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Bronchiolitis: A Systematic Approach to Evaluation, Treatment, and Prevention

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Bronchiolitis begins with symptoms of a mild upper respiratory infection, but it rapidly may develop into a critical, life-threatening event for the at-risk infant. The hypoxic child experiences increased work of breathing, and respiratory failure may develop with startling rapidity. Bronchiolitis remains the most common admission to a pediatric service, with more than 125,000 admissions per year in the United States.[1] Eleven to 15 percent of children will see their physician on an outpatient basis for this common infectious pulmonary event.[2]

During the winter respiratory care season, the evaluation and treatment of this viral infectious disease syndrome is an everyday event for the primary care, emergency, and urgent care physicians, as well as hospitalists and intensivists. There is, however, little consensus on many aspects of the diagnostic workup and subsequent treatment of bronchiolitis. There are questions concerning the utility of radiographs, viral assays and cultures, and common blood tests. Significant controversy exists concerning the use of commonly employed aerosolized bronchodilators, systemic steroids, and antibiotics. Not only do the published studies often contradict each other, they often are contrary to established community standards of therapy. Criteria for hospital admission vary within institutions and across geographical regions. The variation in approach has been shown to drive increasing admission rates and the wide disparity in resource utilization without discernible clinical benefits.[3,4]

Several studies have illustrated successful utilization of practice guidelines based on consensus best practice for inpatient care. Despite clearly derived benefits in cost control, quality of care, and family satisfaction, these guidelines have not been widely implemented.[5,6] Clinicians practicing in the outpatient setting find the majority of clinical studies directed at infants in the inpatient setting. Despite numerous articles published on the individual components of outpatient management of bronchiolitis, a similar systematic implementation of outpatient practice guidelines with effective analysis of clinical, economic, and family satisfaction outcomes has not been performed.

No universal consensus will come of any review of the current literature concerning the diagnosis and treatment of bronchiolitis. Therefore, it is important to look behind these studies and examine their results in light of their methodology and interaction with the underlying pathophysiology of bronchiolitis. This monograph recognizes these necessities and reviews the literature in light of current controversies to extract pertinent information for the emergency department (ED) and urgent care specialist to employ in their management of these potentially ill infants.

—The Editor

Introduction

The signs and symptoms of respiratory infection are the most common cause of acute childhood illness visits to the primary care physician.[2] These infections also frequently are seen in busy EDs and urgent care centers during the winter cold season. Practitioners in these settings have a unique opportunity to effectively intervene in the evaluation and treatment of these patients. Acute bronchiolitis is the most common lower respiratory tract infection (LRTI) seen in the general pediatric population.[7] Effective outpatient evaluation and management of this entity can reduce hospital admissions while directing intensive therapy to those truly in need. The admission rate for bronchiolitis doubled between 1988 and 1996.[1] The diagnosis of bronchiolitis led to 47% of all childhood lower respiratory tract hospitalizations in 1996 and fully 16% of all pediatric admissions in the same year.[1] It results in an economic burden of more than $300 million annually.[8]

Bronchiolitis is defined pathologically as inflammation of the terminal bronchioles in infants and young children, usually as the result of viral infection. The inflammation leads to airway obstruction as a result of edema and accumulation of mucus and cellular debris within the small airways. The obstructive pulmonary disease may lead to air trapping and hyperinflation while work of breathing is increased by dramatic narrowing of the airways.[9] Hypoxemia occurs early in the process, with respiratory failure and hypercapnia being rare and late events.[9] The mechanism of illness drives traditional approaches to therapy. The fundamental role of inflammation leads to consideration of anti-inflammatory therapy with steroids. The role of smooth muscle contraction and bronchoconstriction is controversial and leads to debate over the well-established use of bronchodilators. Viral infection triggers lead one to consider antiviral agents.

Respiratory syncytial virus (RSV) infection is the leading cause of acute bronchiolitis. Overall prevalence is estimated at 43-74% of cases of bronchiolitis throughout the year, with a dramatic increase during winter months.[1] During these annual winter epidemics, it may account for 90% of cases.[10] The season is relatively long (12-16 weeks), with poorly defined boundaries in contrast to the brief, intense, sharply defined influenza season. The overwhelming importance of RSV infection has led to the erroneous synonymous use of RSV infection and bronchiolitis, and many clinical trials restrict inclusion criteria to confirmed RSV inflection. Other agents may cause bronchiolitis. Parainfluenza virus 3, adenovirus, and rhinovirus can provoke the same inflammatory response and account for year-round presentation of children with bronchiolitis.[11,12] Influenza virus, *Mycoplasma pneumoniae*, and *Chlamydia trachomatis* are important causes of infant interstitial pneumonias which may present with virtually identical signs and symptoms in the infant. Coinfection of these agents with RSV in up to 10% of cases has been cited.[13,14]

Human metapneumovirus recently has been identified as a serious pathogen causing viral respiratory tract infections. It apparently has a similar epidemiologic picture to RSV but the clinical syndrome generally milder. It may cause severe illness in younger at-risk infants.[15]

RSV infection occurs in 50% of children in the first year of life, with the remainder seroconverting by age 3.[16] Natural immunity is short-lived, and reinfection occurs throughout life with most illness relatively mild beyond the first two years. Eleven to

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15 percent of children are brought to the attention of their physicians for RSV LRTI in the first year of life. Ten percent of these children will require admission.[1] More than 80% with severe illness will be between 6 weeks and 6 months of age. Boys are represented nearly 2:1 among serious cases, and lower socioeconomic classes are disproportionately represented.[8,9,12]

There are an estimated 5000 deaths per year due to RSV infection in the United States.[17] Case fatality rates for hospitalized children may be up to 10-15% in children with multiple risk factors, 3-4% with underlying cardiac and pulmonary disease, and between 1% and 2% for all hospitalized children.[18,19] While children with risk factors represent a higher risk for serious disease and subsequent hospitalization, the vast majority—80%—of admissions occur in healthy children without antecedent illness or risk factors.

Historically, management of bronchiolitis has been limited to supportive measures with the inherent course of illness dictating hospital admission and utilization of medical resources. Despite the vast number of children with acute bronchiolitis and intense interest in diagnosis and treatment, there had been little consensus on the most appropriate treatment and vast differences in approaches to management within individual medical centers. Recent meta-analyses of treatment options and institution of consensus guidelines have served to reinforce the role of supportive therapy as the critical basis of care despite widespread use of bronchodilators, systemic steroids, and antibiotics in the outpatient setting by experienced, responsible practitioners.

Some of this dichotomy comes from practice setting. Most reported therapeutic trials have centered on the moderately to severely ill hospitalized patient representing one in 10 affected children. Recent trials centered on the clinical approach to the acutely infected child in the outpatient setting present strong evidence for guided use of systemic steroids and sympathomimetic aerosols in bronchiolitis. These therapies effectively may reduce hospital admissions and provide symptomatic relief for ill children.

It long has been observed that infants who recover from acute bronchiolitis have an increased frequency of recurrent wheezing. The reported variance is high, from 29% to 83%.[20] The relationship between recurrent infant wheezing with later development of asthma and of early viral infection is the subject of intense scrutiny. Two theories have emerged and represent alternative ways of accounting for long-term sequelea. The first approach postulates direct damage to the respiratory epithelium by the infecting virus. The second states that the symptoms are a result of a predisposed host with an exaggerated response to the viral infection through either decreased pulmonary function or tendency to atopic disease. The relationship between infant viral infections, wheezing, and the subsequent development of asthma continually is evolving and is beyond the scope of this article.

An examination of the Tucson Children's Respiratory Study data may shed some light on the interaction between host characteristics and the inciting virus.[21] In doing so, one may begin to discern how reputable practitioners observing individual patients may come to different conclusions from studies assuming the homogenous nature of children with the diagnosis of bronchiolitis or those studies dependent on meta-analysis.

In a prospective study of nearly 20 years, more than 800 children were followed for incidence of lower respiratory disease with wheezing. Four groups evolved:

1) Those who never wheezed;

2) Those who wheezed late in childhood but not before age 3;

3) Those who wheezed early in life but did not wheeze after age 6 (early wheezers); and

4) Those who wheezed early and persisted (persistent wheezers).

The latter two groups are of interest. They present in the first year of life with wheezing and commonly are diagnosed as having bronchiolitis. Several important characteristics differentiate the two groups. Early wheezers had impaired pulmonary function tests by squeeze technique in infancy, indicating smaller airways. Viral infection producing airways edema had a more significant effect on work of breathing in these infants compared to normal infants with larger airways. While recurrence of wheezing occurred, the infants were well between events and the process resolved as the children grew. They had relatively smaller airways than normal at 6 years but did not develop classic asthma. There were 113 infants in this category.[21-23]

Persistent wheezers had normal pulmonary functions as infants but had an increased IgE level at 9 months, increased occurrence of atopy by skin test and occurrence of eczema, and were more likely to have a maternal history of asthma. Exacerbations of wheezing in the first years were triggered by viral infection, but they wheezed and required more therapy between acute events. Pulmonary functions had deteriorated by age 6. They may be interpreted as early presentation of (ongoing) asthma. There were 164 infants in this category.[22,23] (*See Table 1.*)

Infants in the first group had wheezing secondary to airway edema and narrowing and would not be expected to respond to classic beta-adrenergic medication. Infants in the second group with early asthma may be expected to benefit from classic asthma therapy. The practicing physician has no reliable way to identify these patient groups with first occurrence of wheezing illness. They all will be diagnosed as having bronchiolitis. Any study cohort of first-episode wheezers will have the two groups represented. One group may respond significantly to a specified therapy with the effect diluted with inclusion of the second group. The clinical trial will show no significant therapeutic effect even though there was a salutary effect on one of the two groups. In contrast, the caring, observing practitioner would see and record the significant effect on the individual patient. The practitioner will elect to employ the therapy widely, despite the clinical trial suggesting little efficacy for the treatment modality.

The long-term relationship between early infection with RSV and asthma will be the subject of debate for the foreseeable future. One recent study of mild RSV bronchiolitis indicates no increased risk for airway hyper-reactivity with observable pulmonary function abnormalities resolved by age 8-12 years.[24] Other studies on long-term implications of bronchiolitis frequently are confounded by inclusion of the two distinct groups identified in the Tucson study.

Readers should bear in mind these two groups when discussion of diagnosis and treatment ensues. The focus will be on the

Table 1. Tucson Children's Respiratory Study[21-23]

	NUMBER	PFT–INFANCY VMAX	PFT–6 YEARS VMAX	IGE 9 MO.	IGE 6 YEAR	ATOPY–% 6 YEARS
Never wheezers	425	120	1260	3.5	30	32
Early wheezers	164	78*	1100*	3.7	32	34
Late wheezers	124	110	1160	4.0	41	56*
Persistent wheezers	113	115	1140*	5.8*	65*	52*

*P < .01

child with first episode of wheezing without strong indication of atopy. Children with recurrent wheezing, evidence of atopy, and strong familial history of allergy and/or asthma potentially represent early manifestation of classic asthma with chronic airways inflammation and secondary bronchospasm as opposed to the infectious acute airway inflammation and edema of bronchiolitis.

Clinical Presentation

The viral infections leading to bronchiolitis present in a protean fashion. For most children, the infection with RSV, adenovirus, or parainfluenza virus appears as a mild to moderate upper respiratory infection requiring little more than parental concern and support.[9] The infection typically begins with 1-3 days of clear rhinorrhea and upper respiratory congestion. A low-grade fever may be present, and the child may appear ill, with decreased appetite. The clinical course generally peaks by day 4 and rapidly resolves without sequelea. Physician or parental preference may dictate over-the-counter symptomatic relief. There is no evidence that antihistamine/decongestant therapy either affects the course of the illness or appreciably reduces upper respiratory symptoms.[25] While there is wide regional variation in use of these agents, many physicians discourage their use in routine viral infections, particularly in patients younger than 6 months.

The onset of significant cough, typically worse at night, indicates progression to more serious lower respiratory involvement. Wheezing is a clinical signature of bronchiolitis. It erroneously has been ascribed to bronchospasm, but the origin generally is attributed to airway narrowing from inflammation and edema.[2] Hypoxemia develops with more serious disease, and accessory muscles of ventilation become involved with prolonged expiratory phase and signs of obstructive lung disease. Pulmonary infiltrates may be seen on x-ray, leading to the diagnosis of viral pneumonia as an alternative or in addition to bronchiolitis. The primary infectious and pathophysiologic process is the same and the distinction is one of nomenclature and often is arbitrary.[9] The therapeutic approach is similar and the appearance of infiltrates does not necessarily indicate the presence of bacterial pneumonia and is not an indication for the use of antibiotics. As the illness progresses, the child may show obvious signs of respiratory distress, with carbon dioxide retention leading to frank respiratory failure.[2,9,10]

Young infants may present with apnea as the sole manifestation.[26] They often have a history of premature birth with an elevated incidence of pulmonary complications. It is uncertain whether the apnea is central or peripheral, and treatment is supportive with mechanical ventilation occasionally required. Central stimulants are not employed, as the apnea resolves promptly as the viral infection clears.

Secondary otitis media is not uncommon in bronchiolitis, approaching an incidence of 15-30%,[2] and a dramatic increase in fever, irritability, or perceived pain should initiate a diligent examination of the tympanic membrane. The observed high frequency of otitis raised the question of whether RSV itself was a primary pathogen in otitis media. One study examined 42 successive children with bronchiolitis in the ED of the University of Pittsburgh with performance of tympanocentesis. Sixty-two percent had acute otitis media on presentation. They found only 14% of the children remained free of bacterial otitis during the course of their illness with the usual spectrum and frequency of bacterial pathogens. They concluded that RSV was not the primary pathogen in the development of the otitis, and bacterial otitis media should be treated with antibiotics.[27]

Clinical Evaluation

The physician in the acute care setting is charged with evaluating infants with acute respiratory infection and determining who has serious illness and needs intervention with pharmacologic and/or hospital support. The classic medical paradigm of a good history and thorough, directed physical examination supplemented by limited laboratory and radiological support is the approach of choice. The diagnosis of bronchiolitis is clinical by nature.

The advent of effective, though costly, prophylaxis for RSV has led to the identification of a high-risk group of infants who tend to have a more serious clinical course. These infants have inherent compromise to the cardio-respiratory system. They include children with congenital heart disease and cystic fibrosis, and premature infants with chronic lung disease. Children with compromised immune systems and uncomplicated premature infants younger than 6 months of age also are at risk.[28] (*See Table 2.*) The attending physician is well advised to take a careful history looking for these risk factors. While these children bear a disproportionate burden of disease, it always should be remembered that the vast majority of serious outcomes with bronchiolitis occur in previously healthy children without identifiable risk factors.

The physical examination is directed to evaluation of the respiratory status of the infant as well as general physical well-

Table 2. Risk Factors for Severe RSV Disease

- Congenital heart disease
- Primary pulmonary hypertension
- Cystic fibrosis
- Chronic lung disease—bronchopulmonary dysplasia
- Congenital or acquired immunodeficiency
- Major congenital anomalies
- Prematurity < 32 weeks
- Prematurity < 36 weeks and age < 6 months
- Age < 6 weeks

being. The experienced clinician's judgment of toxicity is invaluable. An estimation of dehydration helps to determine the general health of the infant. The infant using his strength for air exchange may not have the reserve or desire to maintain hydration with the work of feeding. The classic physical findings of tachypnea, cyanosis, fine crackles, and inspiratory and expiratory wheezing may be difficult to appreciate in an irritable, fearful child. The clinician must develop a feeling for air exchange in these children, looking for physical evidence of distress such as retractions, flare, and audible grunting, as well as recording the auscultatory findings. Often the chest is quiet, as the minimal air exchange cannot produce the classic wheezing of the bronchiolitic infant.[28]

Recognizing the difficult task of evaluating sick infants, one study prospectively evaluated more than 200 infants presenting for ED evaluation. They isolated six variables associated with serious illness requiring admission that have met with wide acceptance.[29] (*See Table 3.*) The time course of clinical symptoms is important. The severity of RSV bronchiolitis reaches a peak in 48-72 hours. An otherwise healthy infant seen after that time period is unlikely to develop more severe disease.[2]

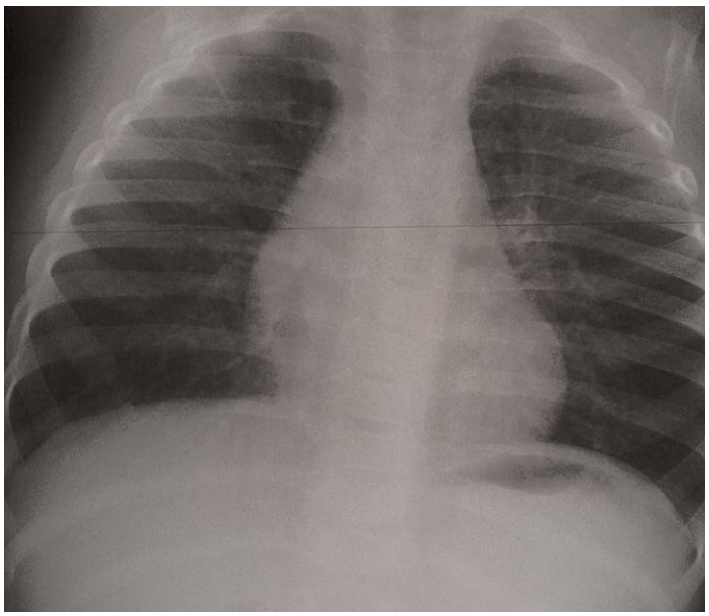
Oxygen saturation by pulse oximeter in a quiet, feeding baby was the most important objective measure predictive of severe illness and need for hospitalization. It also is useful for objectively measuring response to either bronchodilator or oxygen therapy. An infant will defend oxygen saturation late into respiratory failure.[22] An arterial blood gas determination of rising pCO_2 may be necessary to assess impending respiratory failure in a toxic, tired infant. As with older children with asthma, tachypnea should drive down the pCO_2, and a normal pCO_2 may indicate severe respiratory distress. The risk of significant hypoxemia and CO_2 retention generally begins with a respiratory rate greater than 60, and persistent tachypnea alerts one to the possibility of respiratory failure.[2]

Routine laboratory evaluation of complete blood count, metabolic panel, and urinalysis seldom add to the evaluation and are not recommended.[30] The nasopharyngeal swab for RSV assay has come into wide acceptance, with sensitivities approaching 90%.[31] Use of the test in selected sick infants with clinical bronchiolitis has several potential clinical benefits. Children with bronchiolitis appear to be at low risk for serious, invasive bacterial infection.[32] One study examined 211 infants younger than 3 months of age with bronchiolitis and found no evidence of bacteremia, meningitis, or urinary tract infection.[33] A positive assay, therefore, may be used to postpone a sepsis workup in a sick infant. RSV bronchiolitis will have associated serious bacterial illness by sepsis workup in fewer than 2% of cases.[34] Algorithms for institution of sepsis evaluation and institution of systemic antibiotics are keyed to the febrile child without identifiable focus of infection. The use of antibiotics for presumed sepsis has been reported to be associated with an increased frequency of subsequent bacterial infection and emergence of resistant organisms.[35] The individual practitioner may elect to perform a septic workup for serious bacterial illness on a young toxic infant, but the diagnosis of RSV bronchiolitis predicts a very low yield. Confirming this viewpoint is a recent study looking at the use of intravenous broad-spectrum antibiotics in all children admitted for documented RSV bronchiolitis/pneumonia with classical symptoms and physical findings. A study reviewed 2396 children retrospectively who represented seven years' worth of admissions for RSV bronchiolitis in a Texas children's hospital. Broad-spectrum IV antibiotics were initiated in 70.5% of the patients and continued to discharge in 97% of these children. Performance of septic workup was at the discretion of the attending physicians. Unfortunately, data are not available on the number of septic workups performed. Twelve children had positive blood cultures all deemed skin contaminates. There were no positive cerebrospinal fluid (CSF) cultures.[32] The 27 positive urine cultures—1.1%—is comparable to published series of asymptomatic bacteruria.[36] Hoberman reported incidence of urinary tract infection of 3.5% in febrile infants with an identifiable source of fever (i.e., bronchiolitis, otitis media [OM], etc.).[37] The authors concluded that asymptomatic bacteruria could not account for all the positive urine cultures and some must represent serious bacterial infection. There was no evidence of missed sepsis or meningitis in the hospitalized infants.

A negative RSV assay may suggest evaluation for influenza that typically runs concurrently with the RSV season. Influenza produces an interstitial pneumonia difficult to distinguish on clinical grounds from bronchiolitis in the infant and is a potentially treatable viral illness of significant mortality in a vulnerable infant. Recent studies of epidemiology of influenza have highlighted the importance of this respiratory pathogen with seasonal hospitalization rates comparable to those of RSV infections.[38] Studies in Japan have registered higher rates of influenza hospitalization during epidemic years.[39] Rapid laboratory diagnosis of influenza with good sensitivity and specificity is available on a Clinical Laboratories Improvement Act (CLIA) waived basis.[40] Treatment using traditional amantadine antiviral agents and the neuraminidase inhibitors is effective with prompt diagnosis.[41] When confirmed influenza is in the community, there is an increasing premium on consideration of specific viral etiologic diagnosis. A negative RSV assay of an ill child with LRTI should trigger consideration of influenza and appropriate diagnosis and treatment.[42]

The identification of specific viral etiology is a topic that will continue to interest clinicians as progress in diagnostic technolo-

Figure 1. Chest X-ray of Typical Infant with Bronchiolitis



Note hyperinflation without focal infiltrates.

Table 3. Predictors of Severe RSV Disease in Otherwise Normal Infants

- Ill or toxic appearance
- Oxygen saturation < 95% determined by pulse oximetry
- Gestational age < 34 weeks
- Respiratory rate > 70 breaths/minute
- Atelectasis on chest radiograph
- Age younger than 3 months

gy improves turnaround time while reducing costs, and specific antiviral therapies come to market. The use of a rapid direct fluorescent antibody (DFA) screen for respiratory virus pathogens was examined during a two-year period at the University of Utah. DFA-positive patients received fewer days of intravenous antibiotics, fewer days of oral antibiotics, and fewer discharge prescriptions of outpatient follow up antibiotics. As the staff became more comfortable with specific viral diagnosis fewer DFA-positive patients had intravenous antibiotics initiated during the second winter season than the first (26% vs 44%).[43] Multiplex polymerase chain reaction (PCR) also was evaluated at the same institution, with excellent clinical correlation with viral shell cultures.[44] While clinically useful on an individual basis, routine use of RSV assay has not been included in recent guideline implementation with significant monetary savings and little negative clinical impact.[5]

Chest x-ray findings in bronchiolitis are relatively nonspecific. Hyperinflation, peribronchial cuffing, atelectasis, and diffuse interstitial disease often are seen.[45] (*See Figure 1.*) Many films are read as normal, and the radiologic findings do not correlate with disease severity. The most compelling reason to order a film with the first episode of wheezing is to rule out confounding diagnoses that may present with the same picture of respiratory distress.[46] Congenital heart disease with failure, cystic fibrosis, foreign body aspiration, and pneumonia from other infectious agents all may present similarly. Recent practice guidelines discourage routine utilization of the chest film without clinical repercussion.[5] The emergency medicine literature has contributed to advocating decreased use of routine chest films in first episodes of bronchiolitis.[47] In a survey of 140 infants with clinical bronchiolitis in Atlanta and Knoxville, 17 had abnormal films that led to three interventions with additional therapy. These were opacities leading to use of antibiotics for pneumonia possibly of bacterial origin. The one case of congestive heart failure visualized was predicted by physical examination. The authors conclude that routine first-time x-ray is not warranted, but commentary suggests the practice will not be abandoned soon.[47]

Treatment and Controversy

Recommended treatment of bronchiolitis has focused on supportive care of the patient ensuring adequate oxygenation and hydration as the viral illness runs its course.[28] Fever with increased respiratory rate increases fluid requirements while oral intake declines with the general malaise of illness. The difficulty of suckling with tachypnea and hyperpnea also reduces the ability to maintain fluid balance. Hypoxemia is managed with supplementary oxygen to maintain oxygen saturations greater than 92%. The transition from outpatient to inpatient management often hinges on the ability of the infant to maintain adequate fluid intake and oxygenation.

In the hospital, care is directed at fluid support with restriction of oral intake, with respiratory rates greater than 60-80 to prevent aspiration.[48,49] Evaluation for syndrome of inappropriate secretion of antidiuretic hormone (SIADH) proceeds as clinical condition warrants. Warm, humidified oxygen is administered with cannula, mask, or tent as tolerated by the infant. More intensive observation for apnea is warranted in the very young infant, particularly those with premature birth and subsequent lung disease. Intensive care with intubation and mechanical ventilation is indicated for respiratory failure. Respiratory failure may be defined by clinical observation with retained CO_2 and inability to maintain oxygen saturation greater than 92-95% with inspired oxygen of 50% as laboratory support for more intensive treatment.[50]

Clinicians have long recognized many similarities in clinical presentation of infants with acute bronchiolitis and older children with acute asthma. The infants wheeze with prolonged expiratory phase and decreased air exchange. Searching for active intervention in the disease process, physicians have tended to treat bronchiolitis in a fashion resembling therapy of older children with the bronchospasm and airway inflammation of asthma. Supportive care has been augmented by the use of corticosteroids, aerosolized bronchodilators, antiviral agents, chest physiotherapy, and antibiotics.[4] Recent surveys of common clinical practice reveal this approach, yet compendiums of best clinical practice, hospital guideline publications, and most meta-analyses of individual components[51,52] recommend limited utilization of these therapies.[5,6,53] They correctly point out the pathologic basis of

respiratory distress in bronchiolitis is small airways edema and inflammation from acute viral infection, not the bronchospasm of acute asthma.[54]

This widespread disparity of treatment in the light of inconclusive studies highlights the dilemma for the practitioner. The treating physician must examine each component of care in light of his or her own clinical observation and experience and take into account the presentation of each individual patient to implement a reasonable evidence-based plan of care.

Bronchodilators. There have been many studies attempting to determine efficacy of bronchodilator treatment in acute bronchiolitis. Many early studies have been faulted for small sample sizes, use of sedated convalescent infants, and use of subjective clinical scoring systems. They have been performed on sick inpatients. Individually, these studies failed to show a convincing evidence for use of nebulized beta-adrenergic medication in bronchiolitis.[55-58] Meta-analysis is a critical reappraisal of prior clinical studies and has been used draw further conclusions from these studies. Data are pooled, and statistically significant conclusions may be reached if study design parameters are appropriately matched. In 1996, one study examined the effect of all bronchodilators on bronchiolitis and found a significant, though mild, effect on clinical scoring. The researchers did not find a change in admission rate.[51] The study often is faulted for inclusion of all bronchodilators, including anticholinergics. When limited to only beta-adrenergic medication, the results remain the same. While this study and others have demonstrated modest improvement in clinical scoring from beta-adrenergic medications, the relative benefits and lack of effect on critically relevant parameters such as admission rate, length of stay, and oxygen utilization have led consensus reports to discourage the routine use of albuterol aerosol in routine bronchiolitis.[5,6,53]

The potential for the potent alpha adrenergic effect of epinephrine to relieve airway edema has led to reconsideration for the treatment of bronchiolitis. Early work published by Kristjansson suggested a positive effect on clinical scores and oxygen saturation with use of aerosolized epinephrine vs. saline.[59] Further studies on sedated inpatients showed clear preference for epinephrine vs. albuterol in clinical score and pulmonary mechanics.[60] Reijonen demonstrated epinephrine as a safe medication with more rapid improvement in clinical score (Respiratory distress Assessment Instrument-RDAI) compared to albuterol.[61] In 1995, the *Journal of Pediatrics* published a study of 42 infants younger than 12 months with first-time wheezing in the ED. L-epinephrine was compared with albuterol. The control group had greater length of stay in the ED, greater need for ongoing nebulization with albuterol, and decreased clinical scores relative to the epinephrine. Furthermore, 81% of the albuterol group required admission vs. 33% of the L-epinephrine group.[62]

The use of epinephrine in hospitalized infants with bronchiolitis was investigated in a placebo controlled multi-center trial in Australia. It was reported in the *New England Journal of Medicine* in July 2003. There was no difference in length of stay, vital signs, or respiratory effort.[63] The accompanying editorial by highlights the uncertainty in the medical community over the use of aerosolized bronchodilators in bronchiolitis. They acknowledged that neither epinephrine nor albuterol routinely can be recommended for the treatment of infant bronchiolitis, but stated there is some evidence epinephrine reduces airway resistance and increases clinical scores compared to albuterol.[64]

There have been no studies published showing a clear clinical role for the use of anti-cholinergic medications either alone or in conjunction with other bronchodilators for the treatment of acute bronchiolitis.[65]

Emergency Department and Outpatient Issues

It may be concluded that epinephrine is safe and effective for the treatment of acute bronchiolitis in the ED. It would appear to offer significant clinical advantage over albuterol. It also would be prudent to insist on formal clinical assessment of the patient before and after treatment before routine use of any bronchodilator is prescribed and restrict utilization to those who show a clear clinical benefit. If there is no ascertainable improvement in clinical score, vital signs, or sense of well-being, it is sensible to discontinue the use of aerosol bronchodilators rather than increase the frequency of a demonstrably ineffective therapy.

Glucocorticoids. Bronchial inflammation is the fundamental pathophysiologic event in bronchiolitis. The presence of this inflammation with clinical similarity to asthma has led to widespread interest in the use of anti-inflammatory systemic corticosteroids in acute bronchiolitis. Up to 60% of children with bronchiolitis will receive systemic steroids.[53] Despite the teleological attractiveness of this hypothesis, early studies have failed to show clinical benefit. A study published in *Pediatrics* in 1983 did suggest the use of albuterol with dexamethasone was superior to placebo or either albuterol or dexamethasone alone in acutely wheezing infants. The broad entry criteria limit its usefulness in application to uncomplicated bronchiolitis.[66] In a 1991 study of 29 previously healthy infants admitted for bronchiolitis, dexamethasone or placebo was added to standardized therapy of oxygen fluids, albuterol, and ipraprotium aerosols. No difference in oxygen saturation, clinical score, or pulmonary function was noted on day 3.[67]

Randomized controlled trials published in *Lancet* in 1997 failed to show benefit from either oral or intramuscular dexamethasone in acute bronchiolitis.[68] In 1997, a controlled study examined the addition of dexamethasone to albuterol in hospitalized children with bronchiolitis. Seventy-two infants were enrolled, and no differences were detected in length of stay, clinical scores, or need for further intervention.[69] As a result, recent compendiums of clinical guidelines published in *Clinical Evidence* as well as guidelines used in clinical studies of hospitalized patients have all recommend against routine use of steroids in bronchiolitis.[5,6,53]

A meta-analysis published in *Pediatrics* in 2000 reviewed six randomized, controlled trials. The analysis attempted to ask two significant questions:

1) Is systemic steroid therapy associated with a decreased length of hospital stay?

2) Does such therapy provide symptomatic improvement?

Differences in rating scales, endpoints, dosing, and starting

points led to difficulty with selection of papers to review. All subjects were younger than 24 months. Exclusion criteria included previous wheezing and chronic cardio-respiratory disease. Two studies limited patients to positive RSV laboratory tests. Corticosteroid use was oral, intramuscular, or intravenous. Compounds used included dexamethasone, prednisone, prednisolone, and hydrocortisone. None of the studies reported adverse effects of the use of the steroids, and meta-analysis showed the use of corticosteroids in the treatment of bronchiolitis in infants may be more effective than previously acknowledged. There were differences in symptom scores within 24 hours of treatment. As symptom reduction is associated with discharge from the hospital, the length of stay differential was 0.43 days per patient in favor of treatment. The authors were careful to recommend further study and suggested a research design to resolve the question over a broad range of disease severity. They did comment on the long history of safe and effective steroid therapy in asthma and concluded systemic steroids should be considered in the therapy of infants with bronchiolitis.[52]

Confirming the ongoing controversy within academic pediatrics concerning steroid use in inpatients with bronchiolitis, the September 2002 issue of *The Pediatric Infectious Disease Journal* offers a review of the literature from researchers at Turku University of Finland. They conclude that current evidence does not support the use of systemic steroids in wheezing children with primary bronchiolitis younger than 2 years.[70]

Attempts to explain study conclusion differences based on dosing, outcome measures selected, selection of steroid used, route of administration, inpatient vs. outpatient setting, and stage of illness at time of intervention are useful speculations for future trials, provide substrate for academic musings and ferment, and will alter structure of future meta-analysis, but cannot be used to draw firm conclusions concerning general use of steroids in uncomplicated bronchiolitis.

Emergency Department and Outpatient Issues

The majority of patients with bronchiolitis are not admitted to the hospital and are treated as outpatients by parents, primary care physicians, and ED physicians. These practitioners often use oral corticosteroids in bronchiolitis. Most studies of steroids in bronchiolitis have been performed on inpatients. Early in 2002, the Hospital for Sick Children in Toronto published a study of ED utilization of dexamethasone. The objective was to investigate use of a single dose of oral dexamethasone (1.0 mg/kg) in children younger than 2 years of age. Seventy children were enrolled in the study. They were given medication or placebo and observed for four hours in the ED. At four hours, the decision to admit or discharge was made. Outpatients were continued on a daily dose of dexamethasone 0.6 mg/kg with albuterol by small volume nebulizer (SVN). The treatment group had significantly fewer admissions—17% vs. 41%—and fewer children with a poor clinical response over four hours measured by a Respiratory Assessment Change Score (RACS). There was no difference in hospitalization following discharge from the ED, and clinical scores were similar seven days following ED visit.[71]

The authors were conservative in their conclusions, suggesting further research. An editorial by Dr. John T. McBride in the same issue of *The Journal of Pediatrics,* while complementing the authors on their study, offered alternative explanation for the results and urged caution before implementing the protocol in clinical practice.[72] However, the study is straightforward and directed at answering a specific clinical question of great importance, and the data are compelling.

Further evidence supportive of routine corticosteroids in children with mild to moderate bronchiolitis was published in 2002, following a study of outpatients with clinically defined first episode of bronchiolitis seen in the ED of the University of South Alabama. Subjects were randomized to either prednisolone (2 mg/kg/day divided bid × 5 days) with aerosol or oral albuterol or albuterol alone. Fifty-one subjects were enrolled and followed with a clinical bronchiolitis score. There were early significant differences favoring the treatment group. No difference in hospitalization was noted.[73]

Physicians in the outpatient setting are well-advised to read the articles and associated editorial and apply the evidence to their clinical practice as their experience dictates.

Long-term Considerations of Corticosteroid Use. The association between infant bronchiolitis and subsequent wheezing long has been observed. It would be attractive to intervene early to prevent subsequent episodes of bronchospasm. Investigators have looked at this issue in several controlled studies. Van Woensel and colleagues treated hospitalized infants with either prednisolone 1 mg/kg/day for seven days or placebo. There was no difference in respiratory symptoms recorded by telephone interviews during the next three years.[74] There was no difference in nocturnal cough, daily respiratory symptoms, or pulmonary functions in infants younger than 12 months of age who were administered inhaled fluticasone vs. placebo administered for three months following hospital discharge.[75] With a first-time bronchiolitis admission, nebulized budesonide was given for 14 days after discharge. No difference between treatment and control groups in need for respiratory medication, physician visits, or re-hospitalization was found for nine months.[76] While there may be short-term benefits with the use of steroids in bronchiolitis, studies do not support the idea that they prevent further episodes of wheezing.

Antibiotics. The syndrome of bronchiolitis is a consequence of an acute viral infection. Other than the recognized and relatively common occurrence of bacterial otitis media, super-infection with bacteria causing systemic infection is uncommon.[30,34] Some authors suggest the use of RSV assay may eliminate the need for a sepsis workup in a young infant with clinical bronchiolitis. Incidence of serious bacterial infection is fewer than 2% in an infant younger than 2 months of age with a positive RSV assay. A positive RSV immunoassay associated with signs and symptoms of clinical bronchiolitis was considered evidence of RSV infection.[34] In recent series evaluating evidence-based guidelines, more than 50% of infants admitted for bronchiolitis received antibiotics.[5] Antibiotics are indicated for documented bacterial infection and the comorbid otitis media common in

RSV infections. Routine coverage for possible bacterial infection in the lungs and upper airway is not indicated or necessary.[30]

Antiviral Therapy. An effective antiviral agent theoretically would be of benefit in the treatment of acute viral bronchioles. Ribavirin is a synthetic nucleoside virostatic agent that inhibits messenger RNA and prevents replication of many viruses, including RSV.[77] It has been promoted heavily for the treatment of seriously ill infants with RSV infection. Following early studies indicating clinical improvement but no effect on length of stay in non-ventilated patients, a 1991 clinical trial was published of ventilated patients indicating reduced time of mechanical ventilation, decreased length of stay, and decreased mortality.[78] The American Academy of Pediatrics (AAP) recommended use in high-risk infants with serious RSV infection.[76] A multi-center trial published in *The Journal of Pediatrics* in 1996 showed no difference in length of ventilation or mortality.[79] A subsequent review of 10 small trials failed to confirm early findings of efficacy.[80] The questions of toxicity and possible teratogenetic effects to health care workers, significant expense, and questionable efficacy have led the AAP to modify its stance. Ribavirin is to be considered on a case-by-case basis for infants with immunosuppression or immunodeficiency. Infants with underlying chronic lung disease, congenital heart disease, and cystic fibrosis are candidates for treatment.[81] A recent randomized trial in previously healthy infants with severe RSV bronchiolitis showed markedly fewer episodes of reactive airways disease and respiratory admissions in the year following the signal event.[82] Enthusiasm for the treatment seems to center within specific institutions with common usage in certain centers and virtually no use in others. Its role remains controversial.

Studies of the use of RSV immunoglobulin and monoclonal antibody to RSV in sick children with RSV bronchiolitis have indicated rapid clearing of viral burden with active agent. However, the rapid clearing of virus from tracheal aspirates was not associated with change in clinical course.[83] Once infection initiates a serious host inflammatory reaction, specific antiviral therapy has been of little benefit. Studies of antiviral therapy early in the course of illness have not been published.

Chest Physiotherapy. Despite widespread use of chest physiotherapy in the hospital treatment of bronchiolitis, there is very little evidence of efficacy. In a small inpatient study of infants with mild to moderate bronchiolitis, there was no difference in pulmonary compliance, resistance, or work breathing before and after chest physiotherapy.[84] There virtually are no data on implementation with infants with demonstrated atelectasis or those intubated with mechanical ventilation.

Hospital Guidelines—The Cincinnati Experience

Physicians at the Children's Hospital Medical Center in Cincinnati published their experience with hospital care guidelines for epidemic bronchiolitis in December 1999. Examination of hospital records between 1993 and 1997 had revealed a wide variation in criteria for admission and subsequent treatment. Admission rates had risen 15% per year during the period. The principal indication for admission appeared to be the physician decision to employ the use of beta-adrenergic inhalation therapy. A growing body of evidence had cited lack of efficacy for bronchodilator therapy. A study team of 12 participants, including pulmonologists, community physicians, hospital physicians, nurses, respiratory therapists, and members of the hospital division of Health Policy and Clinical Effectiveness, was formed in 1996 to study the problem.[5]

The committee was charged with development of clinical guidelines based on academically supported best practice. The population was limited to infants younger than 1 year of age with a first episode of classic bronchiolitis. The guidelines were implemented on a voluntary basis during the ensuing year with daily monitoring by the study coordinator, chief resident, and head nurse. Extensive physician detailing with presentation at grand rounds, meeting of community physicians, house staff training sessions, and nurse education forums supported the effort.

Table 4 highlights the basis of the guidelines and principles of therapy.

The data was analyzed following 1997. Thirteen hundred historical controls from the prior four-year period were compared with 229 infants admitted that winter season with classic initial bronchiolitis. Demographic characteristics of both groups were similar. Seventy-nine percent of admissions were admitted through the voluntary protocol. Admissions rate declined 29%, with length of stay decreasing from 2.9 to 2.4 days. Readmission

Table 4. Summary of Guidelines Principles

OVERVIEW	
Typical bronchiolitis is to be viewed as an infectious self-limited disease based on airway edema. Treatment is to be based on provision of adequate oxygenation and hydration.	
DIAGNOSTIC STUDIES	
Chest x-ray, nasal washings for RSV assay, and arterial blood gases are to be ordered based on specific need and not obtained routinely.	
RESPIRATORY THERAPY	
Cool mist, chest physiotherapy, induced cough, and suctioning are not recommended.	
Steroid aerosol, saline aerosol are not recommended.	
Routine use of bronchodilator therapy by SVN is not recommended. Utilization of epinephrine by SVN in selected patients considered with demonstrated response greater than 60 minutes by standardized reporting instrument.	
MONITORING	
Discontinue electronic monitoring in a timely manner to facilitate discharge.	
ISOLATION	
Employ respiratory/contact isolation using one-week cohorting.	

Key:
SVN = small volume nebulizer

rates remained constant at 3%, and patient satisfaction surveys showed 93% approval for those treated by guideline vs. 77% admitted off-guideline.[5]

Bronchodilator therapy utilization declined from 69% to 43% post implementation. Multiple doses fell from 57% to 28%. Curiously, neither nurses, respiratory therapists, nor house staff embraced the use of a formal scoring systems for post bronchodilator improvement. RSV assay utilization fell from 89% to 43%, and chest x-ray utilization fell from 70% to 56%. There was no change in the 6-8% use of blood gas determination or the 56-57% incidence of antibiotic usage.[5]

Extensive analysis was done to ensure hospitalization and utilization rates were not affected by a mild RSV season. Utilization of other hospitals and confounding admitting diagnosis by practitioners anxious to avoid the guidelines were not detected. Hospital costs per admission fell 37% during the study period, with a 77% decrease in respiratory therapy costs. There was no increase in morbidity and mortality with these savings. Parents were surveyed for satisfaction during the trial, and the guideline group was significantly more pleased with their experience than families treated outside the guidelines. A preference also was noted with comparison to existing historical hospital patient satisfaction surveys.[5]

Cincinnati Follow Up

The successful initiation of a clinical guideline program with the full backing of the hospital administration, academic medical departments, nursing and resident staff is a significant achievement. Maintaining utilization of the guidelines while letting them evolve to meet changing staff expectations while incorporating new clinical data is another and perhaps more difficult task. The group from Cincinnati reported their experience of three years following introduction of guidelines in October 2000.[85]

The guidelines had evolved, reflecting increasing evidence of the efficacy of epinephrine aerosol in the ED in preventing subsequent admission. This led to insertion of a somewhat ambiguous algorithm recommendation in 1998, which was interpreted by many practitioners as a direct endorsement of epinephrine aerosol for all patients. The evidence was left in the working documents, but implementation of the algorithm was removed in 1999.

Admission rates continued to drop through 1998 and 1999 at a rate of 21% and 23%, respectively, for a total reduction in three years of 56%. Length of stay continued to drop, reaching 2.1 days in 1999. Total reduction of hospital costs fell almost 15% from $3297 before implementation to $2825 following. Rates of ancillary service utilization remained low with continued broad support in the physician and parent community.[85]

Multisite Extension of Cincinnati Guidelines

The success of the Cincinnati Guidelines led to their implementation in the 11 Child Health Accountability Hospitals in the winter of 1998-1999. Infants younger than 1 year with first-episode bronchiolitis were included. Oxygen use, radiologic evaluation, and arterial blood gas (ABG) determinations showed insignificant declines. RSV assay utilization rose slightly. The number of children initially treated with bronchodilators fell 9%, with numbers of multiple treatments dropping from a median of eight doses to three doses post implementation. Ipraprotium use fell from 17% to 0.2%, while trials with epinephrine aerosol increased from 13% to 34% of patients. Systemic steroid used dropped 44%.[86]

It is notable with multiple contemporary publications citing lack of efficacy of aerosolized bronchodilators and significant protocol emphasis on objective limited use of said treatments that attending physicians continued to employ this therapy on a regular, consistent basis during the observed periods. Selection and duration of use showed significant learning experiences. The report's conclusion cites the difficulties in implementing evidence-based medicine in multiple centers and highlights the necessity for effective local leadership and physician buy-in by department chairmen and local thought leaders. The difficulty in changing embedded practice is highlighted.[86]

Cleveland Clinic and Upstate Medical Center

Implementation of guidelines in the Cincinnati experiences was limited by design to infants with a first episode of clinical bronchiolitis. A later examination of a more heterogeneous group of children with documented RSV disease, including children with prior episodes of wheezing (30%) indicated a broader implementation of guidelines is both feasible and beneficial. The Upstate Medical Center in Syracuse, with Cleveland Clinic faculty input, implemented guidelines for RSV bronchiolitis through physician and ancillary detailing in November 1997. Based on the same body of evidential data, the guidelines were remarkably similar to those used in Cincinnati. Documentation of RSV was required in this case, and the use of nebulized albuterol was limited to patients with documented improvement in clinical status with trial nebulization or, less frequently, at the discretion of the treating physician. Results were compared with historical controls of the prior respiratory disease season.[6]

Children admitted after institution of the guidelines were less likely to receive supplemental oxygen or cardiac monitoring and

Table 5. Recommendations for Palivizumab (Synagis) Prophylaxis Candidates

1. Bronchopulmonary dysplasia/chronic lung disease in children younger than 2 years with requirement of medical therapy in the past six months.
2. < 28 weeks gestation and < 12 months of age at beginning of RSV season
3. < 32 weeks gestation and < 6 months of age at beginning of RSV season
4. Infants born at 32-35 weeks gestation < 6 months of age with one or more risk factors for RSV:
   a. Passive tobacco exposure
   b. Routine daycare
   c. Multiple siblings attending daycare or school
5. Palivizumab has not been approved for infants with cardiac disease and utilization often is left to the discrimination of individual cases by the attending pediatric cardiologist.

were more likely to have a documented statement of efficacy of albuterol therapy. There was a trend to shortened stay on both the ward and pediatric intensive care unit (PICU) with a significant decrease in children sent home with SVN for nebulized albuterol. Utilization of ABGs and chest radiographs were unaffected but had been low before implementation. There was no change in morbidity or mortality.[6]

Discussion and Implications for Emergency Department Organization and Therapy

In 1999, a study of 804 children at 10 children's hospitals identified significant variability in approach to treatment of RSV bronchiolitis. The variation in resources utilized was not explained by disease severity or practitioner specialty and experience had no effect on patient morbidity or mortality. It did account for the variation in length of stay and was the primary driver of hospital expense.[4] It was postulated that a rationalized common approach to bronchiolitis could positively affect clinical outcome while directly reducing resource utilization without negatively affecting patient/parental satisfaction or professional values. The studies at Children's Cincinnati and Upstate Medical Center in Syracuse would appear to confirm this hypothesis for inpatient treatment associated with admissions through community primary care practitioners.[4,5]

The ED is the medical system point of contact for many infants with bronchiolitis. Community practitioners will refer sick children to the ED for professional evaluation. Many families see the ED as their primary source of care for their ill children. The department staff initiates and dictates early evaluation and treatment of bronchiolitis, with the decision to admit resting in the hands of the emergency physician and staff. Curiously, representatives of the EDs were not listed on the committees that produced either of the cited protocols for bronchiolitis care.

Implementation of a coordinated guideline between inpatient hospitalists and the ED would appear to be a logical progression from the above trials. It is clear that standardized approaches to the treatment of bronchiolitis as inpatients may reduce admissions and resource utilization. Establishment of similar protocols for early intervention in the ED with common criteria for admission would appear to be a fruitful place for ongoing investigation. A recent survey of admission rates for bronchiolitis in a Canadian metropolitan area showed significantly different admission rates between the general EDs and the pediatric EDs. After controlling for age, clinical severity, socioeconomic status, and co-morbidity, admission rates were 24% for the dedicated pediatric ED vs. 43% for the general EDs.[87] Coordination across multiple outpatient delivery sites also may be a useful tool for more efficient delivery of interventional care.

Prevention

The prevention of RSV infection would be the key to an effective public health program to reduce the burden of infant bronchiolitis. Early attempts to immunize with formalin inactivated whole virus vaccine failed to produce protection against infection.[88] Moreover, vaccinated infants exposed to native virus showed increased morbidity and mortality. Attempts to produce an effective vaccine proceed with use of subunit, recombinant DNA, live attenuate, and peptide vaccine technology has failed to produce a safe and effective vaccine.[88] Clinical trials continue.

The Pediatric Red Book on Infectious Diseases cites traditional nursing interventions of cohort segregation in the hospital, hand washing, and the use of gowns, masks, and gloves as useful for prevention of nosocomial spread of RSV infection in the hos-

Table 6. Summary Recommendations for Urgent Care and Emergency Practitioners

1. Consensus panels with guideline implementation have been demonstrated effective in standardizing care with significant cost savings and increased parental satisfaction with maintenance of clinical standards in inpatient settings. ED physicians should promote similar guidelines within the ED, conferring with local admitting physicians as well as hospitalists and intensivists. Standardization of care within the ED for admission criteria, diagnostic testing, radiology utilization, use of steroids and bronchodilators, and follow up criteria may streamline busy winter RSV/influenza seasons in the ED while providing effective, efficient care.
2. The diagnostic workup should depend on the classic paradigm of history and directed physical examination:
    a. Routine lab work is not indicated (i.e., CBC, UA, electrolytes).
    b. Chest x-ray is not routine but is to be considered for first wheezing event to assist rule out cystic fibrosis, congential heart disease with failure, foreign body or congenital anomaly.
    c. RSV assay is useful for epidemiology and etiology.
        i. If positive, it eliminates need for routine sepsis evaluation in infant
        ii. If negative, it promotes search for alternative infectious agent, i.e., influenza, chlamydia, pertussis.
3. Evidence-based therapy for bronchiolitis:
    a. Bronchiolitis is an infectious self-limited disease where therapy is based on support, hydration, oxygenation, and fever control.
    b. Aerosolized sympathomimetic therapy may be initiated. Continued employment is dependent on objective evidence (i.e., respiratory rate, saO_2, air exchange) of improvement.
    c. Epinephrine has been demonstrated most effective in primary bronchiolitis.
    d. Systemic steroids may be beneficial for preventing admissions in mild to moderate bronchiolitis.
    e. Antibiotics are reserved for documented bacterial infection.
    f. Antivirals are not indicated for routine care.
4. Prevention
    a. Palivizumab (Synagis) prophylaxis is used for selected high-risk infants.
    b. No effective vaccine is on the immediate horizon.

Key:
CBC = complete blood count; UA = urinalysis

pital. Three studies found lower transmission with introduction of cohort segregation alone, hand washing alone, and goggles alone.[89-91] Another found no significant difference with added gowns and masks.[92]

RSV Immune Globulin (RSVIG, Respigam) has been demonstrated to reduce hospital admissions in select high-risk populations.[93] Monthly infusion during RSV season requires IV access, and volume can become an issue in at-risk infants with failure from congenital heart disease. Use of RSVIG has declined significantly with introduction of monoclonal antibody to RSV.

Palivizumab (Synagis), the humanized mouse antibody to RSV, is administered intramuscularly monthly during the RSV season. A controlled trial of more than 1500 infants with gestational age younger than 35 weeks or with diagnosed bronchopulmonary dysplasia (BPD) demonstrated decreased hospitalizations, hospital days, and ICU days as well as number of documented RSV infections. Admissions of children with BPD were reduced 38% and of premature infants by 78%. Based on the results of trials, the AAP has released recommendations for candidates for prophylaxis.[94] (*See Table 5.*)

Summary

Bronchiolitis is an acute viral infection with respiratory distress based on airway edema in peripheral bronchioles. Traditional therapy has been supportive, with common use of beta-adrenergic bronchodilators and considered use of steroids without firm clinical trial basis for either therapy. Controlled implementation of evidence-based treatment has shown to be cost effective while increasing parent satisfaction and reducing morbidity. Recent publications have indicated early outpatient use of steroids and epinephrine sympathomimetic treatment have solid scientific basis for use in the ED and urgent care setting. (*See Table 6.*)

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Physician CME Questions

231. The pathophysiologic basis for bronchiolitis is:
    A. smooth muscle contraction causing bronchospasm.
    B. epithelial necrosis by direct viral invasion.
    C. inflammation of the terminal bronchioles in infants and young children, usually as the result of viral infection.
    D. secondary bacterial superinfection.

232. Viruses associated with bronchiolitis include:
    A. rhinovirus.
    B. parainfluenza.
    C. respiratory syncytial virus.
    D. adenovirus.
    E. All of the above

233. Most therapeutic trials for treatment of bronchiolitis have focused on:
    A. the inpatient setting.
    B. the outpatient setting.
    C. combined extended therapy.
    D. mildly ill patients.

234. The aerosolized therapy demonstrated to be most effective in bronchiolitis is:
    A. albuterol.
    B. epinephrine.
    C. ipratropium bromide.
    D. L-albuterol.

235. Significant predictors of severe course of bronchiolitis in the clinical setting are:
    A. history of premature birth.
    B. low oxygen saturation by oximeter.
    C. respiratory rate greater than 70.
    D. toxic appearance.
    E. All of the above

236. An arterial blood gas determination of rising pCO_2 may be necessary to assess impending respiratory failure in a toxic, tired infant.
    A. True
    B. False

237. The chest x-ray in bronchiolitis:
    A. is diagnostic.
    B. invariably is abnormal.
    C. is ordered routinely in consensus panels.
    D. shows findings that are relatively nonspecific.

CME Instructions

Physicians participate in this continuing medical 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 evaluate 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 that will be provided at the end of the semester and return it in the reply envelope provided to receive a certificate of completion.* When your evaluation is received, a certificate will be mailed to you.

238. Bronchodilator use in bronchiolitis has been shown to:
   A. reduce hospital length of stay.
   B. reduce hospital admission rate.
   C. reduce oxygen utilization as inpatients.
   D. reduce mortality.
   E. have none of the effects listed above.

239. Systemic steroid use has been shown to reduce admission rate in mild to moderate bronchiolitis in the ED.
   A. True
   B. False

240. Antiviral therapy for RSV infection:
   A. is routinely employed in severely ill children.
   B. has been successful in reducing viral titers in tracheal aspirates.
   C. has been shown to be routinely effective in clinical practice.
   D. is recommended by the AAP for all high-risk inpatients.

In Future Issues: **Sepsis**

CME Answer Key

231. C	236. A
232. E	237. D
233. A	238. E
234. B	239. A
235. E	240. B

Emergency Medicine Reports
CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- apply state-of-the-art diagnostic and therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur.

SARS Audio Program Updates Guidelines

Leading epidemiologists say a global return of severe acute respiratory syndrome (SARS)—which wreaked havoc on the health care systems that had to deal with it—is almost inevitable. The current overriding concern is that SARS will resurface as a seasonal illness along with influenza and other respiratory infections. Indeed, it would be a surprising development if the emerging coronavirus did not return, said Julie Gerberding, MD, MPH, director of the Centers for Disease Control and Prevention in Atlanta.

"As an infectious disease expert, I can say in my experience, I've never seen a pathogen emerge and go away on its own," Gerberding said. "I think we have to expect that somewhere, some time, this coronavirus is going to rear its ugly head again; and that's the whole purpose of all this preparedness effort."

What would happen today if a patient with suspect or probable SARS were admitted to your hospital? To help you prepare for the threat, Thomson American Health Consultants offers the upcoming audio conference: The Resurgence of SARS: Why your hospital may not be as prepared as you think, on Dec. 9, from 2:30-3:30 EST. Let our experts help you answer that and many other critical questions with practical tips and solutions to detect first cases and protect other patients and health care workers.

Our speakers are **Allison McGeer, MD**, director of infection control at Mount Sinai and Princess Margaret Hospitals in Toronto. A veteran epidemiologist, McGeer dealt first hand with SARS patients and occupationally infected workers during the prolonged outbreak in Toronto. Hear the lessons learned by somebody who has dealt with this novel emerging pathogen on the frontlines.

If SARS returns, hospital emergency rooms will certainly be on those frontlines. To provide valuable guidance and critical insight in that setting, **Susan E. Shapiro, PhD, RN, MSN, CEN**, will outline valuable tips and procedures, in addition to addressing and clarifying recently updated CDC recommendations for SARS. Shapiro is a Post Doctoral Fellow in Risk Assessment and Intervention Research with Individuals and Families at Oregon Health & Science University School of Nursing in Portland. A career ED nurse and nurse manager before recently completing a doctoral program, Shapiro is the Emergency Nurses Association's representative to the CDC's SARS task force.

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Emergency Medicine Reports®
The Practical Journal for Emergency Physicians

Bronchiolitis

Tucson Children's Respiratory Study

	NUMBER	PFT–INFANCY VMAX	PFT–6 YEARS VMAX	IGE 9 MO.	IGE 6 YEAR	ATOPY–% 6 YEARS
Never wheezers	425	120	1260	3.5	30	32
Early wheezers	164	78*	1100*	3.7	32	34
Late wheezers	124	110	1160	4.0	41	56*
Persistent wheezers	113	115	1140*	5.8*	65*	52*

*P < .01

Risk Factors for RSV Disease

- Congenital heart disease
- Primary pulmonary hypertension
- Cystic fibrosis
- Chronic lung disease—bronchopulmonary dysplasia
- Congenital or acquired immunodeficiency
- Major congenital anomalies
- Prematurity < 32 weeks
- Prematurity < 36 weeks and age < 6 months
- Age < 6 weeks

Predictors of Severe RSV Disease in Otherwise Normal Infants

- Ill or toxic appearance
- Oxygen saturation < 95% determined by pulse oximetry
- Gestational age < 34 weeks
- Respiratory rate > 70 breaths/minute
- Atelectasis on chest radiograph
- Age younger than 3 months

Recommendations for Palivizumab (Synagis) Prophylaxis

1. Bronchopulmonary dysplasia/chronic lung disease in children younger than 2 years with requirement of medical therapy in the past six months.
2. < 28 weeks gestation and < 12 months of age at beginning of RSV season
3. < 32 weeks gestation and < 6 months of age at beginning of RSV season
4. Infants born at 32-35 weeks gestation < 6 months of age with one or more risk factors for RSV:
   a. Passive tobacco exposure
   b. Routine daycare
   c. Multiple siblings attending daycare or school
5. Palivizumab has not been approved for infants with cardiac disease and utilization often is left to the discrimination of individual cases by the attending pediatric cardiologist.

Summary of Guideline Principles

OVERVIEW

Typical bronchiolitis is to be viewed as an infectious self-limited disease based on airway edema. Treatment is to be based on provision of adequate oxygenation and hydration.

DIAGNOSTIC STUDIES

Chest x-ray, nasal washings for RSV assay, and arterial blood gases are to be ordered based on specific need and not obtained routinely.

RESPIRATORY THERAPY

Cool mist, chest physiotherapy, induced cough, and suctioning are not recommended.

Steroid aerosol, saline aerosol are not recommended.

Routine use of bronchodilator therapy by SVN is not recommended. Utilization of epinephrine by SVN in selected patients considered with demonstrated response greater than 60 minutes by standardized reporting instrument.

MONITORING

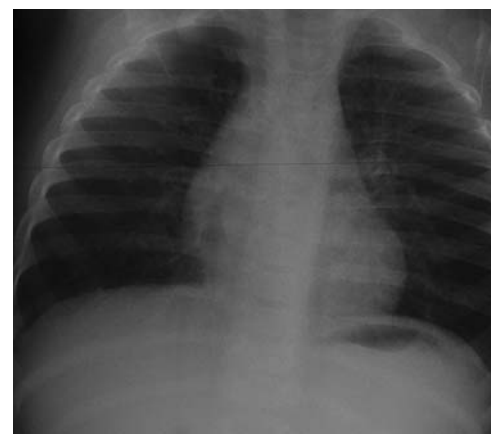
Discontinue electronic monitoring in a timely manner to facilitate discharge.

ISOLATION

Employ respiratory/contact isolation using one-week cohorting.

Key:
SVN = small volume nebulizer

Chest X-ray of Typical Infant with Bronchiolitis



Note hyperinflation without focal infiltrates.

Summary Recommendations for Urgent Care and Emergency Practitioners

1. Consensus panels with guideline implementation have been demonstrated effective in standardizing care with significant cost savings and increased parental satisfaction with maintenance of clinical standards in inpatient settings. ED physicians should promote similar guidelines within the ED, conferring with local admitting physicians as well as hospitalists and intensivists. Standardization of care within the ED for admission criteria, diagnostic testing, radiology utilization, use of steroids and bronchodilators, and follow up criteria may streamline busy winter RSV/influenza seasons in the ED while providing effective, efficient care.
2. The diagnostic workup should depend on the classic paradigm of history and directed physical examination:
   a. Routine lab work is not indicated (i.e., CBC, UA, electrolytes).
   b. Chest x-ray is not routine but is to be considered for first wheezing event to assist rule out cystic fibrosis, congential heart disease with failure, foreign body or congenital anomaly.
   c. RSV assay is useful for epidemiology and etiology.
      i. If positive, it eliminates need for routine sepsis evaluation in infant
      ii. If negative, it promotes search for alternative infectious agent, i.e., influenza, chlamydia, pertussis.
3. Evidence-based therapy for bronchiolitis:
   a. Bronchiolitis is an infectious self-limited disease where therapy is based on support, hydration, oxygenation, and fever control.
   b. Aerosolized sympathomimetic therapy may be initiated. Continued employment is dependent on objective evidence (i.e., respiratory rate, saO_2, air exchange) of improvement.
   c. Epinephrine has been demonstrated most effective in primary bronchiolitis.
   d. Systemic steroids may be beneficial for preventing admissions in mild to moderate bronchiolitis.
   e. Antibiotics are reserved for documented bacterial infection.
   f. Antivirals are not indicated for routine care.
4. Prevention
   a. Palivizumab (Synagis) prophylaxis is used for selected high-risk infants.
   b. No effective vaccine is on the immediate horizon.

Key:
CBC = complete blood count; UA = urinalysis

Supplement to *Emergency Medicine Reports*, November 17, 2003: "Bronchiolitis: A Systematic Approach to Evaluation, Treatment, and Prevention." *Author:* **Stephen R. Luber, MD, FAAP,** Pediatrician, Rockwood Clinic, Spokane, WA; Clinical Faculty, University of Washington Medical School.

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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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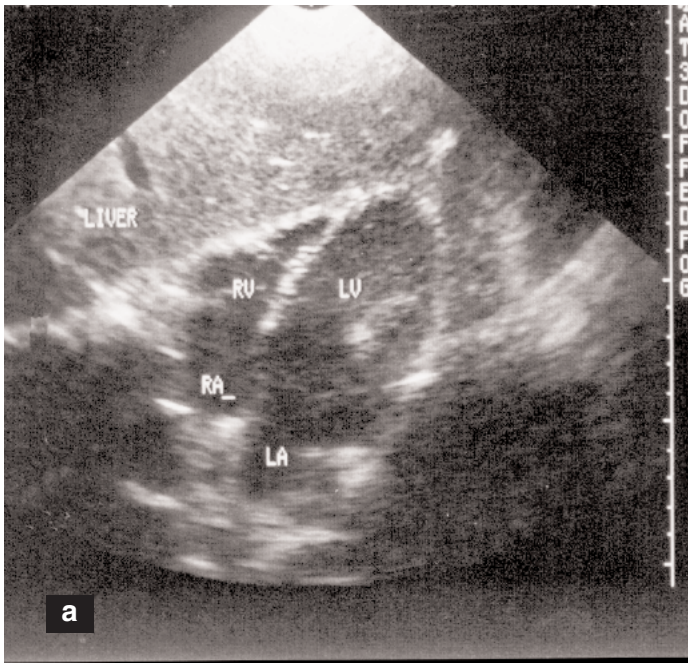
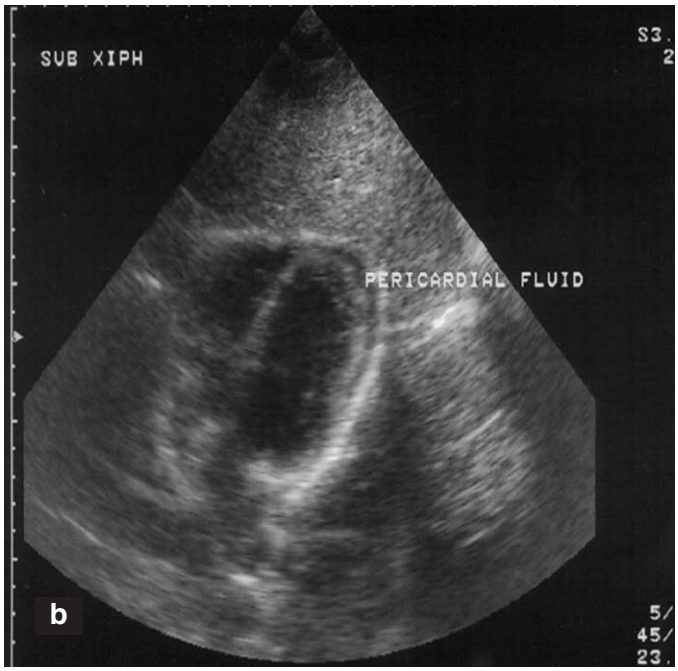
Figures 1a and 1b. The Pericardial Window





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]

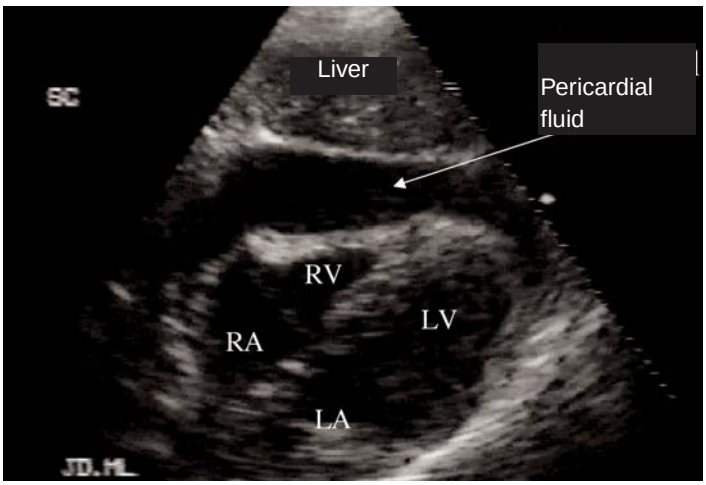
Pericardial Tamponade. Pericardial tamponade may result from gunshot wounds or stab wounds. Stab wounds commonly cause pericardial tamponade (80% of cases).[8] Pericardial tamponade can be characterized by Beck's triad (hypotension, distended neck veins, and muffled heart tones).[9] However, this triad 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]

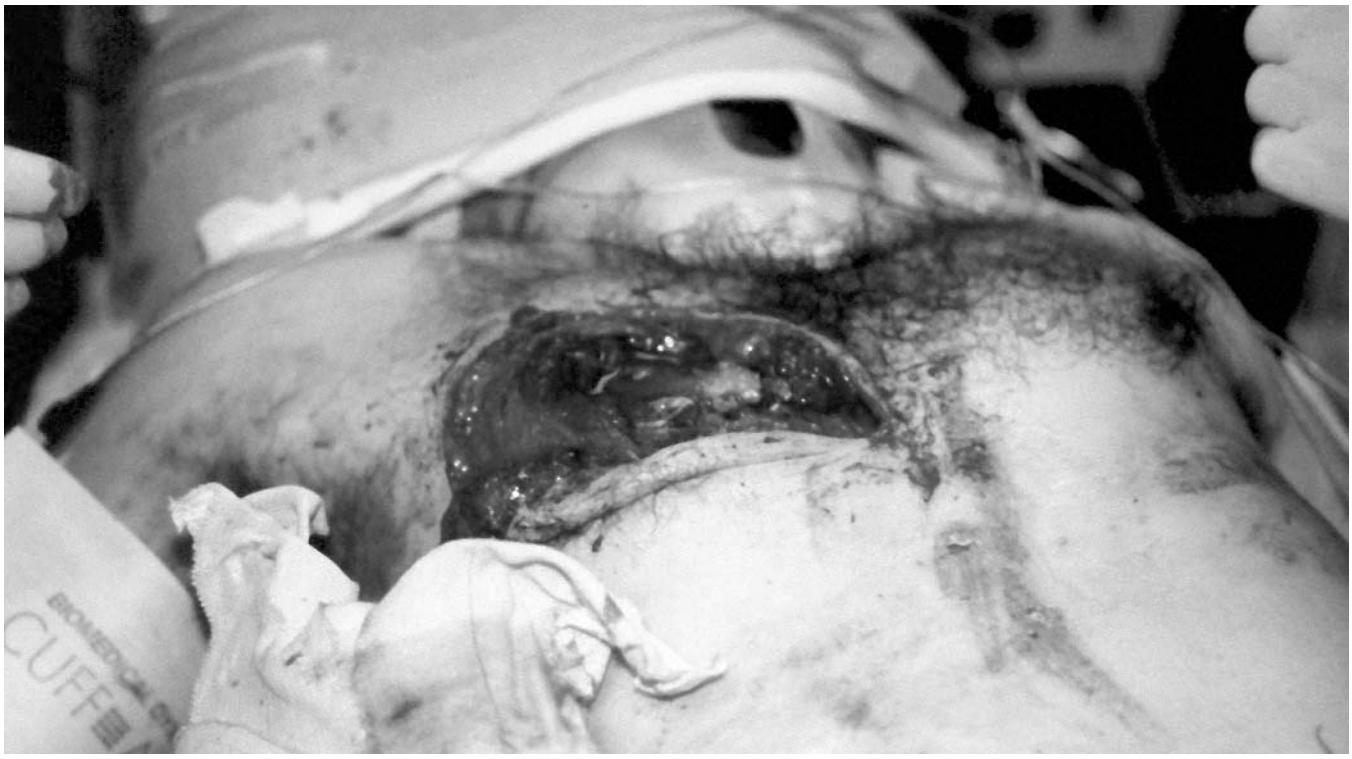
Because the chest is a large potential space, volume losses can be equally impressive and rapid. Each hemithorax can contain approximately 50% of the patient's blood volume (2.5 liters of blood for the average 70 kg person) before it becomes obvious.[3] Patients in extremis with isolated chest penetrating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

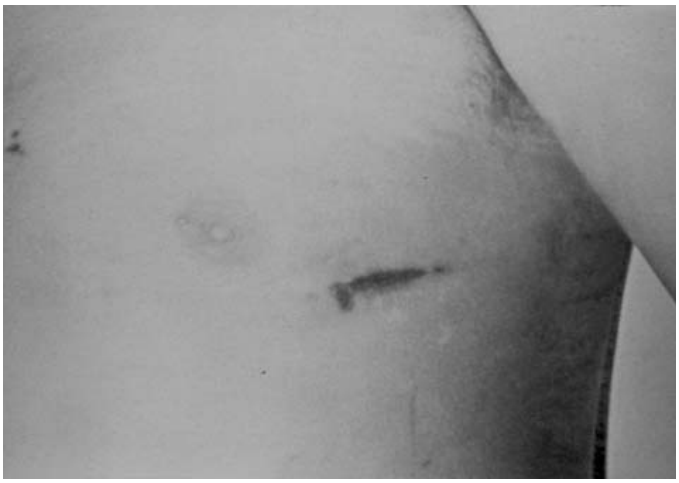
Technical Aspects of the ED Thoracotomy

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.

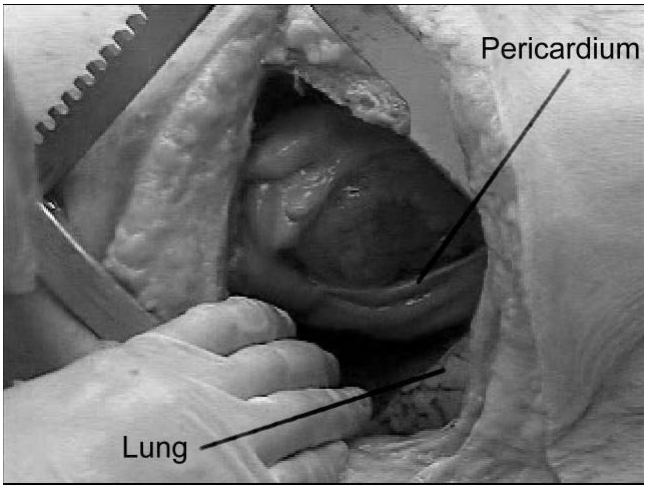
After occlusion of the aorta and aggressive fluid resuscitation the blood pressures should be monitored closely as this provides important prognostic information. If the systolic blood pressure remains below 70 mmHg, it is unlikely that the patient will survive.[12,13,15,16,22,23] On the other hand, 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%.

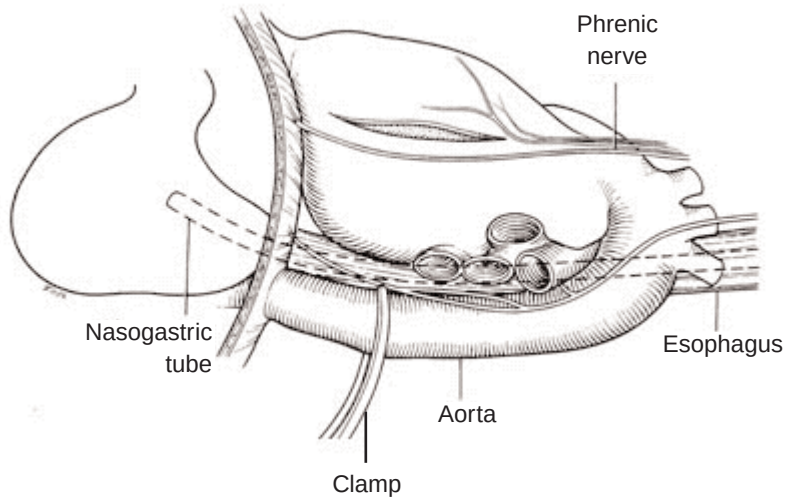
Complications

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



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

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.

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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

Sourcebook Guides You Through Final EMTALA Rule

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* What are the rules concerning off-campus clinics?

Edited by **James R. Hubler, MD, JD, FACEP, FAAEM, FCLM,** attending physician and clinical assistant professor of surgery, Department of Emergency Medicine, OSF Saint Francis Hospital and University of Illinois College of Medicine, Peoria, and reviewed by **Kay Ball, RN, MSA, CNOR, FAAN,** Perioperative Consultant/Educator, K&D Medical, Lewis Center, OH, *EMTALA: The Essential Guide to Compliance* draws on the knowledge and experience of physicians, nurses, ED managers, medicolegal experts, and risk managers to cover the EMTALA topics and questions that are most important to you, your staff, and your facility.

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