WILDERNESS & ENVIRONMENTAL MEDICINE, 28, S69–S73 (2017)

TACTICAL COMBAT CASUALTY CARE: TRANSITIONING BATTLEFIELD LESSONS LEARNED TO OTHER AUSTERE ENVIRONMENTS

Treatment of Thoracic Trauma: Lessons From the Battlefield Adapted to All Austere Environments

Lanny F. Littlejohn, MD

From the Naval Hospital Camp Lejeune, Norfolk, VA.

Severe thoracic trauma in the backcountry can be a formidable injury pattern to successfully treat. Traumatic open, pneumo-, and hemothoraces represent some of the most significant patterns for which advanced equipment and procedures may help leverage morbidity and mortality, particularly when evacuation is delayed and environmental conditions are extreme. This paper reviews the development of successful techniques for treating combat casualties with thoracic trauma, including the use of vented chest seals and the technique of needle thoracentesis. Recommendations are then given for applying this knowledge and skill set in the backcountry.

Keywords: chest trauma, chest seals, needle thoracentesis, prehospital, combat trauma, pneumothorax

Introduction

Thoracic injury accounts for 25% of all trauma mortality,[1] and pneumothorax (PTX) is the single most common manifestation of intrathoracic blunt chest injury.[2] Overall the vast majority of patients with thoracic trauma does not require thoracic surgery but may require other invasive procedures to manage their injury successfully. Traumatic PTX is a leading cause of preventable mortality in multitrauma patients.[3] In combat settings, hemopneumothorax is the second leading cause of preventable combat death and the third leading cause of mortality overall.[4]

The presence of PTX reduces the physiologic negative pressure of normal respiratory mechanics and makes spontaneous breathing progressively more difficult and inefficient. In the backcountry, the risk of thoracic trauma is most likely blunt trauma from falls, but penetrating mechanisms such as gunshot wounds and animal attacks also are possible and have been reported.[5] In civilian settings, most thoracic trauma is managed with supplemental oxygen, tube thoracostomy, and mechanical ventilation when necessary. None of these are likely to be available or advisable in austere settings. Thus, wilderness medicine providers must look to practical means to treat chest trauma when resources are limited and evacuation is prolonged.

Corresponding author: Lanny F. Littlejohn, MD, Naval Hospital Camp Lejeune; e-mail: eoddmo@gmail.com.

Presented at the Tactical Combat Casualty Care: Transitioning Battlefield Lessons Learned to Other Austere Environments Preconference to the Seventh World Congress of Mountain & Wilderness Medicine, Telluride, Colorado, July 30–31, 2016.

Mechanisms and Patterns of Injury

Both penetrating and blunt mechanisms can lead to air and blood accumulating in the chest. Three conditions that lead to the most morbidity and mortality in the combat setting will be reviewed, framing treatment considerations in all austere settings.

An open PTX entails the collapse of the lung due to an associated chest wall defect that allows air to enter during inspiration and exit during expiration (eg, the "sucking" chest wound). It is possible that an open PTX may lead to respiratory compromise or even failure, depending on the size of the defect.[6] The larger the defect, the more likely that air will enter the thorax through the wound during inspiration rather than through the trachea.

A hemothorax occurs from bleeding of the chest wall, lung parenchyma, or pulmonary vessels and is more likely than a PTX to occur in the context of blunt trauma from falls.[7] Rib fractures due to thoracic trauma are in the range of 40 to 80% in blunt thoracic trauma cases.[8] Decompression of the pleura and re-expansion of the lung may allow for parenchymal bleeding to tamponade and ventilation-perfusion to be matched more closely. Thus, decompression of the pleura is beneficial in the treatment of both hemo- and pneumothoraces.

A tension PTX occurs when an injury to the lung, bronchi, or trachea allows a continuous leakage of air into the pleural space and progressively collapses the lung when air cannot escape. This is a disease entity that is different in the awake patient versus the ventilated patient and should be taught in this manner. When a casualty is under positive pressure ventilation, development of a tension PTX can be rapid. With normal pulmonary mechanics (relative negative pressure), the development takes much longer. In spontaneously breathing animal models and human case reports, the pathophysiology is hypoxia from pulmonary shunting and parenchymal collapse,[9] with decompensation occurring from progressive respiratory failure and respiratory arrest.[10,11] The disease in awake, spontaneously breathing patients is progressive and primarily respiratory in clinical appearance, with hypoxemia predominating over hypotension.[12] In ventilated patients, the fall in hemoglobin saturation indicated by pulse oximetry (SpO_2) happens abruptly and is followed quickly by hypotension.[13] Thus, hypotension from a tension PTX in the spontaneously breathing patient is a rare but ominous finding.

Diagnosis

Any patient who presents with penetrating inferior neck or thoracoabdominal trauma could have wounds that result in an open, pneumo-, or hemothorax. Those with blunt trauma with obvious rib fractures or signs of significant trauma to the thorax (abrasions, ecchymosis) also may have a pneumo- or hemothorax. Usually a patient will report pain and progressive dyspnea. Asymmetric chest movement with respirations, decreased breath sounds, subcutaneous emphysema, or paradoxical movement of a flail chest segment may be seen. Application of a portable pulse oximeter may show hypoxia and tachycardia well before a fall in blood pressure in the spontaneously breathing patient. Hypotension is not common in awake patients, and its presence should signal the hunt for other causes. Tracheal deviation and jugular venous distension are rare findings and indicate imminent circulatory collapse.

Treatment

A small PTX may not require treatment but is important to recognize in the context of positive pressure ventilation or air transport requirements, in which there will be changes in ambient pressure. Under those conditions, a simple PTX can develop tension. For an open PTX, chest seals are recommended. These are manufactured as occlusive or ventilated (one-way valve) seals. For hemo- or pneumothoraces, decompression of the pleural space must be performed to maximize oxygenation and cardiac output. This can be accomplished with needle thoracentesis (NT) or via simple thoracostomy.

CHEST SEALS

Recently, a 3-sided occlusive dressing was advocated by the Advancd Trauma Life Support Curriculum.[14] This fell out of favor after a thorough review of the literature found no evidence for, or against, this practice. At that time, occlusive chest seals were recommended for the treatment of open PTX.

Legitimate concerns were raised that chest seals might fail due to coagulation, vent malfunction, or poor skin adherence. Thus the 2 most common chest seals at the time, the Bolin (H&H Medical, Williamsburg, VA) and the Asherman (Teleflex Medical, Morrisville, NC), were tested by Arnaud et al at the US Navy Medical Research Center. In a spontaneously breathing swine model, a 1.5-cm hole was made (held open with a cut portion of a 10-mL syringe) and air was introduced to obtain a 20% fall in mean arterial pressure (average, 372 mL). A total of 1500 mL of air was introduced unilaterally with no fall in mean arterial pressure when the seals were applied. Both vented CS worked well as described and adherence was good on dry skin, but on wet/soiled skin the Bolin profoundly outperformed the Asherman.[15] The Bolin's hydrogel, layered on a rugged polyurethane disc, not only adhered well but allowed for easier relocation and "burping" of the seal if it failed to vent.

In 2012, the Committee on Tactical Combat Casualty Care (CoTCCC) began to question the practice of treating a usually nonlethal condition (open PTX) in a manner predisposed to a lethal condition (tension PTX). Kheirabadi et al looked at this question by comparing the use of vented and nonvented chest seals[16] using a model similar to that of Arnaud et al.[15] They compared the unvented HALO (Progressive Medical, Fenton, MO) to the vented Bolin. In all instances, the HALO led to a tension PTX after injection of air, whereas the Bolin did not. For both seal types, application to an open PTX led to immediate improvement in respiratory function and oxygenation. This model did not test a hemopneumothorax, however, and concerns over clotting remained.

Kotora et al would next compare 3 vented CS in a model that tested the decompression of a tension PTX and then a hemopneumothorax with a 10% autologous