between heat index and PPR; PPR increases by 3% for every 10-degree increase in the heat index [Perron AD et al. *Prehosp Emerg Care.* 2005].

Dr Ossmann outlined 4 echelons of care that need to be addressed at any mass gathering: frontline staff (eg, ushers and security officers), mobile treatment teams, on-site medical facilities, and transfer arrangements with local hospitals.

A 25-year review of mass gathering events characterized them by size, number of off-site medical transports, and sudden cardiac deaths. Variables that best predicted medical usage, specific injury patterns, and levels of care included event type and ambient temperature [Milsten AM et al. *Prehosp Disaster Med.* 2003].

Dr Ossmann concluded by highlighting some key features of a robust event plan: accessible and functional first aid equipment, a large network of cardiopulmonary resuscitation–trained personnel, a dedicated event control center, a published communication plan, on-site physicians with experience and training, and internal and external surveillance and coordination.

Minimizing Compression Interruptions Key to Good Outcomes

Written by Muriel Cunningham

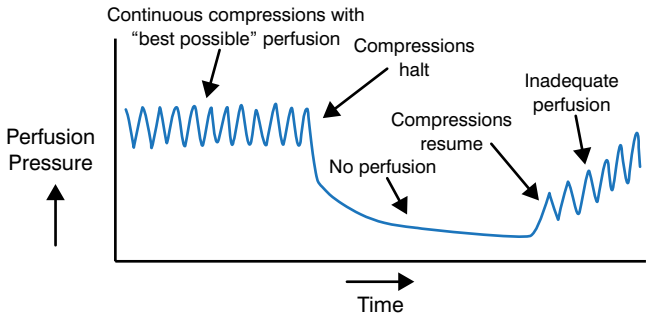
J. Scott Wieters, MD, Texas A&M University, Temple, Texas, USA, reviewed data confirming the importance of minimizing compression pauses during defibrillation of patients in cardiac arrest.

Although the 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care recommend stopping compressions during defibrillation, Dr Wieters stated that there is no convincing evidence in the literature to support this practice. From 1986 to 1990, 13 injuries were reported with "hands-on" defibrillation, most consisting of mild shocks or burns. In experimental models of hands-on defibrillation, current leakage (mean, 283 ± 140 μA; range, 18.9 to 907 μA) was within the acceptable safety limits [Lloyd MS et al. *Circulation.* 2008].

At the same time, there is substantial evidence indicating that interruptions in chest compression pauses should be avoided at all costs. Cardiac perfusion drops off dramatically when chest compressions stop, and perfusion takes time to rise when compressions resume (Figure 1). In one study, if the preshock pause was > 10 seconds, return of spontaneous circulation (ROSC) decreased by 50% [Eftestøl T et al. *Circulation.* 2002].

In another study, when preshock pauses were < 3 seconds, the ROSC was 6 times higher. Keeping postshock pauses to < 6 seconds led to 18 times more ROSC [Edelson DP et al. *Resuscitation.* 2006]. In a large multicenter trial of 815 patients with out-of-hospital (OOH) cardiac arrest, patients with a preshock pause of < 10 seconds had 50% less mortality when compared with patients with a preshock pause > 20 seconds [Cheskes S et al. *Circulation.* 2011]. Every 5-second delay led to 18% mortality.

Figure 1. Effect of Compression Pauses on Cardiac Perfusion



Adapted from *American Journal of Emergency Medicine*, 30, Cunningham LM et al, Cardiopulmonary resuscitation for cardiac arrest: the importance of uninterrupted chest compressions in cardiac arrest resuscitation, 1630-1638. Copyright (2012), with permission from Elsevier.

Dr Wieters emphasized that compressions should certainly continue during preshock charging and that, after defibrillation, end tidal carbon dioxide should be employed in place of pulse checks to monitor perfusion.

In a prospective observational cohort study of 506 cases of OOH cardiac arrest, the best survival (28.7%) was seen when the compressions were performed 60% to 80% of the total resuscitation time [Christensen J et al. *Circulation.* 2009]. Physicians should therefore aim for a chest compression fraction > 80%. Dr Wieters concluded by stating that a shock delivered with perfusion pressure at its peak will more likely result in ROSC.

Latest Drugs and Guidelines for Treating Hemostasis

Written by Toni Rizzo

Nilesh Patel, DO, St Joseph's Regional Medical Center, Paterson, New Jersey, USA, presented the latest drug developments and guidelines regarding hemostasis. He discussed tranexamic acid (TXA) for the reversal of bleeding in target-specific oral anticoagulants (TSOAs) and the use of thromboelastography (TEG) for monitoring hemostasis in trauma and critically ill patients.

TXA is an antifibrinolytic agent that reversibly binds plasminogen, blocks plasmin, stabilizes clot formation, and combats hyperfibrinolysis. It is administered intravenously or orally at variable doses. TXA is more potent than aminocaproic acid. It prolongs thrombin time and can be monitored with TEG. TXA is used for patients undergoing surgery to decrease bleeding and blood transfusions and for patients with bleeding disorders.

The CRASH-2 trial [Shakur H et al. *Lancet.* 2010] of TXA included 20 211 trauma patients at risk for significant hemorrhage, and it demonstrated that TXA significantly reduced all-cause mortality (14.5% vs 16%; $P = .0035$) with similar vascular occlusive events (1.7% vs 2%) as compared to placebo. The military study MATTERs [Morrison JJ et al. *Arch Surg.* 2012] included 896 patients with combat injuries, and it found that patients treated with TXA vs no TXA had reduced mortality (17.4% vs 23.9%; $P = .03$). However, in a single-center US study, mortality in high-risk trauma patients treated with TXA was 27%, as compared with 17% without TXA ($P < .05$) [Valle EJ et al. *J Trauma Acute Care Surg.* 2014]. The PATCH study [Mitra B et al. *Emerg Med Australas.* 2014] is evaluating early administration of TXA in severely injured patients to try to resolve some of this uncertainty.

A 1983 *New England Journal of Medicine* study in patients with acute upper gastrointestinal bleeding found that in-hospital mortality was 6.3% with TXA vs 13.5% with placebo ($P = .0092$). A review of TXA treatment in patients with upper gastrointestinal bleeding found significant improvements in death rates but no significant improvement in rebleeding rates, need for transfusion, or surgery [Morgan A, Jeffrey-Smith A. *Emerg Med J.* 2012]. TXA for routine use in gastrointestinal bleeds needs more prospective data before widespread use can be endorsed.

TSOAs include the direct thrombin inhibitor, dabigatran, and the factor Xa inhibitors rivaroxaban and apixaban. Dabigatran does not require monitoring, but increasing numbers of patients receiving dabigatran have developed bleeding complications. In a prospective review of patients admitted to the hospital for dabigatran- or warfarin-induced bleeding, patients receiving dabigatran had more gastrointestinal bleeding (80% vs 48%), less intracranial bleeding (0% vs 32%), and a shorter hospital stay (3.5 vs 6.0 days) [Berger R et al. *Ann Emerg Med.* 2013].

Dr Patel stated that while the TSOAs do not need laboratory monitoring and have lower rates of drug-drug and drug-food interactions, there is no established antidote for patients who are bleeding or need surgery. Routine supportive care, activated charcoal within 2 hours, and hemodialysis have been suggested for patients on direct thrombin inhibitors with bleeding emergencies [Kaatz S et al. *Am J Hematol.* 2012]. Prothrombin complex concentrate has been shown experimentally to reverse the anticoagulant effect of rivaroxaban but not dabigatran in healthy patients [Eerenberg ES et al. *Coagulation.* 2011]; prothrombin complex concentrates can be considered for use in bleeding emergencies related to both drug classes based on limited data.

TEG is a coagulation test that provides rapid assessment of the coagulation cascade. Studies of TEG have found that test abnormalities correlate with increased transfusion rates and mortality [Carroll RC et al. *Transl Res.* 2009] and that TEG results were accurate when compared with a formal assay [Reed MJ et al. *Eur J Emerg Med.* 2013]. TEG may find nice use in bleeding trauma patients to guide coagulation-directed therapy and resuscitation.

Dr Patel concluded that more data are needed for treating hemostasis with TXA outside of trauma populations. More data are also needed to find the optimal reversal strategies for bleeding emergencies on TSOAs. Finally, TEG is an upcoming coagulation assay that shows promise in our bleeding patients.

ACEP Clinical Policy Updated for New and Refractory Seizures

Written by Emma Hitt Nichols, PhD

Up to 5% of individuals in the United States will experience a nonfebrile seizure during their lifetime. Yet, the accurate diagnosis of seizure can be challenging for emergency department (ED) physicians. Jordan Bonomo, MD, University of Cincinnati Medical Center, Cincinnati, Ohio, USA, discussed the American College of Emergency Physicians' (ACEP) 2014 update regarding the evaluation and management of adult patients presenting to the ED with seizures [Huff JS et al. *Ann Emerg Med.* 2014].

In this update of the 2004 clinical policy, seizure definitions were modified. For example, status epilepticus (SE) was defined as clinical or electroencephalographic (EEG) seizure activity for >5 minutes, continuously or recurrently, without full recovery between events. SE is categorized as convulsive, nonconvulsive, or refractory. Dr Bonomo pointed out that most seizures do not meet these criteria because seizure activity lasts <5 minutes.

The 2014 policy provides level B recommendations that all patients presenting with seizure should be evaluated with blood glucose and sodium levels. In addition, all women should be tested for pregnancy, and immunocompromised patients should undergo lumbar puncture. Although the policy does not provide level