The New Year is here. This will be the 1st issue where we have gone to a quarterly publication schedule. We are very lucky to have additions to our editorial staff. Mary Claire O’Brien, MD is now managing editor. She is an emergency physician and completed an EMS fellowship in Philadelphia. We also now have an editorial board which has also helped review the articles included in this issue.

We have quite a few articles this month. NCCEP has approved a new interfacility protocol for the transfer of patients receiving thrombolytics for stroke. Darrell Nelson has written a nice summary of the new protocol and its rationale. Mary Wittler, an emergency physician and a toxicologist has written a nice article on portable devices for detecting carbon monoxide in a patient’s blood. A huge issue in the recent literature has been the new research done on therapeutic hypothermia. I have tried to put together a summary of the most recent studies addressing this. Kim Askew and Brian Hiestand, both emergency physicians have also put together a great case report and EKG for a patient with pericarditis. Chris Watford, EMT-P and recent 2 time winner of the NC state paramedic competition, has also summarized some recent guideline changes for pelvic trauma. In addition we are lucky that we have an article written by 3 trauma surgeons at Carolina Medical’s Center in Charlotte on needle decompression. The authors are Paul Colavita, Ronald Sing, and Peter Fischer. This is a high risk procedure so please read this article carefully. Mike Clumpner has also put together a great review on how EMS should approach an active shooter situation. Mike is a fire captain in Charlotte, a tactical medic, and is currently completing his PhD in homeland security policy. Tammy Rush, RN and Randy Edwards, RRT have written a great summary of pediatric asthma. Given how widespread non-invasive positive pressure ventilation has become John Gaillard and emergency physician and ICU doc has put together a review of CPAP for us.

I am also trying to put together a twitter feed at @NorthCarEMS in order to share any pertinent NC EMS news sooner.

I also want to take a moment to remind everyone that the Office of EMS is now taking public comments on the proposed rules that will regulate both EMS and trauma
centers in the state of NC. The list of meetings can be found at this link: http://www.ncems.org/pdf/RulesProjectPublicNotice12-10-13.pdf

As always please feel free to submit articles or questions. I can be reached at jwinslow@wakehealth.edu. Also remember that this publication does not represent the views, positions, or opinions of the NC Office of EMS.

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For this issue we also extend a special thanks to Simon Mahler, MD, Justin Sempsrott, MD, and Andrew Asimos, MD who in addition to the people listed above helped with peer review. All the articles have been peer reviewed.
Stroke is now recognized as a time dependent condition where EMS can have a profound impact upon morbidity and mortality. Prompt recognition of stroke and early activation of the 911-system can quickly place a pre-hospital professional in the position to rapidly identify stroke, assess time of onset and triage to the appropriate facility for intervention.

Stroke costs North Carolina over 1 billion dollars and results in 4000 – 5000 deaths annually. North Carolina is part of the Stroke Belt and has the 7th highest stroke mortality in the nation. In both North Carolina and the nation stroke remains the leading cause of disability.  

Similar to major trauma and STEMI, regionalization of stroke care has positively impacted morbidity and mortality. Appropriate triage of the stroke patient now results in increased utilization of the thrombolytic Alteplace or tPA. EMS is called upon to treat these patients when transfer to a stroke center is required during or following administration of tPA. While some agencies have developed policy to direct care in these situations, most agencies have performed monitoring of tPA without a specific protocol.

In late 2011 several members of the stroke community approached the NCCEP EMS Committee concerning the possibility of protocol development. Unfortunately this came at a time when the 2012 NCCEP document was being finalized and EMS agencies were actively adopting and training. Given these circumstances the protocol was tabled until all agencies had adopted the 2012 document.

Recently an Alteplace (tPA) Transfer protocol has been developed and vetted across several stroke stakeholders in North Carolina. The new protocol is now approved by the NCCEP EMS Committee after much review, comment and revision and is included in this article. The protocol is intended to be optional and flexible for any changes required at the local level. Agencies are encouraged to utilize this protocol only after reviews by your local stroke capable hospital and regional stroke center to ensure coordinated stroke care.
Altelplace (tPA) is a controversial subject within the emergency medicine community and opinions vary widely by individual physicians. While much time can be invested reviewing the available literature basically patients receiving tPA have the following expected outcomes: 12 % improve, 6 % worsen (typically due to bleeding) and the remaining patients have little change.

Before incorporating this protocol into your armamentarium, system wide training on the utilization of tPA should be undertaken. Review and hands-on practice with IV infusion pumps used by your stroke capable hospital is encouraged, as they are not widely used by EMS. Several strategies in tPA administration are available including “drip and ship,” having a RN accompany the patient along with EMS during transport for infusion monitoring and completing the infusion at the stroke capable hospital before transfer is initiated. Remember the most important historical information you can gather is the exact time of symptom onset or when the patient was last seen normal. Intervention is wholly dependent upon this time and you may be the only person capable of determining that moment before the window for intervention closes.

References:
**Activase / t-PA IV Transfer**

**Signs / Symptoms consistent with CVA**
- Transfer of patient
- Patient receiving t-PA (Alteplase)

**Obtain / Document VS prior to transport**

**SBP ≥ 185**
- **YES**
- **NO**

**Assess / Document GCS / Pupil exam**
- Nothing by mouth / NPO
- Prehospital Stroke Screen
- Maintain SaO2 ≥ 94%
- Cardiac Monitor
- Verify Activase / t-PA bolus dose
- Verify total dose
- Document time infusion initiated
- Document estimated time of dose completion
- Reassess VS every 15 minutes
- Repeat neurological exam every 15 minutes
- Obtain BP readings in limb without infusion

**SBP ≥ 180**
- **YES**
- **P**
- **NO**

**Labetalol 10 mg IV**
- Contact Medical Control or Receiving Facility

**Remove drip chamber from Activase / t-PA bag**
- Spike Activase / t-PA drip chamber to NS bag
- Restart infusion to complete medication remaining in IV tubing

**Stop IV Activase / t-PA infusion**
- Reassess VS every 15 minutes
- Repeat neurological exam every 15 minutes
- Obtain BP readings in limb without infusion

**Notify Destination or Contact Medical Control**

**Patient must be stabilized BEFORE transport initiated**
- **SBP < 185**
- **DBP < 110**

**SBP ≥ 180**
- **YES**
- **P**
- **NO**

**Stop Anti-hypertensive agent**
- Continue Activase / t-PA infusion

**SBP < 140**
- **YES**
- **P**
- **NO**

**Labetalol: Increase 2 mg/min every 10 minutes**
- Maximum 8 mg/min.
- Or
- **Nicardipine: Increase 2.5 mg/hr every 5 minutes**
- Maximum 15 mg/hr
- Titrate to SBP < 180
- DBP < 105
- Titrate to SBP < 180
- DBP < 105

Any of following:
- Severe headache
- Worsening of neurological exam
- Nausea / Vomiting
- Allergic reaction
- Excessive bleeding

Notify Destination or Contact Medical Control
Activase / t-PA IV Transfer

Hypertension During t-PA Infusion:
When SBP is ≥ 180 and DBP is ≥ 105 Labetalol 10 mg IV push may be administered. Medical Control or receiving Stroke Center (preferred) should be contacted for further orders concerning blood pressure management after this initial dose. This assumes anti-hypertensive medications are not infusing from transferring hospital.

Nicardipine (Cardene):
Common antihypertensive which may be initiated by transferring facility. Used for blood pressure management.
Calcium channel blocker.

<table>
<thead>
<tr>
<th>Common reactions:</th>
<th>Adverse reactions:</th>
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<tbody>
<tr>
<td>Headache</td>
<td>AV Block</td>
</tr>
<tr>
<td>Peripheral edema</td>
<td>MI</td>
</tr>
<tr>
<td>Dizziness</td>
<td>Ventricular Tachycardia</td>
</tr>
<tr>
<td>Nausea / vomiting</td>
<td>Angina exacerbation</td>
</tr>
<tr>
<td>Tachycardia / palpitations</td>
<td>Allergic reactions</td>
</tr>
</tbody>
</table>

Target SBP and DBP during t-PA administration:
A SBP of 170 – 180 and a DBP of 95 – 105 is a reasonable target range. Main target is to keep SBP < 180 and DBP < 105 during and following tPA administration. While aggressive blood pressure control is warranted during t-PA administration, episodes of hypotension give rise to increased morbidity and mortality. Be cautious in titrating antihypertensive medications with the idea that slow and steady reduction is key. Wide and quick swings in blood pressure can worsen condition.

Hypotension:
Hypotension should be aggressively treated as this can worsen cerebral perfusion pressure and outcomes.

Unless contraindicated keep Head of Bed elevated 20 to 30 degrees.

Pearls
- This protocol is optional and given only as an example. Agencies may and are encouraged to develop their own.
- This protocol is intended for interfacility transfer patients only. Medication must be started at initial treating hospital.
- Recommended Exam: Mental Status, HEENT, Heart, Lungs, Abdomen, Extremities, Neuro
- Items in Red Text are key performance measures used in protocol compliance.
- The Reperfusion Checklist should be completed for any suspected stroke patient.
- Onset of symptoms is defined as the last witnessed time the patient was symptom free (i.e. awakening with stroke symptoms would be defined as an onset time when the patient went to sleep or last time known to be symptom free.)
- The differential listed on the Altered Mental Status Protocol should also be considered.
- Be alert for airway problems (swallowing difficulty, vomiting/aspiration).
- Hypoglycemia can present as a localized neurologic deficit, especially in the elderly.
- **Medication dosing safety:**
  When IV Activase / t-PA dose administration will continue en route, verify estimated time of completion. Verify with sending hospital that excess Activase / t-PA has been withdrawn from the bottle and wasted. This ensures the bottle will be empty when the full dose is finished. For example, if the total dose is 70 mg, then 30 cc should be withdrawn and wasted since a 100 mg bottle of Activase / t-PA contains 100 mL of fluid when reconstituted. Sending hospital should apply a label to Activase / t-PA bottle with the number of mL of fluid that should be in the bottle in case of pump failure during transit.
- **Allergy / Anaphylaxis:**
  Activase / t-PA, is structurally identical to endogenous t-PA and therefore should not induce allergy, single cases of acute hypersensitivity reactions have been reported.
  **Angioedema:**
  Rapid swelling (edema) of the dermis, subcutaneous tissue, mucosa and submucosal tissues. Typically involves the face, lips, tongue and neck.
  Almost always self limiting but may progress to interfere with airway / breathing so close monitoring is warranted. Utilize the Allergy / Anaphylaxis Protocol as indicated and also for angioedema. Infusion should be stopped. Give all medications related to the Allergy / Anaphylaxis Protocol by IV route only as the stroke / CVA patient should remain NPO.
Focus on Carbon Monoxide

Mary Wittler, MD

Editor’s note: Dr. Wittler is boarded in toxicology and is an emergency physician.

Winter is upon us and so is the silent killer, carbon monoxide (CO). CO is an odorless, colorless gas that can pass through porous material of finished walls and ceilings to seep from room to room or dwelling to dwelling in a multi-unit building. Approximately 500 deaths occur yearly in the United States from non-fire related CO exposures with the majority occurring in winter. CO causes nonspecific symptoms that require a high index of suspicion to make the diagnosis.

Carbon monoxide (CO) is generated during the combustion of fossil-fuel by any piece of equipment or machinery with a combustion engine. Home sources include malfunctioning fuel-burning appliances, such as gas furnaces, ranges, or water heaters. In the winter, people get into trouble by bringing charcoal grills, gas space heaters, or fires indoors. Fireplaces with blocked or faulty chimneys or flues can cause dangerous amounts of CO accumulation. Additionally, during power outages, portable generators operated inside a house, crawlspace, or garage cause deaths every year. North Carolina law requires carbon monoxide detectors in every dwelling having a fossil-fuel burning heater or appliance, fireplace, or an attached garage. However, noncompliance and lack of education about the dangers of CO are problematic.

CO binds to hemoglobin and blocks the ability of hemoglobin to carry oxygen. Because of the vague symptoms, CO can be misdiagnosed as an influenza-like illness or gastroenteritis. Common symptoms include headache, nausea, dizziness, ataxia, and weakness. Prolonged exposure or higher levels may result in confusion, seizures, loss of consciousness, or coma. The gold standard for diagnosis is measuring the blood CO (COHg) level using a co-oximeter. Since COHg levels decline after removal from exposure, normal or slightly elevated COHg levels do not rule out exposure. Pulse oximetry is of limited value because COHg is misinterpreted as oxyhemoglobin.

The Rainbow SET Rad-57 (Masimo Corporation) is a device that allows non-invasive determination of COHg levels. Several studies have reviewed the performance of this device compared to gold standard co-oximetry for measuring COHg. In three studies, the reported sensitivity and specificity ranged between 48 – 94% and 77-99%, respectively. For this device, sensitivity measures exposures that are correctly identified; specificity measures non-exposures that are correctly identified. The perfect test would be 100% sensitive and 100% specific. In these studies, several reasons may explain the discrepancy in the performance of this device, including inappropriate placement or size of sensor, patient motion, light interference, etc. Ultimately, this device is a tool; a normal level should not replace clinical suspicion for exposure.
In suspected exposures, initial resuscitative efforts focus on removing the patient from the exposure source, stabilization of vital signs, and administration of 100% oxygen. Patients should be transported to the nearest facility for continued care.

Pearls

- CO exposure is common
- CO is misdiagnosed as the flu or gastroenteritis
- Be vigilant for exposures in winter. If more than one person is experiencing similar symptoms or if symptoms occur spatially related to a potential source, then consider CO exposure
- Pulse-oximeter is inaccurate for detecting CO

References:

Review of Recent Research Regarding Prehospital Hypothermia

JE “Tripp” Winslow, MD MPH

Review of New England Journal of Medicine Article:
Targeted Temperature Management at 33 degrees Celsius versus 36 after Cardiac Arrest

**Background:** An article titled “Targeted Temperature Management at 33 degrees Celsius versus 36 after Cardiac Arrest” recently appeared in the New England Journal of Medicine. The objective of the study was to compare the outcomes of two groups of survivors of out-of-hospital cardiac arrest. One group had a target temperature of 33 degrees Celsius and the other group had a target temperature of 36 degrees Celsius. The study was carried out across 36 sites in multiple countries. Patients with all rhythms were entered not just ventricular fibrillation or ventricular tachycardia without a pulse.

**Methods:** Other inclusion criteria included GCS less than 8, age of 18 years and older. Cooling had to be started within 240 minutes of the cardiac arrest. Patients were randomized to a targeted temperature of 33 or 36 degrees Celsius. The target temperatures were achieved with ice-cold fluids, ice packs, or surface management temperature devices. At 28 hours the patients were re-warmed. Patients were only paralyzed as needed for shivering. It does not appear that the cooling took place prehospital.

**Results:** Between 2010 and 2013 there were 950 patients entered into the study. There were 473 patients in the 33 degree group and 466 assigned to the 36 degree group. Cooling was achieved with an intravascular cooling catheter in 24% of the patients and a surface cooling system in 76%. In the 33 degree group 235 of the 473 patients died which means that 50% died. For the 36 degree group 225 of the 466 patients died which is 48%. These differences were not statistically significant. There was also no difference in the two groups in regards to neurological outcome at 180 days.

**Conclusion:** As stated above there was no statistical difference between the two groups in death or neurological outcome. The study also found no harm in using a targeted temperature of 33 degrees Celsius. The study authors make the point that a major difference between their trial and other past research looking at hypothermia is that in both treatment groups they controlled the temperature. In the 33 degree group they kept the patient hypothermic. In the 36 degree group they basically kept the patient at a normal temperature by actively keeping the patient’s temperature at 36 degrees (normal body temperature is 37 degrees) which also prevented
fever. The accompanying editorial makes the point that the biggest benefit to the various hypothermia protocols might actually be that they prevent fever. Fever, especially fever in the first 2 days after cardiac arrest is known to have a bad effect on neurological outcome.

**Comments from accompanying editorial in the New England Journal of Medicine:** Many patients in the non-intervention or normothermic arms of older trials became febrile which can be very bad for neurological outcome. The editorial goes on to caution that the medical community should “not regress to a 2002 style of care that does not manage temperature at all”.


Review of article from Journal of the American Medical Association:

**Effect of Prehospital Induction of Mild Hypothermia on survival and Neurological Status Among Adults With Cardiac Arrest**

**Background:** The best time to begin therapeutic hypothermia has not been well studied. This study sought to determine if therapeutic hypothermia should be initiated in the prehospital setting or if should be started later.

**Methods:** This study took place at King County EMS in Washington State. Inclusion criteria included return of spontaneous circulation, tracheal intubation, intravenous access, placement of an esophageal temperature probe, and unconsciousness. Exclusion criteria included traumatic arrest, age less than 18, being alert/awake, and having an initial temperature of less than 34 degrees Celsius. Subjects were randomized to receive standard care or standard care plus mild hypothermia. Patients in the hypothermia group all received a very long acting paralytic, diazepam, and 2 liters of cold saline. The saline was infused via a pressure bag (300 mm Hg pressure) through at least an 18 gauge catheter.

**Results:** The study took place between the years 2007 and 2012. There was no difference in neurological outcomes between the two groups. There was also no difference in survival to hospital discharge for the two groups. Achievement of targeted temperature was faster for the EMS hypothermia group. The incidence of re-arrest for the EMS hypothermia group was higher as well as the rate of pulmonary edema and use of diuretics during the first 12 hours.
Survival to Discharge

<table>
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<tr>
<th></th>
<th>EMS hypothermia group</th>
<th>non EMS hypothermia group</th>
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<tr>
<td>With ventricular fibrillation</td>
<td>62.7% survived to discharge</td>
<td>64.3% survived to discharge</td>
</tr>
<tr>
<td>Without ventricular fibrillation</td>
<td>19.2% survived to discharge</td>
<td>16.3% survived to discharge</td>
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Note: none of these results show statistical significance

**Conclusion:** Prehospital induction of therapeutic hypothermia does increase the speed at which the target temperature is achieved. There was no difference in outcomes for the two groups. There was a statistically significant increase in the use of diuretics and pulmonary edema in the prehospital hypothermia group but these complications did resolve on their own. There was also an increase in re-arrest for the EMS hypothermia group but as stated above no difference in mortality. The authors make the point that rapid infusion of 2 liters of cold saline may have had a negative effect.


Link to Journal Article
Link to accompanying editorial

**Take away points from NC Journal of Prehospital Care reviewer:**
- These articles do not necessarily mean that we should not continue prehospital induction of therapeutic hypothermia
- We are not at the point where we should stop prehospital cooling
- Therapeutic hypothermia is only one part of the care of the cardiac arrest patient
- In the second study reviewed they used pressure bags to infuse their cold saline and paralyzed all their patients. Most systems in NC do not bolus their fluids so quickly or give paralytics (Dr. Myers mentioned this to me).
- Any changes in practice should always be done only with local medical direction.
- Further research is likely in this area.

Link to EMCrit Blog with interviews of the investigators and an interview with author of the editorial

Additional interview from emcrit of Dr. Stephen Bernard one of the initial hypothermia investigators. He is a little like the father of hypothermia for cardiac arrest.
EMS is called to the home of a 40 year old gentlemen due to the complaint of chest pain. The patient states that his pain began about 1 hour prior to EMS arrival after he awoke from sleep. He describes the pain as being in the center of his chest, and radiates to his right arm and between his shoulder blades. He has associated nausea, sweating, but no shortness of breath. He also admits to fatigue and body aches, and recent increased caffeine intake due to stress at his job. He also states that he had a vomiting and diarrhea illness 2 weeks ago; however, he has no other recent symptoms. He has no medical history, does not smoke or use drugs, nor is there a family history of cardiac disease.

On initial assessment, he describes his pain as 7 out of 10. Vitals show a heart rate of 70, BP 150/96, respiration rate of 16, and pulse oximetry is 99% on room air. On exam, he is no acute distress. His heart has a regular rhythm and his lungs are clear to auscultation bilaterally. He has mild tenderness to palpation in the epigastric region of his abdomen; otherwise his abdominal exam is benign. His extremities show no edema. The rest of the assessment shows no physical exam abnormalities.

The EMS crew obtains a 12 lead ECG shown below:
What is your interpretation of the ECG?

Sinus Rhythm; Rate 65; Normal intervals; ST segment elevation in leads I,II, aVF, V2-V6

When ST segment elevation is not due to ischemia/injury

The above ECG shows changes that are consistent with pericarditis, or an inflammation of the fibrous layer covering the heart. Pericarditis can be caused by several processes. In the US, the most common cause is idiopathic / presumed viral. In the developing world, tuberculosis causes a majority of pericarditis cases. Occasionally, an inflammatory pericarditis can occur after myocardial infarction. The majority of cases of pericarditis occur in men aged 20-50, and pericarditis represents approximately 1% of the cases of ST elevation in the Emergency Department.

The pain of pericarditis is often worse with lying down and with swallowing, due to irritation of the posterior wall of the pericardial sac as it contacts the esophagus.

A pericardial rub can occasionally be heard – if present, it is nearly diagnostic for pericarditis. Unfortunately, the pericardial rub can be transient, and difficult to auscultate in noisy settings. The presence of jugular venous distension, hypotension, and muffled heart sounds suggests that there is an accompanying pericardial effusion that has progressed to cardiac tamponade.
The electrocardiogram has several features to suggest pericarditis:

1. The ST segment elevation is diffuse, involving almost all lead fields (anterior, inferior, lateral), not just one anatomic distribution.
2. There are no reciprocal changes or ST depression on the electrocardiogram.
3. Although not present on this ECG, sometimes PR depression will be noted.
4. There is no flattening of the ST segments or loss of concavity (the ST segments maintain a playground slide configuration).

If there is ST segment depression in leads other than V1 or aVr (where it may be physiologic), the patient should be assumed to be experiencing acute coronary syndrome until proven otherwise.

If there is loss of concavity or flattening of the ST segment, the patient should be assumed to be experiencing acute coronary syndrome until proven otherwise.

In treating this patient, if there is concern for ischemia, then continue to treat based on the chest pain protocols. Transmission of the ECG to online medical control and notification of receiving facility should also be performed to ensure further patient centered care.

Treatment for pericarditis includes non steroidal anti-inflammatory medications and evaluation for cause and its appropriate treatment. Most idiopathic pericarditis is benign and resolves with time and NSAIDS. Higher risk features include the presence of fever, autoimmune disease, or underlying cancer.

Editor’s note: Below is an additional EKG showing pericarditis. This is a patient that the editor saw a few weeks ago. The patient was a young male. Note the widespread ST segment elevation, PR depression, and lack of reciprocal changes.
Ectopic pregnancy is the leading cause of maternal mortality in the first trimester. Physicians are taught that a patient with an ectopic pregnancy will present with vaginal bleeding and abdominal pain after a period of amenorrhea (missing their period). This presentation is actually not all that common, however, manifesting in only about 15% of patients with ectopic pregnancy (Rivers). This being the case, any woman with a positive pregnancy test and any symptom of vaginal bleeding, abdominal pain, or syncope, should have ectopic pregnancy considered in her differential. Basically, for the prehospital provider, this means that any time you encounter a woman of child-bearing age with abdominal pain, ectopic pregnancy should be a consideration.

There are many risk factors for ectopic pregnancy, including history of previous ectopic pregnancy, intrauterine device (IUD) use, history of pelvic inflammatory disease, and history of tubal scarring (previous tubal ligation, etc.). These all very important factors to consider when assessing patient risk for ectopic, but approximately 50% of patients with ectopic pregnancy have no identifiable risk factors (Rivers).

Overall, if identified and treated early, patients with ectopic pregnancy tend to do very well. If an ectopic pregnancy is not detected on evaluation serious disability or death could result. The reason for this is that an unrecognized ectopic can rupture as the embryo grows. This can cause potentially deadly hemorrhage for the patient. It is important to remember that there are no “classic profile” of risk factors or presenting symptoms that can reliably rule in or rule out this condition. Thus, any woman with a positive pregnancy test and concerning symptoms must be considered to have an ectopic until proven otherwise. The take away point is that
providers should be very concerned if a woman with child-bearing age and abdominal pain wishes to refuse treatment. Without a negative pregnancy test they have an ectopic pregnancy until proven otherwise.

Prehospital Needle Decompression for 
Suspected Tension Pneumothorax
From Metrolina Trauma Advisory Committee

Paul D. Colavita, MD

Ronald F. Sing, DO

Peter E. Fischer, MD, MS, NREMT-P

Introduction

Pneumothorax is the presence of air in the thorax between the chest wall and the lungs themselves. As this pocket of air increases in size, it causes compression and collapses the lung on the same side and pushes the mediastinum towards the opposite side. (Figure 1) The mediastinum can be shifted to the point that the major veins bringing blood back to the heart, the superior vena cava and the inferior vena cava, are compressed. This decrease in the return of blood to the heart causes a decrease in cardiac output resulting in a drop in blood pressure as well as oxygen saturation. Pneumothorax is the presence of air in the hemithorax outside of the lung, but it becomes classified as a tension pneumothorax (a life-threatening condition) when blood pressure or oxygenation decrease.

Blunt and penetrating trauma can both cause pneumothorax. Blunt trauma most commonly causes pneumothorax by way of rib fractures injuring the lung parenchyma, whereas penetrating trauma
damages the lung parenchyma itself. Pneumothoraces can also occur spontaneously, such as when a lung bleb in a patient with emphysema ruptures. All 3 mechanisms can lead to pneumothoraces by way of air leaking from the respiratory tree into the hemithorax, between the lung and the chest wall. Pneumothorax is easily detected by chest x-ray, but is more difficult to diagnose based on physical exam alone. Unfortunately, in the prehospital setting, x-ray is not available and physical exam in conjunction with the patient's vital signs guide treatment.

- **A pneumothorax is the presence of air between the chest wall and the lung**
- **A pneumothorax can cause compression on the lung and the mediastinum resulting in hypotension and hypoxia (tension physiology)**

Assessment

Physical exam findings for pneumothorax include decreased breath sounds on the affected side and tympani to percussion. As tension physiology develops, the trachea may deviate away from the affected side and the patient's blood pressure and/or oxygen saturation will drop. When the systolic blood pressure is less than 90mmHg or the oxygen saturation is less than 90%, a patient with suspected pneumothorax requires intervention. Decreased breath sounds on one side with systolic blood pressure above 90mmHg and oxygen saturation greater than 90% do not require emergent treatment in the prehospital setting. Careful assessment must be performed on intubated patients, as a right mainstem intubation can create a false picture consistent with tension pneumothorax. A right mainstem intubation will lead to decreased left chest breath sounds as well as decreased oxygenation, and possibly hypotension when large tidal volumes are given via mechanical assistance.

- **A pneumothorax alone does not require treatment in the prehospital setting**
- **A pneumothorax in the presence of hypotension and/or hypoxia (tension pneumothorax) requires prehospital intervention**

Treatment

Initial treatment for tension pneumothorax in the prehospital setting is needle decompression. The technique of needle decompression is relatively simple, but can have dire consequences if performed on the wrong patient. Hence correct patient selection is the first key to success. With physical exam alone it can be difficult to differentiate a pneumothorax from other conditions such as hemothorax or diaphragm rupture. Therefore decompression should only be performed when tension physiology is present. The site of decompression for tension pneumothorax is in the 2nd intercostal space above the third rib in the midclavicular line. To find this space, you must remember that the first palpable rib on the patient's anterior chest is the second rib. If available, the area should be prepped with antiseptic material prior to decompression. A 14-gauge, long angiocath should be placed over the needle and attached to a syringe. The syringe plunger should be used to perform aspiration as the needle and angiocath are inserted perpendicular to the skin. Advance until air returns, then advance the angiocath to the hub, removing the needle. Once needle decompression is performed, definitive treatment with a larger, formal chest tube can be performed in the hospital setting.
- Needle decompression should be performed immediately above the third rib (2nd intercostal space) along the midclavicular line
- A longer angiocath should be used for adequate chest penetration.

Pitfalls

Potential pitfalls of needle decompression include creating a pneumothorax by introducing air into the chest, damaging a non-collapsed lung or part of the bronchial tree. Improperly performed decompressions have led to damage to veins, arteries, or even the heart itself resulting in massive accumulation of blood in the chest, as well as death. A number of vital structures including the subclavian vein, subclavian artery, and the internal mammary arteries are in close proximity to the correct place for decompression. In addition to selecting the correct patient, meticulous attention to technique is key to a successful chest decompression.

- Needle decompression can cause a pneumothorax when performed in the wrong patient
- Chest decompression if performed incorrectly can cause damage to surrounding structures leading to patient death

Conclusions

It is a vital procedure in our armamentarium because of this lifesaving capability, but because of the potential pitfalls, it must be only performed in appropriate patients with correct instruments and technique. In the prehospital setting, careful assessment must be used to identify those who meet the criteria of suspected tension pneumothorax with hypotension or hypoxia in order to perform needle decompression in the appropriate patients.

References:
Fig 1. Tension Pneumothorax
Serious pelvic fractures are associated with a high mortality rate. This literature review by a prehospital consensus group provides an evidence based guide to the field management of pelvic fractures.

A Medline Database keyword search was performed for prehospital management of pelvic injuries, pelvic circumferential compression devices, pelvic binders, SAM pelvic sling, T-POD, PelvicBinder, Geneva belt and London pelvic sling. Each of the 17 identified articles that met their inclusion criteria was reviewed by the expert panel.

The result of this literature review was the creation of nine recommendations for prehospital personnel and the design of an algorithm to determine the appropriate use of a pelvic binder. Of the nine recommendations, four stand out in terms of their importance or challenge to the status quo.

1. A pelvic binder is a treatment intervention rather than a packaging intervention, and should be applied early.

   This is the authors' most important recommendation. Identifying patients with hemodynamic compromise from a likely pelvic fracture can enable prehospital personnel to provide early stabilization of the pelvis. A pelvic binder is a hemorrhage control device; it decreases internal blood loss and promotes clot formation.

2. In the presence of significant hemodynamic compromise, an attempt should be made to reduce associated femoral fractures.

   This important recommendation challenges the status quo. The authors' review of the literature found no evidence of increased harm of femoral reduction with concomitant use of pelvic binders. However, any use of traction devices should not apply midline pressure to the pelvis. If attempts at femoral traction worsen the patient's condition they should be abandoned and the extremity left “as found.”
3. Patients with suspected pelvic fractures should not be log rolled or transported via spine boards.

The authors challenge tradition, by stating that the pressure and movement associated with logrolling and spine boards must be avoided if the pelvis is unstable. Further, there is no data to support the routine logrolling of blunt trauma patients. If extrication is required, a scoop stretcher or alternative extrication device should be considered. If the patient must spend time on a rigid extrication device, use of appropriate padding is required.

4. Earlier application of a pelvic binding device is preferred.

No controlled trials or studies looked at the difference early versus late application of a device made, but the authors concluded it was reasonable to assume the earlier stability was achieved, the less likely extrication of the patient would disrupt clot formation. While they were unable to choose one device over another, the SAM pelvic sling and the T-POD devices both had strong support in the literature.

Author’s conclusion: A pelvic binder is a treatment option for major hemorrhage, not a packaging device. Pre-hospital providers should apply pelvic binding early for internal hemorrhage control in the hemodynamically unstable patient with a suspected pelvic fracture.
Active shooter events have steadily increased in the United States in the last 20 years (Department of Homeland Security, Federal Bureau of Investigation [DHS, FBI], 2012; Newman & Fox, 2009). On October 20, 2013, United States Attorney General Eric Holder stated that active shooter events have tripled since 2009 and the lethality of these events have increased 150% (Farr, 2013). Attorney General Holder also stated that public safety agencies must create new and aggressive strategies to respond to these events (Farr, 2013).

From the first day in emergency medical technician (EMT) school, students are taught that responder safety is an overarching principle that supersedes all priorities of medical care. While this axiom holds true for the majority of routine responses, fire and emergency medical services (EMS) personnel are now finding themselves at a crossroads of divergent opinions regarding active shooter response. Polarized on two opposite ends, one group advocates for fire and EMS personnel to continue to stage in a safe location until potentially dangerous scenes are secured by law enforcement. On the other end, practitioners are advocating for fire and EMS personnel to aggressively enter into potentially hostile environments to treat and extract casualties.

The concept of EMS integrating into potentially hostile events is not new. In 2008, the Department of Homeland Security published the handbook Active Shooter: How to Respond. This handbook was distributed to public safety agencies across the United States. The handbook states, “The first officers to arrive to the scene will not stop to help injured persons. Expect rescue teams comprised of additional officers and emergency medical personnel to follow the initial officers. These rescue teams will treat and remove and injured persons” (DHS, 2008, p. 5). Following a dramatic increase of active shooter and active assailant events in the last decade, fire service personnel and EMS personnel understand that strategies and tactics must change.

The catalyst for a change in active shooter response began in 1999 at the Columbine attacks. The Columbine Governor’s Review Commission found that law enforcement personnel set up a
perimeter and essentially waited for the attack to end before making entry (CNN, 2001). The incident commander did not rush to send in medical help. Two gunshot victims died of treatable wounds 90 minutes and 180 minutes after the attack ended (Kennedy, 2002). Law enforcement quickly recognized a need to radically change tactics, and thus began the creation of rapid deployment training and direct-to-threat active shooter training (Moore, 2011). Unfortunately, fire and EMS providers did not see a need to change their tactics, and have continued to stage a safe distance away until law enforcement declares the scene safe.

There is compelling research and data to suggest that fire and EMS personnel must rethink the theory of staging until the all clear is given by law enforcement. Morissey (2011) found that it takes law enforcement an average of one to two hours to declare an active shooter scene safe from any obvious threat. Lipkiss (2011) found similar results at the large-scale Urban Shield drill conducted in California. At Columbine, the scene remained unsafe for 72 hours while bomb squads swept the building and defused explosive devices. Delays of care more than 10 minutes to active shooter victims can be deadly.

Approximately half of the victims at an active shooter event will have moderate to severe ballistic injuries (Kaplowitz, et al., 2007). This statistic proved true at the Virginia Tech shooting, where 12 of the 24 patients had moderately critical to critical injuries (Kaplowitz, et al., 2007). Ballistic injury research from combat found that casualties with a major ballistic injury have a 67% mortality rate if the casualty does not receive basic medical care and evacuation within 30 minutes of injury (Strawder, 2006). Half of these combat casualties died from preventable exsanguinating injuries (Strawder, 2006). A significant body of combat casualty research has found that simple point-of-wounding medical care would prevent 10% to 15% of combat casualties (Champion, et al., 2003).

At the Virginia Tech shooting, the mortality was 3.8% for patients who were alive when an EMT reached them (Kaplowitz, et al., 2007). Only one of the 26 patients died after an EMT made patient contact. The same survivability has been demonstrated time and again at events such as the Fort Hood shooting, Orlando Gateway Center shooting, Aurora Theater shooting, the Boston Bombing, and many others.
Providing medical care at hostile events is not a Herculean task. First, advanced life support medical care is not typically required. The United States Department of Defense has no reported incidents of preventable battlefield deaths in Iraq and Afghanistan with Special Forces soldiers trained in a 16-hour basic tactical combat casualty care course (Callaway, et al., 2011). Lieberman and colleagues (2000) performed a meta-analysis of basic life support (BLS) and advanced life support (ALS) care for trauma patients. They found the odds of death were 2.59 times higher if trauma patients were treated by ALS providers when compared to BLS providers even with adjusted injury severity scores (Lieberman, et al., 2000). Lieberman (2003) again examined trauma patient mortality in a two-tiered ALS/BLS system. He found the trauma patients with ALS care had a 29% mortality rate compared to an 18% mortality rate, again with an adjusted injury severity score. Advanced life support care certainly has proven merits; however, the benefit of initial ALS care in trauma patients and hostile environments has a questionable, unproven efficacy.

Medical providers also do not need to be tactical medics to operate in high-threat environments; however, medical providers do require basic awareness level tactical training. Research demonstrates that without basic tactical training, civilian medical providers frequently attempt to use EMS procedures that have no place in tactical medicine (Giduck, 2008). Examples of inappropriate care in high-threat environments include spinal immobilization, establishing intravenous access, performing intubation, attempting to transport patients up and down stairs on stretchers, and other tactical errors performed with good intentions. Basic tactical awareness for EMS providers can be accomplished through an eight or sixteen-hour course, such as the Tactical Combat Casualty Care (TCCC) course or the Tactical Emergency Casualty Care (TECC) course.

This new model does not advocate fire and EMS personnel to run blindly into potentially violent situations. However, fire and EMS providers are strongly encouraged to work and train with local law enforcement to operate in areas that are cleared from obvious threats, but still in potentially hostile areas. Essentially, law enforcement operates where the threat is, and fire/EMS operates where the threat isn’t. In these areas, fire and EMS providers will always work with the security of law enforcement to provide care and extract casualties. For this new life-saving model to work, fire and EMS agencies must train with local law enforcement. Active shooter
events are very dynamic, chaotic, and require tremendous communication, command, and control.

At active shooter events there is a risk to responders. However, the risk is relatively low and the benefit is extremely high for patients actively dying (Coleman, 2004). Regardless of the uniform, the expectation today is that public safety personnel will assume a level of risk at emergency scenes to save lives (Coleman, 2004). As Dawn Anna, mother of Columbine victim Lauren Townsend said in a 60 Minutes’ interview, “If you are going to tell my child to stay put, you’re going to go get her. Either go in there and do something or take off your uniform and find another job. On this day, it was your job to put her life above yours.”

*This is Part 1 of a three part series on emergency medical operations in hostile environments to be published in the North Carolina Journal of Prehospital Medicine.*
References


Giduck, J. (2008). Terror in America’s schools: The need to prepare first responders to defend our nation’s children. *Journal of Emergency Medical Services 33*:4-10


**About the Author:**

Michael Clumpner is the President and Chief Executive Officer at Threat Suppression Incorporated, a public safety consulting firm based in Charlotte, North Carolina. Mr. Clumpner is a career fire captain/paramedic with a metropolitan fire department and a SWAT operator / senior tactical paramedic with a large, urban law enforcement agency. Mr. Clumpner has been a paramedic for 19 years including the last nine years as a helicopter flight paramedic. Mr. Clumpner is currently completing a PhD in homeland security policy and is writing his dissertation on joint public safety response at active shooter events. Mr. Clumpner is a member of multiple local, state, and federal active shooter planning and response taskforces. He can be reached at mclumpner@ThreatSuppression.com.
Asthma is a chronic inflammatory airway disease with varying levels of severity characterized by intermittent functional lower airway obstruction and frequent symptom exacerbations (McNeily et al, 2011). The disease causes constriction of the bronchial tree and small airway inflammatory changes that prevents full exhalation. Asthma affects all ages but most often begins during childhood and is more prevalent in females. Approximately 9.6 million children under the age of 18 have been diagnosed with asthma at some point during their lifetime.

Common triggers for an asthma attack may include irritants such as aerosol sprays, perfumes, and smoke. In addition, allergens such as animal dander, mold, mildew, and pollens can trigger an attack. Environmental and physical conditions such as weather changes, emotional events, and viral infections can also promote asthma symptom exacerbations.

The classic findings in patients with an asthma exacerbation are diffuse expiratory wheezing, a prolonged expiratory phase and an increased respiratory rate. However, other signs that may be noticed in pediatric patients include intercostal retractions and nasal flaring. In severe cases, exam of the lungs may show a “quiet” chest, resulting from such profound constriction of the bronchial tree that the wheezing sound cannot be produced. The key to the pediatric lung exam is to not only listen to the lungs, but look at the chest wall and work of breathing.

Treatment includes the use of short-acting bronchodilators such as albuterol. Some protocols may add ipratropium which offers the patient a more sustained period of adequate air exchange. Young children often resist a mask or any type of equipment near their face, therefore, keeping the child calm with distraction from a toy and allowing the parent to hold the child can make our assessments and treatment easier. In other words if you keep them happy and distracted it will be much easier to put the mask near their face. Creativity is the key!!!! Continuous nebulization of bronchodilator medication is often utilized. Intubation of these patients should be avoided at all costs unless signs of impending respiratory failure are present. These signs include inability to speak, decreased mental status, and worsening fatigue.
Corticosteroids, such as prednisone or methylprednisolone are often used in asthmatic patients to assist in decreasing inflammatory changes in the lungs.

The goals for treating asthma exacerbations include correction of hypoxemia and increase airflow. Interventions include the administration of oxygen (O2) to keep O2 saturations greater than or equal to 94%, administration of Albuterol or Ipratropium by nebulizer along with oral administration of corticosteroids for mild to moderate management of acute asthma.

Pediatric Advanced Life Support. 2011. American Heart Association

Asthma Education Review. 2011. Kettering National Seminars
Continuous positive airway pressure (CPAP) has revolutionized the pre-hospital care of acute hypoxic respiratory failure. Since its inception into pre-hospital medicine in the 1990s, countless numbers of patients have been able to avoid intubations due to CPAP.

**When should CPAP be used?** CPAP should be used in patients that are awake and alert enough to protect their own airway. The patients must be breathing spontaneously. CPAP has the greatest benefit in acute cardiogenic pulmonary edema. Although it can be used as a bridge until tracheal intubation, CPAP should be preferentially used in patients that have a relatively quickly (12-24 hours) reversible cause for their hypoxia.

**How does CPAP work?** CPAP works by providing a constant positive pressure that keeps lung alveoli from closing at the end of expiration. CPAP is interchangeable with Positive End Expiratory Pressure (PEEP). By keeping the alveoli from closing, CPAP will increase the volume of air remaining in the lungs at the end of the respiratory cycle, i.e. Functional Residual Capacity (FRC). This leads to a decrease in the patient’s work of breathing. CPAP will also improve a patient’s cardiac output: CPAP causes an increase in intrathoracic pressure, which leads to a decreased venous return to the right heart. With less blood flow returning to the right heart, less blood will go through the lungs and get to the left ventricle. This leads to a decrease in the left ventricular transmural pressure and end diastolic pressure. The overall effect is a drop in the left ventricular afterload against which the left ventricle must pump.

**Avoid CPAP in which patient population?** Any patient that is not spontaneously breathing or is not able to protect his/her airway should not be started on CPAP. Patients with hypotension are not candidates for CPAP. If there is evidence of facial trauma or significant facial abnormalities, it will be difficult to obtain a tight mask seal, so these patients should not be placed on CPAP.

**Potential problems with CPAP?** It is imperative to closely monitor patients on CPAP. If a patient’s mental status decreases so that he/she cannot protect their airway, CPAP should be removed. Patients may occasionally vomit due to the amount of air that is being pushed into the stomach. This will often lead to aspiration. Any patient that is vomiting should be taken off of CPAP. It is possible for patients on CPAP to develop a pneumothorax due to high positive pressure. Patients that need a prolonged expiratory time (COPD patients) may develop worsening respiratory failure due to auto-peep or breath stacking, a condition in which the patient is not able to fully exhale and get good gas exchange.

CPAP is a potentially lifesaving tool when used in the correct patient population. For patients that are awake, able to protect their airway and have acute hypoxic respiratory failure, it should be the initial treatment of choice.
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<td>0%</td>
<td></td>
</tr>
<tr>
<td>74</td>
<td>115</td>
<td>17</td>
<td>8</td>
<td>3</td>
<td>9</td>
<td>0%</td>
<td>0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>115</td>
<td>178</td>
<td>53</td>
<td>33</td>
<td>0</td>
<td>14</td>
<td>0%</td>
<td>0%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>178</td>
<td>122</td>
<td>16</td>
<td>3</td>
<td>2</td>
<td>3</td>
<td>7</td>
<td>13%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>122</td>
<td>148</td>
<td>17</td>
<td>12</td>
<td>2</td>
<td>2</td>
<td>4</td>
<td>12%</td>
<td>0%</td>
<td></td>
</tr>
<tr>
<td>148</td>
<td>159</td>
<td>54</td>
<td>22</td>
<td>1</td>
<td>0</td>
<td>2</td>
<td>2%</td>
<td>0%</td>
<td></td>
</tr>
</tbody>
</table>

#### Specialty Care Sub-total

<table>
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<tr>
<th>Identifier</th>
<th>911 Service</th>
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<th>1</th>
<th>1</th>
<th>1</th>
<th>1</th>
<th>1</th>
<th>100%</th>
<th>0%</th>
</tr>
</thead>
<tbody>
<tr>
<td>420</td>
<td>233</td>
<td>12</td>
<td>6</td>
<td>16</td>
<td>1</td>
<td>75</td>
<td>3%</td>
<td>1%</td>
<td></td>
</tr>
</tbody>
</table>

#### Grand Total

| Identifier | 1032 | 412 | 68 | 17 | 42 | 11 | 200 | 7%  | 2% |

---

**Note:**
- **Grand Total** includes all forms submitted to NCOEMS from Jan 1, 2013 to Dec 31, 2013.
- **911 Service** represents the breakdown of forms submitted by 911 operators.
- **Specialty Care Transport Agencies** shows the breakdown for specialty care transport agencies.

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**Airway Forms Received** includes:
- **Intubation due to trauma**
- **Nr Unsuccessful Intubations**
- **Greater than 3 Intubation Attempts**
- **Missing ETCO2 value post airway**
- **Secondary Airway Failed**
- **Requires Medical Dir Review**
- **Pct Unsuccessful Intubations**
- **Pct Greater than 3 Intubation Attempts**