Neurotrauma & Critical Care NEWS

AANS/CNS Section on Neurotrauma & Critical Care

Fall 2018

In This Issue…

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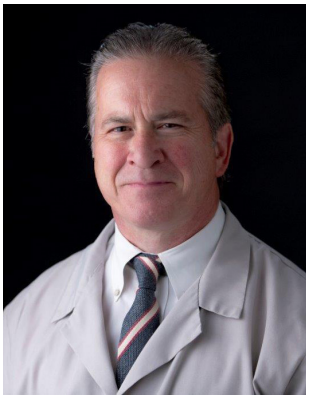
CHAIR'S MESSAGE

Dear Colleagues,



Julian E. Bailes Jr., MD, FAANS

I would like to congratulate Dan Michael, MD, PhD, FAANS, on his recent exemplary service as chair of the AANS/CNS Section on Neurotrauma and Critical Care (SNTCC). Dan's enthusiasm, expertise and commitment to organized neurosurgery have continuously shone during his SNTCC leadership, and we have all benefited from his insight and dedication in advancing the delivery of trauma care.

In assuming the leadership of the SNTCC, I foresee several reforms on our immediate horizon, which will influence the delivery of trauma care. By far, the largest of these is the drive to reduce health care spending, which is being stimulated by pressure to lower the price of care but not necessarily the cost of care. New methods and delivery networks are vital to reform, but also will influence the delivery of trauma care.

The fourth edition of the Brain Trauma Foundation (BTF) Guidelines for the management of Severe Traumatic Brain Injury were published online in 2016 and the Journal of Neurosurgery published them in print in 2017. Originally published in 1995, studies have shown that adherence to these guidelines can improve neurological outcomes and reduce mortality. Numerous stakeholders have embraced and maintained the BTF Guidelines, which have been translated into more than 15 languages and utilized on several continents. The intention is to make the BTF Guidelines a living document, so it is continuously and dynamically updated. However, much work is needed concerning the adherence to BTF Guidelines, which has varied dramatically across various subject areas and geographic location, mainly due to the strength of the association of benefit. There remains much work to be done across the globe to conduct the research needed to validate and standardize these guidelines further as well as to improve compliance and care patterns. An example of this is that although ICP monitoring has become a standard of care for adults and pediatric patients in most trauma centers, there is not universal compliance and no agreement on which type of monitor is superior or which factors are associated with the best outcomes. This is a direct result of a lack of complete data in this area – more research needs to be carried out.

There are many new and emerging aspects to the care delivery and achievement of better outcomes for the traumatic brain injury (TBI) patient. These include, but are not limited to, assessment and imaging of cerebrovascular functioning; inpatient and outpatient rehabilitation and resource utilization; and implementation of the electronic medical record in the trauma setting. It is estimated that 50 million people in the United States live one hour or more from a trauma center, so access to specialty care remains paramount.

Continued on the next page

Editor: Martina Stippler, MD, FAANS

Chair's Message (Continued)

Telemedicine has been proposed as one technological aspect for the improved delivery of trauma care, particularly as those who live in rural areas are at increased risk of both traumatic injury and trauma-related mortality. There are few rural hospitals that are staffed to provide diagnosis and care for the neurological or spinal-injured patient. Recent analyses have shown that telemedicine systems in trauma care may improve quality, reduce length of stay and increase patient and family satisfaction. Several studies have shown a decrease in unnecessary transfers and thus cost savings. In addition, telemedicine applications to mass casualty trauma care could be important in the future, and advances must include aspects such as improved communication, increased wireless coverage, up-linkage of digital data and provider availability among others.

Concussion continues to be an important and current topic for civilians, military service members and athletes. The diagnosis remains elusive, as a concussion is ordinarily a subjective presentation and there are no outward or visible signs of injury. Concussion diagnosis remains one of the most challenging tasks facing the clinician and emerging technologies are using neuropsychological testing, ocular function assessment, electroencephalography network patterns, brain pulsatility and other methods. A recent study by Adrian et al. utilized the biomarkers UCHL1 and GFAP to predict the presence of intracranial lesions on CT scans, representing the first FDA approved blood test to document a mild TBI. This technology is not, however, a concussion test nor approved for use in pediatric patients.

There are many contemporary aspects of neurosurgical trauma coverage for hospitals. These include establishment of trauma care networks, the issues of call coverage, involvement of trauma specialties, gun violence, encouragement of younger neurosurgeons' involvement and other factors. Immediate care centers are going to impact how some trauma victims enter the hospital systems. The landscape may continue to change as urgent and immediate care centers proliferate, particularly regarding neurosurgical issues that include stroke and intracerebral hemorrhage. "Urgent care can play a pivotal role as a first responder for trauma care, especially in areas where access to hospital-based services are limited," says Frank Alderman, MD, Chairman of MedExpress Urgent Care, the leading provider of walk-in health care, with more than 250 neighborhood medical centers in 19 states. "Urgent care facilities that are appropriately staffed with full medical teams and higher level diagnostics have the ability to rapidly evaluate and effectively stabilize trauma patients and then expedite care coordination to the most appropriate follow-up setting, in most cases saving both time and money."

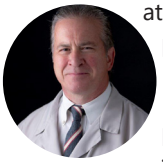
Decision support tools, TBI care advancement, involvement with policy making, and funding for trauma lectureships are all works-in-progress for this upcoming year. The SNTCC continues to be on the cutting edge and very relevant to our members and the public they serve.

Julian E. Bailes Jr., MD, FAANS
Chair, AANS/CNS Section on Neurotrauma and Critical Care

Committee Updates

Sports Injury Committee Report

by Julian E. Bailes Jr., MD, FAANS



The involvement by neurosurgeons in neurological sports medicine continues at a rapid and profound pace, dating back to the beginnings of the Sports Medicine Committee becoming active in the mid-1980s. At that time, neurosurgeons were essentially the sole providers of care for athletes with known or suspected brain or spinal injuries. Fifty years ago, Richard Schneider, who was professor and chairman of the University of Michigan Department of Neurosurgery, produced several important books and papers concerning the identification, management and prevention of neurological sports injuries. During the last several decades, neurosurgeons have held leadership positions in sports organizations at all levels of play in numerous sports, particularly football. From youth sporting leagues, such as Pop Warner Football, to high school, college and professional sports, neurosurgeons are now active in player management, rules committees, safety initiatives and other aspects. Allen Sills, MD, FAANS, is the new NFL Chief Medical Officer, while Nick Theodore, MD, FAANS, heads up the NFL Head, Neck and Spine Committee. Tanvir Chowdhri, MD, FAANS, now leads the SNTCC Sports Medicine Committee, Anthony Petraglia, MD, is vice-chair and Hamad Farhat, MD, FAANS, is also serving on the committee. There are numerous opportunities for further advancement of prevention and management strategies and for neurosurgeons to become more involved in recreational and scholastic sports programs throughout their communities; for their local schools and recreational leagues. Numerous sports are evolving rules, playing styles and methods of injury assessment, leading to a dynamic state of affairs in which neurosurgeons should wholeheartedly participate.

Membership Report

by Martina Stippler, MD, FAANS

Currently, the AANS/CNS Section of Neurotrauma and Critical Care has 479 active members, 84 international members and 229 lifetime members. The lifetime membership is currently free for retired neurosurgeons. We are working on changing the rules and regulations of the Section to be able to allow for a one-time fee for the privilege to become a lifetime member, which still allows access to memberships benefits. Over the last year, we were able to add more membership perks. In the addition to the biannual section newsletter, there is also now a membership-only section on our website. There one can find neurotrauma-related slideshows – for example, a summary of the fourth edition of the Guidelines for the Management of Severe Traumatic Brain Injury. These section-approved slideshows can be used for your own education or to give TBI-related talks.

Scientific Program Committee Report

by Alan Hoffer, MD, FAANS

The AANS/CNS Section on Neurotrauma and Critical Care is excited to announce the neurotrauma offerings for the 2018 Congress of Neurological Surgeons meeting in Houston this fall. This year's program covers the full spectrum of traumatic injuries to the central nervous system, from mild injuries like concussion to the most severe brain and spine injuries. In a variety of settings, including weekend courses with hands-on experiences, debates, didactic sessions and case discussions, we will explore the most recent developments in diagnostic tools, treatment options and controversies in neurotrauma. These sessions are designed to enrich neurosurgeons from every practice setting, from general to specialized practices. Session topics include concussion diagnosis and management, management of chronic subdural hematoma, ethical controversies on neurotrauma and severe TBI update. We look forward to seeing you in Houston!

CSNS Neurotrauma and Emergency Neurosurgery Committee Report

by Adair Prall, MD, FAANS

The CSNS Neurotrauma and Emergency Neurosurgery Committee has been busy this year. In addition to a number of other projects, a comprehensive survey and review of emergency and trauma call coverage among American neurosurgeons was conducted last year. These data will be published in Neurosurgery in the next several months and will serve as the first published baseline criteria for our colleagues to consider when discussing call coverage criteria with their own facilities. This landmark study includes both topic-related data as well as demographic and center-specific data. First author Maya Babu, MD, MBA, will also be engaged in a forthcoming project, looking at billing and utilization practices among neuro-ICUs in this country. The hope is to identify best practices for neurosurgeons currently engaging in neurocritical care in both academic and community settings.

Publication Committee Update

by Martina Stippler, MD, FAANS

The Section is now on twitter! It is about time. Please follow us to learn about CME opportunities, TBI controversies as our EC members see it and the newest TBI research updates. We also have slideshows for our members to download on our website: http://www.neurotraumasection.org/member-corner. Use them to learn or give talks about trauma. Contact Kristin Zerfas at kmz@aans.org if you need a password. We are also working on our next newsletter. If you want to get involved, contact me at Mstipple@bidmc.harvard.edu. I also want to take this opportunity to introduce the new co-chair of the publication committee: Dr. Laura B. Ngwenya, MD, PhD. She is an assistant professor at the department of neurosurgery at the University of Cincinnati, where she holds the title of director of the Neurotrauma Center of the University of Cincinnati Gardner Neuroscience Institute.

Hemostasis Update: Reversal Strategies for Antithrombotics in Patients with Traumatic Intracranial Hemorrhage

David Dornbos III, MD;
Shahid M Nimjee, MD, PhD

While anti-platelet and antithrombotic therapeutics are necessary for numerous cardiovascular conditions, they pose significant treatment difficulty for neurosurgeons in the setting of acute traumatic intracranial hemorrhage (ICH). For patients undergoing elective neurosurgical procedures, the transition off of antithrombotics can be done in a controlled manner, allowing natural resumption of innate coagulation. Patients presenting in an emergent fashion with traumatic, life-threatening hemorrhages (Figure 1) often need rapid reversal of anticoagulants to allow appropriate hemostasis to facilitate the prevention of hemorrhage progression or take the patient to the operating room. The advent of newer, more powerful anti-platelet and anticoagulant medications has presented new challenges in developing reversal protocols; however, new antidotes and reversal agents have shown significant promise.

Anti-platelet agents, namely aspirin and clopidogrel (Plavix), are among the most ubiquitous antithrombotics agents encountered in the general population. Lose dose (81 mg) aspirin carries no significant increase in frequency when compared to the general population with <1 percent of ICH overall.[1] However, full dose aspirin, particularly when combined with clopidogrel, shuts down the functionality of affected platelets for the lifespan of the platelet, which can lead to significant propagation of traumatic ICH. Additionally, other P2Y12 inhibitors (ticagrelor, prasugrel), thromboxane inhibitors (dipyridamole), phosphodiesterase inhibitors (cilostazol) and PAR-1 inhibitors (vorapaxar) are used with increasing frequency in the general population with similar anti-platelet effects that can have profound implications in traumatic ICH. Unfortunately, no reversal agents exist for any of the aforementioned platelet antagonists and cessation of these medications requires production of new platelets to restore physiologic platelet function. While controversial, platelet transfusion with or without desmopressi may be used in emergency settings to restore normal platelet function.

Despite the advent of newer agents, warfarin remains the most frequently encountered oral anticoagulant, used in the treatment of venous thromboembolism, stroke prevention in atrial fibrillation and with mechanical heart valves. When surgical intervention for traumatic ICH is not immediately imminent, discontinuation of warfarin alone with administration of oral or intravenous vitamin K may be warranted. Situations involving significant, life-threatening ICH require the immediate replacement of the inhibited coagulation factors, which may be done with either fresh frozen plasma (FFP) or prothrombin complex concentrate (PCC).

While reversal strategies for anti-platelet agents and warfarin have remained relatively similar over the past several years, the development of Novel (New) Oral Anticoagulants (NOACs) – direct factor Xa and thrombin inhibitors – have posed significant difficulties pertaining to their reversal in the setting of severe traumatic ICH. While low molecular weight heparin (enoxaparin) can be reliably reversed with protamine in a dose and time-dependent fashion, reversal of other factor Xa inhibitors (fondaparinux, apixiban, rivaroxaban, edoxaban) has been significantly more challenging. Over the past several years, these newer agents could only be reversed in an emergent setting with PCC. While this will provide anticoagulant reversal, it also increases the risk of developing a myocardial infarction, stroke or pulmonary embolism.

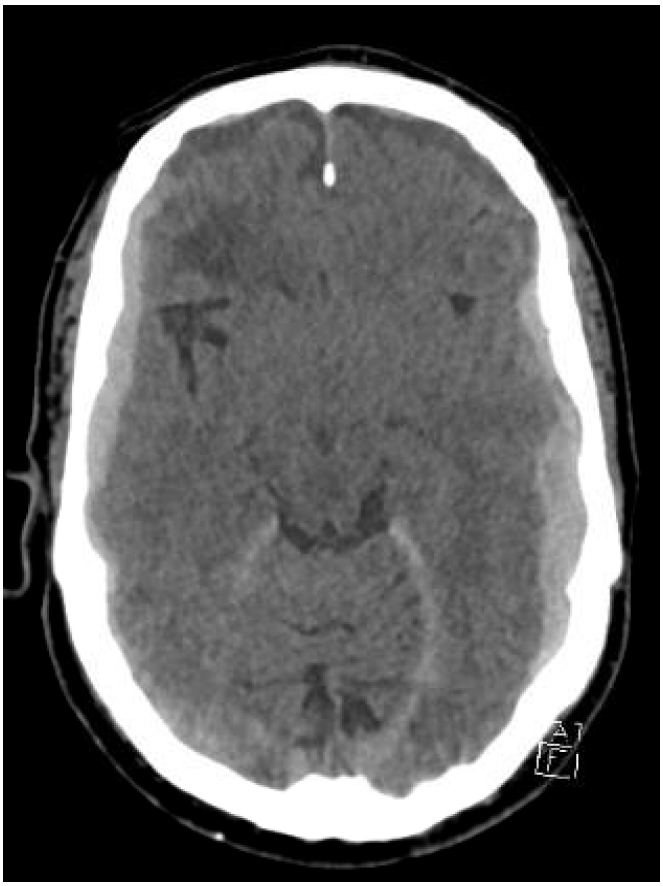


Figure 1

Recently, the Food and Drug Administration (FDA) approved andexanet alfa for routine clinical use. Andexanet is a recombinant modified human factor Xa decoy protein that binds the active site of factor Xa inhibitors with high affinity. Given its highly selective nature, andexanet alfa is able to completely reverse factor Xa inhibitors within minutes of administration.[2] While this antidote is new to the market, it provides a rapid

and robust treatment to ameliorate potentially life-threatening propagation of a traumatic ICH.

Similarly, dabigatran (Pradaxa®) is an oral direct thrombin inhibitor. For patients presenting with traumatic ICH and one of these anticoagulants, further worsening of the traumatic hemorrhage is likely. In severe, life-threatening cases, PCC can be used, but its ability to reverse the effect of dabigatran is limited. Fortunately, an antidote, idarucizumab, has recently been developed. An antibody fragment, this antidote is able to reverse the anticoagulant effects of dabigatran within minutes of infusion.[3]

Although the effects of antiplatelet and anticoagulation therapeutics can be catastrophic in patients following traumatic ICH, numerous strategies exist to safely reverse their effects and restore normal hemostasis. Fortunately, for anticoagulants previously without adequate reversal strategies, new antidotes have been developed that can limit potential hemorrhage propagation and provide a modicum of safety for neurosurgeons to operate emergently, if needed. A thorough understanding of these drug classes and their reversal strategies is imperative for both operative and non-operative hemorrhages to provide patients with optimal hemostasis, limit hemorrhage size and enhance surgical safety.

David Dornbos III, MD;

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Spreading Depolarizations: A Window into the Black Box of the Traumatically Injured Brain

Jed A. Hartings, PhD

A major obstacle in advancing treatment of acute brain trauma is the lack of mechanistic targets for personalized treatment. Most current neuromonitoring techniques measure variables that reflect injury processes, but are not themselves neuronal mechanisms of worsening. Recent developments in the study of spreading depolarizations (SD) offer hope to improve this situation. SDs, first discovered in 1944,[1] are pathologic mass waves of complete cellular depolarization in cerebral gray matter and are the largest known disturbance of still viable tissue. Their disruptive impact is seen at all levels of cellular and tissue function, including neurochemistry, organelle ultrastructure, blood-brain barrier, microvascular flow and metabolism. Based mainly on studies of ischemic stroke in rodents, SDs have long been recognized as an important mechanism driving the toxic changes that lead to infarction. However, it was not until neurosurgeon Anthony Strong, MD (King's College Hospital, London), first observed these events in the human brain in 2002 that they gained clinical and translational relevance.

SDs are monitored in patients by bedside recording from subdural electrode strips placed during a craniotomy to treat acute brain injury. That research has shown that a surprising 50-60 percent of brain trauma patients and over 80 percent of patients with aneurysmal subarachnoid hemorrhage (aSAH), experience SDs as a secondary injury process. Yet, the success in translation is not only in finding that SDs occur, but in finding that they have the same or even more harmful effects as shown in animals. For instance, in many patients – not all – SDs are seen not just sporadically, but as a continuously recurring pattern for days or more. In brain trauma, the peak incidence lasts about 72 hours, while aSAH patients often have a delayed peak coinciding with the period of risk for delayed cerebral ischemia. (By comparison, an episode of migraine aura is thought to be mediated by just a single SD wave lasting about 20 minutes.) Through these long series of SDs, there is often clear evidence of their effect to cause progressive deterioration of tissue function and energy status. For instance, brain activity becomes more depressed with each wave until it persistently 'flatlines', and these patterns correspond to the development of new brain lesions.[2] Thus, for the first time we can now monitor not only the development of secondary injury at the tissue level, but also the mechanism that causes it.

> "...for the first time we can now monitor not only the development of secondary injury at the tissue level, but also the mechanism that causes it."

SDs cause injury not only by short-circuiting electrochemical gradients, but also by restricting vital blood flow at the microvascular level. This phenomenon, known as spreading ischemia, was discovered 20 years ago when Jens Dreier, MD, PhD (University Medicine Berlin, Berlin), studied the effects of erythrocyte breakdown products on vascular responses to SD. Instead of the large increase in blood flow that SD evokes in the intact brain (e.g. in migraine aura), the presence of high potassium and free hemoglobin instead causes a severe vasoconstriction. This decrease in blood flow, triggered by and propagating with the SD wave, is sufficient to cause brain infarction. Clinical studies have now shown that the same mechanism of spreading ischemia occurs in patients with poor-grade aSAH and after severe brain trauma. As for the progressive deterioration of brain function measured by subdural electrodes, blood flow measures similarly show a progression of worsening tissue ischemia induced by a series of repetitive SDs.

What do these insights mean for the future treatment of patients with these conditions? To answer this, it will be important to investigate how patterns of SDs relate to other clinical assessment tools, management decisions, patient courses and outcomes. Thus far, it appears that

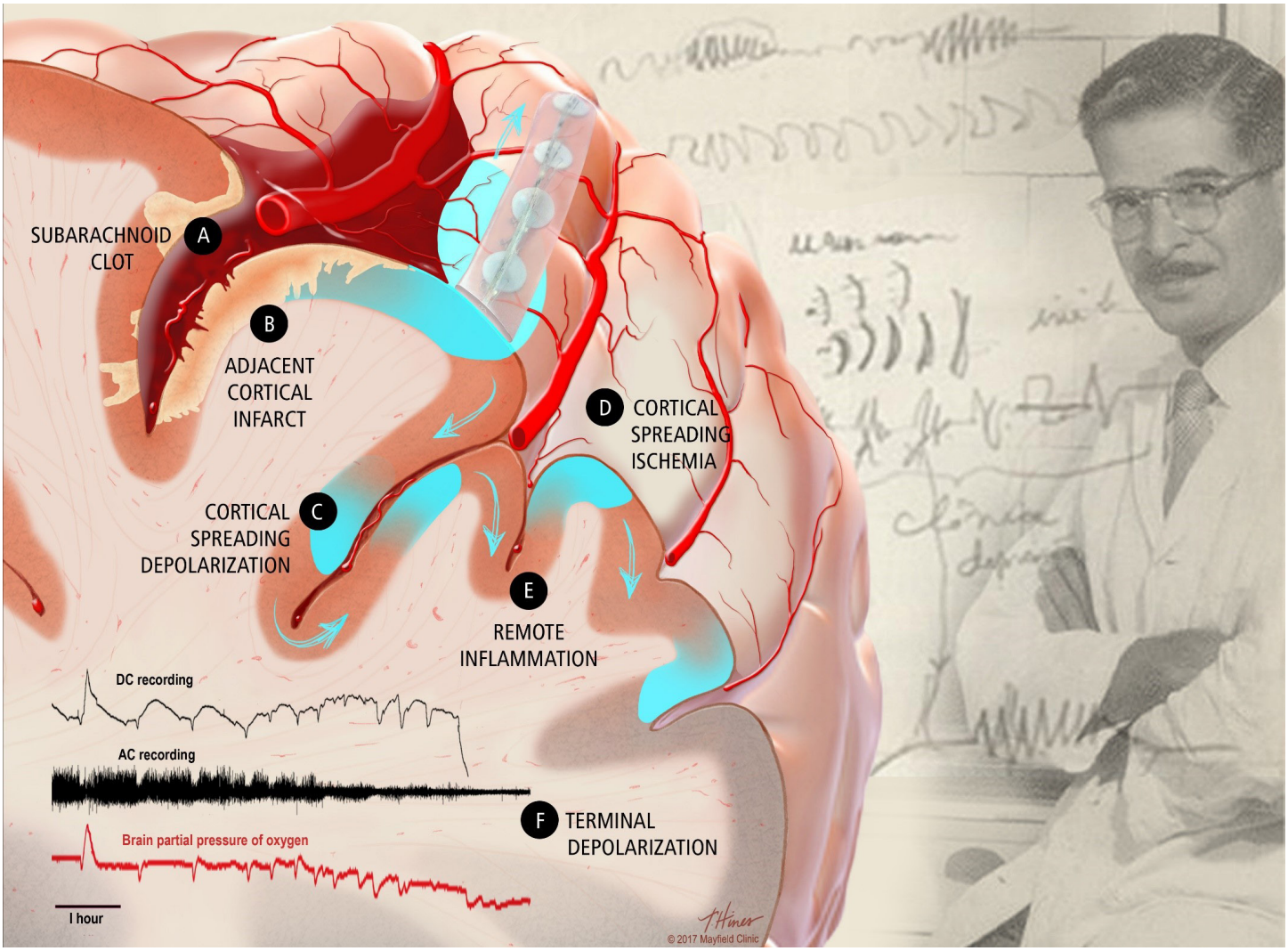


Figure: Illustration of spreading depolarizations provoked by subarachnoid blood. Blood accumulation in sulci (A) provokes adjacent cortical infarction (B) and repetitive waves of spreading depolarizations (C). Depolarizations induce spreading ischemia (D) and are a marker and mechanism of infarction. Each depolarization causes a progressive decrease in brain activity and brain tissue oxygenation (F). Depolarizations also trigger pathologic changes such as inflammation and blood-brain-barrier opening in remote, viable tissue (E). Spreading depolarizations were discovered by Aristides Leão, who is shown in the background with original sketches of his recordings from rabbit cortex. *Used with permission, (c) Mayfield Clinic*

SDs are independent predictors of worse outcome and can serve as a surrogate endpoint or early warning of a worsening course.[3] We found, for instance, that trauma patients who underwent primary decompressive craniotomy with lesion evacuation had fewer and less severe SDs than those who underwent traditional craniotomy with bone flap replacement; they also had better outcomes. Such results naturally raise the question of whether SDs can be stopped and whether doing so would improve outcomes. A retrospective review of analgo-sedative drugs used at multiple centers across the U.S. and Europe suggested that ketamine, an NMDA receptor antagonist, may be an effective agent against SDs and a recent prospective randomized trial seems to confirm this result. This is interesting since ketamine is already seeing increased use in emergency medicine.

On the other hand, it may be too simplistic to think of SD as a single mechanism that would indicate a single treatment. There are likely many tissue pathologies that result in SD provocation, and it is just as important to consider amelioration of these conditions as it is to block the SD itself.[4] For instance, if SD is triggered by ischemic conditions, it is important to determine the cause of ischemia and reverse it. In other words, we must think of SD within the full systems context of patient care and brain health, and learn

Continued on the next pages.

YOUR DAY TR NEUROTRAUMA

OCTOBER 6 SATURDAY

PRACTICAL COURSE
8:00 am–4:00 pm
PC01 | Severe TBI Update: Trends and Tools (pg 28)

OCTOBER 7 SUNDAY

PRACTICAL COURSE
8:00–11:30 am
PC25 | Concussion: Advances in Mild Brain Injury Science (pg 35)

OCTOBER 8 MONDAY

LUNCH SEMINAR
12:15–1:45 pm
M07 | Management of Chronic Subdural Hematoma (pg 42)

CASE-BASED DISCUSSION
4:15–6:15 pm
Cased-based Discussion Session—Challenging Cases: TBI (pg 47)

OCTOBER 9 TUESDAY

7:00–8:30 am
Sunrise Science and Late Breaking Abstract Session

LUNCH SEMINAR
12:15–1:45 pm
TI7 | Combat and Mass-casualties: Neurosurgery Under Fire (pg 52)

SECTION SESSION
2:45–4:15 pm
Section on Neurotrauma and Critical Care (pg 54)

CASE-BASED DISCUSSION
4:15–6:15 pm
Case-based Discussion Session: Ethical Controversies in Neurotrauma (pg 56)

OCTOBER 10 WEDNESDAY

LUNCH SEMINARS
12:15–1:45 pm
W27 | Sports-related Injuries (pg 62)

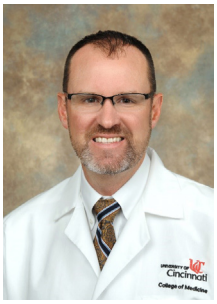
W28 | Pediatric Head Trauma (pg 62)

Look for the TR throughout the program for sessions and science that pertain to your specialty.

Spreading Depolarizations (Continued)

how to integrate SD data within this context. Toward this end, a multi-center observational clinical study is currently being conducted, with Department of Defense funding, as an adjunct to the well-known national initiative, Transforming Research and Clinical Knowledge in Traumatic Brain Injury (TRACK-TBI). Goals of this study include the development of less invasive and more automated methods to measure SDs as well as comparison of SD measures with other advanced assessment candidates, such as blood-based biomarkers and advanced neuroimaging. A similar observational study of SDs in aSAH patients, Depolarizations in Ischemia after Subarachnoid Hemorrhage-1 (DISCHARGE-1), is also being conducted in Germany and is nearing completion.

Another exciting aspect about SDs is the potential for bidirectional translation research, since SDs appear to be a widely conserved feature across the animal kingdom. Motivated by clinical findings, for instance, studies in the gyrencephalic swine brain have become more common in recent years. Imaging the exposed hemisphere has shown the complex nonlinear reaction-diffusion patterns of SD propagation that are possible when sulci and gyri are present. These include patterns of waves splitting, colliding, annihilating and cycling in re-entrant loops, providing critical insight into complex patterns evidenced in clinical recordings. Swine studies have also shown that blood accumulation in the subarachnoid space is sufficient to induce SDs and cause infarction of adjacent cortex. Lower down in the 'great chain of being', SDs even occur in the nervous systems of invertebrates, such as locusts and fruit flies (Drosophila melanogaster). In these species, they appear to be a survival mechanism that shuts off organism functions in response to environmental threats, such as extreme temperatures or food deprivation. It is not hard to imagine how comparative physiology could provide insight into the causes and consequences of SDs and the pathophysiology of coma. Furthermore, Drosophila offers a powerful genetics platform to better understand the molecular mechanisms of these phenomena. These and related developments have collectively cultivated a new and growing translational science community to better understand the role of SDs in neurological disorders. This community initially started as the Co-Operative Study on Brain Injury Depolarizations (COSBID), but annual meetings are now known as the International Conference on Spreading Depolarizations (iCSD). This year's meeting is September 22-24 in Boca Raton, Fla., as an official satellite of the Annual Meeting of the Neurocritical Care Society. The program includes talks running the gamut from single ion channel physiology all the way to clinical outcomes, with more than 100 attendees from a dozen countries expected. We invite the neurosurgery community to learn more about SDs by visiting the COSBID website and attending future meetings. Without the keen interest and participation of a few pioneering neurosurgeons 15 years ago, this story would never have had a beginning. It will be interesting to learn how the next chapters unfold.



Jed A. Hartings, PhD, Associate Professor, Department of Neurosurgery, University of Cincinnati

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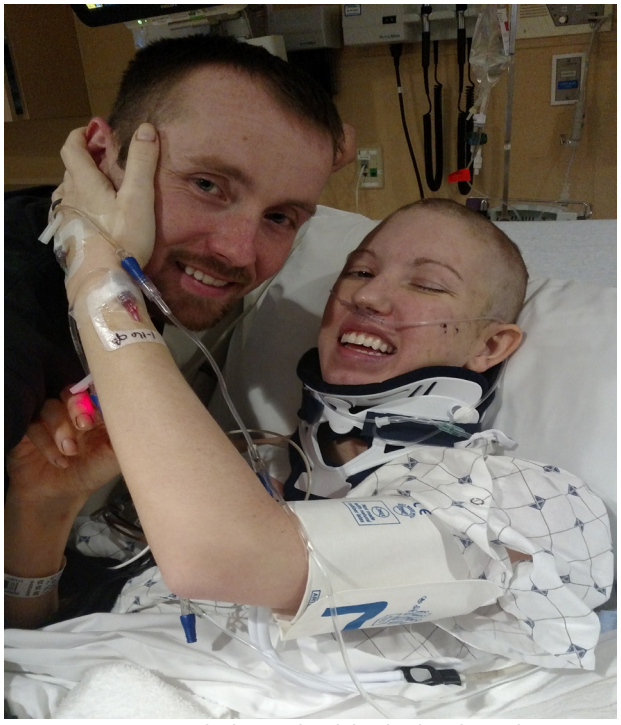
Life Day: A Patient Story

Laura B. Ngwenya, MD, PhD

On a January morning, a "trauma stat" was called and a 26-year-old woman arrived in the emergency room from the scene of a motor vehicle collision. Emergency care reported that she was GCS 3 at the scene with bilaterally unreactive pupils. In the trauma bay, ATLS protocol resuscitation ensued, she received hypertonic saline, and her neurologic exam improved to normal flexion on her motor exam and sluggishly reactive pupils. Imaging revealed a catalog of injuries: evolving right epidural hematoma with mass effect, multifocal acute cerebral artery dissections, cavernous carotid fistula, C1 lateral mass fracture, right clavicle fracture, bilateral pneumothoraxes, pulmonary contusions, grade 1 splenic injury, left femur fracture and multiple facial fractures including left zygomaticomaxillary complex, bilateral pterygoid plates, mandible fracture and nasal bone fracture. She was taken emergently for evacuation of the epidural hematoma. She later underwent diagnostic and interventional cerebral angiography and surgical fixation of her orthopedic fractures. For the physicians involved in her care, this was a typical day and course of events at a Level I trauma center. For Kourtney Hurst, this was her "Life Day".



Kourtney in the hospital with her husband Brandon.

Kourtney Hurst was a happy newlywed on the fateful morning. On her commute to work, her pickup truck slid on a patch of black ice and she collided with a telephone pole. Her husband, Brandon, called Kourtney to make sure she had arrived at work safely when he heard about the bad road conditions. He did not get a response. He "had a feeling" that something was wrong, so he got in his vehicle and drove Kourtney's route to work. He found her crashed vehicle, called 911 and witnessed the emergency response efforts.

Kourtney's head had hit the driver's side window. The truck windows had to be broken and the doors removed in order to retrieve her from the vehicle. She was transported to the Level I trauma center after a prolonged extrication.

One year after her injury, Kourtney visited with the team that took care of her during her hospital stay

Because of her multiple injuries, Kourtney had a two-week hospital stay (image 1). On the day she left for the inpatient rehabilitation hospital, she was able to take a few steps and converse with the team. She spent many weeks in rehabilitation, participated in physical and occupational therapy for months and sought counseling for PTSD symptoms.

After six months of determined recovery, Kourtney returned to her job as a cardiac catheter nurse at a neighboring hospital. She now has a new outlook on life. "January 11 is a new birthday for me. We call it my Life Day," says Hurst. She celebrates the 11th of every month as a reminder to herself and others of the fragility

Continued on the next page.

One year after her injury, Kourtney visited with the team that took care of her during her hospital stay.

of life. A green ribbon, for Traumatic Brain Injury Awareness, marks each 11th-day-of-the-month on her kitchen calendar. "Since my accident, I've really learned the value and importance of life, and understand that we are blessed to wake up every morning." On January 11, 2018, one year after her almost fatal injury, Kourtney returned to the ICU and neurosciences unit to thank her team of providers (Images 2a & 2b). She also shared the exciting news that she and her husband were expecting their first child. In June, Kourtney and Brandon welcomed their new baby girl, who she says is "our greatest miracle yet!" She continues to celebrate each month of her renewed life.

Kourtney stands by her new truck. She has returned to work and she advocates for Traumatic Brain Injury awareness.

Kourtney Hurst was treated at University of Cincinnati Medical Center. More information on her story can be found at the following resources and online websites:

Ann Heise Kult. "It takes a team: Thankful to be alive" Lead Cincinnati Magazine.

Dama Ewbank. "Young woman celebrates life after accident nearly took hers." UC Health Central Line Newsletter. January 2018, page 5.

UC Health Patient Profile: Kourtney Hurst. YouTube.

Liz Bonis. "Woman seriously injured year ago returns to hospital to thank team who saved her." WKRC Cincinnati. Thursday, January 11, 2018.

If you have a patient story you would like to share for a future newsletter, contact the publication committee: Laura.Ngwenya@uc.edu and/or Martina.Stippler@partners.org.

Laura B. Ngwenya, MD, PhD

University of Cincinnati, Department of Neurosurgery
Assistant Professor, Director Neurotrauma Center

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