Over the past decade prehospital care has undergone a transformation toward evidence-based medicine. Advances in research have changed how prehospital providers perform CPR, use lights and sirens, and manage patients experiencing heart attacks and strokes. Unfortunately, since prehospital care was founded on the idea of applying good ideas that seemed like common sense, some of the age-old skills and practices performed in EMS remain based on little more than cultural acceptance rather than research-proven medicine. This month’s CE column is dedicated to debunking such myths regarding care of the trauma patient and explaining true best practices for these patients.

Myth #1: Never Insert Nasal Airways in Head-Injured Patients

The myth explained: While the placement of nasal airways, nasal gastric tubes and nasal intubations is common in emergency medicine, EMS classrooms have long taught that whenever a head injury is suspected, particularly a basilar skull fracture, placing anything in the nares is likely to violate the soft bone of the cribriform plate (skull) and penetrate the cranium, rather than being placed in the airway.

The Evidence

A comprehensive review on the use and indications for nasopharyngeal airways was published in a 2005 *Journal of Emergency Medicine*. This review acknowledged it is widely taught that skull fractures are a contraindication for NPA placement; it went on to identify only two case reports, as of 2005, in all of published medicine. One report was released in 1991 in *Anesthesiology*, and the other in the *Journal of Trauma* in 2000. These authors concluded placing an NPA through the skull is extremely rare and is likely associated with improper technique as well as injuries that already have significant morbidity and mortality.
This position was followed by a 2006 case report and letter in the Journal of Emergency Medicine from three physicians in London. In their letter, these physicians reported another case of an NPA in the cranium following severe facial trauma in a patient whose airway could otherwise not be managed due to a clenched jaw. The authors agreed the placement of an NPA into the cranium is exceedingly rare and most likely results from poor placement techniques. It was their opinion that airway management should take precedence over the risk of an extremely rare consequence. Further, NPA placement is recommended for military prehospital providers even in the case of head injury. David Steinbruner, MD, et al., identified that the military position is that proper placement of the NPA to establish a patent airway offers significantly greater benefit from improved airway management than the relatively small risk the NPA may penetrate the skull.

Multiple different sources support the use of proper nasal airway placement for patients with head injuries; the key here being using proper technique and stopping when resistance is met and directing the airway along the base of the nasal cavity as shown in Figure 1a. Directing any nasally placed device upward toward the eyes increases the likelihood of complications. Keep in mind, these papers are encouraging the use of nasopharyngeal airways and not nasal intubation or nasogastric tubes in particular may have a greater likelihood of compromising the basilar skull.

The Bottom Line
Nasopharyngeal airway placement can safely be performed in patients with head injury when airway management is needed. The benefit of establishing an airway outweighs the incredibly small risk of the NPA entering the brain.

Myth #2: Lidocaine in RSI Prevents Increased ICP in the Head-Injured Patient
The myth explained: It is known that laryngeal manipulation and endotracheal intubation are both associated with a transient rise in intracranial pressure (ICP), and patients who have a head injury with increased ICP are considered at an increased risk for secondary brain injury. Since a transient ICP rise could decrease cerebral perfusion pressures, every effort is made to control ICP. In theory, lidocaine prevents this transient ICP rise, which helps prevent a secondary brain injury. Evidence for lidocaine as a preventive measure first appeared in 1980, when Robert Bedford, et al., published their paper, “Lidocaine prevents increased ICP after endotracheal intubation,” when they monitored 20 patients who were undergoing elective neurosurgery. Their results showed that while patients who received lidocaine did experience a rise in ICP, it was significantly less than in patients who received a placebo. Ultimately, these results were extrapolated to use lidocaine as part of the medication sequence for intubation, although this study did not address this question at all.

The Evidence
Drs. Mike Clancy and Neil Robinson reviewed all of the available literature regarding the use of lidocaine in RSI and published their findings in 2001. After an exhaustive literature review, they only found six papers that addressed lidocaine and intracranial pressure changes. No paper studied lidocaine during RSI. These authors concluded there is no evidence to support the use of lidocaine as a clinical intervention during RSI and recommended its administration be limited to clinical trials.

Although there is no evidence to support the use of lidocaine, is there evidence that suggests that lidocaine causes harm? A 2012 paper in the American Journal of Emergency Medicine determined lidocaine causes harm. A 2012 paper in the American Journal of Emergency Medicine determined lidocaine causes harm.
Lidocaine had no impact on the hemodynamic stability of patients receiving RSI following traumatic injury and determined the drug was safe to administer during RSI.

However, one potential problem is that lidocaine administration is time-dependent. Some authors suggest that to have any potential benefit for the patient receiving RSI, lidocaine must be administered at least 2 minutes prior to laryngoscopy. Waiting an additional 2 minutes to intubate a patient with a head injury may be its greatest risk, as during this time patients can remain hypoxic, potentially aspirate and continue to fight, which further exacerbates an increased ICP. Lidocaine has a dose-dependent effect as well and too much lidocaine can be detrimental. While minimal arterial blood pressure changes occur when patients received 1.5 mg/kg of lidocaine prior to neurosurgery, significant blood pressure declines occur when the dose is increased to 2 mg/kg.

The Bottom Line
Lidocaine is not proven to prevent a rise in ICP during RSI. While it may not directly harm patients, its administration does delay the completion of RSI, which puts the patient at risk for continued hypoxia.

Myth #3: Backboards are Helpful
The myth explained: Every year, as many as 5 million patients are immobilized with a cervical collar and backboard using the same techniques that have been taught in EMS classes for over 30 years. Three small straps are used to secure a human chest and hips to a flat rigid board and head blocks then prevent head movement. This, in theory, was meant to prevent patient movement and further injury and reduce morbidity.

The Evidence
In any population studied, the frequency of spine injury is low. In multi-system trauma patients, rates of spine column injury range between 2%–5%, while cord injury occurs in less than 2% of patients. In minor incidents, such as falls and motor vehicle collisions with restraint use, rates are as low as 1.2%. Thus, it is well understood that the majority of the time patients are immobilized there is no actual injury that the immobilization is theoretically stabilizing.

This is one of the many reasons the American College of Surgeons ATLS course textbook recommends patients be removed from a backboard as quickly as possible. James Morrissey and his colleagues published their 2014 paper demonstrating our current approach to immobilization does not help the overall population and provided evidence that limiting any spine immobilization to patients who cannot pass a spine examination, and then using immobilization strategies that avoid the long spine board, reduces unnecessary immobilizations and increases patient comfort.

One of the most significant reviews of backboard use was the 2013 joint position paper from the National Association of EMS Physicians and the American College of Surgeons Committee on Trauma, which stated there is no proven benefit to rigid spine immobilization. This paper went on to discuss that immobilization can cause pain, patient agitation, respiratory compromise and decreased tissue perfusion where the patient’s skin presses against the board, which can potentially cause pressure ulcers. It is also known that in healthy patients, full spine immobilization has been shown to cause sacral tissue ischemia capable of producing a pressure ulcer within 30 minutes of longboard application. Further, immobilization causes a significant enough increase in pain that neurological exams become less reliable when the patient remains immobilized.

In the same paper, the NAEMSP identified that patients with penetrating trauma to the head, neck and torso without spine deficit do not need immobilization and that immobilization actually causes a delay in transport that can be significant enough to increase patient morbidity and mortality. Further, even when performed properly, backboards do not provide any additional benefit. When patients in two large trauma systems were compared—one in New Mexico, where prehospital immobilization occurred, and the other in Malaysia, where no prehospital immobilization occurs—there was no difference in neurological disability between the patient groups. The authors determined that in blunt spinal injuries immobilization has little to no benefit on patient outcomes.
The Bottom Line
Backboards have no proven benefit for the trauma patient and can be harmful by compromising a patient’s ventilations, placing them at risk for pressure ulcers and delaying transport to definitive care. That being said, spinal motion restriction in trauma patients is good practice.

Myth #4: Trendelenburg Position
The myth explained: During the early part of the 20th century American physiologist Walter Cannon suggested the head down-legs up position pioneered by German surgeon Friedrich Trendelenburg could, in theory, displace blood from the lower extremities during hemorrhagic shock to enhance venous return to central circulation. The original Trendelenburg position was intended to permit an improved field of vision during abdominal surgery by displacing its contents cephalad and was never designed for blood pressure changes.

The Evidence
The 2010 American Heart Association First Aid guidelines clearly states all recommendations on the use of Trendelenburg position are extrapolated from volume expansion studies and there is no direct evidence to its benefit in shock. Margo Halm completed an excellent review of the studies that have been completed on the Trendelenburg position and published the review in a 2012 American Journal of Critical Care. Just over 20 studies have been completed. Raising a patient’s legs in hemorrhagic shock effectively returns 1.4% of the blood volume to central circulation, resulting in a mild transient cardiac output increase lasting anywhere from 1–5 minutes. Note, this increase is in cardiac output; the effect on blood pressure is negligible. Further, she found several studies demonstrated Trendelenburg position actually decreased cerebral blood flow, oxygen saturations, upper extremity blood flow and the lungs’ functional reserve capacity.

Raising the patient’s legs and pelvis can cause the abdominal contents to shift and increase pressure against the diaphragm and the inferior vena cava, limiting the lungs’ ability to expand and placing increased pressure on baroreceptors within the vena cava. By compressing the baroreceptors the body actually can be fooled into thinking the blood volume is higher and as a result depress the body’s compensatory efforts.

The Bottom Line
The evidence demonstrates that not only does the Trendelenburg position not help patients experiencing hemorrhagic shock, but it can actually be harmful because of effects on both ventilatory and circulatory systems.
Myth #5: Patient Extrication with the KED Prevents Spine Movement

The myth explained: Following major motor vehicle collisions, prehospital providers are taught that a patient should remain still within the vehicle and allow properly trained rescuers to control their movements to remove the patient from their vehicle and place them on a long backboard. Prehospital providers are taught several techniques, ranging from rapid extrication to application of a Kendrick Extrication Device (KED). We’re literally taught the patients may kill themselves if they move on their own with an unrecognized spine injury.

The Evidence

In 2009, Jeffery Shafer and Rosanne Nauheim teamed up to compare the differences in spine motion when patients remove themselves from a severely damaged vehicle compared to when the patient is removed with assistance from prehospital professionals. Using motion tracking cameras and strategically placed sensors on their volunteer patients, these authors performed four tests: self-extrication with and without a cervical collar, and extrication by rescuers directly onto a long spineboard, and via KED. In each test the spine motion was recorded for both the overall change from start to finish as well as the range of motion. Self-extrication without a cervical collar produced an average change of 8.7 degrees of motion (standard deviation 11.9°) in the cervical spine with a range of motion over 31°; the application of a cervical collar reduced the overall change to 1.4° (SD 4°) with a range of motion of 6.4°. Standard extrication onto a longboard produced an average of 1° (SD 4.5°); however, the range of motion was 26.6°, and when a KED was used to assist with extrication the patient’s mean overall change was 2° (SD 2.3°) with a range of motion of 31.1°.

Based on this study the authors concluded patient manipulation to apply a KED and slide them onto a longboard directly from a vehicle causes more spine motion than does applying a cervical collar and allowing the patient to extricate themselves and walk to a stretcher. A similar study was repeated by jack Engsberg and his colleagues and published in the Journal of Emergency Medicine in 2013. These researchers found the application of a KED during the extrication process causes more spine motion than assisted extrication directly onto a spineboard and compared to a patient self-extricating after a cervical collar is applied.

The Bottom Line

The KED increases spinal column motion during the extrication process; alternative methods of extrication need to be considered and explored.

Myth #6: Never Remove a Dressing from a Bleeding Wound; When It Bleeds Through Just Add on More Dressings!

The myth explained: Every major first aid resource says to apply a dressing on any uncontrolled hemorrhage, and if it soaks with blood to add more dressings on top of the original but not remove the blood-soaked dressing. The claim is removal of the original dressing may disrupt clots that are forming, causing the patient to continue to bleed.

The Evidence

To start addressing this myth it’s important to understand the basics of hemostasis, the process through which clots form. When an injury to a blood vessel occurs, collagen and von Willebrand factors (vWF) are exposed and promote platelets to attach to the wounded surface. As platelet aggregation occurs, a plug develops which stops the bleeding. Platelet aggregation occurs along human tissue. Once a platelet plug occurs and hemorrhage stops, a fibrin mesh begins to form that stabilizes the clot and strengthens it.

When a dressing is applied to a wound, the goal is to stop the bleeding. This is successfully completed 95% of the time with well-aimed direct pressure directly into the injured tissue. The key to well-aimed direct pressure is to apply adequate pressure into the injured tissue and on the injured vessel. When pressure is applied generally around the wound there is typically inadequate pressure to stop the hemorrhage. Well-aimed direct pressure means targeting the pressure directly into the hemorrhaging tissue.

There is not one clinical trial that demonstrates removing a blood-soaked dressing will cause clot removal or cause the clotting process to start over. When large bulky dressings are applied over hemorrhage sites it becomes very difficult to apply well-aimed direct pressure into the wound and as a result more generalized pressure around the injury occurs. At this point, the dressing becomes a source for collecting the lost blood and is actually doing very little to control hemorrhage.

Dressings that quickly become saturated with blood are an indication adequate pressure has not been applied to the bleeding site. Simply adding more dressings on top of the injury will do little more than absorb more blood; it will not help control bleeding or support clot formation. When a dressing becomes soaked with blood, remove it and apply better aimed pressure with a clean dressing.

In 2014, the American College of Surgeons released a position paper on prehospital hemorrhage management strategies. In this paper they identify that well-aimed direct pressure is likely to control hemorrhage in most instances. When hemorrhage cannot be controlled with well-aimed direct pressure, they recommend the next step be tourniquet placement for extremity injuries. In instances when a tourniquet cannot be
applied they recommend a hemostatic agent be added to a pressure dressing. Hemostatic dressings must be applied directly to the source of the bleeding in order to work.17

**The Bottom Line**
If initial direct pressure fails to control hemorrhage, remove the dressing and apply well-aimed direct pressure onto the hemorrhage location. When this fails, a tourniquet or hemostatic agent should be used.

**Myth #7: Radial Pulse Means a Systolic Blood Pressure of 80 mmHg; a Carotid Signifies a Systolic Blood Pressure of at Least 60 mmHg**
The myth explained: The 80/70/60 rule for radial, femoral and carotid pulse points to predict blood pressure has long been taught in both prehospital medicine and Advanced Trauma Life Support courses. The theory states the presence of a radial pulse indicates the systolic blood pressure is at least 80 mmHg, and a femoral and carotid pulse then indicate systolic pressures of 70 mmHg and 60 mmHg respectively.

**The Evidence**
This myth was challenged in a paper titled “Accuracy of the Advanced Trauma Life Support Guidelines for Predicting Systolic Blood Pressure Using Carotid, Femoral, and Radial Pulses: Observational Study” published in the British Medical Journal. This paper studied major trauma patients and evaluated their blood pressure when their radial, then femoral, then carotid pulses were lost. Without exception, all patients first lost their radial and then femoral pulses in sequence prior to losing carotid pulses. However, the actual values of these were quite surprising. Eighty-three percent of patients with radial pulses had a systolic blood pressure <80 mmHg (mean 72.5 mmHg), and 83% of patients with femoral pulses (radial absent) had systolic blood pressures <70 mmHg (mean 66.4 mmHg). No patients with only carotid pulses had the predicted blood pressure of >60 mmHg.18

In another study on critically ill ICU patients where radial and femoral blood pressures can be measured simultaneously, researchers found the mean arterial pressure (MAP) at the radial artery provided on average a bias of 4.27 mmHg higher than the femoral; however, nearly a third of the measurements found a bias greater than 10 mmHg. Keeping in mind MAP is calculated by the equation [(2 x diastolic)+systolic]/3, this bias could be a difference in systolic blood pressure that is significantly larger.19 The authors concluded peripheral readings become falsely high in critically ill patients because of the catecholamine release that occurs as the body tries to compensate during shock. This further impacts the systolic blood pressure estimation myth by suggesting that acutely hemorrhaging patients may have a falsely elevated peripheral pressure as the body tries to compensate for its blood loss; it is important to consider the central pressure may be lower than peripheral blood pressures suggest.

**The Bottom Line**
Using the 80/70/60 rule for peripheral pulses overestimates a hemorrhaging patient’s blood pressure and may put them at risk for delayed intervention. Obtain accurate blood pressures. There is a key component of common sense here though—if you cannot feel your patient’s radial pulse they are likely to be very hypotensive and ill.

**Myth #8: The Golden Hour**
The myth explained: Following major injury, patients have one hour to arrive at a definitive care facility before their chances of death significantly rise.

**The Evidence**
Craig Newgard, et al., published a paper challenging the truth of the golden hour in the January 2015 Annals of Emergency Medicine. In their research they evaluated trauma patients, 778 of whom were in...
shock, and 1,239 patients with traumatic brain injury who presented to level I and II trauma centers from 81 different EMS systems. They compared the patients’ 28-day mortality and 6-month Glasgow Outcome Scale-extended score (for patients with traumatic brain injury). The researchers found no suggestion that arriving at a trauma center greater than 60 minutes following injury impacted long-term outcomes. This paper supports a 2010 *Annals of Emergency Medicine* paper that reviewed over 3,600 trauma patients in shock—22% of whom died—and found shortened out-of-hospital times did not reduce a patient’s risk for inhospital death. They found increased EMS response, scene, transport or total EMS time did not increase mortality.20

**FOCUS ON DETERMINING WHETHER OR NOT YOU HAVE THE TOOLS TO STABILIZE A PATIENT.**

One subgroup proved the exception in Newgard, et al. Patients who presented in hemorrhagic shock during prehospital care and required emergent trauma center intervention did have better 28-day mortality when they arrived at a trauma center within 60 minutes. The same outcome improvement was not noted in head-injured patients.20

These two papers have come out challenging the golden hour since a 2001 literature review could find no clinical evidence for the instruction of the concept.21 Acknowledging the golden hour doesn’t exist doesn’t mean there is no time sensitivity to trauma center care. But it does mean there is not a definitive 60-minute threshold for decreasing morbidity or mortality. Challenging this myth helps push the notion that there are patients who only have minutes for EMS to bring lifesaving trauma care to their side.

Rather than thinking about a mythical 60-minute benchmark, focus on determining whether or not you have the tools to stabilize a patient, and if you do not then emergently transporting the patient directly to a trauma center that can provide such stabilization may outweigh the risks of emergency transport, which include provider and further patient injury in the event of a motor vehicle collision.

**The Bottom Line**
Delivery of patients suffering from a traumatic injury to a trauma center within 60 minutes of their incident does not improve their outcomes, unless they present in hemorrhagic shock. Safe transport to a trauma center is more important than rapid transport.

**Myth #9: MAST Improves Outcomes**

The myth explained: First introduced during the Vietnam War, military anti-shock trousers (MAST) and pneumatic anti-shock garments (PASG) continue to be used in many EMS systems for the acute management of severe hemorrhagic shock and the stabilization of suspected pelvic fractures. Since their war-driven introduction, MAST have been popular, albeit poorly studied prehospital interventions. The theory behind MAST is that their compression of the distal and then proximal lower extremities—and abdomen, if necessary—increases venous return to central circulation, which increases cardiac output.

**The Evidence**

When all compartments of MAST are inflated to 90 mmHg, cardiac output does rise.23 However, with time, cardiac output, systemic vascular resistance and venous return once again lower. No benefit is seen when MAST are inflated with less than 40 mmHg, and at most there is a 5% increase in central blood volume with full inflation.22

While some studies have demonstrated MAST application does provide some ability to control otherwise uncontrolled hemorrhage within the MAST application region—in the legs or abdomen—the opposite effect is seen when the hemorrhage location is not within the areas MAST compress. In these animal models survival time decreased from 60 to 10–18 minutes following MAST application.22 The correlation to a human would be that if a patient is experiencing shock from a hemothorax, the MAST could worsen their hemorrhage.

Only a few human patient studies have evaluated the use of MAST in the prehospital or emergency department settings for patients experiencing decompensated hemorrhagic shock. Two of these studies demonstrated no increased survival with MAST application, and one prospective randomized trial demonstrated a 6% increase in mortality with application.23 Overall, while there are select instances where MAST can provide hemorrhage control and increase the odds of a patient being admitted to the hospital, there is little to no data suggesting they improve patient outcomes. This is because MAST have no impact on blood flow proximal to the renal arteries.23 Finally, a Cochrane review determined MAST do not decrease a patient’s hospital or ICU stay, and they have no impact on patient morbidity or mortality.24

**The Bottom Line**
MAST likely have no benefit for trauma patients, although there is limited information about patients with long transport times and lower extremity trauma and pelvic fractures. Given the paucity of evidence for any additional benefit, traction splints and pelvic binders appear to be better focused interventions for these injuries.

**Summary**
Evidence-based medicine will continually change the paradigm in which emergency medicine is practiced. Fifteen years ago tourniquets were a last resort and often considered a guaranteed way to lose a limb; today they are a gold standard in hemorrhage control. Believing in, and having practiced, medicine we later learn to be false doesn’t make someone a bad provider, nor does it make them wrong. It simply means emergency medicine and EMS will continue to develop as a profession, and our body of evidence will continue to grow as we learn more about prehospital care.

As we prepare to retire MAST, backboards and lidocaine, and realize the gold-
en hour as a concept rather than a definitive 60 minutes, it’s important to keep a critical eye out for the next intervention that truly will help patients during their prehospital care.

REFERENCES