



44th ANNUAL MEETING
March 2-7, 2014
Steamboat Springs, Colorado

WTA FUTURE MEETINGS

March 1-6, 2015
Telluride, Colorado

Direct all correspondence to:

Western Trauma Association
1741 Newnan Crossings Blvd E
Ste O, #204
Newnan, GA 30265
Phone: 317.538.1953
Fax: 678.668.7312
Email: info@westerntrauma.org
www.westerntrauma.org

ACCREDITATION STATEMENT

This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the American College of Surgeons and the 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 18 *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 13.5 credits meet the requirements for Self-Assessment.



American College of Surgeons
Division of Education

SPEAKER DISCLOSURE INFORMATION

In compliance with 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. All reported conflicts are managed by a designated official to ensure a bias-free presentation. Please see the insert to this program for the complete disclosure list.

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.

LEARNING OBJECTIVES

This activity is designed for physicians of all specialties who are involved in the care of trauma patients. Meeting attendees will hear a wide range of scientific information covering the topics discussed here plus many others. Using that information, attendees can make appropriate adjustments to their own, and their institutions' current practices to align new scientific data. Upon completion of this course, attendees will be able to better understand:

1. What new studies topics are being researched
2. The pathophysiology after trauma
3. The pros and cons of damage control surgery
4. When to use radiographic studies to diagnose and manage the patients
5. The pros and cons of ordering too many tests
6. How to systematically manage penetrating chest trauma
7. How to better manage duodenal injuries
8. How to improve lifestyle and professional career by incorporating family into their life
9. Hemorrhagic shock and its understanding of basic pathways that contribute to pathophysiology

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2013-2014 OFFICERS & COMMITTEE CHAIRS

Officers

President	David H. Livingston, MD
President-Elect	Christine S. Cocanour, MD
Vice President	Thomas Scalea, MD
Secretary	Carl J. Hauser, MD
Treasurer	Dennis W. Vane, MD
Historian	Harold F. Sherman, MD
Immediate Past President	Mark T. Metzdorff, MD

Board of Directors

Karen J. Brasel, MD
Michaela A West, MD, PhD
Alicia J. Mangram, MD
Richard S. Miller, MD
R. Lawrence Reed, MD
Hasan B. Alam, MD
Mark T. Metzdorff, MD
Martin A. Schreiber, MD

Term Ends

2014
2014
2015
2015
2015
2016
2016
2016

Program Committee

Peter Rhee, MD, *Chair*

Publications Committee (Term Ends)

Ajai Malhotra, MD, *Chair* (2014)

Nominating Committee

Mark Metzdorff, MD, *Chair*

Multi-Center Trials Committee

Rosemary A. Kozar, MD, *Chair*

Algorithms Committee

Walter L. Biffl, MD, *Chair*

2013-2014 COMMITTEES

Program Committee

Peter Rhee, MD, *Chair*
Carlos Brown, MD
David Ciesla, MD
Mitch Cohen, MD
Charles Fox, MD

Randall Friese, MD
John Holcomb, MD
James Murray, MD
Henry Sagi, MD
David Shatz, MD

Publications Committee

Ajai Malhotra, MD, *Chair*
Chad Ball, MD
Denis Bensard, MD
Walt Biffi, MD
Rajan Gupta, MD
Riyad Karmy-Jones, MD
Robert Maxwell, MD
Mark Metzdorff, MD
Preston Miller, MD
Richard Miller, MD

Jordon Weinberg, MD
Amy Wyrzkowski, MD
Erik Barquist, MD
Bryan Collier, DO
James Haan, MD
Kenji Inaba, MD
Mathew Martin, MD
Nicholas Namias, MD
Jason Sperry, MD

Nominating Committee

Mark Metzdorff, MD, *Chair*
R. Lawrence Reed, MD,

Andrew Michaels, MD
Steve Ross, MD

Multi-Center Trials Committee

Rosemary A. Kozar, MD, *Chair*

Algorithms Committee

Walter L. Biffi, MD, *Chair*
Raul Coimbra, MD
Martin Croce, MD
Riyad Karmy-Jones, MD
Robert McIntyre, MD

Ernest E. Moore, MD
Nick Namias, MD
Martin Schreiber, MD
Jason Sperry, 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. Neviason, 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
Harold F. Sherman, MD	2006	Big Sky
Frederick A. Moore, MD	2007	Steamboat Springs
James W. Davis, MD	2008	Squaw Valley
Grace S. Rozycki, MD	2009	Crested Butte
Robert C. Mackersie, MD	2010	Telluride
M. Gage Ochsner, MD	2011	Big Sky
R. Lawrence Reed, MD	2012	Vail
Mark T. Metzdorff, MD	2013	Snowmass
David H. Livingston	2014	Steamboat Springs

NEW MEMBERS

**Western Trauma Association
Welcomed the Following New Members at the
2013 Annual Meeting**

Megan Brenner, MD
Baltimore, MD
Critical Care
Active Member

David Notrica, MD
Phoenix, Arizona
Pediatric Surgery
Active Member

Susan Biffi, MD
Denver, Colorado
Physical Medicine & Rehabilitation
Active Member

Kimberly Peck, MD
San Diego, California
General Surgery
Active Member

Bryan Collier, MD
Roanoke, Virginia
Critical Care
Active Member

Christian Schinkel, MD
Memmingen, Germany
Orthopedics
Active Member

Rajesh Gandhi, MD
Fort Worth, Texas
Critical Care
Active Member

David Sugerman, MD
Atlanta, Georgia
Emergency Medicine
Active Member

Oscar Guillamondegui, MD
Nashville, Tennessee
Critical Care
Active Member

George Testerman, MD
Kingsport, Tennessee
General Surgery
Senior Member

Todd Neideen, MD
Milwaukee, Wisconsin
General Surgery
Active Member

Nicholas Wetjen, MD
Rochester, Minnesota
Neurosurgery
Active Member

WESTERN TRAUMA FOUNDATION DONORS

CURRENT LIFETIME ACCUMULATION STATUS

Couloir Society (\$5,000 And Above)

Christine Cocanour	David Livingston	Thomas Scalea
James Davis	Robert Neviaser	Steven Shackford
Barry Esrig	Scott Petersen	Dennis Vane
David Feliciano	R. Lawrence Reed	
Founder (Anonymous)	Grace Rozycki	

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

Denis Bensard	Robert Mackersie	R. Stephen Smith
Marilu Bintz	Andrew Michaels	Harvey Sugerman
Kimberly Davis	J. Scott Millikan	Herbert Thomas, III
David Kappel	Steven Ross	

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

John Adams	Enrique Ginzburg	Frederick Moore
James Benjamin	K. Dean Gubler	Steve Moulton
Walter Biff	Gregory Jurkovich	M. Gage Ochsner
Miriam Bullard	Krista Kaups	Patrick Offner
David Ciesla	Guy Lanzi	Anne Rizzo
Thomas Cogbill	Manuel Lorenzo	David Shatz
Raul Coimbra	Barbara Mainville	Harold Sherman
Matthew Davis	Matthew Martin	Keith Stephenson
Doreen DiPasquale	James McCarthy	Steven Wald
George Dulabon	Mark Metzdorff	Michaela West
Soumitra Eachempati	Ernest Moore	

Blue Trail Associate (\$500 - \$999)

Scott Armen	John Hall	J. Bradley Pickhardt
Carlos Brown	Carl Hauser	Basil Pruitt
Howard Champion	David Hoyt	Peter Rhee
Roy Cobean	Riyad Karmy-Jones	Eric A. Toschlog
Alan Corcos	Brent King	Jennifer Watters
Clay Cothren-Burlew	Rosemary Kozar	R. Christie Wray, Jr.
James Cushman	William Long	
Richard Gamelli	Richard Miller	
Larry Gentilello	Frank Nastanski	

Green Trail Associate (\$250 - \$499)

Karen Brasel	M. Margaret Knudson	Edmund Rutherford
Gregory Campbell	Barbara Latenser	Aaron Scifres
Rajan Gupta	Richard Leone	Mark Shapiro
Michael Hauty	Charles Mains	
James Hebert	M. Ashraf Mansour	

Friends of the WTA (\$1 - \$249)

Anonymous	Charles Fox	George Pierce
Bonny Baron	Warren Gall	Susan Rowell
Donald Carter	Jay Johannigman	Carol Schermer
Mitch Cohen	Alan Marr	Gary Vercruysse
Charles Cook	Robert Maxwell	WTA MCT
Jody Digiacomo	Charlene Nagy	Amy Wyrzykowski
John Fildes	Nicholas Namias	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

IN MEMORIAM – M. GAGE OCHSNER, MD

THANK YOU TO THE WTA MEMBERS WHO MADE DONATION TO THE WESTERN TRAUMA FOUNDATION IN GAGE OCHSNER'S MEMORY



We will miss your extraordinary kindness and humor
Jan and Tom Cogbill

Shine on brightly
Carl Hauser

I hope we get to go turkey hunting in heaven
Fred Moore

Gage was the ideal surgeon role model
Christine Cocanour

In memory of Gage Ochsner
Karen Brasel

To a wonderful fly fishing instructor, I will miss you but I don't think the fish will
Harvey Sugerman

Colleague, friend and grower of the most extraordinary lemons. You will be missed.
David and Debbie Livingston

A role model, always taking the time to go fishing, you will be missed
Peter Rhee

To a man who represents best what we should all aspire to in medicine and in life...
Matt and Sharon Davis

In memory of M. Gage Ochsner, MD, loyal friend, example of family, patriot, and devoted leader of WTA
David Feliciano

In memory of Gage, one of my first friends (and favourite dance partners) at the WTA. Although too brief, he made my life richer.
Barbara Mainville

Gage: Your dedication to family, patients, and trainees is iconic of the WTA
Ernest Moore

Fair winds and following seas from all your military family and friends.
Gregory Campbell

In Memory of Gage Ochsner
Clay Cothren-Burlew

In Memory of Gage Ochsner
Larry Gentilello

In memory of our dear friend Gage Ochsner
Grace Rozycki

IN MEMORIAM – R. CHRISTIE WRAY, MD

THANK YOU TO THE WTA MEMBERS WHO MADE DONATION TO THE
WESTERN TRAUMA FOUNDATION IN CHRIS WRAY'S MEMORY



Your intellect, friendship and endless enthusiasm will be missed
Jan and Tom Cogbill

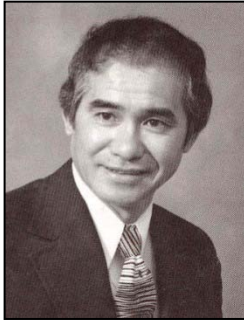
Shine on brightly
Carl Hauser

To a great chairlift buddy, I will miss our chit chats and trying to keep up with you as we zing
down the slopes
Harvey Sugerman

Your indomitable spirit and good cheer serves as an inspiration to us all
David and Debbie Livingston

We will miss you and your attitude
Peter Rhee

**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

<u>Resident</u>	<u>Institution</u>	<u>Year</u>
Joseph Schmoker, MD	University of Vermont	1991
Joseph Schmoker, MD	University of Vermont	1992
Charles Mock, MD	University of Washington	1993
Gino Travisani, MD	University of Vermont	1994
Phillip C. Ridings, MD	Medical College of Virginia	1995
David Han, MD	Emory University	1996
Preston R. Miller, MD	Wake Forest University	1997
Geoffrey Manley, MD, PhD	University of California, San Francisco	1998
James M. Doty, MD	Medical College of Virginia	1999
D.J. Ciesla, MD	Denver Health Medical Center	2000
Ricardo J. Gonzales, MD	Denver Health Medical Center	2001
Scott C. Brakenridge, MD	Cook County Hospital	2002
Adena J. Osband, MD	UMDNJ-New Jersey Medical School	2003
Cindy Lee, MD	UMDNJ-New Jersey Medical School	2004
Ernest A. Gonzalez, MD	University of Texas at Houston	2005
Jennifer M. Watters, MD	Oregon Health & Science University	2005
Jennifer J. Wan, MD	University of California, San Francisco	2006
Jennifer J. Wan, MD	University of California, San Francisco	2007
Keir J. Warner, MD	University of Washington	2008
T. W. Constantini, MD	University of California, San Diego	2009
C. Anne Morrison, MD	Baylor College of Medicine	2010
Marlin Causey, MD	Madigan Army Medical Center	2011
Phillip Letourneau, MD	University of Texas at Houston	2011
Gerard De Castro, MD	University of Maryland	2011
Matthew E. Kutcher, MD	University of California, San Francisco	2012
Kimberly Song, MD, MA	UMDNJ – New Jersey Medical School	2013

“PAINT THE CEILING” LECTURESHIP

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”.

<u>Presenter</u>	<u>Year</u>	<u>Location</u>
G. Jerry Jurkovich, MD	1997	Snowbird
John W. McGill, MD	1998	Lake Louise
William T. Close, MD	1999	Crested Butte
Jimmy Cornell	2000	Squaw Valley
Geoff Tabin, MD	2001	Big Sky
James H. “Red” Duke, MD	2002	Whistler
David V. Shatz, MD	2003	Snowbird
Susan and Tim Baker	2004	Steamboat Springs
Alex Habel, MD	2005	Jackson Hole
Andrew Schneider	2006	Big Sky
Ernest E. Moore, MD	2007	Steamboat Springs
Pamela Kallsen	2008	Squaw Valley
Sylvia Campbell, MD	2009	Crested Butte
William Schecter, MD	2010	Telluride
Jeff McKenney, MD	2011	Big Sky
Larry M. Gentilello, MD	2012	Vail
Neil L. Barg, MD	2013	Snowmass
Ziad Sifri, MD	2014	Steamboat Springs

FOUNDERS' BASIC SCIENCE LECTURESHIP

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.

<u>Presenter</u>	<u>Year</u>	<u>Location</u>
Raul Coimbra, MD	2009	Crested Butte
Lawrence Diebel, MD	2010	Telluride
Carl Hauser, MD	2011	Big Sky
Fred Moore, MD	2012	Vail
Steve Shackford, MD	2013	Snowmass
Hasan Alam, MD	2014	Steamboat Springs

SPECIAL SESSIONS

POINT/COUNTERPOINT: DAMAGE CONTROL SURGERY DEAD OR ALIVE?

Tuesday, March 4, 8:20 am – 9:00 am

Peter Rhee, MD

University of Arizona, Tucson, AZ

John Holcomb, MD

The University of Texas Medical School at Houston, Houston, TX

PRO/CON SESSION: PAN SCAN

Wednesday, March 5, 8:20 am – 8:50 am

James McCarthy, MD

The University of Texas Medical School at Houston, Houston, TX

Jordan Weinberg, MD

The University of Tennessee Health Science Center, Memphis, TN

MEMORIAL: GAGE OCHSNER

Wednesday, March 5, 4:00 pm – 4:10 pm

Jim Davis, MD

UCSF Fresno, Fresno, CA

MEMORIAL: CHRIS WRAY

Wednesday, March 5, 4:10 pm – 4:20 pm

Tom Cogbill, MD

Gunderson Lutheran, LaCrosse, WI

PANEL OF EXPERTS

Wednesday, March 5, 4:20 pm – 5:00 pm

Moderator: Andrew Michaels, MD

Legacy Emanuel Hospital, Portland, OR

Panel: Randall Friese, MD

University of Arizona, Tucson, AZ

Martin Schreiber, MD

Oregon Health & Science University, Portland, OR

Charles Fox, MD

Denver Health Medical Center, Denver, CO

CRITICAL DECISIONS: THORACIC VASCULAR TRAUMA

Thursday, March 6, 8:00 am – 8:20 am

Riyad Karmy-Jones, MD

Southwest Washington Medical Center, Vancouver, WA

CRITICAL DECISIONS: DUODENAL TRAUMA

Thursday, March 6, 8:20 am – 8:40 am

Ajai Malhotra, MD

VCU Medical Center, Richmond, VA

PRESIDENTIAL ADDRESS

“FELLOWSHIP OF THE SNOW”

Tuesday, March 4

5:00 pm – 6:00 pm

David H. Livingston, MD

New Jersey Medical School, Rutgers University

Newark, NJ



PAINT THE CEILING LECTURE

WHEN REALITY IS BETTER THAN THE DREAM: THE STORY OF ISHI

Thursday, March 6

5:00 pm – 6:00 pm

Ziad C. Sifri, MD, FACS

New Jersey Medical School, Rutgers University

Newark, NJ



Ziad C. Sifri, MD, FACS, is an Associate Professor of Surgery and Associate Director of Surgical Clerkship and Director of Trauma Clerkship at the New Jersey Medical School, Rutgers University, in Newark, New Jersey. In addition, he is a Trauma and Critical Care Attending at University Hospital. Dr. Sifri attended Medical School at McGill University in Montreal, Canada. He completed his General Surgery Training at John Hopkins University. He then completed a two year Trauma/ Critical Care and Research fellowships in Newark NJ and then joined the NJ Trauma Center as a Trauma/Critical Care Attending in 2003.

Dr. Sifri is a recipient of the NJMS Excellence in Teaching award and a seven-time recipient of the Golden Apple Teaching Award.

In 2009, Dr. Sifri co-founded and is currently the President of International Surgical Health Initiative (ISHI), a humanitarian non-profit organization that provides free surgical care to underserved communities internationally through volunteer services provided by surgeons and other health care professionals. ISHI is a 501c3 organization based in New Jersey; and has completed 12 surgical missions and performed more than 900 surgeries in Haiti, Sierra Leone, Ghana, Guatemala and the Philippines.

Dr. Sifri has personally led six such missions, including an emergency mission to Haiti right after the Earth Quake in 2010. He has given talks on Global Surgery at NJMS, NJ Chapter of the ACS and at various national surgical meetings. He supported the creation of the ISHI NJMS Club and helped create a non-credit elective on Global Surgery. His research interests in Global Surgery include: Surgical Burden, Impact of Surgery and Surgical Outcome in Rural Hospitals in Low Income Countries.

Dr. Sifri has authored over 30 peer reviewed publications, 40 published abstracts and has co-authored 2 book chapters related to Acute Care Surgery.



A TRAUMA SURGEON'S JOURNEY THROUGH THE WORLD OF BASIC RESEARCH

Friday, March 7

8:00 am – 8:40 am

Hasan Alam, MD

University of Michigan Hospital

Ann Arbor, MI

Dr. Hasan B. Alam is the Norman Thompson Professor of Surgery and Head of General Surgery (seven divisions) at the University of Michigan Hospital. He is a busy Acute Care Surgeon who received his surgical training at the Washington Hospital Center in Washington DC, followed by a post-doctoral research fellowship at the Uniformed Services University of Health Sciences (USUHS) in Bethesda, MD. He then served as a faculty member at the Georgetown University as well as USUHS before joining the Massachusetts General Hospital (MGH) in 2005. There he was rapidly promoted to the position of Professor of Surgery at the Harvard Medical School before moving to Ann Arbor in 2012.

His research focuses on hemorrhagic shock, traumatic brain injuries, resuscitation techniques, novel cell preservation strategies, modulation of response to lethal insults, therapeutic hypothermia, hemorrhage control, and development of new treatments for sepsis. This research has been funded by large federal grants (totaling >\$20 million) including NIH R01 grants. He has published about 200 manuscripts, numerous book chapters and holds 6 patents.

He will present selected research projects as case studies, and draw on his experience as a surgeon-scientist to highlight strategies that practicing trauma surgeons can exploit to succeed in the increasingly competitive world of basic research.

AGENDA

Sunday, March 2

5:00 pm – 7:00 pm	Registration Open	Registration Booth
5:00 pm – 7:00 pm	Welcome Reception	Sunshine Peak
5:00 pm – 7:00 pm	Children's Reception	Twilight
6:30 pm – 7:00 pm	WTA Foundation Board Meeting	Moonlight
7:00 pm – 8:00 pm	WTA Past Presidents Meeting	Boardroom

Monday, March 3

6:30 am – 9:30 am	Registration & Exhibits Open	Grand Ballroom Foyer
6:30 am – 8:00 am	Attendee Breakfast	Grand Ballroom Foyer
7:00 am – 9:00 am	Scientific Session I	Grand Ballroom
7:30 am – 9:00 am	Friends & Family Breakfast	Sevens Restaurant
3:30 pm – 6:30 pm	Registration & Exhibits Open	Grand Ballroom Foyer
4:00 pm – 6:00 pm	Scientific Session II	Grand Ballroom
6:00 pm – 7:00 pm	Residents Reception	Villa Gallery
6:00 pm – 8:00 pm	WTA Board of Directors Meeting	Aspen Boardroom
6:30 pm – 7:30 pm	Family Tubing Night Session One	Howelsen Hill
7:30 pm – 8:30 pm	Family Tubing Night Session Two	Howelsen Hill
8:30 pm – 9:30 pm	Family Tubing Night Session Three	Howelsen Hill

Tuesday, March 4

6:30 am – 9:30 am	Registration & Exhibits Open	Grand Ballroom Foyer
6:30 am – 8:00 am	Attendee Breakfast	Grand Ballroom Foyer
7:00 am – 9:00 am	Scientific Session III	Grand Ballroom
7:30 am – 9:00 am	Friends & Family Breakfast	Sevens Restaurant
10:00 am – 12:00 pm	WTA Ski Race	Lower Bashor
12:00 pm – 1:30 pm	Mountain BBQ	Sevens Deck
3:30 pm – 6:30 pm	Registration & Exhibits Open	Grand Ballroom Foyer
4:00 pm – 5:00 pm	Scientific Session IV	Grand Ballroom
5:00 pm – 6:00 pm	Presidential Address	Grand Ballroom
6:00 pm – 7:30 pm	Multi-Center Trials Meeting	Grand Ballroom

Wednesday, March 5

6:30 am – 9:30 am	Registration & Exhibits Open	Grand Ballroom Foyer
6:30 am – 8:00 am	Attendee Breakfast	Grand Ballroom Foyer
7:00 am – 9:00 am	Scientific Session V	Grand Ballroom
7:30 am – 9:00 am	Friends & Family Breakfast	Sevens Restaurant
3:30 pm – 6:30 pm	Registration & Exhibits Open	Grand Ballroom Foyer
4:00 pm – 6:00 pm	Book Club	Villa Gallery
4:00 pm – 5:00 pm	Scientific Session VI	Grand Ballroom
5:00 pm – 6:00 pm	Business Meeting (Members only)	Grand Ballroom
6:00 pm – 7:00 pm	Algorithms Committee Meeting	Aspen Boardroom

AGENDA (continued)

Thursday, March 6

6:30 am – 9:30 am	Registration & Exhibits Open	Grand Ballroom Foyer
6:30 am – 8:00 am	Attendee Breakfast	Grand Ballroom Foyer
7:00 am – 9:00 am	Scientific Session VII	Grand Ballroom
7:30 am – 9:00 am	Friends & Family Breakfast	Sevens Restaurant
3:30 pm – 6:30 pm	Registration & Exhibits Open	Grand Ballroom Foyer
4:00 pm – 5:00 pm	Scientific Session VIII	Grand Ballroom
5:00 pm – 6:00 pm	Paint the Ceiling Lecture	Grand Ballroom
6:30 pm – 7:30 pm	Reception	Grand Ballroom Foyer
7:00 pm – 10:00 pm	Children's Party	Twilight
7:30 pm – 10:00 pm	Banquet	Grand Ballroom

Friday, March 7

6:30 am – 9:30 am	Registration & Exhibits Open	Grand Ballroom Foyer
6:30 am – 8:00 am	Attendee Breakfast	Grand Ballroom Foyer
7:00 am – 9:00 am	Scientific Session IX	Grand Ballroom
7:30 am – 9:00 am	Friends & Family Breakfast	Sevens Restaurant
3:30 pm – 6:30 pm	Registration & Exhibits Open	Grand Ballroom Foyer
4:00 pm – 6:00 pm	Scientific Session X	Grand Ballroom
6:00pm	End of Meeting	

SCIENTIFIC PROGRAM

MONDAY, MARCH 3 – MORNING SESSION

7:00 am – 9:00 am

Scientific Session I: Papers 1-5, Case Reports: #6-7 (Grand Ballroom)

Moderator: David H. Livingston, New Jersey Medical School, Rutgers University

7:00 am - 7:20 am	*1. THE WHOLE IS GREATER THAN THE SUM OF ITS PARTS: HEMOSTATIC PROFILES OF WHOLE BLOOD VARIANTS Lucy Kornblith UCSF/SFGH, San Francisco, CA	PAGE 35
7:20 am - 7:40 am	*2. PATHOLOGIC VERSUS PHYSIOLOGIC FIBRINOLYSIS: RECONSIDERATION OF EMPIRIC ANTI-FIBRINOLYTIC THERAPY FOR SEVERELY INJURED PATIENTS Hunter Moore Denver Health, Denver, CO	PAGE 37
7:40 am - 8:00 am	*3. ATTRIBUTION: WHOSE COMPLICATION IS IT? Jason Murry Methodist Dallas Medical Center, Dallas, TX	PAGE 39
8:00 am - 8:20 am	*4. DISTRACTING INJURIES IN THE TRAUMA BAY: A TRUE CONTRAINDICATION TO CLINICAL CLEARANCE OF THE CERVICAL SPINE? Mario Gomez Lutheran Medical Center, Brooklyn, NY	PAGE 41
8:20 am - 8:40 am	*5. RISK FACTORS FOR DELIRIUM IN OLDER TRAUMA PATIENTS ADMITTED TO THE SURGICAL INTENSIVE CARE UNIT Sarah B. Bryczkowski Rutgers, New Jersey Medical School, Newark, NJ	PAGE 43
8:40 am - 8:50 am	6. CASE REPORT SEAT BELT CAUSING BILATERAL FLANK ABDOMINAL WALL HERNIAS, MULTIPLE INTESTINAL TRANSECTIONS, DISSECTION OF LEFT COMMON ILIAC ARTERY, LUMBAR BURST FRACTURE, AND SHOCK; ROLE OF A MOBILE SURGICAL TRANSPORT TEAM SUPPORTING A RURAL TRAUMA CENTER Ameen Ramzy Legacy Emanuel Trauma Center, Portland, OR	PAGE 45
8:50 am - 9:00 am	7. CASE REPORT KETAMINE USE IN A FIELD AMPUTATION: MAKING THE TERRIFYING TOLERABLE Jayson Aydelotte University Medical Center Brackenridge, Austin, TX	PAGE 47

**Denotes Earl Young Award Competition Paper*

SCIENTIFIC PROGRAM (continued)

MONDAY, MARCH 3 – AFTERNOON SESSION

4:00 pm – 6:00 pm

Scientific Session II: Papers 8-13 (Grand Ballroom)

Moderator: John Holcomb, The University of Texas Medical School at Houston, Houston, TX

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|-------------------|---|---------|
| 4:00 pm - 4:20 pm | *8. APPLICABILITY OF THE NATIONAL HEALTHCARE SAFETY NETWORK'S SURVEILLANCE DEFINITION OF VENTILATOR ASSOCIATED EVENTS IN THE SURGICAL INTENSIVE CARE UNIT: A ONE-YEAR REVIEW
Colleen Stoeppel
UT Southwestern
Dallas, TX | PAGE 49 |
| 4:20 pm - 4:40 pm | *9. MELATONIN INHIBITS THERMAL INJURY INDUCED HYPERPERMEABILITY IN MICROVASCULAR ENDOTHELIAL CELLS
Katie Wiggins-Dohlvik
Scott and White Memorial Hospital
Temple, TX | PAGE 51 |
| 4:40 pm - 5:00 pm | *10. IMPLEMENTATION OF A SURGICAL ICU SERVICE IS ASSOCIATED WITH IMPROVED OUTCOMES FOR TRAUMA PATIENTS
Amanda Klein
University of Texas Southwestern - Austin,
UMC Brackenridge
Austin, TX | PAGE 53 |
| 5:00 pm - 5:20 pm | *11. DOES SIMPLE IMAGING HOLD THE KEY: USING CXR AS A SCREENING TOOL TO IDENTIFY SEVERE PULMONARY CONTUSION
Elizabeth Helmer
Memorial University Medical Center
San Diego, CA | PAGE 55 |
| 5:20 pm - 5:40 pm | *12. URINARY TRACT INFECTION IN ELDERLY TRAUMA PATIENTS: REVIEW OF THE TRAUMA QUALITY IMPROVEMENT PROJECT DATABASE IDENTIFIES THE POPULATION AT RISK
Stephanie Polites
Mayo Clinic
Rochester, MN | PAGE 57 |
| 5:40 pm - 6:00 pm | *13. BELOW-THE-KNEE ARTERIAL INJURY: THE TYPE OF VESSEL MAY BE MORE IMPORTANT THAN THE NUMBER OF VESSELS INJURED
Joseph Scalea
University of Maryland
Baltimore, MD | PAGE 59 |

**Denotes Earl Young Award Competition Paper*

SCIENTIFIC PROGRAM (continued)

TUESDAY, MARCH 4 – MORNING SESSION

7:00 am – 9:00 am

Scientific Session III: Papers 14-17, Point/Counterpoint (Grand Ballroom)

Moderator: Carlos Brown, University of Texas Southwestern – Austin, University Medical Center Brackenridge, Austin, TX

7:00 am - 7:20 am	*14. MISLED BY TEG? THE EFFECTS OF ALCOHOL ON COAGULATION IN TRAUMA PATIENTS Benjamin Howard UCSF/SFGH San Francisco, CA	PAGE 61
7:20 am - 7:40 am	*15. MODULATION OF INTESTINAL MUCUS PHYSIOCHEMICAL PROPERTIES AND PROTECTION AGAINST OXIDANT INJURY: THE FIRST LINE OF INTESTINAL BARRIER DEFENSE BY ESTROGEN Mark E. Diebel Wayne State University Detroit, MI	PAGE 63
7:40 am - 8:00 am	*16. CRYSTALLOID ADMINISTRATION DURING TRAUMA RESUSCITATION: DOES LESS REALLY EQUAL MORE? John Sharpe University of Tennessee Health Science Center Memphis, TN	PAGE 65
8:00 am - 8:20 am	17. ACUTE RESPIRATORY DISTRESS SYNDROME RISK FACTORS FOR INJURED PATIENTS UNDERGOING DAMAGE CONTROL LAPAROTOMY: AAST MULTICENTER, PROSPECTIVE, OBSERVATIONAL TRIAL Martin Zielinski Mayo Clinic Rochester, MN	PAGE 67
8:20 am – 9:00 am	Point/Counterpoint: DAMAGE CONTROL SURGERY DEAD OR ALIVE? Peter Rhee University of Arizona, Tucson, AZ John Holcomb The University of Texas Medical School at Houston, Houston, TX	

**Denotes Earl Young Award Competition Paper*

SCIENTIFIC PROGRAM (continued)

TUESDAY, MARCH 4 – AFTERNOON SESSION

4:00 pm – 6:00 pm

Scientific Session IV: Papers 18-20, Presidential Address (Grand Ballroom)

Moderator: Henry Sagi, Florida Orthopaedic Institute, Tampa, FL

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|-------------------|--|---------|
| 4:00 pm - 4:20 pm | *18. THE EFFECT OF LACTATE ON PULMONARY MORBIDITY IN FEMORAL SHAFT FRACTURES TREATED WITH EARLY TOTAL CARE: DOES PERSISTENT OCCULT HYPOPERFUSION MATTER?
Justin Richards
Vanderbilt University Medical Center
Nashville, TN | PAGE 69 |
| 4:20 pm - 4:40 pm | *19. EFFECT OF PHARMACOLOGICAL RESUSCITATION ON BRAIN GENE EXPRESSION PROFILES IN A LARGE ANIMAL MODEL OF COMBINED TRAUMATIC BRAIN INJURY AND HEMORRHAGIC SHOCK
Simone Dekker
University of Michigan Hospital
Ann Arbor, MI | PAGE 71 |
| 4:40 pm - 5:00 pm | *20. DETECTION OF LOW VOLUME BLOOD LOSS: THE COMPENSATORY RESERVE INDEX VERSUS
Camille Stewart
University of Colorado, School of Medicine
Aurora, CO | PAGE 73 |
| 4:00 pm – 5:00 pm | Presidential Address:
“FELLOWSHIP OF THE SNOW”
David H. Livingston
New Jersey Medical School, Rutgers University
Newark, NJ | |

**Denotes Earl Young Award Competition Paper*

SCIENTIFIC PROGRAM (continued)

WEDNESDAY, MARCH 5 – MORNING SESSION

7:00 am – 9:00 am

Scientific Session V: Papers 21-25, Pro/Con (Grand Ballroom)

Moderator: Randall Friese, University of Arizona, Tucson, AZ

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|-------------------|---|---------|
| 7:00 am - 7:20 am | *21. DIFFERENCES IN ACUTE KIDNEY INJURY AND DEATH BETWEEN BLUNT AND PENETRATING TRAUMA AFTER RESUSCITATION WITH HETASTARCH
Casey Allen
University of Miami Miller School of Medicine
Miami, FL | PAGE 75 |
| 7:20 am - 7:40 am | 22. A CRITICAL ANALYSIS OF SECONDARY OVERTRIAGE TO A LEVEL-I TRAUMA CENTER
Andrew Tang, MD FACS
University of Arizona
Tucson, AZ | PAGE 77 |
| 7:40 am - 8:00 am | 23. SEASONAL VARIATIONS IN POST-TRAUMATIC OSTEOMYELITIS AFTER OPEN EXTREMITY FRACTURES
Henry Sagi
Florida Orthopaedic Institute
Tampa, FL | PAGE 79 |
| 8:00 am - 8:20 am | 24. CELL NECROSIS INDEPENDENT SUSTAINED MITOCHONDRIAL AND NUCLEAR DNA RELEASE FOLLOWING TRAUMA SURGERY
Zsolt Balogh
John Hunter Hospital and University of Newcastle
Newcastle, Australia | PAGE 81 |
| 8:20 am – 8:50 am | PRO/CON Session: PAN SCAN
James McCarthy
The University of Texas Medical School at Houston, Houston, TX
Jordan Weinberg
The University of Tennessee Health Science Center, Memphis, TN | |
| 8:50 am - 9:00 am | 25. FAMILY ABSTRACT: BUGABOOS, CARIBOOS, AND WTA YAHOOOS: THE SPIRIT OF THE WTA CELEBRATED IN THE CANADIAN ROCKIES.
Hunter Moore
Denver Health Medical Center
Aurora, CO | PAGE 83 |

**Denotes Earl Young Award Competition Paper*

SCIENTIFIC PROGRAM (continued)

WEDNESDAY, MARCH 5 – AFTERNOON SESSION

4:00 pm – 5:00 pm

Scientific Session VI: Memorial, Panel of Experts (Grand Ballroom)

Moderator: Charles Fox, Denver Health Medical Center, Denver, CO

4:00 pm – 4:10 pm

Memorial: GAGE OCHSNER

Jim Davis

UCSF Fresno, Fresno, CA

4:10 pm – 4:20 pm

Memorial: CHRIS WRAY

Tom Cogbill

Gunderson Lutheran, LaCrosse, WI

4:20 pm – 5:00 pm

Panel of Experts

Moderator: Andrew Michaels

Legacy Emanuel Hospital, Portland, OR

Panel: Randall Friese

University of Arizona, Tucson, AZ

Martin Schreiber,

Oregon Health & Science University

Portland, OR

Charles Fox

Denver Health Medical Center, Denver, CO

5:00 pm – 6:00 pm

Business Meeting (Members Only)

SCIENTIFIC PROGRAM (continued)

THURSDAY, MARCH 6 – MORNING SESSION

7:00 am – 9:00 am

Scientific Session VII: Papers 26-30 (Grand Ballroom)

Moderator: David Ciesla, University of South Florida, Tampa, FL

7:00 am - 7:20 am	26. TRANSMISSION-BASED CONTACT PRECAUTIONS FOR MULTIDRUG RESISTANT ORGANISMS: FEWER DAYS IN ISOLATION WITH NO INCREASE IN HOSPITAL ACQUIRED INFECTIONS Carlos VR Brown University of Texas Southwestern - Austin UMC Brackenridge Austin, TX	PAGE 85
7:20 am - 7:40 am	27. MEASURING THROMBIN GENERATION AS A TOOL FOR PREDICTING HEMOSTATIC POTENTIAL AND TRANSFUSION REQUIREMENTS FOLLOWING TRAUMA Jessica Cardenas University of Texas at Houston Houston, TX	PAGE 87
7:40 am - 8:00 am	28. THE PEDIATRIC TRAUMA AND TRANEXAMIC ACID STUDY (PED-TRAX) Matthew Eckert Madigan Army Medical Center Tacoma, WA	PAGE 89
8:00 am - 8:20 am	CRITICAL DECISIONS: PENETRATING THORACIC TRAUMA Riyad Karmy-Jones Southwest Washington Medical Center Vancouver, WA	PAGE 123
8:20 am - 8:40 am	CRITICAL DECISIONS: DUODENAL TRAUMA Ajai Malhotra VCU Medical Center Richmond, VA	PAGE 127
8:40 am - 8:50 am	29. CASE REPORT: BIOSCAFFOLD REPLACEMENT IN PEDIATRIC VASCULAR INJURY: A CASE REPORT Caitlin Smith University of California at Davis Sacramento, CA	PAGE 91
8:50 am - 9:00 am	30. CASE REPORT: EXTRACORPOREAL MEMBRANE OXYGENATION IN SEVERE INTRACRANIAL HEMORRHAGE: A CASE REPORT Robert Jean University of Tennessee-Chattanooga Chattanooga, TN	PAGE 93

SCIENTIFIC PROGRAM (continued)

THURSDAY, MARCH 6 – AFTERNOON SESSION

4:00 pm – 6:00 pm

Scientific Session VIII: Papers 31-33, Paint the Ceiling Lecture (Grand Ballroom)

Moderator: James Murray, University of Southern California, Long Beach, CA

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|-------------------|---|---------|
| 4:00 pm - 4:20 pm | 31. BLUNT ABDOMINAL AORTIC INJURY: A MULTICENTER EXPERIENCE
Sherene Shalhub
University of Washington
Seattle, WA | PAGE 95 |
| 4:20 pm - 4:40 pm | 32. ANGIOGRAPHY IS AN IMPORTANT TOOL TO CONSIDER IN HIGH GRADE SPLEEN INJURIES
Louis Capecci
Allegheny General Hospital
Pittsburgh, PA | PAGE 97 |
| 4:40 pm - 5:00 pm | 33. HUMAN AND PILOT ERROR IN HELICOPTER EMERGENCY MEDICAL SERVICES CRASHES: WHAT ROLE DOES OWNERSHIP PLAY?
Fahim Habib
Broward Health Medical Center
Ft Lauderdale, FL | PAGE 99 |
| 5:00 pm - 6:00 pm | Paint the Ceiling Lecture
WHEN REALITY IS BETTER THAN THE DREAM: THE STORY OF ISHI
Ziad C. Sifri
New Jersey Medical School, Rutgers University
Newark, NJ | |

SCIENTIFIC PROGRAM (continued)

FRIDAY, MARCH 7 – MORNING SESSION

7:00 am – 9:00 am

Scientific Session IX: Papers 34-38, Basic Science Lecture (Grand Ballroom)

Moderator: Mitch Cohen, UCSF/SFGH, San Francisco, California

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|-------------------|---|----------|
| 7:00 am - 7:20 am | 34. HISTONE DEACETYLASE III AS A POTENTIAL THERAPEUTIC TARGET FOR THE TREATMENT OF LETHAL SEPSIS
Hasan Alam
University of Michigan Hospital
Ann Arbor, MI | PAGE 101 |
| 7:20 am - 7:40 am | 35. DEATH AFTER DISCHARGE: PREDICTORS OF MORTALITY IN OLDER BRAIN-INJURED PATIENTS
Kimberly Peck
Scripps Mercy Hospital
San Diego, CA | PAGE 103 |
| 7:40 am - 8:00 am | 36. PROSPECTIVE VALIDATION OF THE BRAIN INJURY GUIDELINES: MANAGING TRAUMATIC BRAIN INJURY WITHOUT NEUROSURGICAL CONSULTATION
Bellal Joseph
The University of Arizona
Tucson, AZ | PAGE 105 |
| 8:00 am - 8:40 am | Founders' Basic Science Lecture: A TRAUMA SURGEON'S JOURNEY THROUGH THE WORLD OF BASIC RESEARCH
Hasan Alam
University of Michigan Hospital
Ann Arbor, MI | |
| 8:40 am - 8:50 am | 37. CARING FOR YOUR PET RATTLESNAKE....BITE. A REVIEW OF CROTALIDAE POLYVALENT IMMUNE FAB USE AND OUTCOME IN A SNAKE INFESTED STATE.
Anne Rizzo
United Regional Hospital
Wichita Falls, TX | PAGE 107 |
| 8:50 am - 9:00 am | 38. THE NATURAL HISTORY OF FRACTURING FOUR EXTREMITIES
Jordan Jacobs
The Ryder Center at University of Miami
Phoenix, AZ | PAGE 109 |

SCIENTIFIC PROGRAM (continued)

FRIDAY, MARCH 7 – AFTERNOON SESSION

4:00 pm – 6:00 pm

Scientific Session X: Papers 39-43 (Grand Ballroom)

Moderator: Peter Rhee, University of Arizona, Tucson, AZ

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|-------------------|--|----------|
| 4:00 pm - 4:20 pm | 39. 1:1 RECONSIDERED : PATIENTS MEETING THE CRITICAL ADMINISTRATION THRESHOLD (CAT+) FARE BETTER WITH MATCHED TRANSFUSIONS
Stephanie Savage
University of Tennessee Health Science Center
Memphis, TN | PAGE 111 |
| 4:20 pm - 4:40 pm | 40. MODERATE ELEVATIONS IN INR SHOULD NOT LEAD TO DELAYS IN NEUROSURGICAL INTERVENTION IN PATIENTS WITH SEVERE TBI
Tori Lennox
Oregon Health & Science University
Portland, OR | PAGE 113 |
| 4:40 pm - 5:00 pm | 41. THE EVOLUTION OF A HIGH RELIABILITY REGIONAL PROGRAM OF EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO) FOR ADULTS WITH TRAUMA, CARDIOVASCULAR COLLAPSE AND REFRACTORY HYPOXEMIC ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)
Andrew Michaels
Legacy Emanuel Medical Center
Portland, OR | PAGE 115 |
| 5:00 pm - 5:20 pm | 42. RESVERATROL AMELIORATES MITOCHONDRIAL DYSFUNCTION BUT INCREASES THE RISK OF HYPOGLYCEMIA FOLLOWING HEMORRHAGIC SHOCK
Hao Wang
The Trauma Center at Penn
University of Pennsylvania, PA | PAGE 117 |
| 5:20pm - 5:40pm | 43. Alcohol Withdrawal Syndrome in Critically Ill Patients: Protocolized vs Non-Protocolized Management
Christine Cocanour
University of California, Davis Medical Center
Sacramento, CA | PAGE 119 |
| 5:40 pm - 6:00 pm | 44. OBESITY-INDUCED HYPERCOAGULABILITY PREVENTS ACUTE TRAUMATIC COAGULOPATHY
Belinda McCully
Oregon Health & Science University
Portland, OR | PAGE 121 |

THE WHOLE IS GREATER THAN THE SUM OF ITS PARTS: HEMOSTATIC PROFILES OF WHOLE BLOOD VARIANTS *

LZ Kornblith, BM Howard, CK Cheung, Y Dayter, S Pandey, MP Busch, S Pati, RA Callcut, RF Vilardi, BJ Redick, MF Nelson, MJ Cohen
UCSF/SFGH, San Francisco, California

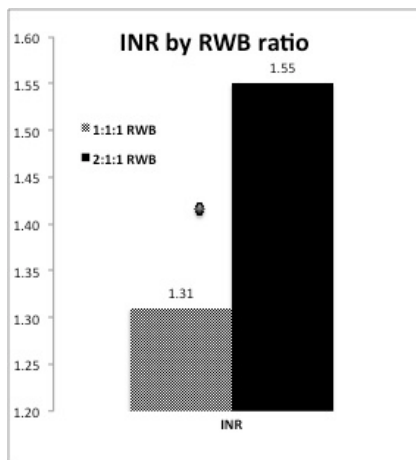
Presenter: Lucy Kornblith

Senior Sponsor: Mitchell Jay Cohen

INTRODUCTION: Mounting evidence highlighting the benefits of hemostatic resuscitation has led to a renewed interest in whole blood (WB) and reconstituted whole blood (RWB). However, no data exists to characterize the clotting profiles of these variants. This study characterizes banked WB variants and RWB in standard 1:1:1 and 2:1:1 transfusion ratios of packed red blood cells (RBC), fresh frozen plasma (FFP), and platelets (PLTS). We hypothesized that the global hemostatic profile of 1:1:1 RWB is superior to 2:1:1 RWB, and platelet-modified WB (MWB) is superior to 1:1:1 RWB.

METHODS: 23 units of RBC, FFP, and PLTS were obtained from the regional blood collection center and mixed to create 23 1:1:1 and 23 2:1:1 RWB units. Freshly donated WB units were obtained and used to create 11 of each: non-modified (NMWB) (room temperature and cooled) and platelet-modified (MWB) (room temperature and cooled) variants. MWB units were created by adding PLTS in a 6 WB:1 PLT ratio. INR/PTT, CBC, functional studies, and an extensive panel of pro- and anti-coagulant factor assays were performed on all products.

RESULTS: 1:1:1 RWB had significantly lower INR and PTT (1.31 vs. 1.55, $p=0.0029$, Figure; 42s vs. 50s, $p=0.0008$) and higher activity of factors II, V, VII, VIII, IX, X, anti-thrombin III, protein C, and higher fibrinogen levels than 2:1:1 RWB (factor IX 86% vs. 70%, $p=0.0313$; fibrinogen 242mg/dL vs. 202mg/dL, $p=0.0385$). There were no differences in INR/PTT or factor activity between MWB and NMWB. However, MWB had greater maximum clot firmness (MCF) by EXTEM than NMWB (MCF 61mm vs. 50mm, $p=0.0031$). MWB also had greater MCF by EXTEM than 1:1:1 RWB (MCF 61mm vs. 45mm, $p=0.0005$).



CONCLUSIONS: Although 1:1:1 RWB had a superior clotting profile relative to 2:1:1 RWB, MWB exhibited even better global hemostasis than 1:1:1 RWB. Characterization of factor-level and functional clotting differences between WB variants is imperative for understanding the clinical benefits of hemostatic resuscitation.

PATHOLOGIC VERSUS PHYSIOLOGIC FIBRINOLYSIS: RECONSIDERATION OF EMPIRIC ANTI-FIBRINOLYTIC THERAPY FOR SEVERELY INJURED PATIENTS *

HB Moore, EE Moore, M Chapman, TL Chin, E Gonzalez, A Banerjee, A Ghasabyan, C Gravitz, A Sauaia
 Denver Health, Denver, Colorado

Presenter: Hunter Moore

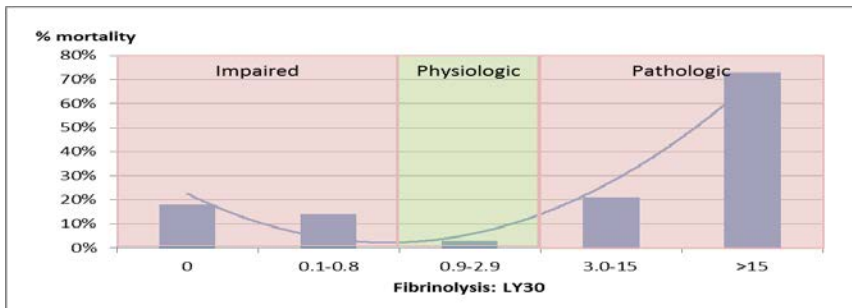
Senior Sponsor: Ernest Moore

INTRODUCTION: Fibrinolysis is an physiologic counterbalances of coagulation to maintain patency of the microvasculature during low flow states. However, this essential function has been proposed as a pathologic mechanism of trauma induced coagulopathy associated with increased mortality. We hypothesize that a low level of fibrinolysis confers a survival benefit to severely injured patients.

METHODS: Thrombelastography and clinical data were prospectively collected on patients with severe injury (ISS>15) admitted to an urban level 1-trauma center from 2010 to 2013. Fibrinolysis at 30 minutes (LY30) was stratified into 5 groups: severe: >15, major: 15-3, moderate: 2.9-0.9, mild: 0.8-0.1 and no lysis: 0). Statistical analysis was performed using Mann-Whitney test for continuous variables and Fischer exact test for categorical data.

RESULTS: 193 patients with median age of 42 years (interquartile range, IQR: 28-55), of whom 19% died were included in the study. Most (70%) were men, and 30% had penetrating injuries. Median ISS was 29 (IQR 22-36), base deficit 9 mEq/L (IQR 6-13). Distribution of fibrinolysis was: no lysis 41%, mild 22%, moderate 20%, major 10% (20/193) and severe 7% (14/193). Mortality rates are shown in Figure. Physiologic lysis (moderate) mortality was significantly reduced compared to pathologic fibrinolysis (severe + major p=0.002) and impaired fibrinolysis (mild and no lysis p=0.027). There were no statistical differences between age, ISS, and base deficit between groups.

CONCLUSIONS: We have identified a U shaped distribution of death related to the fibrinolysis system in response to major trauma. Our data demonstrate a nadir in mortality with ly30 level between 0.9 and 2.9 suggesting a physiologic protection from death. Exogenous inhibition of the fibrinolysis system in severely injured patients requires careful selection as it may have an adverse effect on survival.



ATTRIBUTION: WHOSE COMPLICATION IS IT? *

JS Murry, GE Hambright, MS Truitt, EL Dunn
Methodist Dallas Medical Center, Dallas, Texas

Presenter: Jason Murry

Senior Sponsor: Michael Truitt

INTRODUCTION: American healthcare and its delivery continue to change. Healthcare models aimed at curbing skyrocketing costs and improving quality are being deployed. The most prevalent of these are Accountable Care Organizations (ACO). In order to improve quality, these programs need to determine the part of the healthcare system most “responsible” for a complication. This is referred to as attribution. This may also provide a framework to compare physicians by providing information to patients and third-party payers. Traditionally, the attribution of complications has been to the admitting physician. While this has been a simple solution, it may misidentify the physician “responsible” for the complication. This is especially difficult in trauma patients who have multiple providers. We hypothesized that the current mechanism for attributing complications in trauma patients is inadequate and will need to be modernized.

METHODS: All trauma admissions over a 12 month period were reviewed. We excluded patients with single system trauma who were admitted to and underwent an operation by the admitting service. We reviewed our trauma database for mechanism of injury, complications and readmissions. The trauma director and the medical director (TD/MD) of our ACO reviewed all complications and attributed them to the appropriate healthcare provider. These were compared to the hospital decisions using the traditional definition.

RESULTS: The trauma service had 1526 admissions. After exclusions, 1019 patients were reviewed. These patients were admitted to the acute care surgery (ACS) service with subspecialty input when appropriate. One hundred-twenty five complications occurred in 73 patients. Using the traditional definition, the ACS service was assigned all 125 complications. Using the TD/MD method the neurosurgical attending accounted for 36% (45/125) of complications. The ACS attending was responsible for 34% (43/125) of complications, and orthopedic surgery was identified as the causative factor in 22% (27/125). The remaining 8% (10/125) were attributed to various other services. Urinary tract infections were the most frequent complication with 41% (9/22) attributed to the neurosurgery service, 32% (7/22) to the ACS, and 18% (4/22) to orthopedics. Wound infections were attributed to neurosurgery in 41% (7/17), orthopedics in 35% (6/17) plastics in 18% (3/17) and ACS in 6% (1/17). Seven patients had unexpected readmissions. The majority (6/7) of these were related to orthopedics.

CONCLUSIONS: Hospital complications are now being assigned to individual surgeons. Which physician is responsible for each complication will be a controversial matter. Criteria for attribution will be important as the data will be publically reported and used in the determination of quality and pay for performance. Without a critical review process with physician input, up to two-thirds of complications occurring in trauma patients could be attributed incorrectly to the admitting ACS. The attribution process needs to be refined and it is important for ACS to lead the discussion on this important matter.

DISTRACTING INJURIES IN THE TRAUMA BAY: A TRUE CONTRAINDICATION TO CLINICAL CLEARANCE OF THE CERVICAL SPINE? *

M Gomez, S Oven, FY Vincas

Lutheran Medical Center, Brooklyn, New York

Presenter: Mario Gomez**Senior Sponsor: Ajai Malhotra**

INTRODUCTION: The purpose of this study was to retrospectively evaluate the sensitivity and specificity of clinically screening for cervical spine injury in blunt trauma patients who are alert, awake with coexisting distracting injuries.

METHODS: : Data from the 15 month period from July 2012 to October 2013, from all blunt trauma patients older than 18 years were analyzed with standard cervical spine examination by the trauma team at our urban level 1 trauma center. As previously described in the literature, our protocol dictates that all patients with distracting injuries warrant a cervical spine computerized Tomographic (CT) scan in spite of a negative cervical spine clinical exam. Data collected from patients that were awake and alert with a Glasgow Coma Score (GCS) 14 with distracting injuries were analyzed. Distracting injuries was categorized as described in the literature into different anatomic regions such as Head, Torso injuries and Long bone fractures. Patients without distracting injuries or minor injuries were not selected for this study.

RESULTS: During the 15-month study period, data from 306 blunt trauma patients with GCS 14 and at least one distracting injury were analyzed. Thirty one patients (10%) with distracting injuries had a positive C-spine examination of which 20 (64%) patients were diagnosed with c-spine injury. Two hundred seventy five patients (90%) were clinically cleared initially, but one patient was found to have a c-spine injury on CT scan. Our analyses yield a sensitivity 95% (20/21) and negative predictive value of 99% (274/275) for clinically detecting cervical injury in awake and alert blunt trauma with distracting injuries.

CONCLUSIONS: Our data provides additional support to the recent movement described in the literature in that cervical spine imaging is unnecessary in blunt trauma patients that are awake, alert with distracting injuries. In these patients clinical examination alone can be used to clinically clear the cervical spine in asymptomatic patient with excellent sensitivity and specificity. Expanding the trauma protocol to allow the utilization of clinical examination to clear the cervical spine of blunt trauma patient will both decrease radiation exposure and by limiting the number of CT scans would be economically advantageous for the hospital.

RISK FACTORS FOR DELIRIUM IN OLDER TRAUMA PATIENTS ADMITTED TO THE SURGICAL INTENSIVE CARE UNIT *

SB Bryczkowski, MC Lopreiato, PP Yonclas, JJ Sacca, AC Mosenthal
Rutgers, New Jersey Medical School, Newark, New Jersey

Presenter: Sarah B. Bryczkowski

Senior Sponsor: David Livingston

INTRODUCTION: Adults (age >50) admitted to the SICU are at high risk for delirium. Little is known about the role traumatic injury plays in the development of delirium as these patients have often been excluded from studies. Identification of specific risk factors for delirium among older adults following injury would be useful to guide prevention strategies. We attempted to identify modifiable factors that would predict delirium in an older trauma population admitted to the SICU.

METHODS: Data were collected prospectively from July 2012 - August 2013 at a Level I trauma center on consecutive trauma patients, age >50 years, admitted to the SICU. Patients who died in the SICU were excluded. Delirium was assessed every 12 hours using the Confusion Assessment Method - ICU scale. Demographic, injury, social, and clinical variables were reviewed. Bivariate analysis determined significant factors associated with delirium. A multivariate logistic regression model was used to predict delirium risk. After preliminary results, additional analysis compared patients with chest injury (defined as chest AIS ≥ 3) to those without.

RESULTS: 115 patients met criteria with a mean age 67 years, Injury Severity Score (ISS) 19, and Glasgow Coma Scale (GCS) 14. The incidence of delirium was 61%. Variables present on admission that were positive predictors of delirium were: Age, ISS >17, GCS <15, substance abuse, and traumatic brain injury (TBI; defined as AIS head ≥ 3). Chest injury (defined as AIS chest ≥ 3) was a negative predictor of delirium. Significant risk factors influenced by clinical treatment included: doses of opioids and propofol, restraint use, and hours deeply sedated (RASS ≤ -3). Clinical treatments with negative predictability were: ventilator-free days/30 (vent-free), benzodiazepine-free days/30 (benzo-free) and restraint-free days/30. In a regression model considering age, vent-free days, chest injury, TBI, GCS, benzo-free days, and hours sedated, only age [Odds Ratio 1.1, 95% Confidence Interval (1.01 - 1.1), $p=0.03$] was a predictor of delirium, while vent-free days [OR 0.79 (CI 0.65 - 0.96), $p=0.02$] and chest injury [0.3 (0.09 - 0.83), $p=0.02$] were significant negative predictors of delirium. Patients with chest injury had lower delirium incidence (44%) versus those without (75%), $p=0.002$ despite similar GCS, ISS, and clinical variables.

CONCLUSIONS: Delirium is common in older trauma patients admitted to the SICU and for every year over the age of 50, the chance of delirium increases by 10%. While higher ISS increases delirium risk, we identified several modifiable treatment variables including days patients were: deeply sedated, mechanically ventilated, and physically restrained. Interestingly, patients with chest injury experienced less delirium, despite similar injury severity and clinical variables, perhaps due to frequent healthcare provider interactions.

NOTES

SEAT BELT CAUSING BILATERAL FLANK ABDOMINAL WALL HERNIAS, MULTIPLE INTESTINAL TRANSECTIONS, DISSECTION OF LEFT COMMON ILIAC ARTERY, LUMBAR BURST FRACTURE, AND SHOCK; ROLE OF A MOBILE SURGICAL TRANSPORT TEAM SUPPORTING A RURAL TRAUMA CENTER

Al Ramzy, E Duret, S Izenberg, C Wang, W Long
Legacy Emanuel Trauma Center, Portland, Oregon

Presenter: Ameen Ramzy

Senior Sponsor: William Long

INTRODUCTION: Although protective, seat belts are associated with abdominal wall disruption, intestinal perforations, major vascular injuries, spine injuries, and hemorrhagic shock. A surgeon at a rural level 4 trauma center with a limited blood bank supply may hesitate to perform damage control surgery to control bleeding if a coagulopathy from a lack of blood products (platelets) compromises resuscitative efforts. This case report describes the value of a Mobile Surgical Transport Team (MSTT) to support a severely injured patient in a rural trauma center.

METHODS: Case Review.

RESULTS: A 56 year-old executive was a restrained passenger in a high speed two car, frontal impact crash on a rural highway. EMS took her to the nearest trauma center (an Oregon state level 4) 32 miles away. CAT scan showed hemoperitoneum, probable multiple bowel injuries, left common iliac artery dissection/thrombosis, L4 burst fracture, and bilateral lower abdominal wall traumatic hernias. The surgeon requested our MSTT to assist with a damage control laparotomy for 3 small bowel transections, a sigmoid colon rupture and left iliac artery injuries. After resection of devitalized bowel and stapling transected bowel ends, the rural surgeon and MSTT surgeon used a Fogarty catheter to restore adequate blood flow to the leg. Platelets brought by the MSTT corrected the coagulopathy. The MSTT transported the patient to our level one center 71 air miles away. A vascular surgeon repaired the left iliac artery injuries. In a staged fashion, we performed anastomoses of the small bowel and colon, and did a loop transverse colostomy. The abdomen was closed in stages. The patient was discharged to home in another state, and several months later had takedown of the loop colostomy and repair of the abdominal wall hernias.

CONCLUSIONS: An MSTT, consisting of a trauma surgeon, an OR scrub nurse, and critical care nurse, can bring to the rural trauma center both trauma expertise and blood components in sufficient quantity to support 3 exchange transfusions with all the appropriate clotting factors to correct or prevent a major coagulopathy. This patient with complex abdominal and spine injuries from a seat belt had improved chances for survival by timely damage control surgery, active blood component transfusion therapy, and subsequent definitive surgery at a level 1 trauma center.

NOTES

KETAMINE USE IN A FIELD AMPUTATION: MAKING THE TERRIFYING TOLERABLE

JD Aydelotte

University Medical Center Brackenridge, Austin, Texas

Presenter: Jayson Aydelotte**Senior Sponsor: Carlos Brown**

INTRODUCTION: The majority of the field amputations described in the literature discuss pain control and anesthesia for the patient in terms of narcotics with or without the addition of benzodiazepines or propofol as the primary anesthetic agent. But two major problems exist with this anesthetic/pain control regimen: 1) hypovolemia-associated hypotension and 2) inability for a patient-controlled airway, both of which become magnified in a field setting.

METHODS: We were called to a scene at a box-making factory where a worker's leg was caught in a 16-ton roller. The patient was stuck in a standing position. Emergency crews had stemmed most of his bleeding with a thigh tourniquet but he had lost an estimated 2 liters of blood. He was conscious and able to have a conversation with the surgeon. The roller could not be moved or disassembled and the patient could not be freed from the roller. The decision was made to amputate his left leg at the knee and free him from the machine.

RESULTS: Because he was in a standing position, controlling his airway was limited to supplemental O₂ via facemask and jaw thrust, if necessary. We gave the patient 25 mg (0.25 mg/kg) of Ketamine IV and the patient immediately dissociated. His amputation was completed with him seemingly conscious, controlling his airway, watching the procedure. Several more doses of Ketamine were given during the standing-phase of the operation as well as when his bleeding was controlled while he was laying down on a gurney. The patient never lost his airway. His oxygen saturation never fell below 95%. The patient was then transported to the Level I trauma center where his amputation was washed out and then formalized over the course of the next several days. The patient had no recall of the procedure but could vividly remember everything up to the initial ketamine dose.

CONCLUSIONS: Ketamine use for field amputation has not been described in the literature. Its usefulness as a dissociative anesthetic as well as an analgesic should prompt surgeons and other providers who need to perform major procedures in the field to use it as a first line agent.

APPLICABILITY OF THE NATIONAL HEALTHCARE SAFETY NETWORK'S SURVEILLANCE DEFINITION OF VENTILATOR ASSOCIATED EVENTS IN THE SURGICAL INTENSIVE CARE UNIT: A ONE-YEAR REVIEW *

C Stoeppel, E Eriksson, K Hawkins, A Eastman, S Wolf, J Minei, CT Minshall
UT Southwestern, Dallas, Texas

Presenter: Colleen Stoeppel

Senior Sponsor: Steven Wolf

INTRODUCTION: Last year, the National Healthcare Safety Network (NHSN) presented a new surveillance definition for ventilator-associated events (VAE) as a mechanism to objectively define worsening pulmonary status in ventilated patients. VAE subcategories, including ventilator-associated condition (VAC), infection-related ventilator associated condition (IVAC) and probable ventilator-associated pneumonia (PrVAP) were vetted predominantly in medical intensive care unit patients, and it is unclear how well it will characterize respiratory dysfunction in a surgical population. Surveillance using the NHSN VAE definition started in January of 2013. Our goal was to evaluate how well the NHSN's new surveillance definition characterizes pulmonary complications in a critically ill surgical patient population.

METHODS: Since 9/2012, all intubated patients admitted to our surgical intensive care unit (SICU) have been screened prospectively for VAE using the new NHSN criteria and its subcategories. In parallel, all patients were also monitored for sustained respiratory dysfunction requiring intervention that did not meet VAE criteria. We diagnosed ventilator-associated pneumonia using a clinical definition: clinical pulmonary infection score > 6 and catheter directed bronchoalveolar lavage cultures with >104 CFU per milliliter of pathogenic organisms.

RESULTS: During the 12-month study period, 704 intubated patients were admitted to our SICU. 437 were intubated for > 2 days (mean age 46 +/- 18 years, 65% male, 38% trauma, 13% burn, 21% neuro, 28% surgery, median ventilator days 4 [2, 9], median SOFA 8 [5, 10]). Evaluation of these patients using the NHSN criteria for VAE identified 37 patients with a VAC, 31 with an IVAC and 22 with PrVAP. While, the remaining 400 patients did not meet NHSN VAE, we identified 111 (28%) patients that received sustained non-physiologic elevation of ventilator support, and 98 (25%) were diagnosed with clinical pneumonia. Subsequent analysis of the 111 patients with respiratory deterioration identified 85 (77%) patients that never had a period of stable/decreasing oxygenation, as they required elevated FiO2, PEEP or mean airway pressure upon initiation of mechanical ventilation preventing them from meeting VAE criteria. Furthermore, of the 98 pneumonia patients, 10% had sustained respiratory deterioration requiring elevation in mean airway pressure, however, they also did not meet VAE criteria, as the PEEP and FiO2 were not elevated. 27% of these patients never had a period of stable/decreasing oxygenation, thus they also did not meet VAE criteria. 58% of the pneumonia patients had < 2 days of respiratory deterioration. Agreement between PrVAP and the diagnosis of clinical pneumonia was 77.3% ($\kappa = 0.243$, $p < 0.001$).

CONCLUSIONS: The applicability of the new NHSN categories of VAE to critically ill and injured surgery patients is limited, and agreement between the new diagnosis of PrVAP and clinical pneumonia in critically ill surgical patients is poor. Because most surgical patients are not well categorized by these new definitions, a better method of surveillance must be created for this patient population.

MELATONIN INHIBITS THERMAL INJURY INDUCED HYPERPERMEABILITY IN MICROVASCULAR ENDOTHELIAL CELLS *

K Wiggins-Dohlvik, MS Han, HW Stagg, ML Davis, B Tharakan
Scott and White Memorial Hospital, Temple, Texas

Presenter: Katie Wiggins-Dohlvik

Senior Sponsor: Matt L Davis

INTRODUCTION: Burns are known to induce intense systemic inflammatory reactions and hyperpermeability. The mechanisms that regulate this process are unclear but it is known that the breakdown of endothelial cell adherens junctions is integral and that reactive oxygen species play a large role in initiating this process. We hypothesized that burn induced junctional damage and hyperpermeability could be attenuated with the use of the antioxidant Melatonin.

METHODS: After IACUC approval, Sprague Dawley rats were assigned to either sham or burn groups (30% total body surface area). FITC-albumin was administered intravenously. Mesenteric post capillary venules were examined with intravital microscopy to analyze and determine the flux of the albumin from the intravascular space to the interstitium. Fluorescence intensities were measured and serum was collected. Rat lung microvascular endothelial cells were then grown as monolayers on Transwell inserts. Wells were divided into four groups (n=six) and sham serum, burn serum, Melatonin plus sham serum and Melatonin plus burn serum were applied. FITC-albumin flux across the monolayer was obtained as an indicator of permeability. Wells were again divided into four groups (as previously described). Immunofluorescence staining for the adherens junction proteins β -catenin and VE Cadherin, and rhodamine phalloidin staining for F-actin were performed. Images were obtained. Statistical analysis was conducted with a student's t-test ($p < 0.05$).

RESULTS: Data from intravital microscopy revealed a significant increase in vascular hyperpermeability following burn trauma ($p < 0.05$). Monolayer permeability was increased with burn serum when compared with sham ($p < 0.05$). However, this increase in permeability was attenuated with Melatonin treatment ($p < 0.05$). Immunofluorescence showed that damage of rat lung microvascular endothelial cell adherens

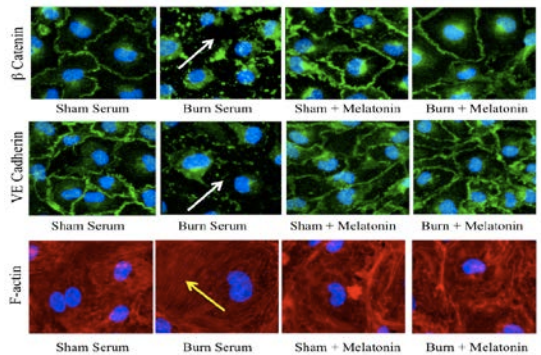


FIGURE 1: White arrows indicate damaged adherens junction proteins; yellow arrow indicates F-actin stress fibers

junctions occurred with exposure to burn serum and Melatonin restored integrity (figure 1). Rhodamine phalloidin staining showed an increase in F-actin stress fiber formation following exposure to burn serum and Melatonin decreased this (Fig 1).

CONCLUSIONS: Burns induce microvascular hyperpermeability and damage endothelial adherens junctions: Melatonin attenuates this process. This insight into the mechanisms of burn induced fluid leak confirms the role of reactive oxygen species but more importantly hints at the possibility of exciting new treatments to combat vascular hyperpermeability in burn.

IMPLEMENTATION OF A SURGICAL ICU SERVICE IS ASSOCIATED WITH IMPROVED OUTCOMES FOR TRAUMA PATIENTS *

AL Klein, CVR Brown, S Ali, M Makkhouk, J Aydelotte, B Coopwood
University of Texas Southwestern - Austin, UMC Brackenridge, Austin, Texas

Presenter: Amanda Klein

Senior Sponsor: Carlos Brown

INTRODUCTION: Trauma patients admitted to an intensive care unit (ICU) may be managed by a variety of intensivists including anesthesiology, pulmonary, and surgical intensivists. Our trauma service recently transitioned from a pulmonary ICU service to a surgical ICU service. We hypothesized that a newly formed surgical ICU service could provide comparable outcomes to the existing pulmonary ICU service. Specific aim of this study was to compare outcomes of trauma patients admitted to the ICU before and after implementation of a surgical ICU service.

METHODS: We performed a retrospective study of trauma patients admitted to the ICU of our urban, ACS-verified, level 1 trauma center over a four-year period (2009-2012). Patients managed by the pulmonary ICU service (PulmICU group, 2009-2010) were compared to patients managed by a surgical ICU service (SICU group, 2011-2012). The PulmICU service was composed of 10 rotating pulmonary intensivists providing coverage of trauma ICU patients 24/7, while the SICU service was composed of four rotating general/trauma surgeons covering trauma ICU patients 24/7. The primary outcome was mortality while secondary outcomes included complications (pulmonary, infectious, cardiac, and thromboembolic), hospital and ICU length of stay, ventilator days, and need for reintubation.

RESULTS: There were 2,253 trauma patients admitted to the ICU during the study period, 1,124 and 1,129 managed by the PulmICU and SICU service, respectively. Patients in the SICU group were older (47 vs. 44 years old, $p = 0.01$) and more often sustained blunt trauma (91% vs. 87%, $p = 0.002$) but there was no difference in race, gender, admission physiology, need for mechanical ventilation, injury severity, or distribution of injuries. When comparing outcomes for SICU and PulmICU patients there was no difference in mortality (11% vs. 13%, $p = 0.41$), but patients managed by the SICU service had fewer pulmonary complications (3% vs. 6%, $p < 0.001$), fewer days on the ventilator (3 vs. 4, $p = 0.002$), and less often required reintubation after extubation (4% vs. 9%, $p < 0.001$). There was no difference for other categories of complications, hospital or ICU length of stay. Similar outcomes were seen for subgroups of patients ≥ 65 years old and patients requiring at least one day of mechanical ventilation.

CONCLUSIONS: Transition from a pulmonary ICU service to a surgical ICU service at our institution was associated with no change in mortality but an improvement in pulmonary complications, ventilator days, and reintubation rates. Trauma centers currently staffed with a pulmonary ICU service should feel comfortable converting to surgical ICU service and should expect comparable or improved outcomes for trauma patients admitted to the ICU.

DOES SIMPLE IMAGING HOLD THE KEY: USING CXR AS A SCREENING TOOL TO IDENTIFY SEVERE PULMONARY CONTUSION *

EJ Helmer, MG Ochsner

Memorial University Medical Center, San Diego, California

Presenter: Elizabeth Helmer**Senior Sponsor: M. Gage Ochsner**

INTRODUCTION: In blunt chest trauma, pulmonary contusion (PC) is the most frequent injury identified. Previous studies have shown that complications occur more frequently when PC are greater than 20-24% of total lung volume. Subsequent complication rates including ventilator dependent respiratory failure (VDRF), acute respiratory distress syndrome (ARDS), and pneumonia are up to 50%. Our prior work with blunt chest trauma demonstrated the ability of chest CT to accurately estimate the total lung volume of the affected parenchyma in adult trauma patients. On CT each anatomic lobe represents approximately 20% to the total lung volume (TLV) with the exception of the right middle lobe (10% of TLV). Plain film chest x-ray (CXR) has been shown to underestimate the degree of PC compared to chest CT. We set out to determine if the visualization of PC on CXR could be used as a screening tool to identify patients who had sustained PC $\geq 20\%$. Complication risk between PC $< 20\%$ and those $\geq 20\%$ was also determined.

METHODS: This is a prospective study of adult Level 1 blunt trauma patients. By protocol all patients underwent an admission CXR and chest CT. The initial CXR was read to determine if there was evidence of a PC. Using standardized lung volumes, chest CT allowed for quantification, in percentage, of the PC. We determined the sensitivity and specificity of the admission CXR to identify a pulmonary contusion size of $\geq 20\%$ as defined by the chest CT. To ensure pulmonary complications were not due to associated injuries, patients were excluded if they had a coexisting traumatic brain injury (TBI), GCS

RESULTS: 125 patients met inclusion criteria and were included in this study. Using standard lung volumes, PC volumes were quantified with an average at 13.4% of TLV. 88 patients had PC

CONCLUSIONS: The simple presence of PC on the initial CXR is an adequate screening tool to identify severe PC, or PC $\geq 20\%$. These patients have an increased risk of complications and greater resource utilization. Using this information, resources can be utilized more effectively with patients quickly and appropriately triaged to a trauma center.

URINARY TRACT INFECTION IN ELDERLY TRAUMA PATIENTS: REVIEW OF THE TRAUMA QUALITY IMPROVEMENT PROJECT DATABASE IDENTIFIES THE POPULATION AT RISK*

MD Zielinski, KM Thomsen, M Amr, M Alzghari, DH Jenkins, AM Pakula, SP Zietlow, EB Habermann

Mayo Clinic, Rochester, MN

Presenter: Stephanie Polites

Senior Sponsor: Scott Zietlow

INTRODUCTION: Elderly trauma patients are at high risk for urinary tract infection (UTI). Despite this, UTI has been deemed a potentially preventable problem, and therefore not reimbursable, by the Centers for Medicare and Medicaid Services (CMS). Early identification of UTI in these patients should lead to prompt treatment, improved outcomes and cost savings. Risk factors for UTI development in this population, however, must be elucidated in order to realize these goals.

METHODS: The Trauma Quality Improvement Project (TQIP) database, a partnership of over 180 trauma centers, was used to analyze elderly patients (≥ 65 years) admitted as a result of injury during 2011. Patients with genitourinary injuries or dialysis prior to admission were excluded. Multivariable logistic regression analysis was conducted to identify UTI risk factors. Mean cost of UTI was calculated based on the assumption of \$862 - \$1,007 per UTI. Data are presented as odds ratios (OR) with 95% confidence intervals (CI).

RESULTS: In total, 34,693 patients were identified; 1492 developed UTI (4.3%). Multiple significant risk factors were identified, including: age > 75 years, female sex, ascites, moderate head injury, impaired sensorium, congestive heart failure (CHF), and prolonged duration of hospital stay (**Table**). Urinary catheterization statistics are not captured in the TQIP database. Assuming UTIs diagnosed on hospital day 1 were pre-existing, the cost of UTI to TQIP hospitals ranged from \$1,280,959 to \$1,496,434 per year.

Variable		OR	95% CI
Age (vs 65-69 years)	70-74	1.13	0.92-1.38
	*75-79	1.40	1.16-1.70
	*80-84	1.41	1.17-1.70
	*85-89	1.58	1.30-1.91
*Female sex		1.87	1.65-2.11
Injury Severity Score (vs 0-9)	10-16	0.98	0.85-1.13
	17-25	0.97	0.83-1.13
	26+	1.20	0.98-1.46
Glasgow Coma Scale (vs 3-8)	13-15	1.12	0.89-1.42
	*9-12	2.03	1.49-2.77
*Hx of ascites		3.4	1.04-11.2
*Hx of CHF		1.2	1.02-1.44
*Hx of impaired sensorium		1.26	1.08-1.47
Duration of stay (vs 3-5 days)	*1	0.13	0.05-0.35
	*2	0.43	0.27-0.68
	*6-8	2.34	1.94-2.82
	*9-14	5.10	4.3-6.11
	*15-21	9.39	7.62-11.6
	*22+	18.0	14.7-22.1
*p<0.05			

CONCLUSION: Prolonged duration of stay has a profound impact on the development of UTIs in elderly trauma patients but overall severity of injury does not. In addition, multiple non-modifiable risk factors were identified prompting the possibility for increased screening of occult UTIs. Reimbursement for care of UTI in this complicated patient population should be revisited. The TQIP database must incorporate urinary catheter data.

BELOW-THE-KNEE ARTERIAL INJURY: THE TYPE OF VESSEL MAY BE MORE IMPORTANT THAN THE NUMBER OF VESSELS INJURED *

JR Scalea, RS Crawford, S Scurci, J Danquah, J O'Connor, R Sarkar, TM Scalea
University of Maryland, Baltimore, Maryland

Presenter: Joseph Scalea

Senior Sponsor: Thomas Scalea

INTRODUCTION: Introduction: Little is written about limb salvage following below-the-knee (BTK) arterial trauma. Based on published data and clinical experience we hypothesized that any single, patent vessel BTK would allow for limb viability and salvage.

METHODS: Methods: The Trauma Center registry was retrospectively queried, from 2007 through 2012, for patients presenting with BTK vascular injuries (BTKVI), we defined as injury below the popliteal artery. Cox regression, ANOVA, and student's T-Test were used to analyze data.

RESULTS: Results: 122 patients were identified. The mean age was 35 years, 84% were male, 43% were non-Caucasian. 75 (61%) sustained blunt and 39 (32%) penetrating injury. 51(41%) had an injury to a single BTK vessel and only 23.5% of these underwent attempted repair. 100% of patients with 2 vessel, and 50% of 3 vessel BTKVIs had attempted repair. No patient had endovascular repair. Amputation need was not associated with ISS, gender, or age ($p=NS$). Patients with blunt injury had higher amputation rates (26.8% vs 7.5%, $p=0.01$). The amputation rate for injuries to a single BTK vessel was 17.6%; when either 2 or 3 vessels were injured, amputation rates were 29% and 50%, respectively. Of those with single vessel injuries, 25 were posterior tibial (PT) artery injuries, 17 anterior tibial (AT) and 9 had a peroneal (P) injury. In patients with a single-vessel injured following blunt trauma, an injured AT was associated with a higher amputation rate when compared with those patients with either PT or P injuries (37.5% vs 8.8%, $p=0.045$). The odds ratio of requiring an amputation after an injury to the AT alone was 22.4 ($p=0.02$). None of the 3 patients (0%) with combined PT and P injuries (intact AT) required amputation.

CONCLUSIONS: Conclusions: BTKVIs are uncommon. Multiple BTKVIs are more likely to have repair. Amputation was more common following blunt versus penetrating trauma. In contrast to the commonly taught surgical dogma which suggests that any intact single vessel BTK is associated with limb salvage, single AT vessel injuries were associated with much higher rates of amputation when compared with P or PT injuries.

MISLED BY TEG? THE EFFECTS OF ALCOHOL ON COAGULATION IN TRAUMA PATIENTS *

BM Howard, LZ Kornblith, BJ Redick, RF Vilardi, KS Balhotra, JM Crane, MR Forde, MF Nelson, RA Callcut, MJ Cohen
UCSF/SFGH, San Francisco, California

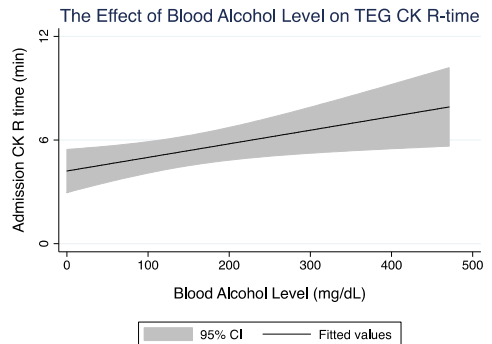
Presenter: Benjamin Howard

Senior Sponsor: Mitchell Jay Cohen

INTRODUCTION: The effects of alcohol on coagulation after trauma remain unclear. In vitro studies suggest that alcohol may decrease clot strength and inhibit fibrinolysis. Observational data in healthy patients has shown that alcohol leads to altered thromboelastography (TEG) parameters, with impaired clot formation and hypocoagulability. Studies of traumatic brain injured patients, however, indicate that alcohol may have protective effects on outcome, which are modulated through protection against coagulopathy. We hypothesized that critically injured patients with elevated blood alcohol levels (EtOH) would appear hypocoagulable by TEG, but show no other evidence of coagulopathy or its clinical sequelae.

METHODS: Longitudinal plasma samples were prospectively collected from 415 critically-injured trauma patients at a single Level 1 Trauma Center and were matched with demographic and outcome data. Citrated kaolin (CK) TEG and standard coagulation measures were performed in parallel. Coagulopathy by standard measures was defined by INR ≥ 1.3 . Massive transfusion was defined by ≥ 10 units of pRBCs transfused in 24 hours.

RESULTS: 264 patients (63.3%) had detectable EtOH ($>10\text{mg/dL}$). These alcohol-positive patients were primarily male (87% vs. 79%), bluntly injured (77% vs. 59%), and had lower median GCS (9.5 vs. 14, all $p<0.05$) than EtOH-negative patients; however, there were no notable differences in pH (7.29 vs. 7.31, $p=\text{NS}$) or injury severity (median ISS 11 vs. 14, $p=\text{NS}$) between groups. Alcohol-positive patients had a prolonged TEG CK R-time (5.91m vs. 4.43m, $p=0.013$), prolonged K-time (1.77m vs. 1.43m, $p=0.036$), and decreased alpha angle (66.5° vs. 70.2° , $p=0.001$). In multiple linear regression, for every 10mg/dL increase in EtOH, R-time was prolonged by 3.84s ($p=0.015$, Figure), and alpha angle decreased by 0.11° ($p=0.013$). However, in multiple logistic regression analyses, EtOH was a negative predictor of coagulopathy by INR (>1.3), and was not predictive of transfusion requirements or early or late mortality.



CONCLUSIONS: Patients with elevated blood alcohol levels present with impaired clot formation as assayed by TEG, but this does not appear to correlate to standard measures of coagulopathy or to outcome, measured by transfusion requirement or mortality. Reliance on TEG for determining coagulopathy in intoxicated trauma patients may lead to a misperceived “hypocoagulable state” and subsequent inappropriate resuscitation and transfusion. TEG appears to be affected by alcohol in a previously unreported way, warranting further investigation.

MODULATION OF INTESTINAL MUCUS PHYSIOCHEMICAL PROPERTIES AND PROTECTION AGAINST OXIDANT INJURY: THE FIRST LINE OF INTESTINAL BARRIER DEFENSE BY ESTROGEN *

ME Diebel, LN Diebel, DM Liberati, CW Manke
Wayne State University, Detroit, Michigan

Presenter: Mark E. Diebel

Senior Sponsor: Lawrence N. Diebel

INTRODUCTION: The intestinal epithelial barrier and the intestinal mucus layer may be protective against trauma/hemorrhage (T/HS) induced injury in females. This effect is related to estradiol (E) concentrations and varies with the menstrual cycle. We examined the ability of E to impact the physiochemical properties of intestinal mucus and to protect against oxidant related injury to the mucus and underlying intestinal epithelial barrier in an in vitro model.

METHODS: Nonmucus producing (HT29) and mucus producing (HT29-MTX) intestinal epithelial cells (IEC) were exposed to E (90 pg/ml) or no E for 3 days in tissue culture flasks and then grown to confluence on transwell plates. Non-adherent and adherent mucus content was indexed by analysis of mucin using an ELISA. In other experiments, mucus viscosity (cp) and elasticity (G') were determined by rheometry. In additional experiments IEC groups were exposed to hydrogen peroxide (H₂O₂, 250µM for 30 min.) and IEC apoptosis and permeability (FITC-Dextran, FD4) determined. Oxidative damage to the mucus layer was also determined by measuring lipid hydroperoxide, LPO, and protein carbonyl content, PC.

RESULTS: Mean ± S.D. (N = 5 for each group)

	Non-adherent mucus (ng/ml)	Adherent mucus (ng/ml)	Viscosity (cp)	Elasticity (G') (dyne/cm ²)
HT29	0	0	1.1 ± 0.1	0
MTX	4.9 ± 0.2*	6.4 ± 0.3*	5.4 ± 0.2*	10,400 ± 170*
MTX + E	8.9 ± 0.6*#	10.1 ± 0.6*#	10.5 ± 0.2*#	74,500 ± 200*#
	Apoptosis (%)	Perm. (nmol/cm ² /hr)	LPO (pmol/mg)	PC (nmol/mg)
MTX	4.6 ± 0.4	0.28 ± 0.02	1.6 ± 0.1	1.3 ± 0.1
MTX + H ₂ O ₂	14.8 ± 1.0#	0.62 ± 0.05#	7.8 ± 0.3#	3.9 ± 0.1#
MTX + H ₂ O ₂ + E	6.5 ± 0.5\$	0.35 ± 0.04\$	2.2 ± 0.3\$	2.5 ± 0.2\$

*p<0.001 vs. HT29, #p<0.001 vs. MTX, \$p<0.001 vs. MTX + H₂O₂

CONCLUSIONS: Treatment with the female sex hormone E increased mucin content and altered viscosity and elasticity of the mucus layer. These physiochemical changes were associated with a protective effect against oxidant damage to the mucus layer and underlying IEC barrier. Gender differences in gut barrier function following T/HS may in part be related to differences in intestinal mucus content and the resultant physiochemical and oxidant resistant properties of the mucus layer.

CRYSTALLOID ADMINISTRATION DURING TRAUMA RESUSCITATION: DOES LESS REALLY EQUAL MORE? *

JP Sharpe, LJ Magnotti, TJ Schroepfel, EM Paulus, MA Croce, TC Fabian, JA Weinberg
Department of Surgery, University of Tennessee Health Science Center, Memphis, Tennessee

Presenter: John Sharpe

Senior Sponsor: Jordan Weinberg

INTRODUCTION: Current direction in trauma resuscitation has diverged from historical ATLS guidelines, emphasizing early administration of blood products while minimizing crystalloid resuscitation. Although evidence suggests that higher crystalloid volume over the first 24 hours is associated with negative outcomes, the effect of crystalloid administration during initial resuscitation remains unclear. The purpose of this study was to evaluate the impact of the ratio of crystalloid to RBC (C:RBC) in the resuscitation room on mortality.

METHODS: Over 6.5 years at a level 1 trauma center, prospective data were collected on patients that received > 1 RBC unit in the resuscitation room. C:RBC was defined as the ratio of crystalloid infused in liters (L) to the units of RBCs transfused in the resuscitation room. Patients were stratified by high-ratio (>0.75) vs. low-ratio (<0.75). Stepwise multiple logistic regression analysis (MLR) was performed to determine variables significantly associated with mortality.

RESULTS: 383

patients met study criteria: 192 (50%) high-ratio and 191 (50%) low-ratio. The high-ratio group received significantly more crystalloid (4 vs. 1.3 L, $p < 0.05$) and less RBC (2.7 vs. 4 units, $p < 0.05$)

	In-hospital Mortality	24-hour Mortality
C:RBC	0.98 (0.57 - 1.69)	0.90 (0.49 - 1.66)
Blunt Injury	1.12 (0.57 - 2.18)	0.79 (0.37 - 1.68)
Admission Heart Rate	0.99 (0.99 - 1.01)	1.00 (0.99 - 1.01)
Injury Severity Score	1.05 (1.03 - 1.07)*	1.04 (1.02 - 1.07)*
Admission Base Excess	0.95 (0.91 - 0.99)*	0.95 (0.90 - 1.00)
Colloid in Resus room	1.24 (0.69 - 2.24)	1.32 (0.66 - 2.55)
Plasma in Resus Room	0.83 (0.62 - 1.13)	0.96 (0.66 - 1.39)
Platelets in Resus Room	0.53 (0.18 - 1.54)	<0.001 (<0.001 - >999)
RBC over 24 hours	1.02 (1.01 - 1.04)*	0.99 (0.97 - 1.02)
Time in Resus Room	1.01 (1.01 - 1.03)*	1.01 (1.00 - 1.03)

compared to the low-ratio group. While the high-ratio group had higher admission heart rate (118 vs. 112, $p < 0.05$) and Injury Severity Score (ISS) (32 vs. 27, $p < 0.05$), no difference was seen with respect to ventilator free days (3.4 vs. 3.8, $p = 0.51$), PaO₂:FiO₂ at 48 hours (234 vs. 287, $p = 0.12$), in-hospital mortality (34% vs. 28%, $p = 0.16$), or 24-hour mortality (21% vs. 17%, $p = .38$). MLR found ISS, admission base excess, 24-hour transfusion, and time in the resuscitation room, but not C:RBC, to be significantly associated with in-hospital mortality, and only ISS was significantly associated with 24-hour mortality (Table).

CONCLUSIONS: In this cohort of patients receiving RBC in the resuscitation room, factors related primarily to injury severity were associated with mortality, but C:RBC was not. Pertaining to initial resuscitation, the purported benefit of crystalloid limitation was not observed.

NOTES

ACUTE RESPIRATORY DISTRESS SYNDROME RISK FACTORS FOR INJURED PATIENTS UNDERGOING DAMAGE CONTROL LAPAROTOMY: AAST MULTICENTER, PROSPECTIVE, OBSERVATIONAL TRIAL

MD Zielinski, DH Jenkins, BA Cotton, K Inaba, G Vercruysse, R Coimbra, CVR Brown, L Allen, J DuBose, TM Scalea
Mayo Clinic, Rochester, Minnesota

Presenter: Martin Zielinski

Senior Sponsor: Thomas Scalea

INTRODUCTION: Severely injured patients undergoing damage control laparotomy (DCL) have multiple risk factors for Acute Respiratory Distress Syndrome (ARDS) making it challenging to differentiate the contributions of individual features. We aimed to determine the relative contributions of ARDS risk factors hypothesizing that greater volumes of crystalloid would be a leading feature.

METHODS: Analysis of the prospectively collected AAST Multi-institutional Open Abdomen Database was performed. Inclusion criteria were any patient, age ≥ 18 years, who underwent DCL at one of the 14 participating Level I trauma centers. Univariable and multivariable Cox regression analyses were performed to determine the association of variables with the development of ARDS during hospitalization. Data are summarized as mean \pm standard deviation, percentages, and hazard ratios (HR) with 95% confidence intervals (CI). A p-value < 0.05 was considered significant.

RESULTS: A total of 562 patients (78% men, age 40 ± 18 years) were identified of whom 77 developed ARDS (14%). Overall mortality was 23% with a 39% mortality rate for ARDS patients. Univariable analysis demonstrated that Injury Severity Score (ISS; HR 1.03, 95% CI 1.02-1.05), intraoperative (IO) estimated blood loss (EBL; HR 1.09, 95% CI

Features	Hazard Ratio	95% CI
Age (per 10 years)	1.07	0.91-1.26
Female sex	1.43	0.67-3.08
ISS* (per point)	1.03	1.01-1.05
Hemoglobin (grams/deciliter)	1.05	0.95-1.16
IO red blood cell Tx (per liter)	1.00	0.86-1.17
IO plasma Tx (per liter)	1.13	0.87-1.46
IO platelet Tx (per liter)	1.06	0.80-1.41
IO EBL (per liter)	1.01	0.90-1.13
IO crystalloid (per liter)	1.01	0.88-1.16
IO plasma (per liter)	1.13	0.87-1.46
Total IO fluid balance (per liter)	0.99	0.89-1.10
24-hour colloid volume (per liter)	1.02	0.97-1.07
24-hour crystalloid volume (per liter)*	1.01	1.00-1.01
Table	*statistically significant	

1.04-1.13), IO plasma transfusion (Tx; HR 1.17, 95% CI 1.10-1.25), 24-hour colloid volume (HR 1.07, 95% CI 1.04-1.10) and 24-hour crystalloid volume (HR 1.01, 95% CI 1.00-1.01) were associated with the development of ARDS. Multivariable analysis of clinically and statistically significant features, inclusive of patients surviving > 24 hours but excluding ARDS prior to hospital day 2, are presented in the Table.

CONCLUSIONS: Severity of injury and greater crystalloid resuscitation volume at 24 hours were independently associated with ARDS development but blood product volumes were not. Avoiding large volumes of crystalloid during resuscitation may reduce rates of ARDS in this critically injured population.

NOTES

THE EFFECT OF LACTATE ON PULMONARY MORBIDITY IN FEMORAL SHAFT FRACTURES TREATED WITH EARLY TOTAL CARE: DOES PERSISTENT OCCULT HYPOPERFUSION MATTER? *

JE Richards, P Matuszewski, OD Guillamondegui, SM Griffin, DM Koehler, RV O'Toole, MJ Bosse, WT Obremsky, JM Evans
Vanderbilt University Medical Center, Nashville, Tennessee

Presenter: Justin Richards

Senior Sponsor: Rick Miller

INTRODUCTION: Lactate is often utilized as a marker of hemodynamic resuscitation in trauma patients. Previous literature suggests that occult hypoperfusion (OH), defined as lactate ≥ 2.5 mmol/L, prior to reamed intramedullary nailing (IMN) of femur fractures was predictive of increased complications. We hypothesize that elevated lactate levels are associated with increased post-operative pulmonary morbidity in multi-trauma patients requiring IMN.

METHODS: Retrospective cohort study over a 5-year period from three academic trauma centers. Inclusion criteria: age ≥ 18 years, Injury Severity Score (ISS) ≥ 17 , femoral shaft fracture treated within 24 hours with a reamed IMN, admission lactate ≥ 2.5 mmol/L. Demographics, injury characteristics, and inpatient data as well as admission and preoperative serum lactate values were collected. Persistent OH was considered for a preoperative lactate ≥ 2.5 mmol/L. Primary outcome: duration of mechanical ventilation ≥ 5 days. Secondary outcomes: initial 24-hour packed red blood cells (PRBC) and total ventilator days. Multivariable logistic regression modeling was performed to evaluate the effect of admission and preoperative lactate on pulmonary morbidity after controlling for confounding variables.

RESULTS: 414 patients identified; 294/414 (71.0%) with admission lactate ≥ 2.5 mmol/L. No difference in primary outcome among the groups (86/294, 29.3% vs. 28/120, 23.3%; $p=0.22$). Mean ISS was 27.0 (SD: 8.9). Median admission lactate: 3.7 (IQR: 3-4.6) and median preoperative lactate: 2.8 (IQR: 1.9-3.5); 184/294 (62.6%) demonstrated OH prior to fracture fixation. No significant difference in persistent OH and admission Glasgow Coma Scale (GCS) score (13.5 ± 3.2 vs. 12.7 ± 4.1 , $p=0.06$), ISS (26.2 ± 8.7 vs. 27.6 ± 9.1 , $p=0.19$), or the primary outcome (50/184, 27.2% vs. 36/110, 32.7%; $p=0.31$). There was no difference in primary outcome when OH was considered separately for a preoperative lactate ≥ 3.0 (34/123, 27.6% vs. 52/171, 30.4%; $p=0.61$), ≥ 3.5 (21/79, 26.6% vs. 65/215, 30.2%; $p=0.54$), or ≥ 4.0 (14/50, 28.0% vs. 72/244, 29.5%; $p=0.83$). No difference in persistent OH and secondary outcomes: PRBC (1.4 ± 4.5 vs. 1.9 ± 3.4 , $p=0.34$) or vent days (4.8 ± 9.9 vs. 3.9 ± 6.0 , $p=0.41$). Multivariable regression demonstrated admission lactate was significantly associated with the primary outcome (OR: 1.28, 95% CI: 1.05-1.56) after controlling for age, admission GCS, chest Abbreviated Injury Scale (AIS) score, abdominal AIS, and admission glucose; however, preoperative lactate was not a risk factor (OR: 0.88, 95% CI: 0.68-1.13) for pulmonary morbidity.

CONCLUSIONS: Contrary to previous studies, we demonstrate that persistent OH prior to reamed IMN is not associated with pulmonary morbidity in multi-trauma patients with a femoral shaft fracture treated with early definitive fixation.

EFFECT OF PHARMACOLOGICAL RESUSCITATION ON BRAIN GENE EXPRESSION PROFILES IN A LARGE ANIMAL MODEL OF COMBINED TRAUMATIC BRAIN INJURY AND HEMORRHAGIC SHOCK *

SE Dekker, T Bambakidis, M Sillesen, B Liu, CN. Johnson, G Jin, Y Li, HB Alam
University of Michigan Hospital and Department of Anesthesiology, Ann Arbor, Michigan
VU University Medical Center, Amsterdam, the Netherlands

Presenter: Simone Dekker

Senior Sponsor: Hasan Alam

INTRODUCTION: We have previously shown that addition of valproic acid (VPA, a histone deacetylase inhibitor) to hetastarch (HEX) resuscitation significantly decreases lesion size in a swine model of traumatic brain injury (TBI) and hemorrhagic shock (HS). However, the precise mechanisms have not been well defined. As VPA is a transcriptional modulator, the aim of this study was to investigate its effect on brain gene expression profiles.

METHODS: Swine were subjected to controlled TBI and HS (40% blood volume), kept in shock for two hours, and resuscitated with HEX or HEX+VPA (n=5/group). Following six hours observation, brain RNA was isolated, and gene expression profiles were measured using a Porcine Gene ST 1.1 microarray (Affymetrix, CA). Pathway analysis was done using network analysis tools Gene Ontology (GO), Ingenuity Pathway Analysis (IPA), and Parametric Gene Set Enrichment Analysis (PGSEA). Real-time polymerase chain reaction (PCR) was employed to verify the key microarray findings.

RESULTS: 1668 probesets mapping to 370 known genes were differentially expressed between the HEX and HEX+VPA groups. Expression of apoptotic genes differed between groups (Fig.1A), and biological function analysis predicted a significant down-regulation of apoptosis ($p=1.29 \times 10^{-12}$), cell death ($p=8.46 \times 10^{-12}$) and necrosis ($p=9.07 \times 10^{-11}$). Pathway analysis indicated a significant modulation of pathways involved in cell signaling, dendritic cell response, and the complement system (Fig. 1B).

CONCLUSIONS: This is the first high-throughput analysis of cerebral gene profiling following TBI+HS. It shows that treatment with VPA significantly alters early transcription of pathways related to cell survival and inflammation, which may explain its neuroprotective effects.

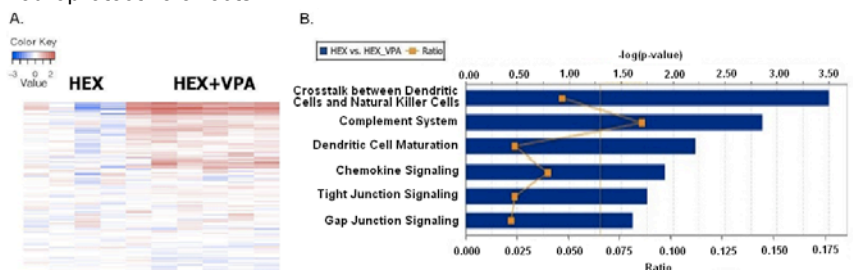


Figure 1:
A. Heat map from the microarray data. Each row represents a gene related to apoptosis. Each column represents an individual sample from the HEX or HEX+VPA group. Up- and down-regulated genes are shown in shades of red and blue colors, respectively.
B. Genetic pathways with the greatest modulation in gene expression between HEX and HEX+VPA groups. P-values indicate significance of mapping to the pathway while ratio (yellow line) indicates number of identified genes divided by total number of genes in the pathway.

DETECTION OF LOW VOLUME BLOOD LOSS: THE COMPENSATORY RESERVE INDEX VERSUS*

CL Stewart, J Mulligan, GZ Grudic, VA Convertino, SL Moulton
University of Colorado, School of Medicine, Aurora, Colorado

Presenter: Camille Stewart

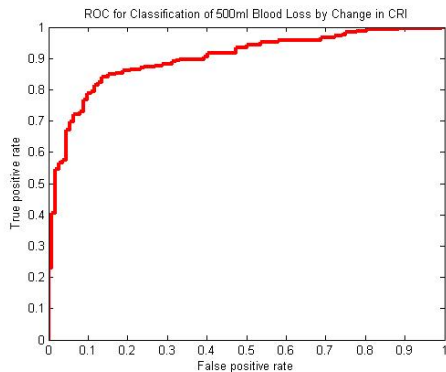
Senior Sponsor: Steve Moulton

INTRODUCTION: Humans are able to compensate for acute blood loss through various neural, hormonal, and respiratory mechanisms. We hypothesized that a novel computational algorithm, which analyzes pulse oximetry waveforms to continuously estimate the Compensatory Reserve Index (CRI), could detect low volume blood loss. CRI is a physiological measure of compensation, which corresponds with changes in central volume. A CRI of “1” represents supine normovolemia and “0” represents the circulatory volume at which hemodynamic decompensation will occur; values between 1 and 0 indicate the proportion of reserve remaining.

METHODS: Subjects underwent voluntary blood donation (~450 ml). Demographics and continuous noninvasive vital sign waveform data were collected, including HR, pulse oximetry, SBP, cardiac output, and stroke volume. The volume of blood outflow was continuously measured and synchronized with the pulse oximetry waveforms. The pulse oximetry waveforms were later processed by the algorithm to estimate continuous, beat-to-beat CRI values.

RESULTS: Data were collected from 244 healthy volunteer subjects (79 males/165 females) with a mean age (\pm SD) of 40.1 ± 14.2 years. A control subgroup of 122 subjects (29 males/83 females) with a mean age 37.5 ± 13.3 was monitored for > 4 minutes before their blood draw. Using a change in CRI of 0.05 or more between the beginning and end of monitoring as the measure for classification of 450 ml of blood loss, the sensitivity of this classifier

is 0.84 and the specificity is 0.86, while the area under the ROC curve is 0.90 (Figure). CRI significantly decreased in $92\% \pm 4\%$ of subjects (0.05α), whereas significant changes in SBP, HR, CO and SV were observed in 52%, 65%, 47% and 74% of the subjects respectively, when sampled in a statistically similar population of 204 subjects.



CONCLUSIONS: CRI values derived from pulse oximetry waveforms effectively identify low volume blood loss (450 ml) and CRI is more discriminative than traditional physiological measures. These findings warrant further evaluation of the CRI algorithm in actual trauma settings.

NOTES

DIFFERENCES IN ACUTE KIDNEY INJURY AND DEATH BETWEEN BLUNT AND PENETRATING TRAUMA AFTER RESUSCITATION WITH HETASTARCH *

CJ Allen, EJ Valle, JM Jouria, N Namias, AS Livingstone, CI Schulman, and KG Proctor
University of Miami Miller School of Medicine, Miami, Florida

Presenter: Casey Allen

Senior Sponsor: Nicholas Namias

INTRODUCTION: In June 2013, the FDA issued a Black Box Warning on the use of hydroxyethyl starch (HES) based on reports of increased mortality and acute kidney injury (AKI) in critically ill patients. In our previous studies, we observed that HES reduced transfusion requirements and early mortality in penetrating trauma patients. We test the hypothesis that a one-time, low-volume HES bolus during initial resuscitation of trauma patients effects the overall rate of AKI and mortality.

METHODS: Every trauma admission from October 2011 to December 2012 was reviewed. Endpoints were the development of AKI (RIFLE criteria) and mortality from 90 days of admission. Patients who died within 24 hours were excluded. After factors associated with mortality and AKI were identified, multiple logistic regression analysis defined the individual predictors. Factors included in the regression model were initial systolic blood pressure (SBP), heart rate, hematocrit, base deficit, Injury Severity Score (ISS), presence of traumatic brain injury (TBI), blood transfusion, urgent operative intervention, and use of HES.

RESULTS: 1410 patients were reviewed; average age 42 yrs, 77% male, 32% penetrating trauma, average ISS 14.8, with an overall mortality of 6.7%. Within the first 24 hours of arrival, 227 patients received HES (0.5-1.5L). Results of multiple logistic regression analysis are below.

	Blunt (n=952)				Penetrating (n=458)			
	AKI		Death		AKI		Death	
	OR	p=	OR	p=	OR	p=	OR	p=
SBP	1.02	NS	0.98	.023	1.00	NS	0.95	<.001
Hematocrit	0.91	<.001	0.91	<.001	0.88	<.001	0.86	<.001
ISS	1.03	.01	1.05	.001	1.12	<.001	1.11	.002
TBI	1.95	.045	3.21	.01	0.62	NS	0.0	NS
Transfusion	.82	NS	1.1	NS	0.22	.048	0.48	NS
HES	2.47	.004	2.52	.023	1.9	NS	3.5	NS

NS: Not Significant

CONCLUSIONS: HES is an independent risk factor in the development of AKI and death in the blunt, but not the penetrating, population.

A CRITICAL ANALYSIS OF SECONDARY OVERTRIAGE TO A LEVEL-I TRAUMA CENTER

A Tang, A Hashmi, B Zangbar, N Kulvatunyou, B Joseph, R Frise, DJ Green, G Vercruysse, L Gries, T O'Keeffe, P Rhee
University of Arizona, Tucson, Arizona

Presenter: Andrew Tang

Senior Sponsor: Peter Rhee

INTRODUCTION: Trauma centers often receive transfers from lower-level trauma centers or non-trauma hospitals. The aim of this study was to analyze the incidence and pattern of secondary overtriage to our Level-I trauma center.

METHODS: We performed a 2-year retrospective analysis of all trauma patients transferred to our level-I trauma center and discharged within 24-hours of admission. Reason for referral, referring specialty, mode of transfer, and intervention details were collected. Major procedure was defined as surgical intervention in the operating room. Minor procedures were defined as procedures performed in the emergency department.

RESULTS: All patients were transferred from non-trauma facilities, as our hospital is the only regional trauma center for a population of over 1 million. A total of 1,846 patients were transferred to our level-I trauma center, of which 440 (24%) were discharged within 24-hours of admission. The mean age was 35 ± 21 ; 66% were male; and mean Injury Severity Score (ISS) 4 ± 4 . The most common reasons for referral were extremity fractures (31%), followed by head injury (23%) and soft tissue injuries (13%). Of the 440 patients discharged within 24 hours, 380 (86%) patients required only observation ($n=268$, 61%) or minor procedure (112, 25%). Fracture management ($n=47$, 42%) and wound care ($n=65$, 58%) were the most common minor procedures performed. The mean interfacility transfer distance was 45 ± 46 miles. Mean hospital charges per transfer were $12,549 \pm 5,863$ \$.

Transfers discharged within 24-hours	
N=440	
Referring Specialties	
Emergency Medicine	262 (59.3%)
Family Medicine	74 (16.7%)
Internal Medicine	35 (7.9%)
Interventions	
Observation	268 (60.9%)
Minor Procedures	112 (25.4%)
Major Procedures	61 (13.8%)
Consultations Obtained	
Radiology	340 (77%)
Orthopedics	80 (18%)
Facial Plastics	50 (11%)

CONCLUSIONS: A significant number of patients transferred to our trauma center were discharged within 24-hours, most of them required observation and/or minor procedures. Appropriately increasing primary hospital resources, in addition to inter-hospital outreach in the form of education or telemedicine should be considered in order to decrease the number of avoidable transfers.

SEASONAL VARIATIONS IN POST-TRAUMATIC OSTEOMYELITIS AFTER OPEN EXTREMITY FRACTURES

HC Sagi, G Herzog, S Cooper, D Donahue, S Marberry, B Steverson
Florida Orthopaedic Institute, Tampa, Florida

Presenter: Henry Claude Sagi

Senior Sponsor: Henry Claude Sagi

INTRODUCTION: The vast majority of the orthopedic trauma literature has dealt with risk of infection as a function of time to debridement and severity of open fracture. According to Carsenti-Etesse, the incidence of infection is 0-9% for grade I fractures, 1-12% in grade II fractures, and 9-55% in grade III fractures. With the increasing incidence of community acquired MRSA infections there is an unquestionable shift in the causative organisms associated with post-traumatic wound infections and osteomyelitis seen with open fractures. The goal of this analysis was to determine if the either the incidence or causative organism of post-traumatic infection varies with the season in which the open fracture occurred. No such study has been previously published. **Hypothesis:** The incidence and causative organisms related to post-traumatic wound infection after open fractures of the extremity will vary with the season in which the injury occurs. **Setting:** Level One Regional Referral Trauma Center.

METHODS: Retrospective chart review of all skeletally mature patients sustaining an open fracture of either the upper or lower extremity from 2007 to 2012. Charts were reviewed to extract information regarding date of injury, type of injury, Gustillo-Anderson grade of open fracture, prophylactic antibiotics administered, surgical treatment, any subsequent treatment (operative or non-operative) for a post-traumatic wound infection, and the causative organisms. Patients were placed into one of four groups based on the time of year: Group 1 (Spring) March, April, May; Group 2 (Summer) June, July, August; Group 3 (Fall) September, October, November; and Group 4 (Winter) December, January, February. Statistical analysis was performed to assess whether any observed differences were of significance.

RESULTS: 1,129 open fractures were treated between 2007 and 2012. There were 58 total infections for an overall incidence of 5.1%. The incidence of infection was 6.7% for the Spring, 5.6% for the Summer, 3.8% for the Fall, and 4.0% the Winter ($p=0.0184$ and 0.0595). The incidence of infection was 6.4% for Spring/Summer and 3.9% for Winter/Fall ($p=0.0023$). The most common causative organisms were: Gram Positive Cocci for Spring, Summer, and Fall, and Gram Negative Rods for Winter.

CONCLUSIONS: A seasonal variation exists regarding the incidence of infection and causative organisms for post-traumatic wound infection following open extremity fractures. For the Southeast region of the US, the incidence of infection is significantly higher in the spring and summer months. Gram Negative organisms are the major causative organism in the winter. This data should be taken into consideration when prophylaxis is administered and patient counseling is undertaken.

NOTES

CELL NECROSIS INDEPENDENT SUSTAINED MITOCHONDRIAL AND NUCLEAR DNA RELEASE FOLLOWING TRAUMA SURGERY

DJ McIlroy, M Bigland, A White, BM Hardy, N Lott, D Smith and ZJ Balogh
Department of Traumatology, John Hunter Hospital and University of Newcastle,
Newcastle, Australia

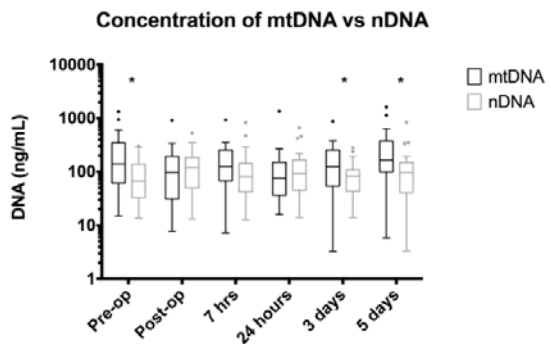
Presenter: Zsolt Balogh

Senior Sponsor: Zsolt Balogh

INTRODUCTION: Mitochondrial DNA (mtDNA), a potent pro-inflammatory damage associated molecular pattern (DAMP) has been shown to be released in large titres following injury. The mtDNA titers rapidly decrease after trauma. The effect of surgical interventions on mtDNA concentration in major trauma patients is unknown. We hypothesised that increases in mtDNA would be related to the degree of tissue injury sustained in surgery and due to rapid degradation of cell-free DNA in plasma, would decrease rapidly following surgery in uncomplicated recovery.

METHODS: Plasma was obtained from 35 trauma patients who underwent orthopaedic surgical intervention during their admission. Samples were obtained at 6 peri-operative time points (Pre-operatively then 7hrs, 24hrs, 3 days and 5 days post-operatively). DNA was extracted and the mtDNA, nuclear DNA (nDNA) were assessed using quantitative polymerase chain reaction (qPCR). Markers of cell necrosis were assayed including CK (direct skeletal muscle injury/necrosis), LDH and AST (remote secondary liver inflammatory injury/necrosis) for possible correlation with mtDNA plasma levels. Intra-operative fluid administration was also evaluated for correlation with post-operative mtDNA levels. *

RESULTS: There were high levels of free plasma mtDNA and nDNA compared to healthy controls. MtDNA and nDNA concentration remained elevated during the 5 days post-operatively, without evidence of further tissue trauma related cell necrosis (no correlation with CK, LDH or AST). MtDNA was significantly elevated compared to nDNA at Pre-Op ($p=0.003$), 3 days ($p=0.003$) and 5 days ($p=0.0014$) Pre-Op mtDNA levels were significantly higher with a shorter time from injury to surgery ($p=0.0085$). Post-operative mtDNA levels were negatively correlated with intra-operative crystalloid infusion ($p=0.0017$).



CONCLUSIONS: Serum mtDNA and nDNA concentration remains high up to 5 days following major orthopaedic trauma surgery. The free serum DNA is primarily mtDNA. Trauma DAMP studies should report surgical interventions and resuscitation volumes. The persistence of circulating DNA is not associated with further tissue necrosis. Systemic inflammation after major trauma surgery could propagate further mtDNA release of inflammatory rather than cell necrosis origin.

NOTES

BUGABOOS, CARIBOOS, AND WTA YAHOOOS: THE SPIRIT OF THE WTA CELEBRATED IN THE CANADIAN ROCKIES.

H Moore, C Bensard, E Moore, P Moore, D Ciesla, B Pickhardt, J Pickhardt, A Pickhardt, D Bensard

University of Colorado, Aurora, Colorado

Presenter: Hunter Moore

Senior Sponsor: Ernest E. Moore

INTRODUCTION: Family, friends, and fraternity embody the spirit of the WTA.

METHODS: In 1978 Ernest “Gene” E. Moore became a member of the WTA. He was elected president in 1989. Two of his residents Brad Pickhardt and Denis Bensard, who also happened to be good friends, attended the meeting and committed someday they too would become members. Similarly, David Ciesla, a resident at the University of Colorado received the Earl Young Award in 2000 and like his mentors committed to joining the WTA upon completion of training. All have remained friends, colleagues through the years attending nearly every WTA meeting with their families with the never ending competition to out ski one another

RESULTS: The annual gathering was equally important to our children who like their dads were determined to out ski everyone. Ironically some have chosen to enter a field of medicine: Hunter Moore –R4 Surgery, Peter Moore-R1 Internal Medicine; Jack Pickhardt-EMS/Fire, Anna Pickhardt-Child counselor; Claire Bensard- MS1 MD-PhD

CONCLUSIONS: In Vail we decided as a group to celebrate our friendship, in part due to our annual gathering at the WTA, with our children in a new adventure: heli-skiing in British Columbia. Here’s our story.



TRANSMISSION-BASED CONTACT PRECAUTIONS FOR MULTIDRUG RESISTANT ORGANISMS: FEWER DAYS IN ISOLATION WITH NO INCREASE IN HOSPITAL ACQUIRED INFECTIONS

CVR Brown, L Watkins, S Ali

University of Texas Southwestern - Austin, UMC Brackenridge, Austin, Texas

Presenter: Carlos VR Brown**Senior Sponsor: Carlos Brown**

INTRODUCTION: Multidrug-resistant organisms (MDRO's) including methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycin-resistant enterococci (VRE), and extended-spectrum β -lactamase producing organisms (ESBL's), can lead to hospital-associated infections that may be difficult or impossible to eradicate even with broad-spectrum antibiotics. Many institutions routinely place any patient with a history or diagnosis of an MDRO in contact isolation when admitted to their facility. Our hospital transitioned from routine to selective contact isolation of patients based not on history or diagnosis of MDRO, but rather on the likelihood the patient was soiling their environment. Indications for contact isolation became one or more of the following: diarrhea, an actively draining wound, or uncontrolled respiratory secretions. We hypothesized that the change to true transmission-based contact isolation would not be associated with an increase in hospital-associated infections with MDROs.

METHODS: We performed a retrospective study of trauma admissions to our ACS-verified, urban, level 1 trauma center during two time periods: the first time period (March - August 2011, PRE group) prior to, and the second time period (March - August 2012, POST group) after implementation of the new transmission-based contact isolation guidelines. We compared the PRE and POST groups for admission demographics and physiology, and mechanism and severity of injury. The primary outcome was the number of patients placed in contact isolation. Secondary outcomes included days spent in contact isolation and the development of a hospital-acquired MDRO infection.

RESULTS: There were 1,465 and 1,617 trauma admissions during the PRE and POST time periods, respectively. The PRE and POST groups were similar for age, gender, admission physiology, mechanism of injury, and injury severity. Overall, 3.3% (n = 49) of patients in the PRE group were placed in contact isolation as compared to 1.7% (n = 25) in the POST group (p = 0.001). Patients who required contact isolation spent an average of five days in isolation (PRE: 5 + 9 days vs. POST: 5 + 7 days, p = 0.91). More than double the number of patient days were spent in isolation in the PRE time period than the POST time period [3.8% (n = 246 days) vs. 1.7% (n = 131 days), p < 0.001]. Despite fewer patients being placed in isolation and fewer total days in isolation in the POST group, there was no difference between the PRE and POST groups in rates of developing hospital-acquired MDRO infections [0.2% (n = 3) vs. 0.2% (n = 4), p = 0.99]. All seven patients who developed a hospital-acquired MDRO infection grew MRSA in culture.

CONCLUSIONS: Transitioning from routine MDRO contact precautions to transmission-based MDRO contact precautions can reduce the number of trauma patients requiring isolation without an increase in hospital-acquired MDRO infections.

MEASURING THROMBIN GENERATION AS A TOOL FOR PREDICTING HEMOSTATIC POTENTIAL AND TRANSFUSION REQUIREMENTS FOLLOWING TRAUMA

JC Cardenas, LA Baer, N Matijevic, BA Cotton, JB Holcomb, CE Wade
University of Texas at Houston, Houston, Texas

Presenter: Jessica Cardenas

Senior Sponsor: Charles Wade

INTRODUCTION: Traumatic injury results in dysregulation of the coagulation system that can predispose patients to excessive hemorrhage or increase the risk of future thromboembolic events. Thrombin is the central coagulation protease that activates other clotting proteins, triggers platelet aggregation, and converts fibrinogen to fibrin. The early enzymatic phases of coagulation are crucial, as the ability to generate thrombin is necessary for the formation of a stable fibrin clot during hemorrhage. The relationships between thrombin generation (TG) and clinical outcomes have yet to be defined in trauma patients. We hypothesized that the potential for generating thrombin would be predictive of transfusion requirements and outcomes in critically injured patients.

METHODS: This study was performed following Institutional Review Board approval and informed consent was obtained for individual patients. Plasma was collected upon admission to the emergency department from patients meeting highest-level trauma activation. Demographics, resuscitation volumes, laboratory values and injury severity (ISS) were collected through direct observation and electronic medical record query. In addition, plasma was collected from 30 healthy donors to serve as controls. TG was measured by calibrated automated thrombography (CAT) as peak thrombin (nM), which is the highest concentration of thrombin generated in the plasma sample. Injured patients and healthy controls were compared using Mann-Whitney test and are expressed as median (IQR). Relationships between TG and outcomes were analyzed by multivariate linear regression and expressed as beta coefficient, p value.

RESULTS: A total of 406 patients were included in this study with a median age of 30 (28,52) and ISS of 17 (9-26). Trauma patients generated substantially more thrombin [315.8 nM (269.7, 355.5)] compared to healthy controls [124.6 nM (91.1, 156.2)], $p < 0.001$. TG was inversely correlated with ISS (-0.04 , $p < 0.001$) and significantly affected by mechanism of injury as patients with blunt injuries had the lowest TG ($p = 0.001$). TG was inversely related to transfusion requirements as those patients with the lowest TG capacity required more units of red blood cells (-0.021 , $p = 0.001$), plasma (-0.02 , $p = 0.003$), platelets (-0.02 , $p = 0.003$), and crystalloid (-6.28 , $p = 0.03$). Finally, peak thrombin was highly correlated with every thrombelastography (TEG) parameter tested suggesting TEG is sensitive to thrombin production and activity.

CONCLUSIONS: We have shown that TG potential is critically important following traumatic injury. While the physiologic response to injury is to upregulate plasma procoagulant activity, patients with defective or deficient TG required more transfusions to manage their bleeding. Measuring TG may provide an exquisitely sensitive tool for detecting disturbances in the enzymatic phases of coagulation in critically injured patients.

THE PEDIATRIC TRAUMA AND TRANEXAMIC ACID STUDY (PED-TRAX)

MJ Eckert, TM Wertin, SD Tyner, MJ Martin
Madigan Army Medical Center, Tacoma, Washington

Presenter: Matthew Eckert

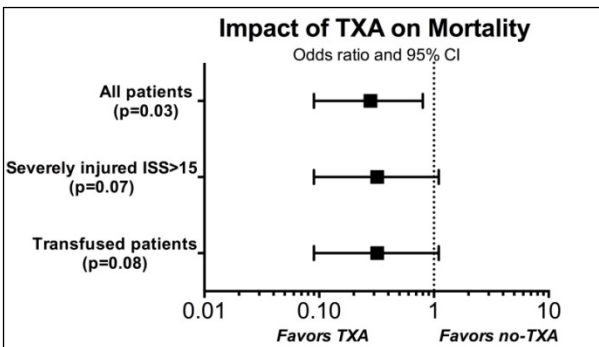
Senior Sponsor: Matthew Martin

INTRODUCTION: Early administration of TXA has been associated with reduction in mortality and blood product requirements in severely injured adults. It has also shown significantly reduced blood loss and transfusion requirements in major elective pediatric surgery but no published data has examined the use of TXA in pediatric trauma.

METHODS: Retrospective review of all pediatric trauma admissions to the NATO Role 3 Hospital, Camp Bastion, Afghanistan, from 2008 to 2012. Univariate and logistic regression analyses of all patients and select subgroups was performed to identify factors associated with TXA use and mortality. Standard adult dosing of TXA was used in all patients.

RESULTS: There were 766 injured patients ≤ 18 yrs old: mean age 11 ± 5 yrs, 88% male, 73% penetrating injury, ISS 10 ± 9 , GCS 12 ± 4 . 35% of patients required transfusion in the first 24 hrs, 10% received massive transfusion, and 76% required surgery. Overall mortality was 9%. 66 of 766 patients (9%) received TXA. The only independent predictors of TXA use were severe abdominal or extremity injury (AIS ≥ 3) and a base deficit >5 (all $p < 0.05$). Patients who received TXA had greater injury severity, hypotension, acidosis, and coagulopathy versus the no-TXA group. Unadjusted mortality was 15% TXA vs 9% no-TXA ($p = 0.07$). After correction for demographics, injury type and severity, vitals, and lab parameters, TXA use was independently associated with decreased mortality among all patients (OR 0.3, $p = 0.03$), and showed similar trends for subgroups of severely injured (ISS >15) and transfused patients (see Figure). There was no significant difference in venothromboembolic complications or other cardiovascular events.

CONCLUSIONS: TXA was used in approximately 10% of pediatric combat trauma patients, typically in the setting of severe abdominal or extremity trauma and metabolic acidosis. TXA administration was independently associated with decreased mortality. There were no adverse safety or medication-related complications identified.



BIOSCAFFOLD REPLACEMENT IN PEDIATRIC VASCULAR INJURY: A CASE REPORT

CA Smith, DL Dawson, CR Schermer, JM Galante
University of California at Davis, Sacramento, California

Presenter: Caitlin Smith

Senior Sponsor: Carol Schermer

INTRODUCTION: Introduction: Vascular injury in children presents a challenge in terms of operative repair, surveillance, and long-term implications, especially when primary repair is not an option.

METHODS: Case Description: Patient is an 8-year-old girl in a rollover motor vehicle crash. She was transferred to our pediatric Level 1 trauma center after initial stabilization and evaluation at two outside institutions. Physical exam on arrival to our center demonstrated a large seatbelt sign across her abdomen with an extensive soft tissue defect on her left lateral flank and peritoneal signs. Right common iliac artery occlusion and an L2 Chance fracture were noted on the CT scan. She was taken to the operating room for exploratory laparotomy where a large mesenteric defect and 40 cm of devascularized distal ilium was found and resected. A retroperitoneal hematoma was explored to expose the thrombosed right common iliac artery. Thrombectomy and endarterectomy of an intimal flap was performed. Primary anastomosis was attempted, but an intraoperative duplex ultrasound scan showed abnormal flow patterns and elevated velocities at the site of repair, indicative of a severe stenosis. The arteriotomy was reopened and pale thrombus was removed from the endarterectomy surface. The arterial closure was revised with a patch angioplasty using CorMatrix, an extracellular matrix biomaterial. The final intraoperative duplex scan and post-operative follow up confirmed a good technical result. Three months after the injury the child is doing well with normal pedal and groin pulses.

RESULTS: Case Report. Please see above.

CONCLUSIONS: Conclusion: This is an example of a complex vascular injury in a child where a satisfactory primary repair could not be performed. CorMatrix, a bioscaffold that allows native tissue remodeling, was used to complete the repair. The bioscaffolding allows native cells to incorporate into the repair and remodel or regenerate as the child grows. Use of Cormatrix in such a manner has been described in carotid artery repair, and may be an alternative to implantation of prosthetic material or use of autologous vein in injured pediatric patients or in a potentially contaminated operative field.

EXTRACORPOREAL MEMBRANE OXYGENATION IN SEVERE INTRACRANIAL HEMORRHAGE: A CASE REPORT

RJ Jean, PW Smith, RA Maxwell, DE Barker

University of Tennessee-Chattanooga, Chattanooga, Tennessee

Presenter: Robert Jean**Senior Sponsor: Robert A. Maxwell**

INTRODUCTION: Extracorporeal membrane oxygenation (ECMO) is a useful adjunct for the treatment of acute respiratory distress syndrome (ARDS) refractory to other ventilatory strategies in select adult trauma patients. ECMO was first introduced in 1972 in a young adult trauma patient with severe ARDS. However, the mortality in the early years was prohibitive in this population and ECMO has primarily been used in the neonatal population. Recently, ECMO has been increasingly used in the adult ARDS population, mainly with influenza, but is also being utilized more frequently in the adult trauma population. Traumatic intracranial hemorrhage (ICH) has been considered a contraindication to ECMO due to the need for systemic heparin anticoagulation and the associated risk of worsening intracranial bleeding. We report what we believe is the first successful case of adult venovenous ECMO with full systemic heparinization in a patient with severe traumatic intracranial hemorrhage.

METHODS: A 19 year-old man presented after a 25-foot fall with severe, multifocal ICH and an initial Glasgow Coma Scale (GCS) of 4 (E1 V1 M2). He developed refractory intracranial hypertension and, on hospital day (HD) 6, required decompressive craniectomy with evacuation of intracranial clot. He subsequently developed severe ARDS (PaO₂:FiO₂ ratio 38) with hypoxemia refractory to conventional mechanical ventilatory support. He was placed on venovenous ECMO with full systemic heparinization five days after decompressive craniectomy (HD 11).

RESULTS: During 119 hours of continuous venovenous ECMO with full systemic heparinization, he showed progressive improvement of infiltrates on chest x-ray, improved oxygenation, and resolution of ARDS (PaO₂:FiO₂ ratio 218) without computed tomography (CT) evidence of an increase in intracerebral hemorrhage. He was subsequently removed from ECMO after 5 days (HD 16) and placed back on conventional mechanical ventilatory support within ARDSnet guidelines. He was liberated from mechanical ventilator support ten days after cessation of ECMO (HD 26) and subsequently discharged to a neurologic rehabilitation facility sixteen days after cessation of ECMO (HD 32) with a GCS of 11 (E4 V1 M6) and without evidence of increased ICH.

CONCLUSIONS: Although considered extremely high risk, venovenous ECMO with full systemic heparinization can be successfully used in select patients with ICH and ARDS.

BLUNT ABDOMINAL AORTIC INJURY: A MULTICENTER EXPERIENCE

S Shalhub, RA Kozar, BW Starnes, I Kenji, D Skiada, M Brenner, B Zarzaur, C Nawaf, A Azizzadeh, W Biffi, EA Eriksson, SM Fakhry, JS Paul, KL Kaups, K Cagle, DJ Ciesla G Natalie, SR Todd, MJ Seamon, L Capano-Wehrle, J Jurkovich
University of Washington, Seattle, Washington

Presenter: Sherene Shalhub

Senior Sponsor: Rosemary Kozar

INTRODUCTION: Blunt Abdominal Aortic Injury (BAAI) is rare and most of the literature comprised of case series. The objective of the current study was to examine the presentation and management of this rare injury at a multi-intuitional level

METHODS: The Western Trauma Association Multi-Center Trials conducted a study of patients presenting with BAAI from 1996 to 2011. Data collected included demographics, mechanism of injury, associated injuries, ISS, type of injury, interventions and complications.

RESULTS: 116 patients with BAAI presented at 12 major trauma centers (67% male, median age 38 years old, range 6-89, median ISS 34, range 16-75). BAAI incidence was 0.03% of blunt trauma injuries and the leading cause was motor vehicle collisions (61%). Hypotension on arrival was documented in 47% of the cases. The most commonly associated injuries were spine fractures (44%), solid organ (42%), and hollow viscus injuries (41%). BAAI presented as free rupture (30%), pseudoaneurysm (15%), and injuries without external contour abnormality such as large intimal flap (36%) or minimal aortic injury (19%). Open and endovascular repairs were undertaken in 44% and 16% of cases respectively. Choice of management varied by type of BAAI with 70% of minimal aortic injuries managed non-operatively and 96% of aortic rupture undergoing open repairs. Overall mortality was 41% with 28% occurring in the first 24 hours due to hemorrhage or cardiac arrest. Mortality associated with free aortic rupture was 82%. Of those discharged alive, 38% had at least one follow up visit.

CONCLUSIONS: This is the largest BAAI series reported to date. BAAI is rare and presents as a spectrum of injury ranging from minimal aortic injury to aortic rupture. Non-operative management is successful in uncomplicated cases without external contour abnormality. Highest mortality occurs in those with free aortic rupture, suggesting that alternative measures of early hemorrhage control are needed. Evaluation of newer technology for non-compressible torso hemorrhage, such as aortic balloon occlusion, may be warranted.

NOTES

ANGIOGRAPHY IS AN IMPORTANT TOOL TO CONSIDER IN HIGH GRADE SPLEEN INJURIES

E Jeremitsky, LM Capecci, FH Philp, RS Smith
Allegheny General Hospital, Pittsburgh, Pennsylvania

Presenter: Louis Capecci

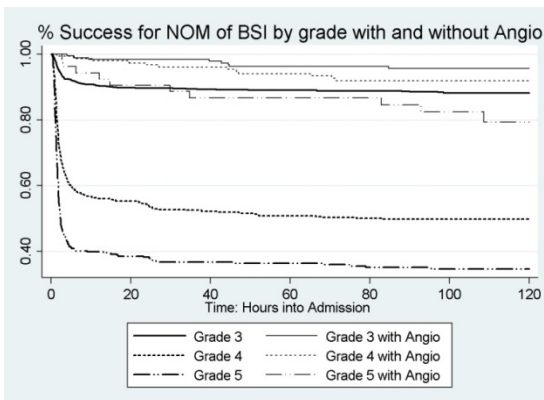
Senior Sponsor: Stephen Smith

INTRODUCTION: Non-operative management (NOM) for blunt splenic injury (BSI) is well established. Several single institution studies have demonstrated an improved success rate of NOM in BSI with the use of angiography (ANGIO) and embolization (EMBO) modalities. Treatment protocols for NOM are not currently standardized and vary widely between institutions. We hypothesize that angiography improves the success of NOM for BSI.

METHODS: A large multi-institutional statewide database (2007-2011) was used to generate the patient cohort. All BSI patients (age >12) were included in the study. Patient demographics, grade of BSI and the use of ANGIO and/or EMBO were compared between the failed and successful NOM patients. Univariate, Kaplan-Meier survival curves and multivariable Cox proportional hazard ratios for splenectomy (failed NOM) were performed.

RESULTS: Of the 5491 patients with BSI, 1131 patients underwent splenectomy (21% of the patient cohort). Patients who had splenectomy were older (41.7 ± 19.2 vs. 38.5 ± 21 , p55 (HR 1.76, CI 1.44-2.14), angiography (HR 0.34, CI 0.23-0.50), and using injury grades 1,2 as a reference; grade 3 (HR 2.84, CI 2.10-3.89), grade 4 (HR 14.08, CI 10.39-19.10) and grade 5 (HR 17.08, CI 12.36-23.62).

CONCLUSIONS: There was a clear improvement in successful NOM for BSI when angiography was incorporated. This was especially profound for higher grades of splenic injury with spleen salvage rates doubling. Using angiography for BSI resulted in a risk reduction for splenectomy of 66%. When NOM is considered for higher grades of BSI, ANGIO should be strongly considered.



HUMAN AND PILOT ERROR IN HELICOPTER EMERGENCY MEDICAL SERVICES CRASHES: WHAT ROLE DOES OWNERSHIP PLAY?

FA Habib, D Shatz, Al Habib, I Puente, M Bukur, J Catino, R Farrington
Broward Health Medical Center, Ft Lauderdale, Florida

Presenter: Fahim Habib

Senior Sponsor: David Shatz

INTRODUCTION: The National Transportation Safety Board (NTSB) ranks Helicopter Emergency Medical Services (HEMS) as the most dangerous occupation in the United States, and ranks improvements in HEMS as one of the highest priority targets among its 10 Most Wanted Improvements. Considering that a sizeable proportion of injured patients are transported to trauma centers by HEMS, these concerns are of particular relevance to the trauma community. While of arguable benefit, the use of HEMS is associated with a heightened degree of inherent risk. We hypothesized that this risk is not uniform, and varies with the entity providing HEMS, specifically, corporate versus public providers.

METHODS: The NTSB accident and incident database was queried to identify all helicopter crashes operating under 14 CFR Parts 91 and 135 for the 15-year period 1998-2012. Each accident synopsis was reviewed to identify HEMS involved events. The detailed NTSB investigation report was reviewed to obtain details on geography, time, weather, injury severity, helicopter damage, and probable cause. These were analyzed on the basis of HEMS ownership: corporate, not-for-profit, and public. Statistical analyses were performed using ANOVA and Fisher's Exact Test as appropriate. A $p < 0.05$ was used to determine statistical significance.

RESULTS: Over the 15-year study period, 137 of 2040 (6.7%) crashes under the relevant codes involved HEMS and occurred across 133 cities in 37 states, killing 113 and seriously injuring another 54. Of the 137 events, there were 116 corporate, 14 not-for-profit and 7 public HEMS crashes. When analyzed in 5 year blocks, no decrease in crash incidence was seen (9.6 ± 1.67 , 8.4 ± 3.91 , and 8.4 ± 1.82 , $p = 0.7$, ANOVA). Crashes tended to be more common in the latter part of the week (39 vs. 70). Overall, weekends, nighttime, meteorological conditions, and weather were not different among corporate and public HEMS crashes (all $p = NS$). In contrast, human error and pilot error were significantly more common among corporate HEMS than in public HEMS (89 of 116 vs. 2 of 7, $p = 0.013$, and 72 of 116 vs. 1 of 7, $p = 0.017$, Fisher's Exact Test). Similar results were obtained when corporate and not-for profits together were compared against public HEMS. Severity of damage of the helicopter was similar in both groups ($p = 0.22$). Review of the narrative identified conditions encountered for which training was not adequate, limited resources, inadequate equipment and the undertaking of suboptimal trips as common themes. Trauma patients were involved in 33 (24.1%) transports, with fatal or serious outcomes in 44 patients on 11 flights.

CONCLUSIONS: The occurrence of potentially preventable human and pilot error-related HEMS crashes are significantly more frequent with corporate than public providers. Deficiencies in training, reduced availability of equipment and resources as well as questionable flight selection appear to play a key role.

HISTONE DEACETYLASE III AS A POTENTIAL THERAPEUTIC TARGET FOR THE TREATMENT OF LETHAL SEPSIS

T Zhao, Y Li, B Liu, RT Bronson, GC Velmahos, HB Alam
University of Michigan Hospital, Ann Arbor, Michigan

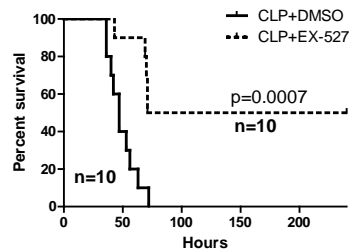
Presenter: Hasan Alam

Senior Sponsor: Hasan Alam

INTRODUCTION: We have recently demonstrated that inhibition of histone deacetylase (HDAC) class I, II and IV with non-specific HDAC inhibitors (HDACi) improves survival in a mouse model of lethal cecal ligation and puncture (CLP). However, the consequence of HDAC class III (distinct from other HDAC) inhibition is unknown in this model. The aims of present study were to explore the effect of EX-527, a selective SIRT1/HDAC III inhibitor, on survival in the lethal model of CLP-sepsis, and to assess the impact of the treatment on inflammatory cytokine production, coagulopathy and bone marrow atrophy during severe sepsis.

METHODS: Experiment I: C57BL/6J mice were subjected to CLP, and 1 h later intraperitoneally injected with either EX-527 (47 mg/kg) dissolved in dimethyl sulfoxide (DMSO), or DMSO only (n=10/group). Survival was monitored for 10 days. Experiment II: One hour after CLP, animals were randomly treated with: (i) DMSO vehicle, and (ii) EX-527 (n=10-15/group). Sham-operated animals (no CLP, no treatment) served as a normal control. Peritoneal fluid and blood samples were collected for measurement of cytokines at 24 or 48 h. Blood at 48 h was also used to evaluate coagulation status using Thrombelastography. In addition, long bones (femurs and tibias) were harvested from animals (n=5-6/group) at 48 h to determine morphological changes of bone marrow by H&E staining. Atrophy percentages of bone marrow were then graded by a pathologist blinded to group allocation of the samples. Experiment III: Normal primary splenocytes were cultured, and treated with lipopolysaccharide (LPS) in the presence or absence of EX-527 (10 μ M) for 6 h to assess cytokine production (n=4/group).

RESULTS: EX-527 significantly improved survival (figure), and attenuated levels of cytokines in blood (TNF- α : 298.3 \pm 24.6 vs. 55.3 \pm 8.0 pg/ml, P=0.0049; IL-6: 583.8 \pm 83.8 vs. 216.1 \pm 135.6 pg/ml, P=0.0398) and peritoneal fluid (IL-6: 704.8 \pm 67.7 vs. 378.4 \pm 128.4 pg/ml, P=0.0388) compared to the vehicle control. It also decreased TNF- α and IL-6 production by splenocytes in-vitro (TNF- α : 68.1 \pm 6.4 vs. 40.5 \pm 5.1 pg/ml, P=0.0152; IL-6: 73.1 \pm 4.2 vs. 45.8 \pm 4.8 pg/ml; P=0.0091). Animals subjected to CLP displayed prolonged fibrin formation and fibrin cross-linkage time, and decreased clot formation speed, platelet function and clot rigidity (coagulopathy markers). Selective inhibition of SIRT1 was associated with dramatic improvements in fibrin cross-linkage, platelet function and clot rigidity, but without a significant impact on the clot initiation parameters. Moreover, inhibition of SIRT1 decreased bone marrow atrophy significantly (64.0 \pm 4.0 vs. 35.0 \pm 12.6%, P=0.0456).



CONCLUSIONS: Selective inhibition of Class III histone deacetylase (SIRT1) significantly improves survival, attenuates “cytokine storm” and sepsis-associated coagulopathy, and decreases bone marrow atrophy in a lethal septic model.

NOTES

DEATH AFTER DISCHARGE: PREDICTORS OF MORTALITY IN OLDER BRAIN-INJURED PATIENTS

K Peck, RY Calvo, C Dunne, J Badiie, J Johnson, B Sise, S Shackford, M Sise
Scripps Mercy Hospital, San Diego, California

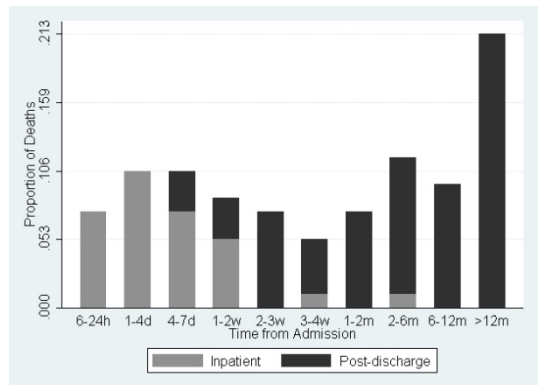
Presenter: Kimberly Peck

Senior Sponsor: Kimberly Peck

INTRODUCTION: Older patients with traumatic brain injury (TBI) may be at high risk of death after hospitalization. To identify factors that predict mortality after discharge, we characterized long-term mortality of older patients with TBI who survived to discharge. We hypothesized that predictors of post-discharge mortality differ from those of inpatient mortality.

METHODS: A 5-year historical prospective cohort study was performed on TBI patients aged >55 years admitted to our Level I trauma center. Post-discharge deaths were identified by matching patient data with local vital records up to 12/31/11, when data collection was terminated (censoring). All patients were categorized by age, ISS, head AIS, initial TBI type, need for mechanical ventilation, pre-hospital living status and discharge location. Factors predictive of inpatient death were compared to predictors of post-discharge death. The effect of risk factors on mortality was evaluated by Cox proportional hazards modeling.

RESULTS: Of 353 patients, 322 (91.2%) survived to discharge. Post-discharge mortality was 19.8% (n=63) for the study period. Of post-discharge deaths, 54% died within 6 months and 68.3% died within 1 year. Median days to death after discharge or censoring were 149 or 410, respectively. Factors associated with death after discharge were age, pre-injury anticoagulant use, higher Charlson Index, presence of more than one initial TBI type, discharge to a care facility, and moderate/severe disability. Factors predictive of inpatient death (ISS, initial GCS, head AIS, pre-injury antiplatelet agent use) did not predict post-discharge mortality.



CONCLUSIONS: Older TBI patients who survive to discharge have a significant risk of death the following year. Predictors of post-discharge mortality and inpatient death differ. Whereas measures of injury severity predict inpatient death, death after discharge is largely a function of overall health status. Continued aggressive management of comorbidities after discharge may be essential in determining long-term outcomes.

PROSPECTIVE VALIDATION OF THE BRAIN INJURY GUIDELINES: MANAGING TRAUMATIC BRAIN INJURY WITHOUT NEUROSURGICAL CONSULTATION

B Joseph, H Aziz, V Pandit, N Kulvatunyou, A Tang, T O'Keeffe, DJ Green, G Vercruysse, RS Friese, P Rhee

The University of Arizona, Tucson, Arizona

Presenter: Bellal Joseph

Senior Sponsor: Peter Rhee

INTRODUCTION: In order to optimize neurosurgical resources, guidelines were developed at our institution allowing the acute care surgeons (ACS) to independently manage traumatic intracranial hemorrhage (ICH) less than or equal to 4 millimeter (≤ 4 mm). The aim of our study was to validate our established Brain Injury Guidelines (BIG). We hypothesized that ACS can manage Traumatic Brain Injury (TBI) with ICH without neurosurgical consultation (NC).

METHODS: We formulated the BIG guidelines based on 4 year retrospective chart review of 1,232 TBI patients with an ICH. We then prospectively implemented our guidelines to analyze all TBI patients with ICH managed without neurosurgical consultation (No-NC). Propensity scoring matched patients with No-NC to a similar cohort of patients managed with NC prior to implementation of our BIG guidelines in a 1:1 ratio for age, gender, neurologic exam Glasgow Coma Scale (GCS) Score, mechanism of injury, head Abbreviated Injury Scale (h-AIS) Score, Injury Severity Score (ISS, skull fracture, and ICH (type and size). Primary outcome measure was need for neurosurgical intervention and 30 day re-admission rates.

RESULTS: A total of 384 TBI patients (142: NC and 142: No-NC) were included in the analysis. The mean age was 40 ± 23 years, 62% were male, median GCS was 15[12-15], and median head AIS was 2[1-3]. There was no neurosurgical intervention or 30 day re-admission in both the groups. In the No-NC group 8% of the patients had post discharge ED visits compared to 2% of the NC group ($p=0.5$). All patients were discharged home from the emergency department.

Table 1. Outcomes

Outcomes	No-NC (n=142)	NC (n=142)	<i>p</i>
ICU Admission	14%	32%	0.006
Repeat Head CT	19%	81%	<0.001
Post Discharge Head CT	3%	11%	0.049
Hospital Cost	6246.8 \pm 7735	12953 \pm 12131	<0.001

CONCLUSIONS: We validated our Brain Injury Guidelines and demonstrated that implementation of the BIG guidelines is safe and cost effective. A national multi-institutional prospective evaluation is warranted.

CARING FOR YOUR PET RATTLESNAKE....BITE.**A REVIEW OF CROTALIDAE POLYVALENT IMMUNE FAB USE AND OUTCOME IN A SNAKE INFESTED STATE.**

AG Rizzo, K Darden, L Pressler, L Smith, LC Mercer
United Regional Hospital, Wichita Falls, Texas

Presenter: Anne Rizzo

Senior Sponsor: Anne Rizzo

INTRODUCTION: World wide, snakebites are considered a public health emergency. The World Health Organization (WHO) estimates 5 million bites, 500,000 amputations and 125,000 annual deaths. With the emergence of new antivenins the case-fatality rate has decreased from a high of 40% to its present estimate at 1.25%. The crotalidae venomous snakes to include: rattlesnakes, copperheads and cottonmouths/water moccasins inhabit the state of Texas. Many municipalities host "round-ups" to curtail the burgeoning population of these reptiles and to protect their mammalian populations.

METHODS: Retrospective review of a protocol-based treatment plan with crotalidae polyvalent immune fab (CroFab, BTG international) for rattlesnake envenomation was performed. Administration of CroFab is based on measurement of limb and area of swelling along with clinical symptoms. All snakebites from 2009-2013, at a Level II Trauma center in north east Texas were reviewed. Charts were abstracted by two independent observers. Seventy rattlesnake bites and one rat snake (*Pantherophis obsoletus*) bite, with no envenomation were identified. Demographic data, laboratory values, CroFab administration and hospital course were documented. All rattlesnake bites were grade 1-3.

RESULTS: The average age of patients bitten was 42 (range 2-81). Twice as many males were bitten compared to females (n=48:23). The 40-60 year old range were bitten almost twice as commonly as the 100, 14 patients had a d-dimer >4000, this did not correlate with vials of CroFab administered, but did correlate with grade of bite and surgical intervention. Interestingly the one case with no envenomation also had a d-dimer of 823. The most common areas bitten were finger (n=26), hand (n=17) and foot (n=10). There was an even bodily distribution of bites throughout the age groups. Men had twice as many upper versus lower extremity bites (n=36:12), women were the opposite (n=7:16).

CONCLUSIONS: Rattlesnake bites cause morbidity but not mortality in the United States. There is no correlation between abnormal lab values, length of stay or amount of antivenom delivered. Using a protocol for treatment decreases the amount of antivenom used. Surgical intervention is related to the grade of envenomation. Men grab snakes, women step on them.

THE NATURAL HISTORY OF FRACTURING FOUR EXTREMITIES

JV Jacobs, JR Rivas, BR Robinson, N Attias, SR Petersen, N Namias, L Pizano
 St. Joseph's Hospital and Medical Center, Phoenix, Arizona

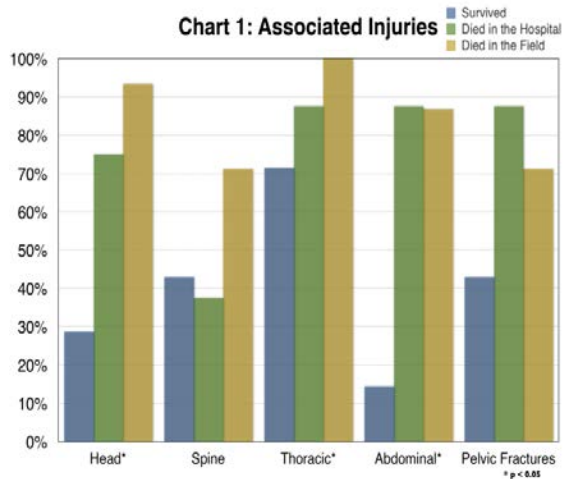
Presenter: Jordan Jacobs

Senior Sponsor: Nicholas Namias

INTRODUCTION: Injuries leading to fractures in all four extremities have never been studied. The objective of this study is to report population and hospital based survival rates and rates of life-threatening associated injuries for trauma victims with a fracture in all four extremities.

METHODS: A retrospective cohort analysis was performed over a 10-year period of 46,471 consecutive trauma admissions at an urban Level 1 trauma center. Additionally, an analysis of 2,993 autopsy reports from every major blunt traumatic death (motor vehicle crashes, motorcycle crashes, and pedestrians/bicyclists hit by car) in the surrounding county was completed. The cohort having all four extremities fractured was stratified by location of death. Comparisons were made using chi-squared analysis.

RESULTS: There were 60 trauma victims with a fracture in all four extremities identified. Among this cohort, 75% (45 of 60) were pronounced dead in the field, and 25% (15 of 60) were transported to the county's trauma center. Of those transported 53% (8 of 15) died in the hospital, creating an overall survival rate of 12% (7 of 60). The incidence of fracturing all four extremities among major blunt traumatic death was 1 in 56. Associated injury rates demonstrated 83% (50 of 60) with head injuries, 95% (57 of 60) with thoracic injuries, 78% (47 of 60) with abdominal injuries, 70% (42 of 60) with pelvic fractures, and 63% (38 of 60) with spine injuries. Rates of life-threatening associated injuries stratified by location of death can be found in Chart 1. Survivors had significantly less head, thoracic, and abdominal injuries.



CONCLUSIONS: An injury which leads to a fracture in all four extremities is highly lethal and almost universally associated with life-threatening injuries to the head or trunk. Efforts should focus on the prevention of these severe blunt injuries, and vigilance towards identifying and treating the associated injuries is critical in efforts to save those who survive to the hospital.

1:1 RECONSIDERED : PATIENTS MEETING THE CRITICAL ADMINISTRATION THRESHOLD (CAT+) FARE BETTER WITH MATCHED TRANSFUSIONS

SA Savage, BL Zarzaur, MA Croce, TC Fabian

University of Tennessee Health Science Center, Memphis, Tennessee

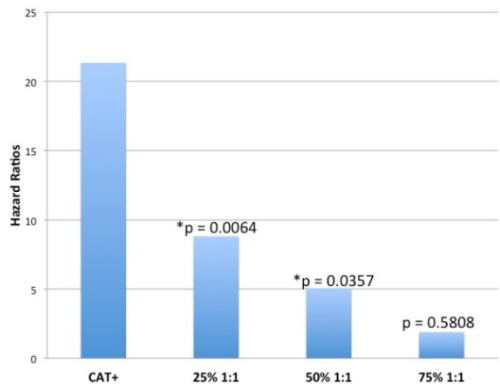
Presenter: Stephanie Savage

Senior Sponsor: Stephanie Savage

INTRODUCTION: The practice of 1:1 transfusion, administering packed red blood cells (PRC) with fresh frozen plasma (FFP), has shown an association with improved survival in several reports. However, the reported ratios are the result of mathematical averages over 24 hours and do not necessarily represent concurrent administration. This raises concerns of “survival bias.”. Using a critical administration threshold (CAT+) of > 3 units of PRC/hr (JOT Feb. 2013) to identify large volume transfusion patients, this study evaluates the effect of concurrent administration of PRC:FFP on patient survival.

METHODS: CAT+ patients identified retrospectively were eligible for analysis. Exact timing (minutes) of administration of each unit of PRC and FFP was known for every patient. Each PRC was then matched to a corresponding unit of FFP given within five minutes before or after, with no FFP counted more than once. Ideal 1:1 ratios (IR) were calculated for each hour over the first day of admission. IR = # times 1:1 achieved in an hour/total units PRC in same hour. Hourly IR classes were created (IR reached 25%, 50%, 75% of transfusion opportunities) and evaluated as time-varying covariates in subsequent analysis. A Cox proportional hazard ratio (HR) was used to determine risk of mortality and Student’s t-test or Wilcoxon signed-rank test were used to compare patient groups.

RESULTS: 169 patients were analyzed (70% NISS > 10). 77 patients were identified as CAT+. There were no clinical differences between groups in this study. Total blood volumes transfused over 24 hours were also similar between groups (25% 15.5 units, 50% 17.2 units, 75% 19.1 units). Patients who reached the 1:1 ratio 25% of the available transfusion opportunities had an HR of 8.806 (95% CI 1.845, 42.034, p=0.0064). Patients meeting the 1:1 ratio 50% of opportunities had an HR of 5.062 (95% CI 1.115, 22.982, p=0.0357). Patients meeting the 1:1 ratio 75% of opportunities had an HR of 1.888 (95% CI 0.198, 18.035, p=0.5808).



CONCLUSIONS: It has previously been shown that CAT+ patients represent the trauma subset at highest risk of mortality. This group also represents those that may benefit from a more focused blood product resuscitation. While all IR classes had an elevated risk of death, as would be expected in patients receiving large volume blood resuscitations, patients who were able to meet the 1:1 ratio more often had a noticeable decrease in risk of death compared those who achieved less 1:1 transfusions. Administering FFP concurrently with PRC is associated with a decrease in mortality in CAT+ patients.

MODERATE ELEVATIONS IN INR SHOULD NOT LEAD TO DELAYS IN NEUROSURGICAL INTERVENTION IN PATIENTS WITH SEVERE TBI

TC Lennox, RR Barbosa, KA Fair, AJ Rao, SJ Underwood, MA Schreiber, SE Rowell
Oregon Health & Science University, Portland, Oregon

Presenter: Tori Lennox

Senior Sponsor: Susan Rowell

INTRODUCTION: Management of severe traumatic brain injury (sTBI) frequently involves invasive intracranial monitoring or cranial surgery. In our institution, intracranial procedures are often deferred until an international normalized ratio (INR) of < 1.4 is achieved, potentially resulting in increased plasma transfusions and delay in neurosurgical intervention (NI). There is no evidence that a moderately elevated INR is associated with increased risk of bleeding in sTBI. Thrombelastography (TEG) provides a functional assessment of clotting and has been shown to be a better predictor of clinically relevant coagulopathy compared to INR. We hypothesized that in patients undergoing NI, an elevated INR would not be associated with coagulation abnormalities based on TEG and would result in increased time to NI.

METHODS: Data were prospectively collected in trauma patients with intracranial hemorrhage (ICH) that underwent NI (defined as cranial surgery or intracranial pressure monitoring) within 24 hours of arrival. Time from admission to NI was recorded. TEG, INR, prothrombin time (PTT), fibrinogen and platelet count were obtained on admission. Patients on warfarin and clopidogrel were excluded. Patients were considered hypocoagulable based on INR if their admission INR was > 1.4 (high-INR). Patients were considered hypocoagulable based on TEG if any of the following parameters were observed on the admission TEG: $R > 9$ min, $K > 3$ min, $Angle > 8$ min.

RESULTS: Sixty-one patients (median AIS head=5) met entry criteria of which 16% had high-INR. Demographic, physiologic, and injury scoring data were similar between groups. High-INR patients had significantly different admission lab values: INR 1.7 vs 1.1 ($p < 0.001$); PTT 33 vs. 27 ($p = 0.004$). There were no differences in platelet count or fibrinogen levels. High-INR patients were transfused more plasma than patients with an $INR \leq 1.4$ (2 vs 0 units; $p = 0.01$). There was no correlation between an elevated INR and hypocoagulability based on TEG. The median time to NI was longer in patients with high-INR (358 minutes vs 184 minutes; $p = 0.027$).

CONCLUSIONS: Patients with an $INR > 1.4$ often have NI delayed waiting for plasma transfusions. Time to NI was twice as long in patients with an $INR > 1.4$ compared to those with an $INR \leq 1.4$. Many of these patients may not have had significant coagulopathy. Utilizing TEG to assess coagulation status may decrease time to NI and decrease transfusion requirements in patients with sTBI.

THE EVOLUTION OF A HIGH RELIABILITY REGIONAL PROGRAM OF EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO) FOR ADULTS WITH TRAUMA, CARDIOVASCULAR COLLAPSE AND REFRACTORY HYPOXEMIC ACUTE RESPIRATORY DISTRESS SYNDROME (ARDS)

AJ Michaels, JG Hill, WB Long, BP Young, SM Wanek, JC Dalthorp, TR Shanks, LJ Morgan
Legacy Emanuel Medical Center, Portland, Oregon

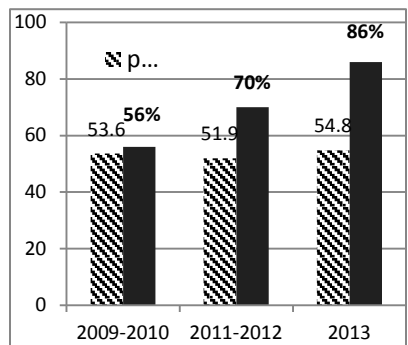
Presenter: Andrew Michaels

Senior Sponsor: Andrew Michaels

INTRODUCTION: The evolution of a high reliability program in medicine depends upon both experience and the efficient use of organizational and clinical processes. We have treated adults with extracorporeal membrane oxygenation (ECMO) for over 25 years. Initially, ECMO was used for resuscitation from cardiac events and hypothermia. With the advent of heparin bonded circuits, acutely injured patients were supported on ECMO during resuscitation and early recovery. With the H1N1 pandemic of 2009, refractory hypoxemic acute respiratory distress syndrome (ARDS) became the primary indication for ECMO. We describe the evolution of a regional ECMO program from an experienced yet episodic process to a high volume, high reliability program providing ECMO to adults with ARDS, trauma and cardiovascular collapse.

METHODS: The registries of the Trauma and ECMO programs were queried for all patients treated between 1985 and 2013 (to date). Cases after 2008 are reported with age, gender, preECMO PaO₂:FiO₂ ratio (prePF), preECMO days of ventilation (ventDAY), hours of ECMO support (ECMOhrs), postECMO PaO₂:FiO₂ ratio (postPF), primary diagnosis and outcome (survival to discharge). Prior to 2009, the physiologic data were not collected.

RESULTS: The first case of ECMO in the institution was in 1985 for hypothermia. In the pre2009 era, 103 patients received ECMO. These cases were mostly resuscitative and treated with veno-arterial (VA) ECMO. From 2009 forward, ECMO was resuscitative for 4 patients and 52 patients were treated with ECMO for ARDS. In this latter group, patients were 38.5 ± 1.9 years old and 51% male, had 3.1 ± 0.33 ventDAYs and a prePF of 52.9 ± 2.4 . They had 182.13 ± 24.1 ECMOhrs and, in survivors, a postPF of 321.5 ± 13.7 . They were referred from other medical centers in 91%, transferred by the ECMO transport team with ECMO support in 69%, recovered to wean from ECMO in 76% and survived to discharge in 70%. For adults treated with primarily venovenous (VV) ECMO for ARDS, prePF and survival rates during three time periods since 2008 are listed in Table 1.



CONCLUSIONS: For even a mature ECMO program, the effect of increased clinical volume combined with the conscious establishment of formal organizational processes including protocols, credentialing, crew resource management (CRM) based culture, timely performance improvement (PI) and education are associated with progressively improved clinical outcomes that compare favorably to institutional historical controls and international benchmarks.

RESVERATROL AMELIORATES MITOCHONDRIAL DYSFUNCTION BUT INCREASES THE RISK OF HYPOGLYCEMIA FOLLOWING HEMORRHAGIC SHOCK

H Wang, Y Guan, AL Widlund, LB Becker, JA Baur, CA Sims

The Trauma Center at Penn, University of Pennsylvania

Department of Critical Care Medicine, Qilu Hospital of Shandong University, Jinan, China

Presenter: Hao Wang

Senior Sponsor: Rosemary A. Kozar

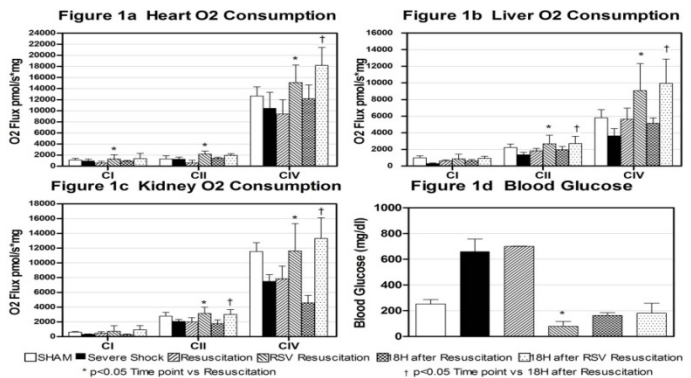
INTRODUCTION: Hemorrhagic shock (HS) may contribute to organ failure by profoundly altering mitochondrial function. Resveratrol (RSV), a naturally occurring polyphenol, has been shown to promote mitochondrial function and regulate glucose homeostasis in diabetes. We hypothesized that, RSV during resuscitation ameliorates HS-induced mitochondrial dysfunction and improves hyperglycemia following acute blood loss.

METHODS: Using a decompensated hemorrhagic shock model, male Long-Evans rats were bled to a mean arterial pressure (MAP) of 40 mmHg until the blood pressure could no longer be maintained without fluid infusion. A MAP of 40 mmHg was then sustained by incrementally infusing lactated Ringer's (LR) until 40% of the shed volume had been returned (Severe Shock). Animals were resuscitated with 4X the shed volume in LR with or without RSV (30 mg/kg) over 60 minutes. Animals (n=6 per group) were sacrificed prior to hemorrhage (Sham), at Severe Shock, following Resuscitation, and 18 hours post-resuscitation. At each time point, mitochondria were isolated from tissue samples (liver, kidney and heart) in order to assess individual respiratory complexes (CI, CII, and CIV) using high resolution respirometry. Blood glucose was recorded at each time point. Hormones that influence blood glucose, including insulin, corticosterone and total glucagon-like peptide-1 (GLP-1), were measured by ELISA.

RESULTS: RSV supplementation following hemorrhagic shock significantly improved cardiac mitochondrial function in all complexes and restored CII and CIV respiratory capacity in liver and kidney tissues (Figure 1a, 1b and 1c). RSV

treated animals had significantly lower blood glucose (116.0 ± 20.2 mg/dl vs. 359.0 ± 79.5 mg/dl, $p < 0.01$) following resuscitation (Figure 1d). RSV was associated with increased GLP-1 (376.1 ± 193.6 ng/ml vs. 194.5 ± 20.8 ng/ml, $p = 0.05$) and decreased plasma insulin (1.4 ± 1.5 ng/ml vs. 12.8 ± 13.7 ng/ml, $p = 0.04$), but had little effect on corticosterone levels.

CONCLUSIONS: Resuscitation with RSV restores mitochondrial function, but increases the risk of hypoglycemia which may be mediated by increased GLP-1 secretion and insulin sensitivity.



Alcohol Withdrawal Syndrome in Critically Ill Patients: Protocolized vs Non-Protocolized Management

CS Cocanour, AJ Berry, P Ghayyem, JJ Duby

University of California, Davis Medical Center, Sacramento, California

Presenter: Christine Cocanour**Senior Sponsor: Christine Cocanour**

INTRODUCTION: Approximately 18-25% of patients with alcohol use disorders admitted to the hospital develop alcohol withdrawal syndrome (AWS). Historically benzodiazepines (BZDs) have been administered in a continuous or scheduled fashion and gradually tapered following a 5-7 day course for AWS. Symptom-triggered dosing of BZDs appears to lead to shorter courses of treatment, lower cumulative BZD dose and more rapid control of symptoms. This study compares the outcomes of critically ill patients with AWS when treated using a protocolized, symptom-triggered, dose-escalation approach versus a non-protocolized approach (standard care).

METHODS: This is a retrospective before-after study of patients >18 years with AWS admitted to an ICU. The Pre-intervention cohort (PRE) was admitted between 2-2008 and 2-2010. The Post-intervention cohort (POST) was admitted between 2-2012 and 1-2013. PRE were treated by standard care and were compared to POST that were given escalating doses of BZDs and/or phenobarbital according to an AWS protocol, titrating to light sedations (RASS of 0 to -2). Baseline characteristics such as gender, age, history of AWS, history of seizure, SOFA score on admit, urine toxicology on admit, and history of psychosis were measured in both groups. 1° outcome measure was ICU LOS. 2° outcomes included BZD use, phenobarbital use, duration of sedation, requirement of mechanical ventilation, and ventilator days.

RESULTS: There were 135 episodes of AWS in 132 critically ill patients. POST (n=75) were younger (50.7 vs 55.7 years) than PRE (n=57). SOFA scores were higher in PRE (6.0 vs 3.8). There was a significant decrease in mean ICU LOS from 9.6+10.5 to 5.3+6.4 days ($p=0.0004$) in the POST group. The POST group also had significantly fewer ventilator days (5.6 ± 13.9 vs 1.31 ± 5.6 days, $p<0.0001$) as well as a significant decrease in BZD usage (319.4 vs 92.8 mg, $p=0.002$). There were significant differences between the two cohorts with respect to need for continuous sedation ($p<0.001$), duration of sedation ($p<0.001$), and intubation secondary to AWS ($p<0.001$). In all of these outcomes, the POST cohort had a notably lower frequency of occurrence.

CONCLUSIONS: A protocolized treatment approach of AWS in critically ill patients involving symptom-triggered, dose escalations of diazepam and phenobarbital may lead to a decreased ICU length of stay, decreased time spent on mechanical ventilation, and decreased benzodiazepine requirements.

OBESITY-INDUCED HYPERCOAGULABILITY PREVENTS ACUTE TRAUMATIC COAGULOPATHY

BH McCully, RK Dean, SP McCully, MA Schreiber
Oregon Health & Science University, Portland, Oregon

Presenter: Belinda McCully

Senior Sponsor: Martin Schreiber

INTRODUCTION: Obesity and hemorrhagic shock (HS) following trauma are two predictors of mortality, but their effects on coagulation are opposed. HS leads to acute traumatic coagulopathy (ATC), producing a hypocoagulable state. Conversely, obesity promotes clotting and impairs fibrinolysis, yielding a hypercoagulable state. High rates of organ failure, thromboembolic disease and early mortality in obese patients may be related to hypercoagulability. We hypothesized that the obesity-induced hypercoagulable state prevents the development of ATC.

METHODS: Male Sprague-Dawley rats (8 weeks) were fed a control (10%kcal/fat) or high fat diet (33%kcal/fat) for 6 weeks. Anesthetized rats were subjected to an uncontrolled splenic hemorrhage. Hypotension (mean arterial pressure; MAP 30-40mmHg) was maintained by intermittent bleeding from a femoral artery catheter over 30 min to induce HS. Physiologic parameters, blood loss, and thrombelastogram (TEG) were measured before and after HS.

RESULTS: Obese rats weighed more than lean rats ($606 \pm 9g$ vs. $485 \pm 4g$; $p < 0.05$). At baseline, obese rats exhibited a shorter time to clot initiation (R), elevated clot strength (MA), and higher coagulation index (CI) compared to lean rats ($p < 0.05$), indicating a hypercoagulable state. Shock was verified by elevated lactate ($p < 0.05$) and BD ($p < 0.05$) in both groups. Post-HS MAP, blood loss, lactate, and BD did not differ between groups. In lean rats, HS prolonged the R and time to 20mm clot (K), while decreasing the rate of clot formation (α -angle) and CI (all $p < 0.05$). In obese rats, HS lowered MA, but did not affect the other TEG parameters. In addition, the R remained lower in obese rats compared to lean rats ($p < 0.05$).

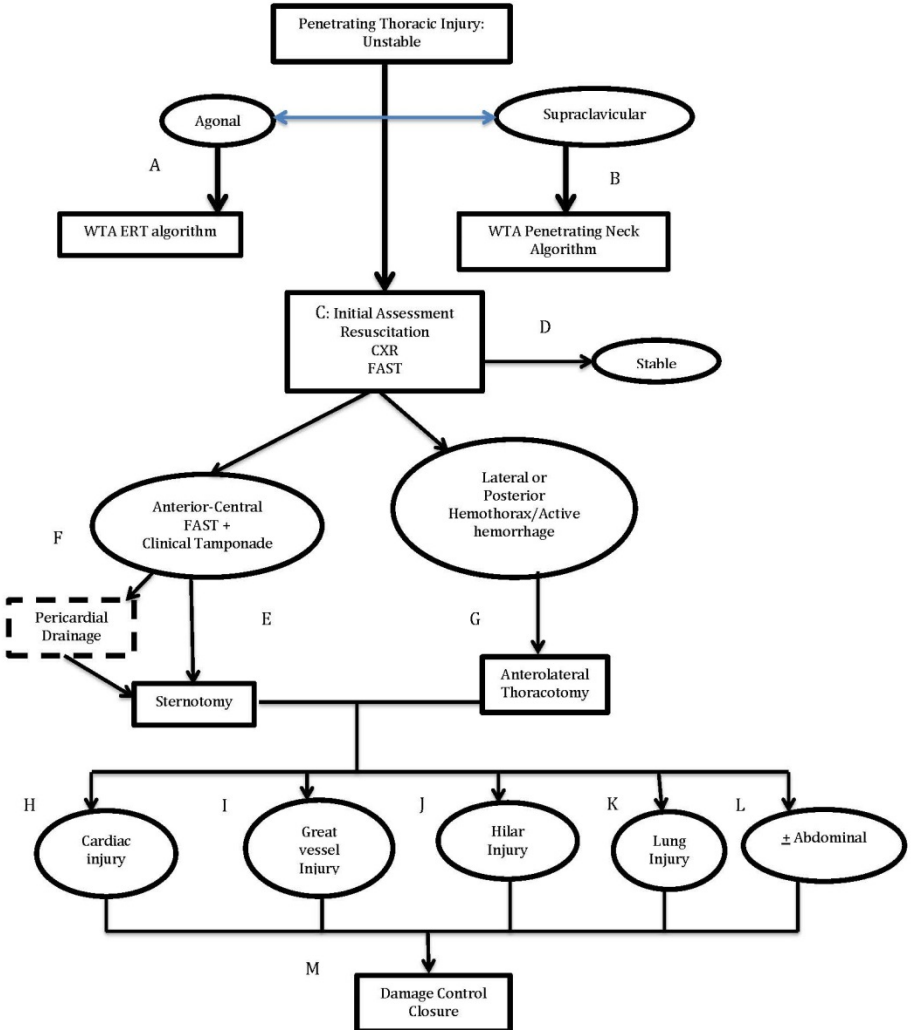
CONCLUSIONS: For the same degree of HS, obesity-induced hypercoagulability prevents the development of ATC. Furthermore, the complications associated with obesity following HS may be mediated by the hypercoagulable state.

	LEAN (n=6)		OBESE (n=6)	
	Baseline	Shock	Baseline	Shock
MAP (mmHg)	116 ± 12	36 ± 1*	114 ± 10	34 ± 2*
Blood loss (ml)	0	7.1 ± 0.4	0	9.8 ± 1.7
Lactate (mmol/L)	0.4 ± 0.1	3.6 ± 0.7*	0.6 ± 0.2	4.7 ± 0.6*
Base Excess (mmol/L)	5.7 ± 0.6	-2.3 ± 1.0*	4.7 ± 0.6	-2.9 ± 1.4*
R (min)	4.7 ± 0.3	6.1 ± 0.3*	2.9 ± 0.6 [†]	3.7 ± 0.9 [†]
K (min)	1.3 ± 0.1	1.6 ± 0.1*	1.0 ± 0.1	1.2 ± 0.9
α -angle (deg)	71 ± 2	68 ± 2*	76 ± 1	73 ± 3
MA (mm)	72 ± 1	67 ± 2	76 ± 1 [†]	69 ± 2*
CI	2.9 ± 0.3	1.0 ± 0.5*	4.8 ± 0.4 [†]	3.4 ± 1.1

Table 1: Data are presented as means ± SE. * $p < 0.05$ vs baseline within group. [†] $p < 0.05$ vs lean within time point.

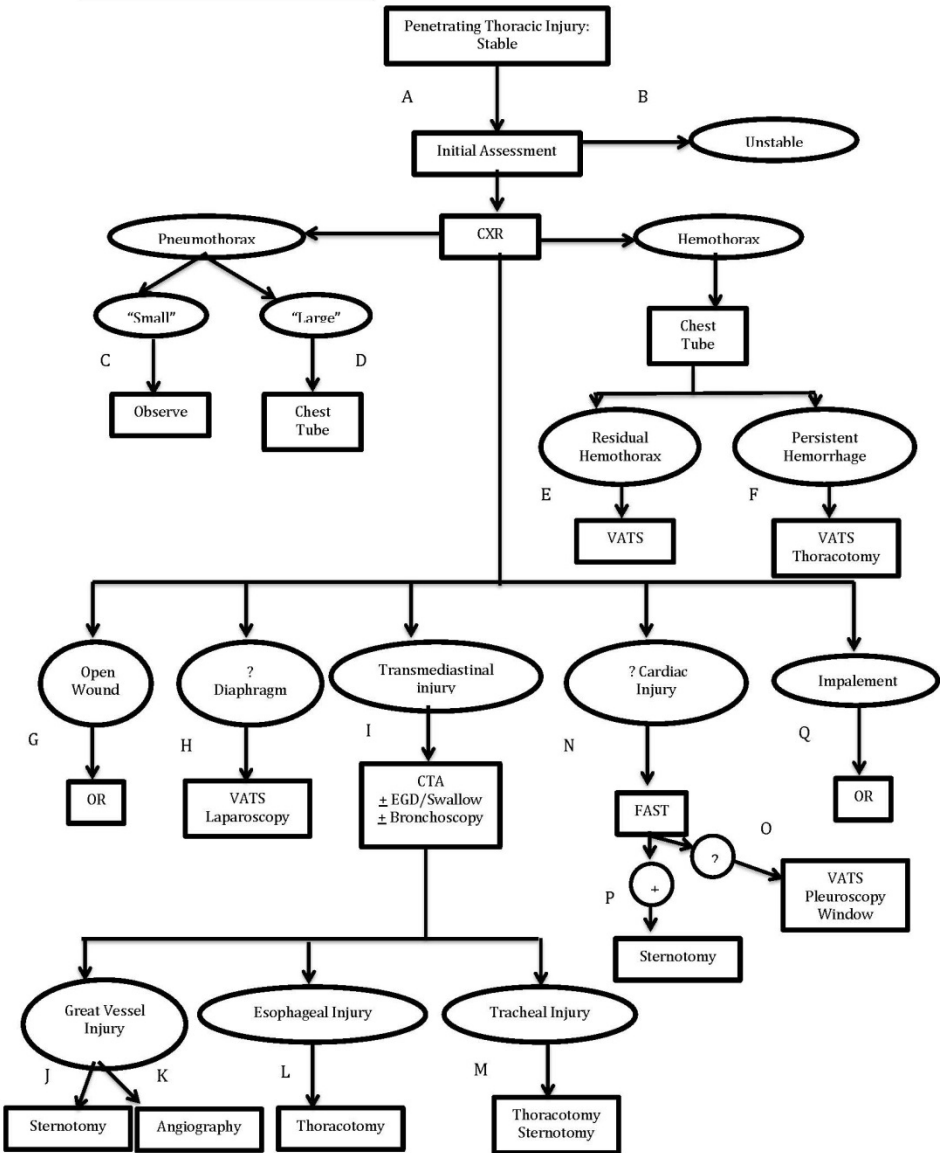
CRITICAL DECISIONS: PENETRATING THORACIC TRAUMA

Figure 1: Assessment and management strategies in the unstable patient

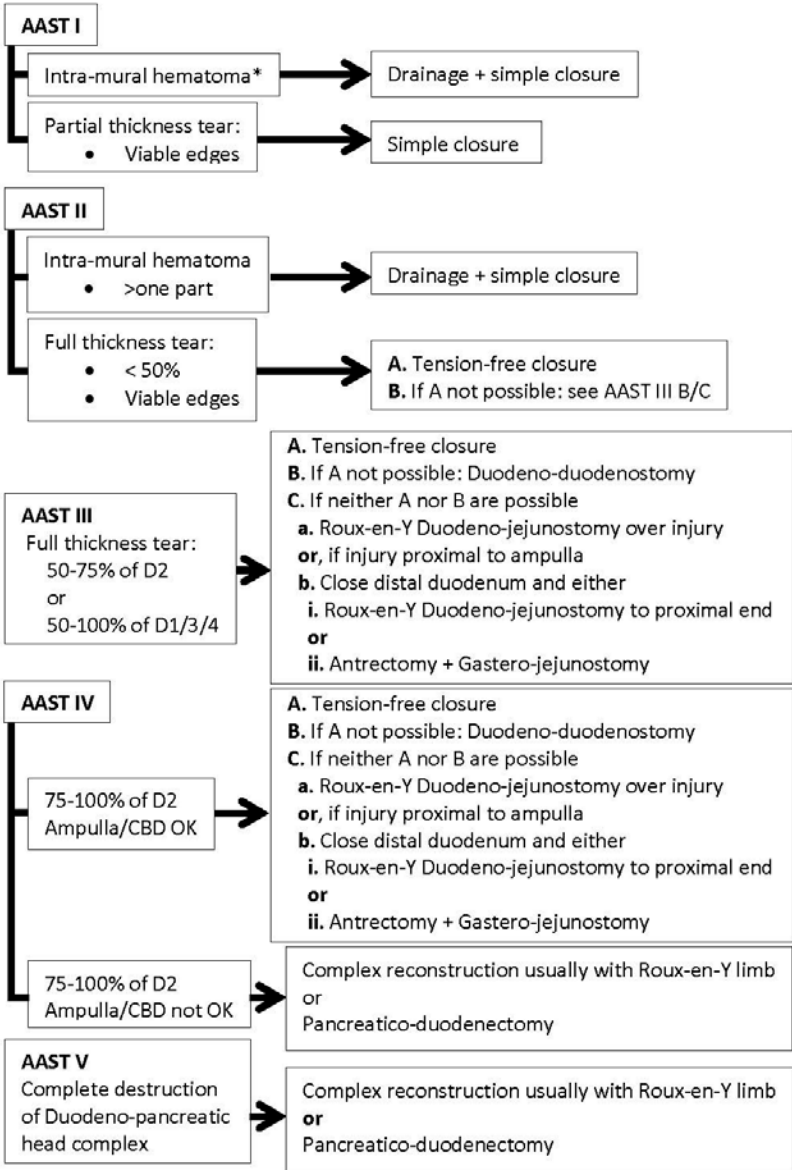


CRITICAL DECISIONS: PENETRATING THORACIC TRAUMA

Figure 2: Stable patient



CRITICAL DECISIONS: DUODENAL TRAUMA



*If detected by CT and no other indication to operate: observe with gastric decompression. If no resolution at 2 weeks – operate and manage as AAST I