SAVE THE DATE
Western Trauma Association
49th Annual Meeting
March 3-8, 2019
Westin Snowmass Resort
Snowmass, Colorado
FORTY-EIGHTH ANNUAL MEETING

February 25 – March 2, 2018
Whistler, British Columbia
Dear Members, Friends and Guests:

On behalf of the Officers and Board of Directors, I want to warmly welcome you to the 48th Annual Meeting of the Western Trauma Association in Whistler, British Columbia, Canada. We last held our meeting here in 2002 and the changes to the town and mountain are amazing. Since the Winter Olympics were held here the town has grown exponentially boasting a myriad of new shops and restaurants. There are activities and sources of entertainment for everyone. The Mountains (Whistler and Blackcomb) are now linked by an incredible tram which can be accessed from either base by gondola. Even non-skiers can take advantage of the breathtaking views from the summits of either peak. With almost 13 square miles of skiable terrain, Whistler Blackcomb is the largest downhill ski area in North America. The Fairmount Hotel is a prime property and the spacious conference center is more than adequate for all our functions without leaving the hotel. The facility is ski in and ski out with ski lockers for your convenience, as well as a complementary hot chocolate when you finish skiing.

The Program Committee under the leadership of Ajai Malhotra has created a first-rate scientific program that’s available at www.westerntrauma.org and in the program book. 34 Abstracts were selected for oral presentation including several WTA multicenter trial results. The algorithms committee will suggest pathways for The Open Abdomen, Clearance of the Cervical Spine, ICP management in the ICU and Gunshot Wounds to the Abdomen. This year’s Pro-Con Debates will be titled, “TxA is the Elixir of Life and Should Be Administered to All Potentially Bleeding Patients”, and “REBOA is an Established Tool and Should Be Available in Every Surgeon’s Armamentarium.”

This year’s Founders Lecture will be given by Dr. Gene Moore and is titled “Trauma Research: Trials and Tribulations of a Triceratops”. Dr. Steve Shackford will give the Paint the Ceiling Lecture titled “Against Empathy”. My Presidential address is titled “It’s a Privilege, not a Right”. We will of course have our usual Panel Discussions where our young faculty take on more “experienced” (old) faculty.
The Social Program includes classic WTA events like the Sunday Welcome Reception, the Resident’s Reception, The Mountain Barbeque, The Book Club (Wine Tasting) and of course the Thursday Banquet. No fears the NASTAR race will be held in its usual Wednesday position.

The WTA is science, collegiality, family and friends. It is a place for us to learn, teach, ski, make new friends and new colleagues, and help build young surgical careers. The Mountains have a 120’ base and snow is predicted almost every day until the meeting starts.

Jerrie and I look forward to hosting you and hope you have a great educational, family, and collegial experience in Whistler!

**Dennis Vane, MD**  
*President, Western Trauma Association*
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CONTINUING MEDICAL EDUCATION CREDIT INFORMATION

Accreditation
This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME) through the joint providership of the American College of Surgeons and Western Trauma Association. The American College of Surgeons is accredited by the ACCME to provide continuing medical education for physicians.

**AMA PRA Category 1 Credits™**
The American College of Surgeons designates this live activity for a maximum of 19.25 AMA PRA Category 1 Credits™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Of the AMA PRA Category 1 Credits™ listed above, a maximum of 17.00 credits meet the requirements for Self-Assessment.

Of the AMA PRA Category 1 Credits™ listed above, a maximum of 19.25 credits may qualify as Trauma.*

* The content of this activity may meet certain mandates of regulatory bodies. ACS has not and does not verify the content for such mandates with any regulatory body. Individual physicians are responsible for verifying the content satisfies such requirements.
CME INFORMATION

TO CLAIM CME
You will receive an email with instructions on completing the meeting evaluation, taking self-assessment tests and obtaining your CME Certificate. These instructions will be sent to the email used to register you for the meeting. Instructions will also be posted on the WTA website. The self-assessment tests will be available at the end of each day.

MEETING APP INSTRUCTIONS
Download the WTA Meeting App on your iOS or Android device. The Schedule of Events, Attendee List, Abstracts and Self-Assessment tests can be found on the app.

View the Vimeo video for downloading an app on iOS - first time users - https://vimeo.com/155553890

Downloading the app is easy on iOS and Android! Instructions:
1. Visit http://my.yapp.us/WTAMEETING on your device and follow instructions on the page
2. You’ll be asked to install Yapp from the app store. (if you don’t have it already)
3. Open Yapp and tap “Download an existing Yapp” and your app will appear.

Don’t have an iOS or Android device?
You can view this app from your desktop browser by clicking the my.yapp.us URL above.
LEARNING OBJECTIVES

This activity is designed for physicians of all specialties who are involved in the care of trauma patients.

Upon completion of this course, attendees will be able to:

• Compare the use of needs assessment in trauma center placement vs structure/process based designation.

• Describe the pros/cons of lower level (Level III and IV) designation in the overall care of the trauma patient.

• Apply scientific data in developing local protocols for the reversal of anti-thrombotic therapy for elderly patients with traumatic intra-cranial bleeds

• Assess the importance of pre-existing and/or asymptomatic DVT for appropriate therapy for the individual patient and the financial implications for the institution

• Interpret the existing literature regarding the cellular and sub-cellular mechanisms driving traumatic coagulopathy
  • Implement algorithms for the following conditions:
    • Open abdomen
    • Cervical spine clearance
    • Intra-cranial pressure management
    • Abdominal wall stab wounds
DISCLOSURE INFORMATION

In accordance with the ACCME Accreditation Criteria, the American College of Surgeons, as the accredited provider of this activity, must ensure that anyone in a position to control the content of the educational activity has disclosed all relevant financial relationships with any commercial interest. Therefore, it is mandatory that both the program planning committee and speakers complete disclosure forms. Members of the program committee were required to disclose all financial relationships and speakers were required to disclose any financial relationship as it pertains to the content of the presentations. The ACCME defines a ‘commercial interest’ as “any entity producing, marketing, re-selling, or distributing health care goods or services consumed by, or used on, patients”. It does not consider providers of clinical service directly to patients to be commercial interests. The ACCME considers “relevant” financial relationships as financial transactions (in any amount) that may create a conflict of interest and occur within the 12 months preceding the time that the individual is being asked to assume a role controlling content of the educational activity.

ACS is also required, through our joint providership partners, to manage any reported conflict and eliminate the potential for bias during the activity. All program committee members and speakers were contacted and the conflicts listed below have been managed to our satisfaction. However, if you perceive a bias during a session, please report the circumstances on the session evaluation form.

Please note we have advised the speakers that it is their responsibility to disclose at the start of their presentation if they will be describing the use of a device, product, or drug that is not FDA approved or the off-label use of an approved device, product, or drug or unapproved usage.

The requirement for disclosure is not intended to imply any impropriety of such relationships, but simply to identify such relationships through full disclosure and to allow the audience to form its own judgments regarding the presentation.
## DISCLOSURE INFORMATION

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**WTA MISSION STATEMENT**

The Western Trauma Association is committed to the improvement of trauma care through research, education, sharing of clinical experiences, and the development of physicians of all specialties who are involved in the care of trauma patients. The goals of the Association are not only the intellectual growth attained through increased knowledge, but also the emotional growth attained through camaraderie and interaction with family and friends in an environment conducive to winter sports.
2017-2018 OFFICERS & COMMITTEE CHAIRS

Officers
President                    Dennis W. Vane, MD
President-Elect              Roxie M. Albrecht, MD
Vice President               David V. Shatz, MD
Secretary                    Rosemary Kozar, MD
Treasurer                    Robert McIntyre, MD
Historian                    Mark Metzdorff, MD
Immediate Past President     Carl J. Hauser, MD

Board of Directors
Christine S. Cocanour, MD    Term Ends
Nick Namias, MD               2018
Brent King, MD                2018
Thomas M. Scalea, MD          2019
Carlos Brown, MD              2019
Enrique Ginzburg, MD          2019
Carl J, Hauser, MD            2020
Bonnie Baron, MD              2020
Riyad Karmy Jones, MD         2020

Program Chair
Ajai Malhotra MD             Term Ends
2019

Publications Chair
Rochelle Dicker, MD          Term Ends
2018

Multi-Center Trials Chair    Term Ends
Mitch Cohen, MD              2018

Algorithms Chair
Kenji Inaba, MD              Term Ends
2019

Nominating Chair
Carl J, Hauser, MD           Term Ends
2018
2017-2018 COMMITTEES

Program Committee
Ajai Malhotra, MD, Chair
Charles Fox, MD
Gary Vercruysse, MD
James Haan, MD
Jordan Weinberg, MD
Megan Brenner, MD
Nirav Patel, MD
Oliver Gunter, MD
Richard Miller, MD
Stephanie Savage, MD
Rochelle Dicker, MD, ex-officio
Dennis W. Vane, MD, ex-officio

Publications Committee
Rochelle Dicker, MD, Chair
Megan Brenner, MD
Clay Cotheren-Burlew, MD
Marc DeMoya, MD
Alex Eastman, MD
Oliver Gunter, MD
Anastasia Kunac, MD
Bob Letton, MD
James McCarthy, MD
Laura Moore, MD
Jasmeet Paul, MD
Justin Richards, MD
Mark Shapiro, MD
Jason Sperry, MD
Rob Todd, MD
Jennifer Watters, MD
Ben Zarzaur, MD

Nominating Committee
Carl J. Hauser, MD, Chair
Christine S. Cocanour, MD
Thomas M. Scalea, MD
Aaron Scifres, MD
Eric Toschlog, MD

Multi-Center Trials Committee
Mitch Cohen, MD, Chair

Algorithms Committee
Kenji Inaba, MD, Chair
Hasan Alam, MD
Karen Brasel, MD
Carlos Brown, MD
Dave Ciesla, MD
Marc DeMoya, MD
Rochelle Dicker, MD, ex-officio
Rosemary Kozar, MD, ex-officio
Ajai Malhotra, MD, ex-officio
Matthew Martin, MD
Ernest "Gene" Moore, MD, ex-officio
Jack Sava, MD
Gary Verycrusse, MD
WTA PRESIDENTS

Robert G. Volz, MD 1971 Vail
Robert G. Volz, MD 1972 Vail
Peter V. Teal, MD 1973 Vail
William R. Hamsa, MD 1974 Aspen
Arthur M. McGuire, MD 1975 Sun Valley
Lynn Ketchum, MD 1976 Snowmass
Fred C. Chang, MD 1977 Park City
Glen D. Nelson, MD 1978 Steamboat
Gerald D. Nelson, MD 1979 Snowmass
Kevin G. Ryan, MD 1980 Snowbird
David S. Bradford, MD 1981 Jackson Hole
Erick R. Ratzer, MD 1982 Vail
William R. Olsen, MD 1983 Jackson Hole
Earl G. Young, MD 1984 Steamboat Springs
Robert B. Rutherford, MD 1985 Snowbird
Rudolph A. Klassen, MD 1986 Sun Valley
Robert J. Neviaser, MD 1987 Jackson Hole
Robert C. Edmondson, MD 1988 Steamboat Springs
Ernest E. Moore, MD 1989 Snowbird
Stephen W. Carveth, MD 1990 Crested Butte
George E. Pierce, MD 1991 Jackson Hole
Peter Mucha, Jr., MD 1992 Steamboat
David V. Feliciano, MD 1993 Snowbird
R. Chris Wray, MD 1994 Crested Butte
David A. Kappel, MD 1995 Big Sky
Thomas H. Cogbill, MD 1996 Grand Targhee
G. Jerry Jurkovich, MD 1997 Snowbird
James B. Benjamin, MD 1998 Lake Louise
Herbert J. Thomas III, MD 1999 Crested Butte
Barry C. Esrig, MD 2000 Squaw Valley
Steven R. Shackford, MD 2001 Big Sky
James A. Edney, MD 2002 Whistler-Blackcomb
J. Scott Millikan, MD 2003 Snowbird
Harvey J. Sugerman, MD 2004 Steamboat Springs
Scott R. Petersen, MD 2005 Jackson Hole
# WTA Presidents

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<tr>
<td>Thomas M. Scalea, MD</td>
<td>2016</td>
<td>Squaw Valley</td>
</tr>
<tr>
<td>Carl J. Hauser, MD</td>
<td>2017</td>
<td>Snowbird</td>
</tr>
<tr>
<td>Dennis W. Vane, MD</td>
<td>2018</td>
<td>Whistler</td>
</tr>
</tbody>
</table>
NEW MEMBERS

Western Trauma Association Welcomed the Following New Members at the 2017 Annual Meeting

Andrew Dennis, DO, FACS, FACOS
Chicago, IL
Surgical Critical Care
Active Member

Jennifer Hartwell, MD
Indianapolis, IN
General Surgery
Active Member

Eric Ley, MD
Los Angeles, CA
Surgery
Active Member

Michelle McNutt, MD
Houston, TX
General Surgery
Active Member

Eugene Reilly, MD, FACS
West Reading, PA
Informatics
Active Member

Justin Richards, MD
Baltimore, MD
Anesthesiology
Active Member

David Zonies, MD, MPH, FACS, FCCM
Portland, OR
Surgical Critical Care
Active Member
WESTERN TRAUMA FOUNDATION DONORS

Current lifetime accumulation status based on 2017 year end

**Summit**
($25,000 and up)

Barry Esrig
Ernest Moore

Thomas Scalea
Robert Volz

**Extreme**
($10,000-24,999)

James Davis
David Feliciano

David Livingston
Grace Rozycki

**Couloir Society**
($5,000 – $9,999)

Roxie Albrecht
Christine Cocanour
Mark Metzdorff
Andrew Michaels
J. Scott Millikan

Robert Neviaser
Scott Petersen
R. Lawrence Reed
Steven Shackford
Dennis Vane

**Double Black Diamond Club**
($2,500 - $4,999)

John Adams
Denis Bensard
Marilu Bintz
Kimberly Davis
Soumitra Eachempati
Enrique Ginzburg
James Haan
Gregory Jurkovich
David Kappel

Krista Kaups
David Kissinger
Robert Mackersie
Matthew Martin
Robert McIntyre, Jr.
Steven Ross
R. Stephen Smith
Harvey Sugerman
Herbert Thomas, III
WESTERN TRAUMA FOUNDATION DONORS

Black Diamond Circle
($1,000 - $2,499)

James Benjamin
Walter Biffl
Karen Brasel
Megan Brenner
Carlos Brown
Miriam Bullard
Gregory Campbell
David Ciesla
Thomas Cogbill
Mitch Cohen
Raul Coimbra
Marc de Moya
Rochelle Dicker
Doreen DiPasquale
George Dulabon
Charles Fox
K. Dean Gubler
Carl Hauser
Natasha Keric
Brent King
M. Margaret Knudson
Rosemary Kozar
Guy Lanzi
Robert Letton
Manuel Lorenzo
Barbara Mainville
Ajai Malhotra
James McCarthy
Richard Miller
Frederick Moore
Steve Moulton
Nicholas Namias
M. Gage Ochsner
Patrick Offner
Peter Rhee
Anne Rizzo
Susan Rowell
Martin Schreiber
David Shatz
Harold Sherman
Keith Stephenson
Ali Tabatabai
Michael Truitt
Steven Wald
Jennifer Watters
Michaela West

Blue Trail Associate
($500 - $999)

Hasan Alam
Scott Armen
Bonny Baron
Howard Champion
Roy Cobean
Alain Corcos
Clay Cothren-Burlew
James Cushman
Bruce Ferris
Richard Gamelli
Rajesh Gandhi
Larry Gentilello
John Hall
David Hoyt
Riyad Karmy-Jones
Richard Leone
Alicia Mangram
M. Ashraf Mansour
Frank Nastanski
Raminder Nirula
David Notrica
J. Bradley Pickhardt
Basil Pruitt
Andrew Rosenthal
Henry Sagi
Kevin Schuster
Aaron Scifres
Mark Shapiro
George Testerman
Brian Tibbs
S. Robb Todd
Eric A. Toschlog
R. Christie Wray, Jr.
WESTERN TRAUMA FOUNDATION DONORS

Green Trail Associate
(up to $499)

Christopher Baker
Allison Berndtson
Donald Carter
Charles Cook
Todd Costintini
Martin Croce
Matthew Davis
Jody Digiacomo
Julie Dunn
Brian Eastridge
Matthew Eckert
John Fildes
Alfonso Fonseca
Warren Gall
Ernest Gonzalez
Rajan Gupta

Michael Hauty
James Hebert
Kenji Inaba
Jay Johannigman
Laura Johnson
Olga Kaslow
Barbara Latenser
David Leshikar
Heather MacNew
Charles Mains
Alan Marr
Robert Maxwell
Laura Moore
Charlene Nagy
Michael Norman
Keith O’Malley

Jasmeet Paul
Erik Peltz
George Pierce
Bruce Potenza
Edmund Rutherford
Jack Sava
Carol Schermer
Henry Schiller
Ronald Tesoriero
Ricard Townsend
Daniel Vargo
Gary Vercruysse
Charlie Wade
Amy Wyzykowski
Ben Zarzaur
IN MEMORIAM

Earl G. Young, MD
February 27, 1989

Gerald S. Gussack, MD
August 25, 1997

Peter Mucha, Jr., MD
August 9, 2006

W. Bishop McGill, MD
October 14, 2007

Ronald P. Fischer, MD
January 25, 2013

M. Gage Ochsner, MD
April 26, 2013

George Cierny, MD
June 24, 2013

R. Christie Wray, MD
November 18, 2013

Robert B. Rutherford, MD
November 22, 2013

Doreen DiPasquale, MD
January 7, 2014

Barbara Latenser, MD
June 15, 2015

Matthew L Davis, MD
September 3, 2015
EARL YOUNG AWARD

Earl G. Young, MD
(1928-1989)

RESIDENT PAPER COMPETITION

Dr. Earl G. Young of Minneapolis was a founding member of the Western Trauma Association and its 14th President. He died of a myocardial infarction, Monday, February 27, 1989, while skiing at Snowbird during the 19th Annual Meeting of the Association.

Dr. Young received his medical degree from the University of Rochester, N.Y. and Ph.D. in surgery from the University of Minnesota. He completed advanced training in cancer research at Harvard, a fellowship in cardiovascular surgery at Baylor University in Houston and studied microvascular surgery at the University of California–San Diego.

He was a clinical professor of surgery at the University of Minnesota Medical School, and a practicing general and vascular surgeon at the Park-Nicollet Clinic in Minneapolis from 1960. He was nationally known and was actively involved in research and education throughout his career. In 1988, one year before his untimely death, he received the Owen H. Wangensteen Award for Academic Excellence from the University of Minnesota Health Science Center. It was awarded by an unprecedented unanimous vote of all 72 surgical residents.

The Residents Paper competition was begun in 1991 as a tribute to Dr. Young’s memory and his “spirit of inquiry, love of learning … and commitment in service to mankind.”* The award is given to the best resident paper presented at the Annual Meeting.

*Dr. John Najarian characterizing Earl at a memorial service in his honor at the University of Minnesota.
## EARL G. YOUNG AWARD RECIPIENTS

<table>
<thead>
<tr>
<th>Resident</th>
<th>Institution</th>
<th>Year</th>
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</thead>
<tbody>
<tr>
<td>Joseph Schmoker, MD</td>
<td>University of Vermont</td>
<td>1991</td>
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<tr>
<td>Joseph Schmoker, MD</td>
<td>University of Vermont</td>
<td>1992</td>
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<tr>
<td>Charles Mock, MD</td>
<td>University of Washington</td>
<td>1993</td>
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<tr>
<td>Gino Travisani, MD</td>
<td>University of Vermont</td>
<td>1994</td>
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<tr>
<td>Phillip C. Ridings, MD</td>
<td>Medical College of Virginia</td>
<td>1995</td>
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<tr>
<td>David Han, MD</td>
<td>Emory University</td>
<td>1996</td>
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<tr>
<td>Preston R. Miller, MD</td>
<td>Wake Forest University</td>
<td>1997</td>
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<tr>
<td>Geoffrey Manley, MD, PhD</td>
<td>University of California, San Francisco</td>
<td>1998</td>
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<tr>
<td>James M. Doty, MD</td>
<td>Medical College of Virginia</td>
<td>1999</td>
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<tr>
<td>David J. Ciesla, MD</td>
<td>Denver Health/University of Colorado</td>
<td>2000</td>
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<tr>
<td>Ricardo J. Gonzales, MD</td>
<td>Denver Health/University of Colorado</td>
<td>2001</td>
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<tr>
<td>Scott C. Brakenridge, MD</td>
<td>Cook County Hospital</td>
<td>2002</td>
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<tr>
<td>Adena J, Osband, MD</td>
<td>UMDNJ-New Jersey Medical School</td>
<td>2003</td>
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<tr>
<td>Cindy Lee, MD</td>
<td>UMDNJ-New Jersey Medical School</td>
<td>2004</td>
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<tr>
<td>Ernest A. Gonzalez, MD</td>
<td>University of Texas at Houston</td>
<td>2005</td>
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<tr>
<td>Jennifer M. Watters, MD</td>
<td>Oregon Health &amp; Science University</td>
<td>2005</td>
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<tr>
<td>Jennifer J. Wan, MD</td>
<td>University of California, San Francisco</td>
<td>2006</td>
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<tr>
<td>Jennifer J. Wan, MD</td>
<td>University of California, San Francisco</td>
<td>2007</td>
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<tr>
<td>Keir J. Warner, MD</td>
<td>University of Washington</td>
<td>2008</td>
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<tr>
<td>T. W. Constantini, MD</td>
<td>University of California, San Diego</td>
<td>2009</td>
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<tr>
<td>C. Anne Morrison, MD</td>
<td>Baylor College of Medicine</td>
<td>2010</td>
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<tr>
<td>Marlin Causey, MD</td>
<td>Madigan Army Medical Center</td>
<td>2011</td>
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<tr>
<td>Phillip Letourneau, MD</td>
<td>University of Texas at Houston</td>
<td>2011</td>
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<tr>
<td>Gerard De Castro, MD</td>
<td>University of Maryland</td>
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<tr>
<td>Matthew E. Kutcher, MD</td>
<td>University of California, San Francisco</td>
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<tr>
<td>Kimberly Song, MD, MA</td>
<td>UMDNJ – New Jersey Medical School</td>
<td>2013</td>
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<tr>
<td>Lucy Kornblith, MD</td>
<td>UCSF/SFGH, San Francisco</td>
<td>2014</td>
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<tr>
<td>Hunter B. Moore, MD</td>
<td>Denver Health/University of Colorado</td>
<td>2015</td>
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<tr>
<td>George Black, MD</td>
<td>Madigan Army Medical Center</td>
<td>2016</td>
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<tr>
<td>Morgan Barron, MD</td>
<td>Madigan Army Medical Center</td>
<td>2017</td>
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</table>
PRESIDENTIAL ADDRESS

A PRIVILEGE NOT A RIGHT

Tuesday, February 27
5:00 pm - 6:00 pm

Dennis W. Vane, MD
Wadmalaw Island, South Carolina

WTA President, Dennis William Vane, attended medical school at the Universite Libre de Bruxelles in Brussels Belgium and did his general surgical residency at Indiana University. At Indiana he met Jay L. Grosfeld, perhaps the most internationally renowned Pediatric Surgeons in the world. He fell in love with the specialty and was fortunate enough to be accepted to the Pediatric Surgery Fellowship at Ohio State University, where he studied under the tutelage of H. William Clatworthy, and Thomas Boles, two of the pioneers in the field. Dr. Vane returned to Indiana University in 1985, after his fellowship to work with Dr. Grosfeld at Riley Children’s Hospital. While there he received several grants to fund the Kiwanis Pediatric Trauma Center at Riley Hospital,
the first such funded center outside of the original Kiwanis Pediatric Trauma Center started by Dr. Burton Harris at the Boston Floating Hospital. Dr. Vane was recruited to the University of Vermont in 1990 by Dr. Steven Shackford, to re-establish the Pediatric Surgery Service. He was appointed Professor of Surgery and Pediatrics in 1996 and the first Surgeon-in-Chief of the Vermont Children’s Hospital in 2002. He obtained his MBA in 1999 and remained at the University of Vermont until 2008. Dr. Vane was then recruited to Saint Louis University to become the J. Eugene Lewis, Jr, MD, Professor of Pediatric Surgery and the first Surgeon-in-Chief of the Cardinal Glennon Children’s Medical Center. While there he rebuilt the Department and re-established the Pediatric Surgery Fellowship Program. Dr. Vane remained in Saint Louis until July of 2016 when he and his wife “retired” to South Carolina. Dr. Vane holds an appointment as Clinical Professor of Surgery at the Medical University of South Carolina, where he is active in the teaching of Medical Students and Residents.

Dr. Vane attended his first Western Trauma Association meeting and presented his first paper to the organization in 1992. He became a member in 1994 and has attended all but one of the meetings since that time. The WTA is Dr. Vane’s “favorite scientific meeting”. It is to him “not only an excellent scientific educational opportunity, but also an environment where informal contact with colleagues and the family atmosphere provides a professional opportunity not present in any other organization”.

“The honor of being elected WTA President is the greatest privilege I have ever had.”
“PAINT THE CEILING” LECTURE

In 1997, Dr. Gregory “Jerry” Jurkovich delivered his Presidential Address entitled “Paint the Ceiling: Reflections on Illness”. This was a personal account of his battle with non-Hodgkin’s lymphoma. His deep insights were shared from a patient’s perspective, even that of a stained ceiling that he observed while lying on his back. He proposed that future WTA Scientific Programs have some time “dedicated to our patients and to the Art of Medicine”.

<table>
<thead>
<tr>
<th>Presenter</th>
<th>Year</th>
<th>Location</th>
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<tbody>
<tr>
<td>G. Jerry Jurkovich, MD</td>
<td>1997</td>
<td>Snowbird</td>
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<tr>
<td>John W. McGill, MD</td>
<td>1998</td>
<td>Lake Louise</td>
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<tr>
<td>William T. Close, MD</td>
<td>1999</td>
<td>Crested Butte</td>
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<tr>
<td>Jimmy Cornell</td>
<td>2000</td>
<td>Squaw Valley</td>
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<tr>
<td>Geoff Tabin, MD</td>
<td>2001</td>
<td>Big Sky</td>
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<tr>
<td>James H. “Red” Duke, MD</td>
<td>2002</td>
<td>Whistler</td>
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<td>David V. Shatz, MD</td>
<td>2003</td>
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<td>Susan and Tim Baker</td>
<td>2004</td>
<td>Steamboat Springs</td>
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<tr>
<td>Alex Habel, MD</td>
<td>2005</td>
<td>Jackson Hole</td>
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<td>Andrew Schneider</td>
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<td>Ernest E. Moore, MD</td>
<td>2007</td>
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<td>Pamela Kallsen</td>
<td>2008</td>
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<td>Sylvia Campbell, MD</td>
<td>2009</td>
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<td>William Schecter, MD</td>
<td>2010</td>
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<td>Jeff McKenney, MD</td>
<td>2011</td>
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<td>Larry M. Gentilello, MD</td>
<td>2012</td>
<td>Vail</td>
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<td>Neil L. Barg, MD</td>
<td>2013</td>
<td>Snowmass</td>
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<td>Ziad Sifri, MD</td>
<td>2014</td>
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<td>Julie Freischlag, MD</td>
<td>2015</td>
<td>Telluride</td>
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<td>Lewis Rubinson, MD, PhD</td>
<td>2016</td>
<td>Squaw Valley</td>
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<td>Kenneth Waxman, MD</td>
<td>2017</td>
<td>Snowbird</td>
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<tr>
<td>Steven R. Shackford, MD</td>
<td>2018</td>
<td>Whistler</td>
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</table>
PAINT THE CEILING LECTURE

AGAINST EMPATHY

Thursday, March 1
5:00 pm – 6:00 pm

Steven R. Shackford
Coronado, CA

Dr. Steven Shackford is past Chairman of the Department of Surgery in the College of Medicine at the University of Vermont, a post that he held for 18 years. He has authored or co-authored 352 scientific papers, 29 book chapters and 3 monographs, mostly relating to secondary brain injury and the effect of hemorrhagic shock on the injured brain. He was funded by the NIH from 1988 through 1995. He is on the editorial board of several surgical journals and is Associate Editor of the Journal of Trauma. In addition to his interest in trauma he is an active vascular surgeon. Working with a number of other surgeons, Dr. Shackford helped to develop the ultrasound educational program for the American College of Surgeons. He was the first Chair of the National Ultrasound Faculty of the American College of Surgeons. He is a past President of the American Association for the Surgery of Trauma and the Western Trauma Association. He has been married to Ellen Dee Shackford for 41 years and has four children and 3 (soon to be 4) grandchildren.
FOUNDERS’ BASIC SCIENCE LECTURE

Throughout the years, the Western Trauma Association has matured as an academic society while maintaining the cherished elements of friendship, collegiality and family. In honor of this unique spirit, a founding member has generously provided the idea and most of the financial support for an annual Founders’ Basic Science Lectureship. The purpose of this Lecture is to further enhance the educational value of our Scientific Meeting relative to the area of basic science research. This Lecture reflects the vision and dedication of our founding members and will hold a prominent place in all future programs.

<table>
<thead>
<tr>
<th>Presenter</th>
<th>Year</th>
<th>Location</th>
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<tbody>
<tr>
<td>Raul Coimbra, MD</td>
<td>2009</td>
<td>Crested Butte</td>
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<tr>
<td>Lawrence Diebel, MD</td>
<td>2010</td>
<td>Telluride</td>
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<tr>
<td>Carl J. Hauser, MD</td>
<td>2011</td>
<td>Big Sky</td>
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<tr>
<td>Fred Moore, MD</td>
<td>2012</td>
<td>Vail</td>
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<td>Steve Shackford, MD</td>
<td>2013</td>
<td>Snowmass</td>
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<tr>
<td>Hasan B. Alam, MD</td>
<td>2014</td>
<td>Steamboat Springs</td>
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<tr>
<td>Charles S. Cox, Jr. MD</td>
<td>2015</td>
<td>Telluride</td>
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<tr>
<td>Rosemary Kozar, MD</td>
<td>2016</td>
<td>Squaw Valley</td>
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<tr>
<td>Mitchell J. Cohen, MD</td>
<td>2017</td>
<td>Snowbird</td>
</tr>
<tr>
<td>Ernest E. “Gene” Moore, MD</td>
<td>2018</td>
<td>Whistler</td>
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FOUNDERS' BASIC SCIENCE LECTURE

TRAUMA RESEARCH: TRAILS AND TRIBULATIONS OF A TRICERATOPS

Wednesday, February 28
7:40 am – 8:20 am

Ernest E. “Gene” Moore, MD
University of Colorado
Denver, CO

Ernest E. “Gene” Moore, M.D. has been the Editor of the Journal of Trauma and Acute Care Surgery since 2012 and was the Chief of Trauma at the Denver General Hospital for 36 years, Chief of Surgery for 28 years, and the first Bruce M. Rockwell Distinguished Chair in Trauma Surgery. He continues to serve as Vice Chairman for Research and is a Distinguished Professor of Surgery at the University of Colorado Denver.

Under Dr. Moore’s leadership, the Rocky Mountain Regional Trauma Center at Denver General became internationally recognized for innovative care of the injured patient, and its trauma research laboratory has been funded by the NIH for 30 consecutive years.

Dr. Moore has served as president of nine academic societies, including the Society of University Surgeons, American Association for the Surgery of Trauma, International Association for the Trauma and Surgical Intensive Care, and the World Society of Emergency Surgery.

His awards include the Robert Danis Prize from the Society of International Surgeons, Orazio Campione Prize from the World Society of Emergency Surgery, Philip Hench Award from the University of Pittsburgh, Florence Sabin Award from the University of Colorado, the Lifetime Achievement Award from the Society of University Surgeons, the Lifetime Achievement Award for Resuscitation Science from the American Heart Association, the Distinguished Investigator Award the American College of Critical Medicine, the Distinguished Service
FOUNDERS’ BASIC SCIENCE LECTURE

Award from the Shock Society, and the Lifetime Service Award from the International Association for Trauma and Surgical Intensive Care. He has honorary fellowships in the Royal College of Surgeons of Edinburgh, the Royal College of Surgeons in Ireland, the Royal College of Surgeons of Thailand, and the American College of Emergency Physicians; and is an honorary member of the Brazilian Trauma Society, Colombian Trauma Society, Eastern Association for the Surgery of Trauma, European Society for Trauma and Emergency Surgery, and Trauma Association of Canada Dr. Moore is coeditor of the textbook Trauma, now in its 8th edition, Surgical Secrets in its 7th edition, and the textbook Trauma Induced Coagulopathy; he has >1600 publications and has lectured extensively throughout the world.

He is married to Sarah Van Duzer Moore, M.D., an internist at the University of Colorado Denver, and they have two sons; Hunter, a surgical resident at UCD and Peter, an internist at the University of California San Francisco. Dr. Moore’s additional interests include endurance sports, mountaineering, skiing, and wapiti pursuit. He lives by the principle to work hard you must play hard, with the understanding that family is the ultimate priority.
## SUNDAY, FEBRUARY 25, 2018

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>5:00pm - 7:30pm</td>
<td><strong>REGISTRATION OPEN</strong></td>
<td>MacDonald Foyer</td>
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<tr>
<td>5:00pm - 7:00pm</td>
<td><strong>WELCOME RECEPTION</strong></td>
<td>MacDonald DEF</td>
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<tr>
<td>5:00pm - 7:00pm</td>
<td><strong>KIDS WELCOME RECEPTION</strong></td>
<td>Empress AB</td>
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<tr>
<td>6:00pm - 7:00pm</td>
<td><strong>WTA FOUNDATION MEETING</strong></td>
<td>Saskatchewan</td>
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<tr>
<td>7:00pm - 8:00pm</td>
<td><strong>WTA PAST PRESIDENTS MEETING</strong></td>
<td>Saskatchewan</td>
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</table>
Monday, February 26, 2018

6:00am - 9:00am  **Registration Open**
MacDonald Foyer

6:30am - 8:00am  **Attendee Breakfast**
MacDonald Foyer

7:00am - 9:00am  **Scientific Session 1 - Earl Young Competition**
MacDonald ABC
Moderator: Ajai Malhotra

7:00am - 7:20am  1. The procoagulant molecule PAI-1 is associated with injury severity and shock
Mary Condron, MD
Oregon Health & Science University

7:20am - 7:40am  2. Is delaying venous thromboembolism chemoprophylaxis worth the wait?: Dynamic coagulability after injury
Joshua J. Sumislawski, MD
Denver Health Medical Center

7:40am - 8:00am  3. Validating predictive equations to assess energy expenditure in acute spinal cord injury
Christine L Ramirez, MD
St. Luke’s University Health Network

8:00am - 8:20am  4. Acute hyperglycemia exacerbates trauma induced endothelial and glycocalyx injury: An in vitro model
Mark E. Diebel MD
Wayne State University

8:20am - 8:40am  5. Artificial intelligence can predict daily trauma volume and average acuity
David P Stonko
Vanderbilt

8:40am - 9:00am  6. Blunt traumatic scapular fractures are associated with great vessel injuries in children
Ibrahim Abdelshafy
Maimonides
7:30am - 9:00am  **FRIENDS & FAMILY BREAKFAST**  
Mallard

3:30pm - 6:00pm  **REGISTRATION OPEN**  
MacDonald Foyer

4:00pm - 6:00pm  **Scientific Session 2 – EARLY YOUNG COMPETITION**  
MacDonald ABC  
Moderator: Charles Fox, MD

4:00pm - 4:20pm  
7. **TRAUMATIC RECTAL INJURIES: IS THE COMBINATION OF COMPUTED TOMOGRAPHY AND RIGID PROCTOSCOPY SUFFICIENT?**  
Jacob Paul Veith, MD  
University of Texas at Austin Dell Medical School  
Surgery Residency

4:20pm - 4:40pm  
8. **GOAL DIRECTED PLATELET TRANSFUSIONS CORRECT PLATELET DYSFUNCTION AND IMPROVE MORTALITY IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY**  
Elisa Furay MD  
UT Austin Dell Seton Medical Center

4:40pm - 5:00pm  
9. **PERHAPS IT’S NOT THE PLATELET: RISTOCETIN UNCOVERS THE POTENTIAL ROLE OF VON WILLEBRAND FACTOR IN PLATELET DYSFUNCTION FOLLOWING TRAUMATIC BRAIN INJURY**  
Lucy Zumwinkle Kornblith MD  
University of California San Francisco, Zuckerberg San Francisco General Hospital

5:00pm - 5:20pm  
10. **THE NOVEL ORAL ANTICOAGULANTS (NOACS) HAVE WORSE OUTCOMES COMPARED TO WARFARIN IN PATIENTS WITH TRAUMATIC BRAIN INJURY**  
Muhammad Zeeshan  
University of Arizona

5:20pm - 5:40pm  
11. **QUETIAPINE PROTECTS THE BLOOD-BRAIN BARRIER IN TRAUMATIC BRAIN INJURY**  
Bobby Darnell Robinson, MD  
Scott and White Medical Center - Temple

5:40pm - 6:00pm  
12. **EXTENDING THE GOLDEN HOUR FOR ZONE 1 REBOA: IMPROVED SURVIVAL AND REPERFUSION INJURY WITH INTERMITTENT VERSUS CONTINUOUS REBOA IN A PORCINE SEVERE TRUNCAL HEMORRHAGE MODEL**  
John Kuckelman DO  
Madigan Army Medical Center
### AGENDA

**MONDAY, FEBRUARY 26, 2018**

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<tr>
<th>Time</th>
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<tr>
<td>6:00pm - 7:00pm</td>
<td><strong>RESIDENT RECEPTION</strong></td>
<td>Lombard</td>
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<td>6:00pm - 8:00pm</td>
<td><strong>WTA BOARD MEETING (Invitation only)</strong></td>
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**TUESDAY, FEBRUARY 27, 2018**

6:00am - 9:00am  **REGISTRATION OPEN**  
MacDonald Foyer

6:30am - 8:00am  **ATTENDEE BREAKFAST**  
MacDonald Foyer

7:00am – 9:00am  **Scientific Session 3**  
MacDonald ABC  
Moderator: Gary Vercruysse, MD

7:00am - 7:20am  
**13. NO WIRE? NO PROBLEM: RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA (REBOA) CAN BE PERFORMED EFFECTIVELY AND MORE RAPIDLY WITH A WIRE-FREE DEVICE**  
Anna N. Romagnoli, MD  
RA Cowley Shock Trauma Center

7:20am - 7:40am  
**14. AORTIC BRANCH VESSEL FLOW DURING RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA (REBOA)**  
Melanie Hoehn, MD  
RA Cowley Shock Trauma University of Maryland

7:40am - 8:00am  
**15. DOES SELECTIVE USE OF TRANEXAMIC ACID MATTER: FIBRINOLYSIS PHENOTYPES AND ASSOCIATED OUTCOMES**  
Hunter Burroughs Moore  
University of Colorado

8:00am - 8:20am  
**16. SEVERELY INJURED TRAUMA PATIENTS WITH ADMISSION HYPERFIBRINOLYSIS; IS THERE A ROLE OF TRANEXAMIC ACID? FINDINGS FROM THE PRAGMATIC, RANDOMIZED OPTIMAL PLATELET AND PLASMA RATIOS (PROPPR) TRIAL**  
Muhammad N. Khan  
University of Arizona

8:20am - 8:45am  
**PRO/CON DEBATE**  
**TRANEXAMIC ACID (TXA) IS THE ELIXIR OF LIFE AND SHOULD BE ADMINISTERED TO ALL POTENTIALLY BLEEDING PATIENT**  
Pro: Michel Aboutanos; Con: James Haan

8:45am - 9:00am  
**ALGORITHM #1 - OPEN ABDOMEN**  
Jack Sava, MD
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<tr>
<td>7:30am - 9:00am</td>
<td><strong>FRIENDS &amp; FAMILY BREAKFAST</strong></td>
<td>Mallard</td>
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<td>10:00am - 12:00pm</td>
<td><strong>SKI WITH AN OFFICER</strong></td>
<td>Mountain</td>
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<td>3:30pm - 6:00pm</td>
<td><strong>REGISTRATION OPEN</strong></td>
<td>MacDonald Foyer</td>
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<td>4:00pm - 6:00pm</td>
<td><strong>Scientific Session 4</strong></td>
<td>MacDonald ABC</td>
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<td>Moderator: Oliver Gunter, MD</td>
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<td>4:00pm - 4:20pm</td>
<td><strong>7. SALVAGE OF RIB STABILIZATION HARDWARE WITH ANTIBIOTIC BEADS</strong></td>
<td>Page 81</td>
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<tr>
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<td>Michelle S. Junker, MD Mayo Clinic</td>
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<td>4:20pm - 4:50pm</td>
<td><strong>PANEL OF EXPERTS</strong></td>
<td>Page 83</td>
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<td></td>
<td>Case presenter: Ajai Malhotra, MD; Panelists: Mitch Cohen MD, Stephanie Savage MD, Nirav Patel MD, Oliver Gunter MD</td>
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<td>4:50pm - 5:00pm</td>
<td><strong>FAMILY ABSTRACT</strong></td>
<td>Page 85</td>
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<td>18. OBSTETRICAL HEMORRHAGE - UTILIZATION OF TRAUMA RESOURCES TO SAVE ONE OF OUR OWN</td>
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<td>Elizabeth M. Windell, DO Legacy Emanuel Medical Center</td>
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<td>5:00pm - 6:00pm</td>
<td><strong>PRESIDENTIAL ADDRESS - A PRIVILEGE NOT A RIGHT</strong></td>
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<td></td>
<td>Dennis Vane, MD</td>
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**AGENDA**

**WEDNESDAY, FEBRUARY 28, 2018**

**6:00am - 9:00am**  
**REGISTRATION OPEN**  
MacDonald Foyer

**6:30am - 8:00am**  
**ATTENDEE BREAKFAST**  
MacDonald Foyer

**7:00 am - 9:00 am**  
**Scientific Session 5**  
MacDonald ABC  
Moderator: Ajai Malhotra, MD

**7:00am - 7:20am**  
**19. HIDDEN BURDEN OF VENOUS THROMBOEMBOLISM AFTER TRAUMA: A NATIONAL ANALYSIS INCLUDING READMISSION TO A DIFFERENT HOSPITAL**  
Joshua Parreco MD  
University of Miami

**7:20am - 7:40am**  
**20. NOT ALL DVT IS CREATED EQUAL: INCIDENCE OF PRE-EXISTING DVT AMONG HIGH-RISK TRAUMATIZED PATIENTS**  
Amy Tefft, MD  
University of Vermont Medical Center

**7:40am - 8:20am**  
**BASIC SCIENCE LECTURE**  
**TRAUMA RESEARCH: TRIALS AND TRIBULATIONS OF A TRICERATOPS**  
Ernest E. “Gene” Moore, MD

**8:20am - 9:00am**  
**SPECIAL SESSION**  
Deborah Kuhls, MD, Paul Chestovich MD,  
Chris Fisher, MD, Sean Dort, MD
AGENDA

7:30am - 9:00am  FRIENDS & FAMILY BREAKFAST
Mallard

10:00am - 12:00pm  WTA SKI RACE
Mountain

11:30pm – 1:30pm  WTA MOUNTAIN PICNIC
GLCC – Whistler Village

3:30pm - 6:00pm  REGISTRATION OPEN
MacDonald Foyer

4:00pm – 6:00pm  WTA BOOK CLUB
Deerhurst

4:00pm – 6:00pm  Scientific Session 6
MacDonald ABC
Moderator: Ajai Malhotra, MD

4:00pm - 4:20pm  21. MACHINE LEARNING WITHOUT BORDERS? AN
ADAPTABLE TOOL TO OPTIMIZE MORTALITY
PREDICTION IN DIVERSE CLINICAL SETTINGS
S. Ariane Christie MD
University of California San Francisco

4:20pm - 4:30pm  22. THE HISTORY AND TRADITION OF ALPINISM IN
THE WESTERN TRAUMA ASSOCIATION AND CLIMBING
THE MATTERHORN WITH YOUR MENTOR
Gregory A. Magee, MD MSc
University of Southern California

4:30pm - 5:00pm  MILITARY PANEL
Case presenters: Mathew Martin MD and
Jennifer Gurney MD
Panelists: Kim Peck MD; David Feliciano MD;
Warren Dorlac MD; Racheal Callcut MD

5:00pm - 6:00pm  WTA BUSINESS MEETING (MEMBERS ONLY)

6:00pm - 8:00pm  WTA FAMILY NIGHT
Tubing at Coca-Cola Tube Park
THURSDAY, MARCH 1, 2018

6:00am - 9:00am  **REGISTRATION OPEN**
MacDonald Foyer

6:30am - 8:00am  **ATTENDEE BREAKFAST**
MacDonald Foyer

7:00 am – 9:00 am  **Scientific Session 7**
MacDonald ABC
Moderator: Stephanie Savage, MD

7:00am - 7:20am  23. OPIOID DEPENDENCY IS INDEPENDENTLY ASSOCIATED WITH INFERIOR CLINICAL OUTCOMES AFTER TRAUMA
Walter Robert Hsiang
Yale School of Medicine

7:20am - 7:40am  24. CRISIS UNDER THE RADAR: METHAMPHETAMINE USE IS REACHING EPIDEMIC PROPORTIONS AND CONTRIBUTING TO RESOURCE OVER-UTILIZATION AT A LEVEL 1 TRAUMA CENTER
Vincent A. Gemma, MD
St. Joseph’s Hospital and Medical Center

7:40am - 8:00am  25. PREVALENCE AND TREATMENT OF DEPRESSION AND POSTTRAUMATIC STRESS DISORDER AMONG TRAUMA PATIENTS WITH NON-NEUROLOGICAL INJURIES
Ben L. Zarzaur, MD, MPH
Indiana University School of Medicine

8:00am - 8:20am  26. TIME TO STROKE: A WESTERN TRAUMA ASSOCIATION MULTICENTER STUDY OF BLUNT CEREBROVASCULAR INJURIES
Clay Cothren Burlew, MD
Denver Health Medical Center

8:20am - 8:45am  **PRO/CON DEBATE**
REBOA IS AN ESTABLISHED TOOL AND SHOULD BE AVAILABLE IN EVERY SURGEONS’ ARMAMENTARIUM
Pro: Megan Brenner MD; Con: Gary Vercruysse MD

8:45am - 9:00am  **ALGORITHM #2**
CERVICAL SPINE CLEARANCE
David Ciesla, MD
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<tr>
<td>7:30am - 9:00am</td>
<td><strong>FRIENDS &amp; FAMILY BREAKFAST</strong></td>
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<td>MacDonald Foyer</td>
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<td>4:00pm - 6:00pm</td>
<td><strong>Scientific Session 8</strong></td>
<td>MacDonald ABC</td>
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<td><strong>Moderator: Jordan Weinberg, MD</strong></td>
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<td>4:00pm - 4:20pm</td>
<td>27. RISK OF INCREASED MORTALITY ASSOCIATED WITH STOPPING AT LEVEL III AND IV TRAUMA CENTERS: A PROPENSITY SCORE MATCHED STUDY</td>
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<td>Patrick C Bonasso MD, MBA</td>
<td>University of Arkansas for Medical Sciences</td>
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<td>4:20pm - 4:40pm</td>
<td>28. NEEDS-BASED ASSESSMENT OF TRAUMA SYSTEMS: A SURVEY OF THE MEMBERSHIP OF THE WESTERN TRAUMA ASSOCIATION</td>
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<td>R Stephen Smith MD FACS</td>
<td>University of Florida</td>
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<td>5:00pm - 5:05pm</td>
<td><strong>CASE REPORT</strong></td>
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<td>29. INNOVATIVE SURGICAL TREATMENTS AND ADVANCED PROSTHETICS FOR UPPER EXTREMITY AMPUTEE: A CASE REPORT</td>
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<td>Dr. Susan Rowell</td>
<td>Oregon Health &amp; Science University</td>
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<td>5:05pm - 5:20pm</td>
<td><strong>ALGORITHM #3</strong></td>
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<td>CP MANAGEMENT</td>
<td>Hasan Alam, MD</td>
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<td>5:20pm - 6:00pm</td>
<td><strong>PAINT THE CEILING LECTURE AGAINST EMPATHY</strong></td>
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<td>Steven R, Shackford MD FACS</td>
<td>University of Vermont College of Medicine</td>
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6:30pm – 7:00pm  **COCKTAIL RECEPTION**  
Frontenac Foyer

6:30pm – 10:00pm  **KIDS PARTY**  
Empress AB

7:00pm – 9:30pm  **AWARDS BANQUET**  
Frontenac
**FRIDAY, MARCH 2, 2018**

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<td>MacDonald Foyer</td>
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<td>6:30am - 8:00am</td>
<td>ATTENDEE BREAKFAST</td>
<td>MacDonald Foyer</td>
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<td>7:00 am - 9:00 am</td>
<td><strong>Scientific Session 9</strong></td>
<td>MacDonald ABC</td>
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<td>Moderator: James Haan, MD</td>
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<td>7:00am - 7:20am</td>
<td><strong>30. THE WHY &amp; HOW OUR TRAUMA PATIENTS DIE</strong></td>
<td>Page 125</td>
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<td>A PROSPECTIVE WESTERN TRAUMA ASSOCIATION MULTICENTER TRIALS COMMITTEE STUDY</td>
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<td>Rachael A Callcut, MD, MSPH</td>
<td>UCSF/San Francisco General Hospital</td>
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<td>7:20am - 7:40am</td>
<td><strong>31. THE IMPACT OF IN-HOSPITAL PALLIATIVE CARE CONSULTATION ON HEALTHCARE AT THE END OF LIFE FOR OLDER TRAUMA PATIENTS WHO DIE AFTER DISCHARGE</strong></td>
<td>Page 127</td>
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<td>Elizabeth J Lilley, MD, MPH</td>
<td>Rutgers Robert Wood Johnson Medical School</td>
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<td>7:40am - 8:00am</td>
<td><strong>32. A DIFFERENT FORM OF INJURY PREVENTION: SUCCESSFUL SCREENING AND REFERRAL FOR HIV AND HEPATITIS C IN A TRAUMA POPULATION</strong></td>
<td>Page 129</td>
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<td>Benjamin White, MD</td>
<td>Medical University of South Carolina</td>
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<td>8:00am - 8:20am</td>
<td><strong>33. INABILITY TO PREDICT SUB-PROPHYLACTIC ANTI-FACTOR XA LEVELS IN TRAUMA PATIENTS RECEIVING EARLY LOW-MOLECULAR-WEIGHT HEPARIN</strong></td>
<td>Page 131</td>
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<td>Michael W Cripps, MD</td>
<td>University of Texas Southwestern Medical Center</td>
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<td>8:20am - 8:40am</td>
<td><strong>34. RIVAROXABAN VERSUS ENOXAPARIN FOR VENOUS THROMBOPROPHYLAXIS IN AN ADULT TRAUMA POPULATION</strong></td>
<td>Page 133</td>
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<td>Stephanie A. Savage, MD, MS</td>
<td>Indiana University School of Medicine</td>
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<tr>
<td>Time</td>
<td>Session Details</td>
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| 8:40am - 8:45am | **CASE REPORT**  
35. “X MARKS THE SPOT:” A NOVEL APPROACH TO OPERATIVE FIXATION OF RIB FRACTURES UTILIZING COMPUTED TOMOGRAPHY (CT) BIOPSY GRIDS FOR PREOPERATIVE LOCALIZATION OF TRAUMATIC RIB FRACTURES  
Cressilee Butler, MD  
University of Oklahoma |
| 8:45am - 9:00am | **ALGORITHM #4**  
ABDOMINAL GSW  
Matthew Martin, MD |
| 7:30am - 9:00am | **FRIENDS & FAMILY BREAKFAST**  
MacDonald Foyer |
| 3:30pm - 6:00pm | **REGISTRATION OPEN**  
MacDonald Foyer |
| 4:00pm - 6:00pm | **Scientific Session 10**  
MacDonald ABC  
Moderator: Megan Brenner |
| 4:00pm - 4:20pm | **36. THE IMPACT OF ACS LEVEL I PEDIATRIC TRAUMA CENTERS ON ADOLESCENT MOTOR VEHICLE FATALITIES, 1999-2015**  
David M. Notrica, MD  
Phoenix Children's Hospital |
| 4:20pm - 4:40pm | **37. PEDIATRIC OUTREACH IN TRAUMA: APPROPRIATE TIMING AND TECHNIQUES FOR RETENTION**  
Tiffany Willard MD  
University of Colorado Health - Memorial Hospital |
| 4:40pm - 5:00pm | **38. UTILITY OF DIAPHRAGM PACING IN THE MANAGEMENT OF ACUTE CERVICAL SPINAL CORD INJURY**  
Andrew J. Kerwin, MD  
University of Florida College of Medicine-Jacksonville |
| 5:00pm - 5:20pm | **39. A SUBSET OF FIVE HUMAN MITOCHONDRIAL FORMYL PEPTIDES MIMIC BACTERIAL PEPTIDES AND FUNCTIONALLY DEACTIVATE HUMAN NEUTROPHILS (PMN)**  
Kiyoshi Itagaki, PhD  
Beth Israel Deaconess Medical Center |
5:20pm - 5:40pm

40. EARLY ENTERAL NUTRITION IMPROVES MORBIDITY IN CRITICALLY INJURED ADULT MALES DESPITE LOWER SARCOPENIA INDEX SCORES
Jennifer L. Hartwell
Indiana University

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5:40pm - 6:00pm

41. INJURY PATTERNS AND SUBSEQUENT INTERVENTION OF HANGING MECHANISM PATIENTS: HOW MUCH WORK-UP IS NEEDED?
David Berke, MD
The University of Kansas School of Medicine - Wichita

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NOTES

Figure 1. PAI-1 level box-plot distributions by ISS group. Boxes represent quartiles; Circles represent comparisons of means.
THE PROCOAGULANT MOLECULE PAI-1 IS ASSOCIATED WITH INJURY SEVERITY AND SHOCK
M Condron, S Rowell, E Dewey, D Farrell, H Hinson
Oregon Health & Science University

Presenter: Mary Condron, MD
Sponsor: Susan Rowell, MD

Introduction: Traumatic injury is associated with dysregulation of the plasmin system and abnormal fibrinolysis. PAI-1 is a procoagulant molecule produced by endothelium and platelets that inhibits tPA/uPA, thrombomodulin, and activated protein C. We hypothesized that elevated PAI-1 levels would be associated with increased Injury Severity Score (ISS) and shock.

Methods: Injured patients requiring ICU admission (n=268) were prospectively enrolled at a level I trauma center. We evaluated demographic, physiologic, and injury scoring data. PAI-1 levels were measured at admission using a Luminex analyte platform. Univariate tests for correlation informed the construction of a multivariate model examining the relationship between PAI-1 and ISS.

Results: PAI-1 was positively associated with ISS at all levels (p <.0001) and was highest in patients with ISS>35 (p<.0001, Figure 1). Patients with isolated body injury and those with both TBI and body injury had higher PAI-1 levels than those with isolated TBI (p <.0001). On univariate analysis, older patients (p=0.0011), hypotensive patients (p=0.0076), and those with elevated blood alcohol (p=0.0024) had higher PAI-1 levels. A significant interaction was also observed between PAI-1 and hypotension (p=0.0242). After adjusting for these covariates, increasing ISS was associated with increasing PAI-1 levels (p<.0001).

Conclusion: Severely injured patients with hypotension have the highest levels of PAI-1. This suggests that elevated PAI-1 may reflect clinically significant endothelial damage. The lower levels observed in patients with isolated TBI may reflect less endothelial damage, despite similar ISS scores. Further studies are needed to clarify the interaction between the plasmin system, shock, and traumatic endotheliopathy.
Introduction: Trauma patients often progress from early hypocoagulable to normal and eventually hypercoagulable states, developing increased risk for venous thromboembolism (VTE). Prophylactic anticoagulation can decrease this risk, but its initiation is often delayed for extended periods due to concerns for bleeding. We characterized the transition from hypo- to hypercoagulability to determine the optimal time to initiate VTE chemoprophylaxis and hypothesized that hypocoagulability resolves within 24 hours after injury.

Methods: Serial blood samples were collected prospectively for 120 hours from critically injured patients at an urban Level I trauma center. Extrinsic thromboelastometry (EXTEM) maximum clot firmness (MCF) was used to classify patients as hypocoagulable (HYPO, <50 mm), normocoagulable (NORM, 50-70), or hypercoagulable (HYPER, >70). Dynamic coagulability and VTE occurrence were analyzed.

Results: 898 patients (median ISS 13, mortality 12%) were enrolled. Upon arrival, 4% were HYPO (88% NORM, 8% HYPER; figure), which increased to 12% at 6 h before downtrending. 96% were NORM by 24 h, and 57% were HYPER at 120 h. Mean MCF began in the NORM range, uptrended gradually, and entered the HYPER range at 120 h. Patients with isolated traumatic brain injury (TBI) followed a similar course, but more (66%) were HYPER at 120 h. Among NORM and HYPER patients at 48 h, 23% developed a symptomatic VTE.

Conclusion: Post-injury hypocoagulability resolves within 24 hours, and patients start to become hypercoagulable at 48 hours. This latter effect is exacerbated after TBI. VTE chemoprophylaxis should be initiated upon resolution of coagulopathy and is appropriate for most critically injured patients by 24 hours.
Paper #3 — EARL YOUNG COMPETITION
Monday, 2/26/2018

VALIDATING PREDICTIVE EQUATIONS TO ASSESS ENERGY EXPENDITURE IN ACUTE SPINAL CORD INJURY
C Ramirez, S Pelekhaty, J Massetti, S Galvagno, L Harmon, I Botwinick, T Scalea, D Stein
St. Luke’s University Health Network

Presenter: Christine Ramirez, MD
Sponsor: Deborah Stein, MD

Introduction: Acute spinal cord injury (SCI) is devastating with morbidities compounded by inadequate nutrition. The American Society for Parenteral and Enteral Nutrition recommends indirect calorimetry (IC) to evaluate energy needs in SCI because no predictive energy equations have been validated. We sought to identify predictive equations that could be used as alternative(s) to measured energy expenditure (MEE) from IC.

Methods: A retrospective review was performed over 2 years. Patients 18 years or older with cervical SCI who received IC were included. Height, weight, maximum temperature and minute ventilation on day of IC, plus MEE and VCO2 from IC were obtained. Estimated energy expenditure (EEE) was calculated using Harris-Benedict (HB), Penn State (PS), Mifflin-St.-Jeor (MSJ), Weir, Ireton-Jones (IJ) and 25 kcals/kg formulas. MEE was then compared to EEE of each method.

Results: Thirty-nine IC studies were done on 20 patients. Weir had the strongest correlation to MEE \( r=0.98 \), followed by PS \( r=0.83 \). Correlations were similar among HB \( r=0.78 \), MSJ \( r=0.75 \) and IJ \( r=0.73 \), and weakest with 24 kcal/kg \( r=0.55 \). All had a \( p<0.001 \). Deming regression confirmed strong correlations between Weir and PS to MEE, with coefficients of 1.03 and 1.43 \( (p<0.001) \), respectively. Other formulas had comparatively higher coefficients and standard errors. Bland-Altman analysis confirmed Weir had the narrowest range of difference, with a mean difference of 25.5 kcal/day, followed by PS (-326.8 kcal/day).

Conclusion: Weir is the best predictive energy equation, with all statistical tests demonstrating a strong correlation between MEE and Weir. The second best predictive equation is the Penn State formula and is a good alternative to indirect calorimetry in acute SCI.
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<th>HLA (μg/ml)</th>
<th>Syncytium (μg/ml)</th>
<th>Glycocalix thickness (Fluorescence intensity)</th>
<th>TM (μg/ml)</th>
<th>EOC (Fluorescence intensity)</th>
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<td>HUVEC control</td>
<td>10.9 ± 2.1</td>
<td>22.7 ± 2.3</td>
<td>192.7 ± 15.8</td>
<td>23.9 ± 1.9</td>
<td>60.7 ± 15.7</td>
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<td>100 mg/dL glucose</td>
<td>13.6 ± 1.4</td>
<td>23.3 ± 2.9</td>
<td>197.6 ± 15.6</td>
<td>26.2 ± 2.5</td>
<td>69.6 ± 11.6</td>
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<td>300 mg/dL glucose</td>
<td>44.8 ± 35</td>
<td>33.5 ± 44</td>
<td>151.8 ± 17.1</td>
<td>47.4 ± 28</td>
<td>52.1 ± 19.2</td>
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<td>HUVEC + HR + Epi</td>
<td>87 ± 4.4</td>
<td>94.2 ± 5.2</td>
<td>121 ± 17.8</td>
<td>103.6 ± 66</td>
<td>123.2 ± 18.3</td>
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<td>HR + Epi + 80 mg/dL glucose</td>
<td>89.3 ± 58</td>
<td>99 ± 58</td>
<td>121 ± 7.2</td>
<td>105.1 ± 9.1</td>
<td>130 ± 15.8</td>
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<td>HUVEC + Epi + 200 mg/dL glucose</td>
<td>152.6 ± 78.4</td>
<td>123 ± 62</td>
<td>92 ± 66</td>
<td>156.8 ± 16.2</td>
<td>160 ± 17.6</td>
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*p<0.05 vs. HUVEC control, #p<0.05 vs. HUVEC + HR + Epi

NOTES
Introduction: Early hyperglycemia is associated with higher mortality in trauma and predicts multiple organ failure. These findings do not appear to be due to nosocomial infections; however the mechanisms remain speculative. Endothelial cell (EC) injury and glycocalyx (GC) degradation occur following traumatic shock and are key factors in the development of trauma-induced coagulopathy, and result in impaired microvascular perfusion and accompanying organ failure. Acute hyperglycemia has been shown to result in the loss of the GC layer, EC inflammation, and activation of coagulation in vivo. These effects are increased by proinflammatory conditions such as diabetes. Microfluidic devices (MFD) provide an improved technology to study endothelial-glycocalyx biology vs. standard culture conditions. We postulated that acute hyperglycemia would exacerbate trauma induced EC injury and GC shedding and integrity. This was studied using a MFD in an in vitro model.

Methods: Human umbilical vein endothelial cells (HUVEC) monolayers established in the microfluidic channels of a MFD well plate were perfused at constant shear overnight. HUVEC monolayers were then exposed to hypoxia/reoxygenation (H/R) and epinephrine followed by the addition of varying concentrations of glucose. Perfusate from MFD outlets was obtained and GC shedding indexed by hyaluronic acid (HLA), and syndecan-1. Glycocalyx thickness was determined by FITC-wheat germ agglutinin. Endothelial injury was indexed by soluble thrombomodulin (TM). Reactive oxygen species (ROS) from EC were measured by a cell permeable dye and fluorescent microscopy.

Results: Table attached.

Conclusion: Exposure to hyperglycemia exacerbated adverse effects on the GC following H/R plus epinephrine exposure and may be related to enhanced production of ROS.
Predicted and Actual: June 30, 2016

Predicted: 9.93 Traumas, Mean ISS: 15.99

Actual: 10 Traumas, Mean ISS: 13.12

Number of Traumas

Mean Daily ISS

NOTES
ARTIFICIAL INTELLIGENCE CAN PREDICT DAILY TRAUMA VOLUME AND AVERAGE ACUITY
D Stonko, B Dennis, A Peetz, R Betzold, O Gunter, O Guillamondegui
Vanderbilt

Presenter: David Stonko, MS
Sponsor: Oscar Guillamondegui, MD MPH FACS

Introduction: The goal of this study was to integrate temporal and weather data in order to create an artificial neural network (ANN) to predict trauma volume, acuity and emergent operative cases at a level 1 trauma center.

Methods: Trauma admission data from TRACS and weather data from the National Oceanic and Atmospheric Administration (NOAA) was collected for all adult trauma patients from July 2013-June 2016. The ANN was constructed using temporal (time, day of week), and weather factors (daily high, active precipitation) to predict four points of daily trauma activity: number of traumas, number of penetrating traumas, average ISS, and number of immediate OR cases per day. We trained a two-layer feed-forward network with 10 sigmoid hidden neurons via the Levenberg-Marquardt backpropagation algorithm, and performed k-fold cross validation.

Results: 10,612 patients over 1,096 days were identified. The ANN accurately predicted the daily trauma distribution in terms of number of traumas, number of penetrating traumas, number of OR cases, and average daily ISS (combined training correlation coefficient $r = 0.9018+/-0.002$; validation $r = 0.8899+/-0.005$; testing $r = 0.8940+/-0.006$). As an example, for June 30, 2016 it predicted 9.93 traumas (actual: 10), and a mean ISS score of 15.99 (actual: 13.12); see figure.

Conclusion: We were able to successfully predict trauma and emergent operative volume, and acuity using an ANN by integrating local weather and trauma admission data from a level 1 center. This may prove useful for predicting trauma needs across the system and hospital administration when allocating limited resources.
BLUNT TRAUMATIC SCAPULAR FRACTURES ARE ASSOCIATED WITH GREAT VESSEL INJURIES IN CHILDREN
I Abd el-shafy, L Rosen, JM Prince, RW Letton, NG Rosen
Maimonides

Presenter: Ibrahim Abdelshafy, MD
Sponsor: Robert W. Letton, MD

Introduction: Patients with stable blunt great vessel injury (GVI) can have poor outcomes if the injury is not identified early. With current pediatric trauma radiation reduction efforts these injuries may be missed. As a known association between scapular fracture and GVI exists in adult blunt trauma patients, we examined whether that same association existed in pediatric blunt trauma patients.

Methods: Bluntly injured patients under 18 years old were identified from 2012-2014 in the National Trauma Data Bank. GVI included all major thoracic vessels and carotid/jugular. Demographics of patients with and without scapular fracture were compared with descriptive statistics. The chi-square test was used to examine this association using SAS v 9.4 (SAS Institute, Inc., Cary, NC).

Results: We found a significant association between pediatric scapular fracture and GVI. Of 291,632 children identified, 1960 had scapular fractures. Children with scapular fracture were ten times more likely to have GVI (1.2%) compared to those without (0.12%, p < 0.0001.) Most common GVI seen were carotid artery, thoracic aorta, and brachiocephalic or subclavian artery. Children with both scapular fracture and GVI were most commonly injured by motor vehicles (57% crash, 26% struck).

Conclusion: Injured children with blunt scapular fracture have a ten-fold greater risk of having a GVI when compared to children without scapular fracture. Presence of blunt traumatic scapular fracture should have appropriate index of suspicion for a significant GVI in pediatric trauma patients.
Figure 1.

107 Patients

- +CT/+RP
  33 (31%)

- +CT/-RP
  3 (3%)

- -CT/+RP
  68 (63%)

- -CT/-RP
  3 (3%)

NOTES
TRAUMATIC RECTAL INJURIES: IS THE COMBINATION OF COMPUTED TOMOGRAPHY AND RIGID PROCTOSCOPY SUFFICIENT?

J Veith, M Trust, C Brown, J Sharpe, T Musonza, J Holcomb, E Bui, B Bruns, A Hopper, M Truitt, C Burlew, M Schellenberg, J Sava, J Vanhorn PA-C,
The AAST Contemporary Management Of Rectal Injuries Study Group
University of Texas at Austin Dell Medical School Surgery Residency

Presenter: Jacob Veith, MD
Sponsor: Carlos Brown, MD, FACS

Introduction: There are no clear guidelines for the best test or combination of tests to identify traumatic rectal injuries. We hypothesize that computed tomography (CT) and rigid proctoscopy (RP) are a sufficient screening combination.

Methods: American Association for the Surgery of Trauma multi-institutional retrospective study (2004-2015) of patients who sustained a traumatic rectal injury. Patients with known rectal injuries who underwent both CT and RP as part of their diagnostic workup were included. Only patients with full thickness injuries (grade II-V) were included. CT findings of rectal injury, perirectal stranding, or rectal wall thickening and RP findings of blood, mucosal abnormalities, or laceration were considered positive.

Results: 107 patients were identified. Mean age was 32 years, 87(81%) were male, and 69(64%) involved penetrating mechanisms. A total of 36 (34%) and 71 (66%) patients had positive CT and RP findings, respectively. Test result combinations are shown in Figure 1. Only 3 (3%) patients had both a negative CT and negative RP. On further review, each of these three patients had intraperitoneal injuries and had indirect evidence of rectal injury on CT scan including pneumoperitoneum or sacral fracture.

Conclusion: As stand-alone tests, neither CT nor RP can adequately identify traumatic rectal injuries. However, the combination of both CT and RP identifies 97% of all traumatic rectal injuries. Intraperitoneal injuries may be missed by both CT and RP, so patients with a high index of suspicion and/or indirect evidence of rectal injury on CT scan may necessitate laparotomy for definitive diagnosis.
Table 1: Serial TEG Variables Following Platelet Transfusion

<table>
<thead>
<tr>
<th></th>
<th>Baseline TEG N= 35</th>
<th>Second TEG N= 35</th>
<th>Third TEG N= 16</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Split point (SP; min), mean ± SD</td>
<td>3.9±1.2</td>
<td>3.8±.81</td>
<td>3.8±7.3</td>
<td>0.92</td>
</tr>
<tr>
<td>Reaction time (r; min), mean ± SD</td>
<td>4.3±1.4</td>
<td>4.2±.87</td>
<td>4.1±8.0</td>
<td>0.80</td>
</tr>
<tr>
<td>Clot formation time (K2; min), mean ± SD</td>
<td>1.6±.75</td>
<td>1.1±.22</td>
<td>.92±.14</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Angle (α; degree), mean ± SD</td>
<td>68.5±6.9</td>
<td>73.7±2.9</td>
<td>76.3±2.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Maximal amplitude (MA; mm), mean ± SD</td>
<td>63.03±6.8</td>
<td>67.8±4.1</td>
<td>71.4±4.4</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>G value (dyynes/cm²), mean ± SD</td>
<td>8.9±2.4</td>
<td>10.8±2</td>
<td>12.9±3.1</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Adenosine diphosphate inhibition (%), mean ± SD</td>
<td>88.6±12.6</td>
<td>69.4±22.1</td>
<td>58.8±24.5</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Arachidonic acid inhibition (%), mean ± SD</td>
<td>40.3±32.6</td>
<td>30±28.4</td>
<td>25.4±29.06</td>
<td>0.19</td>
</tr>
</tbody>
</table>

**NOTES**
GOAL DIRECTED PLATELET TRANSFUSIONS CORRECT PLATELET DYSFUNCTION AND IMPROVE MORTALITY IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY

E Furay, MJ Daley, N Malesa, C Tellinghuisen, S Ali, LH Brown, JD Aydelotte, PG Teixeira, TB Coopwood, CVR Brown

UT Austin Dell Seton Medical Center

Presenter: Elisa Furay, MD
Sponsor: Carlos VR Brown, MD

Introduction: Platelet dysfunction, defined as adenosine diphosphate (ADP) inhibition greater than 60% on thromboelastogram (TEG), is an independent predictor of increased mortality in patients with severe traumatic brain injury (TBI). We changed our practice to transfuse platelets for all patients with severe TBI and platelet dysfunction. We hypothesized that platelet transfusions would correct platelet dysfunction and improve mortality in patients with severe TBI.

Methods: This retrospective review included adult trauma patients admitted to our Level 1 trauma center from July 2015 to October 2016 with severe TBI (Head AIS >/= 3) who presented with platelet dysfunction and subsequently received a platelet transfusion. Serial TEGs were obtained to characterize the impact of platelet transfusion on clot strength. Subsequently, the platelet transfusion group was compared to a group of historical controls with severe TBI patients and platelet dysfunction who did not receive platelet transfusion.

Results: A total of 35 patients with severe TBI presented with platelet dysfunction. Following platelet transfusion clot strength improved as represented by decreased K time, increased angle, maximum amplitude, and G-value, as well as correction of ADP inhibition (table 1). When comparing to 51 historic controls with severe TBI and platelet dysfunction, the 35 study patients who received a platelet transfusion had a lower mortality (9% vs. 35%; p=0.005). In stepwise logistic regression, platelet transfusion was independently associated with decreased mortality (OR 0.23; 95% CI 0.06-0.92; p=0.038).

Conclusion: In patients with severe TBI and platelet dysfunction, platelet transfusions correct platelet inhibition and are independently associated with decreased mortality.
<table>
<thead>
<tr>
<th></th>
<th>TBI (n=54)</th>
<th>Non-TBI (n=179)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>ADP AUC</td>
<td>60 (48-70)</td>
<td>60 (46-71)</td>
<td>0.69</td>
</tr>
<tr>
<td>Collagen AUC</td>
<td>51 (38-63)</td>
<td>49 (40-61)</td>
<td>0.92</td>
</tr>
<tr>
<td>TRAP AUC</td>
<td>99 (82-118)</td>
<td>97 (82-111)</td>
<td>0.50</td>
</tr>
<tr>
<td>ASPI AUC</td>
<td>60 (40-69)</td>
<td>61 (49-71)</td>
<td>0.35</td>
</tr>
<tr>
<td>RISTO AUC</td>
<td>57 (38-77)</td>
<td>69 (46-88)</td>
<td>0.03</td>
</tr>
<tr>
<td>Factor II (% activity)</td>
<td>69 (59-80)</td>
<td>72 (61-84)</td>
<td>0.28</td>
</tr>
<tr>
<td>Factor VII (% activity)</td>
<td>70 (60-90)</td>
<td>83 (62-100)</td>
<td>0.05</td>
</tr>
<tr>
<td>Factor VIII (% activity)</td>
<td>215 (132-346)</td>
<td>156 (97-221)</td>
<td>0.01</td>
</tr>
<tr>
<td>Factor IX (% activity)</td>
<td>122 (105-151)</td>
<td>137 (107-166)</td>
<td>0.21</td>
</tr>
<tr>
<td>Factor X (% activity)</td>
<td>76 (60-83)</td>
<td>79 (66-90)</td>
<td>0.17</td>
</tr>
</tbody>
</table>

*Bolded for p<0.05. Data for skewed variables reported as median with inter-quartile ranges.
Agonist responses to collagen (COL), thrombin receptor-activating peptide-6 (TRAP), adenosine diphosphate (ADP), arachidonic acid (ASPI), and ristocetin von Willebrand complex (RISTO-vWF) were measured and results reported as area under the aggregation curve in units (U), aggregation (AU), velocity (AU/min), and baseline/end impedances (Ω).*

**NOTES**
PERHAPS IT’S NOT THE PLATELET: RISTOCETIN UNCOVERS THE POTENTIAL ROLE OF VON WILLEBRAND FACTOR IN PLATELET DYSFUNCTION FOLLOWING TRAUMATIC BRAIN INJURY

L Kornblith, A Robles, A Conroy, C Hendrickson, R Callcut, M Cohen
University of California San Francisco, Zuckerberg San Francisco General Hospital

Presenter: Lucy Kornblith, MD
Sponsor: Mitchell Jay Cohen, MD

Introduction: Early investigations have identified disruption of the protective blood-brain barrier (BBB) exposing endothelium rich in platelet activating factor and von Willebrand factor (vWF) as a potential driver of platelet dysfunction following traumatic brain injury (TBI). We examined these mechanisms by performing platelet aggregometry in severely injured patients and hypothesized that TBI patients have decreased agonist response consistent with disruption of the BBB.

Methods: Blood was collected from 233 trauma patients with normal platelet counts. Platelet function was assessed using multiple electrode platelet aggregometry. Agonist responses to collagen (COL), thrombin receptor-activating peptide-6 (TRAP), adenosine diphosphate (ADP), arachidonic acid (ASPI), and ristocetin-von Willebrand complex (RISTO-vWF) were measured. Factor activity was measured.

Results: Of the 233 patients, 23% had TBI. There was no difference in baseline impedance or agonist response to ADP, ASPI, TRAP, or COL between TBI and non-TBI patients. However, RISTO-vWF response was significantly lower in TBI patients (TABLE). TBI patients had higher activity of Factor VIII (272% vs. 193%, p<0.05) while all other factor activity was lower/unchanged. In multivariate analysis, low RISTO-vWF was the only agonist significantly associated with TBI (OR 3.05, p<0.05).

Conclusion: Given the central importance of platelet function following injury, elucidating specific drivers and deficits is critical for identifying future therapeutic targets. The decrease in RISTO-vWF response and increase in factor VIII activity in TBI patients may be secondary to an injury-induced deficit in vWF leading to decreased FVIII carrier function. Known treatments for vWF deficits are available and represent a potential therapeutic target to address traumatic induced platelet dysfunction.
<table>
<thead>
<tr>
<th>Outcomes</th>
<th>Warfarin (n=140)</th>
<th>NOACs (n=70)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progression of ICH, % (n)</td>
<td>13% (18)</td>
<td>26% (18)</td>
<td>0.03</td>
</tr>
<tr>
<td>Neurosurgical Intervention, % (n)</td>
<td>9.2% (13)</td>
<td>20% (14)</td>
<td>0.04</td>
</tr>
<tr>
<td>Hospital LOS, median [IQR]</td>
<td>4 [3-8]</td>
<td>5 [4-8]</td>
<td>0.34</td>
</tr>
<tr>
<td>SNiF disposition, % (n)</td>
<td>21% (30)</td>
<td>27% (19)</td>
<td>0.14</td>
</tr>
<tr>
<td>Mortality, % (n)</td>
<td>9% (13)</td>
<td>20% (14)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

NOTES
THE NOVEL ORAL ANTICOAGULANTS (NOACS) HAVE WORSE OUTCOMES COMPARED TO WARFARIN IN PATIENTS WITH TRAUMATIC BRAIN INJURY
M Zeeshan, F Jehan, A Jain, M Hamidi, ER Zakaria, L Gries, A Tang, T O’Keeffe, N Kulvatunyou, B Joseph
University of Arizona

Presenter: Muhammad Zeeshan, MD
Sponsor: Narong Kulvatunyou, MD

Introduction: Novel-oral-anticoagulants (NOACs) use is increasing in trauma patients. The reversal of these agents after hemorrhage is still evolving. The aim of our study was to evaluate outcomes after traumatic brain injury in patients on NOACs.

Methods: 3-year (2014-2016) analysis of our prospectively maintained TBI database. We included all TBI patients with intracranial-hemorrhage (ICH) on anticoagulants. Patients were stratified into two groups; those on NOACs and on Warfarin, and were matched in a 1:2 ratio using propensity score matching for demographics, injury and vital parameters, type, and size of ICH. Outcome measures were progression of ICH, mortality, SNF disposition, and hospital and ICU length of stay (LOS).

Results: We analyzed 1459 TBI patients, of which 210 patients were matched (NAOCs: 70, Warfarin: 140). Matched groups were similar in age (p=0.21), mechanism of injury (p=0.61), GCS (p=0.54), ISS (p=0.62), and type and size of ICH (p=0.09). Patients on pre-injury NOACs had higher rate of progression (p=0.03), neurosurgical intervention (p=0.04), mortality (p=0.04), and longer ICU LOS (p=0.04) compared to patients on warfarin. However, there was no difference in hospital-LOS (p=0.22) and SNF disposition (p=0.14) (Table-1). On sub-analysis of severe-TBI patients (GCS<8), rate of progression (p=0.59), neurosurgical intervention (p=0.62) or mortality (p=0.81) was similar in both groups.

Conclusion: NOACs use is associated with increased risk of progression of ICH, neurosurgical intervention and mortality after a TBI in less severely injured group. Development of reversal agents for the NOACs and implementation of a strict protocol for the reversal of these agents may lead to improve outcomes.
Figure 1A. Intravital images of TBI studies in murine model.

Figure 1B. Analysis of intravital imaging measuring intravascular vs. interstitial fluorescence. $\Delta f = \frac{I_2 - I_1}{I_1}$

$\Delta f$ is blood-brain barrier hyperpermeability; $I_1$ is intravascular fluorescence; and $I_2$ is interstitial fluorescence. $^{* * *} = p < 0.05$

NOTES
QUETIAPINE PROTECTS THE BLOOD-BRAIN BARRIER IN TRAUMATIC BRAIN INJURY
B Robinson, C Isbell, C Anasooya Shaji, S Kurek, B Tharakan
Scott and White Medical Center - Temple

Presenter: Bobby Robinson, MD
Sponsor: Alexander Eastman, MD

Introduction: The integrity of the blood-brain barrier (BBB) is paramount in limiting vasogenic edema following traumatic brain injury (TBI). The purpose of this study was to ascertain if quetiapine, an atypical antipsychotic commonly used in trauma/critical care, protects the BBB and attenuates hyperpermeability in TBI.

Methods: Rat brain microvascular endothelial cells (BMECs) were pretreated with quetiapine (20×M; 1 hour) by an inflammasome activator (20×g/mL chitosan; 2 hours). Immunostaining for tight junction proteins zonula occludens-1 (ZO-1) and adherens junction protein β-catenin was performed. Human BMECs were grown as a monolayer and pretreated with quetiapine (20×M; 1 hour). Permeability was induced with chitosan (20×g/mL; 2 hours), and transendothelial electrical resistance (TEER) was measured. C57BL/6 mice (n=15) underwent mild-moderate TBI (controlled cortical impactor) or sham craniotomy. The treatment group was given 10 mg/kg quetiapine intravenously 10 minutes after TBI. The difference in fluorescence intensity between intravascular and interstitium (ΔI) represents BBB hyperpermeability. Molecular docking was performed with AutoDock Vina to key proteolytic enzymes in TBI.

Results: Quetiapine was protective in vitro and in vivo. Junctional staining of ZO-1 and β-catenin showed retained integrity in quetiapine-treated cells as compared to the chitosan group. Quetiapine attenuated monolayer permeability compared to chitosan group (p<0.05). In the animal studies, there was a significant decrease in BBB permeability when compared between the TBI and TBI plus quetiapine groups (p<0.05). There was no difference between the Sham and TBI plus quetiapine groups (p=NS). In silico studies showed quetiapine thermodynamically-favorable binding to matrix metalloproteinase-9 (MMP-9).

Conclusion: Quetiapine may have novel anti-inflammatory properties to acutely treat TBI.
EXTENDING THE GOLDEN HOUR FOR ZONE 1 REBOA: IMPROVED SURVIVAL AND REPERFUSION INJURY WITH INTERMITTENT VERSUS CONTINUOUS REBOA IN A PORCINE SEVERE TRUNCAL HEMORRHAGE MODEL

J Kuckelman, M Barron, D Moe, M Derickson, C Phillips, J Kononchik, MLallemand, S Marko, M Eckert, M Martin
Madigan Army Medical Center

Presenter: John Kuckelman, DO
Sponsor: Matthew Martin, MD

Introduction: Non-compressible hemorrhage can be controlled using resuscitative endovascular occlusion of the aorta (REBOA). Prolonged ischemia limits REBOA application during Zone 1 deployment. Intermittent inflation/deflation may effectively mitigate this problem.

Methods: A lethal abdominal vascular injury was created in 28 swine. Animals were randomized to controls (n=7), 60min full REBOA (FR, n=5), time-based intermittent REBOA (iRT, n=7), and pressure-based REBOA (iRP, n=9). Intermittent groups had an initial inflation for 15min, followed by 10min inflation:3min deflation cycles (iRT) or an inflate/deflate schedule based on blood pressure (MAP)<40mmHg (iRP). Experiments were concluded after 120min or death (MAP<20mmHg).

Results: Intermittent REBOA animals all survived to 120min versus 15min for controls and 63min for FR(p<0.001, Figure). After 60min, FR animals were more hypotensive(MAP 20mmHg vs 80mmHg(iRP) and 100mmHg(iRT), p<0.001), had lower cardiac output(1.06mL/min vs 5.1L/min(iRP) and 8.2L/min(iRT), p<0.001), higher lactate(12.5mg/dL vs 8.5mg/dL(iRP), p=0.02), and decreased clot firmness on ROTEM than iRP/T(64mm vs 69mm(iRP) and 69mm(iRT), p=0.04). Acidosis was worse in iRT versus iRP at 120min(pH 7.28 vs 7.12, p=0.02), improved lactate(11.9mg/dL vs 16.3mg/dL, p=0.04), and decreased whole blood resuscitation(452cc vs 646cc, p=0.05). Blood loss (clot weight) was higher in controls(2.0kg) versus iRT and iRP(1.16kg and 1.23kg, p<0.01) and not different from FR(0.87kg, p=0.10).

Conclusion: Intermittent REBOA can maintain supraceliac hemorrhage control while decreasing distal ischemia in a swine model. Prolonged survival times, decreased acidosis, and lower resuscitation requirements indicate that this technique could potentially extend Zone 1 REBOA deployment times. Schedules based on MAP may be superior to time-based regimens.
NO WIRE? NO PROBLEM: RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA (REBOA) CAN BE PERFORMED EFFECTIVELY AND MORE RAPIDLY WITH A WIRE-FREE DEVICE.
A Romagnoli, W Teeter, P Wasicek, B Gamble, P Hu, D Stein, T Scalea, M Brenner
RA Cowley Shock Trauma Center

Presenter: Anna Romagnoli, MD
Sponsor: Megan Brenner, MD

Introduction: A wire-free device is available for REBOA providing aortic occlusion (AO) without lengthy platform guide-wires and large sheaths.

Methods: Patients who received REBOA from May 2014-September 2017 were enrolled. Timing of procedural steps was measured in seconds (s) using time-stamped videography.

Results: 74 patients received REBOA; 29 with a platform guidewire, 12F sheath, and balloon catheter (12F group), and 45 with a 7F sheath and wire-free device (7F group). Mean age (p=0.18) and ISS (p=0.95) were similar between groups. 59 patients received REBOA at Zone 1; 15 patients at Zone 3. There was no difference in median [IQR] time to common femoral artery (CFA) access between the 7F (194[98,313]s) and 12F (193[126,280]s) groups (p=0.96). Both median time to AO after CFA access (7F:158[109,264]s vs. 12F:307[222,390]s, p<0.001) and median total procedural time (7F:366[263,596]s vs. 12F:511[441,597]s; p=0.012) were significantly shorter with the wire-free system. The rates of percutaneous versus open CFA access was not different between groups (p=0.48). Both groups had a similar physiologic response to AO as measured by pre- and post-AO SBP (p=0.86). Overall mortality rate was 74%; 90% in the 12F group, and 64% in the 7Fr group (p = 0.027). The procedure-related complication rate was not significantly different between groups (12F:7% vs 7F:9%, p=1.0).

Conclusion: Once CFA access is obtained, AO with a smaller wire-free device reduces procedural time by approximately 50%. When perfusion to proximal organs is essential, the seconds saved to achieve AO may contribute to improved mortality. Time to obtain CFA access is not dependent on introducer sheath size.
NOTES
AORTIC BRANCH VESEL FLOW DURING RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA (REBOA)

M Hoehn, W Teeter, J Morrison, B Gamble, P Hu, D Stein, M Brenner, T Scalea
RA Cowley Shock Trauma University of Maryland

Presenter: Melanie Hoehn, MD
Sponsor: Megan Brenner, MD

Introduction: REBOA (Resuscitative Endovascular Balloon Occlusion of the Aorta) is a torso hemorrhage control adjunct. Aortic branch vessel flow (BVF) during REBOA is unknown and has implications for ischemia-reperfusion (I-R) injury. The aim of this study is to characterize branch vessel flow in hypovolemic shock with and without REBOA.

Methods: Female swine (79-90kg) underwent anesthesia, 40% controlled hemorrhage and sonographic flow monitoring of the carotid, hepatic, superior mesenteric, renal and femoral arteries. Animals were randomized to REBOA (n=5) and no-REBOA (n=5); underwent limited whole-blood resuscitation for 4-hours, followed by full resuscitation and balloon deflation for 1-hour.

Results: All animals were successfully induced into hemorrhagic shock with a mean decrease of flow in all vessels of 50% from baseline (p<0.001). Deployment of REBOA resulted in a 200-400% increase in carotid flow, but near complete abolition of BVF distal to the balloon (Figure). The no-REBOA group saw recovery of BVF to 100% of baseline in all measured vessels, except the hepatic at 50-75%. 2-way ANOVA confirmed a significant difference between the groups throughout the protocol (p<0.001). During resuscitation, the REBOA group saw BVF restore to between 25-50%, but never achieving baseline values. The lactate at 4 hours was significantly higher in the REBOA vs. no-REBOA group (17.240.1 vs. 4.941.4; p<0.001).

Conclusion: REBOA not only abolishes BVF during occlusion, but appears to have a post-REBOA effect, reducing visceral perfusion. This may be a source of REBOA associated I-R injury and warrants further investigation in order to mitigate this effect.
DOES SELECTIVE USE OF TRANEXAMIC ACID MATTER: FIBRINOLYSIS PHENOTYPES AND ASSOCIATED OUTCOMES

H Moore, B Cotton, A Saueria, E Moore, W Dorlac, J Dubose, C Wade, J Morrison
University of Colorado

Presenter: Hunter Moore, MD
Sponsor: Charles Wade, MD

Introduction: The MATTERs studies suggested improved survival in combat casualties receiving tranexamic acid (TXA), however, civilian trauma studies are not in agreement. Recent data suggests TXA may be harmful in trauma patients who present to the hospital with a physiologic level of fibrinolysis. We hypothesize that a post hoc analysis of the MATTERs data would identify a cohort of patients who did not benefit from TXA based on their predicted fibrinolysis phenotype.

Methods: Thrombelastography (TEG) data available in trauma patients requiring a blood transfusion before antifibrinolytics were used to predict abnormal fibrinolysis (elevated or depressed LY30) based on clinical variables that were available in the MATTERs database. This model was then used to predict which patients from MATTERs would have abnormal vs physiologic fibrinolysis. Primary outcomes were survival and thrombotic complications.

Results: 1,142 patients were used to build the propensity model for fibrinolysis abnormalities. Variables matched 545 patients in the MATTERs database. Patients predicted to have abnormal fibrinolysis had decreased mortality associated with TXA (11% vs 26% p=0.033), while those in physiologic fibrinolysis had increased mortality (12% vs 6% p=0.062). Patients in the abnormal fibrinolysis group who received TXA had a higher rate of PE (9% vs 0% p=0.021). Those who received TXA in the physiologic group were more likely to have a DVT (3% vs 0% p=0.001).

Conclusion: This post hoc analysis of MATTERs supports the ongoing need to refine the use of TXA in trauma patients, as this medication in the right patient at the right time appears to be lifesaving, but may cause harm to those when not indicated.
### Table 1. Outcome Measures

<table>
<thead>
<tr>
<th>Outcomes</th>
<th>No-TXA (n=62)</th>
<th>TXA (n=31)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary outcomes</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mortality, % (n)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6-hours mortality</td>
<td>34% (21)</td>
<td>16.1% (5)</td>
<td><strong>0.03</strong></td>
</tr>
<tr>
<td>12-hours mortality</td>
<td>37% (23)</td>
<td>22.6% (7)</td>
<td>0.16</td>
</tr>
<tr>
<td>24-hours mortality</td>
<td>40% (25)</td>
<td>25.8% (8)</td>
<td>0.15</td>
</tr>
<tr>
<td>30-day mortality</td>
<td>50% (31)</td>
<td>45.2% (14)</td>
<td>0.69</td>
</tr>
<tr>
<td>Time to achieve hemostasis, m, median [IQR]</td>
<td>158 [87-243]</td>
<td>139 [86-255]</td>
<td>0.65</td>
</tr>
<tr>
<td>Re-bleeding requiring intervention, % (n)</td>
<td>3.2% (2)</td>
<td>9.7% (3)</td>
<td>0.13</td>
</tr>
</tbody>
</table>

**NOTES**
Paper #16  
Tuesday, 2/27/2018

SEVERELY INJURED TRAUMA PATIENTS WITH ADMISSION HYPERFIBRINOLYSIS; IS THERE A ROLE OF TRANEXEMIC ACID? FINDINGS FROM THE PRAGMATIC, RANDOMIZED OPTIMAL PLATELET AND PLASMA RATIOS (PROPPR) TRIAL

M Khan, F Jehan, J Holcomb, T O’Keeffe, C Wade, E Bulger, B Joseph  
University of Arizona

Presenter: Muhammad Khan, MD  
Sponsor: John B. Holcomb, MD

Introduction: Administration of Tranexemic acid (TXA) in coagulopathy-of-trauma (COT) has gained popularity after the CRASH-2 trial. The aim of our analysis was to analyze the role of TXA in severely injured trauma patients with admission hyperfibrinolysis.

Methods: We reviewed the prospectively collected Pragmatic, Randomized Optimal Platelet and Plasma Ratios (PROPPR) database. We included patients with admission hyperfibrinolysis (Ly30>3%) on thromboelastography. Patients were stratified into two groups (TXA and No-TXA) and were matched in 1:2 ratio using propensity score matching for demographics, admission vitals, and injury severity. Primary outcome measures were 6h, 12h, 24hr, and 30d mortality, time to achieve hemostasis and re-bleeding after hemostasis requiring intervention. Secondary outcome measures were ICU and hospital-length of stay (LOS) and thrombotic complications.

Results: We analyzed 680 patients. 134 patients had admission Ly30>3%, of which 93 patients (TXA: 31; No-TXA: 62) were matched. Matched groups were similar in age (p=0.67), sex (p=0.57), mechanism of injury (p=0.22), ED SBP (p=0.70), ED HR (p=0.68) and ISS (p=0.81) between the two groups. Table 1. Demonstrates our outcome measures. TXA groups had lower 6h mortality (p=0.03), however, there was no difference in 12h (p=0.16), 24h (p=0.15), and 30d (p=0.69) mortality, time to achieve hemostasis (p=0.65) and re-bleeding after hemostasis (p=0.13) between the two groups. There was no difference in the hospital (p=0.22) and ICU LOS (p=0.57) and thrombotic complications (p=0.98).

Conclusion: TXA does not improve outcomes in severely injured trauma patients who develop hyperfibrinolysis. Further studies are required to better define the subset of trauma population, which may benefit from this therapy.
Pro/Con Debate
Tuesday, 3/7/2017

TRANEXAMIC ACID (TXA) IS THE ELIXIR OF LIFE AND SHOULD BE ADMINISTERED TO ALL POTENTIALLY BLEEDING PATIENT

Pro: Michel Aboutanos MD; Con: James Haan MD
ALGORITHM #1 - OPEN ABDOMEN

Jack Sava, MD

Trauma Laparotomy

Indication for Damage Control/Open Abdomen

Open abdomen management

Y N

PRIMARY FASCIAL CLOSURE

Response to resuscitation

Y N

Plan reoperation in 8-72h

Consider ongoing hemorrhage, missed hollow viscus injury, anastomotic leak, abdominal compartment syndrome, organ/limb ischemia. Consider early/emergent reoperation if abdominal source suspected

Relaparotomy

Definitive management of injuries

Indication for continued open abdomen management

N

PRIMARY FASCIAL CLOSURE

Y

Continue open abdomen management. Consider impact of open abdomen on other organs/systems

Successful

Attempt progressive primary abdominal closure

Unsuccessful

ALTERNATIVE ABDOMINAL CLOSURE
Background: We have previously reported our management of infected rib stabilization hardware and suggested an algorithm using antibiotic beads to suppress infection. We have gained further experience with this algorithm and have extrapolated to patients deemed high risk for hardware infection.


Results: 285 patients underwent SSRF, of which 10 (3.5%) developed confirmed hardware infection. Nine had hardware salvaged until rib healing by implanting antibiotic beads. One underwent immediate hardware explantation with no attempt at salvage due to prior experience with difficult removal of certain types of hardware. An additional 8 patients met our guidelines for SSRF, but were judged high risk for hardware infection due to pneumonia, empyema, or chest tube placed in the pre-hospital setting. These patients underwent SSRF with prophylactic antibiotic bead implantation at index operation to suppress infection until rib healing. To date, antibiotic beads and hardware have been removed in 12 of 17 patients at a median time of 166 days from index operation. All patients demonstrated rib fracture union prior to antibiotic bead, plate and screw hardware removal.

Discussion: We identified a 3.5% incidence of hardware infection, prompting development of a treatment algorithm which has successfully salvaged infected hardware until fracture healing. From this, we extrapolated the potential benefits in high risk patients, and have subsequently developed a practice management guideline for patients deemed high risk for hardware infection, comprised of prophylactic antibiotic bead implantation and placement of hardware amenable to future retrieval.
PANEL OF EXPERTS
Tuesday, 2/27/18
Case presenter: Ajai Malhotra, MD

Panelists: Mitch Cohen MD, Stephanie Savage MD; Nirav Patel MD, Oliver Gunther MD
Paper #18 — FAMILY ABSTRACT
Tuesday, 2/27/2018

OBSTETRICAL HEMORRHAGE - UTILIZATION OF TRAUMA RESOURCES TO SAVE ONE OF OUR OWN
E Windell, J Sweeney, K Wilson-Panum, T Green, M Barney, W Long
Legacy Emanuel Medical Center

Presenter: Elizabeth Windell, DO
Sponsor: William Long, MD

Introduction: Placenta accreta is a potentially life-threatening obstetrical complication that occurs ~1/600 deliveries. The placenta implants deep into the uterine wall and is unable to separate after delivery, leading to massive hemorrhage. At our institution, if a massive transfusion protocol (MTP) is initiated, trauma resources are mobilized to assist with management. In this case, our protocol was used to save one of our own.

Methods: A G1P0, 37w0d trauma surgeon went into labor after SROM. Labor was augmented with misoprostol and Pitocin. 37 hrs later a healthy infant was born. Unfortunately, patient had a previously unrecognized placental accreta and 3rd stage of labor prolonged. She had ~1.8L of blood loss. Decision was made to proceed to D&C for removal of the placenta. There was ongoing blood loss of 3.8L and severe hypotension. MTP was initiated, and trauma resources were mobilized to the OR. Patient received a total of 14 units of blood products. Rotem was used to guide resuscitation. Post-operatively, the patient was taken for uterine artery gelfoam embolization due to ongoing hemorrhage. She recovered well and was discharged home 4 days later.

Results: none

Conclusion: Our MTP facilitates the rapid restoration of circulating blood volume for patients in hemorrhagic shock. Trauma resources are utilized to assist any hemorrhaging patient, including obstetrical patients. In this case report, our protocol helped to save the life of our own trauma surgeon. She had to miss WTA 2017 due to this, but my son will forever be a WTA kid!
PRESIDENTIAL ADDRESS
Tuesday, 2/27/18

A PRIVILEGE NOT A RIGHT
Dennis W. Vane, MD
Table 1. Selected protective and risk factors for 1-year readmission with venous thromboembolism, presented as OR (95% CI), p < 0.01.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Risk of readmission</th>
<th>Risk of readmission to a different hospital</th>
</tr>
</thead>
<tbody>
<tr>
<td>Penetrating injury</td>
<td>0.82 (0.77-0.88)</td>
<td>NS</td>
</tr>
<tr>
<td>Income &lt; $38,000</td>
<td>1.09 (1.07-1.12)</td>
<td>1.13 (1.07-1.19)</td>
</tr>
<tr>
<td>Injury Severity Score &gt; 15</td>
<td>1.11 (1.09-1.14)</td>
<td>1.13 (1.07-1.18)</td>
</tr>
<tr>
<td>For-profit hospital</td>
<td>1.11 (1.09-1.14)</td>
<td>1.34 (1.28-1.41)</td>
</tr>
<tr>
<td>Major procedure</td>
<td>1.15 (1.13-1.17)</td>
<td>0.74 (0.71-0.76)</td>
</tr>
<tr>
<td>Medicaid</td>
<td>1.18 (1.14-1.23)</td>
<td>1.17 (1.07-1.28)</td>
</tr>
<tr>
<td>Length of stay &gt; 7 days</td>
<td>1.36 (1.33-1.38)</td>
<td>1.16 (1.11-1.21)</td>
</tr>
<tr>
<td>Discharge to skilled nursing</td>
<td>1.77 (1.73-1.82)</td>
<td>0.88 (0.82-0.91)</td>
</tr>
<tr>
<td>≥ 65 years old</td>
<td>2.09 (2.00-2.18)</td>
<td>NS</td>
</tr>
<tr>
<td>Venous injury</td>
<td>3.62 (3.19-4.10)</td>
<td>NS</td>
</tr>
</tbody>
</table>

NOTES
HIDDEN BURDEN OF VENOUS THROMBOEMBOLISM AFTER TRAUMA: A NATIONAL ANALYSIS INCLUDING READMISSION TO A DIFFERENT HOSPITAL

R Rattan, J Parreco, S Eidelson, N Namias
University of Miami

Presenter: Joshua Parreco, MD
Sponsor: Nicholas Namias, MD

Introduction: Trauma patients are at increased risk for venous thromboembolism (VTE). A large proportion of trauma readmissions occur at a different hospital. There are no national studies measuring readmissions to different hospitals for VTE following trauma. Thus, the national burden in trauma patients with newly diagnosed VTE on readmission is unknown and can provide a benchmark to improve quality of care.

Methods: The Nationwide Readmission Database (2010-2014) was queried for patients ≥18 years non-electively admitted for trauma. Patients with VTE or IVC filter placement on index admission were excluded. Outcomes included 30-day and 1-year readmission to both index and different hospitals with a new diagnosis of VTE. Multivariable logistic regression identified risk factors. Results were weighted for national estimates.

Results: Of the 5,158,390 patients admitted for trauma, 1.2% (n=61,881) were readmitted with VTE in 1 year and of those, 29.6% (n=18,326) were readmitted to a different hospital. While some risk factors, such as admission to a for-profit hospital, Medicaid, or income <$38,000, persisted independent of readmission hospital, others were protective (Table 1). The yearly cost of VTE readmission was $235 million, with $82 million (34.9%) due to different hospital readmission.

Conclusion: Previously unreported, nearly 1 in 3 patients readmitted with VTE after trauma, accounting for over a third of the cost, present to another hospital and are not captured by current metrics. Risk factors are unique. This has significant implications for benchmarking, outcomes, prevention, and policy.
<table>
<thead>
<tr>
<th></th>
<th>No DVT (n=5479)</th>
<th>DVT (n=64)</th>
<th>Acute (n=56)</th>
<th>Non-acute (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>62.09±22.55</td>
<td>57.44±19.04</td>
<td>56.26±19.37</td>
<td>67.33±16.57</td>
</tr>
<tr>
<td><strong>Gender (% M:F)</strong></td>
<td>51:49</td>
<td>70:30</td>
<td>75:25</td>
<td>33:67</td>
</tr>
<tr>
<td><strong>Mechanism (% B:P)</strong></td>
<td>97:3</td>
<td>100:0</td>
<td>100:0</td>
<td>100:0</td>
</tr>
<tr>
<td><strong>ISS</strong></td>
<td>11.80±8.07</td>
<td>18.50±11.60</td>
<td>17.80±11.50</td>
<td>20.70±13.50</td>
</tr>
<tr>
<td><strong>ICULOS (days)</strong></td>
<td>1.86±4.47</td>
<td>8.38±12.00</td>
<td>8.64±12.70</td>
<td>5.00±3.90</td>
</tr>
<tr>
<td><strong>HLOS (days)</strong></td>
<td>7.73±10.92</td>
<td>21.52±13.21</td>
<td>22.14±16.10</td>
<td>11.83±5.30</td>
</tr>
<tr>
<td><strong>Mortality</strong></td>
<td>102 (1.9%)</td>
<td>7 (10.9%)</td>
<td>6 (10.7)</td>
<td>1 (16.7%)</td>
</tr>
</tbody>
</table>

* p<0.05 No DVT vs DVT

# p<0.05 Acute vs Non-acute

NOTES
NOT ALL DVT IS CREATED EQUAL: INCIDENCE OF PRE-EXISTING DVT AMONG HIGH-RISK TRAUMATIZED PATIENTS
A Tefft, T Moss, A Stanley, A Malhotra

Presenter: Amy Tefft, MD
Sponsor: Ajai Malhotra, MD

Introduction: Centers for Medicare and Medicaid Services classifies Deep Vein Thrombosis (DVT) as a Hospital Acquired Condition and can deny payment for care unless the DVT was present on admission. There is paucity of data regarding pre-existing DVT among traumatized patients. In a recent single institution study with surveillance 30% of DVTs may have been pre-existing. We sought to determine rates and patient characteristics of pre-existing DVT without surveillance at our ACS verified level-I trauma center.

Methods: A list of all traumatized patients with stay >/=2 days diagnosed with DVT over 7-year study period ending December 2016 was obtained from the trauma registry. Duplex scans of these patients were reviewed and DVT classified as ‘acute’ and ‘non-acute’ (pre-existing) based on accepted sonographic criteria. Incidence and patient characteristics of the two forms of DVT were compared.

Results: Of 5543 patients meeting criteria 391 underwent at-least one Duplex scan for suspicion of DVT and DVT was diagnosed in 64 (1.1%). Characteristics and outcomes of patients with and without DVT are presented (Table) and follow known patterns. Of 64 patients with DVT, sonographic characteristics classified 56 (87.5%) as ‘acute’, 6 (9%) as ‘non-acute’ (pre-existing) and 2 (3.5%) as ‘indeterminate’. Among 6 patients with pre-existing DVT, 3 (50%) had no known history of DVT.

Conclusion: Despite small numbers the study demonstrates that even without routine surveillance, up to 10% of the DVTs among high risk traumatized patients maybe pre-existing and up to 50% of these are without a known history. This has significant financial implications.
BASIC SCIENCE LECTURE
Wednesday, 2/28/2018

TRAUMA RESEARCH: TRIALS AND TRIBULATIONS OF A TRICERATOPS
Ernest Moore, MD
SPECIAL SESSION
Wednesday, 2/28/2018

Deborah Kuhls, MD, Paul Chestovich MD, Chris Fisher, MD, Sean Dort, MD
NOTES
Introduction: Mortality prediction aids clinical decision-making and is necessary for quality improvement initiatives. Validated metrics rely on pre-specified variables and often require advanced diagnostics which are unfeasible in resource-constrained contexts. We hypothesize that machine learning will generate superior mortality prediction in both high-income (HIC) and low and middle-income country (LMIC) cohorts.

Methods: SuperLearner (SL), an ensemble machine-learning algorithm, was applied to data from three prospective trauma cohorts: a highest-activation cohort in the United States (US), a high-volume center cohort in South Africa (SA), and a multicenter registry in Cameroon. Cross-validation was used to assess model discrimination of discharge mortality by site using receiver operating characteristic curves. SuperLearner discrimination was compared with standard scoring methods. Clinical variables driving SL prediction at each site were evaluated.

Results: Data from 28,212 injured patients were used to generate prediction. Discharge mortality was 17%, 1.3%, and 1.7% among US, SA, and Cameroonian cohorts. SL delivered superior prediction of discharge mortality in the US (AUC 94-97%) and vastly superior prediction in Cameroon (AUC 90-94%) compared to conventional scoring algorithms. It provided similar prediction to standard scores in the SA cohort (AUC 90-95%). Context-specific variables (partial thromboplastin time in the US and hospital distance in Cameroon) were prime drivers of predicted mortality in their respective cohorts, while severe brain injury predicted mortality across sites.

Conclusion: Machine learning provides excellent discrimination of injury mortality in diverse settings. Unlike traditional scores, data-adaptive methods are well-suited to optimizing precise site-specific prediction regardless of diagnostic capabilities or dataset inclusion allowing for individualized decision-making and expanded access to quality improvement programming.
The History and Tradition of Alpinism in the Western Trauma Association and Climbing the Matterhorn with Your Mentor

G Magee, C Fox
University of Southern California

Presenter: Gregory Magee, MD, MSc
Sponsor: Charles Fox, MD

Introduction: The Western Trauma Association (WTA) was founded with a mission to increase knowledge in the care of trauma patients as well as promote camaraderie with family, friends, and colleagues with a shared love of the winter sports and the mountains. Innumerable lasting friendships have been formed through this meeting in a beautiful mountain setting. This past summer one WTA member, Chuck Fox, joined his graduating fellow Greg Magee for a two-week adventure in the Alps. Their ultimate objectives were to climb the Matterhorn and Mont Blanc. While Mont Blanc is the highest summit in the Alps, no mountain in the world can compare to the iconic face and dramatic history of the Matterhorn. In 1865 the Matterhorn remained as the only unclimbed Alpine peak over 4,000 meters, and it became an international competition to conquer it. On July 14, 1865 on their seventh attempt, a party led by Edward Whymper became the first to reach the summit. However, tragedy ensued on the descent when 4 of the 7 climbers fell to their deaths, forever tarnishing their accomplishment. Their first ascent and tragedy marked the end of the golden age of alpinism. Conversely, the golden age of trauma surgery is just beginning. Our adventure solidified our friendship through toil and blood. Great reward comes from great effort. We encourage all those interested in Alpinism to create a cadre of mentors and mentees to climb together and enjoy the bonds provided by education, training, and mountaineering in a rigorous environment.
MILITARY PANEL
WEDNESDAY, 2/28/2018

Case presenters: Mathew Martin MD and Jennifer Gurney MD

Panelists: Kim Peck MD; David Feliciano MD; Warren Dorlac MD; Racheal Callcut MD
<table>
<thead>
<tr>
<th></th>
<th>Length of stay % longer</th>
<th>Ventilator days % longer</th>
<th>Non-home discharge odds ratio</th>
<th>Readmissions odds ratio</th>
<th>Major Complications odds ratio</th>
<th>Mortality odds ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Opioid-naive</td>
<td>0 (Ref)</td>
<td>0 (Ref)</td>
<td>1 (Ref)</td>
<td>1 (Ref)</td>
<td>1 (Ref)</td>
<td>1 (Ref)</td>
</tr>
<tr>
<td>Opioid-dependent</td>
<td>27.6% (5.9%, 41.5%)**</td>
<td>68.5% (1.2%, 180.6%)</td>
<td>1.47 (1.03, 2.11)**</td>
<td>3.80 (1.14, 12.80)**</td>
<td>3.80 (0.94, 1.88)</td>
<td>1.83 (0.64, 5.27)</td>
</tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prescription abuse</td>
<td>17.0% (-9.2%, 26.7%)</td>
<td>97.7% (-5.4%, 313.6%)</td>
<td>2.94 (1.32, 4.64)**</td>
<td>2.93 (0.91, 5.06)</td>
<td>2.09 (0.43, 4.77)</td>
<td>1.63 (0.63, 4.47)</td>
</tr>
<tr>
<td>Illicit abuse</td>
<td>46.0% (10.9%, 87.9%)**</td>
<td>10.1% (-35.9%, 195.8%)</td>
<td>1.77 (0.91, 3.45)</td>
<td>3.53 (0.66, 3.58)</td>
<td>0.83 (0.40, 1.66)</td>
<td>1.02 (0.10, 9.89)</td>
</tr>
<tr>
<td>Chronic pain</td>
<td>44.9% (-6.0%, 66.9%)</td>
<td>71.3% (-15.9%, 191.6%)</td>
<td>1.00 (0.82, 1.25)</td>
<td>1.86 (1.03, 3.32)**</td>
<td>3.59 (0.83, 1.68)</td>
<td>2.21 (0.38, 13.55)</td>
</tr>
</tbody>
</table>

*P<0.05, **P<0.01

NOTES
Introduction: Increased use of opioids has led to higher rates of overdose and hospital admissions. Studies in trauma populations have focused on outcomes associated with acute intoxication rather than addiction. We hypothesize that clinical outcomes after injury would be different between opioid-naïve and opioid-dependent patients.

Methods: We identified all opioid-dependent (OD) adult patients admitted to an academic level I trauma center in 2016 with an Injury Severity Score (ISS) ≥ 5. Patients were further categorized by their pattern of opioid dependency into Prescription Abuse (PA), Illicit Abuse (IA), or Chronic Pain (CP) subgroups. Outcome measures included length of stay (LOS), major complications, mortality, non-home discharge, ventilator days, and readmissions. Regression models were adjusted for patient demographics, insurance, ISS, and comorbidities.

Results: Of the 1450 patients who met the inclusion criteria, 17.9% were OD. Among OD patients, 30.1%, 27.0%, and 42.9% were PA, IA, CP patients, respectively. Compared to opioid-naïve (ON) patients, OD patients had longer LOS, more ventilator days, more non-home discharges, and higher readmission rates (table). Subgroup analysis revealed significant differences among all cohorts when compared to the ON group in LOS, non-home discharge, readmissions, and major complications (table). OD was not associated with mortality.

Conclusion: Opioid dependency was detected in 17.9% of trauma patients and was independently associated with inferior outcomes. The impact of opioid dependency affects each opioid subgroup differently, with the IA cohort having more in-hospital complications and the PA and CP groups having more post-discharge implications. This knowledge may allow for targeted risk assessments and interventions within trauma centers.
Figure 1. Illicit drug and alcohol use by fiscal year at urban level-1 trauma center.

NOTES
CRISIS UNDER THE RADAR: METHAMPHETAMINE USE IS REACHING EPIDEMIC PROPORTIONS AND CONTRIBUTING TO RESOURCE OVER-UTILIZATION AT A LEVEL 1 TRAUMA CENTER
V Gemma, P Goslar, K Chapple, T Thompson, S Petersen, J Weinberg
St. Joseph’s Hospital and Medical Center

Presenter: Vincent Gemma, MD
Sponsor: Jordan Weinberg, MD

Introduction: It is our perception that illicit amphetamine use (METH) is on the rise, and that these patients require care with length of stay (LOS) beyond what would be expected for their injuries. The purpose of this study was to evaluate the impact of METH use in trauma patients with respect to hospital resource utilization.

Methods: 7-year review of registry of level-1 trauma center in metropolitan center. METH patients were identified from toxicology screening and compared to non-METH patients with respect to intubation, ICU admission, LOS, and cost of care.

Results: From 2011 to 2017, METH use increased threefold, surpassing alcohol (Figure 1). Comparing METH patients (n=1,282) to non-METH patients (n=5,754), METH patients were more likely to be intubated (32% vs 17%,p<.001), admitted to the ICU (55% vs. 41%,p<0.001), and have increased average LOS (5.4 days 4.1,p<0.001) and cost of care ($34,667 $27,668, p<0.001). Modelling determined these differences were particular for patients with minor injuries (ISS < 9): increased odds of intubation (OR 3.0;CI 2.2-3.9;p<0.001), odds of ICU admission (OR 1.5;CI 1.2-1.8;p<0.001), as well as 3.9% increased LOS (p= 0.013) and 4.4% increase in hospital cost (p<0.001) were all independently associated with METH. METH patients were also more likely to be “self-pay” status (33% vs. 21%,p<0.001).

Conclusion: METH use has overtaken alcohol intoxication at a metropolitan level 1 trauma center. Hospital resource utilization of METH patients with minor injuries is significant. Trauma centers with similar epidemic growth in proportion of METH patients face a potentially significant resource strain relative to other centers.
### Percentage of patients with mental health condition and percentage receiving treatment.

<table>
<thead>
<tr>
<th>Follow-Up</th>
<th>Depression</th>
<th>PTSD</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Treatment</td>
<td>Treatment</td>
</tr>
<tr>
<td>1 month</td>
<td>53.3</td>
<td>26.6</td>
</tr>
<tr>
<td>2 months</td>
<td>49.9</td>
<td>27.8</td>
</tr>
<tr>
<td>4 months</td>
<td>49.0</td>
<td>29.8</td>
</tr>
<tr>
<td>12 months</td>
<td>50.2</td>
<td>30.0</td>
</tr>
</tbody>
</table>

### NOTES
PREVALENCE AND TREATMENT OF DEPRESSION AND POSTTRAUMATIC STRESS DISORDER AMONG TRAUMA PATIENTS WITH NON-NEUROLOGICAL INJURIES

B Zarzaur, T Bell
Indiana University School of Medicine

Presenter: Ben Zarzaur, MD, MPH
Sponsor: Ben L Zarzaur, MD, MPH

Introduction: Psychological impairment among injury survivors is well documented. Little is known about the prevalence of treatment of psychological impairment, however. We aimed to determine the proportion of injury survivors treated for depression and PTSD in the year after injury as well as to determine potential barriers to treatment.

Methods: Adults (18 and over) admitted to a Level I trauma center with an injury severity score (ISS) greater than 10, but without traumatic brain injury or spinal cord injury were eligible for study inclusion. The Center for Epidemiological Studies-Depression (CES-D) and PTSD CheckList – Civilian Versions (PCL-C) surveys were administered during the initial hospitalization and repeated at 1, 2, 4, and 12 months after injury. Patients were asked if they received treatment specifically for depression or PTSD at each follow-up. Factors associated with treatment were determined using multivariable logistic regression analysis.

Results: 500 injury survivors were enrolled in this prospective observational study. Of those, 68.4% of patients screened positive for depression at some point in the year after their injury (53.3% 1 month, 49.9% 2 month, 49.0% 4 month, and 50.2% 12 month). Only 22.2% of depressed patients reported receiving treatment for depression. 44.4% of patients screened positive for PTSD (26.6% 1 month, 27.8% 2 month, 29.8% 4 month, and 30.0% 12 month), but only 9.8% received treatment for PTSD. Insurance status was significantly associated with depression treatment (OR 1.81, 95%CI 1.16-2.84; p=0.009).

Conclusion: Depression and PTSD are common in non-neurotrauma patients in the year following injury. Greater collaboration between those caring for injury survivors and behavioral health experts may help improve psychological outcomes after injury.
NOTES
TIME TO STROKE: A WESTERN TRAUMA ASSOCIATION MULTICENTER STUDY OF BLUNT CEREBROVASCULAR INJURIES


Presenter: Clay Cothren Burlew, MD
Sponsor: Clay Cothren Burlew, MD

Introduction: Screening for blunt cerebrovascular injuries (BCVI) in asymptomatic high-risk patients has become routine. To date, the length of this asymptomatic period has not been defined. Determining the time to stroke could impact therapy including earlier initiation of antithrombotics in multiply injured patients. The purpose of this study is to determine the time to stroke in patients with a BCVI-related stroke. We hypothesized the majority of patients suffer stroke between 24-72 hours after injury.

Methods: Patients with a BCVI-related stroke from January 2007-January 2017 from 37 trauma centers were reviewed.

Results: During the 10-year study, 492 patients suffered a BCVI-related stroke; the majority were men (61%) with an average age of 41 and ISS of 31. Stroke was present at admission in 182 (37%) patients and occurred during an IR procedure in 6 patients. In the remaining 304 patients, stroke was identified an average of 73.8 hours after admission; 91.8 hours in the 144 patients identified by neurologic symptoms and 57.8 hours in the 160 patients without a neurologic exam/stroke identified on imaging. Of those patients with neurologic symptoms, 88 stroked within 72 hours while 56 stroked after 72 hours; time intervals to stroke are noted (figure). Of the 308 patients who suffered stroke after admission, 65 (22%) patients were being treated with antithrombotics when the stroke occurred.

Conclusion: The majority of patients suffer BCVI-related stroke in the first 72 hours after injury. Time to stroke can help inform clinicians about initiation of treatment in the multiply injured patient.
NOTES
PRO/CON DEBATE
Thursday, 3/1/2018

REBOA IS AN ESTABLISHED TOOL AND SHOULD BE AVAILABLE IN EVERY SURGEONS’ ARMAMENTARIUM

Pro: Megan Brenner; Con: Gary Vercruysse
ALGORITHM #2  
Thursday, 3/1/2018

CERVICAL SPINE CLEARANCE

David Ciesla, MD

Pre-Hospital C-Collar

Normal Exam

Patient Examinable

CT C-Spine

Normal Neck CT

Abnormal Neck CT

C-Spine Fx

MRI C-Spine

NO Ligament Injury

Ligament Injury

Remove Collar

Spine Consultation
Table: Post Matching Outcomes.

<table>
<thead>
<tr>
<th></th>
<th>Non-Transfers (N=1735)</th>
<th>Transfers (N=1735)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mortality</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>% (count)</td>
<td>2.7% (45)</td>
<td>4.3% (72)</td>
<td>0.009*</td>
</tr>
<tr>
<td><strong>Time from injury to destination hospital</strong> (minutes)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (25th percentile, 75th percentile)</td>
<td>96.0;240.0;366.0</td>
<td>223.0;291.0;389.0</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td><strong>Time in Level I/II ED (minutes)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (25th percentile, 75th percentile)</td>
<td>149.0;208.0;284.0</td>
<td>133.0;195.5;273.0</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td><strong>Level III ED Disposition % (Count)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Home</td>
<td>16.9% (292)</td>
<td>19.0% (325)</td>
<td>0.107</td>
</tr>
<tr>
<td>ICU</td>
<td>23.6% (409)</td>
<td>24.1% (414)</td>
<td>0.639</td>
</tr>
<tr>
<td>OR</td>
<td>12.1% (210)</td>
<td>11.7% (200)</td>
<td>0.593</td>
</tr>
<tr>
<td><strong>Intervention % (Count)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Intubation</td>
<td>7.7% (134)</td>
<td>9.7% (158)</td>
<td>0.039*</td>
</tr>
<tr>
<td>Transfusion</td>
<td>4.0% (70)</td>
<td>3.1% (57)</td>
<td>0.167</td>
</tr>
<tr>
<td>Chest tube placement</td>
<td>1.3% (23)</td>
<td>1.2% (20)</td>
<td>0.622</td>
</tr>
<tr>
<td><strong>LOS Hospital (days)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Median (25th percentile, 75th percentile)</td>
<td>1;2.5</td>
<td>1;2.4</td>
<td>0.290</td>
</tr>
</tbody>
</table>

* P-values less than or equal to 0.05 indicate
Paper #27
Thursday, 3/1/2018

RISK OF INCREASED MORTALITY ASSOCIATED WITH STOPPING AT LEVEL III AND IV TRAUMA CENTERS: A PROPENSITY SCORE MATCHED STUDY
P Bonasso, L Gurien, M Saylors, A Porter, M Rettiganti, D Tuggle, R Maxson
University of Arkansas for Medical Sciences

Presenter: Patrick Bonasso, MD, MBA
Sponsor: David Tuggle, MD

Introduction: Triage to the appropriate level trauma center is critical to a trauma system. The purpose of this study was to evaluate outcomes between those transported directly to a Level I&II trauma center versus those transferred from a Level III&IV center.

Methods: Patients from a single state trauma registry (2012-2015) were divided into non-transfer and transfer groups. The non-transfer group presented directly to a Level I&II trauma center. The transfer group was initially seen at a Level III&IV trauma center and transferred to a Level I&II trauma center. Propensity score matching matched patients 1:1 on age, race, gender, injury type, mechanism, transport mode, trauma team activation, hypotension, and GCS. Outcomes post-matching were compared using the signed-rank test or McNemar-Bowker tests as appropriate. P-values < 0.05 were considered statistically significant.

Results: 1,735 patients were matched. Mortality was higher among transfers (4.3% vs. 2.7%; p= 0.009). Transfers had a longer median time from injury to destination hospital (291 vs. 240 minutes, p<.001) and a shorter median length of stay in the level I&II ED (195 vs. 208 minutes; p<0.001) (table). There was no difference hospital stay, proportion discharged home directly from ED, transferred to ICU or ED.

Conclusion: This study illustrates an increased risk of mortality for patients transported to Level III&IV trauma centers and may help modify patient triage in a rural statewide trauma system.
NEEDS-BASED ASSESSMENT OF TRAUMA SYSTEMS: A SURVEY OF THE MEMBERSHIP OF THE WESTERN TRAUMA ASSOCIATION
RS Smith, D Ciesla, N Namias, S Brakenridge, FA Moore
University of Florida

Presenter: R Stephen Smith, MD
Sponsor: R Stephen Smith, MD

Introduction: Proliferation of trauma centers (TC) threaten several trauma systems (TS). The ACSCOT published a Needs-Based Assessment of Trauma Systems (NBATS) tool to assist TS in determining the number of TCs needed. Acceptance of NBATS is limited and some believe that NBATS criteria are not appropriate. WTA members are considered experts in trauma care and TS.

Methods: A survey instrument, based on NBATS, was made available to members of the WTA. If an item received 75% support (agree, strongly agree), it was considered to be a consensus opinion. If 51% agreed or strongly agreed, the item was judged to have majority support.

Results: 167 members (71%) responded. 9 items received consensus support: (1) an assessment tool is needed (2) TC designation should be based on need (3) population needs should be held above stakeholder interests (4) justification should be mandatory before TC designation (5-6) too many or too few TCs adversely affect care (7) distance between TCs should be considered (8) academic level 1 TCs should be preserved (9) the minimum TSA for a Level 2 TC is 600,000. 2 questions received majority support: (1) defer designation of a new TC if a TC is present in a TSA of 1.5 million (2) a new TC should decrease transport time by 15 minutes.

Conclusion: There is broad support for an assessment tool, but the composition is controversial. WTA opinions should be considered in future assessment tools. The WTA should write a position paper to assist TS in determining appropriate number of TCs.
Paper #29 — CASE REPORT
Thursday, 3/1/2018

INNOVATIVE SURGICAL TREATMENTS AND ADVANCED PROSTHETICS FOR UPPER EXTREMITY AMPUTEE: A CASE REPORT
A Chi, R Armiger, C Moran, R McGown, M Schreiber

Presenter: Albert Chi, MD
Sponsor: Susan Rowell, MD

Introduction: This case report presents the first patient who has undergone targeted muscle reinnervation (TMR) and an osseointegration (OI) implant (Compress implant, Zimmer Biomet) in the US following a transhumeral amputation. The patient was fitted with the Modular Prosthetic limb (MPL), the world’s most advanced limb system.

Methods: The MPL was held onto the implant with a custom mechanical interface. The interface provided an adjustable, instrumented friction clutch that collected real-time values for axial torque at the implant interface. Two Myo armbands (Thalmic Labs) streamed a combined 16 channels of raw EMG at 300 Hz over a Bluetooth connection to a controlling laptop producing a fully wireless control interface. On the laptop the data was processed using custom pattern recognition software. In the first multi-day OI fitting and use session, the subjects trained at tasks such as pick and place of objects on overhead shelves and self-care simulations.

Results: On the first of four days, the participant achieved an average classification accuracy of 95.3 % for 6 motion classes plus no movement and 92.4% accuracy with a total of 7 motions plus no movement. On the fourth day, two new motions were added, wrist flexion and extension, for a total of 9 motions and a classification accuracy of 89%. The subject also placed a baseball cap on his head and successfully picked up and placed objects on an overhead shelf.

Conclusion: The MPL system is a safe and improved system over currently available prosthetic upper extremity for individuals who have undergone TMR and OI surgery.
**ICP MANAGEMENT**

*Hasan Alam, MD*

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**Suspected Severe Traumatic Brain Injury (TBI)**

CT Scan and Clinical Evaluation

- **Primary Decompressive Craniectomy**
  - Y: Indications for Primary Decompressive Craniectomy
  - N: Supportive Care & Avoidance of Secondary Brain Injury

Continue Supportive Care and Avoidance of Secondary Brain Injury

- Y: ICP Adequately Controlled (<22 mmHg)
  - N: Repeat CT scan in 6-8 hrs

- Y: Place ICP monitor + Supportive Care and Avoidance of Secondary Brain Injury

Tier 1 Interventions

Continue Tier 1 Interventions

Tier 2 Interventions

- Y: ICP Adequately Controlled
  - N: Repeat CT Scan Based Upon Clinical Judgement

Consider Decompressive Cranietomy

Expand Intracranial Lesion with Mass Effect

Tier 3 Interventions

- Y: ICP Adequately Controlled
  - N: Rescue Interventions

Continue Tier 1-3 Interventions

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48TH ANNUAL WESTERN TRAUMA ASSOCIATION MEETING
PAINT THE CEILING LECTURE
Thursday, 3/1/2018

AGAINST EMPATHY
Steven R, Shackford MD FACS
University of Vermont College of Medicine
NOTES
THE WHY & HOW OUR TRAUMA PATIENTS DIE: A PROSPECTIVE WESTERN TRAUMA ASSOCIATION MULTICENTER TRIALS COMMITTEE STUDY

UCSF/San Francisco General Hospital

Presenter: Rachael Callcut, MD, MSPH
Sponsor: Mitchell J Cohen, MD

Introduction: Historically, hemorrhage has been identified as the leading cause (40%) of early deaths; however, rigorous real-time classification of the cause of death (COD) has been lacking. This study sought to prospectively adjudicate COD.

Methods: 18 centers prospectively enrolled all adult trauma patients at the time of death during 12/2015-8/2017. Immediately following death, attending providers adjudicated the primary and contributing secondary COD using standardized definitions. Data were confirmed by autopsies, if performed.

Results: 1536 patients were enrolled (median age 55, IQR 32-75; 74.5% male). Penetrating mechanism (n=412) patients were younger (32 vs 64, p<0.0001) and more likely male (86.7% vs. 69.9%, p<0.0001). Falls were the most common mechanism (26.6%) with GSWs second (24.3%). The most common overall primary COD was TBI (45%) followed by exsanguination (23%). TBI was non-survivable in 82.2% of cases. Primary COD in blunt patients was more commonly TBI (47.8% vs. 37.4%, p<0.0001) and exsanguination for penetrating patients (51.7% vs 12.5%, p<0.0001). Exsanguination was the predominant prehospital (44.7%) and early COD (39.1%) with TBI most common later. Penetrating mechanism patients died earlier (80.1% on day 0 vs. 38.5%, p<0.0001). Most deaths were deemed disease related (69.3%), rather than from limitation of further care (30.7%). Hemorrhage was a contributing cause to 38.8% of deaths that occurred due to withdrawal of care.

Conclusion: Exsanguination remains the predominant early primary COD with TBI dominating later deaths. Timing and primary COD vary significantly by mechanism. Contemporaneous adjudication of COD is essential to elucidate the true understanding of patient outcome, center performance, and future research.
NOTES
THE IMPACT OF IN-HOSPITAL PALLIATIVE CARE CONSULTATION ON HEALTHCARE AT THE END OF LIFE FOR OLDER TRAUMA PATIENTS WHO DIE AFTER DISCHARGE

E Lilley, J Scott, N Krumrei, A Haider, A Salim, R Gupta, Z Cooper
Rutgers Robert Wood Johnson Medical School

Presenter: Elizabeth Lilley, MD, MPH
Sponsor: Rajan Gupta, MD, MHCDS

Introduction: Elderly trauma patients have substantial post-discharge morbidity and mortality, and many who die experience high healthcare utilization at the end-of-life. Inpatient palliative care consultation (PCC) has been shown to improve post-discharge end-of-life care in non-trauma populations, but its role in trauma remains poorly defined. We hypothesized that PCC would similarly be associated with less aggressive end-of-life care for elderly trauma decedents.

Methods: A retrospective review of Medicare beneficiaries ≥65 years including those who died ≤180 days after at least moderate trauma (ICD-9-CM 800.00-801.9, 803.0-804.9, 850.0-854.1, 959.01 and ISS ≥9). Patients who received PCC during the trauma admission were matched with non-PCC controls using an exact matching algorithm for ISS, survival duration, age, sex, race, and comorbidities. Post-discharge outcomes included readmission, long-term care facility admission, death in an institution, and ICU stay and life-sustaining treatments (LST) during a hospital readmission.

Results: There were 294,665 patients who died ≤180 days after trauma: 6,188 (2.1%) received PCC and 92% of these (n=5,674) were exact-matched with non-PCC controls. PCC was associated with reduced odds of readmission, admission to a long-term care facility, and death in an institutional setting (Figure). For those with hospital readmissions (n = 1,158), PCC was associated with fewer ICU stays (27% vs. 41%, OR 0.53 [0.37-0.74]) and less LST (24% vs. 35%, OR 0.58 [0.41-0.83]).

Conclusion: Inpatient PCC for older patients with moderate to severe trauma was associated with reduced post-discharge healthcare utilization at the end of life. These data support the benefits of palliative care consultation and highlight its underutilization in trauma.
Table 1. Total and new diagnoses of HIV and Hepatitis C amongst trauma patients and initiation of disease follow-up.

<table>
<thead>
<tr>
<th></th>
<th>Total Disease Positive (prior &amp; new diagnosis)</th>
<th>Total Disease Positive Follow-up</th>
<th>New Diagnosis</th>
<th>New Diagnosis Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hepatitis C</td>
<td>54 (6.4%)</td>
<td>77.6%</td>
<td>37 (3.0%)</td>
<td>77.1%</td>
</tr>
<tr>
<td>HIV</td>
<td>16 (1.3%)</td>
<td>99.8%</td>
<td>3 (0.7%)</td>
<td>100%</td>
</tr>
<tr>
<td>Total</td>
<td>70 (5.7%)</td>
<td>81.5%</td>
<td>40 (3.3%)</td>
<td>75.9%</td>
</tr>
</tbody>
</table>

NOTES
A DIFFERENT FORM OF INJURY PREVENTION: SUCCESSFUL SCREENING AND REFERRAL FOR HIV AND HEPATITIS C IN A TRAUMA POPULATION

B White, L Richey, P Ferguson, ED Norcross, A Privette
Medical University of South Carolina

Presenter: Benjamin White, MD
Sponsor: Mitchell Cohen, MD

Introduction: Nationally, millions of people are living with HIV and/or Hepatitis C (0.44% and 1.5%) and many remain undiagnosed. Since highly effective treatments are now available, patient identification is paramount. A single prospective study (1992) demonstrated a greater prevalence of both Hepatitis C and HIV in the trauma population. We hypothesized that a screening program could be applied to trauma activations and that they could be successfully referred for care.

Methods: Hepatitis C and HIV screening tests were added to trauma activation order sets at an academic Level 1 Trauma Center. Confirmatory viral load was sent when indicated. Patients with positive results were referred to disease-specific follow-up. Data was collected prospectively from January 1, 2016 until June 30, 2017. Total and new diagnoses and follow-up referral rates were analyzed.

Results: 1221 patients screened (64.1%): Level A = 75.6%, Level B = 60.2%. 5.7% of all patients screened positive. Rates of both HIV and Hepatitis C were triple the national average. 4.4% screened positive for a new diagnosis. The newly diagnosed Hepatitis C rate was twice the national average. Approximately 80% of all cases were referred for follow-up. (Table)

Conclusion: HIV and Hepatitis C are frequently newly diagnosed in the trauma population. The majority of activations were successfully screened and referred for follow-up. Routine screening of trauma patients should be considered to increase diagnosis rates and intervention programs should be implemented to increase referral for medical management. These efforts would decrease disease transmission to health care providers and within the community as a whole.
INABILITY TO PREDICT SUB-PROPHYLACTIC ANTI-FACTOR XA LEVELS IN TRAUMA PATIENTS RECEIVING EARLY LOW-MOLECULAR-WEIGHT HEPARIN

J Imran, T Madni, A Clark, L Taveras, P Rizk, E Huang, A Christie, A Eastman, M Cripps
University of Texas Southwestern Medical Center

Presenter: Michael Cripps, MD
Sponsor: Alexander L. Eastman, MD, MPH

Introduction: Standard low-molecular-weight heparin (LMWH) dosing may be suboptimal for venous thromboembolism (VTE) prophylaxis. We aimed to identify independent predictors of sub-prophylactic Xa (subXa) levels in trauma patients treated under a novel early chemoprophylaxis algorithm for high VTE/low bleeding risk trauma patients.

Methods: A retrospective analysis of trauma patients from July 2016-June 2017 who received enoxaparin 40 mg BID and had peak Xa levels drawn was performed. Cohorts were divided based on having a subXa (< 0.2 IU/mL) or prophylactic (≥ 0.2 IU/mL) Xa level.

Results: 124 patients were included, of which 38 (31%) had subXa levels and 17 (14%) had Xa levels > 0.4 IU/mL. Of the subXa cohort, 35 (92%) had their dosage increased and the repeat Xa testing that was done in 32 revealed that only 75% reached prophylactic levels. The overall median time to initiation of chemoprophylaxis was 21.9 hours [11.45 – 35.07]. Patients with lower risk for bleeding (no ICH/spine fracture) had shorter time to starting prophylaxis than those at higher risk (18.39 [5.76 – 26.51] vs. 29.5 hours [16.23 – 63.07], p < 0.01). There was no difference in demographics, weight, BMI, creatinine, creatinine clearance, ISS, type of injury, weight-based dose, time to chemoprophylaxis or bleeding complications between the cohorts. Four DVTs and 2 PEs occurred in each group. No independent predictors of a subXa level were identified on multivariable logistic regression.

Conclusion: A significant number of trauma patients fail to achieve prophylactic Xa levels. Intrinsic factors may prevent adequate prophylaxis even with earlier administration and higher LMWH dosing.
<table>
<thead>
<tr>
<th></th>
<th>Rivaroxaban (n=1053)</th>
<th>Enoxaparin (n=1053)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>51 (32-63)</td>
<td>49 (30-63)</td>
<td>0.701</td>
</tr>
<tr>
<td>Female, n(%)</td>
<td>500 (47)</td>
<td>429 (41)</td>
<td>0.002</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>170 (163-180)</td>
<td>173 (163-180)</td>
<td>0.388</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>84 (70-100)</td>
<td>81.8 (70-100)</td>
<td>0.517</td>
</tr>
<tr>
<td>Blunt mechanism of injury, n(%)</td>
<td>1011 (96)</td>
<td>999 (95)</td>
<td>0.210</td>
</tr>
<tr>
<td>Injury severity score</td>
<td>9 (5-10)</td>
<td>8 (5-10)</td>
<td>0.691</td>
</tr>
<tr>
<td>Time to first dose (hours)</td>
<td>28.5 (18.6-34.1)</td>
<td>20.3 (10.4-34.2)</td>
<td>0.002</td>
</tr>
<tr>
<td>SCDs ordered, n(%)</td>
<td>963 (91.4)</td>
<td>798 (75.7)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Drug dosing in mg/kg</td>
<td>n/a*</td>
<td>0.39 (0.32-0.48)</td>
<td>n/a</td>
</tr>
<tr>
<td>Total drug dose per day (mg)</td>
<td>n/a*</td>
<td>60 (40-60)</td>
<td>n/a</td>
</tr>
</tbody>
</table>

NOTES
Introduction: Venous thromboembolism (VTE) often complicates trauma, despite twice daily doses of enoxaparin or equivalents. In some groups, Rivaroxaban has been preferentially recommended over Enoxaperin for routine VTE prophylaxis. There is no evidence, however, demonstrating Rivaroxaban’s effectiveness compared to more commonly used agents. The purpose of this study was to compare the safety and efficacy of Rivaroxaban to Enoxaparin for prevention of VTE in injured patients.

Methods: This retrospective cohort analysis evaluated the rate of VTE in adult patients at a Level 1 Trauma center from 2013-16. Patients receiving prophylactic Rivaroxaban were matched to patients receiving Enoxaparin using propensity score matching. Patients were evaluated for a primary outcome of VTE within 6 months of admission, with secondary outcomes of bleeding, hospital mortality and length of stay.

Results: 5,435 patients met criteria for the study. 1,053 patients were in each group. Table 1 demonstrates baseline characteristics. Overall incidence of VTE was 1.3%, with no significant difference between groups (14 VTE in each group, p =1.0). There was no difference in the incidence of deep venous thrombosis (0.9% in Rivaroxaban (n=10) and 1.1% in Enoxaperin (n=12, p=0.529)) or pulmonary embolism (n=6, 0.6% versus n=2, 0.2% respectively; p=0.157). Incidence of major and minor bleeding episodes was equivalent between groups (p>0.05).

Conclusion: Rivaroxaban demonstrated equivalent protective effect for VTE and no increase in bleeding complications. Administered orally, Rivaroxaban compliance by patients may be superior. Rivaroxaban appears to be a safe and effective alternative for VTE prophylaxis in this high risk population.
Rib fractures affect ~10% of blunt trauma patients resulting in significant morbidity and mortality. Recent advances in operative techniques facilitate the restoration of the mechanical integrity of the chest wall reducing pain, improving respiratory function, and decreasing length of stay. However, accurate intraoperative localization of rib fractures remains a challenge. Case Presentation: A 57-year-old male presented on transfer after an industrial accident with fractures of right ribs 2-11 with significant rib displacement, concern for diaphragm injury, and associated hemothorax. A non-contrast CT chest was obtained prior to intervention for better characterization of his injury. Concurrently, a biopsy grid was placed over the known area of injury. Utilizing standard CT biopsy techniques, each fracture was marked while the patient was in the gantry. Intraoperatively, the corresponding marks were used to identify the rib fractures, which allowed for more accurate localization, a smaller incision, and a less extensive dissection. Discussion: CT biopsy grids are used by radiologists to improve localization during image-guided procedures. CT scans prior to operative fixation of the ribs are common, and the addition of a biopsy grid provides accurate localization with no added risk to the patient. Lack of reliable corresponding external landmarks and varying levels of injury renders the initial CT alone an imprecise modality for localization. We hypothesize that this novel approach utilizing CT biopsy grids for rib fracture localization will increase localization accuracy resulting in smaller incision size, more limited dissection, decreased operative time, and thus decreased morbidity.
ABDOMINAL GSW
Matthew Martin, MD

ABDOMINAL GSW

Exploratory Laparotomy or immed transfer to trauma center

- Y -

Hemodynam unstable
Peritonitis, Evisceration
Hematoma, Gross blood per rectum
Austere/resource limited environment

N

Complete external survey
Rapid bedside imaging (may include):
1. upright CXR
2. FAST exam or FAST exam
3. ab/pelvic x-rays

- Y -

Intrapertitoneal free air
Large volume, multiquadrant free fluid
Pericardial effusion

N

Reliable abdominal examination?
(awake, no severe intoxication, follows
commands, cooperative with exam)

Y

Choose management pathway +
additional anatomic considerations
based on injury location (see inset box)
& estimated trajectory

A. Diagnostic Laparoscopy

Peritoneal penetration or visualized
Intra-abdominal injury?

- Y -

Abdominal exploration

- N -

Observe or discharge

B. Selective Nonoperative Management

Operative injury identified or
suggested?

- Y -

CT Scan

- N -

Abdominal exploration

Serial clinical exams

Worsening exam, vitals, labs?

- Y -

Observe or discharge

- N -
THE IMPACT OF ACS LEVEL I PEDIATRIC TRAUMA CENTERS ON ADOLESCENT MOTOR VEHICLE FATALITIES, 1999-2015

DM Notrica, LW Sayrs
Phoenix Childrens Hospital

Presenter: David Notrica, MD
Sponsor: David Notrica, MD

Introduction: Motor vehicle crashes (MVCs) are a leading cause of adolescent death from trauma. A recent study found that ACS verified pediatric trauma centers (vPTC) were inversely correlated with pediatric mortality. We assess the contribution of verified Level I trauma centers (adult vs. pediatric) and changes in state laws with respect to graduated drivers’ licenses (GDLs) on trends in adolescent MVC mortality for all 50 states from 1999-2015.

Methods: Retrospective data on adolescent motor vehicle fatalities, state driving laws and vPTCs was collected from publically available sources for all 50 U.S. states from 1999-2015. A mixed fixed/random effects multivariate regression model was fitted to assess the relative contribution of GDL and vPTCs (adult vs. pediatric), controlling for state effects and changing variables over time.

Results: The final regression model included pediatric trauma center, GDL, state and year; model fit predicting to fatalities per 100,000 was strong (Adj. R-square=.84). Both GDL [B=1.51; p<.001] and ACS pediatric level I verified [B=1.25; p<.001] contributed to declines in teen fatalities. ACS vATCs were not significant. Fatalities declined 1-2 per 100,000 for each additional verified pediatric Level I trauma center and for each state with GDL. There were significantly stronger effects for GDL after 2007 [B=8.38; p<.001] while the effects from ACS verification remained constant over the time period.

Conclusion: GDLs were associated with reduced rates of adolescent motor vehicle fatalities in the last 2 decades independent of changes in state laws (GDL). The effect was independent of the protective effect of vPTCs.
Introduction: Trauma outreach aimed at pediatric trauma prevention is rarely directed to children. It routine targets parents and is intended to help by telling children to “wear your seatbelt” or “don’t drink and drive,” without explaining the reasoning behind these demands. Fifth grade students are often taught basic human body systems, which provides an opportunity to teach injury prevention efforts directly to children. The objective of this project is to describe a technique for pediatric outreach within the elementary school system.

Methods: Two schools are visited each month. Two 105-minute lectures are performed each day. A moulaged trauma man simulator and porcine dermis sheets are used in the simulation. Expired/unused OR supplies are donated for use at the schools. Consent is obtained from the parents prior to children participating.

Results: 80 schools were visited over 5 years. Each school had 2-4 fifth grade classrooms with 24-32 children per class. The age range was 10-12 years old with equal females vs. males. A total of 7126 children took part. Verbal reports from teachers, parents, and students suggest excellent retention of the materials as far as 5 years out. Unexpected outcomes include improvement in grades, improvement in communication, and 6 child abuse cases were identified.

Conclusion: Despite the mature content, engaging this age group in serious, interactive discussions with a hands-on experience regarding traumatic injuries is an effective way to deter children from making poor choices, to bring awareness to the ramifications of trauma, and to improve school performance.
### TABLE. BIVARIATE OUTCOMES

<table>
<thead>
<tr>
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<th>DPS (n=40)</th>
<th>None (n=61)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Mortality</strong></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Died</td>
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<td>9</td>
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<tr>
<td>Lived</td>
<td>39</td>
<td>52</td>
<td></td>
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<tr>
<td><strong>Hospital LOS</strong></td>
<td>43 ± 24</td>
<td>65 ± 61</td>
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<tr>
<td><strong>ICU LOS</strong></td>
<td>31 ± 14</td>
<td>39 ± 24</td>
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<td><strong>Ventilator Days</strong></td>
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<td>32 ± 24</td>
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<td><strong>Pneumonia</strong></td>
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<tr>
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<td>26</td>
<td>39</td>
<td>0.91</td>
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<tr>
<td>No</td>
<td>14</td>
<td>22</td>
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</tr>
</tbody>
</table>

### NOTES
UTILITY OF DIAPHRAGM PACING IN THE MANAGEMENT OF ACUTE CERVICAL SPINAL CORD INJURY
A Kerwin, B Yorkgitis, D Ebler, F Madbak, A Hsu, M Crandall
University of Florida College of Medicine-Jacksonville

Presenter: Andrew Kerwin, MD
Sponsor: Fred Moore, MD

Introduction: Cervical spinal cord injury (CSCI) is devastating. Respiratory failure, ventilator associated pneumonia (VAP), sepsis, and death frequently occur. Case reports of diaphragm pacing (DPS) have suggested earlier liberation from mechanical ventilation in acute CSCI patients. We hypothesized DPS implantation would decrease VAP and facilitate liberation from ventilation.

Methods: We performed a retrospective review of acute CSCI patients managed at a single level 1 trauma center between 1/2005-5/2017. Routine demographics were collected. Patients underwent propensity matching based on age, ISS, ventilator days, hospital length of stay and need for tracheostomy. Outcome measures included hospital length of stay (HLOS), ICU length of stay (ICU LOS), ventilator days (vent days), incidence of VAP, and mortality. Bivariate and multivariate logistic and linear regression statistics were performed using STATA v10.

Results: Between 7/2011-5/2017 all acute CSCI patients were evaluated for DPS implantation. 40 patients that had laparoscopic DPS implantation (DPS) were matched to 61 who did not (NO DPS). Median time to liberation after DPS implantation was 7 days. HLOS and mortality were significantly lower on bivariate analysis in DPS patients. DPS placement was not found to be associated with statistically significant differences in these outcomes on risk-adjusted multivariate models that included admission year.

Conclusion: DPS implantation in acute CSCI patients can be one part of a comprehensive critical care program to improve outcomes however, the association of DPS with the marked improved mortality seen on bivariate analysis may be due solely to improvements in critical care. Further studies to define the benefits of DPS implantation are needed.
NOTES
A SUBSET OF FIVE HUMAN MITOCHONDRIAL FORMYL PEPTIDES MIMIC BACTERIAL PEPTIDES AND FUNCTIONALLY DEACTIVATE HUMAN NEUTROPHILS (PMN)
E Kaczmarek, CJ Hauser, WY Kwon, L Chen, LE Otterbein, N Sandler, CH Cook, M Marusich, K Itagaki
Beth Israel Deaconess Medical Center

Presenter: Kiyoshi Itagaki, PhD
Sponsor: Carl J. Hauser, MD

Introduction: Trauma causes inflammation by releasing mitochondria (MT) that act as DAMPs. Trauma also causes susceptibility to infection. Human MT contain 13 N-formyl peptides (mtFPs). We studied whether these human mtFPs induce inflammatory responses equally, whether their potency reflects molecular conformation and whether they can suppress PMN function.

Methods: N-terminal sequences of the 13 mtFP were synthesized. Volunteer neutrophils (PMN) were used to assay cell calcium ([Ca2+]i) and chemotactic responses to mtFPs using fura-2AM and transwells. Conformation of N-terminal epitopes was studied using the BLOSUM62 bioinformatics system using the canonical bacterial peptide f-Met-Leu-Phe (fMLP) as comparator.

Results: Only five mtFP (ND6, ND3, ND4, ND5, and COX1) induced [Ca2+]i flux and chemotaxis in descending order of potency. Chemoattractant potency was linearly related to [Ca2+]i flux (R2= 0.96). Molecular similarity to fMLP linearly predicted both [Ca2+]i flux (R2=0.97) and chemotactic potency (R2=0.99). All active mtFPs suppress PMN responses to the physiologic pulmonary chemoattractants GRO-α and LTB4 but prior FPR-1 inhibition by Cyclosporin H (FIG.) prevented the heterologous suppression of PMN chemotaxis by mtFPs.

Conclusion: A subgroup of five mtFPs is responsible for PMN attraction and [Ca2+]i flux activation by MT DAMPs. Molecular modeling shows that their potency reflects their similarity to bacterial f-peptides. We hypothesize that the variable potencies of mtFP DAMPs may predict their clinical relevance. Inhibition of specific mtFP might modulate inflammation or preserve PMN responses to infection after MT release by tissue trauma. These findings suggest new paradigms for prevention of both inflammation and infection after tissue trauma.
NOTES
Paper #40
Friday, 3/2/2018

EARLY ENTERAL NUTRITION IMPROVES MORBIDITY IN CRITICALLY INJURED ADULT MALES DESPITE LOWER SARCOPENIA INDEX SCORES
JL Hartwell, JK Kays, M Li M, L Timsina, BL Zarzaur
Indiana University

Presenter: Jennifer Hartwell, MD
Sponsor: Jennifer L. Hartwell, MD

Introduction: Sarcopenia, the loss of skeletal muscle mass and strength, correlates strongly with disability and death, known primarily from research with elderly and cancer patients. In our prior work, we described that critically injured patients who resolve a protein debt within four days of hospital admission have fewer complications. We hypothesized that critically ill trauma patients with early adequate protein delivery will have fewer complications, despite sarcopenia on admission.

Methods: sliceOmatic CT scan software was used to measure all muscle volume at the L3 level in 162 trauma patients admitted to the ICU in an urban Level 1 trauma center over a one-year period. The Sarcopenia Index (SI) was calculated and relationships between the patients’ SI and age, sex, ISS, morbidity, mortality and nutrition delivery were analyzed using descriptive, bivariate and multivariate analysis.

Results: For each one-year increase in age, the SI decreased by 0.25 units (p<0.001) in males and by 0.33 units (p<0.001) in females. For each unit increase in SI, males had a 10% decreased 30-day mortality (OR: 0.90; 95% CI: 0.82-0.99; p=0.030). Despite having SI 6.47 units lower than the comparison group, males who close their protein debt within four days remain in a group with fewer complications than the group who fails to sustain protein recovery over the first week of critical care admission (p=0.003).

Conclusion: Early, adequate protein delivery in critically injured adult males places them at lower risk for complications despite a worse SI than the comparison group. Increasing SI reduces the risk of death after trauma.
INJURY PATTERNS AND SUBSEQUENT INTERVENTION OF HANGING MECHANISM PATIENTS: HOW MUCH WORK-UP IS NEEDED?
DM Berke, J Reyes, SD Helmer, JM Haan
The University of Kansas School of Medicine - Wichita

Presenter: David Berke, MD
Sponsor: James Haan, MD

Introduction: Survivors of near-hangings suffer sequelae of anoxic brain injuries, but it remains uncertain whether the incidence of associated injuries warrants extensive work-up or trauma team activation. This study examined injuries identified and interventions performed on this population to assess the need for trauma team activation.

Methods: A retrospective chart review was conducted on patients (age ≥ 18), excluding prisoners, who presented to an ACS-verified level 1 trauma center with hanging mechanism from January 1, 2005 through December 31, 2015, and underwent trauma team work-up and management. Data collection included demographics, injury characteristics, interventions, hospitalization details, and outcomes.

Results: A total of 118 patients met inclusion criteria. The majority were white (88.1%) and male (76.3%) with an average age of 30.6 ± 13.3 years. A total of 262 imaging studies were performed and uncovered 11 injuries: 5 thyroid cartilage/hyoid fractures; 4 vertebral column injuries; and 2 cervical vascular injuries. None of these injuries required procedural intervention or contributed to mortality. Anoxic brain injury was diagnosed clinically in 55 patients (46.6%) and was present in all 39 patients (33.1%) who died. Seventy-nine patients (66.9%) survived to discharge with resuscitation and airway management. Brief mechanical ventilation was required in fifty-four patients (45.8%), but none required emergent surgical airway. Only one patient had an intra-abdominal injury requiring surgery by the trauma team.

Conclusion: Patients who present with hanging mechanism have a low incidence of associated injuries. Work-up can be restricted to patients with specific symptoms with trauma team activation for signs of trauma.
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