Does needle thoracostomy provide adequate and effective decompression of tension pneumothorax?

Matthew Martin, MD, Steven Satterly, MD, Kenji Inaba, MD, and Kelly Blair, MD, Tacoma, Washington

BACKGROUND: Tension pneumothorax (tPTX) is a common and potentially fatal event after thoracic trauma. Needle decompression is the currently accepted first-line intervention but has not been well validated. The purpose of this study was to evaluate the effectiveness of a properly placed and patent needle thoracostomy (NT) compared with standard tube thoracostomy (TT) in a swine model of tPTX.

METHODS: Six adult swine underwent instrumentation and creation of tPTX using thoracic CO₂ insufflation via a balloon trocar. A continued 1 L/min insufflation was maintained to simulate an ongoing air leak. The efficacy and failure rate of NT (14 gauge) compared with TT (34F) was assessed in two separate arms: (1) tPTX with hemodynamic compromise and (2) tPTX until pulseless electrical activity (PEA) obtained. Hemodynamics was assessed at 1 and 5 minutes after each intervention.

RESULTS: A reliable and highly reproducible tPTX was created in all animals with a mean insufflation volume of 2441 mL. tPTX resulted in the systolic blood pressure declining 54% from baseline (128–58 mm Hg), cardiac output declining by 77% (7–1.6 L/min), and equalization of central venous pressure and wedge pressures. In the first arm, there were 19 tPTX events treated with NT placement. All NTs were patent on initial placement, but 5 (26%) demonstrated mechanical failure (due to kinking, obstruction, or dislodgment) within 5 minutes of placement, all associated with hemodynamic decline. Among the 14 NTs that remained patent at 5 minutes, 6 (43%) failed to relieve tension physiology for an overall failure rate of 58%. Decompression with TT was successful in relieving tPTX in 100%. In the second arm, there were 21 tPTX with PEA events treated initially with either NT (n = 14) or TT (n = 7). The NT failed to restore perfusion in nine events (43%), whereas TT was successful in 100% of events as a primary intervention and restored perfusion as a rescue intervention in eight of the nine NT failures (88%).

CONCLUSION: Thoracic insufflation produced a reliable and easily controlled model of tPTX. NT was associated with high failure rates for relief of tension physiology and for treatment of tPTX-induced PEA and was due to both mechanical failure and inadequate tPTX evacuation. This performance data should be considered in future NT guideline development and equipment design. (J Trauma Acute Care Surg. 2012;73: 1412–1417. Copyright © 2012 by Lippincott Williams & Wilkins)

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physiology or even death. Beyond these well-described technical limitations of NT, it is unknown whether these devices perform adequately and reliably even when properly placed and patent. The purposes of this study were to develop a clinically relevant and reproducible large animal model of severe tPTX and to test the efficacy of appropriately placed standard NT devices for relieving tension physiology.

**MATERIALS AND METHODS**

The experiments were performed in adherence to the Guidelines on the Use of Laboratory Animals of the National Institutes of Health. Approval for animal research was obtained through the local Institutional Animal Care and Usage Committee and the federal authorities for animal research. Six Yorkshire swine were used for this experiment. Animals were purchased from an approved swine research facility of the US Department of Agriculture and housed following standardized veterinary care. Animals were fasted for 12 h before surgery with restrictions to water the morning of surgery. Each animal was premedicated intramuscularly with a cocktail of ketamine, telazol, and xylazine (50 mg/mL of each drug) dosed at 2.2 to 2.5 mg/kg. Once sedated, each animal was intubated, and general anesthesia was induced and maintained using isoflurane. Pancuronium dosed at 0.1 mg/kg intravenously was administered, followed by 0.2 mg kg\(^{-1}\) h\(^{-1}\) of continuous infusion. Minute ventilation was adjusted to maintain \(\text{PaCO}_2\) between 35 and 45 mm Hg, and \(\text{FiO}_2\) was titrated to maintain an oxygen saturation level higher than 90%. During all experiments, the animals were ventilated using a volume control mode with a tidal volume of 10 mL/kg and the high airway pressure alarm inactivated. After the induction of anesthesia, the right carotid sheath was dissected, and the right common carotid artery and the internal jugular vein were isolated for access. The common carotid artery was then cannulated and connected to a transducer for continuous arterial pressure monitoring. At the same time, the right internal jugular vein was accessed using a 9F introducer sheath and a pulmonary artery catheter inserted for continuous measurement of cardiac parameters.

A midline celiotomy was then performed, and a urinary catheter was inserted into the dome of the bladder. A small defect was then created in the central tendinous portion of the right hemidiaphragm and a 0-Prolene purse-string suture placed around the margins of the defect. A 10-mm balloon-tipped laparoscopic trocar (Covidien, Mansfield, Mass) was then placed through the diaphragmatic defect and secured in place by inflation of the balloon tip and cinching down the foam cuff as well as securing the previously placed purse-string suture around the trocar shaft. A standard 28F chest tube was then placed in the right thoracic cavity through a 1-cm right lateral chest wall incision and secured with a 0-silk purse-string suture. The free end of this tube was clamped with a large hemostat. The thinnest point on the right anterior chest wall was identified by palpation and marked to guide the placement of the NT. The abdomen was then closed with towel clamps. The correct positioning of the tubes and lines was confirmed by chest x-ray (Fig. 1A). Once the instrumentation was completed, vitals were allowed to stabilize for 30 min, and baseline measurements were taken, including mean arterial pressure, heart rate, central venous pressure (CVP), cardiac index, pulmonary artery occlusion pressure (PAOP), arterial blood gas, and lactate.

**Arm 1—Tension Physiology**

All tension events in both study arms were created using thoracic insufflation with CO\(_2\) gas delivered through the trans-diaphragmatic trocar. A standard laparoscopic insufflator and pressure monitoring system was used to control the insufflation pressure and monitor the volume of gas used. Insufflation was performed in 5-mm increments at a flow rate of 5 L/min, with 2 minutes of stabilization between pressure increases. Vital signs were continuously recorded, and CVP, PAOP, and cardiac output were assessed at 1-minute intervals. Tension physiology was defined as a drop of more than 50% of the cardiac output from baseline value. Once tension was achieved (Fig. 1B), a randomized intervention of either NT or tube thoracostomy (TT) was performed. NT involved the percutaneous placement of a standard 3.25-inch 14-gauge angiocatheter into the right thoracic cavity at the...
previously marked optimal point. All NT were placed at a 90-degree angle from the chest wall and inserted to the hub of the catheter, with adequate initial placement confirmed by observing continuous flow of gas from the thoracic cavity. The placement of TT was simulated by releasing the clamp on the previously placed chest tube. After intervention, the insufflation flow was decreased to a rate equivalent to the clamp on the previously placed chest tube. After intervention, the insufflation flow was decreased to a rate equivalent to the clamp on the previously placed chest tube. After intervention, the insufflation flow was decreased to a rate equivalent to the clamp on the previously placed chest tube.

Arm 2—Tension-Induced PEA Arrest

After the completion of the first arm in each animal, a 5-minute stabilization period was given, and a new set of baseline hemodynamic data was obtained. Tension-induced pulseless electrical activity (PEA) was then induced by continuous insufflation at a rate of 10 L/min until complete loss of the arterial line pulsatile wave form was obtained. Insufflation flow was then stopped, PEA was maintained for 30 seconds, and then the randomly assigned intervention of either NT or TT was performed as previously described. After the intervention, insufflation flow was resumed at a rate of 40 mL kg⁻¹ min⁻¹ to simulate a moderate continuous air leak. Each intervention was assessed for primary patency and adequacy of tension relief at 1 and 5 minutes after intervention. In the event of hemodynamic collapse with NT before the 5-minute end point, the TT was opened as a rescue intervention. In the event of an NT failure, the needle was inspected both in vivo and after removal to identify any kinking or other source of luminal obstruction. No additional manipulation of the needle or animal was performed during the 5-minute observation period after the initial NT placement. A 5-minute stabilization period was given between each tension event, and this process was repeated for a maximum of six tension events per animal.

RESULTS

A total of seven animals were used for this study. One developed an unstable cardiac arrhythmia and died during the initial thoracic insufflation, leaving six animals for complete analysis. Five animals survived through all phases of the study, and one animal died during the last round of arm 2. A reliable and highly reproducible tPTX was created in all animals (Fig. 1B), with a mean insufflation volume of 2441 mL and at a mean pressure of 15 mm Hg; tPTX resulted in the systolic blood pressure declining 54% from baseline (128–58 mm Hg, p < 0.01) and the cardiac output declining by 77% (7 L/min to 1.6, p < 0.01). There was a steady increase of CVP and simultaneous decline in the PAOP with progressive insufflation pressure, with a precipitous decline in blood pressure and cardiac output once the CVP and PAOP reached a point of equalization (Fig. 2). Although there was a major effect on hemodynamics, there was a minimal effect on pulmonary function and gas exchange because of the high insufflation pressure allowed to ventilate the animals despite the tension physiology. Oxygen saturation levels remained higher than 92%, with PaO₂ higher than 60 mm Hg in all animals throughout the experiments.

In the first arm, there were 19 tPTX events treated with NT placement and 10 tPTX events treated with TT. In all events treated with TT, there was immediate relief of tension physiology, with the restoration of systolic blood pressure and cardiac output to baseline levels within 30 seconds. Among the 19 tension events treated with NT, all needles were patent on initial placement and at 1 minute. However, 5 NTs (26%) demonstrated mechanical failure (due to kinking, obstruction, or dislodgment) within 5 minutes of placement, all associated with hemodynamic decline that was immediately

Data and Statistical Analysis

All data were collected and entered into separate spreadsheets for each study arm. Each intervention was classified as a success or failure, and the reason for failure (if determined) was noted. Categorical data were analyzed using Fischer's exact test and continuous data with either Student's t test or analysis of variance. Within-animal comparisons of pretension and posttension hemodynamics were performed using a paired-sample t test, and between-group comparisons were performed using a repeated-measures analysis of variance. All statistical analysis was analyzed using the Statistical Package for the Social Sciences version 18.0 (IBM Inc., Armonk, NY).
rescued with unclamping the chest tube. Among the 14 NTs that remained patent at 5 minutes, 6 (43%) failed to relieve tension physiology for an overall failure rate of 58% (Fig. 3).

In the second arm, there were 21 tPTX with PEA events treated initially with either NT (n = 14) or TT (n = 7). The NT failed to restore perfusion in nine events (64%), whereas TT was successful in 100% of events as a primary intervention (Fig. 4). In eight of the nine failures of NT, perfusion was immediately restored with TT as a rescue intervention. The comparison of only the NT and TT successes demonstrated a mean time to restore perfusion of 5 seconds for the TT versus 35 seconds for the NT (p < 0.01).

**DISCUSSION**

PTX from blunt or penetrating trauma represents a significant problem with a reported incidence of up to 10% and 50%, respectively. Population-based data have found that PTX is present in one of five victims of major trauma and is associated with worse hemodynamics and outcomes.10 PTX can rapidly progress to a life-threatening tension physiology, with death due to either hypoxia or cardiac tamponade. Previously, battlefield trauma data suggested a 33% combat-related death rate from tPTX trauma.11 The immediate recognition and treatment of tPTX remains one of the single most important life-saving interventions in the treatment of chest injuries in both civilian and combat settings and is a key part of all trauma training programs for prehospital and in-hospital personnel.

tPTX is a life-threatening condition that develops when air is trapped in the pleural cavity under positive pressure, displacing mediastinal structures and compromising cardio-pulmonary function. Pleural trauma results from a disruption of the air-filled portion of the lung from fragments, blast, or rib injury with the entrapment of air in the intrathoracic pleural space. Without an evacuation route, trapped air results in impaired venous return to the heart, a shift in the heart toward the noninjured side, decreased cardiac function, collapse of the lung, blunt diaphragmatic excursion on the affected side due to an increase in intrathoracic pressure, impaired oxygenation and ventilation, and hypotension.12 Consequently, the patient experiences shortness of breath, hypotension, and complete cardiovascular collapse if left untreated. In addition, in most cases, there is likely an ongoing leakage of air from the injured lung, so any proposed intervention must not only decompress the accumulated air but also adequately control the ongoing air leak. Our model was designed to simulate just such a scenario of a patient with an ongoing slow air leak being ventilated with positive pressure during either the prehospital transportation or the early emergency department phase of care.

Despite the relative frequency of tPTX and the importance of rapid identification and treatment, there is scarce controlled scientific data on the underlying physiology of this disease process. One of the goals of this study was to create a valid and easily reducible large animal model of tPTX. Another goal was to describe the characteristic central hemodynamic changes associated with tension physiology. Our model used commonly available laparoscopic instrumentation and equipment that allowed for the creation of a highly reliable model of tension physiology based on thoracic CO2 insufflation. One of the distinct advantages of this model is the ease of titration of the insufflation pressure, which allows for maintenance of a consistent pleural pressure and for the induction of a wide range of physiologic tension changes from mild to complete PEA arrest. This is in comparison with several other models that have used static and intermittent bolus air infusions into the thoracic cavity.12,13 The hemodynamic response in our animal model recreated the classically described sequelae in humans, including equalization of right and left-sided filling pressures followed by precipitous cardiac collapse. Although a previous model of tPTX in swine found that hypoxemia caused death before hemodynamic collapse, this model differed from ours in using only 21% FiO2, and it was unclear whether consistent tidal volumes were maintained.12 In contrast, our study used 100% FiO2 and maintained a constant tidal volume resulting in sustained adequate oxygenation that allowed us to assess the hemodynamic effect of tPTX to the point of death.

To mitigate the grave consequences of a tPTX, traditional treatments include immediate decompressive NT or TT placement. Historically, NT was used for the rapid treatment of tPTX and lauded by advocates for its plethora of advantages.14 Needles were ubiquitous in prehospital field emergency medical technician kits, emergency departments, and clinic settings.15 Needle placement requires less technical expertise than an emergent TT and uses low-cost and widely available angiocatheters. Currently, an intravenous catheter (14, 16, or 18 gauge and at least 2–3 inches in length) inserted in the midclavicular line at the second intercostal space is advocated by both civilian and military protocols.1,16 Although the use of NT as the primary emergent intervention for tPTX has become widely adopted, particularly in the prehospital or medical evacuation setting, data on the true indications, optimal technique, and efficacy remain ill defined and increasingly debated.

Multiple studies on the efficacy of NT for tension PTX challenge its effectiveness. Disadvantages cited in several studies demonstrated high incidence of inadequate placement due to catheter length, kinking due to flexible material, or occlusion by clots.6,16,17 Improper placement due to equipment inadequacy, technique, or site has been noted in multiple descriptive studies at trauma centers.5,17–19 One
particular concern that has been echoed by multiple authors is the relative thickness of the chest wall at the preferred site for NT. Both autopsy- and CT scan–based studies have demonstrated that more than 50% of standard length NT devices would be expected to be unable to penetrate the thoracic cavity when placed in the second intercostal space at the midclavicular line.4,17,20 A recent study by Inaba et al21 has suggested that the lateral chest wall might be the optimal site for NT, but even this location would have a significant failure rate, particularly in larger patients. In addition, this site is impractical for military applications, as standard body armor makes this location inaccessible in an emergent situation. These studies also assume perfectly perpendicular placement at the correct anatomic location, so they likely underestimate what the true failure to penetrate rate would be in clinical practice. The accumulation of these data prompted both civilian and military trauma systems to adopt a strategy of utilizing longer catheters, but there remains no data on whether simply increasing the length of the catheter will decrease the NT failure rate.6,7

One problematic assumption that is made in many of these studies and in current Advanced Trauma Life Support and Prehospital Trauma Life Support guidelines is that NT catheters are effective when properly placed and patent. Our animal model was created to examine this assumption and to assess the efficacy of NT when ideally placed and patent in a controlled setting. It is surprising that for such a widely used and relied upon technique, there exists very little scientific data confirming the safety of efficacy of NT. Isolated case reports or small series have described persistent tension physiology despite NT decompression, but larger more reliable experiences are lacking.22,23 The practical realities and limitations of emergent trauma care make it difficult to impossible to reliably identify the true incidence of tPTX and success rate of NT in the trauma population, and thus we must rely on animal models to provide this critically important information. Our results raise significant concerns about how effective these devices are in two separate clinical scenarios: (1) the patient with tension-induced hemodynamic collapse and (2) the patient in PEA arrest due to tPTX. Although TT was universally successful in treating these two scenarios, NT demonstrated significantly lower success rates of 42% and 36%. The only previous similar study for comparison that we identified was by Holcomb et al24 and analyzed the efficacy of NT versus TT in a swine model of hemopneumothorax. They found 100% success rate for both NT and TT at relieving tPTX for up to 4 hours. However, they used a much more mild definition of tPTX (20% decline in cardiac output), a significantly slower rate of insufflation (3 mL kg⁻¹ min⁻¹), and spontaneously ventilating animals and placed all catheters in the midaxillary line. In addition, they did not study the efficacy of these devices for relieving full PEA arrest. Rather than conflicting, these results can be interpreted as demonstrating that NT may be effective in lesser degrees of tPTX under controlled conditions but does not perform as well in a more severe model of tPTX compared with standard TT.

We found that even when adequately placed and under controlled conditions with no catheter manipulation or animal movement, many of these NT catheters failed to adequately maintain relief of tension physiology for the short period of 5 minutes. One source of failure was the classically described mechanical issues with these catheters, including kinking, luminal obstruction from tissue or clot, and dislodgement likely due to chest wall motion with ventilation. However, another and potentially more concerning issue is that the source of failure was the inadequacy of the NT to relieve tension physiology or restore perfusion despite remaining patent and functional. This is likely related to the small bore and flow limitations induced by these devices, which was inadequate in the face of a large tPTX with a slow ongoing air leak. Although they likely would have performed better in a model that did not include an ongoing air leak, we feel that this model is likely more clinically relevant to an actual trauma patient with tPTX due to major chest trauma.

This study has several significant limitations that should be considered. This is an animal model with obvious anatomic differences compared with human trauma victims; in addition, there was no concomitant chest wall or lung injury as would likely be seen in real trauma victims. It is unclear to what degree this model would be anatomically and physiologically reproducible in a human model. The TT was prepositioned and the placement was simulated by simply unclamping the tube; hence, the true efficacy and incidence of malposition or other placement problems cannot be inferred from these data. Our model used continuous thoracic insufflation with CO₂, which may not reflect the exact physiology induced by a room air or oxygen-supplemented tPTX. The addition of a continuous air leak provided a more challenging situation for the studied interventions to overcome, and our chosen rate of air leak might overestimate or underestimate what would be seen in the setting of an actual lung laceration with air leak. Finally, all tension events were created in the right hemithorax, and results or hemodynamic patterns may be different if tPTX develops on the left side.

With the data from this study in addition to the large accumulation of literature documenting the concerning limitations and failure rates associated with NT, we believe that a re-evaluation of the role of NT in the emergent treatment of tPTX or traumatic arrest due to suspected tension physiology is in order. At a minimum, the published guidelines and training programs should give a better appreciation of the limitations of these devices and an awareness of the problems of malpositioning or device failure. Although the recently recommended adoption of longer catheters is becoming widely accepted, it has been our observation that these devices are still limited by their flimsy construction and may even have a higher tendency to kink due to the increased length. Alternatively, NT could be changed from a therapeutic maneuver to a purely diagnostic maneuver, with a more definitive intervention mandated if there is a return of air on the initial placement of the angiocatheter. Although several limited series have demonstrated high efficacy and acceptably low complication rates with prehospital chest tube placement,15,25,26 this would need further study before advocating more widespread application. Another option that is being actively investigated is the use of larger bore and stiffer catheters that in theory should perform better than standard NTs but not
carry the higher risk of TT. Preliminary data with these devices are encouraging, but further study is required to validate their role in the treatment of PTPX.

A final option would be to abandon NT altogether in favor of other more reliable options. However, this would need careful consideration and deliberation to weigh the potential benefits of a more reliable intervention with the obvious downsides of introducing a more technically difficult and invasive procedure with a likely greater complication profile. As our study demonstrated, although NT was not nearly as effective as TT, it did relieve tension physiology or PEA arrest in approximately one third of the animals. Thus, it should still be considered as a rescue option for tension physiology when other means are unavailable or would result in significant delay. However, we believe that the significant failure rate that has been reported for NT should be recognized and included in the teaching and hands-on training programs such as Advanced Trauma Life Support, Prehospital Trauma Life Support, and combat medic training programs, and continued efforts at developing safe and effective alternatives to NT are warranted.

AUTHORSHIP

DISCLOSURE
The authors declare no conflicts of interest.

REFERENCES