<table>
<thead>
<tr>
<th>President</th>
<th>Year</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Robert G. Volz, M.D.</td>
<td>1971</td>
<td>Vail</td>
</tr>
<tr>
<td>Robert G. Volz, M.D.</td>
<td>1972</td>
<td>Vail</td>
</tr>
<tr>
<td>Peter V. Teal, M.D.</td>
<td>1973</td>
<td>Aspen</td>
</tr>
<tr>
<td>William R. Haas, M.D.</td>
<td>1974</td>
<td>Sun Valley</td>
</tr>
<tr>
<td>Arthur M. McGuire, M.D.</td>
<td>1975</td>
<td>Snowmass</td>
</tr>
<tr>
<td>Lynn Ketchum, M.D.</td>
<td>1976</td>
<td>Park City</td>
</tr>
<tr>
<td>Fred C. Chang, M.D.</td>
<td>1977</td>
<td>Steamboat</td>
</tr>
<tr>
<td>Glen D. Nelson, M.D.</td>
<td>1978</td>
<td>Snowmass</td>
</tr>
<tr>
<td>Gerald D. Nelson, M.D.</td>
<td>1979</td>
<td>Snowbird</td>
</tr>
<tr>
<td>Kevin G. Ryan, M.D.</td>
<td>1980</td>
<td>Jackson H</td>
</tr>
<tr>
<td>David S. Bradford, M.D.</td>
<td>1981</td>
<td>Vail</td>
</tr>
<tr>
<td>Erick R. Ratzer, M.D.</td>
<td>1982</td>
<td>Jackson H</td>
</tr>
<tr>
<td>William R. Olsen, M.D.</td>
<td>1983</td>
<td>Steamboat</td>
</tr>
<tr>
<td>Earl G. Young, M.D.</td>
<td>1984</td>
<td>Snowbird</td>
</tr>
<tr>
<td>Robert B. Rutherford, M.D.</td>
<td>1985</td>
<td>Sun Valley</td>
</tr>
<tr>
<td>Rudolph A. Klassen, M.D.</td>
<td>1986</td>
<td>Jackson H</td>
</tr>
<tr>
<td>Robert J. Neviaser, M.D.</td>
<td>1987</td>
<td>Steamboat</td>
</tr>
<tr>
<td>Robert C. Edmondson, M.D.</td>
<td>1988</td>
<td>Snowbird</td>
</tr>
<tr>
<td>Ernest E. Moore, M.D.</td>
<td>1989</td>
<td>Crested Bc</td>
</tr>
<tr>
<td>Stephen W. Carveth, M.D.</td>
<td>1990</td>
<td>Jackson H</td>
</tr>
<tr>
<td>George E. Pierce, M.D.</td>
<td>1991</td>
<td>Steamboat</td>
</tr>
<tr>
<td>Peter Mucha, Jr., M.D.</td>
<td>1992</td>
<td>Snowbird</td>
</tr>
<tr>
<td>David V. Feliciano, M.D.</td>
<td>1993</td>
<td>Crested Bc</td>
</tr>
<tr>
<td>R. Chris Wray, M.D.</td>
<td>1994</td>
<td>Big Sky</td>
</tr>
<tr>
<td>David Kappel, M.D.</td>
<td>1995</td>
<td>Grand Targ</td>
</tr>
<tr>
<td>Thomas H. Cogbill, M.D.</td>
<td>1996</td>
<td>Snowbird</td>
</tr>
<tr>
<td>G. Jerry Jurkovich, M.D.</td>
<td>1997</td>
<td>Snowbird</td>
</tr>
</tbody>
</table>

***The 1998 WESTERN TRAUMA ASSOCIATION MEETING will be held at Chateau Lake Louise, Banff, Alberta, Canada, February 22-28, 1998***
OFFICERS:
G. Jerry Jurkovich, M.D. President
James Benjamin, M.D. President-Elect
Herbert Thomas, M.D. Vice President
James A. Edney, M.D. Secretary
Barry C. Eshrig, M.D. Treasurer
Gerald Gussack, M.D. Historian

BOARD OF DIRECTORS:
Bruce G. Ferris, M.D. TERM ENDS
R. Lawrence Reed, II, M.D. 1997
Steven Shackford, M.D. 1997
Fred Moore, M.D. 1998
William Iannacone, M.D. 1999
Dwight A. Webster, M.D. 1999

PROGRAM COMMITTEE:
Alexander S. Rosemurgy II, M.D. Chairman
Bruce H. Broecker, M.D.
James W. Davis, M.D.
Larry Gentilello, M.D.
William Iannacone, M.D.
Jeffrey Landercasper, M.D.
Jeffry A. Metheny, M.D.
Steven L. Wald, M.D.
G. Jerry Jurkovich, M.D., ex-officio

PUBLICATIONS COMMITTEE:
Steven R. Shackford, M.D. Chairman
Jon M. Burch, M.D.
Stephen M. Cohn, M.D.
James C. Hebert, M.D.
Robert E. Kearney, M.D.
Margaret Knudson, M.D.
John W. McGill, M.D.
H. Leon Pachter, M.D.
Patrick J. Offner, M.D.
R. Lawrence Reed II, M.D.
Steven E. Ross, M.D.
David W. Tuggle, M.D.
Dennis W. Vane, M.D.
Steve Wald, M.D.
John R. Zelko, M.D.

NOMINATING COMMITTEE:
Thomas H. Cogbill, M.D. Chairman
Bruce G. Ferris, M.D.
H. Leon Pachter, M.D.
Thomas F. Phillips, M.D.
<table>
<thead>
<tr>
<th>Resident</th>
<th>University</th>
<th>Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Joseph Schmocker, M.D.</td>
<td>University of Vermont</td>
<td>1991</td>
</tr>
<tr>
<td>Joseph Schmocker, M.D.</td>
<td>University of Vermont</td>
<td>1992</td>
</tr>
<tr>
<td>Charles Mock, M.D.</td>
<td>University of Washington</td>
<td>1993</td>
</tr>
<tr>
<td>Gino Travisani, M.D.</td>
<td>University of Vermont</td>
<td>1994</td>
</tr>
<tr>
<td>Phillip C. Ridings, M.D.</td>
<td>Medical College of Virginia</td>
<td>1995</td>
</tr>
<tr>
<td>David Hahn, M.D.</td>
<td>Emory University</td>
<td>1996</td>
</tr>
</tbody>
</table>
### Western Trauma Association

#### Schedule of Events

**Sunday, March 2, 1997**
- 24 Hours/Daily: Speaker Ready Room
- 4:00 - 7:00 PM: Registration/NASTAR Sign-up
- 4:00 - 7:00 PM: Welcome Reception
- 4:00 - 7:00 PM: Kids' Reception
- Coat Room
- 2 Ballroom Sections

**Monday, March 3, 1997**
- 6:30 - 7:00 AM: Breakfast*
- 6:30 - 9:00 AM: Registration/NASTAR Sign-up
- 7:00 - 9:00 AM: Scientific Paper Session I
- 4:00 - 6:00 PM: Scientific Paper Session II
- 6:00 - 7:00 PM: Members' Business Meeting
- 1 Ballroom Section
- 2 Ballroom Sections
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section

**Tuesday, March 4, 1997**
- 6:30 - 7:00 AM: Breakfast*
- 7:00 - 8:00 AM: Scientific Paper Session III
- 8:00 - 9:00 AM: Invited Guest Speaker
- Basil Pruitt, M.D.
- "Improvements in Care of the Burn Patient: The Successful Application of Multi-Disciplinary Integrated Biomedical Research"
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section
- Race Hill
- Race Hill
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section

**Wednesday, March 5, 1997**
- 6:30 - 7:00 AM: Breakfast*
- 7:00 - 8:00 AM: Scientific Paper Session V
- 8:00 - 8:30 AM: Margaret Knudson, M.D.
- "Deep Venous Thrombosis: Diagnosis, Prevention, Treatment"
- 8:30 - 9:00 AM: Harvey Sugerman, M.D.
- "Abdominal Compartment Syndrome"
- 4:00 - 5:00 PM: Scientific Paper Session VI
- 5:00 PM: Business Meeting
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section

**Thursday, March 6, 1997**
- 6:30 - 7:00 AM: Breakfast*
- 7:00 - 9:00 AM: Scientific Paper Session VII
- 4:00 - 5:00 PM: Panel Discussion
- 5:00 - 6:00 PM: Scientific Paper Session VIII
- 7:00 PM: Reception/Dinner
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section
- 2 Ballroom Sections

**Friday, March 7, 1997**
- 6:30 - 7:00 AM: Breakfast*
- 7:00 - 9:00 AM: Scientific Paper Session IX
- 4:00 - 6:00 PM: Scientific Paper Session X
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section
- 1 Ballroom Section

*Spouses', Children's, Significant Others' Continental Breakfast served daily from 8-9 AM in the Golden Cliff Room*
MONDAY AM, March 3, 1997

MODERATOR: G. Jerry Jurkovich, M.D.

7:00 AM #1. "Ascorbic Acid Improves Survival and Decreases Pulmonary Neutrophil Sequestration in Hemorrhagic Shock"
Authors: BW Mays, MD, and JR Wallace, MD
Presenter: Bradley W Mays, MD
Medical College of Wisconsin
**Earl Young Resident Competition

7:20 AM #2. "Predicting Prolonged Ventilator Dependence (PVD) in Thermally Injured Patients"
Authors: B Sellers, MD, B Davis, MD, P Larkin, MD, S Morris, MD, J Saffie, MD
Presenter: Barbara J Sellers, MD
Intermountain Burn Center, University of Utah Health Center
**Earl Young Resident Competition

7:40 AM #3. "Early ID of Psychosocial Factors Limiting Outcome Following Traumatic Injury"
Authors: AJ Michaels, MD, CE Michaels, MD, C Moon, MD, MA Zimmerman, PhD, C Peterson, PhD, JL Rodriguez, MD
Presenter: Andrew J. Michaels, MD
Trauma/Burn/Emergency Surgery Division, Department of Psychology and School of Public Health, University of Michigan
**Earl Young Resident Competition

8:00 AM #4. "Modulation of Cytokine Response to Sepsis Through Sublethal Hemorrhage is Neither Cytokine nor Model Specific"
Authors: E Zervos, MD, J Norman, MD, D Watkins, MD, A Rosemurgy, MD
Presenter: Emmanuel E Zervos, MD
Department of Surgery, University of South Florida
**Earl Young Resident Competition

8:20 AM #5. "Ethanol Confounds Base Deficit Estimation of Injury Severity"
Authors: KT Asgarian, MD, RM Price, PhD, KM Kelly, MD
Presenter: K.T Asgarian, MD
Department of Surgery, Morristown Memorial Hospital

8:40 AM #6. "Early Bone Marrow Unresponsiveness to M-CSF and G-CSF Predisposes Burn Patients to Sepsis"
Authors: K Pellegrin, MD, CH Rundus, MT, CE Hartford, MD, XW Wang, MD, VM Peterson, MD
Presenter: Katharina Pellegrin, MD
Department of Surgery, University of Colorado Health Sciences Center and The Children's Hospital, Denver
**Earl Young Resident Competition

9:00 AM Adjourn
**SCIENTIFIC PAPER SESSION II**

MONDAY PM, March 3, 1997

MODERATOR: Alexander S. Rosemurgy II, M.D.

4:00 PM  
**#7. Early Predictors of the Need for Mechanical Ventilation in Blunt Trauma Patients with Pulmonary Contusion**
Authors: J Collinge, MD, J. Tyburski, MD, S Eachempati, MD, R Wilson, MD
Presenter: J Collinge, MD
Wayne State University  
**Earl Young Resident Competition**

4:20 PM  
**#8. Base Deficit is Superior to Ph In Evaluating Clearance of Acidosis After Traumatic Shock**
Authors: JW Davis, MD, KL Kaups, MD, SN Parks, MD
Presenter: James W Davis, M.D.
Valley Medical Center, Department of Surgery

4:40 PM  
**#9. Initial Routine Labs In Trauma Patients are Neither Clinically Relevant nor Cost Effective**
Authors: D Kavouspour, MD, K Baker, MD, G Jans, RN
Presenter: Dar Kavouspour, MD
University of South Florida, Department of Surgery and Tampa General Hospital Trauma Center

5:00 PM  
**#10. L-Arginine Decreases Alveolar Macrophage Proinflammatory Monokine Production in Endotoxin-Induced Acute Lung Injury**
Authors: DR Meldrum, MD, RC McIntyre, Jr., MD, BC Sheridan, MD, DA Fullerton, MD, A Banerjee, PhD, AH Harken, MD
Presenter: Daniel R Meldrum, M.D.
Department of Surgery, University of Colorado, Denver  
**Earl Young Resident Competition**

5:20 PM  
**#11. Resolution of Pneumothorax with Oxygen Therapy is Dose Dependent**
Authors: GJ England, MD, JD Harrah, BS, RC Hill, MD, GA Timberlake, MD, JF Hill, BS, YL Shahan, MS, M Billie
Presenter: Gregory J England, MD
West Virginia University School of Medicine, Department of Surgery, Morgantown, WV  
**Earl Young Resident Competition**

5:40 PM  
**#12. Effects of Hypothermia on Neutrophil Function**
Authors: JJ Filides, S Fisher, MD, CM Sheaff, MD, PhD, JA Barrett, MD
Presenter: John J Filides, MD
Department of Trauma Surgery, Cook County Hospital

6:00 PM  
Adjourn
SCIENTIFIC PAPER SESSION III

TUESDAY AM. March 4, 1997

MODERATOR: Barry C. Esrig, M.D.

7:00 AM  #13.  "Saturation Kinetic Modeling of the Molecular Response to Altered Circulation Using the Michaelis-Menten Equation (MME)"
Authors:  J St. Louis, MD, R Monroe, MD, RL Reed, MD
Presenter:  James St Louis, MD
Duke University Medical Center
**Earl Young Resident Competition

Authors:  MM Aguilar, MD, FD Battistella, MD, JT Owings, MD, SA Olson, K MacColl
Presenter:  Michael M Aguilar, MD
Department of Surgery, University of California, Davis
**Earl Young Resident Competition

7:40 AM  #15.  "An Episode of Prehospital Hypotension in Trauma Patients: A Marker for Just Crying Wolf?"
Authors:  M Aranda, MD, SR Petersen, MD, Colleen Hunt, Brian Nelson, I
Presenter:  Manual Aranda, MD
Trauma Center, St. Joseph's Hospital and Medical Center, and Texas Tech Unh
Health Sciences Center Texas
**Earl Young Resident Competition

8:00 AM  Basil A. Pruitt, M.D.
"Improvements in Care of the Burn Patient: The Successful Application of Multi-Disciplinary Integrated Biomedical Research"

9:00 AM  Adjourn
SCIENTIFIC PAPER SESSION IV

TUESDAY, March 4, 1997

ODERATOR: James Benjamin, M.D.

00 PM
#16. "Immediate Neutrophil Activation after Hemorrhagic Shock and Resuscitation"
Authors: P. Rhee, MD, MPH, D. Burris, MD, E. Pikoulis, MD,
K. Kaufmann, MD, J. McKenzie, PhD
Presenter: P. Rhee, MD, MPH
Alameda Services University of the Health Sciences, Bethesda

12:20 PM
#17. "Secretory Iga Blocks Hypoxia Augmented Bacterial Passage Across MDCK Cell Monolayers"
Authors: L.N. Diebel, MD, D.M. Liberati, MD, W.J. Brown, MD, S.A. Dulchavsky, MD,
T. Painter, B.S., C.A. Digi, PhD
Presenter: Lawrence N. Diebel, MD
Wayne State University, Departments of Surgery, Pathology and Microbiology

1:40 PM
Authors: J.G. Chipman, MD, S.B. Johnson, MD, D. Scanzaroli, R.P.A., J.F. Fortune, MD
Presenter: Jeffrey Chipman, MD
University of Arizona Health Sciences Center
**Earl Young Resident Competition

2:00 PM
G. Jerry Jurkovich, M.D.
Presidential Address - "Paint the Ceiling"

3:00 PM
Board of Directors Meeting
SCIENTIFIC PAPER SESSION V

WEDNESDAY AM, March 5, 1997

MODERATOR: James A. Edney, M.D.

7:00 AM    #19. "Randomized Prospective Evaluation of the Effects of Increased Preload Cardiopulmonary Function and Visceral Perfusion During Resuscitation"
Authors: PR Miller, MD, MC Chang, MD, JW Meredith, MD
Presenter: Preston R Miller, MD
The Bowman Gray School of Medicine, Wake Forest University
**Earl Young Resident Competition

7:20 AM    #20. "The Effect of a Quality Assurance - Derived Trauma Airway Policy on Reducing Provider-Related Errors in the Emergency Department"
Authors: JG Cushman, MD, GS Rozycki, MD, DV Feliciano, MD
Presenter: JG Cushman, MD
Grady Memorial Hospital/Emory University
**Earl Young Resident Competition

7:40 AM    #21. "Effects of Exogenous Cytokines on Intravascular Clearance of Bacteria In Normal and Splenectomized Mice"
Authors: JC Hebert, MD, M O'Reilly, MD, B Barry, L Shatney, K Sartorelli, MD
Presenter: James C Hebert, MD
University of Vermont College of Medicine

8:00 AM    Margaret Knudson, M.D.
"Deep Venous Thrombosis: Diagnosis, Prevention, Treatment"

8:30 AM    Harvey Sugerman, M.D.
"Abdominal Compartment Syndrome"

9:00 AM    Adjourn
SCIENTIFIC PAPER SESSION VI

WEDNESDAY PM, March 5, 1997

MODERATOR: William M. Iannacone, M.D.

1:00 PM  #22. "Nonoperative Management of Splenic Injury: Are Follow-Up CT Scans of Any Value?"
Authors: BC Thaemert, MD, TH Cogbill, MD, PJ Lambert, RN, CH Miller, III, MD
Presenter: Bradley C Thaemert, MD
Gundersen Lutheran Medical Center
**Earl Young Resident Competition

1:20 PM  #23. "A Cost Effective Method for Bedside Insertion of Vena Caval Filters In Trauma Patients"
Authors: CR Nunn, MD, D Neuzil, MD, JG Bass, BA, R Pierce, RN, JA Morris, Jr., MD
Presenter: Craig R. Nunn, MD
Vanderbilt University Medical Center
** Earl Young Resident Competition

1:40 PM  #24. "Fasciotomy, Chronic Venous Insufficiency and the Calf Muscle Pump"
Authors: K Bermudez, MD, M Knudson, MD, D Morabito, RN, MPH, O Kessel, BS
Presenter: Kenneth M. Bermudez, MD
San Francisco General Hospital, University of California, San Francisco
** Earl Young Resident Competition

5:00 PM  Business Meeting
THURSDAY AM, March 6, 1997

MODERATOR: James W. Davis, M.D.

7:00 AM  
#25. "Significance of Free Fluid on Abdominal CT"
Authors:  K Brasel, MD, C Olson, MD, R Stafford, MD, T Johnson, MD
Presenter: Thomas J Johnson, MD
St. Paul-Ramsey Medical Center, University of Minnesota
**Earl Young Resident Competition

7:20 AM  
Authors:  JT Tokish, MD, JB Benjamin MD, F Walter, MD
Presenter: JT Tokish, MD
The University of Arizona Health Sciences Center, Department of Surgery, Section Orthopedic Surgery
**Earl Young Resident Competition

7:40 AM  
#27. "Cost Analysis of Pediatric Trauma Care by Cost Centers and Macha Injury as a Basis for Injury Prevention and Clinical Guidelines"
Authors:  K Achanta, MD, Q Chu, MD, J Garby, MS, B Simon, MD, T Emhoff, MD, V Fiallo, MD, KF Lee, MD
Presenter: K Achanta, MD
Baystate Medical Center VA
**Earl Young Resident Competition

8:00 AM  
#28. "Ultrasound is Cost-Effective Triage Tool to Evaluate Blunt Abdominal Trauma in the Pediatric Population"
Authors:  DA Partrick, MD, DD Bensard MD, EE Moore, MD, FM Karrer, MD, SJ Terry, BSN
Presenter: David A Partrick, MD
Denver Health Medical Center, University of Colorado
**Earl Young Resident Competition

8:20 AM  
#29. "Hypertonic Saline is Not Beneficial for Resuscitation of Pulmonary Contusion"
Authors:  SM Cohn, MD, BT Fisher, BS, AT Rosenfield, MD
Presenter: Stephen M Cohn, MD
Departments of Surgery and Radiology, Yale University School of Medicine

8:40 AM  
#30. "Diagnostic Laparoscopy for the Evaluation of Penetrating Abdominal Trauma"
Authors:  JE Mazuski, MD, PhD, MJ Shapiro, MD, DL Kaminski, MD, AC Andrus, MD, M Keegan, RN, W Luchtefeld, RN, RM Durham, I
Presenter: Rodney M Durham, MD
Department of Surgery, St. Louis University

9: 00 AM  
Adjourn
SCIENTIFIC PAPER SESSION VIII

THURSDAY PM, March 6, 1997

4:00 PM  
PANEL DISCUSSION

MODERATOR: Fred Moore, M.D.

5:00 PM  
#31. "Content Analysis of Pediatric Trauma in the Media"
Authors: EJ Doolin, MD, AM Browne, MSN
Presenter: Anne M Browne, MSN
Cooper Hospital/University Medical Center, Camden, NJ

5:20 PM  
#32. "Neurosurgeons and Neurotrauma: Facts and Myths"
Authors: TJ Esposito, MD, AM Kuby, AM, AS Zelby, MD, C Unfred, NA,
          HL Young, MS, RL Gamelli, MD
Presenter: Thomas J Esposito, M.D.
Loyola University Medical Center

5:40 PM  
#33. "Management of Self-Inflicted Low-Energy Gunshot Wounds that Violate the Skull Base"
Authors: DJ Kriet, MD, RB Stanley, MD, MS Grady, MD
Presenter: David J Kriet, MD
Harborview Medical Center, University of Washington School of Medicine

6:00 PM  
Adjourn
SCIENTIFIC PAPER SESSION IX

FRIDAY AM, March 7, 1997

MODERATOR: Larry Gentilello, M.D.

7:00 AM  #34. "Patients with Gunshot Wound to the Head Do Not Require Cervical Spine Immobilization and Evaluation"
Authors: KL Kaups, MD, JW Davis, MD
Presenter: KL Kaups, MD
Valley Medical Center, Fresno

7:20 AM  #35. "Compelling Injuries: The Focus of Trauma Triage, Systems, and Care"
Authors: HR Champion, FRCS (Edin), CM Cushing, MD, AC Maltiaris, PhD, WJ Sacco, PhD, LV Lombardo, BS
Presenter: Howard R. Champion, FRCS (Edin)
National Study Center for Trauma and EMS University of Maryland at Baltimore

7:40 AM  #36. "Maxillofacial Injuries in the National Hockey League"
Authors: GL Lanzi, DMD, JE Burke, DMD, AJ Fedeli, DMD
Presenter: GL Lanzi, MD
Cooper Hospital/University Medical Center

8:00 AM  #37. "Cardiopulmonary and Visceral Effects of Operative Decompression on Patients with Intra-Abdominal Hypertension"
Authors: MC Chang, MD, PR Miller, MD, JW Meredith, MD
Presenter: Michael C Chang, MD
The Bowman Gray School of Medicine, Wake Forest University

8:20 AM  #38. "Abdominal Ultrasound is Not a Sensitive Detector of Intra-Abdominal Injury"
Authors: KF O'Malley, MD, H Sariol MD, G Cains, MD, M DiMarcangelo, DI SE Ross, MD
Presenter: Cooper Hospital/University Medical Center
Cooper Hospital/University Medical Center

8:40 AM  #39. "Test-Based Improvements for Mobile Field Surgical Teams"
Authors: AT Putnam, MD, R Karulfl, MD, A Olsewski, MD, D Bradley, PhD, D Kissinger, MD
Presenter: A Tyler Putnam, M.D.
Wilford Hall Medical Center, San Antonio

9:00 AM Adjourn
RIDAY PM, March 7, 1997

ODERATOR: Thomas H. Cogbill, M.D.

8:00 PM #40. “A Changing Perspective of Odontoid Fractures: Implications for Evaluation and Management”
Authors: W Iannacone, MD, J Naranja, MD, WG DeLong, Jr, MD,
C Born, MD, R Dalsey, MD, L Deutsch, MD, J Catalano, MD
Presenter: William M Iannacone, MD, Ph D
Cooper Hospital/University Medical Center

8:20 PM #41. “The Efficacy of the Motor Component of the Glasgow Coma Scale in Trauma Triage”
Authors: SE Ross, MD, C Leipold, MS, C Terrigino, MD, K O’Malley, MD
Presenter: Steven E Ross, MD
Cooper Hospital/University Medical Center

8:40 PM #42. “Evaluation of Alpine Skiing and Snowboarding Injury in a Northeastern State”
Authors: DE Sacco, K Sartorelli, MD, DW Vane, MD
Presenter: Diane E Sacco, MD
University of Vermont College of Medicine

9:00 PM #43. “Can Surgeons Evaluate Emergency Ultrasound Scans for Blunt Abdominal Trauma?”
Authors: MG McKenney, MD, L Fernandez, MD, D Shatz, MD, N Namiot, MD,
D Levi, MD, A Arrillaga, MD, L Martin, MD, J Moylan, MD
Presenter: Mark G McKenney, MD
University of Miami

Authors: DJ McMahon, MBBS, MD Nance, MD, MD Grossman, MD, AK May, MD
DR Kauder, MD, CW Schwab, MD
Presenter: Damian J. McMahon, MD
Division of Trauma and Surgical Critical Care, Hospital of the University of Pennsylvania, Philadelphia

9:40 PM #45. “Randomized Multicenter Prospective Tracheostomy Study”
Authors: HJ Sugerman, MD, FR Pasquale, MD, KF O’Malley, MD
Presenter: Harvey J. Sugerman, MD
Western Trauma Association Multicenter Study Group Medical College of Virginia, Virginia Commonwealth University, Richmond

6:00 PM Adjourn
ASCORBIC ACID IMPROVES SURVIVAL AND DECREASES PULMONARY NEUTROPHIL SEQUESTRATION IN HEMORRHAGIC SHOCK

B.M. Mays, M.D. and J.R. Wallace, M.D., PhD
Medical College of Wisconsin
Milwaukee, WI
B.M. Mays, M.D.
Charles Aprahamian
Milwaukee, WI

OBJECTIVE: To test if the aqueous antioxidant ascorbic acid (AA) given during resuscitation in a model of severe hemorrhagic shock will improve survival and prevent neutrophil sequestration in the lung.

METHODS: Fasted, male Sprague Dawley rats were anesthetized with halothane and body temperature was maintained at 37 degrees Celsius with a warming pad. The femoral artery and vein were cannulated and hemorrhagic shock maintained for one hour at 25mmHg with a gravitational column in the absence of heparin. After one hour of shock the animals (n = 15 in each arm) were randomized by card selection into two groups: 1) 0.9% saline alone or 2) 1 gm/kg AA in isotonic solution and survival was assessed at 72 hours. Total resuscitation volume was 40 ml/kg. Separate animals were treated as above but were sacrificed one hour after resuscitation to evaluate pulmonary neutrophil sequestration utilizing a myeloperoxidase spectrophotometric assay (MPO). Sham operated animals were anesthetized and MPO activity assessed units/gm wet tissue).

RESULTS: Systolic blood pressure and final hematocrit prior to decannulation were not different between the two groups (AA = 63mmHg ± 6.8) versus Saline alone = 55mmHg ± 10.4) and (29% ± 3.0 versus 30% ± 4.0). Resuscitation with AA improved survival (10 lived/5 died 67%) versus Saline alone (3 lived/12 died 20%) p < 0.05 **. Animals given AA had less lung MPO activity 0.89 units/gm tissue, S.E.M.=0.08 versus 1.47 units/gm tissue, S.E.M.=0.13, p < 0.05* in saline treated animals. Sham operated animals had 0.46 units/gm tissue, S.E.M.= 0.26.

CONCLUSION: These data show that ascorbic acid given during resuscitation improves survival and prevents neutrophil sequestration in this model of hemorrhagic shock.

** Chi-square with Yate’s correction
* Student’s unpaired T-test
PREDICTING PROLONGED VENTILATOR DEPENDENCE (PVD) IN THERMALLY INJURED PATIENTS
B Sellers, B Davis, P Larkin, S Morris, J Saffle,
Intermountain Burn Center, University of Utah Health Center
Barbara J. Sellers, MD
Jeffrey R. Saffle, MD
Salt Lake City, UT

Recent studies suggest that when PVD (≥ 14 days) can be predicted in trauma/ICU patients, early tracheostomy may reduce length of stay and pneumonia. This study was performed to develop early criteria predictive of PVD in burn patients.

METHODS: We reviewed all burn patients age ≥16 admitted between 1990-1994 who required ventilator support (VS) for ≥ 3 days. Using the variables total (TBSA) and full-thickness burn size (FTBSA), age, inhalation injury, and worst PaO2/FiO2 on post-burn day 3 (P/F), an equation predicting PVD for each patient was created using logistic regression. The equation was tested by applying it to 1995 patients.

RESULTS: 62 of 110 patients reviewed developed PVD

<table>
<thead>
<tr>
<th>Variable</th>
<th>All pts</th>
<th>VS≤13d (n=48)</th>
<th>VS≥14d (n=62)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TBSA</td>
<td>33 ± 19*</td>
<td>24 ± 16</td>
<td>40 ± 19</td>
</tr>
<tr>
<td>FTBSA</td>
<td>19 ± 18</td>
<td>9.6 ± 9.7</td>
<td>27 ± 19**</td>
</tr>
<tr>
<td>Age (yrs)</td>
<td>41 ± 16</td>
<td>38 ± 14</td>
<td>44 ± 17**</td>
</tr>
<tr>
<td>IN (%)</td>
<td>58/110 (53)</td>
<td>18/48 (38)</td>
<td>40/62 (65)**</td>
</tr>
<tr>
<td>P/F</td>
<td>181 ± 16</td>
<td>212 ± 70</td>
<td>155 ± 62**</td>
</tr>
</tbody>
</table>

* mean ± sd  **p < 0.05 vs VS≤13days

The following equation was created: \( \ln[\text{Prob PVD}/(1-\text{Prob PVD})] = (-2.967) + (0.07)\text{(age)} + (0.116)\text{(FTBSA)} + (-0.014)\text{(P/F)} + (2.013)\text{(IN;1=yes; 2=no)} \). When a probability ≥ 0.5 was considered predictive of PVD, the equation correctly predicted PVD in 84% of the 1990-1994 sample, and 93% of the 1995 sample.

CONCLUSIONS: Prolonged ventilator dependence in burn patients can be predicted using objective variables available in the early post-burn period. These predictions can be used in planning resource utilization, and in selecting patients for prospective studies of early tracheostomy.
EARLY IDENTIFICATION OF PSYCHOSOCIAL FACTORS LIMITING OUTCOME FOLLOWING TRAUMATIC INJURY

AJ Michaels, MD*, CE Michaels, MD*, C Moon*, MA Zimmerman, PhD*, C Peterson, PhD*, and JL Rodriguez MD*

Trauma/Burn/Emergency Surgery Division*, Department of Psychology* and School of Public Health* - University of Michigan
Presenter: AJ Michaels, MD
Sponsor: JW McGill, MD
Ann Arbor, Michigan

The return of an injured patient to meaningful function is the central challenge facing trauma systems. We present a model which predicts psychosocial morbidity that compromises return to work independent of pre-injury function, injury severity, physical recovery, compensation or litigation status.

METHODS: A cohort of 56 patients without severe neurotrauma was evaluated prospectively for return to functional employment. Evaluation included data from the trauma registry [age, injury mechanism, and Injury Severity Score (ISS)], an expanded social history (gender, marital status, income, employment, education, and religious practice), and psychometric testing including: psychological [Brief Symptom Index (BSI), Impact of Events Scale (IES) and Mississippi PTSD scale], social (MSPSS and LES), and general health measures [Sickness Impact Profile (SIP) and SF-36]. Evaluation consisted of interviews and mailed self-report questionnaires at admission, one and four months following injury. Results were analyzed by a stepwise linear regression model and by Chi-square, student T-test and Spearman's correlations. Data are reported as percent (%) or mean ± standard error of the mean. Significance is noted at the 95% confidence level.

RESULTS: 35 patients completed the protocol (64%). This population was 37 ± 2.9 years old, 77% male, 46% married, 52% high school and 28% college educated, and 80% earned >$20,000. Injury mechanism was blunt force in 74%, penetrating in 8% and 17% burns. The ISS was 13.3 ± 1.8, 28% required urgent operation and the length of stay was 7.8 ± 1.2 days. Poor SIP work score was due to physical disability (p=.016) and psychological pathology (p=.015) in a regression analysis of data collected at four months. Premorbid employment status, premorbid psychopathology, and ISS were not significant in this model. A high IES on admission correlated with delayed psychopathology in a X² analysis (P<.001, risk ratio of 4.8). Of interest, patients not completing the protocol were no different by any psychological measure, although they had lower education (p=.001) and income (p=.009) levels, were 90% male and had a higher proportion of penetrating injury.

CONCLUSIONS: Our regression model allowed us to identify several components of poor outcome as measured by the SIP work score. Psychological distress at four months following injury is associated with compromised return to work independent of premorbid employment or psycho-pathology, ISS, incomplete physical recovery or litigation. Significant psychopathology and poor outcome can be predicted at the time of admission by a high IES score. Early identification of risk factors may permit early intervention designed to limit psychological morbidity and potentially improve functional outcome.
MODULATION OF CYTOKINE RESPONSE TO SEPSIS THROUGH SUBLETHAL HEMORRHAGE IS NEITHER CYTOKINE NOR MODEL SPECIFIC
E. Zervos, MD, J. Norman, MD, D. Watkins, MD, A. Rosemurgy, MD
University of South Florida Department of Surgery
E. Zervos, M.D.
A.S. Rosemurgy, M.D.
Tampa, FL

Background: We have previously shown that tolerance to lethal endotoxic challenge can be induced in mice through sublethal hemorrhage. In these studies, LPS-induced serum IL-1β was blunted following hemorrhage. This study was undertaken to determine if hemorrhage induces attenuation of the cytokine response to sepsis beyond IL-1β, whether this response occurs at the level of gene transcription and whether it occurs in a non-murine model. Methods: Twelve Sprague Dawley rats (175-200 g) underwent either sublethal hemorrhage, 1cc/100g by cardiac puncture (Group H), lethal intraperitoneal endotoxin 40mg/kg (Group L) or sublethal hemorrhage followed by lethal endotoxin 24 hours later (Group HL). Animals were sacrificed by decapitation 12 hours following LPS (Groups L and HL) or hemorrhage (Group H). IL-1β and TNF mRNA was determined on total splenic RNA isolates by quantitative differential RT-PCR. Results: * = p<0.05 vs. H and # = p<0.05 vs. L by Mann Whitney U-Test. Data are mean ± SEM.

Rats hemorrhaged prior to lethal LPS mounted a cytokine response greater than rats exposed to hemorrhage alone but less than rats receiving LPS alone.

Conclusions: Sublethal hemorrhage induces early IL-1β and TNF gene expression which blunt their expected rise following subsequent endotoxic challenge. This study demonstrates that this effect is neither model nor cytokine specific. Blunting of inflammatory cytokine gene expression by sublethal hemorrhage prior to LPS challenge supports the host protection and survival benefit observed in the murine model.
ETHANOL CONFOUNDS BASE DEFICIT ESTIMATION OF INJURY SEVERITY
K.T. Asgarian, M.D., R.M. Price, Ph.D., K.M. Kelly, M.D.
Department of Surgery, Morristown Memorial Hospital
K.T. Asgarian, M.D.
K.F. O'Malley, M.D.
Morristown, NJ 07962

The literature supports the use of the base deficit (BD) calculation from arterial blood gas determination as a guide for resuscitation (J Trauma 28:1464, 1988) and as a marker for injury severity (J Trauma 33:417, 1992). Although serum lactate may be more specific for end organ hypoperfusion, the minimum turnaround time for standard wet chemistry lactate analysis is approximately one hour. BD has been described as stoichiometric to lactate (Surg Clin N Am 62:31, 1982). Because of this, our trauma surgeons use BD as one indicator of injury severity. BD may be reflective of many acid-base disorders but during trauma resuscitation abnormal BD levels (BD < -2) are almost always due to hypothermia, hypovolemia or alcohol-induced ketoacidosis. Since no previous authors have described the effect of alcoholic ketoacidosis on BD in the trauma patient, we sought to determine if alcohol (EtOH) ingestion affected BD.

In a retrospective analysis of 104 trauma patients who had serial arterial blood gas determinations, we noted the following: (1) no patients had a core temperature less than 35 °C, i.e., none were hypothermic; (2) for equal severity of injury, the initial BD was significantly (*p < 0.001) lower in those patients with EtOH levels > 10 mg/dl (Table 1).

Table 1: Comparison of Patients With and Without EtOH > 10 mg/dl (mean ± S.D.).

<table>
<thead>
<tr>
<th></th>
<th>EtOH +</th>
<th>EtOH -</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>28</td>
<td>76</td>
</tr>
<tr>
<td>EtOH</td>
<td>197.4±89.2</td>
<td>0.2±0.7</td>
</tr>
<tr>
<td>ISS</td>
<td>23.5±13.0</td>
<td>23.5±9.1</td>
</tr>
<tr>
<td>RTS</td>
<td>6.3±2.0</td>
<td>6.5±1.9</td>
</tr>
<tr>
<td>GCS</td>
<td>11.7±4.8</td>
<td>11.9±4.7</td>
</tr>
<tr>
<td>BD</td>
<td>-6.3±3.8</td>
<td>-3.8±3.3</td>
</tr>
<tr>
<td>BD</td>
<td>-1.5±5.3</td>
<td>-1.7±3.5</td>
</tr>
</tbody>
</table>

*Significance at p<0.001
ISS = Injury Severity Score (1-75)
RTS = Revised Trauma Score (0-7.84)
GCS = Glasgow Coma Score (3-15)
BD = initial BD
BD = final BD after resuscitation

In patients with EtOH < 10 mg/dl, the degree of BD abnormality was consistent with the severity of injury, as previously reported. However, in those patients with EtOH > 10 mg/dl, the degree of BD abnormality was greater than had been previously correlated with the same severity of injury. In all previous studies, more severe derangements in BD were correlated with increased injury severity. Our data show equal injury severity in the two comparison groups as measured by ISS or RTS. In multiple pairwise comparisons (Tukey test), only the initial BD was significantly (*p < 0.001) different between the EtOH + and EtOH - groups.

We conclude that alcoholic ketoacidosis exaggerates BD abnormalities. This is an important consideration during resuscitation since the degree of BD abnormality is often used to judge the degree of hypovolemia and is acted on accordingly.
EARLY BONE MARROW UNRESPONSIVENESS TO M-CSF AND G-CSF PREDISPOSES BURN PATIENTS TO SEPSIS
K Pellegrin, M.D., CH Rundus, M.T., CE Hartford, M.D., XW Wang, M.D., VM Peterson, M.D.
Department of Surgery, University of Colorado Health Sciences Center and The Children’s Hospital, Denver, Colorado
K Pellegrin, M.D.

FM Karrer, M.D.
Denver, Colorado

Previous data in our laboratory indicate that bone marrow failure invariably precedes postburn sepsis. Macrophage-colony-stimulating factor (M-CSF), granulocyte-CSF (G-CSF), and GM-CSF stimulate growth and function of granulocyte and macrophage progenitors. We hypothesized that early alterations in the production of CSFs may predate postburn marrow failure and predict sepsis. Serial serum levels of M-CSF, G-CSF and GM-CSF were obtained in 70 adult burn patients (59 survivors, 11 nonsurvivors; 23 septic patients with mean time to onset of sepsis = 9 days; mean age,

Figure 1 Sepsis and M-CSF levels
39 ± 17; mean burn size, 36 ± 23%) and related CSF levels to burn size, sepsis, survival and white cell count. Sera were assayed for G-CSF and GM-CSF by ELISA, and for M-CSF by radio-immunoassay. Admission M-CSF was 60-fold above normal controls (0.14 ± .018 U/μl), peaked at days 3 & 7, and was higher (p< .05) in septic patients as early as day 3 (Fig 1). Admission and days 2 & 3 G-CSF levels were also higher (p < 0.05) in septic patients (Fig 2). GM-CSF was undetectable in normal and postburn sera. WBC were paradoxically lower in septic than nonseptic burn patients at day 2 (7,500 vs 11,300; p<0.05), despite the fact that serum levels of both M-CSF and G-CSF were greater in septic patients in the early postburn period. In summary, elevations in M-CSF and G-CSF failed to increase WBC production, indicating an early bone marrow failure in patients who subsequently became septic. We therefore conclude that high serum levels of M-CSF and G-CSF, in conjunction with a low WBC, appear to be sensitive, early predictors of postburn sepsis.

Figure 2 Sepsis and G-CSF levels
EARLY PREDICTORS OF THE NEED FOR MECHANICAL VENTILATION IN BLUNT TRAUMA PATIENTS WITH PULMONARY CONTUSION

J. Collinge, M.D., J. Tyburski, M.D., S. Eeachemati, M.D., R. Wilson, M.D.
Wayne State University
J. Collinge, M.D.
R. Wilson, M.D.
Detroit, Michigan

OBJECTIVE: To evaluate early predictors of the need for mechanical ventilation in patients with pulmonary contusion.

METHODS: The medical records and chest x-rays of 103 consecutive trauma patients with the diagnosis of pulmonary contusion admitted to a Level One Trauma Center from 1993 to 1995 were evaluated for Injury Severity Score (ISS), Glasgow Coma Score (GCS), shock on admission, number of rib fractures (RibFx), presence of flail chest (3 or more consecutive ribs broken in more than one place), extent of contusion (mild, moderate, severe) evident within 24 hours on chest x-ray, and initial arterial partial pressure of oxygen to fraction of inspired oxygen ratio (PaO₂/FiO₂). Patients were divided into two groups based on ventilator requirements. A subdivision of ventilated patients with GCS ≥ 9 (n=38) was identified to exclude patients intubated solely on the basis of neurologic status.

RESULTS: Fifty-six patients required mechanical ventilation and 47 did not. The overall mortality rate was 22%. There were no differences in age, sex, or mechanism of injury between the groups. Predictions of the need for mechanical ventilation in these groups were:

<table>
<thead>
<tr>
<th></th>
<th>Pts with ISS ≥20</th>
<th>Pts with Shock on Admisson</th>
<th>Pts with Flail Segment</th>
<th>Mean RibFx ± SEM</th>
<th>Pts with Mod- or Sev. Contusion on CXR</th>
<th>Mean PaO₂/FiO₂ ± SEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-vent (n=47)</td>
<td>14 (30)</td>
<td>2 (4)</td>
<td>13 (28)</td>
<td>2.9 ± 0.4</td>
<td>35 (75)</td>
<td>417 ± 42</td>
</tr>
<tr>
<td>Total Vent (n=56)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vent with GCS ≥ 9 (n=38)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* P = <.01 vs non-vent; #P = NS vs non-vent; +P = <.02; (%) = %

CONCLUSION: Early predictors of the need for mechanical ventilation in patients with pulmonary contusion were ISS ≥20, shock on presentation, moderate or severe contusion on CXR within 24 hours, and a PaO₂/FiO₂ <350. These predictors were accurate independent of intubation for associated neurologic injury. The number of rib fractures and the presence of a flail segment were not predictive of the requirement for intubation. The PaO₂/FiO₂ ratio and the appearance of the CXR are highly predictive of the need for mechanical ventilation in patients with pulmonary contusions.
BASE DEFICIT IS SUPERIOR TO PH IN EVALUATING CLEARANCE OF ACIDOSIS AFTER TRAUMATIC SHOCK
J.W. Davis, M.D., K.L. Kaups, M.D., S.N. Parks, M.D.
Valley Medical Center
J.W. Davis
J.W. Davis
Fresno, California

PURPOSE: Previous studies have demonstrated the utility of admission Base Deficit (BD) in assessing shock, resuscitation and predicting complications. This study was done to evaluate the differences in BD clearance, pH normalization and complications between Survivors (S) and Non-Survivors (NS) after traumatic shock.

METHODS: Patients entered into the trauma registry from 7/80 through 8/95 with arterial blood gases performed within an hour of arrival, and an admission BD ≤ -6, were included. Data is grouped by BD category (Moderate, -6 to -9, Severe ≤ -10) and survival. Group mean ± SEM were compared with two-tailed T test.

RESULTS: Over the study period, 674 patients met entry criteria and had medical record review with recording of serial blood gas data.

<table>
<thead>
<tr>
<th>BD Category</th>
<th>Admit</th>
<th>4 hr</th>
<th>8 hr</th>
<th>16 hr</th>
<th>24 hr</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BD_S</td>
<td>-7.0 ± .1</td>
<td>-5.4 ± .3</td>
<td>-4.2 ± .4</td>
<td>-1.2 ± .3</td>
<td>0.6 ± .3</td>
</tr>
<tr>
<td>BD_NS</td>
<td>-7.3 ± .1</td>
<td>-7.7 ± .9</td>
<td>-6.2 ± .8</td>
<td>-3.5 ± .9</td>
<td>-1.9 ± .9</td>
</tr>
<tr>
<td>p Value</td>
<td>.02</td>
<td>.004</td>
<td>.011</td>
<td>.002</td>
<td>.001</td>
</tr>
<tr>
<td>pH_S</td>
<td>7.32 ± .00</td>
<td>7.37 ± .01</td>
<td>7.38 ± .01</td>
<td>7.41 ± .01</td>
<td>7.43 ± .01</td>
</tr>
<tr>
<td>pH_NS</td>
<td>7.31 ± .01</td>
<td>7.33 ± .02</td>
<td>7.37 ± .02</td>
<td>7.40 ± .02</td>
<td>7.43 ± .02</td>
</tr>
<tr>
<td>p Value</td>
<td>.15</td>
<td>.07</td>
<td>.3</td>
<td>.3</td>
<td>.3</td>
</tr>
<tr>
<td>Severe</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>BD_S</td>
<td>-14.1 ± .4</td>
<td>-7.5 ± .6</td>
<td>-4.9 ± .5</td>
<td>-1.7 ± .4</td>
<td>0.4 ± .5</td>
</tr>
<tr>
<td>BD_NS</td>
<td>-15.9 ± .5</td>
<td>-10.0 ±1.1</td>
<td>-7.1 ±1.1</td>
<td>-5.4 ±1.2</td>
<td>-3.4 ±1.2</td>
</tr>
<tr>
<td>p Value</td>
<td>.002</td>
<td>.03</td>
<td>.034</td>
<td>.001</td>
<td>.001</td>
</tr>
<tr>
<td>pH_S</td>
<td>7.18 ± .01</td>
<td>7.32 ± .01</td>
<td>7.36 ± .01</td>
<td>7.40 ± .01</td>
<td>7.43 ± .01</td>
</tr>
<tr>
<td>pH_NS</td>
<td>7.12 ± .02</td>
<td>7.29 ± .03</td>
<td>7.34 ± .02</td>
<td>7.36 ± .02</td>
<td>7.39 ± .02</td>
</tr>
<tr>
<td>p Value</td>
<td>.001</td>
<td>.134</td>
<td>.3</td>
<td>.024</td>
<td>.041</td>
</tr>
</tbody>
</table>

Multi-organ failure was significantly more frequent in NS vs. S, in both moderate (16% vs 2%, p<0.001) and severe (13% vs. 3%, p<0.001) BD categories.

CONCLUSIONS: S in both the moderate and severe groups had improved their BD by one category at 4 hr and normalized their BD by 16 hr. NS did not improve BD category until 8 hr (severe) and 16 hr (moderate) and did not normalize their BD until 24 hr. BD revealed differences in metabolic acidosis between S and NS not shown by pH and is clearly a better marker of acidosis clearance after shock.
INITIAL ROUTINE LABS IN TRAUMA PATIENTS ARE NEITHER CLINICALLY RELEVANT NOR COST EFFECTIVE

D. Kavouspour, M.D.; K. Baker, M.D.; G. Jans, R.N.
University of South Florida

Dar Kavouspour, M.D.

Alexander Rosemurgy, M.D.

Tampa, Florida

Objective: To determine if routine CBC (WBC, Hgb, HCT, Plt.), Chem7 (Na+, K+, Cl-, HCO3-, BUN, Creatinine, Glucose), PT, PTT, serum alcohol (ETOH) and urine drug screen (UDS) have clinical significance in the initial evaluation of severely injured adults.

Methods: Over 4 months, 193 consecutive adult trauma alert patients were prospectively randomized to Limited Lab Profile (LLP = U/A, Hgb) vs Complete Lab Profile (CLP = U/A, CBC, Chem7, PT, PTT, ETOH/UDS) via an envelope pull method. Age > 60, sternotomy scar, and coumadin history were used as strict criteria to add Chem7 or PT, PTT for those randomized to LLP.

Results: 95 adults randomized to LLP and 98 to CLP. Injuries were 114 (59%) blunt, 47 (24%) penetrating, 7 (4%) burn, and 25 (13%) other. Data are mean ± S.D.

<table>
<thead>
<tr>
<th></th>
<th>#</th>
<th>Male</th>
<th>Age</th>
<th>ISS</th>
<th>RTS</th>
<th>GCS</th>
<th>Hgb(gm/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LLP</td>
<td>95</td>
<td>67(70%)</td>
<td>33±15</td>
<td>12.7±14.5</td>
<td>10.6±2.8</td>
<td>13.2±4.5</td>
<td>13.5 ± 2.9</td>
</tr>
<tr>
<td>CLP</td>
<td>98</td>
<td>75(77%)</td>
<td>33±15</td>
<td>9.9±10.1</td>
<td>11.1±1.9</td>
<td>13.0±3.7</td>
<td>13.6 ± 1.9</td>
</tr>
</tbody>
</table>

Those undergoing CLP had abnormalities in Na+ (8%), K+ (36%), Creatinine (22%), Plt (2%), PT (61%), and ETOH/UDS (63%). Of the 98 patients in CLP, 76 (78%) had abnormal lab values. Correction was undertaken in only 2 patients (3%), that being FFP to correct prolonged PT. The difference in cost between CLP and LLP is $86.91 per patient in our institution, which translates to an estimated annual hospital cost of $174,000.

<table>
<thead>
<tr>
<th></th>
<th>Chem7</th>
<th>Plt</th>
<th>ETOH/UDS</th>
<th>PT</th>
</tr>
</thead>
<tbody>
<tr>
<td># of abnormalities in CLP</td>
<td>35(36%)</td>
<td>2(2%)</td>
<td>62(63%)</td>
<td>60(61%)</td>
</tr>
<tr>
<td># of interventions in CLP</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>2</td>
</tr>
</tbody>
</table>

Conclusion: This prospective randomized study documents that routine labs do not add substantively to the care of a broad range of notably injured trauma patients. Abnormalities on CLP occurred in over 75% of the subjects and corrective intervention was virtually never undertaken, without ill effect. Chemistry profile, CBC, alcohol and drug screens, and coagulation profiles obtained on admission of trauma alert patients are often abnormal and are seldom corrected, thus being generally superfluous and excessively expensive.
L-ARGININE DECREASES ALVEOLAR MACROPHAGE PROINFLAMMATORY MONOKINE PRODUCTION IN ENDOTOXIN-INDUCED ACUTE LUNG INJURY.
DR Meldrum, MD, RC McIntyre, Jr, MD, BC Sheridan, MD, DA Fullerton, MD, A Banerjee, PhD, AH Harken, MD. Department of Surgery, Univ. of Colorado, Denver, CO.
Daniel R. Meldrum, M.D.
Ernest E. Moore, M.D.
Denver, Colorado

Recent clinical reports (Chollet et al, Am J Respit Crit Care Med, 1996) indicate that inhaled nitric oxide (NO) reduces alveolar macrophage proinflammatory monokine (TNFα and IL-1β) production which are purported to promote acute lung injury. We hypothesized that the provision of substrate for local NO production (L-arginine) would reduce alveolar macrophage proinflammatory monokine production following endotoxin (ETX)-induced acute lung injury. The purpose of this study was to determine the effect of L-arginine on tumor necrosis factor α (TNFα) and interleukin 1 β (IL-1β) production in ETX-induced acute lung injury. Methods: Rats received ETX (0.5 mg/kg IP) or vehicle, with or without L-arginine supplementation (300 mg/kg IP). 4 hours later, alveolar macrophage were harvested by bronchoalveolar lavage and incubated at 10^6 cells/ml+1 μg/ml PMA for 24 hours. Cell-free supernatants were collected and assayed (ELISA) for TNFα and IL-1β. Results: L-arginine decreased alveolar macrophage TNFα (Fig 1) and IL-1β (Fig 2) release during acute lung injury (P<0.05, ANOVA and Tukey’s, n=6/group).

![Graphs Fig 1 and Fig 2 showing TNFα and IL-1β levels](image)

We conclude that: 1) L-arginine decreases alveolar macrophage proinflammatory monokine production during ETX-induced acute lung injury; 2) L-arginine is an immunomodulating nutritional supplement; and 3) the provision of exogenous substrate for local NO production may reduce the deleterious clinical effects of acute lung injury.
RESOLUTION OF PNEUMOTHORAX WITH OXYGEN THERAPY IS DOSE DEPENDENT

Gregory J. England MD, Jason D. Harrah BS, Ronald C. Hill MD, Gregory A. Timberlake MD, Jeffrey F. Hill BS, Yvonne L. Shahan MS, Mike Billie
West Virginia University School of Medicine, Department of Surgery, Morgantown, West Virginia, USA

Presenter: Gregory J. England, MD
Senior Sponsor: David Kappel, MD

The treatment of asymptomatic patients with small pneumothoraces has included observation and tube thoracostomy. Our laboratory has shown previously that high dose oxygen therapy (FiO₂ 60%) improves resolution of pneumothoraces significantly when compared with room air. This study was designed to evaluate lower levels of inspired oxygen and to establish a dose response curve in the resolution of experimental pneumothoraces.

Forty New Zealand white rabbits were randomly divided into four groups of inspired oxygen: room air (21%), 30%, 40%, and 50% FiO₂. Experimental pneumothoraces were created in the rabbits and they were placed in cages with the designated level of inspired oxygen. Serial chest x-rays were performed until the pneumothoraces resolved. Statistical analysis was performed using the Jonckheere-Terpstra test for ordered alternatives.

![Graph showing mean hours to resolution of pneumothorax](image)

Results are given as mean ± standard error of the mean. Room air pneumothoraces resolved in 61.7 ± 12.3 hours, 30% FiO₂ in 42.9 ± 6.0 hours, 40% FiO₂ in 35.8 ± 4.3 hours, and 50% FiO₂ in 33.8 ± 4.7 hours.

These results show a statistically significant dose dependent improvement in resolution of pneumothoraces with increasing levels of inspired oxygen (p<0.01). Supplemental oxygen in lower concentrations may be utilized to facilitate the resolution of small pneumothoraces.
EFFECTS OF HYPOTHERMIA ON NEUTROPHIL FUNCTION

J.J. Fildes, M.D.; S. Fisher, M.D.; C.M. Sheaff, M.D., Ph.D; J.A. Barrett, M.D.

Department of Trauma Surgery, Cook County Hospital
J.J. Fildes, M.D.
J.J. Fildes, M.D.
Chicago, Illinois

Objective: We will determine if neutrophil function, as measured by oxidative burst, is decreased by mild to moderate levels of hypothermia.

Methods: Blood was collected from healthy adult volunteers. The neutrophils were isolated and divided into 2 aliquots. One was incubated at the control temperature of 37.5°C, and the other at an experimental temperature of either 30°C, 32°C, 34°C, or 36°C. The peak chemiluminescence and the time required to reach the peak were determined at 1 and 2 hours for each sample. The percent change of the peak chemiluminescence and the time to reach the peak were calculated for each temperature at 1 and 2 hours. These values were compared to control using ANOVA with p <0.05 indicating significance.

Results: The percent change of the peak chemiluminescence was significantly decreased after 2 hours at temperatures of 34°C (-18.76% ± 3.45), 32°C (-28.39% ± 4.19), and 30°C (-35.57% ± 3.54). The percent change of the time required to reach the peak was significantly increased after 1 hour at temperatures of 34°C (31.5% ± 3.40), 32°C (33.7% ± 4.5), and 30°C (32.1% ± 3.61).

Conclusions: Hypothermia (30°-34°C) causes significant levels of neutrophil dysfunction. Neutrophils are the first line of defense against infection. Additional studies are required to determine if this in-vitro observation correlates with increased infection rates.
SATURATION KINETIC MODELING OF THE MOLECULAR RESPONSE TO ALTERED CIRCULATION USING THE MICHAELIS–MENTEN EQUATION (MME)

J. St. Louis, R. Monroe, R.L. Reed
Duke University Medical Center

James St. Louis, M.D.
R. Lawrence Reed, II, M.D.

Durham, NC  27710

Adequate circulatory performance should ensure sufficient oxygen delivery to prevent cell stress. A mathematical model of saturation kinetics, such as MME (VO₂ = [DO₂ x \( \frac{V_{\text{max}}}{[K_m + DO_2]} \)), should accurately define the oxygen delivery (DO₂)-oxygen consumption (VO₂) relationship as well as the cellular response pattern. Heat Shock Proteins (HSP72) are a unique class of proteins believed to be responsible for the prevention of abnormal protein unfolding during cellular stress. Ten Dekalb pigs (30-80kg) were fibrillated and DO₂ controlled by use of Direct Mechanical Ventricular Actuation. VO₂ was measured independently of DO₂ using spirometry. Six additional pigs (50-80kg) were used as controls. RT-PCR was performed on kidney and liver biopsies to analyze the expression of RNA for HSP72. Least-squares regression techniques were used to curve-fit data points to the MM model.

Curve-fitting of VO₂ vs. DO₂ using the MME produced individual VO₂-DO₂ curves with a mean \( R^2 \) of 0.91±0.05, whereas curve-fitting of HSP72 mRNA expression vs. DO₂ using an inverse MME produced a mean \( R^2 \) of 0.8±0.12 for kidney and 0.71±0.18 for liver. Analysis of pooled VO₂-DO₂ data revealed a VO₂max of 10.8 mlO₂/min/kg (fig. #1). An arbitrary point at which HSP72 mRNA expression was greater than two SD above the mean control levels was used as the critical DO₂, corresponding to a DO₂ of 3.2 ml O₂/min/kg for kidney (fig. #2, point B) and 1.4 ml O₂/min/kg for liver. This critical DO₂ extrapolates to a VO₂ that is 29% and 12% of maximal VO₂ in the kidney (fig. 1, point A) and liver, respectively.

Figure #1

![Figure #1 Image]

Figure #2

![Figure #2 Image]

These data confirm that a definable kinetic relationship exists between DO₂ and VO₂ as well as between DO₂ and the expression of HSP72 mRNA. Using such a model, critical DO₂ can be defined based upon the molecular response to anaerobic stress. These current studies also confirm that the kidney appears more sensitive to circulatory insufficiency than the liver.
POST-TRAUMATIC LYMPHOCYTE FUNCTION:  
COMPARISON BETWEEN TISSUE AND PERIPHERAL  
BLOOD T-CELL RESPONSES  
MM Aguilar, MD, FD Battistella, MD, JT Owings, MD,  
SA Olson, MD, K MacColl  
Department of Surgery, University of California, Davis  
Presenter: Michael M. Aguilar, MD  
Sponsor: M. Margaret Knudson, MD  
San Francisco, California

The majority of T-cells (97%) reside in lymphoid tissues and lymphatic channels (tissue T-cells). T-cell response to injury has been assessed largely by sampling blood lymphocytes which represent < 3% of total body T-cells. We hypothesized that tissue T-cells are stressed by injury and hypoperfusion to a greater extent than blood T-cells. Anesthetized adult sheep (n=10) instrumented with a pre-femoral lymphatic fistula were subjected to lower extremity fractures, a fixed volume hemorrhage, and a fixed volume resuscitation. Blood and tissue T-cells were characterized at baseline and 2 hours after trauma using monoclonal antibodies and flow cytometry.

Results: Mean arterial blood pressure and cardiac output fell by 50% with hemorrhage and were restored to baseline with resuscitation. Compared with blood, tissue T-cell composition demonstrated a higher percentage of CD4 (helper) and a lower percentage of CD8 (suppressor) T-cells. A much larger percentage of tissue lymphocytes expressed the adhesion molecule L-selectin (L-Sel). Poly-trauma induced a relative decrease in tissue CD8 and γδ-TCR T-cell subpopulations while blood T-cell subpopulation composition remained unchanged.

<table>
<thead>
<tr>
<th></th>
<th>Blood T-cells</th>
<th>Tissue T-cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>% CD4</td>
<td>31 ± 14</td>
<td>42 ± 10*</td>
</tr>
<tr>
<td>% CD8</td>
<td>40 ± 11</td>
<td>21 ± 9 *</td>
</tr>
<tr>
<td>% γδ-TCR</td>
<td>11 ± 5</td>
<td>12 ± 4</td>
</tr>
<tr>
<td>% L-Sel</td>
<td>44 ± 9</td>
<td>86 ± 3 *</td>
</tr>
<tr>
<td>% MHC II</td>
<td>20 ± 8</td>
<td>20 ± 6</td>
</tr>
</tbody>
</table>

* = p < 0.05, blood vs. lymph; † = p < 0.05, baseline vs. post-trauma (repeated measures ANOVA, mean ± SD).

Conclusions: The trauma induced decreases in tissue CD8 and γδ T-cells suggest that evaluating T-cell response to injury by sampling peripheral blood lymphocytes underestimates the T-cell's role in the post-traumatic inflammatory response. Considering the immunologic activity of T-cells and the direct access lymphatic cells have to the lungs via the thoracic duct, activated tissue lymphocytes may play an integral role in the development of ARDS, SIRS, and post-traumatic immunosuppression.
AN EPISODE OF PREHOSPITAL HYPOTENSION IN TRAUMA PATIENTS: A MARKER FOR DOOM OR JUST CRYING WOLF?

Manuel Aranda, M.D., Scott R. Petersen, M.D., Colleen Hunt, and Brian Nelson, M.D.
Trauma Center, St. Joseph’s Hospital and Medical Center, and Texas Tech University Health Sciences Center Texas
Manuel Aranda, M.D.

Phoenix, Arizona and El Paso, Texas

Background: Overtriage to trauma centers continues to be a problem for trauma systems, especially in blunt injured patients who are triaged by mechanism of injury. In patients who have associated persistent hypotension or other physiologic derangements, the decision to triage to a trauma center is clear.

Study Design: To assess the importance of a single episode of prehospital hypotension on the outcome of blunt injured trauma patients who are triaged primarily by mechanism of injury. Outcomes studied were: need for urgent operative intervention (thoracotomy, laparotomy or craniotomy), ICU admission, ICU and hospital LOS, and survival.

Design: Retrospective, trauma registry based, clinical review.
Setting: ACS Level I Trauma Center
Type of Participants: Blunt injured patients who experienced one episode of prehospital hypotension (SBP ≤ 90) (n=103) which corrected upon arrival to the trauma center were compared to patients who remained normotensive throughout the prehospital phase and upon initial presentation (n=103). The patients were matched for age (mean 31.4 years, range 6-82 years), sex (65.3% males), GCS (13 ± 0.2, range 3-15), and RTS (7.47 ± 0.5, range 2.9304-7.8408)

Results: (mean ± SEM)

<table>
<thead>
<tr>
<th>SBP ≤ 90 mm Hg</th>
<th>SBP &gt; 90 mm Hg</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>31.4 ± 0.6</td>
<td>31.4 ± 1.8</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>76 ± 2</td>
<td>133 ± 2</td>
</tr>
<tr>
<td>RTS</td>
<td>7.3 ± 0.1</td>
<td>7.7 ± 0.1</td>
</tr>
<tr>
<td>GCS</td>
<td>12.2 ± 0.4</td>
<td>13.8 ± 0.3</td>
</tr>
<tr>
<td>ISS</td>
<td>12.9 ± 1.1</td>
<td>8.5 ± 0.8</td>
</tr>
<tr>
<td>LOS (days)</td>
<td>6.2 ± 0.7</td>
<td>3.7 ± 0.6</td>
</tr>
<tr>
<td>ETOH (mg/dl)</td>
<td>80 ± 11</td>
<td>53 ± 10</td>
</tr>
</tbody>
</table>

Although there was no difference in the requirement for urgent operations (6.8% SBP ≤ 90 vs. 1.9% SBP > 90), patients who sustained one episode of prehospital hypotension had longer ICU stays (ICU > 3 days, 21.4% vs. 8.7%; p = 0.025) and higher mortality (5.8% vs. 1.9%; p = 0.05).

Conclusions: An episode of hypotension that responds to prehospital fluid administration and is alleviated upon arrival to the emergency department selects a group of trauma patients that are more seriously injured, have longer hospital and ICU lengths of stays, and have a higher mortality. Even a single episode of hypotension warrants activation of the trauma team.
IMMEDIATE NEUTROPHIL ACTIVATION AFTER
HEMORRHAGIC SHOCK AND RESUSCITATION
P. Rhee, MD, MPH, D.Burris, MD, E. Pilkoulis, MD, K. Kaufmann MD, J. McKenzie, PhD,
Uniformed Services University of the Health
Sciences
Peter Rhee, MD, MPH,
G. Jurkovich, MD,
Bethesda, Maryland

OBJECTIVE: Primed or activated neutrophils play a major role in tissue injury following trauma. Our hypothesis was that neutrophils are primed and activated early during hemorrhagic shock and resuscitation.

METHODS: Eight awake pigs were subjected to a 40% volume hemorrhage (28ml/kg) over 15 minutes, followed by a 60 minute shock period and then resuscitation of 3X blood volume lost (84ml/kg) with Ringer's shock Lactate solution over one hour. Four control animals underwent the same volume of fluid infusion without hemorrhage. Through previously placed pulmonary artery and carotid artery catheters blood was collected serially throughout the experiment. Whole blood were stained with 2',7'-dichlorofluorescein-diacetate and leukocyte burst activity was measured with flow cytometry. CD18b expression was measured utilizing monoclonal antibodies and flow cytometry.

RESULTS: While there was a significant increase in fluorescence of the neutrophils (p<0.05, ANOVA) immediately following hemorrhage, a greater peak in fluorescence was observed after resuscitation (p<0.01). During the same time period, the CD18b expression on neutrophils in blood progressively decreased to 70% of baseline (p<0.05).

CONCLUSIONS: These data demonstrate that circulating neutrophils are activated early during hemorrhage as evidenced by increased oxidative burst activity. However, a greater activation of neutrophils occurred at resuscitation (reperfusion) than during ischemia. The decline in surface adhesion antigens (CD18b) expression during the burst activity may suggest that this burst activity is non-CD18 mediated.
SECRETORY IgA BLOCKS HYPOXIA AUGMENTED BACTERIAL PASSAGE ACROSS MDCK CELL MONOLAYERS

LN Diebel, MD, DM Liberati, MS, WJ Brown, MD, SA Dulchavsky, MD, T Painter, BS, CA Diglio, PhD
Wayne State University, Depts. of Surgery, Pathology, & Microbiology
Lawrence N. Diebel, M.D.
Frederick Moore, M.D.

Detroit, Michigan

Secretory IgA (sIgA) is the primary antibody for mucosal immunity. However, the importance of sIgA in gut barrier function following shock is unknown. Tissue hypoxia is a likely primary contributing factor to bacterial translocation (BT) following shock. We, therefore, studied the impact of hypoxia and sIgA on BT in an in-vitro system.

Madin-Darby Canine Kidney (MDCK) epithelial cells were grown as a polarized monolayer in a two chamber tissue culture system. Monolayer integrity was assessed periodically by measurement of transepithelial resistance (TEER). Tissue culture media was replaced with fresh media or media containing polymeric sIgA (100μg/ml). Stationary growth phase E. coli M14 (10⁶ CFU/ml) was inoculated in the apical chamber. Tissue culture dishes were then placed in a 21% or 5% O₂ incubator environment for 90 minutes followed by a 21% O₂ environment. Aliquots were obtained from the basal compartment of the culture dishes at the end of the hypoxic period (90 minutes) and at intervals thereafter for culture.

<table>
<thead>
<tr>
<th>Group</th>
<th>90 minutes</th>
<th>240 minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>I - Control - no IgA (n=8)</td>
<td>1.51± 0.8</td>
<td>2.91± 0.27</td>
</tr>
<tr>
<td>II - Control + IgA (n=11)</td>
<td>0.58 ± 0.57*</td>
<td>2.16 ± 0.42#</td>
</tr>
<tr>
<td>III - Hypoxia- no IgA (n=22)</td>
<td>1.44 ± 0.83</td>
<td>3.52 ± 0.47$</td>
</tr>
<tr>
<td>IV - Hypoxia + IgA (n=18)</td>
<td>0.52 ± 0.62**</td>
<td>2.16 ± 0.41##</td>
</tr>
</tbody>
</table>

*p < 0.01 Gp I vs. Gp II
** p < 0.01 Gp III vs. Gp IV
by ANOVA

*p < 0.01 Gp I vs. GpII
#p < 0.01 Gp II vs. Gp III
$ p < 0.01 Gp IV vs. Gp III
## p < 0.001 Gp III vs. Gp IV

TEER remained stable throughout the experiment. BT increased with incubation time in all groups. Hypoxia facilitated BT and this effect was blocked by sIgA. Secretory IgA is important in mucosal defense during shock states.
AUTOTRANSFUSION OF ENTERICALLY CONTAMINATED INTRAPERITONEAL SHED BLOOD IN TRAUMATIC GASTROINTESTINAL PERFORATION: IS IT SAFE?
JG Chipman, MD, SB Johnson, MD, D Scanzaroli, RPA, JB Fortune, MD
Section of General Surgery and Trauma
University of Arizona Health Sciences Center
Jeffrey Chipman, MD
James Benjamin, MD
Tucson, Arizona

Introduction: Autologous transfusion of enterically contaminated shed intraperitoneal blood is controversial.

Purpose: To evaluate the risk of autologous shed intraperitoneal blood transfusion on post-operative infections in patients with traumatic gastrointestinal perforations (GIPerf).

Methods: The medical records of all patients admitted to a level 1 trauma center in a 4 year period with traumatic GIPerf were reviewed. Age, ISS, abdominal organs injured, abdominal trauma index (PATI), extent of intraabdominal contamination (EDIC), operative blood loss (EBL), volume of blood transfused (autologous and banked; intraoperative and first 24 hours), and antibiotics were collected. Dependent variables of incidence and type of postoperative infection (excluding C. difficile colitis) and mortality were collected. Patients were divided into: Group 1) GIPerf with autotransfusion, and Group 2) GIPerf without autotransfusion. Data: mean±S.D., significance: p<0.05.

Results: There were 51 patients with traumatic GIPerf who required blood transfusion. Six (11.8%) patients died including 4 intraoperatively. Groups 1 and 2 were similar in age (35±15 vs 31±14), ISS (26±17 vs 21±8), PATI (30±20 vs 24±16), EDIC (2.6±0.9 vs 2.9±0.8), volume of banked blood transfused (4.2±4.1L vs 3.1±3.8L), and total volume of blood transfused (5.4±5.2L vs 3.1±3.8L,p=0.07). Group 1 had significantly greater EBL (6.0±4.6L vs 2.6±2.7L, p>0.01) and received 1.3±1.2L of autologous blood. There was no difference between groups in the incidence of stomach, small bowel or large bowel injuries.

<table>
<thead>
<tr>
<th>Group 1</th>
<th>Group 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>n=16</td>
<td>n=35</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Mortality</th>
<th>Incidence of Infectious Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>All patients</td>
<td>Adequate Antibiotics</td>
</tr>
<tr>
<td>2 (12.5%)</td>
<td>4 (29%)</td>
</tr>
<tr>
<td>4 (11.4%)</td>
<td>18 (56%)</td>
</tr>
</tbody>
</table>

p value ns 0.08 0.004 ns

Conclusion: If adequate antibiotics are administered, autotransfusion of enterically contaminated blood results in fewer infectious complications than banked blood transfusion alone.
RANDOMIZED PROSPECTIVE EVALUATION OF THE EFFECTS OF INCREASED PRELOAD ON CARDIOPULMONARY FUNCTION AND VISCERAL PERFUSION DURING RESUSCITATION
P.R. Miller, M.D., M.C. Chang, M.D., J.W. Meredith, M.D.
The Bowman Gray School of Medicine, Wake Forest University
P.R. Miller, M.D.
M.C. Chang, M.D.
Winston-Salem, North Carolina

Purpose: To evaluate the effects of maintaining increased levels of preload on cardiopulmonary function and visceral perfusion during resuscitation.
Design/Setting: Randomized prospective study of 39 consecutive trauma patients with a low right ventricular ejection fraction (RVEF < 40%) admitted to a university level I trauma center over a 1 year period. Patients were randomized to one of two groups, increased preload (PRE), or normal preload with inotropes (INO). The PRE group received fluid administration to maintain a target right ventricular end diastolic volume index (RVEDVI) of ≥120 mL/m² during resuscitation. The INO group had inotropes added per a prospectively determined protocol, and were maintained at a RVEDVI of 90-100 mL/m².
Main Outcome Measures: Cardiac index (CI), PaO₂/FiO₂ ratio, dynamic compliance (Cdyn), ventilator days, and incidence of Adult Respiratory Distress Syndrome (ARDS). Gut perfusion was assessed by measuring gastric intramucosal pH (pHi). Data are expressed as mean±SD.
Results: PRE patients (n=19) were maintained at significantly higher RVEDVI than INO patients (n=20, Table). There was no difference in CI or studied pulmonary variables between the groups. The incidence of ARDS was not significantly different (PRE 31% vs INO 50%, p>0.01, chi-square) between groups. In the patients who had pHi measured sequentially during resuscitation (PRE=13, INO=17), the final pHi was significantly higher in the PRE group (7.31±0.14 vs 7.16±0.15, p=0.03).
Conclusions: Maintaining higher levels of preload during resuscitation is safe and has beneficial effects on gut perfusion. The development of pulmonary failure is determined by the injury and its metabolic consequences, not the style of resuscitation.

<table>
<thead>
<tr>
<th></th>
<th>PRE</th>
<th>INO</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVEDVI</td>
<td>119±18</td>
<td>102±22</td>
<td>0.01</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>3.9±1.1</td>
<td>3.9±0.9</td>
<td>0.96</td>
</tr>
<tr>
<td>PaO₂/FiO₂</td>
<td>266±115</td>
<td>217±77</td>
<td>0.13</td>
</tr>
<tr>
<td>Cdyn (mL/cmH₂O)</td>
<td>28±9</td>
<td>28±9</td>
<td>0.85</td>
</tr>
<tr>
<td>Vent Days</td>
<td>8±8</td>
<td>11±10</td>
<td>0.26</td>
</tr>
</tbody>
</table>

Unpaired t-test, significance @ p<0.05
THE EFFECT OF A QUALITY ASSURANCE - DERIVED TRAUMA AIRWAY POLICY ON REDUCING PROVIDER-RELATED ERRORS IN THE EMERGENCY DEPARTMENT

J.G. Cushman, M.D. G.S. Rozycki, M.D., D.V. Feliciano, M.D.
Grady Memorial Hospital / Emory University
J.G. Cushman, M.D.

D.V. Feliciano, MD
Atlanta, Georgia 30303

Objective: The purpose of this study was to determine the effect of a QA-derived trauma airway policy (TAP) on reducing provider-related errors in the emergency department in patients who subsequently died of their injuries.

Methods: From 1994-1995, 206 consecutive trauma deaths over a 24 month period at a Level I trauma center were reviewed at a monthly peer review "death audit". The period of study was divided into 8 months prior to implementation of the TAP (Period A: Trauma airway management solely by Emergency Medicine (EM)) and 16 months following implementation (Period B: Trauma airway management shared by EM and Anesthesiology). The TAP was developed after an unacceptable rate of provider-related errors in airway management (error in judgement/delay in intubation / error in technique) was noted during Period A. TAP mandated that an in-house anesthesiologist would perform/supervise intubations in the following circumstances: (1) < 8 years old; (2) predictably difficult airway (inability to intubate in field, severe maxillofacial injury, obesity); (3) systolic blood pressure < 100 mm Hg in ED. Errors in management were defined according to the ACS-COT Resources Document, while the periods were compared using chi-square test to assess statistical significance.

<table>
<thead>
<tr>
<th></th>
<th>Period A</th>
<th>Period B</th>
<th>p-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deaths reviewed</td>
<td>73</td>
<td>133</td>
<td>----</td>
</tr>
<tr>
<td>Blunt/penetrating/other</td>
<td>38/28/7</td>
<td>75/55/3</td>
<td>n.s</td>
</tr>
<tr>
<td>Mean ISS</td>
<td>35.3</td>
<td>34.1</td>
<td>n.s</td>
</tr>
<tr>
<td>Intubation delay errors</td>
<td>20 (27.4%)</td>
<td>13 (9.8%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Conclusions: 1. Concurrent quality assurance data identified provider-related errors in airway management, leading to the development and implementation of a hospital TAP. 2. Reassessment of this policy's effect on airway management demonstrated a significant reduction in intubation delays and errors in technique as possible contributing factors in patients dying from their injuries.
EFFECTS OF EXOGENOUS CYTOKINES ON INTRAVASCULAR CLEARANCE OF BACTERIA IN NORMAL AND SPLENECTOMIZED MICE.

J.C. Hebert, MD, M.O'Reilly, MD, B. Barry, L. Shatney, K. Sartorelli, MD.
University of Vermont College of Medicine
J.C. Hebert, M.D.

Burlington, VT 05401

Exogenous cytokine (IL-1, G-CSF and GM-CSF) pretreatment can improve alveolar macrophage bactericidal activity against S. pneumoniae (pneumococcus). These effects vary in eusplenic and asplenic mice. Likewise, these cytokines have been shown to improve survival following an aerosol pneumococcal challenge. Mice dying in these studies had positive blood cultures and disseminated infection. The purpose of this study was to determine the effect of cytokine pretreatment on intravascular clearance of bacteria from eusplenic and asplenic mice. Two weeks after splenectomy (splx) or sham operation, mice were pretreated for various times with either IL-1, G-CSF, GM-CSF, or their corresponding vehicles. Mice then received tail vein injections of bacteria (0.1 ml) and quantitative blood cultures were performed at various times thereafter. (Table)

<table>
<thead>
<tr>
<th>Log10 Pneumococci in Blood (cfu/ml) (t=30 min)</th>
<th>IL-1</th>
<th>PBS</th>
<th>G-CSF</th>
<th>D5W</th>
<th>GM-CSF</th>
<th>PBS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Splx</td>
<td>3.22±.04</td>
<td>4.05±.01</td>
<td>4.35±.02</td>
<td>4.32±.03</td>
<td>0</td>
<td>4.06±.01</td>
</tr>
<tr>
<td>Sham</td>
<td>3.87±.02</td>
<td>3.75±.01</td>
<td>1.73±.55</td>
<td>3.66±.06</td>
<td>0</td>
<td>2.79±.01</td>
</tr>
</tbody>
</table>

Splx mice had impaired clearance of pneumococci compared to shams (p<.01). IL-1 enhanced clearance in splx mice (p<.001) but not shams (p=ns). G-CSF enhanced clearance in shams (p<.001) but not splx mice (p=ns). GM-CSF enhanced clearance in both groups (p<.001). The net in vitro effects of exogenous cytokine therapy for infections depends upon the state of the host defenses at the time of therapy. These agents may be useful as adjuvants for the treatment of infections but further study is warranted.
NONOPERATIVE MANAGEMENT OF SPLENIC INJURY:
ARE FOLLOW-UP CT SCANS OF ANY VALUE?
B.C. Thaemert, M.D., T.H. Cogbill, M.D.,
P.J. Lambert, R.N., C.H. Miller, III, M.D.
Gundersen Lutheran Medical Center

B.C. Thaemert, M.D.
T.H. Cogbill, M.D.
La Crosse, Wisconsin

Hypothesis: Follow-up abdominal CT scans in patients with splenic trauma managed nonoperatively are not necessary.

Patients: 109 patients with proven splenic injuries treated at a single trauma center from 1990 to 1996. Patients who presented more than 24 hours after injury were excluded.

Methods: Retrospective review of hospital chart and outpatient clinic records. All abdominal CT scans were reviewed by the authors.

Results: Initial management was surgical in 35 (32%) patients and intentionally nonoperative in 74 (68%) patients. Nonoperative management was successful in 46 (92%) of 50 adults and 22 (92%) of 24 children. 61 follow-up abdominal CT scans were obtained in 44 patients. Multiple, asymptomatic intrasplenic pseudoaneurysms were demonstrated on a follow-up CT scan in one patient. Information which affected management was not evident on any of the remaining follow-up CT scans in the absence of a change in clinical findings. Potential savings in hospital charges for these routine follow-up CT scans approaches $100,000.00.

Conclusion: Follow-up abdominal CT scans are not routinely necessary in patients with splenic injuries managed nonoperatively. Clinical indications should dictate the need for follow-up imaging techniques.
A COST EFFECTIVE METHOD FOR BEDSIDE INSERTION OF VENA CAVAL FILTERS IN TRAUMA PATIENTS
CR Nunn, MD, D Neuzil, MD, JG Bass, BA, R Pierce, RN, JA Morris, Jr., MD
Vanderbilt University Medical Center
Craig R. Nunn, MD
John A. Morris, Jr., MD
Nashville, TN

Objective: The benefit of inferior vena cava (IVC) filter placement in select trauma patients has been reported. The charges related to IVC filter placement are substantial. The need for patient transport to the operating room or the radiology suite impacts patient charges, patient comfort and nursing care. We hypothesized bedside, ultrasound guided IVC filter placement to be an acceptable, cost effective alternative.

Design: Prospective cohort study of 55 consecutive trauma patients requiring IVC filter placement at a single level one trauma center.

Methods: During a thirteen month period (8/95 - 9/96), all trauma patients meeting criteria for IVC filter were evaluated for ultrasound guided filter placement. Complications during and after placement were recorded. The financial savings attributable to bedside, ultrasound guided IVC filter placement were determined by subtracting the common charges of all methods of filter placement from the total charges.

Results: Of 3,172 trauma admissions, 55 patients (1.7%) met IVC filter criteria and 49 patients (89.1%) had IVC filters placed at the bedside under ultrasound guidance. 67% percent of the cohort group were male and the mean age was 31 years. The mean ISS was 30.3 (16-54). In six patients (10.9%), ultrasound guided filter placement failed. Failure resulted from non-visualization of the right renal vein in 5 patients and the IVC was too large (>3.4cm) for the available filters in the remaining patient. There were 4 complications (8.2%) in 4 patients including: 1 tilted filter, 1 DVT at the needle puncture site, 1 IVC occlusion and 1 minor filter migration. Over 13 months, charges were reduced by $76,500 when compared to radiology suite placement ($1,500 / pt) and $107,100 when compared to operative placement ($2,100 / pt).

Conclusions: Ultrasound guided, bedside placement of IVC filters is a safe, cost effective method of providing pulmonary embolism prophylaxis in select trauma patients.
FASCIO TOMY, CHRONIC VENOUS INSUFFICIENCY AND THE CALF MUSCLE PUMP
K. Bermudez, M.D., M. Knudson, M.D.,
D. Morabito, RN, MPH, O. Kessel, B.S.
San Francisco General Hospital, University of California, San Francisco
Presenter: K.M. Bermudez, M.D.
Senior Sponsor: M. Margaret Knudson, M.D.
San Francisco, California

Objectives: The long term effects of lower extremity fasciotomy are largely unknown. We hypothesized that fasciotomy may impair the calf muscle pump, which in turn could result in the development of chronic venous insufficiency.

Methods: To test this hypothesis, patients with a history of unilateral lower extremity fasciotomy were invited to participate in the study. All patients received a review of their medical and functional history, a detailed physical exam of both lower extremities, and air plethysmography (APG) studies. The contralateral limb was used as a control.

Results: Of 17 patients who completed the study, eight had vascular injuries, six had fractures, and three had soft tissue infections as a primary diagnosis. The time frame from injury to exam ranged from 6 months to 20 years. The APG data showed significant mean differences between fasciotomy and controlled extremities in ejection fraction (EF) (p=0.000) and residual volume fraction (RVF) (p=0.000), both measures of calf muscle pump function. There were no significant changes in either venous filling index (VFI), which is a measure of venous reflux, or outflow fraction (OF), which correlates with venous obstruction (see graph). Furthermore, there were no differences in APG variables between vascular and orthopedic or soft tissue injuries.

Conclusions: Lower extremity fasciotomy impairs long term calf muscle pump function, as measured by APG, in patients with and without vascular injuries. Fasciotomy independently may lead to the development of chronic venous insufficiency following lower extremity trauma.

![Graph showing mean values for VFI, EF, RVF, and OF between control and injured limbs.]

p = 0.000
SIGNIFICANCE OF FREE FLUID ON ABDOMINAL CT
K Brase MD, C Olson MD, R Stafford MD, T Johnson MD
St. Paul-Ramsey Medical Center, University of Minnesota
T Johnson MD
S Petersen MD
St. Paul, Minnesota

In evaluation of blunt abdominal trauma, free fluid on abdominal CT scan without solid organ injury is considered by many to be evidence of hollow viscus injury requiring laparotomy. Others suggest this is normal or requires only close observation or further diagnostic tests. The purpose of this study was to determine the incidence and significance of free fluid on abdominal CT scan obtained in evaluation of blunt trauma.

All patients with an abdominal CT scan obtained in the initial evaluation of blunt trauma from 8/93-12/95 were identified from the trauma registry at a level 1 trauma center. All CTs were performed on a GE HiSpeed Advantage helical scanner with 10 mm cuts through the abdomen and pelvis. Intravenous contrast was used for all; the use of oral contrast was variable due to a concurrent prospective, randomized study evaluating its use. 1159 CT scans were performed during this period; records of 36 patients were excluded due to incomplete medical records. The remaining 1123 patients comprise this review. Patient records were reviewed for age, sex, ISS, length of stay, ICU length of stay, abdominal injuries, bladder injury, and pelvic fracture. Records were also reviewed for additional evaluation with DPL, whether the patient underwent a laparotomy, and postoperative complications. Official CT reports were reviewed for the presence of free fluid, solid organ injury, bladder injury, and pelvic fracture.

The incidence of free fluid on abdominal CT scan was 12.8%. Free fluid without solid organ injury was found in 3% (34/1123). Two patients had a DPL prior to CT and 4 had bladder rupture accounting for the free fluid. Fluid was attributed to pelvic fracture in 3 patients. Two patients died from severe head injury before laparotomy.

Laparotomy was performed solely because of free fluid in 13 patients. Small bowel injuries were found in 6, and a diaphragm injury in 1. Five patients had non-therapeutic laparotomies (1 without injury, 2 non-significant mesenteric injuries, 1 non-bleeding omental injury, 1 non-bleeding liver laceration, and 1 extraperitoneal pelvic hematoma). Ten patients had trace amounts of free fluid (6 female, 4 male) and did not undergo laparotomy; none had a missed small bowel injury.

In patients with more than a trace of unexplained free fluid on abdominal CT scan the therapeutic laparotomy rate is 54%. This confirms that free fluid without solid organ injury or other explanation should mandate exploratory laparotomy.
CROTALID ENVENOMATION,
THE SOUTHERN ARIZONA EXPERIENCE
JT Tokish, MD, JB Benjamin MD, F Walter MD

The University of Arizona Health Sciences Center,
Department of Surgery, Section of Orthopedic Surgery
JT Tokish, MD
JB Benjamin, MD
Tucson, AZ 85724-5064

INTRODUCTION: To characterize the experience with snakebite injuries, records were reviewed for pre-hospital management, patient demographics, and treatment protocol, including surgical intervention. The charts of all patients admitted to the five major hospitals, including two level one trauma centers, in Southern Arizona following venomous snake bites between 7/90 and 3/96 were reviewed.

RESULTS: Of the 170 patients admitted during the study period, 164 charts were available for review. Seventy-two percent of those admitted were male. A majority of the admissions occurred between April and October and resulted from a single rattlesnake bite. Eighteen patients self-administered field treatment following envenomation, such as “cut and suck” and tourniquets. Thirty-eight percent were transported to treating hospitals via helicopter. Seventy-three percent were admitted to the ICU and the mean hospital stay was 2.8 days (range 1-8). A vast majority (94%), received antivenin and 20% experienced an anaphylactoid reaction resulting from this treatment. Patients were monitored with serial laboratory studies with frequently noted elevation of the PT and PTT as well as abnormal fibrinogen and fibrin spitz product levels. Thrombocytopenia was noted in 47% of patients and was frequently severe. Eleven percent required a surgical procedure as a result of the envenomation including six who required fasciotomy for compartment syndrome. There were no fatalities, however one patient required an amputation.

CONCLUSION: As a result of this review it was felt that field management of snake bites, including tourniquet application and “cut and suck,” was ineffective and often detrimental to outcome. Helicopter transport and ICU was, in many cases, over-utilized. Compartment syndrome is a rare but real complication of rattlesnake envenomation and compartment pressures should be monitored in cases where this diagnosis is considered. Although hospital stays were short, the treatment of snake envenomations utilizes significant medical resources.
COST ANALYSIS OF PEDIATRIC TRAUMA CARE BY COST
CENTERS AND MECHANISMS OF INJURY AS A BASIS
FOR INJURY PREVENTION AND CLINICAL GUIDELINES
K. Achanta, MD, Q. Chu, MD, J. Garb, MS, B. Simon, MD, T.
Emhoff, MD, V. Fiallo, MD, K. F. Lee, MD.
Baystate Medical Center

K. Achanta, MD
H. Sugerman, MD (Richmond, VA)
Springfield, MA

In response to capitated managed care, clinical guidelines are often developed to reduce unnecessary hospital expenditure. Injury prevention programs are also implemented to decrease major trauma cases requiring hospital admission. Yet the financial data detailing potential areas of cost-reduction for these programs are lacking. The purpose of our study was to evaluate the cost profile of pediatric trauma care in order to identify major target areas of cost reduction.

In a retrospective analysis, we studied all pediatric trauma patients aged between 0-18 years admitted to a Level I Trauma Center between 1/93 and 1/96. We used a cost-accounting system based on relative value units assigned to fixed overhead expenses such as salary, institutional operating costs, and capital, as well as variable costs of goods and services which vary with usage. For each patient we calculated the fixed cost (FC), the variable cost (VC), and the total cost of care (TC). We then further divided these costs into cost centers relating to type of patient care: Nursing, Emerg.-Dept., ICU, Operating Room, Radiology, Laboratory, Pharmacy, and Physiatry.

For each cost center (CC) and mechanism of injury (MOI), we calculated the mean TC. Using a repeated measures analysis of variance, we tested the null hypothesis that the differences in costs across CC's and MOI's were due to chance.

There was a significant difference in mean TC among the CC's. The CC's with the highest mean TC were in decreasing order: Nursing ($2,126 ± 203), Operating Room ($584 ± 74), Radiology ($516 ± 26), Laboratory ($483 ± 81), Physiatry ($426 ± 53), Emergency Department ($361 ± 8), Pharmacy ($317 ± 44), and ICU ($302 ± 93). (p<0.0001). The cost of nursing far surpassed the next three highest CC's. Considering that nursing cost is directly related to the length of stay (LOS), these data suggested reduction of LOS as the principal strategy for clinical guidelines. But the evaluation of variable-to-fixed cost ratio (VFR) revealed specific cost-saving strategies for different CC's. Pharmacy had a high variable cost component (VFR 2.53), indicating containment of incremental usage of goods and services as a major cost-saving strategy. Operating Room, Radiology and Laboratory had a relatively high fixed cost component (VFR 0.55 to 0.68), suggesting containment of overhead expenditures as a more effective means of cost reduction.

MOI's with a significantly higher mean TC were gunshot ($11,408 ± 3266) and pedestrian ($9,086 ± 1902) injuries, compared to a MOI with significantly lower mean TC, fall ($4,066 ± 657). (p = 0.0001)

The frequency of individual MOI's varied significantly across age groups. (P<0.05) Analysis of the total cumulative cost (mean TC x frequency) identified a specific MOI that expended the most healthcare dollars for each age group. Age 0-5: fall injuries ($113,440/yr). Age 6-10: pedestrian injuries ($145,755/yr). Age 11-15: bicycle injuries ($90,775/yr). Age 16-18: unbelted motor vehicle injuries ($208,442/yr).

Our study provides a useful cost-accounting model which can assess pediatric trauma care cost based on CC's and MOI's. Prevention strategies should be targeted towards MOI that generates the highest cost in each age group. Development of clinical guidelines should include analysis of variable and fixed costs of each CC. Consideration of economic factors that identify high-expenditure cost centers and mechanisms of injury will become increasingly important for Level I Trauma Centers in the managed care, capitated system.
ULTRASOUND IS A COST-EFFECTIVE TRIAGE TOOL TO EVALUATE BLUNT ABDOMINAL TRAUMA IN THE PEDIATRIC POPULATION
Denver Health Medical Center, University of Colorado
D.A. Partrick, M.D.
E.E. Moore, M.D.

Denver, Colorado

Background: The appropriate cost-effective evaluation of injured children presenting with blunt abdominal trauma (BAT) continues to be a challenge for regional trauma centers. Computed tomography (CT) has been considered the diagnostic modality of choice for pediatric patients with BAT. However, CT is costly, time-consuming, requires sedation, and may be associated with complications in young children. Abdominal ultrasonography (US) is a promising modality in the evaluation of BAT in that it is quick, noninvasive, repeatable, and cost-effective. Although evaluated in adult trauma, US has not been studied as thoroughly in injured children. We hypothesized that emergency department US, performed by trauma surgeons, is a useful triage tool for pediatric BAT that reduces the need for CT.

Methods: An US based key clinical pathway was implemented after an US training program. From 7/94 to 4/96, 188 children (2 months-17 years of age) with suspected BAT were evaluated with emergent US by surgeons. The presence of intraperitoneal (IP) fluid or parenchymal injury was interpreted as a positive result. Subsequent CT scan or exploratory laparotomy was performed as indicated by the key clinical pathway. Data were collected prospectively and are reported as mean ± SEM.

Results: The mean age of the 188 study patients was 10.3 ± 4.0 years, 116 (61%) were male and their mean ISS was 6.5 ± 0.8. Only 8 of these children (4.3%) had documented intraabdominal injuries. Results were compared between US and CT.

<table>
<thead>
<tr>
<th>BAT Evaluation (US and CT)</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>US (-) and no CT</td>
<td>106</td>
</tr>
<tr>
<td>US (+) and CT (-)</td>
<td>74</td>
</tr>
<tr>
<td>US (-) and CT (+)</td>
<td>6</td>
</tr>
<tr>
<td>US (+) and CT (+)</td>
<td>2</td>
</tr>
</tbody>
</table>

All injured children with significant IP fluid were identified by US. Of the six pediatric patients who had a negative US followed by a positive CT scan, five sustained minor solid organ injuries that were managed nonoperatively (three liver hematomas, one renal laceration, and one adrenal hemorrhage). One child had gas in the soft tissues around the bladder, evidenced by CT, and was found to have a bladder neck laceration on exploration. No false negative US exam had significant IP fluid by CT. Extrapolated reductions in hospital charges based on the reduced number of CT scans total $231,080 (106 patients who did not have a CT, [106CT x $2180]).

Conclusions: We have confirmed that pediatric BAT patients referred to an urban level 1 trauma center have a low incidence of intraabdominal injury. US had a positive predictive value of 100% and a negative predictive value of 99.5% for detecting intraabdominal injuries requiring emergent laparotomy. Therefore, similar to the adult trauma population, using US as a triage tool may dramatically decrease the cost of pediatric BAT evaluation while being able to quickly identify injuries that require emergent exploratory laparotomy.
HYPERTONIC SALINE IS NOT BENEFICIAL FOR RESUSCITATION OF PULMONARY CONTUSION

S.M. Cohn, MD, B.T. Fisher, BS, and A.T. Rosenfield, MD
Departments of Surgery and Radiology,
Yale University School of Medicine New Haven, CT 06520

OBJECTIVE: We postulated that hypertonic solutions could minimize the accumulation of lung water and subsequent respiratory derangements which occur after pulmonary contusion in a porcine model.

METHODS: Anesthetized pigs (15-20kg) underwent contusion to the right chest at t=0min; were hemorrhaged (30cc/kg) from t=0-20min; resuscitated with 7.5% NaCl (HTS=4cc/kg, n=10) or 0.9% saline (NS=90cc/kg, n=8) from t=20-40min, and observed to t=240min. Gravimetric lung weights, and spiral CT scan (n=4 scans/group) quantitated lung water.

RESULTS:

<table>
<thead>
<tr>
<th>GROUP</th>
<th>t=0</th>
<th>t=20</th>
<th>t=40</th>
<th>t=120</th>
<th>t=240</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP NS</td>
<td>89±3</td>
<td>50±4*</td>
<td>81±3#</td>
<td>94±4</td>
<td>87±6</td>
</tr>
<tr>
<td>HTS</td>
<td>93±2</td>
<td>52±3*</td>
<td>72±3*</td>
<td>83±3</td>
<td>78±5</td>
</tr>
<tr>
<td>MPAP NS</td>
<td>15±2</td>
<td>13±2</td>
<td>22±2*</td>
<td>17±2</td>
<td>17±2</td>
</tr>
<tr>
<td>HTS</td>
<td>17±2</td>
<td>12±2</td>
<td>18±2</td>
<td>14±1</td>
<td>11±1</td>
</tr>
<tr>
<td>PaO₂ NS</td>
<td>110±12</td>
<td>90±8</td>
<td>82±8*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HTS</td>
<td>100±5</td>
<td></td>
<td></td>
<td>84±3</td>
<td>82±3</td>
</tr>
<tr>
<td>COMP NS</td>
<td>20±1</td>
<td>15±1*</td>
<td>15±1*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>HTS</td>
<td>19±1</td>
<td>16±1*</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Values=means±SEM, *=value vs t=0 within group, # =value vs HTS group, p<0.05 ANOVA-Tukey's test, MAP=mean arterial pressure [mmHg], MPAP=mean pulmonary arterial pressure [mmHg], PaO₂=arterial oxygen tension [mmHg], COMP=static complancice [ml/mmHg].

Lung weights were similar in both groups: %lunng water [g/kg BW]= Right: NS-12.5±2.1 vs. HTS-13.2±1.1, Left: NS-8.4±0.6 vs. HTS-10.3±1.3. CT volumes were not statistically different (NS-5.9±2.7 vs. HTS-4.5±1.0, L/kg BW).

CONCLUSIONS: HTS did not prevent the accumulation of lung water or the subsequent hypoxemia and worsening of compliance after traumatic lung injury. Resuscitation with small volumes of HTS, therefore, does not appear to provide benefit over large volumes of isotonic crystalloids in this porcine pulmonary contusion model.
DIAGNOSTIC LAPAROSCOPY FOR THE EVALUATION OF PENETRATING ABDOMINAL TRAUMA

Department of Surgery, St. Louis University
Presenter: Rodney M. Durham, M.D.
Senior Sponsor: Scott Petersen, M.D.
St. Louis, MO

Diagnostic laparoscopy has been advocated as a means of selecting patients requiring laparotomy for penetrating abdominal trauma. To evaluate the use of laparoscopy for this purpose, we performed a prospective trial in which consenting, hemodynamically-stable patients undergoing exploratory laparotomy for penetrating abdominal trauma had a preliminary diagnostic laparoscopy performed prior to laparotomy. All patients underwent laparotomy regardless of the laparoscopic findings. A total of 77 patients were entered into the trial; 73% had suffered gunshot wounds, 5% shotgun injuries, and 22% stab wounds. The entrance site was the anterior abdomen in 47%, the lower chest in 16%, the flank in 17%, the back in 13%, and multiple sites in 8%. In 53 patients, laparoscopy was considered positive because it revealed peritoneal penetration (74%), hemoperitoneum (70%), or an injury to an abdominal organ (55%). At subsequent laparotomy, injuries requiring operative repair were identified in 47 (89%) of these patients; 6 patients (11%) had nontherapeutic laparotomies. In 24 patients, laparoscopy was considered negative because it did not reveal peritoneal penetration, hemoperitoneum, or an injury to an abdominal organ. At laparotomy, 23 (96%) of these patients had no significant injury. One patient with a gunshot wound to the back and gross hematuria had an isolated injury to the kidney. Overall, 47 of 48 patients requiring operative repair of an injury were identified based on positive laparoscopic findings, resulting in a sensitivity of 98%. In addition, 23 of 29 patients who had nontherapeutic laparotomies were identified by negative laparoscopic findings, giving it a specificity of 79%. However, laparoscopy did not accurately predict which abdominal organs were actually injured. At least one significant injury was missed on initial laparoscopy in 77% of the 47 patients who had therapeutic laparotomies. Thus, laparotomy can be deferred in stable patients with penetrating trauma if laparoscopy shows no evidence of peritoneal penetration, hemoperitoneum, or an abdominal organ injury. Exploration is warranted in patients with any one of these positive laparoscopic findings, even in the absence of an obvious injury.
CONTENT ANALYSIS OF PEDIATRIC TRAUMA IN THE MEDIA

E.J. DOOLIN, M.D., & A.M. BROWNE, M.S.N.
COOPER HOSPITAL / UNIVERSITY MEDICAL CENTER
E.J. DOOLIN, M.D.
S.E. ROSS, M.D.
CAMDEN, NEW JERSEY

OBJECTIVE: To analyze information distributed in local newspapers related to pediatric trauma.

METHOD: Articles related to pediatric trauma were collected from two newspapers for six months (n=400). Data included mechanism, outcome, size and location of article in newspaper. Data was compared to incidence of injuries and outcomes as reported by the National Pediatric Trauma Registry (NPTR).

RESULTS: 400 articles were reviewed, 317 feature and 83 capsule reports. Mechanism of injury and outcome were compared to the NPTR (media/NPTR): assaults (25%/7%), gunshot wounds (24%/5%), stab wounds (4%/5%), motor vehicle crash (7%/19%), bicycle (<1%/9%), pedestrian (5%/15%), falls (2%/26%), recreational injuries (2%/6%), intentional injuries (58%/13%), death (56%/3%), sexual abuse/neglect (13.5%/4%). Article type analysis revealed: case reports (63%), public opinion (33%), objective evaluations (4%). Front page articles (12%) related to violent crimes 70% of the time. Article size was not a function of mechanism of injury, but fewer reports of death appeared in capsule articles (7%) than overall (56%). Safety articles accounted for 5%.

CONCLUSION: Media over reports violent crimes and death, and under reports motor vehicle crashes, falls, and unintentional injuries. Safety and prevention are lacking, anecdotal and opinion oriented articles are the majority. Trauma education efforts must recognize the misrepresentation of this disease.

CONCLUSION: In-house personnel improved efficiency for the less severely injured, and an in-house attending surgeon reduced mortality in the severely injured older patient. None of the other variables were found to have a significant impact on outcome.
Neurotrauma care is integrally related to the pool of neurosurgeons available and willing to treat injured patients. Many factors can influence the commitment of neurosurgeons to participate in trauma care. This study seeks to determine the degree of reluctance to care for trauma patients and those factors associated with such a reluctance among neurosurgeons in comparison to other surgical specialties.

A national sample of 2503 general, orthopedic and neurological surgeons was scientifically selected from the AMA Masterfile of Physicians and surveyed by mail regarding a number of trauma care issues. The neurosurgical sub-sample numbered 215 and is representative of their geographic and professional distribution among all surgeons. Analysis for this study is limited primarily to neurosurgical respondents.

Response rate was 60%. 20% of neurosurgeons reported that they prefer not to care for trauma patients. This was less than the reluctance expressed by other surgical specialties. One third, however, expressed that they would not take trauma call were it not required. Preference to treat varied by geographic region and community size. Neurosurgeons between the ages of 40 and 50 were the most reluctant to treat trauma patients. Compensation was not a major factor associated with reluctance to treat these patients. Perceptions of increased time commitment, personal factors (such as family stress, physical demands and risk of infection) and perceived commitment of the hospital and other providers appeared to be greater factors in a surgeon’s decision. Perception of increased medicolegal risk was also an important factor in reluctance to treat trauma patients, however, neurosurgeons are less likely to be influenced by malpractice litigation than other surgical specialists.

Neurosurgical participation in trauma care is vital both to the injured and the public perception of neurosurgeons as serving community needs. Further efforts to discern myth from fact and dispel myths while addressing true problems, where possible, seem warranted.
MANAGEMENT OF SELF-INFLECTED LOW-ENERGY GUNSHOT WOUNDS THAT VIOLATE THE SKULL BASE
DAVID J. KRIET, M.D., R.B. STANLEY, M.D.,
M.S. GRADY, M.D.
HARBORVIEW MEDICAL CENTER
UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE
D.J. KRIET, M.D.
G.J. JURKOVICH, M.D.
SEATTLE, WASHINGTON

Self-inflicted ballistic wounds aimed at the brain through the mouth or from below the chin are usually shotgun or high-energy gunshot wounds. Management of these wounds, which typically produce large avulsion-type injuries of the bone and soft tissue of the face, has been extensively discussed in the literature. Management of self-inflicted low-energy gunshot wounds with the same sites of entrance has not been specifically addressed. We, therefore, present our experience with nine of these self-inflicted, low-energy gunshot wounds seen during a 2 year period.

All patients underwent a frontal craniotomy, and two developed serious postoperative intracranial complications not related to the brain injury itself. One patient developed an extradural frontal abscess, and one developed progressive pneumocephalus. The common factor for these patients was an injury through the roof of the ethmoid sinus that was not repaired with a bone graft. The patients were retreated using split cranial bone grafts, and both healed without further difficulty. Three other patients with similar injuries repaired initially with bone grafts experienced no complications. Four patients with injuries anterior (floor of frontal sinus) or lateral (orbital roof) to the ethmoid sinus were also successfully treated without bone grafts.

Low-energy submental or transoral gunshot wounds, in particular those created by a handgun, appear less likely to exit through the face without violating the skull base than wounds caused by shotguns or high powered rifles. Instead, they follow the usually intended path through the palate, nose, paranasal sinuses, and skull base into the brain. The lack of external evidence of destruction of the facial skeleton, as well as the need for urgent attention to the brain injury, may lead to an overlooked serious anterior skull base injury. Split cranial bone grafts should be considered as a supplement to dural repair for injuries involving the central third of the anterior skull base, even if a pericranial flap is used. The bone grafts provide the best initial seal of the skull base for protection against ingress of air and/or bacteria from the nose and ethmoid sinuses.
PATIENTS WITH GUNSHOT WOUND TO THE HEAD DO NOT REQUIRE CERVICAL SPINE IMMOBILIZATION & EVALUATION
K.L. Kaups, M.D., J.W. Davis, M.D.

VALLEY MEDICAL CENTER

K.L. Kaups, M.D.

J.W. Davis, M.D.

Fresno, California

Purpose: Cervical spine injury (CSI) has been reported as occurring in up to 20% of head injured patients. Patients with gunshot wounds to the head (GSW-H) do not incur the same forces as those having blunt head trauma, but have customarily had cervical spine immobilization (IMMOB) and subsequent evaluation. This study was undertaken to determine the necessity of IMMOB and cervical spine evaluation in patients with GSW-H.

Methods: Trauma registry records of all patients sustaining GSW to the head from 7/90 to 9/95 were reviewed. Data abstracted from the records included age, sex, ED GCS, other injuries, IMMOB, intubation, survival and autopsy data.

Results: There were 215 patients identified with GSW-H during the study period. The average age was 28 years (range 2-87 years) and the average ED GCS was 8 ± 0.36. There were 123 deaths (57%) and 64 patients (29%) died in the ED.

Of the 215 patients, 180 (84%) had IMMOB. Cervical spine clearance was performed in 199 patients (93%), either clinically (42), radiographically (84) or at autopsy. Twelve suicide patients had no autopsies performed and the autopsy report on one homicide patient could not be located. There was no clinical suspicion of CSI in any of these patients. There were no missed CSI and no CSI from indirect injury. Three patients had CSI from direct bullet injury, but all had CSI suspected based on their wounds.

Field intubation was attempted in 36 patients and was unsuccessful in 16 (42%); all but 2 of these patients had IMMOB during these intubation attempts. Thirty-four IMMOB patients had 49 intubation attempts versus 5 in the 4 non-IMMOB patients ($\chi^2$, p = 0.008). Additionally, six field intubation patients required reintubation at ED arrival; all but one had IMMOB ($\chi^2$, p < 0.001). Twenty-two patients had successful field intubation.

Conclusions: IMMOB of the patient with GSW-H is unnecessary and may delay definitive airway management and inhospital care. In the patient who does not have direct bullet injury to the neck, "clearing" the neck adds unnecessary time and expense to the resuscitation.
COMPELLING INJURIES: THE FOCUS OF TRAUMA TRIAGE, SYSTEMS, AND CARE

H.R. Champion, FRCS(Edin), B.M.
Cushing, MD, A.C. Malliaris, PhD, W.J.
Sacco, PhD, Louis V. Lombardo, BS

National Study Center for Trauma and EMS University of Maryland at Baltimore

H.R. Champion, FRCS(Edin)
Baltimore, MD

No clear definition exists for the anatomical injuries that require the focused attention of and triage to prompt definitive trauma care. Evaluation of triage guidelines has relied on dependent variables such as ISS \( \geq 15 \), need for ICU or surgery, etc., all of which have widely documented problems. In an age of managed care, cost efficiency and accountability, triage guidelines for trauma care need to be more precise. Objective: Define injuries that need prompt definitive trauma care—termed "compelling injuries". Methods: HICDA-9-CM, AIS-85 and AIS-90 anatomical taxonomies were reviewed and, from each a threshold of severity or urgency was used to define "compelling injuries". Each compelling injury in each taxonomy was noted. The definition was tested on two datasets to determine whether it crisply captured the defined patient population and identified predictors of such injuries in automobile crashes subjects. The two datasets were (i) National Accident Sampling System (NASS), a DOT NHTSA dataset of some 15,000 injured patients collected between 1988-1995, and (ii) MTOS Controlled Sites database, composed of about 13,500 patients from four Level I Trauma Centers. Results: In NASS, 4% of all injured occupants had compelling injuries or died. In MTOS, 37% of motor vehicle occupants had compelling injuries or died.

<table>
<thead>
<tr>
<th>Compelling</th>
<th>Non-Compelling</th>
</tr>
</thead>
<tbody>
<tr>
<td>NASS Database of Deaths and Injuries in Towaway Crashes</td>
<td></td>
</tr>
<tr>
<td>Hospitalized &amp; Alive</td>
<td>60.5%</td>
</tr>
<tr>
<td>Dead Prehospital or In-hospital</td>
<td>34.7%</td>
</tr>
<tr>
<td>Transported, but not admitted</td>
<td>4.8%</td>
</tr>
<tr>
<td>MTOS Database of Controlled Site Admitted Auto Crash</td>
<td></td>
</tr>
<tr>
<td>In-hospital mortality</td>
<td>22%</td>
</tr>
</tbody>
</table>

In the NASS database, "compelling injuries" captured all pre- and in-hospital deaths; the MTOS database contains only in-hospital deaths. The death rates are thus highly compatible. Compelling injuries were then used as a dependent variable to redefine relationships with crash signature data, e.g., Delta Velocity, Speed, Direction of Force, Rollover, Occupant Age. The results provide an essential basis for scientific re-evaluation of triage from the scene of automobile crashes and for the Automated Crash Notification systems currently being tested. ( Funded by NHTSA contract
MAXILLOFACIAL INJURIES IN THE NATIONAL HOCKEY LEAGUE: A SINGLE TEAM EXPERIENCE
G.L. Lanzi, DMD, J.E. Burke, DMD, A.J. Fedeli, DMD
Cooper Hospital/University Medical Center
G.L. Lanzi, DMD

Camden, New Jersey

Maxillofacial injuries are commonplace in the National Hockey League. The size and speed of the players along with speed and nature of the game contribute to the likelihood of trauma. Injuries range from simple contusions, lacerations and dental injuries to more complex problems including severe lacerations, fractures, brain injuries and eye injuries. Causative agents include sticks, the puck, skate blades, physical contact (legal and illegal) and of course fighting. Mandatory helmets have helped to reduce injury, and increasing use of mouthguards and face shields throughout all levels of hockey have further lessened the frequency and severity of maxillofacial trauma. One team's recent experience with maxillofacial injuries will be discussed including injury severity, causality, disability relative to playing time and methods of repair. Data from the National Hockey League injury registry will be reviewed.
CARDIOPULMONARY AND VISCERAL EFFECTS OF OPERATIVE DECOMPRESSION ON PATIENTS WITH INTRA-ABDOMINAL HYPERTENSION

M.C. Chang, M.D., P.R. Miller, M.D., J.W. Meredith, M.D.
The Bowman Gray School of Medicine, Wake Forest University
M.C. Chang, M.D.
M.C. Chang, M.D.
Winston-Salem, North Carolina

Objective: Increased intra-abdominal pressure (IAP) compromises cardiopulmonary function and visceral perfusion. Our goal was to characterize acute changes in these subsystems associated with operative abdominal decompression.

Patient Population: Consecutive series of 11 injured patients monitored with a pulmonary artery catheter and nasogastric tonometer in whom operative decompression was performed. Indications for decompression included oliguria or progressive acidosis despite aggressive resuscitation in the presence of an elevated IAP (>25mmHg).

Main Outcome Measures: Studied hemodynamic variables included pulmonary artery occlusion pressure (PAOP), right ventricular end-diastolic volume index (RVEDVI), and cardiac index (CI). Pulmonary variables included shunt fraction (Qs/Qt) and dynamic compliance (C_{dyn}). Visceral perfusion was assessed using urine output 4 hrs pre- and post-decompression (UOP) and gastric intramucosal pH (pHi). Mean values before (PRE) and after (POST) decompression were compared using the paired t-test. Linear regression and Fisher’s z transformation were used to evaluate the relationships between RVEDVI, PAOP, CI, and IAP. IAP was transduced via bladder pressures.

Significance was defined as p<0.05. Data expressed as mean ± std dev.

Results: PRE and POST values of the studied variables are shown in Table 1.

<table>
<thead>
<tr>
<th>Variable</th>
<th>PRE</th>
<th>POST</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>IAP (mmHg)</td>
<td>49±11</td>
<td>19±6.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>RVEDVI (mL/m²)</td>
<td>83±18</td>
<td>110±24</td>
<td>0.01</td>
</tr>
<tr>
<td>CI (L/min/m²)</td>
<td>3.7±0.6</td>
<td>3.9±0.8</td>
<td>0.44</td>
</tr>
<tr>
<td>Qs/Qt (%)</td>
<td>33±12</td>
<td>21±12</td>
<td>0.04</td>
</tr>
<tr>
<td>C_{dyn} (mL/cmH₂O)</td>
<td>13±5.0</td>
<td>24±5.8</td>
<td>.002</td>
</tr>
<tr>
<td>UOP (mL)</td>
<td>422±340</td>
<td>753±507</td>
<td>0.01</td>
</tr>
<tr>
<td>pHI</td>
<td>7.15±.13</td>
<td>7.20±.14</td>
<td>0.01</td>
</tr>
</tbody>
</table>

RVEDVI improved independent of CI, and correlated better (p<0.01) with CI (r=.49, p=.04) than PAOP did (r=-.36, p=.09). PAOP correlated significantly with IAP (r=0.45, p=.04).

Decompression resulted in significant improvements in Qs/Qt, C_{dyn}, UOP and pHI.

Conclusions: Abdominal decompression in patients with increased IAP improves preload, pulmonary function, and visceral perfusion. Elevated IAP has important effects on PAOP, and makes the PAOP an unreliable index of preload in these patients.
50 cc NS in Foley via aspiration port, then needle in port connected to arterial pressure monitor, clamp Foley distally.
ABDOMINAL ULTRASOUND IS NOT A SENSITIVE DETECTOR OF INTRAABDOMINAL INJURY
KF O'Malley MD, H Sariol MD, G Cains MD, M DiMarcangelo DO, and SE Ross MD
Cooper Hospital/University Medical Center
KF O'Malley

Camden, New Jersey

We conducted a prospective study in order to assess the efficacy of abdominal ultrasound (US) by surgeons with limited US training in a Level I trauma center. Eight attending trauma surgeons performed US on 455 patients over an 18 month period. US was followed by CAT scan or diagnostic lavage as clinically indicated. Blinded radiologist ultrasonographers reviewed 400 of these studies. Actual diagnoses were obtained on all cases from medical record. Results are tabulated below:

<table>
<thead>
<tr>
<th></th>
<th>True(+)</th>
<th>False(+)</th>
<th>True(-)</th>
<th>False(-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma</td>
<td>44</td>
<td>7</td>
<td>382</td>
<td>22</td>
</tr>
<tr>
<td>Radiology</td>
<td>35</td>
<td>2</td>
<td>340</td>
<td>23</td>
</tr>
</tbody>
</table>

Sensitivity in both groups was poor (66.6% vs 60.4%). Although most false negatives were trivial injuries accompanied by small amounts of fluid, there were a number of missed injuries which were potentially life threatening. Sensitivity was much better in the 54 unstable patients (87%), presumably due to larger volumes of intraperitoneal blood simplifying the interpretation.

Although abdominal US promises to be a valuable tool in the initial evaluation of trauma victims, particularly those with hemodynamic instability, it is unlikely to completely replace CAT scan or diagnostic peritoneal lavage.
TEST-BASED IMPROVEMENTS FOR MOBILE FIELD SURGICAL TEAMS
A.T. Putnam, M.D., R. Karulf, M.D., A. Olsewski, M.D.,
D. Bradley, PhD., D. Kissinger, M.D.
Wilford Hall Medical Center

A. T. Putnam, M.D.
D. Kissinger, M.D.
San Antonio, Texas

INTRODUCTION: The Mobile Field Surgical Team (MFST) is a five person military field trauma surgical unit. Carrying 300 pounds of surgical equipment and supplies, the team is tasked to perform 10 “damage control” trauma procedures in a 24-hour period. Weight restrictions limit surgical instrumentation and supplies. Before utilization in combat or disaster roles, studies were conducted to evaluate the team’s overall capability, ability to function in a light-limited environment, and a new unstressed disinfection technique. All team surgeons regularly practice “damage control” techniques at a military ACS Level One trauma center.

METHODS: Ten anesthetized multiply-injured swine underwent sequential surgical procedures in a field setting. Surgeons evaluated their ability to adequately perform necessary damage control using only MFST equipment. Aerobic and anaerobic cultures of surgical instruments were obtained following disinfection between cases. Thereafter, in the laboratory, instruments underwent ten serial stool contamination/disinfection cycles, with subsequent cultures. To assess low-light capabilities, damage control procedures were performed on nine anesthetized multiply-injured swine in a darkened environment. Surgeons wearing various models of night vision goggles subjectively assessed their ability to perform the necessary procedures. Operations were timed and compared to procedures in the lighted field environment. In a subsequent evaluation, 25 cm of porcine mid-jejunum was rendered ischemic by mesenteric artery ligation. Surgeons assessed bowel viability by gross visualization, pulse palpation, doppler, intravenous fluorescein with Wood’s lamp illumination, night vision goggles with infrared illumination, and thermal imaging technology. The limits of bowel ischemia were identified for each technique. Bowel segments were then sent for pathologic evaluation/confirmaion.

RESULTS: Surgeons successfully completed necessary damage control procedures on swine in a field setting. Equipment and resuscitative fluid deficiencies were identified. Hypothermia presumably resulted in the early death of one pig. Instrument cultures obtained in the field experiment revealed no growth, while those obtained in the laboratory experiment revealed minimal growth. Utilizing the most advanced night vision goggles, surgeons successfully completed damage control procedures with operative times comparable to those performed in previous field lighting conditions. Earlier generation night vision goggles were inadequate for damage control surgery. Night vision goggles with infrared illumination were ineffective for evaluating bowel ischemia, however, thermal imaging was comparable to traditional techniques.

CONCLUSIONS: Thorough evaluation of revolutionary field surgical concepts have confirmed their feasibility and have suggested additional refinements. Additional testing of MFST equipment and concepts is ongoing.
A CHANGING PERSPECTIVE OF ODONTOID FRACTURES: IMPLICATIONS FOR EVALUATION AND MANAGEMENT
W. Iannace, MD; J. Naranga, MD; W. DeLong, Jr., MD; C. Born, MD; R. Dalsey, MD; L. Deutsch, MD; J. Catalano, MD
Cooper Hospital/University Medical Center

William M. Iannace, M. D., Ph. D.

William M. Iannace, M. D., Ph. D.

Camden, NJ 08103

Introduction: Odontoid fractures have been classified by the Anderson and D’Alonzo method since 1974: Type I involve the tip of the dens; Type II occur at the base, but not into the body; Type III extend into the vertebral body. Management and prognosis have been based on classification of odontoid fractures from plain radiographs. The use of CT scans and tomograms has essentially been ignored in evaluating acute injuries. The purpose of this study was to classify and evaluate odontoid fractures using plain radiographs in conjunction with CT scans and/or tomograms.

Methods: 57 consecutive acute odontoid fractures, occurring between April 1987 and November 1995, were retrospectively evaluated. An analysis of plain radiographs, CT scans and/or tomograms was completed, along with an evaluation of demographics, mechanism of injury, associated injuries, neurologic sequelae, mortality, treatment outcomes and rate of nonunion.

Results: After classifying the fractures, we found a distinctly different incidence of the different subtypes. There were no Type I, 16 (28%) Type II, and 41 (72%) Type III fractures. Several fractures, initially felt to be Type II with a plain radiograph, were re-classified as Type III after review of the CT scan. The primary mechanism of injury (motor vehicle accident) was 73% and multiple injuries were present in 51% of the patients evaluated. Neurologic sequelae were present in 7% and 11 patients died (19%). Halo immobilization was used in 44 cases (77%), Minerva jacket in 3 (5%), Philadelphia collar in 3 (5%), and a SOMI brace in 3 (5%). Four patients underwent posterior spinal fusion (7%). The distribution of treatment outcomes, excluding those who expired, was: 44 (96%) healed within 13 weeks; there was 1 (2%) asymptomatic nonunion; and 1 patient was lost to follow-up. There was no significant difference in age between those who healed (average age – 45) and those with a nonunion (average age – 75). There was a significant difference between those who expired (average age-69) and those who healed (p<0.05).

Discussion and Conclusion: With the use of CT scans and tomograms, a much higher incidence of Type III odontoid fractures was identified. This implies a better prognosis for patients with these fractures. The use of halo immobilization should result in high union rates for those who survive this injury. We were unable to correlate the traditional negative prognostic determinants of healing, such as age, probably because of the limited number of non-unions in this series of patients.
National guidelines recommend that patients with Glasgow Coma Scale (GCS) lower than 14 be taken to trauma centers. Such scaling systems may be too complex for pre-hospital applications. We hypothesised that the motor component (GCSM) of the GCS would as effective as total GCS for head injury triage.

Retrospective review of our two year experience revealed that only 1410 of 3235 adults (44%) brought directly to the center alive, had field GCS and GCSM recorded. 265 had GCS<14, while 252 had GCSM<6. Triage of 129/209 (62%) patients with major AIS-4 or AIS-5 head injury was accomplished by GCS; Similar triage of 127/209 (61%) would have occurred by GCSM [p=ns; chi squared]. GCS identified 92%(56/61) of those with AIS-5 injuries, while GCSM found 90%(55/61) [p=ns]. 24 patients required emergent craniotomy: 17(71%) had GCS<14; 18(75%) had GCSM<6 [p=ns]. None of the 15 with GCS<14 and GCSM=6 required craniotomy.

We conclude that pre-hospital GCSM is equal to the pre-hospital GCS in sensitivity for major head injury. In view of the simplicity of a one dimensional score, we recommend that GCS motor component replace the total GCS for triage.
EVALUATION OF ALPINE SKIING AND SNOWBOARDING INJURY IN A NORTHEASTERN STATE
D.E. Sacco, K. Sartorelli, M.D., D.W. Vane, M.D.
University of Vermont College of Medicine
Diane E. Sacco
Dennis W. Vane, M.D.
Burlington, Vermont

Purpose: To demonstrate the injury patterns of Alpine Skiing and Snowboarding in a northeastern state and evaluate potential risk factors.

Materials and Methods: The medical records of a single pediatric and adult level one trauma center were evaluated from January 1, 1990 through December 31, 1995. All admissions with injuries caused by Alpine Skiing or Snowboarding were reviewed. Those patients arriving from 2 local ski resorts, all of whose injuries are referred to the institution for care were separated out for consideration. Age, sex, type of injury, date of injury, ISS, operations performed and outcome were evaluated. In addition, number of skiers and snowboarders per week was also recorded.

Results: For the six year period of the study approximately 2,978,000 skier and snowboarder days were recorded at the study sites. Approximately 447,000 of those were attributed to snowboarders (15%). In all, 279 patients were admitted for injuries (0.01%), 238 related to skiing (incidence 0.01%) and 41 for snowboarding (incidence 0.01%). Snowboarders were statistically younger (20 years range 4-44) than skiers (29 years range 6-70) (p<.05), and had a significantly lower ISS (15 snowboarders vs 27 skiers, p<.03). One female was injured snowboarding and 68 females were injured skiing. 7% of injured snowboarders and 16% of injured skiers sustained multiple injuries (p<.01). Injury patterns were significantly different. Upper extremity injuries were almost exclusively found in snowboarders *24% vs 7%, and cruciate ligament injuries far more commonly in skiers (*45% vs 4%). Lower extremity injuries were far more common in skiers in general (78%) than snowboarders (17%)*. CNS injuries including head and spine injuries were evenly distributed, although the snowboarders with CNS injuries were significantly younger. In addition, splenic injuries were more common in snowboarders (12% vs 2%*) than skiers. Snowboarding accidents were far more common in December, March and April than other months. 51 patients sustained abdominal or chest trauma necessitating admission. None of these patients required surgical intervention for these injuries. All operative interventions were limited to extremity injuries, injuries of the spine, or placement of an intracranial pressure monitor. (*=p<.05) There were no fatalities from injury recorded during this period in this population, however two patients in each group sustained injuries which left them with permanent significant handicaps. No head injuries occurred in helmeted skiers; spine injuries were only recorded in very young snowboarders and skiers out of control.

Conclusions: Snowboarders and Alpine Skiers are equally prone to injury. Snowboarding accidents are typically less severe and show significantly different injury patterns than skiing accidents. Abdominal and chest injuries in this population generally appear amenable to non-operative management. Injuries in snowboarders occur more frequently when snow conditions are marginal, and skiers are less likely to be on the slopes. Prevention programs are best targeted at safe skiing practices; not skiing or snowboarding in poor conditions, use of helmets for skiers, and restraint of snowboard use in very young children.
Can Surgeons Evaluate Emergency Ultrasound Scans For Blunt Abdominal Trauma?

M.G. McKenney, M.D. L. Fernandez, M.D. D Shatz, M.D. N. Namias M.D. D. Levi, M.D. A. Arrillaga, M.D. L. Martin, M.D. J. Moylan M.D.

Objective: To determine if surgeons and surgical residents with minimal training can accurately evaluate emergency ultrasound examinations (US) for blunt abdominal trauma (BAT).

Methods: Over 7 months we conducted a prospective study comparing the evaluation of emergency US for BAT by surgeons and attending radiologists. The surgical team evaluated the ultrasound from the real time images and recorded the results prior to the radiologist. US readings from the surgical team and the radiologist were correlated with patient outcome. The surgical team consisted of 2 attending surgeons, 2 trauma fellows and 2 surgery residents. Minimal training in US was provided to the surgical team. All surgical interpretations of US were completed from the real time images before a radiologist was present.

Results: One hundred-twelve patients were evaluated with US by the surgical team and radiology service. Ninety-two patients had an US read as negative by the surgical and radiology services with no subsequent injuries identified. Eighteen patients had an US deemed positive by the surgical and radiology services. Injuries were confirmed in this group by operation or computed tomography. One patient had an US deemed positive by the surgical team and subsequently negative by the radiologist. A subsequent DPL was performed which was negative. Another patient had an US interpreted as negative by the surgical evaluator and positive by the radiologist. Exploratory laparotomy was negative for intraabdominal hemorrhage or organ injury. Overall results reveal an accuracy of US reading of 99% for the surgical team and 99% for the radiology attending.

Conclusion: Surgeons and residents at different levels of training can accurately interpret emergency ultrasound examinations from the real time images, saving critical time in the work up of blunt abdominal trauma. Minimal training is required to accurately evaluate ultrasound scans. Surgeons can utilize ultrasound even if not supported by the radiology service.
Objective: Rates of operative splenic salvage (OSS) greater than 50% are reported. We sought to measure the rate and circumstances of OSS in a large population of bluntly injured patients to determine if contemporary management reflected these reports. Method: All patients age > 12 yr with blunt abdominal trauma and splenic injury (AIS ≥ 2) admitted to one of 25 Pennsylvania trauma centers over a 3.5 year period were included. Data was extracted from the state trauma registry. Injury grades I and II (AIS = 2) were combined. Results: From 56,226 pts there was 1540 instances of splenic injury. The mode of management, by injury grade, is displayed in Figure 1. There was 813 non-operative (NOP), 135 OSS and 592 splenectomy (S). The grade of injury did not impact on the rate of OSS. NOP and OSS had similar physiology except for SBP at presentation, while the S pts were significantly different. The splenectomy rate doubled when another abdominal injury was present, (23.8% Vs 48.3%) but at laparotomy an isolated splenic injury was 50% more likely to be conserved (14% Vs 21%). There was 5 OSS that became S (3.7%) at separate operations. The average trauma center experience with OSS is 1.5 cases per year. Conclusion: OSS is uncommon in the operative management of blunt splenic injury across all grades of injury. Similarities between NOP and OSS groups suggest OSS will become even less frequent.
RANDOMIZED MULTICENTER PROSPECTIVE
TRACHEOSTOMY STUDY
HJ Sugerman, MD Pasquale, FR Rogers, KF O'Malley
Western Trauma Association Multicenter Study Group
Medical College of Virginia, Virginia Commonwealth
University, Richmond, Virginia
Harvey J. Sugerman, M.D.
Harvey J. Sugerman, M.D.
Richmond, Virginia

5 institutions participated in this multicenter, prospective trial of early (days 3 to 5) or late randomization (days 10 to 14) to tracheostomy (T) in injured or critically ill patients. The primary goals were: pneumonia frequency, duration of ICU stay and death; secondary goals were frequency of pharyngeal, vocal cord or laryngeal injury at T or extubation and long-term injury. **Methods:** Data obtained included Apache I Score (A), Glasgow Coma Score (GCS), Emergency Room Trauma Score (ERTS), Injury Severity Score (ISS), Acute Injury Score (AIS). Patients were to undergo laryngoscopy at extubation and 3-5 mos after discharge. **Results:** 155 patients were randomized; however, only 101 patients had completed data forms for the primary study goals: 58 head trauma (HT) patients (28 T+, 30 T-), 31 non-head injury trauma (NHT) patients (12 T+, 19 T-), and 12 non-trauma (NT) patients (5 T+, 7 T-). The A was higher (p < 0.05) in the HT T+ (64 ± 4) than T- (50 ± 4) and NT T+ (93 ± 6) than T- (68 ± 7) but equivalent in NHT. GCS, ERTS, ISS, AIS were not significantly different in any of the groups. There were no significant differences in ICU stay, frequency of pneumonia or death.

<table>
<thead>
<tr>
<th></th>
<th>HT</th>
<th>NHT</th>
<th>NT</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>ICU Stay</td>
<td>T-</td>
<td>T+</td>
<td>T-</td>
<td>T+</td>
</tr>
<tr>
<td>Pneumonia</td>
<td>17/28</td>
<td>16/30</td>
<td>31/12</td>
<td>26/18</td>
</tr>
<tr>
<td>Death</td>
<td>0/28</td>
<td>3/30</td>
<td>7/19</td>
<td>6/12</td>
</tr>
</tbody>
</table>

Only 40 patients underwent early post-extubation laryngoscopy. The only significant laryngoscopic findings were a higher (p < 0.05) frequency of vocal cord (17/24 vs 5/14) and laryngeal (10/20 vs 3/14) inflammation in the T- patients. The frequency of vocal cord and laryngeal ulcers were insignificantly higher in the T- vs T+ groups. No patient was seen in this study with late vocal cord or laryngeal stenosis; there were no tracheal-inominate artery fistulae. Six patients had normal 3-5 mos post-extubation laryngoscopy. **Conclusions:** Physician bias limited patient entry into the study. Although there were higher Apache scores in the HT and NHT T+ patients, there were no significant differences in the primary endpoints of ICU stay, pneumonia or death in any of the groups studied. Although long-term follow-up was poor, no late tracheal stenoses were seen.
BYLAWS OF
WESTERN TRAUMA ASSOCIATION

ARTICLE I

Name, Objectives, Organization, and Jurisdiction

SECTION 1: Name
The name of this organization is the Western Trauma Association.

SECTION 2: Objectives
The objectives of the Association are to promote the exchange of educational and scientific information and principles, at the highest level, in the diagnosis and management of traumatic conditions and to advance the science and art of medicine.

SECTION 3: Organization
This is a non-profit membership corporate entity, duly incorporated on this 25th day of January, 1971 under, and by virtue of, the provisions of the laws of the State of Colorado.

SECTION 4: Territory
The territory in which this Association shall act will be the United States of America. It shall not be constrained, however, from holding its annual meetings at any designated site throughout the "free world".

SECTION 5: Governing Board
The affairs of the Association shall be conducted by the Board of Directors.
ARTICLE II

Membership

SECTION 1: Membership Limitation
Membership shall be limited to 100 members. No single specialty shall comprise more than 40% of the total membership of 100.

SECTION 2: Qualifications
Active members shall be limited to Doctors of Medicine who are Board Certified in their particular medical specialty. The Board of Directors is hereby given discretionary powers to interpret if foreign physicians who apply for membership have the credentials comparable to Board Certification. Certified members of other (non-M.D.) health care disciplines with a special interest or expertise in trauma may be elected to associate membership with the approval of the Board of Directors and the membership. Associate members shall have all the rights and privileges and must satisfy the same requirements for election to and retention of membership as active members except the right to vote or hold office. For applications to be considered, candidates must submit a completed application with a letter of support (sponsorship) from a member of the Association, submit an abstract for consideration by the Program Chairman, and attend the entire meeting during which their application is being considered. A new member must attend a prior meeting in which he/she is voted on for membership in the capacity of a resident, physician or certified specialist.

SECTION 3: Membership Retention
To retain membership in the Association, each member must comply with the following:

(a) Be a physician in good standing before his or her professional specialty board.
(b) Attend at least one out of every three consecutive meetings of the Association.
(c) Tender to the Program Chairman for consideration an abstract relating to the diagnosis or management of traumatic conditions within the particular medical specialty of the member at one out of every three consecutive meetings of the Association. An invited active panelist can fulfill this requirement.
(d) Agree to be responsible for annual membership dues and any assessments as set by the Board of Directors at a special or the annual meeting and to remain current in the payment of same.

At age 55, members in good standing will automatically accept the position of senior membership in the Western Trauma Association. A senior member must pay dues annually and retains all voting privileges and rights of active members, but does not have to attend the meetings, or submit an abstract once every three years, and his membership is not counted as part of a given specialty’s membership quota or the total membership number.

SECTION 4: Board Action Concerning Membership
Applicants to the Association can obtain membership on a two-thirds vote of the Board of Directors.

Termination of membership can only be obtained on a two-thirds vote of the Board of Directors for a violation of one or more of the items set forth in Article II, Section 3 of the By-Laws of this association.
ARTICLE III

Meetings

SECTION 1: Annual Meetings
There shall be an annual meeting of the membership of the Association held in some suitable location chosen by the Board of Directors. Funds shall be made available for the conduct of the scientific program at the annual meeting (the exact amount of the funds shall be set by the Board of Directors).

SECTION 2: Special Meetings
Special meetings of the Association may be called by the Board of Directors or two-thirds of the membership in good standing, entitled to vote. The location for a special meeting of the Association shall be chosen by the Board of Directors.

SECTION 3: Notice
Notice of the time and place of the annual or special meetings of the Association shall be mailed by the secretary of the Association to each and every member at his address as it last appears on the records of the Association with postage thereon prepaid. Notice shall be deemed delivered when deposited in the United States Mail, so addressed to the respective member.

SECTION 4: Quorum
One-fourth of the membership present at any meeting of the Association shall constitute a quorum.

ARTICLE IV

Meetings of the Directors

SECTION 1: Annual Meetings
The annual meetings of the Board of Directors shall be held on the same day or days and at the same place as the annual meeting of the Association.

SECTION 2: Special Meetings
Special meetings of the Board of Directors may be held at any time and place upon the call of the president, or a majority of the Board providing ten days prior written notice shall be given to each director, stating the time, place and purpose of the special meeting. Notices of special meetings shall be mailed to the directors by the secretary of the Association in the same form and manner as provided above for mailing notices of meetings for the general membership of the Association.

SECTION 3: Quorum
A majority of the Board of Directors shall constitute a quorum.
ARTICLE V

Registration, Fees, Dues, and Assessments

SECTION 1: Registration Fees
Registration fees for annual meetings shall be paid and used to defray the cost of the functions of the annual meeting. The amount of the registration fee shall be determined by the treasurer and president and notice thereof shall be sent to the membership along with the written notice of the annual meeting.

SECTION 2: Dues
Dues of the Association shall be set by the Board of Directors. Each member shall pay dues to the treasurer of the Association prior to the annual meeting. Failure to pay dues shall be considered cause for termination of membership.

SECTION 3: Assessments
A two-thirds majority vote of the Board of Directors of the Association can institute a special assessment of the general membership. Special assessments can be voted by the Board of Directors only for the promotion of scientific programs at the annual meetings, research papers or other purposes designed to achieve the exchange of ideas and principles pertaining to the diagnosis and management of traumatic injuries and conditions. Notice of any special assessment of the membership so voted by the Board of Directors shall be sent to respective members at their last address on record with the Association, postage pre-paid.

SECTION 4: Waiver of Dues
All requirements for retention of membership including payment of dues, submission of abstract, and attendance at meetings may be waived by the Board of Directors upon petition. Eligibility for such waivers shall include inductions into the Armed Forces of the United States on a temporary basis, physical disability, or other reasons which would place unreasonable hardship, physical disability, or other reason upon the petitioner.
ARTICLE VI

Voting

SECTION 1: Voting Rights
Each active or senior member in good standing shall be entitled to one vote on each matter submitted to a vote of the membership.

SECTION 2: Majority
A majority of the votes entitled to be cast on a matter at a meeting at which a quorum is present shall be deemed necessary for the adoption of such matters unless otherwise noted in the Bylaws.

SECTION 3: Manner of Voting
Each member of the Association is entitled to vote in one of three following manners:

(1) In person.
(2) By United States Mail, postage pre-paid, addressed to the secretary of the Association at the Association’s registered office, postmarked on or before the date of the meeting of the membership where the vote is to be taken.
(3) By proxy duly executed in writing by the member or his authorized attorney-in-fact. No voting member in attendance at a meeting shall hold or vote more than one duly executed proxy for absent members.

SECTION 4: Cumulative Voting
Cumulative voting shall not be allowed.

SECTION 5: Amendments
As to the Articles of Incorporation, consolidation or dissolution of the Association shall be passed only in the event of a two-thirds vote of the members in good standing.

SECTION 6: Elections
Elections and all other matters raised to a vote of the membership cannot be held unless a quorum is present and shall be by majority vote.

ARTICLE VII

Officers

SECTION 1: Officers
The officers of the corporation shall consist of the President, President-Elect, Vice-President, Secretary, Treasurer, Historian, and such other officers as from time to time may be appointed by the Board of Directors. The President, President-Elect, Vice-President, Secretary, Historian, and Treasurer shall be elected at the annual meeting of the members.

SECTION 2: Terms and Vacancies
The Secretary, Historian, and Treasurer shall each hold office for the term of three years. The remaining officers shall be elected at the annual meeting of the members. In the event that an officer cannot fill his term, his successor shall be chosen by the Board of Directors to fill the vacancy for the unexpired term of the office.

SECTION 3: Removal
Any officer may be removed, with or without cause, by a vote of a majority of the members of the Board of Directors present at any meeting for that purpose.

SECTION 4: Resignation
Any officer may resign at any time by giving written notice to the Board of Directors and receiving their approval.
ARTICLE VIII

Duties of Officers

SECTION 1: President
Following his ascension to the chair, the president shall preside at all meetings of the members and shall serve as ex-officio member at all committees. The president shall be Chairman of the Board of Directors and shall serve as the liaison to the American Association for the Surgery of Trauma.

SECTION 2: President-elect
The president-elect shall plan and organize the next annual meeting and assume whatever responsibilities the president shall assign to him.

SECTION 3: Vice President
The vice president shall preside at all business meetings in the absence of the president.

SECTION 4: Secretary
The secretary shall keep the minutes of all meetings of the members and the Board of Directors; shall keep all records and information pertaining to the history of the Association; and be responsible for applications for membership, approvals, and deletions as well as communications to the membership, especially those whose membership is in jeopardy.

SECTION 5: Treasurer
The treasurer shall have the following duties:

1. Shall keep the books of account of the Association and shall cause to be prepared an annual audit for presentation at the annual meeting.
2. Shall have custody of, and be responsible for all funds, securities, and other properties of the Association and shall deposit all such funds in the name of the Association in such banks or other depositories as shall be selected by the Board of Directors.
3. Shall assist the secretary in keeping the roster of the membership which is current and accurate.
4. Shall engage a certified public accountant, approved by the president to audit annually the books of the Association. The accountant’s report shall be reviewed by the auditing committee.

SECTION 6. Historian
The Historian should maintain and safeguard archives of the Association. The Historian shall be an ex-officio member of the Board of Directors. In case of a vacancy by reason of death, resignation, or otherwise, the vacancy may be filled by the Board of Directors until the next annual meeting of the members. The historian shall keep a continuous account of the history of the Association for the use of the membership. This shall include significant information concerning each annual meeting, including the site of the meeting, recipients of honors, invited lecturers, highlights of the scientific program, and important actions arising from the Business Meeting. The historian shall also record significant action of the Board of Directors at its meeting. Each five years the historian shall prepare the history of the Association from the time of the last recorded history to be part of the archives of the Association. Memorabilia of the Association shall be retained by the Historian.
ARTICLE IX
Board of Directors

SECTION 1: Composition
The Board of Directors of the Association shall consist of the following individuals:

(1) The president, president-elect, vice president, secretary, and treasurer, immediate past president, and six members-at-large.
(2) Two members of the Association in good standing shall be elected annually to replace two existing members-at-large of the Board unless the membership should, by majority vote, elect to retain the then existing Board of Directors.
(3) The tenure of elected members of the Board of Directors shall be for no more than three years unless such member shall be elected to a position as an officer in the Association.

SECTION 2: Powers
Subject only to the limitations of the provisions of the Colorado Nonprofit Corporation Act, all corporate powers shall be exercised by or under the authority of, and the affairs and activities of the corporate shall be controlled by, or under the authority of, the Board of Directors.

ARTICLE X
Committees

SECTION 1: Nominating Committee
The Nominating Committee shall be composed of three (3) members of the Association appointed by the President. These individuals should represent General Surgery, Orthopedic Surgery, and another specialty. The Chairman of this Committee shall be the immediate past president. This committee shall submit a slate of nominees for the various offices of the Association to the annual meeting of the members.

SECTION 2: Program Committee
The Program Committee shall consist of a Chairman and a Committee including a General Surgeon, and Orthopedic Surgeon, another specialist, and the Chairman of the Publications Committee (ex-officio), all appointed by the President. The Chairman is appointed for a two-year term. This Committee will be responsible for the organization and conduct of the program at the annual meeting.

SECTION 3: Membership Committee
The Membership Committee shall consist of the Board of Directors. The secretary shall present to the Board of Directors at its annual meeting a list of candidates who have satisfied the requirements for membership. Upon approval of the Board of Directors, this group shall be then presented to the membership for its approval as previously outlined.

SECTION 4: Publications Committee
The Publications Committee will consist of a Chairman and a Committee including a General Surgeon, an Orthopedic Surgeon, a Plastic Surgeon, another specialist, and the Chairman of the Program Committee (ex-officio), all appointed by the President. This committee will be responsible for reviewing all manuscripts submitted in association with presentations at the annual meeting and for choosing those which will be submitted to The Journal of Trauma. The Chairman will serve as the liaison to The Journal of Trauma. Should the Chairman not be an Editorial Consultant to The Journal of Trauma, the Chairman will consult with a member of the Editorial Board of The Journal of Trauma designated by the President.
ARTICLE XI

Conduct and Order of Business

SECTION 1: Business Sessions of the Members
There shall be an annual business meeting of the members during the annual meeting. It shall be preceded by a meeting of the Board of Directors also held during the annual meeting of the Association.

SECTION 2: Order of Business
The President shall set the agenda and where possible should follow Robert’s Rules of Order.

ARTICLE XII

Amendments

These Bylaws may be amended at any annual meeting of the Association provided that a notice stating the purpose of each proposed amendment and the reason therefore, and a copy of the proposed amendment is sent to every member in good standing not less than thirty (30) days prior to the date of the meeting at which the proposed amendment is to be voted upon. It shall require a two-thirds vote of a quorum of the membership present at the meeting to amend a Bylaw.
AMMONS, Mark A.  
(Ice Anne)  
2005 Franklin  
Midtown II, #410  
Denver, CO  80205  
O: 303-832-6165  
H: 303-355-5709  
Gen/Thoracic Surgery

*APRAHAMIAN, Charles  
(Patricia)  
8700 W. Wisconsin Ave.  
Milwaukee, WI  53226  
O: 414-257-5022  
H: 414-781-2209  
Surgery

BARTON, Richard G.  
(Janet)  
50 N. Medical Dr.  
#3B313  
Salt Lake City, UT  84132  
O: 801-581-4314  
H: 801-582-4202  
Surgery

BENJAMIN, James  
(Laurie)  
Univ. of Arizona Health Sciences Ctr.  
1501 N. Campbell Avenue  
Tucson, AZ  85724-5064  
O: 520-626-4024  
H: 520-297-9418  
F: 520-626-2473  
Orthopaedics

BERGSTEIN, Jack M.  
8700 W. Wisconsin Avenue  
Milwaukee, WI  53214  
O: 414-257-5023  
H: 414-962-0949  
Surgery

BINTZ, Marilu  
610 E. Taylor St.  
Prairie du Chien, WI  53821  
O: 608-236-6466  
H: 608-326-4306  
Surgery

BOYD, Allen D., Jr.  
(Claire)  
601 Elmwood Avenue  
Box 665  
Rochester, NY  14642  
O: 716-275-7938  
H: 716-264-9489  
Orthopaedics

BROECKER, Bruce H.  
1901 Century Blvd.  
Atlanta, GA  30345  
O: 404-320-9179  
H: 404-325-2297  
Urology

Burch, Jon. M.  
(Rita)  
Dept. of Surgery  
Denver Gen'l Hosp.  
777 Bannock Street  
Denver, CO  80204-4507  
O: 303-436-6570  
H: 303-989-4035  
Surgery/Vascular

CARTER, Donald R.  
8200 East Belleview  
Suite 400  
Englewood, CO  80111-2807  
O: 303-740-7760  
H: 303-671-0259  
Head & Neck Surgery

*CARVETH, Stephen  
(Kay)  
6200 Old Cheney Rd.  
Lincoln, NE  68516  
O: 402-489-6553  
H: 402-423-1768  
Thoracic/  
Cardiovascular Surgery
<table>
<thead>
<tr>
<th>Name</th>
<th>Institution/Medical Center</th>
<th>Address</th>
<th>City, State</th>
<th>Zip</th>
<th>Phone</th>
<th>Home Phone</th>
<th>Specialty</th>
</tr>
</thead>
<tbody>
<tr>
<td>F.R.C.S</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Maria)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>*CHANG, Frederic C.</td>
<td></td>
<td>35 Via Roma, Wichita, KS</td>
<td>67230</td>
<td></td>
<td>O: 316-263-0296</td>
<td>H: 316-733-0627</td>
<td>General Surgery</td>
</tr>
<tr>
<td>(Jan)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Robin)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COBEAN, Roy A.</td>
<td>Maine Medical Center</td>
<td>Surgical Associates</td>
<td>229 Vaughan Street, Portland, ME</td>
<td>04102</td>
<td>O: 207-774-2381</td>
<td>H: 207-781-4735</td>
<td>Surgery</td>
</tr>
<tr>
<td>(Linda K. Rathburn)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COCANOUR, Christine S.</td>
<td></td>
<td>6431 Fannin, MSB 4.167, Houston, TX</td>
<td>77030</td>
<td></td>
<td>O: 713-792-5407</td>
<td>H: 713-432-0253</td>
<td>Gen/Trauma Surg/Crit Care</td>
</tr>
<tr>
<td>(David McCloskey)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>COGBILL, Thomas H.</td>
<td>Gunderson Clinic, Ltd.</td>
<td>1836 South Avenue, La Crosse, WI</td>
<td>54601</td>
<td></td>
<td>O: 608-782-7300</td>
<td>H: 608-788-7808</td>
<td>Gen/Vascular Surgery</td>
</tr>
<tr>
<td>(Jan)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Kelly)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DAVIS, James W.</td>
<td>Tampa General Hospital</td>
<td>PO Box 1289, Room F220, Tampa, FL</td>
<td>33601</td>
<td></td>
<td>O: 813-251-7968</td>
<td>H:</td>
<td>Gen/Trauma Surgery</td>
</tr>
<tr>
<td>(Amy)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Janet)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>J*EDMONDSON, Robert C.</td>
<td></td>
<td>921 Cleveland Street, Woodland, CA</td>
<td>95695</td>
<td></td>
<td>O: 916-666-1631</td>
<td>H: 916-662-7856</td>
<td>Internal Medicine (Oncology)</td>
</tr>
<tr>
<td>(Ann)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDNEY, James A.</td>
<td>Univ. of Nebraska Medical Center</td>
<td>Dept. of Surgery</td>
<td>600 S. 42nd Street, Box 983280, Omaha, NE</td>
<td>68198-3280</td>
<td>O: 402-559-7272</td>
<td>H: 402-493-0705</td>
<td>General Surgery</td>
</tr>
<tr>
<td>(Debbi)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
ESPOSITO, Thomas J.  
2160 South First Avenue  
Bldg. 54 - Room 260  
Maywood, IL  60153  
O: 708-216-8351  
H: 708-531-1271  
Surgery

ESRIG, Barry C.  
USC Cardiothoracic Surgery  
1510 San Pablo Street  
Suite 415  
Los Angeles, CA  90033  
O: 818-683-9000  
H: 818-355-1893  
F: 213-342-5956  
Thoracic Surgery

FELICIANO, David V.  
Grady Mem. Hospital  
69 Butler Street  
Atlanta, GA  30303  
O: 404-616-5456  
H: 404-875-1648  
Gen/Trauma Surgery

FERRIS, Bruce G.  
(Joan)  
825 N. Hillside  
Wichita, KS  67214  
O: 316-688-7500  
H: 316-733-1241  
Plastic Surgery

FILDES, John  
(Elizabeth)  
Univ of Nevada School of Med  
Department of Surgery  
2040 W. Charleston Blvd  
Suite 601  
Las Vegas, NV  89102  
O:  
H:  
Surgery

*FISCHER, Ronald P.  
(Nancy)  
Chief of Surgery  
LBJ Hospital  
5656 Kelley Street  
Houston, TX  77026  
O: 713-636-5095  
H: 713-827-7925  
Gen/Trauma Surgery

FRAZEE, Richard C.  
(Debbie)  
2401 S. 31st Street  
Desk 4-A  
Temple, TX  76508  
O: 817-724-4976  
H: 817-778-6387  
General Surgery

GALL, Warren  
(Beth)  
100 Langworthy  
Dubuque, IA  52001  
O: 319-589-9551  
H: 319-557-1243  
Thoracic/Cardiovascular Surgery

GENTILELLO, Larry M.  
(Olivia)  
Harborview Medical Center  
325 9th Avenue  
ZA-16  
Seattle, WA  98104  
O: 206-731-3956  
H: 206-641-4191  
General Surgery

GUSSACK, Gerald S.  
(Lynn)  
1365 Clifton Road  
N.E. Room 3303  
Atlanta, GA  30322  
O: 404-778-3976  
H: 404-621-9350  
Otolaryngology

HALL, John R.  
(Mary)  
Cook County Hosp.  
700 S. Wood Street  
B-40  
Chicago, IL  60612  
O: 312-633-8353  
H: 312-348-4722
HARRISON, Paul B.  
(Carolyn)  
3243 Murdock  
Suite 404  
Wichita, KS 67208  
O: 316-685-6222  
H: 316-686-3075  
General Surgery

HAUTY, Michael  
(Rose)  
800 S. W. 13th St.  
Portland, OR 97205  
O: 503-221-0161  
H: 503-294-0754  
Surgery

HEBERT, James C.  
(Mary Ellen)  
Dept. of Surgery  
Fletcher 301  
Medical Ctr. Hosp.  
Burlington, VT 05401  
O: 802-656-5354  
H: 802-985-3937  
Surgery

HELLING, Thomas C.  
(Linda)  
4320 Wornall, #308  
Kansas City, MO 64111  
O: 816-753-7460  
H: 913-649-6164  
General Surgery

HOLEVAR, Michele R.  
Christ Hospital & Medical Center  
Trauma Office 102 T  
4440 West 95th St  
Oak Lawn, IL 60453  
O: 708-364-4255  
H: 312-779-3043  
Emergency Medicine

HOYT, David B.  
(Beth)  
UCSD Medical Ctr.  
200 West Arbor Drive  
San Diego, CA 92103-8896  
O: 619-294-6400  
H: 619-272-5893  
Surgery/Trauma

IANNACONNE, William  
(Jane)  
Three Cooper Plaza  
Suite 411  
Camden, NJ 08103  
O: 609-342-3253  
H: 610-649-8515  
Orthopaedic Surgery

JURKOVICH, G. Jerry  
(Deanne)  
Harborview Medical  
Center  
325 9th Ave., ZA-16  
Seattle, WA 98104  
O: 206-731-5912  
H: 206-232-2153  
Gen/Trauma Surgery

KAPPEL, David A.  
(Charl)  
40 Medical Park  
Suite 200  
Wheeling, WV 26003  
O: 304-242-0590  
H: 304-277-3018  
Plastic Surgery

KARRER, Frederick M.  
(Debra)  
The Children's Hospital  
1950 Ogden, B323  
Denver, CO 80218  
O: 303-861-6571  
H: 303-322-4328  
Pediatric Surgery

KEARNEY, Robert E.  
(Becky)  
Harbourside Medical Tower,  
#730  
4 Columbia Drive  
Tampa, FL 33606  
O: 813-259-0982  
H: 813-281-1250  
General Surgery

KING, Brent R.  
(Rosemary Kozar)  
Dept. of Emergency Medicine  
Med. College of PA  
3300 Henry Avenue  
Philadelphia, PA 19129  
O: 215-427-5006  
H: 215-782-1448  
Pediatrics/Emerg Med
KISSINGER, David P. General Surgery Department
Lackland AFB
1622 Hawkes Ridge
San Antonio, TX 78248-1711
O: (210) 670-5906
H: (210) 493-6980
General Surgery/Trauma

*KLASSEN, Rudolph A. (Frieda)
Mayo Clinic
200 First St., S.W.
Rochester, MN 55905
O: 507-284-3662
H: 207-288-4879
Orthopaedics

KNUDSON, Mary Margaret (Stephen A. Delateur)
SF General Hospital
Ward 3A
1001 Potrero
San Francisco, CA 94110
O: 415-206-8814
H: 415-948-3419
Gen/Trauma Surgery

LANDERCASPER, Jeffrey (Betty)
Gundersen Clinic, Ltd.
1836 South Avenue
La Crosse, WI 54601
O: 608-782-7300
H: 507-895-6222
General Surgery

LANZI, Guy L. (Maureen) (Associate Member)
15 E. Euclid Avenue
Haddonfield, NJ 08033
O: 609-429-1711
H: 609-427-0722
Oral and Maxillofacial Surgery

LATENSER, Barbara A. 1400 Locust Street
Pittsburgh, PA 15219
O: 412-232-5612
H: 412-361-6808

LAU, Jeffrey M. (Diane)
1329 Lusitana St.
Suite 108
Honolulu, HI 96813
O: 808-537-1974
H: 808-595-7039
Thoracic/Cardiovascular Surgery

LEWALLEN, David G. (Marti)
Mayo Clinic
200 First St., S.W.
Rochester, MN 55905
O: 507-284-4896
H: 507-292-4463
Orthopaedics

LINDEN, Christopher H. (Jeanne)
Univ. of Mass. Medical Center
55 Lake Ave. North
Worcester, MA 01655
O: 508-856-4101
H: 508-756-3993
Emergency Medicine

LUCIE, Stephen R. (Sharon)
3804 McGirts Blvd.
Jacksonville, FL 32210
O: 904-346-3465
H: 904-387-3604
Orthopaedics

MACKERSIE, Robert C. (Katherinc)
Ward 3A, Dept. of Surgery
SF GH
1001 Potrero Ave.
San Francisco, CA 94110
O: 415-206-8814
H: 415-731-8466
Gen/Trauma Surgery

MCAULEY, Clyde E. 1044 Belmont Avenue
Youngstown, OH 45501-1790
O: 216-480-3907
H: 412-947-9030

*MCGILL, J. Bishop (Betty)
152 Sanborn Road
Stowe, VT 05401
O: None.
H: 802-253-4081
**MCALLISTER, John W.**
(Edward)

**MCDONALD, Robert O.**
(Netty)

**MCDONALD, Robert A.**
(Donna)

**MCNARY, Donald L.**
(Joan)

**MCGILL, John W.**
(Juliette Fournet)

**MCGUIRE, Arthur M.**
(Peggy)

**MCKINLEY, Richard C.**
(Crystal)

**MEHRHOF, Austin I., Jr.**
(Trudi)

**METHENY, Jeffery**

**METZDOFF, Mark T.**
(Marie-Louise)

**MILLIKAN, J. Scott**
(Ann)

**MOORE, Ernest E.**
(Sarah)

**MOORE, Fred**
(Paula)

**MOORE, John B.**
(Debbie)

**MORRIS, John A., Jr.**
(Julina)

**MUCHA, Peter, Jr.**
(Sonja)

---

Hennepin County Medical Center
701 Park Avenue, S.
Minneapolis, MN 55415

O: 612-337-7393
H: 612-825-4281
Emergency Medicine

4200 E. 9th Avenue
Campus Box C-313
Denver, CO 80262

O: 303-270-7673
H: 303-837-9048
General Surgery

P. O. Box 199
Augusta, MO 63332

O: 314-482-4548
H:
Addiction Medicine

Box 154, MCV Station
Richmond, VA 23298

O: 804-786-9318
H: 804-794-6329
Gen/Plastic Surgery

2020 Sutter Place
Suite 104
Davis, CA 95616

O: 916-750-5900
H: 916-758-0597
Orthopaedics

2226 NW Pettygrove Street
Portland, OR 97210

O: 503-226-6321
H: 503-243-1088
Gen/Trauma Surgery

1138 Moon Valley Road
Billings, MT 59105

O: 406-238-2770
H: 406-256-8434
Thoracic/Cardio-vascular
Surgery

Denver General Hospital
777 Bannock Street
Denver, CO 80204

O: 303-436-6558
H: 303-335-9717
Gen/Trauma Surgery

UT-Houston Medical School
6431 Fannin, Suite 4.286
Houston, TX 77030

O: 713-792-5463
H: 713-346-2376
Trauma/Critical Care

9351 Grant Street
Suite 400
Thornton, CO 80229

O: 303-452-0059
H: 303-467-2321
General Surgery

S-2304 Medical Center, No.
Vanderbilt Univ.
Nashville, TN 37232

O: 615-936-0175
H: 615-292-0483
Gen/Trauma Surgery

RR #9 Bingen Road
Bethlehem, PA 18015

O: 717-272-6621
H:
Gen/Trauma Surgery
NEVIAER, Robert J.  
(Doris)  
2150 Pennsylvania Avenue, NW  
Washington, DC 20037  
O: 202-994-4386  
H: 301-869-1919  
Orthopaedics (Hand)  

OCHSNER, M. Gage  
(Judy)  
Chief, Trauma & Surg. Critical  
Svs.  
P. O. Box 22084  
Savannah, GA 31403-2084  
O: 912-350-7384  
H: N/A  
Gen/Trauma Surgery  

OFFNER, Patrick J.  
(Kelly Greene)  
Gen'l Surgery Svs.  
Madigan Army Med. Center  
Tacoma, WA 98431  
O: 206-968-2364  
H: 206-232-4729  
Surgery  

OLSEN, William R.  
(John)  
3387 Herron Road  
Frankfort, MI 49635  
O: None.  
H: 616-352-4815  
Retired  

O'MALLEY, Keith F.  
Three Cooper Plaza  
Suite 411  
Camden, NJ 08103  
O: 609-342-3013  
H: 609-234-0253  
Gen/Trauma Surgery  

OSBORNE, Robert W., Jr.  
(Martha)  
502 N. Stadium Way  
Tacoma, WA 98405  
O: 206-383-3325  
H: 206-593-4694  
Vascular Surgery  

PACHTER, H. Leon  
(Rena)  
530 First Avenue  
NY/6C  
New York City, NY 10016  
O: 212-340-7302  
H: 212-679-9633  
Gen/Trauma Surgery  

PETERSEN, Scott R.  
(Elizabeth)  
St. Joseph's Hospital & Medical  
Center  
350 W. Thomas Rd.  
Phoenix, AZ 85013  
O: 602-406-3157  
H: 602-992-1648  
Surgery  

PHILLIPS, Thomas F.  
(Bonnie)  
P. O. Box 158  
Eureka, CA 95502  
O: 209-826-1973  
H: 707-822-1648  
Gen/Orthopaedic Surgery  

PICKARD, Laurens  
(Bonnie New, M.D.)  
Seurlock Tower  
6560 Fannin  
Suite #1612  
Houston, TX 77030  
O: 713-797-1211  
H: 713-669-9722  
Gen/Thoracic/Pediatric Surgery  

PIERCE, George  
(Carolyn)  
Univ. Of Kansas Medical Center  
3901 Rainbow Blvd.  
Kansas City, KS 66103  
O: 913-588-6128  
H: 913-268-5631  
Gen/Thoracic/Vascular Surgery  

POLACK, Edward P.  
(Wendy)  
40 Medical Park, Suite 200  
Wheeling, WV 26003  
O: 304-242-0590  
H: 304-233-6132  
Plastic Surgery
<table>
<thead>
<tr>
<th>Name</th>
<th>Address</th>
<th>Phone Numbers</th>
<th>Specialties</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Jeanne)</td>
<td>Littleton, CO 80121</td>
<td></td>
<td></td>
</tr>
<tr>
<td>REED, R. Lawrence, II</td>
<td>Box 3501</td>
<td>O: 919-681-5080, H: 919-489-1413</td>
<td>Gen/Trauma Surgery</td>
</tr>
<tr>
<td>(Geraldine)</td>
<td>Dept. of Surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Duke Univ. Medical Center</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Durham, NC 27710</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Sara)</td>
<td>Charlotte, NC 28207</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROSEMURGY, Alexander</td>
<td>Tampa General Hospital</td>
<td>O: 813-251-7393, H: 813-932-9167</td>
<td>Gen/Trauma Surgery</td>
</tr>
<tr>
<td>(Kathryn)</td>
<td>P.O. Box 1289</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Tampa, FL 33601</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Mary)</td>
<td>Suite 400</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Thornton, CO 80229</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROSS, Steven E.</td>
<td>3 Cooper Plaza</td>
<td>O: 609-342-3014, H: 609-427-4352</td>
<td>Gen/Trauma Surgery</td>
</tr>
<tr>
<td>(Carolyn)</td>
<td>Suite 411</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Camden, NJ 08103</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ROZYCKI, Grace S.</td>
<td>Dept. of Surgery</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Grady Memorial Hospital</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>69 Butler Street</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Atlanta, GA 30303</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RUTHERFORD, Edmund J.</td>
<td>2100 Pierce Avenue</td>
<td>O: 615-936-0175, H: 615-298-5775</td>
<td>General Surgery/ Critical Care</td>
</tr>
<tr>
<td>(Eloise)</td>
<td>243 MCS</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Division of Trauma</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Nashville, TN 37212</td>
<td></td>
<td></td>
</tr>
<tr>
<td>*RUTHERFORD, Robert B.</td>
<td>Box C-312</td>
<td>O: 303-394-8552, H: 303-322-3790</td>
<td>Gen/Thoracic/Vascular Surgery</td>
</tr>
<tr>
<td>(Kay)</td>
<td>4200 E. 9th Avenue</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Denver, CO 80262</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAPPLE, Jeffrey R.</td>
<td>50 N. Medical Drive</td>
<td>O: 801-581-3595, H: 801-582-6603</td>
<td>Gen/Trauma Surgery</td>
</tr>
<tr>
<td>(Susan)</td>
<td>#3B-306</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Salt Lake City, UT 84132</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SCALEA, Tom M.</td>
<td>SUNY HSC at Brooklyn</td>
<td>O: 718-245-4790, H: 718-403-0924</td>
<td>Gen/Trauma Surgery</td>
</tr>
<tr>
<td></td>
<td>450 Clarkson Avenue</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Box 1228</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Brooklyn, NY 11203</td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Gisela)</td>
<td>Ward 3A</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>San Francisco, CA 94110</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
SEIBERT, Charles E.  
(Mary)  
One Cimarron Drive  
Littleton, CO 80121  
O: 303-761-7940  
H: 303-781-7760  
Radiology  

SHACKFORD, Steven R.  
(V: 4)  
301 Fletcher House  
Medical Center Hospital of Vermont  
Burlington, VT 05401  
O: 802-655-5354  
H: 802-983-1145  
Gen/Trauma Surgery  

SHARP, Kenneth W.  
(Filen)  
TVC 3662  
Vanderbilt Univ.  
Nashville, TN 37232-5732  
O: 615-322-0259  
H: 615-377-1978  
Gen/Trauma Surgery  

SHERMAN, Harold  

STOTHERT, Joseph C.  
(Jean)  
Div. Multisystem Trauma  
Mercy Hospital of Pittsburgh  
1400 Locust Street  
Pittsburgh, PA 15219  
O: 412-232-7786  
H: 412-681-7744  
Gen/Trauma Surgery  

STREET, David E.  
(Karen)  
818 N. Emporia, Suite 200  
Wichita, KS 67214  
O: 316-263-0296  
H: 315-682-7012  
Gen Surg  

SUGERMAN, Harvey J.  
(Betsy)  
Box 980519  
Medical College  
Virginia Station  
Richmond, VA 23298-0519  
O: 804-828-9516  
H: 804-741-2764  
Gen Surgery  

JAWES, Roy L.  
(Joyce)  
1828 El Camino Real  
Suite #601  
Burlingame, CA 94010  
O: 415-697-3408  
H: Not Available  
Vascular Surgery  

TEAL, Peter V.  
(Aunie)  
1232 N. 30th Street  
Billings, MT 59101  
O: 406-245-3149  
H: 406-245-0060  
Orthopaedics  

THOMAS, Herbert J., III  
(Klasina)  
799 East Hampden Avenue  
Suite 400  
Englewood, CO 80110  
O: 303-789-2663  
H: 303-696-4586  
Orthopaedics  

TUGGLE, David W.  
(Judy)  
940 N.E. 13th Street  
Room 2B204  
Oklahoma City, OK 73126  
O: 405-271-5922  
H:  
Pediatric Surgery  

VANE, Dennis W.  
(Jerrie)  
Univ. of Vermont  
Given Building  
Room D319  
Dept. of Surgery  
Burlington, VT 05404  
O: 802-656-4274  
H: 802-425-4086  
Pediatric Surgery
VOLZ, Robert G. (Anne)  
P. O. Box 2298  
Breckenridge, CO 80424  
O: 303-453-7780  
H:  
Orthopaedics

WALD, Steven L. (Linda)  
Univ. Health Ctr.  
One South Prospect Street  
Burlington, VT 05401  
O: 802-656-3072  
H: 802-985-2582  
Neurosurgery

WEBSTER, Dwight A. (Constance)  
550 Harrison Court  
Syracuse, NY 13202  
O: 315-742-2015  
H: 315-446-1802  
Orthopaedics

WHITLEY, Ronald  
The Atrium - #1100  
1401 Johnston-Willis Drive  
Richmond, VA 23235  
O: 804-560-5964  
H: 804-598-2195  
General Surgery

WILSON, Robert F. (Jacqueline)  
376 Wattles Road  
Bloomfield Hills, MI 48103  
O: 313-745-3485  
H: 313-644-1091  
Gen/Thoracic/CV Surgery

WITTMANN, Dietmar H (Heidi)  
Medical College of Wisconsin  
Dept. of Surgery  
8700 W. Wisconsin Avenue, DH205  
Milwaukee, WI 53226  
O: 414-454-5837  
H: 414-797-9190  
Surgery and Trauma

WRAY, R. Chris (Rocky)  
601 Elmwood Avenue  
Box 661  
Rochester, NY 14642  
O: 716-275-5818  
H: 716-385-3454  
Plastic Surgery

ZELKO, John R. (Katherine)  
Metropolitan Clinic  
265 N. Broadway St.  
Portland, OR 97227  
O: 503-288-5381  
H: 503-241-9125  
Surgery

ZIETLOW, Scott P. (Jill Swanson)  
Mayo Clinic  
Dept. of Surgery  
200 First St., SW  
Rochester, MN 55905  
O: 507-255-6960  
H: 507-285-0074  

*Denotes senior members  
|Denotes honorary members
WESTERN TRAUMA ASSOCIATION
TWENTY-SEVENTH ANNUAL MEETING
SNOWBIRD, UTAH

CHANGE OF ADDRESS FORM

NAME

SPouse
NAME

MAILING
ADDRESS

CITY STATE ZIP

OFFICE PHONE HOME PHONE

SPECIALTY

Return form to:

James A. Edney, M.D.
University of Nebraska Medical Center
42nd and Dewey
Omaha, NE 68150