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INSIDE

D-dimer in the Diagnosis of DVT

ABSTRACT & COMMENTARY

Source: Wells PS, et al. Evaluation of D-dimer in the diagnosis of suspected deep-vein thrombosis. *N Engl J Med* 2003;349:1227-1235.

THE AUTHORS OF THIS RANDOMIZED, CONTROLLED STUDY SOUGHT to determine whether incorporation of a D-dimer testing strategy in patients with suspected deep-vein thrombosis (DVT) would reduce the need for ultrasound (US) imaging without compromising patient safety. Adult patients were recruited from thrombosis units and emergency departments (EDs), and were excluded if they were terminally ill, pregnant, taking warfarin, or if a pulmonary embolism (PE) was suspected.

The study design was complex and employed four different treatment algorithms. The clinical likelihood of DVT was first determined using previously validated criteria.[1] Patients were randomized to a control group in which conventional US testing was performed, or to a D-dimer group in which US was performed selectively. Control group patients with low pre-test DVT likelihood underwent US imaging alone, while low-risk D-dimer group patients underwent US only if the D-dimer test was positive. Control group patients with high pre-test DVT likelihood underwent US, and those with negative studies had a repeat US at one week. D-dimer group patients with high DVT likelihood all underwent US; those with negative US and negative D-dimers had no further testing done, while those with negative US and positive D-dimers had a repeat US study at one week. D-dimer testing was performed using either the SimpliRED assay (American Diagnostica Inc., Greenwich, CT) or the IL-Test (Instrumentation Laboratory, Lexington, MA). The study endpoint was a thromboembolic event (development of a proximal DVT or PE) within three months in a patient in whom DVT had been ruled out using the appropriate testing strategy.

The authors enrolled 1096 patients with an overall DVT prevalence of 16%. Among the 530 patients in the control group (US only), six (1.4) had a thromboembolic event within three months after initially having a DVT ruled out. Among the 566 patients in the D-dimer group, two (0.4%) had a thromboembolic event within three

months after initially having a DVT excluded. This difference was not significant. Thirty-nine percent of the patients in the D-dimer group required no US testing—that is, they were low-risk and had negative D-dimer results—and only two of these 218 non-imaged patients ultimately had a thromboembolic event. Furthermore, follow-up testing was necessary for only 18% of patients in the D-dimer group, while 35% of control patients required further study at one week. The authors conclude that DVT safely can be excluded in low-risk patients with negative D-dimer tests, and that no US is necessary in such patients.

COMMENTARY BY DAVID J. KARRAS, MD

This study is powerful and may have a great impact on ED practice. However, great care is necessary to avoid misapplication of its findings. It is important to recognize that all patients determined to be at high risk for DVT underwent US study. Thus, familiarity with the Wells risk stratification model is essential.[1] Second, the authors used only D-dimer tests known to have extremely high sensitivity for DVT—the SimpliRED assay (a qualitative whole-blood agglutination assay) and the IL-test (a quantitative latex agglutination assay). Multiple D-dimer tests exist, some with sensitivities as low as 80%, and there is no standardization of results between the assays.[2] The testing strategies employed in this study cannot be extrapolated to sites using less sensitive D-dimer tests.[3]

Provided that emergency physicians carefully consider the test being utilized and select appropriate patients, the results of this study make it feasible for EDs to initiate D-dimer-based DVT evaluation policies in an effort to save time and cost and to eliminate the need for follow-up testing in up to one-third of patients. As an afterward, the three references listed below are outstanding clinical reviews of approach to the patient in whom a DVT or PE is suspected. ❖

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CURE, Clopidogrel, and Aspirin: Good Study, Nice Adjunct, and the Real Deal

ABSTRACT & COMMENTARY

Source: Peters RJ, et al. Effects of aspirin dose when used alone or in combination with clopidogrel in patients with acute coronary syndromes: Observations from the Clopidogrel in Unstable angina to prevent Recurrent Events (CURE) study. *Circulation* 2003;108:1682-1687.

THE AUTHORS STUDIED THE BENEFITS AND RISKS OF adding clopidogrel to different doses of aspirin in the treatment of patients with acute coronary syndrome (ACS). Patients with ACS who were using daily aspirin of varying doses were randomized to receive clopidogrel or placebo for up to one year. The patients were divided into three groups based on the daily dose of aspirin—up to 100 mg, 101-199 mg, and 200 mg or more. Patients were eligible for inclusion if they had ACS symptoms

without ST segment elevation, class IV heart failure, or recent therapeutic cardiac catheterization or coronary artery bypass grafting. The patients needed to have evidence of ischemia other than ST segment elevation, or cardiac enzymes (including troponin) of at least twice the normal level.

Clopidogrel was given as a loading dose of 200 mg orally followed by 75 mg per day for 3-12 months. The dose of the aspirin was left up to the discretion of the local investigator. The outcome measured was the combined incidence of cardiovascular death, myocardial infarction, or stroke. This combined outcome measure was reduced by clopidogrel regardless of aspirin dose, as follows: up to 100 mg, 10.5% vs. 8.6% (relative risk [RR], 0.81 [95% CI, 0.68-0.97]); 101-199 mg, 9.8% vs. 9.5% (RR, 0.97 [95% CI, 0.77-1.22]); and 200 mg or more, 13.6% vs. 9.8% (RR, 0.71 [95% CI, 0.59-0.85]).

The incidence of major bleeding increased with increasing aspirin dose both in the placebo group (1.9%, 2.8%, and 3.7%, respectively; P = 0.0001) and the clopidogrel group (3.0%, 3.4%, and 4.9%, respectively; P = 0.0009); thus, the excess risk with clopidogrel was 1.1%, 1.2%, and 1.2%, respectively. The adjusted hazard ratio for major bleeding for the highest vs. the lowest dose of aspirin was 1.9 (95% CI, 1.29-2.72) in the placebo group, 1.6 (95% CI 1.19-2.23) in the clopidogrel group, and 1.7 (95% CI 1.36-2.20) in the combined group.

In patients with ACS, adding clopidogrel to aspirin is beneficial regardless of aspirin dose. However, bleeding risks increase with increasing aspirin dose, regardless of the clopidogrel. Higher doses of aspirin are no more effective than lower doses in prevention of the combined outcome measure. These findings suggest that with or without clopidogrel, the optimal daily dose of aspirin may be between 75 mg and 100 mg. Overall, the greatest improvement in outcome measures was seen at the lowest dose of aspirin and clopidogrel, although the latter had only a fractional effect.

COMMENTARY BY RICHARD J. HAMILTON, MD, FAAEM, ABMT

Clopidogrel is an adenosine 5′-diphosphate (ADP) inhibitor and is classified as an antiplatelet agent. Recently, experts and consensus panels have been suggesting its use in the patient suspected of acute coronary syndrome.[1] In addition, *Clinical Evidence Concise* suggests that clopidogrel is likely to be beneficial in unstable angina patients.[2] I was interested in this article because I have yet to become completely comfortable with its use in the emergency department (ED). This study provided me with some fairly helpful data to guide safety issues in using clopidogrel and—more importantly—aspirin. I will be using the "baby" dose (81 mg) of aspirin and clopidogrel to maximum benefit for my patients. By the way, *Clinical Evidence Concise* lists only aspirin as proven to be beneficial. I think everyone in the ED who has any complaint remotely related to ACS should be given a baby aspirin. Aspirin is the horse that pulls the risk reduction cart in ACS—everything else is just helping out. ❖

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Isolation: Don't Let 'Out of Sight' Mean 'Out of Mind' When Using this Precaution

ABSTRACT & COMMENTARY

Source: Stelfox HT, et al. Safety of patients isolated for infection control. *JAMA* 2003;290:1899-1905.

Patient safety and medical errors have become a major focus of the provision of health care during the last decade. Critics of transmission-based precautions question whether isolation affects the quality of care provided to these patients. This study examined whether the isolation of patients to prevent nosocomial transmission of disease leads to patient neglect and medical errors.

This retrospective, case-controlled study examined two separate cohorts of consecutive patients isolated for methicillin-resistant *Staphylococcus aureus* (MRSA) colonization or infection: 1) a general cohort of patients admitted for any diagnosis; and 2) a disease-specific cohort of patients admitted for congestive heart failure (CHF). The investigators then selected two matched controls for each isolated patient. The MRSA isolation policies used at the study institutions included private rooms, gloves and gowns for all visitors, dedicated equipment, and restriction of the patient to the room except for essential movement. The main out-

come measures included several measures of quality of care, patient outcomes, and patient satisfaction.

In the general cohort, 78 isolated patients and 156 control patients were enrolled. In the disease-specific CHF cohort, 72 isolated patients and 144 control patients were enrolled. The cases and controls were well-matched, with very similar baseline characteristics.

The isolated patients had more days without vital signs recorded (5% vs 1%, $p = 0.02$) or with vital signs not recorded as ordered (51% vs 31%, $p < 0.01$). Isolated patients also were more likely to have days without physician progress notes (26% vs 13%, $p < 0.01$). Isolated patients with CHF were far less likely to have a stress test or angiogram if they had angina (14% vs 45%, $p < 0.01$). Isolated patients with CHF were less likely to have documentation of CHF education (29% vs 51%, $p < 0.01$) and to have timely follow-up appointments scheduled (24% vs 46%).

Isolated patients were twice as likely as controls to experience adverse events (31 vs 15 events per 1000 days, $p < 0.01$), with this difference reflecting a greater rate of preventable adverse events (20 vs 3 per 1000 days, $p < 0.01$), but no difference in non-preventable adverse events (11 vs 12 per 1000 days). Isolated patients were eight times more likely to experience supportive care failures such as pressure ulcers, falls, and fluid or electrolyte disturbances. No difference in total hospital mortality was observed.

In addition, isolated patients expressed greater dissatisfaction with their treatment than controls. More isolated patients lodged unsolicited formal complaints to the hospital than controls (8% vs 1%). These complaints were related to negative perceptions of treatment, access to staff, and communication.

The authors conclude that these results demonstrate a strong relationship between patient isolation and shortfalls in processes, outcomes, and satisfaction. They suggest that isolation policies could be examined to keep the components most important for infection control, and to perhaps discard those most deleterious to patient safety.

COMMENTARY BY JACOB W. UFBERG, MD

Perhaps the one positive note in this study for emergency physicians is the fact that in the CHF group, ED treatment of cases and controls was identical. No differences were noted in the rates of patients receiving ECGs, chest radiographs, general bloodwork, and cardiac enzyme measurement.

The decision to admit a patient to an isolation bed frequently is made by the emergency physician, or is made by an admitting physician based on the fact that the ED isolated the patient. For emergency physicians, there are two major take-home points in this study: 1) We should be sure to check on our isolated patients in the ED frequently; and 2) we should be careful to isolate only the patients who truly need it, as isolation clearly carries risk. ❖

The Right Thermometer for Tiny Infants: Rectal Proves Most Reliable

ABSTRACT & COMMENTARY

Source: Callanan D. Detecting fever in young infants: Reliability of perceived, pacifier, and temporal artery temperatures in infants younger than 3 months of age. *Pediatr Emerg Care* 2003;19:240-243.

Fever in babies is a major cause for parental alarm and frequently leads to ED evaluation. Families and physicians need accurate measurements of body temperature to expedite evaluation of infants thought to have fever, an indicator of possible serious bacterial illness. Callanan analyzed three methods of temperature measurement among babies in an ED population to ascertain performance criteria for means of fever determination.

Two hundred infants younger than 3 months of age were studied at an urban pediatric ED in San Antonio. Parents were queried as to their perception of the presence or absence of fever (perc-T) in their children, regardless of presenting complaint. Each patient then had temperature measurement by three methods: pacifier thermometer (pac-T), temporal artery thermometer (temp-T), and rectal thermometer (rect-T). Fever was defined as temperature exceeding 38.0° C (100.4° F) by rect-T determination.

Based on rect-T determinations in 179 infants, 91% of 23 febrile infants were accurately detected as having fever greater than 38.0° C by tactile perception (perc-T) among parents. Yet 21% of 156 nonfebrile infants also were thought to have fever by parental perc-T estimation. Hence, for the perc-T method, sensitivity was 91%, specificity 79%, positive predictive value (PPV) 39%, and negative predictive value (NPV) 98%.

Pacifier thermometry among 149 infants, 21 (14%) of whom were febrile, revealed a sensitivity of 48%, specificity of 99%, PPV of 91%, and NPV of 92%. Difficulty was encountered with inability or refusal of many infants

to suck on the pacifier device for times sufficient to reach endpoints for measurement.

For 187 infants evaluated by temporal artery thermometry, including 23 (12%) febrile patients, sensitivity and specificity of temp-T were 83% and 86%, respectively, while PPV and NPV were 45% and 97%, respectively. The author concludes that parents are accurate in perc-T detection of fever among infants younger than 3 months of age, but that the pac-T and temp-T tools tested in this study are unacceptably variable compared to rect-T measurements.

COMMENTARY BY MICHAEL FELZ, MD

Unfortunately, the same perc-T method by which parents reliably detected fever also overestimated temperature in one of five infants tested. Pac-T, while attractive to caregivers, is highly dependent on the infant's willingness to suck on an artificial nipple device for prolonged, sometimes unachievable, periods of time, which sharply lowers sensitivity. The temp-T method, recently available for home use, seems promising based on the ease and rapidity of sweeping the probe across the forehead, but is plagued with misclassification of febrile infants as normal, and healthy infants as febrile, in 15-20% of cases.

My impression of this data is that perc-T still matters, for parents traditionally are the unsurpassed "front line" in recognizing fever in their infants and seeking ED or office evaluation appropriately. This study emphatically demonstrates the tactile reliability of parental detection of fever, with 91% sensitivity. But the pacifier and forehead methods, by comparison, have a long way to go to equal the performance of "mother's touch" in detecting fever, much as has been the case for axillary and tympanic thermometry devices when tested in young infants. Rectal thermometry remains the best method of detecting or excluding fever when considering urgent sepsis workups in infants younger than 3 months of age in the ED or office.

At our tertiary care center, ED physicians doggedly insist on rectal temperatures for the tiny tot who feels hot. I further would suggest that fever as indicated by all noninvasive devices for measurement of temperature in tiny babies be accurately confirmed by rectal methodology before proceeding with complete blood count, blood and urine cultures, chest films, lumbar punctures, antibiotics, and/or hospital admission. I suppose you could view the fever-in-infants issue this way: determination of fever requires clinical precision far exceeding a 50-50 coin toss, so in the ED, it is heads (forehead, pacifier methods) you lose, and tails (rectal thermometer) you win. ❖

Special Feature

The Electrocardiographic Diagnosis of LV Aneurysm

By William J. Brady, MD

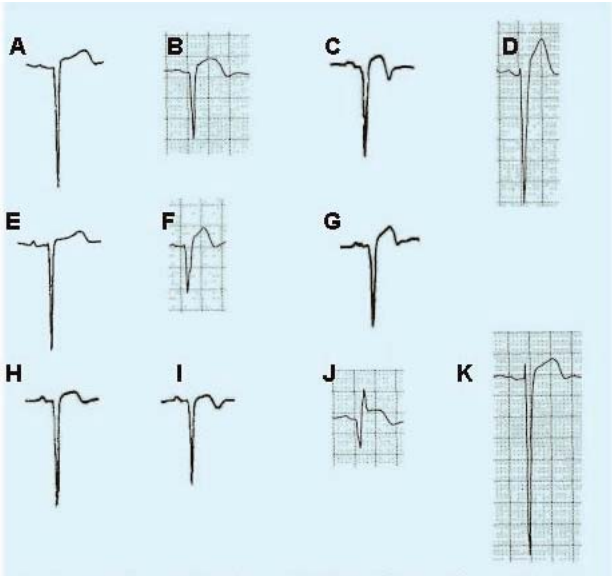
Left ventricular (LV) aneurysm is defined as a localized area of infarcted myocardium that bulges outward during the entire cardiac cycle. LV aneurysm most frequently is seen after extensive anterior wall myocardial infarction (MI), but also may be encountered following inferior and posterior wall MIs. In most instances, the LV aneurysm is manifested electrocardiographically by some degree of ST segment elevation (STE); approximately 60% of patients with completed MI who manifest persistent ST segment abnormality will demonstrate ventricular aneurysm.

Among ED patients with STE, LV aneurysm is an uncommon cause of the ST segment abnormality, accounting for only 3-4% of all patients with STE.[1,2] While STE secondary to aneurysm is rare, this pattern of STE is an important cause of error in diagnosis and management of ED chest pain patients with ST segment abnormality.[3,4]

Patients who tend to develop LV aneurysm include those individuals with a previous large MI, particularly in those instances with resultant LV ejection fractions less than 50%. A marked gender difference is encountered in these patients, with a male-to-female ratio of 4:1.[5] At coronary angiography, LV aneurysm is seen in approximately 8% of post-MI patients. The incidence of LV aneurysm following MI ranges from 3% to 15%, as determined at autopsy.[6] The vast majority of LV aneurysms result from MI, yet other etiologies are encountered, including blunt chest injury with myocardial contusion, Chagas disease, and cardiac sarcoidosis. Approximately three-quarters of LV aneurysms involve the anterior wall, and are attributable to MI due to obstruction of the left anterior descending artery.[7]

Regarding electrocardiographic pathophysiology, the ST segment abnormalities result from either an injury current originating from viable yet ischemic myocytes in the aneurysm, or from mechanical wall stress caused by traction on the adjacent, normal myocardium.[8] Pathologically, the aneurysm is characterized by transmural fibrosis—clearly delineated from, and markedly thinner than, the adjacent myocardium.[9] Associated post-mortem findings include significant multi-vessel coronary artery disease, scarred papillary muscle, pulmonary edema, pericardial thickening, and mural thrombi.

Figure. ECG Features of LV Aneurysm



Various examples of ST segment elevation resulting from LV aneurysm. The ST segment elevation may be minimal and concave in morphology; alternatively, the elevation may be pronounced with a convex shape. Q waves, in varying forms (QS and Qr configurations), also may be seen.

The diagnosis of LV aneurysm best is made via an analysis of the history (past MI) and physical examination (evidence of congestive heart failure) findings, as well as the results of cardiac imaging studies beyond the electrocardiogram (ECG). The patient may be asymptomatic, however, with a markedly abnormal ECG. Echocardiography and coronary angiography are excellent modalities that can demonstrate the anatomic and functional features of LV aneurysm. Echocardiography has a sensitivity and specificity of 93% and 94%, respectively, for detecting LV aneurysm, representing the most frequently and easily applied test for this anatomic abnormality. Coronary angiography, however, remains the gold standard for diagnosis.

Electrocardiographic Features of LV Aneurysm

The electrocardiographic features of LV aneurysm include the following: 1) STE, most often less than 3-4 mm; 2) diminished or inverted T waves; 3) QS or Qr complexes preceding the STE in the right-to-mid precordial leads; and 4) lack of dynamic change of these findings over time on ECG. *(See Figure.)* Inferior aneurysms usually have QR waves and are very difficult to distinguish from acute MI of the inferior wall. LV aneurysm is characterized electrocardiographically by persistent STE seen days to weeks after MI. STE resulting from LV aneurysm may assume varying morphologies, ranging from benign, minimally deviated, concave elevations to more ominous, pronounced, convex ST segment elevation. *(See Figure.)* Fortunately, the vast majority of LV aneurysm patients will demonstrate a concave morphology of the elevated ST segment.[2] It also has been shown that the magnitude of the STE does not correlate with the size of the aneurysm.[10]

Because LV aneurysm frequently is anterior or anterolateral in location, electrocardiographic STE typically is seen in leads I, aVL, and V_1-V_6. STE resulting from LV aneurysm of the inferior wall is noted in leads II, III, and aVF (the inferior leads); the magnitude of the STE in this distribution is less pronounced than that seen in the anterior wall aneurysm pattern. Posterior wall aneurysm is more difficult to detect electrocardiographically from the perspective of the traditional 12-lead ECG; if used, the posterior leads V_8 and V_9 may demonstrate STE.

ST segment depression, when observed in the setting of ST segment elevation elsewhere on the ECG, may represent reciprocal change and strongly suggest the presence of STE acute MI. ST segment depression, however, may also be seen in lead aVL in the patient with inferior wall LV aneurysm; caution is therefore advised.

Patients with LV aneurysm also frequently will have significant Q waves in the same distribution. In fact, it has been demonstrated that patients with larger LV aneurysms have a wider distribution of Q waves.[10] The loss of R wave deflection in standard leads also may correlate with the presence of LV aneurysm.[11] Initially, the QRS complex duration usually is normal in the setting of electrocardiographic LV aneurysm, assuming that intraventricular conduction is normal; however, the QRS complex width may increase with the age of the aneurysm.[10]

Lastly, the ratio of maximum T-wave amplitude to the QRS complex amplitude is important, with lower values occurring in association with LV aneurysm. Smith and colleagues have reported that this ratio (T-wave amplitude/QRS complex amplitude), when less than 0.36, is associated highly with LV aneurysm-related STE.[12] In the patient with STE acute MI, the T-wave frequently is quite prominent—termed the hyper-acute T-wave of acute MI. In acute MI, the ratio of the T-wave to the QRS complex, therefore, usually is a larger value, occasionally approaching 1.0. ❖

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Physician CME Questions

1. **Which of the following statements regarding the electrocardiographic diagnosis of LV aneurysm is true?**
   a. The simultaneous presence of Q waves and ST segment elevation confirms the diagnosis of LV aneurysm.
   b. Approximately 60% of post-MI patients with persistent ST segment elevation have LV aneurysm.
   c. Co-existent ST segment depression, particularly in lead aVL, rules out the possibility of LV aneurysm.
   d. A high value (approaching 1.0) of the ratio comparing the amplitude of the T wave to the QRS complex strongly suggests LV aneurysm.

2. **Magnitude of ST segment elevation on the electrocardiogram is predictive of the anatomic size of the left ventricular aneurysm.**
   a. True
   b. False

3. **Clopidogrel _____ in some sub-populations of patients with acute coronary syndrome.**
   a. is a preferred alternative to heparin
   b. is equivalent to ticlopidine
   c. may be a useful adjunct to aspirin
   d. carries a significantly higher risk of intracranial bleeding when compared to aspirin alone

4. **Certain highly sensitive D-dimer assays can be used to exclude deep vein thrombosis without concomitant lower extremity Doppler testing:**
   a. if the patient population is at low risk as determined by the Wells criteria pre-test assessment.
   b. in patients who have normal ventilation-perfusion scans.
   c. in patients with prior history of cancer or deep vein thrombosis.
   d. if the patients have normal computed tomography studies of the chest.

5. **Patients isolated for infection control are more likely to:**
   a. experience adverse events while in the hospital.
   b. die during hospitalization.
   c. have daily physician progress notes recorded in the chart.
   d. have their vital signs reported as ordered.

6. **The most reliable method for documenting the presence or absence of fever in infants younger than 3 months of age is:**
   a. tympanic canal thermometry.
   b. pacifier thermometry.
   c. rectal thermometry.
   d. temporal artery thermometry.

Answer Key: 1. b; 2. b; 3. c; 4. a; 5. a; 6. c.

CME Objectives

To help physicians:

- Summarize the most recent significant emergency medicine-related studies;
- Discuss up-to-date information on all aspects of emergency medicine, including new drugs, techniques, equipment, trials, studies, books, teaching aids, and other information pertinent to emergency department care; and
- Evaluate the credibility of published data and recommendations.

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 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 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.

Bigeminal Term Misuse

By Ken Grauer, MD

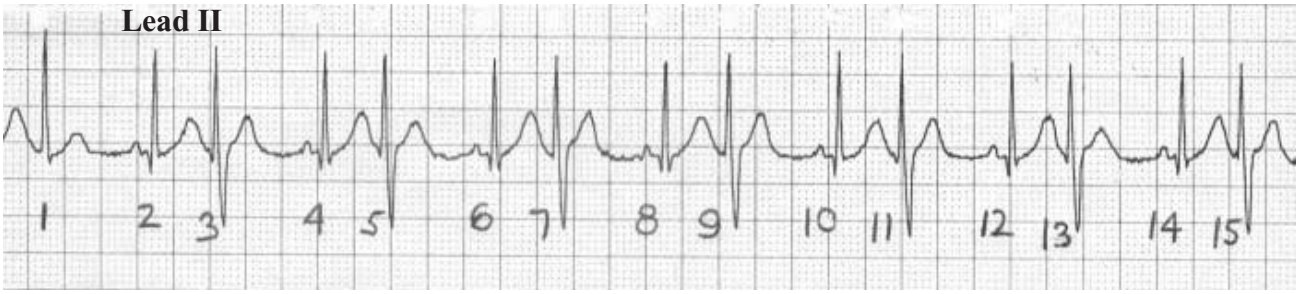

Figure. Telemetry tracing interpreted as showing a bigeminal rhythm.

Clinical Scenario: The telemetry tracing shown in the Figure was interpreted as showing "bigeminy," in that every other beat was thought to be a PVC. Do you agree with this interpretation?

Interpretation: A bigeminal rhythm is one in which every other beat is ectopic, with a fixed relation to each preceding beat. This definition is satisfied in the Figure. However, it is unlikely that the bigeminal rhythm seen in this Figure reflects the presence of premature ventricular contractions (PVCs).

Although reference to the term "bigeminy" is most often associated with a sinus rhythm in which every other beat is a PVC, it is important to appreciate that there are *other* causes of this type of patterned beating. Thus, instead of ventricular bigeminy there may be atrial or junctional bigeminy if every other beat on the tracing is early and arises from a premature atrial contraction (PAC) or premature junctional contraction (PJC).

Rather than ventricular bigeminy, one should suspect that the rhythm in the Figure represents *atrial* bigeminy. The reason the QRS complex of early occurring beats looks different than the QRS of sinus-conducted beats is that each PAC in this tracing (i.e., beats 3, 5, 7, 9, 11, 13, and 15) is conducted with aberration, a consequence of PAC occurrence during the relative refractory period.

The most helpful criterion for distinguishing between PVCs and PACs that conduct with aberration is detecting of a *premature* P wave preceding the widened and abnormal appearing early QRS complex. Sometimes identification of this premature P wave is easy, as would be the case when there is an obvious spiked deflection deforming the T wave that precedes the anomalous beat. At other times, evidence of the "telltale" premature P wave is much more subtle. Such is the case in the Figure, in which the key clue to the etiology of this bigeminal rhythm lies with inspection of the T wave of beat No. 1 on the tracing. Note how the amplitude of this T wave is smaller than that of each of the sinus-conducted beats (i.e., beats 2, 4, 6, 8, 10, 12, and 14). We suspect this may be due to the hidden presence of a premature P wave in the T wave of each of the sinus beats. Further support for a supraventricular etiology for each bigeminal beat comes from the fact that although different in appearance, the QRS complex of bigeminal beats is only minimally widened.

As is often the case, certainty of diagnosis is lacking on the basis of a single rhythm strip. Additional rhythm strips and ideally a 12-lead ECG obtained during the bigeminal rhythm would be needed to confirm QRS width and morphology of the bigeminal beats. The slightly different appearance of the QRS complex of beat No. 1 (which is taller and lacking the small initial q wave of the sinus beats) raises other questions (junctional etiology for this beat?). Nevertheless, the key point to emphasize is that not all bigeminy is ventricular, and there are several indications on this tracing that the bigeminal beats seen here are most likely to be PACs with aberrant conduction rather than PVCs. ❖

In Future Issues: Shoulder Reduction Techniques

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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

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Introduction

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

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

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

Historical Perspective

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

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

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

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

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

Rationale for Use of the ED Thoracotomy

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

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

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

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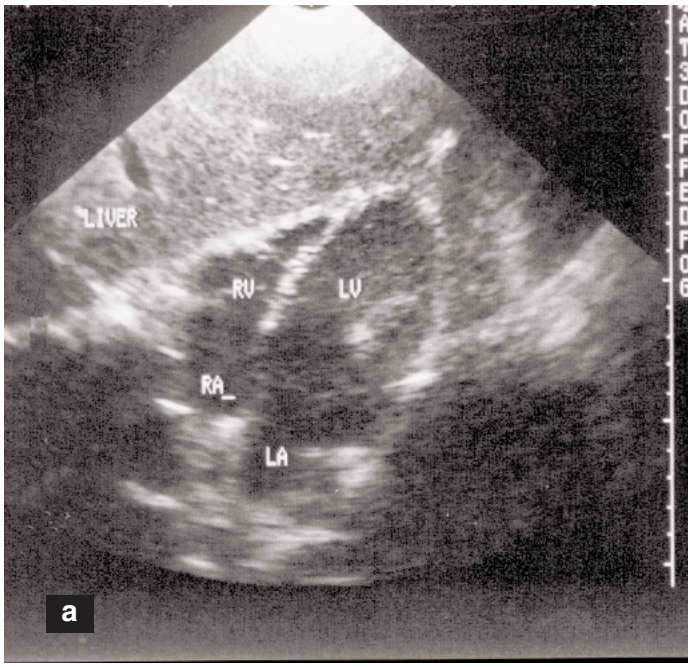
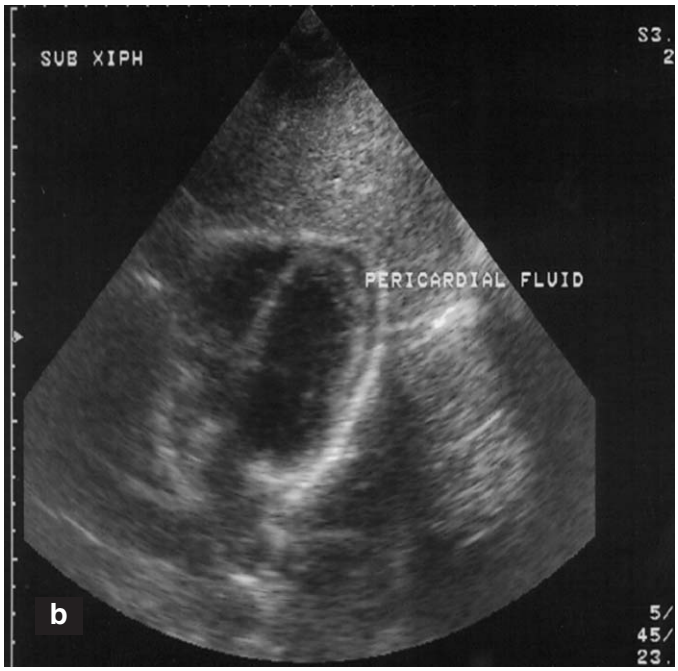
Figures 1a and 1b. The Pericardial Window





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

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

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

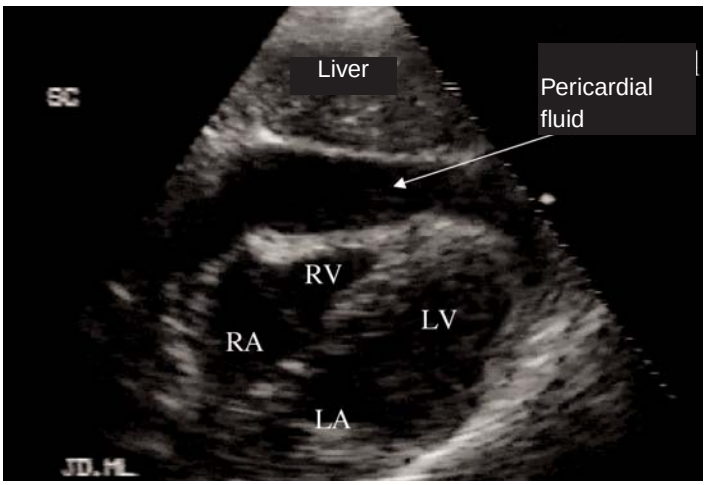
Pericardial Tamponade. Pericardial tamponade may result from gunshot wounds or stab wounds. Stab wounds commonly cause pericardial tamponade (80% of cases).[8] Pericardial tamponade can be characterized by Beck's triad (hypotension, distended neck veins, and muffled heart tones).[9] However, this triad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

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

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

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

Figure 2. Subcostal Image of Traumatic Hemopericardium



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

Table 1. Thoracotomy Equipment

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

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

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

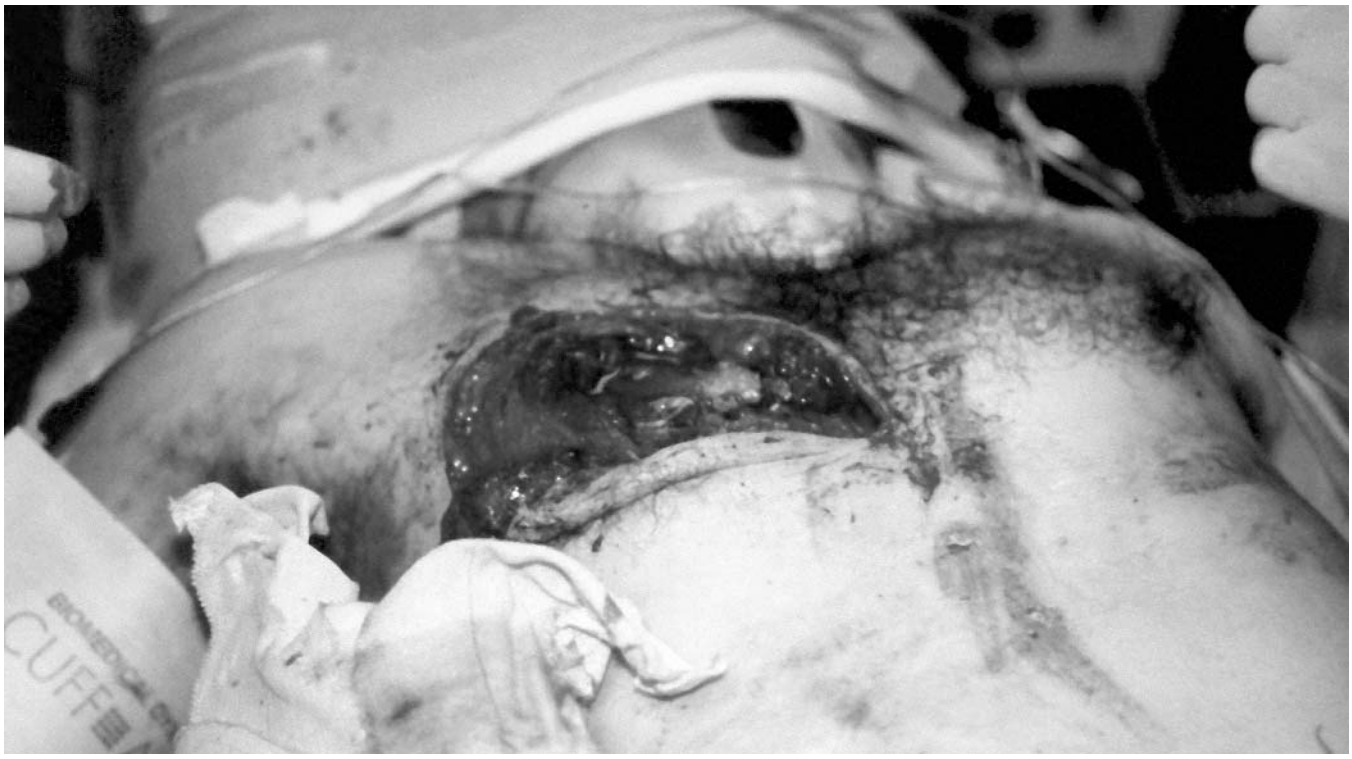
Because the chest is a large potential space, volume losses can be equally impressive and rapid. Each hemithorax can contain approximately 50% of the patient's blood volume (2.5 liters of blood for the average 70 kg person) before it becomes obvious.[3] Patients in extremis with isolated chest penetrating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

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

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

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

Figure 3. Penetrating Wound to the Chest



Patient who sustained a penetrating wound to the chest.

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

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

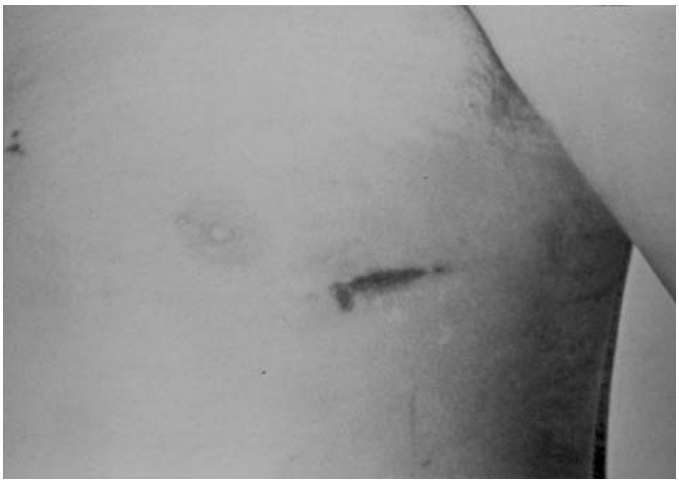
Technical Aspects of the ED Thoracotomy

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

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

The left anterolateral thoracotomy incision is the pre-

Figure 4. Isolated Stab Wound to the Chest



This male received an isolated stab wound to his chest.

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

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

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

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

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

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

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

Figure 5. Cardiac Massage

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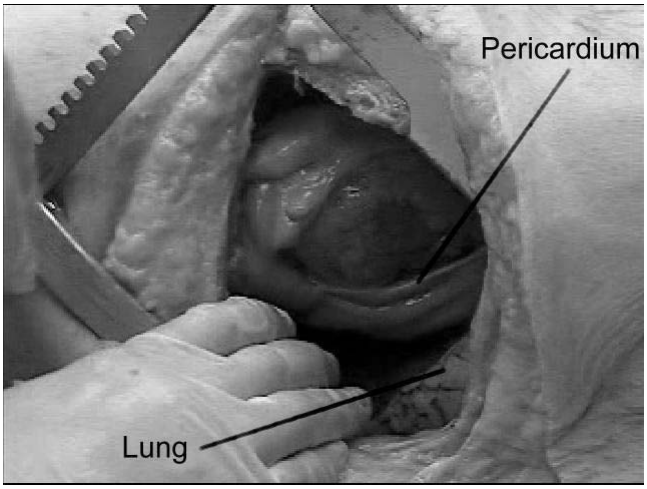
Figure 6. EDT Landmarks



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

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

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

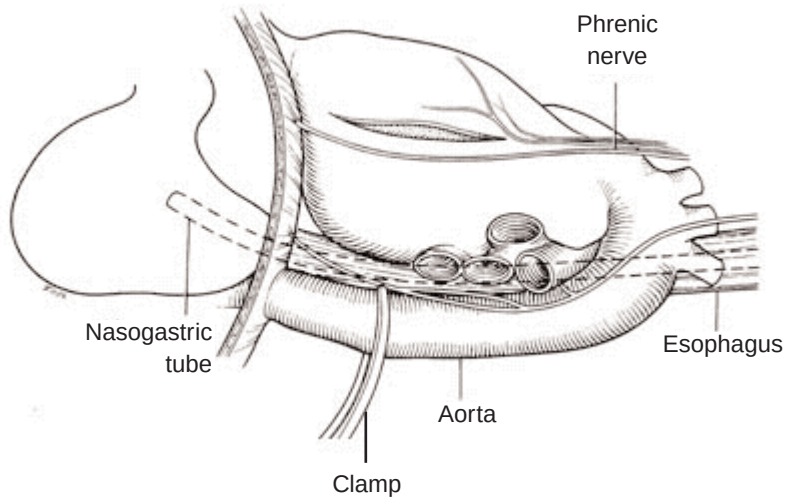
Complications

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

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

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

Figure 7. Cross-clamp of the Aorta



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Current Guidelines for the Use of ED Thoracotomy

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Conclusions

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

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

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

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

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

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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

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