

MIGRAINE WITHOUT AURA

1. Have at least five attacks fulfilling Criteria Nos. 2-4.
2. Headache attacks lasting 1-48 hours
3. During an attack, the headache has at least two of the following characteristics:
   a. Bilateral or unilateral location
   b. Pulsating quality
   c. Moderate to severe intensity
   d. Aggravation by routine physical activities
4. During an attack, the headache is accompanied by one of the following symptoms:
   a. Nausea and/or vomiting
   b. Photophobia and/or phonophobia

Commonly Used Medications for the Treatment of Migraine Headaches in Children

MEDICATION	PEDIATRIC DOSE	ADULT DOSE
Dihydroergotamine	Not established	1-2 mg/dose or 6 mg/day IM or IV
Metoclopramide	0.1-0.2 mg/kg/dose IV	10-20 mg/dose IV
Chlorpromazine	> 6 months: 0.5-1 mg/g/dose PO 10 mg/m^2/dose IM, IV	 10-25 mg/dose PO 25-50 mg/dose IM, IV
Prochlorperazine (>2 y/o or 10 kg)	0.1 mg/kg/dose PO or PR	5-10 mg/dose IM 5-10 mg/dose PO 25 mg/dose PR
Promethazine	0.25-0.5 mg/kg/dose PO, IV, IM, or PR	12.5-25 mg/dose
Sumatriptan	Not established	5 mg/dose IN 6 mg/dose SQ 100 mg/dose PO
Acetaminophen	10-15 mg/kg/dose PO	325-650 mg/dose PO
Ibuprofen	10 mg/kg/dose PO	400-800 mg/dose PO
Naproxen	5-7 mg/kg/dose PO	250-500 mg/dose PO
Ketorolac	0.5 mg/kg/dose IM, IV Max 120 mg/day	60-90 mg/dose IV or IM 10 mg/dose PO

Supplement to *Pediatric Emergency Medicine Reports*, November 2003: "Pediatric Migraine: Recognizing and Managing Big Headaches in Small Patients." *Author:* **Raymond D. Pitetti, MD**, Assistant Professor, Division of Pediatric Emergency Medicine, Department of Pediatrics, Children's Hospital of Pittsburgh and the University of Pittsburgh School of Medicine, Pittsburgh, PA. *Peer reviewer:* **Ronald M. Perkin, MD, MA,** Professor and Chairman, Department of Pediatrics, The Brody School of Medicine, East Carolina University, Greenville, NC.

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Evidence-Based Medicine for the ED

Trauma Reports®

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Nov./Dec. 2003

Emergency department thoracotomy (EDT) is the most invasive and dramatic procedure that can be performed in the resuscitation of a trauma patient. The increased availability of rapid pre-hospital assessment and transportation of trauma patients has allowed patients who would never have survived in the past to be transported to the ED. ED physicians and trauma surgeons then are placed in the critical position of determining the etiology of the arrest, reversing any correctable processes, and deciding if an EDT is indicated. Lack of oxygen to the brain longer than 4-10 minutes does not bode a meaningful outcome. Therefore, the ED physician and trauma surgeon must have evidence-based information on indications for EDT that can be determined rapidly, easily accessible equipment, and the ability to recognize situations in which EDT clearly is not in the patient's best interest.

—The Editor

ED Thoracotomy Revisited: A Complete Reassessment of its Past, Present, and Future

Authors: **Victor V. Dizon, DO,** Grant Medical Center Trauma Fellow, 2001-2002, Columbus, OH; **Steven A. Santanello, DO,** Grant Medical Center Trauma Program Director, Columbus, OH.

Peer Reviewer: **Corey M. Slovis, MD, FACP, FACEP,** Professor of Emergency Medicine and Medicine, Department of Emergency Medicine, Vanderbilt University School of Medicine, Nashville, TN; Medical Director, Metro Nashville Fire Department, Nashville, TN.

Introduction

The EDT remains one of the most dramatic tools in the trauma surgeon's armamentarium. This technique has been practiced for years, although controversy has surrounded its use. As medicine has evolved, the indications for EDT have become more sophisticated. Settings where it has been used vary, and include penetrating thoracic and thoracoabdominal trauma.

The literature also reports its use in patients presenting in cardiopulmonary arrest secondary to isolated blunt trauma. Increasingly, medicine is required to answer many complicated questions regarding utility, ethics, and cost/risk-to-benefits ratios. Should we be performing a costly procedure that has a low rate of success? What is the benefit in saving a patient who survives with severe neurologic impairment, and what financial burden does that place on society?

Finally, does the diminutive survival benefit of such a procedure outweigh the potential for injury or transmission of disease to those performing and assisting in EDT? To completely understand the evolution of the EDT and improve our vision of its place in the future, it is necessary to identify the many historical events that shaped medicine and our world, making this procedure possible.

Historical Perspective

By the turn of the 20th century, America had established itself as a world power. The West was won. The frontier was no more. The continent was settled from coast to coast.

This was a time when J.P. Holland invented the first torpedo boat, elevating the United States Navy into a world-wide maritime force. King Camp Gillette developed the double-edged safety razor. The nation recovered from the assassination of its 25th president, William McKinley, and embraced Theodore Roosevelt as its new leader. Inventions revolutionized home travel, as well. Henry Ford introduced the Model T to the world, and the Wright brothers astonished us with the first powered, manned flight. With all that was going on in the world, medicine, too, was evolving.

In terms of chest surgery, four notable physicians demonstrated the feasibility of chest exploration for the treatment of injury. In 1874, Dr. Schiff first suggested open cardiac massage as a resuscitative measure for chloroform-induced cardiac arrest.[1] Then, in 1882, Dr. Block demonstrated the reality of opening the chest to repair cardiac injury in his canine experiments involving heart lacerations.[1-3] However, it wasn't until 1889 when the first successful open cardiac resuscitation was performed by Tuffier.[1] Dr. Rehn followed suit with the successful repair of a penetrating right ventricular injury in a human.[2,4] One year later, Dr. Ingelsrod successfully revived a post-injury cardiac arrest patient using open cardiac massage.[1] Claude Beck popularized open cardiac massage, and for the next 50-60 years, this became the standard of care for cardiac arrest in the operating room.[1,5] In 1947, he ultimately established the precedent of electrical defibrillation in the operating room and boasted a 29% survival rate for open cardiac massage on 1200 patients.[5] During the following years, exploration of the chest became a more common practice. Shortly thereafter, this practice fell out of vogue.[1]

Several key events gave rise the EDT's near elimination. In 1943, Drs. Alfred Blalock and Michael M. Ravitch (more well known for their contributions to pediatric surgery) perfected the technique of pericardiocentesis and advocated its use for the treatment of pericardial tamponade.[6] A decade later, Michael Zoll demonstrated the practicality of external defibrillation for life-threatening arrhythmias.[1,7] In the 1960s, Drs. Kownhoven, Jude, and Knickerbocker introduced closed-chest massage.[1] These new concepts and techniques shifted the medical tide away from the use of the EDT.

However, while history was staging itself for the near elimination of the EDT, other concepts in chest trauma were being discovered as a result of World War II. Heart-lung machines pioneered by Dr. John Gibbons allowed surgeons like Dr. Michael DeBakey of Baylor University in Houston to refine cardiothoracic techniques. Occlusion of the thoracic aorta now was possible in patients exsanguinating from abdominal trauma. Ultimately, this led to the revival of EDT.

Rationale for Use of the ED Thoracotomy

With refined cardiothoracic techniques and the ability to cross-clamp the aorta, the EDT became more commonplace for patients in extremis with traumatic chest and/or abdominal injury. Since reversal of underlying causes of trauma arrest, which consists of hypovolemia, rapid hemorrhage or pericardial tamponade, is critical to patient survival, EDT is a valuable adjunct to a readily available surgical staff and definitive surgical repair. Guidelines were identified and more clearly defined to dictate the appropriateness of its use. The term "no signs of life," defined as no detectable blood pressure, papillary reactivity, respiratory effort, or cardiac electrical activity, clearly became a contraindication for EDT. However, physicians caring for patients with evidence of signs of life despite no vital signs still could make a valid argument for EDT.

Clearly, the decision to undertake such a formidable task should be based on scientific information directed toward

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The intended audience for this publication is emergency, family, osteopathic, and general practice physicians and nurses who have contact with trauma patients.

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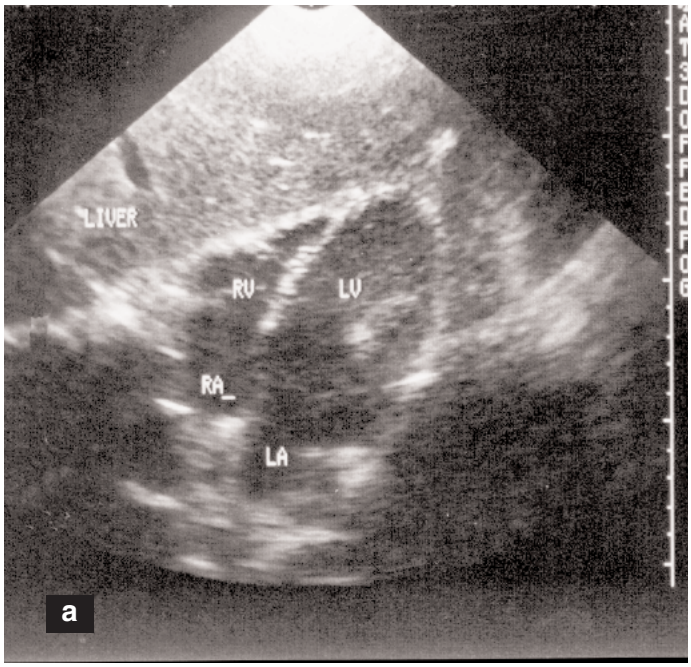
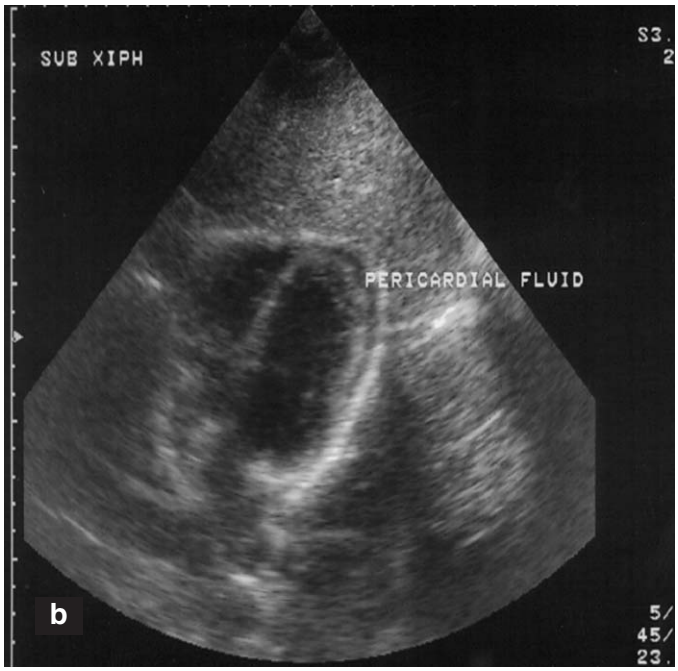
Figures 1a and 1b. The Pericardial Window





1a. A normal pericardial window.
RV = right ventricle; LV = left ventricle; RA = right atrium; and LA = left atrium.

1b. Pericardial window that shows blood separating the visceral and parietal layers of the pericardium.

identifying and temporarily stabilizing specific correctible injuries. There are five basic motives for performing an EDT: 1) to release pericardial tamponade; 2) to control intra-thoracic vascular and/or cardiac bleeding; 3) to control massive air embolism or bronchopleural fistula; 4) to permit open cardiac massage; and 5) to provide temporary occlusion of the descending thoracic aorta to diminish intra-abdominal hemorrhage and optimize blood flow to the brain and heart.[1,3]

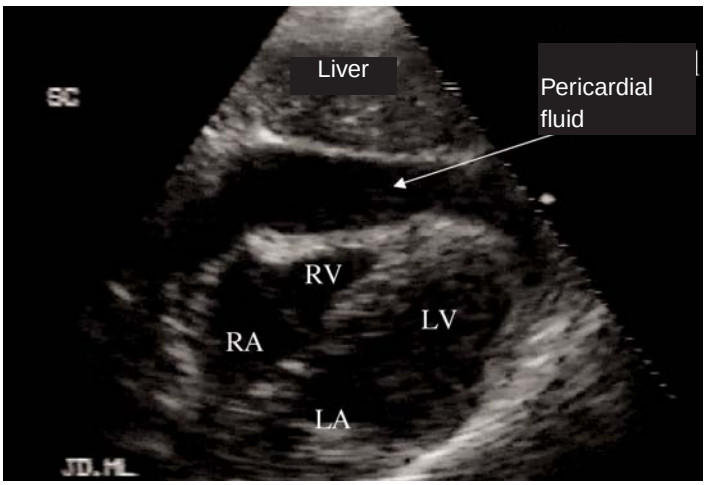
Pericardial Tamponade. Pericardial tamponade may result from gunshot wounds or stab wounds. Stab wounds commonly cause pericardial tamponade (80% of cases).[8] Pericardial tamponade can be characterized by Beck's triad (hypotension, distended neck veins, and muffled heart tones).[9] However, this triad has been demonstrated to have low specificity and sensitivity. More commonly, pericardial tamponade presents as a subtle constellation of symptoms with gradual progression of diminishing cardiac function. Often in trauma, the patient decompensates before the diagnosis is firmly established. Hence, it is important to understand the progressive three stages of pericardial tamponade that lead to death.

In stage one of traumatic pericardial tamponade, blood accumulates around the heart within the pericardial sac, resulting in increased pericardial pressures. *(See Figures 1a, 1b, and 2.)* This restricts ventricular diastolic filling and subendocardial blood flow. The body compensates for this by increasing heart rate, systemic vascular resistance, and central venous pressure. This represents an effective concerted effort of the body to maintain cardiac output. During this stage in traumatic pericardial tamponade, treatment consists of securing a patent airway, aggressive volume resuscitation, and pericardiocentesis.[6]

Stage two of traumatic pericardial tamponade results in further restriction of ventricular diastolic filling, stroke volume, and coronary perfusion from progressive increases in pericardial blood accumulation. Although blood pressure usually is maintained by the same stage-one compensatory mechanisms, clinical signs of shock begin to emerge. These signs may include anxiety, confusion, or unconsciousness; diaphoresis; pallor; diminished capillary refill and urinary output; tachycardia; and increased thirst. Strict control of the airway, aggressive volume resuscitation, and pericardiocentesis again are paramount in the treatment of this particular stage of traumatic pericardial tamponade. In addition, a sybxiphoid pericardial window made popular by Dr. J.K. Trinkle can be performed by the clinician to diagnose and treat pericardial tamponade.[6,9]

When the pericardial pressure approaches or exceeds the ventricular diastolic filling pressure, blood flow becomes ineffective. Failure of compensatory mechanisms results in global hypotension and severe coronary hypoperfusion. These events characterize this third and final stage of traumatic pericardial tamponade.[10] Without immediate treat-

Figure 2. Subcostal Image of Traumatic Hemopericardium



Key: RV= right ventricle; LV=left ventricle; RA=right atrium; LA=left atrium.
Image courtesy of Michael J. Lambert, MD.

Table 1. Thoracotomy Equipment

- Scalpel with #10 blade
- Mayo scissors
- Metzenbaum scissors
- Rib spreaders (Finichetto's)
- Lebsche's knife and mallet or Gigali's saw (for transecting sternum)
- Tooth forceps (2)
- Vascular clamps (2, Satinsky)
- Long needle holder (2, Hegan)
- 2.0 or larger silk strands
- 3.0 cardiovascular ethibond suture
- Suture scissors
- Aortic clamp (DeBakey or other)
- Tonsil clamps (4)
- Foley catheter (20 french, 30 mL balloon)
- Chest tube
- Towel clips
- Towels
- Laparotomy pads
- Teflon patches (different sizes)
- Internal fibrillation paddles

ment, cardiac arrest ensues. EDT is indicated to ensure the immediate evacuation of pericardial blood and to control the source of the bleeding.

Intrathoracic Hemorrhage. The presence of persistent intrathoracic bleeding is another reason to pursue an EDT. Intrathoracic bleeding can result from penetrating or blunt trauma. *(See Figures 3 and 4.)* The incidence of life-threatening intrathoracic bleeding, however, is less for blunt chest trauma compared to penetrating chest trauma (1-3% vs 3-5%) and usually is due to bleeding from the lung.[3,4,7,11-13] The highest salvage rates with cardiopulmonary resuscitation via EDT occur in patients who have sustained stab wounds to the heart and who go into cardiac arrest just before or soon after arrival to the ED.[3,4,7,11,13,14]

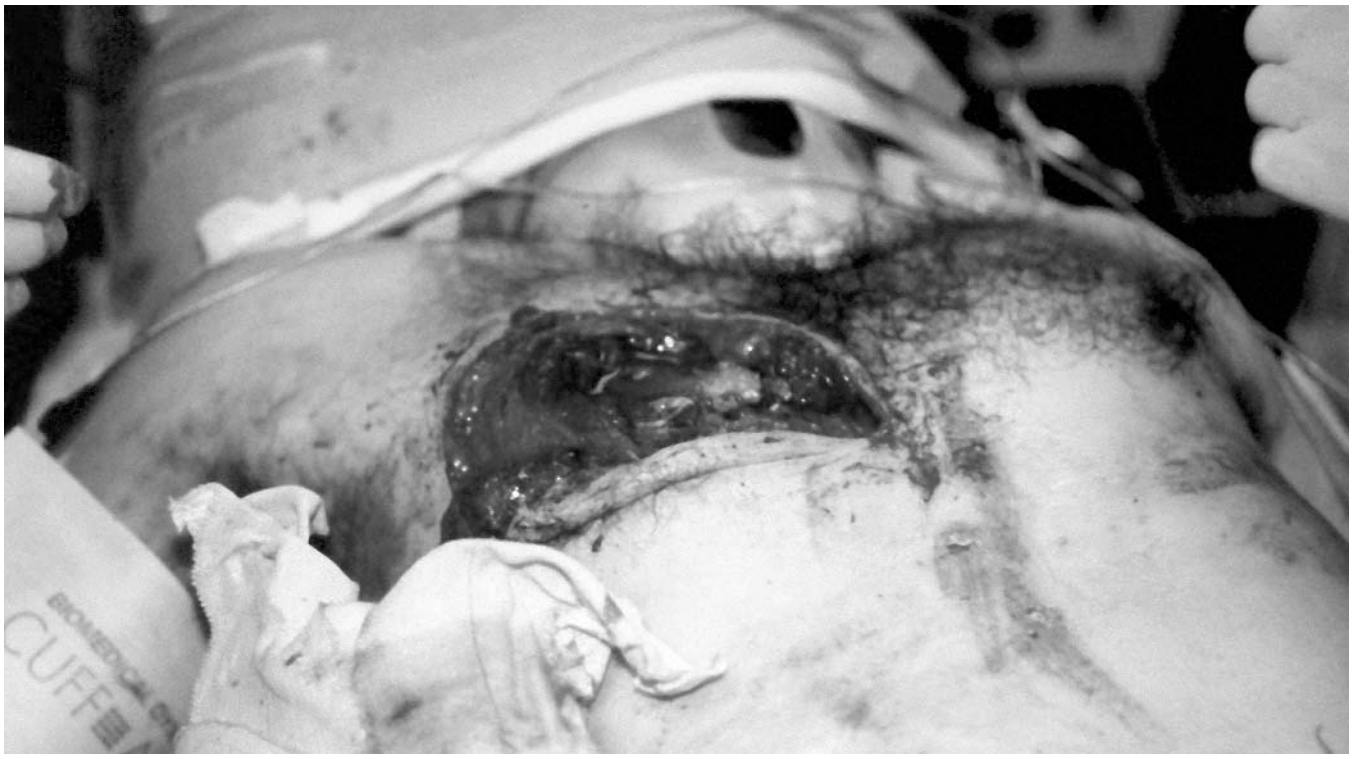
Because the chest is a large potential space, volume losses can be equally impressive and rapid. Each hemithorax can contain approximately 50% of the patient's blood volume (2.5 liters of blood for the average 70 kg person) before it becomes obvious.[3] Patients in extremis with isolated chest penetrating trauma should undergo an EDT to stop the bleeding.[3,7,11-14]

Massive Air Embolism. Air embolism in the setting of trauma is a subtle clinical finding and often is missed. Typically, patients have sustained penetrating chest trauma. After successful endotracheal intubation and positive-pressure ventilation, these patients usually develop precipitous shock. This results when air from the alveolovenous communication shower into the coronary arterial circulation. Myocardial hypoperfusion develops, followed by rapid and global myocardial ischemia. If an EDT is not judiciously performed, cardiac arrest ensues. The goal of the EDT is to cross-clamp the pulmonary hilum on the side of injury to prevent more air from entering the vascular tree. The air should be vented from the ventricles and aorta with the patient in the Trendelenburg position.[15]

Open Cardiac Massage. Open cardiac massage first was proposed by Dr. Schiff in 1874.[1,13] Almost 100 years later, Drs. Kownhoven, Jude, and Knickerbocker introduced closed-chest massage.[1] Since then, both techniques have been scrutinized. There is scientific data to support the rationale of use of both techniques. Overall, open cardiac massage has been shown to be superior to closed-chest compressions.[16] *(See Figure 5.)* Properly performed external cardiac compression can provide up to 10-20% of baseline cardiac output, 3-10% of cerebral perfusion, and 3-10% of coronary perfusion.[17] This allows for reasonable salvage only up to15 minutes, with diminishing survival rates at 30 minutes of cardiopulmonary resuscitation.[16,17] This data pales in comparison to that generated from open cardiac massage in euvolemic patients. Open cardiac massage can deliver up to 60% of baseline (pre-arrest) aortic pressures and cardiac outputs often can be maintained at 50-70% of baseline. This allows for adequate cerebral and coronary perfusion, and hence, reasonable salvage at 30 minutes.[5,17] Because of these studies, there has been increasing discussion about returning to open cardiac massage for resuscitation.

The trauma population is unique in that the hypovolemic patient is more prevalent than in the general medical population. In 1989, Luna and associates demonstrated that external cardiac compressions in the face of hypovolemia and reduced ventricular filling provided inadequate coronary and cerebral perfusion.[15] Animal research clearly demonstrates a marked hemodynamic improvement with open cardiac massage vs. closed-chest compressions (especially beyond two

Figure 3. Penetrating Wound to the Chest



Patient who sustained a penetrating wound to the chest.

minutes).[5] Finally, direct intra-arterial pressure monitoring during external compressions in patients has consistently demonstrated that the maximal aortic pressures generated during precordial compression correlate poorly with cardiac output.[5] These studies solidify the argument for open cardiac massage over closed-chest compressions.

Intra-abdominal Hemorrhage. Performance of EDT for patients with intra-abdominal exsanguinations has been under much debate. Occluding the thoracic aorta could prevent further volume losses below the diaphragm and redistribute blood flow to organs of highest priority—namely, the brain and the myocardium. Studies have shown that clamping the thoracic aorta doubles the mean arterial pressure and cardiac output during hypovolemic shock, allowing these organs to be adequately perfused. However, providing adequate blood flow to these organs comes at a steep price. In the euvolemic patient, this maneuver increases afterload (systemic vascular resistance) and, thus, the oxygen demands placed on the myocardium. It also reduces blood flow by 90% to the abdominal viscera, the spinal cord, and the kidneys. Cross-clamp times up to 30 minutes in elective cases have been well-tolerated. Beyond this time, significant ischemia is encountered. Anaerobic metabolism gives rise to acidemia, which potentiates the typical cascade of events intimately linked to multiple organ dysfunction. Although the idea of temporary aortic clamping to reduce intra-abdominal blood losses and redistribute blood flow to vital organs is sound, there is little current data to suggest that it significantly improves the patient's overall survival rate.[10,18,19]

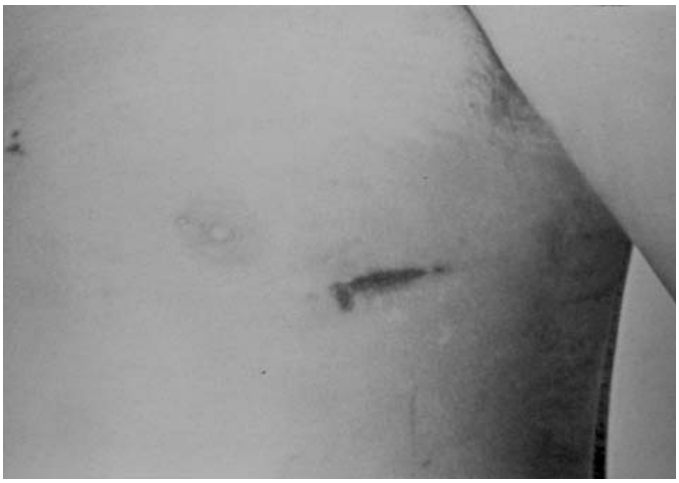
Technical Aspects of the ED Thoracotomy

Preparation. Before performing an EDT, it is necessary to ensure preparedness. A staff skilled in performing an EDT and providing post-EDT resuscitation is a necessity.[20] An EDT tray should be available at all times. This tray should include a scalpel with a No. 10 blade, curved Mayo's and Metzenbaum's scissors, a Finichetto's chest retractor, a Lebsche's knife and mallet or Gigali's saw, long Debakey's vascular forceps, a Satinksky's vascular clamp, Debakey's aortic clamp, a needle driver, non-absorbable suture, pledgets, a Foley balloon, silk ties, sterile towels, and laparotomy pads. The staff should be familiar with the contents of this tray and should observe universal precautions during the procedure. *(See Table 1.)*

The Procedure. As with all surgical procedures, the approach to the EDT should be very systematic. The stepwise approach consists of exposure, pericardiotomy, repair of cardiac injury, open cardiac massage, aortic occlusion, and pulmonary hilar cross clamping (if necessary). Definitive management should be accomplished in the operative theater with optimal lighting, equipment, and sterility.

The left anterolateral thoracotomy incision is the pre-

Figure 4. Isolated Stab Wound to the Chest



This male received an isolated stab wound to his chest.

ferred approach for open cardiac massage. This incision can be extended across the sternum into the right chest to provide exposure of both pleural spaces and virtually all mediastinal structures. *(See Figure 6.)* It is initiated by a swift incision at the level of the fourth to fifth intercostal space (in most cases). A right-sided thoracotomy is reserved for the hypotensive patient with an isolated right-sided penetrating injury. Partial division of the overlying perctoralis and serratus muscles help in exposing the fifth intercostal space. The intercostal muscles and parietal pleura are then divided with heavy curved Mayo's scissors along the superior rib edge so as not to injure the inferiorly positioned intercostal neurovascular bundle. The Finichetto's rib retractor is placed with the handle positioned posteriorly to prevent repositioning if a trans-sternal incision is required. This can be done with a Lebsche's knife and mallet or a Gigali's saw. Be aware that the internal mammary vessels lie approximately 0.5-1 cm lateral to the lateral margin of the sternum. Care must be given to identifying these vascular structures and tying them off. Inadvertently lacerating these vessels can lead to significant blood loss and consume valuable time needed for definitive therapy.

Once adequate exposure is established, the pericardial sac should be opened longitudinally on the anterior surface so as not to injure the pericardiacophrenic complex.

The tense pericardial sac may be difficult to grasp and cut with scissors. It is best to make a small nick in the pericardium with a knife, then carefully extend the pericardiotomy with scissors. The pericardiotomy should extend along the ascending aorta to the top of the pericardium and inferiorly to the level of the diaphragm. This will provide maximum exposure and prevent cardiac strangulation. Blood clots should be evacuated rapidly from the pericardium. In the event of cardiac arrest, bimanual open cardiac massage should be initiated as described by Moore, et al.[18,21] This is done with the palms of the hands hinged together and the fingers providing compression of the ventricles from the apex to the base of the heart. The pads of the fingers never should be used to provide cardiac compression. This technique minimizes the risk of myocardial perforation. If the sternum is intact, open cardiac massage alternatively can be performed by compressing the heart up against the sternum.

Bleeding sites from the heart usually are controlled with light digital pressure. The suturing should be done rapidly with 3-0 non-absorbable sutures prior to defibrillation. Partially occluding clamps can be used to control bleeding from the atrium or great vessels. Ventricular exsanguination can be controlled by inserting a Foley catheter into the ventricular defect. The balloon is inflated, and the catheter is bolstered in place with a non-absorbable purse-string suture. The Foley catheter also can be used for intra-cardiac high volume resuscitation. Definitive repair of ventricular wounds should be performed in the operative theater with 2-0 non-absorbable horizontal mattress sutures buttressed with Teflon pledgets. Posterior cardiac wounds are very treacherous due to limited exposure. Attempts at repair must be made only in the operative theater with optimal lighting and equipment. These injuries usually are associated with a very high mortality rate. Cardiopulmonary bypass should be considered early if there is massive bleeding and/or cardiac irritability every time the heart is lifted to view or repair the posterior injury.

If the heart is void of gross injury and open cardiac massage and/or internal defibrillation do not restore vigorous cardiac activity, the descending thoracic aorta should be occluded inferior to the left pulmonary hilum. It is not necessary to encircle the aorta with the Satinsky's or Debakey's vascular clamp. The aorta can be dissected away from the esophagus anteriorly by incising the mediastinal pleura and away from the prevertebral fascia posteriorly. Encircling the aorta only will increase the likelihood of esophageal injury.

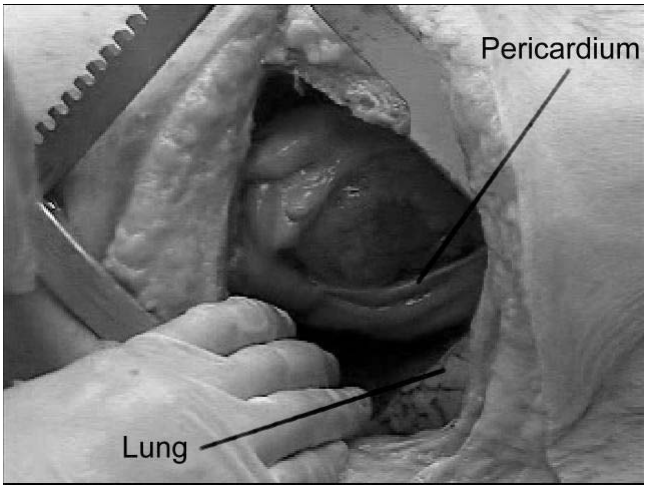
After occlusion of the aorta and aggressive fluid resuscitation the blood pressures should be monitored closely as this provides important prognostic information. If the systolic blood pressure remains below 70 mmHg, it is unlikely that the patient will survive.[12,13,15,16,22,23] On the other hand, if the systolic blood pressure exceeds 160-180 mmHg, the resultant strain on the left ventricle can lead to acute left ventricular distension/failure and pulmonary edema. The clamp should be removed as soon as an effective systemic arterial pressure has been achieved. When aortic cross clamp times exceed 30 minutes, the metabolic penalty becomes exponential. This especially is true in multisystem trauma.

If coronary or systemic air emboli are present, the pulmonary hilum should be clamped to prevent further embolism. Retracting the lung inferiorly can provide adequate exposure of the pulmonary hilum for clamping from a superior to inferior approach. Air can then be aspirated from the apex of the ventricle and the aorta with the patient in a Trendelenburg position.

Figure 5. Cardiac Massage

Used with permission from: ED thoracotomy. Trauma.org, June 2001. WWW.trauma.org/thoracic/EDToperative.html (Accessed 10/8/2003.)

Figure 6. EDT Landmarks



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A systematic approach to the EDT should be honored every time this procedure is performed. Adherence to these basic steps will minimize delays in diagnosis/repair and minimize injury to self and the trauma team.

The Aftermath. If spontaneous cardiac function resumes, the resuscitation priorities shift to maximizing oxygen delivery to the injured tissues. The after-affects of EDT usually results in direct cardiac injury, myocardial ischemia, circulation of cardiac depressants, pulmonary hypertension, and reperfusion injury.[10] Declamping the aorta causes a washout of metabolic by-products and inflammatory mediators into the systemic circulation that may initiate a cascade of events resulting in shock and triggering the systemic inflammatory response. Thus, it becomes paramount to address issues of non-delivery dependent oxygen consumption (VO_2). This is accomplished by raising oxygen delivery (DO_2) until oxygen consumption is supranormal and/or will not rise further with increases in DO_2.

Oxygen delivery is a function of the cardiac output and the oxygen concentration of blood (oxygen carrying capacity). Cardiac output (CO) is related to stroke volume and heart rate. The oxygen concentration of blood is largely related to the hemoglobin concentration (Hgb) and oxygen saturation (SaO_2). To optimize DO_2, the circulating blood volume should be increased until the cardiac index is 4-405 L/min/m^2 or until the cardiac output will not increase with further elevation of end diastolic volume (EDV). The oxygen concentration of blood can be maximized by increasing the hematocrit levels above 35-40%. Fleming and colleagues clearly have demonstrated that if these strategies fail to increase VO_2 to at least 150 cc/min/m^2 within 12 hours of injury, there is an increased incidence of multiple organ failure. In addition, they demonstrated that using supranormal CI, DO_2, and VO_2 parameters can decrease mortality from 50% to 20%.

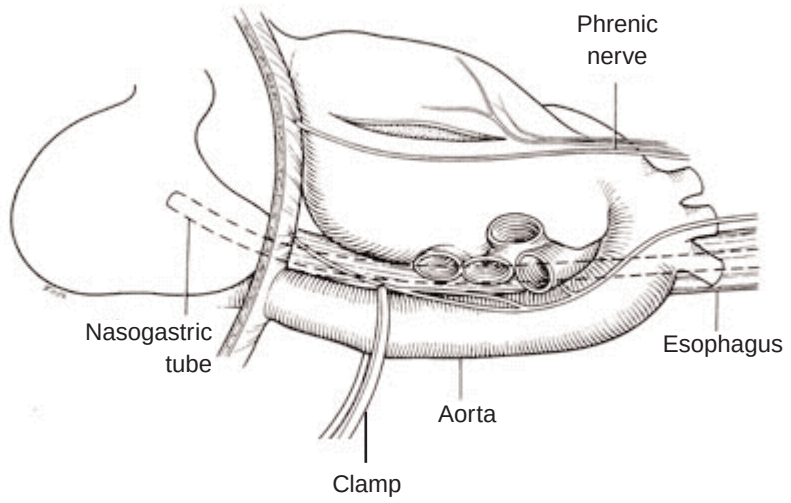
Complications

The EDT can be fraught with hazards in every step of the procedure. Technical complications may involve virtually every intrathoracic structure. Reported complications include injury of the heart, coronary arteries, aorta, intercostals arteries, phrenic nerves, esophagus, and lungs.[21] Adhesions from previous thoracotomies can make performing an EDT extremely challenging and represents a relative contraindication to EDT. Nonetheless, a midline sternotomy for cardiac injuries still remains a viable option for safe exposure.

Other very important, often overlooked and undermentioned complications include accidental injury or disease transmission to the surgeon, assistant and trauma team. Oftentimes, the initial trauma assessment can be chaotic and confusing. This is the perfect environment for injury and blood borne disease transmission. In this setting, it is necessary to regroup thoughts prior to making the initial skin incision and proceed swiftly, safely, and systematically with caution.

For those patients who survive EDT, the most common postoperative complications include atelectasis, pneumonia, recurrent bleeding, diffuse intravascular coagulation, empyema, infections, and sternal dehiscence.[21] The management of these individual problems will not be discussed, as this is beyond the scope of this paper.

Figure 7. Cross-clamp of the Aorta



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Current Guidelines for the Use of ED Thoracotomy

In mid-1999, the American College of Surgeons—Committee on Trauma employed a Working Group, Ad Hoc Subcommittee on Outcomes to embark on the monumental task of reevaluating the use of the EDT. The five questions that they set out to answer included: 1) Which patients should be subjected to EDT? 2) What are the valuable physiologic predictors of favorable outcomes? 3) What is the true survival rate of this procedure? 4) How may survivors succumb to severe neurologic impairment? 5) How can we ensure that those performing EDT are qualified?[22,24]

Literature from 1966 to 1999 was meticulously reviewed and separated based on data classification. There were no Class I (prospective randomized controlled) trials identified. There were 29 Class II (clearly reliable data collected prospectively and retrospectively analyzed) and 63 Class III (retrospectively collected data) studies identified.[21-28]

Which patients should be subjected to EDT? In April 2001, the ACS-COT Subcommittee on Outcomes gave their final recommendations regarding EDT.[24,26] *(See Table 2.)* As expected there was insufficient evidence to support a Level I recommendation for this practice guideline. Their Level II recommendations are as follows:

• EDTs should be performed rarely in patients sustaining cardiopulmonary arrest secondary to blunt trauma due to the unacceptably low survival rate and poor neurologic outcomes;[22]

• EDT should be limited to those that arrive with vital signs at the trauma center and experience a witnessed cardiopulmonary arrest;[16]

• EDT is best applied to patients sustaining penetrating cardiac injuries who arrive at trauma centers after a short scene and transport time with witnessed signs of life;[12,13]

• EDT should be performed in patients sustaining penetrating non-cardiac thoracic injuries.[12,13,15,16,22,23] They did acknowledge the difficulty in ascertaining whether the thoracic injury was cardiac or non-cardiac and promoted the use of EDT to establish the diagnosis; and

• EDT should be performed in patients sustaining exsanguinating abdominal vascular injuries although these patients experience a low survival rate.

The above Level II recommendations also are applicable to the pediatric trauma population.

What is the true survival rate of this procedure? Of studies reporting EDT, 7035 procedures were performed with a survival rate of 7.83%. These procedures were stratified by the mechanism of injury. The survival rate for EDT based on penetrating trauma was 11.16%. The survival rate for EDT based on blunt trauma was 1.6%. The survival rate for EDT based on penetrating cardiac injury was 31.1%[22,25,26,29]

Four series included pediatric trauma patients. The overall survival rate for 142 patients who required an EDT was 6.3%. When stratified by the mechanism of injury, the survival rate for penetrating trauma was 12.2% vs. 2.3% for blunt trauma. There was no reliable data reporting penetrating cardiac injuries in the pediatric population.

How may survivors succumb to severe neurologic impairment? Of the series reporting neurologic outcomes, 4520 patients were subjected to EDT. There was a 5% overall survival rate. Of these survivors, 15% survived with severe neurologic impairment.

What are the valuable physiologic predictors of favorable outcomes? Physiologic predictors of outcomes for EDT have been identified. In 1983, Cogbill and associates determined four statistically significant indicators that portend a dismal outcome. They are: 1) no signs of life at the scene; 2) no signs of life in the ED; 3) no cardiac activity at the time of EDT; and 4) persistent hypotension (SBP < 70 mmHg) despite aortic occlusion. Five years later, Branney and his group determined that the absence of vital signs in the face of blunt trauma also led to a poor outcome.[22,25,26,29]

How can we ensure that those performing EDT are qualified? Although reports of a successful roadside resuscitative thoracotomy in a man sustaining a stab wound to the left lower lobe of the lung has been published by Wall et al,[20] enthusiasm for the use of EDT should be tempered by the receiving hospital's ED resources and the surgical experience of their physicians. Currently, a certification course for EDT does not exist. The technical aspects of EDT is taught at the level of surgical residency. There is much debate regarding the qualification of emergency medicine

Table 2. ACS-COT Subcommittee on Outcomes: Recommendations on EDT

- EDTs should be performed rarely in patients sustaining cardiopulmonary arrest secondary to blunt trauma due to the unacceptably low survival rate and poor neurologic outcomes.
- EDT should be limited to those that arrive with vital signs at the trauma center and experience a witnessed cardiopulmonary arrest.
- EDT is best applied to patients sustaining penetrating cardiac injuries who arrive at a trauma center after a short scene and transport time with witnessed signs of life.
- EDT should be performed in patients sustaining penetrating non-cardiac thoracic injuries.
- EDT should be performed in patients sustaining exsanguinating abdominal vascular injuries although these patients experience a low survival rate.

(The above Level II recommendations also are applicable to the pediatric trauma population.)

physicians to perform this procedure. The optimal benefit of the EDT is achieved at a trauma center by a trauma-trained surgeon or surgeon experienced in the management of major intrathoracic injuries. The emergency medicine physician should not hesitate to perform an EDT, provided that a trauma-trained surgeon is available readily to deliver definitive surgical care. Provision for emergency medicine physicians to perform EDT to temporize problems without the immediate availability of the surgeon is, quite honestly, a waste of time and resources and a significant risk of injury/disease to the trauma team. Be that as it may, the prerequisites for performing EDT should include: 1) a physician experienced in performing thoracotomies and open cardiac massage; and 2) an ED/surgery system that rapidly can provide surgical support.

Conclusions

Chest surgery for open cardiac massage and the repair of injury was first demonstrated at the turn of the 20th century—a time of American ingenuity and innovation in modern medicine. The EDT as a technique for resuscitation of moribund thoracic trauma patients became popular in the 1960s. Enthusiasm for this procedure subsequently led to the employment of EDT in the setting of extrathoracic penetrating trauma and blunt trauma. However, interest in EDT for blunt trauma waned as data (largely retrospective) accumulated demonstrating minimal survival benefit from this procedure.

The rationale for use of EDT includes the release of pericardial tamponade, control of intrathoracic bleeding, control of massive air embolism, open cardiac massage, and temporary occlusion of the descending thoracic aorta to diminish intra-abdominal hemorrhage and optimize blood flow to the brain and the heart. Following successful EDT, the primary goal of resuscitation then focuses on maximizing oxygen delivery to tissues that have been deprived and injured. This is done by optimizing cardiac function and oxygen-carrying capacity at supranormal levels. Evidence exists to validate the utility of these goals, and the newer pulmonary artery catheters can assist in achieving these endpoints.

The literature is replete with data regarding all controversies and questions surrounding this formidable procedure. The issues that have been raised include EDT candidates, survival determinants of patients undergoing EDT for blunt vs. penetrating trauma, the neurologic sequelae of EDT, and quality issues of those performing this procedure. In one of the most complete recent assessments of EDT by the American College of Surgeons Committee on Trauma, these issues were addressed. The committee identified 167,735 studies from trauma centers across the nation, and conducted a strict selection process that narrowed the number of studies to 92. Those studies were then classified according to the scientific evidence and formulation of recommendations scheme. Ultimately, the ACS-COT practice management guidelines recommended EDT's best utility is in those patients sustaining penetrating non-cardiac injuries and exsanguinating abdominal vascular injuries. These same recommendations held true for both the adult and pediatric trauma population.

As medicine faces further scrutiny by the public regarding suitable appropriation of limited resources, it becomes even more critical to identify which patients face mortality and/or severe neurologic impairment. The future will focus on defining nonsalvageability early in the resuscitative effort. Currently, work is underway to identify markers of brain metabolic activity that may assist physicians in earlier termination of futile efforts prior to the consumption of our valuable limited resources.

Other current areas of focus strive to attenuate reperfusion injury, limit the generation of oxidant metabolites during reperfusion, decrease the elaboration of harmful cytokines produced by endothelial cells and macrophages during tissue injury, and pacify primed neutrophils that play a vital role in the inflammatory cascade. The new millennium brings exciting innovations and possibilities in reference to trauma resuscitation. It will be exciting to witness how these discoveries will change the face of our current decision algorithm for the selective use of resuscitative thoracotomy in the ED.

References

1. Barber RF, Wadden JL. Historical aspects of cardiac resucitation. *Am J Surg* 1945;70:135.
2. Blatchford JW, Ludwig R. The first successful cardiorrhaphy. *Ann Thoracic Surg* 1985:39:492.
3. Mittal V. Penetrating cardiac injuries. *Am Surg* 1998;65: 444-448.
4. Rizoli SB. Penetrating heart wounds. *Int Surg* 1993;78: 229-230.
5. Boczar ME, Howard MA, Rivers EP, et al. A technique revisited: Hemodynamic comparison of closed and open chest cardiac massage during human cardiopulmonary resuscitation in

dogs. *Resuscitation* 1984;12:147.
6. Blalock A, Ravitch MM. A consideration of the nonoperative treatment of cardiac tamponade resulting from wounds of the heart. *JAMA* 1960;173:1064.
7. Arreola-Rise C. Factors influencing outcome in stab wounds of the heart. *Am J Surg* 1995;169:553-555.
8. Asfaw I, Austin A. Penetrating wounds of the pericardium and heart. *Surg Clin North Am* 1977;57:37-48.
9. Beck CS. Two cardiac compression triads. *JAMA* 1935:104: 714-716.
10. Arom KV, Richardson JD, Webb G, et al. Subxiphoid pericardial window in patients with suspected traumatic pericardial tamponade. *Ann Thorac Surg* 1977;23:545-549.
11. Shoemaker WC, Carey JS, Rao ST, et al. Hemodynamic alterations in acute cardiopulmonary tamponade after penetrating injuries to the heart. *Surgery* 1970;67:754.
12. Millham FH. Survival determinants in patients undergoing emergency room thoracotomy for penetrating chest injury. *J Trauma* 1993;24:332-336.
13. Tavares S, Hankins JR, Moulton AL, et al. Management of penetrating cardiac injuries: The role of emergency room thoracotomy. *Ann Thorac Surg* 1984;38:83.
14. Trinkle J, Toon R, Franz J, et al. Affairs of the wounded heart: Penetrating cardiac wounds. *J Trauma* 1979:19:467-472.
15. Brown SF. Penetrating chest trauma: Should indications for emergency toom thoracotomy be limited? *Am Surg* 1996;62: 530-534.
16. Graham JM, Beall AC, Mattox KL, et al. Systemic air embolism following penetrating trauma to the lung. *Chest* 1977;72:449.
17. Mattox KL, Feliciano DV. Role of external cardiac compression in truncal trauma. *J Trauma* 1982;22:934-935.
18. Sanders AB, Kern KB, Gordon AE. Time limitations for open chest cardiopulmonary resuscitation for cardiac arrest. *Critc Care Med* 1985;13:897.
19. Moore JB, Moore EE, Thompson JS. Abdominal injuries associated with penetrating trauma. *Am J Surg* 1980;140:724.
20. Sankaran S, Lucas C, Walt A. Thoracic aortic clamping for prophylaxis against sudden cardiac arrest during laparotomy for acute massive hemoperitoneum. *J Trauma 1975;*15:290.
21. Wall RJ. Successful roadside resuscitative thoracotomy: Case report and literature review. J *Trauma* 1994;36:131-134.
22. Moore EE, Moore JB, Galloway AC, et al. Postinjury thoracotomy in the emergency department: A critical evaluation. *Surgery* 1979;86:590.
23. Ivatury RR. Directed emergency room thoracotomy: A prognostic prerequisite for survival. *J Trauma* 1991;3:1076-1082.
24. Washington B, Wilson RF, Steiger Z. Emergency thoracotomy: A four-year review. *Ann Thorac Surg* 1985;40:188.
25. ACOS-COT: Practice management guidelines for emergency department thoracotomy. *J Am Coll Surg* 2001;2:303-307.
26. Branney SW, Moore EE, Feldhaus KM, et al. Critical analysis of two decades of experience with postinjury emergency department thoracotomy in a regional trauma center. *J Trauma* 1988;45:87.
27. Cogbill TH, Moore EE, Millikan JA, et al. Rationale for selective application of emergency department thoracotomy in trauma. *J Trauma* 1983;23:453.
28. Mazzorana V. Limited utility of emergency department thoracotomy. *Am Surg* 1994;60:516-521.
29. Vij D, Simoni E, Smith RF, et al. Resuscitative thoracotomy for patients with traumatic injury. *Surgery* 1983;94:554.
30. Kavolius J. Predictors of outcome in patients who have sustained trauma and who undergo emergency thoracotomy. *Arch Surg* 1993;128:1158-1162.

CE/CME Questions

1. Which of the following is a situation in which EDT may be beneficial to the patient?
   A. Release pericardial tamponade.
   B. Control intrathoracic vascular /or cardiac bleeding.
   C. Control massive air embolism.
   D. Permit open cardiac massage.
   E. All of the above

2. The incidence of life-threatening intrathoracic bleeding is less for blunt chest trauma compared to penetrating chest trauma.
   A. True
   B. False

CME Objectives

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

a.) Quickly recognize or increase index of suspicion for traumatic injuries that may require ED thoracotomy;

b.) Be educated about how to correctly and quickly perform an EDT;

c.) Understand situations where an EDT will not be beneficial; and

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

CE/CME Instructions

Physicians and nurses participate in this continuing medical education/continuing education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge. To clarify confusion surrounding any questions answered incorrectly, please consult the source material. **After completing this activity, you must complete the evaluation form provided and return it in the reply envelope provided in order to receive a certificate of completion.** When your evaluation is received, a certificate will be mailed to you.

3. The highest salvage rates for EDT occur in patients with stab wounds to the heart who go into cardiac arrest just before or soon after arrival in the ED.
   A. True
   B. False

4. Which of the following is true regarding air embolism?
   A. Air embolism, in the setting of trauma, is usually very obvious.
   B. Typically the patient has sustained blunt trauma.
   C. Following intubation and positive pressure ventilation, patients with this disease develop precipitous shock.
   D. Shock results from blood loss into the pericardium.

5. Overall, open cardiac massage has been shown to be superior to closed chest compressions.
   A. True
   B. False

6. Which of the following are prerequisites to performing an EDT?
   A. Skilled staff
   B. Easy availability of an appropriately equipped EDT tray
   C. Familiarity with the tray and the procedure
   D. Use of universal precautions
   E. All of the above

7. The left anterolateral thoracotomy incision is the preferred approach for open cardiac massage.
   A. True
   B. False

8. Which of the following are possible complications of an EDT?
   A. Cardiac injury
   B. Atelectasis
   C. Pneumonia
   D. Infection
   E. All of the above

9. EDT should be performed in all patients sustaining cardiopulmonary arrest secondary to blunt trauma.
   A. True
   B. False

10. EDT is best applied to patients sustaining penetrating cardiac injuries who arrive at trauma centers after a short scene and transport time with witnessed signs of life.
   A. True
   B. False

Answer Key

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

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