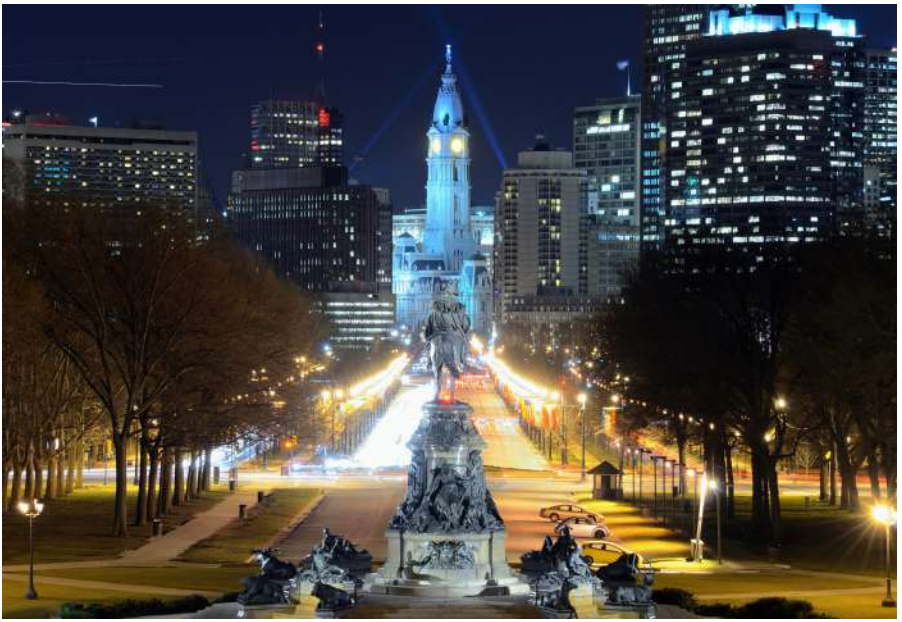
73rd Annual Meeting of the American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery

September 10 — September 13, 2014



PHILADELPHIA MARRIOTT DOWNTOWN

PHILADELPHIA, PA

HISTORICAL BACKGROUND

AAST

The American Association for the Surgery of Trauma started with conversations at the meetings of the Western Surgical Association and Southern Surgical Association in December, 1937. The 14 founders, who were present at one or both of these meetings, subsequently invited another 68 surgeons to a Founding Members meeting in San Francisco on June 14, 1938. The first meeting of the AAST was held in Hot Springs, Virginia, in May, 1939, and Dr. Kellogg Speed's first Presidential Address was published in *The American Journal of Surgery* (47:261-264, 1940). Today, the Association holds an annual scientific meeting, owns and publishes *The Journal of Trauma and Acute Care Surgery*, initiated in 1961, and has approximately 1,300 members from 30 countries.

EDUCATIONAL GRANTS:

The American Association for the Surgery of Trauma wishes to recognize and thank the Childress Institute for Pediatric Trauma for their ongoing support through an educational grant.

The American Association for the Surgery of Trauma wishes to recognize and thank Children's Hospital of Philadelphia for their support through in-kind grant donation.

American Association for the Surgery of Trauma
(AAST)

Annual Meeting of AAST and Clinical Congress of Acute Care Surgery
Learning Objectives and Outcomes

- Exchange knowledge pertaining to current research practices and training in the surgery of trauma.

- Design research studies to investigate new methods of preventing, correcting, and treating acute care surgery (trauma, surgical critical care and emergency surgery) injuries.

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 American Association for the Surgery of Trauma. 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 31.75 *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 6 credits meet the requirements for self-assessment.*

"In order to receive self-assessment credit, you must take and pass the self-assessment test within ten (10) business days of session."

American College of Surgeons
Division of Education

** Self-assessment credits are available for:*
*Wednesday: Acute Care Surgery-Maintenance of Certification (3 hrs), Pediatric Trauma Simulation session (4 hrs)** and Military Symposium (4 hrs)*
Thursday and Friday: Lunch Sessions only (1 hour each)
Total maximum number of self-assessment credits available: 6 hours

** The Pediatric Trauma Simulation Symposium will have a separate email and a separate certificate for the course. If you attend this course, you will receive TWO emails for the AAST Annual Meeting, one on Wednesday afternoon/Thursday morning and the overall meeting evaluation on Saturday morning.

STATEMENT OF ATTENDANCE FORM
THE AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA
73rd Annual Meeting of AAST and Clinical Congress of Acute Care Surgery
Philadelphia Marriott Downtown, Philadelphia, PA, September 10-13, 2014

As a participant in this educational activity, indicate by marking (x) by each session you attended. To receive your CME certificate, follow the instructions below for completing the online evaluation. By September 13, 2014 all registered participants will receive an email with instructions for claiming CME credit. **No paper forms will be accepted. The boxes below are for record-keeping purposes only.**

WEDNESDAY, SEPTEMBER 10, 2014 (total for day 10.5, including 4 for self-assessment credit)

_____ Optional Session: ACS-MOC (3)*
_____ Optional Session: Military (4)*
_____ Optional Session: Pediatric Symposium (4)*
_____ Session I: Papers 1-8 (2.75)
_____ Session II: Master Surgeon Lecture I (.5)
_____ Session III: Challenging Cases Panel I (1.25)
_____ Session IV: Posters (2)

**Self-assessment is available*
*** You can only check one Optional Session*

THURSDAY, SEPTEMBER 11, 2014 (total for day 8.25, including 1 self-assessment credits)

_____ Session V: Master Surgeon Lecture II (.5)
_____ Session VI: Papers 9-12 (1.5)
_____ Session VIII: Papers 13-16 (1.25)
_____ Session IX: Presidential Address (1)
_____ Lunch Sessions (1)*
_____ Session XA: Papers 17-25 (3)
_____ Session XB: Papers 26-34 (3)

**Self-assessment is available*
*** You can only check XA or XB, you cannot check both.*
If you attended one of the six lunch sessions, check here: □

FRIDAY, SEPTEMBER 12, 2014 (total for day 9, including 1 for self-assessment credit)

_____ Session XI: Panel II (.5)
_____ Session XII: Papers Quick Shots (3)
_____ Session XIII: Fitts Lecture (1)
_____ Lunch Sessions (1)*
_____ Session XIVA: Papers 35-44 (3.5)
_____ Session XIVB: Papers 45-54 (3.5)

**Self-assessment is available*
*** You can only check XVA or XVB, you cannot check both.*
If you attended one of the six lunch sessions, check here: □

SATURDAY, SEPTEMBER 13, 2014 (total for day 4)

_____ Session XV: Papers 55-66 (4)

Total hours available: 31.75
Self-assessment hours will be uploaded into the system by AAST Staff.

TOTAL CME HOURS CLAIMING:_____
TOTAL SELF-ASSESSMENT HOURS:_____
***** Please note the Pediatric Symposium on Wednesday will be sent out separately and have its own certificate.

If you are a member of the American College of Surgeons, your completed CME information will be sent to "MY CME Portal Page" and will be updated with the credits within six (6) months of this activity. ACS ID #_____ - you will need this when completing the online evaluation.

ONLINE CME INFORMATION

All registered participants can obtain CME online only. To receive your CME for the 2014 Annual Meeting of AAST and Clinical Congress of Acute Care Surgery, please read over the instructions below. All CME forms must be completed within 30 days after the meeting (by October 13, 2014). *To be eligible for self-assessment credit you MUST take AND pass the self-assessment test within ten (10) business days of the session (September 26, 2014).*

By Saturday, September 13th, the email address on file (the email address you submitted on your registration form) will be sent an email with information for claiming your CME for the 2014 Annual Meeting.

If you are an AAST Member and used the same email address in the AAST membership system to register for the meeting, your information is already in the AAST database and you have an account on the AAST website at www.aast.org. To claim CME please click on the link on the home page once you have signed in using the "log in" button on the top right hand corner.

If you are not an AAST member, but have created an account on www.aast.org and used the same email address to register, you can claim CME by logging in using the account you created at www.aast.org and clicking on the "log in" button on the top right hand corner.

If you are not an AAST member and have not created an account, you will need to create an account. It is suggested that you create an account prior to Saturday, September 13th. However, if you choose not to do that, once you receive the email for CME, you will need to create an account. To create an account after September 13, go to www.aast.org and click on "Create an Account" in the top right hand corner, once your account is created, please refer to the instructions emailed to you to claim CME.

AMERICAN COLLEGE OF SURGEONS | DIVISION OF EDUCATION
JOINT SPONSORSHIP PROGRAM

Disclosure Information

73rd Annual Meeting of AAST and Clinical Congress of Acute Care Surgery

September 10-13, 2014

Philadelphia, PA

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

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

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

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

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
A. Brit Christmas	No	
A. Peter Ekeh	No	
Aaron Jay Dawes	No	
Adil H Haider	No	
Adil Hussain Haider	No	
Ajai Malhotra	No	
ALBERTO FEDERICO GARCIA	No	
Alex Valadka	No	
Ali Cheaito	No	
Ali Salim	No	
Allan B Peetz	No	
Amy Virginia Gore	No	
Anahita Dua	No	
Andre Campbell	No	
Andrew James Kerwin	No	
Andrew Kerwin	No	
Andrew Kirkpatrick	Yes	KCI Corp, research dollars

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Andrew M Nunn	No	
Andrew Peitzman	No	
Angela Neville	No	
Ann Marie Warren	Yes	Baylor University Medical Center; grant support
Anna Ledgerwood	No	
Anne Mosenthal	No	
Ari Leppäniemi	No	
Arun Saini	Yes	Heamonetics, assay support
Ashley B Hink	No	
Ashley Zander	No	
Avery Nathens	No	
Barbara Gaines	No	
Basil Pruitt, Jr.	No	
Bellal Joseph	No	
Bijan Shams Kheirabadi	No	
Bradley Moffat	No	
Brian J Daley	No	
Brian Zuckerbraun	No	
Bryan A Cotton	No	
C. William Schwab	No	
Carl Hauser	No	
Carlos A Ordonez	No	
Carlos V.R. Brown	No	
Carnell Cooper	No	
Carrie Sims	No	
Carrie Valdez	No	
Catherine Garrison Velopulos	No	
Catherine Juillard	No	
Cathy A Maxwell	No	
Chad G. Ball	No	
Charles Adams, Jr.	No	
Charles Lucas	No	
Charles Townsend Harris	No	
Chih-Yuan Fu	No	
Chris Cribari	No	
Chris Salvino	No	
Christine Cocanour	No	
Christopher Cameron McCoy	No	
Christopher Matthew Lamb	No	
Christopher Michetti	No	
Christopher Richardson	No	
Ciara Rosemary Huntington	No	
Clay Cothren Burlew	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Clayton Charles Petro	Yes	Bard, medical supplies, consulting fee, Lifecell, medical supplies, consulting fee
Connie Michell DeLa'O	No	
Corrado Paolo Marini	No	
Cristiane Alencar Domingues	No	
Daithi S Heffernan	No	
Daniel J. Weber	No	
Darren Malinoski	No	
David Bar-Or	No	
David Efron	No	
David Feliciano	No	
David Gomez Jaramillo	No	
David Harrington	No	
David Hoyt	No	
David King	No	
David Livingston	No	
David Spain	No	
Deborah Kuhls	No	
Deborah Stein	No	
Denise M Torres	No	
Dennis Kim	No	
Dennis Wayne Ashley	No	
Dennis Yong Kim	No	
Diogo Freitas Valeiro Garcia	No	
Donald Jenkins	No	
Donald Norris Reed	No	
Donald Trunkey	No	
Eileen Bulger	No	
Elizabeth Alexandra Zubowicz	No	
Ellen Corman	No	
Emily Elizabeth Kenefick Murphy	No	
Eric Ley	No	
Eric Toshlog	No	
Erika Lu Rangel	No	
Ernest Moore	Yes	Haemonetics: Research support, PI. TEM: Research support, PI
Evert Austin Eriksson	No	
Fahim Habib	Yes	Acell; consultant fees
Forrest Bryan Fernandez	No	
Frank Butler	No	
Frederick B Rogers	No	
Frederick Luchette	No	
Frederick Moore	No	
Gabriel E. Ryb	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Gail Toshie Tominaga	No	
Galinos Barmparas	No	
Garth Utter	No	
George Kasotakis	No	
George Velmahos	No	
Glenn Tinkoff	No	
Grace Rozycki	No	
Gregory Jurkovich	No	
Gregory Victorino	No	
Gustavo Fraga	No	
H. Gill Cryer	No	
Hans-Christoph Pape	No	
Hasan Alam	No	
Haytham Kaafarani	No	
Heena P Santry	No	
Heidi Frankel	No	
Henry Cryer	No	
Herb A Phelan	No	
Hiba Abdel-Aziz	No	
Hisatake Matsumoto	No	
Howard Champion	No	
Ilan Rubinfeld	No	
Isadora C Botwinick	No	
J. Jason Hoth	No	
Jacob Robert Peschman	No	
James Davis	No	
James Dunne	No	
James Forrest Calland	No	
James M Haan	No	
James Tyburski	No	
Janeen Rene Jordan	No	
Jasmeet Singh Paul	No	
Jason L Sperry	No	
Jason W Smith	No	
Jay Doucet	No	
Jeffrey David Sedlack	No	
Jennifer C Roberts	No	
Jennifer Lang Mooney	No	
Jeremy Cannon	No	
Jeremy Jon Johnson	No	
Jeremy W. Cannon	No	
Jody M Kaban	No	
John Como	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
John Fildes	No	
John Holcomb	No	
John P Sharpe	No	
Jon Groner	No	
Jon M Gerry	No	
Jose Diaz	No	
Jose L Pascual	No	
Jose Pascual Lopez	No	
Joseph F Rappold	No	
Joseph Galante	No	
Joseph J DuBose	No	
Joseph Minei	No	
Joseph Tepas	No	
Joshua Paul Hazelton	No	
Juan A. Asensio	No	
Juan Carlos Puyana	No	
Juan Duchesne	No	
Julia Grabowski	No	
Junya Tsurukiri	No	
Karen Brasel	No	
Karen Macauley	No	
Karim Brohi	No	
Kathi Ayers	No	
Kazuhide Matsushima	No	
Kenji Inaba	No	
Kenneth G Proctor	No	
Kimberly Davis	No	
Kimberly Joseph	No	
Kimberly Nagy	No	
Kirby Gross	No	
Kiyoyuki W Miyasaka	No	
Krista Kaups	No	
Kristan Lea Staudenmayer	No	
Laura J Moore	No	
Laura Nadine Godat	No	
Lena Napolitano	No	
Lenworth Jacobs, Jr.	No	
Leonard Weireter	No	
Leopoldo Cancio	Yes	Arcos, Inc.; intellectual property rights
Leslie Kobayashi	No	
Lewis Kaplan	No	
Linda Maerz	No	
Lisa Ferrigno	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Lucy Kornblith	No	
Lucy Ruangvoravat	No	
Luke Leenen	No	
Lynne Moore	No	
M. Margaret Knudson	Yes	Nova Nordisk; research grant
Mack Drake	No	
Marc de Moya	No	
Margaret Hedgecock Lauerman	No	
Maria Fernanda Jimenez	No	
Maria Lima Guerreiro	No	
Mark Malangoni	No	
Mark R. Hemmila	Yes	Blue Cross Blue Shield Of Michigan; salary support
Mark Shapiro	No	
Marko Bukur	No	
Markus Tyler Ziesmann	No	
Martin Allan Schreiber	No	
Martin Croce	No	
Martin Donald Zielinski	No	
Martin Schreiber	No	
Martin Zielinski	No	
Mary Elizabeth Schroeder	No	
Mary Fallat	No	
Mary Frances Stuever	No	
Mathew Edavettal	No	
Matthew Bradley	No	
Matthew E Kutcher	No	
Matthew Joseph Delano	No	
Matthew L. Davis	No	
Matthew Martin	No	
Matthew Rosengart	No	
Matthew Wall	No	
Mauricio Velasquez	No	
Megan Brenner	No	
Megan Colleen Reynolds	No	
Michael C Soult	No	
Michael Chang	No	
Michael Charles Reade	No	
Michael J. Sise	No	
Michael Kalina	No	
Michael Nance	No	
Michael Patrick Chapman	Yes	Haemonetics; research support
Michael Truitt	No	
Mitchel Cohen	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Muhammad Jabran Younus	No	
Nicholas Namias	No	
Nicole Stassen	No	
Nobuyuki Saito	No	
Norman McSwain	No	
Oliver Lee Gunter	No	
Orlando Kirton	No	
Oscar Guillamondegui, MD	No	
Patel Vihas	No	
Patricia Ayoung-Chee	No	
Patrick Langdon Bosarge	No	
Patrick Reilly	No	
Paula Ferrada	No	
Peep Talving	No	
Peter Noon Dietrich	No	
Peter Rhee	No	
Philip C. Spinella	Yes	Entegrion; consultant fees; Octapharma; consultant fees;
R. Lawrence Reed	No	
R. Todd Maxson	No	
Rachel A. Hight	No	
Rachel Ann Jacobs	No	
Raminder Nirula	No	
Ramyar Gilani	No	
Randall Friese	No	
Randeep S Jawa	No	
Rao Ivatury	No	
Raul Coimbra	No	
Raymond Fang	No	
Rebecca Schroll	No	
Rebecca Warner Schroll	No	
Reuven Rabinovici	No	
Richard Falcone	No	
Richard Gonzalez	No	
Richard Miller	No	
Richard Mullins	No	
Robert Cooney	No	
Robert David Winfield	No	
Robert John Winchell	No	
Robert Mackersie	No	
Robert Shayn Martin	No	
Robert T Russell	No	
Rodrigo Fabricio Alban	No	
Ronald Gross	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Ronald Maier	No	
Ronald Simon	No	
Ronald Stewart	No	
Ronald Tompkins	No	
Rosemary Kozar	No	
Roxie Albrecht	No	
Russ Kotwal	No	
Salvatore Docimo	No	
Saman Arbarbi	No	
Sapan S Desai	No	
Sarah Balderston Murthi	No	
Sarah Christiaans	No	
Sarah Lombardo	No	
Sarah Majercik	Yes	DePuy Synthes; consulting fees
Scott Sagraves	No	
Sean Patrick McCully	No	
Shabnam Hafiz	No	
Shahid Shafi	No	
Sharon Henry	No	
Sharon Moran	No	
Shelley Timmons	No	
Sonlee Denise West	No	
Stefano Magnone	No	
Stephanie Carol Montgomery	No	
Stephanie Savage	No	
Stephen Barnes	No	
Stephen Flaherty	No	
Stephen S Kaminski	No	
Stephen Wayne Davies	No	
Steven Shackford	No	
Suresh Agarwal	Yes	Acute Innovations; research support and intellectual property rights
Susan Briggs	No	
Susan Rowell	No	
Sylvia Demetra Hobbs	No	
Takashi Fujita	No	
Terry Paul Nickerson	No	
Therese Duane	No	
Thomas Alexander Mitchell	No	
Thomas Scalea	No	
Thomas W Carver	No	
Timothy D Browder	No	
Timothy Fabian	No	
Timothy Patrick Plackett	No	

SPEAKER/MODERATORS/ CHAIRS/DISCUSSANTS	NOTHING TO DISCLOSE?	DISCLOSURE (as it pertains to the content of the presentation)
Tina L Palmieri	No	
Todd Owen Mckinley	No	
Todd Rasmussen	No	
Tomohito Hirao	No	
Tyler Putnam	No	
Vanessa P Ho	No	
Vicente Gracias	No	
Victor Yeewai Kong	No	
Vincent Edward Chong	No	
Walter Biffl	No	
Wendy Wahl	No	
Wenjun Z Martini	No	
William Cioffi	No	
Yasuhiro Otomo	No	
Zara Cooper	No	
Zsolt Balogh	No	
PROGRAM COMMITTEE		
Raul Coimbra, Chair	No	
William Cioffi	No	
Martin Croce	No	
Mary Fallat	No	
David Harrington	No	
Orlando Kirton	No	
David Livingston	No	
Ernest E. Moore	Yes	TEM and Haemonetics, Research Support
Thomas Scalea	No	
George Velmahos	No	

SCHEDULE

(TAB #1)

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SEVENTY-THIRD ANNUAL MEETING
OF THE
AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA
AND
CLINICAL CONGRESS OF ACUTE CARE SURGERY
SEPTEMBER 10-13, 2014
PHILADELPHIA MARRIOTT DOWNTOWN
PHILADELPHIA, PA
GENERAL & SCIENTIFIC PROGRAM SCHEDULE

Wednesday, September 10, 2014

6:30 am - 5:30 pm		**REGISTRATION** Location: Grand Ballroom Salon G-L Foyer
12:30 - 1:00 PM		**WELCOME** Location: Grand Ballroom Salons G-L Presiding: William Cioffi, MD, AAST President
1:00 - 3:40 PM		**SESSION I**: **PLENARY PAPERS** 1-8 Location: Grand Ballroom Salons G-L Moderator: William Cioffi, MD Recorder: Raul Coimbra, MD, PhD
1:00 PM	Paper # 1	THE SPLENIC INJURY OUTCOMES TRIAL: AN ASSOCIATION FOR THE SURGERY OF TRAUMA MULTI-INSTITUTIONAL STUDY Presenter: Ben Zarzaur, MD, MPH Discussant: Andrew Peitzman, MD
1:20 PM	Paper # 2	A CONTROLLED RESUSCITATION STRATEGY IS FEASIBLE AND SAFE IN HYPOTENSIVE TRAUMA PATIENTS: RESULTS OF A PROSPECTIVE RANDOMIZED PILOT TRIAL Presenter: Martin Schreiber, MD Discussant: Thomas Scalea, MD
1:40 PM	Paper # 3	GETTING IT RIGHT: ADHERENCE TO AN ESTABLISHED DIAGNOSTIC THRESHOLD FOR VENTILATOR-ASSOCIATED PNEUMONIA CONTRIBUTES TO LOW FALSE-NEGATIVE RATES IN TRAUMA PATIENTS Presenter: John Sharpe, MD, MS Discussant: Christopher Michetti, MD
2:00 PM	Paper # 4	REDUCING SECONDARY BRAIN INJURY IN TRAUMA PATIENTS: THE EFFECT OF REMOTE ISCHEMIC CONDITIONING Presenter: Bellal Joseph, MD Discussant: Deborah Stein, MD, MPH

2:20 PM	Paper # 5	REGIONAL COLLABORATIVE QUALITY IMPROVEMENT FOR TRAUMA REDUCES COMPLICATIONS AND COSTS Presenter: Mark Hemmila, MD Discussant: David Hoyt, MD
2:40 PM	Paper # 6	MITOCHONDRIAL DAMPS FROM FRACTURES SUPPRESS PULMONARY IMMUNE RESPONSES VIA FORMYL PEPTIDE RECEPTORS 1 AND 2 Presenter: Haipeng Li, MD Discussant: Ronald Maier, MD
3:00 PM	Paper # 7	THE AAST PROSPECTIVE OBSERVATIONAL VASCULAR INJURY TREATMENT (PROOVIT) REGISTRY: MULTICENTER DATA ON MODERN VASCULAR INJURY DIAGNOSIS, MANAGEMENT AND OUTCOMES Presenter: Joseph DuBose, MD Discussant: Howard Champion, FRCS
3:20 PM	Paper # 8	INTRACRANIAL PRESSURE MONITORING AND INPATIENT MORTALITY IN SEVERE TRAUMATIC BRAIN INJURY: A PROPENSITY-SCORE MATCHED ANALYSIS Presenter: Aaron Dawes, MD Discussant: Alex Valadka, MD

3:40 - 4:10 PM	**SESSION II: MASTER SURGEON LECTURE I** Location: Grand Ballroom Salons G-L "*Managing the TBI Patient: Too Much? Too Little? When is it Just Right?"* Alex Valadka, MD

4:10 – 5:25 PM	**SESSION III**: **Challenging Cases Panel** Location: Grand Ballroom Salons G-L Panelists: Roxie Albrecht, MD, Jose Diaz, MD, Ari Leppaniemi, MD, Angela Neville, MD, Matthew Rosengart, MD, MPH Moderator: Thomas Scalea, MD

5:30 - 7:30 PM	**SESSION IV**: Poster Session and Exhibit Hall Open Location: Franklin B

Poster	Professors	Category
1-10	Luke Leenan, MD Carlos Brown, MD	Abdominal Trauma/Burns/Soft Tissue
11-20	Charles Lucas, MD Eric Toschlog, MD	Acute Care Surgery
21-30	Lewis Kaplan, MD Andrew Kerwin, MD	Critical Care
31-40	Vicente Gracias, MD Ajai Malhotra, MD	Neurological Trauma
41-50	Adil Haider, MD Zsolt Balogh, MD	Outcomes/Guidelines I
51-60	A. Peter Ekeh, MD Randall Friese, MD	Outcomes/Guidelines II
61-70	Hans-Christoph Pape, MD Joseph Rappold, MD	Outcomes/Guidelines/Extremity Trauma/Vascular Trauma
71-80	Karen Brasel, MD, MPH Kristan Staudenmayer, MD	Trauma Education/Trauma Systems
81-90	R. Todd Maxson, MD Darren Malinoski, MD	Pediatric Trauma/Shock/Transfusion
91-100	R. Lawrence Reed, MD Krista Kaups, MD	Socieconomics/Ethics/Trauma Systems
101-110	Ronald Stewart, MD Christopher Cribari, MD	Trauma Systems
111-120	Carnell Cooper, MD Glen Tinoff, MD	Trauma Prevention & Epidemiology
121-130	Matthew Wall, Jr., MD James Tyburski, MD	Thoracic Trauma
131-140	Bryan Cotton, MD Juan Carlos Puyana, MD	Shock/Transfusion

6:30 - 8:30 PM	**JOURNAL OF TRAUMA EDITORIAL MEETING** Location: Grand Ballroom Salon C

8:30 - 10:00 PM	**JOURNAL OF TRAUMA RECEPTION** Location: JW's

Thursday, September 11, 2014

6:15 – 7:30 AM	COMMITTEE MEETINGS	VARIOUS LOCATIONS
Acute Care Surgery Committee Meeting		Conference Room 404
Critical Care Committee Meeting		Conference Rooms 401-402
Disaster Ad Hoc Committee Meeting		Conference Rooms 414-415
International Relations Committee Meeting		Conference Room 406
Multi-Institutional Trials Committee Meeting		Conference Rooms 411-412
Prevention Committee Meeting		Conference Rooms 407-408

6:15 am – 7:30 am	**RESIDENT, MEDICAL STUDENT, AND IN-TRAINING FELLOW BREAKFAST** Location: Grand Ballroom Salon C Moderator: Thomas Scalea, MD *" The Making of A Trauma Surgeon: Perspectives on a 35 Year Odyssey"*

6:30 am - 4:00 pm	**REGISTRATION** Location: Grand Ballroom Salon G-L Foyer
7:00 am - 3:30 pm	**EXHIBITS** Location: Franklin B
7:00 am - 8:30 am	**BREAKFAST IN EXHIBIT HALL** Location: Franklin B

7:30 - 8:00 AM	**SESSION V: MASTER SURGEON LECTURE II** Location: Grand Ballroom Salons G-L *"Acute Cholecystitis: When to Operate and How to do it Safely"* Andrew Peitzman, MD

8:00 - 9:20 AM	**SESSION VI: PLENARY PAPERS 9-12** Location: Grand Ballroom Salons G-L Moderator: Grace Rozycki, MD, MBA Recorder: Raul Coimbra, MD, PhD

8:00 AM	Paper # 9	RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA (REBOA) IS A FEASIBLE ALTERNATIVE TO RESUSCITATIVE THORACOTOMY IN TRAUMA PATIENTS WITH NON-COMPRESSIBLE TRUNCAL HEMORRHAGE AND PROFOUND HEMORRHAGIC SHOCK Presenter: Laura Moore, MD Discussant: Timothy Fabian, MD
8:20 AM	Paper # 10	EVALUATION OF THE SAFETY AND FEASIBILITY OF RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA IN JAPAN Presenter: Nobuyuki Saito, MD Discussant: Matthew Wall, Jr., MD

8:40 AM	Paper # 11	THE EARLY EVOLVING SEX HORMONE ENVIRONMENT IS ASSOCIATED WITH SIGNIFICANT CLINICAL OUTCOME AND INFLAMMATORY RESPONSE DIFFERENCES POST-INJURY Presenter: Samuel Zolin, BS Discussant: Reuven Rabinovici, MD
9:00 AM	Paper # 12	SYSTOLIC BLOOD PRESSURE CRITERIA IN THE NATIONAL TRAUMA TRIAGE PROTOCOL FOR GERIATRIC TRAUMA: 110 IS THE NEW 90 Presenter: Joshua Brown, MD Discussant: Frederick Luchette, MD
9:20 – 9:40 AM		**SESSION VII: SCHOLARSHIP PRESENTATIONS** Location: Grand Ballroom Salons G-L Presiding: William Cioffi, MD, AAST President
9:40 – 10:00 AM		***Break in Exhibit Hall***
10:00 - 11:20 AM		**SESSION VIII: ACUTE CARE SURGERY PAPERS 13-16:** Location: Grand Ballroom Salons G-L Moderator: Kimberly Davis, MD, MBA Recorder: Joseph Minei, MD
10:00 AM	Paper # 13	ARTERIOGRAPHY FOR LOWER GI BLEEDING: DOES A PRECEDING ABDOMINAL CT ANGIOGRAM IMPROVE BLEED IDENTIFICATION AND OUTCOME? Presenter: Christina Jacovides, BS Discussant: Leslie Kobayashi, MD
10:20 AM	Paper # 14	SAME DAY COMBINED ERCP AND CHOLECYSTECTOMY: ACHIEVABLE AND COST EFFECTIVE Presenter: Jeffrey Wild, MD Discussant: Michael Chang, MD
10:40 AM	Paper # 15	DEFINING THE ACUTE CARE SURGERY CURRICULUM Presenter: Therese Duane, MD Discussant: Ronald Stewart, MD
11:00 AM	Paper # 16	PROTOCOLIZED MANAGEMENT OF ADHESIVE MECHANICAL SMALL BOWEL OBSTRUCTION: MOVING IT ALONG. Presenter: Janeen Jordan, MD Discussant: Clay Cothren Burlew, MD
11:30 AM - 12:30 PM		**SESSION IX: PRESIDENTIAL ADDRESS** Location: Grand Ballroom Salons G-L Presiding: Thomas Scalea, MD, AAST President-Elect *"Responsibility"* William Cioffi, MD, AAST President

12:30 – 1:45 PM LUNCH SESSIONS I-VI LOCATIONS LISTED ON TICKET

Session I: Management of Complications in the Bariatric Patient by the Acute Care Surgeon
Moderator: Therese Duane, MD
Speakers: John Como, MD, MPH, Mark Shapiro, MD

Session II: Topical Hemostatics in Trauma and Emergency Surgery: When, What and How
Moderator: Stephen Barnes, MD
Speakers: Martin Schreiber, MD, Matthew Martin, MD

Session III: Penetrating Thoracic Injuries: The Cases, the Pitfalls and the Challenges
Speakers: Juan Asensio, MD, Thomas Scalea, MD

Session IV: Writing and Reviewing for the Journal II: Critical Skills
Moderator: Steven Shackford, MD
Speakers: Ernest Moore, MD, Steven Shackford, MD

Session *V:* Recognition and Management of Pediatric Acute Care Emergencies
Moderator: Mary Fallat, MD
Speakers: R. Todd Maxson, MD, Jon Groner, MD

Session VI: REBOA: New Kid on the Block
Moderator: John Holcomb, MD
Speakers: Timothy Fabian, MD, Laura Moore, MD, Todd Rasmussen, MD

1:45 – 2:00 PM ***Break in the Exhibit Hall***

2:00 - 5:00 PM **SESSION XA: PAPERS 17-25**
Location: Grand Ballroom Salons G-L
Moderator: Martin Croce, MD
Recorder: Christine Cocanour, MD

2:00 PM	Paper # 17	NOT ALL BELLIES ARE THE SAME: A COMPARISON OF DAMAGE CONTROL SURGERY FOR INTRA-ABDOMINAL SEPSIS VERSUS TRAUMA Presenter: Jason Smith, MD, PhD Discussant: Andre Campbell, MD
2:20 PM	Paper # 18	THE IMPACT OF TRANEXAMIC ACID ON MORTALITY IN INJURED PATIENTS WITH HYPERFIBRINOLYSIS Presenter: John Harvin, MD Discussant: Ernest Moore, MD

2:40 PM	Paper # 19	OVERWHELMING TPA RELEASE, NOT PAI-1 DEGRADATION, IS RESPONSIBLE FOR HYPERFIBRINOLYSIS IN MASSIVELY TRANSFUSED TRAUMA PATIENTS Presenter: Michael Chapman, MD Discussant: Yasuhiro Otomo, MD
3:00 PM	Paper # 20	LUNG PROTECTIVE VENTILATION (ARDSNET) VERSUS APRV: VENTILATORY MANAGEMENT OF A COMBINED MODEL OF ACUTE LUNG AND BRAIN INJURY Presenter: Stephen Davies, MD, MPH Discussant: Orlando Kirton, MD
3:20 PM	Paper # 21	RECONSTITUTION FLUID TYPE DOES NOT AFFECT PULMONARY INFLAMMATION OR DNA DAMAGE FOLLOWING INFUSION OF LYOPHILIZED PLASMA Presenter: Sean McCully, MD, MS Discussant: Frederick Moore, MD
3:40 PM	Paper # 22	CLEARLY DEFINING PEDIATRIC MASSIVE TRANSFUSION: CUTTING THROUGH THE FOG AND FRICTION WITH COMBAT DATA Presenter: Lucas Neff, MD Discussant: Michael Nance, MD
4:00 PM	Paper # 23	ANGIOTENSIN INHIBITION DECREASES MULTIPLE ORGAN FAILURE IN OBESE TRAUMA PATIENTS Presenter: Robert Winfield, MD Discussant: Robert Cooney, MD
4:20 PM	Paper # 24	TIME FOR CLOSURE: A DEDICATED TRAUMA ICU IS ASSOCIATED WITH LOWER POST INJURY COMPLICATION RATES AND DEATH AFTER MAJOR COMPLICATIONS Presenter: Marko Bukur, MD Discussant: Charles Adams, Jr., MD
4:40 PM	Paper # 25	FACTOR VIIA ADMINISTRATION IN TRAUMATIC BRAIN INJURY: AN AAST-MITC PROPENSITY SCORE ANALYSIS Presenter: Sarah Lombardo, MD, MSc Discussant: M. Margaret Knudson, MD
2:00 - 5:00 PM		**SESSION XB: PAPERS: 26-34** Location: Grand Ballroom Salons A, B & F Moderator: Ari Leppaniemi, MD Recorder: Robert Mackersie, MD
2:00 PM	Paper # 26	OBESITY AND CLOTTING: BMI INDEPENDENTLY CONTRIBUTES TO HYPERCOAGULABILITY AFTER INJURY Presenter: Lucy Kornblith, MD Discussant: Hasan Alam, MD

2:20 PM	Paper # 27	THE NEW METRIC TO IDENTIFY LARGE-VOLUME HEMORRHAGE: RESULTS OF A PROSPECTIVE STUDY OF THE CRITICAL ADMINISTRATION THREHSOLD Presenter: Stephanie Savage, MD Discussant: John Holcomb, MD
2:40 PM	Paper # 28	A PHARMACOLOGIC APPROACH TO VAGAL NERVE STIMULATION PREVENTS MESENTERIC LYMPH TOXICITY AFTER HEMORRHAGIC SHOCK Presenter: Koji Morishita, MD Discussant: William Cioffi, MD
3:00 PM	Paper # 29	FIBRINOGEN CONCENTRATE ADMINISTRATION INHIBITS ENDOGENOUS FIBRINOGEN SYNTHESIS IN PIGS AFTER TRAUMATIC HEMORRHAGE Presenter: Wenjun Martini, PhD Discussant: Peter Rhee, MD, MPH
3:20 PM	Paper # 30	ARE WE MEASURING THE RIGHT THING? CARDIAC DYSFUNCTION, NOT CARDIAC OUTPUT, IS PREDICTIVE OF OUTCOME IN CRITICALLY ILL SURGICAL PATIENTS Presenter: Sarah Murthi, MD Discussant: Paula Ferrada, MD
3:40 PM	Paper # 31	POST-RESUSCITATIVE HYPERCHLOREMIC METABOLIC ACIDOSIS IS ASSOCIATED WITH ACUTE KIDNEY INJURY Presenter: Dennis Kim, MD Discussant: Lena Napolitino, MD
4:00 PM	Paper # 32	NATIONAL ESTIMATES OF PREDICTORS OF OUTCOMES FOR EMERGENCY GENERAL SURGERY Presenter: Adil Shah, MD Discussant: Kristan Staudenmayer, MD
4:20 PM	Paper # 33	IMPLEMENTATION OF AN ACUTE CARE SURGERY IN A COMMUNITY HOSPITAL: IMPACT ON HOSPITAL EFFICIENCY AND PATIENT OUTCOMES Presenter: Michael Kalina, DO Discussant: Lewis Kaplan, MD
4:40 PM	Paper # 34	COMPARISON OF ATRIOCAVAL SHUNTING WITH PERIHEPATIC PACKING VS PERIHEPATIC PACKING ALONE FOR RETROHEPATIC VENA CAVA INJURIES IN A SWINE MODEL Presenter: Joshua Hazelton, DO Discussant: David Feliciano, MD

Friday, September 12, 2014

6:15 – 7:30 AM	COMMITTEE MEETINGS	VARIOUS LOCATIONS
ACS Program Directors Meeting		Conference Room 403
AAST Board of Managers Meeting		Conference Room 410
Education/CME Committee Meeting		Conference Room 404
Geriatric Trauma Ad Hoc Committee Meeting		Conference Rooms 407-408
Injury Assessment and Outcome Committee Meeting		Conference Rooms 414-415
Military Liaison Committee Meeting		Conference Rooms 401-402
Pediatric Trauma Committee Meeting		Conference Rooms 411-412
Publications and Communication Committee Meeting		Conference Room 406

6:15 – 7:30 AM		**INTERNATIONAL ATTENDEE BREAKFAST** Location: Grand Ballroom Salon C Presenter: Hans-Christoph Pape, MD
7:00 AM - 3:00 PM		**REGISTRATION** Location: Grand Salon G-L Foyer
7:00 AM - 2:00 PM		**EXHIBITS** Location: Franklin B
7:00 AM - 8:30 AM		**BREAKFAST IN EXHIBIT HALL** Location: Franklin B
7:30 - 8:00 AM		**SESSION XI: DEMYSTIFYING GOVERNMENT RESEARCH** Location: Grand Ballroom Salons G-L Presenter: Todd Rasmussen, MD
8:00 - 10:55 AM		**SESSION XII: QUICKSHOTS** Location: Grand Ballroom Salons G-L Moderator: C. William Schwab, MD
8:01 AM	QS # 1	CONTEMPORARY MANAGEMENT AND OUTCOMES OF BLUNT THORACIC AORTIC INJURY: A MULTICENTER RETROSPECTIVE STUDY Presenter: Megan Brenner, MD Discussant: Michael Sise, MD
8:08 AM	QS # 2	EARLY WHOLE BLOOD AUTO-TRANSFUSION: AN UNDEFINED PRACTICE IN CIVILIAN TRAUMA Presenter: Peter Rhee, MD, MPH Discussant: Juan Duchesne, MD
8:15 AM	QS # 3	DEFINING THE EXCESS MORBIDITY AND MORTALITY ATTRIBUTABLE TO EMERGENCY GENERAL SURGERY Presenter: Allan Peetz, MD Discussant: Shahid Shafi, MD

8:22 AM	QS # 4	INDUCING ACUTE TRAUMATIC COAGULOPATHY IN VITRO Presenter: Benjamin Howard, MD, MPH Discussant: Walter Biffl, MD
8:29 AM	QS # 5	INHALED, NEBULIZED SODIUM NITRITE PROTECTS AGAINST TRAUMA/HEMORRHAGIC SHOCK INDUCED TISSUE INJURY AND INFLAMMATION Presenter: Benjamin Kautza, MD Discussant: Rosemary Kozar, MD, PhD
8:36 AM	QS # 6	TWO YEAR CESSATION OF RESIDENT TEAMWORK TRAINING IMPACTS TRAUMA RESUSCITATION PERFORMANCE AND EFFICIENCY Presenter: Charles Harris, MD Discussant: Joseph Galante, MD
8:43 AM	QS # 7	OPERATIVE VERSUS NON-OPERATIVE MANAGEMENT OF MULTIPLE RIB FRACTURES Presenter: Mauricio Velasquez, MD Discussant: Marc de Moya, MD
8:50 AM	QS # 8	A COMPARISON OF DIAGNOSTIC PERITONEAL LAVAGE TO COMPUTED TOMOGRAPHY IN THE DIAGNOSIS OF THORACO-ABDOMINAL STAB WOUNDS Presenter: Reza Salabat, MD Discussant: Leonard Weireter, Jr., MD
8:57 AM	QS # 9	THE IMPACT OF IMAGE-SHARING ON THE EVALUATION OF TRAUMA TRANSFER PATIENTS IN A RURAL TRAUMA SYSTEM Presenter: Tanveer Zamani, MD Discussant: Sharon Henry, MD
9:04 AM	QS # 10	LEVEL I ACADEMIC TRAUMA CENTER INTEGRATION AS A MODEL FOR SUSTAINING COMBAT SURGICAL SKILLS: THE RIGHT SURGEON IN THE RIGHT PLACE FOR THE RIGHT TIME Presenter: Rachel Hight, MD Discussant: Nicholas Namias, MD
9:11 AM	QS # 11	RISK ASSESSMENT MODELS FOR PREDICTING VENOUS THROMBOEMBOLISM IN TRAUMA PATIENTS ARE NOT ADEQUATE Presenter: Jan-Michael Van Gent, DO Discussant: Avery Nathens, MD, PhD, MPH
9:18 AM	QS # 12	IMPACT OF SOCIO-ECONOMIC STATUS ON HOSPITAL LENGTH OF STAY FOLLOWING INJURY: A MULTICENTER COHORT STUDY Presenter: Lynne Moore, PhD Discussant: Karen Brasel, MD, MPH

9:25 AM	QS # 13	REPAIR VERSUS LIGATION OF MAJOR VENOUS INJURY AFTER PENETRATING TRAUMA: IS THERE A DIFFERENCE IN DEVELOPMENT OF PULMONARY EMBOLISM? Presenter: Casey Allen, MD Discussant: Ronald Simon, MD
9:32 AM	QS # 14	ULTRASOUND VS. PALPATION GUIDED RADIAL ARTERY CATHETERIZATION: A RANDOMIZED CONTROLLED TRIAL Presenter: Lucy Ruangvoravat, MD Discussant: Christine Cocanour, MD
9:39 AM	QS # 15	A PROSPECTIVE EVALUATION OF SURGICAL RESIDENT INTERPRETATION OF CT SCANS FOR TRAUMA Presenter: Reinaldo Morales, MD Discussant: David Efron, MD
9:46 AM	QS # 16	WELCOME BACK: FACTORS ASSOCIATED WITH EARLY READMISSION FOLLOWING TRAUMA Presenter: Jennifer Roberts, MD, MS Discussant: David King, MD
9:53 AM	QS # 17	FIBRINOLYSIS DOES NOT OCCUR FOLLOWING RESUSCITATION IN SEVERE HEMORRHAGIC SHOCK REQUIRING IMMEDIATE OPERATION Presenter: Mona Taleb, MD Discussant: Mitchell Cohen, MD
10:00 AM	QS # 18	INTRACRANIAL PRESSURE MONITORING DURING THERAPEUTIC CEREBROSPINAL FLUID DRAINAGE: "ONLY PART OF THE STORY?" Presenter: Raymond Fang, MD Discussant: Susan Rowell, MD
10:07 AM	QS # 19	COMPLICATIONS OF DAMAGE CONTROL AND DEFINITIVE LAPAROTOMY IN COMBAT: 2002-2011 Presenter: Thomas Mitchell, MD Discussant: C. William Schwab, MD
10:14 AM	QS # 20	STRESS-INDUCED HYPERGLYCEMIA IS ASSOCIATED WITH HIGHER MORTALITY IN SEVERE TRAUMATIC BRAIN INJURY Presenter: Patrick Bosarge, MD Discussant: Jay Doucet, MD
10:21 AM	QS # 21	DETERMINING FACTORS THAT IMPROVE RESPONSE TO INPATIENT REHABILITATION IN PATIENTS WITH MODERATE TO SEVERE TRAUMATIC BRAIN INJURY Presenter: Fred McLafferty, BA Discussant: Garth Utter, MD

10:28 AM	QS # 22	THE DISPARITY GAP IN TRAUMA DOES NOT NARROW FOR UNINSURED ADULTS SUFFERING SEVERE INJURY Presenter: Jon Gerry, MD Discussant: Adil Haider, MD
10:35 AM	QS # 23	BLUNT DUODENAL TRAUMA, IS NON-OPERATIVE MANAGEMENT SAFE? Presenter: Brandon Bonds, MD Discussant: Anna Ledgerwood, MD
10:42 AM	QS # 24	MICROARRAY ANALYSIS OF GENE EXPRESSION PROFILES IN TRAUMATIC BRAIN INJURED PATIENTS: IMPACT OF PREHOSPTIAL FLUID RESUSCITATION Presenter: Matthew Delano, MD, PhD Discussant: Eric Ley, MD
10:49 AM	QS # 25	CAN MESENCHYMAL STEM CELLS REVERSE CHRONIC STRESS-INDUCED IMPAIRMENT OF WOUND HEALING FOLLOWING TRAUMATIC INJURY? Presenter: Amy Gore, MD Discussant: Gregory Victorino, MD
10:55-11:15 AM		***Break in the Exhibit Hall***
11:15 AM - 12:15 PM		**SESSION XIII: FITTS LECTURE** Location: Grand Ballroom Salons G-L *"Genomics of Injury - The Glue Grant Experience"* Ronald Tompkins, MD

12:15-1:30 PM LUNCH SESSIONS VII-XII LOCATIONS LISTED ON TICKET

Session VII: Managing Unexpected Findings at Emergency Laparotomy
Moderator: Clay Cothren Burlew, MD
Speakers: Hasan Alam, MD, Patrick Reilly, MD

Session VIII: What's New in Reversal of Systemic Anticoagulation: New Drugs, New Challenges
Moderator: Mitchell Cohen, MD
Speakers: Donald Jenkins, MD, Bellal Joseph, MD

Session IX: Surgical Management of Arterial and Venous Pelvic Hemorrhage
Speakers: Raul Coimbra, MD, PhD, David Feliciano, MD

Session X: What Every Trauma Surgeon Should Know About Managing an Unexpected, Multiple Casualty Diaster
Moderators: Joseph Rappold, MD and Susan Briggs, MD
Speakers: Jay Doucet, MD, M. Margaret Knudson, MD, George Velmahos, MD

Session XI: Death, Dying and Futile Care in the ICU, ED and OR - What Have We Learned?
Moderator: Linda Maerz, MD
Speakers: Bryan Cotton, MD, Richard Miller, MD, Anne Mosenthal, MD

Session XII: Assessment of Geriatric Patient After Falls
Moderator: Frederick Luchette, MD
Speakers: Saman Arbabi, MD, Ellen Corman, MD, Zara Cooper, MD

1:30 - 4:50 PM		**SESSION XIVA: PAPERS 35-44** Location: Grand Ballroom Salons G-L Moderator: Donald Trunkey, MD Recorder: David Spain, MD
1:30 PM	Paper # 35	CORRELATION OF CEREBRAL OXYGEN DYNAMICS AND METABOLIC CRISIS IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY (sTBI) Presenter: Michael Stiefel, MD Discussant: Jose Pascual Lopez, MD
1:50 PM	Paper # 36	A COST ANALYSIS OF SURGICAL STABILIZATION VERSUS CONVENTIONAL MANAGEMENT OF SEVERE RIB FRACTURES Presenter: Sarah Majercik, MD, MBA Discussant: Suresh Agarwal, MD
2:10 PM	Paper # 37	CAN WE EVER STOP WORRYING ABOUT VENOUS THROMBOEMBOLISM AFTER TRAUMA? Presenter: Laura Godat, MD Discussant: Ali Salim, MD
2:30 PM	Paper # 38	VARIATIONS IN IMPLEMENTATION OF ACUTE CARE SURGERY: RESULTS FROM A NATIONAL SURVEY OF UNIVERSITY-AFFILIATED HOSPITALS Presenter: Heena Santry, MD Discussant: John Fildes, MD
2:50 PM	Paper # 39	IS IT A MYTH THAT THE WHOLE-BODY COMPUTED TOMOGRAPHY IMPROVES THE OUTCOME FOR POLY-TRAUMA PATIENTS WITH AN ISS 16 AND OVER ? : A PROPENSITY-ADJUSTED ANALYSIS Presenter: Takashi Fujita, MD Discussant: H. Gill Cryer, MD, PhD
3:10 PM	Paper # 40	THE ANATOMIC SEVERITY OF CHEST WALL INJURIES DOES NOT PREDICT POST-RECOVERY PULMONARY SYMPTOMS: A PROSPECTIVE COHORT STUDY Presenter: Tejveer Dhillon, MD Discussant: Raminder Nirula, MD, MPH
3:30 PM	Paper # 41	A GERIATRIC SPECIFIC RIB FRACTURE PROTOCOL SIGNIFICANTLY IMPROVES MORTALITY Presenter: Sean Monagahan, MD Discussant: Ronald Gross, MD

3:50 PM	Paper # 42	INTRODUCTION OF A MOBILE DEVICE BASED TERTIARY SURVEY APPLICATION REDUCES MISSED INJURIES: A MULTI-CENTER PROSPECTIVE STUDY Presenter: Bradley Moffat, MD Discussant: Leopoldo Cancio, MD
4:10 PM	Paper # 43	PRIORITIZING QUALITY IMPROVEMENT IN ACUTE CARE SURGERY Presenter: Christopher McCoy, MD Discussant: David Harrington, MD
4:30 PM	Paper # 44	DOES RESUSCITATION WITH PLASMA INCREASE THE RISK OF VENOUS THROMBOEMBOLISM? Presenter: Ashley Zander, DO Discussant: Heidi Frankel, MD
1:30 - 4:50 PM		**SESSION XIVB: PAPERS 45-54** Location: Grand Ballroom Salons A, B & F Moderator: Robert Winchell, MD Recorder: David Livingston, MD
1:30 PM	Paper # 45	USING A VIDEO DECISION-SUPPORT TOOL TO INFORM SURROGATE DECISIONS IN THE SURGICAL INTENISIVE CARE UNIT Presenter: Zara Cooper, MD Discussant: David Livingston, MD
1:50 PM	Paper # 46	LETHAL NOW OR LETHAL LATER: THE NATURAL HISTORY OF GRADE IV BLUNT CEREBROVASCULAR INJURY Presenter: Margaret Lauerman, MD Discussant: Martin Croce, MD
2:10 PM	Paper # 47	THE VALIDITY OF ABDOMINAL EXAMINATION IN BLUNT TRAUMA PATIENTS WITH DISTRACTING INJURIES Presenter: Jack Rostas, MD Discussant: Andrew Kirkpatrick, MD, CD, MHSc
2:30 PM	Paper # 48	SURGEON PERFORMED ULTRASOUND (SPUS) IN PREDICTING WOUND INFECTIONS: NO COLLECTION, NO INFECTION. Presenter: Christopher Barrett, MD Discussant: Nicole Stassen, MD
2:50 PM	Paper # 49	AN ANALYSIS OF THE EFFECTIVENESS OF A STATE TRAUMA SYSTEM: TREATMENT AT DESIGNATED TRAUMA CENTERS IS ASSOCIATED WITH AN INCREASED PROBABILITY OF SURVIVAL Presenter: Dennis Ashley, MD Discussant: Gregory Jurkovich, MD

3:10 PM	Paper # 50	A REASSESSMENT OF THE IMPACT OF TRAUMA SYSTEMS CONSULTATION ON REGIONAL TRAUMA SYSTEM DEVELOPMENT Presenter: Robert Winchell, MD Discussant: Richard Mullins, MD
3:30 PM	Paper # 51	INTIMATE PARTNER VIOLENCE – RISKS GO BEYOND THE VIOLENCE: ASSOCIATION OF INTIMATE PARTNER VIOLENCE WITH MENTAL ILLNESS AND SUBSTANCE ABUSE AMONG FEMALES ADMITTED TO A RURAL LEVEL-I TRAUMA CENTER Presenter: Ashley Hink, MD, MPH Discussant: James Davis, MD
3:50 PM	Paper # 52	IS TRAUMATIC VIOLENCE GETTING BETTER OR WORSE? NON-FATAL GUN VIOLENCE AT AN URBAN TRAUMA CENTER Presenter: Vincent Chong, MD, MS Discussant: Lenworth Jacobs, Jr., MD, MPH
4:10 PM	Paper # 53	TRAUMA PATIENT READMISSIONS: WHY DO THEY COME BACK FOR MORE? Presenter: Laura Petrey, MD Discussant: Mark Malangoni, MD
4:30 PM	Paper # 54	MINIMALLY INVASIVE IS MAXIMALLY EFFECTIVE: THERAPEUTIC AND DIAGNOSTIC LAPAROSCOPY FOR PENETRATING ABDOMINAL INJURIES Presenter: Paul Chestovich, MD Discussant: Rao Ivatury, MD

4:50 - 5:00 PM	**MILITARY AWARDS CEREMONY** Location: Grand Ballroom Salons G-L Presiding: William Cioffi, MD, AAST President
5:00 - 6:15 PM	**AAST ANNUAL BUSINESS MEETING** **Members Only** Location: Grand Ballroom Salons G-L
7:30 - 10:00 PM	**AAST RECEPTION AND BANQUET** Location: Grand Ballroom Foyer and Salons A-F

Saturday, September 13, 2014

7:00 - 8:00 AM		**NEW FELLOWS BREAKFAST** Location: Conference Rooms 411-412
7:30 – 10:00 AM		**REGISTRATION** Location: Grand Salon G-L Foyer
7:30 – 9:00 AM		**BREAKFAST** Location: Grand Salon G-L Foyer
8:00 AM - 12:00 PM		**SESSION XV: Papers 55-66** Location: Grand Ballroom Salons G-L Moderator: Thomas Scalea, MD Recorder: Susan Briggs, MD, MPH
8:00 AM	Paper # 55	TWO ARE BETTER THAN ONE: SYNERGY OF BETA-BLOCKADE AND STATIN THERAPY ON SURVIVAL IN SEPSIS Presenter: Irada Ibrahim-zada, MD, PhD Discussant: Carl Hauser, MD
8:20 AM	Paper # 56	SAVING LIVES AND SAVING MONEY: HOSPITAL-BASED VIOLENCE PREVENTION IS COST-EFFECTIVE Presenter: Randi Smith, MD, MPH Discussant: Carnell Cooper, MD
8:40 AM	Paper # 57	PROSPECTIVE, MULTICENTER DERIVATION OF A CLINICAL DECISION RULE FOR THORACIC AND LUMBAR SPINE EVALUATION AFTER BLUNT TRAUMA Presenter: Kenji Inaba, MD Discussant: Carrie Sims, MD
9:00 AM	Paper # 58	THE EVIL OF GOOD IS BETTER: MAKING THE CASE FOR BASIC LIFE SUPPORT TRANSPORT OF PENETRATING TRAUMA VICTIMS IN AN URBAN ENVIRONMENT Presenter: Joseph Rappold, MD Discussant: Norman McSwain, Jr., MD
9:20 AM	Paper # 59	MORTALITY FOLLOWING EMERGENCY SURGERY CONTINUES TO RISE AFTER DISCHARGE IN THE ELDERLY Presenter: Erika Rangel, MD, MS Discussant: George Velmahos, MD, PhD
9:40 AM	Paper # 60	PREVENTING MOTOR VEHICLE CRASHES THROUGH GRADUATED DRIVING LICENSING LAWS IN MASSACHUSETTS: A POPULATION-BASED STUDY Presenter: Haytham Kaafarani, MD, MPH Discussant: Barbara Gaines, MD

10:00 AM	Paper # 61	THE EFFECT OF TISSUE DAMAGE VOLUME ON SYSTEMIC INFLAMMATION AND ORGAN FAILURE Presenter: Travis Frantz, MS4 Discussant: Basil Pruitt, Jr., MD
10:20 AM	Paper # 62	DOES SIZE MATTER, OR IS EXPERIENCE WHAT REALLY COUNTS? ANNUAL CLINICAL EXPERIENCE (PER SURGEON) IS ASSOCIATED WITH IMPROVED SURVIVAL Presenter: Richard Doty Discussant: Joseph Minei, MD
10:40 AM	Paper # 63	THE CLINICAL SIGNIFICANCE OF SOLUBULE RAGE IN PATIENTS WITH SEVERE SEPSIS Presenter: Hisatake Matsumoto, MD Discussant: Eileen Bulger, MD
11:00 AM	Paper # 64	THE PEDIATRIC TRAUMA CENTER AND THE INCLUSIVE TRAUMA SYSTEM: IMPACT ON SPLENECTOMY RATES Presenter: Emily Murphy, MD Discussant: Mary Fallat, MD
11:20 AM	Paper # 65	EMERGENCY GENERAL SURGERY OUTCOMES IN TEACHING VERSUS NON-TEACHING HOSPITALS Presenter: Syed Nabeel Zafar, MD, MPH Discussant: Gregory Jurkovich, MD
11:40 AM	Paper # 66	PNEUMOMEDIASTINUM FOLLOWING BLUNT TRAUMA: WORTH WHILE A WORKUP? Presenter: Konstantinos Chouliaras, MD Discussant: Oscar Guillamondegui, MD

AAST INFORMATION

(TAB #2)

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Hartford, Connecticut

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2013-2014

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Memphis, Tennessee

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FUTURE AAST MEETINGS

74th Annual Meeting of the American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery

September 9-12, 2015
Wynn Las Vegas
Las Vegas, NV

75th Annual Meeting of the American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery

September 14-17, 2016
Hilton Waikoloa Village
Waikoloa, HI

76th Annual Meeting of the American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery

September 13-16, 2017
Baltimore Marriott Waterfront
Baltimore, MD

77th Annual Meeting of the American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery

September 26-29, 2018
Manchester Grand Hyatt
San Diego, CA

78th Annual Meeting of the American Association for the Surgery of Trauma and Clinical Congress of Acute Care Surgery

September 18-21, 2019
Sheraton Dallas
Dallas, TX

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1948	Chicago, Illinois	Casper F. Hegner, M.D.
1947	Atlantic City, New Jersey	Ralph G. Carothers, M.D.
1946	San Antonio, Texas	Grover C. Penberthy, M.D.
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1944	Chicago, Illinois	Charles S. Venable, M.D.
1943	No Meeting Due to War	Henry C. Marble, M.D.
1942	Boston, Massachusetts	Henry C. Marble, M.D.
1941	Montreal, Canada	Fraser B. Gurd, M.D.
1940	Atlantic City, New Jersey	Edgar L. Gilcreest, M.D.
1939	Hot Springs, Virginia	Kellogg Speed, M.D.

ABSTRACTS OF PAPERS

(TAB #3)

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SEVENTY-THIRD MEETING OF THE AMERICAN ASSOCATION FOR THE SURGERY OF TRAUMA AND CLINICAL CONGRESS OF ACUTE CARE SURGERY

Category 1 credit hours will be awarded based upon actual hours attended. Total number of hours will be calculated from information individual physicians provide in the online CME evaluation forms.

WELCOME
Wednesday, September 10, 2014, 12:30 PM -1:00 PM
GRAND BALLROOM SALONS G-L
PRESIDING: William G. Cioffi, M.D., AAST President

SESSION I: Plenary – Papers #1- #8
Wednesday, September 10, 2014, 1:00 PM – 3:40 PM
GRAND BALLROOM SALONS G-L
PRESIDING: William Cioffi, M.D.
RECORDER: Raul Coimbra, M.D., Ph.D.

Session: I: Plenary Papers 1-8
Paper 1: 1:00 - 1:20 PM

THE SPLENIC INJURY OUTCOMES TRIAL: AN AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA MULTI-INSTITUTIONAL STUDY

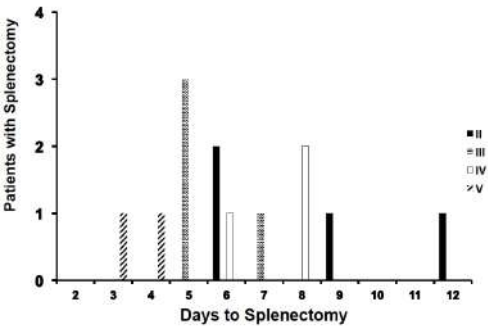
Ben L. Zarzaur* MD,MPH, Rosemary Kozar* MD,Ph.D., John G. Myers* MD, Jeffrey A. Claridge* MD, MS, Thomas M. Scalea* MD, Todd A. Neideen* MD, Adrian A. Maung* MD, Louis Alarcon* MD, Aaron Scifres* MD, Alain Corcos* MD, Andrew Kerwin* MD, Raul Coimbra* MD,Ph.D., AAST Multi-Institutional Trials Committee

Invited Discussant: Andrew Peitzman, MD

Introduction: Delayed splenic rupture resulting in delayed splenectomy (DS) after attempted non-operative (NON-OP) management of blunt splenic injury (BSI) is a feared complication, particularly in the outpatient setting. Significant healthcare resources, including angiography (ANGIO) and follow-up computed tomography (CT), are utilized in an effort to prevent DS. However, no prospective, long-term data exists to determine the actual risk of DS. Understanding the actual risk of DS could help limit resource utilization and radiation exposure. The purpose of this multi-institutional trial was to ascertain the 180-day risk of DR after 24 hours of successful NON-OP management of BSI and to determine factors related to DS.

Methods: 11 Level I trauma centers participated in this prospective study. Adults ≥ 18 with BSI successfully managed NON-OP for 24 hours were eligible. Patients were followed for 180-days. Demographic, physiologic, radiographic and injury related information was obtained. Any spleen related interventions were recorded. Univariate and bivariate analyses were used to determine factors associated with DS.

Results: 383 patients were enrolled. 30, 90, and 180-day follow-up were 95%, 88%, and 87% respectively. 12 patients (3.1%) suffered in-hospital splenectomy between 24 hours and 9 days post-injury. 4 patients died (none were spleen related) and 1 withdrew leaving 366 patients discharged with a spleen. 1 (0.27%) required readmission for splenectomy on post-injury day 12. No Grade I injuries suffered DS. High-grade injuries tended to have earlier DS and lower grade injuries had later DS (Figure). Overall splenectomy rate after NON-OP management for 24 hours was 1.5 per 1000 patient-days. Only extravasation from the spleen (ADMIT-BLUSH) at time of admission was associated with DS (OR 3.6; 95% CI 1.4, 12.4). Of patients with ADMIT-BLUSH (n=49), 17 (34.7%) did not have ANGIO with embolization (EMBO) and 2 of those (11.8%) underwent splenectomy; 32 (65.3%) underwent ANGIO with EMBO and 2 of those (6.3%) required splenectomy.



Conclusions: The need for splenectomy after 24 hours of successful NON-OP management is rare. After the initial 24 hours, no additional interventions are warranted for patients with Grade I injuries. For grade II – V BSI close observation is indicated for 10 – 14 days as this is the time of greatest risk of DS. Extravasation of contrast from the spleen at the time of admission is a strong predictor of DS and may be an area where aggressive use of ANGIO and EMBO is warranted and should be the focus of future prospective studies. Use of CT to follow splenic healing after discharge is not indicated in patients without symptoms.

NOTES

Session: I: Plenary Papers 1-8
Paper 2: 1:20-1:40 PM

A CONTROLLED RESUSCITATION STRATEGY IS FEASIBLE AND SAFE IN HYPOTENSIVE TRAUMA PATIENTS: RESULTS OF A PROSPECTIVE RANDOMIZED PILOT TRIAL

Martin A. Schreiber* MD, Samuel A. Tisherman* MD, Eric N. Meier MS, Jeffrey Kerby* MD,Ph.D., Craig Newgard MD, Karen Brasel* MD, Debra Egan MPH, MSc, William Witham MD, Carolyn Williams RN, BSN, Jeff Beeson DO, FACEP, Delores Kannas RN, MS, Susanne May Ph.D., Barbara McKnight Ph.D., David B. Hoyt* MD, And The ROC Investigators, Oregon Health & Science University

Invited Discussant: Thomas Scalea, MD

Introduction: Optimal resuscitation of hypotensive trauma patients has not been defined. A prior prospective randomized trial performed in hypotensive patients with penetrating torso wounds showed a survival advantage in patients who received delayed resuscitation. The current trial was performed to determine the feasibility and safety of controlled resuscitation (CR) vs standard resuscitation (SR) in hypotensive patients with blunt and penetrating trauma.
Methods: Patients were enrolled and randomized in the prehospital setting using exception from informed consent. Nine-teen EMS systems and 10 hospitals in 6 regions of the Resuscitation Outcome Consortium participated in the study. Eligible patients suffered blunt or penetrating trauma, were ≥ 15 years old or > 50kg if their age was unknown, had a prehospital systolic blood pressure (SBP) ≤ 90mmHg and were transported to a participating trauma center. EMS units carried blinded boxes containing either two 250cc bags of normal saline (NS) and a 500cc weight (CR) or 1 liter of NS (SR). Patients in the CR group received fluid only if they had no radial pulse or a SBP ≤ 70mmHg. SR group patients received 2 liters initially and additional fluid as needed to maintain a SBP ≥ 110mmHg. Patients could receive blood products as indicated. The crystalloid protocol was maintained until either hemorrhage was controlled or for 2 hours after hospital arrival.
Results: Between March 2012 and April 2013, 192 patients were randomized (97 to CR and 95 to SR). The CR and SR groups were similar at baseline in terms of mean age 42 (20)* vs 42 (19) years, median ISS 9 (2-19) vs 9 (2-24), median initial SBP 82 (72-92) vs 84 (74-94) mmHg and rate of penetrating injury 33% vs 35%, respectively. Average crystalloid resuscitation given during the study period was 1.0 (1.5) liter in the CR group and 2.0 (1.4) liters in the SR group, a difference of 1.0 liter (95% CI: 0.6 to 1.4). Admission mean SBP was 99 (32) mmHg in the CR group and 105 (34) mmHg in the SR group. ICU free days, ventilator free days, renal injury and renal failure did not differ between groups at 28 days. At 24 hours after admission, there were 5 deaths (5%) in the CR group and 14 (15%) in the SR group (adjusted odds ratio 0.39 (95% CI: 0.12, 1.26). Among patients with blunt trauma, there was a significant difference in 24 hour mortality, with rates of 3% (CR) and 18% (SR) and an adjusted OR of 0.17 (0.03, 0.92). There was no difference among patients with penetrating trauma: 9% vs 9%, adjusted OR 1.93 (0.19, 19.17).
Conclusion: Conclusions: Controlled resuscitation is achievable in prehospital and hospital settings and may offer an early survival advantage. A large-scale, Phase III trial to examine its effects on survival and other clinical outcomes is warranted.

*Means are given with their standard deviations and medians are given with their interquartile ranges.

NOTES

Session: I: Plenary Papers 1-8
Paper 3: 1:40-2:00 PM

GETTING IT RIGHT: ADHERENCE TO AN ESTABLISHED DIAGNOSTIC THRESHOLD FOR VENTILATOR-ASSOCIATED PNEUMONIA CONTRIBUTES TO LOW FALSE-NEGATIVE RATES IN TRAUMA PATIENTS

John P. Sharpe MD, MS, Louis J. Magnotti* MD, Jordan A. Weinberg* MD, Joseph M. Swanson MD, Thomas J. Schroeppel* MD, Timothy C. Fabian* MD, Martin A. Croce* MD, University of Tennessee Health Science Center - Memphis

Invited Discussant: Christopher Michetti, MD

Introduction: For more than two decades, diagnosis of ventilator-associated pneumonia (VAP) in our institution has followed an established diagnostic threshold (DT) of $\geq 10^5$ CFU/mL on bronchoalveolar lavage (BAL) based on our previous experience (PS). Because mortality from VAP is related to treatment delay, some have advocated a lower DT. The purpose of the current study (CS) was to evaluate the impact of adherence to this DT for VAP on false-negative (FN) rates and mortality in trauma patients.

Methods: Consecutive patients over 9 years with VAP (defined as $\geq 10^5$ CFU/mL in the BAL effluent) subsequent to the previous study (PS) were identified. Data regarding timing and frequency of BAL and the colony counts of each organism identified were recorded. A false negative BAL was defined as any patient who had $<10^5$ CFU/mL and developed VAP with the same organism up to 7 days after the previous culture. The CS was then compared to the PS.

Results: Over 9 years, 1679 patients underwent 3202 BALs. Of these, 79% were male, 88% followed blunt injury, and mean age and Injury Severity Score (ISS) were 44 years and 31, respectively. Overall there were 73 FN BALs (2.3%) in the CS compared to 3% in the PS (p=0.09). No FN BALs were identified in patients with $<10^4$ organisms on BAL. In those patients with 10^4 organisms, the FN rate was reduced (7.5% vs 11%, p=0.045) and mortality unchanged (5.4% vs 8.3%, p=0.36) in the CS compared to the PS. Use of $\geq 10^5$ resulted in a cumulative reduction in antibiotic charges of $1.57 million.

Conclusion: Continued adherence to the diagnostic threshold of $\geq 10^5$ for quantitative BAL in trauma patients has maintained a low incidence of FN BALs and reduced patient charges without impacting mortality. The purported benefit of a lower threshold (reducing mortality by shortening treatment delays) is not supported. In addition, the potential sequelae of increased resistant organisms, antibiotic-related complications and costs associated with prolonged unnecessary antibiotic exposure are minimized.

NOTES

Session: I: Plenary Papers 1-8
Paper 4: 2:00-2:20 PM

REDUCING SECONDARY BRAIN INJURY IN TRAUMA PATIENTS: THE EFFECT OF REMOTE ISCHEMIC CONDITIONING

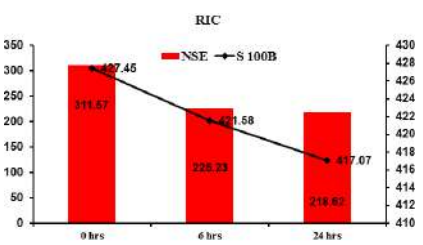
Bellal Joseph* MD, Viraj Pandit MD, Ammar Hashmi MD, Narong Kulvatunyou* MD, Terence O'Keeffe* MD, MBChB, Bardiya Zangbar MD, Andrew Tang MD, Donald J. Green* MD, Lynn Gries MD, Randall S. Friese* MD, Peter Rhee* MD,MPH, University of Arizona - Tucson

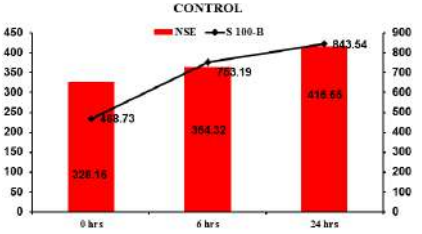
Invited Discussant: Deborah Stein, MD, MPH

Introduction: Management of traumatic brain injury is primarily focused on preventing secondary brain injury. Remote ischemic conditioning (RIC) is an established treatment modality that has been shown to improve patient outcomes secondary to inflammatory insults. The aim of our study was to assess the effects of RIC in trauma patients with severe TBI.

Methods: This prospective consented interventional trial included all TBI patients with an intracranial hemorrhage (ICH) and a Glasgow coma scale (GCS) score of ≤ 8 on admission. In each patient, four cycles of RIC were performed on admission. Each cycle consisted of five minutes of controlled upper limb ischemia followed by five minutes of reperfusion. Serum bio-markers of acute brain injury, S-100B and Neuron specific enolase (NSE) (sensitivity=80%, specificity=73%) were measured at 0 hrs, 6 hrs, and 24-hours. RIC was performed after collection of 0hr serum marker. The established elevated serum levels of NSE and S100B in TBI patients were reconfirmed using patients without RIC (the baseline value of these markers is zero in patients without neuronal injury). Outcome measure was reduction in the level of serum biomarkers post RIC.

Results: A total of 20 patients were enrolled. The mean age was 46.15±18.64 years, median GCS was 8[3-8] and median head abbreviated injury (h-AIS) scale score was 3[3-5]. The mean 0hr S-100B value was 427.45±201.15 pg/ml while mean NSE value was 311.57±146.81 pg/ml. There was a significant reduction in the levels of S-100B (p=0.033) and NSE (p=0.031) at 6hrs and 24hrs in comparison to the 0hr level **(Figure 1)**. The overall mortality was 18.18% (n=4).





Conclusion: This study highlights the novel therapeutic role of RIC for preventing secondary brain insults in TBI patients. There was a significant reduction in the biomarkers of acute brain injury post RIC. Further research assessing the impact of RIC on patient outcomes is warranted.

NOTES

Session: I: Plenary Papers 1-8
Paper 5: 2:20-2:40 PM

REGIONAL COLLABORATIVE QUALITY IMPROVEMENT FOR TRAUMA REDUCES COMPLICATIONS AND COSTS

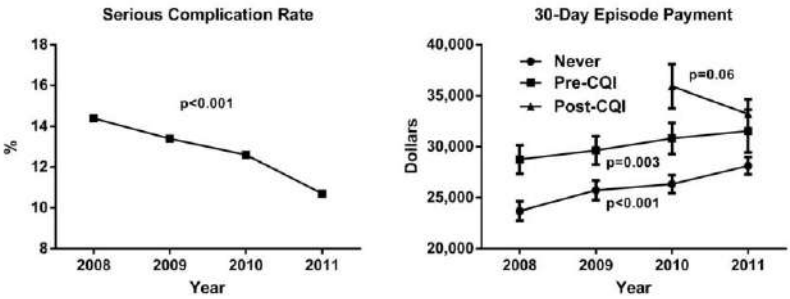
Mark R. Hemmila* MD, Peter C. Jenkins MD, Wendy L. Wahl* MD, Wayne E. Vander Kolk MD, Judy N. Mikhail MSN, Nancy J. Birkmeyer Ph.D., University of Michigan

Invited Discussant: David Hoyt, MD

Introduction: While evidence suggests that quality improvement to reduce complications for trauma patients should decrease costs, studies have not addressed this question directly. In our state, trauma centers and a private payer have created a regional collaborative quality improvement (CQI) program. This CQI program began as a pilot in 2008 and was expanded to a formal program in 2010. We examined the relationship between outcomes and expenditures for trauma patients treated in collaborative participant and non-participant hospitals.

Methods: Payer claims and collaborative registry data (2008-2011) were analyzed for 30-day episode payments and complications in patients admitted with trauma diagnoses. Patients were categorized as treated in hospitals that had different CQI status: 1) never participated (Never), 2) collaborative participant, but patient treated prior to CQI initiation (Pre-CQI), or 3) active collaborative participant (Post-CQI). ICD-9 codes were cross-walked to AIS 2005 codes. Episode payment data were risk adjusted (age, gender, comorbidities, type/severity of injury, and year of treatment) and price standardized. Outcome data was adjusted for risk and reliability. A serious complication consisted of one or more of the following occurrences: abdominal compartment syndrome, acute lung injury/ARDS, acute kidney injury, cardiac arrest with CPR, decubitus ulcer, DVT, enterocutaneous fistula, extremity compartment syndrome, mortality, myocardial infarction, pneumonia, pulmonary embolism, severe sepsis, stroke/CVA, unplanned intubation, or unplanned return to OR.

Results: The risk-adjusted rate of serious complications declined from 15.6% to 12.2% ($p<0.001$) in participating hospitals (Post-CQI; n=23). Average episode payments decreased by $2,697 ($35,927 to $33,230, p=0.06) among patients (n=4,084) treated in Post-CQI centers, whereas patients treated at Pre-CQI (n=9,931) or Never (n=28,777) institutions had a significant year-to-year increase in payments (Figure). A savings of $11 million in total episode payments from 2010 to 2011 was achieved for Post-CQI treated patients.



Conclusion: This study confirms our hypothesis that participation in a regional collaborative quality improvement program improves outcomes and reduces costs for trauma patients.

NOTES

Session: I: Plenary Papers 1-8
Paper 6: 2:40-3:00 PM

MITOCHONDRIAL DAMPS FROM FRACTURES SUPPRESS PULMONARY IMMUNE RESPONSES VIA FORMYL PEPTIDE RECEPTORS 1 AND 2

Haipeng Li MD, Kiyoshi Itagaki Ph.D., Nicola Sandler MD, David Gallo BS, Elzbieta Kaczmarek Ph.D., Amanda Galenkamp BS, Leo Otterbein Ph.D., Carl J. Hauser* MD, Beth Israel Deaconess Medical Center

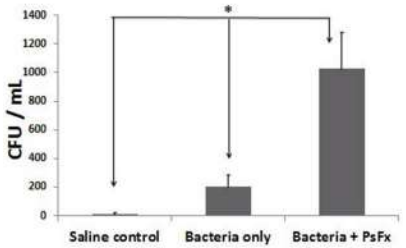
Invited Discussant: Ronald Maier, MD

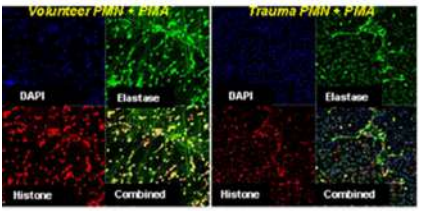
Introduction: Pneumonia (PNA) is a common cause of morbidity & mortality after trauma but no known biologic mechanisms link PNA with tissue injury. Neutrophils (PMN) are the dominant innate immune effector cells that control bacterial growth in the lung. But to clear bacteria from the lung, circulating PMN must get to the alveoli using chemotaxis (CTX) to G-protein coupled chemoattractants and then must kill bacteria in Neutrophil Extracellular Traps (NETs). We previously found that clinical trauma suppresses PMN CTX and predicted trauma might suppress other antimicrobial functions. Mitochondrial damage molecules (DAMPs) from femur reamings alter PMN phenotype. We now hypothesize DAMPs from fracture injury caould predispose to PNA by decreasing PMN traffic to the lung and suppressing the antimicrobial function of PMN once they get there.

Methods: **Expt 1:** Rats (n=10 /condition) were divided into 4 groups; a) naïve controls; b) pulmonary contusion (PC induced by percussion); c) pseudo-fracture (PsFx: injection of the thigh with crushed bone supernatant containing DAMPs), and d) PC plus PsFx. **Expt 2:** Rats were put in three groups: a) controls - saline injection in the thigh; b) intratracheal (*i.t.*) injection of $5x10^4$ *S. aureus*; c) PsFx followed by *S. aureus i.t.* Bronchoalveolar lavage (BAL) was performed 16h after treatment. BAL bacteria and PMN were counted. **Expt 3:** Human clinical trauma PMN were assayed for CTX (in transwells) and NETosis (by fluorescent confocal microscopy).

Results:**: Expt 1:** PC increased and PsFx markedly decreased PMN traffic to the lung ($P<0.01$, ANOVA). **Expt 2:** PsFx markedly suppressed lung bacterial clearance ($P<0.01$, Fig. **Lt**) with BAL bacteria increasing as PMN decreased. DAMPs from human bone activated PMN $[Ca^{2+}]_i$ flux. This response was inhibited by mAbs to formyl peptide receptors (FPR) 1&2. **Expt 3:** Clinical trauma strongly suppressed PMN CTX to IL-8 and LTB4 (not shown) as well as NETosis responses to PMA (Fig. **Rt**).

Conclusion: Fracture DAMPs act via FPR1/2 to decrease PMN traffic to the lung and to decrease their ability to clear bacteria. PNA after injury reflects DAMP mediated decreased PMN traffic to the lung and decreased antimicrobial function once there **.**





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Session: I: Plenary Papers 1-8
Paper 7: 3:00-3:20 PM

THE AAST PROSPECTIVE OBSERVATIONAL VASCULAR INJURY TREATMENT (PROOVIT) REGISTRY: MULTICENTER DATA ON MODERN VASCULAR INJURY DIAGNOSIS, MANAGEMENT AND OUTCOMES

Joseph J. DuBose* MD, Stephanie A. Savage* MD, Timothy Fabian* MD, Jay Menaker MD, Thomas Scalea* MD, John Holcomb* MD, Nathaniel Poulin MD, Konstantinos Chouliaras MD, Kenji Inaba* MD, Xian Luo-Owen MD,Ph.D., Thomas A. O'Callaghan* MD, Andreas Larentzakis MD, George Velmahos* MD, George Dulabon MD, Todd E. Rasmussen* MD, University Of Texas Health Science Center - Houston

Invited Discussant: Howard Champion, FRCS

Introduction: There is a need for a prospective registry designed to capture trauma-specific, in-hospital and long-term outcomes related to vascular injury.

Methods: The AAST PROspective Vascular Injury Treatment (PROOVIT) registry was used to collect demographic, diagnostic, treatment and outcome data on vascular injuries.

Results: 542 injuries from 14 centers (13 ACS Level I, 1 ACS Level II) have been captured since February 2013. The majority of patients are male (70.5%); ISS ≥ 15 among 32.1%. Penetrating mechanisms account for 36.5%. Arterial injuries to vessels of the head/neck (26.7%), thorax (10.4%), abdomen/pelvis (7.8%), upper extremity (18.4%) and lower extremity (26.0%) were identified, along with 98 major venous injuries. Hard signs of vascular injury, including hypotension (SBP < 90, 11.8%), were noted in 28.6%. Pre-hospital tourniquet use for extremity injuries occurred in 20.2% (47/233). Diagnostic modalities included exploration (28.8%), CTA (38.9%), duplex US (3.1%) and angiography (10.7%). Arterial injuries included transection (24.3%), occlusion (17.3%), partial transection/flow limiting defect (24.5%), pseudoaneurysm (9.0%) and other injuries including intimal defects (22.7%). Non-op management was undertaken in 276 (50.9%), with failure in 4.0%. Definitive endovascular and open repair were utilized in 40 (7.4%) and 126 (23.2%) patients. Damage control maneuvers were used in 57 (10.5%), including ligation (31, 5.7%) and shunting (14, 2.6%). Re-intervention of initial definitive repair was required in 42 (7.7%). Amputation was performed in 7.7% of extremity vascular injuries and overall hospital mortality was 12.7%. Follow-up ranging from 1-7 months is available for 48 patients via a variety of modalities, with re-intervention required in only 1 patient.

Conclusions: The PROOVIT registry provides a useful contemporary picture of the management of modern vascular injury. This resource promises to provide needed information required to answer questions about optimal diagnosis and management of these patients – including much needed long-term outcome data.

NOTES

Session: I: Plenary Papers 1-8
Paper 8: 3:20-3:40 PM

INTRACRANIAL PRESSURE MONITORING AND INPATIENT MORTALITY IN SEVERE TRAUMATIC BRAIN INJURY: A PROPENSITY-SCORE MATCHED ANALYSIS

Aaron J. Dawes MD, Greg D. Sacks MD,MPH, H. Gill Cryer* MD,Ph.D., Christy Preston RN, Deidre Gorospe RN, Matt Garrett MD, David McArthur MPH,Ph.D., Melinda Maggard Gibbons MD, MSHS, Marcia M. Russell MD, Clifford Ko MD, MS, MSHS
University of California, Los Angeles
Invited Discussant: Alex Valadka, MD

Background: Despite being recommended as standard of care by the Brain Trauma Foundation, the benefit of intracranial pressure (ICP) monitoring in severe traumatic brain injury (TBI) remains controversial. Moreover, the effect of ICP monitoring on mortality may be overstated if more severely injured patients die without undergoing monitor placement. Our study aim was to examine the impact of ICP monitor placement on in-patient mortality within a regional trauma system, and to attempt to correct for selection bias through propensity-score matching.

Methods: Data were prospectively collected from all severe TBI cases presenting to 14 trauma centers within a single trauma system during the two-year study period (2009-2010). Inclusion criteria were: blunt injury, Glasgow Coma Scale (GCS) ≤ 8 in the emergency room, and at least one intracranial injury on head CT. Patients who died in the emergency department or underwent emergency craniotomy were excluded. A multivariate logistic regression model was used to predict inpatient mortality after controlling for demographics, trauma center, severity of injury, comorbidities, and TBI-specific variables (GCS, pupil reactivity, initial INR, and 10 specific head CT findings). We then calculated both unadjusted and risk-adjusted odds ratios for mortality based on ICP monitor placement. To examine for differences between the treatment and non-treatment groups, we developed a logistic model to predict ICP monitor placement using the variables in our initial multivariate model predicting inpatient mortality. Using observed associations between covariates and the likelihood of undergoing treatment, we then developed a propensity-score matched model to explore the underlying effect of ICP monitoring on in-hospital mortality.

Results: During the two-year study period, 635 patients met inclusion criteria. In-patient mortality was 39.2%, and 38.0% of patients had an ICP monitor placed within the first 72 hours. Both unadjusted and risk-adjusted odds of mortality were significantly lower in the ICP monitoring group (Table). Predicted mortality rates were significantly higher in the non-treated group (42.8% vs. 33.7%, p=0.006), suggesting the possibility of selection bias in ICP monitor placement. Based on our treatment model, ICP monitor placement was positively associated with injury severity score, pupil reactivity, and subarachnoid hemorrhage, and negatively associated with age, GCS, obesity, elevated INR, and compression of basal cisterns on head CT. After adjusting for treatment selection by propensity score matching, ICP monitor placement remained significantly associated with mortality with recipients displaying a 13% reduction in the odds of death compared to those without an ICP monitor (Table).

	Odds of inpatient mortality for ICP monitor placement (vs. not)	95% Confidence Interval
Unadjusted data	0.49	0.35-0.69
Risk-adjusted model	0.37	0.17-0.77
Prospensity-score matched	0.87	0.77-0.98

Conclusions: ICP monitor placement occurred in only 38% of eligible patients, but was significantly associated with decreased mortality after adjusting for baseline risk profile and the propensity to undergo monitor placement. The decreased magnitude of the effect between logistic and propensity-score matched models indicates that significant selection bias exists in the placement of ICP monitors. As individual benefits of ICP monitoring may vary, future efforts should center on determining the appropriate use of invasive monitoring techniques.

NOTES

WEDNESDAY, SEPTEMBER 10, 2014, 3:40 PM

SESSION II: MASTER SURGEON LECTURE I

LOCATION: GRAND BALLROOM SALONS G-L

"Managing the TBI Patient:
Too Much? Too Little? When Is It Just Right?"

Alex B. Valadka, M.D.

Chairman and Chief Executive Officer
of the Seton Brain and Spine Institute
Adjunct Professor of Psychology
University of Texas at Austin
Austin, TX

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WEDNESDAY, SEPTEMBER 10, 2014, 4:10 PM - 5:25 PM

SESSION III:
PANEL I: CHALLENGING CASES

LOCATION: GRAND BALLROOM SALONS G-L

MODERATOR: THOMAS M. SCALEA, M.D.

Roxie Albrecht, M.D.

Jose Diaz, M.D.

Ari Leppaniemi, M.D.

Angela Neville, M.D.

Matthew Rosengart, M.D., M.P.H

SESSION IV:

POSTER SESSION/OPENING RECEPTION

WEDNESDAY, SEPTEMBER 10, 2014, 5:30 PM – 7:30 PM

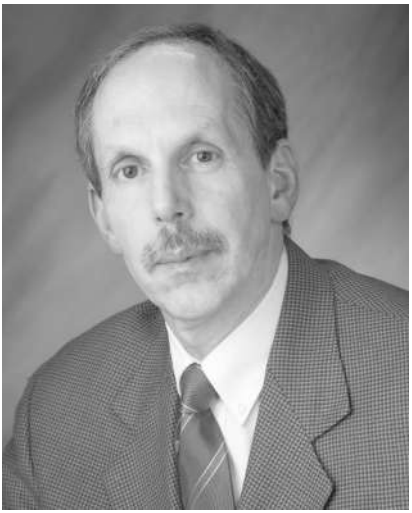
LOCATION: FRANKLIN B

Poster #	Professors	Category
1-10	Luke Leenen, M.D. Carlos Brown, M.D.	Abdominal Trauma/Burns/Soft Tissue
11-20	Charles Lucas, M.D. Eric Toschlog, M.D.	Acute Care Surgery
21-30	Lewis Kaplan, M.D. Andrew Kerwin, M.D.	Critical Care
31-40	Vicente Gracias, M.D. Ajai Malhotra, M.D.	Neurological Trauma
41-50	Adil Haider, M.D. Zsolt Balogh, M.D.	Outcomes/Guidelines I
51-60	A. Peter Ekeh, M.D. Randall Friese, M.D.	Outcomes/Guidelines II
61-70	Hans-Christoph Pape, M.D. Joseph Rappold, M.D.	Outcomes/Guidelines/Extremity Trauma/Vascular Trauma
71-80	Karen Brasel, M.D., M.P.H. Kristan Staudenmayer, M.D.	Trauma Education/Trauma Systems
81-90	R. Todd Maxson, M.D. Darren Malinoski, M.D.	Pediatric Trauma/ Shock/Transfusions
91-100	R. Lawrence Reed, M.D. Krista Kaups, M.D.	Socioeconomics/Ethics/Trauma System
101-110	Ronald Stewart, M.D. Christopher Cribari, M.D.	Trauma Systems
111-120	Carnell Cooper, M.D. Glen Tinkoff, M.D.	Trauma Prevention & Epidemiology
121-130	Matthew Wall, Jr., M.D. James Tyburski, M.D.	Thoracic Trauma
131-140	Bryan Cotton, M.D. Juan Carlos Puyana, M.D.	Shock/Transfusions

THURSDAY, SEPTEMBER 11, 2014, 7:30 AM – 8:00 AM

SESSION V: MASTER SURGEON LECTURE II

LOCATION: GRAND BALLROOM SALONS G-L



"Acute Cholecystitis:
When to Operate and How to Do it Safely"

Andrew B. Peitzman, M.D.

Distinguished Professor of Surgery
Mark M. Ravitch Professor and Vice-Chairman
Department of Surgery
UPMC Vice-President for Trauma and Surgical Services
University of Pittsburgh School of Medicine
Pittsburgh, PA

SESSION VI:

PLENARY PAPERS #9 - #12

THURSDAY, SEPTEMBER 11, 2014, 8:00 AM – 9:20 AM

GRAND BALLROOM SALONS G-L

MODERATOR: GRACE ROZYCKI, M.D., M.B.A.

RECORDER: RAUL COIMBRA, M.D., Ph.D.

Session: VI: Plenary Papers 9-12
Paper 9: 8:00-8:20 AM

RESUSCITATIVE ENDOVASCULAR BALLOON OCCLUSION OF THE AORTA (REBOA) IS A FEASIBLE ALTERNATIVE TO RESUSCITATIVE THORACOTOMY IN TRAUMA PATIENTS WITH NON-COMPRESSIBLE TRUNCAL HEMORRHAGE AND PROFOUND HEMORRHAGIC SHOCK

Laura J. Moore* MD, Megan Brenner MD, Rosemary A. Kozar* MD,Ph.D., Jason Pasley DO, Charles Wade* Ph.D., Thomas Scalea* MD, John B. Holcomb* MD, University of Texas Health Science Center-Houston

Invited Discussant: Timothy Fabian, MD

Introduction: Hemorrhage remains the leading cause of death in trauma patients. Proximal aortic occlusion, usually performed by direct aortic cross-clamping via thoracotomy, can provide temporary hemodynamic stability, permitting definitive injury repair. Resuscitative endovascular balloon occlusion of the aorta (REBOA), utilizes a minimally-invasive, trans-femoral balloon catheter which is rapidly inserted retrograde and inflated for aortic occlusion, may control inflow and allow time for hemostasis. We compared resuscitative thoracotomy with aortic cross-clamping (RT) to REBOA in trauma patients in profound hemorrhagic shock.

Methods: Trauma registry data was utilized to compare all patients undergoing RT or REBOA over an 18 month period from two Level one trauma centers. Groups were compared using a t-test, p value <0.05 considered significant.

Results: There was no difference between RT (n=72) and REBOA groups (n=24) in terms of demographics, mechanism of injury, injury severity score (40.6 vs. 31.4, p=0.17), admission base deficit (12.9 vs. 11, p=0.31), or initial blood pressure (70.9 vs. 55.8, p=0.15). REBOA had fewer early deaths (see table) and improved overall survival as compared to RT (37.5% vs. 9.7%, p=0.003).

	Resuscitative Thoracotomy (RT) Deaths (n=65)	**REBOA Deaths (n=15)**	**p value**
Died in ED (%)	69.2%	26.7%	0.002
Died in OR (%)	9.3%	20%	0.234
Died in ICU (%)	21.5%	53.3%	0.013

Conclusions: REBOA is a feasible and controls non-compressible truncal hemorrhage in trauma patients in profound shock. Patients undergoing REBOA have improved overall survival and fewer early deaths as compared to patients undergoing RT.

NOTES

Session: VI: Plenary Papers 9-12
Paper 10: 8:20-8:40 AM

Evaluation of the Safety and Feasibility of Resuscitative Endovascular Balloon Occlusion of the Aorta in Japan

Nobuyuki Saito MD, Takanori Yagi MD, Hisashi Matsumoto MD,Ph.D., Kunihiro Mashiko* MD,Ph.D., Chiba Hokusoh Hospital, Nippon Medical School

Invited Discussant: Matthew Wall, Jr, MD

Introduction: Resuscitative endovascular balloon occlusion of the aorta (REBOA) is ultimately an invasive procedure for managing severe subphrenic torso injury, but it is less invasive than resuscitative open aortic cross clamping and therefore its clinical application is expected. We retrospectively evaluated the safety and clinical feasibility of REBOA sets (IABO:intra-aortic occlusion balloon; MERA, Tokyo, Japan) using the Seldinger technique.
Methods: Of 5230 trauma patients admitted to our Japanese trauma center between 2007 and 2013, 52 underwent REBOA for first-line resuscitation and 24 who underwent REBOA independently was included in the analysis. The indication for REBOA was a pelvic ring fracture (PRF) or hemoperitoneum with impending cardiac arrest. Emergency hemostasis was performed during REBOA in all patients.
Results: All 24 patients suffered blunt mechanism trauma and median age was 59.5 (interquartile range: 41.5–71) years, median injury severity score was 47.5 (37–52), and 30-day survival rate was 29.2% (n=7). The probability survival rate of this group of all patients was less than 0.5. Indications for REBOA were hemoperitoneum in 15 cases, PRF in 15 cases. In 10 cases of death, the balloon could not be deflated in 5 cases. In 19 cases in which the balloon could be deflated, median duration of aortic occlusion was shorter in surviving cases than in cases of death (21 min vs 35 min, P=0.05). The mean systolic blood pressure was significantly increased by REBOA (53.1±21 mmHg to 98.0±26.6 mmHg, P<0.01). The balloon was inserted within around 20 minutes from arrival in the emergency room. Percutaneous puncture was employed in 23 (95.8%) insertion techniques; surgical cut-down was performed in only 1 case. There were 3 cases of complications (12.5%), 1 of vascular injury and 2 of limb ischemia, in which lower limb amputation was needed in all cases. Acute kidney injury developed in all 3 cases, but the failure was not persistent.
Conclusion: REBOA appears to be feasible for trauma resuscitation and should improve survivorship. There were serious complications of limb ischemia, so it is necessary to improve the safety of REBOA.

NOTES

Session: VI: Plenary Papers 9-12
Paper 11: 8:40-9:00 AM

THE EARLY EVOLVING SEX HORMONE ENVIRONMENT IS ASSOCIATED WITH SIGNIFICANT CLINICAL OUTCOME AND INFLAMMATORY RESPONSE DIFFERENCES POST-INJURY

Samuel J. Zolin BS, Yoram Vodovotz Ph.D., Raquel M. Forsythe* MD, Rosengart Matthew* MD,MPH, Rami Namas MD, Andrew P. Peitzman* MD, Timothy R. Billiar* MD, Jason L. Sperry* MD,MPH, University of Pittsburgh

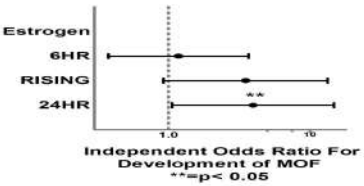
Invited Discussant: Reuven Rabinovici, MD

OBJECTIVE(S): Clinical research characterizing the mechanisms responsible for gender based outcome differences post-injury remain conflicting. Currently lacking is an understanding of the early sex-hormone milieu of the injured patient (< 6 hour from injury) and the effects these early hormone differences have on clinical outcomes and the innate immune response following injury. We hypothesized that the early sex-hormone environment would be associated with significant differences in clinical outcomes and the early innate immune response.

METHODS: A prospective cohort study was performed over an 18 month period. Blunt injured patients requiring ICU admission were enrolled while patients with isolated brain and spinal cord injuries were excluded. Blood samples were collected within 6 hours and at 24 hours post-injury and were analyzed for total testosterone (TT) and estradiol (EST) concentrations. Hormone variables were dichotomized into HIGH and LOW groups and into those patients with increasing hormone levels between 6hr and 24hr measurements (RISING). Outcomes of interest included Multiple Organ Failure (MOF, Marshall MODscore > 5), nosocomial infection (NI), mortality and serial cytokine/mediator measurements. Multivariate logistic regression was utilized to determine the independent risks associated with early sex hormone measurements after controlling for differences in demographics, injury characteristics, shock severity and resuscitation requirements.

RESULTS: In 288 prospectively enrolled patients, 68% were male with a median ISS of 16 [IQR 10,21]. Prevalence of MOF, NI and mortality was 12.5% , 29.9% and 4.1%, respectively. After controlling for important confounders, HIGH TT levels at 6hrs were associated with elevated IL-6 levels and cytokine/mediator measurements (22 out of 26 measured). RISING TT levels were significantly associated with over a 5-fold and 2-fold higher independent risk of MOF and NI, respectively (OR 5.2, p=0.02, 95%CI 1.2-22.3, OR 2.1, p= 0.03, 95%CI 1.02-4.2). At 24hrs HIGH TT was no longer associated with poor outcome while HIGH EST was significantly associated with almost a 4-fold higher independent risk of MOF (OR 3.9, p=0.04, 95% CI 1.05-13).

CONCLUSIONS: Early elevations and increasing testosterone levels over the initial 24hrs are associated with an exaggerated inflammatory response and a significantly greater risk of MOF and NI. HIGH Estrogen levels at 24hrs are independently associated with a greater risk of MOF. Inflammation is known to result in the peripheral conversion of androgens to estrogens. The current analysis suggests an early evolving testosterone to estrogen hormonal environment is associated with a significantly higher independent risk of poor outcome following traumatic injury.



NOTES

Session: VI: Plenary Papers 9-12
Paper 12: 9:00-9:20 AM

SYSTOLIC BLOOD PRESSURE CRITERIA IN THE NATIONAL TRAUMA TRAIGE PROTOCOL FOR GERIATRIC TRAUMA: 110 IS THE NEW 90

Joshua B. Brown MD, Mark L. Gestring* MD, Raquel M. Forsythe* MD, Nicole A. Stassen* MD, Timothy R. Billiar* MD, Andrew B. Peitzman* MD, Jason L. Sperry* MD,MPH, University of Pittsburgh

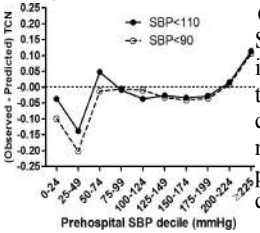
Invited Discussant: Frederick Luchette, MD

Introduction: Under-triage following injury continues to be a concern in the geriatric trauma population. The latest revision of the national trauma triage protocol (NTTP) recognized that a systolic blood pressure (SBP) <110mmHg may represent shock in those over 65 years of age, but this change has not been studied and the current triage trigger remains <90 for all adult age groups. The study objective was to evaluate performance of the NTTP when the current SBP<90 is replaced with <110 for triage.

Methods: Subjects undergoing scene transport in the NTDB (2010-12) were included. NTTP physiologic (Step1) and anatomic (Step2) criteria were determined using prehospital vital signs and ICD-9 diagnosis codes. Imputation was used for missing data. Trauma center need (TCN) was defined as ISS>15, ICU admit, urgent OR, or ED death. Geriatric (age>65) and adult (age 16-65) patients were compared. Test characteristics were calculated to predict TCN. The area under the curve (AUC) was compared between SBP<110 and SBP<90. Logistic regression was used to generate a predicted probability of TCN using SBP<110 or SBP<90, controlling for other NTTP criteria. Observed vs predicted TCN was compared to evaluate calibration. Optimal SBP cutoffs for TCN and mortality were identified as the SBP cutoff maximizing sensitivity and specificity. Logistic regression was also used to determine the effect of geriatric vs adult age group on mortality in patients who would be triaged positive with this change applied (SBP 90-109mmHg).

Results: 1,555,944 subjects were included. Table 1 shows test characteristics for SBP<110 and SBP<90, individually and within the NTTP for the geriatric and adult cohorts. As individual criteria, the AUC was higher for SBP<110 both in the geriatric cohort (p<0.01) and adult cohort (p<0.01). Within the first 2 steps of the NTTP, AUC was still higher using SBP<110 compared to SBP<90 in the geriatric cohort, but slightly lower in the adult cohort (p<0.01). The difference between observed and predicted TCN across SBP deciles for the model using SBP<110 compared to SBP<90 was lower in the geriatric cohort (Figure) than in the adult cohort, indicating better calibration of SBP<110 in geriatric patients than adult patients. The optimal cutoff SBP was 122 for TCN and 118 for mortality in the geriatric cohort, while it was 118 for TCN and 106 for mortality in the adult cohort. In patients with SBP 90-109, geriatric age group was associated with a nearly 10 fold increase in the odds of mortality (OR 9.7; 95%CI 8.7-10.8, p<0.01).

	Sensitivity	Specificity	PPV	NPV	AUC
Geriatric (age>65)					
SBP<110	13%	93%	50%	68%	0.532
SBP<90	5%	99%	66%	67%	0.519
NTTP Step 1+2 using SBP<110	44%	71%	44%	71%	0.575
NTTP Step 1+2 using SBP<90	40%	75%	45%	71%	0.574
Adult (age 16-65)					
SBP<110	23%	90%	63%	61%	0.564
SBP<90	10%	98%	79%	59%	0.539
NTTP Step 1+2 using SBP<110	67%	62%	58%	70%	0.641
NTTP Step 1+2 using SBP<90	62%	67%	59%	69%	0.646



Conclusion: SBP<110 trades specificity for sensitivity. Substituting SBP<110 has better discrimination and calibration in the geriatric cohort. Geriatric patients triaged positive under this change have a nearly 10 fold increased odds of mortality compared to adult patients. This change in SBP criteria appears merited in geriatric patients and may be valuable in all adult patients, warranting further study to consider elevating this change to a step 1 criterion in the NTTP.

NOTES

SCHOLARSHIP PRESENTATIONS

BY 2012-2013 AAST RESEARCH SCHOLARSHIP RECIPIENTS

THURSDAY, SEPTEMBER 11, 2014, 9:20 AM – 9:40 AM

GRAND BALLROOM SALONS G-L

PRESIDING: WILLIAM G. CIOFFI, M.D., AAST PRESIDENT

9:25 AM – 9:30 AM Robert David Winfield, M.D.
Washington University School of Medicine
St. Louis, MO
AAST Research & Education Foundation Award (2013-2014)
Project Title: Adipose Tissue and Post-Injury Immune Dysfunction in the Obese

9:31 AM – 9:36 AM Susan Evans, M.D.
University of North Carolina
Chapel Hill, NC
AAST Research & Education Foundation Award (2013-2014)
Project Title: Mitochondrial-Targeted Antioxidants Prevent Cell Death and Organ Dysfunction and Improve Survival Following Severe Hemorrhage

SESSION VIII:

ACUTE CARE SURGERY

PAPERS #13 - #16

THURSDAY, SEPTEMBER 11, 2014, 10:00 AM –11:20 AM

GRAND BALLROOM SALONS G-L

MODERATOR: KIMBERLY DAVIS, M.D., M.B.A.

RECORDER: JOSEPH MINEI, M.D.

Session: VIII: Papers 13-16: Acute Care Surgery
Paper 13: 10:00-10:20 AM

ARTERIOGRAPHY FOR LOWER GI BLEEDING: DOES A PRECEDING ABDOMINAL CT ANGIOGRAM IMPROVE BLEED IDENTIFICATION AND OUTCOME?

Christina L. Jacovides BS, Gregory Nadolski MD, Steven R. Allen MD, Niels D. Martin* MD, Daniel N. Holena* MD, Patrick M. Reilly* MD, Scott O. Trerotola MD, Lewis J. Kaplan* MD, Jose Pascual* MD,Ph.D., FRCSC University of Pennsylvania

Invited Discussant: Leslie Kobayashi, MD

Introduction: The substantial morbidity of lower gastrointestinal hemorrhage (LGIH) can be reduced with timely localization of the bleeding site. Recently, CT angiography (CTA) has been proposed as an adjunct in LGIH localization. We hypothesized that preceding visceral arteriography (VA) with CTA in acute LGIH would increase the rate of identification and embolization of bleeds, but worsen renal function due to a greater total contrast load.

Methods: An institutional policy was enacted in our tertiary academic center in 1/2009 effectively disallowing the use of VA in any LGIH patient without a prior positive imaging study (e.g. CTA, nuclear bleeding scan [NBS]). An Interventional Radiology database was queried to identify all subjects undergoing VA for LGIH (1/2005 to 12/2012). Exclusion criteria included upper GI bleeding and recent unrelated lower GI procedures. Demographics, imaging used (VA, NBS, CTA), arteriography parameters (fluoroscopy time, contrast volume), creatinine, hemoglobin, and interventions were abstracted. Patients were grouped by imaging performed; parametric statistics were used for intergroup comparisons.

Results: 161 patients underwent VA during the study period (78 pre- and 83 post-2009, 48% male, mean age of 70±20 years). 140 underwent imaging prior to VA (NBS: 91, CTA: 35, both: 14), while 21 underwent VA first. Obtaining VA without prior imaging resulted in less frequent identification of bleeding (62 vs. 94%, $p<0.001$) and fewer imaging studies but similar rates of embolization (28%) or surgical intervention (20%). CTA performed before VA resulted in more total intravenous contrast exposure, but a lower creatinine increase and shorter fluoroscopy time (Table). Pre-VA imaging with CTA instead of NBS resulted in fewer imaging studies, increased VA fluoroscopy time, and tended to increase bleed localization and embolization on VA.

Group	Number imaging studies (N)	Bleed found any study (%)	Bleed localized on VA (%)	Embolization at VA (%)	Total contrast (ml)	Fluoroscopy time (min)	Creatinine increase (%)
VA only	1.3±0.7	42.9	42.8	28.6	160±80	32.2±34.9	300±400
CTA before VA	2.5±0.8	91.8	40.8	36.7	230±80	26.3±16.8	170±60
P-value	***<0.0001***	***<0.0001***	1.0	0.6	***0.001***	***0.01***	***0.006***
CTA only before VA	2.1±0.3	94.3	45.7	40.0	220±80	27.8±17.2	160±50
NBS	2.5±0.8	94.5	26.4	23.1	130±70	17.8±14.8	200±300
P-value	***0.01***	1.0	***0.05***	0.07	***<0.0001***	***0.003***	0.1

Conclusion: Performing abdominal CTA prior to arteriography in the management of acute LGIH increases bleed identification while reducing fluoroscopy time. Compared to NBS, CTA prior to angiography reduces the need for repeated imaging and increases the proportion of positive angiographic studies without worsening renal function.

NOTES

SAME DAY COMBINED ERCP AND CHOLECYSTECTOMY: ACHIEVABLE AND COST EFFECTIVE

Muhammad J. Younus MD, Mohsen M. Shabahang MD, Denise Torres MD, Kenneth Widom MD, DiAnne Leonard* MD, Joseph Blansfield MD, William Strodel MD, Jeffrey L. Wild MD, Geisinger Health System

Invited Discussant: Michael Chang, MD

Introduction: It is estimated that choledocholithiasis is present in 5-20% of patients at the time of laparoscopic cholecystectomy (LC). Several European studies have found decreased length of stay (LOS) when performing LC and intra-operative endoscopic retrograde cholangio-pancreatography (ERCP) on the same day. In the US, presence of CBD stones is usually managed pre-operatively and often on a separate day than LC. Our aim was to evaluate LOS between same day versus separate day ERCP and cholecystectomy.

Methods: This is a retrospective study of patients undergoing ERCP and cholecystectomy during the same admission for the management of choledocholithiasis from 2010-2014. Group one had ERCP and cholecystectomy performed on the same day under one general anesthesia and group two had ERCP at least 1 day prior to cholecystectomy and underwent two general anesthesias. Primary outcome measured was length of stay.

Results: The study population included 245 patients. Average age in group one was 60.7 years versus 62.4 years in group two. There were 67 patients in group one and 178 patients in group two. Overall LOS for group one was 3.8 days versus 6.6 days in group two ($p < 0.0001$). There was no difference in conversion rates to open cholecystectomy between the two groups (15% within each group). 10% of group one required skilled nursing facility versus 20% in group two ($p = 0.08$).

Conclusions: Combined ERCP and cholecystectomy performed under one anesthesia requires coordination between GI, Anesthesia, and Surgery. ERCP/cholecystectomy under the same anesthesia is feasible. Same day procedures decrease hospital LOS by almost 3 days and patients have a higher probability of being discharged home. Future goals include a multidisciplinary protocol to study outcomes in larger numbers.

ERCP	**Sample Size n**	**Hospital day to surgery (days)**	**LOS (days)**
Separate day	178	3.2	6.6
Same day	67	1.3	3.8

NOTES

Session: VIII: Papers 13-16: Acute Care Surgery
Paper 15: 10:40-11:00 AM

DEFINING THE ACUTE CARE SURGERY CURRICULUM

Therese M. Duane* MD, Christopher J. Dente* MD, John J. Fildes* MD, Kimberly A. Davis* MBA,MD, Gregory J. Jurkovich* MD, J W. Meredith* MD, L D. Britt* MD,MPH, Virginia Commonwealth University

Invited Discussant: Ronald Stewart, MD

Introduction:The Acute Care Surgery (ACS) case log system was designed to track operative cases performed by ACS trainees to quantify the depth and breadth of the operative experience within the ACS training paradigm. An initial analysis revealed an experience weighted towards general surgery abdominal cases but with wide variability in exposure to the essential and desirable cases of the ACS curriculum. This follow up study was designed to better identify and define the gaps in essential and desirable (E/D) case volumes that may prompt re-evaluation of the ACS curriculum, or restructuring of the training provided.

Methods:Review of the first two years of ACS case log entry (7/11-6/13) was performed. Individual trainee logs were evaluated to determine how often they performed each case on the E/D list. Trainees described cases using CPT codes, which had been previously mapped to the E/D list. Comparisons were made between the first and second year to determine if any differences could be identified.

Results:There were a total of 29 trainees from 15 programs (year 1) and 30 trainees from 13 programs (year 2) who participated in case log entry with some overlap between the years. There were a total of 487 fellow-months of data with an average of 14.6 CPT codes per month and 175.5 per year for cases on the E/D list vs. 12 and 143.5 for cases not on the E/D list, respectively. Overall the most common essential cases were laparotomy for trauma (1463; 705 year 1, 758 year 2), tracheostomy (665; 372 year 1, 293 year 2) and gastrostomy tubes (566; 289 year 1, 277 year 2). There are a total of 73 types of essential operations and 45 types of desirable operations in the current curriculum. There were 16 (13.6%) distinct codes never used of which 6 overlapped with other codes. Based on body region the ten E/D codes never used by any fellow are shown in the table.

Conclusion:The current ACS trainees lack adequate head/neck and pediatric experience as defined by the ACS curriculum. Restructuring rotations at individual institutions and a focus on novel educational modalities may be needed to augment the individual institutional exposure. Finally, re-evaluation of some aspects of the curriculum, particularly as it relates to the management of pediatric injuries and elective neck explorations may be warranted.

Case category	Essential (E) or Desirable (D)	Number of codes not used	Total number of codes in case category	% of total codes not used	Case specifics
Head/Face	D	1	5	20	Lateral canthotomy
Neck	D	5	12	41.7	All related to elective neck dissection
Thoracic	E	1	16	6.25	Vascular trauma to chest
Pediatric	D	3	5	60	Inguinal hernia repair, SBO treatments

NOTES

Session: VIII: Papers 13-16: Acute Care Surgery
Paper 16: 11:00-11:20 AM

PROTOCOLIZED MANAGEMENT OF ADHESIVE MECHANICAL SMALL BOWEL OBSTRUCTION: MOVING IT ALONG.

Janeen R. Jordan MD, Trina Bala RN, Scott Brakenridge MD, Chasen A. Croft MD, Lawrence Lottenberg* MD, Linda Atteberry* MD, Winston Richards MD, David Mozingo* MD, Alicia Mohr* MD, Frederick A. Moore* MD, University of Florida - Gainesville

Invited Discussant: Clay Cothren Burlew, MD

Introduction:Differentiating between a partial small bowel obstruction (SBO) likely to resolve with medical management and a complete obstruction requiring intervention remains elusive. For quality improvement, we implemented a standardized protocol for management of SBO and the purpose of this study is to evaluate its performance.
Methods:Patients with symptoms and X-ray findings of SBO were admitted for IV fluid resuscitation, bowel rest, nasogastric tube (ngt) decompression and exams every 4 hours. Labs and a CT scan of the abdomen and pelvis with IV contrast only were obtained. Patients with peritonitis or CT imaging findings suggestive of bowel compromise were taken to the operating room (OR) for exploration following resuscitation. All other patients were than given 120mL of diluted 2:1 gastrograffin (GG). KUBs were obtained at 4, 8, 12 and the 24th hour. If contrast did not reach the colon in 24 hours, then the patient was counseled and operative intervention was performed.
Results:Over a year, 101 patients were admitted for SBO. 26 patients went directly to the OR due to imaging or clinical findings suggesting bowel compromise (49% required bowel resection). Seventy-five patients were enrolled in the GG protocol of which 45.3% of patients underwent surgery. The average time to surgery was within 1 day for those not on the protocol and 2 days for those treated with GG

	LOS	Time of GG to colon (min)
GG+no surgery(n=41)	3($p<0.0001$)	300($p<0.0002$)
GG+surgery(n=34)	14(p=1)	480(p=1)
no GG+surgery(n=26)	12(p=0.2)	N/A

Conclusion:Differentiating between patients with a partial bowel obstruction vs. complete obstruction remains arduous. Institution of the GG protocol standardized our management algorithm with the goal to identify those patients with complete obstructions early and intervene more rapidly. The protocol may have led to a more rapid resolution of a partial obstruction. The administration of GG did not comparatively increase the risk of bowel ischemia or significantly increase the rate of bowel resection.

NOTES

THURSDAY, SEPTEMBER, 11, 2014, 11:30 AM

SESSION IX: AAST PRESIDENTIAL ADDRESS

LOCATION: GRAND BALLROOM SALONS G-L

"Responsibility"

William G. Cioffi, M.D., President
American Association for the Surgery of Trauma

Surgeon-in-Chief, Department of Surgery
Rhode Island Hospital
J. Murray Beardsley Professor & Chairman, Department of Surgery
The Warren Alpert Medical School at Brown University
Providence, RI

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Presiding: Thomas M. Scalea, M.D.

AAST President-Elect, 2013-2014

SESSION XA:

PAPERS #17 - #25

THURSDAY, SEPTEMBER 11, 2014, 2:00 PM – 5:00 PM

GRAND BALLROOM SALONS G-L

MODERATOR: MARTIN CROCE, M.D.

RECORDER: CHRISTINE COCANOUR, M.D.

Session: XA: Papers 17-25
Paper 17: 2:00-2:20 PM

NOT ALL BELLIES ARE THE SAME: A COMPARISON OF DAMAGE CONTROL SURGERY FOR INTRA-ABDOMINAL SEPSIS VERSUS TRAUMA

Jason W. Smith* MD,Ph.D., Nick A. Nash MD, Levi Procter MD, Matthew V. Benns MD, Keith R. Miller MD, Glen A. Franklin* MD, J D. Richardson* MD, Andrew C. Bernard* MD, Brian G. Harbrecht* MD, University of Louisville

Invited Discussant: Andre Campbell, MD

Introduction: Damage control surgery (DCS) has found a prominent place in the armamentarium of the acute care surgery. Developed to manage exsanguinating trauma patients it has been applied to the management of peritoneal sepsis and abdominal catastrophes. However, these entities are quite different and yet few manuscripts compare the outcomes of these surgeries on disparate patient populations

Methods: A multi-institutional three group 1:1:1 propensity score matched case cohort study comparing penetrating trauma (PT-DCS), blunt trauma (BT-DCS) and intra-peritoneal sepsis (IPS-DCS) patients treated between 2008-2013 with damage control surgery was performed. Propensity scoring was performed utilizing demographic and presenting physiologic data. Outcome variables were collected and analyzed utilizing univariate and multivariate techniques with a priori significance at $p< 0.05$.

Results: Four hundred and twelve (412) patients were treated with DCS during study period across the two institutions. After propensity matching for presenting physiologic and demographic variables, 80 patients per group were identified for comparison. Neither method of temporary abdominal closure nor institution predicted time to closure or rate of primary fascial closure. Rate of primary fascial closure was lowest in the IPS-DCS patients and highest in the PT-DCS patients which correlated with fewer numbers of operations and an increased time to closure.

Intra-abdominal complication rates (including abscess and EC fistula) were highest in the IPS-DCS group and correlated with an increased time to abdominal closure across all groups. (RR 1.8; 1.3-2.2; $p<0.03$) Mortality at 90 days was highest in the IPS-DCS patients and patients delayed >8 days were at more than twice the risk of death at 90 days across all groups.

(RR: 2.15; 1.2-3.5; $p<0.002$).

Conclusion: Expected outcomes after the use of DCS in trauma and emergency general surgery are very different. Despite these great differences, patients treated with prolonged open abdomens had a higher intra-abdominal complication rate and mortality at 90 days post-surgery. Regardless of etiology, prompt abdominal closure at the earliest possible opportunity affords the best outcome in patients managed via damage control surgery.

Table1: Selected outcome variables of patients treated using DCS

	PT- DCS (n=80)	BT- DCS (n=80)	IPS-DCS (n=80)
Time to closure(days)	**4.3 ± 1.9**	**6.9 ± 2.7 ***	**8.3 ±4.1‡†**
# of operations	**3 ± 2**	**5 ± 2 ***	**5 ± 5 ‡**
Primary fascial closure	**61 (79%)**	**49 (64%) ***	**40 (50%)‡†**
Abdominal complications	**18 (23%)**	**33 (41%) ***	**36 (45%)‡**
90 day Mortality	**10 (13%)**	**13 (16%)**	**21 (26%)‡†**

‡ denotes $p<0.05$ between IPS-DCS and PT-DCS †$p<0.05$ between IPS-DCs and BT-DCS, * denotes $p<0.05$ between PT-DCs and BT-DCS

NOTES

Session: XA: Papers 17-25
Paper 18: 2:20-2:40 PM

THE IMPACT OF TRANEXAMIC ACID ON MORTALITY IN INJURED PATEINTS WITH HYPERFIBRINOLYSIS

JOHN A. HARVIN MD, MARK M. MIMS BS, JESSICA HUDSON MD, JEANETTE PODBIELSKI RN, CHARLES E. WADE Ph.D., JOHN B. HOLCOMB* MD, BRYAN A. COTTON* MD,MPH, University of Texas Health Science Center-Houston

Invited Discussant: Ernest Moore, MD

Introduction:Tranexamic acid has been shown in a randomized trial to improve survival among trauma patients if given in the first three hours after injury. However, given the numerous issues and concerns surrounding the design and results of that study, the indications for its use remain uncertain. Supported by data from two separate trauma centers, we implemented a protocol to administer TXA in trauma patients with evidence of hyperfibrinolysis on admission. The purpose of this study was to examine whether the use of TXA in patients with fibrinolysis >3.0% by admission thrombelastography (TEG) was associated with improved survival.

Methods: Following IRB approval, we evaluated all trauma patients >15 years of age admitted to our center between 09/09 and 09/13. We excluded patients with admission fibrinolysis <3.0%, determined by TEG LY-30. Our protocol for TXA with LY30>3% was implemented in 09/11. The patients were then divided into those who received TXA (TXA group) in the emergency department and those who did not receive TXA (no TXA group). In addition to admission rapid TEG values, we evaluated follow-up rapid TEG values obtained within the first 6 hours. Univariate and multivariate analyses were performed. A logistic regression model was developed *a priori* to evaluate to evaluate the impact of TXA on in-hospital mortality (controlling for age, gender, ISS, arrival physiology, and base deficit).

Results: 1032 patients met study criteria. 98 (10%) of patients received TXA and 934 (90%) did not. TXA patients were younger (median 37 vs. 32, p=0.018), more severely injured (median ISS 29 vs. 14, p<0.001), more hypotensive (arrival SBP 103 vs. 125, p<0.001) and were more likely to be in shock (BE -5 vs. -2, p<0.001). Al arrival rapid TEG values were more hypocoagulable on admission (ACT 128 vs 113, alpha 69 vs. 71, mA 57 vs. 63, and LY30 5.3 vs 4.3, all p<0.001). However, with the exception of ACT (136 vs. 121, p=0.047) there were no differences in repeat rapid TEG values obtained in the first 6 hours (all p>0.05). Only 6% of TXA patients had LY30>3% on repeat rapid TEG (vs. 0% in the no TXA group, p<0.001). With respect to outcomes, mortality was significantly higher in the TXA group (40% vs. 17%, p<0.001) while there were no differences in venous thromboembolic events (3.3% vs. 3.8%). Controlling for age, gender, injury severity, shock, and hypotension, regression analysis failed to find a difference in mortality among those receiving TXA upon admission (odds ratio 1.74, 95% C.I. 0.38-1.40). Consistent with the implementation of our protocol in 09/11, use of TXA increased over the study period from 3% in the first to years to 21% in the last year. However, when evaluating outcomes by year and increasing TXA compliance, there were no mortality differences observed by univariate (23% vs 22%) or multivariate analyses (odds ratio 1.35, 95% C.I. 0.42-4.26).

Conclusion: In the current study, use of TXA upon arrival resulted in correction of hyperfibrinolysis in 94% of patients. However, this correction did not translate into a reduction in mortality. Depsite a protocol in place, trauma faculty chose not to administer TXA to 90% of patients. Further studies are needed to better define who will benefit (and who might be harmed) by administration of TXA.

NOTES

Session: XA: Papers 17-25
Paper 19: 2:40-3:00 PM

OVERWHELMING TPA RELEASE, NOT PAI-1 DEGRADATION, IS RESPONSIBLE FOR HYPERFIBRINOLYSIS IN MASSIVELY TRANSFUSED TRAUMA PATIENTS

Michael P. Chapman MD, Ernest E. Moore* MD, Eduardo Gonzalez MD, Hunter B. Moore MD, Theresa L. Chin MD, Arsen Ghasabyan MPH, CCRC, Fabia Gamboni Ph.D., Sanchayita Mitra MS, Anirban Banerjee Ph.D., Angela Sauaia MD,Ph.D., Christopher C. Silliman MD,Ph.D., Denver Health Medical Center

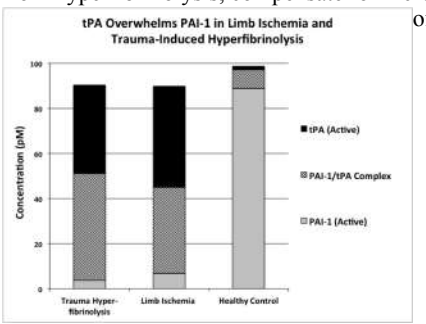
Invited Discussant: Yasuhiro Otomo, MD

Introduction: Hyperfibrinolysis is a highly lethal component of trauma-induced coagulopathy (TIC), but its mechanism is poorly understood. Plasminogen activator inhibitor-1 (PAI-1) and tissue plasminogen activator (tPA) are mutually inhibitory, existing in equilibrium with an inactive covalent complex. Degradation of PAI-1 by activated protein C (aPC) has been proposed as the initiator of hyperfibrinolysis in TIC. However, we have observed increased resistance to exogenous tPA in most trauma patients. Thus, we hypothesized that the normal reaction to traumatic injury is a protective elevation of PAI-1 and that cases of hyperfibrinolysis in TIC are caused by an overwhelming release of tPA from ischemic tissues.

Methods: Consecutive trauma patients activating our massive transfusion protocol (MTP, n=22) had blood collected on admission. Functional ELISAs for active tPA and PAI-1 as well as the inactive PAI-1/tPA complex were performed. Hyperfibrinolysis was defined as a tranexamic acid-reversible clot lysis after 30 minutes ≥3% by thrombelastography. Trauma patients were compared to healthy volunteers (n=3) before and after 45 minutes of upper extremity ischemia.

Results: 41% of MTP patients (median ISS 34, IQR 25-41), had hyperfibrinolysis. Total and free tPA were dramatically elevated in these patients compared to controls: total 86.8±20.0 vs. 9.9±1.2 picomolar (pM); (p= 0.024, t-test) and free 39.4±5.2 pM vs. 1.7±0.6 pM; (p=0.001). Total PAI-1 levels did not change significantly. Instead, the PAI-1 population shifted from 91% free PAI-1 to 7% in hyperfibrinolysis, the remainder complexed to tPA. The same pattern was observed after limb ischemia. In non-fibrinolytic trauma patients, total tPA was still markedly elevated, but was compensated for by a 20-fold increase in total PAI-1.

Conclusion: Hyperfibrinolysis in trauma with hemorrhagic shock is driven by a massive increase in tPA levels. Excess tPA inactivates PAI-1 by driving formation of the covalent PAI-1/tPA complex. Enzymatic degradation of active PAI-1 is not a significant feature of trauma-induced hyperfibrinolysis. Severely injured trauma patients who do not suffer from hyperfibrinolysis, compensate for increased tPA levels with a massive upregulation of PAI-1, which suppresses tPA activity.



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Session: XA: Papers 17-25
Paper 20: 3:00-3:20 PM

Lung Protective Ventilation (ARDSNet) Versus APRV: Ventilatory Management of a Combined Model of Acute Lung and Brain Injury

Stephen W. Davies MD,MPH, Kenji L. Leonard MD, Randall K. Falls Jr., MD, Ronald P. Mageau MD, Joseph P. Hollowell BA, Wayne E. Trainer II, BS, Hilal A. Kanaan MD, Robert C. Hickner Ph.D., Robert G. Sawyer MD, Nathaniel R. Poulin MD, Brett H. Waibel* MD, Eric A. Toschlog* MD, Brody School of Medicine at East Carolina University

Invited Discussant: Orlando Kirton, MD

Introduction: Each year, over 300 thousand Americans suffer concomitant traumatic brain and lung injury resulting in significant morbidity and mortality. Lung protective ventilation (ARDSNet) has become the standard for managing acute respiratory distress syndrome (ARDS); however, the resulting permissive hypercapnea may compound traumatic brain injury. Airway pressure release ventilation (APRV) offers an alternative strategy for managment of this patient population. The purpose of this study was to evaluate the effects of APRV compared to ARDSNet protocol on a swine model with concomitant lung and brain injury.
Methods: Yorkshire swine were randomized to either ARDSNet or APRV. Lung injury was induced using 0.1N hydrochloric acid (4cc/kg) via the endotracheal tube. Intracranial hypertension (brain injury) was induced by inflating an intracranially placed, catheter to an intracranial pressure (ICP) between 30-40mmHg. Ventilatory settings and pulmonary parameters, vitals, arterial and venous blood gases, quantitative histopathology, and cerebral microdialysis were compared between groups.
Results: 22 swine (17male, 5female), weighing 24.2±5.6kg, were randomized to APRV (n=9), ARDSNet (n=12), or sham (n=1). Baseline characteristics (while on pressure support) were similar between groups. Following lung and brain injury, static compliance (APRV 11.7±1.1L/cmH_2O versus ARDS Net 10.9±4.7L/cmH_2O), P/F ratio (165±66 versus 181±52), and cerebral perfusion pressure (CPP)(65±14 versus 78±21mmHg) dropped significantly, while ICP (40±19 versus 31±6mmHg) increased significantly compared with baseline. Peak inspiratory pressure was significantly greater among ARDSNet recipients; however, static complicance, P/F ratio, CPP, and ICP were not significantly different between groups throughout the duration of the study. Additionally, preliminary review of histopathology and cerebral microdialysis did not differ significantly between groups; however, cerebral biomarkers glucose, glycerol, pyruvate, and lactate/pyruvate ratio trends suggest reduced cerebral ischemia with ARDSNet (Table).
Conclusion: Previous studies have not evaluated the effects of APRV in this population. While macroscopic parameters did not observe a significant difference between groups, microdialysis data suggest a trend toward cerebral ischemia associated with APRV. Additional and future studies should focus on extending the time interval for observation to deliniate differences between groups.

Table: Cerebral Microdialysis Biomarkers

	Mode	Baseline	1.5hr	2.5hr	3.5hr	4.5hr	5.5hr	6.5hr
Glucose (mmol/L)	APRV ARDSNet	1.0±0.79	0.84±0.65 1.4±1.2	0.23±0.071 1.7±1.3	ND 2.2±2.0	ND 3.1±2.2	ND 1.8±1.1	ND 1.6±1.0
Lactate (mmol/L)	APRV ARDSNet	4.0±4.3	5.6±2.9 5.1±2.5	6.9±1.9 7.3±4.9	6.5±1.4 7.4±4.8	6.2±0.60 4.7±2.6	6.5±1.1 5.2±2.4	6.6±1.2 7.3±3.9
Glycerol (µmol/L)	APRV ARDSNet	112±131	63±26 118±79	102±118 122±72	156±254 122±66	185±292 95±73	355±454 90±73	262±403 145±145
Pyruvate (µmol/L)	APRV ARDSNet	237±426	130±70 128±46	102±110 255±236	147±148 126±84	126±153 128±87	68±38 149±90	96±56 183±147
Lac/Pyr	APRV ARDSNet	23±10	58±44 38±14	174±132 40±18	106±82 86±99	145±115 67±70	124±69 62±59	93±52 68±78

NOTES

Session: XA: Papers 17-25
Paper 21: 3:20-3:40 PM

RECONSTITUTION FLUID TYPE DOES NOT AFFECT PULMONARY INFLAMMATION OR DNA DAMAGE FOLLOWING INFUSION OF LYOPHILIZED PLASMA

Sean P. McCully MD, MS, Tim H. Lee MD, MS, Belinda H. McCully Ph.D., Elizabeth A. Rick BS, Nathan W. Anderson BS, David A. Hampton MD, MEng, Scott G. Louis MD, Martin A. Schreiber* MD, FACS Oregon Health & Science University

Invited Discussant: Frederick Moore, MD

Introduction: Dysfunctional inflammation following severe trauma and hemorrhagic shock can lead to multiple organ failure, and death. Compared to lyophilized plasma (LP) buffered with other acids, LP reconstituted in sterile water with ascorbic acid (AA) in our polytrama swine model restores hemostasis, suppresses systemic inflammation, and attenuates DNA damage. It is unknown whether the inflammatory response is affected by the type of fluid used to reconstitute LP. We hypothesize that commonly used reconstitution fluids: sterile water (SW), lactated Ringers (LR), normal saline (NS), or Hextend (HX) will yield similar profiles of pulmonary and systemic inflammation, and DNA damage following resuscitation with LP.

Methods: This was a randomized, prospective, blinded animal study. Donor plasma was collected from swine and lyophilized. LP was reconstituted to 50% of original volume with SW, LR, NS, or HX buffered with 15mM AA. Forty swine were subjected to a validated model of polytrauma, hemorrhagic shock, Grade V liver injury, and resuscitated with LP. Physiologic data was collected. Serum IL-6, IL-10 and plasma 8-hydroxy-2-deoxyguanosine concentrations were assessed for systemic inflammation and DNA damage at baseline, 2 and 4 hours following liver injury. Lung inflammation was evaluated by RT-PCR.

Results: Physiologic parameters and degrees of shock were not different between groups. The pH of reconstituted LP prior to resuscitation was similar between all groups. In all groups, serum IL-6 and IL-10 increased at 2- and 4-hours compared to baseline ($p \leq 0.05$), with no difference between groups. Serum IL-10 peaked between 1 and 4 hours following liver injury. In animals resuscitated with LP reconstituted with NS, LR and SW, DNA damage (Figure 1) increased from baseline to 2 hour, baseline to 4 hour and 2 to 4 hours, $p \leq 0.05$ (*). In animals resuscitated with LP reconstituted with HX, increased DNA damage occurred only at 4 hours versus baseline, $p \leq 0.05$ (Ψ). DNA damage was not different between groups at any time point. Lung tissue inflammation was similar between groups.

Conclusions: Reconstitution fluid type does not affect inflammatory cytokine profiles or DNA damage. Based on these findings and its universal availability, sterile water appears to be the fluid of choice for reconstitution of LP.

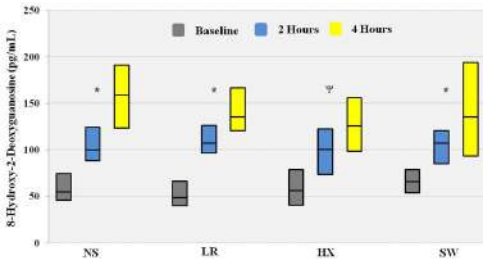


Figure 1: Plasma 8-Hydroxy-2-Deoxyguanosine concentration. Medians (IQR) are provided for each fluid group across time.

NOTES

Session: XA: Papers 17-25
Paper 22: 3:40-4:00 PM

CLEARLY DEFINING PEDIATRIC MASSIVE TRANSFUSION: CUTTING THROUGH THE FOG AND FRICTION WITH COMBAT DATA

Lucas P. Neff MD, Jeremy W. Cannon* MD, Jonathan J. Morrison MB, Philip C. Spinella MD, Matthew A. Borgman MD, San Antonio Military Medical Center

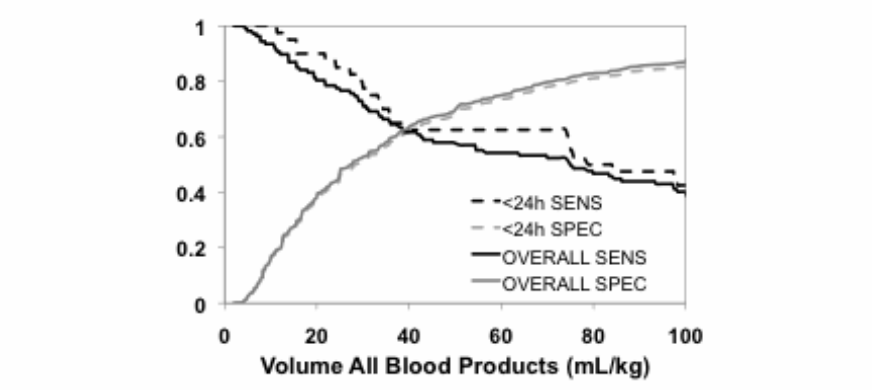
Invited Discussant: Michael Nance, MD

Introduction: Massive transfusion (MT) in pediatric patients remains poorly defined. Using the largest existing collection of transfused pediatric patients, we sought to identify an MT threshold which serves as an accurate surrogate for risk of death from trauma.

Methods: The Department of Defense Trauma Registry (DoDTR) was queried from 2001-2013 for pediatric trauma patients (<18 years). Burns, drowning, isolated head trauma, and missing injury severity score (ISS) were excluded. MT was evaluated as a weight-based volume of *all* blood products given at any time in the first 24 hours including packed red blood cells, whole blood, plasma, platelets, and cryoprecipitate. Mortality at 24 hours and in-hospital was calculated for increasing transfusion volumes. Sensitivity and specificity curves for predicting mortality were used to identify an optimal MT threshold. Patients above and below this threshold (+MT and –MT, respectively) were compared.

Results: The DoDTR yielded 4,990 combat-injured pediatric trauma patients of whom 1,340 were excluded. An additional 2,536 were not transfused. The remaining 1,114 transfused patients comprised the study cohort. Sensitivity and specificity for 24-hour and in-hospital mortality were optimal at 40.4 mL/kg and 38.8 mL/kg total blood products in the first 24 hours, respectively (Figure). Using a pragmatic threshold of 40 mL/kg, patients were divided into +MT (n=436) and –MT (n=678). On univariate analysis, +MT patients were more often in shock (68.1% vs. 47.3%, $p<0.0001$), hypothermic (12.9% vs. 3.6%, $p<0.0001$), coagulopathic (44.2% vs. 30.2%, $p=0.0002$), and thrombocytopenic (10.9% vs. 5.0%, $p=0.0008$) on presentation. +MT patients also had a higher ISS, longer ICU and hospital length of stay, more mechanical ventilator days, and a higher 24-hour (5.7% vs. 2.2%, $p=0.002$) and in-hospital mortality (15.1% vs. 6.0%, $p<0.0001$).

Conclusion: Based on this large cohort of transfused combat-injured pediatric patients, a threshold of 40 mL/kg of *all* blood products given at any time in the first 24 hours reliably identifies critically injured children at high risk of early and in-hospital death. This evidenced-based definition will provide a consistent framework for future research and protocol development in pediatric resuscitation.



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Session: XA: Papers 17-25
Paper 23: 4:00-4:20 PM

ANGIOTENSIN INHIBITION DECREASES MULTIPLE ORGAN FAILURE IN OBESE TRAUMA PATIENTS

Robert D. Winfield MD, Robert E. Southard MD, Anja Fuchs Ph.D., Kelly Bochicchio MSN, Bradley D. Freeman MD, Douglas J. Schuerer* MD, Grant V. Bochicchio* MD,MPH, Washington University School of Medicine

Invited Discussant: Robert Cooney, MD

Introduction: Obese patients fare poorly following severe blunt injury, and are more likely to develop multiple organ failure (MOF) than lean patients. The pathophysiology of obesity includes an overactive, adipose tissue-derived, renin-angiotensin-aldosterone system (RAAS); this affects inflammatory responses via leukocyte angiotensin receptors. We hypothesized that obese patients taking pre-injury ACE Inhibitors (ACE-I) or angiotensin receptor blockers (ARB) would show a decrease in multiple organ failure and differences in immune cell frequencies.

Methods: We analyzed data contained within the "Inflammation and the Host Response to Injury" trauma related database. Patients taking pre-injury ACE-I and ARB were characterized as obese (BMI $\geq$30) or nonobese (BMI<30). Groups were then age, gender, and ISS matched against patients in the database who were not taking ACE-I or ARB. Patients were compared on the basis of demographic information, two MOF scores (Marshall Multiple Organ Dysfunction Score and Denver-2 Postinjury MOF Score), and leukocyte surface markers on T cells and monocytes as measured by flow cytometry.

Results: 1,932 patients were evaluated. Of these, 110 took pre-injury ACE-I (n=80) and/or ARB (n=31) with 94 patients (55 obese, 39 non-obese) having data available to calculate BMI. These patients were compared to patients not taking ACE-I or ARB (102 obese, 75 nonobese). Obese patients taking ACE-I/ARB showed maximum Marshall (5.83 ± 2.87) and Denver-2 (2.45 ± 2.32) scores similar to nonobese patients taking or not taking ACE-I/ARB, while obese patients not taking ACE-I/ARB had significantly higher Marshall (6.49 ± 2.57, $p=0.009$) and Denver-2 (3.33 ± 2.21, $p=0.006$) scores. Leukocyte analysis demonstrated multiple differences, most notably lower frequencies of GITR ($p<0.001$) and CD328 ($p<0.001$) positive T cells in obese patients taking ACE-I/ARB when compared to obese not taking these medications.

Conclusions: Obese patients taking pre-injury ACE-I/ARB show post-injury organ failure scores similar to nonobese patients, while obese patients not taking these medications show significantly greater post-injury organ failure. Leukocyte analysis demonstrates differences in T cell and monocyte surface marker expression, and this may indicate improved regulation of the immune system. Further study is needed to investigate the connection between these findings, and to clarify the role of angiotensin and adipose RAAS in post-injury immune dysfunction.

NOTES

Session: XA: Papers 17-25
Paper 24: 4:20-4:40 PM

TIME FOR CLOSURE: A DEDICATED TRAUMA ICU IS ASSOCIATED WITH LOWER POST INJURY COMPLICATION RATES AND DEATH AFTER MAJOR COMPLICATIONS

Marko Bukur* MD, Ivan Puente MD, Joe Catino MD, MIchael Parra MD, Margaret Crawford RN, MSN,CCRN,CEN, Robyn Farrington MBA,RN, BSN, Fahim Habib MD,MPH, Delray Medical Center

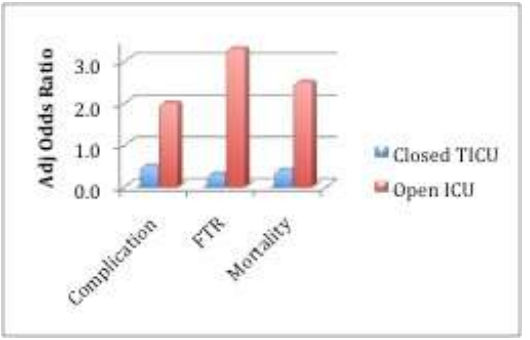
Invited Discussant: Charles Adams, Jr., MD

Introduction: Recent data suggests that injured patients admitted to a dedicated trauma intensive care unit (TICU) have better outcomes than those admitted to mixed ICUs. The cause for this apparent discrepancy has not been well established. We hypothesized that trauma patients admitted to a dedicated TICU would have a lower frequency of post injury complications, as well as, death after major complication (failure to rescue [FTR]).

Methods: We performed a retrospective review of patients admitted to the respective ICUs of two Level I trauma centers covered by the same group of trauma and surgical intensivists over the past 5 years. One center has a dedicated TICU, while the other has a mixed, open ICU. Patients were excluded if they had non-survivable injuries or an ICU stay of less than 48 hours. Relevant demographic and clinical characteristics were abstracted and stratified into TICU and ICU groups for comparison. The primary outcomes were overall post-injury complication rate and FTR between the respective ICU models. Multivariate regression was used to derive factors associated with complication rate(s) and FTR.

Results: During the 5 year study period, 2,567 patients were admitted to the TICU and 1,266 to the mixed ICU respectively. TICU patients were older (Mean Age 57.8 vs. 47.0, $p<0.0001$), had more co-morbidities (Charlson score 2 vs. 1, $p=0.001$), were more likely to be admitted with severe head injuries (Head AIS $\geq$ 3, 50.0% vs. 37.5%, $p<0.0001$), and have a greater overall injury burden (ISS>16 49.6% vs. 38.6%, $p<0.0001$) than those admitted to the mixed ICU. Need for operative intervention was similar between the two groups (18.0% vs. 17.6%, $p=0.788$). Complications were significantly higher in trauma patients admitted to the mixed ICU (27.5% vs. 17.0%, $p<0.0001$), as well as, FTR (3.7% vs. 1.8%, $p<0.0001$). After adjusting for confounding factors, trauma patients admitted to a dedicated TICU had a significantly lower chance of developing a post-injury complication (AOR 0.5, 95%CI [0.4,0.6], $p<0.0001$), FTR (AOR 0.3, 95%CI [0.2,0.5], $p<0.0001$), and overall mortality (AOR 0.4, 95%CI [0.3,0.5], $p<0.0001$).

Conclusion: Admission of critically ill trauma patients to a dedicated TICU staffed by a surgical intensivist is associated with a significantly lower risk of developing p ost-injury complications and death after major complication. Factors such as trauma nursing experience, education, and unit structure should be further explored to elucidate the observed improved outcomes.



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Session: XA: Papers 17-25
Paper 25: 4:40-5:00 PM

FACTOR VIIA ADMINISTRATION IN TRAUMATIC BRAIN INJURY: AN AAST-MITC PROPENSITY SCORE ANALYSIS

Sarah Lombardo MD, MSc, Tom H. Greene Ph.D., Molly L. McFadden MS, Thomas M. Scalea* MD, Gregory J. Jurkovich* MD, Jason Sperry* MD,MPH, Iman Feiz-Erfan* MD, Patrick O'Neill* MD,Ph.D., Raul Coimbra* MD,Ph.D., Raminder Nirula* MD,MPH, University of Utah

Invited Discussant: M. Margaret Knudson, MD

Introduction: Recombinant Factor VIIa (rFVIIa) is FDA-approved for treatment of uncontrolled bleeding in patients with hemophilia. There are numerous accounts of off-label rFVIIa use as an adjunct in the reversal of warfarin therapy and management of hemorrhage in the setting of trauma. Only a handful of these reports are rigorous studies, from which results regarding safety and effectiveness have been mixed. There remains no clear consensus as to the role of rFVIIa in traumatic brain injury.

Methods: Eleven Level 1 trauma centers provided clinical data and head CT scans of patients with a GCS<=13 and radiographic evidence of TBI. CTs were blindly graded according to the Marshall classification. A propensity score (PS) to receive rFVIIa in those surviving 2 days or greater was calculated for each patient based upon patient demographics, comorbidities, physiology, injury severity score (ISS), admission GCS, and treatment center. Patients who actually received rFVIIa within 24hr hours of admission were matched to patients who did not receive rFVIIa for outcomes assessment.

Results: There were 4284 patient observations; 129 (3.3%) received rFVIIa within 24 hours of admission. Those receiving rFVIIa tended to be older, male, have more comorbidities, on warfarin therapy, and had higher ISS and head AIS scores. Groups were comparable after matching. No differences in mortality or morbidity were seen in association with rFVIIa. GCS at discharge was significantly lower among those receiving rFVIIa in the setting of polytrauma (-1.40, 95% CI: -2.572, -0.221), and a trend towards decreased GCS was seen among primary head injury patients receiving rFVIIa (-1.00, 95% CI: -2.934, 0.934).

Conclusion: Use of rFVIIa in early management of the traumatic brain injured patient is not associated with a decreased risk of mortality or morbidity, and may negatively impact recovery and functional status at discharge.

	Pre-matching		**Post-matching, all comers**		**Post-matching, primary head injury**	
	OR/coef	*95% CI*	*OR/coef*	*95% CI*	*OR/coef*	*95% CI*
Mortality	3.45	2.416, 4.931	1.57	0.731, 3.379	0.56	0.161, 1.919
Morbidity	0.61	0.367, 1.006	0.66	0.298, 1.475	1.84	0.387, 8.767
VAP	0.61	0.307, 1.207	1.00	0.330, 3.035	--	--
ARDS	0.56	0.176, 1.781	3.10	0.314, 30.608	--	--
DVT	0.48	0.118, 1.973	0.48	0.085, 2.741	--	--
CRBSI	0.34	0.047, 2.460	--	--	--	--
Abscess	6.68	1.429, 31.244	--	--	--	--
Meningitis	1.06	0.143, 7.853	1	0.061, 16.342	--	--
GCS at discharge	-1.39	-2.076, -0.703	-1.40	-2.572, -0.221	-1.00	-2.934, 0.934

NOTES

SESSION XB:

PAPERS #26 - #34

THURSDAY, SEPTEMBER 11, 2014, 2:00 PM – 5:00 PM

GRAND BALLROOM SALONS A, B & F

MODERATOR: ARI LEPPANIEMI, M.D.

RECORDER: ROBERT MACKERSIE, M.D.

Session: XB: Papers: 26-34
Paper 26: 2:00-2:20 PM

OBESITY AND CLOTTING: BMI INDEPENDENTLY CONTRIBUTES TO HYPERCOAGULABILITY AFTER INJURY

Lucy Kornblith MD, Benjamin Howard MD,MPH, Ryan Kunitake BA, Brittney Redick BA, Mary Nelson RN, MPA, Mitchell Cohen* MD, Rachael Callcut* MD, MSPH University of California, San Francisco

Invited Discussant: Hasan Alam, MD

Introduction: The role of obesity as a mediator in coagulation after injury remains unknown. However, obese patients exhibit chronic low-grade inflammation and have high rates of thrombosis after injury. We hypothesized that BMI is independently associated with increased measures of hypercoagulability longitudinally after injury.

Methods: Demographics, outcomes, and laboratory measures were prospectively collected on arrival and up to 28 days for 377 consecutive highest-level trauma activation patients with a body mass index (BMI) ≥ 18.5, not anti-coagulated, and without liver failure at a single Level 1 Trauma Center. Standard coagulation measures, citrated kaolin and functional fibrinogen thromboelastography (TEG), and an extensive panel of clotting factors were measured at 0, 6, 12, 24, 48, 72, 96, and 120h. Multiple linear regression was used at each time point to examine the relationship of BMI with clotting measures. Multiple logistic regression was used to define predictors of thromboembolic complications. BMI categories were defined by standard cutoffs: normal weight (BMI 18.5-24.99 kg/m^2), overweight (BMI 25-29.99 kg/m^2), and obese (BMI ≥ 30 kg/m^2).

Results/progress: The 377 patients were mostly male (81%) and bluntly injured (61%), with a median BMI of 25.8 kg/m^2. 42% were normal weight (median BMI 22.5 kg/m^2), 32% were overweight (median BMI 27.1 kg/m^2), and 26% were obese (median BMI 33.0 kg/m^2). There were no differences in age, gender, ISS or base deficit between groups, but the obese patients had lower rates of blunt injury and head injury than the normal weight patients (blunt injury rate 54.6% vs. 71.5%p=0.007; median AIS-head 0 vs. 2, p=0.008). There were no differences in admission INR/PTT or factors II, V, VII, VIII, X, ATIII, or protein C across BMI groups. However, obese patients had significantly higher admission platelet counts (302.69 vs. 268.58 x 10^9/L, p=0.004), factor IX (134 vs. 119 % activity, p=0.04), and lower D-dimer (1.88 vs. 4.00 ug/mL, p=0.004) than normal weight patients. Measured by TEG, clot strength (MA) and functional fibrinogen level (FLEV) were also higher on admission for obese patients (MA 65.7 vs. 63.4 mm,p=0.016; FLEV 406.53 vs. 350.73 mg/dL, p=0.008). In multiple linear regression, the relationship of BMI to clot strength, FLEV, and FIX persisted through 24h (Table). Similarly, the relationship of BMI and platelet count persisted through 120h (all$p$<0.05). In multiple logistic regression, for every 5kg/m^2 increase in BMI, there was an 85% increase in odds of developing a thromboembolic complication (OR 1.85, CI 1.13-3.08, p=0.017).

Conclusions: Following injury, obese patients are hypercoagulable compared to their similarly injured normal weight counterparts. This hypercoagulability persists to at least 24h after injury as demonstrated by relative thrombocytosis, stronger clot, increased FIX activity, and increased functional fibrinogen levels with increasing BMI. The clinical significance of this hypercoagulability needs to be elucidated to guide management of anticoagulation in this at-risk group.

Table. Multiple linear regression for each 5kg/m2 increase in BMI from 0-24h

	0h	6h	12h	24h
MA (mm)	1.01 (p=0.002)	1.24 (p=0.004)	1.17 (p=0.005)	0.95 *($p=0.032$)*
FLEV (mg/dL)	23.20 (p=0.001)	19.92 (p=0.047)	19.85 (p=0.016)	15.96 (p=0.049)
Factor IX (% act)	7.79 (p=0.015)	13.88 (p=0.003)	9.30 (p=0.016)	7.83 (p=0.133)
Platelet (x10^9/L)	10.94 (p=0.013)	23.30 (p=0.020)	33.33 (p<0.001)	10.57 (p=0.002)

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Session: XB: Papers: 26-34
Paper 27: 2:20-2:40 PM

THE NEW METRIC TO IDENTIFY LARGE-VOLUME HEMORRHAGE: RESULTS OF A PROSECTIVE STUDY OF THE CRITICAL ADMINISTRATION THREHSOLD

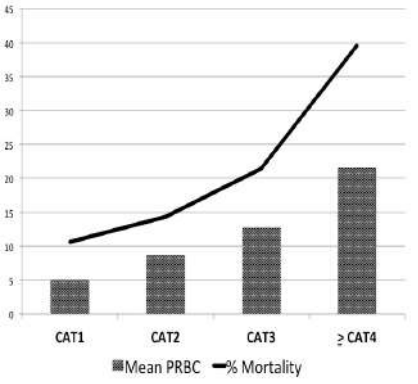
Stephanie Savage* MD, MS, Joshua J. Sumislawski BS, Ben L. Zarzaur* MD,MPH, Wesley P. Dutton BS, Martin A. Croce* MD, Timothy C. Fabian* MD, University of Tennessee Health Science Center - Memphis

Invited Discussant: John Holcomb, MD

Introduction: The current definition of massive transfusion (MT, $\geq$ 10 units PRBC in 24 hours) focuses on a static volume over fixed time and is a crude estimate of acute hemorrhage. This arbitrary volume-definition leads to survival bias and fails to identify the 'massively' transfused patient. In prior retrospective work, the critical administration threshold (CAT) was created to incorporate both rate and volume of transfusion; CAT proved a superior predictor of mortality compared to traditional MT (*J Trauma Acute Care Surg, 2013*). The purpose of this study is to prospectively validate CAT in a larger population of trauma patients. We hypothesize that CAT is a highly sensitive method to identify actively bleeding patients.

Methods: Patients receiving at least one unit of blood within the first 24 hours of admission were identified prospectively from June 2012 to May 2013. Patients with isolated head injury or cardiac arrest upon arrival were excluded. Administration time of each blood unit was recorded in minutes from time of injury. CAT status, defined as receipt of at least 3 units of blood in a 60-minute period, was identified for each hour of the first 24 hours. CAT+ patients were further quantified by number of times CAT+ was reached: once (CAT1), twice (CAT2), three times (CAT3) or 4 or more times (CAT4). A multivariable Cox Proportional Hazard model with a time-varying covariate was used to quantify risk of death with increasing CAT status.

Results: 432 patients were prospectively identified. 308 met inclusion criteria, 163 were CAT+. 76% were male with mean age of 38 years, 46% penetrating injury, mean injury severity score (ISS) of 18.6 and 21% mortality. CAT+ showed a 3-fold increased risk of death (HR 3.099, 95% CI 1.187, 8.091), and multiplicative increases in mortality risk with each CAT+.



Subdividing CAT+ patients by number of events showed a consistent increase in gross mortality (*Figure*). 95 patients were CAT+ and received less than 10 units of blood, thereby failing to meet criteria for MT (CAT+/MT-). CAT+/MT- had significant injury patterns with a mean ISS of 18.1 (blunt ISS 22, penetrating ISS 13.3) and 11% mortality. Mean time to initial CAT+ in the CAT+/MT- group was 236 minutes.

Conclusion: The prospective nature of CAT allows early identification of injured patients at greatest risk of death. Encompassing both rate and volume, CAT is a more sensitive tool than common massive transfusion definitions. Further, CAT allows identification of hemorrhaging patients excluded by static definitions of MT. Studies examining large-volume blood transfusions should utilize CAT, not traditional MT definitions, to accurately identify cohorts of interest.

NOTES

Session: XB: Papers: 26-34
Paper 28: 2:40-3:00 PM

A PHARMACOLOGIC APPROACH TO VAGAL NERVE STIMULATION PREVENTS MESENTERIC LYMPH TOXICITY AFTER HEMORRHAGIC SHOCK

Koji Morishita MD,Ph.D., Akinori Ueno Ph.D., Brian Eliceiri Ph.D., Todd W. Costantini MD, Raul Coimbra* MD,Ph.D., University of California, San Diego

Invited Discussant: William Cioffi, MD

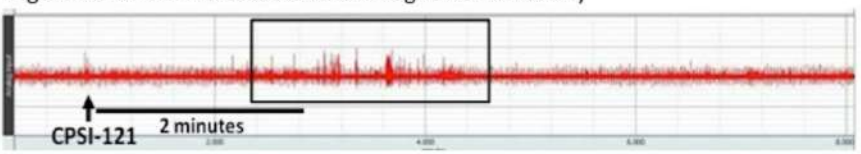
Introduction: Electrical stimulation of the vagus nerve (VN) prevents gut inflammation and mesenteric lymph (ML) toxicity in animal models of injury. While electrical stimulation of the VN currently has limited translational applicability, a pharmacologic approach to stimulating the VN could easily be administered to injured patients. We have previously shown that treatment with CPSI-121, a guanylhydrazone-derived compound, prevents gut barrier failure after burn injury. While the structure of CPSI-121 predicts that it will activate parasympathetic signaling, its ability to stimulate the VN is unknown. The aims of this study were to 1) measure the ability of CPSI-121 to induce VN activity, 2) determine whether CPSI-121 causes significant hemodynamic effects, and 3) further define the potential for CPSI-121 to limit the systemic inflammatory response to injury.

Methods: Male Sprague Dawley rats were given 1 mg/kg of CPSI-121 intravenously while blood pressure, heart rate, and efferent VN electrical activity was recorded. Rats also underwent cannulation of the mesenteric lymph duct prior to trauma/hemorrhagic shock (T/HS, 60 min at a mean arterial pressure of 35 mmHg). Following T/HS, animals were resuscitated with shed blood and normal saline. A separate cohort of animals received CPSI-121 after the HS phase. Gut tissue was harvested at 2 hours after injury for histologic analysis. The ability of mesenteric lymph to prime neutrophils was assessed by measuring in vitro oxidative burst using flow cytometry.

Results: Blood pressure was not altered after treatment with CPSI-121, while heart rate decreased only slighty. Recording of efferent VN electrical activity revealed an increase in discharge rate starting at 2 minutes after administration of CPSI-121 that lasted for several minutes (See Figure). T/HS caused histologic gut injury which was prevented in animals treated with CPSI-121 ($p< 0.05$). Treatment with CPSI-121 following T/HS attenuated neutrophil priming after exposure to ML, with decreased neutrophil oxidative burst compared to T/HS alone ($p < 0.05$).

Conclusion: CPSI-121 causes efferent vagus nerve output and limits gut injury and ML toxicity after T/HS. CPSI-121 is a candidate pharmacologic approach to vagus nerve stimulation aimed at limiting the inflammatory response in patients following severe injury.

Figure: CPSI-121 increases efferent vagus nerve activity



NOTES

Session: XB: Papers: 26-34
Paper 29: 3:00-3:20 PM

FIBRINOGEN CONCENTRATE ADMINISTRATION INHIBITS ENDOGENOUS FIBRINOGEN SYNTHESIS IN PIGS AFTER TRAUMATIC HEMORRHAGE

Wenjun Z. Martini Ph.D., Michael Dubick* Ph.D., US Army Institute of Surgical Research

Invited Discussant: Peter Rhee, MD, MPH

Introduction:
Fibrinogen plays a central role in coagulation and has been shown to fall to critical levels early after trauma. Administration of fibrinogen concentrate (FC) to improve hemostasis after bleeding under various pre-clinical and clinical conditions seems beneficial, but it is unclear whether its use introduces overly abundant fibrinogen with potential risk of thrombosis. This study investigated changes of endogenous fibrinogen metabolism from FC administration following traumatic hemorrhage in pigs.
Methods:
Anesthetized, instrumented pigs were randomized into LR and FC groups (n=7 each). Femur fracture was induced using the captive bolt stunner at mid shaft of the pigs' left legs, followed by hemorrhage of 60% total blood volume and resuscitated with LR (3x bled volume, LR group) or LR plus FC at 250 mg/kg in the FC group. Afterwards, a primed constant infusion of stable isotope 1- ^{13}C-phenylalanine (phe, 6h) and d_5-phe (3h) was performed with hourly blood sampling and subsequent gas chromatography and mass spectrometry analysis to quantify fibrinogen synthesis and breakdown rates, respectively. Hemodynamics was continuously monitored. Blood samples were taken at phases for blood and coagulation measurements using Thrombelastograph ®. Animals were euthanized at the end of the 6 h isotope period.
Results:
MAP decreased from baseline (BL) 75 ± 3 mmHg to 37 ± 2 mmHg by the hemorrhage but returned to near BL within 1h after resuscitation in both groups. Hemorrhage and LR resuscitation reduced total protein, Hct, fibrinogen and platelets to 50% of BL; decreased fibrinogen concentration from 207 ± 6 mg/dL to 132 ± 7 mg/dL and clot strength from 72 ± 2 mm to 63 ± 2 mm in both groups (both $p<0.05$). FC administration in the FC group recovered plasma fibrinogen concentration and restored clot strength within 15 min, while no changes observed in LR group. Fibrinogen synthesis rate in FC was reduced to 1.3 ± 0.2 mg/kg/h, compared to 3.1 ± 0.5 mg/kg/h in LR ($p<0.05$). Fibrinogen breakdown rates were similar between FC and LR.
Conclusion:
In addition to acute recoveries of fibrinogen concentration and clotting strength, administration of FC after traumatic hemorrhage inhibited fibrinogen synthesis without affecting fibrinogen breakdown. These data suggest a feedback mechanism in regulating host fibrinogen availability and an unlikely risk of thrombosis from FC administration.

NOTES

Session: XB: Papers: 26-34
Paper 30: 3:20-3:40 PM

ARE WE MEASURING THE RIGHT THING? CARDIAC DYSFUNCTION, NOT CARDIAC OUTPUT, IS PREDICTIVE OF OUTCOME IN CRITICALLY ILL SURGICAL PATIENTS

Sarah B. Murthi* MD, Samuel M. Galvagno DO,Ph.D., Jacob J. Glaser MD, Thomas M. Scalea* MD, University of Maryland Medical Center

Invited Discussant: Paula Ferrada, MD

Introduction: Hemodynamic monitors use cardiac output (CO) as the sole assessment of cardiac function. We sought to determine the *i*) accuracy of CO in detecting cardiac dysfunction (CDF) and *ii*) the association between CDF and outcome.

Methods: The Focused Rapid Echocardiographic Evaluation (FREE) assesses CO by CI and stoke volume index (SVI). The functional assessments are; left ventricular (LV) ejection fraction (EF), LV diastolic function, and right ventricular (RV) function. The area under the Receiver-Operator Characteristic (AUROC (95% CI)) was used to determine the accuracy of CO (CI and SVI) in detecting moderate and severe CDF (EF <30%, diastolic dysfunction (DD), and RV dysfunction (RVD)). In addition we determined the association of CI, EF, DD and RVF on mortality and other measures of outcome.

Results: From 9/2009-3/2013, 1059 patients had a FREE performed for clinical indications. The average age was 60 yrs (±) 18, 60% were male and, 52% had undergone surgery. The overall prevalence of dysfunction (EF <55%, DD and RVD) was 24%, 32% and 15%. The prevalence of moderate and severe dysfunction was, 12% for EF<30%, 19% for DD, and 9% for RVD. Both CI and SVI were inaccurate detectors of all forms of moderate to severe dysfunction. Using a CI of 2.2 L/min/M ^{2} as a threshold value, the AUROC was 0.66 (0.62 -0.71), 0.60 (0.56-0.63) and 0.59 (0.55-0.63) for EF, DD and RVD. In addition, using a threshold value of 33 ml/M^2/beat for SVI it was 0.53 (0.50-0.55), 0.50 (0.49-0.52) and 0.52 (0.50-0.54). Furthermore, the associated risk of death was higher with each form of moderate to severe CDF; 1.43 (CI 0.97-2.1, P=0.07) for EF <30%, 1.52 (1.10-2.11, P=0.01) for DD and 1.28 (0.9-1.8, P=0.2) for RVD.

Conclusion: Cardiac output is a poor measure of EF, DD and RVD. Patients with DD have a significantly increased risk of mortality, which has not been previously demonstrated. If cardiac dysfunction is suspected, an echo should be performed. Further studies are needed to determine if function-based treatment, directed by echo, is superior to catheter based care.

NOTES

Session: XB: Papers: 26-34
Paper 31: 3:40-4:00 PM

POST-RESUSCITATIVE HYPERCHLOREMIC METABOLIC ACIDOSIS IS ASSOCIATED WITH ACUTE KIDNEY INJURY

Dennis Y. Kim MD, Kyle Mock MD, Brian Nguyen MD, James Cunningham MD, Allison Forbes RN, Amy Kaji MD,Ph.D., Scott Bricker MD, Fred Bongard* MD, Angela Neville* MD, Brant Putnam* MD, David Plurad* MD, Harbor-UCLA Medical Center

Invited Discussant: Lena Napolitano, MD

Introduction: Hyperchloremic metabolic acidosis (HMA) is a common finding following the acute resuscitation of trauma patients. The clinical consequences of this metabolic abnormality are questionable. Recent data suggest an association between HMA and adverse outcomes including acute kidney injury (AKI). We hypothesized that HMA would be associated with an increased risk for early AKI in trauma patients.

Methods: We performed a 4 year retrospective analysis of all adult patients with an injury severity score (ISS) >15 admitted to the surgical intensive care unit for >48 hours at our Level 1 trauma center. Records were reviewed for arterial blood gases, serum chemistry, 24 hour admission fluid and blood product composition, volumes, and balance. AKI was defined according to the Acute Kidney Injury Network criteria. Patients with an HMA (pH <7.35; chloride >110; normal adjusted anion gap) were compared to patients without an acidosis. The primary outcome of interest was early AKI (developing ≤48 hours of admission). Multivariable logistic regression analysis was performed to identify independent predictors of AKI.

Results: Of 494 patients, 208 (42%) developed an HMA. Patients with an HMA had a higher ISS (28 ± 10 vs. 23 ± 8, $p<0.0002$), received more fluids in the first 24 hours (4L [IQR=3L-7L] vs. 2L [1L-3L], $p<0.0001$), and required an emergent operation (41% vs. 30%, $p=0.009$) and massive transfusion (7% vs. 3%, $p=0.04$) more frequently than patients without an acidosis. The overall incidence of AKI was 18%, which occurred more commonly in patients with an HMA (26% vs. 8% [OR=4.0; 95% CI 2.3-7.1, $p<0.0001$]). On multivariate logistic regression analysis, after controlling for confounding variables and known risk factors for AKI, HMA was found to be the only independent predictor of early AKI (OR=3.9; 95% CI=2.2-6.9, $p<0.0001$).

Conclusion: Post-resuscitative hyperchloremic metabolic acidosis is independently associated with the development of early AKI. Efforts to minimize the administration of chloride rich crystalloids may potentially reduce the incidence of AKI among critically injured patients.

NOTES

Session: XB: Papers: 26-34
Paper 32: 4:00-4:20 PM

NATIONAL ESTIMATES OF PREDICTORS OF OUTCOMES FOR EMERGENCY GENERAL SURGERY

Adil A. Shah MD, Adil H. Haider* MD,MPH, Diane A. Schwartz MD, Elliott R. Haut* MD, Syed N. Zafar MD,MPH, Zain G. Hashmi
MD, Eric B. Schneider Ph.D., Catherine G. Velopulos MD, Shahid Shafi* MD,MPH, Hasnain Zafar MBBS, FRCS, David T. Efron*MD, Johns Hopkins School of Medicine

Invited Discussant: Kristan Staudenmayer, MD

Introduction: Identification of predictors of complications and mortality have enabled improvements in outcomes for a variety of surgical conditions. However, similar work has yet to be done to identify factors affecting outcomes following emergency general surgery (EGS). Our objective was to determine the predictors of in-hospital complications and mortality among EGS patients.

Methods: The Nationwide Inpatient Sample (2003–2011) was queried for patients with conditions encompassing EGS as determined by the American Association for Surgery of Trauma (AAST) and categorized into the 24 defined EGS groups using ICD9 codes. Our primary outcomes of interest were the incidence of a major complication (defined as pneumonia, pulmonary emboli, urinary tract infections, myocardial infarction, sepsis or septic shock) and in-hospital mortality. Separate multivariate logistic regression analyses for complications and mortality were performed, to identify risk-factors of either outcome from the following domains: patient demographics (age, sex, insurance type, race, and income quartile), comorbidities, and hospital characteristics (location, teaching status and bed size).

Results: We analyzed 6,836,764 visits for EGS conditions. The average age was 58 years with a slight female preponderance (55%). Uninsured patients were more likely to die (OR[95% CI]:1.24[1.19- 1.29]), whereas patients in the highest income quartile had the least likelihood of mortality(OR[95% CI]:0.85[0.84-0.87]). Old age was an independent predictor of mortality for all EGS sub-diagnoses (table). The overall mortality rate was 1.8%. The overall complication rate was 15%. Of the patients who died, 62% suffered at least one major complication. Risk adjusted mortality rates were highest for patients requiring resuscitation for sepsis & shock. Patients with septic shock had a (OR[95%CI]:71.6[69.9-73.4]) had markedly high odds of death compared to those with no complications.

Table: Odds likelihood of mortality for the top 5 EGS conditions

	Odds Likelihood of Mortality [95% Confidence Interval]				
	Soft tissue pathologies	**Biliary pathology**	**Gastrointestinal bleeding**	**Colorectal pathology**	**Intestinal Obstruction**
Female	0.94 [0.89-0.98]	0.94 [0.89-0.98]	0.85 [0.83-0.88]	0.94 [0.89-1.01]	1.01 [0.98 -1.05]
Age Categories (years) (Referent: 16-25)					
• 66 - 75	12.0 [8.1-17.9]	9.1 [5.4-15.2]	3.6 [2.4-5.3]	7.1 [3.5-14.4]	3.2 [2.4-4.3]
• 76-85	18.2 [12.2-27.1	15.8 [9.4-26.5]	4.8 [3.2-7.2]	11.1 [5.5-22.5]	5.3 [4.0-7.1]
• >85	28.0 [18.8-41.7]	29.9 [17.8-51.1]	8.0 [5.4-11.8]	20.2 [10.1-41.0]	10.6 [7.9-14.2]
Race (Referent: White)					
• Black	1.09 [1.01-1.18]	1.01 [0.90-1.13]	0.84 [0.79-0.88]	0.89 [0.78-1.01]	1.05 [0.98-1.11]
• Hispanic	0.96 [0.87-1.05]	0.82 [0.73-0.91]	0.90 [0.84-0.96]	0.79 [0.69-0.92]	0.93 [0.85-1.01]
• Others/Unknown	1.10 [0.97-1.24]	1.07 [0.99-1.16]	0.96 [0.93-1.01]	0.94 [0.87-1.04]	1.06 [1.01-1.11]
Insurance Type (Referent: Private)					
• Government	1.15 [1.07-1.25]	1.34 [1.21-1.48]	1.01 [0.96-1.07]	1.45 [1.30-1.61]	1.10 [1.03-1.16]
• Uninsured	1.07[0.92-1.25]	1.17 [0.95-1.44]	1.24 [1.13-1.37]	1.05 [0.80-1.37]	1.19 [1.02-1.40]
Income Quartile (Referent: Lowest)					
• Highest	0.84 [0.78-0.90]	0.78 [0.71-0.86]	0.85 [0.81-0.89]	0.84 [0.80-0.97]	0.85 [0.80-0.89]
Teaching Hospital	0.94 [0.89-0.99]	1.00 [0.94-1.07]	1.00 [0.97-1.03]	1.00 [0.93-1.07]	1.04 [1.00-1.09]
Urban location	1.11 [1.03-1.19]	1.02 [0.94-1.11]	0.92 [0.88-0.96]	0.91 [0.82-1.00]	0.91 [0.86-0.96]

Conclusion: EGS patients have death patterns that can be discerned using administrative datasets. Understanding patterns of mortality and complications would allow for hospital benchmarking on similar lines to trauma surgery.

NOTES

Session: XB: Papers: 26-34
Paper 33: 4:20-4:40 PM

IMPLEMENTATION OF AN ACUTE CARE SURGERY SERVICE IN A COMMUNITY HOSPITAL: IMPACT ON HOSPITAL EFFICIENCY AND PATIENT OUTCOMES

Michael Kalina DO, Steven A. Johnson MD, Capital Health

Invited Discussant: Lewis Kaplan, MD

Introduction: Incorporating an acute care surgery service led by acute care surgeons into the management of critically ill surgical, emergency general surgery, and trauma patients has resulted in improved efficiency and patient outcomes at university hospitals and Level 1 trauma centers. Our goal was to determine if implementing an acute care surgery service led by acute care surgeons, entitled the Surgical Trauma and Acute Resuscitative Service (STARS), in a community hospital improved hospital efficiency and patient outcomes.

Methods: After IRB approval was obtained, 492 patient charts were retrospectively reviewed, 230 prior to implementation of the STARS, from August 2012 to March 2013, (pre-STARS or control group) as compared to 262 after implementation, from April 2013 to December 2013, (post-STARS or study group). Demographic data included age, gender, APACHE 2 score, and medical co-morbidities including diabetes (DM), hypertension (HTN), coronary artery disease (CAD), pulmonary disease (PD), liver failure (LF), and renal failure (RF). Hospital efficiency data included emergency department length of stay (ED-LOS), surgical intensive care unit length of stay (SICU-LOS), and hospital length of stay (H-LOS). Patient outcome data included mortality. Data were analyzed using Student's t-test, Chi-square, and multivariate logistic and linear regression analyses with bootstrapping. Statistical significance was denoted by $p \leq 0.05$.

Results: Of 492 patients, the average age was 64.1 ± 16.4 years, range of 18–98 years. 255 were males (51.83%) and 237 were females (48.17%). The average APACHE 2 score was 11.9 ± 5.8. There were no significant differences in age, APACHE 2 score, DM, CAD, PD, LF or RF. There was a significant difference with respect to HTN and gender ($p=0.02$, $p=0.001$ respectively). Mean ED-LOS was 9.7 ± 9.6 hrs, pre-STARS vs. 6.6 ± 4.5hrs, post-STARS. Mean SICU-LOS was 5.3 ± 9.6 days, pre-STARS vs. 3.5 ± 4.8 days, post-STARS. Mean H-LOS was 12.4 ± 12.7 days, pre-STARS vs. 11.4 ± 11.3 days, post-STARS. Mortality was 8.7%, pre-STARS vs. 4.9%, post–STARS. Adjusting for confounders revealed an average decrease in ED-LOS of 2.9 hours ($p=0.17$; 95% CI: -7.0, 1.2), an average decrease in SICU-LOS of 6.3 days ($p<0.001$; 95% CI: -9.3, -3.2), and an average decrease of H-LOS of 7.6 days ($p=0.001$; 95% CI: -12.1, -3.1) in the post-STARS group. Adjusting for confounders revealed that patients in the post-STARS group had an odds of survival that was 3.4 times greater than those in the pre-STARS group ($p=0.04$; 95% CI: 1.1, 10.7).

Conclusions: Implementation of the Surgical Trauma and Acute Resuscitative Service resulted in statistically significant decreases in surgical intensive care unit and hospital length of stay and increases in the odds of survival. Therefore, implementation of an acute care surgery service led by acute care surgeons in a community hospital improved hospital efficiency and improved patient outcomes.

NOTES

Session: XB: Papers: 26-34
Paper 34: 4:40-5:00 PM

COMPARISON OF ATRIOCAVAL SHUNTING WITH PERIHEPATIC PACKING VS PERIHEPATIC PACKING ALONE FOR RETROHEPATIC VENA CAVA INJURIES IN A SWINE MODEL

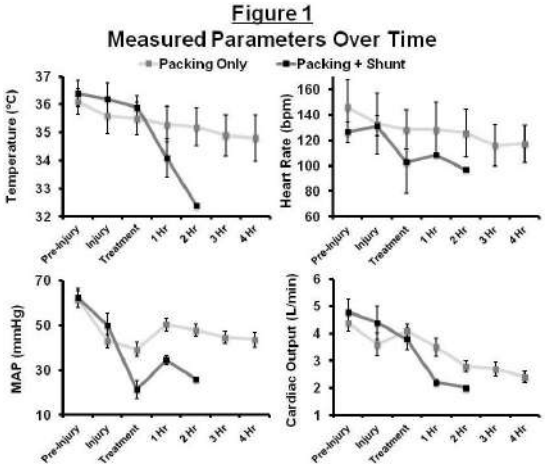
Joshua P. Hazelton DO, Rachel L. Choron MD, Gregory M. Dodson DO, Jeffrey A. Gerritsen MD, Sophia Khan MD, Kathryn E. VanOrden MD, Lisa M. Capano-Wehrle MPH, Ludmil V. Mitrev MD, Marc C. Torjman Ph.D., Roy D. Goldfarb Ph.D., Steven E. Ross* MD, Mark J. Seamon* MD, Cooper University Hospital

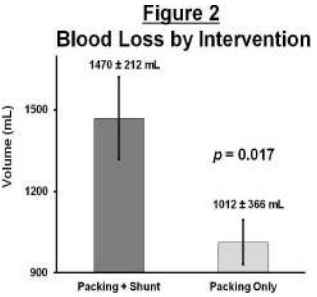
Invited Discussant: David Feliciano, MD

Introduction: Retrohepatic vena cava (RVC) injuries are technically challenging and often lethal. Atriocaval shunting has been promoted as a modality to control hemorrhage from RVC injuries, but evidence supporting its benefit is lacking. We hypothesized that atriocaval shunting with perihepatic packing (ACS) is more effective than perihepatic packing alone (PPA) in controlling hemorrhage after RVC injury.

Methods: After a survivable atriocaval shunting model was refined in 4 swine without RVC injury, 13 additional female Yorkshire swine were randomized into either ACS (n=7) or PPA (n=6) treatment arms following the creation of a standardized, 1.5 cm stab RVC injury. Hemodynamic parameters, intravenous fluid, and blood loss were recorded until mortality or euthanization after 4 hours. Data were analyzed using one-way ANOVA with repeated measures, the Student's t-test, and Mann-Whitney U test to compare differences between the 2 groups. A $p<0.05$ was set for statistical significance.

Results: Immediately before and after RVC injury, no difference in temperature, heart rate, mean arterial pressure, or cardiac output was detected (all $p>0.05$) between ACS and PPA groups. Although the RVC injury did affect measured parameters in PPA swine over time (Figure 1), hemodynamic compromise (Figure 1) and blood loss (Figure 2) were significantly greater in ACS than PPA swine. After a 45% greater mean blood loss, all ACS swine died (0% survival; mean survival time, 20 ± 34 min) while all 6 PPA swine survived (100%, $p=0.001$) the entire 4 hour study period.





Conclusion: Our results indicate that atriocaval shunting with perihepatic packing is ineffective compared to perihepatic packing alone in controlling hemorrhage after RVC injury. Further study is warranted to compare perihepatic packing to other surgical strategies for the intraoperative management of RVC injuries.

NOTES

FRIDAY, SEPTEMBER 12, 2014, 7:30 AM—8:00 AM

SESSION XI:
PANEL II: DEMYSTIFYING GOVERNMENT RESEARCH

LOCATION: GRAND BALLROOM SALONS G-L

PRESENTER:
Col. Todd E. Rasmussen, M.D.

Colonel USAF MC
Director US Combat Casualty Care Research Program
Fort Detrick, MD
Harris B. Shumacker, Jr. Professor of Surgery
The Uniformed Services University
Bethesda, MD
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SESSION XII:

QUICKSHOTS

FRIDAY, SEPTEMBER 12, 2014, 8:00 AM – 10:55 AM

GRAND BALLROOM SALONS G-L

MODERATOR: C. WILLIAM SCHWAB, M.D.

Session: XII: Quickshots
Paper 1: 8:01-8:07 am

Contemporary Management and Outcomes of Blunt Thoraic Aortic Injury: A multicenter retrospective study

Joseph J. DuBose* MD, Sam Leake BS, Megan Brenner MD, Tom Scalea* MD, Jason Pasley MD, Thomas O'Callaghan MD, Xian Luo-Owen MD,Ph.D., Marc D. Trust MD, Jennifer Mooney MD, Herb Phalen* MD, Omar Rivera MD, Kenji Inaba* MD, Martin Zielinski* MD, Gary Verycrusse* MD, Ali Azizzadeh MD, University of Texas Health Science Center-Houston

Invited Discussant: Michael Sise, MD

Introduction: Blunt thoracic aortic injuries (BTAI) comprise a spectrum from intimal tear to rupture; yet optimal management and ultimate outcome has not been clearly established.

Methods: Retrospective multicenter study of BTAI from Jan 2008 to Dec 2013. Demographics, diagnosis, treatment and in-hospital outcomes were analyzed.

Results: Nine ACS Level I trauma centers contributed data from 453 patients with BTAI. After exclusion of patients expiring prior to imaging (58), and transfers (13), 382 patients with imaging diagnosis were available for analysis (SVS Grade 1 = 94, Grade 2 = 68, Grade 3 = 192, Grade 4 =28). Hypotension (SBP<90 mm Hg) was present on admission in 56 (14.7%). CTA alone was utilized for diagnosis in 94.5% of patients. Non-operative management (NOM) was selected in 32%, with only 2 in-hospital failures (Grade 1, Grade 4) requiring endovascular (TEVAR) salvage. Open repair (OR) was successfully completed in 61 (16%). TEVAR was conducted in 198 (52%) at a mean of 3.1 days after injury; with 41% of these undergoing left SCA coverage. Complications following TEVAR included endograft malposition (6, 3.0%) endoleak (5, 2.5%), paralysis (1, 0.5%) and stroke (2, 1.0%). Six TEVAR failures were treated by repeat TEVAR (2) or OR (4). Overall in-hospital mortality was 18.8% (NOM 34.4%, OR 19.7%, TEVAR 8.6%), with an aortic-related mortality of 6.5% (NOM 9.8%, OR 13.1%, TEVAR 2.5%)[Grade 1 = 0%, Grade 2 = 2.9%, Grade 3 = 5.2%, Grade 4 = 46.4%]. The majority of aortic-related deaths (18/25) occurred prior to the opportunity for repair. Independent predictors of aortic-related mortality among all BTAI patients were advanced chest AIS, greater SVS Grade and greater ISS; with TEVAR proving protective (p = 0.03, OR 0.25 [0.07-0.87].

Conclusions: Based on in-hospital outcomes, failures and aortic-related mortality of NOM following BTAI SVS Grade –1-3 injuries are rare. Early complications of TEVAR have substantially decreased relative to prior reports. Prospective long-term follow-up data is required to better refine indications for intervention following BTAI.

NOTES

Session: XII: Quickshots
Paper 2: 8:08-8:14 am

EARLY WHOLE BLOOD AUTO-TRANSFUSION: AN UNDEFINED PRACTICE IN CIVILIAN TRAUMA

Bellal Joseph* MD, Kenji Inaba* MD, Viraj Pandit MD, Stefano Siboni MD, Gary Vercruysse* MD, Narong Kulvatunyou* MD, Andrew Tang MD, Terence O'Keeffe* MD, MBChB, Donald J. Green* MD, Lynn Gries MD, Randall S. Friese* MD, Demetrios Demetriades* MD, Peter Rhee* MD,MPH, University of Arizona - Tucson

Invited Discussant: Juan Duchesne, MD

Introduction: The changing transfusion practices advocate early and liberal use of blood products for resuscitation in trauma patients. However; the use of whole blood resuscitation in civilian trauma practice remains a rare phenomenon. The aim of this study was to assess outcomes in trauma patients receiving whole blood auto-transfusion (AT).

Methods: This 6 year multi-institutional retrospective study included all trauma patients requiring transfusions on presentation in the emergency department. Patients that received AT were matched to patients that did not receive AT (No-AT) using propensity score matching in a 1:1 ratio for demographics, injury severity score (ISS), systolic blood pressure (SBP), and international normalized ratio (INR). AT was defined as transfusion of blood from patient's chest and/or abdominal cavity. Outcome measures were: in-hospital complications and mortality. In-hospital complications were defined as acute respiratory distress syndrome (ARDS), sepsis, disseminated intravascular coagulation (DIC), renal insufficiency, and transfusion related acute lung injury (TRALI).

Results: A total of 272 patients (AT: 136, No-AT: 136) were included. There was no difference in age (p=0.81), injury severity (p=0.56), head AIS (p=0.42), SBP (p=0.48), and INR (p=0.21) between the two groups. AT was initiated in the emergency department (ED) in 48.5% patients while remaining (n=70) received AT in both ED and operating room.

Table 1: Outcomes

Variables	AT (n=136)	No-AT (n=136)	*p*
PRBC	10.3±7.3	12.1±9.7	0.03
Platelets	5.2±4.2	7.9±5.4	0.04
FFP	6.1±3.9	8.2±6.5	0.24
24 post admission INR	1.3±1.1	1.1±1	0.31
In-hospital Complications	12.5%	10.3%	0.61
Mortality	18.3%	15.4%	0.51
Cost of Therapy	8,794±6,531	10,427±8,621	0.03
Hospital Cost	42,156±40,981	43,963±40,528	0.04

Conclusion: Early whole blood AT is a safe and effective practice in severely injured trauma patients. Auto-transfusion resulted in lower packed red blood cell and platelet transfusions as well as lower costs without increasing complications or mortality.

NOTES

Session: XII: Quickshots
Paper 3: 8:15-8:21 am

DEFINING THE EXCESS MORBIDITY AND MORTALITY ATTRIBUTABLE TO EMERGENCY GENERAL SURGERY

Allan B. Peetz MD, Ali Salim* MD, Zara Cooper MD, Edward Kelly* MD, Reza Askari MD, Jonathan Gates MBA,MD, Gally Reznor MS, Joaquim Havens MD, Brigham and Womens Hospital

Invited Discussant: Shahid Shafi, MD

Introduction: Emergency general surgery (EGS) carries a disproportionate burden of risk from medical errors, complications and death compared to non-emergency general surgery (NEGS). Previous studies have been limited by patient and procedure heterogeneity, but suggest worse outcome due to preoperative risk factors. We hypothesize that the disproportionate morbidity and mortality in EGS patients are not fully explained by these factors but are due to the EGS itself.

Methods: We retrospectively analyzed data from patients in the American College of Surgery National Surgical Quality Improvement Program (ACS-NSQIP) database that underwent one of 14 selected procedures between 2008 and 2013. The procedures represented general and vascular surgery operations common to both emergency and elective settings including; visceral resection, abdominal wall hernia repair, ileo-mesenteric bypass, and aortic reconstruction. Patients were stratified based on emergency status. The primary outcome was death within 30 days of operation. Secondary outcomes included post-operative complications. 42 preoperative variables were analyzed from the ACS-NSQIP preoperative risk assessment which includes demographic data, functional and dependent status, smoking and alcohol history, comorbidities, presence of sepsis, acute renal failure, revascularization/amputation, recent dialysis, impaired sensorium, pneumonia, recent ventilator dependence, steroid use, bleeding disorders, preoperative blood transfusions, laboratory data, and American Society of Anesthesia classification. Additionally, wound class and 23 post-operative outcomes were analyzed. This included the ACS-NSQIP post-operative occurrences (wound, respiratory, urinary tract, central nervous system, cardiac, hematologic, and septic). A Chi-square test was used to compare categorical variables and the Wilcoxon rank sum test for continuous variables. Multivariate logistic regression analysis was performed to identify independent risk factors for mortality and complications.

Results: Of 66,665 patients, 24,068 underwent emergency procedures and 42,597 were non emergent. Death occurred in 12.5% of EGS patients and 2.7% of NEGS patients ($p<.0001$). Post-operative complications occurred in 48.2% of EGS patients and 27.5% of NEGS patients ($p<.0001$). When preoperative risk factors, type of procedure, and post-operative complications were controlled, EGS was independently associated with death (Odds Ratio (OR) 1.13 $p<.0001$) and complications (OR 1.09 $p<.0001$). EGS was also independently associated with wound (OR 1.03 $p=0.07$) and respiratory complications (OR 1.17 $p<.0001$).

Conclusion: EGS is an independent risk factor for death and post-operative complication. The disproportionate morbidity and mortality observed in EGS is independent of patient factors, type of operation, and type of post-operative complication. Research seeking to improve EGS patient outcomes should include investigating the cause of this excess burden of risk with special consideration for identifying modifiable factors.

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Session: XII: Quickshots
Paper 4: 8:22-8:28 am

INDUCING ACUTE TRAUMATIC COAGULOPATHY IN VITRO

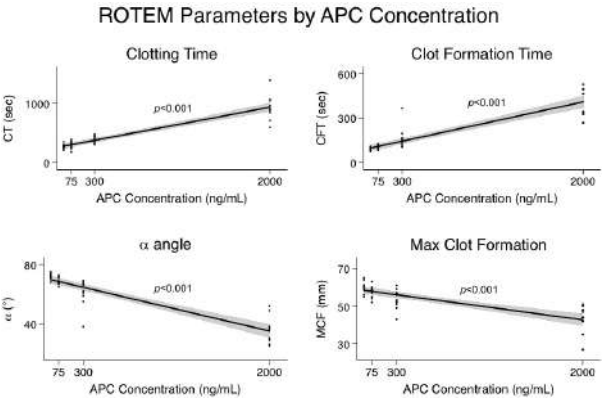
Benjamin M. Howard MD,MPH, Lucy Z. Kornblith MD, Christopher K. Cheung BA, Matthew E. Kutcher MD, Ryan F. Vilardi MS, Byron Miyazawa BS, Mitchell J. Cohen* MD, San Francisco General Hospital; University Of California, San Francisco

Invited Discussant: Walter Biffl, MD

Introduction: Nearly one third of critically injured patients present with acute traumatic coagulopathy, independent of iatrogenic causes. This coagulopathy has been associated with shock and tissue injury, and may be mediated via activation of the protein C pathway. Patients with acute traumatic coagulopathy have prolonged PT and PTT, and decreased activity of factors V and VIII; they are also hypocoagulable by thromboelastometry (ROTEM) and other viscoelastic assays. To test the etiology of this phenomenon, we hypothesized that such coagulopathy could be induced *in vitro* in healthy human blood with the addition of activated protein C (APC).

Methods: Whole blood was collected from ten healthy human subjects, and was "spiked" with increasing concentrations of purified human APC (control, 75, 300, 2000 ng/mL). PT/PTT, factor activity assays, and ROTEM were performed on each sample. Statistical differences were assessed across and between APC concentration groups, and linear regression was performed to assess the association of APC concentration with PT/PTT, factor activity, and ROTEM parameters.

Results: In all subjects, increasing concentrations of APC produced ROTEM tracings consistent with traumatic coagulopathy. ROTEM EXTEM parameters differed significantly by APC concentration, with stepwise prolongation of clotting time (CT) and clot formation time (CFT), decreased alpha angle (α), and reduced maximum clot firmness (MCF). Significant prolongation of PT and PTT and decreased activity o f factors V and VIII were observed at higher APC concentrations. Linear regression demonstrated that for every 100 ng/mL increase in APC concentration, CT prolonged by 32.9 seconds, CFT prolonged by 22.2 seconds, α decreased by 1.7 degrees, and MCF decreased by 0.8 mm (all $p<0.001$; see Figure); PTT was prolonged by 2.1 seconds, factor V activity decreased by 1%, and factor VIII activity decreased by 2.7% (all $p<0.01$).



Conclusion: In this study, we reproduced a phenotype of acute traumatic coagulopathy in healthy blood by the *in vitro* addition of APC alone, as evidenced by viscoelastic measures and confirmed by conventional coagulation assays and factor activity. This lends further mechanistic insight to the etiology of coagulation abnormalities in trauma, supporting the central role of the protein C pathway. Our findings also represent a novel model for future investigations in the diagnosis and treatment of traumatic coagulopathy.

NOTES

Session: XII: Quickshots
Paper 5: 8:29-8:35 am

INHALED, NEBULIZED SODIUM NITRITE PROTECTS AGAINST TRAUMA/HEMORRHAGIC SHOCK INDUCED TISSUE INJURY AND INFLAMMATION

Benjamin Kautza MD, Daniel Escobar MD, Ana Botero MD, Hakon Haugaa MD, Michael Pinsky MD, Sruti Shiva Ph.D., Hernando Gomez MD, Andrew Peitzman* MD, Brian Zukerbraun MD, University of Pittsburgh

Invited Discussant: Rosemary Kozar, MD, PhD

Introduction:
Trauma and hemorrhage results in tissue injury and inflammation that contributes to morbidity and mortality. Organ injury from trauma and hemorrhage occurs in part from the development of cellular shock and oxidative injury. Endogenous adaptive responses to tissue hypoxia are helping to guide the development of resuscitative adjuncts to limit tissue injury, including investigations into nitrite/nitrite reductase/nitric oxide signaling pathway. The purpose of these investigations was to test the hypothesis that inhaled, nebulized sodium nitrite protects against the development of shock and tissue injury in models of trauma and hemorrhagic shock.

Methods:
Male C57BL/6 mice were randomized to sham surgery or soft tissue injury+hemorrhage to 25mmg Hg for 2 hours followed by resuscitation with lactated ringers (2X volume of shed blood). In each group, some mice were further treated with inhaled nebulized sodium nitrite ($NaNO_2$; 30 mg in 5ml into environment over 20 minutes) or vehicle at the time of resuscitation. Mice were sacrificed 4 hours after resuscitation. Yorkshire Durock pigs (30-35kg) underwent vascular cannulation and were randomized to anesthesia only or hemorrhage to 30mmHg and bled to maintain a pressure between 30-40 for 90 minutes. Shocked pigs were randomized to receive vehicle or inhaled nebulized $NaNO_2$ 11mg in 2.5 mL PBS. Pigs were sacrificed 4 hours after resuscitation. Hemodynamics, blood, and microdialysis specimens were collected throughout.

Results:
In a murine model, trauma/hemorrhage resulted in organ injury and inflammation as demonstrated by increased serum ALT, Cystatin C and IL-6. Nebulized nitrite limited these responses ($P<0.05$). Additionally, nitrite limited oxidant injury as determined by lipid peroxidation. In a porcine model of hemorrhagic shock, nebulized nitrite was associated with significant decreases in blood, muscle, and peritoneal fluid lactate concentrations ($P<0.05$), and significant decreases in glycerol release into peritoneal fluid ($P<0.05$). Additionally hemorrhage resulted in mitochondrial injury as determined by decreased respiratory control ratio in both muscle and platelets. Nitrite prevented this injury. Furthermore, hemorrhage induced platelet activation was limited by nitrite.

Conclusion:
Resuscitation adjuncts to limit the development of shock, oxidant stress, and thus tissue injury hold the promise of improving outcomes. Nebulized NaNO2 was a protective resuscitative adjunct in both murine and porcine models of hemorrhagic shock and resuscitation, and may prove useful in human trauma and hemorrhage, however further investigations are warranted.

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Session: XII: Quickshots
Paper 6: 8:36-8:42 am

TWO YEAR CESSATION OF RESIDENT TEAMWORK TRAINING IMPACTS TRAUMA RESUSCITATION PERFORMANCE AND EFFICIENCY

Charles T. Harris MD, Ellen Harvey RN, DNP, Andrea Wright RN, MSN, Dallas Taylor BS,RN, Carol Gilbert* MD, Bryan Collier* DO, Carilion Roanoke Memorial Hospital

Invited Discussant: Joseph Galante, MD

Introduction: Prior studies suggest structured teamwork training improves trauma team resuscitation performance and outcomes. This study examines the impact of *cessation* of resident training as a component of a multidisciplinary TeamSTEPPS simulation-based training program on previous performance gains noted in the trauma resuscitation setting.

Methods: This prospective, observational follow-up study of trauma team resuscitation performance was conducted in a Level 1 trauma center. Trained evaluators scored team performance during trauma resuscitations using the validated Trauma Team Performance Observation Tool (TPOT). Possible scores ranged 21 to 105. Efficiency (minutes) data were gathered from the trauma registry for ED dwell time, time to FAST, and time to CT scan during the same time period TPOT scores were collected. Data collected two years after cessation of training were compared to pre-training, post training, and one year post ongoing training periods. For each outcome variable, the data were rank-transformed and analyzed using the analysis of variance. If a significant overall group effect was detected ($p < 0.05$), a Dunnett's Multiple Range Test was performed.

Results: TPOT scores increased significantly from pre-training (N=33) to post training (N=40) (62.2 to 74.7, $p<.0001$) and sustained one year later (N=32) (72.5, $p<.0001$) with ongoing training. TPOT scores at 2 years cessation (N=43) declined close to pre-training levels (66.6,$p=.0735$) and were significantly lower than the scores at post training and one year post ongoing training ($p<0.05$). A decline in efficiency in minutes (post training vs. 2 year post cessation) was also noted in ED dwell time (190 vs. 323; $p<0.0001$), time to FAST (10.11 vs. 14.03; $p<0.0001$), and time to CT scan (26.5 vs. 29.2; $p=.0008$).

Conclusion: A 2 year cessation of resident teamwork training is associated with deterioration of trauma team performance scores, increased times to critical radiologic trauma exams (FAST and CT), and overall prolonged ED dwell times placing trauma patient outcomes at risk.

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Session: XII: Quickshots
Paper 7: 8:43-8:49 am

OPERATIVE VERSUS NON-OPERATIVE MANAGEMENT OF MULTIPLE RIB FRACTURES

Mauricio Velasquez MD, Carlos Ordonez* MD, Michael Parra* MD, Andres Dominguez MD, Juan C. Puyana* MD, Fundacion Valle Del Lili

Invited Discussant: Marc de Moya, MD

Introduction: Multiple ribs fractures (MRF) are associated with high rates of morbidity and mortality. Our goal was to compare outcomes between the traditional non-operative management and that of a comprehensive surgical approach.

	Control group	Operative group	p_value
n	20	20	
Age, *	44.5 (36-54.5)	51.1 (41-63)	0,2153
Número of fractured ribs*	5 (4-6.5)	5 (4-8)	0,065
Ventilator days*	10 (6-16)	2 (1-3)	0,000
ICULOS*	8 (6-10.5)	4.5 (1-8)	0,0199
HLOS*	16 (11-22)	6 (4-10)	0,0001
Respiratory morbidity, n(%)	13 (65)	5 (16.7)	0,000
CTLOS*	9 (7-12)	3 (2-5.5)	0,000
ISS*	13 (9-17)	9 (9-16)	0,357
CHEST AIS	3	3	
Mortality,n(%)	2 (10)	0 (0)	0,155

*** Median (IQR)**

Methods: Retrospective analysis of adult MRF injuries from June 2011 to December 2013 at a regional level I trauma center. Patients with concurrent severe closed head injuries (Glasgow Coma Score <8) were excluded. Data was collected prospectively in the operative group and was compared to a historic non-operative control group matched for age, ISS and number of ribs fractured from the same institution. Outcome variables included were duration of mechanical ventilation (DMV), intensive care unit length of stay (ICULOS), hospital length of stay (HLOS), chest tube length of stay (CTLOS), incidence of pneumonia and mortality. Surgical stabilization of the rib fractures was performed with the Strasbourg Thoracic Osteosyntheses System (STRATOS [MedXpert GmbH, Heitersheim, Germany]). This system consists of titanium screw less clips which facilitates a less invasive surgical fixation and stabilization of the rib fractures.

Results:MRF were identified in a total of 40 patients and evenly divided among both groups. The operative group demonstrated a significant reduction in DMV (median of 10 (6-16) vs. 2 (1-3) [p<0,000]), ICULOS (median of 8 (6-10,5) vs. 4,5 (1-8) [p<0,0199]), HLOS (median of 16 (11-22) vs. 6 (4-10) [p<0,0001]), CTLOS (median of 9 (7-12) vs. 3 (2-5,5) [p<0,000]), pneumonia rates (65% vs. 16.7% [p<0,000]), and overall mortality (2 vs. 0). Death was due to pulmonary sepsis in one case and ARDS in the other

Conclusion: As compared with non-operative therapy, operative fixation of MRF is associated with reductions in DMV, HLOS, CTLOS and overall pneumonia rates.;

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Session: XII: Quickshots
Paper 8: 8:50-8:56 am

A COMPARISON OF DIAGNOSTIC PERITONEAL LAVAGE TO COMPUTED TOMOGRAPHY IN THE DIAGNOSIS OF THORACO-ABDOMINAL STAB WOUNDS

Reza Salabat MD, Andrew Dennis DO, John Kubasiak MD, Adelaide Kaczynski BS, Samuel Kingsley MD, Elizabeth Gwinn MD, Kimberly Joseph* MD, Frederic Starr MD, Dorion Wiley MD, Faran Bokhari MD, Kimberly Nagy* MD, Cook County Hospital

Invited Discussant: Leonard Weireter, Jr., MD

Introduction: Studies have reported 27% occult diaphragmatic injury due to stab wounds (SW) to the thoraco-abdomen (TA), with nearly 36% mortality if found incarcerated. The computed tomography (CT) is a widely used initial diagnostic tool, but it lacks the sensitivity to detect diaphragm injuries. We assessed the application of diagnostic peritoneal lavage (DPL) in determining missed abdominal and/or diaphragmatic injuries by CT and the ability to predict a therapeutic laparotomy in patients with SW to TA region.
Methods: Data was collected prospectively from 2006 to 2011. Inclusion criteria consisted of hemodynamic stability and patients who underwent both CT and DPL. Patients were excluded if they were hemodynamically unstable, had evisceration or peritonitis, or had only one of the above tests. A positive DPL was defined as RBCs>10,000/mm3. A positive CT was defined as free air, intra-abdominal organ injury/laceration or hematoma adjacent to an organ. Sensitivity (SN), specificity (SP) were calculated independently for DPL and CT. A therapeutic exploratory laparotomy was defined as presence of an injury, which required intervention.
Results: During the study period, there were 192 patients with SW to TA of which 58 met our criteria. DPL resulted in 12 true positive (TP), 0 false positive (FP), 1 false negative (FN) and 45 true negative (TN) results for detection of injury, whereas CT scan was shown to have 7 TP, 0 FP, 6 FN, and 45 TN tests. Sensitivity (SN) was demonstrated to be 92% vs. 53% for DPL vs. CT, respectively. Specificity was 100% for both groups. When only therapeutic lapatotomy was considered, we found 12 TP, 0 FP, 0 FN, and 45 TN DPL results, compared to CT scan which had 6 TP, 0 FP, 6 FN, and 45 TN tests. Sensitivity was demonstrated to be 100 vs. 50% for DPL vs. CT, respectively. Specificity was 100% for both groups. Notably, CT missed 1 colon, 2 splenic, 3 hepatic, and 7 diaphragmatic injuries. Four of the diaphragmatic injuries were found on the left anterior and lateral aspects
Conclusion: DPL was found to be superior to CT both for detection of intra-abdominal organ injury and for prediction of therapeutic exploratory laparotomy. CT missed all diaphragmatic injuries as well as 6 other intra-abdominal injuries. We conclude that CT, despite improvements in technology, remains a poor test for evaluating the diaphragm after penetrating TA trauma. When attempting to rule out potentially operative injuries to the diaphragm, DPL continues to be a useful test that should remain in the arsenal of every trauma surgeon.

NOTES

Session: XII: Quickshots
Paper 9: 8:57-9:03 am

THE IMPACT OF IMAGE-SHARING ON THE EVALUATION OF TRAUMA TRANSFER PATIENTS IN A RURAL TRAUMA SYSTEM

Tanveer Zamani MD, FRCSI, Chris Wargo MSN, James Dove BA, Jeffery L. Wild MD, Kenneth A. Widom MD, DiAnne Leonard* MD, FACS, Susan M. Baro DO, Aalpen Patel MD, Kim L. Roadarmel Geisinger Health System

Invited Discussant: Sharon Henry, MD

Introduction: Many rural trauma centers receive patients that are first evaluated at local hospitals (LH). LH transferring patients to regional trauma centers (TC) often obtain computed tomography (CT) scans to diagnose injuries and justify transfer. Many studies have shown that these studies are often repeated for a variety of reasons, including inadequate technique of acquisition of images and software incompatibilities. Repeating these studies adds a tremendous cost and radiation exposure to trauma patients. This study was performed to determine how the implementation of image-sharing systems impacted the rate of repeat CT scans during interfacility transfers at a rural Level I trauma center.

Methods: All trauma alert patients age greater than 15 years, transferred with prior CT imaging to a rural Level I trauma center from January 1, 2009 until December 31, 2012 were retrospectively reviewed. Data abstracted included CT scans performed at LH and CT scans repeated at the TC. The data was further divided into three time periods: pre-image sharing implementation (2009), peri-implementation (2010-2011), and post-implementation (2012). We compared the incidence of repeat CT scans with the implementation of image sharing system during these periods by using Cochran-Armitage trend test. We used Head CT (HCT) as a model study tool to review the impact of imaging on the cost and radiation exposure.

Results: During the study period, 1259 patients were transferred to the Level I TC. Of the total study population, 1003 (79.6%) patients underwent CT imaging prior to transfer. During this 4-year period, 554 (55.2%) patients underwent repeat CT imaging. When compared to the pre-implementation period, where 126/180 (70%) patients with prior imaging had CT scans repeated and 209/439 (47.6%) pre-transfer CT scans were repeated, in the post-implementation period, the number of patients with prior imaging who had repeat CT scans was decreased to 131/294 (44.6%) and only 201/751 (26.8%) pre-transfer CT scans were repeated which has p value of <.001. This saved $784,531.00 in 2012, as compared to $335,397.00 in 2009. Focusing on HCT, in the pre-implementation period, 142 out of 180 patients with pre-transfer imaging had HCT available, out of which 82/142 (57.7%) patients had HCT repeated, 60/142 (42.2%) had no repeat HCT; saving $70,020.00 and preventing patients from further radiation exposure with the saving of total 4,392 mGy of radiation dose. In the post-implementation period, 228 out of 294 patients with prior imaging on transfer had HCT performed, out of which only 69/228 (30.3%) patients had HCT repeated while 159/228 (69.7%) did not have HCT repeated. This saved $185,553.00, prevented 69.7% of the patients from repeat radiation exposure and saved total 11,639 mGy of radiation dose consumption which signifies p value of <0.001.

Conclusion: Seventy-nine percent of interfacility transfer patients underwent CT scans prior to transfer. Since the implementation of imaging-sharing systems, the number of repeat CT scans has significantly decreased. This has resulted in increased cost saving and decreased radiation exposure to the patient. Image-sharing is an innovative practice of quality improvement and a safety measure in the rural trauma system.

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Session: XII: Quickshots
Paper 10: 9:04-9:10 am

LEVEL I ACADEMIC TRAUMA CENTER INTEGRATION AS A MODEL FOR SUSTAINING COMBAT SURGICAL SKILLS: THE RIGHT SURGEON IN THE RIGHT PLACE FOR THE RIGHT TIME

Rachel A. Hight MD, Sean P. Martin MD, Edgardo S. Salcedo MD, Ho H. Phan MD, Garth H. Utter* MD, Christine S. Cocanour* MD, Joseph M. Galante* MD, University of California, Davis

Invited Discussant: Nicholas Namias, MD

Introduction: As North Atlantic Treaty Organization (NATO) countries begin troop withdrawal from Afghanistan, military medicine needs programs for combat surgeons to retain the required knowledge and surgical skills. Existing programs for each military branch at various Level I academic trauma centers deliver pre-deployment training and provide a robust trauma experience for deploying surgeons. Outside of these successful programs there is no system-wide mechanism for non-deploying military surgeons to care for a high volume of critically ill trauma patients on a regular basis in an educational environment that promotes continued professional development.

Methods: We describe our military/university relationship for integrating military surgeons into a civilian trauma practice. We characterized the Level I practice using the number of trauma resuscitations, operative trauma/acute care surgery procedures, number of work shifts, operative density (defined as the ratio of operative procedures/days worked) and frequency of educational conferences. The same parameters were collected from two NATO Role III hospitals in Afghanistan during the peak of Operation Enduring Freedom (OEF). Data for two civilian Level II trauma centers, two civilian Level III trauma centers, and a Continental United States (CONUS) Military Treatment Facility (MTF) without trauma designation were collected.

Results: The number of trauma resuscitations, operative density, and educational conferences are shown in the table for the Level I trauma center compared to the different institutions. Civilian center trauma resuscitations and operative density were highest at the Level I trauma center and were only slightly lower than what was seen in Afghanistan. Level II and III trauma centers had lower numbers for both. The Level I trauma center provided the most frequent educational opportunities.

Facility Type	Trauma Designation and Number of Facilities Evaluated	Trauma Resuscitations per year	Average Number of 24hr Shifts per month	Operative Density (procedures/day)	Dedicated Educational Conferences
Civilian trauma centers	Level I - one	3000	6-10	3.53	Daily
	Level II - two	2100	2-3	1.92	Weekly
	Level III - two	600	2-3	0.83	Monthly
Military treatment facilities (MTF)	MTF, no trauma designation, CONUS - one	N/A	Clinic practice with a surgeon on-call daily	2.15	Weekly
	NATO Role III MTF, Afghanistan - two	3600	15-30	4.68	Weekly

Table: Civilian trauma center characteristics contrasted with CONUS MTF and NATO trauma centers in Afghanistan

Conclusions: In a level I academic trauma center integrated program, military and civilian surgeons have the same clinical and educational responsibilities: rounding and operating, managing critical care patients, covering trauma/acute care surgery call, and mentoring surgery residents in an integrated residency program. The Level I trauma center experience most closely mimics the combat surgeon experience seen at NATO Role III hospitals in Afghanistan compared with other civilian trauma centers. At high-volume Level I trauma centers, military surgeons will have a comprehensive trauma practice including dedicated educational opportunities. We recommend integrated programs with Level I academic trauma centers as the primary mechanism for sustaining military combat surgical skills for in the future.

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Session: XII: Quickshots
Paper 11: 9:11-9:17 am

RISK ASSESSMENT MODELS FOR PREDICTING VENOUS THROMBOEMBOLISM IN TRAUMA PATIENTS ARE NOT ADEQUATE

Ashley Zander DO, Jan-Michael Van Gent DO, Erik Olson MD, Steven Shackford* MD, Jayraan Badiee MPH, Beth Sise RN, Michael Sise* MD, Scripps Mercy Hospital Trauma Service

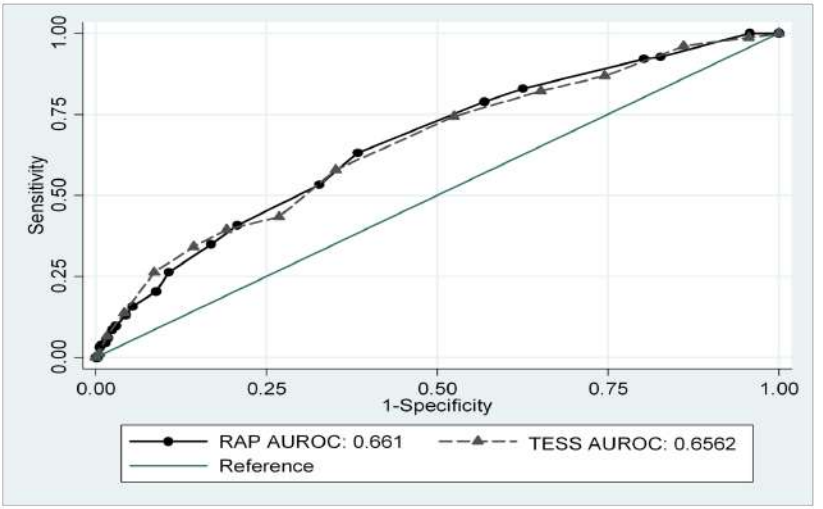
Invited Discussant: Avery Nathens, MD, PhD, MPH

Introduction: Venous thromboembolism (VTE) risk assessment models exist to stratify patients at risk for VTE and guide surveillance and prophylaxis. We evaluated two models developed specifically for trauma patients: the Trauma Embolic Scoring System (TESS) and the Risk Assessment Profile (RAP).

Methods: Clinical and demographic data of patients admitted between 7/2006-12/2011 who underwent surveillance lower extremity duplex ultrasound (LEDU) were recorded. Patients received prophylaxis according to American College of Chest Physicians guidelines. TESS and RAP scores were calculated retrospectively and compared between patients with VTE and patients without VTE. High risk, as defined by the models, is TESS≥7 and RAP≥5.

Results: 2,868 patients received surveillance LEDU. TESS was calculated for 2,140 patients; 215 developed VTE, 110 (51%) of whom had TESS<7. The sensitivity and specificity at a cut point of 7 were 49% and 72%, respectively. RAP was calculated for 1,505 patients; 152 developed VTE, 26 (17%) of whom had RAP<5. The sensitivity and specificity at a cut point of 5 were 83% and 37%, respectively. The receiver operating characteristic curves for the models were similar (Fig.).

Conclusion: A clinically significant number of patients who developed VTE were classified as low-risk by both TESS and RAP. The indications for VTE surveillance and chemoprophylaxis should not be based exclusively on these scores. These results suggest that additional variables should be sought to improve risk assessment for VTE following trauma.



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Session: XII: Quickshots
Paper 12: 9:18-9:24 am

IMPACT OF SOCIO-ECONOMIC STATUS ON HOSPITAL LENGTH OF STAY FOLLOWING INJURY: A MULTICENTER COHORT STUDY

Lynne Moore Ph.D., Brahim Cisse MSc, Brice L. Batomen MSc, Gilles Bourgeois MD, Laval University

Invited Discussant: Karen Brasel, MD, MPH

Introduction: In the USA, around 1.9 million injury hospitalizations are recorded annually with 19.5$ billion in acute care hospital costs alone, representing 7% of total hospital costs. Several studies have identified predictors of hospital length of stay (LOS) in trauma patients but despite the documented impact of socio-economic status (SES) on LOS in other patient populations such as cardiovascular disease, the impact of SES on LOS in trauma patients in unknown. This study aimed to examine the effect of SES on acute care LOS following injury in a setting with universal health insurance.

Methods: We conducted a multicenter cohort study involving the 56 adult trauma centers in a Canadian provincial trauma system using trauma registry and hospital discharge data collected between 2005 and 2012. SES was determined using ecological indices of material and social deprivation. Geometric Mean Ratios (GMR) and 95% confidence intervals (CI) adjusted for age, gender, comorbidities, injury severity, body region of the most severe injury, and the number of admissions in the 12 months prior to injury were generated using multivariate linear regression with a correction for hospital clusters. Analyses were stratified for age, discharge destination, and payor status.

Results: The cohort consisted of 52,122 patients with a mean LOS of 13.4 days. Patients in the highest quintile of material deprivation had a mean length of stay 4% higher than those in the lower quintile (95% CI 2%-6%). Patients in the highest quintile of social deprivation had a mean length of stay 5% higher than those in the lower quintile (95% CI 3%-7%). Patients in the highest quintile of both social and material deprivation had a mean length of stay 12% higher than those in the lowest quintile for both measures of SES (95% CI 7%- 18%).

Conclusion: Patients admitted for traumatic injury who suffer from high social and material deprivation have a longer acute care LOS in a universal-access health care system. This research suggests that interventions to facilitate access to post-discharge care for these patients may improve the efficiency of acute trauma care.

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Session: XII: Quickshots
Paper 13: 9:25-9:31 am

REPAIR VERSUS LIGATION OF MAJOR VENOUS INJURY AFTER PENETRATING TRAUMA: IS THERE A DIFFERENCE IN DEVELOPMENT OF PULMONARY EMBOLISM?

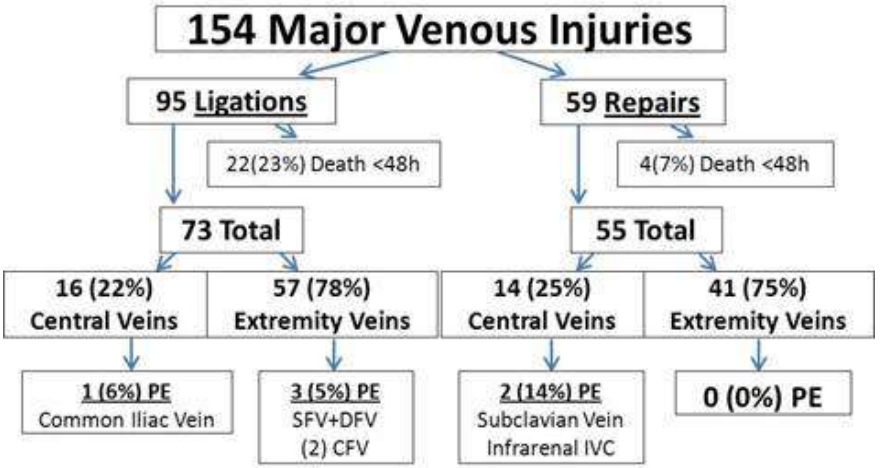
Casey J. Allen MD, Albert Hsu MD, Evan Valle MD, Nicholas Namias* MD, Alan Livingstone MD, Edward Lineen* MD, Kenneth G. Proctor* MD, University Of Miami Miller School Of Medicine

Invited Discussant: Ronald Simon, MD

Introduction: With improved rates of limb salvage and resolution of edema and phlegmasia, guidelines recommend repair of major venous injury (MVI) from penetrating trauma if the patient is hemodynamically stable. However, few studies have eveluated the rate of venous thrombotic complications following major venous injury from penetrating trauma. Repair could provide a nidus for thrombosis with subsequent propagation to the lungs; would ligation prevent this morbid complication? Our study evaluates the rate of pulmonary embolism (PE) between patients who underwent either a repair or ligation.

Methods: All MVI from penetrating trauma requiring an operation from 2003-2012 were reviewed. Information obtained includes vessels injured, repair versus ligation, presence of deep vein thrombosis (DVT) prophylaxis, and mortality. The development of DVT was obtained from venous duplex ultrasound results; PE was diagnosed by computed tomography results and cause of death analysis.

Results: The population comprised 154 patients who underwent ligation (62%) or repair (38%) of MVI. The characteristics were 88% male, age 32±12 years, 74% gunshot wound, ISS 16±8, length of stay 17±29 days, with an overall mortality of 21.4%. Comparing ligation vs repair groups, initial GCS was 12±5 vs 14±3 (p=0.009), initial BE -9±8 vs -5±5 (p=0.006), ISS 19±12 vs 16±11 (p=NS), units PRBCs transfused 16±12 vs 12±13 (p=NS). Of the 95 patients with ligation, 73 (77%) survived past 48 hours, and 4 (5.5%) subsequently developed PE. Of the 59 patients with repair, 55 (93%) lived past 48 hours, and 2 (3.6%) developed a PE. No cause of death was suspected from PE. In those that developed a PE, all were receiving standard DVT chemoprophylaxis. 5 patients (3 ligation, 2 repair) received antiplatet therapy in addition to standard chemoprophylaxis, and none of these patients developed a DVT, PE, nor died. 7 patients received IVC filters (4 after confirmed PE, 3 after confirmed DVT (2 ligation, 1 repair)).



Conclusions: Following penetrating trama, ligation of MVI does not prevent the development of PE. In fact, this population is at a heightened risk of PE. The repair of deep extremity veins has not been associated with PE. Patients that received antiplatelet therapy in addition to standard chemoprophylaxis did not develop a DVT nor PE.

NOTES

Session: XII: Quickshots
Paper 14: 9:32-9:38 am

ULTRASOUND VS. PALPATION GUIDED RADIAL ARTERY CATHETERIZATION: A RANDOMIZED CONTROLLED TRIAL

Lucy Ruangvoravat MD, Tobias Zingg MD, Matthew Band PA-C, Linda Maerz MD, Christopher Erb MD, Peter Marshall MD,MPH, Michael Ditillo* DO, Kimberly Davis* MBA,MD, Kevin Schuster* MD, Yale School of Medicine

Invited Discussant: Christine Cocanour, MD

Introduction:

Catheterization of the radial artery (CRA) is commonly done via a "blind" method with palpation of the arterial pulse guiding puncture. We hypothesized that ultrasound guidance would increase success in CRA and decrease time to CRA.

Methods:

We conducted a prospective randomized trial of ultrasound guided CRA in the Surgical and Medical Intensive Care units of a tertiary care academic medical center. Patients requiring CRA were randomly assigned to the palpation-guided (PP) or the ultrasound-guided (US) method. Each attempt was defined as time from skin puncture to appearance of an arterial waveform or withdrawal from the skin. After three failed attempts the operator would switch to the alternate method for three attempts. After six failed attempts the procedure was either aborted or attempted by a more experienced operator. Data collected included radial pulse quality, blood pressure, body mass index (BMI), need for vasopressors, and anticoagulant use. Analysis was by Chi-square, Fisher's exact test and Wilcoxon signed rank test with $p<0.05$ significance.

Results:

Forty six patients were enrolled; 2 randomized to PP withdrew after randomizing. Of the remaining 44, 26 were randomized to US and 18 to PP. There were no differences between groups in BMI, use of vasopressors, anticoagulation, limb edema or pulse quality (all $p>0.05$). Pulse was weak or absent in 69.2% of US patients and 61.1% of PP patients. The average time for US CRA was 666s and 591s for PP. 38.4% in the US group were successful on the first attempt and 22.2% in the PP group ($p=0.256$). In the PP group 50% achieved CRA in 3 attempts compared to 80.8% in the US group ($p=0.013$). There were an average of 2.04 attempts in the US group compared to 2.39 in the PP group ($p=0.141$). Of those that failed PP and crossed over to US, 66.6% were successful compared with 0% of US crossing over to PP ($p=0.070$). All rescue CRAs were by US; 71.4% were successful. Overall ultrasound was attempted in 35 patients (32 successful, 94.4%) and palpation was attempted in 23 (9 successful, 39.1%). The overall probability of success with ultrasound was therefore 7.10 (95% CI 2.29 – 22.00, $p<0.001$) times that of palpation alone.

Conclusions:

With a limited number of attempts US is more likely to result in successful CRA and is also more likely to result in ultimate success. Trends toward longer operating time and higher rescue rates were observed with US suggesting a group of patients where only US will result in successful catheterization.

NOTES

Session: XII: Quickshots
Paper 15: 9:39-9:45 am

A PROSPECTIVE EVALUATION OF SURGICAL RESIDENT INTERPRETATION OF CT SCANS FOR TRAUMA

Reinaldo Morales MD, Rachel M. Drake M.Ed, Stephen D. Helmer Ph.D., The University Of Kansas School Of Medicine - Wichita

Invited Discussant: David Efron, MD

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction: In trauma centers across the United States, surgery residents are utilized to initiate trauma care. These residents are often relied upon to read imaging studies which direct patient care. In many cases, hospitals pay a substantial amount to outside teleradiology firms to evaluate imaging that arrives after the radiology attending is no longer in-house. The objective of this study was to compare the interpretations made by senior surgical residents to those provided by an outside teleradiology firm.

Methods: All trauma patients arriving at an American College of Surgeons verified Level 1 trauma center between 2200 and 0600 hours requiring a CT scan for evaluation of possible injury between December 2012 and December 2013 were included. Once the patient had been scanned, a trauma imaging assessment form was filled out by the senior surgical resident providing their interpretation of the CT study. At a later date, the interpretation of the outside radiologist was recorded and compared to the resident's interpretation.

Results: A total of 135 patients underwent CT evaluation during the study period. The CT interpretation of the senior surgical resident was compared to the outside radiologist. The outside radiologist and senior surgical resident rarely disagreed on the interpretation of a CT. When compared to an outside radiologist, residents were primarily in agreement with CT of the head (96.4%; κ=.927, $p<.001$), cervical spine (90.9%; κ=.773, $p<.001$), thoracic and lumbar spine (50.0%; κ=.475, $p<.001$), abdomen and pelvis (84.2%; κ=.745, $p<.001$), and chest (77.3%; κ=.737, $p<.001$). Overall, there were discrepancies in image interpretation between the resident and outside radiologist in 34 patients (25.2%). Of those 34 patients, residents missed 7 (5.2%) injuries that were clinically significant and resulted in a change of management. These missed injuries included T3 compression fracture (n=1), T6 spinous process fracture (n=1), L2 and L3 transverse process fractures (n=2), pelvic fracture (n=1), renal contusion (n=1), and a subarachnoid hemorrhage (n=1).

Conclusion: Senior surgical residents have a high rate of accuracy in reading CT imaging. Although no life-threatening injuries were missed, resident education in reading radiographic imaging in spinal regions needs to be emphasized.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

NOTES

Session: XII: Quickshots
Paper 16: 9:46-9:52 am

WELCOME BACK: FACTORS ASSOCIATED WITH EARLY READMISSION FOLLOWING TRAUMA

Jennifer C. Roberts MD, MS, Jon Gipson MD, Joseph S. Farhat MD, Patty Reicks RN, Gregory Beilman* MD, University of Minnesota Dept of Surgery

Invited Discussant: David King, MD

Introduction: The purpose of this study was to identify patient and system-based risk factors associated with 30 day readmission at an urban level one trauma center.

Methods: A retrospective review of all adult trauma registry patients readmitted to the hospital within 30 days of discharge between 10/2011-9/2013 was performed. Patient demographics, injury data, length of stay, discharge medications, and reason for readmission were abstracted from the medical record. Descriptive statistics were calculated where appropriate using SPSS software.

Results: 5063 patients were admitted during the study period, of those 238 (4.7%) patients were readmitted within 30 days. 107 were excluded from analysis as the admissions were planned orthopedic procedures leaving 131 eligible for analysis. 51.9% of patients where male, with a mean age of 68, median ISS of 9, and mean length to readmission of 12 days. 63.3% of patients had multiple medical comorbidities (DM,HTN, CRI, or CHF), 20.6% had dementia, and 27.5% had an axis I mental illness excluding dementia. The most common mechanism of injury was falls (74%), followed by motor vehicle crashes (16%) and penetrating injury (4%). The most common reason for readmission was wound issues, followed by cardiac and pulmonary complications (table 1).

Table1. Readmission Data by System

	Wound	Cardiac	Bleeding	Pulmonary	Pain	GI	VTE	Stroke	Other
patients (%)	30 (23)	21 (16)	17 (13)	11 (8)	9 (7)	7 (5)	7 (5)	3 (1)	26 (19)
Ave. days to readmission (range)	14 (7-30)	12 (1-30)	12 (2-30)	13 (2-30)	2 (0-7)	12 (2-27)	16 (10-22)	13 (10-14)	
Diagnosis	18 SSI 6 wound breakdown	6 CHF 5 MI 5 arrhythmia	10 SDH 5 GIB	9 PNA		2 N/V 2 PSBO	5 PE 2 dvt		9 falls 8 confusion 5 UTI
Sent from skilled nursing (%)	14/30 (50)	15/21 (72)	15/17 (88)	9/11 (81)	5/9 (56)	0/7 (0)	2/7 (28)	3/3 (100)	16/26 (62)

9 patients were readmitted with a new fall. The mean time to readmission was 12 days. In 102/131 patients, the trauma service was not notified of readmission. Following readmission, fewer patients were discharged home and more required home health resources (Table 2). Changes were made in home medications in 113/131 patients: 75/131 new opioids, 44/131 new anticoagulation, 31/131 new blood pressure/diuretic medication.

Table 2. Discharge Location by Admission

	Original disposition	Readmission Disposition
Home (%)	46/131 (35)	30/131 (25)
Home Health (%)	7/131 (5)	20/131 (14)
Skilled Nursing Facility (%)	78/131 (60)	81/131 (61)

Conclusions: In contrast to the trauma population as a whole, patients readmitted within 30 days at our institution were elderly men or women with multiple comorbidities who sustained falls prior to admission. The trauma team is infrequently notified of these readmissions. Improved pain control prior to discharge, patient education of pain expectations, and earlier wound surveillance regardless of discharge location may help reduce these readmissions. Many readmissions occur at least 14 days following discharge. These are likely related to elderly patient comorbidities and may represent alterations in patient physiology following trauma. Commonly, medication changes are made at the time of initial discharge and could be a cause of readmission. Closer follow up with a primary care provider after discharge may be helpful in this population.

NOTES

Session: XII: Quickshots
Paper 17: 9:53-9:59 am

FIBRINOLYSIS DOES NOT OCCUR FOLLOWING RESUSCITATION IN SEVERE HEMORRHAGIC SHOCK REQUIRING IMMEDIATE OPERATION

Mona Taleb MD, Anna M. Ledgerwood* MD, Wayne State University

Invited Discussant: Mitchel Cohen, MD

INTRODUCTION: Hemorrhagic shock (HS) causes immediate hypofibrinogenemia and fibrinolysis. This study assesses fibrinolysis following resuscitation in patients (pts) taken directly to surgery for control of severe hemorrhage.

METHODS: 317 measurements of fibrinogen (FI), fibrin split product (FSP), and fibrin monomer (FM) were made in 268 pts following resuscitation for HS in the OR 3.9 hrs after injury (32 pts), postoperatively at 20 hrs (153 pts), at 35 hrs (94 pts), at 52 hrs (64 pts), at 71 hrs (53 pts), and 19 days (8 pts). During OR they received 14.3 RBC units, 851 ml FFP, and 11.5 L balanced electrolyte solution (BES).

RESULTS: OR FI levels were low, rose to low normal levels by 20 hrs and high normal by 35 hrs, and increased to supernormal levels by 52 hrs, 71 hrs, and day 19. FSP and FM were absent during operation and did not appear until FI levels were within the normal range or greater than normal (Table).

Table	OR (4 hrs)	20 hrs	35 hrs	52 hrs	71 hrs	19 days
FI (mg/dL)*	159±13	231±13	373±18	448±15	604±42	500±47
FSP (pos/#pts)$^{+}$	0/25	1/34	8/35	12/37	24/36	10/15
FM (pos/#pts) $^{+}$	0/29	0/37	2/41	1/31	4/11	3/8

Mean ± SE; $^{+}$ Positive value/# pts

CONCLUSION: Low FI during OR and at 20 hrs is due to bleeding, dilution from BES, and extravascular relocation; lysis is absent. Significant lysis does not occur until FI levels are supernormal. Early antifibrinolytic therapy after resuscitation is not indicated.

NOTES

Session: XII: Quickshots
Paper 18: 10:00-10:06 am

INTRACRANIAL PRESSURE MONITORING DURING THERAPEUTIC CEREBROSPINAL FLUID DRAINAGE: "ONLY PART OF THE STORY?"

Raymond Fang* MD, Catriona Miller Ph.D., Shiming Yang Ph.D., Yao Li MS, Colin F. Mackenzie M.B.,Ch.B., Hegang Chen Ph.D., Katharine R. Colton BS, Thomas M. Scalea* MD, Deborah M. Stein* MD,MPH, Peter F. Hu Ph.D., US Air Force

Invited Discussant: Susan Rowell, MD

Introduction: The 2007 Brain Trauma Foundation Guidelines for management of severe traumatic brain injury (TBI) recommends intracranial pressure (ICP) monitoring utilizing an intraventricular catheter (IVC) for salvageable patients with an abnormal head computed tomography scan. IVCs not only provide accurate and reliable ICP measurements, but they also enable therapeutic drainage of cerebrospinal fluid (CSF). Because standard IVCs cannot simultaneously drain CSF and measure ICP, ICP is then often only measured hourly during brief manual closures of the circuit. We hypothesized that unrecognized, but clinically significant, ICP fluctuations occur during periods of CSF drainage in the care of these patients.

Methods: Adult admissions to a Level I urban trauma center between 2008 and 2010 were reviewed to seek patients with concurrent IVC and intraparenchymal ICP monitor (IPM) placements. Time periods when ICP data were recorded from both devices were analyzed and compared. While IPM ICP measurements were autonomously recorded at 6 second intervals, IVC ICP values were abstracted from nursing entries into the patients' vital signs flow sheet and then presumed to remain static until the next IVC ICP measurement was recorded.

Results: Eighty-one patients were identified with concurrent IVC and IPM ICP data over 5,579 hours of monitoring. The mean of all the differences between ICP values measured by IVC vs. IPM showed an IVC bias of -2.1 mm Hg (SD +/- 6 mm Hg). Limiting analysis to periods when IPM ICP $\geq$ 15 and $\geq$ 20 mm Hg, the IVC bias increased to -6.6 (+/-7.8) and -11.1 (+/-9.6) mm Hg.

Conclusions: Neurocritical care practice using IVCs for therapeutic CSF drainage with hourly ICP measurements undervalues ICP when compared to continuous IPM measurement. This difference is more pronounced in patients with intracranial hypertension and may contribute to additional secondary brain injury and suboptimal functional outcomes.

NOTES

Session: XII: Quickshots
Paper 19: 10:07-10:13 am

Complications of Damage Control and Definitive Laparotomy in Combat: 2002-2011

Thomas A. Mitchell MD, Christopher White MD, Lorne Blackbourne MD, John Holcomb* MD, San Antonio Military Medical Center

Invited Discussant: C. William Schwab, MD

Introduction: Damage control laparotomy (DCL) in an austere environment is an evolving surgical treatment modality. We evaluated the demographics, organ injury burden, complications, and mortality for United States (US) soldiers undergoing definitive laparotomy (DL) and DCL in Iraq and Afghanistan.

Methods: A retrospective evaluation of all patients surviving 24 hours who underwent a laparotomy from 2002-2011 during Iraq and Afghanistan was performed. Utilizing ICD-9 Procedure codes, DCL was defined as: a patient undergoing operative procedures at two distinct North American Treaty Organization (NATO) Role III medical treatment facilities (MTFs); a NATO Role II and III MTFs, and/or having the ICD-9 procedure code 54.12, for Re-Opening of Recent Laparotomy Site. Definitive laparotomy (DL) was defined as patients undergoing one operative procedure at one NATO Role II or III MTF. Demographic, mortality, intra-or-retro-peritoneal operative interventions, and complications were compared.

Results: DCL comprised 26.4% (n=330) of all 1,248 laparotomies performed between March 2002 to September 2011. Patients undergoing DCL compared to DL had an ISS of 28.0 (IQR 19, 36) versus 22 (IQR 14, 30), respectively ($p<0.05$). DCL patients had more intra-or-retroperitoneal organs requiring operative intervention, 1.8 ± 1.4 versus 1.0 ± 1.0 ($p<0.05$). Colonic operative procedures occurred in 50% (n=165) of DCLs compared to in 24.2% (n=222) of DLs ($p<0.05$). DCL occurred in 21.7% (n=177) and 35.3% (n=153) of all laparotomies in Iraq and Afghanistan ($p<0.001$). DCL occurred in 37.1% (n=117) and 24.6% (n=201) of all firearm and explosive device injuries ($p<0.05$). Abdominal and extremity/pelvic abbreviated injury scores were directly proportional to predicting DCL compared to DL ($p<0.05$). Intra-abdominal, acute respiratory distress, and thromboembolic complications for DCL versus DL were 8.5% and 5.6% ($p=0.07$), 2.1% and 0.8% ($p=0.06$), and 1.5% and 0.7% ($p=0.17$), respectively. Theater mortality for DCL and DL were 1.5% and 1.4% ($p=1.0$).

Conclusions: Implementation of DCL in an austere environment is a safe and effective means to care for severely injured combat wounded across distinct geographic locations; utilization of this data will provide the foundation to codify a DCL theater clinical practice guideline for future deployed military surgeons.

NOTES

Session: XII: Quickshots
Paper 20: 10:14-10:20 am

STRESS-INDUCED HYPERGLYCEMIA IS ASSOCIATED WITH HIGHER MORTALITY IN SEVERE TRAUMATIC BRAIN INJURY

Patrick L. Bosarge MD, Russell L. Griffin Ph.D., Jeffrey D. Kerby* MD,Ph.D.,
University of Alabama Birmingham

Invited Discussant: Jay Doucet, MD

Introduction: An association between stress induced hyperglycemia (SIH) and increased mortality has been demonstrated following trauma. Experimental animal model data regarding the association between hyperglycemia and outcomes following traumatic brain injury (TBI) is inconsistent suggesting hyperglycemia may be harmful, neutral, or beneficial. The purpose of this study is to examine the effects of SIH vs diabetic hyperglycemia (DH) on severe TBI.

Methods: Admission glycosylated hemoglobin (HbA1c), glucose levels, and comorbidity data were collected over a 4-year period from September 2009 to December 2013 for patients with severe TBI (i.e., admission GCS of 3-8). Diabetes mellitus (DM) was determined by patient history or admission HbA1c of 6.5% or more. SIH was determined by absence of DM and admission glucose of 200 mg/dL or more. A Cox proportional hazards model adjusted for age, sex, injury mechanism, and injury severity score was used to calculate hazard ratios (HRs) and associated 95% confidence intervals (CIs) for the association between SIH and the outcomes of interest.

Results: During the study period, 986 trauma patients with severe TBI were admitted and had available glucose, HbA1c, and comorbidity data. A total of 286 patients were admitted with hyperglycemia; 228 patients (79.7%) were diagnosed with SIH and 58 patients (20.3%) were diagnosed with DH. Compared to normoglycemic, non-diabetic, severe TBI patients, SIH patients had a two-fold increased risk of death (HR 1.95, 95% CI 1.55-2.44), and DH patients had a 50% increase in mortality risk (HR 1.48, 95% CI 1.00-2.19).

Conclusion: Hyperglycemia is associated with higher mortality after severe TBI. This association is stronger in patients with SIH. Further research is warranted to identify mechanisms causing hyperglycemia and subsequent worse outcomes after TBI.

NOTES

Session: XII: Quickshots
Paper 21: 10:21-10:27 am

DETERMINING FACTORS THAT IMPROVE RESPONSE TO INPATIENT REHABILITATION IN PATIENTS WITH MODERATE TO SEVERE TRAUMATIC BRAIN INJURY

Fred S. McLafferty BA, Galinos Barmparas MD, Pamela Roberts Ph.D., Alicia Ortega BS, Miriam Nuño Ph.D., Chirag G. Patil MD, MS, Eric Ley* MD, Cedars-Sinai Medical Center

Invited Discussant: Garth Utter, MD

Introduction: We set out to determine factors that improve response to inpatient rehabilitation among patients with moderate to severe traumatic brain injury (TBI).

Methods: A retrospective cohort study of moderate to severe TBI patients who were admitted to the inpatient rehabilitation service at an academic, level I trauma center between 2002 and 2012. Each patient was assigned a level, 1-7, which was determined by equally spaced intervals of functional independence measure (FIM) scores. A patient with improvement by 2 levels was considered a responder. A forward logistic regression was utilized to identify factors associated with response to inpatient rehabilitation.

Results: Over the 10-year study period, a total of 2,211 patients were treated at our facility for TBI, of which 192 (9%) were admitted to inpatient rehabilitation. Of those, a total of 102 (55.7%) were considered responders, with the largest proportion improving in mobility (73.2%), followed by self-care (60.7%), sphincter control (42.1%), and communication/social cognition (21.3%). Patients that responded to rehab were significantly younger (52.3 years vs. 61.6, $p<.0001$) and had on average longer rehab stays (15.3 days vs. 12.3, $p<.0001$) than their non-responding counterparts Once we adjusted for injury severity score (ISS), hospital length of stay (LOS), and ICU LOS, being younger than age 65 (<65, HR=0.24, $p<.0003$), having a higher head abbreviated injury scale (5 vs. 4, HR=3.06, $p<0.02$), and having a longer rehabilitation LOS (>14 days, HR=3.86, $p<.001$) were all associated with increased response to inpatient rehabilitation. Overall injury severity, as measured by the ISS, had no association with determining responders to inpatient rehabilitation (HR=1.00, $p=0.32$).

Conclusion: Of patients who qualify for inpatient rehabilitation, only one out of two responds to treatment. Mobility and self-care are the major components of overall improvement in functional independence. Younger age and longer rehabilitation times are associated with response to rehabilitation. Further efforts should be placed on identifying and facilitating transfer of traumatic brain injury patients to inpatient rehabilitation.

NOTES

Session: XII: Quickshots
Paper 22: 10:28-10:34 am

THE DISPARITY GAP IN TRAUMA DOES NOT NARROW FOR UNINSURED ADULTS SUFFERING SEVERE INJURY

Jon M. Gerry MD, Thomas G. Weiser MD, David A. Spain* MD, Kristan L. Staudenmayer MD, Stanford University, Trauma And Acute Care Surgery

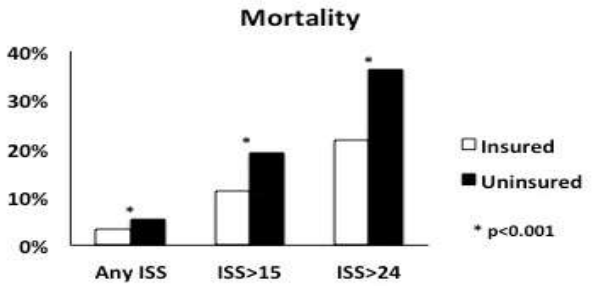
Invited Discussant: Adil Haider, MD

Introduction: Lack of access to trauma care for the uninsured results in worse outcomes. However, once patients arrive at a trauma hospital, the care received should be the same. We hypothesized that disparities in outcomes would lessen with increases in injury severity.

Methods: We performed a retrospective analysis of the 2010 National Sample Program from the National Trauma Databank. We included adults (18 to 64 years) treated at level I or II trauma centers, and excluded patients with unknown insurance status, injury severity scores (ISS), or hospitaldisposition. Patients with insurance were defined collectively as patients with private insurance, Medicare or Medicaid. The primary outcome was in-hospital mortality. Unadjusted and adjusted analyses were performed. All observations are presented as weighted numbers.

Results: There were 310,000 injured patients included in the analysis and 90,000 (29%) were uninsured. The uninsured were younger (35 vs. 41 yrs, $p<0.001$) and more often male (81% vs. 68%, $p<0.001$). Uninsured patients were less severely injured as measured by mean injury severity score (ISS 9.6 vs. 10.7, $p=0.007$) and more often injured by a penetrating mechanism (26% vs. 10%, $p<0.001$). The uninsured were more likely to die regardless of injury severity (11% vs. 19%, ISS>15, $p<0.001$; 22% vs. 36%, ISS>24, $p<0.001$). Admissions directly to the ICU and mean ventilator free days were not significantly different between the two groups. In the adjusted analysis, uninsured status was associated with higher mortality, even at high injury severity (OR 1.67 for uninsured vs insured with ISS>15, $p=0.003$).

Conclusion: Contrary to our hypothesis, being uninsured is independently associated with higher mortality across the ISS spectrum, suggesting increased vulnerability of this population throughout U.S. trauma systems. Since all patients in this study did not suffer fromlack of access to trauma care and because these patients likely received similar care based on their injury severity and ICU utilization, this suggests patient-level factors may be playing an important role in driving poor outcomes.



NOTES

Session: XII: Quickshots
Paper 23: 10:35-10:41 am

BLUNT DUODENAL TRAUMA, IS NON-OPERATIVE MANAGEMENT SAFE?

Matthew Bradley MD, Brandon Bonds MD, David Dreizin MD, Katie Colton BS, Kathirkamanthan Shanmuganathan MD, Thomas Scalea* MD, Deborah Stein* MD,MPH, R Adams Cowley Shock Trauma Center

Invited Discussant: Anna Ledgerwood, MD

Introduction: Clear signs of duodenal injury (DI), pneumoperitoneum and/or oral contrast extravasation mandate laparotomy. However, appropriate management when CT has indirect evidence of DI (duodenal hematoma or periduodenal fluid) is unclear. We evaluated the ability of indirect signs to identify DI and evaluated success of expected management, hypothesizing patients with indirect evidence of DI on CT can be safely managed non-operatively.

Methods: We retrospectively reviewed all patients with a diagnosis of blunt DI and CT scan with duodenal hematoma or periduodenal fluid collection treated between January 2003 and January 2013. Children (age <18 years) and penetrating trauma were excluded. Laboratory values, operative findings, Injury Severity Score (ISS) and Abbreviated Injury Score (AIS) were recorded. Patients having immediate laparotomy were compared to those initially managed conservatively. Student t-tests were used to compare groups.

Results: We identified 84 patients. Nine had findings diagnostic for DI and were excluded from further analysis. 36 patients with indirect signs (48%) underwent immediate operative exploration and 39 (52%) were initially managed non-operatively. There was no difference in admission laboratory values, but mean (±SD) ISS (35±12 vs 26±12, $p<0.001$) and abdominal AIS (3.3±1 vs 2.5±0.8, $p<0.001$) were higher in those with immediate operation. The incidence of DI requiring operative repair was 12% (9 of 75). Seven of 36 (19%) explored urgently had a DI requiring surgical repair while 29 of 36 (81%) had no DI or minor injury not requiring surgical therapy. Of those managed non-operatively, 7 of 39 (18%) failed observation but only two (5%) of those required duodenal repair. Both had worsening findings on interval CT scan. There was no significant difference in ICU (8.6±11 vs 10.4±14, $p=0.38$) and hospital (20.7±15 vs 22.9±21, $p=0.49$) lengths of stay between those that failed non-operative management and those operated on immediately. None of the patients that failed conservative management died.

Conclusion: Observation of patients with indirect sign of DI fails in about 20% of patients, but failure rate due to DI is low. Conservative management in the appropriately selected patient is reasonable and follow-up CT scan may help. Higher ISS may suggest the need for urgent operation.

NOTES

Session: XII: Quickshots
Paper 24: 10:42-10:48 am

MICROARRAY ANALYSIS OF GENE EXPRESSION PROFILES IN TRAUMATIC BRAIN INJURED PATIENTS: IMPACT OF PREHOSPTIAL FLUID RESUSCITATION

Matthew J. Delano MD,Ph.D., Maria Y. Shiu MD, Shawn Rhind Ph.D., Carl Virtanen Ph.D., Joseph Cuschieri* MD, Eileen Bulger* MD, University of Washington

Invited Discussant: Eric Ley, MD

Introduction: Traumatic brain injury (TBI) is the leading cause of death following blunt trauma. Therapeutic intervention focuses on minimizing secondary brain injury by supporting systemic perfusion and reducing intracranial pressure with the administration of hypertonic fluids. Cerebral recruitment of blood leukocytes exacerbates endogenous neuroinflammatory pathways and potentiates secondary brain injury. However, the impact of hypertonic fluid on leukocyte genomic expression is unknown. We hypothesize that peripheral blood leukocyte gene expression profiles vary based on the type of hypertonic fluid administered and may influence the extent of secondary brain injury.
Methods: In a multicentre, 3-arm, double blinded, randomized controlled trial. Patients with severe TBI were administered either 7.5% hypertonic saline (HS), 7.5% hypertonic saline + 6% dextran 70 (HSD), or 0.9% normal isotonic saline (NS) as the initial prehospital resuscitation fluid. Seventy-nine patients and ten age-matched healthy adult volunteers were enrolled. Upon hospital admission peripheral blood samples were drawn directly into PAXgene tubes for mRNA stabilization and extraction. Microarray hybridization was carried out with Agilent Genotypic Technologies.
Results: Hierarchic clustering analysis demonstrated 11788 probes differentially expressed between TBI patients and controls (FDR0.05). Of the 11788 probes, 3236 probes exhibited a two-fold or greater change across all cellular, molecular and biological categories, with immune modulation, inflammatory mediation and wounding responses most represented. Supervised analysis between hypertonic fluid treatment groups revealed 2082 probes that were differentially expressed (FDR0.05). Principle component analysis based on the 2082 probes was able to differentiate the genomic expression patterns between treatment groups. Further analyses between treatment groups revealed 834 probes differentially expressed between NS and HS groups, 259 between the NS and HSD groups, and 460 between the HSD and HS groups. Pathway analysis discovered that many probes represented purine base metabolism and DNA packaging and assembly.
Conclusion: In this study we demonstrated that peripheral blood leukocyte genomic signatures vary significantly with the type of prehospital hypertonic fluid administered. Moreover, the genomic alterations represent immune modulatory and inflammatory pathways that may impact and predict the evolution of secondary brain injury and allow more timely intervention to improve outcome following severe TBI.

NOTES

Session: XII: Quickshots
Paper 25: 10:49-10:55 am

CAN MESENCHYMAL STEM CELLS REVERSE CHRONIC STRESS-INDUCED IMPAIRMENT OF WOUND HEALING FOLLOWING TRAUMATIC INJURY?

Amy V. Gore MD, Letitia E. Bible MD, Kim J. Song MD, Walter D. Alzate MS, Alicia M. Mohr*MD, David H. Livingston* MD, Ziad C. Sifri* MD, University of Medicine and Dentistry New Jersey

Invited Discussant: Gregory Victorino, MD

Introduction: One week following unilateral lung contusion (LC), rat lungs demonstrate full histologic recovery. When animals undergo LC plus the addition of chronic restraint stress (CS), wound healing is significantly delayed. Mesenchymal stem cells (MSC) are pluripotent cells capable of immunomodulation that have been the focus of much research in wound healing and tissue regeneration. We hypothesize that the addition of MSCs will abrogate this impaired healing in the setting of CS.

Methods: Male Sprague-Dawley rats (n=6-7/group) were subjected to LC/CS with or without the injection of MSCs. MSCs were given as a single IV dose of 5×10^6 cells in 1mL IMDM media at the time of LC. Rats were subjected to two hours of restraint stress on days 1-6 following LC. Seven days following injury, rats were sacrificed and lungs examined for histologic evidence of wound healing using a well-established histologic lung injury score (LIS) to grade injury. LIS examines inflammatory cells/high power field (hpf) averaged over 30 fields, interstitial edema, pulmonary edema, and alveolar integrity with scores ranging from 0-11. Data analyzed by ANOVA followed by Tukey's multiple comparison test, expressed as mean ± SD.

Results: As previously shown, seven days following isolated LC, LIS has returned to 0.83 ± 0.41, with a subscore of zero for inflammatory cells/hpf. The addition of CS results in a LIS score of 4.4 ± 2.2, with a subscore of 1.9 ± 0.7 for inflammatory cells/hpf. Addition of MSC to LC/CS decreased LIS score to 1.7 ± 0.8 with a subscore of zero for inflammatory cells/hpf.

LIS Total and Subgroup Score Seven Days Following Injury					
Group	**Inflammatory cells/hpf**	**Interstitial Edema**	**Pulmonary Edema**	**Alveolar Integrity**	**Total LIS**
LC	**0 ± 0**	**0.75 ± 0.5**	**0 ± 0**	**0 ± 0**	**0.8 ± 0.4**
LC/CS	**1.9 ± 0.7***	**1.4 ± 0.5**	**0.4 ± 0.5**	**0.7 ± 0.8**	**4.4 ± 2.2***
LC/CS + MSC	**0 ± 0**	**1.2 ± 0.4**	**0 ± 0**	**0.5 ± 0.5**	**1.7 ± 0.8****

Data presented as mean score ± standard deviation; * p<0.05 vs LC **p<0.05 vs LC/CS

Conclusion: Stress-induced impairment of wound healing is reversed by addition of MSCs given at the time of injury in this rat lung contusion model. This improvement in lung healing is associated with a decrease in the number of inflammatory cells. Further study into the mechanisms by which MSCs hasten wound healing is warranted.

NOTES

40TH WILLIAM T. FITTS, JR., M.D., LECTURE

William T. Fitts, Jr., M.D.
October 6, 1915 - June 17, 1984

William T. Fitts, Jr. was born on October 6, 1915, in Jackson, Tennessee. He received his A.B. degree from Union University in Jackson in 1937 and his M.D. degree from the University of Pennsylvania in 1940. He was an intern resident, Harrison Fellow in Surgical Research, Rockefeller Foundation Fellow in Surgery and Instructor in Surgery at the University of Pennsylvania from 1940-1942 and from 1945-1947. From 1942-1945, he was a Surgical Ward Officer in the Affiliated Unit of the University of Pennsylvania, the 20th General Hospital, in the China-Burma-India Theatre of World War II. He became an Assistant Professor of Surgery in 1949, Associate Professor of Surgery in 1952, and was John Rhea Barton Professor of Surgery and Chairman, Department of Surgery, University of Pennsylvania, from 1972-1975. He spent his entire career at the University of Pennsylvania. Because of his long service to the organization, the Fitts Lecture was established by the American Association for the Surgery of Trauma in 1974 and first presented by Curtis P. Artz, M.D. at the 35th AAST Meeting in Scottsdale, Arizona.

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American Association for the Surgery of Trauma:
Secretary, Vice-President, President-Elect, 1957-1964
President, 1964-1965
Editor, Journal of Trauma, 1968-1974

American College of Surgeons:
Vice-Chairman, Committee on Trauma, 1965-1966
Chairman, Pennsylvania Committee on Trauma, 1955-1967
National Safety Council Surgeon's Award for Distinguished Service to Safety, 1971

American Trauma Society:
President, 1972-1973

FRIDAY, SEPTEMBER 12, 2014, 11:15 AM – 12:15 PM

AAST 40TH WILLIAM T. FITTS, JR. LECTURE

LOCATION: GRAND BALLROOM SALONS G-L

"GENOMICS OF INJURY – THE GLUE GRANT EXPERIENCE"

Ronald Tompkins, M.D., Sc.D.
Division Chief, Surgery, Science & Bioengineering
Massachusetts General Hospital
Boston, MA

****

PREVIOUS FITTS ORATORS

1. 1975 Curtis P. Artz, M.D.
Charleston, SC

2. 1976 Francis D. Moore, M.D.
Boston, MA

3. 1977 G. Tom Shires, M.D.
New York, NY

4. 1978 Lloyd D. MacLean, M.D.
Montreal, Quebec, Canada

5. 1979 Mr. Peter S. London
Birmingham, England

6. 1980 Carl T. Brighton, M.D.
Philadelphia, PA

7. 1981 John W. Kinney, M.D.
New York, NY

8. 1982 Thomas W. Langfitt, M.D.
Philadelphia, PA

9. 1983 Col. Robert Scott, L/RAMC
London, England

10. 1984 F. William Blaisdell, M.D.
Sacramento, CA

11. 1985 Donald P. Becker, M.D.
Los Angeles, CA

12. 1986 Sheng Chih-Yong, M.D.
Woods Hole, MA

13. 1987 Paul Dudley Hart
Woods Hole, MA

14. 1988 Roderick A. Little, M.D.
Manchester, United Kingdom

15. 1989 Prof. Martin Allgower, M.D.
Switzerland

16. 1990 Philip R. Lee, M.D.
San Francisco, CA

17. 1991 Donald D. Trunkey, M.D
Portland, OR

18. 1992 Basil A. Pruitt, Jr., M.D.
Fort Sam Houston, TX

19. 1993 John H. Davis, M.D.
Burlington, VT

20. 1994 John R. Border, M.D.
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21. 1995 Jonathan E. Rhoads, M.D.
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22. 1996 Susan P. Baker, M.P.H.
Baltimore, MD

23. 1997 George F. Sheldon, M.D.
Chapel Hill, NC

24. 1998 Leonard Evans, Ph.D.
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25. 1999 Barbara Barlow, M.D.
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26. 2000 Johannes A. Sturm, M.D.
Hannover, Germany

27. 2001 Janet Reno
Washington, DC
(Cancelled)

28. 2002 C. James Carrico, M.D.
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29. 2003 Ellen J. MacKenzie, Ph.D.
Baltimore, MD

30. 2004 Colonel John Holcomb, M.D.
Ft Sam Houston, TX

31. 2005 Sylvia D. Campbell, M.D.
Tampa, FL

32. 2006 Sten E.V. Lennquist, M.D., Ph.D.
Linkoping, Sweden

33. 2007 Thomas M. Scalea, M.D., FCCM
Baltimore, MD

34. 2008 Charles E. Lucas, M.D.
Detroit, MI

35. 2009 Frederick P. Rivara, M.D., M.P.H.
Seattle, WA

36. 2010 Charles N. Mock, M.D., Ph.D., M.P.H.
Seattle, WA

37. 2011 H. Leon Patcher, M.D..
New York, NY

38. 2012 David B. Hoyt, M.D.
Chicago, IL

39. 2013 Frank R. Lewis, Jr., M.D.
Philadelphia, PA

SESSION XIVA:

PAPERS #35 - #44

FRIDAY, SEPTEMBER 12, 2014, 1:30 PM – 4:50 PM

GRAND BALLROOM SALONS G-L

MODERATOR: DONALD TRUNKEY, M.D.

RECORDER: DAVID SPAIN, M.D.

Session: XIVA: Papers 35-44
Paper 35: 1:30-1:50 PM

CORRELATION OF CEREBRAL OXYGEN DYNAMICS AND METABOLIC CRISIS IN PATIENTS WITH SEVERE TRAUMATIC BRAIN INJURY (sTBI)

Michael Stiefel MD, Christy Stoller MD, Nicole Eiden MD, Corrado P. Marini* MD,
Westchester Medical Center

Invited Discussant: Jose Pascual Lopez, MD

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction:Despite successful maintenance of cerebral oxygen dynamics (COD) a percentage of patients with sTBI will have metabolic derangements as detected by cerebral microdialysis (CMD). This study evaluates possible factors contributing to metabolic crisis (MC) in the presence of maintenance of COD.
Methods:Prospective monitoring of 12 sTBI patients with retrospective data analysis. Minimum of three consecutive days multimodality monitoring and targeted therapy to maintain intracranial pressure (ICP) ≤ 20 mmHg, cerebral perfusion pressure (CPP) ≥ 60 mmHg, cerebral partial pressure of oxygen ($PbtO_2$) ≥ 20 mmHg and cerebral oxygen content ≥ 55% measured by bi-frontal Near-infra red spectroscopy (NIRS). CMD was done via dual lumen catheter to sample the interstitial fluid of the brain; it measured glucose (mmol/L), lactate (mmol/L), pyruvate (µmol/L), glutatmate (µmol/L), glycerol (µmol/L), and Lactate:Pyruvate ratio. CMD measurements were averaged hourly. MC was defined as ischemic if L/P 25 with pyruvate < 120 or secondary to mitochondrial dysfunction (MD), if L/P 25 with pyruvate > 120. The multimodality recorded minutes (MRM) and CMD were combined in excel according to time of the measurements. Statistical analysis was done with student t-test and chi-square as applicable. Data are presented as mean ± SD. Significance was accepted to correspond to a $p < 0.05$.

Results:There were a total of 34,907 consecutive MRM. Of these minutes, 5 (0.01%) had CPP < 60 with NIRS < 55 (MRM1), 780 (2.23) had CPP < 60 with PbtO2 < 20 (MRM2), and 234 (0.67) had PbtO2 < 20 with NIRS < 55 (MRM3) for a total abnormal MRM of 1019/34,907 (2.92%). MRM stratified by survival status showed 614/17,808 (3.4%) MRM of the cerebral oxygen dynamics to be abnormal in non-survivors (n=4) compared to 405/37603 (1.1%) in survivors (n=8), $p < 0.05$. 474/951 (49.8%) of the CMD samples collected simultaneously with the multimodality monitoring had L/P > 25; of these 389/474 (82%) had MC from MD in contrast to 85/474 (18%) from ischemia. There were only 16 events of MC occurring simultaneously with abnormal cerebral oxygen dynamics:1/16 with MRM1, 9 with MRM2 and 6 with MRM3 physiologic pattern.

Conclusion: Based on the results of this study we conclude: 1. MC can occur despite the achievement of endpoints of targeted therapy in patients with sTBI; 2.COD events are associated with increased mortality;3. The majority of the MC events are due to mitochondrial dysfunction not to ischemia.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

NOTES

Session: XIVA: Papers 35-44
Paper 36: 1:50-2:10 PM

A COST ANALYSIS OF SURGICAL STABILIZATION VERSUS CONVENTIONAL MANAGEMENT OF SEVERE RIB FRACTURES

Sarah Majercik* MBA,MD, Scott Gardner PA-C, Steven R. Granger MD, Donald H. Van Boerum MD, Emily Wilson MS, Justin Dickerson MBA,Ph.D., Thomas W. White MD, Intermountain Medical Center

Invited Discussant: Suresh Agarwal, MD

Introduction: Rib fractures are common, and contribute significantly to healthcare costs and socioeconomic burden. Surgical stabilization of rib fractures (SSRF) is increasingly used for flail chest and severely displaced fractures. One factor that has precluded the wide adoption of SSRF is the perception that it is too expensive to surgically fix an injury that will eventually heal without intervention. The purpose of this study was to compare hospital and professional charges for 137 patients with SSRF to a series of propensity-matched, non-operatively managed rib fracture patients (NON-OP) at a single Level One Trauma Center.

Methods: All patients who were admitted to the hospital with rib fractures between January 2009 and June 2013 were identified. Patient demographics, injury mechanism, injury severity score (ISS), chest acute injury severity (AIS), hospital length of stay (LOS), ICU LOS, and charge data were collected for each patient. Propensity score matching was used to identify NON-OP patients whose rib injuries were similar to the SSRF patients. The 2:1 match was based on propensity scores from a model including age, sex, chest AIS, and hospital LOS. Using the matched dataset, zero-inflated negative binomial regression was conducted to assess the relationship between SSRF and ICU LOS. A subset of charge codes relevant to the medical care provided for the thoracic injury was identified. Charge information for each group was compared using Wilcoxon rank sum tests.

Results: 411 patients (137 SSRF, 274 NON-OP) were included in the analysis. As expected, SSRF and NON-OP patients were not significantly different with regard to age, sex, ISS, or chest AIS. By binomial regression, ICU LOS was 1.65 days less for SSRF patients as compared to NON-OP. SSRF had less ventilator days than NON-OP(1.7 vs 3.6 p=0.01). NON-OP patients accrued less total relevant hospital and professional charges than SSRF patients. ($112,606 vs $132,232, $p<0.001$).

Conclusion: Overall relevant hospital and professional charges for SSRF patients are higher than for similarly injured NON-OP patients. SSRF patients have shorter ICU LOS and less ventilator days, but similar hospital LOS as compared to NON-OP. Further cost-effectiveness research in this patient population will determine whether improved quality of life and ability to return to meaningful activity sooner outweighs the increased costs of the acute care episode for SSRF patients.

NOTES

Session: XIVA: Papers 35-44
Paper 37: 2:10-2:30 pm

CAN WE EVER STOP WORRYING ABOUT VENOUS THROMBOEMBOLISM AFTER TRAUMA?

Laura N. Godat MD, Leslie Kobayashi MD, David C. Chang MBA,MPH,Ph.D., Raul Coimbra* MD,Ph.D., University of California, San Diego

Invited Discussant: Ali Salim, MD

Introduction: Trauma patients with pelvic fractures, vertebral fractures and spinal cord injury are known to be at increased risk for venous thromboembolism (VTE). However, the risk of developing VTE may change over time following injury. Determining the time period in which patients are at increased risk of developing VTE may have an impact on prophylaxis, cost and quality of care.

Methods: The California Office of Statewide Health Planning and Development (OSHPD) hospital discharge database was searched between 1995-2009 for all patients admitted with ICD-9 diagnosis codes for traumatic pelvic fractures and vertebral fractures with and without associated spinal cord injury. Those patients were then searched for the ICD-9 diagnosis codes for pulmonary embolism or deep vein thrombosis. Kaplan-Meier and Cox Proportional Hazards analyses were used to assess the timing of VTE events and their association with mortality. Factors studied included; age, gender, race, insurance status, injury type, Survival Risk Ratio, Charlson co-morbidity index, and hospital type.

Results: During the study period 267,743 trauma patients met the injury criteria, of those 10,633 or 3.97% developed VTE. The occurrence of VTE was a significant predictor of mortality, (HR 1.18, $p<0.001$). Compared to pelvic fractures, patients with spine fractures without cord injury were less likely to develop PE or DVT (HR 0.85, $p=0.002$). However, patients with spine fractures and cord injury were more likely to develop VTE (HR 3.18, $p<0.001$); this remained true when in combination with a pelvic fracture (HR 2.12, $p=0.001$). Patients with spine fractures and cord injury at the cervical or thoracic level were significantly more likely to develop VTE, (HR 1.53, $p=0.028$ and HR 1.88, $p=0.001$ respectively), compared to those with lumbar injury.

In the first 3 months after injury the incidence of VTE is 10.3%. This rate drops to 0.5% by the end of 6 months post injury. Subsequently, the rate falls to 0.2% at one year, 0.14% at 18 months and remains low at 0.12%, at 2 years.

Conclusions: The cumulative incidence of VTE is 3.97% in this subset of patients; the highest risk is during the first three months after injury. Between 18 and 21 months the rate returns to that of the general population at 0.1%. These results may guide management strategies such as duration of VTE prophylaxis and removal of IVC filters, which may have an impact on quality of care.

NOTES

Session: XIVA: Papers 35-44
Paper 38: 2:30-2:50 PM

VARIATIONS IN IMPLEMENTATION OF ACUTE CARE SURGERY: RESULTS FROM A NATIONAL SURVEY OF UNIVERSITY-AFFILIATED HOSPITALS

Heena P. Santry* MD, Courtney E. Collins MD, Didem Ayturk MS, Timothy A. Emhoff* MD, George C. Velmahos* MD,Ph.D., MSEd, L D. Britt* MD,MPH,
Catarina I. Kiefe MD,Ph.D., University of Massachusetts

Invited Discussant: John Fildes, MD

Introduction: Acute Care Surgery (ACS) was espoused as a surgical subspecialty over a decade ago to re-invigorate interest in trauma and critical care and improve access to care for general surgery emergencies. Since then many institutions have implemented ACS. We undertook a national survey to determine predictors of ACS implementation and variations emergency general surgery (EGS) processes of care.

Methods: We surveyed surgeons responsible for EGS coverage at University HealthSystems Consortium hospitals (representing >90% of our nation's academic medical centers and their affiliates) using an 8-page postal/email questionnaire querying respondents on attitudes toward EGS coverage, clinical vignettes, hospital resources and infrastructure, and EGS processes of care. Survey responses were analyzed using descriptive statistics, univariate comparisons, and multivariable logistic regression models.

Results: 258 of 319 potential respondents completed surveys (81% response rate). 81 hospitals (31%) had implemented ACS while 134 (52%) had a traditional general surgeon on-call model (GSOC). 38 (15%) hospitals had another model (Other). The table compares hospitals by their reported characteristics. In multivariable modeling, hospital type, setting, and trauma center verification were predictors of ACS implementation. EGS processes of care varied with 28% GSOC having EGS block time vs 67% ACS (p<0.0001); 45% GSOC critically ill patients cared for in a surgical specialty ICU vs 93% ACS patients (p<0.0001); GSOC sharing call among 5.7 (+/- 3.2) surgeons vs 7.9 (+/-2.3) ACS surgeons (p<0.0001); and 13% GSOC taking in-house EGS call vs 75% ACS (p<0.0001). Among ACS hospitals there were variations in patient cohorting (25% EGS patients alone; 21% EGS+trauma; 17% EGS+elective; 30% EGS+trauma+elective), data collection (only 26% had prospective EGS registries), and patient handoffs (only 56% had attending surgeon presence). ACS surgeons took 4.8 (+/- 1.3) calls per month with 60% providing extra call stipend and 40% freeing overnight surgeons from clinical responsibility the following day.

Conclusions: ACS has been adopted by nearly 1/3 of our nation's university-affiliated hospitals with the model more prevalent among urban, university-based level-1 trauma centers. Thus, the potential of the new specialty on the national crisis in access to EGS care is not fully met. Furthermore, variations in EGS processes of care among adopters of ACS suggest that standardized criteria for ACS implementation, much like trauma center verification criteria, may be beneficial.

Table. Hospital Characteristics by Patient Care Model*

	General Surgeon on Call (N=134)	Acute Care Surgery (N=81)	Other (N=38)	Univariate p-value†	OR for ACS Implementation¥ (95% CI)
Hospital Type N (%)					
University-based	**18 (13)**	**55 (68)**	**23 (61)**	**<0.0001**	**Ref**
Community-based	**96 (72)**	**6 (7.4)**	**9 (24)**		**0.24 (0.06, 0.99)**
State/County/City/Public	**8 (6)**	**16 (20)**	**2 (5.2)**		**1.9 (0.55, 6.6)**
Other	**2 (1.5)**	**2 (2.5)**	**1 (2.6)**		**4.2 (0.09, 189)**
Setting N (%)					
Urban	**35 (26)**	**65 (80)**	**19 (50)**	**<0.0001**	**Ref**
Suburban	**52 (39)**	**7 (8.6)**	**10 (26)**		**0.24 (0.08, 0.76)**
Rural	**37 (28)**	**7 (8.6)**	**6 (16)**		**0.53 (0.16, 1.8)**
Teaching Status N (%)					
Teaching	**68 (51)**	**78 (97)**	**31 (82)**	**<0.0001**	**Ref**
Non-teaching	**56 (42)**	**1 (1.2)**	**4 (11)**		**0.41(0.03, 5.4)**
Trauma Center Verification N (%)					
Level 1	**14 (10)**	**71 (88)**	**21 (55)**	**<0.0001**	**Ref**
Level 2	**15 (11)**	**2 (2.5)**	**5 (13)**		**0.11(0.02, 0.59)**
Level 3	**20 (15)**	**2 (2.5)**	**1 (2.6)**		**0.09 (0.01, 1.5)**
Non-designated	**75 (56)**	**3 (3.7)**	**7 (18)**		**0.03 (0.00, 0.21)**
Inpatient Bed Capacity N (%)					
>500	**14 (10)**	**46 (57)**	**16 (42)**	**<0.0001**	**Ref**
401-500	**8 (6)**	**12 (15)**	**4 (11)**		**13.1(0.97, 176)**
301-400	**17 (13)**	**12 (15)**	**6 (16)**		**0.52 (0.11, 2.5)**
201-300	**24 (18)**	**5 (6.2)**	**4 (11)**		**0.50 (0.16, 1.5)**
101-200	**25 (19)**	**4 (5)**	**0**		**0.59 (0.18, 1.9)**
< 100	**36 (27)**	**0**	**5 (13)**		**--**

'Row category' percentages may not equal 100 due to missing responses. †Pearson Chi² test (or Fisher exact test for cell sizes <5). ¥Logistic regression model for outcome "ACS implementation" adjusted for all other hospital characteristics.

NOTES

Session: XIVA: Papers 35-44
Paper 39: 2:50-3:10 PM

IS IT A MYTH THAT THE WHOLE-BODY COMPUTED TOMOGRAPHY IMPROVES THE OUTCOME FOR POLY-TRAUMA PATIENTS WITH AN ISS 16 AND OVER ? : A PROPENSITY-ADJUSTED ANALYSIS

Takashi Fujita* MD, Taichiro Tsunoyama MD, Yasuyuki Uchida MD, Maki Kitamura MD, Kahoko Nakazawa MD, Hideki Ishikawa MD, Ichiro Kaneko MD, Yasuhiko Ajimi MD, Hiroto Ikeda MD, Tetsuya Sakamoto MD, Teikyo University School of Medicine

Invited Discussant: H. Gill Cryer, MD, PhD

Introduction:

The efficacy of whole-body computed tomography (WBCT) has recently been reported for patients with an injury severity score (ISS) of 16 or over. However, the precise prediction of the injury severity is impossible, and we frequently order WBCT as part of a conservative and self-defensive practical behavior. The objective of this study was to evaluate the outcomes of WBCT for blunt-injured adults.

Methods:

We used the datasets from the Japan Trauma Data Bank (JTDB) 2004-13 to obtain data for injured adult patients with blunt trauma with an ISS of 16 and over who were transported directly to the hospital, who did not lack any data related to their vital signs on admission or their outcome at discharge. The patients' demographic data and outcomes on discharge were compared between the WBCT group (WG) and non-WBCT group (nWG). A propensity-adjusted regression analysis was used to determine the association of these factors with the survival discharge for the two groups.

Results:

Of the 25,326 patients evaluated, 10,109 (40%) were evaluated using WBCT (WG), while and 15,195 (60%) were not (nWG). The mean age (95%CI) was 57(56-57) in the nWG versus 54(53-54) in the WG ($p<0.001$). The mean ISS was 24.6(24.5-24.8) vs. 26.8(26.6-27.0) ($p<0.001$), respectively. The mean RTS was 6.88(6.85-6.90) vs. 6.82(6.80-6.85) ($p=0.006$), respectively for the WG and nWG. The crude survival rate was 0.82(0.81-0.82) vs. 0.83(0.82-0.84) ($p=0.002$). The propensity-adjusted odds ratio for the survival discharge rate between the WG and nWG was 1.037(95%C.I., 0.972-1.106; $p=0.275$).

Conclusion:

We failed to demonstrate a significant increase in the survival discharge rate due to the use of WBCT in this study. This result is in contrast to the previous reports about the use of WBCT for patients with an ISS of 16 and over, and suggests that further prospective studies should be performed with strict indication protocols.

NOTES

Session: XIVA: Papers 35-44
Paper 40: 3:10-3:30 PM

THE ANATOMIC SEVERITY OF CHEST WALL INJURIES DOES NOT PREDICT POST-RECOVERY PULMONARY SYMPTOMS: A PROSPECTIVE COHORT STUDY

Tejveer S. Dhillon MD, Joseph M. Galante* MD, Edgardo S. Salcedo MD, Garth H. Utter* MD MSc University of California, Davis

Invited Discussant: Raminder Nirula, MD, MPH

Introduction: Although thoracic trauma is common, little is known about which factors lead to poor long-term functional outcomes. We sought to determine whether the anatomic severity of the chest wall injury predicts post-recovery pulmonary symptoms.

Methods: We conducted a prospective cohort study of patients with thoracic injuries who participated in a single-center randomized trial involving chest tube management. At 60 days after injury, a blinded member of our team conducted standardized interviews to assess: dyspnea severity (visual analog scale, score 0-10); dyspnea frequency (score 0-4); Modified Medical Research Council dyspnea scale (MMRC) (score 0-4); St. George's Respiratory Questionnaire (SGRQ) (score 0-100); and Medical Outcomes Study Short Form 36 physical component score (SF-36 PCS) (normalized score 0-100). To account for both the severity and frequency of dyspnea, we calculated a measure of "dyspnea burden" by multiplying the severity by the frequency. We evaluated the severity of the chest wall injury [number of rib fractures, presence of flail chest, and chest Abbreviated Injury Scale (AIS) score] as a predictor of pulmonary symptoms using linear regression, adjusting for potential confounding factors.

Results: At 60 days after injury, of 244 patients with chest wall injuries who participated in the parent trial, 21 had died, 18 were lost to follow up, 13 were unable to communicate, and 3 declined to be interviewed. Among the remaining 189 evaluated patients, the mean age was 42 ± 18 years and 77% sustained blunt trauma. Prior to injury, 41% were current smokers, 13% had asthma, and 4% had chronic obstructive pulmonary disease (COPD). The mean Injury Severity Score (ISS) was 26 ± 13, chest AIS score was 3.5 ± 0.9, and number of radiographically apparent rib fractures was 5 ± 4. At 60 days, 23% of patients reported dyspnea ≥5 on a 0-10 scale, and 28% experienced dyspnea within the past 24 hours.

Patient-reported outcomes 60 days after chest wall injury*

	Dyspnea Burden (range 0-40, worst=40)	MMRC (range 0-4, worst=4)	SGRQ (range 0-100, worst=100)	SF-36 PCS (range 0-100, worst=0)
Change per additional rib fracture	0.0 (-0.4 to +0.3)	0.0 (0.0 to +0.1)^	+0.1 (-0.7 to +1.0)	-0.5 (-1.0 to 0.0)†
Change associated with flail chest	+1.4 (-2.8 to +5.6)	+0.4 (-0.3 to +1.0)	+10.0 (-0.3 to +20.3)	-6.4 (-12.3 to -0.4)
Change per unit of chest AIS score	+0.2 (-1.4 to +1.8)	0.0 (-0.3 to +0.2)	+1.6 (-2.3 to +5.4)	+0.9 (-1.3 to +3.1)

* Mean change in scores after adjusting for age, sex, cigarette pack-years, asthma, COPD, mechanism of injury, and ISS.
^ 95% CI contains 0.
† 95% CI does not contain 0 but the upper bound is indicated as 0 purely due to rounding.

Conclusion: The anatomic severity of chest wall injuries does not predict worse dyspnea symptoms 60 days post-injury, but it does predict impaired patient-reported overall physical health. The lack of association with pulmonary symptoms suggests that treatment targeted at restoring anatomy (e.g., plating of rib fractures) thus may not address the salient consequences of such injuries.

NOTES

Session: XIVA: Papers 35-44
Paper 41: 3:30-3:50 PM

A GERIATRIC SPECIFIC RIB FRACTURE PROTOCOL SIGNIFICANTLY IMPROVES MORTALITY

Sean F. Monagahan MD, Charles A. Adams* MD, Michael D. Connolly MD, Andrew H. Stephen MD, Stephanie N. Lueckel MD, David T. Harrington* MD, William G. Cioffi* MD, Daithi S. Heffernan MD, Brown University Rhode Island Hospital

Invited Discussant: Ronald Gross, MD

Introduction:The trauma population of the United States continues to age rapidly. Minor injuries, normally well tolerated by younger patients, often prove fatal among the geriatric trauma population. Despite an apparently minor anatomic impact, two rib fractures carry tremendous associated morbidity and mortality among geriatric patients. Protocols and management strategies specific to these older patients must be developed. To this end, we adopted a geriatric specific standardized rib fracture protocol.
Methods:All admitted geriatric (>/=65 years old) trauma patients who sustained rib fractures over the 10 year period 2004-2013 were reviewed. The protocol was started in 2009 and consisted of 1)immediate ICU admission for every geriatric patient whose injuries included at least 2 rib fractures and 2)prophylactic analgesics including epidural analgesia and frequent pulmonary toilet. Patients were divided into pre-protocol (2004-2008) and post-protocol (2009-2013). Demographics, injuries, hospital course and outcome data were collected. Categorical data was assessed using Fisher's exact test and continuous data was compared using a t-test. Alpha was set to 0.05.
Results:Over the ten-year period a total of 619 geriatric patients with at least 2 rib fractures were admitted, 121 in the pre-protocol and 498 patients in the post-protocol phase. Groups were equal with respect to age (79 vs 79 yrs;p=0.9), gender, bunt mechanism, (fall being the most frequent mechanism), and ISS (10.4 vs 11.6;p=0.07). Following 2009, rib fracture protocol patients were more likely admitted directly to the ICU from the ED (64.4% vs 24.8%; p<0.001), resulting in shorter ICU length of stay (5.5 vs 8 days), and fewer patients requiring mechanical ventilation (14% vs 43%;p<0.001). Despite similar probability of survival (92% vs 91%;p=0.9), mortality was significantly lower in rib fracture protocol patients (9% vs 24%;p=0.01).
Conclusion: With the aging trauma population it is critical that we understand the unique burden minor trauma imposes upon an often fragile physiology. We noted that using an aggressive standardized protocol with a large emphasis on prophylactic pain control, pulmonary toilet and a willingness to utilize scarce resources, resulted in significantly lowering the mortality associated with rib fractures. Adherence to geriatric specific protocols across a spectrum of injuries will continue to improve outcomes of geriatric trauma patients.

NOTES

Session: XIVA: Papers 35-44
Paper 42: 3:50-4:10 PM

INTRODUCTION OF A MOBILE DEVICE BASED TERTIARY SURVEY APPLICATION REDUCES MISSED INJURIES: A MULTI-CENTER PROSPECTIVE STUDY

Bradley Moffat MD, Kenji Inaba* MD, Neil G. Parry* MD, Daryl K. Gray MD, Richard Malthaner MD, Christopher Martin MD, Demetrios Demetriades* MD,Ph.D., Kelly N. Vogt MD, London Health Sciences Center

Invited Discussant: Leopoldo Cancio, MD

Introduction: Missed injuries during the initial assessment are a major cause of morbidity after trauma. Though the tertiary survey is designed to capture missed injuries, there is wide variation in how the tertiary survey is applied and documented. We designed a novel mobile device application (Physician Assist Trauma Software [PATS]) to standardize performance and documentation of the tertiary survey. The PATS program facilitates the documentation of a complete head to toe examination, the consolidation of investigations, and prompts the user to take action based on tertiary survey findings. This study was undertaken to assess the feasibility of introducing PATS into routine clinical practice, as well as its capacity to reduce missed injuries at two distinct level I trauma centers.

Methods: Prior to implementation of PATS, the missed injury rates at a higher-volume (5000 annual admissions) and a lower-volume (500 annual admissions) level I trauma center were assessed. Missed injuries were defined as those identified after the completion of the tertiary survey and prior to hospital discharge. The PATS program was implemented simultaneously at both centers. Clinical house staff responsible for completing the tertiary survey were trained on the use of the new app, and a one-month acclimatization period allowed before data collection began. Missed injuries were then prospectively tracked during the study period. Compliance and tertiary survey completion rates were evaluated as a marker of feasibility.

Results: A total of 503 and 104 patients were admitted at the higher- and lower-volume centers respectively during the study period. At the higher-volume trauma center, the missed injury rated decreased from 1% to 0% with the introduction of the PATS program ($p = 0.04$). At the lower-volume trauma center, the missed injury rate decreased from 9% to 2% ($p = 0.01$). Prior to implementation of PATS, 68% of patients at the higher-volume center had a complete tertiary survey documented. After implementation of PATS, the compliance rate increased to 100%. At the lower-volume center, prior to implementation, no formal tertiary survey was documented; after implementation documentation of a tertiary survey increased to 60%.

Conclusions: The implementation of a mobile tertiary survey app significantly reduced missed injuries at both a higher- and lower-volume trauma center. The use of this app resulted in a significant improvement in compliance with documentation of the tertiary survey.

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Session: XIVA: Papers 35-44
Paper 43: 4:10-4:30 PM

PRIORITIZING QUALITY IMPROVEMENT IN ACUTE CARE SURGERY

Christopher C. McCoy MD, Brian Englum MD, Jeffrey Keenan MD, Steven N. Vaslef* MD,Ph.D., Mark L. Shapiro* MD, John E. Scarborough MD, Duke University

Invited Discussant: David Harrington, MD

Introduction: The relative contribution of emergency surgery to overall surgical mortality and the impact of specific postoperative complications on mortality after emergency operations have not been previously described. Identifying specific contributors to postoperative mortality following acute care surgery will allow for significant improvement in the care of these patients.

Methods: Patients from the 2005-2011 American College of Surgeons National Surgical Quality Improvement Program (ACS-NSQIP) database who underwent an emergency operation for one of eight common general surgical diagnoses (acute appendicitis, gallbladder disease, gastroduodenal ulcer disease, intestinal ischemia, intestinal obstruction, intestinal perforation, diverticulitis, and abdominal wall hernia) were included for analysis. Postoperative complications were chosen based upon surgical outcome measures monitored by national quality improvement initiatives and national regulatory bodies. Regression techniques were used to determine the independent association between five specific postoperative complications and subsequent 30-day mortality, after adjustment for a robust array of patient- and procedure-related variables and surgical diagnosis. To account for possible survivor bias, the association between specific complications and postoperative mortality was also determined for patients who died on or after postoperative day 7.

Results: Emergency operations accounted for only 14.9% of the approximately 1.2 million general surgery procedures that are included in ACS-NSQIP, but for over 53.5% of the 19,094 postoperative deaths. 100,829 emergency general surgery patients were included in our analysis. The incidences of five specific postoperative complications are shown in the Table. Of these complications, only pneumonia and myocardial infarction were significantly associated with subsequent mortality.

Complication	# (%) With Complication	# (%) Died	AOR (95% CI) Any Death	AOR (95%CI) Late Death*
Incisional SSI (superficial or deep)	4,209 (4.2%)	117 (2.8%)	0.39 (0.31,0.49) p<0.001	0.67 (0.54,0.84) p<0.001
Pneumonia	2,751 (2.7%)	544 (19.8%)	1.61 (1.41,1.84) p<0.001	2.75 (2.39,3.17) p<0.001
Urinary Tract Infection	1,519 (1.5%)	115 (7.6%)	0.52 (0.41,0.65) p<0.001	0.85 (0.67,1.08) p=0.19
DVT/PE	1,198 (1.2%)	129 (10.8%)	0.71 (0.56,0.89) p=0.003	1.10 (0.87,1.40) p=0.44
Myocardial Infarction	450 (0.5%)	143 (31.8%)	2.96 (2.27,3.86) p<0.001	2.80 (2.07,3.80) p<0.001

***Excludes patients who died before postoperative day 7.**

Conclusion: Given its disproportionate contribution to overall surgical mortality, emergency surgery represents an ideal focus for quality improvement initiatives. Of the specific postoperative complications that are typically targeted by such initiatives, only pneumonia and myocardial infarction have an independent association with subsequent mortality. These complications should therefore receive priority as targets for surgical quality improvement initiatives.

NOTES

Session: XIVA: Papers 35-44
Paper 44: 4:30-4:50 PM

DOES RESUSCITATION WITH PLASMA INCREASE THE RISK OF VENOUS THROMBOEMBOLISM?

Ashley Zander DO, Erik Olson MD, Jan-Michael Van Gent DO, Jesse Bandle MD, Richard Calvo Ph.D. (c), Steven Shackford* MD, Kimberly Peck* MD, Beth Sise RN, Michael Sise* MD, Scripps Mercy Hospital Trauma Service

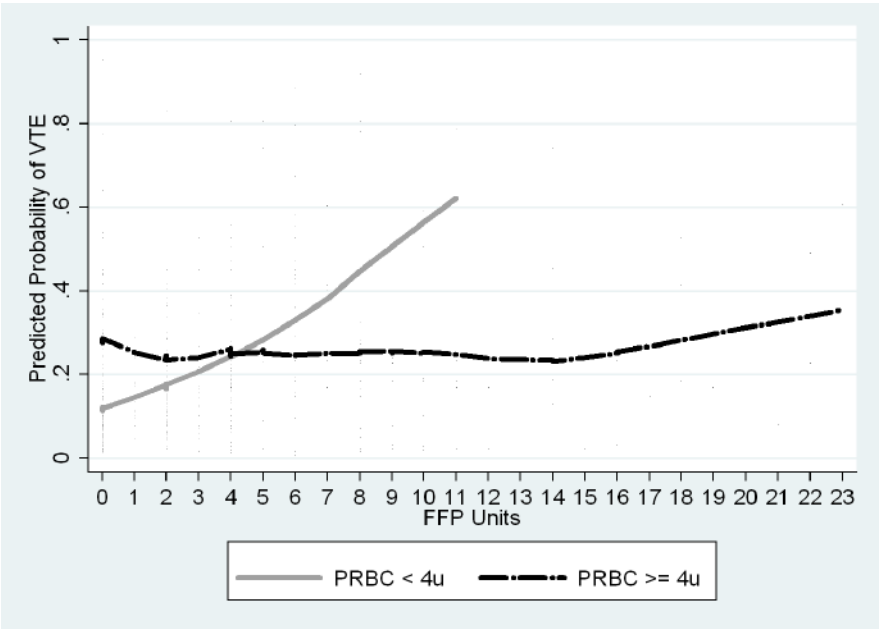
Invited Discussant: Heidi Frankel, MD

Introduction: Resuscitation with blood products improves survival in patients with traumatic hemorrhage. However, the risk of venous thromboembolic (VTE) complications associated with fresh frozen plasma (FFP) administration is unknown. We hypothesized that a higher ratio of FFP to packed red blood cells (PRBCs) given during acute resuscitation increases the risk of VTE independent of severity of injury and shock.

Methods: The records of patients admitted between 4/2007-12/2011 who had surveillance lower extremity duplex ultrasounds were retrospectively reviewed. Patients who received at least 1 unit of PRBCs within 24 hours of admission were included. Patients who died without VTE were excluded. The relationship between FFP and VTE was evaluated using logistic regression.

Results: 387 patients met inclusion criteria, of whom 74 (19%) developed VTE. In patients who required <4 units of PRBCs, increasing units of FFP were associated with an increasing risk for VTE, with each unit of FFP having an adjusted OR=1.25 (95%CI 1.03-1.52, p=0.027) (Fig.). Conversely, in patients who required ≥4 units of PRBCs, FFP in equal or greater ratios than PRBCs was negatively associated with VTE (PRBC=FFP aOR=0.25, 95%CI 0.07-0.90, p=0.035 and PRBCFFP as the reference).

Conclusion: Each unit of FFP increased VTE risk by 25% in patients who required <4 units of PRBCs. In patients who required ≥4 units of PRBCs, FFP administration conferred no increased risk of VTE. This suggests that FFP should be used cautiously when early hemodynamic stability can be achieved with <4 units of PRBCs.



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SESSION XIVB:

PAPERS #45 - #54

FRIDAY, SEPTEMBER 12, 2014, 1:30 PM – 4:50 PM

GRAND BALLROOMS SALONS A, B & F

MODERATOR: ROBERT WINCHELL, M.D.

RECORDER: DAVID LIVINGSTON, M.D.

Session: XIVB: Papers 45-54
Paper 45: 1:30-2:10 PM

USING A VIDEO DECISION-SUPPORT TOOL TO INFORM SURROGATE DECISIONS IN THE SURGICAL INTENISIVE CARE UNIT

Zara Cooper* MD, MSc., Rae M. Allain MD, Rebecca Kallman MD, Elizabeth C. Williams MD, Stephanie Whitener MD, Yuchiao Chang Ph.D., Angelo Volandes MD,MPH, Brigham and Womens Hospital

Invited Discussant: David Livingston, MD

Introduction: Effective communication with surrogates in the surgical ICU (SICU) can be particularly challenging given the complexities of formulating prognosis for high-risk surgical patients and the multidisciplinary nature of the providers involved. Videos are innovative tools to standardize communication and improve knowledge among surrogates making difficult medical decisions. In this pilot randomized controlled trial, we investigated the impact of a video decision support tool on knowledge of routine treatments in the SICU among surrogates of critically ill surgical patients.

Methods: We used a convenience sample of English-speaking surrogates of patients $\geq$ 50 years old who were admitted to the SICU from the acute care surgery service of a major tertiary hospital, and who were expected to stay in the SICU for more than 48 hours. Proximate to SICU admission, physicians used a universal consent form to obtain permission to perform 8 procedures which could be routinely performed during the SICU including: tracheal intubation and mechanical ventilation, arterial line, central venous line, pulmonary artery catheterization, bronchoscopy, blood product transfusion, and tube thoracostomy. In the standard care (SC) arm, physicians provided verbal explanations of the procedures, and their risks and benefits during the consent process. Alternatively, in the intervention (video) arm, surrogates received a verbal explanation and viewed a 12 minute video decision-support tool including images and a voice-over describing these same procedures and their risks and benefits during consent. Surrogate and patient demographic data were obtained through chart review and survey questionnaires. Surrogates in each arm completed a 24-question knowledge test (score range of 0 to 24, with higher score indicating more knowledge) after receiving verbal explanation or viewing the video. Surrogates in the video arm also completed questions about the perceived value of the video. We used two-sample t-tests, wilcoxon rank sum tests or Fisher's exact tests as appropriate.

Results: We enrolled 24 surrogates, 10 in the video arm and 14 in the SC arm. There were no significant differences in patient or surrogate characteristics between groups. Out of a possible 24 points on our knowledge test, surrogates in the video arm scored 22.3 points (SD $\pm$ 1.1) and surrogates in the SC arm scored 19.1 points (SD $\pm$ 3.4), p= 0.005. Eight of 10 surrogates found the video very helpful, and all 10 surrogates would recommend the video to others. There was no difference in consent rates for treatments between groups.
Conclusion: Surrogates who viewed the video had higher knowledge scores, found the video helpful, and would recommend it to others. Those who viewed the video were equally likely to give consent for treatment. Video has the potential to improve the informed consent process for surrogates making complex decisions for critically ill surgical patients.

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Session: XIVB: Papers 45-54
Paper 46: 1:50-2:10 PM

LETHAL NOW OR LETHAL LATER: THE NATURAL HISTORY OF GRADE IV BLUNT CEREBROVASCULAR INJURY

Margaret H. Lauerman MD, Timothy Feeney BS, MS, Clint W. Sliker MD, Nitima Saksobhavivat MD, Brandon R. Bruns MD, Adriana Laser MD, Ronald Tesoriero MD, Megan Brenner MD, Thomas M. Scalea* MD, Deborah M. Stein* MD,MPH, R Adams Cowley Shock Trauma Center

Invited Discussant: Martin Croce, MD

Introduction: Treatment of Grade IV blunt cerebrovascular injury (BCVI4), complete occlusion of the internal carotid (ICA) and/or vertebral artery (VA), by preventing thrombus propagation and encouraging dissolution with antiplatelet agents and anticoagulation optimizes outcomes. We sought to describe the natural history of BCVI4 with regards to stroke and outcomes.

Methods: We retrospectively reviewed patients with ICA or VA BCVI4 from July 2009 to August 2013. Demographic, clinical, and admission and subsequent radiographic information was collected. Rates of BCVI4-related stroke and stroke-related mortality were calculated.

Results: Eighty-two patients sustained BCVI4 (13 ICA and 69 VA). In patients with ICA BCVI4 surviving to repeat imaging, the stroke rate was 70%, with stroke present on initial imaging in 30%, repeat imaging in 40%, and a 20% stroke-related mortality. All patients with ICA BCVI4 developing a BCVI4-related stroke after admission had evidence of cerebral embolus and new BCVI4 recanalization on peri-stroke imaging, with 50% of recanalizations present at stroke diagnosis and 50% of recanalizations present on the first cervical follow-up imaging after stroke. VA BCVI4 was associated with a 2.9% BCVI4-related stroke rate and no stroke-related mortality. Both patients with VA BCVI4 who developed a BCVI4-related stroke had new VA recanalization present at stroke diagnosis, and one patient had evidence of cerebral embolus. BCVI4-related stroke often presented early in the hospital course, with VA and ICA BCVI4-related stroke occurring between 8.5-9 hours and 2-120 hours after admission, respectively.

Conclusion: BCVI4 are not static, frequently recanalize, often soon after injury, and are associated with BCVI4-related stroke. Emergent endovascular vessel occlusion should be considered to prevent recanalization in patients with contraindications to antiplatelet agents or anticoagulation.

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Session: XIVB: Papers 45-54
Paper 47: 2:10-2:30 PM

THE VALIDITY OF ABDOMINAL EXAMINATION IN BLUNT TRAUMA PATIENTS WITH DISTRACTING INJURIES

Jack Rostas MD, Benjamin Cason Jon Simmons MD, Mohammad Frotan MD, Sidney Brevard MD, Richard Gonzalez* MD, University of South Alabama

Invited Discussant: Andrew Kirkpatrick, MD, CD, MHSc

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction:Physicians who care for blunt trauma patients often disregard abdominal clinical examination in the presence of extra-abdominal distracting injuries. Furthermore, many trauma centers mandate a computed tomography (CT) scan of the abdomen in these patients. Ignoring the clinical examination in this patient population may incur undue expense and radiation exposure. The purpose of this study was to assess the efficacy of abdominal clinical examination in patients with distracting injuries.

Methods:During a 1 year period, all awake and alert blunt trauma patients with GCS of 14 or 15 were entered into a prospective study at a Level 1 Trauma Center. Abdominal clinical examination was performed and documented prospectively on all patients entered. Abdominal clinical examination consisted of subjectively questioning the patient for the presence of abdominal pain and physical examination of the abdomen. Physical examination included a four-quadrant anterior abdominal palpation, flank palpation, lower thoracic palpation, pelvis examination, and palpation of and lumbar spine. Following documentation of the clinical examination, all patients underwent CT scan of the abdomen with IV contrast.

Results: Eight hundred and three patients were enrolled during the study period. Four hundred and fifty-one patients had distracting injuries, and 352 patients had no distracting injuries. Of the 352 patients without distracting injuries, one (0.3%) patient was found to have free intra-abdominal fluid that did not require surgical intervention. Of the 451 patients with distracting injuries, 232 patients had a positive abdominal examination, 62 (26.7%) of whom had an intra-abdominal injury diagnosed by CT scan. Of the 219 patients with negative abdominal examination and distracting injuries, 7 (3.2%) were diagnosed with an intra-abdominal injury. All of the seven missed injuries were solid organ injuries, none of which required surgical intervention or blood transfusion. The sensitivity and negative predictive value of abdominal clinical examination for patients with distracting injuries was 89.9% and 96.8%, respectively. The sensitivity and negative predictive value of abdominal examination for surgically significant and transfusion requiring injuries were both 100%.

Conclusion:Distracting injuries do not diminish the efficacy of clinical abdominal examination in the diagnosis of clinically significant abdominal injury. Clinical examination of the abdomen is valid in awake and alert blunt trauma patients, regardless of the presence of other injuries.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

NOTES

Session: XIVB: Papers 45-54
Paper 48: 2:30-2:50 PM

SURGEON PERFORMED ULTRASOUND (SPUS) IN PREDICTING WOUND INFECTIONS: NO COLLECTION, NO INFECTION.

Christopher D. Barrett MD, Arthur Celestin MD,MPH, Emily Fish MD, Alok Gupta MD, Carl J. Hauser* MD, Beth Israel Deaconess Medical Center

Invited Discussant: Nicole Stassen, MD

Introduction: Surgical wound infections (SWI) after emergency abdominal surgery are an important source of morbidity and are highly associated with sepsis and hospital readmission. Primary wound closure is desireable but increases SWI risk. Thus closing high-risk wounds mandates careful observation. Nonetheless, early signs of SWI are commonly missed even by experienced observers and delayed discovery can lead to wound sepsis, dehiscence, prolonged admissions, expensive outpatient wound care, readmissions and late hernia formation. We hypothesized surgeon-performed ultrasound (SPUS) done at the bedside would detect wound fluid collections and that the presence of a wound collection on SPUS would predict SWI better even than careful clinical examination. If so, SPUS might alter early management and improve outcomes.

Methods: A prospective, single-institution study was conducted on adult patients undergoing high-risk open abdominal surgery. After informed consent, SPUS was performed on post-op day (POD) 2-4, then prior to hospital discharge (or on POD 30). Images were obtained by a Surgery PGY1 or 2 who received only one day of training on a smartphone based US system (MobiUS SP-1, Mobisante, Inc.). The primary surgical team was blinded to SPUS results and delivered standard wound care. SWI was diagnosed if patients were begun on antibiotics for the wound, if the wound was opened or had drains placed, if there was any intervention for SWI at the request of the treating physicians or if patients had SWI adjudicated at POD 30 by NSQIP wound-care nurse review. Results were compared by chi-square test with significance set at a $P< 0.05$.

Results: Fifty-four patients were studied. Twenty patients had detectable ($\gtrsim$ 1cm) incisional collections found on SPUS at POD 2-4. Nine went on to develop a clinical SWI. In 34 patients no collection was seen: only 3 of these developed an infection. SWI was clearly associated with early fluid collections on SPUS ($p = 0.002$). The relative risk of SWI in those with a collection was 5.1 (1.5 - 22.3) compared to those with no collection. The negative predictive value of SPUS was 91.2% (0.81-0.98).

Conclusion: SPUS is a highly effective screening tool for detection of SWI in post-op patients. Using current technology and with minimal training, PG1-2 residents detected early collections better than far more experienced examiners. About half of patients with a collection will go on to develop SWI and in the absence of a collection progression to SWI is very unlikely. In effect, without a collection there is little concern for infection. Also, assuming that early management ('formal' imaging or wound interventions) can resonably be based on this data, the number needed to treat (NNT) would only be 2.8 (1.9-11.4). Given the high morbidity and costs associated with delayed diagnosis and treatment, SPUS may be a useful and cost-effective modality for determining the need for early therapy in post-operative patients at risk of developing SWI. Further study with larger sample sizes, advancing technology and greater expertise will evaluate the characteristics of wound collections in more detail and undoubtedly improve accuracy. Randomized interventional studies are also warranted to validate the results of this preliminary study and definitively determine the clinical utility of SPUS in guiding early wound interventions.

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Session: XIVB: Papers 45-54
Paper 49: 2:50-3:10 PM

AN ANALYSIS OF THE EFFECTIVENESS OF A STATE TRAUMA SYSTEM: TREATMENT AT DESIGNATED TRAUMA CENTERS IS ASSOCIATED WITH AN INCREASED PROBABILITY OF SURVIVAL

Dennis W. Ashley* MD, Etienne E. Pracht Ph.D., Regina S. Medeiros DNP,MHSA, RN, Elizabeth Atkins RN, A R. Bayakly Ph.D., Jeffrey M. Nicholas* MD, Medical Center of Central Georgia/Mercer University School of Medicine

Invited Discussant: Gregory J. Jurkovich, MD

Introduction: States struggle to continue support for recruitment, funding and development of designated trauma centers. The purpose of this study was to evaluate the probability of survival for severely injured patients treated at designated trauma centers (DTC) versus non-trauma centers (NTC).

Methods: We reviewed 188,348 patients from the state's hospital discharge database and identified 14,612 severely injured patients admitted to either DTC or NTC between 2008-2012. ICD-9 Injury Severity Scores (ICISS), an accepted indicator of injury severity, was assigned to each patient. Severe injury was defined as an ICISS < 0.85 (indicating ≥ 15% probability of mortality). Three sub-groups of the severely injured patients were defined as most critical, intermediate critical and least critical. A full information maximum likelihood bivariate probit model was used to determine the differences in the probability of survival for matched cohorts.

Results: After controlling for injury severity, injury type, patient demographics, comorbidities, and insurance type and status, severely injured patients treated at a DTC have a 9.5 percent increased probability of survival. The largest improvement was seen in the intermediate subgroup.

	Improvement in probability of survival when treated at a DTC versus NTC	P-Value
All severe trauma (ICISS < 0.85)	9.5%	<0.01
Most critical (ICISS < 0.25)	16.5%	<0.01
Intermediate critical (0.25 ≤ ICISS < 0.5)	22.0%	<0.01
Least critical (0.5 ≤ ICISS < 0.85)	8.3%	<0.01

Conclusion: Treatment of severely injured patients at a DTC is associated with an improved probability of survival. This argues for continued resources in support of designated trauma centers within a defined statewide network.

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Session: XIVB: Papers 45-54
Paper 50: 3:10-3:30 PM

A REASSESSMENT OF THE IMPACT OF TRAUMA SYSTEMS CONSULTATION ON REGIONAL TRAUMA SYSTEM DEVELOPMENT

Robert J. Winchell* MD, Nels Sanddal Ph.D., REMT, Jane Ball RN, DrPH, Holly Michaels BA, Chrisoph R. Kaufmann* MD,MPH, Rajan Gupta* MD, Thomas J. Esposito* MD,MPH, Haris Subacius MA Trauma Systems Evaluation And Planning Committee, American College Of Surgeons Committee On Trauma

Invited Discussant: Richard Mullins, MD

Introduction: Many prior studies have shown that trauma systems decrease morbidity and mortality after injury, but progress in system development has been slow and inconsistent. The Trauma Systems Evaluation and Planning Committee (TSEPC) of the American College of Surgeons Committee on Trauma has developed a process to provide expert consultation to facilitate regional trauma system development, and has conducted consultative visits to 35 regions (commonly states) since 1999. This study evaluated the progress in regional system development that occurred after a consultative visit conducted by the TSEPC in 20 state or regional systems, expanding upon a previous study that demonstrated significant progress in six regional systems following consultations done between 2004 and 2006.

Methods: The study group consisted of regional trauma systems that had consultative visits by expert teams from the TSEPC conducted between 2004 and 2010. System status was assessed using a set of 16 objective indicators. Six systems visited between 2004 and 2006 had baseline scoring done retrospectively as part of the prior study, while the 14 regions visited after 2006 had baseline scores calculated by the consult team as part of the visit. Post-consultation status was assessed during facilitated teleconferences, conducted by members of the original consultation team and current key representatives from each system. Progress was assessed by comparing changes in both aggregate and individual indicator scores.

Results: This study showed a statistically significant increase in aggregate indicator scores following consultation. When each of the sixteen indicators was analyzed individually, significant improvements were seen in fourteen of the sixteen indicators. Indicators showing the greatest gain included those related to trauma system standards, improved linkages with public health, data systems, performance improvement, and prehospital triage criteria. This represented a change from the original study, in which the largest gains were in system planning and quality assurance. As was found in the original study the two indicators related to financing for the trauma system showed no improvement, and showed deterioration in several cases. There was a trend toward better overall improvement in regions with stable funding. In contrast to the initial study, system improvement did not continue to increase with time, but showed a tendency toward deterioration as the length of time after consultation increased.

Conclusions: The TSEPC trauma system consultation process continues to be associated with improvements in regional trauma system development. Gains may not be self-sustaining as there was a trend toward deterioration of aggregate score over time, suggesting that a repeat consultation may be beneficial. A trend showing greater progress in regions with established funding was found. As demonstrated in the original study there was no statistically significant progress area of system funding, and system funding was the area most likely to suffer setbacks over the study period. The ongoing lack of stable trauma system funding may be an even more critical given the indentified trend linking funding and system development.

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Session: XIVB: Papers 45-54
Paper 51: 3:30-3:50 PM

INTIMATE PARTNER VIOLENCE – RISKS GO BEYOND THE VIOLENCE: ASSOCIATION OF INTIMATE PARTNER VIOLENCE WITH MENTAL ILLNESS AND SUBSTANCE ABUSE AMONG FEMALES ADMITTED TO A RURAL LEVEL-I TRAUMA CENTER

Ashley B. Hink MD,MPH, Eric A. Toschlog* MD, Brett Waibel MD, Michael Bard* MD, Brody School of Medicine at East Carolina University

Invited Discussant: James Davis, MD

Introduction: Intimate partner violence (IPV) is a major public health problem and significant contributor to intentional injuries and homicides among women. Despite this, it remains under-recognized in the trauma surgery setting and its association to other risk factors for both intentional and unintentional injuries remains poorly defined. This study aims to assess the prevalence of IPV and its association with alcohol abuse (AA), illicit substance use (ISU), selected mental illnesses (MI) and other risk factors for injury among females admitted to a Level-I trauma center.

Methods: This is a prospective study enrolling adult female patients at a rural, Level-I trauma center over a seven-month period in 2013 to participate in face-to-face structured interviews utilizing a formal survey instrument. Lifetime IPV (LIPV) was identified with self-report items, and past-year or current IPV was identified with the Partner Violence Screen (PVS) and the Woman Abuse Screening Tool (WAST). Self-report items and validated instruments assessed AA, ISU and MI. Other collected data include demographics, insurance status, access to primary care, previous injuries, past IPV screening, possession of firearms, and presenting mechanism of injury (MOI). Bivariate analyses were performed with Chi-square, Mantel-Haenszel odds ratios, and independent-samples t-tests. Multivariate analysis was performed with binary logistic regression.

Results: 107 patients met the inclusion criteria and 81 (76%) were enrolled. 41 (50.6%) reported LIPV and 25 (30.9%) reported past-year IPV. Women with LIPV and past-year IPV were more likely to be under the age of 50, in a current relationship, uninsured, and report previous IPV screening compared to those who have not experienced IPV. Participants with a past-year history of IPV were more likely to have a significant other possessing a firearm (OR 4.6, 40% vs. 12.5%, $p=0.005$). Although it did not reach statistical significance, the odds of having a MOI of assault or self-inflicted injury among those with a history of LIPV was 3.4 times higher than those without (15.8% vs. 5.3%, $p=0.13$). Suicidal ideation was significantly associated with LIPV (OR 9.5, $p = 0.015$). LIPV and past-year IPV were significantly associated with self-reported MI and positive detection of MI (Table 1). LIPV was significantly associated with ISU (OR 4.2, 31.7% vs. 10%, $p=0.016$), and past-year IPV was significantly associated with AA (OR 5, 28% vs. 7.1%, $p=0.011$). These associations remained significant when controlling for relationship status, although the associations between IPV, ISU and AA were significant only among women under the age of 50. On further bivariate analysis, MI was not associated with ISU, AA, firearm access or suicidality. Logistic regression models identified that partner possession of a firearm, lifetime IPV exposure, self-reported MI and AA were significant predictors of past-year IPV.

Table 1. Associations Between IPV, Mental Illness and Substance Abuse

	LIPV (n=41)	No LIPV (n=40)	*p*-value OR (95% CI)	Past-Year IPV (n=25)	No Past-Year IPV (n=56)	*p*-value OR (95% CI)
Self-Report Past-Year MI (n=23)	16 (39%)	7 (17.5%)	*0.032** *OR = 3 (1.1 – 8.4)*	12 (48%)	11 (19.6%)	*0.009** *OR = 3.8 (1.4 – 10.5)*
Positive MI MINI (n=26)	20 (48.8%)	6 (15%)	*0.001** *OR = 5.4 (1.9-15.6)*	13 (52%)	13 (23.2%)	*0.01** *OR = 3.6 (1.3 – 9.7)*
Alcohol Abuse AUDIT > 8 (n=11)	7 (17.1%)	4 (10%)	*0.35* *OR = 1.9 (0.5-6.9)*	7 (28%)	4 (7.1%)	*0.011** *OR = 5 (1.3 – 19.3)*
Past-Year Illicit Substance Use (n=17)	13 (31.7%)	4 (10%)	*0.016** *OR = 4.2 (1.2-14.2)*	8 (32%)	9 (16.1%)	*0.104* *OR = 2.4 (0.8 – 7.3)*
Past-Year Illicit SA DAST > 5 (n=8)	6 (14.6%)	2 (5%)	*0.15* *OR = 3.3 (0.6 – 17.2)*	4 (16%)	4 (7.1%)	*0.2* *OR = 2.5 (0.6 – 10.8)*

*Significance defined as $p < 0.05$. MI = Mental Illness, MINI = MINI International Neuropsychiatric Interview, AUDIT = Alcohol Use Disorder Identification Test, SA = Substance Abuse, DAST = Drug Abuse Screening Test

Discussion: The prevalence of IPV among women admitted to a Level-I, rural trauma center was significantly higher than the reported lifetime prevalence of 20-30% in the general population, and IPV was significantly associated with MI, ISU and AA in addition to high-risk scenarios for intentional injury including suicidal ideation and concurrent IPV and firearm ownership by a significant other. These findings further inform the potential value of IPV screening in the inpatient trauma setting, and suggest that IPV, MI, and substance abuse should be considered as associated entities in secondary prevention and recidivism reduction efforts in the female trauma population.

NOTES

Session: XIVB: Papers 45-54
Paper 52: 3:50-4:10 PM

IS TRAUMATIC VIOLENCE GETTING BETTER OR WORSE? NON-FATAL GUN VIOLENCE AT AN URBAN TRAUMA CENTER

Vincent E. Chong MD, MS, Wayne Lee MD, Gregory P. Victorino* MD, University of California San Francisco - East Bay

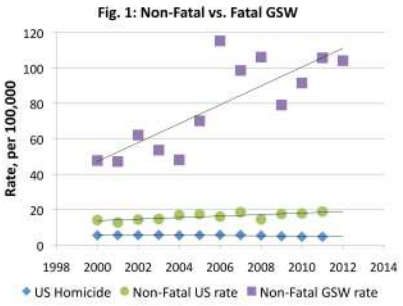
Invited Discussant: Lenworth Jacobs, Jr., MD, MPH

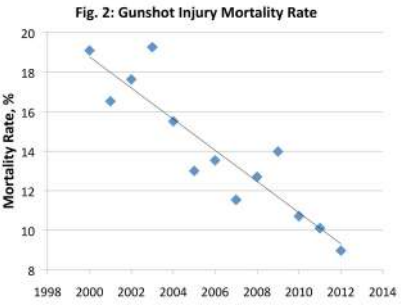
Introduction: National homicide trends suggest violence is getting better. Local data is less clear. Homicide rates in major American cities have diverged in the past decade, with some cities worsening while others improve. Rates of non-fatal firearm injury complicate the picture, suggesting that gun violence is becoming more prevalent, but with varying severity. We hypothesized that non-fatal gunshot injuries were increasing in extent and severity at our urban trauma center.

Methods: We identified patients in our trauma registry who presented as a result of interpersonal violence from 2000-2012. Demographic information and variables of interest (ISS, # of non-fatal gunshot injuries, mortality rate) were trended by year. Non-fatal gunshot injury rates were calculated using population estimates from the FBI Uniform Crime Reporting Program (UCR) and correlated to local municipal police agency data.

Results: We treated 10,082 patients due to interpersonal violence. Of these, 43% (N=4,376) were due to firearms. Rates of non-fatal gunshot injuries rose from 47.7 to 105.7 per 100,000 population ($p<0.001$; Fig. 1). These rates calculated from our registry correlated with rates from the local municipal police agency ($R^2=0.92$; $p<0.001$). Interestingly, the proportion of patients with non-fatal injuries who presented with a severe injury (ISS>25) decreased from 19% to 12% ($p=0.02$) and our gunshot injury mortality rate decreased from 19% to 9% ($p<0.001$).

Conclusion: Homicide rates do not tell the whole story. At our trauma center, non-fatal gunshot injuries are becoming more common, but less lethal. This contrasts with data from other American cities, which show gun violence becoming more prevalent and more lethal. The rate of increase in our non-fatal firearm injury rate outpaced the national trend. Comparison among other major American cities is needed to better understand firearm violence trends in the United States.





NOTES

Session: XIVB: Papers 45-54
Paper 53: 4:10-4:30 PM

TRAUMA PATIENT READMISSIONS: WHY DO THEY COME BACK FOR MORE?

Laura B. Petrey MD, FACS, Alan D. Cook MD, FACS, Richard Gilder RN, MS, Monica Bennett Ph.D., Megan C. Reynolds MS, Jo Weddle MD, Michael L. Foreman* MD, FACS, Ann Marie Warren Ph.D., Baylor University Medical Center

Invited Discussant: Mark Malangoni, MD

Introduction: Unplanned hospital readmissions are a significant focus of healthcare reform. There is speculation that rates of readmission following a traumatic injury will be publically disclosed and penalties will incur. We hypothesize identifying reasons for readmission will help develop targeted patient interventions directed toward high-risk patients to reduce their hospital utilization and healthcare spending.

Methods: Retrospectively, data was collected for 2027 unique trauma patients admitted to a Level I ACS-certified trauma center over a year, with readmissions identified following one year from the index admission. A regional database encompassing 15,000 square miles including 75 hospitals was queried for readmissions. Outcomes of all readmission encounters were analyzed using a binary logistic regression model including demographic, diagnosis, ISS, procedure, Elixhauser comorbidity, insurance, and disposition data. The Regional Enterprise Master Patient Index (REMPI) was also included in the model and is a probabilistic tool that matches patient encounters across hospitals and allows identification and analysis of patient activity regardless of encounter location or payer. Subset analysis of 174 encounters that had 3 or more readmissions was also performed to look for different patterns in the frequent fliers.

Results: 474 (23%) patients were readmitted during the study period. There were 821 re-admit encounters, (range 1-21) averaging 2.03 re-encounters, with a median of one re-encounter. Of the 474, 88 patients were readmitted for a trauma specific related diagnosis. The trimmed model includes significant "independently predictive" variables with a receiver operating characteristic curve of 0.770, which is characteristic of a strong model.

Independent Predictors for 3 or More Readmissions (above 75th percentile)	Odds Ratio	Beta value	p value
Septicemia with extreme risk of mortality	5.147	1.639	0.001
Elixhauser diabetes mellitus complicated or uncomplicated	2.824	1.038	0.000
Medicaid	2.446	0.894	0.002
Congestive heart failure	1.954	0.670	0.006
Elixhauser weight loss	1.864	0.623	0.008
Elixhauser psychosis	1.724	0.545	0.040
REMPI sequence > 14	2.970	1.088	0.000
Increasing age	0.998	-0.012	0.036

Conclusion: In our study, race, ethnicity, sex, and socioeconomic status were not found to be significant factors for readmission. Septicemia was found to be the most significant risk factor (515%) for readmission. Increasing age was actually found to slightly decrease the risk of readmission. Readmissions occur more frequently in patients with comorbidities such as diabetes and congestive heart failure. Having Medicaid funding increased the risk of readmission by 245%. Weight loss and psychosis were also found to increase the risk of readmission. The REMPI sequence above 14 carries a 297% risk of readmission. Multi-disciplinary discharge planning, patient and family discharge education, and arranging outpatient follow-up has been shown to reduce readmissions. Identification of population factors for readmission following injury may allow trauma centers to target risk factors preemptively and hopefully minimize readmission. This is the first step for developing interventions for the reduction of resource utilization and healthcare cost.

NOTES

Session: XIVB: Papers 45-54
Paper 54: 4:30-4:50 PM

MINIMALLY INVASIVE IS MAXIMALLY EFFECTIVE: THERAPEUTIC AND DIAGNOSTIC LAPAROSCOPY FOR PENETRATING ABDOMINAL INJURIES

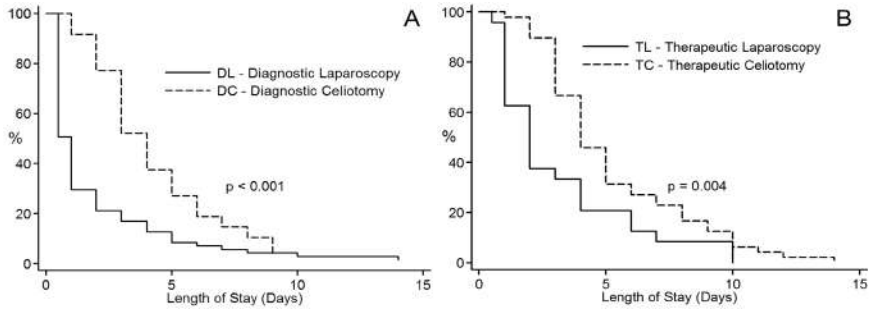
Paul J. Chestovich MD, Timothy D. Browder* MD, Shawna L. Morrissey DO, Douglas R. Fraser MD, Nichole K. Ingalls MD, John J. Fildes* MD, University of Nevada School of Medicine

Invited Discussant: Rao Ivatury, MD

Introduction: Minimally invasive techniques and equipment have evolved allowing increased operative capabilities within most subspecialties of general surgery. There remains limited evidence supporting laparoscopic techniques in managing the injured patient. We hypothesized that laparoscopy is effective for the diagnosis and treatment of penetrating abdominal injuries.

Methods: We retrospectively reviewed all patients undergoing abdominal exploration following penetrating trauma at our Level 1 trauma center over a 6-year period from 1/1/08 to 12/31/13. Demographic and resuscitation data were obtained from our trauma center patient registry. Charts were reviewed for operative details, hospital course and complications. Hospital length of stay (LOS) and complications were primary endpoints. Patients were classified as having non-therapeutic diagnostic laparoscopy (DL), non-therapeutic diagnostic celiotomy (DC), therapeutic laparoscopy (TL), or therapeutic celiotomy (TC). TL patients were matched 1:2 with TC patients having similar intra-abdominal injuries.

Results: 518 patients, including 281 (55%) stab wounds (SW) and 237 (45%) gunshot wounds (GSW) were identified. Celiotomy was performed in 379 (73%) patients, laparoscopy in 139 (27%), with 44 (32%) of those converted to celiotomy. The initial comparison group were non-therapeutic explorations with 119 patients (23%) including 71 DL and 48 DC with similar injury severity (ISS 5.31 vs. 3.83, p=NS). LOS was shorter in DL compared to DC (Fig A, p<0.001). There were no missed injuries. Wound infections (8% vs. 0%, p=0.013) and ileus (10% vs. 0%, p=0.005) were more common after DC than DL. The therapeutic comparison group consisted of 399 (77%) patients, including 375 (72%) with celiotomy, and 24 (4.6%) with laparoscopy (TL). Laparoscopic repairs included liver, stomach, small and large intestine, diaphragm, bladder, minor hemorrhage and abdominal wall defects. The TC included 48 patients with similar injuries and ISS (8.85 vs. 7.95, p=NS). LOS was shorter in the TL group than TC (Fig B, p=0.004). There were no missed injuries. Wound infections were more common in TC than TL (15% vs. 0%, p=0.049).



Conclusion: We have demonstrated the safety of laparoscopy following penetrating abdominal trauma. The use of laparoscopy resulted in shorter hospitalization, fewer wound complications and no missed injuries. Laparoscopy should be the initial procedure of choice in stable patients with penetrating abdominal injuries.

NOTES

MILITARY AWARDS

FRIDAY, SEPTEMBER 12, 2014, 4:50 PM – 5:00 PM

GRAND BALLROOM SALONS G-L

PRESIDING: WILLIAM CIOFFI, M.D., AAST PRESIDENT

AAST ANNUAL BUSINESS MEETING *(FELLOWS ONLY)*

FRIDAY, SEPTEMBER 12, 2014, 5:00 PM – 6:15 PM

GRAND BALLROOM SALONS G-L

AAST BANQUET RECEPTION

FRIDAY, SEPTEMBER 12, 2014, 7:30 PM – 8:00 PM

GRAND BALLROOM FOYER

AAST BANQUET *(BLACK TIE)*

FRIDAY, SEPTEMBER 12, 2014, 8:00 PM – 10:00 PM

GRAND BALLROOM SALONS A-F

PETER C. CANIZARO, M.D.
June 30, 1935 - September 3, 1990

Peter C. Canizaro was born on June 20, 1935, in Vicksburg, Mississippi. He received his B.A. degree from the University of Texas, Austin, in 1956 and his M.D. degree from the University of Texas Southwestern Medical School, Dallas, in 1960. Following an internship at Parkland Memorial Hospital/UTSMS, he spent two years as a Captain in the Surgical Research Unit, Brooke Army Hospital, Fort Sam Houston. Following another year as a NIH Research Fellow, he completed his surgical residency at Parkland/UTSMS from 1964-1968. He remained on staff at Parkland/UTSMS from 1968-1974, and then subsequently served on the faculty at the University of Washington (1974-1976) and Cornell University Medical Center (1976-1981) where he became Professor of Surgery. Dr. Canizaro became Professor and Chairman of the Department of Surgery at the Texas Tech University Health Sciences Center in 1982 and remained there until his untimely death in 1990. Dr. Canizaro was an innovative surgical scientist who made multiple contributions to the field of trauma and resuscitation. Examples of topics covered in his published manuscripts include the following:

1960	Distribution changes in extraceullular fluid during acute hemorrhage (with G. Tom Shires, M.D.)
1963	Use of dextran
1963	Use of hypertonic glucose
1969	Diagnostic abdominal paracentesis in trauma
1970	Fluid resuscitation of hemorrhagic shock
1971	Use of Ringer's lactate during shock
1974	Oxygen-hemoglobin dissociation curve

1975 Stroma-free hemoglobin

1985 Ultrasound detection of fluid collection

1986 Endopeptidase in human lung

In recognition of Dr. Peter Canizaro's outstanding contributions to the science of trauma, the AAST has presented the Canizaro Award since 1993 to the best paper by a new member in their first two years of membership.

PETER C. CANIZARO AWARD

1993 Philip S. Barie, M.D., M.B.A.

1994 Frederick A. Luchette, M.D.

1995 Patrick J. Offner, M.D.

1996 Rodney M. Durham, M.D.

1997 Ronald J. Simon, M.D.

1998 Charles N. Mock, M.D., M.P.H., Ph.D.

1999 David A. Spain, M.D.

2000 John T. Owings, M.D.

2001 Hans-Christoph Pape, M.D.

2002 Karen J. Brasel, M.D., M.P.H.

2003 James Jeng, M.D.

2004 Eileen M. Bulger, M.D.

2005 Carnell Cooper, M.D.

2006 Saman Arbabi, M.D.

2007 Kari Hansen, M.D.

2008 Randall S. Friese, M.D.

2009 Andrew C. Bernard, M.D.

2010 Oscar D. Guillamondegui, M.D.

2011 Jay Manaker, M.D., FACEP

2012 Stephanie Savage, M.D.

2013 Jason Smith, M.D.

SESSION XV:

PAPERS #55 - #66

SATURDAY, SEPTEMBER 13, 2014, 8:00 AM – 12:00 PM

GRAND BALLROOM SALONS G-L

MODERATOR: THOMAS SCALEA, M.D.

RECORDER: SUSAN BRIGGS, M.D., M.P.H.

Session: XV: Papers 55-66
Paper 55: 8:00-8:20 AM

TWO ARE BETTER THAN ONE: SYNERGY OF BETA-BLOCKADE AND STATIN THERAPY ON SURVIVAL IN SEPSIS

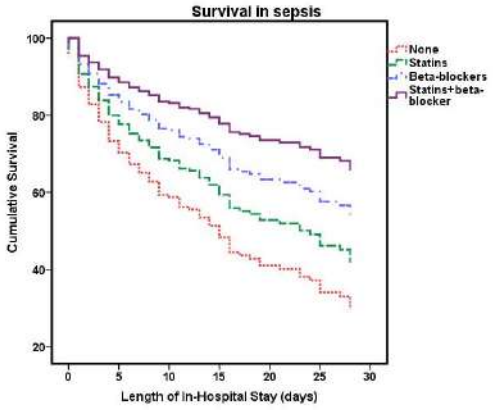
Irada Ibrahim-zada MD,Ph.D., Peter Rhee* MD,MPH, John Santoro Jr., Irina Maskaykina BS, Lynn Gries MD, Terence O'Keeffe* MD, Randall Friese* MD, University of Arizona - Tucson

Invited Discussant: Carl Hauser, MD

Introduction:Current evidence suggests the importance of immunomodulatory therapy in sepsis. Recent clinical trials have demonstrated survival benefits of statins and beta-blocker therapies in sepsis via anti-inflammatory mechanisms. The aim of the study is to evaluate the effect of synchronous administration of beta-blockade and statin therapy in sepsis. We hypothesize that combined administration is associated with increased survival in patients with sepsis.
Methods: This is a single-institution retrospective cohort study on patients with sepsis hospitalized in the ICU at our urban tertiary referral center from 1/1/2008 through 3/31/2011. Records were cross-referenced with pharmacy database to identify patients on beta-blockers (BB), statins (ST), both (BB+ST), and none. Primary outcome is in-hospital 28-day mortality. Kaplan-Meier and Cox-regression analyses were utilized to identify survival benefits adjusted by gender and APACHE II scores.
Results: 304 patients were identified in our database. 101(33%) patients received BB only, 14(5%) received ST only, and 36(12%) patients had BB+ST. Mean APACHE II score was 19.45±7.5 with no difference between groups(p=0.5). Mean survival was 15, 18, 20 and 22 days in groups with none, ST, BB, and BB+ST, respectively. Cox-regression analysis showed that synchronous administration of BB and ST during ICU stay improved in-hospital survival in patients (HR=0.35, 95%CI 0.18 to 0.67, p=0.002) compared to patients without therapy whereas BB only group had HR of 0.51 (p=0.001).

Therapy	p value	HR	95%CI
None	1.0 (REFERENCE)		
Statins	.488	.72	0.29; 1.80
Beta-blockers	.001	.51	0.35; 0.76
Statins+beta-blockers	.002	.35	0.18; 0.67
APACHE II	.000	1.11	1.08; 1.14
Gender	.027	.67	0.47; 0.96

Conclusion:Beta-blockade combined with statin therapy during ICU stay has additive effect and is associated with a further 1.5-fold reduction in mortality in septic patients. Randomized clinical trials are warranted to evaluate their synergistic benefits on survival and explore immunomodulatory mechanisms.



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Session: XV: Papers 55-66
Paper 56: 8:20-8:40 AM

SAVING LIVES AND SAVING MONEY: HOSPITAL-BASED VIOLENCE PREVENTION IS COST-EFFECTIVE

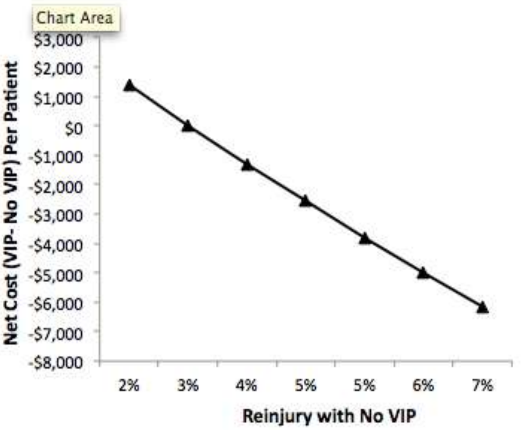
Catherine Juillard MD,MPH, Nancy Anaya MS, Randi Smith MD,MPH, Arturo Garcia MD, James G. Kahn MD,MPH, Rochelle A. Dicker* MD, San Francisco General Hospital

Invited Discussant: Carnell Cooper, MD

Introduction: Victims of violence are at significant risk for injury recidivism and 20% risk of future fatality. We previously demonstrated that our hospital-based violence intervention program (VIP) resulted in four-fold reduction in injury recidivism; trauma care costs $41,000 per patient. Given limited trauma center resources, VIP cost-effectiveness is fundamental when evaluating exportability of these programs to other institutions. This study aims to determine the cost-effectiveness of a hospital-based VIP using a cost utility analysis.

Methods: We used Markov methodology to analyze cost-utility for a hypothetical cohort of violently injured subjects, comparing VIP versus not having VIP at a trauma center. Quality adjusted life-years (QALYs) were calculated using health state utilities based on literature data. Cost of care, cost of VIP, and risk of recidivism with and without VIP were obtained from institutional data. Outcomes were based on QALYs gained and cost of VIP and trauma care over a 5-year horizon. Sensitivity analyses were done using variable values for costs and recidivism rates to predict outcomes over a range of values.

Results: VIP results in 25.6 average QALYs saved and average costs (program cost and cost of care for recidivists) of $5,892 per patient. This is less than the cost of care associated with recidivism for no VIP ($5,923), demonstrating that the reduction in recidivism offsets the cost of VIP. In the sensitivity analysis, net QALYs gained with VIP nearly triple when the injury recidivism rate without VIP is highest. When analyzed over extreme values of injury cost, the cost of having no VIP increases to $85,691 at the highest recorded institutional cost of care. The graph represents potential cost or savings of VIP at our institution's VIP-associated annual injury recidivism rate of 0.9% over a range of recidivism rates with no VIP, at fixed injury cost. Cost-effectiveness remained robust over a range of values; $6,000 net cost savings occur when 5-year recidivism rate without VIP is at 7%.



Conclusion: VIP costs less than having no VIP with significant improvement in QALYs gained. Having no VIP has the most economic consequence when injury recidivism is high. Across a range of plausible values at which VIP would be least cost-effective (lower injury recidivism, cost of injury, and program effectiveness), VIP still results in acceptable cost per health outcome gained. VIP is effective and cost-effective and should be considered in any trauma center that takes care of violently injured patients. This analysis can be used by other trauma centers to determine VIP feasiblity in their setting.

NOTES

Session: XV: Papers 55-66
Paper 57: 8:40-9:00 AM

PROSPECTIVE, MULTICENTER DERIVATION OF A CLINICAL DECISION RULE FOR THORACIC AND LUMBAR SPINE EVALUATION AFTER BLUNT TRAUMA

Kenji Inaba* MD, Lauren Nosanov BA, Jay Menaker* MD, Patrick Bosarge MD, David Turay MD, Riad Cachecho* MD, Marc DeMoya* MD, Marko Bukur* MD, Jordan Carl BS, Leslie Kobayashi* MD, Stephen Kaminski MD, Alec Beekley MD, Mario Gomez DO, Dimitra Skiada MD, And The TL-Spine Multicenter Study Group LAC+USC Medical Center

Invited Discussant: Carrie Sims, MD

Introduction: Unlike the C-Spine, where NEXUS/Canadian C-Spine Rules can be used, evidence based TL-spine clearance guidelines do not exist. The aim of this study was to develop a clinical decision rule for evaluating the injured TL-spine.

Methods: Adult (≥15yo) blunt trauma patients were prospectively enrolled at 13 US trauma centers (01/12-01/14). Exclusion criteria: C-Spine injury with neurologic deficit, pre-existing paraplegia/tetraplegia, unevaluable examination. The remaining evaluable patients underwent TL-Spine imaging and were followed to discharge. The primary endpoint was a clinically significant TL-Spine injury requiring TL-Spine Orthoses/surgical stabilization. Regression techniques were used to develop a clinical decision rule. Decision rule performance in identifying clinically significant fractures was tested.

Results: Of 12,479 patients screened, 3,068 (24.6%) met inclusion criteria [age 43.5+/-19.8 years (15-103), ISS 8.8+/-7.5, male gender 66.3%]. The majority underwent CT (93.3%), 6.3% only plain films and 0.2% MRI exclusively. TL-Spine injury was identified in 502 patients (16.4%), of which 268 (8.7%) were clinically significant. The most common clinically significant injury was compression fracture (67.4%) followed by a burst fracture (17.2%) and a fracture dislocation (5.0%). The predictive ability of clinical examination (midline tenderness, step-off or neurologic deficit), age and mechanism were examined. A positive clinical examination resulted in a sensitivity of 78.4% and specificity of 72.9%. Addition of age ≥ 60 and a high risk mechanism (MVC with ejection or rollover, pedestrian struck by auto, fall from height, torso crush, jump from moving vehicle, non-enclosed vehicle crash) increased the sensitivity to 98.5% with a specificity of 29.0%.

Conclusion: Clinical examination alone is insufficient for determining need for imaging in evaluable patients at risk of TL-Spine injury. Addition of age and high risk mechanism results in a clinical decision making rule with a sensitivity of 98.5% for clinically significant injuries. Utilization of this clinical decision rule will significantly lower the negative imaging rate.

NOTES

Session: XV: Papers 55-66
Paper 58: 9:00-9:20 AM

THE EVIL OF GOOD IS BETTER: MAKING THE CASE FOR BASIC LIFE SUPPORT TRANSPORT OF PENETRATING TRAUMA VICTIMS IN AN URBAN ENVIRONMENT

Joseph F. Rappold* MD, Kathryn A. Hollenbach Ph.D., Thomas Santora* MD, Dania Beadle BS, Elizabeth Dauer MD, Lars Sjoholm MD, Pathak Abhijit* MD, Amy Goldberg* MD, Temple University School Of Medicine Department Of Surgery

Invited Discussant: Norman McSwain, Jr., MD

Introduction: Controversy remains over the ideal way to transport penetrating trauma victims in an urban environment. Both advanced (ALS) and basic life support (BLS) transport are utilized in most urban environments depending on availability and proximity to the victim.

Methods: A retrospective cohort study was conducted at an urban Level I Trauma Center utilizing the Pennsylvania Trauma Outcomes Study Data Registry. Information on all trauma admissions from January 2008 through November 2013 with penetrating injuries that were transported by ALS, BLS or police were included. Standard demographics and injury related variables were abstracted. Patient survival by mode of transport was analyzed using logistic regression to control for confounding effects. A secondary analysis was conducted by level of care provided (ALS vs. BLS).

Results: During the study period, 1490 penetrating trauma patients were transported to our institution via ALS (44.8%), BLS (15.6%), or police (39.6%) personnel. The majority of injuries were gunshot wounds (72.9% for ALS; 66.8% for BLS; 90% for police) and occurred most frequently in males (91%). Mean transport minutes were significantly longer for ALS (17.0±7.9) than for BLS (15.7±6.7) transports ($p < 0.05$).

Descriptive Characteristics of Penetrating Trauma admissions, January 2008 – November 2013

	ALS (n = 668)		BLS (n = 232)		Police (n = 590)	
	n	**%**	**n**	**%**	**n**	**%**
ISS 0-15	362	54.2	140	60.3	268	45.4
16-30	194	29.0	64	27.6	208	35.3
31-45	48	7.2	11	4.7	53	9.0
46-60	5	0.8	1	0.4	7	1.2
61-75	59	8.8	16	6.9	54	9.2
Care: BLS	64	9.7	232	100.0	590	100.0
ALS	596	90.3	0		0	
Died prior to discharge	174	26.1	38	16.4	196	39.6
Died in ED	119	17.8	27	11.6	125	21.2
Overall Death Rate		43.9		28.0		60.8

After adjusting for transport time, among victims with an ISS of 0-30, there was a 2.3 fold increased odds of death (95% CI = 1.3, 4.1) if transported by ALS as compared to BLS. With ISS >30, this relationship did not exist (OR = 0.9; 95% CI = 0.3, 2.5). Similar relationships were observed when comparing police to BLS transport (OR = 3.4; 95% CI = 2.0, 5.9) for ISS 0-30 and OR=0.9 (95% CI = 0.3, 2.3) for ISS >30. When ALS and BLS transports were evaluated by type of care provided, patients with ISS 0-30, who received ALS support were 3.6 times more likely to die than those who received BLS support (95% CI = 2.0, 6.3). Among those with ISS >30, this relationship was not significant (OR = 1.3; 95%CI = 0.5, 3.3).

Conclusion: In an urban environment our data demonstrate that ALS care offers no survival benefit for victims of penetrating trauma regardless of severity and may lead to harm in those with moderate injury. A prospective, randomized study is warranted.

NOTES

Session: XV: Papers 55-66
Paper 59: 9:20-9:40 AM

MORTALITY FOLLOWING EMERGENCY SURGERY CONTINUES TO RISE AFTER DISCHARGE IN THE ELDERLY

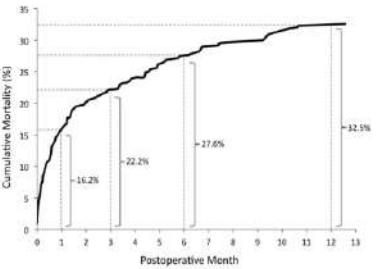
Erika L. Rangel MD, M.S., Gifty Kwakye MD, Christopher Calahan BS, Zara Cooper* MD, M.Sc., Ali Salim* MD, Mohammad Sarhan MD, Joseph Hanna MD,Ph.D.,
Brigham and Womens Hospital

Invited Discussant: George Velmahos, MD, PhD

INTRODUCTION: It is well known that the need for emergency surgery in the elderly is associated with high short term mortality. Longer term outcomes, which would be helpful for counseling patients, are not well described. For elderly patients undergoing emergency operations, we hypothesized that 30-day mortality may underestimate the true burden of operative mortality. The purpose of this study was to characterize cumulative postoperative mortality rates extending to one year following emergency abdominal surgery. In addition, we sought to identify independent preoperative predictors of one year mortality.

METHODS:This is a retrospective study of all elderly patients (age≥70) who underwent emergency general surgery for an acute abdominal condition between 2006-2011 at a major teaching hospital. Medical records were reviewed for demographic characteristics, functional status, preoperative vital signs, body mass index (BMI), laboratory values, Charlson scores, comorbid conditions, ASA classification, type of operation, transfusion, and duration of surgery. In-hospital death was determined from medical records and post-discharge death determined from the Social Security Death Index. Multivariate logistic regression analysis with stepwise selection was used to determine independent predictors of one-year mortality. The area under the receiver operator characteristic (ROC) curve was calculated to assess model performance.

RESULTS: 390 patients met our inclusion criteria. The mean age was 78.8 ± 6.1 years and 44% were men. Postoperative mortality was 16.2% at 30 days, 22.2% at 3 months ($p<0.0001$), 27.6% at 6 months ($p<0.0001$), and 32.5% at one year ($p<0.0001$), reflecting a doubling of mortality between 30 days and one year. Independent preoperative predictors of one year mortality were: Charlson score (OR 1.40; 95% CI 1.18-1.67), serum albumin (OR per unit increase in albumin: 0.51, 95% CI 0.34-0.77), BMI <18.5 (OR 4.45, 95% CI 1.06-18.78), high ASA grade (III:OR 3.18, 95% CI 1.12-9.03, IV:OR 10.74, 95% CI 3.28-35.20), immunosuppression (OR 2.49, 95% CI 1.18-5.24), and acute kidney injury (OR 3.19, 95% CI 1.50-6.78). Our composite model with these variables had excellent predictive value with an area under the ROC curve of 0.87 (95% CI 0.83-0.90).



CONCLUSION: Thirty-day mortality in the elderly emergency surgery population is an inadequate measure of postoperative outcome, as it markedly underestimates the mortality burden suffered by this cohort during the first postoperative year. We identified a constellation of preoperative clinical markers that were highly predictive of one year mortality. Further efforts are needed to explore the causes of late term mortality in the context of these variables. Future studies will develop a clinical scoring tool that can be applied at the bedside to allow more effective and pragmatic perioperative counseling for this high risk cohort.

NOTES

Session: XV: Papers 55-66
Paper 60: 9:40-10:00 AM

PREVENTING MOTOR VEHICLE CRASHES THROUGH GRADUATED DRIVING LICENSING LAWS IN MASSACHUSETTS: A POPULATION-BASED STUDY

Haytham Kaafarani MD,MPH, Catrina Cropano BS, Yuchiao Chang Ph.D., Jarone Lee MD,MPH, Toby Raybould MS, Alice Gervasini Ph.D.,RN, Laurie Petrovick CPHQ, MSc, Christopher DePesa RN, MS, George Velmahos* MD,Ph.D., Peter Masiakos MD, Massachusetts General Hospital

Invited Discussant: Barbara Gaines, MD

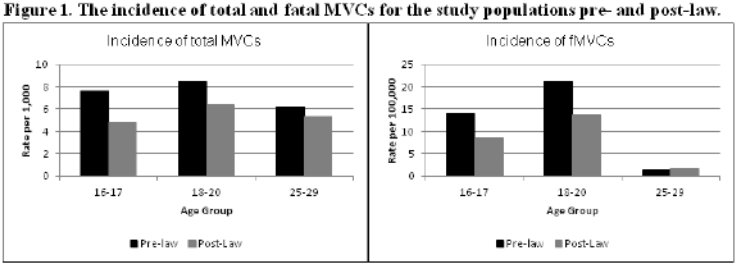
Introduction: Graduated Driving Licensing (GDL) programs phase in driving privileges for teenagers. We aimed to evaluate the effect of the 2007 GDL law on the incidence of motor vehicle crashes (MVCs) and fatal MVCs (fMVCs) among teenagers in Massachusetts.

Methods: The Fatality Analysis and Reporting System, the Missouri Census Data Center and the Massachusetts Department of Transportation databases were all used to create and compare the incidence of MVCs and fMVCs pre- (2002-2006) and post- (2007-2011) law. Three driver age groups were studied: 16-17 (evaluating the law effect), 18-20 (evaluating the sustainability of the effect), and 25-29 (control group) years old. As a sensitivity analysis, we compared the incidence rates per population and per licenses issued.

Results: MVCs decreased following the law for all three age groups (16-17: 7.6 to 4.8 per 1,000 people, $p<0.0001$; 18-20: 8.5 to 6.4 per 1,000 people, $p<0.0001$; 25-29: 6.2 to 5.2 per 1,000 people, $p<0.0001$) [Figure 1]. The subset of fMVCs decreased in the 16-17 (14.0 to 8.6 per 100,000 people, $p=0.0006$) and 18-20 (21.2 to 13.7 per 100,000 people, $p<0.0001$) age groups [Figure 1], but remained unchanged in the control group. All the results were confirmed in sensitivity analyses.

Conclusions: The 2007 Massachusetts GDL was effective in decreasing the incidence of teenager MVCs including fatal MVCs; the effect was sustainable. This study provides further support to develop, implement, enforce and maintain GDL programs aimed at preventing MVCs and their related mortality in the young novice driver population.

Figure 1. The incidence of total and fatal MVCs for the study populations pre- and post-law.



NOTES

Session: XV: Papers 55-66
Paper 61: 10:00-10:20 AM

THE EFFECT OF TISSUE DAMAGE VOLUME ON SYSTEMIC INFLAMMATION AND ORGAN FAILURE

Travis L. Frantz MS4, Scott D. Steenburg MD, Greg E. Gaski MD, Timothy Pohlman* MD, Todd O. Mckinley MD, Robert L. Reed* MD, Indiana Univesity School of Medicine

Invited Discussant: Basil Pruitt, Jr., MD

Introduction:The Systemic Inflammatory Response Syndrome (SIRS) can lead to organ failure and death in multiply injured patients (MIPs). SIRS results primarily from an immune response to endogenous molecules, Damage Associated Molecular Patterns (DAMPs) felt to be liberated from damaged tissue. However, it is not known how the magnitude of tissue damage affects the subsequent inflammatory response and organ dysfunction. This purpose of this study was to quantify how the volume of tissue damage affected the magnitude of inflammation and organ dysfunction in MIPs.

Methods:Data from 36 MIPs (ISS $\geq$ 18), admitted to the ICU for a minimum of 6 days, were used to calculate daily SIRS scores (0 to 4) and daily Sequential Organ Functional Assessment scores (SOFA; 0 - 24). A novel radiographic index, the Tissue Damage Volume Score (TDVS), was calculated by making volumetric measurements of every injury in each patient detected on admission CT scans and plain X-rays. Individual injury volumes were summed to generate a total body TDVS for each patient. Regression analyses evaluated correlations between TDVS and both inflammatory and organ dysfunction scores.

Results: Two distinct patient populations were identified comparing TDVS to organ dysfunction and SIRS. High-risk patients: SOFA = 0.0043 TDVS + 3.72; Cumulative SIRS = 0.031TDVS + 22. Low-risk patients: SOFA = 0.0006 TDVS + 2.68; Cumulative SIRS = 0.014TDVS - 6.1. SOFA vs. TDVS slope was 7.2X higher in high-risk patients ($p = 0.0007$) and SOFA vs. Cumulative SIRS slope was 2.2X higher in high-risk patients ($p = 0.002$) compared to low-risk patients.

Conclusion: These results demonstrate a dichotomous response of how MIPs tolerate tissue damage. High-risk patients developed over twice the magnitude of inflammation per tissue damage volume compared to low-risk patients. The accentuated inflammatory repsonse extrapolated into a 7X increase in the amount of organ dysfunction per tissue damage volume. Further investigations are required to elucidate the underlying pathomechanistic pathways on how tissue damage causes inflammation and organ dysfunction.

NOTES

Session: XV: Papers 55-66
Paper 62: 10:20-10:40 AM

DOES SIZE MATTER, OR IS EXPERIENCE WHAT REALLY COUNTS? ANNUAL CLINICAL EXPERIENCE (PER SURGEON) IS ASSOCIATED WITH IMPROVED SURVIVAL

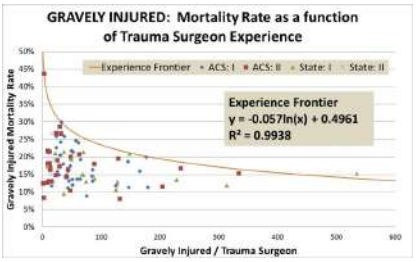
Richard W. Doty Jeffrey S. Young* MD, James F. Calland* MD, University of Virginia

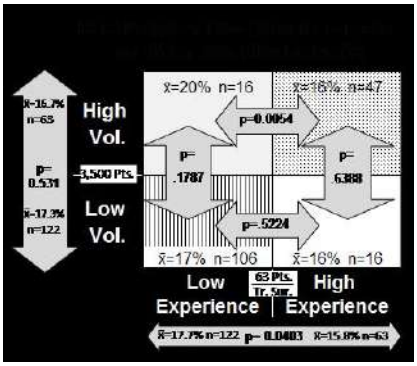
Invited Discussant: Joseph Minei, MD

Introduction: Severely injured trauma patients derive a clear mortality benefit from treatment at trauma centers, but the relationship between trauma center volume and outcome is less well understood. This study seeks to discern the relationship between trauma center volume and trauma surgeon "experience" in patients with varying severity of anatomic injury.

Methods: Patient outcomes for all adult blunt-trauma patients 16-64 years old in the combined 2011 and 2012 NSP Datasets (n=1,200,439 admissions) were cross-tabulated against facility size, verification status, trauma surgeon experience (patients treated per surgeon / year stratified by injury severity score level), and injury severity score (ISS). Data were evaluated and analyzed using two-tailed Student's t-tests and r2 tests of correlation using Microsoft excel.

Results: Trauma centers in which surgeons were annually responsible for > 63 ISS25+ patients per year tended to demonstrate mortality rates that were 12% lower (15.8% vs. 17.7%, $p<0.05$) than centers with surgeons that have a diluted annual "experience" of <63 patients per year. In low volume centers (<3,500 admissions per year) low "experience" did not seem to influence the mortality of ISS25+ patients (16% vs. 17%, $p=0.52$). In high volume centers, however (>3,500 patients treated annually) high experience seemed to result in a 20% lower annual relative observed mortality rate (20% vs. 16%, $p<0.01$). At high experience, increasing facility volume seemed to exert no influence on mortality (16% vs 16%, $P=0.31$).





Conclusions: Trauma surgeon annual "experience" may positively influence the mortality risk of gravely injured patients. Increasing annual center volume, on the other hand, does not improve performance. High "experience" centers seem to produce unexpected survivors, especially in high volume centers that admit >3,500 patients per year.

NOTES

Session: XV: Papers 55-66
Paper 63: 10:40-11:00 AM

The clinical significance of solubule RAGE in patients with severe sepsis

Hisatake Matsumoto MD, Naoya Matsumoto Ph.D., Hiroshi Ogura* Ph.D., Junya Shimazaki Ph.D., Kazuma Yamakawa MD, Takashi Muroya MD, Junichiro Nakagawa MD, Tomoki Yamada MD, Takeshi Shimazu* Ph.D., Department Of Traumatology And Acute Critical Medicine, Osaka University Graduate School Of Medicine

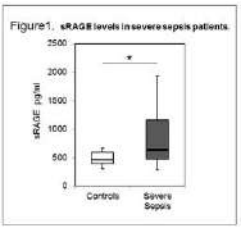
Invited Discussant: Eileen Bulger, MD

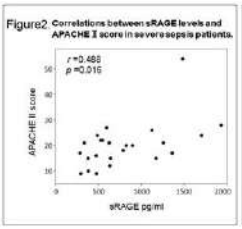
Introduction: Severe sepsis is a major clinical challenge, especially to clinicians working in intensive care units. The receptor for advanced glycation end products (RAGE) is a pattern- recognition receptor, which is involved in the pathogenesis of several inflammatory disease. RAGE has secretory isoforms referred to as soluble RAGE (sRAGE). The role of sRAGE has not been thoroughly clarified in the pathogenesis of critically ill patients. The objective of this study was to investigate circulating sRAGE in patients with sepsis.

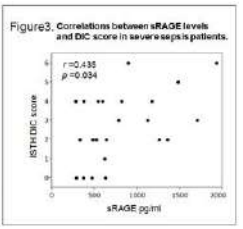
Methods: This prospective observational study was conducted from November 2012 to September 2013. The criteria for inclusion were patients with severe sepsis and age greater than 18 years. Blood samples were collected from patients within 24 hours after the diagnosis of sepsis and from healthy volunteers. sRAGE, high-mobility group box 1 (HMGB1), interleukin 6 (IL-6), and Plasminogen activator inhibitor-1 (PAI-1) were measured with an enzyme-linked immunosorbent assay kit. APACHE II score and SOFA score were assessed at the enrollment time of sepsis patients. We used ISTH overt DIC diagnostic criteria algorithm for assessing DIC.

Results: During the study, 24 sepsis patients and 12 healty volunteers were included. Sepsis patients and controls were similar with respect to age and sex. In the overall analysis of participants, sRAGE levels in the serum were significantly increased in sepsis patients compared with healthy controls ($p<0.05$) (Figure.1). Significant correlations were found between sRAGE levels and APACHE II score (Figure.2) ($p<0.05$), as well as between sRAGE levels and SOFA score ($p<0.05$). sRAGE levels showed significant dependency on ISTH DIC score (Figure.3) ($p<0.05$). Significant correlations were found between sRAGE levels and IL6 levels, as well as between sRAGE levels and PAI-1 levels ($p<0.05$). sRAGE levels also had a tendency to correlate with HMGB-1 levels.

Conclusion: We demonstrated for the first time that sRAGE increased with the progression of DIC in the sepsis patiants, suggesting that sRAGE may reflect the severity of coagulative activity. We also found that sRAGE showed a correlation with APACHE II and SOFA score, suggesting the possibility that sRAGE may play a role as a new sepsis marker.







NOTES

Session: XV: Papers 55-66
Paper 64: 11:00-11:20 AM

The Pediatric Trauma Center and the Inclusive Trauma System: Impact on Splenectomy Rates

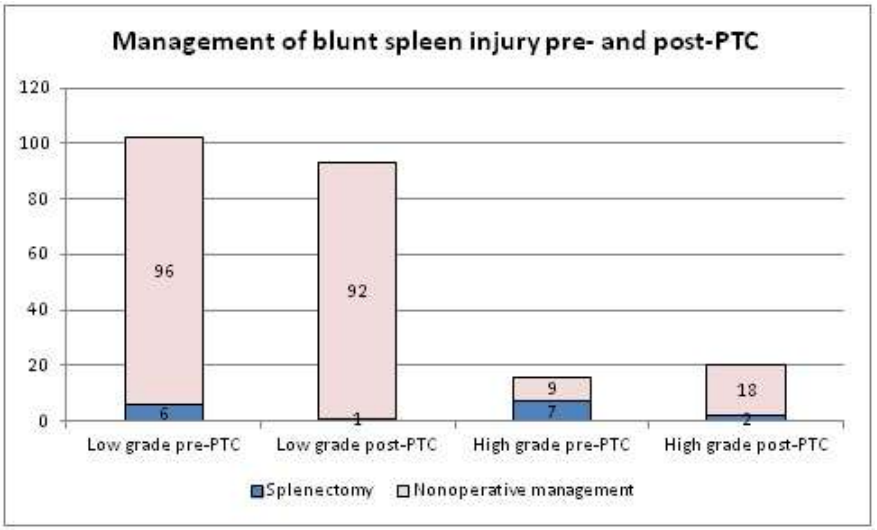
Emily E. Murphy MD, Stephen G. Murphy MD, Mark D. Cipolle* MD,Ph.D., Glen H. Tinkoff* MD, Christianacare Health Services

Invited Discussant: Mary Fallat, MD

Introduction: Prior to 2006, the Delaware Trauma System (DTS) did not include a designated pediatric trauma center (PTC). In 2006, AI DuPont Hospital for Children, a free-standing children's hospital, was designated by the DTS and verified by the ACS Committee on Trauma verification/ consultation program as a PTC. We sought to assess the impact of the addition of this PTC into the pre-existing trauma system on splenectomy rates.

Methods: The DTS trauma registry was queried for all children younger than 16 years of age with spleen injury (ICD9 diagnoses codes 865.0-865.5) from January 1998 through December 2012. This cohort was categorized into two groups, pre-PTC (1998-2005) and post-PTC (2006-2012). Penetrating injuries were excluded. These groups were compared for age, gender, length of stay, organ specific injury grade, injury severity score, incidence of polytrauma, splenectomy rate, and admitting hospital. Management, operative versus nonoperative, of low grade (OIS 1-3) and high-grade (OIS 4-5) were also compared. Pearson's chi-square analysis was performed for categorical variables. Continuous variables were reported as mean +/- standard deviation and compared by Student's t-test for independent normally distributed samples. Mann-Whitney U test was used for non-normally distributed variables. A p value of <0.05 was considered significant.

Results: Of the 231 pediatric spleen injuries, 118 occurred pre-PTC and 113 occurred post-PTC. There were no significant differences in age, gender, length of stay, ISS, OIS grade and incidence of polytrauma. Splenectomy rate decreased from 11% pre-PTC to 2.7% post-PTC (13 vs. 3, p=0.014).



Conclusion: The addition of an ACS-verified PTC within an inclusive trauma system that was previously without one was associated with a significant reduction in the rate of blunt-trauma-related splenectomy. Integration of a verified PTC is an influential factor in achieving spleen-preservation rates equivalent to published APSA benchmarks within a trauma system.

NOTES

Session: XV: Papers 55-66
Paper 65: 11:20-11:40 AM

EMERGENCY GENERAL SURGERY OUTCOMES IN TEACHING VERSUS NON-TEACHING HOSPITALS

Adil A. Shah MD, Syed N. Zafar MD,MPH, David T. Efron* MD, Zain G. Hashmi MD, Elliott R. Haut* MD, Eric B. Schneider Ph.D., Diane Schwartz MD, Catherine G. Velopulos* MD, Adil H. Haider* MD,MPH, Johns Hopkins School of Medicine

Invited Discussant: Gregory J. Jurkovich, MD

Introduction: Prior analyses demonstrate teaching hospitals to have worse outcomes and higher costs, raising concerns for quality of care. The purpose of this study is to compare outcomes between teaching and non-teaching hospitals for emergency surgical conditions in a national sample.

Methods: The Nationwide Inpatient Sample (2005–2011) was queried for patients with conditions encompassing EGS as determined by the American Association for Surgery of Trauma (AAST) Committee on Severity Assessment and Patient Outcomes in 2011 and grouped into the 24 different EGS categories. Outcomes of in-hospital mortality, length of stay and cost of care were compared between patients presenting to teaching versus non-teaching hospitals. Both groups were matched on age, gender, clinical diagnosis, comorbidities, disease diagnosis, disease severity, payer status and insurance utilizing propensity scores. Multivariate regression analyses were performed further adjusting for hospital level factors including EGS volume.

Results: A total of 3,707,465 patient were included. 41% (n=1,520,358) of patients were treated at teaching hospitals. After propensity matching and adjustment, there were no significant differences in the overall mortality, LOS and cost between teaching and non-teaching hospitals (Table). Similar results were observed for rural and urban healthcare facilities.

	Overall	Urban Hospitals	Rural Hospitals
	Mortality (OR [95% CI])		
Teaching Hospitals (Non-teaching as referent)	1.04 (1.02-1.06)	1.04 (1.02-1.06)	1.08 (0.99-1.16)
	Length of Stay (Days with 95%CI)		
Teaching Hospitals	5.03 [4.98-5.09]	5.14 [5.08-5.21]	4.37 [4.3-4.44]
Non-teaching Hospitals	5.22 [5.16-5.29]	5.31 [5.26-5.38]	4.87 [4.59-5.16]
	Cost ($ with 95% CI)		
Teaching Hospitals	12,304 [12,290-12318]	12,507 [12,492-12,523]	11,004 [10,974-11,034]
Non-teaching Hospitals	12,846 [12,827-12,865]	13,078 [13,059-13,098]	11,237 [11,135-11,340]

[OR; Odds Ratio, CI; Confidence Intervals]

Conclusion: National estimates of outcomes for EGS conditions demonstrate comparable results between teaching and non-teaching hospitals. Concerns regarding quality of care and health care costs at teaching hospitals are unfounded.

NOTES

Session: XV: Papers 55-66
Paper 66: 11:40-12:00 PM

PNEUMOMEDIASTINUM FOLLOWING BLUNT TRAUMA: WORTH WHILE A WORKUP?

Konstantinos Chouliaras MD, Elias Bench BS, Peep Talving* MD,Ph.D., Aaron Strumwasser MD, Elizabeth Benjamin MD,Ph.D., Lydia Lam* MD, Kenji Inaba* MD, Demetrios Demetriades* MD,Ph.D., LAC+USC Medical Center

Invited Discussant: Oscar Guillamondegui, MD

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction: Pneumomediastinum is a common radiological finding following blunt upper torso injury, however, its clinical significance is poorly defined. The purpose of this study was to define the incidence of aerodigestive injuries in patients with pneumomediastinum after blunt upper torso injury.

Methods: After IRB approval, a retrospective review of all blunt chest and neck trauma patients admitted to Level I urban trauma center between 1/2007 and 12/2012 was performed. All patients with pneumomediastinum on radiological investigations were enrolled. Data accrued included demographics, admission clinical data, injury severity patterns, incidence of aerodigestive injuries, operative findings, morbidity, mortality, and ICU and hospital lengths of stay.

Results: A total of 9,946 patients were studied. Overall, 258 patients (2.6%) had a pneumomediastinum, 65 (25%) and 193 (75%) diagnosed on a chest x-ray or on a computed tomography (CT) scan, respectively. Almost half of the patients (49%) were injured in motor vehicle collisions with 76% being male. A total of 21 patients (8.1%) had a workup including bronchoscopy, esophagogram or esophagoscopy. Overall, 4 aerodigestive lesions (1.6%) were diagnosed. Three tracheobronchial injuries were identified on CT scan, and one esophageal injury was diagnosed on an esophagogram. Two of the tracheobronchial injuries required surgery while the remaining aerodigestive lesions were managed conservatively. The overall mortality in this cohort was 10.9%. After adjusting for significant confounders age, Injury Severity Score, and Glasgow Coma Scale Score were found to be independent predictors of mortality.

Conclusion: Pneumomediastinum is a poor predictor of aerodigestive injury following blunt trauma. Selective workup in this clinical setting is warranted.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

NOTES

POSTERS

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Poster # 1

EFFECT OF TRAUMA SPLENECTOMY ON LONG TERM RISK OF DEATH DUE TO INFECTIONS

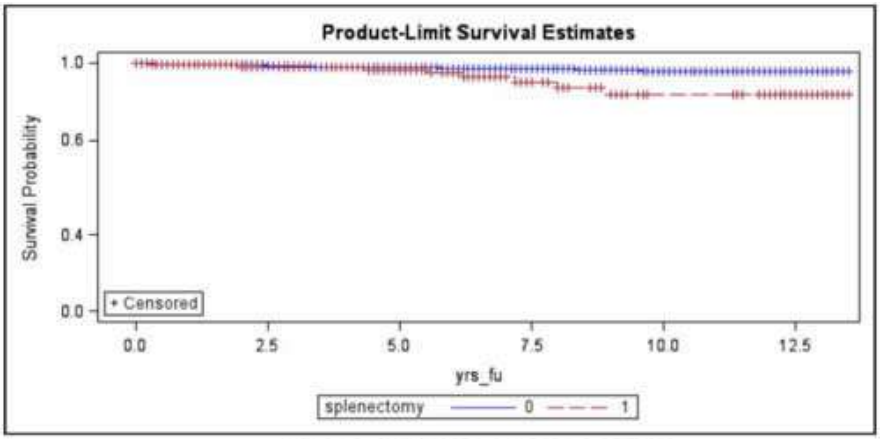
Gabriel E. Ryb* MD,MPH, Gordon S. Smith MD,MPH, Patricia C. Dischinger Ph.D., Kimberly Auman MS, Joseph A. Kufera MA, Thomas M. Scalea* MD, Baltimore C-STARS/ R Adams Cowley Shock Trauma Center

Introduction: Long term outcomes after splenectomy in injured adults have not been clearly determined. We ascertained whether splenectomy after trauma in adults increases the risk of post-discharge long-term natural death.

Methods: Adults with blunt splenic injury admitted to a Level I Trauma Center between July 1995 and December 2008 were identified in the trauma registry. Determination of death, cause of death and date of death were obtained from the National Death Index through 12/31/2008. Differences in the 30 day-post-discharge mortality (natural, infectious related and non-infectious natural causes) in relation to splenectomy were determined by comparing Kaplan-Meier survival curves and using the log rank test. Cox proportional hazard regression was used to adjust for confounders (age, gender, injury severity, race and alcohol use) (alpha=0.05).

Results: 2589 patients with blunt splenic injury were examined. Subjects who died during the initial hospitalization (n=327) and within 30 days after discharge (n=296) were excluded. Those treated with splenorrhaphy (n=67) were excluded due to their small number, leaving a total of 2176 (267 with and 1890 without splenectomy) for analysis. 164 deaths occurred during the 12.5 year follow up period (median 5.1 years). 119 died from natural causes, 22% of which (n=26) were due to infection. Survival analysis revealed that those with splenectomy had significantly higher natural and infectious cause related mortality (p= 0.004 and 0.01, respectively), but no effect with respect to non-infectious natural causes (p=0.06). Cox proportional hazard models adjusting for age, gender, race, ISS and alcohol use revealed significant associations of splenectomy with natural death [OR 1.70 (1.07-2.72)] and with infection related deaths [OR 2.50 (1.02-6.16)].

Conclusion: Injured patients treated with splenectomy have an increased long term natural cause mortality that, in part, is attributable to infections. Splenic preservation should be attempted where possible. Patients should be aware of the increased risk of infection related mortality after splenectomy. Interventions to reduce this increased risk need to be studied.



INFECTION RELATED MORTALITY

Poster # 2

SELF EXPANDING POLYURETHANE FOAM FOR THE TREATMENT OF ABDOMINAL EXSANGUINATION: UNANSWERED QUESTIONS

David R. King* MD, Adam Rago BS, Andreas Larentzakis MD, John Marini BS, Michael J. Duggan DVM, John Beagle BS, Greg Zugates Ph.D., Rany Busold Ph.D., Marc Helmick BS, George Velmahos* MD,Ph.D., Peter Fagenholz MD, Upma Sharma Ph.D., Massachusetts General Hospital

Introduction:Noncompressible abdominal bleeding is a significant cause of preventable death with no effective pre-surgical therapies. We have previously described the hemostatic efficacy of a percutaneously-administered, self-expanding polyurethane foam in several lethal hemorrhage models. Prehospital diagnosis of severe abdominal hemorrhage, use in the presence of a diaphragmatic injury, effects on spontaneous respiration, efficacy after prolonged shelf life and at temperature extremes remain important unanswered questions.

Methods:: Experiment (Exp.) 1: Across a range of efficacy models, diagnostic blood aspiration was attempted through a Veress Needle at the umbilicus prior to foam deployment. Exp. 2: Foam was deployed in naïve, spontaneously breathing animals. Effects on PCO2 and respiration were characterized. Exp. 3: In a model of lethal hepatoportal hemorrhage, a one cm2 full thickness injury was created in the left diaphragm. Foam was deployed ten minutes after injury (n=6) or animals received fluid resuscitation alone (n=6) and were monitored. Exp. 4: In the same model, foam was delivered with a robust, field relevant delivery system (n=5), after conditioning at 50°C for two months to simulate one year shelf life (n = 6), or after conditioning to 10°C (n=6) or 50°C (n=6) and compared to controls (n=12).

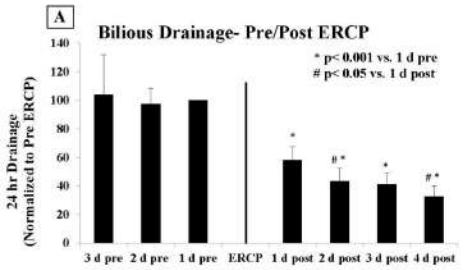
Results:Foam was tested in 76 swine across these studies. Exp.1: Foam was tested over a range of injury models in more than 40 animals. Blood was successfully aspirated from a Veress Needle in 70% of lethal iliac artery injuries and 100% of lethal hepatoportal injuries. Blood aspiration was unsuccessful (0%) when less than 500 mL of intraperitoneal blood was present. Exp. 2: Animals were allowed to breathe spontaneously following foam treatment. Hypercarbia was observed: PCO2 was 48 ± 9.4 mmHg at baseline and 65 ± 14 mmHg at 60 minutes. Exp. 3: When deployed in the presence of a diaphragm injury, between 0 and 50cc of foam was found to migrate through the diaphragmatic injury. Foam treatment resulted in a significant survival benefit relative to the control group at one hour ($p=0.03$). Exp. 4: Foam delivered from a fieldable delivery system improved survival relative to the control group ($p=0.006$). Foam efficacy was maintained under simulated one year shelf life conditions: 3hr survival was 83% ($p<0.05$). Foam treatment also resulted in a significant benefit when conditioned to operational temperature extremes: 3hr survival was 83% and 67% in the low and high temperature groups, respectively ($p<0.05$). Temperature extremes did not result in hypothermia-related coagulopathy or thermal damage.

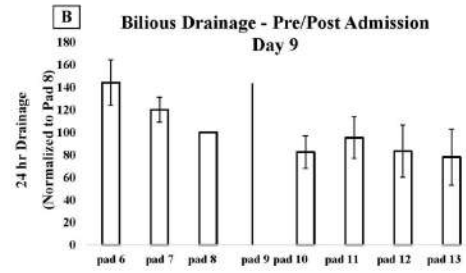
Conclusion:Blood aspiration through a Veress Needle may be an effective diagnostic technique to confirm peritoneal access, confirm massive hemorrhage at point of injury, as well as prevent inappropriate foam use for non-massive hemorrhage. Foam therapy remains an effective pre-surgical hemostatic intervention under a variety of possible field conditions.

Poster # 3

SHOULD ALL BILE LEAKS AFTER LIVER INJURY BE TREATED WITH ERCP DECOMPRESSION?

Rachel Eisenstadt BA, Nicole Krumrei MD, Joshua Marks MD, Michael Kochman MD, Gregory Low Steven Allen MD, Niels Martin* MD, Daniel Holena* MD, C. William Schwab* MD, Patrick Reilly* MD, Jose Pascual* MD,Ph.D., University of Pennsylvania





Introduction: Certain patients with complex liver injuries develop persistent bile leaks managed with perihepatic drains placed either at laparotomy or percutaneously. Endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy +/- stent may reduce biliary sphincter resistance promoting forward bile flow, yet no study to date has evaluated its effect on daily bile leakage after liver injury. We hypothesized that ERCP in patients with liver injury and persistent bilious leakage would reduce drainage volume and time to drain removal.

Methods:Adult patients with liver injuries from a level 1 trauma center (06/01/2002 to 06/01/2013) were retrospectively reviewed. Patients with perihepatic drains found to contain bile were divided into those who did or did not undergo ERCP. Patients who died within 48 hours of presentation, had an ERCP performed < 4 days after admission, for whom drainage was non-bilious or where volume data was missing were excluded. Daily drainage volumes (ml/24 hrs) were collected from 3 days after admission until drain removal or patient discharge. Demographics, time to drain removal and length of stay (LOS) were recorded. The Students t-test was used to compare means with $p<0.05$ deemed significant.

Results: 1238 liver injury patients were identified of whom 58 (4.7%) met inclusion criteria for persistent bile leakage. 31 (53.4%) underwent ERCP (mean: 9 days post-injury) and 27 (46.6%) were managed expectantly (NoERCP). ERCP patients were younger (27±1 vs. 34±2 yrs, $p=0.03$) and more often had penetrating injuries (100 vs. 76.9%, $p<0.01$). Both groups had similar gender, AIS-abdomen scores and mortality. Bilious drainage decreased by half the day after ERCP and decreased further in subsequent days ($p<0.05$)(Fig A). Drainage in the non-ERCP group remained unchanged over the same post-injury time frame (Fig B). Time to drain removal (46.8±12.1 vs. 43.6±7.5 days, $p=0.82$) and hospital LOS (27±3.9 vs. 26±4.5 days, $p=0.83$) were similar in NoERCP and ERCP groups.

Conclusion: ERCP is highly effective in decreasing daily bilious drain output after liver injury. Nonetheless, this does not appear to translate into faster drain removal or patient discharge. Prospective investigation is needed to determine in which injured populations this can expedite bile leak resolution and recovery from complex liver injuries.

Poster # 4

THE POTENTIAL UTILITY OF DIRECT TRANSFERS FROM THE PREHOSPITAL SETTING TO THE HYBRID OPERATING ROOM: HOW OFTEN WOULD WE GET IT RIGHT?

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Introduction: Prehospital hypotension following injury often leads to sustained hypotension within the trauma bay. Given that patient dispositions and therefore treatment plans are time critical with regard to both ongoing hemorrhage and/or end organ ischemia, selecting the appropriate destination (operating theater, angiography suite, intensive care unit) can be extremely challenging in some clinical scenarios. Although the rapidity of these decisions are based on clinical acumen, pattern recognition, and clinician experience, it is clear that the initial destination of choice is not infrequently incorrect. The purpose of this study was to define the flow and interventions of persistently hypotensive injured patients with regard to their initial destination of choice after leaving the trauma bay (including patients who required multiple treatment destinations).

Methods: The Alberta Trauma Registry and chart reviews were employed to perform a retrospective cohort study describing the route and flow of all persistently hypotensive (2 or more systolic blood pressures (sBP) less than 90 mmHg) severely injured ($ISS \geq 12$) patients (1995 to 2012) over their first 24 hours of their admission to a level-one, tertiary care trauma referral center. Standard statistical methodology was utilized ($p<0.05$).

Results: Of 913 patients with an initial sBP less than 90 mmHg (prehospital, referring hospital, or initial trauma bay reading), 56% remained persistently hypotensive on subsequent trauma bay measurements. These patients had a mean age of 41 years, were 73% male, and sustained blunt injury mechanisms in 87% of cases. Of these persistently hypotensive patients, 53% were transferred directly to the operating theater, 29% to the intensive care unit, 13% to the trauma ward after resuscitation and diagnostic imaging (i.e. responders), and 5% to the interventional angiography suite. Of all hypotensive patients, 68% underwent cross sectional imaging with computed tomography either before or after initial transfer from the trauma bay. Of the patients who were transferred to the operating theater, 64% were subsequently transferred to the intensive care unit, 23% to the trauma ward and 14% died within the theater itself. Within the operating theater, 99% of patients underwent an operative intervention (194 (79%) laparotomies). A total of 7% of patients required both emergent operative and angiographic intervention. Although varied, these were most commonly patients with ongoing hemorrhage secondary to pelvic fractures or major hepatic lacerations. The mean hospital length of stay was 24 days with an associated mean intensive care unit stay of 7 days. The overall mortality of all persistently hypotensive patients was 22% within the first 24 hours.

Conclusion: This descriptive study at a high volume, level-one, tertiary care trauma referral center confirms that the majority of persistently hypotensive patients are transferred directly from the trauma bay to the operating theater for operative intervention. At least 7% of patients in this cohort would benefit from the efficiency of a single hybrid operating theater such as the RAPTOR (Resuscitation with Angiography, Percutaneous Therapy and Operative Repair) suite. Given the high percentage of patients with an initial prehospital hypotensive blood pressure measurement that normalized with a second reading and/or short resuscitation (i.e. responders), direct transfer of patients from ground or rotary wing ambulance bays to the RAPTOR suite must remain based on experienced clinical acumen and patient assessment.

Poster # 5

UNSTABLE HEMODYNAMICS DO NOT ALWAYS MAKE COMPUTED TOMOGRAPHY SCANS UNFEASIBLE IN THE MANAGEMENT OF PATIENTS WITH MULTIPLE TORSO INJURIES

Chih-Yuan Fu MD, Chih-Po Hsu MD, Department Of Trauma And Emergency Surgery, Chang Gung Memorial Hospital

Introduction:

Torso computed tomography (CT) scans have been used worldwide to evaluate patients with multiple torso injuries. However, torso CT scans have traditionally been considered as a part of a secondary survey that can only be performed after hemodynamics have been stabilized. In this study, we attempted to evaluate the role of the CT scan in managing patients with unstable hemodynamics.

Methods:

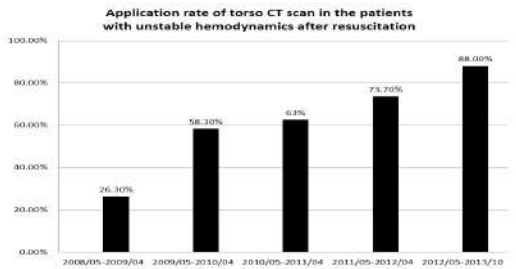
Patients who fulfilled the criteria of major torso injuries in our institution were treated according to the Advanced Trauma Life Support guideline. The selection of diagnostic modalities for patients with stable and unstable hemodynamics was discussed. Furthermore, patients with unstable hemodynamics who received hemostasis procedures were the focus of our analysis. We also delineated the influence of CT scans on the time interval between arrival and definitive treatment for these patients.

Results:

During the study period, 909 patients were enrolled in this study. Ninety-one patients (10.0%, 91/909) had a systolic blood pressure (SBP) less than 90 mmHg after resuscitation. Fifty-eight of the patients (63.7%) received torso CT scans before they received definitive treatment. There was no significant difference in the application rates of torso CT scan evaluation between patients with unstable and stable hemodynamics (63.7% vs. 68.8%, p=0.343). Among the 79 patients with unstable hemodynamics who underwent a hemostasis procedure (surgery or angioembolization), there was no significant difference in the time between arrival and definitive hemostasis between the patients received torso CT scans and those who did not (surgery: 57.8±6.4 vs. 61.6±14.5 minutes, p=0.218; angioembolization: 147.0±33.4 vs. 139.3±16.7 minutes, p=0.093)

Conclusion:

The traditional priority of diagnostic modalities used to manage patients with torso injuries should be reconsidered because of advancements in facilities and concepts. With shorter scanning times and transportation distances, unstable hemodynamics do not always make performing a CT scan unfeasible.



Poster # 6

POST-RESECTION PANCREATIC DYSFUNCTION IN TRAUMA: 18-YEAR REPORT FROM A LEVEL I TRAUMA CENTER

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Introduction: Post-resection pancreatic dysfunction in trauma is not well characterized. The aim of this study was to examine the incidence of new-onset endocrine and exocrine dysfunction following traumatic pancreatic resection.

Methods: After IRB approval, all patients sustaining a pancreatic injury from 1/96-12/13 were identified. Patients with preexisting diabetes were excluded. Survivors were divided into three groups according to the extent of anatomic resection- distal, head or total pancreatectomy. Endocrine function including blood glucose levels, insulin requirements and discharge medications were abstracted from clinical records and analyzed.

Results: During the 18-year period, 331 (0.5%) trauma admissions presented with a pancreatic injury; 109 (32.9%) required pancreatectomy and 84 (77.1%) survived to hospital discharge. Four (4.8%) were excluded: 3 incomplete charts and 1 for preexisting diabetes. Of 80 cases, 73 (91.3%) received distal resection, 7 (8.8%) head and none underwent a total pancreatectomy. Distal resection mean age was 27 years (8-62), ISS 23 and BMI 27. Thirty-eight (52.1%) required insulin postoperatively, with the majority (47.4%) requiring insulin for ≤1 day; no patients were discharged on insulin. Head resection mean age was 29 years (17-45), ISS 28 and BMI 32. Six (85.7%) required insulin postoperatively and 28.6% were discharged on insulin. For both distal and head resections, none had evidence of exocrine dysfunction or required pancreatic enzyme supplementation at discharge.

Conclusion: Exocrine dysfunction following distal or head pancreatectomy for trauma is rare. The incidence of new-onset endocrine dysfunction following traumatic distal pancreatectomy is rare, but not uncommon following head resection.

Poster # 7

DESCRIBING COMPLICATIONS OF NON-THERAPEUTIC LAPAROTOMY USING THE COMPREHENSIVE COMPLICATION INDEX

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Introduction: The reported high morbidity rates of non-therapeutic laparotomy (NTL) in trauma has shifted practice toward more conservative management of suspected abdominal injury. Complications associated with the procedure are frequent and typically are reported as an overall complication rate. The Clavien-Dindo Grading System is used to grade complications by severity. The Comprehensive Complication Index (CCI) generates for each patient a weighted sum of all complications on a scale from 0 to 100. These tools were applied to a cohort of trauma patients who underwent non-therapeutic laparotomy.

Methods: The registry of a single level one trauma center was used to identify patients with NTL, either as their only major procedure (NTL group) or as one of several procedures (NTL+). Complications during admission were tabulated and scored using Clavien-Dindo grades, and then used to calculate a CCI score. Patients who died in <8hours, had incomplete records, or had laparotomy in the emergency room were excluded.

Results: 54 patients were identified, 36 had NTL only and 18 had NTL as well as additional operations (NTL+). The complication rate for NTL patients was 33% with a mean CCI score of 6.4 +/- 2.0; the complication rate for the NTL+ cohort was 72% with a mean CCI score of 29.6 +/- 6.8. Increases in injury severity correlated with increased complication rates and mean CCI (Table 1).

Conclusion: Overall morbidity rate was high after NTL, consistent with previous reports. However, analysis with more sophisticated grading tools suggests that the inpatient complication burden of isolated NTL may be relatively low.

	N	Complication Rate (%)	Grade I	Grade II	Grade III	Grade IV	Grade V	Mean CCI
All	54	46	11	11	11	6	1	14.1
NTL	36	33	6	5	0	2	0	6.4
NTL+	18	72	5	6	11	4	1	29.6
ISS								
< 9	31	35	7	4	0	2	0	6.7
9 - 15	10	40	3	1	1	1	0	10.8
> 15	13	69	1	6	10	3	1	34.2

Table 1. Complication rate, Clavien-Dindo classification of complication severity, and mean CCI for NTL and NTL+ along with increasing ISS. ISS: injury severity score.

Poster # 8

INTRAOPERATIVE FEEDING OF THE BURNED PATIENT IS SAFE AND EFFICACIOUS

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Introduction:Severely burned patients are hypermetabolic and hypercatabolic and can remain so for upto one year following injury. The goal of nutrition is to provide adequate calories, protein, and micronutrients to meet their ever-evolving metabolic needs. Historically, enteral nutrition is withheld in the perioperative period for aspiration concerns. As a result, burn patients who require multiple operative debridements and skin grafting procedures risk accumulating significant caloric deficit. We describe our burn center's experience with intraoperative feeds.

Methods:Case series describing a single-institution experience of intraoperative feeding in burn patients with major thermal injuries defined as a cutaneous burn ≥ 20% total body surface area (TBSA). In patients with a tube-secured airway, a large bore salem sump nasogastric tube and a smaller bore feeding tube were placedwith enteral feeds initiated within 18 hours of ICU admission. All patients with major thermal injury undergoing surgery had tube feeds continued intraoperatively provided they had a secure airway and radiographic confirmation of a post-pyloric feeding tube. For those with only a gastric feeding tube, feeding was stopped and the stomach decompressed with a salem sump tube on call to the operating room. These patients received continuous intraoperative tube feeds at the discretion of the attending anesthesiologist. Any patient undergoing surgical tracheostomy was NPO for 8 hours prior to surgery. Post operatively, tube feeds were re-initiated within 2 hours of return from the OR at the previously tolerated rate. Oxandrolone and beta-blockers were also given in patients who met requirements in both groups.

Results:Sixteen patients with a total of 116 trips to the operating room without intraoperative feeding were matched by TBSA to 16 patients with a total of 118 trips to the operating room with intraoperative feeding. The primary outcome of days to goal tube feeds was on average 3 days less for those who received intraoperative feeding (3.4 verses 6.2 days). The total percent of prescribed caloric and protein needs achieved during the hospital stay was also higher in those patients what were fed intraoperatively. With no intraoperative feeding the percent of goal caloric and protein needs that were achieved was 88 and 86, respectively, and increased to 96 and 97 percent, respectively, in those patients who were fed intraoperatively. There were no intraoperative aspiration events or regurgitations events as documented by the anesthesiologist in all 112 non-tracheotomy surgeries. This is the first time patients were fed intraoperatively in the prone and lateral positions with no adverse effects. Pre-albumin increased by 48% in those who were not fed intraoperatively and by 61% in those who were fed. CRP values decreased 25% in the patients who were not fed intraoperatively and 41% in those who were. The total LOS, ICU days, and ventilator days, on average, also decreased in the group that was fed intraoperatively. (58 to 53, 49 to 45, and 45 to 33).

Conclusion:Intraoperative nasoduodenal feeding can be administered safely in supine, prone and lateral positions in severely burned patients with a tube-secured airway. Early initiation and continuation of feeds has led to reaching goal caloric needs at half the number of days and achieving overall higher percentages of their goal prescribed needs. This has shown improvement in prealbumin and CRP levels. Trends toward decreased total LOS, total ICU days, and ventilator days can also be seen possibly due to the improvement in nutritional support.

Poster # 9

IMPACT OF TRAUMA ON BURN MORTALITY

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Introduction: Factors influencing mortality in burns and trauma have been evaluated independently, but they are rarely evaluated together. Outcomes in combined burn/trauma injuries have assumed increased importance due to recent military conflicts. The purpose of this study is to assess the additional impact of trauma on burn outcomes.

Methods: We analyzed the American Burn Association National Burn Repository (NBR) 2009 release containing of 286,293 admissions. Analysis was restricted to years after 2000. Records missing mortality, age, burn size (total body surface area-TBSA), inhalation injury, or length of hospital stay, as well as readmissions, non-burn injuries, and duplicates were eliminated from analysis. We used generalized estimating equation with a logistic regression to model the effect of trauma, burn size, inhalation injury and age on mortality. We fit a series of models consisting of main effects and two-way interactions for Burn Only and for Burn/Trauma. Two-way interactions were analyzed for TBSA, inhalation injury, and age for Burn and Burn/Trauma. Models were compared on the significance of predictors ($p < 0.05$) and QICu values.

Results: Of the 95,579 records meeting screening requirements, 2,868 patients had documented trauma/burn injuries. Comparing the parameter estimates from all models revealed: 1. Burn Only Model: mortality was dependent on burn size, age and inhalation injury 2. Burn/Trauma Model: trauma increased mortality ~62%. On two-way interaction, trauma increased mortality, with magnitude dependent on burn size and age. Similarly, inhalation injury increased mortality, with magnitude of effect depenent on burn size and age. (Table 1) Without trauma, the odds ratio increased 2.21 for every 10 year increase in age; with trauma this increase is only 1.87. For inhalation injury, the odds ratio increased 1.05 for every 10 year age increase with inhalation injury and 2.21 per 10 years of age without inhalation injury.

Conclusion: Trauma influences burn mortality variably based on burn size and inhalation injury. Mortality for patients with <40% TBSA burn is increased with concomitant trauma. However, for larger burns (>40%) burn size is the primary determinant of mortality, and trauma does not add significantly to overall mortality for these larger burns. Treatment of combined burn/trauma should be predicated on factors influencing mortality.

Table 1. Odds ratio for impact of trauma and inhalation injury on burn mortality

Burn Size (%TBSA)	Trauma	Inhalation
0-20	8.76	56.04
20-40	5.68	20.64
40-60	4.40	9.37
60-80	3.59	6.64
80-100	1.59	6.66

Poster # 10

Predictors of Death and Development of Clavien IV Complications in Patients with Necrotizing Fasciitis

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Introduction:Necrotizing soft tissue infections are associated with a high mortality and morbidity rate. Prompt management requires a high index of suspicion from the surgeon. We sought to identify admission characteristics associated with increased incidence of adverse outcomes.

Methods:Patients with necrotizing fasciitis from 2005 – 2012 were identified using the ACS NSQIP database. Complications were stratified based on the Clavien classification. Clavien IV (Life –threatening complications requiring ICU management) were identified. Univariate analyses were performed to identify clinical characteristics associated with high incidence of Clavien IV complications post operatively. A logistic regression was deployed to identify independent predictors of mortality.

Results:A total of 1,906 patients underwent debridement for necrotizing fasciitis. Overall mortality was 12.6% while 38.1% developed Clavien IV complications. Increasing frailty index and functional status prior to admission were associated with an increasing incidence of Clavien IV complications ($p<0.001$). COPD, hypertension and emergency operations were associated with higher incidence of life threatening complications (58.8% vs. 36.0%, 40.0% vs. 35.7% and 43.6% vs. 28.9% respectively, $p<0.05$). DM, African American race and tobacco use did not prove to increase the incidence of serious complications. Multivariate analysis identified frailty index and emergency operation as the only independent factors associated with increased mortality [AOR (95% CI): 28.9 (11.8, 71.0) and 2.1 (1.5, 2.9) respectively].

Conclusion:Frailty index is an accurate predictor of outcomes for patients presenting with necrotizing fasciitis.

Poster # 11

THE SURGICAL ACUITY SCORE-GALLBLADDER (SAS-G): A STANDARD CLASSIFICATION FOR THE SEVERITY OF GALLBLADDER DISEASE IN THE EMERGENCY GENERAL SURGERY

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Introduction: Unlike the Injury Severity Score (ISS), a uniform severity scoring system for emergency general surgery (EGS) does not exist. We developed two scoring systems for the gallbladder: one based on anatomic criteria only, and the Surgical Acuity Score – Gallbladder (SAS-G) which adds measures of physiology and comorbidities to the anatomic criteria. Our aim was to evaluate and compare the performance of these two models in predicting complications, duration of stay (LOS), and mortality.

Methods: Patients ≥18 who underwent cholecystectomy or cholecystostomy tube placement for cholecystitis or biliary colic at our institution between July 2012 and December 2013 were identified. Anatomic, Physiologic, and Comorbidity scores (**Table**) were determined, squared, and added together to create the SAS-G. The outcomes of extended LOS (>75th percentile), complications, and mortality were recorded. Area under the receiver operating characteristic curve (AUROC) analysis was performed.

Results: There were 307 patients with a mean (SD) age of 62 (20) years (51% male). 85% underwent cholecystectomy during the index hospital admission (77% laparoscopic). There was an 18% complication rate, and an extended LOS (>6 days) rate of 19%. Mortality was 4%. The median (IQR) anatomic, physiologic and comorbidity scores were 2 (2-3), 2 (0-2), and 2 (0-3), respectively for an overall median SAS-G of 13 (8-22). AUROC analysis demonstrated that the SAS-G was more highly associated than the anatomic score for complications (0.68 vs. 0.58), extended LOS (0.82 vs. 0.67), and mortality (0.92 vs. 0.60).

Conclusions: The SAS-G is a reliable tool to categorize severity of gallbladder disease and is superior to anatomic criteria alone. EGS scoring systems that include physiologic and comorbidity criteria will allow enhanced standardization of disease severity between institutions. SAS-G should be validated in a prospective, multi-institutional study.

Score	Anatomic	Physiologic	Comorbidity
0	Normal	Normal	Charlson 0
1	Cholelithiasis	SIRS	Charlson 1-2
2	Cholelithiasis w/ RUQ pain >4 hours, gallbladder wall thickening, or pericholecystis edema Choledocholithiasis	Sepsis	Charlson 3-4
3	Choledocholithiasis w/ cholangitis, pancreatitis, Mirizzi syndrome	Severe sepsis	Charlson 5-6
4	Gallbladder necrosis Perforation with localized abscess	Septic shock	Charlson 7-8
5	Biliary peritonitis	MODS	Charlson ≥9

Poster # 12

ALL OUR EGS IN ONE BASKET? EXAMINING THE COST OF EMERGENCY GENERAL SURGERY BY PAYER STATUS

Catherine G. Velopulos MD, MHS, Xuan Hui MD, Eric B. Schneider Ph.D., Shahid Shafi* MD,MPH, David Ciesla* MD, Oliver Gunter MD, Shalini Selvarajah MD, Elliott R. Haut* MD, David T. Efron* MD, Adil H. Haider* MD,MPH, Johns Hopkins School of Medicine

Introduction:

Acute Care Surgery is a new discipline encompassing Trauma, Critical Care, and Emergency General Surgery (EGS). The American Association for the Surgery of Trauma (AAST) has defined the scope of practice for EGS, but the burden in cost is unknown, as well as who pays for it. As these patients become concentrated within a specialized group of providers, we must delineate implications for reimbursement. We sought to determine the annual inpatient costs incurred by EGS patients by payer status.

Methods:

We conducted a retrospective analysis of patients with EGS admissions from the Nationwide Inpatient Sample (NIS) for 2006-2010, selected by primary diagnosis ICD-9-CM codes defined by AAST as associated with EGS. After adjustment to 2010 US dollars, hospital cost-to-charge ratios (CCR) were used to convert charges to costs. The data was then reweighted to provide national estimates of mean cost per patient, and total yearly costs for EGS.

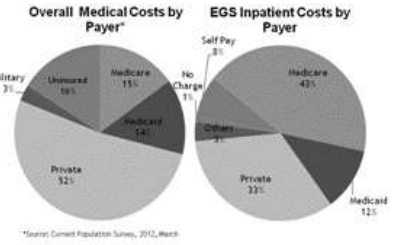
Results:

After weighting the NIS data to the US population, on average 3.9 million patients per year were admitted with a primary diagnosis encompassing EGS. Overall mean cost per EGS patient was $9,711.75, yielding a total estimated yearly cost of $37.62 billion. Compared to privately insured, mean cost per patient was significantly *lower* for self-pay and patients who were not charged, and significantly *higher* for those with Medicare, Medicaid, and other forms of insurance (Table 1). Self-pay and no-charge patients comprised 9% of EGS costs compared to 16% of the overall medical costs incurred by uninsured patients. In contrast to representing 15% of overall medical costs, Medicare patients represented 43% of costs related to EGS (Figure 1).

Table 1. Mean Cost per patient and mean overall cost per year by Payer Status

Payer	Yearly Mean Cost/ Patient*	95% CI	Weighted Population Estimate (%)	Mean Cost per Year* (%)
Medicare	$10,982.25	($10948.21, $11016.29)	1,645,629 (42.6)	$18,072,703,654.87 (48.0)
Medicaid	$9,461.45	($9394.81, $9528.09)	461,380 (11.9)	$4,365,323,338.27 (11.6)
Other insurance	$9,176.12	($9070.97, $9070.97)	137,617 (3.6)	$1,262,788,991.74 (3.4)
Private	$8,722.52	($8690.94, $8754.09)	1,293,818 (33.4)	$11,285,352,171.45 (30.0)
No Charge	$8,514.97	($8356.76, $8673.17)	36,643 (0.9)	$312,013,003.39 (0.8)
Self Pay	$7,779.58	($7727.13, $7832.04)	299,060 (7.7)	$2,326,560,039.26 (6.2)
Overall Mean	**$9711.75**	**Total Yearly National Emergency General Surgery Inpatient Cost**		**$37,624,741,198.98**

*Costs in 2010 US Dollars



*Source: Current Population Survey, 2012, March

Figure 1. Distribution of payers for overall medical costs compared to inpatient emergency general surgery (EGS) costs, showing that burden of uninsured patients is less in EGS, while EGS bears an increased share of Medicare cost. Medicare patients are also disproportionately represented in emergency care.

Conclusion:

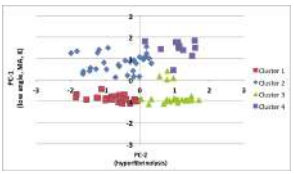
The financial burden of EGS in the US is estimated to be nearly $40 billion/year. Distribution of EGS costs across payers is significantly different from that of the overall healthcare system, with a relatively low proportion of uninsured (self-pay/no charge) patients. Medicare patients are disproportionately represented in EGS costs compared to overall costs of healthcare, which concentrates their care with Acute Care Surgeons as the specialty develops. With the changing reimbursement landscape and uncertainty surrounding current legislation, Acute Care Surgeons may become disproportionately affected. Also, because many EGS conditions are related to early-identifiable diseases that could potentially be managed more electively, this also has implications for potential cost-effectiveness of screening for early detection.

Poster # 13

PHENOTYPES OF TRAUMA-INDUCED COAGULOPATHY

Eduardo Gonzalez MD, Angela Sauaia MD,Ph.D., Ernest E. Moore* MD, Theresa Chin MD, Michael P. Chapman MD, Hunter B. Moore MD, Christopher C. Silliman MD,Ph.D., Anirban Banerjee Ph.D., University of Colorado Denver

Introduction:Protein-C activation, platelet dysfunction, endothelial glycocalyx degradation, and hyperfibrinolysis have been demonstrated to be endogenous drivers of trauma-induced coagulopathy(TIC). Principal component analysis(PCA) of coagulation proteins and of viscoelastic parameters has been validated as a tool that identifies mechanistic pathways leading to TIC. PCA has identified that depletion coagulopathy(principal component 1, PC-1) is independent from fibrinolytic coagulopathy(PC-2). Whether these biological mechanisms translate into phenotypes that are associated with distinct clinical outcomes remains to be elucidated. We hypothesized that phenotypes of trauma-induced coagulopathy could be identified based on cluster analysis of PCA of viscoelastic parameters. **Methods**:Trauma patients admitted to our level 1 academic trauma center(Sept. 2010-Oct. 2013) that met criteria for massive transfusion protocol activation, and received $\geq$1unit of packed red blood cells (PRBC) were studied. Rapid thrombelastography(r-TEG) values were obtained within the first 6 hours post-injury. Based on TEG variables the degree of PC-1(x-axis) and PC-2(y-axis) each patient had was plotted for cluster analysis(median method). Primary outcomes: mortality, and blood product requirements.Continuous values were expressed by median(IQR). Clusters were compared using the Kruskal-Wallis, Chi-square, or Wilcoxon test. **Results**:98 patients were studied; 24 patients were categorized as cluster-1(low PC-1, low PC-2), 30 as cluster-2(low PC-1,high PC-2), 26 as cluster-3(high PC-1,low PC-2), 12 as cluster-4(high PC-1,high PC-2). Median ACT,angle,MA,and LY30 for cluster-1: 117.0 sec, 72.1 degrees, 57.7 mm, 0%; cluster 2: 124.5sec,69.9degrees,57mm,1%; cluster 3: 140.0sec,53.9degrees,41.6mm,0%; cluster 4: 148.0sec,47.3degrees,35.4mm,28.6%. There were no significant differences in age,sex,admission pH,base deficit, and lactate. There was a trend($p=0.09$) towards decreased mortality in cluster-2(13%), compared to any of the other clusters(cluster 1: 20%,cluster 3: 34%,cluster 4: 33%).Cluster-4 had more PRBC, plasma, cryoprecipitate, and platelet blood product requirements (18, 9, 1.5, 1, units, respectively) within the first 6h compared to any of the other clusters(cluster-1: 5, 1.5, 0, 0; cluster-2: 8, 4, 0, 0; cluster-3: 10, 5.5, 1.0, 1)($p<0.001$). Cluster-2 had more red-cell, plasma, and cryoprecipitate requirements compared to clusers-1 and 2($p<0.001$). ISS was significantly higher in clusters-3 and 4(34 and 38 respectively) compared to that of cluster-1 and 2(24 and 25)($p=0.002$). Head injury was less common in patients in cluster-2(3.3%) compared to clusters-1, 3, and 4(25.0%,34.6%,and 33.3% respectively($p=0.002$). There were no significant differences for other injury patterns(chest, abdominal, extremity). **Conclusion:**Cluster analysis of viscoelastic components demonstrates unique patterns of coagulopathy that represent phenotypes associated with distinct clinical outcomes, allowing for more individualized management.



Poster # 14

TRENDS IN ACUTE CARE SURGERY WORKFORCE DEMANDS 2003-2013

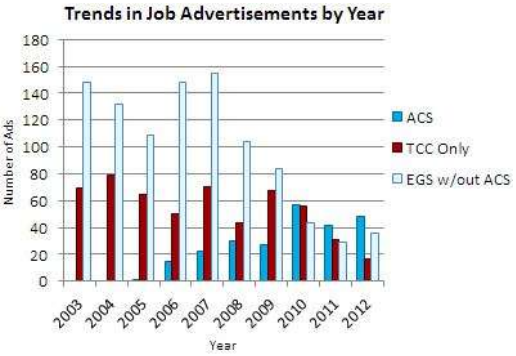
Angela Wood Pawan Barot BS, Braden Price BS, Timothy A. Emhoff* MD, Heena P. Santry* MD, University of Massachusetts

Introduction: Early in the 21st century, leading US trauma surgeons formally convened to discuss growing dissatisfaction with trauma/critical care (TCC), which was increasingly becoming a non-operative specialty. Envisioning the transformation of TCC into the new specialty of acute care surgery (ACS) addressed both the crisis in their ranks and the shrinking pool of general surgeons to take emergency general surgery (EGS) call. We examined advertisements for such positions in the years since to better understand the evolution of ACS workforce demands.

Methods: We reviewed the listings for open TCC and ACS positions in each issue of the Journal of the American College of Surgeons and Journal of Trauma from January 2003 through March 2013. A database was created to record key variables such as name of position (eg. trauma/critical care surgeon, acute care surgeon, emergency general surgeon), geographic region, and type of practice (eg. academic, public, private). Trends in numbers and types of positions available were determined using Cochran Armitage trend tests and linear regression. Job descriptions were analyzed using Nvivo qualitative software for salient themes used to market the position.

Results: We identified 1806 individual ads across both journals. The total number of ads per year decreased from 217 in 2003 (EARLY) to 101 in 2012 (LATE)($p=6.2x10^{-3}$).

The proportion of ads described only as TCC was 32% EARLY and 17% in LATE ($p=8.4x10^{-3}$). Meanwhile, the proportion of ads using ACS nomenclature was 0% EARLY and 48% in LATE ($p=6.2x10^{-5}$), and the proportion of EGS without ACS nomenclature was 68% EARLY and 36% in LATE ($p=1.2x10^{-3}$). The figure to the right shows these proportions across all years.The figure to the right shows these proportions across all years.



Of all ads identified, 30.5% used TCC, 14% ACS, and 55.4% EGS. Differences in overall proportions according to practice type and trauma center verification were analyzed. It was noted that 39% of private practice ads used TCC; only 9.8% used ACS, and 51.2% used EGS. Level I trauma centers used 18.2% ACS, 30.1% TCC, and 51.4% EGS, while Level II used 7.2% ACS, 23.5% TCC, and 69.3% EGS.

Conclusions: The high proportion of EGS ads in early years reflects the shrinking pool that helped prompt the shift from TCC to ACS. The significant increase in the proportion of ACS positions and corresponding decreases in TCC and EGS ads indicate that the workforce demands followed the formal transformation from TCC to ACS. The overall decrease in ads per year may indicate that the demands were being better met as acute care surgeons addressed the needs of both TCC and EGS. Based on these trends, it is likely that in the coming years ACS job demand will wholly eclipse TCC and EGS.

Poster # 15

POSTERIOR COMPONENT SEPARATION AND TRANSVERSUS ABDOMINIS MUSCLE RELEASE (TAR) FOR COMPLEX INCISIONAL HERNIA REPAIR IN PATIENTS WITH A HISTORY OF AN OPEN ABDOMEN

Clayton C. Petro MD, John J. Como* MD, MS, Sydney Yee BS, Ajita S. Prabhu MD, Yuri W. Novitsky MD, Michael J. Rosen MD, Case Surgery

Introduction: Closing large fascial defects following management of the open abdomen remains a daunting challenge. The best reconstructive approach in those patients has not been elucidated to date. We have utilized posterior component separation with transversus abdominus muscle release (PCS/TAR) as the procedure of choice for the vast majority of our complex abdominal wall reconstructions. Herein, we aimed to evaluate our outcomes using this approach in a complex cohort of patients with a previous open abdomen.

Methods: Patients with a history of an open abdomen - defined as an inability to close the fascia after an initial laparotomy – who ultimately underwent complex hernia repair with PCS/TAR from 2010-2013 were identified in our prospective database and analyzed.

Results: Of 37 patients (mean age 54, BMI 32) only one achieved fascial closure during the same hospital stay as their open abdomen. Twenty patients underwent a previous hernia repair, including four anterior component separations, before developing a recurrence. Our operations consisted of 24 (65%) contaminated cases, including five enterocutaneous fistula takedowns, four stoma revisions, and two excisions of infected mesh. The mean hernia size was 445cm ^{2} (range 216-1152) with a mean width of 18cm (range 10-32). Mesh reinforcement included a macroporous polypropylene mesh in 19 (51%) patients and biologic mesh in 18 (49%) patients, all placed in the retrorectus position. Anterior fascial coverage was achieved in 35 (95%) cases. Wound morbidity consisted of 13 (35%) surgical site occurrences: two wound dehiscences, two hematomas, one seroma, seven surgical site infections (19%: four superficial, two deep, and one organ space) and one enterocutaneous fistula that closed spontaneously. There have been no mesh excisions to date. With a mean follow-up of 10.5 (range 3-40) months, there have been three (8%) parastomal recurrences, one associated with a concurrent epigastric recurrence.

Conclusion: Patients with a history of an open abdomen represent a challenging reconstructive problem. To our knowledge, this is the first report describing the use of PCS/TAR in patients with a history of open abdomen. We have demonstrated that this approach is associated with low significant perioperative morbidity and low recurrence. We advocate our approach for patients with complex hernias after an open abdomen.

Poster # 16

INADEQUATELY MARKETING OUR BRAND: MEDICAL STUDENT AWARENESS OF ACUTE CARE SURGERY

Stephanie C. Montgomery MD, Alicia R. Privette MD, Pamela Ferguson Ph.D., Meena Mirdamadi BS, Samir M. Fakhry* MD, Medical University of South Carolina

Introduction: Despite focused national efforts to promote Acute Care Surgery (ACS) as a specialty, little is known about medical student awareness of ACS as a career choice. The impending shortage of general surgeons emphasizes the need to increase interest in a comprehensive surgical specialty such as ACS. The goal of this study was to determine whether medical students would be more likely to consider choosing ACS if they were aware of the specialty and its benefits.

Methods: An anonymous survey was distributed electronically to all medical students at our institution (N=699), a level I trauma center with an active ACS service. The survey included questions regarding specialty choice and factors that were used in making that decision. Also included were questions regarding their familiarity and affinity for ACS.

Results: The survey was returned by 524 students (response rate 75%). Each medical school year and gender were proportionately represented. 21% of students reported surgery as their career choice, but women were half as likely to choose surgery as men. When asked to define ACS, only 23% of all students gave the correct response. Only 8.9% of student in the pre-clinical years correctly defined ACS. Even in the clinical years, 54% were unaware of ACS as a specialty choice. Students reported the top three factors that influenced their choice of specialty were controllable lifestyle, predictable schedule, and a positive role model during medical school. When asked to identify a factor that would make ACS more appealing as a career choice, a 50 hour work week was deemed the most influential. When given the correct definition of ACS as well as approximate pay scale and on-call hours, 41.5% of all medical students and 75% of those interested in surgery would be very likely or somewhat likely to choose ACS as a career.

Medical School Class	Aware of Acute Care Surgery	Very Likely/ Somewhat Likely to Choose ACS
1st Year	(12/101) 11.88 %	(60/101) 59.41 %
2nd Year	(26/113) 23.01 %	(61/113) 53.98 %
3rd Year	(60/123) 48.78 %	(40/124) 32.26 %
4th Year	(53/124) 42.74 %	(31/124) 25.00 %

Conclusion: This study highlights that awareness of ACS as a specialty choice among medical students at our institution is lacking. This may reflect inadequate marketing of our "brand" both locally and nationally. Focused efforts at familiarizing medical students with ACS (especially in pre-clinical years) and increased efforts at role-modeling may increase interest in ACS as a career choice. Special attention should be paid to female medical students as they represent an unrealized source of new surgeons.

Poster # 17

ADVERSE EVENTS IN EMERGENCY GENERAL SURGERY

Oliver L. Gunter* MD, Oscar D. Guillamondegui* MD,MPH, Bradley M. Dennis MD, Naji N. Abumrad MD, Daniel A. Barocas MD,MPH, Vanderbilt University Medical Center

Introduction: Outcomes research in emergency general surgery (EGS) is limited by patient population ambiguity. Teaching hospital status has previously been associated with increased morbidity and mortality in surgical patients. We hypothesized that EGS patients undergoing major abdominal surgery at teaching hospitals have an increased risk of surgical adverse events (SAE) and mortality.

Methods: Retrospective cohort study of 2010 Nationwide Inpatient Sample, restricted to adults, non-elective admissions, and APRDRG for major small and large bowel procedures. Primary outcome was SAE, defined as diagnoses/procedures derived from AHRQ patient safety indicators and NSQIP postoperative wound occurrences. Secondary outcome was hospital mortality. Survey logistic regression was utilized for outcomes with hospital teaching status as principle exposure controlling for demographics, socioeconomic status, hospital features, and comorbidities.

Results: National estimate was 39 million discharges in the US for 2010; the study population represented 156,313 cases. Mean age was 63.2 [95% CI, 62.7-63.7], 54% female, 27% nonwhite minority, 59% govt insured, 44% cared for at teaching hospitals. SAEs occurred in 14%, mortality was 5%. Pre-existing coagulopathy and weight loss were independently associated with increased odds of SAE (O.R. 1.59 [95% CI, 1.40-1.81] and 1.84 [95% CI, 1.84-2.02], respectively). Odds of SAE and mortality at teaching hospitals were 1.35 [95% CI, 1.22-1.49] and 1.16 [95% CI, 1.04-1.36].

Conclusion: Coagulopathy and nutritional status are the strongest comorbidity predictors for postoperative SAEs in EGS patients. Teaching hospitals are independently associated with increased morbidity and mortality for patients undergoing emergency major abdominal operations. Comorbidity does not account for disease severity or patient physiology and may be insufficient for risk-adjusted modeling of EGS outcomes.

Poster # 18

THE MODERN TRAUMA AND EMERGENCY SURGEON: CHARACTERIZING AN EVOLVING SURGICAL NICHE

Brent C. Pottenger MHA, Joseph M. Galante* MD, David H. Wisner* MD, University of California, Davis

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction:Trauma and emergency surgery continues to evolve as a surgical niche. The simple fact that *The Journal of Trauma* is now entitled *The Journal of Trauma and Acute Care Surgery* captures this reality. We sought to characterize more richly the niche that trauma and emergency surgeons have operated within during the transition to the acute care surgery model.

Methods: We analyzed the UHC-AAMC Faculty Practice Solutions Center database for the years 2007 to 2012 for specific Current Procedural Terminology (CPT) codes. This database includes coding and billing data for more than 90 academic medical centers throughout the United States. We analyzed frequency counts and wRVUs generated for specific codes to characterize the average trauma and emergency surgeon's work experience over time.

Results:We found that trauma and emergency surgeons generated 42.36% of wRVUs from procedural work and 57.56% from cognitive work. For procedural work, laparoscopic cholecystectomies produced the most wRVUs (2.44% of total), and placement of a non-tunneled catheter was the most frequently performed procedure (42.22 per year). For cognitive work, critical care services generated the most wRVUs (24.19% of total), and subsequent hospital care was the most frequently performed activity (1,236.55 per year). Moreover, the average trauma surgeon performed far more splenectomies per year (4.88) than splenorrhaphies (0.40); less than one (0.63) video-assisted thoracic (VAT) surgery operation per year; one or two (1.42) skin/muscle flap repairs on the trunk; 4.69 reducible and 2.11 strangulated hernia repairs per year; 7.71 wound vacuum device changes per year; 19.99 appendectomies and 29.62 cholecystectomies per year; and, 2.61 drainage of perirectal abscess procedures per year.

Conclusion:The modern trauma and emergency surgeon is a hybrid of critical care medicine physician and ever-evolving surgical interventionalist who continues to do traditional trauma work while increasingly performing acute care surgeries that fill in the gaps amongst and overlap with other surgical specialties, serving a valuable niche in the healthcare system.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

Poster #19

WITHDRAWN

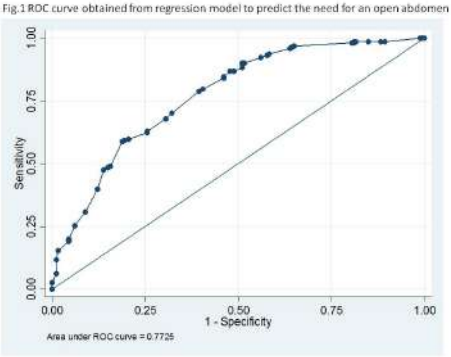
Poster # 20

WHO DO WE LEAVE OPEN IN SEVERE ABDOMINAL SEPSIS?

Carlos A. Ordonez* MD, Fernando Miñan MD, Michael W. Parra MD, Marisol Badiel MD,Ph.D., MSc, Luis F. Pino MD, Fernando Rodriguez MD, Cristina Vernaza MD, Juan C. Puyana* MD, Fundacion Valle del Lili

Introduction:The aim of this study was to develop a prediction model in patients with abdominal sepsis that could select out those that would benefit from an open abdomen.
Methods:A retrospective review was performed of all adult (Age> 18) non-trauma patients who underwent damage control laparotomy (DCL) with an open abdomen (OA) for abdominal sepsis from January, 2004 to December, 2010. Patients with abdominal sepsis secondary to trauma or primary pancreatic disease were excluded. Patients were further divided into two groups: those that were not managed with an open abdomen (Non-OA) and those that were (OA). Data including indications and outcome were collected and analyzed. Variables were selected based on previous reports and common clinical sense and screened in a univariable regression analysis to identify those associated with the need for relaparotomy. Variables with the strongest association were considered for the prediction model which was constructed after backward elimination in a multivariable regression analysis. The discriminatory capacity of the model was expressed with the area under the curve (AUC).
Results:A total of 401 patients were included of which 180 (44.9%) were managed Non-OA and 221 (55.1%) underwent an OA. Both groups were similar demographically. The median age was 55 years (IQR=38-68). The most common source of the abdominal sepsis was the colon in 140 (34.9%) patients, followed by the small bowel in 129 (32. 2%). A total of 52 (13%) patients developed post-operative complications of which the most common was the entero-cutaneous fistula (10% in the OA group vs. 0,5% in the Non-OA group, p= <0.0001). The overall mortality was 17.5%, which was noticeably less in the Non-OA group (13% vs. 21%, p=0.0497). The prediction model included abdominal sepsis from a small bowel source, abdominal sepsis from a colon source, diffuse peritonitis and an APACHE II score >15 as indicatives for the need to leave the abdomen open. We also discovered that age >60 and acute perforated appendicitis were associated with higher complication rates when managed with an OA approach. Our predictive model had the capacity to determine 71.3% (*goodness-of-fit test,* p= 0.4035) and to predict the need for an open abdomen in 77.3% of cases.

Fig.1 ROC curve obtained from regression model to predict the need for an open abdomen



Conclusion:We identified that an elevated APACHE II score >15, a colon or small bowel etiology of the abdominal sepsis, and the presence of diffuse peritonitis are the most common factors indicating the need for an open abdomen approach in severe intra-abdominal sepsis. Our data also showed that age >60 and acute perforated appendicitis were associated with higher complication rates when managed with an OA approach.

Poster # 21

MECHANISMS OF HYPERCOAGULABILITY AFTER TRAUMA: A FIBRIN OR PLATELET PROBLEM?

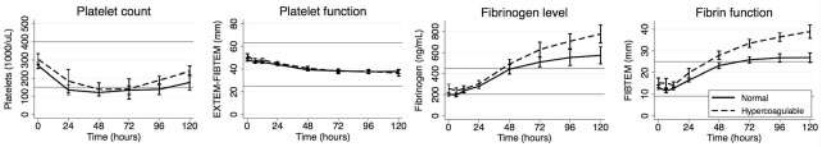
Matthew E. Kutcher MD, Lucy Z. Kornblith MD, Benjamin M. Howard MD,MPH, Sara Moore Ryan F. Vilardi Brittney J. Redick Mary F. Nelson RN, Alan Hubbard Ph.D., Mitchell J. Cohen* MD, University of California, San Francisco

Introduction: Hypercoagulability after trauma is common, leading to thromboembolic complications. The two major contributors to clot strength are fibrin polymerization and platelet function; however, their relative contributions to hypercoagulability and its clinical sequelae such as venous thromboembolism (VTE) are unknown.

Methods: Longitudinal citrated whole blood samples were collected from 177 highest-level trauma activation patients admitted to a Level I urban trauma ICU. Clot strength was assayed using ROTEM® rotational thromboelastometry in response to tissue factor (EXTEM); fibrin function was assayed using tissue factor in the presence of the platelet inhibitor cytochalasin D (FIBTEM). Platelet function was calculated as maximal clot formation (MCF) in EXTEM minus FIBTEM tests. A total of 934 measurements (median 4, inter-quartile range 4-7 samples per patient) were analyzed and matched to standard laboratory values and outcomes.

Results: EXTEM MCF increased throughout ICU stay, becoming hypercoagulable in 52 patients (29.4%) at a median of 72h. Patients who developed hypercoagulability had lower admission pH and INR, and higher platelet count, platelet function, fibrinogen level, and fibrin function (all $p<0.05$), but had similar age and injury characteristics. Fibrin function (OR 1.18, $p=0.038$) on admission was the only adjusted predictor of later hypercoagulability. Platelet function and count decreased over time, and did not differ by hypercoagulability; however, mean fibrin function and fibrinogen levels increased significantly into the supranormal range by 72h, and were significantly higher in hypercoagulable patients from 48h onwards ($p<0.05$). Initiation of VTE prophylaxis was associated with a decline in platelet count and function, but did not impede increasing fibrinogen level and function. Admission fibrinogen (OR 1.01, $p=0.019$) and fibrin function (OR 1.14, $p=0.037$) were identified as injury-adjusted predictors of later VTE.

Conclusion: Hypercoagulability was common in this critically injured population, and occurred early. Patients who later developed hypercoagulability had elevated fibrin and platelet levels and function on admission; however, only fibrin function was a multivariate predictor of early hypercoagulability and later VTE. While mean platelet function and platelet count decreased, fibrin function and fibrinogen levels increased during ICU stay; this trend was not reversed by initiation of VTE prophylaxis. This suggests that hyperfibrinogenemia may play a previously unrecognized - and currently undertreated - critical role in the hypercoagulable state after traumatic injury.



Poster # 22

ELIMINATING BENZODIAZEPINES IN THE TRAUMA INTENSIVE CARE UNIT (TICU): APPLICATION OF AN ATYPICAL ANTIPSYCHOTIC-BASED SEDATION PROTOCOL

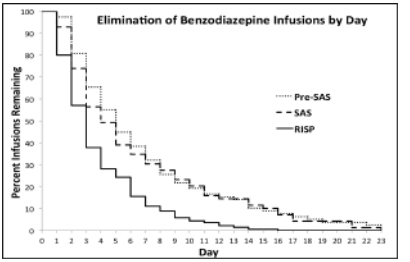
Robert S. Martin* MD, James M. Taylor BS, Melissa K. Barney MD, Gerald J. Rebo PharmD, Nathan T. Mowery* MD, Preston R. Miller* MD, Amy N. Hildreth* MD, Wake Forest University School of Medicine

Introduction: Injured, mechanically ventilated patients are given sedation to increase comfort and decrease ventilator dyssynchrony. Benzodiazepines are often administered for this purpose, but this class of drugs is associated with increased risk of ICU delirium, prolonged cognitive disfunction, and even post-traumatic stress disorder. Risperidone is an atypical antipsychotic that has demonstrated benefit in the setting of delirium. Given the evidence of the dangers of benzodiazepine administration, we designed a protocol to systematically administer risperidone within our approach to sedation. The purpose of this study was to determine the impact of this protocol on benzodiazepine administration. Consistent with national standards we had previously adopted a nurse driven sedation protocol with daily interruption (SAS), and this was also accounted for in the analysis.

Methods: Adult patients admitted to the TICU who required mechanical ventilation for >24 hours were retrospectively enrolled from three time periods- 1/2001-12/2002, 1/2006-12/2007, and 1/2011-12/2012. These time periods were chosen to include a pre-sedation protocol group (Pre-SAS), a sedation protocol group (SAS), and an antipsychotic group (RISP). The RISP group was managed using a sedation protocol that included standard ICU methods to limit sedation in addition to daily titration of risperidone (from 1 to 8 mg BID) until benzodiazepines were no longer needed. Drugs and doses administered to these groups were determined through chart review. Benzodiazepine administration (midazolam/lorazepam) was compared between groups.

Results: Seven hundred forty six patients were included in the study. Over 48 % of patients in the RISP group received risperidone by protocol. Total ICU administration of benzodiazepines decreased across all three groups including a significant decrease from the SAS to the RISP groups (Table). Similarly, the mean daily dose administered by infusion decreased significantly from the SAS to the RISP group. A Kaplan Meier curve demonstrates the early discontinuation of benzodiazepine infusions in the RISP group compared to the Pre-SAS and SAS groups (Figure).

	Pre-SAS (1)	SAS (2)	RISP (3)	p-value (2 vs 3)
Number	178	291	277	
Risperidone percent	0	0	48.5	
Total benzo (mg)	292.5	137.0	88.0	**0.029**
Drip dose per day (mg)	62.6	50.2	27.8	**< 0.001**



Conclusion: Implementation of a sedation protocol that employs atypical antipsychotics was associated with a significant reduction in the administration of benzodiazepines in ventilated TICU patients. We present a protocol that uses these medications independent of measured delirium status and suggest that this approach would benefit from prospective study.

Poster # 23

NSQIP CLINICAL CRITERIA FOR DIAGNOSIS OF NOSOCOMIAL PNEUMONIA ARE ACCURATE AND SPECIFIC

Andrew J. Kerwin* MD, Jhun De Villa MD, Jin H. Ra MD, J. Bracken Burns Jr., DO, Indermeet S. Bhullar* MD, David J. Skarupa MD, Joseph J. Tepas* III, MD, University of Florida, Jacksonville

Introduction:Postoperative pneumonia (PNA) remains a costly complication of surgical care and a metric for quality assessment increasingly embraced by federal and private payers. In light of our policy of aggressive broncho-alveolar lavage (BAL) to confirm suspected PNA by the presence of pathogenic organisms, we assessed the accuracy of NSQIP clinical criteria used to identify PNA by comparing BAL results to NSQIP registry data.

Methods:The registry records of 2,543 surgical patients reviewed in our NSQIP program were analyzed. Microbiology results of BAL cultures obtained during the study period were matched to the NSQIP population. Incidence of pathogenic organisms >100,000 cfu/ml was determined for patients identified by NSQIP as PNA, and for all other NSQIP patients who underwent BAL and were not recorded as having PNA. Positive (PPV) and negative (NPV) predictive values as well as positive and negative Likelihood ratios (LR +, LR-) were calculated for NSQIP clinical criteria.

Results:Over 22 months 2,543 surgical cases were reviewed. NSQIP clinical criteria identified 25 PNA, 21 of whom were confirmed by BAL. Two of the other four had positive sputum cultures and two had no bacterial confirmation. During the same period 455 BAL procedures were performed on surgical patients, 51 of which were part of the NSQIP review cohort. The 33 additional NSQIP patients with BAL results included 28 (85%) with negative results, 70% of which were sterile or mixed flora while 30% had sub threshold quantities of pathogenic organisms. The 5 positive culture patients included 2 confirmed on second BAL with organisms not present on initial BAL. NSQIP criteria screening with confirmed pathogenic organism generated a PPV of 92% with a LR+ of 194.2, and a NPV of 99% with LR- of .0052.

Conclusion:These results confirm the accuracy of NSQIP screening criteria for identifying patients with probable PNA. The 83% incidence of negative BAL may reflect overaggressive therapy, especially in light of the high incidence of negative results. Using NSQIP clinical criteria as part of fever evaluation could allow clinicians to make more accurate decisions regarding the need for BAL and empiric antibiotic use in patients with possible PNA.

Poster # 24

SPLENECTOMY IS ASSOCIATED WITH A HIGHER RISK FOR VENOUS THROMBOEMBOLISM

Debora Lee BS, Galinos Barmparas MD, Nicole Fierro BS, Douglas Liou MD, Alex W. Lamb BS, Brandon Nguyen BS, Rex Chung MD, Eric J. Ley* MD, Cedars-Sinai Medical Center

Introduction: Thrombocytosis following splenectomy is a common post-operative finding. Whether thrombocytosis leads to a higher risk of venous thromboembolism (VTE) is unclear. The aim of this investigation was to determine if splenectomy is associated with the development of VTE.

Methods: This was a prospective, observational study conducted at a 24-bed SICU. All patients admitted from 1/2011 to 11/2013 after undergoing a splenectomy, a bowel resection/repair or a combination of both were followed. Demographics and relevant clinical data were collected and results of venous Duplex studies and CT angiographies were recorded. The primary outcome measure was development of VTE, including deep venous thrombosis (DVT) and pulmonary embolus (PE). The three groups were compared using analysis of variance and the bowel resection/repair group was used as a reference group for comparison.

Results: Over the 34 month study period, 2,308 patients were admitted to the SICU: 341 (14.8%) after a bowel resection/repair, 33 (1.4%) after a splenectomy and 14 (0.4%) after a combination of both. The mean±SD age was 63±19 years and 51% were male. The three groups (bowel resection/repair, splenectomy, and combination) did not differ in regards to age, gender, need for mechanical ventilation (61% v. 58% v. 79%, p=0.378) or pressor support (28% v. 18% v. 36%, p=0.380). No differences were noted for past medical history, including diabetes mellitus, cardiac history or renal failure. Trauma patients constituted 7% of the study population (5% v. 27% v. 29%, p<0.001). There was no difference in utilization of chemical prophylaxis between the three groups (74% v. 73% v. 76%, p=0.913). Overall, 34% underwent imaging to rule out VTE (32% v. 42% v. 71%, p=0.005). The incidence of VTE was highest among patients undergoing a combination of splenectomy and bowel resection/repair (11% v. 21% v. 57%, p<0.001). Splenectomy was associated with a higher adjusted risk for VTE compared to the bowel repair/resection group (AOR [95% CI]: 5.33 [1.73, 16.43], p=0.004). Combination of splenectomy and bowel resection/repair also increased the adjusted risk for VTE (AOR [95% CI]: 14.21 [3.64, 55.56], p<0.001).

Conclusion: Splenectomy significantly increases the risk for VTE, especially when performed on patients undergoing a bowel resection/repair. A high index of suspicion should be maintained for early recognition of VTE in splenectomy patients and a more aggressive policy for prophylaxis should be considered.

Poster # 25

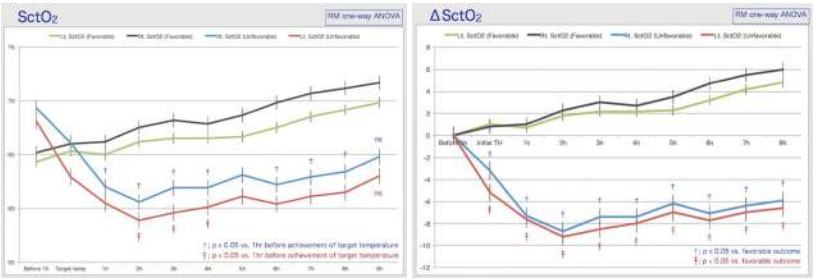
Dynamic changes of cerebral tissue oxygen saturation ($SctO_2$) using a Near Infrared Spectroscopy (NIRS) during therapeutic hypothermia for patients with post-resuscitated ischemic brain damage.

Junya Tsurukiri MD,Ph.D., Katsuhiro Nagata MD, Jun Oda* MD,Ph.D., Tetsuo Yukioka* MD,Ph.D., Tokyo Medical University

Introduction:The energy metabolism of the brain is often heterogeneous after successful resuscitation and induction of therapeutic hypothermia (TH) in comatose patients with post-resuscitated ischemic brain damage changes the balance between oxygen delivery and supply. Recently, cerebral tissue oxygen saturation ($SctO_2$) using a Near Infrared Spectroscopy (NIRS) can reflects the balance between cerebral metabolic supply and oxygen demand.

Methods:This study included post-resuscitated comatose patients treated with TH, monitored with NIRS at the intensive care unit (IC U). SctO2 was non-invasively and continuously measured every one minute to 72 hours and calculated average of every one hour in 16 comatose patients during inducing TH and TH maintenance phase and active rewarming. A targeting temperature (TT) was at 33.5°C and rewarming was commenced at 24 hours after TH (0.5°C/4hrs).

Results:In patients with favorable outcome for a cerebral performance category (CPC) score of 1 or 2, significant increase or decrease of bilateral $SctO_2$ did not show. In contrast, bilateral $SctO_2$ in patients with unfavorable outcome for a CPC score of 3-6 significantly decreased within the first 2 hours after achievement of TT compared with those at 1 hour before achievement of TT. These significant changes were observed within 8 hours after achievement of TT (Figure 1,2). After 9 hours from achievement of TT, $SctO_2$ gradually returned to baseline values, with no differences between favorable and unfavorable outcomes to 72 hours.



Conclusion: Dynamic changes of $SctO_2$ represented by simple numerical values using a NIRS can be used to evaluate the metabolic balance of ischemic brain damage for post-resuscitated comatose patients during TH at the ICU.

Poster # 26

DECREASING MAINTENANCE FLUIDS IN NORMOTENSIVE TRAUMA PATIENTS IS SAFE AND REDUCES LENGTH OF STAY

Galinos Barmparas MD, Debora Lee BS, Nicole Fierro BS, Douglas Liou MD, Tri Tran BS, Sogol Ashrafian BS, Danielle Tran BS, Eric J. Ley* MD, Cedars-Sinai Medical Center

Introduction: Excessive fluid administration is associated with a prolonged hospital course and worse outcomes. We provided the minimum basal fluid rate, "to keep open (TKO)", to normotensive trauma patients admitted to the surgical intensive care unit (ICU) to determine the impact on outcomes.

Methods: This study was conducted in a 24-bed dedicated surgical ICU at a Level 1 trauma center. In June 2013, all normotensive trauma patients admitted to the surgical ICU were administered crystalloids at 30 cc/hr (TKO group) and were compared to patients admitted during the preceding 6 months who were placed on a rate of 125cc/hr (non-TKO group). Changes in the maintenance fluid rate and boluses were at the discretion of the attending intensivist. Net fluid balance was collected until day of ICU transfer or ICU day 5, whichever occurred first. Exclusions included initiation of vasopressors and brain death. Primary outcomes included ICU and hospital stay and ventilation days.

Results: During the 12-month study period, a total of 134 trauma patients met inclusion criteria: 51 (38%) in the TKO and 83 (62%) in the non-TKO group. Overall the two groups were well balanced in regards to age (47.9 ± 23.4 vs. 48.6 ± 22.6 years, p=0.87), ISS (16.5 ± 11.2 vs. 15.6 ± 9.5, p=0.63), APACHE IV scores (36.2 ± 28.4 vs. 40.3 ± 29.4, p=0.42), the need for mechanical ventilation (41.2% vs. 43.4%, p=0.80) and the need for exploratory laparotomy (5.9% vs. 8.4%, p=0.74). TKO patients were more likely to be male (88.2% vs. 67.5%, p<0.01). As expected, TKO patients required significantly less fluid per day (1.9 ± 1.3 vs. 2.6 ± 1.8 liters, p=0.03). After adjusting for differences between the two groups, TKO patients had a lower overall hospital stay (adjusted mean difference AMD [95% CI]: -2.7 [-13.5, -2.91] days, p<0.01). Differences in ICU stay (-1.9 [-3.5, -0.25] days, p=0.07) and ventilation days (-0.99 [-4.0, -0.07] days, p=0.06) did not reach significance. In a forward logistic regression model, TKO was associated with ICU stay ≤ 48 hours (Adjusted Odds Ratio (AOR) [95% CI]: 2.45 [1.09, 5.53]; p=0.03).

Conclusion: A protocol that mandates the admission basal fluid rate starts at TKO in normotensive trauma patients reduces fluid intake and predicts a shorter hospital course. Decreasing basal fluid administration in normotensive trauma patients is encouraged.

Poster # 27

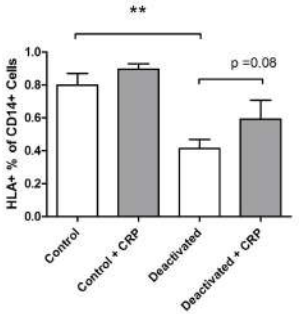
MONOCYTE DEACTIVATION IS ATTENUATED BY CRP AND MAY BE INFLUENCED BY FCγRIIA POLYMORPHISMS

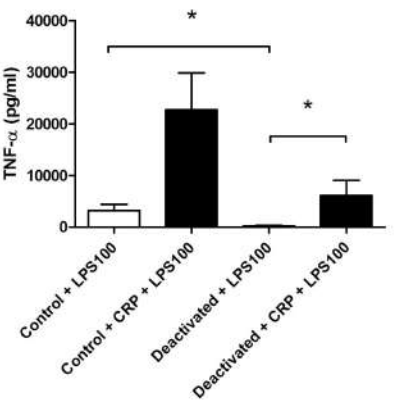
Sonlee D. West MD, Michael Krencicki BS, Carolyn Mold Ph.D., University of New Mexico

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction:We have previously shown that a single nucleotide polymorphism of the FcγRIIa receptor is associated with increased risk of sepsis in trauma patients. Additionally, we noted a correlation between monocyte deactivation and the FcγRIIa SNP in trauma patients. We sought to further identify the effects of the FcγRIIa on monocyte deactivation in an in vitro model. FcγRIIa is the receptor for CRP and IgG located on monocytes. The different polymorphisms affect receptor affinity and specificity and may represent a risk factor for certain diseases, either at the level of disease susceptibility or at the level of disease severity.
Methods:Monocytes were cultured from healthy volunteers and incubated IL-10 and TGF-β to create deactivated monocytes. Normal and deactivated monocytes were incubated with acute phase levels of C reactive protein followed by a 4 hour incubation with LPS (10 ng/ml). HLA-DR expression was then determined by flow cytometry and supernatants were collected to determine TNF-α production in response to LPS.
Results:We found that acute phase levels of CRP attenuated monocyte deactivation as demonstrated by increasing TNF-alpha production in response to LPS and a trend toward increased HLA-DR expression.

C**onclusion**:In conclusion, monocyte deactivation, an important risk factor for post-traumatic sepsis, may be influenced by the FcγRIIa polymorphism of the host.





All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

Poster # 28

METHICILLIN-RESISTANT STAPHYLOCOCCUS AUREUS COLONIZATION STATUS MAY GUIDE EMPIRIC TREATMENT OF HEATHCARE ASSOCIATED PNEUMONIA IN NEUROTRAUMA PATIENTS

Sharon Moran MD, Richard Moore MD, Caesar M. Ursic* MD, Susan Steinemann MD, Maimona Ghows MD, The Queen's Medical Center

Introduction: Patients with critical neurologic injury frequently develop nosocomial pneumonia. The etiology is multifactorial and may include endotracheal intubation, impaired cough reflex, diaphragm paresis, and reduced mucocilliary function due to hypothermia or barbiturates. Due to concern about methicillin-resistant Staphylococcus Aureus (MRSA) infection, vancomycin is often started empirically for suspicion of nosocomial pneumonia. The purpose of this study was to determine the predictive value of admission MRSA nasal swab for subsequent development of MRSA pneumonia. With this information, we hope to develop a tailored approach to empiric antibiotic therapy in the high risk neurotrauma population.

Methods: We retrospectively reviewed the charts of all patients admitted to our neurosurgical intensive care unit (NSICU) with a traumatic brain or spinal cord injury between May 2010 and August 2012. The study group was comprised of patients started on empiric antibiotic therapy for presumed pneumonia. Our standard trauma admission protocol involves nasal swab testing for MRSA upon admission. We compared MRSA colonization status and sputum culture results in neurotrauma patients who developed healthcare associated pneumonia during their hospital course.

Results: 220 patients were admitted to the NSICU with a traumatic neurologic injury during the study period. 201 patients had MRSA screening; 14 (7%) were positive. Of those screened, 98 patients had a sputum culture that was positive for a dominant organism. The negative predicitve value of nasal culture was 95% and the positive predictive value was 64% for development of MRSA pneumonia.

Conclusion: In this population, a negative MRSA nasal swab was associated with the absence of MRSA in sputum cultures. The more discriminate use of vancomycin has been recommended to decrease evolutionary pressure for developing drug-resistant infections. Given the high negative predictive value of nasal swabs in our study, one might rationally omit the empiric use of vancomycin in critically injured neurotrauma patients with clinical evidence of pneumonia. In addition to reducing the appearance of drug-resistant organisms, this tailored approach would result in substantial cost savings and potential avoidance of allergic reactions or adverse side effects. Further study is need into whether decolonization can prevent the development of MRSA pneumonia.

Poster # 29

Penetrating Thoracic Trauma is not a Risk Factor for Pneumonia in Non-Intubated Patients

Rhiana Menen MD, Wayne S. Lee MD, Vincent E. Chong MD, MS, Gregory P. Victorino* MD, University of California San Francisco - East Bay

Introduction: The American Thoracic Society estimates that the incidence of nosocomial pneumonia is between 0.5-1.0%, with the incidence of ventilator-associated pneumonia (VAP) being 6 to 20 fold higher. While the association of mechanical ventilation and pneumonia is well accepted, there are few studies that characterize non-ventilator-associated pneumonia (NVAP) in non-intubated patients following penetrating chest trauma. Because of the direct physical injury to the lung and possible bacterial seeding of the wound track, our hypothesis was that penetrating chest injuries was a risk factor for developing NVAP.

Methods: A retrospective chart review was performed on all trauma patients with a stab wound (SW) or gunshot wound (GSW) to the chest admitted to our university based urban trauma center between 2004 and 2013. Because hospital acquired pneumonia is thought to take 2-3 days to develop, patients with a hospital length of stay ≤ 2 days were excluded. Patient characteristics and outcomes were extracted from a trauma database and analyzed.

Results: Of 683 patients who met inclusion criteria, 24 patients (3.5%) had hospital-acquired pneumonia (VAP+NVAP). Of the 130 patients (19%) that were intubated, 17 patients (13.1%) had VAP. Of the 553 patients (81%) that did not require intubation, 7 patients (1.3%) had NVAP. Overall post-trauma pneumonia was more common in GSW patients than SW patients (4.4% vs. 1.1%; p=0.03). There was a higher rate of VAP in GSW patients (3.2% vs. 0.5%; p=0.04). However, there was no difference in the rate of NVAP between the two mechanisms (1.2% vs. 0.5%; p=0.6). Logistic regression demonstrated that GSW mechanism, ISS, GCS, intubation, length of stay, and length of ICU stay are all independent variables that are associated with hospital-acquired pneumonia.

Conclusion: Penetrating thoracic trauma patients have similar NVAP rates compared to the incidence of nosocomial pneumonia estimated by the American Thoracic Society. Higher rates of overall post-trauma pneumonia were seen in GSW patients compared to SW. This was mainly due to a higher rate of VAP. Despite direct injury to the lung, penetrating thoracic injuries are not associated with a higher rate of NVAP.

Poster # 30

PRE-OPERATIVE SICU ADMISSION DOES NOT IMPROVE CLINICAL PARAMETERS PRIOR TO OPERATIVE DEBRIDEMENT OF NECROTIZING SOFT TISSUE INFECTION

Angela Neville* MD, Jessica Keeley MD, Amy Kaji MD,Ph.D., Andrew Nguyen MD, Dennis Kim MD, Christian DeVirgilio MD, Brant Putnam* MD, Scott Bricker MD, Frederic Bongard* MD, David Plurad* MD, Harbor-UCLA Medical Center

Introduction: Early surgical debridement of necrotizing soft tissue infection (NSTI) is the cornerstone of management of this morbid condition. The role for pre-debridement surgical intensive care unit (SICU) admission for goal directed resuscitation is unclear. The purpose of this study was to evaluate whether pre-surgical SICU admission improves mortality or laboratory markers of end-organ perfusion.

Methods: We analyzed all patients treated for NSTI at our county funded, academic medical center between 2008-2013. Pre-operative admission to the SICU, time to operation, admission and pre-operative laboratory values, and mortality were assessed. Admission laboratory values were compared to pre-operative values for those patients admitted to the SICU prior to surgery.

Results: During the five-year study period, 138 patients were admitted with an NSTI, of which twenty (14.5%) died. Thirty-one patients were admitted to the SICU for resuscitation via a standardized sepsis protocol prior to undergoing therapeutic debridement; 107 proceeded directly to the operating room from the emergency department (ED). There was no difference in median time to debridement in either group (8.0 hours SICU group vs. 8.8 hours ED group, p=0.9). The rate of severe sepsis or septic shock was 80.6% in the SICU group, while it was 47.7% in the ED group ($p<0.001$). Despite SICU admission, there was no improvement in patients' white blood cell (WBC) count, bicarbonate, creatinine, or lactate levels prior to surgery (Table 1). Patients admitted to the SICU pre-operatively had higher mortality rates compared to those who proceeded directly to surgery (OR =2.9, 95% CI 1.1-7.8, p=0.03). As the SICU-resuscitated cohort may have been inherently more severely ill, subgroup analysis was performed for patients with severe sepsis or septic shock. In this subgroup, there was no difference in mortality (p=0.2) or laboratory markers of end-organ perfusion for SICU patients versus those that went directly to the operating room.

Table 1: Laboratory values for SICU-resuscitated patients with NSTI

Variables	Admission	Pre-operative	p-value
WBC	16.5 ± 9.7	16.5 ± 9.1	0.6
Bicarbonate (mmol/L)	19.1 ± 4.4	18.3 ± 4.3	0.2
Lactate (mmol/L)	2.6 ± 1.8	2.0 ± 1.6	0.3
Creatinine (mg/dL)	2.0 ± 1.4	1.6 ± 1.2	0.06

Conclusion: Pre-operative resuscitation in a SICU prior to surgical debridement was not associated with a decrease in mortality or improvements in laboratory markers of end-organ perfusion. SICU admission prior to surgery is an unnecessary step in the management of NSTI, supporting existing literature advocating immediate surgical source control.

Poster # 31

THE BRAIN INJURY GUIDELINES:A PROSPECTIVE ANALYSIS

Bellal Joseph* MD, Viraj Pandit MD, Bardiya Zangbar MD, Narong Kulvatunyou* MD, Andrew Tang MD, Terence O'Keeffe* MD, MBChB, Donald J. Green* MD, Lynn Gries MD, Randall S. Friese* MD, Peter Rhee* MD,MPH, University of Arizona - Tucson

Introduction: The role of acute care surgeons (ACS) for management of traumatic brain injury (TBI) is evolving. We implemented (March 2012) the Brain Injury Guidelines (BIG) at our institution for managing patients with TBI without neurosurgical consultation. The aim of this study was to compare outcomes in TBI patients before and after the implementation of the BIG guidelines.

Methods: We performed a 2-year (2011-2012: pre guideline and 2012-2013: post guideline) prospective cohort study of all patients with TBI presenting to our level 1 trauma center. Patients with skull fracture and/or intracranial hemorrhage were included. Outcome measures were: number of neurosurgical consultations, repeat head computed tomography (RHCT) scan, 30-day readmission rate and hospital cost.

Figure 1: Brain Injury Guidelines

BRAIN INJURY GUIDELINES			
Variables	**BIG 1**	**BIG 2**	**BIG 3**
LOC	Yes/No	Yes/No	Yes/No
Neurologic Examination	Normal	Normal	Abnormal
Intoxication	No	Yes/No	Yes/No
CAMP	No	No	Yes
Skull Fracture	No	Non-displaced	Displaced
SDH	≤ 4 mm	5 – 7 mm	≥ 8 mm
EDH	< 4 mm	5 – 7 mm	> 8 mm
IPH	< 4 mm, 1 location	3 – 7 mm, 2 locations	> 8 mm, Multiple Locations
SAH	Trace	Localized	Scattered
IVH	No	No	Yes
	THERAPEUTIC PLAN		
Hospitalization	No, Observation (6hrs)	Yes	Yes
RHCT	No	No	Yes
NSx	No	No	Yes

Results:A total of 796 patients (Pre: 415, Post: 381) were included. The implementation of the BIG guidelines resulted in overall savings of $2,326,000 in hospital cost.

Table. Demographics and Outcomes

	Pre-BIG (n=415)	Post-BIG (n=381)	*p*
Age	39.5±23	39.6±26.2	0.8
Male	61%	65%	0.7
AP/AC	43%	40.6%	0.8
GCS	13[8-15]	13[9-15]	0.7
Head AIS	2[2-3]	2[2-3]	0.7
Outcomes			
NSx consultation	90.6% (376)	56.2% (214)	0.01
RHCT	91.8% (381)	45.7% (174)	0.01
Hospitalization	88.2% (366)	46.7% (178)	0.02
ICU admission	47.7% (198)	29.4% (112)	0.04
Mortality	16.6% (69)	16.3% (62)	0.6
30-day re-admission	8.9% (37)	8.1% (31)	0.4
Hospital cost/patient, ($)	14,926±11,518	10,154±9,201	0.02

AP/AC, antiplatelet/anticoagulants; GCS, Glasgow coma scale; AIS, abbreviated injury scale; NSx, neurosurgical consultation, RHCT, repeat head computed tomography, ICU, intensive care unit

Conclusion: Implementation of BIG guidelines is safe, effective, and reduces healthcare cost. The BIG guidelines define the management of TBI patients without the need for neurosurgical consultation. Establishing a national multi-institutional study implementing the BIG guidelines is warranted.

Poster # 32

EMERGENT OPERATION FOR SEVERE TRAUMATIC BRAIN INJURY: DOES TIME MATTER?

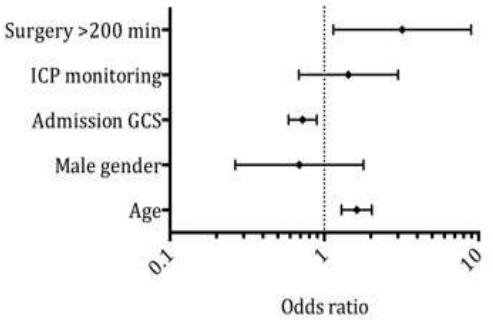
Kazuhide Matsushima MD, Kenji Inaba* MD, Stefano Siboni MD, Gregory Magee MD, Aaron Strussmasser MD, Gene Sung MD,MPH, Elizabeth Benjamin MD,Ph.D., Lydia Lam* MD, Demetrios Demetriades* MD,Ph.D., LAC+USC Medical Center

Introduction: It remains unclear whether the timing of neurosurgical intervention impacts the outcomes of patients with isolated severe traumatic brain injury (TBI). We hypothesized that decreased time between emergency department (ED) presentation to neurosurgical intervention would improve patient survival.

Methods: Our institutional trauma registry was queried for adult patients (>=18 years) who required emergent surgical intervention for TBI. We included all patients with altered mental status upon presentation to the ED (GCS<=8). Patients with associated severe injuries (AIS>=3) were excluded. The optimal cut-off value for the time interval between ED presentation and emergent operation was explored. In-hospital mortality for patients in the early operation group was compared with the late group using univariate and multivariate analyses.

Results: A total of 161 patients were identified between 2003 and 2012. The majority of patients were male (83.2%), median age 48 years (range:18-94). Median ISS was 25 (range:9-43), median GCS 4 (range:3-8). Median time between ED presentation and surgical intervention was 137 min (range:39-284). In the early operation group (<=200 min), significantly lower in-hospital mortality rate was identified (34.5% vs. 59.1%, p=0.034). After clinically important covariates (age, sex, GCS, ICP monitoring) were adjusted in a logistic regression model, the early neurosurgical intervention was associated with a significantly higher odds of patient survival (OR: 3.2, 95% CI: 1.14-8.94, p=0.027).

Conclusion: Our data suggest that the survival rate for isolated severe TBI patients who required emergent neurosurgical intervention may be time-dependent. Specific factors leading to this delay and methods of expediting surgical intervention warrant further examination.



Poster # 33

THE EFFECT OF β-BLOCKADE ON SURVIVAL IN A SWEDISH COHORT OF ISOLATED SEVERE TRAUMATIC BRAIN INJURY

Shahin Mohseni MD, Peep Talving* MD,Ph.D., Eric Thelin MD, Göran Wallin MD,Ph.D., Olle Ljungqvist MD,Ph.D., Louis Riddez MD,Ph.D., KAROLINSKA UNIVERSITY HOSPITAL

Introduction: Several North-American studies have shown that β-blocker exposure has positive effect on survival in patients suffering from traumatic brain injuries (TBI). The purpose of this study was to evaluate the effect of β-blockade after isolated severe TBI in a Swedish population.

Methods: For the proposed study, the trauma registry of an urban academic trauma center was queried to identify patients with a blunt isolated severe TBI between 1/2007 and 12/2011. Isolated severe TBI was defined as an intracranial injury with an abbreviated injury scale of (AIS) ≥3 excluding all extracranial injuries AIS ≥3. Multivariable logistic regression analysis was used to adjust for differences between the groups to determine whether exposure to β-blockers was protective in patients suffering isolated severe TBI.

Results: Overall, 874 patients met the study criteria. Of these, 33% (n=287) were exposed to β-blockers during their hospital admission. The exposed patients were significantly older (62 ±16 years vs. 49 ±21 years, $p<0.001$),and more severely injured based on their admission GCS, ISS and AIS scores (GCS ≤8: 32% vs. 28%, $p=0.007$; ISS ≥16: 71% vs. 59%, $p=0.001$; head AIS ≥4: 60% vs. 45%, $p<0.001$). The unadjusted mortality was higher in patients who did not receive β-blockers (17% vs. 11%, $p=0.007$). The predominantly utilized β-blockers were Labetolol (49%) and Metoprolol (45%). The mean time of initiation of β-blockade was 3 ±4 days. The majority of patients (75%) were exposed to β-blockers within 60 hours of admission. After adjustment for significant confounders between the groups, patients who had not been exposed to β-blockers had a 5-fold increased risk of mortality (AOR 5.0, CI 95% 2.7-8.5, $p=0.001$). We could not detect a difference in survival in regards to the type of β-blocker used.

Conclusion: β-blocker exposure after isolated severe TBI is associated with improved survival. Prospective evaluation of our results is warranted.

Poster # 34

Validation of a new prognostic model in patients with severe traumatic brain injury: a multicenter observational study.

Tomohito HIRAO MD,Ph.D., Kazuhisa YOSHIYA MD,Ph.D., Akinori WAKAI MD, Hiroki MATSUDA MD, Satoshi FUJIMI MD,Ph.D., Daikai SADAMITSU MD,Ph.D., Takeshi SHIMAZU MD,Ph.D., Osamu TASAKI* MD,Ph.D., Nagasaki University Hospital Emergency Medical Center

Introduction:
We established and reported a new mathematical prognostic model for severe traumatic brain injury (STBI) (Acute Medicine & Surgery, 2014). The purpose of the present study was to validate our prediction model and compared the predictive value to previously established models.

Methods:
One hundred and nine patient with a Glasgow Coma Scale score of <9 were enrolled in this multicenter cohort study consisting of four tertiary critical care medical centers in Japan. Our prognostic model included the variables of age, pupillary light reflex on admission, intracranial pressure on ICU admission, subarachnoid hemorrhage and midline shift on CT scan within 24 hours of injury. Outcome was assessed prospectively 6 months after injury according to the Glasgow Outcome Scale. GR and MD were considered to be favorable outcomes. SD, PVS, and D were considered unfavorable. The predictive accuracy was compared to the two models derived from International Mission for Prognosis and Analysis of Clinical Trials in TBI (IMPACT) or Corticosteroid Randomisation After Significant Head Injury (CRASH).

Results:
Out of 109 patients, 25 (22.9%) had favorable outcome, and 84 (77.1%) had unfavorable outcome. The area under the receiver operating characteristic curve of our model was 0.813. That of IMPACT and CRASH was 0.768 and 0.787, respectively. If the cut off value of probability of unfavorable outcome was imposed at 0.51 in our model, the positive predictive value was 87.5%, negative predictive value was 66.7%, and total predictive value was 83.5%. Sensitivity was 91.7%, and specificity was 56.0%.

Conclusion:
Our prognostic model was shown to have high predictive value on external validation, and superior to IMPACT or CRASH models. It will be useful for decision-making of treatment strategy, family counseling, and review of treatment in patients with severe traumatic brain injury.

Poster # 35

NEURONAL CELL CYTOTOXICITY DUE TO BETA ADRENERGIC AGONISM INCREASES AFTER OXYGEN-GLUCOSE DEPRIVATION AND DIFFUSE AXONAL INJURY

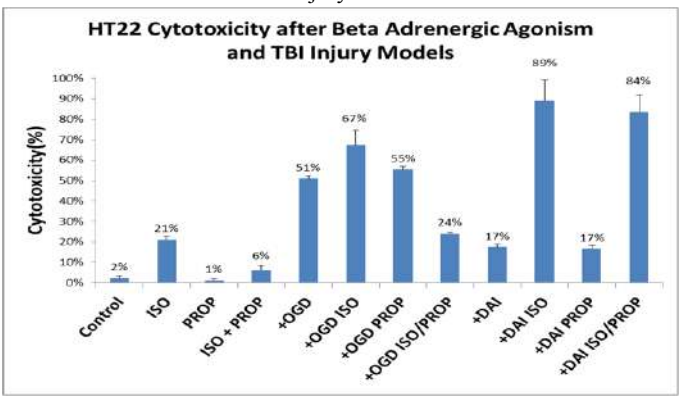
Alexander W. Lamb BS, Galinos Barmparas MD, Brandon Nguyen BS, Kevin Chen BS, Patrick Lyden MD, Eric J. Ley* MD, Cedars-Sinai Medical Center

Introduction: Beta adrenergic receptor (BAR) activity modulates the related immune deficiency that occurs after traumatic brain injury (TBI). The aim of this study was to determine the effect of BAR agonist and/or antagonist on neuronal cells before and after oxygen glucose deprivation (OGD) or diffuse axonal injury (DAI).

Methods: The BAR agonist isoproterenol (ISO) and/or the antagonist propranolol (PROP) were distributed to wells of HT22 cells, an immortalized mouse hippocampal cell line. OGD was performed using glucose free media, N2/CO2 gas mixture for 10 minutes, and then anoxic incubator for 2 hours. DAI was performed by stretch injury using a Cell Injury Controller II at 7 PSI. Cytotoxicity was measured from supernatant Lactate Dehydrogenase (LDH) as a percentage of total lysed cell LDH.

Results: While ISO was cytotoxic to HT22 compared to control (21.1% v. 2.4%, $p < 0.0001$), PROP did not alter cytotoxicity (1.2% v. 2.4%, p=0.38). ISO induced cytotoxicity was inhibited by PROP (21.1% v. 6.2%, p=0.001). (21.1% v. 6.2%, p=0.001). OGD increased cytotoxicity compared to control (50.9% v 2.4%, $p < 0.0001$). While ISO increased cytotoxicity in OGD conditions (50.9% v. 67.3%, p=0.005), PROP did not (50.9% v. 55.3%, p=0.083). When PROP was added to ISO in OGD conditions, a reduction in cytotoxicity was observed compared to ISO alone (67.3% v 24%, $p<0.0001$). DAI increased cytotoxicity compared to control (17.4% v 2.4%, $p<0.0001$). While ISO added to DAI increased cytotoxicity compared to DAI alone (89% v. 17.4%, $p<0.0001$), PROP did not (16.6% v. 17%, p=0.54). When PROP was added to ISO after DAI, no reduction in cytoxicity was observed (89%. v 84%, p=0.45). Although OGD injury was greater compared to DAI (50.9% v 17.4%, $p< 0.0001$), the addition of ISO to DAI increased cytotoxicity compared to OGD with ISO (89% v 67.3%, p=0.01).

Conclusion: BAR agonism is lethal to neuronal cells and this effect increases after cell models of TBI. While BAR agonism could be inhibited after OGD, no inhibition occurred after the mechanical injury from DAI.



Poster # 36

DESTINED TO FAIL? PREDICTORS OF EXTUBATION FAILURE IN PATIENTS WITH TRAUMATIC BRAIN INJURY

Andrew Nguyen MD, David Plurad* MD, Amy Kaji MD,Ph.D., Scott Bricker MD, Fred Bongard* MD, Angela Neville* MD, Brant Putnam* MD, Dennis Kim MD, Harbor-UCLA Medical Center

Introduction: Extubation failure is a morbid complication associated with increased mortality in critically ill patients. The physiologic insult of failed extubation may be more pronounced in patients with traumatic brain injury (TBI). The purpose of this study was to identify risk factors for extubation failure in patients with TBI.

Methods: We performed a 4-year retrospective analysis of our Level 1 trauma center database to identify all adult blunt TBI patients requiring intubation for >48 hours. Extubation failure was defined as the need for reintubation within 48 hours of initial extubation. Variables analyzed included demographics, injury patterns and severity, weaning parameters, and outcomes. Patients that failed extubation were compared to patients that were successfully extubated. Multivariate logistic regression analysis was performed to identify independent risk factors for extubation failure.

Results: Of 127 patients, 26 patients (20.5%) failed extubation. On univariate analysis, extubation failure patients were older and more likely to have sustained multiple rib fractures. There were also significant differences in pre-exubation weaning parameters (Table). The median time to reintubation was 14 (8-19) hours. Patients that failed extubation required a greater duration of mechanical ventilation (14 vs. 3 days, $p<0.0001$), intensive care unit (17 vs. 6 days, $p<0.0001$), and overall length of stay (21 vs. 16 days, $p=0.02$). Patients in the extubation failure group were at an increased risk for pneumonia (OR=2.8; 95% CI, 1.1-7.3, $p=0.03$). On multivariate analysis, after controlling for age, weaning parameters, and ≥4 rib fractures, a rapid shallow breathing index (RSBI) ≥87 (OR=6; 95% CI 1.2-29.8, $p=0.03$) and ≥4 rib fractures (OR=10.4; 95% CI 1.0-111.3, $p=0.05$) were the only independent risk factors for extubation failure.

Variable	Extubation Failure	Extubation Success	p value
Age	58 (28-74)	48 (31-62)	<0.0001
≥4 Rib fractures	3 (12%)	1 (1%)	0.03
PaO2	122 (92-148)	136 (107-171)	0.04
RR	23 (20-29)	18 (14-24)	0.01
RSBI	50 (36-88)	39 (26-51)	0.01

Conclusion: Extubation failure is a common complication among patients with TBI. Application of a more conservative RSBI in TBI patients may minimize the risk for reintubation in this patient population.

Poster # 37

CERVICAL SPINE CLEARANCE IN CLINICALLY UNEVALUABLE PATIENTS: A POOLED ANALYSIS OF COMBINED CT AND MRI

Timothy P. Plackett DO, Franklin Wright MD, Anthony Baldea MD, Michael J. Mosier MD, Casey Thomas DO, Fred Luchette* MD, Hieu H. Ton-That MD, Thomas J. Esposito* MD,MPH, Loyola University Medical Center

Introduction: The role of cervical spine magnetic resonance imaging (MRI) in the evaluation of clinically unevaluable blunt trauma patients has been called into question by recent studies. Proponents of MRI note a superior sensitivity for detection of soft tissue and/or ligamentous injury compared to computed tomography (CT) scan. However, opponents highlight the low incidence of finding a significant missed injury and the high risk for developing soft tissue breakdown from use of immobilization devices.

Methods: A PubMed search was performed to identify all studies describing clinically unevaluable patients imaged with both CT and MRI to evaluate the cervical spine for injury. Results were combined for both radiologic findings and clinical outcomes. A pooled analysis was performed of the aggregate data. Subgroup analysis was performed to evaluate for any effects associated with improved computed tomography technology or timing of the MRI.

Results: A total of 1,714 clincally unevaluable patients were identified from 17 studies. All of the patients had a negative CT scan of the cervical spine and went on to receive a MRI. 271 (16%) patients had a new finding on MRI, with the majority being a ligamentous injury. Seventy-two patients (27%) were maintained in a cervical collar after the MRI and five patients (2%) underwent a surgical intervention. The remainder of the patients (71%) had the cervical collar removed. The propensity of MRI to find additional findings or direct treatment has not improved with increasing number of CT scan slices. Delaying MRI beyond 4 days after injury was associated with a significantly decreased incidence of new findings on MRI and lower probability for continued application of a cervical collar, however it had no effect on the incidence of surgery. Analysis of the patients requiring surgical intervention after the MRI questions whether these should have been noted on CT, the acuity of the finding, and whether the MRI findings were a contributing factor to the decision to operate.

Conclusion: This study demonstrates that MRI identifies additional injuries. However, these tend to be of minor clinical significance. The practice of routine MRI after a negative CT in clinically unevaluable patients is not supported by this study and should only be performed on an individual case basis.

Poster # 38

EVIDENCE BASED TIMING FOR DVT CHEMOPROPHYLAXIS IN TBI PATIENTS

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Introduction: Multiple studies in the literature have addressed timing of DVT chemoprophylaxis (CP) in traumatic brain injury. However, experts indicate that a precise time for safe and effective CP is uncertain.

Methods: A comprehensive brain injury literature review was performed to delineate temporal rates for 1) spontaneous intracranial hemorrhage (ICH) progression, 2) post-CP ICH expansion, and 3) post-CP DVT. Nineteen publications were found and included 5,163 patients.

Results: Spontaneous ICH expansion at 24 hours was 14.8% in 1,437 patients from CP studies and 29.9% in 1,257 patients not involved in a CP study ($p < 0.0001$). With low risk ICH (n=136), 99% of spontaneous ICH expansion occurred within 48 hours. With moderate or high risk ICH (n=109), 18% of spontaneous ICH expansion occurred after day 3. If patients with pre-CP ICH expansion are included, the post-CP ICH expansion rate was 5.6% in 1,258 with CP on days 1-3 and 1.5% in 401 with CP after day 3 ($p = 0.0116$). If patients with pre-CP ICH expansion are excluded, the post-CP ICH expansion rate was 3.1% in 1,570 with CP on days 1-3 and 2.8% in 582 with CP after day 3 ($p = 0.7769$). With DAI (n=188), the post-CP ICH expansion rate was 1.6% with CP after day 3. DVT rates were: no CP, 2.4% in 913; CP on days 1-3, 2.6% in 2,384; and CP after day 3, 3.4% in 930 ($p = 0.3430$).

Conclusion: Spontaneous ICH expansion rates at 24 hours substantially vary between CP and non-CP studies. CP should not be given within 3 days of injury for moderate or high risk ICH. CP is reasonable, when low-risk patients have not developed ICH expansion within 48 hours post-injury. CP is also acceptable after day 3, when low-risk patients develop ICH expansion within 48 hours post-injury. In DAI patients who have not developed an ICH within 72 hours, CP is reasonable.

Poster # 39

AN ANALYSIS OF THE PROTECTIVE BENEFITS OF BETA-BLOCKERS ON TBI PATIENTS

Mathew Edavettal MD,Ph.D., Katelyn J. Rittenhouse BS, Jo Ann Miller RN, BSN, Frederick B. Rogers* MD, MS Lancaster General Hospital

Introduction: The cerebroprotective benefits of beta-blockers (BB) in TBI patients are supported by a growing body of literature; however, the mechanism through which this effect is mediated is not well-understood. We sought to determine if selective BB (SBB) and nonselective BB (NSBB) impact TBI outcome in the same way.

Methods: In a PA-verified level II trauma center, all admissions ≥45 years of age and GCS ≤13 from October 2011 to May 2013 were queried. The impact of BB, both pre-injury and in-hospital, on stability at discharge was analyzed controlling for demographic and injury-specific variables in a multivariate logistic regression model. Stable at discharge was defined as neither moribund nor deceased at discharge A $p<0.05$ was significant.

Results: A total of 270 patients met study inclusion criteria. There were a total of 49 patients on selective BB (SBB) pre-injury and 17 patients on nonselective BB (NSBB) pre-injury. Additionally, a total of 80 patients were administered SBB in hospital and 39 patients were administered NSBB in hospital. When controlling for age, arrival GCS, ISS, ventilator use, and pre-existing conditions (PECs), SBB and NSBB administration in hospital as well as SBB pre-injury were associated with increased odds of stability at discharge for TBI patients. NSBB pre-injury was not found to significantly impact odds of stability at discharge.

Conclusions: Our data indicate that BB pre and post-injury can be beneficial to TBI patients. Interestingly, while SBB and NSBB both have protective effects when administered in hospital, only SBB pre-injury are beneficial to TBI patients.

	Adjusted* Odds Ratio (95% CI)	p-value
BB Pre-Injury		
None	Reference	-
Selective	4.90 (1.25-19.22)	0.023
Nonselective	0.96 (0.09-10.13)	0.974
BB In Hospital		
None	Reference	-
Selective	3.09 (1.06-8.99)	0.038
Nonselective	9.78 (2.28-42.0)	0.002

*Adjusted for age, arrival GCS, ISS, Vent use, & PECs.

ROC: 0.90

Poster # 40

HEAD COMPUTED TOMOGRAPHIC MEASUREMENT: A PREDICTOR OF OUTCOME IN PATIENTS WITH SUBDURAL HEMATOMA WITH CEREBRAL EDEMA

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Introduction: This study aimed to evaluate whether differences in head computed tomographic (CT) measurements in Hounsfield units (HU) of white matter (WM) and gray matter (GM) can be used as a predictor of outcome in patients with subdural hematoma with cerebral edema.

Methods: We evaluated 34 patients who had subdural hematoma with cerebral edema and had undergone head CT within a few hours of admission. We divided the patients into two groups according to outcome: survival (S group, n=24) and death (D group, n=10). We obtained HU measurements of the WM and GM at 6 points (injury and non-injury site at each of the frontal, temporal, and occipital lobes). We also measured the displaced distance from the median (DDM). We assessed the correlation between outcome and HU measurements of the WM and GM or DDM. The paired t-test was used to calculate statistical significance. Data are shown as the mean (SD).

Results: Causes of head injury were motor vehicle accident (n=19), fall (n=9), and other cause (n=6). Operations for hematoma removal and external decompression were performed on 24 patients. All patients who died underwent these operations, and cause of death in each patient was brain herniation due to cerebral edema. At the injury site, HU of GM was 38.4 (2.9) in the S group and 36.8 (3.1) in the D group ($p=0.45$). The HU of WM at the injury site in the S group was significantly higher than that in the D group (34.2 [2.5] vs. 29.5 [3.4]) ($p<0.01$). The HU of GM at the non-injury site was 38.9 (2.9) in the S group and 36.8 (3.1) in the D group ($p=0.92$), and the HU of WM was 34.2 (2.5) in the S group and 32.9 (3.4) in the D group. There was no significant difference in the DDM between the S and D groups ($p=0.15$).

Conclusion: This study suggests that measurement in HU of WM at the injury site may be useful as a predictor of outcome in patients with subdural hematoma with cerebral edema.

Poster # 41

NOSOCOMIAL PNEUMONIA IS INDEPENDENTLY ASSOCIATED WITH WORSE FUNCTIONAL OUTCOME IN BRAIN INJURY PATIENTS 5 YEARS AFTER INJURY

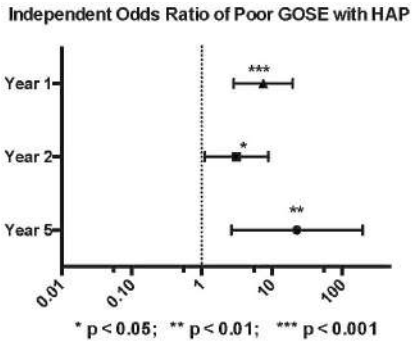
Matthew R. Kesinger BA, Raj G. Kumar MPH, Amy K. Wagner MD, Juan C. Puyana* MD, Andrew P. Peitzman* MD, Timothy R. Billiar* MD, Jason L. Sperry* MD,MPH, University of Pittsburgh

Introduction: Long term outcomes following traumatic brain injury (TBI) are known to correlate with initial head injury severity. Hospital-acquired pneumonia (HAP) is a common complication in patients with TBI. However, little information exists regarding the significance of infectious complications and their effect on long term outcome following TBI. We sought to characterize the risks associated with HAP on long term neurological outcome following severe TBI.

Methods: A retrospective analysis was performed utilizing data derived from the merger of a single institution trauma registry and TBI Model Systems outcome data. Individuals with severe head injuries (Abbreviated Injury Scale ≥ 4), who survived to rehabilitation were analyzed. Primary outcome was Glasgow Outcome Scaled Extended (GOSE) at 1, 2, and 5 years after discharge. GOSE was dichotomized into LOW (GOSE < 6) and HIGH (GOSE ≥ 6) groups. Logistic regression was utilized to determine the independent risk of a LOW GOSE score associated with HAP after controlling for differences in age, head and overall injury severity, cranial surgery, Glasgow Coma Scale (GCS), early intubation status, and other important confounders.

Results: A total of 141 individuals met inclusion criteria, with a 30% incidence of HAP. Individuals with and without HAP were similar in demographics, presenting vitals, head injury severity and the need for cranial surgery. Individuals with HAP were more likely to be intubated and had a corresponding lower presenting GCS. After controlling for potential confounders, logistic regression demonstrated that HAP was independently associated with LOW GOSE scores at 1 year, (OR = 7.5, p<0.001, 95%CI 2.85-19.91), at 2 years, (OR = 3.1, p=0.031, 95%CI 1.11-8.84), and at 5 years (OR = 22.76, p=0.004, 95%CI 2.64-196.5). After stratifying the analysis by the need for early intubation, HAP remained a significant independent predictor of LOW GOSE at 1 year even in patients who required early intubation.

Conclusion: HAP is independently associated with poor neurological outcome in individuals with severe TBI. This significant greater risk of poor neurological outcome extends out to 5 years post-injury. This study suggests that precautions should be taken to significantly reduce the risks of HAP, and other infectious complications, in those individuals with severe TBI.



Poster # 42

PRE-ADMISSION DO NOT RESUSCITATE (DNR): AN INDEPENDENT PREDICTOR OF DEATH FOLLOWING TRAUMA?

Randeep S. Jawa MD, Jane E. McCormack RN, Daniel N. Rutigliano DO, Emily C. Huang MS, Steven Sandoval MD, Michael F. Paccione MD, Marc J. Shapiro* MD, James A. Vosswinkel MD, Stony Brook University Hospital

Introduction: Hospitals are required to inquire about advanced directives on patient admission. There is very limited literature on the influence of DNR status on outcomes in trauma; it is not commonly used in evaluating hospital case mix or outcomes. We hypothesized that pre-admission DNR status is an independent risk factor for death following trauma. **Methods**: We reviewed the trauma registry for patients over age 40 admitted between 2008-2013 at all hospitals in a suburban county of 1.5 million people. This county-based registry includes data from state designated non-trauma centers, area trauma centers, and 1 regional trauma center. Isolated hip fractures are not included. Statistical analyses were performed using parametric tests, nonparametric tests, and multivariate logistic regression; $p \leq 0.05$ was considered significant.

Results:*$p \leq 0.05$, IQR - interquartile range, LOS- length of stay

	Pre-admission DNR	No DNR
Patients with age>40 year (n)	330	7748
Gender (male)	37.9	51.3*
Age (years, median with IQR)	87 (81-91)	69 (54-82)*
Dementia (%)	43.1	8.6*
On anticoagulants (%)	28.1	17.2*
Respiratory Disease (%)	14.7	8.5*
Fall (%)	94.2	65.1*
MVC/pedestrian struck/bicycle (%)	3.4	27.2*
Injury Severity Score (median, IQR)	14 (9-17)	11 (9-17)*
Head AIS $\geq$3 (%)	67.6	47.1*
Hospital LOS (days, median, IQR)	7 (4-11)	6 (4-11)
ICU stay (%)	39.4	36
mechanical ventilation (%)	16.2	31.8
mechanical ventilation days (median, IQR)	3 (2-6)	5 (2-12)*
Pneumonia (%)	12.5	5.9*
Acute MI (%)	3.4	1.3*
Comfort care (withdrawal of care) (%)	9.5	2.8*
In-hospital mortality (%)	33.6	5.9*

Multivariate logistic regression identified multiple significant risk factors for in-hospital mortality, in order of odds ratio with 95% CI: mechanical ventilation 27.7 (19.9-38.5), pre-admission DNR 7.56 (5.4-10.7), ICU stay 1.8 (1.3-2.4), pre-admission respiratory disease 1.5 (1.1-2.2), male sex 1.5 (1.2-1.9), pre-admission anticoagulation 1.3 (1.0-1.7), ISS 1.1 (1.1-1.1), and age 1.1 (1.0-1.1). Injury mechanism, head AIS$\geq$3, dementia, the presence of complications (grouped together) were not significant. **Conclusion**: After mechanical ventilation, pre-admission DNR status is the strongest independent predictor for in-hospital mortality. Only a fraction of the mortality was because of subsequent change in patient status to comfort care. Consideration should be given to adding DNR status to trauma outcome scoring systems and in benchmarking hospital quality measures following trauma, with its attendant financial implications.

Poster # 43

"TIER 3": A NOVEL ADDITION TO A CONVENTIONAL TWO-TIERED TRIAGE SYSTEM DESIGNED TO EXPEDITE CARE OF GERIATRIC TRAUMA PATIENTS

Forrest B. Fernandez MD, Ryan Spinka BS, Susan Butler RN, Adrian Ong* MD,
Reading Health System

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction: Prior to February 2009, patients presenting to the emergency department (ED) were either evaluated by the trauma team as "activations" if they met formal activation criteria or as "consults". To improve the efficiency of triage in the ED, a three-tiered system was introduced in February 2009. This additional triage tier (T3) utilized a time-targeted evaluation and treatment protocol aimed at patients who remained at risk for life threatening injuries but did not meet formal activation criteria. T3 required initial evaluation by a rapid response team headed by the ED physician using resources intrinsic to the ED without trauma team involvement. Criteria for T3 included patients without hemodynamic alteration but had risk factors including antiplatelet or anticoagulant agents, as well as elderly patients with low energy injury mechanisms that did not meet conventional activation criteria.

Methods: All adult patients from two time periods, one prior to introduction of T3 (Pre T3, Nov 2007-Jan 2009) and one after (postT3, Nov 2010- Jan 2012) were compared with respect to admission systolic blood pressure (SBP) , Glasgow Coma Score (GCS), Injury Severity Score (ISS) and mortality, using univariable analyses. Patients above 60 years old were similarly compared between the two periods separately. A p value of 0.05 indicated statistical significance.

Results: The trauma team evaluated 2682 and 3360 patients in the PreT3 and PostT3 period respectively. In the PostT3 period, 23% of patients were seen as T3 patients initially by the ED team. The proportion of all trauma patients requiring trauma team activation declined after T3 introduction (74 vs. 62%, $p<0.001$). Compared to PreT3 patients, PostT3 patients had a greater mean age (54.1±24.9 vs 47.2±24.1 years, $p<0.001$), lower mean GCS (14.2± 2.4 vs 14.4±2.3, $p=0.02$), and a lower mean ISS (7.5±7.8 vs 9.3±9.1, $p<0.001$). Mortality was similar (3.1 vs 3.3%, $p=0.6$). Patients above 60 years comprised a greater proportion of all patients in the postT3 period (44 vs 31%, $p<0.001$). Compared to PreT3, PostT3 elderly patients had similar mean GCS, lower SBP (147.8±35.1 vs 151.4±35.3 mmHg, $p<0.001$) and lower mean ISS (8.0±6.8 vs 10.8±8.9, $p<0.001$). Mortality was lower in the postT3 elderly cohort (4.6 vs 7.0%, $P=0.02$).

Conclusion: Addition of a new triage level harnessing resources intrinsic to the ED reduced the need for trauma team activations and was associated with a lower mortality rate in elderly patients. As elderly patients with low energy injury mechanisms form a significant proportion of trauma patients, this triage level potentially can expedite trauma evaluations while sparing trauma team resources.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

Poster # 44

TIME TO DEATH FOLLOWING INJURY -- 20 YEARS WITH LITTLE PROGRESS

Carrie Valdez MD, Babak Sarani* MD, Hanna Young BA, Richard Amdur Ph.D., James Dunne* MD, Lakhmir Chawla MD, George Washington University

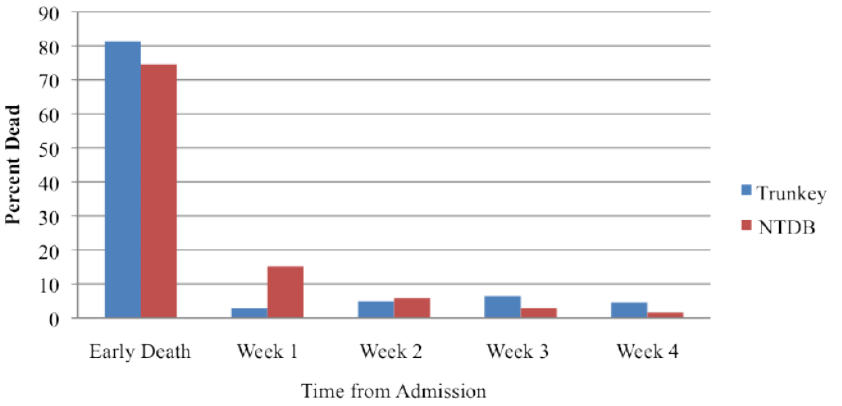
Background: The trimodal distribution of death following injury was first described by Trunkey in 1983. Subsequent studies have found that admission to a trauma center significantly decreases the probability of death following injury. However, there have not been any recent studies to determine the time to death following injury. Given advances in trauma care, we postulate that the time to death histogram described has shifted to the right. This study seeks to determine the timing of trauma-associated mortality and to describe injury or combination of injuries that are associated with early or late death versus survival.

Methods: A retrospective analysis was conducted on the National Trauma Data Bank (version 7.2) from 2002 to 2006. Pediatric patients (age < 18) and burn victims were excluded. Early death was defined as dead on arrival or died within 24 hours of admission. Pearson's χ^2 test was used to compare region of injury to mortality. Multivariate logistic regression was conducted to show the independent effect of region of injury on mortality while controlling for demographic factors and injury type.

Results: The cohort includes 898,982 patients. The mean injury severity score (ISS) was 10.54 ± 10.11. Overall mortality rate was 5.14%, 54% died early. The majority of all deaths occurred between day 0 and 1 with 41% of occurring on day 1. No difference was noted in time to death relative to Trunkey's report (Figure 1). Torso injuries were more prevalent among early deaths (7.78% v 5.43%, $p<0.001$). Survivors were more likely to have a blunt mechanism of injury (89% v 11%, $p<0.001$) and had a lower ISS (10 ± 9 v 28 ± 17, $p<0.001$). These results did not change on multivariate regression modeling.

Conclusion: The time to death following injury has not changed since 1983. In addition to stressing injury prevention, ample opportunity remains to impact mortality in the first 24 hours following injury, particularly following penetrating or torso injuries. Studies directed at early treatment of these injuries are needed.

Figure 1: p = NS



Poster # 45

DETERMINATION OF THE RELATIONSHIP BETWEEN MILD TRAUMATIC BRAIN INJURY DIAGNOSIS AND THE DEVELOPMENT OF POSTTRATUMATIC STRESS DISORDER IN CIVILIAN TRAUMA SURVIVORS

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INTRODUCTION: The development of posttraumatic stress disorder (PTSD) in civilian trauma survivors significantly decreases quality of life. In these patients, mild traumatic brain injury (mTBI) is often undiagnosed. The goals of the present study were to establish the missed rate of mTBI and determine the relationship between mTBI and PTSD.

METHODS: Records of all patients 18 - 96 years with blunt mechanism trauma who were admitted at a Level I trauma center and reported a PTSD Checklist-Civilian version (PCL-C) score 6 months post injury were reviewed. A PCL-C score of >44 was used as the standard for diagnosis of PTSD symptoms. Patients with clinically diagnosed mTBI based on ICD-9 codes and DSM-V criteria during admission were recorded. A missed mTBI diagnosis was defined using World Health Organization criteria of loss of consciousness and/or neurological symptoms and Glasgow Coma score ≥13. Statistical analysis was performed using ANOVA, chi squared, t-test and linear regression.

RESULTS: Of 347 trauma patients, 215 (62%) were male and 132 (38%) were female with an average age of 52 years. Assaultive mechanism of injury was present in 28 (8%) of patients. 31 (9%) had clinically recognized mTBI and 48 (13.8%) had a missed mTBI. Patients with a clinical and missed diagnosis of mTBI (n=79) reported significantly higher PCL-C scores (p=.001) and significantly higher incidence of PCL-C score >44 (p=.019, relative risk=1.58) than those without an mTBI. Using a linear regression of younger age, male gender, assaultive injury mechanism and mTBI, mTBI diagnosis significantly predicted a higher PCLC score$F(4,374)=19.445$, $p<.0005$, $R^2=.172$, with all four variables adding significantly to the model, $p<.05$.

mTBI Diagnosis	Mean PCL-C score (SD)	Patients with PCL-C Score >44
Missed and clinical (n=79)	41.09 (19.16)*	28 (35.4%)*
Absent (n=268)	33.68 (16.42)	60 (22.4%)

*p<.05

CONCLUSION: Having a diagnosis of mTBI predicted clinically significant PTSD scores and increased the relative risk of screening positive for PTSD symptoms. Given the underdiagnosis of mTBI and its associated risk for PTSD, screening for mTBI may help identify at risk patients for early intervention.

Poster # 46

FROM TQIP TO THE GERIATRIC TRAUMA INSTITUTE: DEVELOPING AN INNOVATIVE CARE MODEL FOR THE COMING STORM

Connie M. DeLa'O MD, Russell D. Dumire* MD, Aurelio Rodriguez* MD, Thomas J. Simunich MS Conemaugh Memorial Medical Center, General Surgery Residency

Introduction: By 2030, the US geriatric population is projected to steadily increase to 20.6% . The increased burden to trauma services will be significant. Advanced age is a risk factor for adverse outcomes following trauma. An inverse relationship exists with mortality and age. These facts have led leading US trauma organizations to establish practice guidelines. The American College of Surgeons Committee on Trauma has published triage criteria in an attempt to address the need for a more specialized treatment approach in these patients. This study sought to transform the existing geriatric trauma care model into one that is more effective, efficient, financially sustainable, and capable of absorbing the anticipated increased demand.

Methods: The study goals were to improve the geriatric trauma care process - for patient and hospital, to detail its creation, development, implementation, and to provide a formative evaluation of the result. A team, comprised of stakeholders, both internal and external to the hospital, was assembled and included clinicians from multiple disciplines and administrative and technical staff. During 18 months, tools and concepts of process redesign and Lean Six Sigma were applied to create, develop, implement, and evaluate the resulting novel model of care. Retrospectively, formative evaluation was accomplished by comparing pre-GTI data time matched with that from the first eight months post-GTI initiation.

Results: The intense process redesign produced the Geriatric Trauma Institute (GTI). This novel multidisciplinary care model has achieved 100% involvement of institution orthopedists with 100% of geriatric trauma admissions being converted to the GTI. In the 8 months after GTI inception, geriatric trauma service admissions increased 26.6%, from 338 to 460 patients, with a 78.2% decrease in non-trauma service admissions. A 28.2% decrease in transfers to other hospitals was seen. The analysis revealed a 26.1% increase of patients dispositioned to home, a 47.2% decrease to rehabilitation facilities, and notable decreases to both skilled nursing and transitional care facilities.

Conclusion: The GTI has succeeded as evidenced by the quantifiable benefits to both the patient and the hospital. During the development process, new work processes, tools, and staff training helped to boost the utilization of the trauma service regarding geriatric trauma care via a novel multidisciplinary approach. The GTI has demonstrated sustained and continuous quality improvement in geriatric trauma care. The trauma service maintains the performance gains through the trauma service performance improvement initiative.

Poster # 47

PREOP CT IN PATIENTS MEETING ATLS DIRECT-TO-OR CRITERIA FOR EXPLORATORY LAPAROTOMY: RISKY BUSINESS?

Michael J. Sise* MD, Christopher P. Foran MD, Casey E. Dunne MPH, Jayraan Badiee MPH, Carol B. Sise RN, Kimberly A. Peck* MD, William D. Dutton MD, Steven R. Shackford* MD, Scripps Mercy Hospital Trauma Service

Introduction: Previous studies suggest that preop CT in patients with abdominal trauma who meet criteria to go directly to the OR for exploratory laparotomy (Ex Lap) results in worse outcomes. We tested that hypothesis.

Methods: Patients who underwent Ex Lap for abdominal trauma over a 7-year interval at a Level I trauma center were retrospectively evaluated using ATLS criteria to identify those who should go directly to the OR. Patients who actually went directly to the OR (D-OR) were compared to those who received CT prior to going to the OR (CT-OR). Injury data, time in CT, and operative procedures were reviewed. Death and complication rates were determined to compare outcomes.

Results: Of the 402 patients who underwent Ex Lap, criteria to go directly to the OR were met in 157 (39%). D-OR occurred in 123 (78%) and CT-OR in 34 (22%). In CT-OR, mean time in CT was 27 minutes. Mean age was lower in D-OR compared to CT-OR (28.9 vs. 36.5, p=0.002). Penetrating mechanism was more frequent in D-OR vs. CT-OR (76% vs. 47%, p=0.001). Mean ISS and AIS-abdomen were similar in D-OR and CT-OR (22.6 vs. 23.6, p=0.75; 3.3 vs. 3.5, p=0.49). Mean AIS-chest was 1.6 in both groups. Operative procedures and outcomes for D-OR and CT-OR are displayed in the Table. Rates of D-OR vs. CT-OR neither varied over the study interval (p=0.09) nor among trauma surgeons (p=0.67).

Conclusion: Preop CT in patients who met direct-to-OR status by ATLS criteria was not associated with worse outcomes. CT findings may be important in effective operative management. This merits further study.

	D-OR	CT-OR	P-Value
N (%)	123 (78%)	34 (22%)	
Non-therapeutic Ex Lap	6 (5%)	0	0.34
Mean units PRBC	4.8	4.6	0.85
Resuscitative Thoracotomy	14 (12%)	0	0.041
Damage Control Ex Lap	44 (36%)	6 (18%)	0.045
Death in the OR	10 (8%)	1 (3%)	0.46
Deaths Predicted / Observed	18% / 15%	16% / 9%	0.67 / 0.57
Complications	29%	18%	0.20

Poster # 48

RECRUITMENT OF TRAUMA/SURGICAL CRITICAL CARE FACULTY REVERSES DECLINE OF PATIENT OUTCOMES NOTED WITH PREVIOUS FACULTY ATTRITION

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Introduction: Attrition of surgeons occurs with frequency due to a high rate of burnout. Disruption of a trauma/critical care service may have an impact on quality measures. We examined the effect of the attrition and recruitment of a trauma/surgical care team on trauma patient outcomes.

Methods: A retrospective review was performed encompassing fiscal years (FY) (Oct-Sept) 2010-2012 at an 825-bed Level I trauma center. In 2010, surgical faculty included a full complement of trauma surgeons. During 2011, 7 trauma/surgical critical care faculty departed. Between September 2011 and January 2012 four full time trauma/surgical critical care faculty members were recruited. Total number of trauma admissions, Injury Severity Scores, and both infectious and non-infectious clinical outcomes were reported across this time of evolution. Fiscal year 2010 was compared to 2011 (*attrition*) and 2011 was compared to 2012 (*recruitment*). Student t-tests and Chi squared were applied where appropriate. P-values < 0.05 were significant.

Results: 6,633 patients were evaluated with no ISS change over time. During *attrition* an increase in ICU days was observed (5.9 vs. 6.9; $p<0.05$). Trends toward greater episodes of VTE (115 vs. 142; $p=0.02$), sepsis (42 vs. 60; $p=0.09$), unplanned ICU readmissions (37 vs. 45; $p=0.42$), and mortality (4.5% vs. 5.4%; $p=0.20$) were observed. During *recruitment*, sepsis cases (60 vs. 38; $p=0.02$), pressure ulcers (25 vs. 13; $p=0.04$), ICU readmissions (45 vs. 29; $p<0.049$), VTEs (142 vs. 83; $p=0.0001$), ventilator days (8.5 vs. 5.4; $p<0.0001$), ICU days (6.9 vs. 5.2; $p<0.0001$), and hospital days (6.3 vs. 5.4; $p<0.001$) improved. Trends toward lower episodes of VAP (7 vs. 2; $p=0.09$) and mortality (5.4% vs. 4.2%; $p=0.07$) were observed.

Conclusion: Disruption of a dedicated trauma/surgical critical care service impacts trauma patient quality measures and ICU throughput. Commitment to recruitment/retention appears to reverse the associated decline in quality and resource utilization within 1 year. A symbiotic relationship between hospital and trauma surgeon personnel is required to obtain excellent patient outcomes and optimal use of resources.

Poster # 49

THE DIFFERENCE BETWEEN THE PERFORMANCE OF PARAMETERS OF PROBABILITY OF SURVIVAL IN DEVELOPED AND DEVELOPING COUNTRIES

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Introduction: Despite its known limitations, the TRISS methodology remains the "gold standard" for the analysis of probability of survival in trauma. Several efforts have been taken to increase its accuracy. TRISS has been criticized for being based on North American data, which may not be applicable to low and middle income countries. Therefore, we evaluated the accuracy of three models of probability of survival in both a high (HIC) and a middle income country (MIC) to compare the performance of the three new adjustments to the TRISS equation models (NTRISS-like, TRISS SpO_2 e NTRISS-like SpO_2) when derivatives and applied to different groups of trauma patients.

Methods: This is a two center, retrospective study of trauma victims admitted to a university medical center in South America and a Level-1 university-based trauma center in in the US during the period between January 1 st, 2006 and December 31st, 2010. Patient data were grouped into two different databases: derivation and testing; the first served to derive the equations and the second was used to validate the equations initially generated. The model coefficients were established by logistic regression analysis. Receiver Operating Characteristic curves (ROC) were used to evaluate the performance of the models and the De Long et al. algorithm was used to compare the areas under the curves (AUC).

Results: 2,416 patients from the MIC and 8,172 patients from the HIC were studied. The models applied were NTRISS-like which included the Best Motor Response (BMR), Systolic Blood Pressure (SBP), New Injury Severity Score (NISS) and age; TRISS SpO_2 which included Glasgow Coma Scale (GCS), SBP, peripheral oxygen saturation (SpO_2), Injury Severity Score (ISS) and age; and NTRISS-like SpO_2 (BMR + SBP + SpO_2 + NISS + age). All equations had adjusted coefficients for blunt and penetrating trauma. The performance of the models was different when applied to patients from the MIC compared to the HIC. Regardless of the population where the equation was generated it had better performance when applied to HIC patients (AUC from 0.911 to 0.982) compared to MIC patients (AUC from 0.840 to 0.852).

Conclusion: Models of probability of survival derived from injury data collected in HIC are not reliable when used in data obtained in MIC, where trauma care may not be organized in the same manner. The results of the study suggest that other factors besides physiologic and anatomic data may have an impact on final outcome and should be identified in each environment if they are to be used in the development of a trauma performance improvement process in MIC.

Poster # 50

ANTICOAGULANT AND ANTIPLATELET AGENTS: NO PROBLEM FOR THE ELDERLY WITH A LOW-LEVEL FALL

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Introduction: Falls are the leading cause of injury in older patients. Use of antiplatelet (AP) and anticoagulant (AC) drugs in this population is common and thought to complicate the care of these patients. We hypothesized that AP and AC use in older patients with low-level falls worsens outcomes.

Methods: We studied all patients ≥60 years admitted to our level II trauma center with low-level falls during 2012-2013. Use of AC or AP medications, labs, outcomes, type of fall, and injuries were extracted from the electronic record and trauma registry. Fischer exact and Student t-tests were applied, where significance was $p \leq 0.05$.

Results: 900 patients had a low-level fall with an overall mortality of 5.6%. AC/AP patients were older but with similar outcomes as non AC/AP patients (see table). For head-injured patients, AC/AP mortality was the same as for those not taking these medications ($p=0.45$). The mean head AIS was higher for those who died, 4.5 compared to 3.3, $p<0.001$. The percentage of AC/AP use was the same in survivors (55%) as non-survivors (53%). For AC patients only, there was a slightly longer LOS at 5.3 days ($p<0.05$), but similar ISS, age and mortality to patients not on AC/AP medications. Our hospital severity of illness measure, which scores premorbid medical conditions, was worse for patients who died whether in the AC/AP or no medication group ($p<0.0001$).

Conclusion: Despite our clinical bias that elderly patients who take AC/AP agents are more likely to sustain higher degrees of injury and worse outcomes after low impact falls, AC/ AP patients had the same overall injury severity and mortality as their non-AC/AP peers. Mortality was associated with severity of medical comorbidities at admission.

Group	Age years	ISS	Brain AIS (N)	Hospital LOS days	Mortality N (%)
AC/AP (N= 493)	83±9	8.7±5	3.4 (60)	4.8±4	27 (6)
No AC/AP (N=407)	80±10	8.5±5	3.5 (34)	4.5±4	24 (6)
P value	<0.001	NS	NS	NS	NS

Poster # 51

EFFECT OF PREHOSPITAL INTUBATION ON SCENE TIMES IN THE URBAN SETTING

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Introduction:
Prehospital intubation is often necessary to support the respiratory physiology of the critically injured patient. It is however skill and time intensive. We hypothesized that need for Prehospital intubation would lead to prolonged scene times delaying arrival to definitive care.

Methods:
A retrospective analysis of trauma registry data at our urban level I trauma center was performed to identify patients who arrived with an advanced airway intervention for the 10-year period, 2003-2012. Data abstracted included: demographics, injury type and severity, physiologic variables, scene and prehsopital time, and outcome. Statistical analysis was performed using the student's t test, ANOVA, and chi square test as appropriate. A p value of <0.05 determined statistical significance.

Results:
Over the 10-year period, complete data was available in 844 of 919 patients who arrived having received active advanced airway intervention in the Prehospital setting. The majority were male (648 vs. 196, p=0.31), and had sustained blunt trauma (679 vs. 155, p=0.31). Scene times were significantly longer in patients who were either intubated in the field or had undergone a failed attempt at intubation compared to those brought with bad-mask ventilation (17.66 ± 9.59 vs. 15.1 ± 10.15 vs. 13.3 ± 7.32 minutes, respectively, $p<0.0001$, ANOVA). No difference in total Prehospital time was noted (33.62 ± 11.23 vs. 30.17 ± 11.45 vs. 29.76 ± 10.26 minutes, p-0.15, ANOVA). Patients arriving by air were significantly more likely to be intubated ($p<0.00001$). Intubated patients were significantly more likely to be in shock/extremis on arrival, have no signs of life, and have a GCS < 8. They were more severely injured (ISS 27.22 ± 19.1 vs. 24.08 ± 15.35 for intubated vs. bag-mask, $p<0.0005$, Student's t test). Mortality was highest in the intubated group (65.5% for intubated, 61.2% for failed intubation, and 54.6% for bad-mask group, p=0.01, chi square test).

Conclusion:
Prehospital intubation prolongs scene times and potentially delays arrival to definitive care for the more severely injured patients in whom it is more often employed. Intubated patients arrive with a greater degree of hemodynamic instability and have higher mortality rates .Bag-mask ventilation when adequate reduces scene time. When inadequate, the use of supra-glottic devices should be explored as a means of achieving the goal of expeditious transport while maintaining physiology.

Poster # 52

WHAT SYSTEM-LEVEL FEATURES, IF ANY, PREDICT BETTER OUTCOMES FOR PATIENTS IN TRAUMATIC CARDIAC ARREST?

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Introduction: Many perceive resuscitation is futile in established cardiac arrest due to trauma as outcomes are so poor. This position is supported by Guidelines from the American College of Surgeons Committee on Trauma, which recommend withholding resuscitation in blunt trauma patients who are apneic, pulseless and without organized ECG activity, and in penetrating trauma if signs of life are absent. Expected transport time to an emergency department of more than 15 minutes implies a non-survivable condition. In contrast, several published case series report better outcomes, especially in selected patients and when prioritizing hemorrhage control, relief of tension pneumothorax and airway opening over conventional cardiopulmonary resuscitation. We wished to assess the effectiveness of our ambulance service protocol for traumatic cardiac arrest and identify system-level features associated with improved outcomes.

Methods: We identified all adult patients (≥16 years old) with pre-hospital cardiac arrest secondary to trauma were in our ambulance service Out-of-Hospital Cardiac Arrest (OHCA) Database 2000-2012. We sought predictors of sustained return of spontaneous circulation (ROSC) to hospital arrival in those patients for whom resuscitation was attempted. Our ambulance service protocol for traumatic arrest mandates resuscitation be attempted for at least 20 minutes in all but patients with an obvious non-reversible condition (such as massive head injury), with airway control, chest decompression, hemorrhage control and IV fluid prioritized over chest compressions and defibrillation. Transport is not attempted until ROSC.

Results: We identified 45,742 adult OHCA, with the leading cause being cardiac disease (65.8%), followed by trauma (7.9%). Compared to non-trauma arrests (n=42,124), trauma arrests (n=3,617) were more likely to occur in younger (median 41 vs. 68 years old) males (78% vs. 67%). Of the 3,617 traumatic arrests, resuscitation was attempted in 1,277 (35.3%). Traumatic OHCA patients in whom resuscitation was attempted attained ROSC in 17.6% of cases; not significantly different from ROSC in non-traumatic arrest (22.8%). Asystole was the most common initial rhythm in traumatic OHCA (70.1%). In multivariable analysis, significant predictors of ROSC in traumatic OHCA were age>65 (OR 2.2; p=0.003)(possibly due to a lesser magnitude of trauma), shockable rhythm (OR 2.0; p=0.022), ambulance response time ≤ 8minutes (OR1.8; p=0.009), attendance of an intensive care paramedic or physician (OR 5.8; p<0.001) and time on scene >15 minutes (OR 2.7; p<0.001). Attempted cardiopulmonary resuscitation by a bystander prior to ambulance arrival did not predict ROSC.

Conclusion: Attempted resuscitation of patients with traumatic OHCA treated with our ambulance service protocol is not futile. Reducing ambulance response times, augmenting standard ambulance crews with advanced-care clinicians, and emphasizing that transport should not be attempted until ROSC is attained might all further improve outcomes. Bystanders would be better advised to facilitate early ambulance attendance, and perhaps to attempt airway and hemorrhage control, than to undertake conventional cardiopulmonary resuscitation. Further information is needed on survival to hospital discharge and neurological function post-resuscitation.

Poster # 53

TRANSPORTATION TIME IN A RURAL ENVIRONMENT FOLLOWING SPLENIC INJURY: DOES TIME MATTER

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Background: Failure rates remain high following attempted non-operative treatment of spleen injuries despite progress made in identifying risk factors. In the past, transportation times were excluded from predictive models although rapid transportation is advocated to improve patient outcomes. For patients living in a rural environment, this time may prove critical. The purpose of this study was to evaluate survival rates and hospital length of stay for patients selected to receive non-operative versus operative treatment of splenic injury, inclusive of transportation time.

Methods: A 10-year retrospective review was conducted of patients > 18 years presenting to an ACS-verified level 1 trauma center from January 1, 2003 to December 31, 2012. Failed non-operative management (FNOM) was defined as angioembolization or planned operation > 2 hours. Proportional hazard Cox regression and logistic regression analysis were conducted to identify factors associated with hospital length of stay (H-LOS) and mortality. The possible factors include: age, gender, injury severity score (ISS), injury type (blunt or penetrating), treatment modality (non-operative management (NOM), FNOM, or immediate operation (OR) within 2 hours), and transportation time from the time EMS received the phone call to admission.

Results: Among the 364 patients included in the final analysis, 11.0%(n=40) died before hospital discharge. The median transport time was 64 minutes (average=92.6 ± 81 minutes, range=6 to 480 minutes). Majority (92.9%, n=338) of patients underwent NOM, with 7.1% (n=26) receiving OR < 2 hours. Among those 338 NOM patients, 92.3% (n=312) remained NOM after 2 hours, and others had FNOM after 2 hours (7.7%, n=26). Those who received operative intervention<2 hours or NOM before 2 hours were associated with 45.5% and 47.4% of the transportation time being less than 60 minutes, respectively. After 2 hours, those who initially received an immediate OR within 2 hours, remained NOM, or qualified as FNOM had an average ISS score of 23.83, 21.96, and 28.07. Proportional hazard Cox regression analysis reported that ISS score was the only significant predictor for H-LOS. Logistic regression revealed that ISS score and age were associated with mortality. Transport time was not statistically associated with H-LOS or mortality.

Conclusion: While not predictive of H-LOS or mortality, transportation time demonstrated that in rural environments prolonged transportations allow physiologic symptoms to manifest prior to admission. This resulted in decreased FNOM, where the majority (96%) occurred < 6 hours following admission and 100% < 48 hours. Recommendations call for intensive observation < 24 hours following admission, with less robust surveillance through hospital day 2. Discharge can be considered on hospital day 3 based on other injuries.

Poster # 54

VIOLENT TRAUMA RECIDIVISM: UNUSUAL BUT DEADLY

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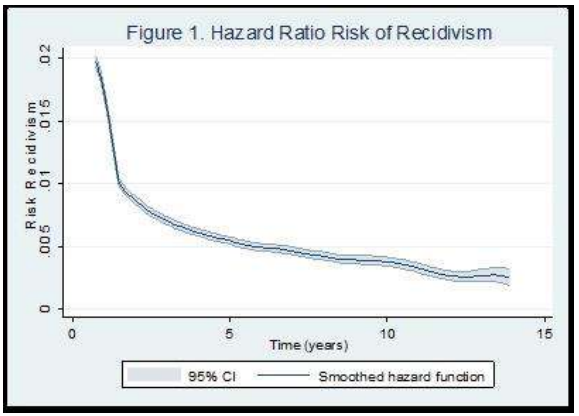
Introduction: Recurrent violence is a significant problem, however studies of recidivism following violent trauma are difficult due to the poor follow up and the migratory nature of trauma patients. We sought to identify risk factors for recidivism following admission for violent trauma within the state of California, and the effect of recidivism on mortality.

Methods: The California Office of Statewide Health Planning and Development (OSHPD) hospital discharge database was searched for all patients admitted between 1995-2010 with E-codes for violent assaults. Recidivists were defined as patients with repeat admissions for violent assaults. Multivariate analysis was used to compare recidivists to non-recidivists. Factors included; admission year, age, gender, race, insurance status, injury type, Survival Risk Ratio, Charlson co-morbidity index, hospital type, and county. Mortality differences between recidivists and non-recidivists were also compared.

Results: 168,814 patients met inclusion criteria, the majority of patients, 84.5%, were male. Penetrating trauma accounted for 43.6% of patients. Recidivism was seen in 6.8% of patients; the majority (43.6%) occurring within the first 6 months, with a median follow up of 7 years (Figure 1). Migration among recidivists was high with 41.9% of patients presenting to different hospitals for subsequent admissions. A significantly ($p<0.001$) increased risk of recidivism was seen in those of African American race (OR 1.25), MediCal (OR 1.37), Medicare (OR 1.43), alcohol (OR 1.36) or illicit substance use (OR 1.25) and combined alcohol/illicit substance use (OR 1.52). Patients in Alameda (OR 1.17), San Francisco (OR 1.19), and Los Angeles (OR 1.68) county were more likely to be recidivists compared to those in San Diego County. 2,925 (1.73%) patients died during the study period. Being a recidivist significantly ($p<0.005$) increased risk for mortality (OR 1.1).

Conclusion: Recidivism following violent assaults is rare, however, it is associated with increased mortality. Risk factors for recidivism include male gender, poor insurance, African American race and intoxication with alcohol or illicit substances suggesting this subgroup of patients may benefit most from violence intervention programs.



Poster # 55

PRE-INJURY COGNITIVE AND PHYSICAL FUNCTION OF INJURED OLDER ADULTS: DOES A BRIEF SCREENING INSTRUMENT IDENTIFY PATIENTS AT RISK?

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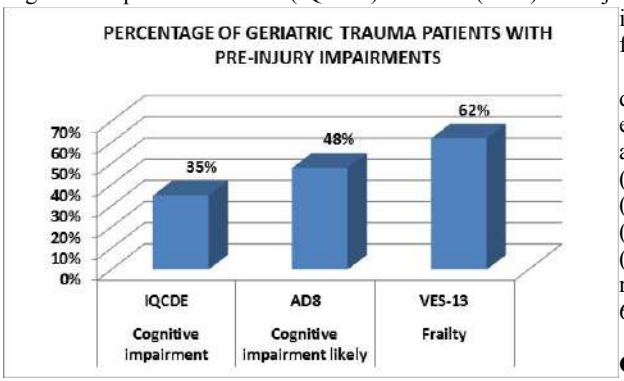
Introduction: Pre-injury cognitive and physical function impairments predict functional decline after injury in older adults. Validated admission screening tools for pre-injury impairment can establish patients' baseline and goals. Thus, the purpose of this study was to evaluate the use of validated brief screening instruments to identify pre-injury impairments in geriatric trauma patients.

Methods: Design: Prospective cohort study. Sample: Patients ≥ age 65 (targeted n = 200) admitted to a level I trauma center between October 2013 and March 2014 with a mechanism of injury and primary injury diagnosis. Instruments: *Cognition:* Informant Questionnaire on Cognitive Decline in the Elderly (IQCDE), AD8 Dementia Screening Interview. *Physical Function:* Vulnerable Elder Survey (VES-13), Paffenbarger Physical Activity Questionnaire (PPAQ). Procedure: Patients and/or surrogates were screened by trained research assistants within 48 hours of admission to determine pre-injury cognition and physical function. Data Analysis: Frequencies (%), measures of central tendency.

Results: 150 of 325 (46%) patients (with surrogates) enrolled thus far. Mean (SD) age: 77.3 (8.9); 57% female. Surrogate-administered instruments identified pre-injury cognitive impairment in 35% (IQCDE) and 48% (AD8) of subjects; the VES-13 identified pre-injury frailty in 62% of subjects. 75% (PPAQ) of patients did not engage in weekly exercise; median daily activity (hours): vigorous (0.0, IQR: 0-0), moderate (0.0, IQR: 0-4), light (4.0. IQR: 2-6), sitting (8.3, (IQR: 5-12), reclining (8.0, IQR: 6-10).



Conclusion: A significant percentage of geriatric trauma patients are admitted with pre-injury cognitive and physical function impairments. Few patients engage in exercise, with most time spent sitting or reclining. Admission screening is a recommended quality indicator. Our next steps include: 1) determining the feasibility of incorporating these screening instruments into provider workflow; and 2) utilization of pre-injury impairment measures for geriatric trauma predictive modeling.

Poster # 56

THROMBOELASTOGRAPHY DOES NOT DETECT PRE-INJURY ANTIPLATELET THERAPY IN ACUTE TRAUMA PATIENTS

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Introduction: As life expectancy in the United States continues to rise, the aging trauma population presents with a higher incidence of pre-existing medical conditions, including the use of antiplatelet agents (APA). Thromboelastography (TEG) with platelet mapping has been proposed as an assay to detect the presence of APA to trigger reversal strategies, yet no study has evaluated TEG markers of platelet dysfunction in acute trauma patients stratified by the use of pre-injury APA. We hypothesized that patients on pre-injury APA would demonstrate prolonged TEG markers of platelet dysfunction compared to those not on pre-injury APA.

Methods: This retrospective chart review evaluated all trauma patients admitted to an urban, level 1 trauma center from February 2011 to April 2013 who received a TEG within the first 24 hours of admission. Patients were classified as receiving pre-injury APA or no APA if their documented medications prior to admission included either aspirin or adenosine diphosphate (ADP) antagonists, including clopidogrel, prasugrel, ticagrelor.

Results: A total of 139 patients were included (APA, n=38; no APA n=101). The time from admission to the first TEG was similar between groups (APA 206±313 minutes vs. no APA 244±347 minutes, p=0.55). There was no significant difference in TEG markers of platelet dysfunction, including maximum amplitude (MA; APA 64.3±6.04 mm vs. no APA 60.8±10.2 mm; p=0.05), % ADP inhibition (APA 60.9±25.4% vs. no APA 60.9±25.4%; p=0.84), % arachidonic acid (AA) inhibition (APA 58.4±31.4% vs. no APA 54.4±33.8%; p=0.54). Both groups had similar proportion of severe platelet dysfunction, defined as ADP inhibition greater than 70% (APA 34% vs. no APA 32%; p=0.82) and AA inhibition greater than 70% (APA 43% vs. no APA 36%; p=0.42).

Conclusion: TEG markers of platelet dysfunction did not identify the use of pre-injury APA in acute trauma patients, which may be explained by early platelet dysfunction following major trauma. There is a continued need for an assay to detect the presence of APA to trigger reversal strategies.

Poster # 57

VALIDATION OF A BRIEF, 2 QUESTION DEPRESSION SCREEN IN TRAUMA PATIENTS: NOW WHAT'S YOUR EXCUSE?

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Introduction: Increasingly, depression following traumatic injury is being recognized as a complication of injury that can last long after the physical injury has healed. Unlike mandated screening for risky alcohol use in trauma centers, screening for psychological risks, such as depression, is not required by the American College of Surgeons Committee on Trauma (ACS-COT). Limited resources and time constraints are commonly given reasons against routine screening for psychological risks. The purpose of this study was to determine if a shorter, two item screen was as valid as the standard eight question screening instrument for depression.

Methods: This prospective longitudinal study consisted of patients admitted to a Level I trauma center. 421 patients were given the Patient Health Questionnaire-8 (PHQ-8) during initial hospitalization to assess depression. A cut off score ≥10 (possible range of 0-24) on the PHQ-8 is routinely used as diagnostic for depression. The PHQ-2, a two item screen (possible range 0-6) , is derived from the first two questions of the PHQ-8 and contains items assessing sad mood and loss of interest/pleasure over the previous two weeks. A cut off score ≥3 was considered to be a positive screen for depression. Using the PHQ-8 as the standard; sensitivity, specificity, and positive predictive values were calculated. Demographic and injury related variables (e.g., etiology of injury, injury type) were also collected.

Results: The sample was predominantly male (65%) and Caucasian (67%). The majority (85%) sustained a blunt trauma, and the primary cause of injury was motor vehicle collision (37%), with a mean Injury Severity Score of 11.6. One hundred and forty two subjects (34%) were positive for depression on the PHQ-8. When comparing the PHQ-2 to the PHQ-8 in this sample, a sensitivity of 76.1 and specificity of 92.8 were found, as well as a positive predictive value of 84.4.

Conclusions: The result of our study confirms that depression is a frequently occurring condition (34%) among individuals who sustain physical injury requiring hospital admission. Screening using the two item PHQ-2 appears to have acceptable sensitivity and specificity to identify depression in this population. The use of a two item screening questionnaire is a minimal addition to the evaluation of patients after injury. Early identification of depression allows earlier intervention and hopefully better outcomes.

Poster # 58

IMPACT OF ATLS GUIDELINES INTRODUCTION ON 24 HOURS MORTALITY IN SEVERE TRAUMA IN A BUSY ITALIAN METROPOLITAN HOSPITAL

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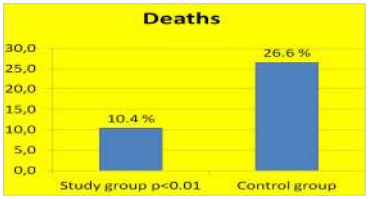
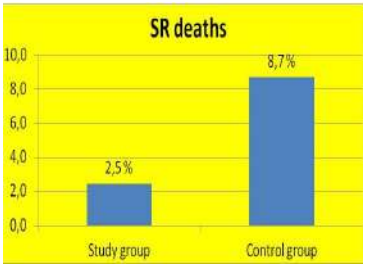
Introduction: ATLS guidelines in the initial management of trauma patients are widely accepted and implemented. Our hospital started to apply them, together with the establishment of a Trauma Team, in April 2011. Aims of this study are to evaluate changes in mortality in the first 24 hours both in the Shock Room (SR) and after admission.

Methods: This is a retrospective study based on patients admission for trauma. Study period was from 4/2011 to 12/2012 and control period was from 1/2007 to 3/2011. Patients were identified by first diagnosis (ICD 9-CM), excluding traumatic brain injuries, peripheral lesions (ie limb fractures) and burns and stratified by admission ward, considering only ICU, General Surgery and Traumatology.

Results: There were 207 patients in the control group (CG) and 163 in the study group (SG). The two groups were not different for age (mean 51,8 years old in the CG, vs 50,2 in the SG), or gender (75,4% males in the CG vs 78,8% in the SG). The died patients were not different in terms of systolic blood pressure (mean 98 mmHg in the CG, vs 103 mmHg in the SG), metabolic acidosis (mean base excess -10,1 units in the CG vs -12,9 units in the SG) or Packed Red Blood Cell consumption (mean of 8,8 units in the CG, vs 10 in the SG). Mortality was significantly better in the SG: 55 patients died in the CG, accounting for 26,6% and 17 in the SG, 10,4% (p< 0,01, OR 0.32, 95% CI 0,17-0,60).



Mortality in the Shock Room was significantly lower in the SG: 2,5% vs 8,7 (p = 0,012, OR 0.26, 95% CI 0.07-0.08).



Conclusion: The introduction of ATLS guidelines and Trauma Team had a good impact on first 24 hours mortality both in the SR and after admission.

Poster # 59

TRENDS IN THE MANAGEMENT OF PELVIC RING FRACTURES

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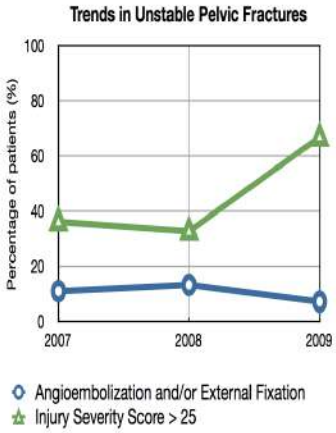
Introduction: The management of unstable and bleeding pelvic fractures involves multiple modalities including surgery, angioembolication (AE), and external fixation (EXFIX). Trends in pelvic fracture management across the U.S. are unknown. We sought to determine the use of AE and EXFIX for pelvic fractures throughout the U.S. in both trauma and non-trauma centers.

Methods: The Nationwide Emergency Department Sample from 2007-09 was queried for all patients who were admitted to the hospital with the diagnosis of a pelvic ring fractures and who had an Injury Severity Score (ISS) >15. We excluded patients who were treated at centers that never performed AE or EXFIX. All reported numbers represent weighted values. Trend analysis over time was performed using ANOVA and Poisson regression.

Results: A total of 6,416 patients met the inclusion criteria. Mean age was 46 years and 50 % were female. AE and EXFIX were mostly performed at trauma centers (98.6% of all patients), however, for patients treated at non-trauma centers, there was no significant difference in rate of AE or EXFIX (10.3% in non-trauma centers vs. 8.6% in trauma centers, p=0.57). Over the study period, the rate of AE and EXIFX decreased (10.9% in 2007 to 7.1% in 2009, p<0.001). This occurred despite an increase in the proportion of patients with an ISS>25 (36.1% in 2007 vs. 66.8% in 2009, p<0.001). Over the same time, the mean age of patients increased (40 ± 21 in 2007 vs. 58 ±21 in 2009, p<0.001). There was also substantial variability in the rate of AE or EXFIX by U.S. region (6.2% in South vs. 14.2% in Northeast, p<0.001).

Conclusion: The rate of AE and EXFIX decreased over the study period, despite increases in injury severity. This trend was associated with an increasing age. These findings suggest that trends in the management of pelvic fractures in severely injured patients are due to changes in patient demographics rather than injury severity.



Poster # 60

THE IMPACT OF A MASSIVE TRANSFUSION PROTOCOL ON TRANSFUSION RATIOS AND PATIENT OUTCOMES

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Introduction: Combat experience has demonstrated transfusion ratios of 1:1:1 (PRBC to FFP to platelet units) improves outcomes for resuscitation of massive hemorrhage. A massive transfusion protocol (MTP) was implemented to guide the resuscitation of the acutely injured in a 1 PRBC:1 FFP:1 platelet fashion. The purpose of this study is to assess the effectiveness of the MTP on transfusion ratios and outcomes

Methods: A retrospective review was conducted of all trauma admissions which received ≥ 10 units of PRBCs in the first 24 hours of hospitalization from 2004 to 2012. Standard demographic data including blood products administered in the first 24 hours, mortality, ISS, GCS, hospital length of stay, ICU length of stay, and ventilator days were collected from the trauma registry. Patients before (PRE) and after (POST) the protocol implementation in May, 2008, were compared.

Results: During the 9 year period, 447 trauma patients required massive transfusion. There was no difference in age, gender, or mechanism between the PRE and POST groups. The PRE and POST transfusion ratios are displayed in the table. In survivors, the hospital length of stay was less in POST compared to PRE (26 vs. 31 days, p= 0.04) as was the ICU length of stay (12 vs. 16 days, p=0.02). Linear regression identified the POST group as an independent predictor of decreased ventilator days after adjusting for age, GCS, and chest AIS (p<0.0001).

	PRE (n=239)	POST (n=208)	p-value
Mean PRBCs	19.72	19.76	0.9639
Mean FFP	14.28	16.17	0.0791
Mean Platelets	**2.60**	**3.43**	**0.0006**
FFP:PRBC	**0.72**	**0.80**	**0.0221**
Platelet:PRBC	**0.13**	**0.18**	**<0.0001**
Mortality	47.28%	40.87%	0.1732
ISS	**26.05**	**29.90**	**0.0021**

Conclusion: The MTP improved our blood product ratios during massive transfusions. There was no change in mortality in the POST group despite an increased ISS. Ventilator days, ICU days, and hospital LOS were all decreased post-MTP implementation.

Poster # 61

INJURY SEVERITY AND COMORBIDITIES ALONE CANNOT BE USED TO PREDICT FUTILTY OF CARE AFTER INJURY FOR ELDERLY TRAUMA PATIENTS

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When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction:Counseling families of elderly trauma patients is a difficult part of delivering these patients' care. When making decisions about the aggressiveness of interventions, surrogates ask for prognostic information which in turn is often based on the clinician's anecdotal experience rather than data. While multiple models have attempted to predict rates of mortality after injury, we undertook this study in an attempt to specifically predict futility of care in the elderly trauma patient in order to facilitate objective prognoses using easily available parameters: Injury Severity Score (ISS) and preinjury comorbidities.

Methods:Two age cohorts (70-79 years and 80 years or older) were constructed from The National Trauma Data Bank (NTDB) for the years 2007-2011. Subjects with a preexisting advanced directive limiting care or an ISS of 75 were excluded. Clinically relevant comorbid conditions were tabulated for each patient. Mortality rates at every ISS score were tabulated for subjects with 0, 1, or $\geq$2 comorbidities. Futility was defined a priori as an in-hospital mortality rate of $\geq$95% in a cell with more than five patients.

Results:A total of 570,442 subjects were identified (age 70-79 years, n=217,384; age $\geq$80 years, n=352,608). Overall mortality rates for the groups were 5.3% for ages 70-79 and 6.6% for patients aged 80 or older. No individual ISS score was found to have a mortality rate of 95% or greater for any number of comorbidities in either age cohort. The highest mortality rate seen in any cell with an adequate number of observations was for an ISS of 66 in the 80 year old cohort with no listed comorbidities (93.3%). Even at very high ISS levels mortality rates, while high, did not approach our definition of futility of care regardless of the number of pre-injury comorbidities. The table represents mortality rates at the upper extremes of ISS when aggregated into deciles (NA= $\leq$5 patients per cell).

ISS	40-49	40-49	40-49	50-59	50-59	50-59	>60	>60	>60
#comorbidities	0	1	2	0	1	2	0	1	2
70-79 yo (%mortality)	45.5	42.6	46.8	62.3	58.3	56.6	73.9	NA	NA
80 or older (%mortality)	64.8	60.3	60.9	76.1	71.9	81.4	93.3	NA	NA

Conclusion:ISS and preinjury comorbidities alone cannot be used to predict futility of care in elderly patients. Future attempts to predict futility in these age groups may benefit from incorporating a measure of physiologic distress.

Poster # 62

ALL IT'S CRACKED UP TO BE? FALLOUT OF TQIP IN HIGH VOLUME PENETRATING TRAUMA CENTERS

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Introduction: High volume urban trauma centers care for more penetrating trauma patients in whom emergency department thoracotomies (EDT) are indicated. This patient population has a very high mortality outcome. The Trauma Quality Improvement Project (TQIP) performs inter-institutional benchmarking by comparing mortality outcomes. We hypothesize that a trauma center in an urban environment with high volume penetrating trauma will have a significantly higher overall penetrating mortality outcome due to the increased rate of mortality for EDT patients.

Methods: A 5-year retrospective analysis was performed for all adult penetrating trauma patients who presented to an urban ACS-verified Level 1 trauma center between 2009-2013**.** Patient demographics, mechanism of injury, injury severity score, and mortality outcome were analyzed. Mortality outcomes were studied for all patients and compared with mortality outcomes after exclusion of EDT patients. Data was analyzed with mortality risk ratios, odds ratios, and t-tests using Stata12 software

Results: Over the 5-year period 3,544 patients met inclusion criteria. These patients were mostly young (mean age 32±12.2), African-American (2,659, 73.62%) males (3,079, 86.9%). EDT was performed for 84 (2.37%) patients, comprising almost 17 EDTs per year. The odds of death was more than 300 times more likely in EDT patients (OR=325, Chisq=733.3, 95% CI 108.0-979.7). When these 84 EDT patients were removed from the cohort, the mortality outcome was significantly decreased compared to overall penetrating traumas (mortality 7.66%±0.3 vs 9.76%±0.3, $p<0.005$).

Conclusion: At our institution, EDTs are performed frequently and significantly impact our mortality rate. These patients would likely be considered dead or unsalvageable at lower volume institutions where EDT and high volume penetrating trauma care is not often performed, therefore being excluded from their overall mortality rates. National benchmarking projects must take this into account when performing inter-institutional comparison. New benchmarking standards for high volume, high penetrating trauma centers should be considered.

Poster # 63

RECREATIONAL ACTIVITIES IN ELDERLY TRAUMA PATIENTS: IS PRE-INJURY BLEEDING TENDENCY ASSOCIATED WITH WORSE OUTCOMES?

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INTRODUCTION: With an increase in life expectancy, the aging population is participating in recreation traditionally considered activities of youth. Elderly trauma patients have many reasons for higher morbidity and mortality after trauma, one of which may be the use of anticoagulant agents. Our aim was to determine whether pre-injury bleeding tendency had an adverse influence on outcomes in geriatric patients engaging in certain "high risk" recreational activities.

METHODS: A retrospective review of the American College of Surgeons National Trauma Data Bank from 2007-2010 for patients admitted to a Level I or II trauma center, age 65 years or older, and with specific ICD-9 E-codes was performed. Activities included riding motorcycles, bicycles, snowmobiles or ATVs, and horses or other animals, as well as skiing and snowboarding. Patients with a pre-injury comorbidity of "bleeding disorder," which includes warfarin and clopidogrel but excludes aspirin use, were compared to those without. After coarsened exact matching was used to balance patient and injury covariates, a multivariate regression analysis was performed to determine differences in outcomes.

RESULTS: Two hundred sixty eight patients with a bleeding disorder were matched to 2900 without. The bleeding disorder group had increased adjusted odds of blood product transfusion $\geq$ 5 units and of deep vein thrombosis. No statistically significant differences in length of stay, pulmonary embolism, or mortality were observed.

Complications and Outcomes (Matched Data)

	No Bleeding Disorder (n = 2,900)	Bleeding Disorder (n = 268)	Odds Ratio (95% CI)	p-value
Hospital Length of Stay	4 (6)	5 (6)		0.700
$\geq$ 5 Units Transfused	2.1%	7.0%	4.7 (2.2-9.9)	< 0.001
Pulmonary Embolism	1.3%	2.2%	1.7 (0.6-5.2)	0.374
Deep Vein Thrombosis	3.8%	7.6%	2.1 (1.2-3.9)	0.018
Mortality	4.2%	5.6%	1.6 (0.8-3.2)	0.197

CONCLUSION: Elderly patients with bleeding tendencies, including use of warfarin and clopidogrel, who participate in certain recreational activities do not have increased mortality, but are more likely to have greater transfusion requirements. Increased risk of deep vein thrombosis in this group may reflect aggressive treatment for or correction of the bleeding disorder. Further studies to better characterize the risks associated with use of anticoagulant medications during some recreational activities are warranted.

Poster # 64

HEALTHCARE DISPARITIES & RISK FACTORS FOR READMISSION AFTER EMERGENCY GENERAL & VASCULAR SURGICAL PROCEDURES

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Introduction: The Affordable Care Act of 2010 mandates healthcare organizations to enact programs to reduce readmission rates. However, risk factors for readmission in emergency surgical patients have not yet been adequately studied. With this project we aim to identify factors associated with 30-day readmission days after non-elective surgery and assess if differences exist across populations at risk.

Methods: Data were obtained from the 2011-2013 Boston University Surgical Quality Improvement Program. Patients undergoing elective procedures, had a length of stay > 30 days and those that expired before any readmission were excluded. Demographic (age, gender, race), socioeconomic (profession, income, insurance status, distance from hospital), pre- (comorbidities, smoking, steroid use, body mass index, routine laboratories, wound & ASA class) and postopoperative (complications, length of stay, discharge destination) variables with $p<0.1$ on univariate analysis were included in a backward elimination logistic regression model selection, to determine the factors that independently predict readmission.

Results: After excluding 44 subjects, 441 patients underwent emergency general or vascular surgical procedure and were prospectively monitored by the program between 01/2011-11/2013. Approximately 13% were readmitted in 10.2±7.3 days after discharge, most commonly for healthcare-associated infections (38.5%), gastrointestinal (17.9%) or cardiopulmonary (12.8%) complications. African American race was independently associated with readmission [Odds Ratio 3.3 (95% C.I. 1.38–7.9), p=0.007], and so were postoperative surgical site infections [6.5 (2.2-19.3), p=0.001], highest white count [1.1 (1-1.3), p=0.007], and ASA class [2.5 (1.3-4.8), p=0.006]. Lengthier hospital stays conferred a protective effect [0.89 (0.82–0.97), p=0.007]. Key socioeconomic differences between African American and other ethnicities treated at our institution may help explain the effect of race on readmission rates and healthcare disparities.

Conclusion: Pre-operative and pre-discharge efforts to limit re-admission risk may be focused based upon socioeconomic factors. Improved access to healthcare and preexisting comorbidity control could be targeted to improve readmission rates after general surgical procedures.

	African American	**Non-African American**	**p-value**
N=	174 (39.7%)	266 (60.3%)	
Median income (by residence zipcode) ($1,000)	48.4 ± 17.1	59.3 ± 22.6	**<0.001**
# of comorbidities	1.2 ± 1	0.9 ± 1	**0.037**
Preop Creatinine (mg/dL)	1.3 ± 1.3	0.9 ± 0.6	**<0.001**
ASA class	2.5 ± 0.8	2.3 ± 0.8	**0.045**
Discharge to facility	20.6	15.3	0.159
Readmission	17.6	10.5	0.075

Poster # 65

OBSERVATION IS AN INADEQUATE APPROACH FOR INJURED EXTREMITIES WITH SINGLE TIBIAL VESSEL RUN-OFF

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Introduction: Trauma patients with sudden loss of distal perfusion due to tibial injuries are frequently not offered vascular interventions if a single vessel retains patency. Given that acutely injured patients have minimal preexisting collateral circulation, we hypothesized that sudden loss of tibial vasculature would result in increased non-operative failure and higher amputation rates.

Methods: Traumatically injured patients with CT-Angiogram (CTA) confirmed tibial (AT), posterior tibial (PT), or peroneal artery injury were included. Demographics, injury severity score (ISS), mechanism of injury, distal vessel patency, management approach (observation, procedural), and 30 day limb salvage outcome were recorded. Failed observation was defined as requiring a revascularization procedure or a primary/secondary amputation after attempting conservative vascular management. Statistical analysis was completed using descriptive statistics, chi-squared testing, and univariate analysis.

Results: From 2009 to 2012, 437 patients were admitted with arterial extremity injury of which 234 (53%) were lower extremity. From this group, 84 (36%) patients were identified with (CTA) confirmed limited or no flow in the tibial (AT, PT, or peroneal) arteries. From the 84 patient cohort, acute intervention was performed in 57% (48) and non-operative observation was instituted in 43% (36). In the acute intervention group, bypass/interposition placement was performed in 66% (32), stenting 6% (3), primary repair 15% (7), embolization/ligation 9% (4), primary amputation 4% (2). The secondary amputation rate in the acute intervention arm was 12% (6) with a mean time to amputation of 5.8 days (range 1-16). Initial observation (n = 36) failed in 16% (6). 83% (5) underwent a revascularization procedure (mean time to revascularization 5.6 days [range 1-15]). One patient from this revascularization sub-group underwent a secondary amputation (necrotic toes) on day 19 post injury. One patient from the failed observation cohort underwent a primary amputation (below knee amputation) on day 8 post injury. 44% with 0 or 1 tibial vessel failed observation while only 8% failed if they had 2 or 3 patent vessels. Patients with 0 or 1 patent tibial vessels were significantly more likely to fail initial observation compared to those with 2 or 3 patent vessels ($p<0.05$). Patency of the tibial vessels impacted limb salvage rates of the overall cohort ($P<0.001$). The number of open tibial vessels was associated with limb salvage, with 2.7 open tibial vessels in the limb salvage group compared to 1.1 in the amputation group ($P<0.05$).

Conclusion: Patients who failed an initial trial of observation were significantly more likely to have only 0 or 1 tibial vessels patent. The number of open tibial vessels is significantly associated with limb salvage. These data suggest that the common practice of avoiding revascularization in acutely injured trauma patients with single tibial run-off may contribute to early limb loss.

Poster # 66

Early fixation for mid-shaft femur fractures: variation in practice across TQIP centers

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Introduction: Early definitive stabilization of mid-shaft femur fractures has been associated with decreased rates of complications, shorter length of stay, and lower costs when compared to delayed fixation. Deviations from optimal practices with delayed fixation are often attributed to patient or injury factors. We postulated that institutional factors might also be responsible for delays to definitive fixation. To test this hypothesis, we evaluated the extent of variation in the rates of delayed fracture fixation across trauma centers (TC) participating in the American College of Surgeons Trauma Quality Improvement Program (TQIP).

Methods: Data were derived from the TQIP analytic dataset (01/2010-03/2013). Adults with mid-shaft femur fractures who underwent definitive internal fixation (ORIF) were included. Delayed fracture fixation was defined as $\geq$ 24 hours from ED arrival. The crude rate of delayed fracture fixation was calculated at each TC. To adjust for case-mix, hierarchical logistic regression modeling was used to estimate the TC-specific odds of delayed fracture fixation. Patient, injury and TC characteristics were included in the model. To quantify the extent of variability present across TCs, we calculated the median odds ratio (MOR). In addition, we quantified the proportion of the total variance explained by patient factors as well as the variance explained by TC factors alone.

Results: We identified 3,342 patients over 93 TC's meeting inclusion criteria. The median time to fixation was 16 hours (IQR 7 – 27 hours) and 28% of patients underwent late (>=24 hours) fixation. There was marked variation in the rates of delayed fixation across TCs (median 29%, IQR 20 - 36%). Six TCs were identified as having a significantly lower than expected rate of delayed fixation and seven centers had higher rates of delayed fixation given their respective case mixes. The MOR for delayed fracture fixation across TCs was 1.95; suggesting that the odds of delayed fixation were 1.95-fold greater if the same patient was admitted to a randomly selected TC as opposed to another. After multivariate multilevel analysis, patients who were 65 years old or older, had history of ischemic heart disease or hypertension, sustained a fall-related injury or had significant injury (AIS $\geq$3) in the head, chest, or abdomen were more likely to undergo delayed fixation. At the hospital level, TC designation level, teaching status, number of orthopedic surgeons per hospital, and volume of patients were not independently associated with timing of fixation. Our hierarchical model, which included both patient and TC factors, explained 46.3% of the variability in the rates of delayed fracture fixation across TCs. Patient factors accounted for 37.4% while TC factors accounted for 8.9% of the explained variation. In other words, TC factors were responsible for 20% of the explained variation in our model.

Conclusions: Differences in rates of delayed mid-shaft femur fracture fixation were observed across TQIP centers. Institutional factors were in part, driving these differences. Specific causal factors including resource availability, critical care practices, or orthopedic commitment, among others, need to be further evaluated.

Poster # 67

ANTIBIOTICS AND OPEN FRACTURES OF THE LOWER EXTREMITY: LESS IS MORE

Michael Truitt* MD, Brittany Bankhead-Kendall MD, Jason Murry MD, Timothy Gutierrez MD, Matthew Weldon MD, Danny Holland MD, Methodist Hospital of Dallas

Introduction: Historically, orthopedic guidelines have recommended Grade II/III open fractures receive a first generation cephalosporin and an aminoglycoside. The use of aminoglycosides in the trauma patient has been criticized for nephrotoxicity with questionable utility. The literature is limited in comparing the outcomes of patients treated with a cephalosporin alone (Group 1) vs a cephalosporin + aminoglycoside (Group 2) for open fractures. At our trauma center we have a unique trauma service where half of our surgeons treat open fractures with a cephalosporin alone and half use a cephalosporin + aminoglycoside. We hypothesized that our rates of infection and need for secondary intervention were the same between the two groups.

Methods: We identified all Grade II/III open fractures of the lower extremity admitted to the trauma service over a 5 year period. Charts were retrospectively reviewed to identify demographic information, injury severity score (ISS), type of antibiotic administered, fracture location, grade of fracture, comorbidities, incidence of acute kidney injury (AKI), wound infection, and hardware removal.

Results: From January 1, 2008 to December 31, 2013 there were 126 grade II/III open fractures of the lower extremity admitted to the trauma service. There were 65 (52%) patients in Group 1 and 61 (48%) in Group 2. Demographics, ISS, fracture grade/location and comorbidities were not different between the two groups. Patients in Group 1 had a 4% incidence of AKI, while the incidence was 10% of patients in Group 2 ($p<.05$). Group 1 had a 6% risk of wound infection, compared to 5% in Group 2. One patient in Group 1 (1.5%) and two patients in Group 2 (3.3%) required hardware removal secondary to infection.

Conclusion: The addition of an aminoglycoside to antibiotic prophylaxis in open lower extremity fractures was associated with a significant increase in AKI with no change in the incidence of wound infection or hardware removal. Cephalosporins alone may be sufficient for prophylaxis in Grade II/III open fractures of the lower extremity. A large scale prospective randomized trial is needed to confirm these findings and inform clinical practice.

Poster # 68

A STATEWIDE, POPULATION-BASED STUDY OF IN-HOSPITAL DEATH AFTER TRAUMATIC BRAIN INJURY: HAS OUTCOME IMPROVED OVER 14 YEARS?

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Introduction:Traumatic brain injury (TBI) accounts for the largest proportion of injury-related disability and death in the United States. Disparities in neurotrauma outcomes exist between regions, age groups and races. The purpose of this study was to analyze patient outcomes from a statewide database and evaluate mortality patterns over the 14 year period of interest.

Methods:Patients, age 16 and older, with TBI (ICD-9 codes 800 to 801, 803 to 804, 850 to 854, and 959.01) were selected from a validated, statewide database of all TBI admissions at nonfederal hospitals in South Carolina, 1998 – 2011. Observation was censored at discharge. Elixhauser comorbidities were determined for each patient based on ICD-9 codes. Injury Severity Score (ISS) and In-Hospital Mortality were determined. Cox regression was performed to examine the risk of in-hospital death. Kaplan-Meier survival curves compared survival probabilities across hospitalization based on admission year.

Results:We identified 42,842 patients with TBI with a mortality rate of 8%. Sixty four percent of patients were male and the median length of stay was 4 days (IQR 2 – 9). Patients who died had higher age (57.8+/- 23.9 vs. 49.3+/- 22.9 years, $p < 0.001$), AIS-H (4.24+/- 0.90 vs. 3.05 +/- 0.95, $p < 0.001$), ISS (22.6+/- 10.3 vs. 13.9+/- 7.8, $p < 0.001$). Relative risk of death increased as severity of TBI increased based on AIS-H =3 [HR 1.651, $p < 0.001$] and AIS-H = 4-6 [HR 3.478, $p < 0.001$] compared to patients with AIS-H =1-2. Adjusted hazard ratios for heart disease (1.254), liver/digestive disorders (1.463), renal disease (1.768), coagulopathy (2.181), and stroke (2.135) were associated with mortality (each with $p < 0.05$). Adjusted hazard ratios for year treated decreased progressively through the study period compared to the index group 1998 – 2000 [2001 – 2003 (0.878), 2004 – 2006 (0.757), 2007 – 2009 (0.668), 2010 – 2011 (0.605)] (figure, $p < 0.05$). There was no difference in outcome between white and black patients.

Conclusion:In-hospital mortality for TBI has progressively improved over the last 14 year period in South Carolina. We could not detect a difference in outcome between white and black patients. Outcomes for geriatric patients especially those with significant co-morbidities lagged and deserve additional attention given the aging of the population. Additional research is needed to elucidate the reasons behind these outcome patterns including the question of why disparities in care exist and how to close the gap for older patients.

Poster # 69

SHOULD TRAUMA SURGEONS PERFORM LIMB REVASCULARIZATION?

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Introduction:Revascularization after extremity vascular injury has long been considered an important skill among trauma surgeons. Increasingly, some trauma surgeons defer vascular repair to vascular surgeons, in response to training or local practice patterns. This study was designed to document results of extremity revascularization surgery when performed by trauma and vascular surgeons.
Methods:The trauma registry of an urban level I trauma center was used to identify all patients from 2003-2013 who underwent an early (<24h) procedure for urgent management of acute injury to axillary, brachial, radial, ulnar, femoral, popliteal, or calf arteries. Patients were managed by trauma (TRA) vs. vascular surgeons (VAS) based on the practice pattern of the on-call trauma surgeon. Injury and outcome variables were recorded, including successful revascularization, postoperative debridement, compartment syndrome, bleeding, thrombosis, and amputation. Patients were excluded if they died within 24 hours of hospitalization, had arterial ligation or solely venous injuries, or were treated by both trauma and vascular surgery.
Results: Of 115 patients, 84 patients were revascularized by trauma surgery and 31 by vascular surgery. Three endovascular surgeries were performed, all by vascular surgery. Complications are displayed in Table 1. There was no difference in type of complication or overall rate.
Conclusion: In appropriately selected patients, trauma surgeons achieve good outcomes after revascularization of injured extremities. Bypass grafting is associated with more complications, presumably due to more complex injury. Open repair remains the mainstay of extremity vascular injury management.

	All patients	**TRA (%)**	**VAS (%)**	***p***
Total	115	84	31	
Complications	19 (17)	11 (13)	8 (26)	0.2
Compartment Syndrome	3 (3)	2 (2)	1 (3)	1
Thrombosis	5 (4)	2 (2)	3 (10)	0.6
Debridement of tissue	5 (4)	3 (4)	2 (7)	1
Postoperative bleeding	3 (3)	2 (2)	1 (3)	1
Wound infection	2 (2)	2 (2)	0	0.5
Amputation	1 (1)	0	1 (3)	0.4

Table 1. Type of complication by service line. TRA- trauma surgery; VAS- vascular surgery.

Poster # 70

INTRAVASCULAR ULTRASOUND ENHANCED AORTIC SIZING FOR ENDOVASCULAR TREATMENT OF BLUNT AORTIC INJURY

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Introduction:Blunt aortic injury (BAI) often occurs in young patients with compliant aorta and resulting hyperdynamic physiology thereby creating potential for significant variation in aortic diameter during the cardiac cycle. Intravascular ultrasound (IVUS) may hold promise as a modality to detect real time variations in aortic diameter for more reliable sizing in patients undergoing endovascular repair (EV) of BAI.
Methods:Retrospective review of a single institution Level 1 trauma registry was performed from January 2004 to January 2014 identifying patients who underwent EV of BAI. Patients were divided into those who underwent EV after CT angiography (CTA) alone (CT Group) and those who had IVUS performed in addition to CTA (IVUS group). Direct comparison between groups using standard statistical methods was performed regarding pre-deployment aortic measurement, size of device implanted, landing zone, and repair outcomes.
Results:In the ten year period, a total of 38 patients underwent EV of BAI. There were 28 patients in the CT group and 10 in the IVUS group. Left subclavian artery (SCA) coverage was performed in 50% (CT group) and 40% (IVUS group) of cases. Mean aortic diameter based on CTA was similar (20.7mm, CT vs. 18.9mm, IVUS) in both groups. In the CT group, average proximal diameter of proximal device implanted was 25.9mm resulting in 20.1% oversizing. With the addition of IVUS, measured maximum aortic diameter was increased by average of 12.4% ($p<0.05$) for the whole group and 21.0% ($p<0.05$) in patients undergoing left SCA coverage resulting in change of device diameter implanted in 40% of patients. The average resulting oversizing of device implanted in the IVUS group was 30% with CTA measurement and 18% with IVUS in the IVUS group. Technical success for of repair for both groups was 100% with no secondary interventions required in either group.
Conclusion:EV repair of BAI is safe with excellent results when performed with CTA alone or with additional IVUS without need for excessive oversizing. However, CTA appears to undersize aortic diameter in reference to IVUS which is most prominent proximal to the left SCA possibly due to impulse variation of aortic diameter. This undersizing may predispose patients to long-term repair failure and continued evaluation of both measurement modalities is necessary.

Poster #71

WITHDRAWN

Poster # 72

VALIDATION OF THE QUALITY OF ULTRASOUND IMAGING AND COMPETENCY (QUICk) SCORE FOR THE FOCUSED ASSESSMENT WITH SONOGRAPHY FOR TRAUMA (FAST) EXAM

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Introduction:

Despite the recent push for medical training to enter an era of "competency-based" assessments, little evidence guides the current credentialing standards regarding point of care ultrasound using the Focused Assessment with Sonography for Trauma (FAST) exam. To date, no tool has been validated for evaluating the quality of image acquisition when performing a FAST examination; we propose to develop and validate such a tool.

Methods:

Two scoring systems were developed by a modified Delphi technique. A nine domain Global Rating Scale (GRS) rated the quality of performance of ultrasound techniques on a five point Likert scale, and a twenty-four point Task Specific Checklist (TSC) served as a binary measure of the successful versus non-successful imaging of important anatomic landmarks. Two cohorts of novice (n=12) and expert (n=12) sonographers were recruited to watch an instructional video and perform a FAST examination on a live volunteer for evaluation by the proposed scoring systems. "Novices" were resident physicians with no formal FAST training while "experts" were staff physicians with credentialed FAST training in accordance with Canadian Emergency Medicine guidelines for independent scanning skill. Performances were scored by two additional experts blinded to participant identities and the scores between cohorts were compared to assess the validity of the scoring metric.

Results:

Experts scored significantly better than novices on the TSC (mean 17.21 vs 11.08, $p<0.01$) with an inter-rater agreement 0.7951. Experts also scored significantly better than novices on all tested domains of the GRS tool, including the mean total GRS scores (mean 29.79 vs. 18.42, $p<0.01$) with an inter-rater agreement of 0.6066. Scoring tools were modeled with univariate logistic regression, and areas under the receiver operating curves (AUROCs) of 0.8988 for the TSC and 0.9762 for the GRS indicated excellent discriminatory power.

Conclusion:

We have successfully developed a quantitative model of FAST exam quality assessment which is able to discriminate with high power between novice and expert sonographers. Such a tool may be useful in helping to define training standards and guiding future FAST research, and also serves as a template for future competency-based ultrasound imaging research.

Poster #73

WITHDRAWN

Poster # 74

SURGEMAN, TRAUMAMAN AND PORCINE MODELS FOR THE SURGICAL SKILLS STATION IN THE ATLS(R) COURSE

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Introduction: Universities and Hospitals are requiring the use of suitable alternatives to animals for training wherever possible. The cost of the currently approved artificial mannequins often effectively makes their use prohibitive in low income countries. A low cost Brazilian artificial mannequin (SurgeMan) has been developed. Our primary objective was to determine if SurgeMan would have equivalent learner and instructor satisfaction scores compared with the currently approved TraumaMan and animal model for the surgical procedures of ATLS ®. Our secondary objective was to determine if user satisfaction scores for Surgeman exceeded 80%.

Methods: Prospective observational cohort study with three models. SurgeMan, TraumaMan and an animal model (Landrace pigs) were used. A convenience sample of 36 students enrolled in ATLS ® courses were divided into 9 groups and were monitored by 1 instructor per group throughout the skills station . Each group participated in all skills in each of the three models. The procedures performed were: Tube Thoracostomy, cricothyroidotomy, pericardiocentesis and diagnostic peritoneal lavage(DPL). Psychometric testing was completed by having students and instructors fill out a Likert scale at the completion of each activity.

Results: Animals and Trauma Man performed better than SurgeMan for all skills except pericardiocentesis, where there was no difference in the models. When no ethical or financial factors were taken in consideration: 58% of the students chosen pigs as their preferred model(p=0.057). When all ethical factors were considered all models were equally recommended p=1.00. For the adequacy of each model for learning ATLS skills, students thought all models were adequate. (81% S.Man;94%T.Man;86% Pigs;p=0.184)

	Thoracostomy	Cricothyroidotomy	DPL
Trauma Man	4.09 (0.66)*	3.9 (0.87)*	3.92 (0.64)#
SurgeMan	3.03 (0.89)	3.00 (1.03)	2.94 (0.86)
Animals	4.10 (0.91)*	3.37 (1.26)	4.39 (0.93)*

*p<0.05, significantly better than all comparator groups.
#p<0.05, significantly better than SurgeMan

Conclusion: TraumaMan performed better than SurgeMan in most procedures. Students found that both TraumaMan and SurgeMan are acceptable for learning ATLS ®surgical skills.

Poster # 75

TRAINING FUTURE TRAUMA SURGEONS; EFFICACY OF THE US DESIGNED ADVANCED SURGICAL SKILLS FOR EXPOSURE IN TRAUMA (ASSET) COURSE FOR UNITED KINGDOM TRAINEES

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When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction: To assess the efficacy and desirability of conducting the ASSET course for United Kingdom (UK) general surgical trainees.

Methods: Prior to the course participants were presented with the course manual, a DVD depicting the surgical exposures, and a pre-course self-efficacy evaluation (pre-score); a measure of confidence in performing the skills with a range of scores from 1 to 5, with 1 being little confidence and 5 extreme confidence.

Fresh frozen human cadavers were used with one trained ASSET faculty per table and four participants. The faculty presented topics that were delivered by PowerPoint presentations and videos. The presentation topics were in five segments: upper extremity, lower extremity, neck and chest, abdomen, and pelvis. After each segment, the participants completed the surgical exposure in the cadaver under supervision. On completion, mean pre-scores were compared with the post-scores scores using the Mann-Whitney U-test.

Results: 27 UK higher surgical trainees (HSTs) were trained on 2 separate courses. HST level of experience ranged from 1-6 years (median 3). The overall mean efficacy scores were 2.26 ±1.34 pre-course versus 3.86 ±0.95 post-course ($p<0.0001$). Across the 5 anatomical areas taught, pre-scores ranged from 2.07 ±1.11 (chest) to 2.37 ±1.14 (lower extremity), with post scores of 3.51 ±0.95 (neck) to 4.07 ±0.67 (pelvis and abdomen). The area of greatest benefit was chest, pre-1.99 ±1.11 to post- 3.96 ±0.78. However all areas demonstrated significant improvement in efficacy scores ($p=<0.0001$).

Conclusion: Within this cohort of UK HSTs the ASSET course produced significant improvements in objective measures of self-confidence with respect to surgical exposure for trauma. In particular, teaching directed at anatomical areas less commonly encountered during routine general surgical practice in the UK yielded the greatest benefits. The ASSET course appears relevant to current UK surgical trainees of all levels.

Poster # 76

ALL FINGERS POINT TO NO DIGITAL RECTAL EXAM IN TRAUMA PATIENTS

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INTRODUCTION: Digital rectal exams (DRE) are routinely used on trauma patients during the secondary survey as recommended by current advanced trauma life support (ATLS) protocols. Gross blood, decreased or absent sphincter tone, and a high-riding prostate are a few findings we search for during the DRE. [1] Recent literature has called the blanket use of the DRE in trauma into question. The purpose of this study is to evaluate the efficacy of the DRE as a diagnostic tool in the setting of urethral, spinal cord, small bowel, colon, and rectal traumatic injuries. **METHODS:** A retrospective review of trauma cases at a Level I trauma center from 2008 to 2012 was performed utilizing ICD-9 coding for small bowel, colon, rectal, urethral, and spinal cord injuries Inclusion criteria included all trauma patients with the above injuries, age of 18 years or older, and a noted DRE. Exclusion Criteria included an age less than 18, patients who received paralytics during intubation, a GCS of 3, and previous history that would make a DRE unreliable (a history of paraplegia, or quadriplegia). The DRE findings were compared to the final diagnosis for each patient. Sensitivity, specificity, and positive and negative predictive value were determined. **RESULTS:** A total of 111 cases (83% male, 17% female) were retrospectively reviewed ranging in age from 18 to 90 years with a mean GCS of 13.7. Ninety-two (82.9%) patients were found to have documented injuries. The majority of cases were level I trauma activations (60.4%) with level II and III (36.9%; 2.7%) following in decreasing frequency. Sixty-two (55.9%) cases were penetrating with the remaining 49 (44.1%) blunt injuries. Seven urethral (6.3%), 24(21.6%) spinal cord, 29(26.1%) small bowel, 19(17.1%) colon, and 3(2.7%) rectal, 4(3.6%) bladder, 5(4.5%) pelvic, and 20(18.0%) intracranial injuries were noted. The DRE missed (false-negative rates) 100% of urethral, 91.7% of spinal cord, 93.1% of small bowel, 100% of colon, and 66.7% of rectal injuries. For injuries confirmed with radiologic modalities, the DRE missed 93.3%. For injuries confirmed on exploratory laparotomy, the DRE missed 94.9%. Positive predictive value (PPV) was poor for urethral (0.0), spinal cord (33.3%), colon (0.0), and rectal (50.0%) injuries. Small bowel injuries demonstrated a PPV of 100% but only a 6.9% sensitivity. For 75 injuries documented by radiology, only 5 had positive findings on DRE (PPV 71.4%, 6.7% sensitivity, 2 false positives). For the 39 injuries confirmed by exploratory laparotomy, only 2 cases had positive findings on DRE (PPV 100%, 5.1% sensitivity, 0 false positives). **CONCLUSION:** From this data we can conclude the DRE does not provide adequate clinical data to warrant its blanket use in the trauma setting. The DRE has poor sensitivity for the diagnosis of urethral, spinal cord, small bowel, and large bowel injury. The DRE was most sensitive in the setting of rectal injuries. When compared to other confirmatory modalities for injury (radiology or surgery), the DRE offers no benefit or predictive value as a diagnostic tool in the setting of traumatic injury. Elimination of the DRE from the secondary survey is a safe option, which will minimize risk to both patients and the trauma team.

REFERENCES

1. American College of Surgeons Committee on Trauma Initial Assessment and Management. Advanced Trauma Life Support for Doctors. 7th ed. Chicago, IL: American College of Surgeons; 2004:18–19.

Poster # 77

An integrated trauma and critical care simulation curriculum for surgical residents

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Introduction: Expectations continue to rise for residency programs to provide integrated simulation training to address clinical competence. How to implement such training sustainably remains a challenge. We developed a compact module for first-year surgery residents integrating theory with practice in high-fidelity trauma and critical care simulations.

Methods: The three-day module features a combination of simulated patient encounters using standardized patients and electronic manikins, didactic sessions, and hands-on training. Manikin-based scenarios developed in-house were used to teach trauma and critical care management concepts and skills. Separate scenarios in collaboration with the regional organ donation program addressed communication in difficult situations such as brain death. Didactic material based on contemporary evidence, as well as skills stations were developed to complement the scenarios. Residents were surveyed before and after training on their confidence in meeting the fourteen learning objectives of the curriculum on a 5-point Likert scale.

Results: Data collected from eleven residents that have completed this training shows an overall improvement in confidence across all learning objectives defined for the module, with median score pre to post-training improving significantly from 3 to 4 (out of 5), $p<0.001$. Greatest improvement was confidence in ability to communicate in ethically challenging or end-of-life situations with a mean score increase of 1.5, followed by confidence in skills of mechanical ventilation, as well as confidence in skills of pulmonary artery catheter placement with mean score increases of 1.4.

Conclusions: We successfully implemented a multimodal simulation-based curriculum that provides skills training integrated with the clinical context of managing trauma and critical care patients, simultaneously addressing a range of clinical competencies. Results to date show consistent improvement in residents' confidence in meeting learning objectives. Development of the curriculum continues for sustainability, as well as measures to embed objective evaluations of resident competence.

Poster # 78

THE ATLS PARTNERSHIP MODEL: AN INNOVATIVE STRATEGY TO EXPAND ATLS TEACHING IN LOW RESOURCES AND UNDERSERVED AREAS

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Introduction: Despite the worldwide reach of ATLS courses since 1980, many areas of the world with high rates of trauma remain untouched The partnership for region 14 (Latin-America) ACS-COT started in October of 2013, a model designed to expand ATLS to countries with limited resources. It consists in the assistance and supervision from countries with mature ATLS programs to countries without ATLS and low resources. The objective of this study is to determine, the components, challenges and impact encountered in planning and executing this model. Methods: We review the process, number of courses and financial information from the three countries where the partnership program started in the Region 14: Belize, Curacao, and Cuba. These results were compared to those from the last two countries, which promulgate ATLS in our Region: Uruguay and Paraguay. Results: Several partnership programs were planned in the Region: Antigua, Belize, Curacao, and Cuba. We held a demonstration course in Belize (December 2013), and two inaugural courses: Curacao (January 2014), and Cuba (February 2014). The time elapsed from the site visit to the inaugural ATLS course, the number of courses/participants in each course, the human resources and expenses, was determined for each country. Table. The partnership model required less time than the promulgation model. The expenses were significantly reduced (in average $60.000 USD) by the financial and time contribution and donations from members of the region, members of other regions, the Brazilian COT, and the ACS-COT compared to the traditional estimated expenses of $118.300- 133.200 USD for the promulgation model.

Country/ Partner	Model	Local Surgical Society	Time to Inaugural Course	Number of ATLS Courses (n) Participants (n)	Local Expenses/ACS and Region 14 contributions (USD)
Uruguay Paraguay	Promulgation		Uruguay/ Paraguay 15 months	2009- 2013 Provider courses: 9/8 Instructor course: 1/0	Total Estimated expenses 118.300- 133.200
Belize/ Costa Rica 2013	Demonstration/Partnership December 01/13	Medical and Dental Association J.Hidalgo P. Arriaga	24 months	Provider course: 1 Participants: 13	20.202/11.676
Curacao/ Colombia 2014	Partnership January Student: 22/01/14 Instructor: 25-26/01/14	St Elizabeth Hospital Michel Berry Sandra Cova,	4 months	Provider course: 1 Participants: 17 Instructor course: 1 Participants: 9	41.140/200
Cuba/ Brazil 2014	Partnership Student: 18-9/02/14 Instructor: 20/02/2014	Sociedad Cubana de Cirugía Martha Larrea	1month	Provider course: 1 Participants: 17 Instructor course: 1 Participants: 9	750/39.328

Conclusion: Three new countries have joined the region 14. We have created a new future - one that would not have occurred without the introduction of the partnership model. This model subsidizes the cost of bringing ATLS to areas that otherwise could not afford it. It could not succeed without the support and the commitment from faculty members of the COT from many regions, the local societies, other region societies, and the ACS-COT.

Poster # 79

THE IMPORTANCE OF TAILORING PHYSICIANS' TRAUMA CARE TRAINING NEEDS IN RURAL ENVIRONMENTS

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Introduction:Trauma training courses for physicians significantly improve technical and non-technical (judgement, management, prioritization) skills, yet gaps in the provision of care exist. These gaps are most evident in the initial evaluation and management of patients first cared for in the most rural settings. The purpose of this work was to explore what unmet educational needs might exist so as to improve the care of patients prior to transfer.

Methods:We designed a survey to evaluate the educational experiences, needs, and preferences of MDs working in emergency departments (EDs) at least 20 miles from a trauma center (TC) level I/II. The survey (n=2563) was disseminated through professional associations and potential respondents were sampled by type of training (specialty certification in Emergency Medicine – EM vs other) and actual practice location (urban vs rural). Responses were stratified by rural/urban (self identified) status of providers.

Results:There were 466 respondents, 372 of whom self identified as rural. Rural MDs had less experience caring for trauma patients, greater exposure to ATLS and were more likely to state that ATLS met their training needs (Table). The educational content most needed was similar for both groups of MDs: pediatric trauma, orthopaedic/peripheral vascular trauma, and airway management were considered priorities. Preferred modes of educational delivery differed across groups: urban MDs preferred didactic and self-learning, while rural MDs preferred case-based discussions and distance education via video-conference. Both felt simulation-based training was the preferred method of learning.

	Urban (%)	Rural (%)	*p*-value
EM certified	64	23	*<0.001*
Annual experience (number of patients)			*<0.001*
< 5	22	51	
5-10	30	27	
>10	48	22	
ATLS meets educational needs	68	82	*.005*
Recent ATLS certification	58	70	*.031*

Conclusion:While ATLS is perceived as important for most MDs in ED's remote from TC, human patient simulators are highly preferred. The preferred learning modalities differed significantly, indicating a requirement to tailor educational experiences to local needs. These findings should allow for improvements in trauma educational programs to prepare physicians working in more rural environments.

Poster # 80

TRAUMA CENTERS AND THEIR INVISIBLE ARCHITECTURE: A WAKE UP CALL FOR SURGICAL LEADERS

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Introduction: Organizational culture (OC), or *invisible architecture*, is defined as a system of shared values held by organizational members that are unique to each organization. OC has been studied extensively in the world of business and has been the subject of increasing attention in the healthcare setting. It is well known that leadership plays a very important role in establishing the culture of an institution. Furthermore, it is important that leadership and employees of all levels perceive culture similarly ensuring that everyone is "on the same page." We postulate that perception of a healthy culture within a trauma center (TC) varies by position and sought to explore this hypothesis using the previously reported Trauma Center Culture Survey (TRACCS).

Methods: We conducted a cross-sectional survey of organizational culture across centers participating in the ACS Trauma Quality Improvement Program (TQIP). TRACCS was administered to members with a variety of roles within each center. We explored differences in responses between those holding leadership positions and those without, as well as responses across roles defined as administrative, physician, or nursing. Overall scores and scores by domains (opportunity, pride and diversity; TC leadership; and employee respect and recognition) are reported. Descriptive statistics are reported as mean (SE).

Results: Responses were obtained from 1,912 in 144 centers. Leaders reported significantly higher total TRACCS score compared to non-leaders (Table 1). The lowest domain scores and largest differences across roles were evident in TC employee respect and recognition. There were significant differences across administrative, nursing and physician roles in total and across all domains.

Conclusion: Within an organization, leaders consistently perceived a healthier OC than non-leaders, particularly in the domain of employee respect and recognition. These differences may not be apparent to TC leadership and can serve as barriers to achieving optimal clinical outcomes. In conclusion, the differences found in perception across domains might have significant impact on organizational performance and mandate further study.

Table 1. Total TRACCS and Factor Scores

	Leadership	Non-Leadership	Leadership vs Non-Leadership	Administration	Physicians	Nurses	Admin vs Physicians	Admin vs Nurses	Physicians vs Nurses
Total TRACCS Score	4.23(0.05)	3.82(0.03)	0.41(0.05)	4.38(0.08)	3.99(0.04)	3.85(0.03)	0.39(0.08)	0.53(0.08)	0.14(0.04)
Factor 1 Opportunity, Pride & Diversity	4.36(0.05)	4.00(0.03)	0.36(0.05)	4.50(0.08)	4.18(0.04)	4.01(0.03)	0.33(0.08)	0.49(0.08)	0.17(0.04)
Factor 2 TC Leadership	4.50(0.07)	4.09(0.04)	0.41(0.06)	4.47(0.11)	4.35(0.06)	4.11(0.05)	0.12(0.11)*	0.36(0.10)	0.24(0.05)
Factor 3 Employee Respect and Recognition	3.78(0.07)	3.22(0.05)	0.56(0.07)	4.02(0.11)	3.49(0.06)	3.26(0.05)	0.54(0.11)	0.76(0.10)	0.23(0.05)

Values are reported as Mean (SE). All groups statistically significant, $p \leq 0.0001$, except*

Poster # 81

PEDIATRIC SEVERE TRAUMATIC BRAIN INURY: HYPO OR HYPERCOAGULABLE STATE?

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Introduction: Coagulopathy after severe traumatic brain injury (sTBI) occurs frequently and is independently associated with worse outcome. Reduced clot strength measured by TEG with platelet mapping (TEG-PM), has been associated with increased mortality in adults. No studies on the frequency and outcomes associated with clot strength abnormalities have been reported in children with sTBI.

Methods: Prospective observational study in children <18 years of age admitted with sTBI (Glasgow Coma Scale score (GCS) ≤ 8) from June 2012 to January 2014. Data collected included; injury severity score (ISS), standard coagulation tests (prothombin time [PT], INR, activated partial thromboplastin time [aPTT], platelet count). TEG-PM was measured daily for 5 days. Outcomes measured were mortality, length of ICU stay, and pediatric functional independence measure [WeeFIM scale]). We defined coagulopathy by standard coagulation tests as platelet count <100,000 per cumm, INR >1.2 or aPTT >36 seconds and by TEG-PM as reaction time (R) >12 minutes, K time >3 minutes or G <6. Data are described as median (interquartile range).

Results: We enrolled 16 children with sTBI with median age of 13.5 years (10.2-16.7). Seventy five percent were male and 37% of patients had isolated sTBI. Median admission GCS was 5 (3-6) and median ISS was 29 (17-41). Coagulopathy after sTBI based on standard coagulation parameters occurred in 62.5% patients in the first 24 hours after injury. However, only one patient had evidence of hypocoagulability on TEG-PM. Instead, 87.5% patients had hypercoagulability (R < 5 minutes). For simultaneously sampled INR and TEG samples, 58% of INR results > 1.2 also had a TEG R-time of < 5 min. Moderate platelet inhibition was found on day 1 with median platelet inhibition on adenosine diphosphate receptor of 54% (24-65%). TEG-PM parameters trended towards normal on serial evaluation over 5 days. One patient died, 50% patients had moderate (WeeFIM 71-84) and 37.5% patients had good functional independence measure (WeeFIM >85) at hospital discharge. There was no correlation between any of the measured coagulation parameters and outcome.

Conclusion: In our preliminary data in children with sTBI, we found divergent results between standard coagulation tests and TEG-PM (specifically, INR-TEG). TEG-PM parameters suggest a mixed coagulopathic state with increased thrombin generation but moderate platelet inhibition. Larger prospective studies are needed to determine whether standard coagulation tests or TEG-PM more accurately identify and classify coagulopathy in children with sTBI.

Poster # 82

PREHOSPITAL IV FLUIDS ARE NOT ASSOCIATED WITH INCREASED MORTALITY IN PEDIATRIC TRAUMA PATIENTS

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Introduction:The association between pre-hospital intravenous fluid (IVF) administration and mortality has been reported with mixed findings in adult trauma patients; some results have shown increased survival and others increased mortality. Because the administration of prehospital IVF is not random, more severely injured patients are more likely to receive IVF. When this confounding by indication is not accounted for, estimates of IVF effect on mortality will be biased by residual confounding, even after risk adjustment. We sought to evaluate whether the observed increase in mortality associated with the use of pre-hospital fluids in our pediatric trauma population is wholly or partly explained by indication bias.

Methods:We performed a retrospective cohort study on all pediatric (0-18 years) trauma patients with an injury severity score (ISS) of 9 or higher who were transported directly from the scene of injury to a Level I pediatric trauma center between January 2008 and June 2011. The outcome of interest was in-hospital mortality and the exposure of interest was amount of pre-hospital IVF administered. This was dichotomously defined as either GT250 (receiving 250ccs or more [exposed]) or LT250 (receiving 0-249 ccs [not exposed]). The cutoff value was determined based on receiver operating characteristic (ROC) curve analysis, which maximized sensitivity and specificity in predicting mortality (AUC=0.7, sensitivity=0.81 and specificity=0.56). Using logistic regression, propensity for exposure assignment for each patient was then determined based on the following pre-hospital variables: mode of transport (air vs. ground), mechanism of injury, patient age, patient weight, intubation status, initial scene vital signs and time (from injury) to Level I trauma center (model c-statistic=0.8, 95%CI:0.76 -.084). Using Cox's regression to minimize survival bias, the independent effect of pre-hospital IVF on mortality was evaluated with and without adjusting for the exposure assignment propensity.

Results:A total of 482 patients met study criteria. Of these, 46.3% (223/449) received 250ccs or more of IVF. In-hospital mortality for the GT250 group was 9.4% (21/223) compared to 2% (5/259) in the LT250 group ($p < 0.05$). After adjusting for ISS, presence of a severe head injury, presence of shock and a penetrating injury, all of which were significant predictors of mortality, receiving 250ccs or more of pre-hospital IVF was significantly ($p=0.0373$) associated with an almost three-fold increase in the risk of in-hospital mortality, hazard ratio (HR) 2.96, 95%CI:1.1-8.2. However, adjusting for the propensity to receive 250ccs of IVF or less (pre-hospital baseline risk) attenuated the effect estimate and resulted in a non-significant ($p=0.3408$) association between pre-hospital IVF and mortality, HR 1.8, 95%CI: 0.5 – 6.2.

Conclusion:Propensity-adjusted survival analysis suggests neither a beneficial nor an adverse effect from pre-hospital resuscitation on mortality after adjusting for confounding variables in our pediatric trauma population. Our data would suggest that IVF resuscitation should not be a reason to delay patient transport to definitive care.

Poster # 83

THE ROLE OF ACTIVATED PROTEIN C IN THE DEVELOPMENT OF COAGULPATHY AFTER PEDIATRIC TRAUMA

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Introduction: Recent evidence for a distinct mechanism of early posttraumatic coagulopathy involves the activation of the anticoagulant protein C pathway. Whether this new mechanism of posttraumatic coagulopathy plays a role in children is still unknown. The purpose of this study was to determine the role of the activation of the protein C pathway in the development of coagulopathy early after severe civilian pediatric trauma.

Methods: We conducted a prospective observational study of pediatric patients after sustaining injury at a level 1 pediatric trauma hospital. Inclusion criteria: highest level trauma activation and arrival within 6 hours of injury. Exclusion criteria: >18 years of age, burns > 20% total body surface area and primary asphyxiation. Blood samples were collected within 20 minutes of arrival to the trauma bay for analysis of partial thromboplastin (PTT), prothrombin times (PT) and activated protein C (aPC).

Results: A total of 49 consecutive patients were enrolled. The mean age was 8.3 ± 4.9 years with 86% sustaining blunt trauma. The mean injury severity score was 20, median pediatric BIG score was 14.9 and overall mortality was 10%. The overall incidence of coagulopathy, defined as a PT ratio >1.2, was 29%. Patients presenting with an early coagulopathy showed a mortality rate of 38%. Non-survivors had a significantly higher PT ratio (2.2 vs 1.06, $p < 0.0001$) and significantly higher PTT levels (178 vs 27.9, $p < 0.0001$). Significantly higher aPC levels were seen in non-survivors versus survivors, 4.32 ng/mL and 3.13 ng/mL respectively ($p=0.033$).

Conclusion:Coagulopathy on arrival is associated with higher mortality in pediatric trauma patients with significantly higher levels of aPC demonstrated in non-survivors. Further evaluation of the mechanisms associated with coagulopathy in pediatric trauma and potential targets for treatment are warranted.

Poster # 84

PREDICTORS FOR EARLY BLOOD PRODUCT ADMINISTRATION IN THE PEDIATRIC TRAUMA POPULATION

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Introduction: Trauma is the leading cause of death in children, and hemorrhage accounts for 20-40% of all early trauma-related mortality. In adult trauma, there is an associated mortality benefit with early activation of transfusion protocols; however, this has not been well studied in the pediatric trauma. Our purpose was to identify predictors available early in the assessment phase that may predict need for transfusion and better assist our blood bank refining the products available at resuscitation.

Methods: Demographics, mechanism of injury, admission vitals, Glasgow Coma Scale score (GCS), and outcome were collected for all pediatric trauma patients retrospectively at a Level I pediatric trauma center from 2008 to 2013. Exclusion criteria included burns, patients with age >16, and those with insufficient data for analysis. We utilize a two tiered trauma activation system: Level 1 trauma includes those patients with intubation prior to arrival, hemodynamic instability, penetrating injury, GCS < 10, deteriorating neurologic status, multi-system injury, and significant soft tissue injury/amputation. All level 1 and level 2 trauma activations were included in the analysis. Transfusion was defined as the administration of any blood product within 24 hours of admission. A logistic regression model estimated odds ratios and 95% confidence intervals for the association between the need for transfusion and the predictors. Also, a sensitivity analysis was performed to assess how accurately these factors predict transfusion.

Results: Of the 1,945 pediatric trauma patients, 8.8% needed transfusion in the first 24 hours. There was a significant difference in mortality between those requiring transfusion and those who did not [28.1% vs. 1.1% ($p < 0.001$)]. Factors associated with transfusion included GCS ≤ 8, Level I activation, low systolic blood pressure, increased heart rate, and age < 3 years (Table 1). The c-statistic for the final model was 0.84. In patients with ≥ 2 predictors, we identified 79% of patients requiring transfusion (sensitivity) and 73% not requiring transfusion (specificity).

Conclusion: GCS ≤ 8 and Level I activation were the most predictive factors for transfusion available early in the pediatric trauma assessment. Prospective validation is warranted.

Table 1. Adjusted odds ratios (ORs) and associated 95% confidence intervals (CIs) for the association between demographic, injury, and clinical characteristics and need for transfusion

	Crude OR (95% CI)	Adjusted* OR (95% CI)
Age <3 years	2.25 (1.59-3.19)	1.87 (1.20-2.90)
GCS ≤8	15.51 (10.85-22.17)	4.29 (2.42-7.59)
Level 1 Activation	14.40 (10.06-20.61)	5.20 (2.91-9.30)
Increased heart rate (age-appropriate values)	1.14 (0.78-1.68)	1.78 (1.06-2.99)
Low systolic blood pressure (age-appropriate values)	5.19 (3.35-8.03)	3.73 (2.02-6.88)

* Adjusted for other variables in table

Poster # 85

ENTERAL ACCESS IN PEDIATRIC AND ADOLESCENT TRAUMA PATIENTS: DISPARITY BETWEEN ADULT AND PEDIATRIC HOSPITALS

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Introduction:Providing enteral access is a necessary component in the rehabilitation process of trauma patients. In the pediatric population, open gastrostomy tubes are often placed in combination with an anti-reflux procedure, which differs from the percutaneous approach that is common in the adult population. There are currently no evidence based recommendations as to the ideal method of providing enteral access in pediatric trauma patients. The aim of this study is to examine differences in the method of enteral access in pediatric and adolescent trauma patients at Level 1 pediatric versus adult trauma centers.

Methods:We performed a retrospective review of all trauma patients age 1 to 21 years who underwent an enteral access procedure between 1/2007 and 6/2013. Infants less than age 12 months were excluded. Patients from a pediatric Level 1 trauma center were compared to patients from an adult Level 1 trauma center. Demographic data, primary diagnosis, technique utilized for feeding tube placement, time to enteral access, time to discharge, and 30 day complications were recorded.

Results:Thirty patients underwent enteral access procedures in this time period. Twelve patients were treated at the children's trauma center with an age range of 20 months to 13 years, with a majority (77%) admitted due to closed head injury. At the children's hospital, surgical gastrostomy was performed in 10 patients, while 2 patients had enteral access placed under fluoroscopic guidance. Of the 10 patients undergoing open surgical gastrostomy, 8 patients (80%) underwent simultaneous fundoplication. Average time to enteral access was 35 days (range 14-63 days) and average time to discharge was 33 days (range 4-104 days). Eighteen patients were treated at the adult trauma center with an age range of 17 to 21 years. Closed head injury was the primary diagnosis in the majority of patients (72%). All patients received a percutaneous gastrostomy tube. Average time to access was 11 days (r 5-29 d) and average time to discharge was 14 days (r 6-32 d). There were no 30 day complications in either group.

Conclusion:There is a disparity in the method of enteral access utilized between adult and pediatric trauma centers. Permanent enteral access can be safely performed in the pediatric and adolescent population through either a percutaneous or surgical approach. There is a need for prospective multi-institutional studies to determine the optimal approach for enteral access and the role of fundoplication in this patient population.

Poster # 86

THE EVOLUTION OF PEDIATRIC TRANSFUSION PRACTICE

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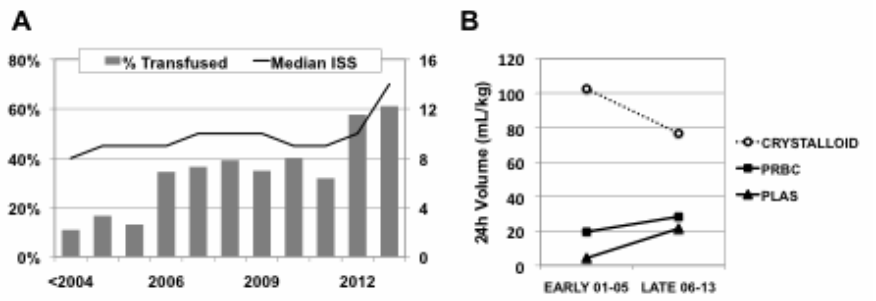
Introduction: The concept of hemostatic resuscitation has significantly changed adult trauma resuscitation. Transfusion practice patterns in pediatric resuscitation likely have changed as well; however, this evolution has not been quantified. We studied pediatric transfusion practices over time within a combat trauma system.

Methods: The Department of Defense Trauma Registry (DoDTR) was queried from 2001-2013 for pediatric trauma patients (<18 y). Burns, drowning, and missing injury severity score (ISS) were excluded. A subset without head injuries (isolated or predominant) was also evaluated. Volumes of packed red blood cells (PRBC), plasma (PLAS), platelets (PLT), cryoprecipitate (CRYO), and whole blood (WB) given in the first 24 hours were calculated per kg body weight. Transfusion practices were then evaluated from 2001-2005 (EARLY) vs. 2006-2013 (LATE) including proportion of transfused patients, volume of blood products administered, ratio of PLAS and PLT to red blood cells (RBC), and tranexamic acid (TXA) use. ICU and hospital length of stay (LOS) and 24-hour and in-hospital mortality were compared.

Results: 4,990 pediatric combat casualties were identified in the DoDTR. 632 were excluded for burns, drowning, and incomplete ISS data. The remaining 4,358 patients comprised the study cohort. Over time, the proportion of transfused patients rose along with an increasing ISS (Figure A). Comparing EARLY vs. LATE, median ISS (9 vs. 10, $p<0.0001$), injuries from explosions (33.8% vs. 47.4%, $p<0.0001$) and isolated or predominant head injuries (12.4% vs. 17.5%, $p<0.0001$) all increased significantly. In the 1,377 transfused patients, mean 24-hour crystalloid volume decreased while PRBC and PLAS volume increased (Figure B). Transfusion of a high ratio of PLAS to RBC (≥ 0.5) increased (17.2% vs. 63.4%, $p<0.0001$). The volume of PLT and CRYO increased while WB was unchanged, and TXA use increased. ICU and hospital LOS decreased in the LATE group. Mortality increased in the overall cohort at 24 hours (2.0% vs. 4.2%, $p=0.0006$) and in-hospital (5.7% vs. 7.8%, $p=0.01$). Excluding those with isolated or predominant head injury (n=708), 24-hour mortality increased (1.2% vs. 2.6%, $p=0.02$) while in-hospital mortality was unchanged (3.6% vs. 5.2%, $p=0.07$) over the study period.

Conclusion: Transfusion practice in pediatric combat casualty resuscitation has shifted towards a more hemostatic approach since 2001. Further study is required to determine if this practice should apply to civilian pediatric resuscitation.



Poster # 87

Racial Bias in the Management of Severe Traumatic Brain Injury: Fact or Fiction?

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Introduction: A complex interplay exists between socioethnic factors and outcome after traumatic brain injury (TBI). While few physicians would claim overt discrimination, several studies have identified racial or ethnic disparity in clinical outcomes and functional recovery following TBI. However, the impact of race on the acute management of patients treated for severe TBI at trauma centers is unknown. Therefore, we asked the question: Is there a racial bias associated with neuro-monitoring and operative intervention in the acute management of patients treated at trauma centers following severe TBI, and does this have an effect on mortality?

Methods: We performed a retrospective analysis of adult patients (aged 18-55) admitted to level 1 & 2 trauma centers between 2007-2011 using the National Trauma Data Bank (NTDB). Patients were included if they had an initial Glasgow Coma Scale (GCS) 3-8 and a head Abbreviated Injury Scale (AIS) of 3-5. Patients were excluded if any anatomical AIS other than head was 3 or greater or if their file contained an empty data field in specific categories (race, mortality, neuro intervention). Coarsened exact matching was performed using age, GCS, Injury Severity Score (ISS), systolic blood pressure and respiratory rate on admission, gender, blunt versus penetrating mechanism of injury, and comorbidities to compare white to non-white patients. Missing data for variables used in the matching was imputed via a multiple regression approach. Outcomes were then analyzed using mixed effects regression analyses with patients nested within facility. Primary outcomes of neuro-monitoring or therapeutic intervention were identified using ICD-9 codes. Secondary outcomes were recorded for intensive care unit days, total length of stay, and mortality.

Results: 7618 patients were identified for analysis. Median age was 36, 81.2% were male and 64.5% were white. Medians for GCS, head AIS, and ISS were 3, 4, and 21 respectively. Rates of neuro-monitoring, operative intervention, and mortality were 8.8%, 16.5%, and 28.5% respectively. 6886 patients were able to be matched and below is the summary of the matched multivariate analysis comparing white to non-white patients. Results were unchanged when patients were stratified by mechanism (blunt and penetrating) and when looking only at blunt mechanism TBI patients with a length of stay > 1 day.

Matched Multivariate Analysis

	White	Non-White	Odds-Ratio	p-value
Hospital LOS - Days, median (IQR)	6 (3)	7 (7)		< 0.001
ICU LOS - Days, median (IQR)	4 (4)	4 (4)		0.071
Pneumonia, %	6.8	6.6	0.97	0.765
Neuro Monitoring, %	9.4	8.1	0.85	0.129
Neuro Intervention, %	16	18	1.04	0.610
Mortality, %	28.8	29.9	1.12	0.096

Conclusion: In a matched analysis using the NTDB, there were no observed differences in neuro-monitoring, operative intervention and mortality between adult white and non-white patients with severe isolated TBI treated at level 1 and level 2 trauma centers. Hospital length of stay was greater for non-white patients when compared to white patients. There does not appear to be a racial bias in the acute management of severe TBI at high level trauma centers. This controversial topic remains an important area for future research.

Poster # 88

PULSE PRESSURE VARIATION MEASURED IN THE ICU FOLLOWING HEMORRHAGE CONTROL DOES NOT IDENTIFY FLUID RESPONSIVENESS IN MECHANICAL VENTILATED TRAUMA PATIENTS

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Introduction: Pulse pressure variation (PPV) predicts fluid responsiveness (FR) in septic shock and high-risk surgery ventilated patients. It has been proposed to guide initial fluid resuscitation in severe trauma patients but its accuracy has not been evaluated in the immediate post-operative trauma patient following hemorrhage control procedures.

Methods: Previous IRB approval, a prospective cross sectional study was designed to evaluate PPV and static preload measurements (PVC and WPP) as predictors of FR in trauma patients with class IV hemorrhagic shock, after controlling bleeding by either surgery or angio embolization, who continued to receive fluid resuscitation in the ICU. All the patients were mechanically ventilated (V_T 6 – 8 mL/Kg), and receiving intravenous sedation and neuro muscular blockade drugs as necessary. A continuous cardiac output (CO) pulmonary artery catheter (Baxter-Edwards, Irvine, Ca) and a radial artery catheter connected to a bedside monitor were used for the assessment of the hemodynamic variables. PPV calculations were made by the freezing and caliper tools available in the monitors. Displayed values were measured by triplicate, averaged and registered before and after the administration of successive 500 mL bolus (up to three) of Gelatine Polisuccinate in Ringer Acetate. ROC-AUC analyses were performed to evaluate discriminating ability of each of the tests, to identify a CO change >15% as an indicator of positive FR.

Results:A total of 54 fluid challenges administered in 19 patients with penetrating (n=13) blunt (n=4) and explosion (n=2) injuries were registered. At the start of the intervention four patients remained hypotensive, three received vasopressors and all included subjects showed biochemical evidence of hypoperfusion. We found that CO increased >15% in 27 of the events (50%). ROC-AUC for predictions of FR were 0.36 (IC 95% 0.21 - 0.52) for WPP, 0.37 (IC 95% 0.21 - 0.52) for CVP and 0.40 (IC 95% 0.25 - 0.56) for PPV.

Conclusion: PPV measured by bedside monitors did not discriminate the responsiveness to IV fluids in trauma patients under resuscitation after surgical control of bleeding.

Poster # 89

TRANSESOPHAGEAL ECHOCARDIOGRAPHY IS EQUAL TO PULMONARY ARTERY CATHETERIZATION FOR TRAUMA AND CRITICAL CARE RESUSCITATION

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Introduction: Pulmonary artery catheter (PAC) use in critically ill populations is being increasingly questioned, with conflicting reports on reliability. A hemodynamic transesophageal echocardiography (hTEE) probe was used to validate its use in resuscitating critically ill trauma patients compared to PAC.

Methods: In a prospective case-series of critically ill trauma patients with PACs, 29 patients received an hTEE probe. Each patient was evaluated with respect to optimal preload (pulmonary arterial occlusion pressure [PaoP] 18-24mm, left ventricular end-diastolic area [LVEDA] 10-12cm2), then with respect to optimal contractility (cardiac index [CI] 2.6, fractional area change [FAC] 40%). Data obtained simultaneously from both modalities was interpreted and compared; treatment was then rendered according to protocol.

Results: In 20 patients, the hTEE supported PAC data in the treatment needed, demonstrating moderate agreement ($\kappa = 0.54$). In 9 patients, the hTEE and PAC data were not synchronous: in 6 of these patients, hTEE favored volume resuscitation, and in the remaining 3 patients PAC favored volume resuscitation. Of these 9 patients, clinical therapy was guided by hTEE in 7, with one mortality and resolution of the clinical picture in the remaining six. In 2 patients, clinical therapy was guided by the PAC, with one mortality and resolution of the clinical picture in the other patient. In 23 patients, data from the hTEE changed management of the patient, 7 of which differed from the recommended PAC treatment. In 3 patients where PAC indicated no change in therapy, the hTEE suggested volume resuscitation.

Conclusion: In trauma patients there is moderate agreement between hTEE and PAC in the ability to diagnose predominant shock etiology and direct therapy, and can help validate PAC data. Further research is warranted to determine if hTEE is a better means of detecting preload problems than PAC.

Poster # 90

INDICES OF INFLAMMATORY RESPONSES AND OXIDATIVE STRESS IN TISSUES FROM PIGS SUBJECTED TO EXSANGUINATION SHOCK

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Introduction: Uncontrolled torso bleeding remains a leading cause of death from potentially survivable injuries in both military and civilian trauma patients. It has been estimated that such massive bleeding and resultant cardiac arrest many account for up to 80% of preventable deaths in combat casualties. We have developed a porcine model of exsanguination cardiac arrest (ECA) to test novel surgical and pharmacological interventions. The present study characterized the inflammatory responses in this model.

Methods: Conscious, sedated swine (n=5/gp) were subjected to a computerized hemorrhage of 80% total blood volume over 20 min and monitored until development of cardiac arrest and death. Then, lung, heart, kidney and liver were collected, frozen and stored at -80 oC for analysis of indices of oxidative stress (thiobarbituric acid reactive substances (TBARS), antioxidant status (total antioxidants, glutathione, antioxidant enzymes) and cytokines (IL-1β, IL-6, TNF-α) plus HSP90. Results were compared to historic sham control animals similarly instrumented (n=4).

Results: Time to cardiac arrest was 18.3 + 0.7 min and blood loss at time of ECA was 71.0 + 1.5%. TBARS were nearly 50% higher in ECA liver and elevated in kidney compared to corresponding sham tissue. In liver, heart and lung, the reduced-to-oxidized glutathione levels were significantly higher in shams than ECA swine. Mn-superoxide dismutase (Mn-SOD) activity was over 40% higher in ECA liver and kidney compared to shams. IL-1β in liver and lung, and TNF-α in lung from ECA animals were markedly elevated compared to shams, whereas IL-6 levels were not significantly different between groups in all tissues. HSP90 levels were significantly higher in all ECA tissue than shams.

Conclusion: These data indicate that ECA induced a significant inflammatory response as indicated by elevations in TBARS, cytokines, HSP90 and Mn-SOD activity and lower ratios of reduced to oxidized glutathione levels. Interestingly, there were differential responses among the tissues analyzed. Taken together with sustained cardiac contractility in ECA animals reported previously, these data suggest that lifesavings interventions to include pharmacologic therapy targeted to the inflammatory response may be beneficial in exsanguination cardiac arrest.

Poster # 91

A PROSPECTIVE ANALYSIS OF URINARY TRACT INFECTIONS AMONG ELDERLY TRAUMA PATIENTS

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Introduction:Catheter-associated urinary tract infections (CAUTI) have been deemed "reasonably preventable" by the Centers for Medicare and Medicaid (CMS) thereby eliminating reimbursement. Elderly trauma patients, however, are at high risk for developing urinary tract infections (UTI) given their extensive comorbidities, immobilization, and environmental changes in the urine which provide the ideal environment for bacterial overgrowth. Whether these patients develop CAUTI as a complication of their hospitalization or have asymptomatic bacteriuria (ASB) or UTI upon admission must be determined in order to justify the "reasonably preventable" classification. We hypothesize that a significant proportion of elderly patients will present with ASB and UTI on admission.

Methods:IRB permission was obtained to perform a prospective, observational clinical trial of all elderly (≥ 65 years) patients admitted to our Level I Trauma Center as a result of injury. Urinalysis (UA) and culture (UCx) were obtained at admission, 72 hours, and, if diagnosed with UTI, at 2 weeks after injury. UTI was defined as a bacteriuria ($\geq 10^5$ colony forming units) with associated symptoms while ASB was defined as a bacteriuria without symptoms. Pyuria was defined as ≥ 4 neutrophils per high power field (HPF) and microscopic hematuria was defined as ≥ 3 RBC per HPF. Mean cost of UTI was calculated based Center for Disease Control estimates of $862 - $1,007 per UTI.

Results:Of 201 eligible patients, 129 agreed to participate (64%). Mean age was 81±8.6 years. All patients had a blunt mechanism of injury (76% falls) with a mean Injury Severity Score of 13.8±7.6. Of the 18 (14%) patients diagnosed with UTI, 14 (78%) were present at admission. Additionally, there were 18 (14%) patients with ASB on admission. Therefore, 32 (25%) patients had a bacteriuria (UTI + ASB) at admission. All of the admission UTIs resolved by day 3 but 2 patients with admission ASB had persistent bacteriuria. The most common bacterial species present on admission urine culture were *E. coli* (24%) and *Enterococcus* (16%). Clinical features associated with bacteriuria on admission included a history of UTI (76% vs 41%, $p<0.01$), positive gram stain (68% vs 1.5%, $p<0.01$), abnormal microscopy (91% vs 45%, $p<0.01$), and pyuria (83% vs 39%, $p<0.01$). The estimated loss of reimbursement for 18 UTIs on admission was $15,516 - $18,126; however, given an estimated cost of $1981 to screen all patients with UA and UCx at admission, $16,144 savings was realized.

Conclusion: Many elderly trauma patients present with concurrent bacteriuria. Most UTIs which would have otherwise been diagnosed in the hospital were in fact present on admission. Screening UA and UCx on admission for elderly trauma patients identifies these UTIs and is cost-effective.

Poster # 92

INCREASING ORGAN DONATION AFTER CARDIAC DEATH IN TRAUMA PATIENTS

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Introduction: Trauma patients form one of the largest pools of organ donors. Organ donation after cardiac death (DCD) is not optimal but still remains a valuable source of organ donation. The aim of this study was to assess national trends in donation after cardiac death from trauma patients.

Methods: We performed a 12 year (2002-2013) retrospective analysis of the United Network for Organ Sharing (UNOS) database. Eligible trauma patients who donated solid organs after cardiac death were analyzed. Outcome measures were: conversion rate (number of donors divided by number of eligible donors), and number and type of solid organs donated.

Results: A total of 120,512 eligible trauma organ donors were reviewed and the conversion rate was 11.36%. Donation after cardiac death resulted in procurement of 16,248 solid organs from 8,724 donors. The number of organs donated per donor remained unchanged over the study period (2 organs/donor in 2002 and 1.8 organs/donor in 2013,p=0.1). Donation after cardiac death increased significantly from 3.1% in 2002 to 14.6% in 2013 (p=0.001). There was a significant increase in the proportion of kidney (2002: 3.4% vs. 2013: 16.3%, p=0.001) and liver (2002: 1.6% vs. 2013: 5%, p=0.041) donation over the study period.

Conclusion: Organ donation after cardiac death from trauma patients provides significant number of solid organs. The rate of organ donation after cardiac death increased significantly over the last 12 years. Increasing education regarding donation in general to the public and to the trauma community may aid in increasing the number of organs available for donation.

Poster # 93

THE UNINSURED, THE HOMELESS AND THE UNDOCUMENTED IMMIGRANT TRAUMA PATIENT. REVEALING HEALTH CARE DISPARITY AT A LEVEL ONE TRAUMA CENTER.

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Introduction: Insurance status has been linked to patient outcome. Differences between patients that are uninsured and have additional disparities such as homeless or undocumented immigrant patients have not been defined. The objective of this project is to review and compare the degree of injury, and financial burden between uninsured trauma patients with and without further disparities.

Methods: The trauma registry was used to evaluate these patients retrospectively from January 1st to December 31 of 2013. Data evaluated included age, mechanism of injury, ISS, hospital length of stay, ICU length of stay, mortality, hospital cost, physicians' charges, hospital charges, and actual payments received. Comparison was made between uninsured patients without further disparities or self –pay (SP), uninsured patients that are homeless (H) and uninsured patients that are undocumented immigrants (UI).

Results: During the study period 2619 patients presented as trauma alerts to a level one trauma center. The total number of uninsured patients was 900. Of those 873 were self-pay (SP), 11 were homeless (H) and 16 were undocumented immigrant (UI) patients. Age was similar between the groups (Mean: SP=34.7, H=36.3, and UI=31. Median: SP=31, H=32, and UI=27). The homeless patients had a median ISS five times higher when compared with the other groups (Mean: SP=8.8, H=21.3, and UI=9.2. Median: SP=5, H=25, and UI=5.5). Homeless patients also had an increased hospital length of stay (Mean: SP=3.9, H=15.4, and UI=8.8 Median: SP=1, H=4, and UI=2) and ICU length of stay (Mean: SP=1.3, H=3.4, and UI=2.9). Mortality in homeless patients was four times higher when compared with undocumented immigrants and five times higher when compared with self-pay patients. (SP=4.47, H=27.27, and UI=6.25). Accordingly, hospital costs were also higher in the homeless group (Mean: SP=15550, H=47370, and UI=28714. Median SP=6298, H= 11937, and UI=7025), as well as physician charges (Mean SP=6533, H=24788, and UI=9780) and hospital charges (Mean: SP=61144, H=170353, and UI=112034). No actual payments were received at all, to cover for the care of homeless trauma patients during the study period (Mean: SP=1890, H=0, and UI=9494).

Conclusions: In this retrospective review of a single center, uninsured homeless trauma patients were sicker and congruently represented more cost for the hospital when compared with other uninsured trauma patients. Interestingly no actual payments have been received to cover for the care of homeless patients . A multi-center trial could be conducted to elucidate the problem better and create awareness, to promote the search for a solution.

Poster # 94

THE IMPACT OF INSURANCE STATUS, DIABETES AND UNDIAGNOSED DIABETES ON OUTCOMES AMONG TRAUMA PATIENTS

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Introduction: Medical factors, such as diabetes, and non-medical factors, such as insurance status,affect outcomes following traumatic injury. We recently demonstrated that when accounting for medical co-morbidities, the effect of insurance status upon trauma outcomes is minimized. Indeed potentially undiagnosed and/or poorly controlled medical co-morbidities appear to exert a tremendous effect upon trauma outcomes. The effect insurance status plays on trauma outcomes may be a reflection of access to health care, more co-morbidities diagnosed and better control of these co-morbidities pre-trauma admission. We wished to assess the impact of insurance status on the pre-trauma diagnosis and management of diabetes, rates of potentially previously undiagnosed diabetes, and whether any undiagnosed diabetes was associated with trauma outcomes.

Methods: A 10 year retrospective review of admitted trauma patients, 18 years and older to a level 1 trauma center. Patients who died within 24 hours were excluded. Charts were reviewed for age, gender, medical co-morbidities, specifically diabetes, insurance status, mechanism, and ISS. Hospital course was reviewed for HbA1c levels, any infectious outcome, length of stay and mortality. Further, charts were reviewed for potentially undiagnosed diabetes. Charts of patients with no known diagnosis of diabetes were reviewed for the four glucose levels prior to discharge as well as any HbA1c measured during their trauma admission. Patients were considered undiagnosed diabetic if they had any of the following; HbA1c>/=6.5%, two fasting glucose levels >125mg/dL, or two random glucose levels >200mg/dL. Chi-squared, t-test and regression analyses were undertaken.

Results:Over the 10 year period 23,220 patients were admitted, 21% of whom were uninsured. Insured patients were older (55.7 vs 36.9 years; $p<0.001$), less likely to be male (59.5% vs 79%; $p<0.01$), but had similar injury severity scores (11.3 vs 11.5; $p=0.3$). Insured patients were more likely to have a pre-trauma diagnosis of diabetes (6.7% vs 3.5%; $p<0.001$). Among these diabetics, insured patients had better pre-trauma diabetes control as reflected by lower average HbA1c levels (7.9% vs 9.6%; $p=0.006$). Of the non-diabetic patients, 34.7% were potentially undiagnosed diabetics. Adjusting for age and gender, insurance status did not affect the possibility of having undiagnosed diabetes (OR=1.05; 95%CI=0.95-1.17). However, adjusting for age, gender and ISS, patients with potentially undiagnosed diabetes were noted to have an increased risk of infection during their hospital stay (OR=1.5; 95%CI=1.06-1.36) and an increased risk of death (OR=2.4; 95%CI=1.9-3.1)

Conclusion: It is becoming increasingly evident that pre-trauma medical health is a large determination of outcomes following admission for traumatic injuries. Insurance status may be affecting trauma outcomes through access to health care and better control of known medical conditions. We have herein demonstrated that potentially a large portion of the trauma population may have un-diagnosed diabetes. Further, undiagnosed diabetes plays a significant role in trauma related outcomes. Trauma remains a leading cause of morbidity and mortality. We believe that better access to health care and screening for controllable medical conditions is crucial to further advances in trauma care.

Poster # 95

FMRI EVIDENCE FOR THE USE OF A 4-ITEM SCREEN FOR PTSD IN INJURED TRAUMA SURVIVORS

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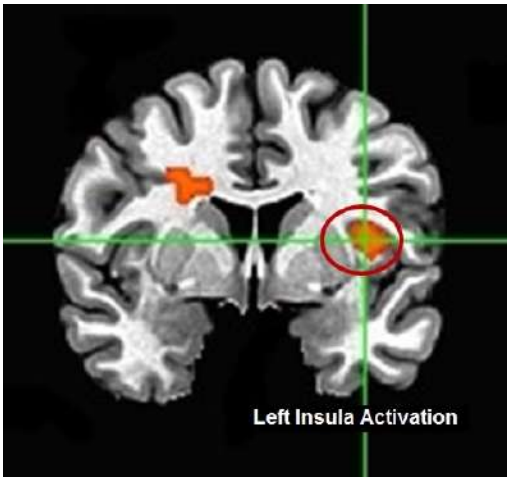
INTRODUCTION: Functional magnetic resonance imaging (fMRI) research suggests that early emotional dysfunction is evident at the neural circuitry level in patients who go on to develop posttraumatic stress disorder (PTSD) at 6 months. The purpose of this study was to determine the relationship between the primary care – PTSD (PC-PTSD) screen and neurologic dysfunction using fMRI technology.

METHODS: Twenty-four motor vehicle crash survivors treated at a Level I trauma center were recruited within 2 weeks post trauma, were given a measure that included the four questions from the PC-PTSD, and underwent fMRI imaging during a trauma narrative paradigm (listening to the story of their motor vehicle crash). Two tailed Pearson Correlations were used to evaluate the relationship between activation of brain structures responsible for emotional processing of fear and the four items from the PC-PTSD.

RESULTS: There was a significant positive relationship between PC-PTSD scores and left insula activation (corrected $p<0.05$, min. cluster size = 12). There was also a significant cluster in left superior temporal gyrus where there was a significant negative relationship between PC-PTSD scores and activation (corrected $p<0.05$, min. cluster size = 12).



CONCLUSION: Early intervention is efficacious in the management of PTSD if at risk patients are identified. While imaging every trauma survivor to determine PTSD risk is not feasible, the use of this 4-item screen would be clinically useful and efficient. This short, easily administered scale appears to be a valid tool for early identification of patients at increased risk of developing PTSD.

Poster # 96

DETERMINANTS OF DRIVING UNDER THE INFLUENCE PROSECUTION IN TRAUMA PATIENTS AT AN URBAN LEVEL 1 TRAUMA CENTER

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Introduction:

We sought to determine the prosecution rate (PR) for alcohol-intoxicated drivers injured in motor vehicle crashes presenting to a Level 1 Trauma Center. This study investigates the differences between those charged and not charged with driving under the influence (DUI).

Methods:
Alcohol-intoxicated drivers from 2009-2012 were identified through the local trauma registry. Patient medical and public judicial records were retrospectively reviewed. Demographic, injury severity, length of stay, and prosecution data were collected.

Results:

1715 patients with prosecution data were identified. Of these, 78 persons were prosecuted yielding an overall prosecution rate (PR) of 4.6%. The PR was significantly higher in females (6.5% vs. 4.0%,p=0.0309) and was significantly higher in blacks (8.3%) and Hispanics (8.8%) than in whites (3.9%) (p=0.0020). We observed no difference in injury severity, length of stay, or patient outcomes. Patients with one or more previous DUI conviction(s), prior vehicular or alcohol infractions, or comorbid intoxication with cocaine or marijuana did not experience higher PR. The association between insurance type and prosecution status exhibited a trend toward significance ($p = 0.0595$), with managed care patients having the highest prosecution rate (9.5%), and Medicare (1.3%) having the lowest. The PR was significantly higher among recidivist patients than among non-recidivist patients (14.3% vs. 5.0%), and the overall rate of recidivism within the study period was 3.9%. There was no significant association between prosecution status and age (even when categorized as < 21 and ≥ 21), type of machine driven, ED disposition, or DMV notification of DUI status by physicians.

In our multivariate analysis, the main predictors for prosecution were gender, race, and level of intoxication. Women were 1.8 times more likely than men to be prosecuted (p=0.0151). Black patients were 2.4 times more likely than whites to be prosecuted (p=0.0020); Hispanic patients were also more likely to be prosecuted, but this did not achieve statistical significance. The likelihood of prosecution increased by 3.7% for every 10 mg/dL increase of BAC.

Conclusion:

PRs for DUI at our trauma center were dismal at 4.6%, and the main predictors for prosecution were female sex, black race, and higher BAC level. Our high recividism rate with almost 4% of patients acquiring a subsequent DUI within the study period highlights the need for urgent multidisciplinary intervention.

Poster # 97

NATIONAL MANDATORY MOTORCYCLE HELMET LAWS CAN SAVE $2.2 BILLION PER YEAR: INPATIENT AND VALUE OF STATISTICAL LIFE ANALYSIS

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Introduction:
As of 2010, 19 states have mandatory motorcycle helmet laws. Two states, Illinois and Iowa, do not have any motorcycle helmet laws. While ample statistics exist regarding the overall rate of fatalities in motorcyclists with and without helmets, a combined inpatient and value of statistical life (VSL) analysis has not previously been reported.
Methods:
Statistical data of motorcycle collisions were obtained from the Centers for Disease Control, National Highway Transportation Safety Board, and Governors Highway Safety Association. The VSL estimate was obtained from the 2002 Department of Transportation calculation. Statistics on helmeted vs. nonhelmeted motorcyclists, death at the scene, and inpatient death was obtained using the 2010 National Trauma Data Bank. Inpatient costs were obtained from the 2010 National Inpatient Sample. Population estimates were generated using weighted samples and all costs are reported using 2010 USD using the Consumer Price Index.
Results:
3,951 fatal motorcycle accidents were reported in 2010, of which 77% of patients died at the scene, 10% in the ER, and 13% as inpatients. 37% of all riders did not wear a helmet, but accounted for 69% of all deaths. Of those motorcyclists who survived to the hospital, the odds ratio of surviving with a helmet was 1.51 compared to those without a helmet ($P<0.001$). 6.5% of hospitalized patients died following motorcycle collision. A VSL analysis ($47,040 per year) yielded $6.8 billion of indirect losses. Total costs for nonhelmeted motorcyclists were 66% greater at $5.5 billion, compared to $3.3 billion for helmeted motorcyclists ($P<0.001$). Direct inpatient costs were 16% greater for helmeted riders ($203,248 vs. $175,006) but led to over 50% greater VSL generated (absolute benefit $602,519 per helmeted survivor).
Conclusion:A cost analysis of inpatient care and indirect costs of motorcycle riders who do not wear helmets leads to an excess of $2.2 billion in losses per year, with almost 1.9 times as many deaths compared to helmeted motorcyclists. The per capita cost per fatality is over $800,000. Institution of a mandatory helmet law can lead to an annual cost savings of over $2.2 billion, plus an additional $2.4 billion generated as a result of a VSL calculation for a total of $4.6 billion net gain per year.

Poster # 98

IMPROVING TRAUMA PATIENT COMPLIANCE WITH FOLLOW UP CLINIC APPOINTMENTS

Jennifer L. Mooney MD, Anna Poullard ACNP-BC, Amanda Theriot APRN, FNP-BC, John P. Hunt* MD, LSU Department of Surgery

Introduction: Compliance with follow up clinic appointments is considered to be quite poor in the trauma population with approximately 40-50% of patients being lost to follow up. [1] The perception that trauma patients do not follow up has implications on patient care that include choice in medication, operative decisions and even the length of time a patient is kept in the hospital. More recent studies have challenged this perception and found that 79% of patients keep at least one scheduled appointment. [2,3] Follow up appointments are an important component in the care of the traumatically injured patient. Malhotra and colleagues found that 17% of trauma patients had significant health issues that were not discovered until follow up visits. [4]

Methods: This study was conducted at an urban level one trauma center. Usual protocol for post inpatient follow up consists of a letter being mailed to the patient supplying them with appointment dates and times. As part of a quality improvement project, all trauma patients discharged during a two-month period were met with by either the nurse practitioner (NP) or surgical intern prior to discharge. During this visit the NP or intern performed a simple intervention; they told the patient when their appointment would be before they left the hospital. A chart review was then performed to ascertain which appointments the patient had kept as well as other demographic information. These patients were then compared to patients admitted during the preceding six months.

Results: Prior to the study intervention there were 465 patients, which made up our control group (PRE). During the two-month study period there were ninety-nine patients admitted and included in the quality improvement initiative (POST). 34% of these patients were victims of penetrating trauma and 66% blunt trauma. The average ISS score was 12.5 $\pm$8.6 and 61% of patients underwent at least one operation during their hospital stay. Overall 38% of all patients were lost to follow up. When looking at the breakdown of specialty clinic 57% were compliant with trauma clinic follow up, 44% with neurosurgery, 69% with orthopedic surgery and 64% with other clinics. When comparing the PRE and POST intervention groups 42% of PRE patients were lost to follow up (58% compliant) and only 24% of POST patients did not keep at least one appointment (76% compliance)($p=0.0027$). Sub group analysis of individual clinics did not show any statistically significant differences between PRE and POST patients. Several factors were analyzed by univariate analysis to see if they had any affect on patient compliance with follow up. Being in the intervention group (POST) ($p=0.0027$), having an operation ($p<0.0001$) and a higher ISS were all associated with increased probability of keeping at least one appointment. Patients who were compliant had an average ISS of 13.9$\pm$8.64, whereas those who were lost to follow up had an average ISS of 10.0 $\pm$ 7.69 ($p<0.0001$).

Conclusions: Aaland and colleagues found that among their trauma patients lost to follow up, 36.8% of them were lost due to errors on part of the physician or hospital. [2] The perspective that trauma patients do not comply with follow up visits may have a negative impact on clinical decision-making. In this study we have shown that by merely providing the patient with the time and date of their follow up appointment prior to discharge, follow up compliance improved significantly from 58% to 76%.

Poster # 99

INTERNAL STAFF SURGE DURING MASS CASUALTY EVENT PRESERVES OTHER HOSPITAL RESOURCES

James M. Corder III, MD, Niki Rasnake RN, Kelly McNutt RN, Scott Smith MD, Blaine L. Enderson* MBA,MD, Brian J. Daley* MBA,MD, University of Tennessee Medical Center, Knoxville

Introduction:Mass casualty events disrupt normal hospital operations that go beyond trauma or surgical services. Certain key resources are invaluable during such events and these appear as bottlenecks that impair patient flow. We reviewed our mass casualty event to assess patient flow and resource consumption as well as the impact on non-trauma services.

Methods:As part of Performance Improvement and with IRB approval, we studied our recent mass casualty event. Twelve elderly patients were injured in a freeway bus crash and brought to the nearest level 1 trauma center after EMS scene triage based on START methodology. The time of patients to ED diagnostic studies and to ICU/Floor transfer were recorded and analyzed. ED non-trauma patient flow and OR patient flow were similarly studied. Patient demographics and outcomes were recorded from the NTRACS database. Outcomes were compared with chi-square analysis and case matched outcomes.

Results:The average age was 69.7 years old and the average ISS was 24.3. ED LOS was 3.4 hours (range 0.6-9 hours). Patients arrived within a 35 minute window midafternoon. Those patients requiring ICU/OR disposition had an ED LOS of 2.3 hours (range 0.6-5.4 hours). Additional resources for patient care were derived from three additional trauma surgeons and one fellow as well as three additional mid-level residents above the designated trauma team (an attending and 4 residents). Four nurses were mobilized from administration and three clerks were shifted to handle non-clinical trauma victim duties. Housekeeping prioritized ICU bed cleaning for 2 hours from on duty personnel. During the time interval, ED patient flow was maintained (238 patients/day, 5 patients/day left without being seen, not different than quarterly data) and OR volume was 67 cases (not significantly different from daily case volume preceding three months). All patients survived, mean LOS was 13.6 days, and 9 patients (75%) were discharged to SNF/Rehab. Survival was improved from case matched controls ($p<0.05$), but LOS was significantly longer ($p<0.05$).

Conclusion:A mature trauma center can handle a mass casualty event with internal resources without delaying other non-trauma services and maintaining sound trauma care. Administration, however, must recognize the value of staffing for surge contingency and maintaining a flexible pool of trained clinicians readily available from other assignments to avoid hospital wide impact.

Poster # 100

On the Feasibility of Using All Payer Claims Data to Monitor Regional Trauma Care Transitions

Sylvia D. Hobbs MPH, Selwyn Rogers* MD,MPH, Frederick H. Millham* MBA,MD, Wenjun Li Ph.D., Center For Health Information And Analysis

BACKGROUND: American College of Surgeons trauma verification includes pre-review of service and referral geographic catchment areas around trauma centers from data collected in hospital and state-level trauma registries. Service area data from such registries based on farthest Euclidean pre-hospital transport distance of ZIP code clusters lacks health exchange information that could quantify out migration of service area patients to surrounding states. Out migration information would facilitate state-level gauging of care fragmentation, assessing regional gaps in specialty taxonomies, and determining to what extent drops in trauma center requisite patient volume are attributable to catchment drift. We sought to determine the feasibility of using Massachusetts (MA) All Payer Claims Database (APCD) to fill this critical gap in state-level and hospital-level information on trauma care navigation by MA residents outside of state boundaries, specifically in bordering New England Region States, and profile referral and transfer patterns in 'in state' and 'out of state' trauma care seekers.

METHOD: An extraction of 1.74 million private-sector health plan beneficiary trauma care (CY 2009-2011) medical claims for 229,557 episodes of care needed by 91,477 MA residents from APCD for MA Level I, II, and III trauma center primary service area ZIP codes were analyzed for out of state inpatient trauma care seeking patterns by service provider specialty taxonomy, referral indicators, covered days, patient outcomes, patient age, and in network payment flags. Statistical tests and patient-level roll up of claims by patient and payer claim control numbers, demographic and diagnoses information, service payment window dates and provider information were performed using SAS (version 9.2). Geographic data visualization of maximum MA resident out of state linear trauma care seeking distance using de-identified aggregated ZIP codes and census attributes were performed using ESRI's ArcMap (version 9.3.1).

RESULTS: Private health plan beneficiary MA residents receiving inpatient trauma care out of state constituted 9% of the patient sample with 50% of that care provided in New England (NE) states bordering MA in order of the following MA patient volume ranking: RI, NH, CT, NY, ME, and VT. Comparing in state care seekers (mean age 53) to NE care seekers (mean age 44), NE patients had increased odds of care destination through referral (OR 1.34, 95% CI 1.28-1.4, $p < 0.0001$ and a 42% higher risk of charges not paid in comparison to MA ($p<0.0001$). Florida and Texas rank as the highest volume non-New England region sites of care. Clinically, NE patients had a higher rate of care sought for open fracture of base of skull with subarachnoid, subdural, and extradural hemorrhage, with loss of consciousness of unspecified duration than non-MA care seekers outside of the NE region. SAS method for patient rollout of claims data was successfully validated against trauma registry volumes for single eligibility beneficiaries. Trauma Registry data had higher quality external cause of injury than claims data.

CONCLUSION: APCD systems in combination with existing clinical registries and pre-hospital data can fill gaps in information needed for care coordination and monitoring disparities in access to care and provide new information on in state and out of state post discharge care settings.

Poster # 101

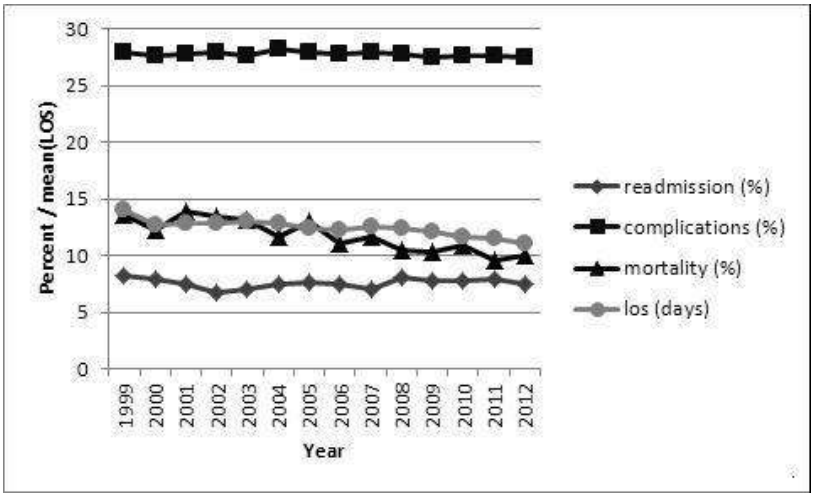
EVOLUTION OF PATIENT OUTCOME IN AN INTEGRATED TRAUMA SYSTEM: 1999-2012

Lynne Moore Ph.D., Henry T. Stelfox MD, Alexis F. Turgeon MD, André Lavoie Ph.D., Gilles Bourgeois MD, Jean Lapointe MD, Xavier Neveu MSc Laval University

Introduction: The introduction of trauma systems in many countries worldwide has been shown to improve outcomes following injury. However, few data are available on the evolution of patient outcomes in fully integrated trauma systems. The objective of this study was to describe the evolution of patient outcomes in a Canadian provincial trauma system from 1999 to 2012.

Methods: This population-based retrospective cohort study was based on patients with major trauma (Injury Severity Score >15) treated in the integrated trauma system of Quebec, Canada between 1999 and 2012. Over 90% of major trauma admissions are treated within the system and this proportion has remained stable over the study period. Data was drawn from the trauma registry linked to the hospital discharge database and mortality files. Performance was evaluated using quality indicators of 30-day mortality, unplanned readmission, length of stay, and complications, derived and validated previously. Performance over time was evaluated using general linear mixed models with a correction for hospital clusters. Analyses were performed for the whole sample then stratified for designation level (levels I and II versus levels III and IV) and patient age (

Results: Risk-adjusted mortality decreased from 13.6% to 10.1% between 2009 and 2012 and mean LOS decreased from 14 days to 11 days (trend-p



Conclusion: The results of this study suggest that there have been important improvements in patient mortality and resource use in the integrated trauma system of Québec over the last decade. Results also suggest that efforts should be made to reduce in-hospital complications and unplanned readmissions. Future research should assess whether improvements have occurred in longer-term outcomes such as functional capacity and quality of life.

Poster # 102

THE GERIATRIC TRAUMA TEAM: A NOVEL APPROACH TO CARING FOR ELDERLY TRAUMA PATIENTS

Mary F. Stuever DO, Kimberly Hendershot MD, Priti Parikh Ph.D., Andrew Russeau
Mary McCarthy* MD, Akpofure P. Ekeh* MD, Wright State University

The Geriatric Trauma Team: A novel approach to caring for elderly trauma patients.

Introduction: Nationwide, geriatric trauma has progressively increased over the last two decades with mechanisms like falls becoming a dominant mechanism of injury. This population often has comorbidities requiring complex concurrent medical management. In view of this rising proportion of elderly trauma patients, in January 2012, at our Level I Trauma Center, we instituted a separate Geriatric Trauma Team – led by a Geriatrician with trauma mid-level practitioners, dedicated to caring for elderly patients with single system injury, who after initial evaluation by the Trauma Team did not require Intensive Care Unit (ICU) management. We studied patients on this new exclusive "team", comparing their outcomes with those patients under the prior traditional model.

Methods: At our ACS verified Level I Trauma Center, trauma registry data of patients admitted to the Geriatric Trauma Team single system injury, age ≥55, not requiring ICU) was collected from the period between January 1- December 31 st, 2012. Data was also collected for the preceding 12-month period (Jan-Dec 2011) – the Pre-Geriatric Team Period. Length of stay (LOS), re-admission rates, Injury Severity Score (ISS), mechanism of injury, injury type, complications and mortality data were recorded. Comparisons were performed for readmissions and LOS based on ISS groupings (0 -9, 10 -15, 16 -24 & ≥ 25). Independent t-test and chi-squared testing were used for continuous and discrete variable comparisons with 0.05 considered statistical significance.

Results: A total of 310 patients were admitted to the Geriatric Team in the 12-month period studied vs. 906 patients ≥ 55 yrs. in the preceding 12 months. Falls and Motor Vehicle crashes were the most common mechanism of injury in both groups. Subdural hematomas, intra-cerebral hemorrhage and spine fractures were the most common injuries in both periods. Mean ISS was expectedly overall higher in the Pre-Geriatric group that consisted of all elderly patients in that 12 month period (12.3 vs. 10.4, $p < 0.0005$) Analyzing four individual ISS groupings stated above, there were no statistically significant differences in the LOS and readmission rates comparing the Geriatric team to the Pre-Geriatric Team period. There was no in hospital mortality on the Geriatric Team during the 12-month period studied.

Conclusion: A team led by a geriatrician exclusively caring for elderly patients with single system injuries after clearance by primary Trauma Team's assessment is equally as effective as the traditional trauma team model with regard to LOS and re-admissions. The ability to specifically focus on the complex medical and social issues potentially proffers an additionally advantage to this population. In the current climate of Trauma personnel shortages and resident hour restrictions at large Trauma Centers, this model and adaptations to it, could present viable options for caring for the increasing ranks of elderly injured patient

Poster # 103

A NOVEL MODEL TO PREDICT ADMISSION VOLUME AT A LEVEL I TRAUMA CENTER

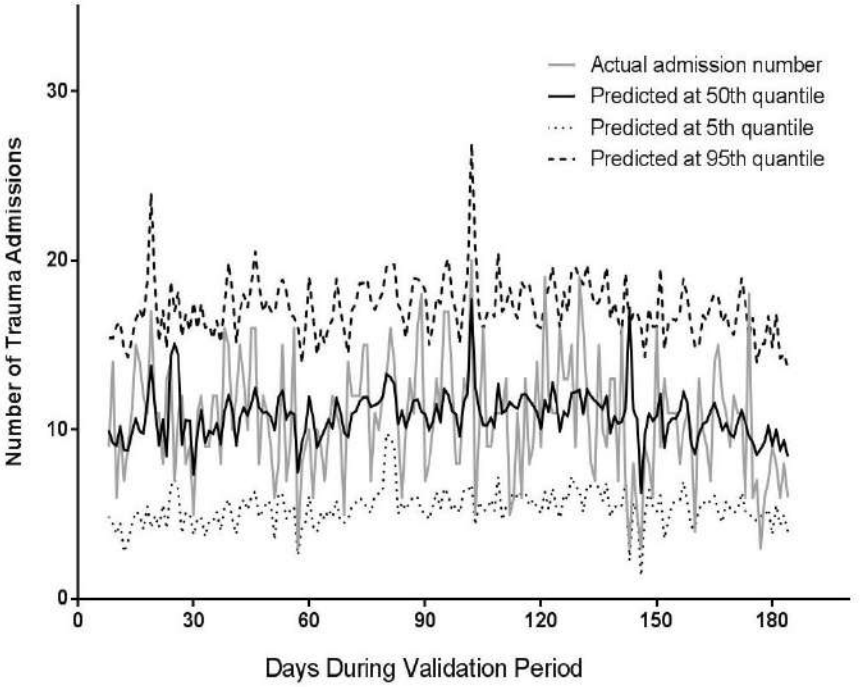
Daniel J. Weber MD, John R. Woods Ph.D., James W. Hardin Ph.D., R. L. Reed* II, MD, Indiana University Department Of Surgery

INTRODUCTION: It is widely believed that multiple factors influence trauma volume including seasonal and weather variations as well as other social and environmental elements. We hypothesized that such factors could be used to create a prediction tool to model trauma volume and injury patterns, making it possible to more accurately predict the number and type of trauma resources that will be necessary on any given shift.

METHODS: A retrospective review of the trauma registry of a level I trauma center from 1/1/2009 to 4/30/2013 was performed, correlating activation and admission data with system factors, local events, and weather data. A prediction model was created using simultaneous quantile regression for the 5th, 25th, 50th, 75th, and 95th quantiles of daily trauma admission volume. This model was then validated on a new data series over a 6-month period from 5/1/2013 through 10/31/2013 where predicted and actual numbers of trauma admissions were compared.

RESULTS: There were 15,873 trauma admissions in the initial series on which the prediction model was developed and 1,940 admissions in the test series on which the model was validated. Factoring in both groups, the number of daily trauma admissions ranged from 2 to 33 with a mean of 10.4 admissions per day. After adjusting for the seasonal, monthly, and weekly cyclic patterns in trauma, several additional parameters including daily high temperature, rain, fog, recurring annual events, and the trauma volume one week and one day prior continued to be independently predictive of daily trauma admissions ($p<0.05$). Model validation revealed an average difference between the predicted and actual number of admissions at the 50th quantile of 2.5 admissions per day. Additionally, well over 90% of trauma admissions fell between the model's 5th and 95th quantiles demonstrating validity for this prediction tool (See Figure).

CONCLUSION: We have presented a novel application of quantile regression to predict the number of daily trauma admissions based on several measurable parameters. This model accurately forecasts both the number of daily admissions as well as a valid range of admissions for the 5th and 95th quantiles. Considering a daily admission range from 2 to 33, the fact that our predictions varied from observed values by an average of only 2.5 is indicative that our model provides reasonable estimates to allow for better allocation of resources, including the number and scheduling of trauma surgeons, residents, and physician extenders.



Poster # 104

EFFECTIVENESS OF GROUND AMBULANCE VERSUS HELICOPTER TRANSPORT FOR INTER-HOSPITAL TRANSFERS IN RURAL TRAUMA

Muhammad J. Younus MD, Chris Wargo MSN, James Dove BA, Denise Torres MD, Kenneth Widom MD, DiAnne Leonard* MD, Susan Baro MD, Joseph Blansfield MD, Mohsen M. Shabahang MD, Jeffrey L. Wild MD, Geisinger Health System

Introduction: The clinical benefits of helicopter transport over ground transport for inter-hospital transfers are unclear. This study compares patient outcomes in helicopter emergency medical services (HEMS) and ground emergency medical services (GEMS) in rural trauma inter-hospital transfers (IHTs).

Method: This is a retrospective cohort study of 3308 IHT patients by HEMS or GEMS between January 2006 and December 2012. Outcomes of interest were hospital length of stay (LOS), ICU LOS, pre-hospital times, and mortality. Patients were divided into minor (ISS<15) and major (ISS≥15) trauma. Multiple logistic regression analysis was performed for patients transferred within 30, 30-59, ≥60 miles from trauma a center (TC).

Results: A total of 1257 (38%) patients were transported by helicopter and 2051 (62%) patients were transported by ground. Patients transported by HEMS were more severely injured: average ISS (18.3 vs 13.1 p<.0001), average GCS (12.7 vs 14.4 p<.0001), and SBP <90 (3.6 % vs 1.4% p<.0001). Patients transported by helicopter were significantly likely to be in the traumatic brain injury (TBI) cohort and were more likely to have pre-hospital intubation. Total pre-hospital time and transportation time were significantly shorter in the helicopter group. Helicopter-transported patients were more significantly likely to be directed to the ICU and operating room from the emergency department. There was no difference in mortality in patients with an ISS <15 and on multivariate analysis there was no difference in survival between the two modes of transport. For patients with an ISS ≥15, helicopter-transported patients had higher mortality (11.4% vs 6.0% p <0.0003). However, on multivariate analysis, patients transferred ≥60 miles from the TC had significant survival benefit in the HEMS group.

Conclusion: Helicopter transport is faster than ground transport in rural trauma IHTs. Patients with major trauma (ISS ≥15) transported by helicopter had survival benefit if transferred ≥60 miles from the TC. This highlights the potential benefit of helicopter-transferred patients with major trauma as the distance increases from a TC.

Poster # 105

SAFE AND COST EFFECTIVE METHOD OF DECREASING TRAUMA SYSTEM OVERTRIAGE WITHOUT OVERBURDENING THE TRAUMA SERVICE

Gail T. Tominaga* MD, Imad S. Dandan* MD, Kathryn B. Schaffer MPH, Christine Wells RN, Marc Sedwitz MD, Melanie Gawlik RN, Scripps Memorial Hospital La Jolla

Introduction: Overtriage and under triage is a balancing act for Trauma Systems. With the changing economics of healthcare reimbursement, efficient utilization of resources without increasing outcome measures is optimal. We implemented a process to decrease overtriage without adversely affecting patient (pt) outcome or overburdening the Trauma Service.
Methods: Patients not meeting strict American College of Surgeons (ACS) trauma criteria for trauma team activation in a mature Trauma System were designated as Trauma Resource (TR) and brought to a designated Trauma Center for expedited evaluation in the Emergency Department (ED) with early involvement of a Trauma Surgeon (TS) as needed. All TR pts were expedited in the ED. A board certified ED physician, trauma nurse and ED nurse met the pt on arrival to the ED. The CT technician, respiratory therapist and lab technician were notified on pt's arrival. CT scans for TR pts were expedited. Data over a 7 month period were collected concurrently and analyzed.
Results: 871 pts meeting ACS trauma criteria and 318 TR pts were treated over the study period. Of the 318 TR pts, 5 pts were upgraded to Trauma Activation (TA) status immediately upon arrival to the ED, 52 pts required TS consultation and hospital admission, 40 pts were admitted to a non-trauma service and 221 pts were evaluated in the ED and discharged home.

Data for Trauma Patients by Triage Group

Patient Group	N	MeanAge	% Males	% Falls	Mean ISS	Hospital LOS(days)	Mortality	Time to definitve care (min)
TA	684	47.2±22.2	66.5	35	9.8±8.5	5.2±10	28(4.1%)	132±74
ED -TS consults	187	57±23.8	58.2	56	9.9±6.0	4.2±4.1	2(1.1%)	98±59
TR upgraded to TA	5	50.6±31.4	40.0	40	15.2±7.8	4.4±4.4	0	74±31
TR admitted to TS	52	60.4±23.7*	61.5	52*	9.7±5.3	3.4±1.6	1(1.9%)	79±24 *^
TR admitted to nonTS	40	70.8±21.9	47.5	82.5	na	na	na	na
TR discharged hom	221	47.4±24.7	61.5	44	na	0	na	na

*Time to definitive care = time from TS contact with pt until admission to IR, OR, ICU, step down unit or floor bed. *Statistically significant difference compared to TA group. ^Statistically significant difference compared to trauma consults from ED group.

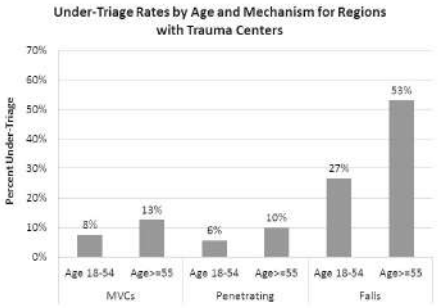
TR pts admitted by the TS were similar to TA pts with regards to gender, mean ISS, hospital LOS and mortality; however, the TR admitted pts were significantly older than TA pts ($p<0.0001$), had more falls as a mechanism of injury ($p=0.0170$) and had a shorter time to definitive care ($p=0.0001$). TR pts admitted by the TS were similar to trauma consult pts from the ED with regards to age, gender, fall mechanism, ISS, LOS, and mortality but had significantly shorter time to definitive care compared to trauma consults from the ED ($p=0.0274$).
Utilization of resources were significantly less in the TR pts compared to TA pts. Charges were significantly different. ED charges for the 52 TR pts admitted to the TS was $253,708 vs $1,168,270 if they had all been trauma activations. There were no ED deaths. The one TR death was a nonagenarian who was made comfort care the following day and expired.
Conclusion: Designating patients as TR pts prehospital with expedited evaluation in the ED with early TS consultation resulted in the utilization of fewer resources, lower hospital charge without increase in hospital LOS, time to definitive care or mortality.

Poster # 106

Parallel Universes for Statewide Trauma Triage: Variability of Trauma System Performance Based on Mechanism of Injury and Age

Kristan L. Staudenmayer* MD, MS, Feng Lin MS, Thomas Weiser MD,MPH, Paul Maggio* MBA,MD, Mackersie C. Robert* MD, David Spain* MD, Renee Hsia MD, Stanford University

Introduction: Trauma system performance is often determined for aggregated populations, which makes it difficult to identify opportunities for improvement. We hypothesized that under-triage is a function of age and mechanism of injury, when controlling for regions with similar access to trauma care.
Methods: We conducted a retrospective analysis of all hospital visits in California using the Office of Statewide Health Planning and Development Database over a 5 year period. All hospital admissions and emergency department visits associated with injury were longitudinally linked. We included patients who were severely-injured patients as defined by an injury severity score (ISS) > 15. Regions were categorized by "Local Emergency Medical Services Agencies" (LEMSAs) with and without trauma centers. The primary outcome was the rate of under-triage,defined by admission to a non-trauma hospital.
Results: A total of 60,182 severely injured patients were included in the analysis. Triage patterns depended on mechanism of injury and age when trauma center access was held constant. In regions with trauma center access, under-triage was low for motor vehicle collisions (MVCs) and penetrating injuries (10-16%), but high for fall injuries in both younger and older adults (27% for 18-54 years and 53% for ≥55 years; Figure). Under-triage was higher in regions without trauma center access, and remained the highest for fall injuries (57% for 18-54 years vs. 78% for ≥55 years). Overall, age ≥55 was associated with a 25-30% increase in under-triage rates, due largely to greater disparities in fall under-triage. Stratifying by access, age, and mechanism identified specific regions that were outliers for high rates of under-triage.
Conclusion: Triage patterns are primarily determined by access to care, mechanism of injury, and age. Fall injuries in all age groups were associated with high rates of under-triage. This suggests that triage guidelines should be refined to better identify severely injured fall patients. Furthermore, stratification of triage patterns by these characteristics allowed the identification of specific regions that were outliers, allowing for targeted education and interventions to improve under-triage.



Poster # 107

EQUAL OUTCOMES FOR ALL: PROPORTION OF DIFFERENT TYPE OR MECHANISM OF INJURY SEEN AT LEVEL 1 TRUMA CENTERS DOES NOT IMPACT SURVIVAL

Zain G. Hashmi MBBS, Syed Nabeel Zafar MD,MPH, Adil A. Shah MBBS, Eric B. Schneider Ph.D., William R. Leeper MD, FRCSC, Elliott R. Haut* MD, Edward E. Cornwell* III, MD, Adil H. Haider* MD,MPH, Johns Hopkins School of Medicine

Introduction: Trauma centers (TCs) differ in the proportions of specific injuries treated. Although there is some evidence suggesting that absolute TC volume does not affect patient outcomes, it is unclear if TCs treating a higher proportion of specific injuries perform better. The objective of this study was to determine if patients treated at level I trauma centers that routinely care for higher proportions of specific type and mechanism of injuries have improved survival.

Methods:Data from National Trauma Data Bank (NTDB) 2007-2011 was analyzed. Patients ≥16 years of age, with blunt/penetrating injuries and an Injury Severity Score (ISS) ≥9 admitted to level I TCs were included. Proportions of patients with specific injuries (penetrating, traumatic brain, spinal cord, thoracic, abdominal and pelvic injuries) treated at each TC were calculated and were used to classify TCs into proportional quintiles for these injuries. To determine if higher proportional quintiles were associated with improved survival, separate multiple logistic regression analyses for each injury were performed, adjusting for known predictors of trauma mortality (age, gender, injury type, pulse on admission, presence of hypotension on admission, total Glasgow Coma Scale, total ISS) as well as overall hospital mortality performance status (high performing, average or low performing).

Results:A total of 172 centers, with 720,563 patients, from an available 223 level I TCs in the NTDB were included. The overall unadjusted mortality rate was 6.7%. On average, a two-fold variation was noted between the lowest and highest proportional quintiles for all injury cohorts. On unadjusted analyses, higher proportional quintiles predicted improved survival for penetrating and traumatic brain injuries versus the lowest quintile. However, this relationship was mitigated after adjusting for patient factors and overall hospital performance (Table).

Conclusion:While level I TCs differ substantially in the proportions of types and mechanisms of injuries treated, these differences do not affect patient survival. Structural factors other than proportions of specific injuries need to be explored for their contribution to overall TC performance.

	Adjusted Odds of Mortality by Quintiles of Proportions of Specific Injuries Treated, OR (95% CI)				
	1 "Lowest Quintile"	2	3	4	5 "Highest Quintile"
Penetrating Injury *n=75,509*	ref	1.20 (0.98-1.48)	1.03 (0.80-1.33)	1.15 (0.96-1.39)	1.26 (1.01-1.57)
Traumatic Brain Injury *n=332,178*	ref	1.04 (0.94-1.16)	1.06 (0.96-1.17)	1.01 (0.90-1.12)	1.04 (0.94-1.15)
Spinal Cord Injury *n=26,883*	ref	0.83 (0.63-1.10)	0.98 (0.76-1.27)	0.93 (0.72-1.22)	0.75 (0.59-0.95)
Thoracic Injury *n=269,123*	ref	1.01 (0.88-1.16)	1.00 (0.86-1.16)	0.99 (0.86-1.14)	0.90 (0.78-1.04)
Abdominal Injury *n=115,332*	ref	0.92 (0.80-1.06)	0.93 (0.81-1.08)	0.93 (0.80-1.08)	0.96 (0.84-1.09)
Pelvic Injury *n=68,895*	ref	0.97 (0.85-1.10)	0.98 (0.85-1.14)	0.93 (0.80-1.08)	0.93 (0.80-1.08)

Poster # 108

LONG DISTANCE MEDICAL EVACUATION FOR TRAUMA: DOES THE "GOLDEN HOUR" APPLY ON THE FRONTIER?

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Introduction: The state of Alaska is set over enormous distances, and is dotted at great intervals with small settlements and villages.. Medical resources have tended to concentrate in the larger towns and cities, and most specialty care in Alaska is delivered in the largest city, Anchorage. The population of the state, therefore, relies on a fairly robust medical evacuation system composed of both fixed wing and rotary aircraft, and medical evacuation distances of up to 1500 miles. This retrospective review evaluates the effect of distance of medical evacuation on outcomes of care for high acuity trauma patients by comparing urban (Anchorage city) high-acuity victims to those with transport distances of greater than 20 miles.

Methods: The trauma database of a single tertiary medical facility was searched for high acuity ("Status 1") trauma patients over the calendar three-year period 2011-2013. The data were examined for geographical location of injury and the distance calculated between injury site and the hospital address. The patients were then divided into two groups –local/urban and remote. These two groups were then compared for demographic, injury severity, mechanism of injury, and survival.

Results: During the three year period 2011-2013, there were 151 patients who entered the trauma facility as a "status 1" trauma. There were 119 males and 32 females. The average age of the group was 35.3 years. There were 74 local/urban patients (transport < 20 miles) and 77 remote (transport > 20 miles). The remote group was transported an average of 83.2 miles (range 20-795 miles). Nineteen of the 77 remote transports were for over 100 miles, and nine were over 200 miles. The transport distances of two of the transports could not be accurately determined as they occurred on ships in the Bering Sea. Transport distance was calculated from first landfall in the Aleutian Islands, where the patients were recovered by fixed wing aircraft. There were several instances of mixed mode transport, including National Guard para-jumper rescue. There were no statistically significant differences between groups for age, gender, mortality, ED mortality (<12 hours), or ISS score. The urban group had a significantly higher proportion of penetrating trauma than the remote group ($p=0.0003$). In the local group there was no difference between genders in mechanism of injury. A trend noted in dispositions from ED, with a greater portion of urban going from ED to OR, and a greater portion of remote patients going from ED to ICU. ($p=0.10$) The length of stay after trauma admission was also significantly longer in the remote group (14 days) than in the urban group (avg. 10 days).

Conclusion: The results were surprising in a number of ways. The authors had anticipated that significant differences in transport time would have resulted in differences in patient findings, either for better or for worse. It was felt that a process of natural selection would have occurred outside of the "golden hour.". Instead, the significant differences were in mechanism of injury and then, reflective of proportional differences in mechanism between the two groups, more urban patients went to the operating room, and more remote patients went to the ICU for initial management. In the local trauma group, there were no gender differences in mechanism of injury Further work is indicated using state transport data to determine whether selection bias and overall death rates for trauma differ between urban and remote populations.

Poster # 109

PREHOSPITAL TOURNIQUET USE AT THE BOSTON MARATHON BOMBINGS: FAILURE TO TRANSLATE

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Introduction:The Boston Marathon bombing was the first major US terrorist event with multiple, severe extremity injuries. This is a common scenario on the battlefield, but uncommon in the homeland. A decade of warfare demonstrates that improvised tourniquets are rarely effective, and that the ubiquitous availability and aggressive use of commercially-available, purpose-made tourniquets has dramatically reduced deaths from extremity exsanguination. In Boston, civilian first responders, including trained professionals and bystanders, rushed to aid the injured. Bleeding limbs had tourniquets applied, but the type and effectiveness of those tourniquets remains unknown.

Methods:A database was created and populated by all the Boston Level I trauma centers following the Boston Marathon bombings. Data regarding specific injuries, limbs affected, demographics, prehospital interventions (including tourniquet types), and outcomes were extracted.

Results:Of 243 injured, 152 patients presented to the ED within 24 hours. There were 66 patients (63.6% female) suffering at least one extremity injury (age 15 to 71 years, ISS median 10 [range 1-38], AIS Extremity median 3 [range 1-4]). Of these 66 patients, 4 had upper limbs affected, 56 lower limbs only, and 6 had combined upper and lower limb injuries. There were 17 lower extremity traumatic amputations in 15 patients. Additionally, there were 10 patients with 12 lower extremities suffering major vascular injuries. Of the 66 patients with limb injuries, 29 patients had recognized extremity exsanguination at the scene. In total, 27 tourniquets were applied: 16 of 17 traumatic amputations, 5 of 12 lower extremities with major vascular injuries, and 6 additional limbs with major soft tissue injury. Eight limbs with severe bleeding had no tourniquet applied. All tourniquets were improvised and no commercially-available/purpose-made tourniquets were identified. Among these 66 patients mortality was 0%.

Conclusion:After the Boston Marathon bombing, extremity exsanguination was either left un-treated or treated with an improvised tourniquet in the prehospital environment. Effective, purpose-made tourniquets (and adequate training) should be made widely available to civilian prehospital responders. The Boston Marathon bombing response represents a failure to translate a valuable lesson learned from war.

Poster # 110

TRAUMA TRANSFERS TO LEVEL 1 CENTERS: MEDICAL NECESSITY OR FISCAL CONVENIENCE?

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Introduction: Level 1 trauma centers are obligated to accept the transfer of trauma victims when a higher level of care is indicated. Based on the forthcoming healthcare access changes through the Affordable Care Act, we hypothesized that transferring institutions subconsciously select trauma transfer cases based on payer status or cost of treatment and injury complexity ratio, and hence transfer patients when perhaps medically unnecessary.

Methods: Our single institution trauma registry was queried to identify trauma transfers and primary trauma patients from December 2002 to May 2013. Demographics, payer status, and injury severity score (ISS) were analyzed to examine any trends associated with transferred patients.

Results: During the study period 6,044 trauma victims were transferred (TTP) to our rural ACS Level 1 trauma center, compared to 11,775 primary trauma patients (PTP). Uninsured patients made up 10% (N=630) of TTP compared with 15% (N=1,804) of PTP. Medicaid recipients comprised 13% (N=630) of TTP compared with 11% (N=1,309) of PTP. Surprisingly 53% (N=3,207) of TTP were Medicare (N=1,326) or HMO (N=1,881) insured, versus 41% of PTP (Medicare=1,528, HMO=3,346) being insured. The discharging services were predominantly subspecialist surgeons (i.e.: General Adult Trauma and Pediatric Trauma comprised <50% of discharges) for all trauma patients. Adult and Pediatric Trauma services accounted for 30% (N=1,781) of TTP versus 45% (N=5,343) of PTP discharges. Mean ISS of TTP was 11.5±0.11 compared with PTP of 11.6±0.10.

Conclusion: Contrary to expectations, these data suggest over half the patients who were transferred to our facility for higher level of trauma care were insured — in fact there were fewer insured patients among our primary trauma population. The notion that trauma transfers increase institutional fiscal burden is unsubstantiated. Transfer of trauma patients should continue based on medical necessity given the availability of subspecialty surgeons.

Poster # 111

TRAUMA PREVENTION TASKFORCE DECREASES FALL ADMISSIONS AT A LEVEL II TRAUMA CENTER

Tracy Evans MD, Katelyn J. Rittenhouse BS, Jo Ann Miller RN, BSN, Ronald Baier BS, EMTP, Roxanne Chandler RHIA, CTR, CSTR, Frederick B. Rogers* MD, MS Lancaster General Hospital

BACKGROUND: Geriatric falls cost the U.S. healthcare system approximately $30 billion in medical costs each year. In 2012, more than 2 million adults 65 and older were treated in emergency departments for falls, 1 million of which required hospital admission. As the paradigm of healthcare in the United States is rapidly changing with the implementation of the Patient Protection and Affordable Care Act, there is a renewed and strong emphasis being placed on preventative medicine. Our trauma team hypothesized that a community outreach program designed to better educate the geriatric population about fall prevention would reduce trauma admissions due to falls and consequently decrease medical costs for the geriatric population.

METHODS: In 2011, a Trauma Prevention Taskforce consisting of a nurse, a trauma surgeon, and a trauma prevention coordinator visited 5 senior living facilities in Lancaster County, PA and presented a protocol to identify patients at high risk for falls as well as helped staff members identify steps to reduce falls. To determine the impact of this intervention, the registry of Lancaster County's only Level II trauma center was queried for all geriatric (age≥65) trauma admissions. Admissions due to falls of patients living at a Lancaster senior living facility with ≥3 falls trauma admissions between 2010 and 2013 and total number of beds data provided by the PA Department of Health were included in our analysis. The fall rates (total falls/total beds) of 2010-2011 were compared to 2012-2013 at the intervention facilities and the control facilities in Lancaster County that were not visited by the Trauma Prevention Taskforce.

RESULTS: There were a total of 23 (5 intervention, 23 control) nursing homes meeting study inclusion criteria. Between 2010 and 2013, there were a total of 2,196 geriatric trauma admissions attributed to falls; 487 (22.2%) of these admissions were attributed to patients living at one of the study nursing facilities. The fall rate was found to have decreased at intervention facilities from 8.9% from 2010-2011 to 8.1% from 2012-2013 ($p<0.001$), and the fall rate was found to have increased at control sites from 5.9% from 2010-2011 to 7.7% from 2012-2013 ($p=0.018$).

DISCUSSION: We have associated a statistically significant decrease in fall admissions from senior living facilities with a visit by a Trauma Prevention Taskforce. Trauma centers should pursue community outreach initiatives as they can significantly benefit public health and decrease unnecessary injury and healthcare costs.

Poster # 112

RELIABILITY OF GGT, MCV, AND BLOOD ALCOHOL LEVEL AS A NEGATIVE PREDICTOR OF CHRONIC ALCOHOL MISUSE IN TRAUMA PATIENTS

Donald N. Reed* Jr., MD, Kevin O'Connor BS, Vickie L. Meyer RN, Annette M. Chard RN, Joel F. Nagel RN, Ronald J. Jones Pharm.D, Gordon H. Bokhart Pharm.D, Katherine E. Kruse MD, William A. Kunkle DO, Lutheran Hospital of Indiana

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction:Alcohol screening is required for verified trauma centers. Verbal questionnaires have been shown to reliably screen for chronic alcohol misuse, but they take time and personnel. Biomarkers of excess alcohol use were considered long ago to have utility for this, but data is lacking. A prospective study was performed to compare several biomarkers with trauma patients' answers to their AUDIT questionnaire results.

Methods:After institutional review board (IRB) approval, consenting adult trauma patients admitted to a level II trauma center from 2010 to 2013 were evaluated for study enrollment. Standard trauma serum laboratory tests gamma-glutamyltransferase (GGT), mean corpuscular volume (MCV), and blood alcohol level were drawn and recorded as positive if elevated (BAL >/=100mg/dl) or negative. With patient consent a blinded trained study operator administered the Alcohol Use Disorders Identification Test (AUDIT) questionnaire. Using AUDIT scores as a “gold standard” for alcohol misuse potential, biomarker results were compared to AUDIT results. Results were evaluated statistically (SPSS and statistics in R) for Cohen’s Kappa coefficient for agreement, and sensitivity, specificity, predictability.

Results:Out of 179 enrolled subjects, 113 were evaluable. The resulting Kappa was 0.30, (95% Confidence Interval 0.118-0.481) suggested “fair agreement” when all 3 markers were compared to AUDIT. Sensitivity and specificity were found to be 0.605 and 0.70 with 74% of negatives truly negative when all markers were negative and a NPV of 0.941 was assessed when an accepted 10% prevalence was applied. Interestingly, if ANY positive ethanol level was applied as positive the result was Kappa 0.374, resulting in sensitivity and specificity of 0.721 & 0.671, respectively with 80% of negatives being true negatives via markers and giving a NPV of 0.956.

Conclusion:While AUDIT is an established tool for alcohol misuse evaluation, it is time consuming and not useful for those with consciousness issues; therefore these biomarkers may have utility. Although as an overall predictor of misuse these biomarkers fall somewhat short, their utility as a possible tool to identify those without ethanol issues is suggested by these results. Our findings demonstrate the potential these three serum tests have to limit the need for further evaluation of biomarker-negative patients and suggest this combination may be a valid preliminary screening mechanism for trauma patients -both conscious and not- in understaffed and resource-limited hospitals.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

Poster # 113

OLDER ADULT PEDESTRIANS NAVIGATING NEW YORK CITY - AGE-ASSOCIATED LIMITATIONS

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INTRODUCTION - In New York City (NYC) older adults (≥65 yrs) comprise 12% of the population but account for 36% of pedestrian fatalities. NYC can be convenient for older adults to access necessary resources, but the busy streets can pose many hazards. We sought to better understand risk factors associated with older adults who are struck by motor vehicles (MV) while walking in NYC.

METHODS - Data were prospectively collected on all pedestrians injured by MV who presented to a NYC level 1 trauma center from 2008 to 2011. Demographics, patient behavior and scene variables were obtained from patient and EMS interviews.

RESULTS - Of the 1,471 patients enrolled, 127 older adults and 803 adult pedestrians (18-64 yrs). Fifty percent of older adults had an ISS>9 and 60.6% required hospital admission. Older adults more frequently had serious injury (49.6% vs 21.3%) and TBI (20.5% vs 5.7%; p=0.0001) compared with younger adults. Older adults were more likely to have unstable gait (11.0% vs 1.1%), hearing (33.0% vs 3.6%) and vision impairment (24.4% vs 5.6%) (p<0.0001). Older adults were less likely to be intoxicated (2.4% vs 17.2%), or distracted by a cell phone (0.8% vs 4.4%) or music (0% vs. 4.4%) (p<0.01). Older adults were less likely to be crossing midblock (13.4% vs 18.9%) or against the signal (6.3% vs 8.7%) (p=0.05). Older adults were more likely to be hit by a vehicle making a turn (41.7% vs 36.0%; p=0.01) and be hit in their home neighborhood (40.2% vs 12.75%; p<0.0001). Multivariate analysis showed that older adult collisions occurring during rush hour and involving pedestrians in the crosswalk or sidewalk were associated with decreased risk of severe injury, although not significant (Table 1).

Table 1. Factors Associated with Severe Injury (ISS>9) in Older Adults

	Unadjusted OR (95%CI)	Adjusted OR (95% CI)
Unstable gait	0.89 (0.3-2.64)	2.05 (0.58-7.23)
Two-way traffic	1.28 (0.57-2.87)	1.11 (0.37-3.34)
Rush hour	0.78 (0.37-1.7)	0.74 (0.28-1.98)
SUV/truck/van	2.11 (0.97-4.59)	2.34 (0.93-5.9)
Bus	0.42 (0.08-2.20)	
Road surface - wet/ice	1.54 (0.64-3.7)	3.15 (0.96-10.3)
Vehicle turning	1.00 (1.00-1.00)	1.00 (1.00-1.00)
Pedestrian in crosswalk/sidewalk	1.34 (0.54-3.32)	0.88 (0.31-2.46)
Distraction (music/cell phone)	0.33 (0.03-3.27)	0.75 (0.10-5.69)

CONCLUSION - Older adults are less likely to engage in risky behavior as they navigate the city, and physical impairments associated with aging may contribute to pedestrian collisions. Future multi-site studies should focus on better defining the extent to which age-related disabilities put older adults at risk for pedestrian injuries and how best to mitigate this risk.

Poster # 114

IT'S NOT ONLY THE STRONG THAT SURVIVE: HOW RESILIENCE EFFECTS TRAJECTORIES OF RECOVERY AFTER INJURY

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Introduction: Chronic, non-healing wounds don't just occur to the flesh. Often, the psychological consequences after injury persist long after physical healing has occurred. These psychological and behavioral incapacities can have significant influence on functional well-being and quality of life. In order to efficiently utilize resources, determining the trajectory of recovery for patients during inpatient hospitalization, as well as factors that may predict those trajectories, may prove beneficial in determining individuals at risk for poorer quality of life outcomes after injury. Four prototypical classes of adjustment (i.e., resilient, chronic, delayed, and recovery) as described by Bonanno (2004) were reproduced.

Methods:This prospective study consisted of 406 subjects who completed assessments while hospitalized, and at three, six, and twelve months post-discharge. The Patient Health Questionnaire-8 (PHQ-8) and the Primary Care Posttraumatic Stress Disorder Screen (PC-PTSD) were used to identify the latent classes (e.g., resilient, chronic, delayed, or recovery). Demographic information, presence of mild traumatic brain injury (mTBI), mental health, pain levels, and Injury Severity Score (ISS) were explored as covariates. Following the process model of resilience, we analysed data from participants over the course of up to a year following treatment at the trauma center and identified four distinct groups that had four different trajectories of adjustment over the year. Data was analyzed using latent growth mixture modelling.

Results: The sample was predominantly male (62.5%) and European-American (65.3%). A four-class model was the best fit for the data. Fifty-seven percent of subjects had stable low levels of depression and 44% had stable low levels of PTSD following injury (i.e., resilient). The three other groups' depression and PTSD analyses reflected high chronic levels of distress (10.!% depression, 24.7% PTSD), distress increasing over time (18% depressed, 11.7% PTSD) or decreasing levels of distress that resolved (15% depressed, 19.1% PTSD). Each non-resilient class represented a minority response type when compared to the resilient class. The inclusion of covariates improved model fit (e.g., gender, income) and the pre-existing mental health variable was a significant predictor of the resilient class.

Conclusion:The majority of survivors following a traumatic event experienced minor and transient symptoms of depression and PTSD, suggesting the most common adjustment pathway after injury was resilience. While encouraging that the majority of trauma patients are resilient, the quarter of patients who showed chronic PTSD and 10% with depression, as well as the 18% of patients who became increasingly depressed over time and the 11.7% who also eventually developed PTSD remain concerning. Demographic factors including race and education level did not predict trajectories of adjustment. Of note, no injury related variable, including etiology of injury, ISS, or mTBI predicted adjustment trajectory. However, patients without a pre-injury mental health diagnosis were significantly more likely to be resilient after injury, This study increases our understanding of psychological adjustment post injury and highlights that severity of injury as well as other injury factors do not predict psychological response.

Poster # 115

ONE-YEAR EVALUATION OF THIRD PARTY VIOLENCE INTERVENTION PROGRAM IN A PUBLIC HOSPITAL

John C. Kubasiak MD, Reza Salabat MD, Andy Wheeler MS, Elizabeth Gwinn MD, Frederic Starr MD, Andrew Dennis DO, Dorion Wiley MD, Kimberly Nagy* MD, Faran Bokhari MBA,MD, Kimberly Joseph* MD, Cook County Hospital

Introduction: Injury prevention is a key component of the modern trauma center; patients who are victims of intentional injury have a 5 year re-injury rate as high as 45%. Recent attempts to decrease this rate have lead to the creation of hospital-based violence intervention programs (VIP). Our publicly-funded level 1 trauma center has been a member of a city wide VIP for 12 months. We sought to evaluate program effectiveness over this period and to investigate areas for improvement.

Methods: Over a 12 month period, patients who were seen at our publicly-funded level 1 trauma center and who were victims of intentional injury were offered enrollment with a third party violence intervention program (VIP) partner during their visit. Our hospital differs from other hospitals participating in the program in that we require written consent from the patient for a referral. The trauma center also had two Masters-candidate Social Work interns on service during that period who interfaced with several of the referred patients. After 12 months of the partnership a phone survey of referred patients was conducted to evaluate program performance. Our primary outcome was successful enrollment with the VIP as evidenced by more than one outpatient follow-up. Secondary outcome measures included patients' subjective feelings regarding the VIP and identification of needs addressed.

Results: A total of 205 patients consented and were referred who were identified as high-risk victims of intentional injury over the 12 month period, with 28 responses (n=22 or 79% GSW, n=2 or 7% SW, n=4 or 14% Blunt trauma). Of those contacted 35% (n=10) had no memory of referral to the VIP and were not contacted once discharged from hospital by the VIP; 9 requested another referral to VIP program. Those with any post-discharge contact by the VIP reported overall adequate follow up and that their needs were addressed. Additionally 32% (n=9) with follow up were "strong supporters"; 42% (n=12) recommended services to similar patients. Patients with no recall saw trauma SW 10% of time (n=1); patients considered strong supporters had trauma SW 55% of time (n=9)

Conclusion: Our primary endpoint demonstrates low enrollment rates in third party VIP program; the need for written consent may have had a significant effect on this outcome. Contact with the hospital-based Social Work interns was highly correlated with successful post-discharge follow up by the VIP, suggesting the benefit of permanently placed in-house personnel to facilitate enrollment. Secondary outcomes were positive in those who successfully enrolled with the VIP. Our data also suggests a role for scheduled follow-up with those patients who are referred but do not have post-discharge contact with the VIP, as the majority of them requested a new referral. Future areas of investigation: survey of patients who declined referral to determine reasons for the initial refusal as well as the willingness to accept a new referral.

Poster # 116

RISKY MOTORCYCLIST BEHAVIOR CORRELATES WITH SMALL MOTORCYCLE (MC) ENGINE SIZE (ES)

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Introduction: The United States has seen an increase in the popularity of MC with larger engines, concurrent with increasing motorcyclist fatalities and injuries. We performed an analysis of patients admitted after a MC crash to evaluate rider behavior by ES. We hypothesized that riders with larger ES would be more likely to exhibit risky behavior.

Methods: We performed a retrospective analysis of adult inpatients after MC accident at a level 1 trauma center from April 2002-March 2007. Demographics, helmet type, helmet fastening, MC licensure, and MC insurance were collected from charts or prospective patient interviews. Risky behavior was determined to be failure to wear a fastened full-face helmet, or unlicensed or uninsured MC operation. ES were categorized as: small (<500 cc), medium (500-850 cc), large (>850 cc), or unknown. Data were analyzed using chi-square and Fisher's exact tests.

Results: 190 inpatients were identified; of these, MC-specific data were obtained for 115 (60.5%). Mean age was 28.3 (SD±8.6) years; 93% were male. 33 (28%) motorcyclists had large engines, 57 had medium (50%), 14 had small (12%), and 11 were unknown (10%). Helmet use and type of helmet used varied by ES (Table). 50% of small engine motorcyclists had fastened helmets versus 85% for riders of large engines, p=0.02. 86% of small ES motorcyclists were unlicensed, whereas 21% of riders with large ES were riding without a license, p<0.001. Large ES riders were more likely to have insurance (82%) compared to small ES riders (7%), p=<0.001.

Conclusion: Within our population, MC riders with larger ES were more likely to mitigate risk by wearing full-faced helmets, fastened helmets, and obtaining proper licensure and insurance than MC riders with small engines. Small MC ES was significantly associated with risky behavior.

Table: Motorcycle Rider Behavior by Engine Size

Factor	Small ES, n=14	Medium ES, n=57	Large ES, n=33	Unknown ES, n=11	p-value
Any Helmet	9 (64%)	53 (93%)	32 (97%)	8 (73%)	0.002
Full-Face Helmet	3 (21%)	38 (67%)	20 (61%)	3 (27%)	0.004
Fastened Helmet	7 (50%)	47 (82%)	28 (85%)	6 (55%)	0.02
License	2 (14%)	42 (74%)	28 (85%)	1 (9%)	<0.001
Insurance	1 (7%)	37 (65%)	27 (82%)	0 (0%)	<0.001

Poster # 117

VISIBLE...IMPACT: RELATIONSHIP BETWEEN COLOR OF VEHICLE AND PROPORTION OF CRASHES BY TIME OF DAY

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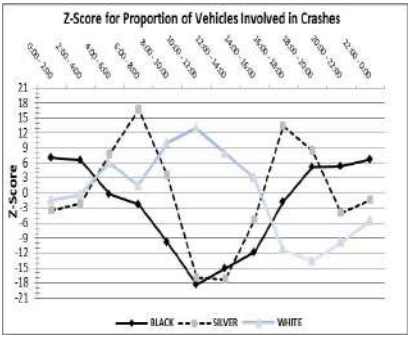
Introduction: Motor vehicle crashes are the leading cause of traumatic injury in the USA, and account for 46% of the total volume of the Arizona State Trauma Registry between 2009-12. Some research has shown that vehicle color may have an impact on the likelihood of crashing, due to the visibility properties of the color, however the published findings are few and inconsistent. We hypothesize that the visibility properties of various vehicle colors are impacting the likelihood of crashes and identify which colors exhibit susceptibility at which times of day.

Methods: The Arizona Safety Data Mart (2009-13) was used to calculate the proportion of vehicles involved in crashes which correspond to each color type and time of day. This dataset is derived from the compiled incident reports produced by responding law enforcement. The proportion of vehicles involved in crashes within each two hour bin was used to normalize for variations in the crash volume caused mainly by changes in vehicle miles traveled by time of day. Variations in that proportion were examined across times of the day to find relationships between visibility and crashes. A statistical test was performed to determine the significance of the variations in proportion relative to the daily average.

Results: There were over one million vehicles involved in crashes during the study period. White was the most common color reported on average (23.65%), followed by black (10.39%), gray (10.36%), aluminum (10.14%), red (8.70%), and blue (8.60%). Crash proportions for certain color types were shown to have a very significant ($p < 0.01$) sensitivity to time of day. The most significant and distinct patterns were found for black, silver, white, and aluminum vehicles. The remaining colors tended to fall into families which exhibited similar time sensitivities to one of those four or else had no statistically significant pattern. Black was highest at night (18:00-4:00) and lowest during the day (8:00-16:00). Silver vehicles peaked at sunrise (4:00-10:00) and sunset (16:00-20:00). White experienced an increase through midday (8:00-16:00) but decreased in the evening (16:00-20:00). Aluminum exhibited an increase from midday through the evening (10:00-0:00), and a decrease during the morning (4:00-8:00). All forms of blue and green exhibited almost no statistically significant variation by time of day.



Conclusions: The results of this analysis lend credence to the hypothesis that vehicle visibility properties interact with ambient lighting conditions to increase susceptibility to crashes. As a matter of public safety care should be taken when consumers select vehicle colors, municipal services pick colors for their vehicle fleets, or manufacturers determine which paint colors to use. Drivers should have an increased awareness of their own visibility and crash risk as a result of vehicle color.

Poster # 118

SIGNIFICANT DIFFERENCES IN OXIDATIVE STRESS BETWEEN HEALHTY CONTROLS AND TBI PATIENTS: A 5-YEAR MULTICENTER PROSPECTIVE OBSERVATIONAL COHORT STUDY

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Introduction: Oxidative stress has been related to the onset or progression of multiple diseases. Significant differences in oxidation-reduction potential (ORP) were previously demonstrated between traumatic brain injury (TBI) patients and non-TBI controls, utilizing technology not suitable for point-of-care use. The objective of this preliminary study was to examine the differences in the point-of-care RedoxSys system ORP measurement between an isolated TBI (iTBI) population and healthy controls.

Methods: Consecutively admitted adult (18 y/o) trauma patients to two Level I Trauma Centers (2008—2012) with an iTBI and ≥5 plasma samples, collected once-daily. Admission samples were taken within 48h, and control samples were obtained from healthy volunteers. Static ORP (sORP, mV) measures the balance between pro and antioxidants, and the capacity ORP (cORP) measures the antioxidant reserves. cORP measures were not normally distributed, thus an inverse transformation was used to achieve normality. Student's T-tests examined differences in the mean ORP values between iTBI and control patients. Higher values of sORP and inverse cORP are indicative of higher oxidative stress. Alpha was set at 0.10.

Results: There were 115 iTBI patients who met inclusion criteria, and 53 healthy controls. Compared to healthy controls, iTBI patients had significantly higher admission sORP (185.2 vs 178.0, p=.08) and inverse cORP values (3.3 vs 2.4, p<.001). Moreover, iTBI patients' maximum ORP values were significantly higher than control samples (Max sORP [Figure 1]: 220.4 vs 178.0, p<.001; Max inverse cORP [Figure 2]: 4.3 vs 2.4, p<.001). Both ORP indices had mean (SD) times from injury to maximum values approximately 5 days after injury (sORP: 5.5d [4.16]; inverse cORP: 5.2d [4.25]).

Conclusion: This preliminary study utilized a novel, point-of-care measurements of oxidative stress and confirmed previous findings observed in the TBI population. These data are consistent with the concept of increased oxidative stress subsequent to TBI. Future investigations are aimed at using ORP values to create an ascorbate-equivalent concentration to aid in clinical decision making.

Figure 1

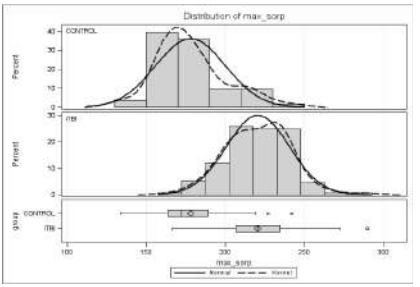
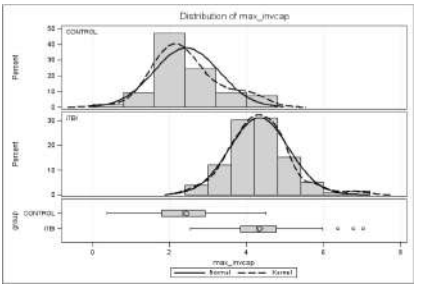


Figure 2



Poster # 119

GERIATRIC NURSING HOME FALLS HAVE INCREASED MORBIDITY AS COMPARED TO THEIR COMMUNITY COUNTERPARTS BUT MORTALITY REMAINS THE SAME

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Introduction: Falls are the leading cause of fatal injury in geriatric patients. Nursing home falls occur at twice the rate of community falls, yet few studies have compared these groups. We hypothesized that nursing home residents admitted for fall would be sicker than their community counterparts on presentation and therefore would have worse outcomes.

Methods: We reviewed 1765 patients, 65 years and older, admitted to our Level 1 trauma center after fall. Demographic data including injury severity score (ISS), admission Glasgow coma scale (GCS), in-hospital complications, length of stay (LOS), operative intervention, and in-hospital mortality was collected. Continuous data was analyzed using Mann Whitney test and categorical data using Fisher exact test. Variables in the univariate tests were analyzed in a multivariate logistic regression.

Results: Table 1 shows comparisons of nursing home and community subgroups. Rates of traumatic brain injury and operative intervention were not significantly different (not shown in Table 1). In a multivariate logistic regression, ISS, GCS and age, but not nursing home status, were significant predictors of in-hospital mortality after fall.

Table 1. (mean +/- SD)	Nursing home n=163	Community n=1545	P value
Age (years)	83 +/- 9	80 +/- 9	0.0002
ISS	9 +/- 7	7+/- 6	NS
GCS	14 +/- 2	15 +/- 2	< 0.0001
Systolic blood pressure	146 +/- 28	155 +/- 28	0.0020
Hemoglobin	11.8 +/- 2.3	12.4 +/- 1.8	< 0.0001
INR	1.3 +/- 0.9	1.2 +/- 0.6	0.0185
LOS (days)	9 +/- 12	8 +/- 12	0.0277
Complications	53 (33%)	313 (20%)	0.0006
Mortality	9 (6%)	55 (4%)	NS

Conclusions: Our study demonstrates that nursing home patients presenting after fall were sicker and had increased morbidity, as evidenced by older age, lower GCS, more anemia, coagulopathy, complications and increased length of stay when compared to their community counterparts. However, mortality was similar between both groups and nursing home residency was not a significant predictor of mortality.

Poster # 120

AN ANALYSIS OF GERIATRIC RECIDIVISM WITHIN AN ACCOUNTABLE CARE ORGANIZATION

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INTRODUCTION: To date, there are almost 500 Accountable Care Organizations (ACOs) across the United States with a strong emphasis on cost-effectiveness of care. Readmission is a huge driver of healthcare cost, and to that end, we sought to determine the factors associated with geriatric trauma readmissions (recidivism) within our institution. We hypothesized that falls and increased age would be significant predictors of geriatric recidivism.

METHODS: All admissions from 2000-2011 attributed to patients age≥65 at our 500 bed Level II trauma center, recently verified by Medicare as an ACO, were queried. Patients were classified as recidivist (RC) or non-recidivist (NRC). The first admissions of recidivist patients were compared to the non-recidivist admissions with respect to gender, age, race, primary insurance, admitting GCS, ISS, hospital LOS, mechanism of injury (MOI), pre-existing conditions, and discharge destination. Factors found to be significant predictors of recidivism in univariate analyses were subsequently incorporated into a multivariate logistic regression model. Additionally, the second admission's MOI was compared to the first admission's MOI, and the proportion of first, second, and third admissions attributed to falls was calculated. A p-value<0.05 was significant.

RESULTS: Between 2000 and 2011, there were a total of 4,963 unique patients admitted to the trauma center age≥65. This population was composed of 287 (5.8%) RCs and 4,676 (94.2%) NRCs. When placed in a multivariate logistic regression, female gender, admitting GCS=15, MOI as fall, history of head trauma, and pre-existing pulmonary disease were identified as significant predictors of recidivism (Table 1). A trend toward increasing proportion of injuries attributed to falls was found with each subsequent trauma admission: 81.5% (234/287) of first admissions, 88.2% (253/287) of second admissions, and 90.5% (19/21) of third admissions.

CONCLUSION: Our study identifies specific factors that should be targeted by social service and prevention resources to inhibit recidivism in the elderly. In the brave new world of ACOs, trauma centers must identify high-risk populations for the consumption of limited resources.

	Recidivism Rate	Adjusted Odds Ratio (95%CI)	p-value
Gender			
Male	3.9%	Reference	
Female	5.8%	1.36 (1.02-1.81)	0.036
Admitting GCS			
GCS 15	5.3%	1.49 (1.03-2.14)	0.034
GCS <15	3.7%	Reference	
MOI			
Fall	5.8%	1.93 (1.36-2.76)	<0.001
Other	2.9%	Reference	
PECs			
Hx Head Trauma	8.3%	1.89 (1.18-3.04)	0.008
Pulmonary Disease	6.8%	1.48 (1.04-2.11)	0.032

Poster # 121

SIRTUIN 1 MEDIATES A PRIMED RESPONSE TO IMMUNE CHALLENGE AFTER TRAUMATIC LUNG INJURY

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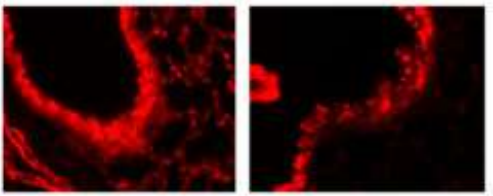
Introduction: Pulmonary contusion (PC) is a common, potentially lethal injury that results in priming for exaggerated inflammatory responses to subsequent immune challenge like infection (2nd hit). The molecular mechanism of priming and the 2nd hit phenomenon after PC remain obscure. Using a mouse model of PC, we have found that Sirtuin 1 (SIRT 1) can regulate the response to immune challenge and hypothesize PC alters SIRT 1 levels and/or activity that in turn, modulates 2nd hit responses.

Methods: Male, 8-9 wk, C57BL/6 mice underwent blunt chest trauma resulting in PC and changes in SIRT 1 were assessed in lung tissue and isolated immune cells at various times after the PC. Injury-primed 2nd hit host responses were tested at 24H after PC by (1) *in vivo* infectious challenge of injured mice or (2) *ex vivo* inflammatory challenge of isolated immune cells from injured mice. SIRT activators or repressors were used to test for SIRT 1 participation in these 2nd hit responses. Data were analyzed using one way ANOVA with Bonferroni multiple comparison post-test with significance defined as $p \leq 0.05$). All experimental protocols were approved by the WFUHS Animal Care and Use Committee.

Results: Immunocytochemistry of the injured lung at 24H showed that PC reduced SIRT 1 levels (Figure). SIRT 1 levels in isolated bronchoalveolar lavage (BAL) cells were quantitated by immunoblot and showed a 50% and 40% decrease in SIRT 1 protein at 3 and 24H after injury, respectively. Injured animals given an infectious challenge by cecal ligation and puncture (CLP) had increased mortality compared injury or infectious challenge alone. To test for SIRT participation in the 2nd hit response to infection, injured animals were treated with a SIRT activator before CLP. Treated mice improved survival to 80%. Isolated BAL cells from injured mice given an *ex vivo* inflammatory challenge with bacterial lipopolysaccharide (LPS) had increased levels of TNF-a mRNA compared to uninjured mice. To test for SIRT participation in the 2 nd hit inflammatory response, BAL cells of injured animals were pre-treated with a SIRT activator before LPS challenge. SIRT activation decreased TNF-a mRNA.



Conclusion: We found that PC decreases SIRT 1 levels in the lung. Host responses to infection or inflammatory stimuli are enhanced in injured mice. Our results suggest that SIRT participates in priming and that increasing SIRT may improve outcomes to the 2 nd hit response after injury.

Poster # 122

NATURAL HISTORY OF A POST PULL PNEUMOTHORAX OR EFFUSION: IS OBSERVATION SAFE?

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Introduction: Placement of a thoracostomy tube (TT) for drainage of hemopneumothorax is the most common intervention in thoracic trauma. Post pull pneumothorax or effusion (PPP/PPE) is common after removal of the TT. The natural history of untreated PPP /PPE after discharge has not been described. This study evaluates the outcomes and management of PPP/PPE after discharge.

Methods: Trauma patients with chest tubes placed from July 1, 2008 to June 30, 2013 were identified from a billing database and our trauma registry. PPP/PPE was defined as the presence of air or fluid in the chest on the last chest image with a TT in place or on a post pull chest image. The electronic medical record (EMR) and final staff radiology interpretation of chest imaging were reviewed to confirm PPP/PPE during the initial admission and on discharge. Subsequent clinical follow up and imaging were reviewed for presence of persistent PPP/PPE. Interventions and readmissions directed towards the PPP/PPE as well as readmissions were recorded for patients with and without a PPP/PPE. A multivariate logistic regression was performed to identify factors a chest related readmission.

Results: Three hundred patients surviving to discharge had one or more TT placed during the study period. Of the 154 (59%) patients with documented PPP/PPE on discharge, 105 patients had follow-up data available. Outpatient imaging was obtained in 34 patients with persistent PPP/PPE noted in 15(44%). Seven patients (6.6%) with available follow up data required readmission and intervention. Patients in the non-PPP/PPE had a lower readmission rate (0.7% vs 6.6%. p=0.02). Multivariate logistic regression noted chest tube days (OR 1.4, p=0.015) and presence of persistent effusion or pneumothorax at clinic follow-up (OR 445, p=0.001) to be associated with readmission.

Conclusion: A PPP/PPE is a common occurrence after removal of a TT, occurring in over half of our patients. While patients discharged with PPE/PPT have a statistically higher readmission and reintervention rate, the absolute value remains low. This should be considered during the decision to treat clinically stable, asymptomatic PPT/PPE.

Poster # 123

AGE NOT THE NUMBER OF RIB FRACTURES AND THE PRESENCE OF PULMONARY CONTUSION DETERMINES THE OUTCOME OF PATIENTS WITH RIB FRACTURES

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Introduction: Because of the growing number of patients age > 75 whose performance status is superior to the former generation of > 65, we decided to evaluate the impact of the number of rib fracture (Rib Fx) and the presence of pulmonary contusion (PC) on the Rib Fx related mortality of the "new old" patients.

Methods: retrospective review of 1529 patients with isolated Rob Fx between 9/1/2010 and 12/31/2013. 316/1529 (20.7%) had PC. Patients were stratified by the number of Rib Fx, age < and > 75, and the presence or absence of PC. Statistical analysis by chi-sqaure included comparison of mortality stratified by age and absence or presence of PC. Data are presented as proportions. Statistical significance was accp[eted to correspond to a p value < 0.05.

Results: Mortality in patients without PC, independent of the number of Rib Fx, was statistically greater in patients > 75 years. The presence of PC did not increase mortality independent of age.

Mortality in patients with and without PC

# Rib Fx	Mortality < 75 No PC	Mortality < 75 PC	Mortality ≥ 75 No PC	Mortality ≥ 75 PC
1-3	19/498 (3.8%)	10/177 (8.5%)	11/87 (12.6%)**	1/9 (11.1)*
4-7	15/34 (4.4%)	5/99 (5.0%)	12/98 (12.2%)**	1/17 (5.9%)
8 or more	13/146 (8.9%)	6/56 (10.7%)	2/41 (4.9%)	1/18 (5.6%)
Total	47/987 (4.8%)	21/272 (7.7%)	25/226 (11.0%)**	3/44 (6.8%)

* $p < 0.05$ versus ≥ 75 No PC; ** $p < 0.05$ versus < 75 No PC

Conclusion: Based on the result of this study we conclude that the presence of PC contusion does not affect rib Fx related mortality independent of age. However, age alone remains a determinant of mortality, independent of the number of rib Fx. Further studies should address the impact of the volume of the contused lung on the outcome of patients with rib Fx.

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

Poster # 124

CLAMSHELL THORACOTOMY: UNDERUTILIZED OR OVERLY AGGRESSIVE?

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Introduction: Resuscitative thoracotomy is a potentially lifesaving operative procedure. Despite the unparalleled exposure and access that a bilateral or clamshell thoracotomy may afford, few studies describe the indications for and the outcomes of patients undergoing this highly morbid procedure. The objective of this study was to review our institutional experience with clamshell thoracotomies. We hypothesized that the performance of a clamshell thoracotomy would be associated with an increased risk for mortality.

Methods: Patients who underwent a resuscitative thoracotomy were identified from our Level I trauma center registry. We performed a 13-year retrospective cohort study comparing demographics, injury patterns and severity, operative procedures, and outcomes between patients undergoing a resuscitative versus clamshell thoracotomy. Multiple logistic regression analysis was performed to determine independent predictors of mortality.

Results: Of 413 patients who underwent a resuscitative thoracotomy, 49 patients (12%) underwent a clamshell thoracotomy. The overall survival rate was 23%. There was no difference in age, sex, or mechanism of injury between patients who underwent a resuscitative versus clamshell thoracotomy. Patients who underwent a clamshell thoracotomy had a higher mean chest AIS (4 ± 2 vs. 3 ± 2, $p \leq 0.05$) and ISS (48 ± 21 vs. 39 ± 22, $p=0.01$). There was a higher incidence of cardiac (45% vs. 29%, $p < 0.05$) and pulmonary (76% vs. 58%, $p < 0.05$) injuries among patients who underwent a clamshell thoracotomy. These patients were also more likely to sustain right-sided and bilateral injuries ($p \leq 0.05$). There was no difference in mortality between patients undergoing a resuscitative versus clamshell thoracotomy (76% vs. 86%, $p=0.13$). On multivariate analysis, an ISS >25 (OR 7.88; 95% CI, 2.70-23.00, $p < 0.001$) and admission GCS ≤8 (OR=23.54; 95% CI, 12.08-45.85, $p < 0.001$) were found to be independently associated with mortality. Clamshell thoracotomy was not associated with an increased risk for mortality.

Conclusion: Clamshell thoracotomy is an invasive yet potentially lifesaving procedure that is not associated with increased mortality among patients requiring a resuscitative thoracotomy. Further study is required to determine the indications and optimal timing for performing this procedure.

Poster # 125

EXTINCTION OF THE PREHISTORIC TRAUMA EVALUATION: ELIMINATION OF THE ROUTINE TRUAMA BAY CHEST X-RAY

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Introduction: Thoracic injuries occur in one third of all trauma patients admitted to major trauma centers. Advanced trauma life support emphasizes the role of portable chest x-ray (CXR) to screen for thoracic injuries. However, with advances in imaging technology, the chest computed tomography (CT) is a better imaging modality for thoracic injury and ultrasound (US) allows for real time evaluation for pneumothorax (PTX). This study sought to determine the role of the routine trauma bay CXR in the evaluation and management of injured patients.

Methods: This study is a retrospective review of a process improvement projects reviewing all trauma alerts at a single institution from April 2013 – February 2014. Patients underwent routine trauma bay CXR, extended focused assessment with sonography for trauma (eFAST), and chest CT. Radiographic images were reviewed by an attending radiologist, ultrasound findings were interpreted by a chief resident and/or a trauma attending.

Results: During the study period 527 patients underwent complete thoracic imaging with CXR, US, and chest CT. The CXR missed an injury in 68 patients, detected an injury that was confirmed on CT in 52 patients, and was read as having an injury in 11 patients that had no injury seen on chest CT. There were two blunt aortic injuries during the study period and neither one had evidence of a widened mediastinum on CXR. Ultrasound detected all PTX seen on CXR. The sensitivity of US for PTX was greater than CXR (29.5% vs 18.1%) when compared to chest CT.

		Computed Tomography		
		Thoracic Injury	No Thoracic Injury	
Chest X-Ray	Thoracic Injury	52	11	PPV- 82.5%
	No Thoracic Injury	68	396	NPV- 84.4%
		Sens- 43.3%	Spec- 97%	Accuracy- 84.2%

Conclusion: The routine use of trauma bay CXR can be safely eliminated from the initial evaluation. Use of the eFAST allows rapid detection of clinically significant pnuemothoracies and expedites acquisition of definitive imaging by chest CT or transport to the operating room. Routine trauma bay CXR has been eliminated from this institution's current trauma evaluation.

Poster # 126

RIB FRACTURES AND MORTALITY: BREAKING THE CAUSAL RELATIONSHIP

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Introduction: Rib fractures have long been considered to be a major contributor to mortality in the blunt trauma patient. Previous studies have shown that the number of rib fractures can be a reliable predictor in patient outcomes in the trauma setting. What has not been determined is whether or not rib fractures contribute to the actual cause of death. We hypothesized that while rib fractures can be an excellent predictor of mortality, they rarely contribute to the cause of death.

Methods: This retrospective chart review evaluated all blunt trauma patients admitted to an urban, level 1 trauma center from January 2008 to April 2013 who sustained one or more rib fractures. Patients who died had their medical records (including trauma peer review summary) reviewed in detail to determine the cause of death. Cause of death was broken down into seven categories (neurological, cardiac, hemorrhage, respiratory, dead on arrival/indeterminable, and other). Deaths were classified as being caused by rib fractures in any of the following cases: any respiratory death, death secondary to pneumonia, death secondary to hemorrhage from rib fractures.

Results: There were 2,514 blunt trauma patients who sustained one or more rib fractures and 130 (5.2%) of them died. Patients with rib fractures who died were an average of 51 years old, 68% male, 65% Caucasian, had an mean admission GCS = 6, systolic blood pressure = 64 mm Hg, pulse = 65 beats per minute, and respiratory rate = 10 breaths per minute. The rib fracture population who died was severely injured with a mean ISS = 40 and Chest AIS = 4. However, rib fractures were the cause of death in only five patients (0.2% of the entire rib fracture population). The cause of death of all five patients who died as a result of their rib fractures was respiratory failure. The other 125 rib fracture patients, who died, died as a result of something other than rib fractures. Cause of death included hemorrhage (42%, n = 53), dead on arrival/indeterminable (28%, n = 35), neurological (15%, n = 19), cardiac 10%, n = 13), infection (2%, n = 3), and other (2%, n = 2). Patients who died as a result of their rib fractures were older (81 years old vs. 51 years old, $p < 0.001$) and had a higher respiratory rate at presentation (25 vs. 9, $p = 0.009$) but were more stable at presentation with a higher admission GCS (14 vs. 6, $p < 0.001$) and systolic blood pressure (122 mm Hg vs. 61 mm Hg, $p = 0.02$). There was no difference in ISS (32 vs. 40, $p = 0.27$) or Chest AIS (4 vs. 4, $p = 0.48$).

Conclusion: Rib fractures after blunt trauma are a marker for severe injury. However, rib fractures rarely actually contribute to mortality. Blunt trauma patients with rib fractures most commonly die from hemorrhagic shock or are dead on arrival.

Poster # 127

PULMONARY CONTUSIONS ARE NOT A CONTRAINDICATION TO RIB FRACTURE STABILIZATION

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Introduction: The indications for operative fixation of rib fractures resulting from trauma to the chest wall remain controversial. Early studies indicated that patients with pulmonary contusions were suboptimal candidates for rib fracture repair. However, imaging modality and ventilatory management have changed considerably over the past decade, and pulmonary contusions that are seen with today's CT scanners may no longer be a valid contraindication to rib stabilization. Given the high rates of morbidity and mortality in patients with multiple rib fractures, more aggressive treatment with rib fixation may be indicated.

Methods: A retrospective review of 106 trauma patients with acute rib fractures who had undergone operative rib stabilization between 8/ 2009 -- 8/2013. The chest wall and pulmonary injury pattern, specifically the degree of lung injury as identified on initial imaging, was standardized and graded by use of the American Association for the Surgery of Trauma Lung Injury Scale (AAST LIS) and AAST Chest Wall Injury Scale (AAST CWIS). Patient demographics, mechanism of injury, ISS, TRISS, complications, ventilator days, ICU and hospital length of stay were measured.

Results: All 106 patients identified underwent rib stabilization, of which 47 (44%) had flail chest. The mean age was 60 (∓ 16.6), and 68% of patients were male. The number of ribs plated ranged from 1-12. Sixty nine patients (64%) had significant (AAST LIS score of 3-4) pulmonary contusions identified on initial imaging. Patient demographics and comorbid pulmonary conditions were similar between AAST LIS score groups. There were no differences in time to repair (p=0.60), OR time (p=0.48), ICU days (p=0.95), hospital LOS (p=0.91) or pneumonia (p=0.92) across AAST LIS scores. The presence or absence of pulmonary contusion did not affect ICU days (p=0.91), hospital LOS (0.98), or rates of pneumonia (0.50) or need for tracheostomy (p=0.65) in patients undergoing rib fracture stabilization.

Conclusion: Trauma patients with multiple rib fractures who have higher AAST lung injury scores can safely undergo operative rib fixation with equivalent outcomes to patients with less severe parenchymal lung injury. Pulmonary contusions should no longer be seen as a contraindication to rib stabilization.

Poster # 128

THE EFFECT OF ISOLATED RIB FRACTURES ON QUALITY OF LIFE SCORES IN TRAUMA PATIENTS

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Introduction: Rib fractures are a common injury and can lead to chronic pain and disability. There are limited data on the effects of rib fractures on quality of life measures. Although poorly characterized, patient perceived quality of life (QOL) following trauma has been shown to be lower when compared to uninjured adults. This study was designed to evaluate the impact of isolated rib fractures on QOL.

Methods: A retrospective review of our trauma registry was performed to identify trauma patients with isolated rib fractures over a four year period. Under our Quality of Life Program Initiative (QOLPI) a SF-36-2 is administered during admission, 1 month, and 6 months after discharge. The SF-36-2 measures QOL across eight domains and reports them as Physical Component Score (PCS) and Mental Component Scores (MCS). The PCS and MCS were compared to established scores in the general adult and adult trauma populations. Linear regression and correlation techniques were performed to determine if any variables predicted lower QOL scores.

Results: 45 of 134 isolated rib fracture patients had SF-36-2 data available. Average age was 62.6 and 53% were male. MVC (48.9%) and falls (33.3%) were the most common mechanisms. No variables were found to predict low QOL scores.

Measure	*Rib Fracture*	*Trauma Population*	*General Population*
PCS initial	24.6 (1.8)	NA	50* (0.2)
PCS 1 month post	26.3(1.3)	32.8 (0.9)	50* (0.2)
PCS 6 months post	28.0(2.3)	41.3 (1.0)	50* (0.2)
MCS initial	18.5 (2.2)	NA	50* (0.2)
MCS 1 month post	16.7 (2.1)	47.5 (1.1)*	50* (0.2)
MCS 6 months post	16.5 (2.3)	47.2 (1.1)*	50* (0.2)

*p<.05 values mean (SD)

Conclusion: Patients with isolated rib fractures have significantly lower SF-36-2 MCS compared to both general trauma patients and the general population, at 1 and 6 months after injury. PCS was not significantly lower than the general trauma population. As MCS scores were dramatically lower compared to norms, further investigation is warranted to evaluate the impact of rib fractures on psychological health and to identify potential treatment options.

Poster # 129

AN AUDIT OF COMPLICATIONS OF INTERCOSTAL CHEST DRAIN INSERTION IN A HIGH VOLUME TRAUMA SERVICE IN SOUTH AFRICA.

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Introduction: Intercostal chest drain (ICD) is a commonly performed procedure in trauma and is associated with significant morbidity.

Method: This was a retrospective review of ICD complications in a major trauma service in South Africa over a 4 year period from January 2010 to December 2013.

Results: A total of 1054 ICDs were inserted in 1010 patients. 966 patients had unilateral ICDs, 44 had bilateral ICDs. Male: 91%, Female: 9%, Median age: 24 (20-29) years. Mechanism of injury: Penetrating: 75% (762/1010), Blunt: 25% (248/1010). Indications: Hemothorax: 30% (314/1054), Hemopneumothorax: 32% (339/1054), Simple Pneumothorax: 25% (268/1054), Tension Pneumothorax: 7% (79/1054), Open Pneumothorax: 5% (54/1054). 235 (22%) complications were identified: 63% (147/235) were insertional complications: Kinked: 24% (26/147), Subcutaneous: 24% (36/147), Too deep: 19% (28/147), Too shallow: 18% (27/147), Inadequate fixation: 10% (14/147), Organ injuries. 37% (88/235) were positional complications: Outside the safety triangle: 94% (83/88), Wrong side: 6% (5/88). 91% (214/235) of complicated drains were inserted by junior doctors and only 9% (21/235) by senior doctors. There was no mortality as a direct result of ICD insertion.

Conclusions: ICD was associated with a high rate of complications with the majority related to insertion. A large number of complications occurred amongst junior doctors. A multifaceted quality improvement program is urgently needed to reduce the complication rate.

Poster # 130

EXPENSE OF ED THORACOTOMY: IS TEMPORARY RETURN OF CIRCULATION THE CULPRIT?

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INTRODUCTION: Resuscitative thoracotomy offers many injured patients one last slim chance for survival. The mean cost per patient of (EDT) has been described, but these studies typically do not discriminate between the thoracotomy and other downstream surgical and critical care. If EDT itself only contributes a small fraction to the hospital costs/charges among patients who undergo the procedure, then the decision of whether or not to perform thoracotomy may not impact costs as much as other decisions in the early hours of care.

METHODS: Using the trauma registry, all itemized charges were documented for patients who underwent EDT at an urban Level One trauma center from 2003-2013. Non-survivors were categorized based on survival time and interventions. Charges were categorized as ED, post-ED, and transfusion-related. All patients taken to OR had spontaneous circulation at time of transfer.

RESULTS: 209 patients were included. Mean total hospital charges were $36,267. Patients pronounced dead in ED had mean charges of $11,524, and charges increased dramatically with time and OR intervention (Table 1). 49/63 patients who went to OR after ED died (78%). 14 patients (6.7%) survived their hospitalization with an average cost of $241,235 and $19,243 in blood transfusion related charges.

CONCLUSION: The charges associated with EDT itself are small compared to the cost of ongoing resuscitation and operative care after temporary return of circulation. To maximize cost effectiveness, efforts to define futile care at/after thoracotomy may be more important than further restriction of the procedure.

	N	ED Charges (thousands $)	Post-ED Charges (thousands $)	Blood Transfusion Charges (thousands $)	Total Charges (thousands $)
All patients	209	10.3 ± 0.5	20.2 ± 4.9	5.8 ± 0.8	36.3 ± 5.2
Died in ED	146	10.7±0.6	0	0.8 ± 0.1	11.5 ± 0.7
OR, died <1 hr	12	10.2 ± 1.7	14.0 ± 1.7	6.8 ± 2.3	31.0± 3.4
OR, died 1-4 hr	23	10.0± 1.3	23.4 ± 3.3	13.7 ± 4.5	47.0 ± 5.5
OR, died 4-8 hrs	8	6.7 ± 1.9	32.8 ± 5.8	23.0 ± 3.9	62.4 ± 8.0
OR, died >8 hrs	6	8.0 ± 1.9	50.4 ± 7.8	35.4 ± 11.5	94.8 ± 15.2
Survivors	14	10.1 ± 1.5	211.9 ±48.9	19.2 ± 3.2	241.2 ± 49.9

Table 1. Mean hospital charges of patients who underwent EDT.

Poster # 131

PREHOSPITAL INTUBATION ADVERSELY AFFECTS OUTCOMES IN PATIENTS RECEIVING MASSIVE TRANSFUSION

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Introduction: Prehospital intubation (PI) remains a controversial intervention provided by Emergency Medical Services (EMS) personnel on severely injured trauma patients. It has been shown to adversely affect mortality in traumatic brain injury and in animal models with penetrating hemorrhagic shock. We hypothesized that PI adversely affects outcomes in patients requiring massive transfusion (MT) in an urban level I trauma center.

Methods: We reviewed our trauma registry for all patients requiring MT (defined as total blood products > 12units/ first 24h) from 2009-2013. Patient demographics, injury severity, body region severity, prehospital airway interventions, scene and transport times and outcomes were extracted from the database. A Mann-Whitney test was used for continuous variables, a Chi-square test for categorical variables and a logistic regression analysis was used to adjust for confounding variables.

Results: A total of 193 patients were identified to have received MT during the study period. PI was performed on 21 patients (11%) vs. 172 patients (89%, no-PI group). Age, gender and initial blood pressure were similar amongst groups. ISS was 24.6 for PI vs. 23.3 for no-PI, p=NS. Mortality was significantly worse for the PI group after both blunt and penetrating trauma; conversely hospital and ICU LOS was shorter (table). Scene and transport times were significantly longer for the PI group: 18.3 min vs. 13.7 for no-PI group, $p<0.05$ and 17.8min vs. 13.3 min, $p<0.05$ respectively. A logistic regression model confirmed that PI was associated with increased mortality despite adjusting for age, ISS, blood units given, transport and scene times: OR 7.5, 95% CI: 2.1-27.6, $P<0.05$.

Conclusion: Prehospital intubation was associated with increased mortality in a cohort of trauma patients requiring massive transfusions despite adjusting for severity of illness, scene and transport times. Other faster, less invasive airway adjuncts combined with rapid transport should be encouraged in this patient population.

	PI n=21	**No PI n=172**	**P value**
Mortality (overall)*	17/21 (81%)	69/172 (40%)	<0.01
Blunt*	13/17 (77%)	46/104 (44%)	<0.01
Penetrating*	4/4 (100%)	23/68 (34%)	<0.01
Hospital LOS* (days)	4.5 +/- 8	17.1 +/- 20	<0.01
ICU LOS* (days)	4.3 +/- 3	8.2 +/- 8	<0.01
Blood products/first 24h (units)	37 +/- 32	29 +/- 24	0.31

Poster # 132

DOES LIMITED PREHOSPITAL RESUSCITATION WITH COLLOIDS OR CRYSTALLOIDS INFLUENCE HEMOSTASIS AND SURVIVAL IN RABBITS WITH AN UNTREATED NON-COMPRESSIBLE HEMORRHAGE

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Introduction: Prehospital, low-volume resuscitation of combat casualties using an artificial colloid (Hextend) has been recommended due to military logistics. We studied hemostatic effects of limited resuscitation with a newer synthetic colloid (Voluven) compared to a natural colloid (albumin) or crystalloids in an uncontrolled hemorrhage model.

Methods: Spontaneously breathing NZW rabbits (3.4±0.1 kg) were anesthetized, instrumented and subjected to a splenic injury with uncontrolled bleeding. 15 min after injury (MAP<40 mmHg), rabbits were resuscitated with colloids [Voluven (V) or 5% albumin (A), 15ml/kg], or crystalloids [normal saline (NS), 30 ml/kg or 5% hypertonic saline (HS), 7.5 ml/kg], given in two bolus IV injections (20 min apart) to achieve a hypotensive pressure (MAP) of 65 mmHg, n=8-9/group (gp). Blood loss (BL) was continuously measured and animals were monitored for 2.5 hrs or until death. Blood samples were collected and analyzed for ABG, CBC, and Coag tests. Data were analyzed by Kruskal Wallis and Chi-square tests and expressed as mean ± SEM.

Results: There were no differences in baseline measures and initial blood loss at 10 min (11±0.3 ml/kg) among gp. Thirty min after fluid resuscitation, MAP was higher and shock indices were lower in colloids *vs.* crystalloids gp (p<0.05). PT changes were minimal but aPTT was ~35% higher than at baseline in all gp except in the V gp which doubled. TEG parameters were most affected by V treatment. Post-resuscitation (30 min) blood test results and final bleeding outcomes are shown in the table. (* P<0.05 vs. others, +P<0.05 *vs*. Crystalloids)

gp	Hct %	MAP mmHg	Base def (mM)	Lactate (mM)	aPTT (sec)	TEG R (mn)	TEG a angle	TEG MA	BL ml/kg	Survival rate
V	20±0.8	32±3+	3.4±0.3+	7.6±1.6+	40±5*	5.5±0.4	62±2*	53±2*	30±3*	2/8
A	24±1.6	41±6+	4.6±1.3+	8.2±1.1+	25±2	5.9±0.5	67±1	62±2	21±2	7/8*
NS	24±0.7	25±1	9.8±1.2	12±1.4	25±2	5.9±0.5	70±1	63±1	27±1	1/9
HS	29±2*	26±2	9.1±1.1	11.6±1	25±3	6.6±0.6	68±1	65±1	22±2	2/9

Conclusion: Small volume resuscitation with crystalloids appeared inadequate to effectively perfuse organs and improve tissue oxygenation and survival. Voluven was effective hemodynamically, but it was most detriment to hemostasis leading to the largest blood loss and poor survival. The best outcomes were achieved with 5% albumin, consistent with our previous observation in this model. These data suggest that plasma protein colloids should be further considered for prehospital, low-volume resuscitation of casualties with uncontrolled hemorrhage.

Poster #133

WITHDRAWN

Poster # 134

BLOOD ON BOARD: PRE-HOSPITAL TRANSFUSION OUTCOMES IN HYPOTENSIVE PATIENTS

Rebecca Schroll MD, Martin J. Carney MS, Jiselle B. Heaney MD,MPH, Norman McSwain* MD, Peter Meade MD,MPH, Juan Duchesne* MD, Tulane School of Medicine

Introduction: Damage Control Resuscitation (DCR) with early high ratio resuscitation has become standard practice in patients with severe hemorrhage. Pre-hospital Blood Transfusion (PBT) as a key component of early DCR has not been well studied in patients with uncontrolled hemorrhage. We hypothesize improved survival in hypotensive patients with PBT by Emergency Medical Services (EMS) en route to a Level 1 trauma center.

Methods: All adult patients with hypotension (systolic blood pressure <100mm Hg) transferred to a Level 1 trauma center from 2003-2013 were included. Patients were divided into two groups based on receipt of blood products during transfer (PBT vs. non-PBT). The two groups were matched by a mean age, ISS, mechanism of injury, transfer Systolic BP, and emergency department SBP. Heart rate (HR) and shock index (SI) were analyzed for transfer and on arrival to the emergency department (ED). The changes in physiologic parameters from transfer to the ED were calculated. Statistical analysis was completed using SAS 9.3. Chi-square, Fisher exact tests, and t-tests were performed.

Results: Of 234 patients, 144 met inclusion criteria with 32 in P-BT group, and the remaining 112 in non-PBT group. T-test between the matched groups PBT vs. non-PBT showed no significant difference between the matched group in regards of age (40.94 vs 40.16, p= 0.81), ISS (19.16 vs 15.97, p= 0.14), transfer SBP (76.34 vs 82.85, p=0.07) transfer HR (96.25 vs 91.59, p=0.41) and mechanism of injury (penetrating 22 vs 11, blunt 21 vs 90, p=0.097), though there was small difference in transfer SI (1.25 vs 1.07) p=0.03. Upon arrival to ED significant differences for PBT vs non-PBT groups were found for ED HR (105.41 vs 94.79, p=0.01*), ED SBP (112.30 vs 121.50, p=0.05*) and ED SI (0.99 vs 0.82, p=0.006*). After both modalities of resuscitation, the changes in physiologic parameters were not significantly different: ΔSBP (35.9 vs 38.7, p=0.93), ΔHR (9.2 vs 3.2, p=0.24), and ΔSI (-0.25 vs -0.25, p=0.90). Chi-square and Fisher exact tests showed no difference in mortality between PBT vs. non-PBT groups: 4/32 (12.5%) vs. 8/112 (7.1%) (p=0.46) respectively.

Conclusion: DCR has been shown to improve outcomes in the military and civilian setting for patients with severe hemorrhage when used in the ED, OR, and ICU. Intuitively this benefit would extend to the pre-hospital resuscitation period. This 10 year retrospective study demonstrated no survival advantage for patients with hypotension that received PBT early en route to the trauma center.

	PBT	Non-PBT	p-value
Transfer			
SBP	76.3	82.7	0.07
HR	96.2	91.5	0.41
SI	1.25	1.07	0.03
ED			
SBP	112.3	121.5	0.05
HR	105.1	94.8	0.01
SI	0.99	0.82	0.006
ED vs Transfer			
ΔSBP	35.9	38.7	0.93
ΔHR	9.2	3.2	0.24
ΔSI	-0.25	-0.25	0.90

Poster # 135

MASSIVE TRANSFUSION POLICIES AT U.S. TQIP TRAUMA CENTERS

Rachel A. Jacobs BA, Mark R. Hemmila* MD, Rosemary Kozar* MD,Ph.D., Grant V. Bochicchio* MD,MPH, H. G. Cryer* MD,Ph.D., Julie C. Leonard MD,MPH, Jennifer Horst MD, Philip C. Spinella MD, Washington University School of Medicine

Introduction: Massive Transfusion Protocols (MTPs) have been developed to implement Damage Control Resuscitation (DCR) principles. The Trauma Quality Improvement Program (TQIP) recently published a best practice guideline for massive transfusion in trauma. We performed a survey of MTP policies at TQIP centers to establish which aspects of DCR are included in massive transfusion guidelines for patients with severe bleeding.

Methods: We administered a cross-sectional electronic survey to 187 American College of Surgeons (ACS) TQIP participants. Surveys were distributed on October 10, 2013 by ACS-TQIP administration.

Results: There were 138 responses to the survey for a 74% response rate. 62% of sites are level I, and 38% are level II ACS trauma centers. 97% of centers reported having a written MTP. The three most common reported triggers for MTP activation were: physician discretion (100%), hypotension (54%), and administration of uncross-matched blood products (54%). 66% of TQIP centers indicated that they have plasma immediately available (within 2 minutes) for MTP activation. For those with immediate use plasma, 52% utilized thawed plasma, (previously frozen), 10% used both thawed and liquid plasma, and 4% used only liquid plasma. For all respondents, plasma products were immediately available in 42% of Emergency Departments (EDs), 34% of Operating Rooms (ORs), and 30% of Intensive Care Units (ICUs). Target packed red blood cells (RBCs) to plasma ratios for the first group of blood products administered during MTP guided transfusion were: 1:1 (54% of sites), 2:1 (15%) and 1.5:1 (10%) Likewise, target ratios for RBCs to platelets were: 1:1 (68%), 2:1 (6%), and no ratio (4%). 17% of sites reported having point of care thromboelastogram (POC TEG) available for use during MTP activations, while only 9% reported using TEG to guide blood use during MTP activation. Among all respondents, POC TEG is located in 13.2 % of ORs, 10% of EDs, and 10% of ICUs. Hemostatic adjuncts incorporated into MTPs included tranexamic acid (48%), cryoprecipitate (48%), recombinant activated factor VII (32%), prothrombin complex concentrates (24%), and fibrinogen concentrates (10%). 63% of sites reported that some or all of their Emergency Medical Services (EMS) prehospital agencies had the ability to administer blood products or hemostatic agents during transport. The most common blood products and hemostatic agents available and administered in transport included: RBCs (27%), TXA (14%), and Plasma (9%). There were no significant differences between level 1 and level 2 ACS trauma centers in RBC:plasma ratio, RBC:platelet ratio and hemostatic agents included in MTPs.

Conclusion: The majority of TQIP trauma centers reported having MTPs that support the use of damage control resuscitation (DCR) principles including plasma:RBC and platelet:RBC ratios >1:2. The immediate availability of thawed plasma and product use by EMS was common, while the use of TEG to guide transfusion was low.

Poster # 136

EFFICACY OF FIBRINOGEN CONCENTRATE, PROTHROMBIN COMPLEX CONCENTRATE AND TRANEXAMIC ACID ON ACUTE TRAUMATIC COAGULOPAYHY

Maria L. Guerreiro MD, Jordi L. Tremoleda Ph.D., Daniel Frith MD,Ph.D., Karim Brohi* MD,Ph.D., Bart's And The London, Queen Mary University, William Harvey Institute

When creating your abstract, the only section headers to be used are listed below and they need to be in this format:

Introduction: Acute traumatic coagulopathy is common in bleeding trauma patients and is a target for therapeutic approaches to reduce haemorrhage and improve outcomes. The consistent delivery of plasma and other blood-derived components can be challenging for even the largest trauma centers, and is even more so in resource-poor environments. Factor concentrates and other therapeutics are therefore attractive alternatives and are utilized in some centers around the world. However their efficacy in reversing acute traumatic coagulopathy has not been fully evaluated.

Methods: Male Wistar rats were subjected to trauma (paramedian laparotomy with crushing of anterior abdominal muscles and bilateral tibia-fibula fracture) and haemorrhage (40± 5% of estimated blood volume) to a target mean arterial blood pressure of 30 ± 5 mmHg. 45 minutes after the start of the bleeding the animal was treated with haemostatic agents (prothrombin complex concentrate 50 IU/kg, fibrinogen 100 mg/kg and tranexamic acid 100mg/kg) or saline and coagulation response was assessed via rotational thrombelastography. Blood samples were taken at the beginning of the experimental period before drug administration and 30 minutes after the treatment. Rats were allocated to therapeutic groups after blind randomisation (Crystalloid (CRYST); Fibrinogen (FIB); Prothrombin Complex (PCC); Tranexamic acid (TXA); and FIB+TXA)

Results:

The shock severity was similar across all studied groups (average lactate at T60= 9 mEq/L; base excess =-20mEq/L and blood withdrawal= 36% of estimated blood volume).

The clot generation improved significantly after treatment with FIB, TXA and FIB+TXA (CA5: CRYST 47.4mm; FIB 52.5mm; TXA 50.7mm; FIB+TXA 52.2mm). Maximum clot firmness also showed significant improvements with all three therapeutics (MCF: CRYST 54.2mm; FIB: 63.3mm; TXA: 62.4mm; 65.9mm). PCC showed little effect on CA5 (47.4mm) or MCF (58.5mm).

Conclusion: Fibrinogen concentrate and tranexamic acid appeared to have the most efficacy in reversing acute traumatic coagulopathy in this experimental model.

All images, charts and tables must be placed and uploaded in the body of your abstract exactly as you want them.

Poster # 137

FILTRATION LESIONS IMPAIR FUNCTIONAL COAGULATION IN BANKED WHOLE BLOOD

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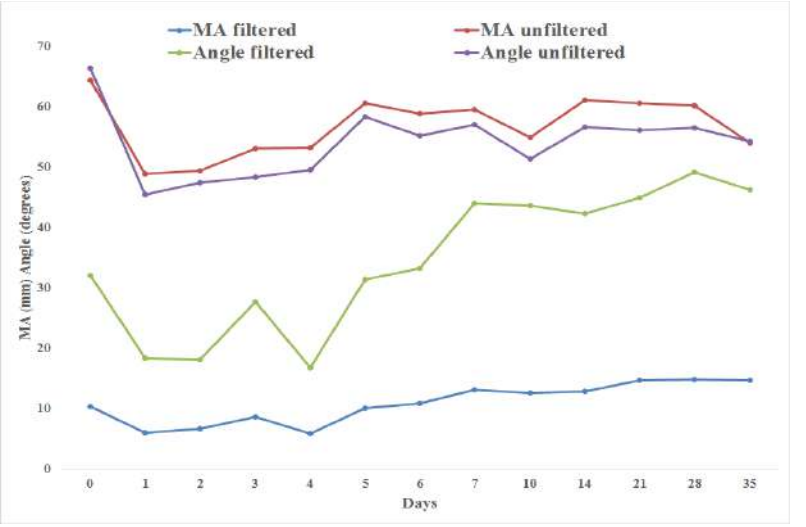
INTRODUCTION: Whole blood (WB) has been proposed as the ideal product for hemostatic resuscitation, but the shelf life and coagulation function have not been determined in leukoreduced banked whole blood. We hypothesized that coagulation impairment occurs during storage in filtered and unfiltered refrigerated WB.

METHODS: Seven donated WB units underwent leukocyte filtration and 7 did not. Units were stored at 4°C and sampled for 35 days for thromboelastogram (TEG) and centrifuged and stored at -80°C for later Calibrated Automated Thrombogram (CAT) and coagulation factor tests. Results were analyzed using t-test and mixed model regression analysis.

RESULTS: K-dependent factors and fibrinogen were low normal, and decreased slightly over 35 days but were similar between groups. Labile factors were better preserved in filtered units. CAT studies showed that thrombin production is largely preserved in both filtered and unfiltered units for 35 days. TEG studies showed that unlike unfiltered blood, filtered blood had significantly decreased clot strength (MA) and rate of clot generation (angle) as seen in the graph. Time to first sign of clot (TEG R) did not differ between filtered and unfiltered units over time.



CONCLUSION: Remarkably, unfiltered banked WB had no impairment of coagulation function over 35 days of storage. However, filtered WB had significantly decreased rate of clot growth, and clot strength and does not appear to be suitable for hemostatic resuscitation as a stand alone product.

Poster # 138

OUTCOMES OF THE MASSIVE TRANFUSION PROTOCOL IN THE NON-TRAUMA POPULATION

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Washington University

Introduction: Massive transfusion protocols (MTPs) have become the standard of care for trauma patients in hemorrhagic shock The adoption of MTP in the non-trauma patient population is a logical extension of a practice with significant survival benefit. However, there currently is little data to support its use. Therefore, we sought to determine the effect of MTP on outcomes in non-trauma patients and to determine differences in outcome between trauma and non-trauma patients undergoing MTP.

Methods: Retrospective data were collected on 159 patients who received a massive transfusion over an 18 month period from June 2012 to December 2013. Patients were stratified by age, gender, severity of illness, indices of shock and blood product transfusions. Severity of illness was assessed by APACHE II scoring.. Univariate and multivariate regression analyses were used to assess outcomes including infection, pulmonary edema, resource utilization and mortality.

Results: The mean age of the study cohort was 57±20, mean APACHE II was 15±8 and 53% were male with 62 % (n=99) being non trauma patients. Non-trauma patients were more likely to be female (59% v 27%, $p<0.0001$), more severely ill (APACHE II 16 v 12, $p=0.023$), have a lower hemoglobin level at time of initiation of MTP (9 v 12 g/dL, $p<0.0001$), a higher PT/PTT pre-MTP (21 v 16 seconds and 49 v 33 seconds, respectively, $p<0.0001$) but equivalent coagulation parameters post-MTP compared to trauma patients undergoing MTP. In addition,, prior to MTP, the non-trauma patients had significantly longer HLOS(5±9 v 0.6±1.6 days, $p<0.01$) and received significantly more red blood cells (PRBC) (3.7±7 v 1.6±0.6 units, $p=0.01$) compared to trauma patients.. Non-trauma patients had a significantly increased incidence of pulmonary edema (16% v 0%, $p=0.001$) and longer ICU and total HLOS (11±16 v 6±9 days and 17±21 v 11±12 days, respectively, $p<0.02$) compared to trauma patients. There was no significant difference in infection rates between the groups.. Logistic regression analysis revealed no significant difference in mortality when r controlling for severity of illness..

Conclusion: The use of MTP is common in the non-trauma patient population.. Non trauma patients undergoing MTP have increased morbidity and resource utilization but not increased mortality when compared to trauma patients. The higher incidence of coagulopaty in non-trauma patients may be due to a delay in activation of MTP as evidence by the lower hemoglobin and longer LOS prior to MTP use in this group. Further study is warranted to determine the true etiology of these differences.

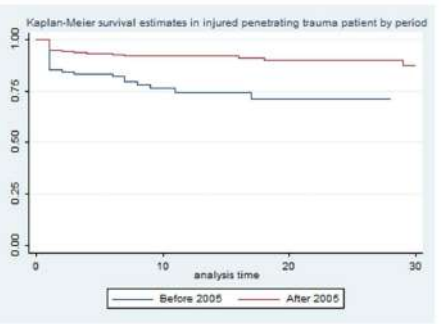
Poster # 139

ASSOCIATION OF CRYOPRECIPITATE WITH IMPROVED SURVIVAL FOLLOWING PENETRATING INJURY

Carlos A. Ordonez* MD, Marisol Badiel MD,Ph.D., MSc, Michael Parra MD, Luis F. Pino MD, Fernando Rodriguez MD, Cristina Vernaza MD, Fernando Miñan MD, Marcela Granados MD, Alvaro I. Sanchez MD, Juan C. Puyana* MD, Universidad Del Valle

Introduction: The approach to managing severe penetrating injuries has undergone major changes over the past several years. The objective of this study was to quantify the impact of fibrinogen containing cryoprecipitate on survival in penetrating trauma patients
Methods:Retrospective observational study comparing major changes in trauma resuscitative management from two separate periods at a large regional level one trauma center between the periods of 1998-2005 and 2005 -2013. All adult patients that suffered penetrating trauma who required immediate operative therapy and that required at least two units of packed red blood cells were included. Data including indications and outcomes were collected and analyzed using a univariable and bivariable regression analysis. Due to the fact that several data points were lacking, 1000 simulations were performed using the bootstrap method to verify the data. The overall survival rates were estimated via the Kaplan-Meier method.

Results: A total of 401 patients were included. The median age was 30±11.6 years and 92.8% were males. Three hundred and forty one (85%) were from gun shot wounds. During the first 24 hours of damage control resuscitation in which the patients received in addition to the packed red blood cells (PRBC's), sixty eight percent received fresh frozen plasma (FFP), 54.6% received platelets(PLT) and 39.4% received cryoprecipitates (CRYO). The mean of transfused units in the first 24 hours was PRBC's 8,3 $\pm$ 8,12, FFP 6,0 $\pm$ 7,5, PLT 7,5 $\pm$ 13,03 and CRYO 3,7 $\pm$ 5,86. The median NISS was 34 (IQR 25-44). The median intravenous fluids infused in the first 24 hours prior to 2005 was 9200cc (IQR 5400 – 13600cc) and after 2005 was 5763cc (3700-8300cc [p =0.0001]). The initial fibrinogen was <200 mg/dl in 78.9% of patients. Seventy six point nine percent of patients received CRYO after 2005 and only 23.1% of patients prior to 2005 ($p < 0.001$). The median intra-operative blood loss was 3000cc (2000-4650cc) prior to 2005 and of about 2000cc (1000-3000cc) after 2005 (p =0.001). The overall mortality was 13.9% (56/401), of which 22/56 (39,2%) were intra-operative. Prior to 2005 the mortality was higher than that of those after this date: 26.4% vs 10.1% (RR 0.38 CI95% 0.24-0.62 [p=0.0001]). The probability of survival by the Kaplan-Meier method at 30 days was 71.2% (Prior to 2005) vs 87.4% (After 2005, [p=0.005]). The possible factors that influenced the decrease in mortality over time were (adjusted according to NISS and age): the use of cryoprecipitate, and the decrease use of intravenous fluids.



Conclusion:Cryoprecipitate may independently add to the survival benefit in the seriously injured penetrating trauma patient requiring transfusion. Additional study is necessary to define the role of fibrinogen in resuscitation from hemorrhagic shock.

Poster # 140

ACHIEVING EARLIER 1:1 HEMOSTATIC RATIO WITH LIQUID PLASMA DURING DAMAGE CONTROL RESUSCITATION: A NON-INFERIORITY ANALYSIS

Juan Duchesne* MD, Rebecca Schroll MD, Peter Meade MD,MPH, Norman McSwain* MD, Tulane School of Medicine

Introduction: Damage control resuscitation (DCR) requires early administration of close ratio of plasma to Packed Red Blood Cells (PRBC) in patients with severe hemorrhage. Rapid acquisition of fresh frozen plasma (FFP) on an as-needed basis can be challenging in a civilian trauma center. We hypothesized that never frozen plasma in the form of Liquid Plasma (LP), when compared to FFP can be a non-inferior alternative in patients with severe hemorrhage managed with DCR.

Methods: All massive transfusion protocols (MTP) from June 1, 2012 to January 1, 2013 were reviewed and analyzed. Patients in which the first plasma product used was LP were identified and compared to those in which FFP was the initial plasma product. Primary outcome analyzed was the time (min.) from initiation of an MTP to the issuance of the first plasma product (LP vs. FFP). Secondary outcomes included: 1.the rapidity with which the first unit of plasma was issued relative to the first unit of PRBC, 2.the number of thawed, type-specific FFP units patients received within an hour of the initial dose of LP and FFP, and 3.overall 24-hour survival between groups.

Results: A total of 42 MTP's were reviewed of which 17 received FFP as the initial plasma product and 25 received LP. Mean time from initiation of the MTP to the issuance of the first plasma product: 5.7 ± 6.4 LP vs. 19.8 ± 7.3 FFP ($p = 0.01$). Mean time between issuance of the first unit of PRBC and the first unit of plasma was: 1.5 ± 3.4 LP vs. 10.9 ± 8.8 FFP ($p = 0.02$). Mean number of type specific units of FFP issued within 1 hour of the first plasma product was 4.3 ± 3.9 FFP vs. 10.4 ± 5.1 LP ($p = 0.04$). No difference in 24-hour survival was noted between groups: 56% LP vs. 53% FFP ($p= 0.58$).

Conclusion: In this non-inferiority analysis Liquid Plasma can provide advantages over fresh frozen plasma in terms of improved turn-around times, earlier 1:1 plasma to PRBC delivery and overall mean number of plasma infused during the first hour of resuscitation with no difference in 24-hour survival. Integration of liquid plasma as the first plasma administered in MTP's should be taken into consideration.

AAST MEMBERS

(TAB #5)

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Plurad, David S.
Putnam, Brant
Van Natta, Timothy

Vacaville
Bandy, W. Christopher
Jacoby, Robert

Ventura
Romero, Javier

Walnut Creek
Baker, Michael S.
Taekman, Howard P.

No City Listed
Batdorf, Jr., John W.
Blaisdell, F. William
Canty, Sr., Timothy G.
Gaspard, Donald J.
Shatney, Clayton

COLORADO

Aurora
Grover, Frederick L.
Hartford, Charles E.
Moulton, Steven L.
Schurr, Michael

Boulder
Greenburg, A. Gerson

Colorado Springs
Berson, Andrew
Butler, Larry J.

2014 GEOGRAPHICAL LISTING

Denver
Aragon, Guillermo E.
Bensard, Denis
Biffl, Walter
Burch, Jon Michael
Cothren Burlew, Clay
Johnson, Jeffrey
Jurkovich, Gregory J.
Moore, Ernest E.
Stahel, Philip

Englewood
Slone, Denetta Sue
Smith, Wade

Evergreen
MacArthur, John D.

Fort Collins
Bauling, Paul C.
Collins, John A.
Metzler, Michael H.

Greeley
Goodwin, Cleon W.

Lakewood
Offner, Patrick J.

Loveland
Cribari, Chris
Dunn, Julie

CONNECTICUT

Bridgeport
Atweh, Nabil A.
Smith, Michael E.

Farmington
Civetta, Joseph M.

Hartford
Brautigam, Robert T.
Browner, Bruce D.
Butler, Karyn L.
Jacobs, Jr., Lenworth M.
Joseph, D'Andrea
Kirton, Orlando
Orlando, III, Rocco
Yeston, Neil S.

New Haven
Davis, Kimberly A.
Maerz, Linda
Maung, Adrian
Schuster, Kevin

Stamford
Barone, James E.
Dwyer, Kevin M.

Waterbury
Dudrick, Stanley J.

DELAWARE

Lewes
Sturtz, Donald L.

Newark
Cipolle, Mark D.
Fulda, Gerard J.
Matsumoto, Teruo N.
Rhodes, Michael
Tinkoff, Glen

Wilmington
Hicks, Barry A.

2014 GEOGRAPHICAL LISTING

DISTRICT OF COLUMBIA

Washington
Burd, Randall S.
Cornwell, III, Edward E.
Dunne, James R.
Jeng, James
Jordan, Marion
Kirkpatrick, John
Sarani, Babak
Sava, Jack
Siram, Suryanarayana M.

FLORIDA

Bradenton
Kimbrell, Brian
Oswanski, Michael F.

Cape Canaveral
Thorbjarnarson, Bjorn

Captiva Island
Reyes, Hernan M.

Celebration
Barquist, Erik

Fort Lauderdale
Bukur, Marko

Fort Myers
Mikulaschek, Andrew

Ft. Pierce
Kurek, Jr., Stanley

Gainesville
Flynn, Timothy C.
Lottenberg, Lawrence
Mohr, Alicia
Moore, Frederick
Mozingo, David W.
Talbert, James L.

Hollywood
Carrillo, Eddy H.
Rosenthal, Andrew A.

Hudson
Norwood, Scott H.

Jacksonville
Bhullar, Indermeet
Crass, Richard A.
Dennis, James W.
Kerwin, Andrew
Tepas, III, Joseph J.

Jupiter
Ruoff, III, Andrew C.

Lutz
Agnew, Samuel G.

Melbourne
Block, Ernest F. J.

Miami
Byers, Patricia
Garcia, George
Ginzburg, Enrique
McKenney, Mark
Namias, Nicholas
Pizano, Louis R.
Schulman, Carl
Sleeman, Danny
Ward, C. Gillon

2014 GEOGRAPHICAL LISTING

Naples
Bubrick, Melvin P.

Nokomis
Wittmann, Dietmar H.

Orlando
Alban, Rodrigo
Bilski, Tracy
Burgess, Andrew R.
Cheatham, Michael

Palm Coast
Schinco, Miren

Palm Harbor
Bednarski, Jeffrey

Pensacola
Durham, Rodney M.

Plantation
Dove, Dennis B.

Port Saint Lucie
Jazarevic, Slobodan

Sanibel
Rowe, Marc I.
Sugerman, Harvey J.

Sarasota
Cass, Alexander S.
Worth, Jr., Melvin H.

Tallahassee
Armstrong, John H.

Tampa
Campbell, Sylvia
Carey, Larry C.
Ciesla, David J.
Hurst, James M.
Paidas, Charles
Rosemurgy, Alexander S.

University Park
Bachulis, Ben L.

Winter Haven
Maurer, Elmer R.

No City Listed
Connolly, John F.
Krizek, Thomas
Thatcher, Donald S.

GEORGIA

Atlanta
Buchman, Timothy G.
Dente, Christopher J.
Henderson, Vernon J.
Nicholas, Jeffrey M.
Subramanian, Anuradha
Wyrzykowski, Amy D.

Augusta
Hawkins, Michael L.
O'Malley, Keith F.
Parrish, Robert A.

Columbus
Hannay, R. Scott

Evans
Law, Edward

Macon
Ashley, Dennis W.
Van De Water, Joseph M.

Morrow
Nallathambi, Manohar N.

Savannah
Boyd, Carl R.
Bromberg, William
Senkowski, Christopher

No City Listed
Farrell, Kevin J.

HAWAII

Haleiwa
Yellin, Albert E.

Honolulu
Edwards, Kurt
Ho, Hao Chih
McNamara, J. Judson
Ursic, Caesar M.
Yu, Mihae

IDAHO

Boise
Mayberry, John C.

ILLINOIS

Chicago
Baker, Robert J.
Barrett, John A.
Bokhari, Faran
Crandall, Marie L.
Fantus, Richard
Flint, Lewis M.
Holevar, Michele R.
Hoyt, David B.
Joseph, Kimberly
Moss, Gerald S.
Nagy, Kimberly K.
Shapiro, Michael
Statter, Mindy B.
Strauch, Gerald O.
Vargish, Thomas

Evanston
Omert, Laurel

Gurnee
Alsikafi, Nejd

Highland Park
Gould, Steven
Sperling, Richard L.

Hines
Luchette, Fred A.

LaGrange Park
Letarte, Peter B.

Maywood
Esposito, Thomas J.
Gamelli, Richard
Santaniello, John

2014 GEOGRAPHICAL LISTING

Naperville
Folk, Frank

New Lenox
Marshall, Wendy J.

North Chicago
Acosta, José A.

Oak Lawn
Doherty, James

Park Ridge
Saletta, John

Quincy
Wilkins, Harry

Springfield
Sutyak, John P.
Wohltmann,
Christopher D.
Zook, Elvin

Willowbrook
Silver, Geoffrey

No City Listed
Collicott, Paul E.
Fry, Donald E.
Wagner, Franklin C.

INDIANA

Carmel
Scherer, III, L. R. Tres

Fort Wayne
Reed, Jr., Donald N.

Indianapolis
Broadie, Thomas A.
Falimirski, Mark
Feliciano, David
Gomez, Gerardo A.
Jacobson, Lewis
Nelson, Paul
Pierce, Jr., Raymond O.
Reed, R. Lawrence
Rozycki, Grace S.
Streib, Erik
Zarzaur, Jr., Ben L.

South Bend
Thomas, Scott

No City Listed
Fryer, Minot Packer

IOWA

Iowa City
Choi, Kent C.
Granchi, Thomas
Kealey, Gerald
Latenser, Barbara
Nepola, James V.
Skeete, Dionne
Thomsen, Timothy A.

No City Listed
Krigsten, William

KANSAS

Kansas City
Moncure, Michael

Leawood
Schloerb, Paul

2014 GEOGRAPHICAL LISTING

Topeka
Baker, Phillip L.

Wichita
Haan, James M.
Harrison, Paul B.

KENTUCKY

Cresent Springs
Alexander, J. Wesley

Lexington
Bernard, Andrew C.
Boulanger, Bernard
Kearney, Paul

Louisville
Cheadle, William G.
Fallat, Mary
Franklin, Glen A.
Garrison, Richard N.
Harbrecht, Brian
Miller, Frank B.
Polk, Jr., Hiram C.
Richardson, J. David
Rodriguez, Jorge L.
Seligson, David
Smith, Jason

LOUISIANA

Baton Rouge
Chapman, Michael W.
Jacome, Tomas

Hammond
Duchesne, Juan

Monroe
Johnson, Lester

New Orleans
Hunt, John P.
Marr, Alan
McSwain, Jr., Norman
Wright, Mary Johanna

Port Allen
Lee, W. Chapman

Shreveport
Owings, John T.
Simpkins, Cuthbert O.

MAINE

Portland
Ciraulo, David Leonard
Cushing, Brad M.
Eddy, Virginia A.
Grindlinger, Gene A.
Sihler, Kristen

MARYLAND

Annapolis
Champion, Howard R.
Ducker, Thomas
Eichelberger, Martin R.

2014 GEOGRAPHICAL LISTING

Baltimore
Chiu, William
Cooper, Carnell
Diaz, Jose
DuBose, Joseph
Efron, David T.
Fang, Raymond
Geis, W. Peter
Gens, David R.
Genuit, Thomas
Haider, Adil
Haller, J. Alex
Haut, Elliott
Henry, Sharon
Lilly, Michael P.
Manson, Paul N.
McQuay, Jr., Nathaniel
Menaker, Jay
Militello, Philip R.
Scalea, Thomas M.
Stein, Deborah M.
Tisherman, Samuel

Bethesda
Bowyer, Mark W.
Rice, Charles L.
Rich, Norman M.
Seyfer, Alan
Westerband, Dany

Cheverly
Ryb, Gabriel E.

Crofton
Myers, Roy A.M.

Elkton
Buckman, Jr., Robert F.

Fort Detrick
Rasmussen, Todd

Glen Burnie
Soderstrom, Carl A.

Lutherville
Gann, Donald S.

New Market
Boyd, David R.

MASSACHUSETTS

Beverly
De Santis, Lindsay

Boston
Ackroyd, Frederick W.
Blackburn, George L.
Briggs, Susan
Burke, John F.
Burke, Peter
Cahill, John M.
Conn, Alasdair K.T.
Cooper, Zara
de Moya, Marc A.
Gates, Jonathan
Hauser, Carl J.
Hechtman, Herbert B.
Kelly, Edward
Kenney, Pardon
King, David
O'Donnell, Thomas F.
Rabinovici, Reuven
Salim, Ali
Schulze, Robert
Sheridan, Robert
Tompkins, Ronald G.
Velmahos, George
Wilmore, Douglas W.

Cambridge
Nauta, Russell J.

Framingham
Cachecho, Riad

Needham
Boyd, Robert

South Weymouth
Driscoll, Robert

Springfield
Gross, Ronald
Patterson, Lisa

Waban
Millham, Frederick H.

Wayland
Marion, Donald W.
Rosenthal, Ronald E.

Worcester
Emhoff, Timothy
Hirsh, Michael P.
Santry, Heena
Silva, Wayne E.

No City Listed
Sherman, Harold

MICHIGAN

Ann Arbor
Alam, Hasan
Anderson, III, Harry
Brandt, Mary-Margaret
Burney, Richard E.
Coran, Arnold G.
Greenfield, Lazar J.
Hemmila, Mark R.
Napolitano, Lena M.
Park, Pauline K.
Raghavendran, Krishnan
Wahl, Wendy Lynn
Wang, Stewart C.

Bear Lake
Wade, Franklin V.

Bloomfield Hills
Lopez, Peter

Detroit
Baylor, III, Alfred
Diebel, Lawrence N.
Dolman, Heather
Dulchavsky, Scott A.
Horst, H. Mathilda
Ledgerwood, Anna M.
Lucas, Charles E.
Patton, Joe
Rubinfeld, Ilan
Steffes, Christopher P.
Tyburski, James
White, Michael T.
Wilson, Robert F.

Grand Blanc
Shapiro, Brian

2014 GEOGRAPHICAL LISTING

Grand Rapids
Iskander, Gaby
Scholten, Donald

Grosse Pointe Farms
Farms Knuth, Thomas

Jackson
Klotz, Jr., Donald

Lansing
Kepros, John

Manistique
Phillips, Thomas F.

Royal Oak
Howells, Greg A.

Three Oaks
Roberts, Roxanne

West Bloom Field
Robb, Herbert J.

No City Listed
Mackenzie, James R.
Robson, Martin C.

MINNESOTA

Duluth
Eyer, Steven D.

Marine on St. Croix
Strate, Richard G.

Minneapolis
Becker, William K.
Chipman, Jeffrey G.
Jacobs, Donald M.
Ney, Arthur L.
Peltier, George
Quickel, Robert R.
Richardson, Chad J.
Rockswold, Gaylan L.

Minnetonka
Croston, J. Kevin

Rochester
Jenkins, Donald
Schiller, Henry
Zielinski, Martin
Zietlow, Scott

St. Cloud
Dorle, Michael J.

St. Paul
Ahrenholz, David H.
Bennett, Bruce A.
Dries, David J.
Larkins, Mark
McGonigal, Michael D.

No City Listed
Beilman, Gregory

MISSISSIPPI

Greenville
Love, Jr., Robert T.

2014 GEOGRAPHICAL LISTING

Jackson
Ahmed, Naveed
Frei, Lonnie W.
Helling, Thomas S.
Martin, Larry C.
Porter, John M.
Timberlake, Gregory A.

Vicksburg
Hopson, Jr.,
William Briggs

MISSOURI

Columbia
Barnes, Stephen L.
Mitchell, Franklin L.

Holts Summit
James, Jr., Paul M.

Kansas City
Bjerke, H. Scott
Hiebert, John M.
Sagraves, Scott

St. Louis
Bochicchio, Grant V.
Brandes, Steven B.
Kirby, John
Mazuski, John
Peick, Ann
Schuerer, Douglas
Srivastava, Anil
Troop, Bryan
Vane, Dennis W.

No City Listed
Trask, Arthur L.

MONTANA

Billings
Hurd, Robert N.

Bozeman
Rinker, Charles F.

Great Falls
Orcutt, Michael

Missoula
Pickhardt, John B.

No City Listed
Diamond, Daniel L.

NEBRASKA

Lincoln
Burton, Reginald

Omaha
Cemaj, Samuel
Hodgson, Paul E.
Schenarts, Paul
Stothert, Jr., Joseph C.

NEVADA

Las Vegas
Browder, Timothy D.
Coates, Jay E.
Fildes, John J.
Kuhls, Deborah A.
McIntyre, Kenneth E.

2014 GEOGRAPHICAL LISTING

NEW HAMPSHIRE

Lebanon
Burchard, Kenneth W.
Gupta, Rajan

NEW JERSEY

Camden
Ross, Steven
Seamon, Mark

Englewood
Siegel, John

Freehold
DiGiacomo, Jody

Hackensack
Barbul, Adrian
O'Hara, Kathleen P.

Jersey City
Lazaro, Eric

Mountainside
Blackwood, James M.

Neptune
Ahmed, Nasim
Davis, John M.
Vernick, Jerome J.

New Brunswick
Gracias, Vicente H.
Lissauer, Matthew
Stafford, Perry W.
Trooskin, Stanley Z.

Newark
Anjaria, Devashish
Deitch, Edwin A.
Livingston, David
Mosenthal, Anne
Padberg, Jr., Frank T.
Sifri, Ziad C.
Swan, Kenneth G.

Park Ridge
LoCurto, Jr., John J.

Paterson
Valenziano, Carl P.

Shourt Hills
Nance, Francis C.

Somerville
Hammond, Jeffrey S.

Stratford
Fallahnejad, Manucher

Trenton
D'Amelio, Louis

Vineland
Slotman, Gus

NEW MEXICO

Albuquerque
Buntain, William L.
Demarest, Gerald B.

Santa Fe
Schiller, William
Wassner, John

2014 GEOGRAPHICAL LISTING

NEW YORK

Albany
Leather, Robert P.
Shah, Dhiraj M.

Bay Shore
Grossman, Michael D.

Bronx
Agarwal, Nanakram
Cayten, C. Gene
Delany, Harry M.
DiRusso, Stephen Michael
Yelon, Jay A.

Brooklyn
Duncan, Albert
Hirshberg, Asher
Horovitz, Joel H.
O'Neill, Patricia
Ramenofsky, Max L.

Buffalo
Bass, Kathryn D.
Flynn, Jr., William J.
Guo, Weidun Alan
Hassett, James M.
Mindell, Eugene R.
Wiles, III, Charles E.

Cooperstown
Borgstrom, David C.

East Meadow
Shaftan, Gerald

Manhasset
Bagdonas, Richard
Coppa, Gene F.

Mineola
Axelrad, Alexander

New Hyde Park
Stylianos, Steven

New York
Barie, Philip S.
Barlow, Barbara A.
Bessey, Palmer Q.
Cooper, Arthur
Eachempati, Soumitra
Frangos, Spiros
Ghajar, Jamshid
Marshall, Gary
Michelsen, Christopher B.
Pachter, H. Leon
Simon, Ronald
Todd, S. Rob
Velcek, Francisca T.
Yurt, Roger W.

Pittsford
Kluge, David

Rochester
Bankey, Paul E.
Cheng, Julius D.
Gestring, Mark L.
Rotondo, Michael F.
Stassen, Nicole
States, John D.

Stanfordville
Madden, Michael R.

Staten Island
Garzon, Antonio A.
Pizzi, Walter
Schulz, III, John

2014 GEOGRAPHICAL LISTING

Stony Brook
Shapiro, Marc
Soroff, Harry S.

Syracuse
Cooney, Robert N.
Hassan, Moustafa
Marx, William H.

Valhalla
Marini, Corrado
Salisbury, Roger
Savino, John

White Plaines
Asensio, Juan

No City Listed
Baue, Arthur E.
Connell, Jr., James F.
Harris, Burton H.
Herbsman, Horace
Mahoney, Jesse W.

NORTH CAROLINA

Asheville
Arrillaga, Abenamar

Chapel Hill
Cairns, Bruce A.
Meyer, Anthony A.
Rich, Preston B.

Charlotte
Bosse, Michael J.
Christmas, A. Britton
Green, John
Huynh, Toan T.
Jacobs, David G.
Kellam, James
Sing, Ronald F.
Stallion, Anthony
Thomason, Michael H.

Durham
Moylan, Jr., Joseph A.
Olson, Steven A.
Shapiro, Mark
Vaslef, Steven

Gastonia
Borzotta, Anthony

Greensboro
Wyatt, III, James

Greenville
Bard, Michael R.
Cunningham, Paul R.G.
Goettler, Claudia E.
Haisch, Carl
Newell, Mark
Rodeberg, David A.
Toschlog, Eric
Waibel, Brett

Hendersonville
Rosner, Michael J.

Raleigh
Oller, Dale W.
Rutherford, Edmund J.
Udekwu, Pascal

2014 GEOGRAPHICAL LISTING

Wilmington
Clancy, Thomas V.

Winston-Salem
Meredith, J. Wayne
Chang, Michael
Hildreth, Amy
Holmes, IV, James
Hoth, James J.
Martin, Robert
Meredith, Jesse H.
Miller, Preston R.
Mowery, Nathan

NORTH DAKOTA

Grand Forks
Szlabick, Randolph

OHIO

Akron
George, Richard
Muakkassa, Farid F.

Cincinnati
Davis, Jr., Kenneth
Falcone, Jr., Richard A.
Johannigman, Jay
Kagan, Richard
Muskat, Peter
Pritts, Timothy
Robinson, Bryce
Tsuei, Betty
Welling, Richard E.
Wulsin, John H.

Cleveland
Clancy, Keith
Claridge, Jeffrey A.
Como, John J.
Fratianne, Richard B.
Likavec, Matt J.
Spirnak, John P.
Treat, Richard C.
Yowler, Charles

Columbia
Yashon, David

Columbus
Bonta, Marco J.
Cook, Charles H.
Falcone, Robert E.
Furste, Wesley
Groner, Jonathan I.
Jones, Larry
Lindsey, David
Miller, Sidney F.
Ruberg, Robert L.
Steinberg, Steven M.
Yaw, Peter B.

Dayton
Ekeh, A. Peter
McCarthy, Mary C.
Saxe, Jonathan
Tchorz, Kathryn M.
Walusimbi, Mbaga S.

Galena
Berggren, Ronald B.

Oregoma
Finley, Jr., Robert K.

Rocky River
Waltz, Robert C.

Toledo
Howard, John M.
Williams, Mallory

Youngstown
Dunham, C. Michael
Ransom, Kenneth

No City Listed
Clare, David W.

OKLAHOMA

Norman
McCullough, Gerald W.

Oklahoma City
Albrecht, Roxie
Bender, Jeffrey S.
Letton, Jr., Robert W.

Tulsa
Ford, Edward G.
Siemens, Roger
Thompson, Lewis W.
Wang, Dennis

No City Listed
Carter, Phillip L.
Fisher, Jr., Robert G.

OREGON

Portland
Barbosa, Ronald
Cole, Jr., Frederic J.
Gubler, K. Dean
Izenberg, Seth D.
Long, William
Malinoski, Darren
Mullins, Richard J.
Ramzy, Ameen I.
Rehm, Christina G.
Rowell, Susan
Schreiber, Martin
Trunkey, Donald D.
Watters, Jennifer
Zonies, David

St. Paul
Livaudais, Jr., West

No City Listed
Martin, Louis F.

PENNSYLVANIA

Allentown
Badellino, Michael M.
Barraco, Robert D.
Pasquale, Michael D.

Altoona
Capella, Jeannette

Bethlehem
De Long, Jr., William
Hoff, William S.
Sharpe, Richard

Bryn Mawr
Templeton, Jr., John M.

2014 GEOGRAPHICAL LISTING

Clarksville
Bergstein, Jack M.

Collegeville
Tortella, Bartholomew

Danville
Leonard, DiAnne Jo
Scorpio, Ronald
Timmons, Shelly D.

Erie
Bales, Charles R.

Glen
Manges, Lewis C.

Hershey
Armen, Scott
Cilley, Robert E.
Indeck, Matthew C.

Johnstown
Dumire, Russell
Rodriguez, Aurelio

King of Prussia
Osterman, A. Lee

Lancaster
Lee, John
Rogers, Frederick B.

Langhorne
Talucci, Raymond C.

Monroeville
Kaufmann, Christoph

Philadelphia
Cohen, Murray
Goldberg, Amy J.
Kaplan, Lewis
Kaplan, Mark
Kim, Patrick
Lewis, Jr., Frank R.
Malangoni, Mark A.
Martin, Niels
Nance, Michael L.
Pascual Lopez, Jose
Pathak, Abhijit S.
Rappold, Joseph
Reilly, Patrick
Rhodes, Robert S.
Rogers, Selwyn O.
Santora, Thomas A.
Schwab, C. Willaim
Simms, H. Hank
Sims, Carrie
Wein, Alan

Pittsburgh
Alarcon, Louis
Billiar, Timothy R.
Copeland, Charles E.
Forsythe, Raquel
Gaines, Barbara A.
Lynch, James M.
Pape, Hans-Christoph
Peitzman, Andrew B.
Philp, Allan S.
Puyana, Juan Carlos
Rosengart, Matthew
Sperry, Jason
Wilberger, James E.
Wilson, Mark A.

Scranton
Shaikh, Khaleel

Swarthmore
Holst, Hazel I.

Towanda
Alpert, Marc

Wayne
Ernst, Calvin B.

West Chester
Gennarelli, Thomas A.

West Reading
Brigham, Robert
Reading Ong, Adrian

Wynnewood
Clarke, John R.

York
Agarwal, Nikhilesh N.

No City Listed
Ralston, Edgar Lee

RHODE ISLAND

Providence
Adams, Jr., Charles
Born, Christopher T.
Cioffi, William G.
Harrington, David
Hopkins, Robert W.
Trafton, Peter.

SOUTH CAROLINA

Charleston
Crookes, Bruce
Fakhry, Samir M.
Norcross, E.
Othersen, Jr., H. Biemann

Columbia
Bell, Richard M.
Bynoe, Raymond
Smith, R. Stephen

Greenville
Gauderer, Michael W.L.
Roettger, Richard H.

Simpsonville
McCormack, Robert M.

Spartanburg
Morrow, Jr., Charles E.

No City Listed
Sprague, Bruce L.

SOUTH DAKOTA

Spearfish
Bee, Tiffany K.

TENNESSEE

Chattanooga
Barker, Donald Edgar
Maxwell, Robert A.

Johnson City
Browder, William
Putnam, Adin

2014 GEOGRAPHICAL LISTING

Kingsport
Hall, John R.
Lasky, Tiffany
Siffring, Corydon
Testerman, George

Knoxville
Daley, Brian
Enderson, Blaine L.

Memphis
Croce, Martin A.
Fabian, Timothy C.
Luce, Edward A.
Magnotti, Louis J.
Maish, III, George
Minard, Gayle
Savage, Stephanie
Schroeppel, Thomas
Weinberg, Jordan

Nashville
Collier, Bryan R.
Guillamondegui, Oscar D.
Gunter, Oliver
May, Addison
Miller, Richard S.
Morris, Jr., John A.
Nunez, Timothy
O'Neill, Jr., James A.
Sharp, Kenneth

Ooltewah
Singh, Iqbal

Rutledge
Kottmeier, Peter

TEXAS

Austin
Brown, Carlos
Garcia, Nilda M.
Peterson, Hugh D.
Tuggle, David W.
Valadka, Alex B.

Bellaire
Andrassy, Richard J.

Brownsville
Barba, Carlos A.

Corpus Christi
Blow, Osbert

Dallas
Dunn, Ernest L.
Eastman, Alexander
Foreman, Michael L.
Hunt, John
Lorenzo, Manuel
Luk, Stephen S.
Megison, Stephen M.
Meyer, Dan M.
Minei, Joseph
Minshall, Christian
Morey, Allen
Murphy, Joseph
Phelan, III, Herbert A.
Shafi, Shahid
Thal, Erwin R.
Truitt, Michael
Wolf, Steven E.

2014 GEOGRAPHICAL LISTING

El Paso
- Bagg, Raymond J.
- Flaherty, Stephen
- Peacock, Jack B.
- Tyroch, Alan H.

Fort Sam Houston
- Martin, Robert
- Renz, Evan
- Bailey, Jeffrey
- Cannon, Jeremy

Galveston
- Cushman, James G.
- Gore, Dennis C.
- Herndon, David N.
- Mileski, William J.

Georgetown
- Gonzalez, Ernest

Helotes
- Blackbourne, Lorne H.

Houston
- Cotton, Bryan A.
- Cox, Jr., Charles
- DeBakey, Michael E.
- Duke, Jr., James H.
- Fischer, Ronald P.
- Holcomb, John B.
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(TAB #6)

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Our Mission
The Mission of the AAST Research and Education Foundation is to promote and advance the optimal care of injured and critically ill surgical patients by obtaining philanthropic support to expand knowledge, advance the art and science, and develop professionals in the field of trauma and acute care surgery.

About The Foundation
The American Association for the Surgery of Trauma Research and Education Foundation was established in April of 1994 by the Association's Board of Managers with the objective to sponsor research scholarships in the fields of burns, trauma, and acute care surgery and to foster advances in education.

The Foundation is currently supported by AAST, private industry donors, and contributions from AAST members. This support has afforded the Foundation the ability to fund numerous scholarships, and with further support, the Foundation hopes to expand its funding of projects into other areas of interest such as multi-institutional trials.

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For the past twenty years the American Association for the Surgery of Trauma, the AAST Research and Education Foundation, and their Educational Partners have awarded over $3.5 Million dollars in scholarships, with nearly $2 Million coming from the AAST Foundation and its Educational Partners.

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IN MEMORY

(TAB #7)

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R. Donald Woodson, M.D.
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Member Since: 1972

WILLIAM R. CLARK, JR., M.D.

William Reeve Clark, 79, of Syracuse died April 18 at University Hospital. A native of Michigan, he has lived in Syracuse since 1974. He graduated from Groton School, Yale University, Boston University School of Medicine, and completed a Surgical Residency Program at Albany Medical Center. A U.S. Army veteran, Dr. Clark served as a flight surgeon in Korea where he started an orphanage, while caring for American troops. In 1969 he was the physician on the USS Manhattan, Humble Oil's Maiden Northwest Passage Voyage.

Dr. Clark was a Professor of Surgery in clinical practice at SUNY Upstate Medical University for 20 years. He founded and was director of the Clark Burn Center at University Hospital and worked tirelessly in the community to promote burn prevention and education. After retiring from UMU, he was employed by the New York State Health Department in the Hospital and Primary Care Program.

While his medical practice was the sole focus prior to his retirement, Dr. Clark was an avid canoe and cross country ski racer. While often in against younger, more experienced competitors, the goal was not so much about winning as about finishing, which he always did.

He served on the Board of Directors of Sonnenberg Gardens and the Frederick Remington Art Museum and worked with Literacy Volunteers. He was a long standing member of the Innominate Club.

He is survived by his wife Laurie; four children and five grandchildren: Michael (Carol) Caspary; Jeffrey (Britt) Caspary and their daughter, Poppy; Beth (William) Davis and their children, Sarah and Mia; Kathryn (Dylan) Dearborn and their children Layla and Lucy; brother Emory (Christina) Clark; sister Carolyn (Gary) Fulcher; and several nieces and nephews.

Funeral services will be held on Thursday, April 25, at 2 p.m. at St. David's Episcopal Church, 14 Jamar Drive, Dewitt, NY. The family will greet friends following the service at the church.

Date of Death: April 18, 2013
Published in Syracuse Post Standard from Apr. 20 to Apr. 21, 2013

FRANK THOMAS PADBERG, SR., M.D.

Frank Thomas Padberg Sr, MD age 96, passed away peacefully surrounded by his immediate family on April 5, 2014. He was born on March 9, 1918 in Canton Oklahoma to Albert F Padberg, the town's general medical practitioner and Mayme Thomas Padberg who taught school.

Graduating Canton High School he attended Wentworth Military Academy. As a professionally trained baritone, he served as Soloist during these years and considered a vocal "career". As the 1937 Honor Graduate from Wentworth he was eligible for appointment to West Point, but elected to pursue a medical career at Northwestern University, Chicago Illinois. He also excelled at this institution, was elected to AOA, the academic medical Honor Society, and graduated in 1943. He was selected for internship at the University of Michigan, Ann Arbor.

Helen Louise Swan, of Konawa Oklahoma, and Frank were married on Feb 6, 1943 and celebrated their 71st anniversary in 2014. As a Captain in the US Army Medical Corps he deployed to Bristol UK, Cherbourg France, and Liege Belgium, from June 1942 to April 1946 with the 298th General Hospital. Returning to Northwestern, he completed a residency in surgery and neurosurgery training with Loyal Davis, author of surgical and neurosurgical textbooks and father of Nancy Reagan.

The family moved to Little Rock bringing the newly formulated specialty of Neurosurgery to the University of Arkansas for Medical Sciences and Little Rock Veterans Affairs Hospitals in 1952. A practice in Neurosurgery was based at St Vincent's Hospital from 1952-1973. During this time he became a Fellow of the American College of Surgeons and served as President of the Arkansas Chapter. The Arkansas State Nurses Association awarded him Honorary Recognition in 1969. He was an active member of Rotary International and The Little Rock Club.

Frank and Helen maintained dual residency in Chicago and Little Rock after he accepted a position as Director of Fellowship and Graduate Medical Education with the American College of Surgeons in 1973. He received the College's Distinguished Service Award in 1988 and retired from the College in 1999. The Surgical Section of the National Medical Association honored him with their Distinguished Service Award that same year. In addition to the ACS, he was a member of other distinguished surgical organizations including the American Surgical Association, the Southern Surgical Association, the Southern Neurosurgical Society and the Congress of Neurological Surgeons.

As an active member of the Second Presbyterian Church, Little Rock, Frank served as an Elder and was an active participant in the Men's Bible class; in Chicago he served as a Deacon in 4th Presbyterian Church, Chicago, Illinois. During church services his projected baritone supplemented the inspiration inherent in the hymns. International travel was a frequent diversion and he managed to visit every continent but Antarctica. He and Frank Jr. went behind the iron curtain to visit eastern Europe and Russia during the era of the cold war. The entire family participated in several reunions with the Familienverband Padberg (the international Padberg family) attending several reunions in St Louis, USA as well as Berlin, Cologne, & Padberg, Germany. Frank maintained a relationship with the families in Canton, Oklahoma and was a contributor to the church. Frank had a distinguished and honest character coupled with a heart for gracious generosity to less fortunate associates and charitable giving.

Frank was predeceased by his older sister Louise Aline Padberg Souders, MD of Rock Island, IL, and both parents. He is survived by his wife, Helen, his daughter, Kristen (Denver CO), his son, Frank Jr, his daughter-in-law, Sharon, and his grandson, Frank III (Berkeley Heights, NJ).

BEN EISEMAN, M.D.

The family and friends of Ben Eiseman congratulate him on a long, productive, and adventurous life, well-lived. Born on November 2, 1917 in St. Louis, MO, his formal education was marked by degrees from John Burroughs School (1935), Yale Universi ty (1939), and Harvard University Medical School (1943). Self-education never ceased. He first served his country as a Lieutenant JG in the US Navy in 1943, on landing crafts as a beach battalion doctor and surgeon. He participated in the Anzio, Norman dy, Peleliu, Philippines, and Okinawa actions. In the days before the Invasion of Normandy, he met a young Englishwoman, Mary Georgina Harding, in Falmouth, England. They were married December 22, 1945. He also served in Viet Nam, and Operation Desert Storm. He retired from the Naval Reserves as a Rear Admiral in 1974. As a surgeon, teacher, researcher, and mentor to generations of physicians, he led many and inspired others. He was the author or co-author of over 450 scientific papers and was principal editor of seven books on general surgery. He facilitated the training of medical students from many nations in American institutions, leading to the proliferation of medical knowledge throughout the world. For these efforts, he received numerous awards and honors, at home and abroad. Professional leadership positions included many with the American College of Surgeons, The American Surgical Association, and the Society of University Surgeons, in addition to being the founding Chairman of the Department of Surgery of the University of Kentucky. Honoraria included fellowships of the Royal College of Surgeons of Thailand and the Royal College of Surgeons of England. As a dedicated outdoorsman and community leader, his non-professional achievements included chairmanship of the Colorado Outward Bound School, the Kent School, and cofounding the Tenth Mountain Trail Association. The Ben Eiseman Hut outside of Vail was built in honor of his contributions to the Association.

Published in Denver Post on Nov. 28, 2012

HARILAOS T. SAKELLARIDES, M.D.

SAKELLARIDES, Harilaos T., M.D., PHD, FAAOS,FACS, FRCS Aug 13,1922 - April 27, 2011 .The worlds of orthopedics and hand surgery have lost one of its giants this past Wednesday with the passing of Dr. Harilaos Sakellarides after a brief hospitalization. Dr. Sakellarides, author of numerous articles concerning techniques of musculoskeletal and hand surgery, and contributor to several of the world's most commonly used orthopedic texts, has been known and renowned among his orthopedics colleagues for more than a half-century. Dr. Sakellarides was born in Greece where he attended medical school, graduating in 1950. He trained as a resident surgeon throughout the decade of the 1950's in Europe's most acclaimed orthopedic programs in both Paris and London, before completing fellowship training at the Hospital for Special Surgery in New York. In 1958, he joined several other Greek and Greek-American surgeons as a founder of the Hellenic Hippocratic Orthopedic Society - where he would later serve as President. He was soon invited to the Mass. General Hospital, where he was one of the founders of the Department of Hand Surgery, and participated as an attending surgeon from 1959 thru 1964. In 1963, he was recognized by both colleagues and journalists for his new and innovative techniques, and was featured in a 2-page article in TIME magazine as one of America's future prominent surgeons. He did not disappoint and of his many designations Fellow American Academy of Orthopedic Surgeons, Fellow Royal College of Surgeons and Hall of Fame, Fellow American College of Surgeons, Pioneer of Hand Surgery -International College of Surgeons, Francais, Academie de Surgerie to name a few. n 1965, he joined the staff at Boston University Medical Center. Assistant Clinical Professor of both Hand and Orthopedic Surgery for the next 3 decades. During this time, he also served as Chief of Hand Surgery at Franciscan Children's Hospital, past President and presently Vice Chairman of the American Academy of Neurologic and Orthopedic Surgeons, and Visiting Professor of Hand/Orthopedic at teaching hospitals in Greece, among them Ioannina Medical School and University of Larissa, and in India, Roumania It has been estimated that he performed more than 20,000 orthopedic and hand surgeries during his career, which spanned more than 6 decades. He was a member of numerous prestigious medical societies, and his resume lists his participation in 39 international and national organizations and societies. He traveled tirelessly to medical conferences around the world. He was dedicated to the welfare and recovery of his patients, welcoming friends and old patients into his office until just days before his passing. His motto was to do no harm. He leaves behind his wife Loukia and daughter Joanne, son Theodore H. of Chestnut Hill, daughter Maria and her husband Dr. Yianni Vlachiotis of Ekali Greece.

Published in The Boston Globe on May 1, 2011

WILLIAM WEI-LIEN SHAW, M.D.

William Wei-Lien Shaw was born March 12, 1942, in China in the middle of World War II, a period of cataclysmic political turmoil. The country had been occupied by the Japanese, and his father, an official in the government of General Chiang Kai-Shek, moved the family throughout China along with other members of the Nationalist government. His family later fled to Taiwan following the Communist revolution and Mao's assumption of power in 1949

Bill attended middle school in Taipei but the family emigrated to Los Angeles when his father joined the faculty of the University of California, Los Angeles. He graduated from Los Angeles High School in 1960 and received his A.B. from the University of California, Los Angeles in 1964 in Biology/Chemistry. He was awarded his M.D. from the University of California, Los Angeles in 1968 and was president of his class.

His introduction to New York came in 1968, when he interned in medicine at the Albert Einstein Medical Center. He then completed a general surgical residency at the University of California, Los Angeles, interrupted by 2 years as a U.S. Army medical officer in orthopedic surgery in Thailand.

He returned to the University of California, Los Angeles in 1989 as Professor and Chief of the Division of Plastic and Reconstructive Surgery. All of us at New York University regretted his departure but were excited for him, as he genuinely wanted to be chief of an academic plastic surgery service. For me, it was a personal loss because we worked so effectively together. Over 15 years, we had become friends and our families had shared several memorable international trips.

He brought to the University of California, Los Angeles a new vision for the education of plastic surgeons and instituted major changes in the didactic part of the curriculum. He developed a premier plastic surgery training program, especially strong in microsurgery, craniofacial surgery, and hand surgery, a residency that attracted the top resident applicants.

He also established one of the first comprehensive breast cancer centers staffed by a full-time faculty. It served as a model for other centers in academic medical centers around the country.

His passions were China, microsurgery, and teaching. I was part of a historic trip to China in 1981. Bill made it all possible, as he was the only American plastic surgeon who spoke Mandarin Chinese. There were other trips to Taiwan, and he guided me through the National Palace Museum on two occasions, proudly pointing out the highlights of the Imperial art treasures. He loved hosting Chinese banquets and would always choose the more unusual or exotic items from the menu.

He was ever the teacher and won the Institute Teacher of the Year Award on several occasions. After he became Chief at the University of California, Los Angeles, he continued his commitment to the training of plastic surgery residents and microsurgery fellows.

His professional accomplishments in microsurgery are enormous. He trained 36 microsurgery fellows, all of whom remained close to him and many of whom have assumed academic leadership positions. He wrote over 170 peer-reviewed journal publications, and many remain classics: lower extremity salvage, autologous breast reconstruction, the gluteus maximus flap, microvascular free flaps, monitoring of microvascular anastomotic patency, head and neck reconstruction, and prefabrication of composite tissue free flaps.

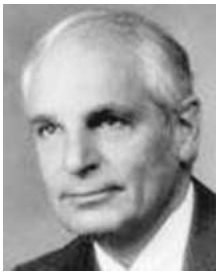
His legacy, however, continues. A microsurgery service at New York University will always bear his mark. All of the faculty, residents, and fellows at New York University and the University of California, Los Angeles, whose lives he touched, will forever be grateful for his teaching and friendship and will always carry special memories of him.

WILLIAM MARTIN STAHL M.D.

WILLIAM MARTIN STAHL, MD - LARCHMONT, N.Y. - William Martin Stahl, MD, passed away on Dec. 22, 2012, at age 90. Bill and his family lived in Burlington for many years, beginning in 1962. Bill was a practicing surgeon, an academic, scientist, master teacher, and lifelong musician, who dedicated much of his professional life to the cause of providing quality health care for the poor. He attended Dartmouth College and Harvard Medical School, graduating from each in three years under the U.S. Navy's wartime accelerated program. He was later commissioned as a captain in the U.S. Army Medical Corps (1951-52) and stationed in the Marshall Islands at Eniwetok Atoll under the auspices of the U.S. Atomic Energy Commission. He received a commendation for his outstanding performance while commanding officer of a mobile surgical hospital - the only doctor for nearly 1,000 men stationed on the island. During his long career, he labored for more than 30 years in New York City hospitals. His residency at Bellevue Hospital (1952-1955) opened his eyes to what was possible and seeded his desire to teach and conduct research. After practicing surgery in Danbury, Conn., he taught at the University of Vermont Medical School (1962-1966) as Associate Professor of Surgery and Vice Chairman of the Department of Surgery. Returning to New York City in 1966, he became a member of the faculty of New York University Medical School and directed one of the surgical services at Bellevue Hospital. In 1977, he joined the faculty at New York Medical College and was appointed Chief of Surgery at Metropolitan Hospital and, in 1980, Director of Surgery at Lincoln Medical and Mental Health Center, where he remained until his retirement in 1997. He became a leader in the fields of trauma and critical care medicine, and his medical publications numbered in the hundreds. He was considered a master teacher by his peers and the legions of students and residents whom he trained. He played clarinet and sang in the Barbary Coast at Dartmouth College, the well-respected Big Band of the time. He later played clarinet with the Hudson Valley Wind Symphony and played soprano sax with the Westchester Saxophone Quartet. His true love was playing tenor sax for 15 years with the Wednesday Night Big Band. He never wasted a moment of his life. He is mourned by his wife, Patricia Maloney; their children, Matyas Stahl and Elizabet Stahl, of Larchmont and Montauk, N.Y.; the children from his first marriage, all of whom attended Burlington public schools, William M. Stahl III "Skip" of Portland, Maine, Katherine A. Stahl of Washington, D.C., and Springs, N.Y., Sarah (Stahl) Jaffe Turnbull of Bridgehampton, N.Y., and Elizabeth Stahl Parkinson of Phoenix, Ariz.; seven grandchildren; four great-grandchildren; and brother, Frederick A. Stahl "Tad" of Boston, Mass. He was predeceased by his son, Jonathan Stahl; and two wives, Mary Elizabeth (Betts) Stahl, and Alice Miller.

Published in The Burlington Free Press on Jan. 28, 2013

JOHN ANTON WALDHAUSEN, M.D.



Dr. Waldhausen was born on May 22, 1920, in New York City as an American citizen to German parents. He grew up in war-torn Germany and returned to the United States nearly penniless in 1947. He benefited from the great kindness of Monsignor Werner, a U.S. Army chaplain whom he met in Germany and who secured for him a train ticket to Montana and a scholarship to the College of Great Falls. He attended medical school at St. Louis University and Johns Hopkins. While at Johns Hopkins, he met Marian Rodney Trescher, who became his beloved wife of 54 years and who preceded him in death on Feb. 14 of this year. Following the completion of his surgical training, he joined the faculty at Indiana University as a cardiac surgeon and while there, developed the subclavian flap angioplasty that for many years became the standard treatment for coarctation of the aorta. His procedure lowered the mortality of the disease from nearly 60 percent to 3 percent. He then moved to the Children's Hospital of Philadelphia (CHOP) and, at the request of C. Everett Koop, developed CHOP's congenital heart program. In 1969, he became the founding chairman of surgery at the newly created Pennsylvania State University College of Medicine in Hershey, Penn., where he led the department for 25 years. From 1972-73 he also served as interim provost and dean of the school of medicine. Dr. Waldhausen was a member of the University of Great Falls Board of Trustees from 2001-2004.

Dr. Waldhausen made significant contributions to the medical field, including over 275 articles, book chapters and books, and helped push the frontiers in congenital heart surgery, as well as artificial organs, with the artificial heart and ventricular assist devises. He also served as the first vice president of the American Surgical Association, the president of the American Association for Thoracic Surgery, the Society of Clinical Surgery and the Thoracic Surgery Directors Association. He served as the editor of the Journal of Thoracic and Cardiovascular Surgery from 1995-2000 and also served on the editorial board of the Journal of Pediatric Surgery. He directed the general and cardiac surgery training programs at Penn State for many years and was a mentor to countless faculty, residents and students during his tenure. Many of his faculty went on to be department chairs and leaders in their surgical fields.

Dr. Waldhausen's work was an inseparable part of him and his family. Providing unwavering support and enthusiasm was his wife Marian. They were completely devoted to each other. They were a team that revealed itself to all who met them: independent, but each at the other's side in everything. They had three sons and instilled their strengths in them by example. His enduring love of reading, history, music and the outdoors as a way to involve his family will forever be a part of us.

He is survived by his sons and their wives John H.T. and Julie, Robert R., and A. Gordon S. and Sherry Waldhausen and his grandchildren, Tessa Mosley, Liesl, Cole, Neil and Henry Waldhausen.

Published in Great Falls Tribune on June 21, 2012

R. DONALD WOODSON, M.D.

Dr. R. Donald Woodson, a surgeon and an early faculty member of the former Medical College of Ohio, died Monday in the Jane Baker House, a skilled care home at the Otterbein North Shore community, Lakeside, Ohio. He was 80. He had cirrhosis of the liver caused by exposure to hepatitis B in his surgical career, his wife, Sharon, said. Dr. Woodson of Ottawa County's Catawba Island Township was also a University of Toledo law school graduate. He was admitted to the Ohio bar in 1984 and reviewed medical malpractice cases for a Port Clinton law firm.

He retired from medicine in the late 1990s after he returned from southern California, where he was on the staff of several hospitals. He became associate professor of surgery at MCO, the current UT Medical Center, on Jan. 1, 1969, hired the previous November by MCO trustees. "He really liked to teach. He wanted to share because he was so enthusiastic," his wife said. "His philosophy [was] there is always a cure." He helped run conferences on chest surgery, recalled Dr. Barney Wisinger, a pulmonologist, an MCO faculty member, and a friend.

"He was very well trained," said Dr. Wisinger, former director of a teaching program at Toledo Hospital. "We got along very well together." Dr. Woodson eschewed painkillers and advocated electrical nerve stimulation devices for relief. He encouraged patients to get moving early in their recovery. "He walked patients down the hall himself with his 3-inch-heel cowboy boots," his wife said. He was on the medical staff at Toledo Hospital, St. Luke's Hospital in Maumee, Bellevue Hospital, Magruder Hospital in Port Clinton, and Firelands Regional Medical Center in Sandusky.

He was born Dec. 24, 1931, in Winfield, Kan., to Ruth and Riley D. Woodson. He was salutatorian of his 1949 class at Shawnee Mission High School. He played basketball as an undergraduate at the University of Kansas and was a 1956 graduate of its medical school. He was an assistant professor of surgery at the University of Illinois medical college when MCO hired him. He called his Navy service as a medical officer aboard the USS Constellation the best time of his life, his wife said. "He was a guy's guy." He liked to sketch and play the piano. He fished in Alaska, Canada, and Mexico, and hunted in Wyoming.

"He was a Mensa man all the way around," his wife said. "There wasn't a subject I could ask that man he didn't know." He was formerly married to Virginia Nalley Woodson and Donna Woodson.

Surviving are his wife, Sharon Pocisk Woodson, whom he married Dec. 31, 1991; sons, Riley D. and Wade C. Woodson; stepson, Jeffrey Pocisk; stepdaughters, Sheri Sobel and Monica Morgan; stepmother, Virginia Maria Woodson; sister, Marjorie Woodson Brownlee, and three grandsons.

Private family services were to be held Saturday in Kansas City, Mo. The family suggests tributes to the Humane Society of Ottawa County.

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AAST
American Association for the Surgery of Trauma
Research and Education Foundation
Promoting and advancing the optimal care of injured and critically ill surgical patients, and inspiring the next generation of surgeons.

73rd Annual Meeting of AAST and Clinical Congress of Acute Care Surgery
Philadelphia, PA - September 10-13, 2014

WED. 9/10/2014	FUNCTION	ROOM
6:30 AM – 5:30 PM	Registration	Grand Ballroom Foyer
7:00 AM – 11:00 AM	Optional Session: ACS-MOC	Grand Ballroom Salons G-L
7:00 AM – 11:00 AM	Optional Session: Military Symposium	Grand Ballroom Salon B
7:00 AM – 11:30 AM	Optional Session: Pediatric Committee Session	Grand Ballroom Salon F
7:00 AM – 11:00 AM	Optional Session: Coalition for Trauma Care Symposium	Grand Ballroom Salon A
7:00 AM – 12:00 PM	Optional Session: TACTIC Meeting	Conference Rooms 401-402
10:30 AM – 12:15 PM	Optional Session: Social Media 101 Tweeting and Facebook	Conference Room 310
12:30 PM – 1:00 PM	Welcome	Grand Ballroom Salons G-L
1:00 PM – 3:40 PM	Session I: Plenary Papers 1-8	Grand Ballroom Salons G-L
3:40 PM – 4:10 PM	Session II: Master Surgeon Lecture I: Alex Valadka, MD	Grand Ballroom Salons G-L
4:10 PM – 5:25 PM	Session III: Challenging Cases Panel I	Grand Ballroom Salons G-L
5:30 PM – 7:30 PM	Session IV: Poster Session & Exhibit Hall Opening	Franklin B
6:30 PM – 8:30 PM	*Journal of Trauma & Acute Care Surgery* Editorial Meeting	Grand Ballroom Salon C
THURS. 9/11/2014	**FUNCTION**	**ROOM**
6:15 AM – 7:30 AM	Resident, Medical Student & In-Training Fellow Breakfast (Ticketed)	Grand Ballroom Salon C
6:15 AM – 7:30 AM	Critical Care Committee Meeting	Conference Rooms 401- 402
6:15 AM – 7:30 AM	Acute Case Surgery Committee Meeting	Conference Room 404
6:15 AM – 7:30 AM	International Relations Committee Meeting	Conference Room 406
6:15 AM – 7:30 AM	Prevention Committee Meeting	Conference Rooms 407-408
6:15 AM – 7:30 AM	Multi-Institutional Trials Committee Meeting	Conference Rooms 411-412
6:15 AM – 7:30 AM	Disaster Ad Hoc Committee Meeting	Conference Rooms 414-415
7:00 AM – 8:30 AM	Continental Breakfast	Franklin B
7:00 AM – 3:30 PM	Exhibits and Posters	Franklin B
6:30 AM – 4:00 PM	Registration	Grand Ballroom Foyer
7:30 AM – 8:00 AM	Session V: Master Surgeon Lecture II: Andrew Peitzman, MD	Grand Ballroom Salons G –L
8:00 AM – 9:20 AM	Session VI: Plenary Papers 9-12	Grand Ballroom Salons G –L
9:20 AM – 9:40 AM	Session VII: Scholarship Presentations	Grand Ballroom Salons G –L
9:40 AM – 10:00 AM	Break	Franklin B
10:00 AM – 11:20 AM	Session VIII: Papers 13-16	Grand Ballroom Salons G –L
11:30 AM – 12:30 PM	Session IX: Presidential Address, William G. Cioffi, MD	Grand Ballroom Salons G –L
12:30 PM – 1:45 PM	Lunch Sessions	See Ticket for Location
1:45 PM – 2:00 PM	Break	Franklin B
2:00 PM – 5:00 PM	Session XA: Papers 17-25	Grand Ballroom Salons G - L
2:00 PM – 5:00 PM	Session XB: Papers 26-34	Grand Ballroom Salons A, B & F
FRI. 9/12/2014	**FUNCTION**	**ROOM**
6:15 AM – 7:30 AM	Military Liaison Committee Meeting	Conference Room 401-402
6:15 AM – 7:30 AM	Injury Assessment Committee Meeting	Conference Room 404
6:15 AM – 7:30 AM	Publications and Communications Committee Meeting	Conference Room 406
6:15 AM – 7:30 AM	Geriatric Trauma Committee Meeting	Conference Rooms 407-408
6:15 AM – 7:30 AM	Pediatric Committee Meeting	Conference Rooms 411-412
6:15 AM – 7:30 AM	Education/CME Committee Meeting	Conference Rooms 414-415
6:15 AM – 7:30 AM	ACS Program Directors Meeting	Conference Room 403
6:15 AM – 7:30 AM	International Attendees Breakfast (Ticketed)	Grand Ballroom Salon C
7:00 AM – 8:30 AM	Continental Breakfast	Franklin B
7:00 AM – 2:00 PM	Exhibits and Posters	Franklin B
7:00 AM – 3:00 PM	Registration	Grand Ballroom Foyer
7:30 AM – 8:00 AM	Session XI: Demystifying Government Research Panel II	Grand Ballroom Salons G-L
8:00 AM – 10:55 AM	Session XII: Quick Shots	Grand Ballroom Salons G-L
10:55 AM – 11:15 AM	Break	Franklin B
11:15 AM – 12:15 PM	Session XIII: Fitts Lecture: Ronald Tompkins, MD	Grand Ballroom Salons G-L
12:15 PM – 1:30 PM	Lunch Sessions	See Ticket for Location
1:30 PM – 4:50 PM	Session XIVA: Papers 35-44	Grand Ballroom Salons G-L
1:30 PM – 4:50 PM	Session XIVB: Papers 45-54	Grand Ballroom Salons A, B & F
4:50 PM – 5:00 PM	Military Awards	Grand Ballroom Salons G-L
5:00 PM – 6:15 PM	AAST Annual Business Meeting	Grand Ballroom Salons G-L
7:30 PM – 8:00 PM	Reception	Grand Ballroom Foyer
8:00 PM – 10:00 PM	Banquet (Black Tie) Ticketed Event	Grand Ballroom Salons A-F
SAT. 9/13/2014	**FUNCTION**	**ROOM**
7:00 AM – 8:00 AM	New Fellows Breakfast (Ticketed Event)	Conference Rooms 411-412
7:30 AM – 10:00 AM	Registration	Grand Ballroom Foyer
8:00 AM – 12:00 PM	Session XV: Papers 55-66	Grand Ballroom Salons G - L

SPEAKER READY ROOM:
Grand Ballroom Level Room 502
Tuesday, September 9th – 4:00 PM – 7:00 PM
Wednesday, September 10th – 6:30 AM – 5:00 PM
Thursday, September 11th – 7:00 AM – 5:00 PM
Friday, September 12th – 7:00 AM – 5:00 PM
Saturday, September 13th – 7:00 AM – 10:00 AM