APSA ANNUAL MEETING
MAY 20 – 23, 2012

APSA-IPSO SYMPOSIUM: **MAY 19 – 20, 2012**

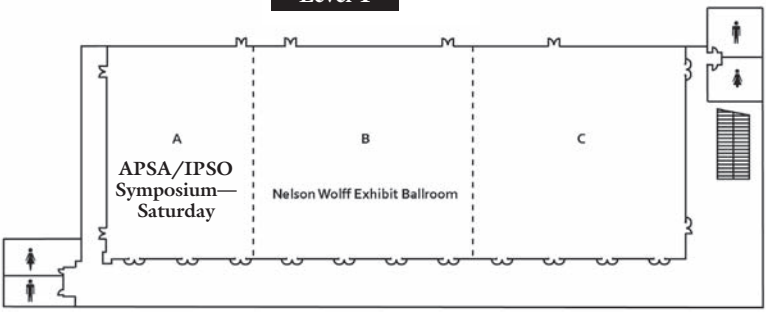
JW MARRIOTT SAN ANTONIO HILL COUNTRY RESORT & SPA
SAN ANTONIO, TEXAS, USA

APSA 43 FORTY-THIRD ANNUAL MEETING

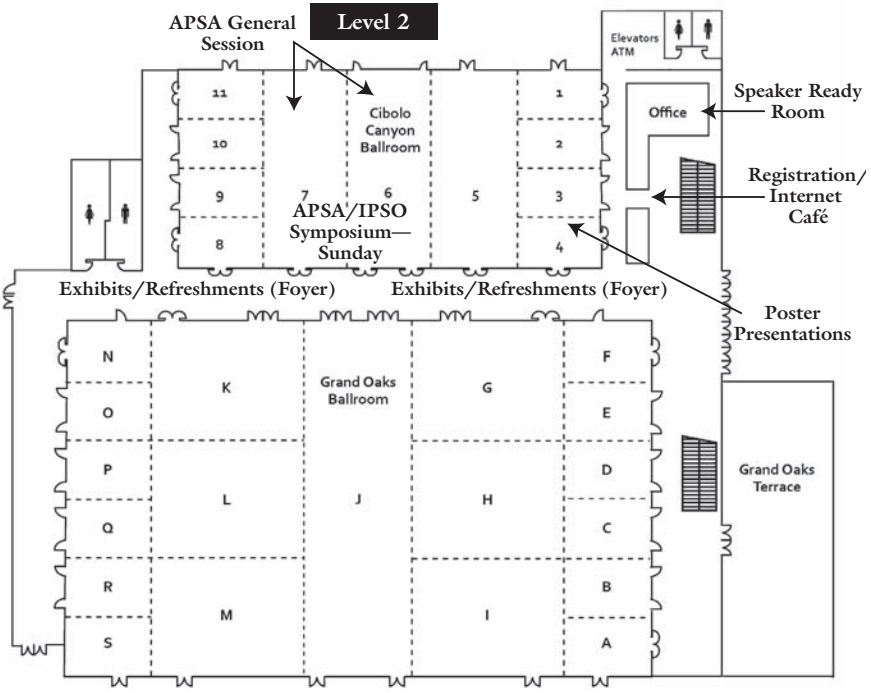
2012 APSA Meeting Program

www.eapsa.org

Level 1



Level 2



Level 3

APSA Committee Meeting Rooms

American Pediatric Surgical Association Mission:

To ensure optimal pediatric surgical care of patients and their families, to promote excellence in the field, and to foster a vibrant and viable community of pediatric surgeons.

We do this by:

- Developing and advocating for standards of care for infants and children and influencing public policy around the surgical care of children
- Encouraging discovery, innovation and improvement of care
- Providing rich venues for the dissemination of up-to-date knowledge
- Offering high quality continuing education to members
- Creating identity and community among pediatric surgeons
- Promoting a supportive health care environment for patients, staff and surgeons and to making certain it is sustained by economic health

American Pediatric Surgical Association
Administrative Offices
111 Deer Lake Road
Suite 100
Deerfield, IL 60015 USA
Telephone: +1-847-480-9576
Fax: +1-847-480-9282
E-mail: eapsa@eapsa.org
website: www.eapsa.org

APSA Headquarters

Lee Ann Clark
Executive Director
lclark@eapsa.org

Marina Petrulla
Administrative Director
mpetrulla@eapsa.org

Liz Freyn
Conference Director
lfreyn@eapsa.org

Christopher Viglione
Conference Registration and Exhibits Manager
cviglione@eapsa.org

Meagan Comerford
Marketing Communications Manager
mcomerford@eapsa.org

Kismet Sağlam, MS Ed.
Education Director
ksaglam@eapsa.org

Liying Zheng
Accountant
lzheng@eapsa.org

Lorna Rubio-Hall
Order Processing Specialist
lrubiohall@eapsa.org

John Waxman
Managing Partner
jwaxman@eapsa.org

Table of Contents

Go to the APSA website at **www.eapsa.org** and:
• Join the discussions on the All-Member Group
• Update your profile
• Connect with APSA colleagues

Tab Page
Remove before Printing

GOVERNANCE

Back of Tab Page
Remove before Printing

Board of Governors 2012–2013

Keith T. Oldham
President 2012-2013
+1-414-266-6557
koldham@mcw.edu

Charles J. Stolar
Treasurer 2011-2014
+1-212-342-8586
cjs3@columbia.edu

Thomas M. Krummel
President-Elect 2012-2013
+1-650-498-4292
tkrummel@stanford.edu

Brad W. Warner
Governor 2010-2013
+1-314-454-6022
brad.warner@wustl.edu

Robert C. Shamberger
Immediate Past President
2012-2013
+1-617-355-8326
robert.shamberger@
childrens.harvard.edu

Kevin P. Lally
Governor 2011-2014
+1-713-500-7300
kevin.p.lally@uth.tmc.edu

Mary L. Brandt
Secretary 2012-2015
+1-832-822-3135
brandt@bcm.edu

Erik D. Skarsgard
Governor 2012-2015
+1-604-875-3744
eskarsgard@cw.bc.ca

APSA Thanks Departing Board Members

Marshall Z. Schwartz
Past President 2011-2012
+1-215-427-5446
mzschwartz@msn.com

Frederick J. Rescorla
Governor 2009-2012
+1-317-274-4681
frescorl@iupui.edu

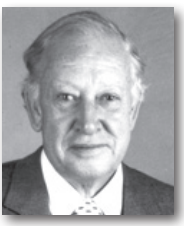
Diana L. Farmer
Secretary 2009-2012
+1-916-734-3190
diana.farmer@ucdmc.ucdavis.edu

APSA Past Presidents

Robert E. Gross
1970-1971

Orvar Swenson
1973-1974

C. Everett Koop
1971-1972



Harvey E. Beardmore
1974-1975

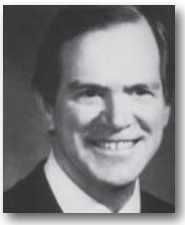
H. William Clatworthy, Jr.
1972-1973

Thomas M. Holder
1975-1976

APSA Past Presidents (cont.)

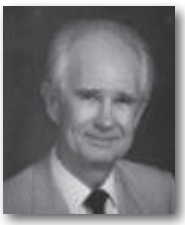
Alexander H. Bill
1976-1977

William B. Kiesewetter
1981



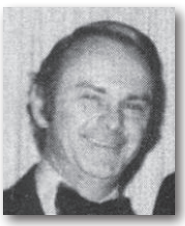
E. Thomas Boles, Jr.
1977-1978

W. Hardy Hendren
1981-1983

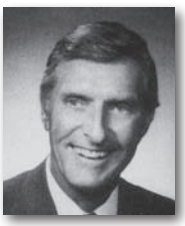


Morton M. Woolley
1978-1979

Lester W. Martin
1983-1984



Robert G. Allen
1979-1980



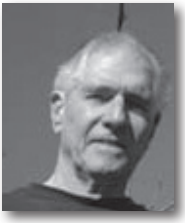
Judson G. Randolph
1984-1985

Thomas V. Santulli
1680-1981

Dale G. Johnson
1985-1986

APSA Past Presidents (cont.)

J. Alex Haller, Jr.
1986-1987



Alfred A. deLorimier
1991-1992

Robert J. Izant, Jr.
1987-1988

Dick G. Ellis
1992-1993

James A. O'Neill, Jr.
1988-1989

Raymond A. Amoury
1993-1994

Eric W. Fonkalsrud
1989-1990

Jay L. Grosfeld
1994-1995

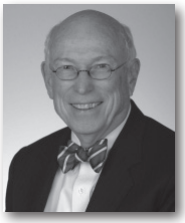
Robert M. Filler
1990-1991

Arvin I. Philippart
1995-1996

APSA Past Presidents (cont.)

Keith W. Ashcraft
1996-1997

Arnold G. Coran
2001-2002



H. Biemann Othersen, Jr.
1997-1998

R. Peter Altman
2002-2003

Marc I. Rowe
1998-1999

Bradley M. Rodgers
2003-2004

Kathryn D. Anderson
1999-2000

Robert J. Touloukian
2004-2005

David Tapper
2000-2001

M. Judah Folkman
2005-2006

Patricia K. Donahoe
2006-2007

Keith E. Georgeson
2009-2010

Moritz M. Ziegler
2007-2008

Marshall Z. Schwartz
2010-2011

Michael R. Harrison
2008-2009

Robert C. Shamberger
2011-2012

Secretary

Thomas M. Holder 1970–1973
Dale G. Johnson 1973–1976
James A. O'Neill, Jr 1976–1979
Robert J. Touloukian 1979–1982
Anthony Shaw 1982–1985
Raymond A. Amoury 1985–1988
Kathryn D. Anderson 1988–1991
Keith W. Ashcraft 1991–1994
Howard C. Filston 1994–1997
Keith T. Oldham 1997–2000
Robert M. Arensman 2000–2003
Donna A. Caniano 2003–2006
Ronald B. Hirschl 2006–2009
Diane L. Farmer 2009–2012

Treasurer

Alfred A. deLorimier 1970–1972
Lucian L. Leape 1972–1975
Robert G. Allen 1975–1978
Dick G. Ellis 1978–1981
J. Alex Haller, Jr 1981–1984
Dick G. Ellis 1984–1987
William P. Tunell 1987–1990
Bradley M. Rodgers 1990–1993
Donald R. Cooney 1993–1996
Robert M. Arensman 1996–1999
Moritz M. Ziegler 1999–2002
Michael D. Klein 2002–2005
Neil J. Sherman 2005–2008
Dennis P. Lund 2008–2011

Governor

Federico A. Arcari 1970–1971
Robert J. Izant 1970–1972
Tague C. Chisholm 1971–1973
Robert G. Allen 1972–1974
Morton M. Woolley 1973–1975
Marc I. Rowe 1974–1976
George W. Holcomb, Jr 1975–1977
Eric W. Fonkalsrud 1976–1978
Dale G. Johnson 1977–1979
Lester W. Martin 1978–1980
Bernard J. Spencer 1979–1981
Harry C. Bishop 1980–1982

Judson G. Randolph 1981–1983
Robert M. Filler. 1981–1984
Keith W. Ashcraft. 1982–1985
Alfred A. deLorimier 1983–1986
Jay L. Grosfeld. 1984–1987
Robert T. Soper. 1985–1988
H. Biemann Othersen, Jr 1986–1989
Robert J. Touloukian. 1987–1990
Arvin I. Philippart 1988–1991
Albert W. Dibbins 1989–1992
Patricia K. Donahoe. 1990–1993
Arnold G. Coran 1991–1994
Moritz M. Ziegler 1992–1995
David Tapper. 1993–1996
Eugene S. Wiener 1994–1997
Samuel H. Kim 1995–1998
R. Peter Altman. 1996–1999
Michael D. Klein 1997–2000
Richard G. Azizkhan 1998–2001
Thomas M. Krummel 1999–2002
Keith E. Georgeson 2000–2003
Marshall Z. Schwartz 2001–2004
John Noseworthy 2002–2005
George W. Holcomb, III 2003–2006
Kurt D. Newman. 2004–2007
Thomas F. Tracy 2005–2008
Robert C. Shamberger. 2006–2009
Mary E. Fallat 2007–2010
Henri R. Ford 2008–2011
Fredrick J. Rescorla 2009–2012

American Board of Surgery

Pediatric Surgery Board
Thomas F. Tracy, Jr., Chair
Mary E. Fallat
Henri R. Ford
Ronald B. Hirschl
David J. Schmeling
Charles J. Stolar
Steven Stylianos

American College of Radiology

Appropriateness Panel on Pediatric Imaging
Charles N. Paidas

American College of Surgeons

Ad Hoc Committee on Membership – Trauma
Max L. Ramenofsky

Ad Hoc Committee on Prevention – Trauma
Barbara A. Barlow
Martin R. Eichelberger

Ad Hoc Legislative Committee on Trauma
James A. O'Neill, Jr.

Advanced Trauma Life Support – Trauma
Max L. Ramenofsky

Advisory Committee on SESAP
Mary E. Fallat

Advisory Council for Pediatric Surgery
Mary E. Fallat
Diana Lee Farmer
Henri R. Ford
Kevin P. Lally
Dennis P. Lund
Marleta Reynolds
Jacqueline M. Saito
Robert C. Shamberger
Thomas F. Tracey, Jr.
Thomas V. Whalen

Advisory Council Program Representatives
Diana L. Farmer

Advisory Council to Board of Regents
Kathryn D. Anderson

AEI Curriculum Committee
Harsh Grewal

AEI Research and Development Committee
Thomas M. Krummel

Board of Governors
Guy F. Brisseau
Mary E. Fallat
Andrea A. Hayes–Jordan
Kevin P. Lally
Don K. Nakayama
Robert C. Shamberger
Joseph J. Tepas, III

Board of Governors Committee on Socioeconomic Issues
Mary E. Fallat
Andrea A. Hayes-Jordan
Robert C. Shamberger
Joseph J. Tepas, III

Board of Regents
Marshall Z. Schwartz
Thomas V. Whalen

Canadian Regents and Governors
Guy F. Brisseau

Coding and Reimbursement Committee
John P. Crow
Samuel D. Smith

Commission on Cancer
Elizabeth A. Beierle
Jed G. Nuchtern

Committee on Allied Health Professionals
Edward M. Barksdale, Jr.

Committee on Ethics
Thomas V. Whalen

Committee on Forum on Fundamental Surgical Problems
Henri R. Ford

Committee on Informatics
Gretchen Purcell Jackson

Committee on Trauma Regional State Committees
Guy F. Brisseau

Committee on Women in Surgery
Stacey D. Moore-Olufemi

Comprehensive Communications Committee
Marshall Z. Schwartz, Chair

COT Regional State Committees
Guy F. Brisseau

Editors: ACS Portal for Pediatric Surgery
Richard G. Azizkhan
Marshall Z. Schwartz

Education Committee
Thomas M. Krummel

Emergency Service – Hospital -Trauma
Martin E. Eichelberger
J. Alex Haller, Jr.

Emerging Surgical Technology and Education Committee
Thomas M. Krummel

Ethics, Committee on
Thomas V. Whalen

Forum on Fundamental Surgical Problems
Henri R. Ford

Governors' Committee on Chapter Relations
Kevin P. Lally

Health Policy Advisory Group
Marshall Z. Schwartz

Information Technology–Trauma
Charles S. Cox, Jr.

Member Services Liaison Committee
Marshall Z. Schwartz

Membership Services Liaison Committee
Marshall Z. Schwartz
Thomas V. Whalen, Chair

Membership – Trauma
David W. Tuggle

National Surgical Quality Improvement Program
Peter W. Dillon, Chair
R. Lawrence Moss
Keith T. Oldham
Charles D. Vinocur
Moritz M. Ziegler

Nominating Committee of the Fellows
Jeffrey S. Upperman

Organization – Board of Regents
Kathryn D. Anderson

Outcomes – Trauma
David E. Wesson

Patient Education Committee
Marshall Z. Schwartz

Performance Improvement –Trauma
Victor F. Garcia

Program Committee
Diana L. Farmer
Henri R. Ford
Gretchen Purcell Jackson

Public Profile and Communications Steering Committee
Kimberly A. Ruscher
Marshall Z. Schwartz

Publications – Trauma
Henri R. Ford
Perry W. Stafford

Quality Assurance – Trauma
Joseph J. Tepas, III

Resident Education
Aaron R. Jensen

Resources – Trauma
Barbara A. Barlow
David P. Mooney
David W. Tuggle

Subcommittee on Resident Education
Diana L. Farmer

Surgical Forum Representative
Henri R. Ford

Surgical Research Committee
Gail E. Besner
Thomas M. Krummel

Trauma Committee
Randall S. Burg
Arthur Cooper
Charles S. Cox, Jr.
Robert T. Maxson
Michael L. Nance
Steven Stylianos

Trauma-Executive Committee
Max L. Ramenofsky
Joseph J. Tepas, III

Trauma Registry
Joseph J. Tepas, III
David E. Wesson

Trauma Systems
Joseph J. Tepas, III

Verification/Consultation – Trauma
David W. Tuggle

Video Based Education Committee
Timothy D. Kane
Todd A. Ponsky

Emergency Medical Services for Children Partnership for Children Stakeholder Group

Arthur Cooper

Relative Value Scale Update Committee (RUC) Board

Samuel D. Smith

Accreditation Council for Graduate Medical Education Surgery Residency Review Committee

George W. Holcomb, III
Marshall Z. Schwartz

Association of Pediatric Surgery Training Program Directors

Max R. Langham, Jr., President
John H.T. Waldhausen, Secretary/ Treasurer

World Federation of Association of Pediatric Surgeons

Richard G. Azizkhan, President
David L. Sigalet, Vice President
George W. Holcomb III, Executive Member
Marshall Z. Schwartz

Audit

Moritz M. Ziegler, Chair, 2010-2013
mmzieglermd@aol.com
Michael D. Klein, 2010-2013
Dennis P. Lund, 2011-2014
Neil J. Sherman, 2010-2013

Cancer

Peter Mattei, Chair, 2011-2013
mattei@email.chop.edu
Andrea A. Hayes-Jordan, Vice Chair, 2011-2013
ahJordan@mdanderson.org
Jennifer Aldrink, 2011-2014
L. Grier Arthur, 2010-2013
Andrew M. Davidoff, 2003-2014
Peter F. Ehrlich, 2010-2013
Gerald Gollin, 2009-2012
Dave R. Lal, 2009-2012
Mary Beth Madonna, 2011-2014
Christopher B. Weldon, 2010-2013
Michael P. LaQuaglia, *Ex Officio* (American Cancer Society)

Childhood Obesity

Thomas H. Inge, Chair, 2010-2012
thomas.inge@cchmc.org
Carroll M. Harmon, Vice Chair, 2010-2012
mac.harmon@chsys.org
Daniel De Ugarte, 2011-2014
Christine M. Finck, 2010-2013
Bradley C. Linden, 2011-2014
Michael A. Helmrath, 2010-2013
Mark J. Holterman, 2008-2014
Marc P. Michalsky, 2010-2013
Mark L. Wulkan, 2011-2014
Jeffrey L. Zitsman, 2008-2013

CME

David M. Powell, Chair, 2010-2013
dmpowell@cnmc.org
George W. Holcomb, III, 2010-2013
Michael B. Ishitani, 2011-2014
Henry E. Rice, 2010-2013
John H. T. Waldhausen, 2010-2013

Education

David M. Powell, Chair, 2010-2012
dmpowell@cnmc.org
Kurt F. Heiss, Vice Chair, 2010-2012
kurt.heiss@choa.org
John J. Aiken, 2009-2012
Marjorie J. Arca, 2011-2014
Clinton Cavett, 2009-2012
Mike K. Chen, 2010-2013
Scott A. Engum, 2008-2014
Kenneth W. Gow, 2011-2014
Harsh Grewal, 2010-2013
Joseph A. Iocono, 2008-2014
Michael B. Ishitani, 2011-2014
Peter C.W. Kim, 2009-2012
Patricia Lange, 2009-2012
Daniel J. Ledbetter, 2009-2012
Craig W. Lillehei, 2009-2012
Gene D. McGahren, 2008-2014
John H.T. Waldhausen, 2009-2012

Ethics and Advocacy

Anthony C. Sandler, Chair, 2011-2013
asandler@cnmc.org
Aviva L. Katz, Vice Chair, 2011-2013
aviva.katz@chp.edu
Gudrun Aspelund, 2009-2012
Elizabeth A. Beierle, 2010-2013
Joy Collins, 2011-2014
Ala S. Frey, 2011-2014
Andrew R. Hong, 2009-2012
Konstantinos Papadakis, 2010-2013
Daniel K. Robie, 2009-2012
Dickens Saint-Vil, 2009-2012
John R. Wesley, 2007-2013

Family and Community Relations

Baird Mallory, Chair, 2011-2013
bairdmallory@yahoo.com
Stephen S. Kim, Vice Chair, 2011-2013
skim@psgkids.com
Lisa Abramson, 2011-2014
Colin Bethel, 2007-2013
James C. Gilbert, 2007-2013
Julie R. Fuchs, 2011-2014
Cynthia Reyes, 2009-2012
Michael Rollins, 2010-2013
Cathy E. Shin, 2007-2013
Roman M. Sydorak, 2011-2014
John R. Wesley, 2008-2014

Fetal Diagnosis and Treatment

Oluyinka Olutoye, Chair, 2011-2014
oolutoye@bcm.tmc.edu
Alan W. Flake, Vice Chair, 2011-2013
flake@email.chop.edu
Terry L. Buchmiller, 2011-2014
Timothy M. Crombleholm, 2009-2012
Brad A. Feltis, 2010-2013
Tracy Grikscheit, 2011-2014
Shinjiro Hirose, 2011-2014
Christopher S. Muratore, 2011-2014
Shaheen J. Timmapuri, 2010-2013
Edmund Y. Yang, 2006-2012

Finance

Charles J. Stolar, Chair, 2011-2014
cjs3@columbia.edu
David J. Schmeling, Vice Chair, 2011-2013
DJS1211@gmail.com
Diana L. Farmer, 2009-2012
George W. Holcomb, III, 2009-2012
Mark J. Holterman, 2010-2013
Frederick J. Rescorla, 2009-2012
Daniel P. Ryan, 2009-2012
Marshall Z. Schwartz, 2010-2013
Robert C. Shamberger, 2010-2013
Eric D. Skarsgard, 2010-2013
Samuel D. Smith, 2010-2013
Thomas F. Tracy, Jr., 2009-2012

Industry Advisory Task Force

Keith E. Georgeson, Chair, 2010-2013
keith.georgeson@providence.org
Carroll M. Harmon, Vice Chair, 2011-2013
mac.harmon@chsys.org
Katherine A. Barsness, 2011-2014
Michael W.L. Gauderer, 2010-2013
Michael R. Harrison, 2010-2013
George W. Holcomb, III, 2010-2013
Thomas M. Krummel, 2010-2013
Michael P. LaQuaglia, 2010-2013
Keith T. Oldham, 2010-2013
Steven S. Rothenberg, 2010-2013
Moritz M. Ziegler, 2010-2013

Informatics and Telemedicine

Jeffrey S. Upperman, Chair, 2008-2012
jupperman@chla.usc.edu
Gretchen Purcell Jackson, Vice Chair, 2009-2012
gretchen.purcell@vanderbilt.edu
Philip L. Glick, 2011-2014
William D. Hardin, Jr., 2009-2012
William P. Ladd, 2010-2013
Samuel M. Mahaffey, 2007-2013
Evan P. Nadler, 2010-2013
Benedict C. Nwomeh, 2010-2013
Oluyinka O. Olutoye, 2010-2013
Todd A. Ponsky, 2010-2013
Mark L. Wulkan, 2011-2014

International Relations

Robert A. Cusick, Chair, 2010-2012
rcusick@chsomaha.org
Jay L. Grosfeld, Vice Chair, 2002-2011
jgrosfel@iupui.edu
A. Alfred Chahine, 2009-2012
Mike K. Chen, 2010-2013
Arnold G. Coran, 2010-2013

Gerald M. Haase, 2010-2013
Sanjay Krishnaswami, 2009-2012
Mark A. Levitt, 2009-2012
Christopher R. Moir, 2009-2012
Steven S. Rothenberg, 2009-2012
David H. Rothstein, 2011-2014
Shawn D. Safford, 2009-2012
Marshall Z. Schwartz, 2010-2013

Membership and Credentials

Stephen E. Dolgin, Chair, 2011-2013
sdolgin@nshs.edu
Michael A. Skinner, 2011-2013
Michael.Skinner@childrens.com
Harry Applebaum, 2010-2013
Robert A. Cowles, 2010-2013
Sherif G.S. Emil, 2009-2012
Harsh Grewal, 2009-2012
Donald C. Liu, 2011-2014
Richard H. Pearl, 2010-2013
Mark L. Silen, 2011-2014
Brian C. Weidner, 2009-2012
Richard G. Weiss, 2009-2012

New Technology

Milissa A. McKee, Chair, 2011-2013
milissa.mckee@yale.edu
Chris K. Breuer, Vice Chair, 2011-2013
christopher.breuer@yale.edu
Sean S. Barnett, 2010-2013
Katherine A. Barsness, 2010-2013
Robert Cina, 2011-2014
Michael J. Goretsky, 2010-2013
Nam X. Nguyen, 2009-2012
Jose M. Prince, 2010-2013
Erik D. Skarsgard, 2009-2012
W. Raleigh Thompson, 2010-2013
Abdalla E. Zarroug, 2011-2014

Nominating

Mary E. Fallat, Chair, 2011-2012
mefall01@louisville.edu
Henri R. Ford, 2011-2012
Peter W. Dillon, 2011-2012
James D. Geiger, 2011-2012
Marleta Reynolds, 2011-2012
Marshall Z. Schwartz, 2011-2012
Keith E. Georgeson, 2011-2012
Michael R. Harrison, 2011-2012

Outcomes and Clinical Trials

Fizan Abdullah, Chair, 2011-2013
fa@jhmi.edu
Saleem Islam, Vice Chair, 2011-2012
islamsa@surgery.ufl.edu
Gudrun Aspelund, 2009-2012
Douglas C. Barnhart, 2010-2013
Martin L. Blakely, 2011-2014
Casey M. Calkins, 2010-2013
Catherine C. Chen, 2009-2012
Cynthia D. Downard, 2010-2013
Adam Goldin, 2010-2013
Eunice Huang, 2009-2012
Shawn J. Rangel, 2010-2013
Shawn St. Peter, 2009-2012
Jacqueline M. Saito, 2011-2014
Laura Cassidy, 2009-2012, *Ex Officio*
Marjorie J. Arca, 2011-2014, *Ex Officio*

Practice

Donald B. Shaul, Chair, 2010-2012
dshaul@chla.usc.edu
J. Duncan Phillips, Vice Chair, 2010-2013
dphillips@wakemed.org
James C. Gilbert, 2010-2013
Philip L. Glick, 2006-2012
Randall M. Holland, 2011-2014
Olajire Idowu, Jr., 2009-2012
Mustafa H. Kabeer, 2009-2012
Kevin P. Lally, 2010-2013
William Middlesworth, 2010-2013
Medo Mirza, 2009-2012
Kevin P. Moriarty, 2011-2014
Don K. Nakayama, 2010-2013
Nam Nguyen, 2011-2014
David M. Notrica, 2008-2014
Ellen M. Reynolds, 2011-2014

Bradley M. Rodgers, 2009-2012
Juan E. Sola, 2009-2012
Dennis W. Vane, 2010-2013

Program

Daniel von Allmen, Chair, 2009-2012
daniel.vonallmen@cchmc.org
Peter F. Ehrlich, Vice Chair, 2011-2013
pehrlich@med.umich.edu
Gail E. Besner, 2009-2012
Terry L. Buchmiller, 2010-2013
J. Ted Gerstle, 2011-2014
Michael A. Helmrath, 2009-2012
Ai-Xuan L. Holterman, 2009-2012
Romeo C. Ignacio, 2011-2014
Wallace W. Neblett, III, 2011-2014
Peter F. Nichol, 2009-2012
Daniel J. Ostlie, 2009-2012
Todd A. Ponsky, 2011-2014
David A. Rodeberg, 2011-2014
David J. Schmeling, 2010-2013
Joel Shilyansky, 2010-2013

PSSAP Sub-Committee

John H.T. Waldhausen, Chair
john.waldhausen@seattlechildrens.org
Marjorie A. Arca
Carroll M. Harmon
Kurt F. Heiss
Craig W. Lillehei
Eugene D. McGahren, III
David M. Powell
Charles L. Snyder

Publications

David L. Sigalet, Chair, 2011-2013
sigalet@ucalgary.ca
Anne C. Fischer, Vice Chair, 2011-2013
anne.fischer@childrens.com
A. Alfred Chahine, 2010-2013
Charles S. Cox, 2011-2014
James C.Y. Dunn, 2009-2012
Henri R. Ford, 2009-2012
John R. Gosche, 2009-2012
Jose L. Iglesias, 2009-2012
Gretchen Purcell Jackson, 2011-2014
Andrea A. Hayes-Jordan, 2011-2014
Eugene S. Kim, 2011-2014
Mark Puder, 2009-2012
Stephen J. Shochat, 2009-2012
Daniel von Allmen, *Ex Officio*

Simulation Sub-Committee

David M. Powell, Chair, 2011-2013
dmpowell@cnmc.org
Scott A. Engum, 2011-2014
Joseph A. Iocono, 2011-2014
Patricia Lange, 2011-2014
Eugene D. McGahren, III, 2011-2014
Andreas H. Meier, 2011-2014

Surgical Quality and Safety

Peter W. Dillon, Chair, 2010-2012
pdillon1@psu.edu
Kurt D. Newman, Vice Chair, 2010-2012
knewman@cnmc.org
David W. Bliss, 2010-2013
Robert Cina, 2011-2014
Gary E. Hartman, 2008-2013
Mary L. Hilfiker, 2010-2013
Saleem Islam, 2010-2013
Brian Kenney, 2009-2012
R. Lawrence Moss, 2007-2013
Don K. Nakayama, 2009-2012
Shawn J. Rangel, 2011-2014
Daniel J. Robertson, 2009-2012
Saad A. Saad, 2010-2013
Joseph J. Tepas, III, 2009-2012
Charles D. Vinocur, 2008-2013

Trauma

Michael L. Nance, Chair, 2011-2013
nance@email.chop.edu
Richard A. Falcone, Jr., 2011-2013
richard.falcone@cchmc.org
Randall S. Burd, 2011-2014
Anthony DeRoss, 2011-2014

Nilda M. Garcia, 2010-2013
Joseph A. Iocono, 2011-2014
Scott E. Langenburg, 2010-2013
Peter T. Masiakos, 2009-2012
William Middlesworth, 2009-2012
David P. Mooney, 2008-2014
Matthew L. Moront, 2010-2013
John K. Petty, 2009-2012
Elizabeth J. Renaud, 2011-2014
F. Dylan Stewart, 2011-2014
Christian J. Streck, Jr., 2011-2014

Workforce

Don K. Nakayama, Chair, 2011-2013
Nakayama.Don@mccg.org
Philip C. Guzzetta, Vice Chair, 2011-2013
pguzzett@cnmc.org
E. Stanton Adkins, 2008-2011
Geoffrey K. Blair, 2008-2011
Brian F. Gilchrist, 2010-2013
George W. Holcomb, III, 2011-2014
Jessica J. Kandel, 2006-2012
Aviva L. Katz, 2011-2014
Dennis P. Lund, 2011-2014
Walt L. Pipkin, 2009-2012
Daniel A. Saltzman, 2010-2013
Marshall Z. Schwartz, 2010-2013
Mark L. Silen, 2009-2012

Alabama

Mike K. Chen, MD
University of Alabama Medical Center
University of Alabama at Birmingham
1600 7th Avenue, South, ACC300
Birmingham, AL 35233
+1-205-939-9751
mike.chen@chsys.org

Arkansas

Samuel D. Smith, MD
University of Arkansas for Medical Sciences
Arkansas Children's Hospital
800 Marshall Street
Little Rock, AR 72202
+1-501-364-2943
smithsamueld@uams.edu

California

Gerald Gollin, MD
Loma Linda University and Children's Hospital
Division of Pediatric Surgery
11175 Campus Street, CP 21111
Loma Linda, CA 92354
+1-909-558-4619
ggollin@llu.edu

Jeffrey S. Upperman, MD
Children's Hospital of Los Angeles
4650 Sunset Boulevard
MS #100
Los Angeles, CA 90027
+1-323-361-7078
jupperman@chla.usc.edu

Craig T. Albanese, MD, MBA
Stanford University Medical Center
Lucile Packard Children's Hospital
780 Welch Road, Suite 206
Stanford, CA 94305-5733
+1-650-724-3664
calbanese@lpch.org

Colorado

Frederick M. Karrer, MD
University of Colorado
Children's Hospital
Department of Pediatric Surgery, B-323
13123 E.16th Avenue
Aurora, CO 80045
+1-303-763-8081
karrer.fritz@tchden.org

Connecticut

Christine M. Finck, MD
University of Connecticut
Connecticut Children's Medical Center
282 Washington Street
Hartford, CT 06106
+1-860-610-4227
cfinck@connecticutchildrens.org

Christopher K. Breuer, MD
Yale-New Haven Medical Center
Yale University School of Medicine
333 Cedar Street
P.O. Box 208062
New Haven, CT 06510
+1-203-785-2701
christopher.breuer@yale.edu

District of Columbia

Timothy D. Kane, MD
George Washington University
Children's National Medical Center
111 Michigan Avenue, N.W.
Washington, DC 20010
+1-202-476-2151
tkane@cnmc.org

Florida

Cathy A. Burnweit, MD
Miami Children's Hospital
3200 SW 60th Court, Suite 201
Miami, FL 33155
+1-305-662-8320
cathy.burnweit@mch.com

Saleem Islam, MD
University of Florida
P.O. Box 100286
Gainesville, FL 32610
+1-352-273-8825
Saleem.islam@surgery.ufl.edu

Georgia

Mark L. Wulkan, MD
Emory University
Egleston Children's Hospital
2040 Ridgewood Drive, N.E.
Atlanta, GA 30322
+1-404-727-3779
mark.wulkan@oz.ped.emory.edu

Illinois

Marleta Reynolds, MD
McGaw Medical Center of
Northwestern University
Children's Memorial Hospital
2300 Children's Plaza, Box 63
Chicago, IL 60614
+1-773-880-4292
mreynolds@childrensmemorial.org

Donald C. Liu, MD
University of Chicago
Comer Children's Hospital
5839 S. Maryland Avenue
MC 4062 (C-411)
Chicago, IL 60637
+1-773-702-6175
dliu@surgery.bsd.uchicago.edu

Indiana

Frederick J. Rescorla, MD
Indiana University School of Medicine
JW Riley Hospital for Children
702 Barnhill Drive, Suite 2500
Indianapolis, IN 46202
+1-317-274-4681
frescorl@iupui.edu

Kentucky

Mary E. Fallat, MD
Louisville-Kosair Children's Hospital
P.O. Box 35070
Louisville, KY 40232-5070
+1-502-629-8638
Mefall01@louisville.edu

Maryland

Paul M. Colombani, MD
Johns Hopkins University Program
Johns Hopkins Hospital
600 N. Wolfe Street, Rm CMSC 7-115
Baltimore, MD 21287
+1-410-955-2717
pc@jhmi.edu

Massachusetts

Craig W. Lillehei, MD
Harvard Medical School
Children's Hospital
Department of Surgery
300 Longwood Avenue
Boston, MA 02115
+1-617-355-3039
craig.lillehei@childrens.harvard.edu

Michigan

Scott E. Langenburg, MD
Wayne State University/Detroit
Medical Center
Children's Hospital of Michigan
3901 Beaubien Boulevard
Detroit, MI 48201
+1-313-745-5881
slangenb@dmc.org

Daniel H. Teitelbaum, MD
University of Michigan
University of Michigan Medical Center
1500 E. Medical Center Drive
F3970 Mott Children's Hospital
Ann Arbor, MI 48109-0245
+1-734-764-6482
dttlbm@umich.edu

Missouri

Daniel J. Ostlie, MD
University of Missouri at Kansas City
Children's Mercy Hospital
2401 Gillham road
Kansas City, MO 64108
+1-816-234-3884
dostlie@cmh.edu

Patrick A. Dillon, MD
Washington University/B-JH/SLCH Consortium
St. Louis Children's Hospital
One Children's Place, Suite 5S60
St. Louis, MO 63110
+1-314-454-6022
dillonp@wustl.edu

Nebraska

Kenneth S. Azarow, MD
University of Nebraska
Children's Hospital and Medical Center
8200 Dodge Street
Omaha, NE 68114
+1-402-955-7400
kazarow@childrensomaha.org

New York

Andrew R. Hong, MD
North Shore-Long Island Jewish Health System
Schneider Children's Hospital
269-01 76th Avenue
New Hyde Park, NY 11040
+1-718-470-3636
hong@lij.edu

Robert Cowles, MD
New York Presbyterian Hospital
Columbia Campus
3959 Broadway
212 North
New York, NY 10032
+1-212-342-8585
rc2114@columbia.edu

Michael G. Caty, MD
SUNY at Buffalo Graduate Medical-Dental Educational Consortium
Children's Hospital of Buffalo
219 Bryant Street
Buffalo, NY 14222
+1-716-878-7785
caty@acsu.buffalo.edu

Ohio

Daniel von Allmen, MD
Cincinnati Children's Hospital Medical Center
3333 Burnet Avenue
Cincinnati, OH 45229
+1-513-636-7365
Daniel.vonAllmen@cchmc.org

Gail E. Besner, MD
Nationwide Children's Hospital
700 Children's Drive
ED-379
Columbus, OH 43205
+1-614-722-3914
gail.besner@nationwidechildrens.org

Oklahoma

David W. Tuggle, MD
University of Oklahoma Health Sciences Center
Children's Hospital at OU Medical Center
P.O. Box 26307
Oklahoma City, OK 73126
+1-405-271-5922
david-tuggle@ouhsc.edu

Oregon

Mark L. Silen, MD
Oregon Health and Sciences University
745 S.W. Gaines Road
CDW-7
Portland, OR 97239
+1-503-494-7764
silen@ohsu.edu

Pennsylvania

Barbara A. Gaines, MD
University of Pittsburgh Medical Center
Children's Hospital of Pittsburgh
Department of Pediatric General & Thoracic Surgery
One Children's Hospital Drive
4401 Penn Avenue
Pittsburgh, PA 15224
+1-412-692-8288
barbara.gaines@chp.edu

Alan W. Flake, MD
Children's Hospital of Philadelphia
34th Street and Civic Center Boulevard
Philadelphia, PA 19104
+1-215-590-2727
flake@email.chop.edu

Rajeev Prasad, MD
St. Christopher's Hospital for Children
Erie Avenue at Front Street
Philadelphia, PA 19134
+1-215-427-4956
rajeev.prasad@tenethealth.com

Rhode Island

Thomas F. Tracy, Jr., MD
Brown University
Brown Medical School
Hasbro Children's Hospital
593 Eddy Street, Room 147
Providence, RI 02903
+1-401-444-7605
Thomas.Tracy@brown.edu

Tennessee

Max R. Langham, Jr., MD
University of Tennessee
Health Science Center
777 Washington Avenue
Suite P-220
Memphis, TN 38105
+1-901-287-6300
mlangham@utmem.edu

Dai H. Chung, MD
Vanderbilt University Medical Center
Monroe Carell Jr. Children's Hospital at Vanderbilt
2200 Children's Way, Suite 7100
Nashville, TN 37232
+1-615-936-1050
dai.chung@vanderbilt.edu

Texas

Kevin P. Lally, MD
University of Texas
6431 Fannin Street
Suite 5.258
Houston, TX 77030
+1-713-500-7300
kevin.p.lally@uth.tmc.edu

Jed G. Nuchtern, MD
Baylor College of Medicine
Texas Children's Hospital
Clinical Care Center, Suite 650
6621 Fannin, MC-CC650
Houston, TX 77030-2399
+1-832-822-3135
nuchtern@bcm.tmc.edu

Michael A. Skinner, MD
University of Texas Southwestern Medical School
Children's Medical Center
D03-310, Y
1935 Motor Street
Dallas, TX 75235
+1-214-456-6040
Michael.Skinner@childrens.com

Washington

John H.T. Waldhausen, MD
Children's Hospital and Regional Medical Center
4800 Sand Point Way, N.E.
Pediatric Surgery, T-0111
Seattle, WA 98105
+1-206-987-1847
john.waldhausen@seattlechildrens.org

Wisconsin

Keith T. Oldham, MD
Children's Hospital of Wisconsin
9000 W. Wisconsin Avenue
Milwaukee, WI 53226
+1-414-266-6557
koldham@mcw.edu

CANADA

Alberta

Andrew L. Wong, MD
University Calgary
Alberta Children's Hospital
1820 Richmond Road, S.W.
Calgary, AB T2T 5C7
+1-403-943-2271
wongal87@yahoo.ca

British Columbia

Erik D. Skarsgard, MD
University of British Columbia
B.C. Children's Hospital
Surgery
4480 Oak Street
Vancouver, BC V6H 3V4
+1-604-875-3744
eskarsgard@cw.bc.ca

Manitoba

B.J. Hancock, MD
Children's Hospital of Winnipeg
AC 401-840 Sherbrook Street
Winnepeg, Manitoba
+1-204-787-1246
bjhancock@exchange.hsc.mb.ca

Nova Scotia

Guy F. Brisseau, MD
Dalhousie University
Department of Surgery
IWK Health Centre
Children's Site
5950-5980 University Avenue
Halifax, NS B3J 3G9
+1-902-470-8114
guy.brisseau@iwk.nshealth

Ontario

J. Ted Gerstle
University of Toronto
The Hospital for Sick Children
Room 1526 Hill Wing
555 University Avenue
Toronto, ON M5G 1X8
+1-416-813-6401
ted.gerstle@sickkids.ca

Shirley Chou, MD
University of Ottawa
General Surgery Program
Children's Hospital of Eastern Ontario
401 Smyth Road
Ottawa, ON K1H 8L1
+1-613-737-7600
pepin@cheo.on.ca

Helene Flageole, MD
McMaster Children's Hospital
1200 Main Street, W.
#4E8
Hamilton, ON L8N 3Z5
+1-905-521-2100 x75244
flageol@mcmaster

Quebec

Pramod S. Puligandla, MD
McGill University
Department of Surgery
Montreal Children's Hospital
2300 Tupper Street
Montreal, QC H3H 1P3
+1-514-412-4438
Pramod.puligandla@muhc.mcgill.ca

Michel Lallier, MD
Universite de Montreal
Departement de Chirurgie
Hopital Sainte-Justine
3175 chemin cote Ste-Catherine
Montreal, QC H3T 1C5
+1-514-345-4931
michel_lallier@ssss.gouv.qc.ca

First: The name of the corporation is The American Pediatric Surgical Association (hereinafter the "Corporation").

Second: The place in this state where the principal office of the Corporation is to be located is in the City of Cleveland, Cuyahoga County, Ohio.

Third: The purposes for which the Corporation is formed are: To encourage specialization in the field of pediatric surgery and in other ways to make available to more people the benefits to be derived from the services of qualified pediatric surgeons; to promote and maintain the quality of education in pediatric surgery through meetings, lectures and the distribution of printed materials; to raise the standards of the specialty by fostering and encouraging research and scientific progress in pediatric surgery, and by establishing standards of excellence in the surgical care of infants and children; to provide a forum for the dissemination of information with regard to pediatric surgery; and to present the common interests of pediatric surgeons in the area of socioeconomic policy development. To accept, receive and acquire by deed, gift, bequest, devise, purchase, lease, or otherwise, property of any sort or nature, without limitation as to amount or value, and to hold, invest, reinvest, manage, use, apply, employ, expand, disburse, or donate the same, whether income or principal or proceeds of sale, exclusively for the purposes hereinabove set forth. To do such other things as are incidental or appropriate in accomplishing the foregoing purposes.

Fourth: The Corporation is organized as a nonprofit corporation under Chapter 1702 of the Ohio Revised Code and shall at all times be operated as a business league within the meaning of Section 501(c)(6) of the Internal Revenue Code of 1986, as amended (the "Code") and, notwithstanding any other provision of these Articles of Incorporation, the Corporation shall not carry on any activities not permitted to be carried on by a corporation exempt from federal income tax under Section 501(a) of the Code by reason of being described in Code Section 501(c)(6).

Fifth: The Corporation shall not make any purchase of property for more than adequate consideration in money or money's worth, shall not sell any of its property for less than an adequate consideration in money or money's worth, and shall not pay compensation in excess of a reasonable allowance for personal services actually rendered. The Corporation shall not lend its property or income, without the receipt of adequate security and a reasonable rate of interest, nor make its services available on a preferential basis. The Corporation shall not engage in any transaction which results in a diversion of its property or income from its purposes as set forth in Article Third. No part of the net earnings of the Corporation shall inure to the benefit of any person except as a proper beneficiary of its said purposes.

Sixth: The Corporation shall not accumulate income to an extent which is unreasonable either in amount or duration in carrying out its purposes set forth in Article Third, shall not use such accumulations for purposes other than such purposes, and shall not invest its funds in any manner as to jeopardize the carrying out of its said purposes.

Seventh: Upon dissolution of the Corporation, or any partial or entire liquidation of its property or assets, all of the Corporation's property of every nature and description shall, after making provision for discharge of all of the liabilities of the Corporation, be paid over and transferred to such one or more organizations or institutions which are then exempt from federal income tax under Section 501(a) of the Code by reason of being described in either Section 501(c)(3) or Section 501(c)(6) of the Code, as shall be selected by a majority of persons who are then members of the Board of Governors of the Corporation.

Eighth: No member of the Board of Governors, officer, or employee of the Corporation, or any other person, shall receive any profit from the operations or liquidation of the Corporation, except as reasonable compensation for services actually rendered to the Corporation.

Ninth: Each reference in these Amended Articles of Incorporation to a section of the Code or the Ohio Revised Code shall include the corresponding provisions of any future Internal Revenue or Ohio laws, respectively.

Tenth: These Amended Articles of Incorporation supersede and take the place of existing Articles of Incorporation of the Corporation as the same may have been amended heretofore.

PREAMBLE

PRINCIPLES OF MEDICAL ETHICS

Members:

1. Shall strive to provide competent medical care to patients with compassion and consideration for their feelings and dignity.
2. Shall strive to maintain existing skills and to develop or acquire new medical and surgical knowledge through continuing practice in order to benefit patients.
3. Shall avoid performing procedures which are beyond their capacity, training or experience.
4. Shall practice medicine with honesty and fairness toward patients, colleagues and all others.
5. Shall seek consultation, assistance or additional talents of other professionals where such might be of value in the care of the patient or where requested by the patient or a concerned representative.
6. Shall choose from equally efficacious treatments and diagnostic procedures those which are the least intrusive, the least painful and the least expensive.
7. Shall recognize a responsibility to participate in activities benefiting the community.

Article I: MEMBERSHIP

SECTION 1. REGULAR MEMBERSHIP

1.1. A regular member must be licensed to practice surgery in the United States or Canada.

1.2. All regular members must be certified by the American Board of Surgery or by the Royal College of Surgeons of Canada. After June 30, 1977, all new members must obtain a Certification of Special Qualifications in Pediatric Surgery by the American Board of Surgery or the Royal College of Surgeons of Canada.

1.3. A regular member must have completed his/her training in an Accreditation Council for Graduate Medical Education-approved training program and must have held the ACGME-approved residency position or equivalent Royal College of Surgeons of Canada approved program.

1.4. An applicant must have a practice devoted entirely to pediatric surgery, except as may be required by emergency care or special circumstance.

1.5. An applicant may not be elected to membership until he or she has practiced pediatric surgery for one year after completion of the required surgical training.

1.6. Any exception to the above criteria for membership must be made by a recommendation from the membership and credentials committee to the board of governors. Subsequent majority approval of the board of governors and an affirmative vote by two-thirds of the voting membership at an annual meeting business meeting is necessary for election.

1.7. The regular member pledges to abide by the obligations and objectives and core values of the association as set forth in the articles of incorporation and the principles of medical ethics as stated in the preamble to the bylaws.

Section 2. Candidate Members

2.1. A candidate member must be currently licensed to practice surgery in the United States or Canada.

2.2. Candidate members must have successfully completed the examination in general surgery given by the American Board of Surgery or by the Royal College of Surgeons of Canada or, they must be eligible for examination by those respective boards.

2.3. Only residents in ACGME-approved pediatric surgical residency programs are eligible for candidate membership.

2.4. An individual may remain a candidate member for five years following completion of an approved pediatric surgical residency program at which time the candidate membership will expire. This five-year period is in addition to the time spent as a candidate member during pediatric surgery residency.
If candidate membership expires, one may still apply for regular membership at any time in the future. Candidate membership is not mandatory in order to qualify for regular membership.

2.5. A candidate member who has completed his/her training in an ACGME-approved pediatric surgery residency position or equivalent Royal College of Surgeons of Canada approved program must practice pediatric surgery exclusively as stipulated in section 1.4. for regular membership.

2.6. Candidate members are not eligible for appointment with voting privileges on standing or ad hoc committees, but may be appointed by the president as consultant members for a period not to exceed two years.

2.7. Candidate members will have the same meeting attendance requirements as regular members, but will not have voting privileges. Candidate members are not eligible to hold office. Candidate members will be subject to 20% of the current regular membership dues and will be governed by all other bylaws applicable to regular membership.

2.8. A candidate member will require sponsorship by a regular member for abstracts submitted for presentation to the annual APSA scientific meeting.

Section 3. Charter Membership

3.1. A charter membership shall be extended to a person actively engaged in the practice of pediatric surgery, who has already amply demonstrated excellence and fitness as a trained specialist in pediatric surgery, who has devoted his practice to pediatric surgery and who is certified by the American Board of Surgery or by the Royal College of Surgeons of Canada.

3.2. A list of charter membership was established and then closed on April 15, 1970.

Section 4. Honorary Membership

4.1. Honorary membership may be conferred upon a physician for outstanding contributions to pediatric surgery by unanimous vote of the board of governors and an affirmative vote by two-thirds of the voting membership attending the annual meeting business meeting.

4.2. Honorary members will be governed by the bylaws as regular members but will not be subject to dues or the meeting attendance requirement and will not be eligible to hold office.

Section 5. International Membership

5.1. A physician who does not live or practice surgery within the Territory of the United States or Canada and who does not otherwise meet criteria for regular membership, may apply to the American Pediatric Surgical Association as an international member. Such applicants must provide documentation that they have successfully completed the established training curriculum in pediatric surgery as required by their respective national or regional agencies. Such applicants must meet the same practice criteria as required of regular members. Letters of recommendation from three APSA members as well as a letter from one local reference must accompany his/her application.

5.2. Applicants for international membership must have attended one annual meeting before they are eligible to apply.

5.3. International members will pay dues and be governed by the bylaws as regular members, but will not be eligible to vote or hold office.

Section 6. Associate Members

6.1. Associate membership shall be extended to a person who has been exclusively engaged in the practice of pediatric surgery for five years, except as may be required by emergency care or special circumstances.

6.2. An associate member requires written endorsement by a regular member sponsor as well as two other members at the time of application.

6.3. Associate member applicants must provide a comprehensive current two-year case log as well as a letter from the chief of surgery at each hospital where he/she practices confirming the validity of the case log and indicating that the applicant is a member of the hospital staff in good standing.

6.4. Associate members shall have all of the rights, privileges and obligations as regular members but may not hold elected office.

6.5. Applications for associate membership will be submitted for consideration to the membership and credentials committee for review and recommendation to the board of governors and membership-at-large. The procedure for election to membership shall be identical as for regular members.

Section 7. Resident Members

7.1. A resident member must be a general surgery resident in good standing in an ACGME-approved residency program or Royal College of Surgeons equivalent.

7.2. Two reference letters are required: One from the general surgery chair or program director and one from an APSA member in good standing.

7.3. The term of membership will be for one year and will automatically expire after one year unless a written request for extension is submitted to and approved by the membership and credentials committee.

7.4. The membership and credentials committee will be solely responsible for all decisions regarding acceptance into the resident group.

Section 8. Application Procedures

8.1. New applications for regular, associate or international membership will be initiated by the prospective member. For regular membership, the procedure may begin prior to the completion of the required one year of pediatric surgery practice. See Article 1, Section 1.5. The application will need supporting letters from three members in good standing. One of these three letters must

be from the training director of the prospective member. At least one sponsor must attest that the applicant exemplifies a high standard of ethical behavior as set forth in the principles of medical ethics in the preamble to the bylaws. Applicants for international membership will require one additional letter of recommendation from a physician who is acquainted with the individual's professional competence and ethics in his/her own practice community.

8.2. Completed applications for membership may be submitted to the membership and credentials committee at any time throughout the year. Applications will be reviewed quarterly by the membership and credentials committee and presented quarterly to the board of governors for approval.

8.3. Upon the recommendation of the membership and credentials committee and approval of the board of governors, the list of applicants shall be circulated to the membership-at-large twice per year for voting. Following the vote of the APSA membership, approved applicants will immediately become members of APSA in their respective categories. Approved applicants will receive their certificates of membership in a ceremony at the subsequent annual meeting.

8.4. All applications for candidate membership will be initiated by the chair of the applicant's pediatric surgery training program, who must also be a regular member in good standing. The sponsoring member will be responsible for completing the candidate member's application form. The completed application will be sent to the chair of the membership and credentials committee. The committee will evaluate the applicant's credentials and make a recommendation concerning membership to the board of governors. Applications for candidate membership will be accepted as outlined in Section 8.2.

8.5. The membership applicant and the sponsor will be notified by mail of the results of the application process.

8.6. The rejection of the membership application by the membership and credentials committee or the board of governors or by the membership of APSA may be appealed within one year of notification of the applicant, if he/she so desires.

8.7. The appeal process is initiated by the membership applicant. He/She can, by written inquiry to the secretary of the board, request an appeal hearing before the board of governors. This hearing will be granted at the time of the next regularly scheduled biannual board of governors meeting, provided the request is received at least three months prior to the next regularly scheduled meeting. This appeals meeting must be attended by the sponsor and a maximum of one other member of the organization. The board of governors may invite other interested parties at their discretion. The membership applicant may attend only upon request of the board of governors.

Section 9. Application Form

9.1. The application shall include:

9.1.1. Curriculum vitae

9.1.2. Bibliography

9.1.3. Applicants for regular or international membership must submit a tabulation, by case, of the operative experience of the applicant during the 12-month period immediately preceding his/her application. Applicants for associate membership must submit a tabulated operative experience covering the 24-month period immediately preceding his/her application. All operative reports must be signed by the chief(s) of surgery where the applicant works.

The report should indicate whether the applicant was surgeon, first or teaching assistant.

9.2. The candidate membership application shall include:

9.2.1. Curriculum vitae

9.2.2. Bibliography

9.2.3. A letter from the chief of the applicant's pediatric surgery training program which attests to his/her satisfactory completion of one or more years of training and suitability for candidate membership. This letter should also confirm that the applicant for candidate membership held the ACGME-approved residency position within the training program (for U.S. trainees or equivalent Royal College of Surgeons of Canada approved program).

Section 10. Resignation

10.1. Any member may submit his/her resignation at any time in writing to the president to be effective on the date of submission.

Section 11. Fiscal Year

11.1. The fiscal year shall be from January 1 to December 31.

Section 12. Dues

12.1. Dues shall be set by the board of governors and approved by the membership at the annual meeting. Dues will be announced by letter by the first day of October and must be paid by the first day of December.

12.2. No annual dues shall be required of a member following his/her 65th birthday or upon retirement from active practice whichever is sooner. (Member will be termed a "senior member.") No annual dues shall be required of any member during any year that person is disabled and unable to practice for six months or more.

12.3. Under special circumstances and by approval of the board of governors, dues may be waived for any member for one calendar year.

12.4. An initiation fee equal to one-half of the annual dues will be levied on all new members at the time of their induction into membership in the organization. This fee must be paid prior to issuing a certificate of membership.

Section 13. Certificate of Membership

13.1. A certificate of membership will be designed and issued to each member, signed by the president and the secretary.

Section 14. Loss of Membership

14.1. A member may be dropped from membership for:

14.1.1. Missing three consecutive meetings without written excuse, submitted to the secretary and considered justifiable by the board of governors. Members over 60 years of age, honorary, international and senior members will be excused from this requirement.

14.1.2. Failure to adhere to the obligations and objectives of the Association set forth in the articles of incorporation and in the bylaws.

14.1.3. Failure to remit dues within six months of the announced date will result in loss of membership in the Association. Members in arrears will receive a

registered letter at least one month prior to the date of loss of membership outlining this action. Reinstatement of membership may be obtained by petitioning the board of governors. Payment of past dues owed as well as a reinstatement fee equal to the initiation fee for the organization will be required to resume membership.

14.2. The board of governors shall act by two-thirds vote to implement Article I, Section 14.1. with due process as specified by Article I, Section 14.3.3. and Article I, Section 14.3.3.7.

14.3. Discipline.

14.3.1. The board of governors may expel, call for the resignation of or otherwise discipline a member if three-quarters of all the members of the board of governors find that the conduct of the member has been injurious to the purposes of the Association as outlined in the bylaws and the preamble entitled principles of medical ethics.

14.3.2. Without limiting the foregoing, the following shall be considered to be conduct or conclusive evidence of conduct injurious to the purposes of the Association:

14.3.2.1. Conviction of a felony or of any crime relating to or arising out of the practice of medicine and involving moral turpitude.

14.3.2.2. Limitation or termination of any right associated with the practice of medicine in any state, province or country.

14.3.2.3. Grossly immoral, dishonorable or unprofessional conduct.

14.3.3. Due process.

14.3.3.1. Questions of discipline shall be investigated by an ad hoc committee, appointed by the president of the APSA.

14.3.3.1.1. The ad hoc committee shall consist of two members-at-large and one member of the board of governors.

14.3.3.1.2. The chair of the ad hoc committee shall be one of the specified members-at-large and shall be designated by the president of APSA.

14.3.3.1.3. The ad hoc committee shall convene for the purpose of investigating the charges within six months of time of its appointment and shall report its recommendation(s) to the board of governors in writing within nine months of the committee's appointment.

14.3.3.1.4. The term of the ad hoc committee includes but does not extend beyond the time of submission of their report.

14.3.3.2. A statement of charges shall be sent by the secretary of APSA for the ad hoc committee. The statement shall be sent to the member's last recorded address, by certified or registered mail, at least thirty days before the designated meeting date for the committee's consideration of the matter.

14.3.3.2.1. The time and place of the meeting shall be indicated.

14.3.3.2.2. The member shall be informed that he/she may appear at the meeting in person and with counsel, if he/she so elects, so as to state his/her response to the charges.

14.3.3.3. The board of governors shall consider the recommendation(s) of the ad hoc committee at its next regular meeting or upon extraordinary session, but no earlier than thirty days from time of the member's notification.

14.3.3.3.1. A statement of the recommendation(s) of the ad hoc committee shall be sent by the secretary to the last recorded address of the member in question, by certified or registered mail, at least thirty days before the date of the meeting when the board of governors shall consider the matter.

14.3.3.3.1.1. The time and place of the meeting shall be indicated.

14.3.3.3.1.2. The member shall be informed that he/she may appear at the meeting in person and with counsel, if he/she so elects, so as to state his/her response to the charges.

14.3.3.4. The board of governors may temporarily suspend any member and defer consideration of disciplinary action during the pending of appeal from a judicial or other governmental decision which forms the basis for disciplinary action as stated in Article I, Section 14.3.2. or during anytime in which he/she is prevented from appearing at a hearing by reasons of health. Upon completion of the exception, the board of governors shall implement Article I, Section 14.3.3.

14.3.3.5. Following consideration by the board of governors, the member shall be informed by the secretary of the result of the deliberations by certified or registered mail to the last recorded address of the member.

14.3.3.6. The result of the deliberations of the board of governors shall be considered final unless the secretary receives in writing within thirty days from the time of issuance of the notification, as stated in Article I, Section 14.3.3.5. a request for appeal to the membership-at-large of the action of the board of governors.

14.3.3.7. Upon request for appeal, the membership shall be presented at the next annual meeting the recommendations of the board of governors. The member may elect, if he/she so desires to personally present his/her argument for the appeal. The membership present shall confirm or refute the recommendation of the board of governors by simple written majority vote. This vote shall be considered binding and final.

14.4. Upon loss of membership, the certificate of membership shall be returned to the secretary.

Article II OFFICERS

Section 1. The Officers

1.1. The officers shall be a president, a president-elect, a secretary and a treasurer.

1.2. The officers shall be elected by written ballot mailed by the nominating committee to the membership three months prior to the annual meeting.

1.3. The nominee for each office obtaining the majority vote by the deadline posted shall be elected.

Section 2. Term of Office

2.1. The terms of each above office shall be:

President	1 year
President-Elect	1 year
Secretary	3 years
Treasurer	3 years

Article III BOARD OF GOVERNORS

Section 1. Membership of the Board of Governors

1.1. The membership of the board of governors shall consist of the president, the president-elect, the secretary, the treasurer, the immediate past president and three elected members-at-large.

1.2. The three at-large members, for the first year of this amendment, shall be elected to serve for one, two and three years respectively. Thereafter, a new member shall be elected for a three-year term each year.

1.3. Election shall be conducted in the same manner as for the officers. See Article II, Sections 1.2. and 1.3.

Section 2. Chair of the Board of Governors

2.1. The president shall be the chair of the board of governors.

Section 3. Functions of the Board of Governors

3.1. It shall generally oversee the activities of the Association and make certain that the spirit and the letter of the articles of incorporation and the bylaws are carried out.

3.2. It shall pass recommendations on candidates for membership to the entire membership.

3.3. It shall approve the meeting place of the annual meeting business meeting at least one year in advance.

3.4. It shall review the report of the membership and credentials committee.

3.5. It shall meet at least once a year or more times, as is appropriate, sufficiently prior (at least four months) to the annual meeting business meeting to allow time for proper action.

3.6. A quorum for official business at a board of governors meeting shall be four.

3.7. Vacancies on the board of governors, other than the presidency, shall be filled by appointment by the president until the next annual meeting business meeting, when a special election will be held.

Article IV DUTIES OF OFFICERS

Section 1. The President

1.1. Shall preside at the annual meeting and at all meetings of the board of governors.

1.2. Shall enforce all rules and regulations of the Association.

1.3. Shall sign all official documents.

1.4. Shall make appropriate committee appointments.

1.5. Shall be an ex-officio member of all committees except the nominating committee.

Section 2. The President-Elect

2.1. Shall preside at the annual meeting in the absence of the president.

2.2. Shall preside at other meetings in the president's absence.

2.3. In the event of the disability or death of the president, shall assume the president's responsibilities.

2.4. Shall become president the next year.

Section 3. The Secretary

3.1. Shall record the proceedings at all meetings.

3.2. Shall notify the membership of all meetings and publish and distribute the agenda of the annual meeting business meeting.

3.3. Shall maintain a registry of membership.

3.4. Shall conduct appropriate correspondence and maintain a file of such.

3.5. Shall submit a report of the minutes of the previous annual business meeting.

3.6. Upon the disability of the president and then the president-elect, shall assume the office of the president automatically—to serve only until the next annual meeting.

Section 4. The Treasurer

4.1. Shall bill to and collect from members all dues and fees pertaining to the Association.
4.2. Shall render disbursements for authorized official expenses.
4.3. Shall maintain a financial ledger.
4.4. Shall maintain records, which shall be available for an annual audit by an appropriate auditing committee of members appointed by the president or by an outside accounting firm.
4.5. Shall present a report to the membership at the business session of the annual meeting.
4.6. Shall maintain at the expense of the Association a surety bond for the treasurer and all others handling Association funds.
4.7. The first treasurer shall be elected for a two-year term.

Article V MEETINGS

Section 1. Annual Meeting

1.1. There shall be an annual meeting, the time and place of which shall be established by the board of governors at least a year in advance.
1.2. There shall be a scientific meeting incorporated into the annual meeting.
1.3. There shall be a business meeting incorporated into the annual meeting, which will be open only to members in good standing and at which official business shall be transacted.
1.4. All meetings shall be guided by the current edition of Robert's Rules of Order.

Section 2. Guests and the Annual Meeting

2.1. The scientific sessions of the annual meeting shall be open to all interested physicians who register for the meeting.
2.2. Interested paramedical professionals may be invited by any member in good standing.
2.3. A registration fee may be required of non-members and guests at the discretion of the program committee.
2.4. The privilege of the floor at the scientific sessions will be restricted to the membership and to others who have been given official designation by letter from the secretary.

Section 3. Quorum

3.1. The members present shall constitute a quorum for business at the annual meeting business meeting and other official committee meetings unless the number is otherwise specifically stated.

Article VI BYLAWS

Section 1. Time of Effect

1.1. The bylaws shall take effect immediately from the time of adoption.

Section 2. Amendments of the Bylaws

2.1. The bylaws may be changed or amended by submitting a written resolution to the board of governors who, in turn, will present the change or amendment to the Membership at least one month prior to the next annual meeting.

2.2. A two-thirds vote of the membership voting at the annual meeting will be necessary for adoption of a change or amendment of the bylaws of the Association.

Article VII PERMANENT COMMITTEES

Section 1. Permanent Committees

1.1. The board of governors shall establish permanent committees to conduct the business and educational affairs of the Association. These permanent committees shall be defined and their duties described in the Association's policies and procedures. Creation, dissolution and modification to the number and duties of the permanent committees shall be by majority vote of the board of governors. Any changes in committees shall be submitted to and ratified by the members of the American Pediatric Surgical Association at the yearly meeting.

Article VIII AD HOC COMMITTEES

Section 1. Membership

1.1. From time to time, the president may establish an ad hoc committee and appoint its membership.

Article IX REPRESENTATION TO OTHER SOCIETIES

The president may appoint liaison representatives to other organizations, societies or associations as seems appropriate.

Article X HISTORIAN

An historian shall be appointed by the president.

Article XI OFFICIAL SEAL

A seal shall be designated and affixed to all official stationery and documents.

Article XII INDEMNIFICATION AND INSURANCE

Section 1. Indemnification

1.1. As provided herein, the Association may, but shall not be required or obligated to, indemnify any governor or officer or any former governor or officer of the Association (and his or her heirs, executors or other personal representatives) against expenses, including attorney's fees, judgments, fines and amounts paid in settlement which are actually and reasonably incurred by such person by reason of the fact that such person is or was a governor or officer in connection with any threatened, pending or completed action, suit or proceedings, whether civil, criminal, administrative or investigative, to the extent and according to the procedures and requirements set forth in the Ohio Non-Profit Corporation law. The decision of whether to indemnify is reserved to the board of governors to be decided by the majority vote of governors who are not involved in or parties to the same or substantially the same claim, action, suit or proceeding. Where a quorum cannot be obtained or the board of governors cannot reach a decision, an independent legal counsel shall be appointed pursuant to Ohio Non-Profit Corporation law to make such decision. The indemnification provided for herein shall not be deemed to restrict the right of the Association to indemnify employees, agents and others as permitted by law.

Section 2. Insurance

2.1. The board of governors may, at its option, purchase and maintain such insurance on behalf of the Association and its governors, officers, employees, agents and others as the board of governors deem appropriate and necessary.

Approved May 30, 2009

Tab Page
Remove before Printing

APSA
FOUNDATION

Back of Tab Page
Remove before Printing

In 1991, a small group of APSA members discussed establishing a foundation for APSA which would foster support for scientific investigation in the field of children's surgery by providing an Annual Enrichment Grant to qualified applicants.

The group led by Dr. Albert H. Wilkinson, Jr., of Jacksonville, FL, included former presidents Kathryn Anderson, James A. O'Neill, Jr., the late Alfred A. de Lorimier, then current President Dick Ellis, the APSA Treasurer Bradley Rodgers and the APSA Secretary Keith Ashcraft. Dr. Wilkinson was the driving force, and with the aid of a Jacksonville law firm, developed the Bylaws and Articles of Incorporation which took effect on October 7, 1992. The Articles of Incorporation of the APSA Foundation were filed with the Secretary of State of Florida on February 2, 1993. Certification followed on March 19, 1993. The initial officers of the Foundation were President Dick Ellis, Secretary, Keith Ashcraft, and Treasurer, Bradley Rodgers, with Dr. Wilkinson serving as the Foundation Agent in the State of Florida. On May 3, 1994, an application for a 501(c)(3) tax-exempt charitable corporation was filed with the Internal Revenue Service and was approved by the U.S. Department of Treasury, Internal Revenue Service on March 15, 1995. At the time, the Foundation Board was led by Jay Grosfeld, APSA President, Donald R. Cooney, Treasurer and Howard Filston, Secretary.

Initially, only 2% of the APSA membership contributed to the fledgling Foundation. The Corpus of the Foundation grew slowly, and the first Enrichment Grant was awarded in 1996 to Dr. Michael Caty in the amount of $9,825. In 1997, the Foundation bylaws were amended and the Board of Directors' membership revised to include (1) the four past presidents of APSA, (2) the APSA secretary, (3) the APSA treasurer and (4) one at-large member, elected to a three-year term by the general membership at the Annual Business Meeting. A formal grant application process with stringent peer review was established as the method for selecting each annual Enrichment Grant recipient.

By its third year, contributors to the APSAF had increased to 7% of the total APSA membership. In 1998, efforts to enhance donor participation were increased by further formalization of the donation process. Dr. Grosfeld was asked to continue as Chairman of the Board of Directors, serving indefinitely at the discretion of the Board. In 2003, Dr. Wilkinson stepped down as Foundation Agent and was replaced by Dr. John Noseworthy. Gift level categories were established including Donor (up to $1,000), Gold Donor ($1,000 or more), Robert E. Gross benefactor ($5,000) and more recently, the William A. Ladd Society level of giving ($10,000) was added. The latter three levels can be achieved by cumulative annual gifts. The active APSA president was considered an *ex officio* member of the Foundation Board, and in 2007, a second at-large member was added to the Board.

APSA Foundation History (cont.)

The corpus of the Foundation has at times exceeded $500,000 despite the fact that less than 20% of the membership have as yet contributed to the Fund. The stipend for each grant has gradually increased, at first to $10,000, and in the past five years to $25,000. Currently, two grants are awarded annually. Since its inception, the APSA Foundation has provided more than $385,000 in grant support for our young pediatric surgeon-scientists (see list below). The return on investment has been extraordinary!

Most of the recipients have used their Enrichment Grants from the APSA Foundation as a springboard from which to acquire significant external funding from the National Institutes of Health (NIH) and other sources.

APSA Foundation Board of Directors

Chairman
Jay L. Grosfeld
Indianapolis, IN

Secretary
Diana L. Farmer
Sacramento, CA

Treasurer
Charles J. Stolar
Santa Barbara, CA

Members
Keith E. Georgeson
Spokane, WA
Michael R. Harrison
San Francisco, CA
Jacob C. Langer
Toronto, ON Canada
Marshall Z. Schwartz
Philadelphia, PA
Thomas F. Tracy, Jr.
Providence, RI

Foundation Agent
John Noseworthy
Jacksonville, FL

***Ex Officio* Member**
Robert C. Shamberger
Boston, MA
APSA President

Your tax-exempt contributions to APSAF have energized young and deserving pediatric surgeons to become some of the leading surgeon-scientists of the future.

2011
Shaun M. Kunisaki, MD
Mesenchymal Stem Cell Regulation of Fetal Lung Development in Diaphragmatic Hernia

Peter Nichol, MD
Using a Genetic Model of Duodenal Atresia to Understand Regenerative Mechanisms within the Intestine

2010
Cynthia D. Downard, MD
Control of Intestinal Microcirculation in NEC

Cassandra M. Kelleher, MD
Extracellular Components Critical to Alveolarization: Contributions of Elastin

2009
Tippi C. MacKenzie, MD
Maternal Immune Response *in Utero* Hematopoietic Stem Cell Transplantation

Kelly A. Miller, MD
The Pathogenic Role of Enteric Glia in Hirschsprung's Enterocolitis

2008
Douglas N. Miniati, MD
Role of Notch4 signaling in Aberrant Pulmonary Vascular Development

2007
Alan M. Goldstein, MD
Role of Sonic Hedgehog in Enteric Nervous System Development

2006
James C.Y. Dunn, MD
Enteric Nervous System Regeneration for Hirschsprung's Disease

2005
Elizabeth A. Beierle, MD
Focal Adhesion Kinase and Vascular Endothelial Growth Factor Receptor-3 in Human Neuroblastoma

Kerilyn K. Nobuhara, MD
Intestinal Dysmotility in Fetal Repair of Gastroschisis

2004
Karl G. Sylvester, MD
Liver Regeneration and Stem Cell Regulation via the WNT Signaling Pathway

Christopher K. Breuer, MD
Do Tissue Engineered Venous Conduits Grow? Investigating the Growth Potential of Tissue Engineered Venous Conduits in a Juvenile Lamb Model

2003
Peter F. Ehrlich, MD
Injury Prevention through Brief Intervention: A Novel Approach to Pediatric Injury Prevention

2002
Mary Beth Madonna, MD
Growth Factor Receptor Signaling and its Relationship to Cell Proliferation and Differentiation in a Neuroblastoma Cell Line

2001
Anthony Stallion, MD
Intestinal Ischemia Reperfusion Injury Contributes to the Initiation of the Systemic Inflammatory Response Syndrome

2000
Edward M. Barksdale, Jr., MD
The Therapy of Neuroblastoma-Induced Disorders of Dendropoiesis of Dendritic Cell Development

1999
Gail E. Besner, MD
Heparin-Binding EGF-Like Growth Factor (HBEGF) and Intestinal Ischemia Reperfusion Injury

The American Pediatric Surgical Association Foundation thanks the following individuals who have contributed to the Foundation*.
*Donations received as of April 15, 2012.

WILLIAM E. LADD SOCIETY BENEFACTORS — $10,000

Bleacher, John C.
Grosfeld, Jay L.
Hays, Daniel M.
Knowles, Joan
Noseworthy, John
Oldham, Keith T.
Raffensperger, John
Wesley, John R.
West, Karen W.

ROBERT E. GROSS BENEFACTORS — $5,000

Altman, R. Peter
Anderson, Glen F.
Ashcraft, Keith W.
Caniano, Donna Anne
Coran, Arnold G.
Donahoe, Patricia K.
Dunn, James C.Y.
Engum, Scott Alan
Filston, Howard C.
Ford, Edward G.
Ford, Henri R.
Gilchrist, Brian F.
Harrison, Michael R.
Hendren, W. Hardy
Holcomb, III, George W.
Klein, Michael D.
Lankau, Jr., Charles
LaQuaglia, Michael P.
Lund, Dennis P.
Meier, Donald E.
Powell, Randall W.
Puranik, Subhash R.
Rescorla, Frederick J.
Rodgers, Bradley M.
Rouse, Thomas M.
Scherer, L.R. (Tres)
Schmeling, David J.
Schwartz, Marshall Z.
Shamberger, Robert C.
Shaul, Donald B.
Sherman, Neil J.
Stylianos, Steven
Toyama, William M.
Tunell, William, P.
Malvin, Weinberger
Ziegler, Moritz M.

GOLD SUPPORTER LEVEL BENEFACTORS — $1,000

Acton, Robert D.
Alaish, Samuel M.
Anderson, Kathryn D.
Andrews, David A.
Arensman, Robert M.
Asch, Morris J.
Azizkhan, Richard G.
Baldwin, Charles E.
Barlow, Barbara Ann
Barnhart, Douglas C.
Beaver, Bonnie L.
Beierle, Elizabeth A.
Besner, Gail E.
Billmire, Deborah F.
Bliss, David W.
Borger, James A.
Bower, Richard J.
Brand, Theodore
Breaux, Jr., Charles W.
Brennom, William
Burnweit, Cathy Anne
Campbell, John R.
Carr, Michael G.
Cavett, Clinton
Chahine, A. Alfred
Chen, Li Ern
Christian, Jeffrey S.
Coln, C. Dale
Consentino, Catherine
Coughlin, John P.
Curci, Michael R.
Curran, Thomas J.
Cusick, Robert A.
DeLorimier, Alfred
Dibbins, Albert W.
Dillon, Peter W.
Dimler, Michael
Dolgin, Stephen E.
Doody, Daniel P.
Doolin, Edward J.
Dorman, George W.
Doski, John J.
Downey, Jr., Earl C.
Drucker, David E. M.
Ehrlich, Peter F.
Ein, Sigmund H.
Ellis, Dick G.
Emmens, Robert W.
Fallat, Mary E.
Farmer, Diana L.
Feins, Neil
Fidler, Christopher D.
Filler, Robert M.
Fiore, Jr., Nicholas F.
Fishman, Steven J.
Folkman, M. Judah
Fonkalsrud, Eric W.
Gandhi, Rajinder P.
Gauderer, Michael W.L.
Gingalewski, Cynthia A.
Ginsburg, Howard B.
Glick, Philip L.
Goodwin, Charles
Diller, B. Groff
Groner, Jonathan I.
Guttman, Frank M.
Guzzetta, Philip C.
Harris, Burton H. & Kathleen
Healey, Patrick J.
Hicks, Barry A.
Hicks, Leon M.
Hill, J. Laurance
Hirschl, Ronald B.
Hoelzer, Dennis J.
Holder, Thomas M.
Holterman, Mark J.
Hong, Andrew R.
Hulka, Frieda Marie
Jan, Dominique M.
Johnson, Dale G.
Jolley, Stephen G.
Kays, David W.
Kessler, Edmund

Kim, Peter C. W.
Kim, Samuel H.
King, Denis R.
Klotz, Jr., Donald
Krummel, Thomas M.
Laberge, Jean-Martin
Ladd, Alan P.
Lally, Kevin P.
Lam, Vinh T.
Langer, Jacob C.
Langenburg, Scott E.
Latchaw, Laurie A.
Ledbetter, Daniel J.
Letton, Robert W.
Lewis, Fawn
Lewis, J. Eugene
Lillehei, Craig W.
Lobe, Thom E.
Loeff, Deborah S.
Long, Julie A.
Luks, Francois I.
Lynch, James M.
Maddox, John R.
Madonna, Mary Beth
Magnuson, David K.
Marr, Clifford C.
Martin, Abigail E.
McGahren, Eugene D.
McGill, Leigh C.
Meagher, David P.
Morgan, William W.
Moriarty, Kevin P.
Moss, R. Lawrence
Murphy, John P.
Newman, Barry M.
O'Neill, James A.
Ortiz-Justiniano, Victor N.
Ostlie, Daniel J.
Othersen, Jr., H. Biemann
Pearl, Richard H.
Pendse, Prabhakar D.
Pinch, Lewis W.
Pittinger, Timothy P.
Powell, David M.
Priebe, Cedric. J
Radhakrishnan, Jayant
Randolph, Judson
Ratner, Michael H.
Raynor, Stephen C.
Reyes, Hernan M.
Rice, Henry E.
Ricketts, Richard R.
Rowe, Marc I.
Ryan, Daniel P.
Ryckman, Frederick C.
Sawin, Robert
Saxton, Mark L.
Schullinger, John N.
Silen, Mark L.
Skinner, Michael A.
Slim, Michael S.
Smith, C.D.
Snyder, Charles J.
Statter, Mindy B.
Stolar, Charles J.
Superina, Riccardo A.
Talbert, James L.
Taylor, Lesli Ann
Teitelbaum, Daniel H.
Templeton, John M.
Tepas, Joseph J.
Touloukian, Robert. J.
Tracy, Thomas F.
Turner, Charles S.
Upperman, Jeffrey S.
Vane, Dennis W.
Vegunta, Ravindra K.
von Allmen, Daniel
Wagner, Charles W.
Waldhausen, John H.T.
Warner, Brad W.
Weber, Thomas R.
Wesson, David E.
Whalen, Thomas V.
Wiener, Eugene S.
Wilkinson, Albert H.

DONORS

Able, Luke
Adamson, William T.
Adelman, Susan H.
Adkins, John C.
Adkins, E. Stanton
Adolph, Vincent R.
Adzick, N. Scott
Alexander, Frederick
Allhouse, Michael J.
Almond, Patrick S.
Alonso, Maria H.
Amoury, Raymond A.
Anderson, Alan E.
Andrassy, Richard J.
Andrews, H. Gibbs
Andrews, Walter S.
Applebaum, Harry
Askew, Allyson A.
Atkinson, James B.
Azarow, Kenneth S.
Baesl, Thomas J.
Bagwell, Charles E.
Bailey, Patrick V.
Bailey, William C.
Barksdale, Jr., Edward M.
Bass, Kathryn D.
Beardmore, Harvey E.
Beck, A. Robert
Bell, Martin J.
Bergman, Kerry S.
Besser, Arthur S.
Bilik, Ron
Birken, Gary
Bishop, Patte J.
Black, Preston R.
Black, Timothy L.
Black, Richard E.
Blair, Geoffrey K.
Bleicher, Michael A.
Bloss, Robert S.
Bodenstein, Lawrence
Boles, Jr., E. Thomas
Bond, Sheldon J.
Boswell, William C.
Bourque, Michael D.
Brandt, Mary L.
Breuer, Christopher K.
Breuer, Julie G.
Brown, Rebeccah L.
Browne, Allen F.
Buchmiller, Terry L.
Buckner, Donald M.
Bufo, Anthony J.
Buntain, William
Burrington, John D.
Butler, Marilyn W.
Cain, Walter S.
Campbell, David P.
Campbell, Timothy J.
Canty, Timothy G.
Cartwright, Karen C.
Casas-Melley, Adela
Cass, Darrell L.
Caty, Michael G.
Chaet, Mark S.
Chang, Chris C. N.
Chang, Jack H.T.
Chen, Steve Chi-Long

Chiu, Priscilla Pul Lam
Chung, Dai H.
Chwals, Walter J.
Cigarroa, Francisco G.
Cobb, L. Mason
Cohn, Bertram
Collins, David L.
Colombani, Paul M.
Connors, Robert H.
Cooke, Ronald W.
Cooney, Donald R.
Cooper, Arthur
Coran, Charles
Coryllos, Elizabeth
Courtney, Richard
Cradock, Thomas V.
Croitoru, Daniel P.
Crombleholme, Timothy M.
Crowe, C. Peter
Crow, John P.
Cywes, Robert
Dahman, Bassam M.
DeRoss, Anthony
D'Angio, Guilio J.
David, Joseph S.
DeCou, James M.
DiFiore, John W.
DuBois, Jeffery J.
Dudgeon, David L.
Eichelberger, Martin R.
El-Shafie, Mohamed
Emil, Sherif G.S.
Escobar, Jr., Mauricio A.
Fagelman, Kerry M.
Falcone Jr., Richard A.
Falla, Anita
Fecteau, Annie H.
Feltis, Brad A.
Figueroa-Otero, Ivan
Fischer, Anne C.
Flageole, Helene
Flake, Alan W.
Foglia, Robert P.
Foster, George L.
Fowler, Carol L.
Frantz, Frazier W.
Friedman, David L.
Frykman, Philip K.
Fullerton, Monte W.
Garcia, Victor F.
Garza, Jennifer
Geissler, Grant H.
German, John C.
Ghory, Mary Jo
Gibbs, David L.
Gibson, Peter
Gittes, George K.
Glasser, James G.
Glynn, Loretto A.
Goldstein, Allan M.
Goretsky, Michael J.
Gosche, John R.
Graham, D. David
Greenfeld, Jonathan Ian
Greenholz, Stephen K.
Grisoni, Enrique R.
Haase, Gerald M.
Hall, Dale G.
Haller, J. Alex
Hamilton, Thomas E.
Hansbrough, Faith
Hardin, William D.
Harmel, Richard P.
Harmon, Carroll M.
Harrison, Marvin W.
Hartman, Gary E.
Haynes, Jeffrey H.
Hebra, Andre V.
Heiss, Kurt F.
Helffrich, Maryanne Dokler
Henderson, Bruce M.
Henderson, Janette A.
Henry, Marion
Hight, Donald W.
Hirsh, Michael P.
Hitch, David C.
Hixson, Douglas S.
Hodin, Earl
Holgersen, Leif
Hollabaugh, Robert S.
Holland, Randall M.
Hollands, Celeste
Hopkins, James W.
Howard, Michael R.
Huang, Yuan-Chao
Hutchins, Carol A.
Idowu, Jr., Olajire
Iocono, Joseph A.
Ishitani, Michael B.
Izant, Robert J.
Jacir, Nabil
Jackson, Richard J.
Jaksic, Tom
Johnson, Frank R.
Jona, Juda Z.
Kanchanapoom, Visut
Karp, Melvyn P.
Karrer, Frederick M.
Kavianian, Ali
Kelly, Robert E.
Kennedy, Alfred P.
Kennedy, Richard
Kenney, Brian
Kim, Hyun Hahk
Kitano, Yoshohiro
Klein, Gerald J.
Klein, Robert L.
Kling, Karen
Konefal, Stanley H.
Koop, C. Everett
Kosloske, Ann M.
Kokoska, Evan R.
Krasna, Irwin H.
Kugaczewski, Jane T.
Kurkchubasche, Arlet G.
Lafer, Dennis J.
Langham, Jr., Max R.
Larson, Shawn D.
Lawrence, John P.
Lazar, Eric L.
Lee, Steven L.
Lemon, David G.
Levitt, Marc A.
Levy, Marc S.
Liebert, Peter S.
Loe, William
LoSasso, Barry F.
Lovvorn, III, Harold N.
Luck, Susan R.
Lynn, Hugh B.
Lynch, Frank P.
Mackie, George G.
Mallory, Baird
Malo, Leslie
Manktelow, Anne
Manning, Peter B.
Martin, Lester W.
Martinez-Frontanilla, Luis A.
McBride, Whitney J.
McGowan, George E.
Meller, Janet L.
Menchaca, John
Meyers, Rebecka L.
Middlesworth, William
Miniati, Douglas N.
Miller, David

APSA Foundation Contributors (cont.)

Miller, James P.
Mirza, Medo
Mooney, David P.
Moore-Olufemi, Stacey
Morden, Robert S.
Morgan, Ross A.
Morton, Jr., Duncan
Moulton, Steven L.
Muenchow, Sharon K.
Musemeche, Catherine A.
Nagaraj, Hirikati S.
Nahmad, Michel H.
Nanagas, Victor N.
Ndiforchu, Fombe
Neblett, Wallace W.
Newman, Kurt D.
Nguyen, Luong T.
Nicolette, Linda A.
Nikaidoh, Hisashi
Noble, H. George S.
Nuchtern, Jed G.
Nuss, Donald
Oiticica, Claudio
Olsen, Margaret M.
Olutoye, Oluyinka O.
Paidas, Charles N.
Palder, Steven
Parker, Paul M.
Pegoli, Jr., Walter
Pena, Alberto
Pettitt, Barbara J.
Philippart, Arvin I.
Pippus, Kenneth G.
Pohlson, Elizabeth C.
Pranikoff, Thomas
Prasad, Rajeev
Price, Mitchell R.
Pulito, Andrew R.
Ramenofsky, Max L.
Rangel, Shawn J.
Ranne, Richard D
Ratner, Irving A.
Reddy, P. Prithvi
Rettig, Arthur
Reynolds, Marleta
Ringer, Jayme
Roback, Stacy
Robertson, Frank M.
Robie, Daniel K.
Rosser, Samuel B.
Rothenberg, Steven S.
Rowe, George A.
Saad, Saad A.
Saites, Constantine G.
Sachs, Barry F.
Saenz, Nicholas C.
Safford, Shawn D.
SanFilippo, J. Anthony
Santos, Mary C.
Sato, Thomas T.
Sauvage, Lester R.
Schaller, Robert T.
Schindel, David T.
Schlatter, Marc G.
Schlechter, Robert D.
Schnitzer, Jay J.
Schuster, Samuel R.
Seashore, John H.
Shafer, Alan D.
Shaw, Anthony
Shilyansky, Joel
Shim, Walton K.T.
Shochat, Stephen J.
Shrock, Peter
Sieber, William K.
Sigalet, David L.
Signer, Richard D.
Skarsgard, Erik D.
Smith, E. Ide
Smith, Melvin D.
Smith, Samuel D.
Seider, Erica
Snyder, Howard M.
Sola, Juan E.
Sonnino, Roberta E.
Stafford, Perry W.
Stallion, Anthony
Stehr, Wolfgang
Steichen, Felicien M.
Stevenson, Richard J.
Stone, Marshall M.
Stovroff, Mark C.
Stringel, Gustavo L.
Swank, Ralph L.
Tagge, Edward P.
Teich, Steven
Telander, Robert L.
Ternberg, Jessie L.
Thompson, W. Raleigh
Torres, Ascension M.
Towne, Barbara H.
Trump, David S.
Uceda, Jorge E.
Uitvlugt, Neal D.
Upp, James Robert
Vacanti, Joseph P.
Valda, Victor
Vaughan, W. Glaze
Velcek, Francisca T.
Wahoff, David C.
Walburgh, C. Eric
Walker, Andrew B.
Webb, H. Warner
Weinberg, Gerard
Weiss, Richard G.
Weissberg, Alan
Weitzman, Jordan
White, John J.
Wilson, Jay Mark
Wilson, Marion Curtiss
Woolley, Morton W.
Wrenn, Jr., Earle L.
Wulkan, Mark L.
Wolf, Stephen A.
Wong, Andrew L.
Yamataka, Atsuyuki
Yedlin, Steven
Zerella, Joseph
Zitsman, Jeffrey S.

Tab Page
Remove before Printing

MEMBERSHIP

Back of Tab Page
Remove before Printing

APSA Distinguished Service Award Recipients

Stephen L. Gans
Marc I. Rowe
Thomas M. Holder
Lucian L. Leape
Harvey E. Beardmore
W. Hardy Hendren

ACS /APSA Executive Leadership Program in Health Policy and Management

Peter W. Dillon - 2012
Patrick V. Bailey - 2011
Aviva L. Katz - 2010
Dennis P. Lund - 2009
George W. Holcomb, III - 2008

APSA /Association of Pediatric Surgeons Training Program Directors M. Judah Folkman Memorial Award Recipients

Best Poster Presentation
2011
Barrie Rich, MD
Predictors of Survival in Childhood and Adolescent Cutaneous Melanoma

2010
Allison L. Speer, MD
Tissue-Engineered Esophagus is a Versatile *in Vivo* Mouse Model with Intact Architecture

2009
Laura A. Boomer, MD
Cholangiocyte Apoptosis During Lamprey Metamorphosis

2008
Henry L. Chang, MD
In Vivo Metastatic/Invasion Assay to Identify Cancer Stem Cells and their Markers

Best Podium Presentation
2011
Amar Nijagal, MD
The Maternal Adaptive Immune Response Against Paternal Antigens Incites Fetal Demise After Fetal Intervention

2010
Mehul V. Raval, MD
Pediatric ACS NSQIP: Feasibility of a Novel Prospective Assessment of Surgical Outcomes - a Phase I Report

2009
Eric Jelin, MD
Effects of Notch4 On Lung Vascular Remodeling

2008
Emily T. Durkin, MD
The Ontogeny of Human Fetal NK Cell Allorecognition: A Potential Barrier to *in Utero* Transplantation

APSA Posters of Distinction

Basic Science
2011
R. Dawn Fevurly, MD
Novel Zebrafish Model Reveals Critical Role for MAPK in Lymphangiogenesis

2010
Hayden W. Stagg, MD
Matrix Metalloproteinase-9 Induces Hyperpermeability Following Traumatic Burn Injury

2009
Francois I. Luks, MD
Reflectance Spectrometry for Realtime Hemoglobin Determination of Placental Vessels During Endoscopic Laser Surgery for TTTS

Clinical
2011
Jesse Gutnick, MD
Circulating Thyrotropin Receptor MRNA for Evalution of Thyroid Nodules and Surveillance of Thyroid Cancer

2010
Diana L. Diesen, MD
Temporal Association Between Blood Transfusion and Necrotizing Enterocolitis in Premature Infants

2009
Henry L. Chang, MD
Mullerian Inhibiting Substance Inhibits Migration of Epithelial Cancer Cell Lines

The APSA Board of Governors and Membership Congratulates Our Newest Members

Regular Members

Acierno, Stephanie P.
Austin, Mary T.
Bae, Jae-O
Chen, Li Ern
Clifton, Matthew S.
Dicken, Bryan J.
Diefenbach, Karen A.
Emran, Mohammad A.
Fairbanks, Timothy J.
Hamner, Chad E.
Jarboe, Marcus D.
Jones, Sarah A.
Kapfer, Stephanie A.
Kawaguchi, Akemi L.
Kelleher, Cassandra M.
Lawlor, David
Lee, Yi-Horng
McMahon, Lisa M.
Prince, Jose M.
Potter, Donald D.
Riehle, Kimberly J.
Rowell, Erin E.
Spurbeck, William W.
Stehr, Wolfgang
Vogel, Adam M.
Walford, Beth

Candidate Members

Akay, Begum
Askegard-Giesmann, Johanna R.
Boutros, John
Chiu, Bill
Crowley, Helena M.
Diesen, Diana L.
Fisher, Jason C.
Gadepalli, Samir K.
Garrett, Deiadra J.
Guner, Yigit S.
Hartin, Charles W., Jr.
Harting, Matthew T.
Heaton, Todd E.
Iqbal, Corey W.
Jones, Stephanie A.
Langer, Monica
Lao, Oliver B.
Lee, Sang Il
Lo, Andrea Y.
Lumpkins, Kimberly M.
Markel, Troy A.
Maxwell, Damien R.
Merianos, Demetri J.
Modi, Biren P.
Murrell, Zaria C.
Naik-Mathuria, Bindi
Piper, Hannah G.
Ricca, Robert L., Jr.
Ruscher, Kimberly A.
Russell, Robert T.
Shah, Ami N.
Taylor, Janice A.
Wall, James K.
Zalieckas, Jill M.

Associate Member

Mon, Rodrigo A.

Resident Members (General Surgery Residents)

Barlow, Meade P.
Bryner, Benjamin S.
Cappiello, Clint D.
Coleman, Alan M.
Coots, Abigail C.
Dalton, Brian G.A.
Fitzwater, John W.
Garcia, Megan L.
Gonzales, Kelly D.
Hartwich, Joseph E.
Jeziorczak, Paul M.
Johnson, Jeremy J.
Jones, Brian A.
Kennedy, Raelene D.
Kiser, Michelle M.
Khozeimeh, Nini
Knott, Erol M.
Leeflang, Elisabeth J.
Lillegard, Joseph B.
Long, Eric L.
Lozada, Joseph S.
Maki, Alexandra C.
McAteer, Jarod P.
Munaco, Anthony J.
Neff, Luke P.
Nice, Tate, R.
Rentea, Rebecca M.
Ryan, Mark L.
Schneider, John G.
Sharp, Nicole E.
Short, Joshua J.
Smith, Jennifer B.
Stone, Matthew L.
Timmons, Jennifer A.
Whitehead, Alia F.
Zamora, Irving J.

Pledge for New Members of the American Pediatric Surgical Association

This pledge will be read before the New Member Induction Ceremony.

As president of the American Pediatric Surgical Association, it is my pleasure to welcome you into regular membership and to stress the obligations that you assume by such membership.

The American Pediatric Surgical Association was founded on April 15, 1970, by 200 surgeons drawn together to encourage specialization in the field of pediatric surgery; to make available the benefits to be derived from the services of qualified pediatric surgeons; to promote and maintain the quality of education in pediatric surgery through meetings, lectures and the distribution of printed materials; to raise the standards of the specialty by fostering and encouraging research and scientific progress in pediatric surgery and by establishing standards of excellence in the surgical care of infants, children and teenagers; and to provide a forum for the dissemination of information with regard to pediatric surgery.

The association expects its new members to support the objectives and obligations of the association as set forth in the Articles of Incorporation and to reflect the values expressed in the Principles of Medical Ethics as stated in the Preamble to the Bylaws. The members are also expected to support the association through active participation in its meetings. We look forward to your contributions in advancing its proud traditions.

If you pledge to exemplify the high ethical and professional standards of the American Pediatric Surgical Association in your practice of surgery, and if you will participate actively in future meetings, please respond by stating "I will." Since you have indicated your intent to become an active and worthy member and since you have been duly elected, I now declare you to be a regular member of the American Pediatric Surgical Association.

I now call upon the current members and guests of the American Pediatric Surgical Association to rise and join me in welcoming our new colleagues.

Gilbert, Michel 1972
Gamion, Robers S. Jr. 1973
Chamberlain, John W. 1974
Snyder, William H. Jr. 1974
Bracey, Altamount 1978
Erwin, James H. 1979
White, Robert F. 1980
Allen, Robert G. 1981
Karn, Gordon M. 1981
Kiesewetter, William B. 1981
Schneider, Keith M. 1982
Hawes, Ernest B. 1984
Lozoya-Solis, Jesus 1984
Soave, Franco 1984
Rosenkrantz, Jens G. 1985
Cresson, Samuel L. 1986
Owings, Richard S. 1986
Pilling, George P. IV 1986
Stewart, David R 1986
Simpson, James Stanley 1988
Gross, Robert E. 1988
Ravitch, Mark M. 1989
Ballantine, Thomas V.N 1990
Ferguson, Colin C. 1991
Mishalany, Henry 1991
Schisgall, Richard M. 1991
David, Ronald 1992
Kaufman, Bruce 1992
Harkins, George A. 1993
Sakaguchi, Shimpei 1993
Segnitz, Richard H. 1993
Gans, Stephen L. 1994
Kumar, A.P. Mahesh 1994
McParland, Felix A. 1994
Pokorny, William J. 1994
Richardson, William R. 1994
Benson, Clifford D. 1995
Lilly, John R. 1995
Riker, William L. 1995
Bill, Alexander H. (Sandy) . . . 1996
Cheu, Henry W. 1996
Danis, Richard K. 1996
Goldstein, I. Richard 1996
Longino, Luther A 1996
Welch, Kenneth J. 1996
Baffes, Thomas G. 1997
Bettex, Marcel 1997
Salzberg, Arnold M.. 1997
Santulli, Thomas V. 1997
Brennan, L. Patrick 1998
Brooks, Benjy F. 1998
Carson, James A. 1998
Hamilton, James P. 1998
Stanley-Brown, Edward G. . . . 1998
Knutrud, Ola. 1999
Warden, M. James 1999
Winslow, Paul 1999
Zachary, R.B 1999
Linkner, Laurance M. 2000
Meeker, Irving A. Jr. 2000
Chisholm, Tague C.. 2000
McAteer, Jerry. 2001
Clatworthy, William 2001
Allen, James E. 2001
Lizarralde, A. Eduardo. 2001
Weitzman, Jordan J.. 2001
Campbell, David P. 2001
Carcassone, Michel 2002
Cohn, Bertram D. 2002
Colodny, Arnold H.. 2002
Eraklis, Angelo J. 2002
Smith, Willard D.. 2002
So, Henry B. 2002
Tapper, David 2002
Zwiren, Gerald T. 2002
Abrams, Martin W. 2003
Harberg, Franklin J. (Jim) 2003
Lynch, Frank P. III 2003
Smith, E. Ide 2003
Rickham, Peter P.. 2003
Huseby, Thomas L. 2004
Izant, Robert 2004
Pickett, Lawrence K. 2004
Bronsther, Burton 2004
Stahl, Nicholas M. 2004
Phillipart, Arlvin I. III 2004
McAlpin, Columbus D. 2004
Lloyd, James R. 2004
Moore, Thomas C. 2004
Rathauser, Frank 2005
Fitzpatrick, John 2005
Able, Luke W. 2006
Andrews, Gibb. 2006
Jewett, Theodore C. 2006

In Memoriam (cont.)

Rothmann, Bruce F. 2006
Wiener, Eugene S. 2006
Beardmore, Harvey E. 2007
Black, Preston R. 2007
Cox, Joseph A. 2007
Exelby, Philip R. 2007
Mollitt, Daniel L. 2007
Ratner, Irving A. 2007
McClenathan, James E. 2007
Pitts, R. Marshall 2007
Wolfson, Philip J. 2007
Folkman, M. Judah 2007
Smith, Melvin D. 2007
McGovern, Bruce 2008
MacDonald, James S. 2008
Campbell, Timothy J. 2008
Votteler, Theodore P. 2008
Cooney, Donald R. 2008
Cooke, Ronald W. 2009
Anderson, Alan E. 2009
de Lorimier, Alfred A. 2009
Fisher, John H. 2009
Mercer, Stanley 2010
Schultz, Lloyd R. 2010
Hayes, Lawrence E. 2010
Besser, Arthur S. 2010
Verhagan, Arie D. 2010
Cloud, Daniel T. 2010
Altman, R. Peter 2011
DeLuca, Frank G. 2011
Schnaufer, Louise. 2011
Stephens, Frank D. 2011
Roback, Stacy A. 2011

Founding Members

Fred Arcari, Royal Oak, MI
E. Thomas Boles, Columbus, OH
John R. Campbell, Portland, OR
Alfred A. de Lorimier, Geyserville, CA
Frank G. DeLuca, Barrington, RI
Robert M. Filler, Toronto, ON, Canada
Eric W. Fonkalsrud, Santa Monica, CA
Edward A. Free, Prescott, AZ
Dale G. Johnson, Rutledge, TN
Peter K. Kottmeier, Salt Lake City, UT
Lucian L. Leape, Boston, MA
Julius Lister, Framingham, MA
John Raffensperger, Sanibel, FL
Mark I. Rowe, Sanibel, FL
William K. Sieber, Yerona, PA
Robert T. Soper, Iowa City, IA
James A. Talbert, Gainesville, FL
Edward S. Tank, Portland, OR

Charter Members

Raymond A. Amoury, Kansas City, MO
H. Paulsen Armstrong, Baton Rouge, LA
A. Robert Beck, New York, NY
Jerrold M. Becker, New Hyde Park, NY
Clifford R. Boeckman, Salem, SC
Scott J. Boley, Bronx, NY
William E. Bomar, Gray Court, SC
John D. Burrington, Colorado Springs, CO
John L. Cahill, Indian Wells, CA
Walter S. Cain, Birmingham, AL
Gordon S. Cameron, Dunas, ON, Canada
Daniel T. Cloud, Phoenix, AZ
David L. Collins, San Diego, CA
Elizabeth Coryllos, Mineola, NY
C. Peter Crowe, Tucson, AZ
Joseph S. David, Eagle, ID
Jean G. DesJardins, Saint-Laurent, QC, Canada
Pieter A. deVries, Larkspur, CA
George W. Dorman, Prescott, AZ

Jacques C. Ducharme, Mont Royal, QC, Canada
Dick G. Ellis, Fort Worth, TX
John H. Fisher, Marshfield, MA
Eric W. Fonkalsrud, Santa Monica, CA
Eugene Garrow, Jersey City, NJ
Marvin Glicklich, Fox Point, WI
Leonard Graivier, Dallas, TX
Jacob A. Haller, Glencoe, MD
Daniel M. Hays, Riverside, CA
Bruce M. Henderson, Corpus Christi, TX
W. Hardy Hendren, Duxbury, MA
Jack H. Hertzler, Franklin, MI
George W. Holcomb, Nashville, TX
Thomas M. Holder, Prairie Village, KS
James W. Hopkins, Windsor Heights, IA
George A. Hyde, Horare, Avondale, Zimbabwe
Patrick F. Jewell, Lincoln, CA
Frank R. Johnson, Woodstock, IL
Kenneth Kenigsberg, Glen Cove, NY
William N. Kincannon, Santa Barbara, CA
Murray R. Kliman, Vancouver, BC, Canada
Charles H. Klippel, Paxton, MA
Irwin H. Krasna, Forest Hills, NY
Dennis J. Lafer, Jacksonville, FL
J. Eugene Lewis, St. Louis, MO
Peter S. Liebert, White Plains, NY
Hugh B. Lynn, Winchester, VA
Enrique Marquez, San Juan, PR
Lester W. Martin, Bellbrook, OH
R. W. Paul Mellish, Dhahran, Saudi Arabia
Ascher L. Mestel, Brooklyn, NY
Richard C. Miller, Jackson, MS
David R. Murphy, Kingston, ON Canada
H. Biemann Othersen, Charleston, SC
Cedric J. Priebe, Stony Brook, NY
Thomas C. Putnam, Rockland, ME
Judson Randolph, Nashville, TN
Lester R. Sauvage, Seattle, WA
Louise Schnaufer, Philadelphia, PA
John N. Schullinger, Woodstock, VT
Lloyd Schultz, Omaha, NE
Samuel R. Schuster, Westboro, MA
Alan D. Shafer, Dayton, OH
Barry Shandling, Toronto, ON, Canada
Anthony Shaw, Pasadena, CA
Walton K.T. Shim, Honolulu, HI
Laurence A. Somers, Lafayette Hill, PA
Bernard J. Spencer, Sanibel Island, FL
Rowena Spencer, New Orleans, LA
Nicholas M. Stahl, Charlestown, RI
Felicien M. Steichen, Mamaroneck, NY
H. Harlan Stone, Glenville, NC
Kamthorn Sukarochana, Pittsburgh, PA
Orvar Swenson, Charleston, SC
Jessie L. Ternberg, St. Louis, MO
Robert J. Touloukian, New Haven, CT
David S. Trump, Grants Pass, OR
Kenneth R. Tyson, Burnet, TX
Arie D. Verhagen, Hamilton, OH
Vollrad J. Von Berg, Hot Springs, AR
Theodore P. Votteler, Dallas, TX
H. Warner Webb, Jacksonville, FL
John J. White, Seattle, WA
Albert H. Wilkinson, Jacksonville, FL
Morton M. Woolley, Rancho Mirage, CA
Earle L. Wrenn, Greensboro, NC

Tab Page
Remove before Printing

SCHEDULE AND PROGRAM

Back of Tab Page
Remove before Printing

Friday, May 18

2:00 – 5:00 p.m.	Pediatric Surgery Training Program Directors Meeting	*Bluebonnet/Dogwood*
2:00 – 6:00 p.m.	Registration Open	*Cibolo Canyon Foyer*
2:00 – 6:00 p.m.	Speaker Ready Room Open	*Cibolo Canyon Foyer*
2:00 – 6:00 p.m.	Internet Café Open	*Cibolo Canyon Foyer*
4:00 – 10:00 p.m.	APSA Board of Governors Meeting	*Sunday House*

Saturday, May 19

6:30 a.m. – 5:00 p.m.	Registration Open	*Cibolo Canyon Foyer*
6:30 a.m. – 5:00 p.m.	Speaker Ready Room Open	*Cibolo Canyon Foyer*
6:30 a.m. – 5:00 p.m.	Internet Café Open	*Cibolo Canyon Foyer*
APSA-IPSO Symposium		
8:00 – 8:15 a.m.	Welcome: APSA and IPSO Presidents	*Exhibit Ballroom Salon A*
8:15 – 10:00 a.m.	**Neuroblastoma Session**	*Exhibit Ballroom Salon A*
10:00 – 10:30 a.m.	Refreshment Break	*Exhibit Ballroom Salon Foyer*
10:30 a.m. – Noon	**Scientific Session I** Oncology	*Exhibit Ballroom Salon A*
Noon – 1:00 p.m.	Lunch (individual arrangements)	
1:00 – 3:00 p.m.	**Liver Tumors Session** Focus on Hepatoblastoma	*Exhibit Ballroom Salon A*
3:00 – 3:30 p.m.	Refreshment Break	*Exhibit Ballroom Salon Foyer*
3:30 – 5:00 p.m.	**Spectacular/Challenging Cases Session**	*Exhibit Ballroom Salon A*
6:00 – 8:00 p.m.	APSA-IPSO Reception	*Event Lawn 3*
6:30 – 10:00 p.m.	Publications Committee Meeting	*Sunflower*

Sunday, May 20

6:00 – 8:00 a.m.	Committee Meetings	*See page 69 for ancillary meeting schedule*
7:00 a.m. – 5:00 p.m.	Registration Open	*Cibolo Canyon Foyer*
7:00 a.m. – 5:00 p.m.	Speaker Ready Room Open	*Cibolo Canyon Foyer*
7:00 a.m. – 5:00 p.m.	Internet Café Open	*Cibolo Canyon Foyer*
7:15 – 7:45 a.m.	Continental Breakfast	*Cibolo Canyon Foyer*
7:45 – 8:00 a.m.	President's Welcome	*Cibolo Canyon Salons 5-11*
8:00 – 10:00 a.m.	Companion Hospitality Room Open for Registered Companions	*Crooked Branch*

Sunday, May 20 (cont.)

APSA-IPSO Symposium in Conjuction with APSA Education Day		
8:00 – 11:00 a.m.	**Education Session I** Management of Metastatic Disease	*Cibolo Canyon* *Salons 5-11*
11:00 – 11:15 a.m.	Refreshment Break	*Cibolo Canyon Foyer*
11:15 a.m. – 12:15 p.m.	**Jay and Margie Grosfeld Lecture** M. James Kaufman, PhD *Health Care Reform – The Impact on Children*	*Cibolo Canyon* *Salons 5-11*
12:15 – 12:30 p.m.	Box Lunch Pick-Up	*Cibolo Canyon Foyer*
12:30 – 2:00 p.m.	Lunch Session—TBA	*Cibolo Canyon* *Salons 5-11*
2:00 – 4:00 p.m.	**Concurrent Session** **Education Session II** Joint Session with APSMA: Billing and Coding Update	*Cibolo Canyon* *Salons 5-11*
	Education Session III Basic Science Update	*Cibolo Canyon* *Salons 1-4*
4:00 – 5:00 p.m.	Poster Wine and Cheese Reception	*Cibolo Canyon Foyer*
4:15 – 6:00 p.m.	**Concurrent Sessions:** **Poster Session I** Basic Science	*Cibolo Canyon* *Salons 5-11*
	Poster Session II Clinical	*Cibolo Canyon* *Salons 1-4*
6:30 – 8:30 p.m.	Welcome Reception	*Event Lawn 2*

Monday, May 21

5:15 a.m.	5K Fun Run – *Pre-Registration Required*	*Sunday House Garden*
6:15 – 7:30 a.m.	APSA Foundation Board Meeting	*Wisteria*
6:30 – 7:30 a.m.	Committee Meetings	*See page 69 for ancillary meeting schedule*
6:30 – 10:00 a.m.	Poster Set Up	*Cibolo Canyon* *Salons 1-4*
6:30 a.m. – 1:00 p.m.	Registration Open	*Cibolo Canyon Foyer*
6:30 a.m. – 2:00 p.m.	Speaker Ready Room Open	*Cibolo Canyon Foyer*
6:30 a.m. – 1:00 p.m.	Internet Café Open	*Cibolo Canyon Foyer*
6:45 – 7:30 a.m.	Continental Breakfast	*Cibolo Canyon Foyer*
6:45 a.m. – 1:00 p.m.	Exhibits Open	*Cibolo Canyon Foyer*
7:30 – 9:00 a.m.	**Scientific Session II** Clinical: Clinical Trials and Quality Improvement	*Cibolo Canyon* *Salons 5-11*
8:00 – 10:00 a.m.	Companion Hospitality Room Open for Registered Companions	*Crooked Branch*

Monday, May 21 (cont.)

8:30 a.m. – 12:30 p.m.	Companion Tour – "Cruisin' and Exporin' San Antonio" Tour *Pre-Registration Required*	*Front Lobby*
9:00 – 10:00 a.m.	**Robert E. Gross Lecture** Daniel M. Green, MD *The Evolution of Treatment for Wilms' Tumor*	*Cibolo Canyon Salons 5-11*
10:00 – 10:30 a.m.	Refreshment Break	*Cibolo Canyon Foyer*
10:00 a.m. – 1:00 p.m.	Posters open for viewing	*Cibolo Canyon Salons 1-4*
10:30 – 11:45 a.m.	**Scientific Session III** Fetal/Neonatal and Trauma	*Cibolo Canyon Salons 5-11*
11:45 a.m. – 12:45 p.m.	**International Guest Lecture** Benno M. Ure, MD *Enthusiasm, Evidence and Ethics: the Triple E of Minimally Invasive Pediatric Surgery*	*Cibolo Canyon Salons 5-11*
1:00 – 2:30 p.m.	Benjy Brooks Meeting and Luncheon *Pre-Registration Required*	*Verbena/Periwinkle*
2:00 – 5:00 p.m.	Tennis Tournament *Pre-Registration Required*	*Meet at the hotel concierge desk*
2:00 – 6:00 p.m.	Golf Tournament *Pre-Registration Required*	*TPC San Antonio, AT&T Oaks Golf Course*
5:00 – 6:30 p.m.	*Journal of Pediatric Surgery* Reception (By Invitation)	*Sunday House*

Tuesday, May 22

6:30 – 8:00 a.m.	Member Business Meeting with Breakfast	*Cibolo Canyon Salons 5-11*
6:30 a.m. – 3:30 p.m.	Registration Open	*Cibolo Canyon Foyer*
6:30 a.m. – 3:30 p.m.	Speaker Ready Room Open	*Cibolo Canyon Foyer*
6:30 a.m. – 3:30 p.m.	Internet Café Open	*Cibolo Canyon Foyer*
7:00 – 8:00 a.m.	Continental Breakfast for Non-Members	*Cibolo Canyon Foyer*
7:00 a.m. – 2:00 p.m.	Exhibits open	*Cibolo Canyon Foyer*
7:00 a.m. – 3:00 p.m.	Posters open for viewing	*Cibolo Canyon Salons 1-4*
8:00 – 9:00 a.m.	***Journal of Pediatric Surgery* Lecture** Brad W. Warner, MD *Adaptation: Paradigm for an Academic Career and the Gut*	*Cibolo Canyon Salons 5-11*
8:00 – 10:00 a.m.	Companion Hospitality Room Open for Registered Companions	*Crooked Branch*
9:00 – 10:15 a.m.	**Scientific Session IV** Basic Science	*Cibolo Canyon Salons 5-11*
9:30 – 11:00 a.m.	Companion's Meeting	*Crooked Branch*
10:15 – 10:45 a.m.	Refreshment Break	*Cibolo Canyon Foyer*

Tuesday, May 22 (cont.)

10:45 – 11:15 a.m.	**APSA Foundation Scholars** Shaun M. Kunisaki, MD *Mesenchymal Stem Cell Regulation of Fetal Lung Development in Diaphragmatic Hernia*	*Cibolo Canyon Salons 5-11*
	Cassandra M. Kelleher, MD *Extracellular Components Critical to Alveolarization: Contributions of Elastin*	
11:15 – 11:30 a.m.	Introduction of New Members	*Cibolo Canyon Salons 5-11*
11:30 a.m. – 12:30 p.m.	**Presidential Address** Robert C. Shamberger, MD *Cooperative Group Trials in Pediatric Oncology: The Surgeon's Role*	*Cibolo Canyon Salons 5-11*
12:30 – 12:45 p.m.	Box Lunch Pick-Up	*Cibolo Canyon Foyer*
12:45 – 1:45 p.m.	**Innovation Session** Abstracts on New and Innovative Techniques and Procedures	*Cibolo Canyon Salons 5-11*
1:45 – 2:45 p.m.	**Video Session**	*Cibolo Canyon Salons 5-11*
2:00 – 5:00 p.m.	Exhibitor Dismantle	*Cibolo Canyon Foyer*
2:45 – 6:45 p.m.	Leisure time	
3:00 – 5:30 p.m.	Poster Dismantle	*Cibolo Canyon Salons 1-4*
6:45 – 7:30 p.m.	President's Reception	*Cibolo Canyon Foyer*
7:30 – 10:00 p.m.	President's Banquet	*Cibolo Canyon Salons 5-11*

Wednesday, May 23

6:30 – 7:30 a.m.	Committee Meetings	*See page 69 for ancillary meeting schedule*
7:00 – 8:00 a.m.	Continental Breakfast	*Cibolo Canyon Foyer*
7:00 – 10:30 a.m.	Speaker Ready Room Open	*Cibolo Canyon Foyer*
7:00 – 11:30 a.m.	Internet Café Open	*Cibolo Canyon Foyer*
7:30 – 11:30 a.m.	Registration Open	*Cibolo Canyon Foyer*
8:00 – 9:15 a.m.	**Scientific Session V** Clinical: Common Problems	*Cibolo Canyon Salons 5-11*
9:15 – 10:15 a.m.	**Outcomes and Clinical Trials Update** NEC; VATS/Empyema	*Cibolo Canyon Salons 5-11*
10:15 – 10:30 a.m.	Refreshment Break	*Cibolo Canyon Foyer*
10:30 a.m. – Noon	**Pediatric Surgery Case Debates and Controversies**	*Cibolo Canyon Salons 5-11*
Noon	Annual Meeting Concludes	

Ancillary Meetings by Day

Committee	Time	Room
FRIDAY, MAY 18		
Association of Pediatric Surgery Training Program Directors	2:00 – 5:00 p.m.	Blue Bonnet/Dogwood
SATURDAY, MAY 19		
Simulation	6:00 – 8:00 a.m.	Larkspur
CME	6:00 – 8:00 a.m.	Blue Bonnet
PSSAP	6:00 – 8:00 a.m.	Dogwood
Education	6:00 – 8:00 a.m.	Indian Paintbrush
Publications	6:30 – 10:00 p.m.	Sunflower
SUNDAY, MAY 20		
Pediatric NSQIP Surgeon Champions	6:00 – 7:00 a.m.	Begonia
Combined Meeting - CME, Education, PSSAP, Simulation Sub-Committee	6:00 – 8:00 a.m.	Wisteria/Sunflower
Fetal Diagnosis and Treatment	6:00 – 8:00 a.m.	Freesia
Outcomes and Clinical Trials	7:00 – 8:00 a.m.	Peony
Practice	6:00 – 8:00 a.m.	Goldenrod
Program	6:00 – 8:00 a.m.	Alyssum
OCHSiC	6:00 – 8:00 a.m.	Blue Bonnet/Dogwood
Family and Community Relations	7:00 – 8:00 a.m.	Indian Paintbrush
Informatics and Telemedicine	7:00 – 8:00 a.m.	Begonia
MONDAY, MAY 21		
APSA Foundation Board Meeting	6:15 – 7:30 a.m.	Wisteria
New Technology	6:00 – 7:00 a.m.	Sunflower
Childhood Obesity	6:00 – 7:30 a.m.	Alyssum
Ethics and Advocacy	6:00 – 7:30 a.m.	Goldenrod
Membership and Credentials	6:00 – 7:30 a.m.	Freesia
Surgical Quality and Safety	6:00 – 7:30 a.m.	Peony
Trauma	6:00 – 7:30 a.m.	Begonia
Workforce	6:00 – 7:30 a.m.	Paintbrush
Children's Steering Committee	6:00 – 7:30 a.m.	Blue Bonnet
Cancer	6:30 – 7:30 a.m.	Dogwood
ACS Advisory Council for Pediatric Surgery	4:00 – 5:30 p.m.	Peony
JPS Reception	5:00 – 6:30 p.m.	Sunday House
BCM Reunion (Baylor College of Medicine Residency Program)	5:00 – 7:00 p.m.	Blue Bonnet/Dogwood
TUESDAY, MAY 22		
Companions' Roundtable Meeting	9:30 – 10:30 a.m.	Crooked Branch
Neonatal Research Network	3:00 – 5:00 p.m.	Begonia/Bottlebrush
WEDNESDAY, MAY 23		
International Relations	6:00 – 8:00 a.m.	Begonia
DHREAMS	6:30 – 8:00 a.m.	Blue Bonnet
Surgical Critical Care	7:00 – 8:00 a.m.	Dogwood
Industry	7:00 – 8:00 a.m.	Bottlebrush

Ancillary Meetings by Group

Committee	Date/Time	Room
ACS Advisory Council for Pediatric Surgery	Monday, May 21, 4:00 – 5:30 p.m.	Peony
APSA Foundation Board Meeting	Monday, May 21, 6:15 – 7:30 a.m.	Wisteria
Association of Pediatric Surgery Training Program Directors	Friday, May 18, 2:00 – 5:00 p.m.	Blue Bonnet/Dogwood
BCM Reunion (Baylor College of Medicine Residency Program)	Monday, May 21, 5:00 – 7:00 p.m.	Blue Bonnet/Dogwood
Cancer	Monday, May 21, 6:30 – 7:30 a.m.	Dogwood
Childhood Obesity	Monday, May 21, 6:00 – 7:30 a.m.	Alyssum
Children's Steering Committee	Monday, May 21, 6:00 – 7:30 a.m.	Blue Bonnet
CME	Saturday, May 19, 6:00 – 8:00 a.m.	Blue Bonnet
Combined Meeting - CME, Education, PSSAP, Simulation Sub-Committee	Sunday, May 20, 6:00 – 8:00 a.m.	Wisteria/Sunflower
Companions' Roundtable Meeting	Tuesday, May 22, 9:30 – 10:30 a.m.	Crooked Branch
DHREAMS	Wednesday, May 23, 6:30 – 8:00 a.m.	Blue Bonnet
Education	Saturday, May 19, 6:00 – 8:00 a.m.	Indian Paintbrush
Ethics and Advocacy	Monday, May 21, 6:00 – 7:30 a.m.	Goldenrod
Family and Community Relations	Sunday, May 20, 7:00 – 8:00 a.m.	Indian Paintbrush
Fetal Diagnosis and Treatment	Sunday, May 20, 6:00 – 8:00 a.m.	Freesia
Industry	Wednesday, May 23, 7:00 – 8:00 a.m.	Bottlebrush
Informatics and Telemedicine	Sunday, May 20, 7:00 – 8:00 a.m.	Begonia
International Relations	Wednesday, May 23, 6:00 – 8:00 a.m.	Begonia
JPS Reception	Monday, May 21, 5:00 – 6:30 p.m.	Sunday House
Membership and Credentials	Monday, May 21, 6:00 – 7:30 a.m.	Freesia
Neonatal Research Network	Tuesday, May 22, 3:00 – 5:00 p.m.	Begonia/Bottlebrush
New Technology	Monday, May 21, 6:00 – 7:00 a.m.	Sunflower
OCHSiC	Sunday, May 20, 6:00 – 8:00 a.m.	Blue Bonnet/Dogwood
Outcomes and Clinical Trials	Sunday, May 20, 7:00 – 8:00 a.m.	Peony
Pediatric NSQIP Surgeon Champions	Sunday, May 20, 6:00 – 7:00 a.m.	Begonia
Practice	Sunday, May 20, 6:00 – 8:00 a.m.	Goldenrod
Program	Sunday, May 20, 6:00 – 8:00 a.m.	Alyssum
PSSAP	Saturday, May 19, 6:00 – 8:00 a.m.	Dogwood
Publications	Saturday, May 19, 6:30 – 10:00 p.m.	Sunflower
Simulation	Saturday, May 19, 6:00 – 8:00 a.m.	Larkspur
Surgical Critical Care	Wednesday, May 23, 7:00 – 8:00 a.m.	Dogwood
Surgical Quality and Safety	Monday, May 21, 6:00 – 7:30 a.m.	Peony
Trauma	Monday, May 21, 6:00 – 7:30 a.m.	Begonia
Workforce	Monday, May 21, 6:00 – 7:30 a.m.	Indian Paintbrush

The APSA Annual Meeting is designed to provide comprehensive continuing education in the field of pediatric surgery. APSA strives to bring together the world's leading pediatric surgery authorities to present and discuss the most recent clinical and research efforts. There will be five scientific sessions with abstract presentations, a video session, an innovation session, case debates and controversies, and a session on outcomes and clinical trials. This year the APSA annual meeting is preceded by the APSA-IPSO Symposium. Starting on Saturday, May 19, and going through 11:00 a.m. on Sunday, May 20, the APSA-IPSO Symposium will feature a neuroblastoma session, oncology research abstracts, a liver tumors session and a spectacular/challenging cases session.

The APSA Annual Meeting covers the breadth of pediatric surgery and is intended to acquaint attendees with the latest research findings, clinical discoveries and trends that influence the day-to-day practice of pediatric surgery. Specific sessions relating to educating members on new developments in medical technology have been added to supplement the traditional sessions on clinical practice and basic science research chosen by the Program and Education Committees. The scientific sessions consist of basic research and practical clinical presentations. The poster sessions are intended to provide young investigators an opportunity to share preliminary clinical research, basic science work and novel ideas. The meeting will begin with the APSA-IPSO Symposium in conjunction with the APSA Education Day program on Sunday, May 20, with a focus on metastatic disease. Afternoon sessions include a joint session with the American Pediatric Surgical Managers Association (APSMA) on billing and coding and a basic science update. Meeting attendees will also view and discuss selected poster presentations.

Accreditation Statement

APSA is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. This live CME activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education (ACCME).

APSA 43rd Annual Meeting
This live activity has been approved for a maximum of 20.5 *AMA PRA Category 1 Credit(s)™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

APSA-IPSO Symposium
APSA designates this live activity for a maximum of 9.75 *AMA PRA Category 1 Credit(s)™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Delegates attending all sessions of the APSA-IPSO Symposium and the APSA 2012 Annual meeting can claim a maximum of 27.25 AMA *PRA Category 1 Credit(s)™*.

Disclaimer: THESE MATERIALS AND ALL OTHER MATERIALS PROVIDED IN CONJUNCTION WITH CME ACTIVITIES ARE INTENDED SOLELY FOR PURPOSES OF SUPPLEMENTING CME PROGRAMS FOR QUALIFIED HEALTH CARE PROFESSIONALS. ANYONE USING THE MATERIALS ASSUMES FULL RESPONSIBILITY AND ALL RISK FOR THEIR APPROPRIATE USE. APSA MAKES NO WARRANTIES OR REPRESENTATIONS WHATSOEVER REGARDING THE ACCURACY, COMPLETENESS, CURRENTNESS, NONINFRINGEMENT, MERCHANTABILITY OR FITNESS FOR A PARTICULAR PURPOSE OF THE MATERIALS. IN NO EVENT WILL APSA BE LIABLE TO ANYONE FOR ANY DECISION MADE OR ACTION TAKEN IN RELIANCE ON THE MATERIALS. IN NO EVENT SHOULD THE INFORMATION IN THE MATERIALS BE USED AS A SUBSTITUTE FOR PROFESSIONAL CARE.

Policy on Faculty Disclosure

It is the policy of the ACCME and APSA that the planning committee and faculty disclose and resolve real or apparent conflicts of interest relating to the content of the educational activity, and also disclose discussions of unlabeled/unapproved uses of drugs or devices during their presentations.

In the case of faculty presentations the following faculty members have disclosed a financial relationship with an industry partner. The relationship was proven not to have an impact on the science presented at this annual meeting. All other faculty indicated that they have no financial relationships to disclose.

Joseph Newsome	Ownership Interest: CoderClass.com, LLC
Robert H. Bartlett	Stockholder/Ownership; MC3, Inc. - Maker of the M-pump, which was used in this study.
Marc Schneider	Stockholder/Ownership; company manufactures the brace.
Joseph R. Moorman	Stockholder/Ownership: Medical Predictive Science Corporation HeRO

Douglas E. Lake	Stockholder/Ownership: Medical Predictive Science Corporation HeRO.
James Holmes	Consulting; Zimmer. Grant/Research Support; Mylan Pharmaceuticals
John Griswold	Grant/Research Support; ReCell

Disclosure forms were provided to and signed by all APSA 2011-2012 committee members. These committee members have reported the following financial relationships and it has been determined that no conflict of interest exists with any of these relationships. All other committee members indicated that they have no financial relationships to disclose.

Arnold G. Coran	Spouse received consulting fee ($4,000) for Surgical Institute Inc.
Charles Cox	Grant/Research Support - Athersys, Celgene ; Consultant - KCI; Ownership Interest - EMIT; Speakers Bureau - CBR
Christopher K. Breuer	Grant/Research Support - Ginze Ltd; Pall Corp. Patents - through Harvard and Yale; one joint patent with Pall Corp.
Daniel Saltzman	Consultant - Botanic Oil Innovations; Ownership/ Interest - Botanic Oil Innovations.
Gerald M. Haase	Ownership Interest: Premier Micronutrient Corp. Entity has no relevance with respect to APSA activities.
Gretchen P. Jackson	Financial/Material Interest: Member of NIH/NLM Literature Selection Technical Committee
J. Duncan Phillips	Consultant: Kimberly-Clark. Speakers Bureau: Fresenius Medical Care. Not receiving any payments.
Jeffrey L. Zitsman	Unlabeled/investigational uses of laparoscopic gastric banding in adolescents.
John Wesley	Consultant: Excelsior Medical Corporation
Kevin P. Moriarty	Financial Interest: Consultant - McKesson Health Solutions
Mark Holterman	Grant/Research Support: $200,000 from Allergan, for patient care only. Plans to discuss unlabeled/investigational commercial products: laparoscopic adjustable gastric band by Allergan
Mark Puder	Consultant: Vernalis. Patent for Omegaven
Mark Wulkan	Grant/Research Support: Allergan Pharmaceuticals. Grant support for a clinical trial, does not receive payments of any kind.

Disclosures

Michael Harrison	Ownership Interest: Magnets-In-Me. Equity Only
Peter Dillon	Consulting and Royalties with Synthes
Romeo Ignacio	Ownership Interest - Abbott Laboratories, Hospira Not relevant.
Thomas H. Inge	Grant/Research Support: Ethicon Endosurgery. Not receiving any direct payments.
Thomas M. Krummel	Salary: CA Water Service Group. Ownership Interest: Visible Productions

Jay & Margie Grosfeld Lecture

Sunday, May 20, 11:15 a.m. – 12:15 p.m.

M. James Kaufman, PhD
Vice President on Public Policy, Children's Hospital Association, Alexandria, VA USA

Health Care Reform – The Impact on Children

Jim Kaufman is vice president for public policy at the Children's Hospital Association (formerly the National Association of Children's Hospitals). The Association supports children's hospitals in addressing public policy issues that affect their ability to serve children and their families. The Association focuses on federal advocacy, policy analysis and communications designed to influence policy makers, both in the White House and within Congress. Each of these efforts focuses on improving children's health through raising awareness of their unique health issues, developing innovative solutions, and effecting change through collaborative action.

Jim's professional experience includes almost twenty years of public policy experience in a range of different organizations. Prior to joining the Association, Jim served as director of government affairs for Johns Hopkins Institutions, which includes both Johns Hopkins University and Johns Hopkins Medicine. Prior to Johns Hopkins, Jim's experience included serving in finance policy for the Maryland Higher Education Commission and budget policy for the Maryland General Assembly.

Jim received his bachelor's degree in Political Science from McDaniel College, his master's degree in Public Administration from the University of Baltimore, and his PhD in Public Policy from the University of Maryland, Baltimore Graduate Schools.

Learning Objectives:

At the conclusion of this lecture, participants will be able to:

- Discuss the latest on health care reform, state exchanges and other new programs and their impact on children's health care
- Discern Medicaid and CHIP's roles as part of health care reform
- Cite the opportunity and challenges health care faces and how the 2012 election may impact children

Robert E. Gross Lecture

Monday, May 21, 9:00 – 10:00 a.m.

Daniel M. Green, MD
Department of Epidemiology, St. Jude's Children's Hospital, Memphis, TN USA

The Evolution of Treatment of Wilms' Tumor

Dr. Daniel M. Green is a faculty member at St. Jude Children's Research Hospital in Memphis, Tennessee. His research interests include the late effects of treatment of children for various malignancies with some emphasis on Wilms' tumor. He has utilized the resources of the Childhood Cancer Survivor Study (CCSS), the National Wilms' Tumor Study Group (NWTSG), of which he was the chair from 1990 to 2000, and St. Jude Children's Research Hospital resources including the St. Jude Lifetime Cohort Study (SJLIFE). The specific outcomes he investigates include fertility, reproductive outcomes, obesity and congestive heart failure.

Dr. Green received his BS from the Massachusetts Institute of Technology and achieved an MD at the St. Louis University School of Medicine. He has served on numerous review boards and panels including the Late Effects of Pediatric Cancer RFA Development Workshop at the Canadian Institute for Health Research, and is a current member of the Pediatric and Adolescent Solid Tumor Steering Committee at the National Cancer Institute.

Learning Objectives

At the conclusion of this lecture, participants will be able to:

- Discuss the historical background for the National Wilms' Tumor Study Group
- Review the results of the National Wilms' Tumor Studies
- Discuss the advantages and disadvantages of pre-nephrectomy chemotherapy for Wilms' Tumor

International Guest Lecture

Monday, May 21, 11:45 a.m. – 12:45 p.m.

Benno M. Ure, MD
Kinderchirurgische Klinik Medizinische Hochschule, Hannover, Germany

Enthusiasm, Evidence and Ethics: the Triple E of Minimally Invasive Pediatric Surgery

Benno Ure, born 1955, is Surgeon-in-Chief and Professor of Pediatric Surgery at Hannover Medical School and the Division Chief of Pediatric Surgery at the Children´s Hospital Bult in Hannover, Germany. He is also the Vice Chief Executive Officer of the University and the Speaker of the University Chairmen. In addition, he is the Vice Chairman of the Scientific Board of DKV, a German Health Insurance.

Benno Ure is board certified in general and pediatric surgery. He is a graduate of the Universities of Vienna and Cologne and received his training in Cologne, Germany. He worked in several African countries and served a consultant at Utrecht University, The Netherlands. His position in Hannover was assumed in 2000.

He serves as board member and scientific chairman of the European Association of Pediatric Surgeons and as board member of the International Pediatric Surgical Research. Benno Ure is the vice president of the International Pediatric Endosurgery Group, overseas council member of the British Association of Pediatric Surgeons and honorary member of numerous international organizations. Since 2001, he is the Editor-in-Chief of the *European Journal of Pediatric Surgery* and serves in the editorial board of numerous other journals.

In addition to his clinical work, Benno Ure is deeply involved in research and is the author of more than 200 journal articles, 50 book chapters and 4 books. His special interests are minimally invasive techniques, neonatal immunology, clinical pathways and ethics.

Benno Ure was the president of a German non-governmental humanitarian organization for many years and served in numerous disaster regions. He was founding member of the European Master Degree of Humanitarian Assistance and is member of the Advisory Board of the Institute for International Law of Peace and Armed Conflict at the University Bochum. Benno Ure also served as Member of the Board of Humanitarian Assistance of the German Ministry of Foreign Affairs.

Learning Objectives:

At the conclusion of this lecture, participants will be able to:

- Review minimally invasive pediatric surgery: from enthusiastic reports to evidence-based medicine
- Discuss the advantages and disadvantages of minimally invasive pediatric surgery today and make recommendations for appropriate indications and future developments will be based on data from clinical trials and experimental research
- Identify the ethical aspects of performing, teaching and reporting on minimally invasive pediatric surgery

Journal of Pediatric Surgery Lecture

Tuesday, May 22, 8:00 – 9:00 a.m.

Brad W. Warner, MD
St. Louis Children's Hospital, St. Louis, Missouri, USA

Adaptation: Paradigm for an Academic Career and the Gut

Dr. Brad W. Warner is currently the Jessie L. Ternberg, MD, PhD, Distinguished Professor of Pediatric Surgery at Washington University School of Medicine and the Surgeon-in-Chief at the St. Louis Children's Hospital. Dr. Warner is a native of St. Louis and completed college and medical school at the University of Missouri-Kansas City School of Medicine's six-year medical program. In 1982, he moved to the University of Cincinnati where he completed a general surgery residency, a two-year research fellowship, and a fellowship in pediatric surgery. He stayed on faculty at the Cincinnati Children's Hospital Medical Center and University of Cincinnati College of Medicine, rising through the ranks to full professor. He has an NIH funded basic science laboratory and has been Program Director for the Pediatric Surgery Fellowship in Cincinnati, as well as Director of Surgical Research.

Dr. Warner is past-president of the Society of University Surgeons and past Chairperson of the Section on Surgery of the American Academy of Pediatrics. He is currently on the Board of Governors of American Pediatric Surgical Association. Dr. Warner came to St. Louis Children's Hospital/Washington University in 2007 where he maintains an active laboratory staffed with multiple surgical residents, PhDs, and pre- and postdoctoral students. Dr. Warner's research and clinical interests include intestinal adaptation, short gut syndrome, ulcerative colitis and advanced gastrointestinal surgery.

Learning Objectives:

At the conclusion of this lecture, participants will be able to:

- Describe intestinal adaptation responses
- Identify the various cell types contributing to adaptation
- Discuss pitfalls associated with basic science research

IPSO

The International Society of Paediatric Surgical Oncology (IPSO) is an international society of surgeons who specialize in the surgical care of children with cancer. IPSO's aims are: to set a global standard for surgical care of children with cancer; to provide a forum and enhance communication between surgeons who specialize in children's cancer; to promote and support clinical trials aimed at improving the outcome in the treatment of children's cancer; and to encourage cooperation with other organizations concerned with children's cancer.

APSMA

The American Pediatric Surgery Managers Association (APSMA) was established to advance the administration and management of health care in academic, hospital and private pediatric surgical entities; to promote the concept of professional management in pediatric surgical administration; and to enhance the continuing educational process for pediatric surgical administrators through sponsorship of meetings, seminars, written communications, and much more.

APSA 43RD ANNUAL MEETING
PROGRAM IN DETAIL

Saturday, May 19, 2012

8:00 a.m. – 8:15 a.m.	Welcome: APSA and IPSO Presidents	*Exhibit Ballroom Salon A*
8:15 – 10:00 a.m.	**APSA-IPSO SYMPOSIUM** **Neuroblastoma Session**	*Exhibit Ballroom Salon A*

Moderators:
Roly Squire, MD; Michael P. LaQuaglia, MD

New Developments in Neuroblastoma Biology
Yael Mosse, MD

International Neuroblastoma Staging System (INRGSS)
Roly Squire, MD

The Role of Surgery in High-Risk Neuroblastoma
Keith Holmes, MD; Michael LaQuaglia, MD

The Role of Surgery in Intermediate Risk Neuroblastoma
Roly Squire, MD

Neonatal Expectant Observation Study
Jed G. Nuchtern, MD

Learning Objectives:
- Update the surgeon on new developments in neuroblastoma biology
- Update surgeons on the INRG staging system
- Define the role of surgery in high and intermediate-risk neuroblastoma
- Update the surgeon on the developing role of observation alone in selected neuroblastoma patients

10:00 – 10:30 a.m.	Refreshment Break	*Exhibit Ballroom Foyer*
10:30 a.m. – Noon	**Scientific Session I** **Oncology**	*Exhibit Ballroom Salon A*

Moderators:
Jan Godzinski, MD; Daniel von Allmen, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:
- Describe the characteristics of invasive neuroblastoma
- Cite the recent changes and challenges with primary surgery for renal tumors
- Cite the evidence for nephron sparing surgery in Wilms' tumor

1 **PRIMARY NEPHRECTOMY AND INTRA-OPERATIVE TUMOR RUPTURE: REPORT FROM THE CHILDREN'S ONCOLOGY GROUP (COG) RENAL TUMORS COMMITTEE**

Peter F. Ehrlich[1], Ken Gow, MD[2], Douglas C. Barnhardt, MD[3], Jessica Kandel, MD[4], Tom Hamilton, MD[5], Mike Chen, MD[6], Mitchell Price[7], Arlene Naranjo, MD[8], Elizabeth Perlman, MD[9], Jeff Dome, MD[10], Elizabeth Mullen[11].

[1]University of Michigan, Ann Arbor, MI, USA, [2]University of Washington, Washington, WA, USA, [3]Primary Children's Hospital, Salt Lake City, UT, USA, [4]Columbia University, New York City, NY, USA, [5]Boston Children's Hospital, Boston, MA, USA, [6]University of Alabama, Birmingham, AL, USA, [7]University of Illinois, Chicago, IL, USA, [8]University of Florida, Ganesville, FL, USA, [9]Childrens Memorial, Chicago, IL, USA, [10]Childrens National Medical Center, Washington, DC, USA, [11]Boston Children's, Boston, MA, USA.

2 **CLINICAL AND MOLECULAR CHARACTERISTICS OF INVASIVE NEUROBLASTOMA**

Kappler Roland, PhD, Nathalie Kremer, MD, Josef Mueller-Hoecker, MD, PhD, Maximilian Stehr, MD, PhD, Dietrich G. von Schweinitz, MD, PhD.

University of Munich, Munich, Germany.

3 **TREATMENT, OUTCOME AND SURGICAL TREATMENT FAILURES IN PATIENTS SUFFERING FROM PARATESTICULAR RHABDOMYOSARCOMA TREATED WITHIN THE COOPERATIVE SOFT TISSUE SARCOMA TRIALS CWS-86, -91-, -96, AND -2002P**

Guido Seitz, MD[1], Joerg Fuchs, MD[1], Tobias Dantonello, MD[2], Daniel Kosztyla[2], Thomas Klingebiel, MD[3], Ewa Koscielniak, MD.[2]

[1]University Children`s Hospital, Tuebingen, Germany, [2]Olgahospital, Stuttgart, Germany, [3]University Children`s Hospital, Frankfurt/Main, Germany.

4 **HIF-1α ACTIVATION MEDIATES RESISTANCE TO ANTIANGIOGENIC THERAPY IN NEUROBLASTOMA XENOGRAFTS**

Joseph E. Hartwich, MD[1], W. Shannon Orr, MD[2], Catherine Y. Ng, MS[3], Yunyu Spence, PhD[3], Christopher Morton, BS[3], Andrew M. Davidoff, MD[3].

[1]Virginia Commonwealth University, Richmond, VA, USA, [2]University of Tennessee Health Science Center, Memphis,TN, USA, [3]St Jude Children's Research Hospital, Memphis, TN, USA.

5 **mTOR INHIBITION PROMOTES OSTEOPROTEGERIN PRODUCTION, DELAYING TIME TO PATHOLOGIC FRACTURE IN AN ORTHOTOPIC XENOGRAFT MODEL OF NEUROBLASTOMA BONE METASTASIS**

Joseph E. Hartwich, MD[1], W. Shannon Orr, MD[2], Catherine Y. Ng, MS[3], Yunyu Spence, PhD[3], Wayne Furman, MD[3], Lisa M. McGregor, MD, PhD[3], Andrew M. Davidoff, MD[3].

[1]Virginia Commonwealth University, Richmond, VA, USA, [2]University of Tennessee Health Science Center, Memphis, TN, USA, [3]St Jude Children's Research Hospital, Memphis, TN, USA.

6 **HEPATOCELLULAR CARCINOMA IN CHILDREN AND ADOLESCENTS: THE INTERNATIONAL THERAPEUTIC EXPERIENCE**

Marcio H. Malogolowkin[1], Piotr Czauderna[2], Beate Haberle[3], Eiso Hiyama[4], Irene Schmid[3], Mark Krailo[5], Rudolf Maibach[6], Howard Katzenstein[7], Giorgio Perilongo[8], Daniel C. Aronson[9], Dietrich VonSchweinitz[3], Rebecka L. Meyers[10].

[1]Childrens Hospital Los Angeles, Los Angeles, CA, USA, [2]Medical University of Gdansk, Gdansk, Poland, [3]Children`s Hospital of the Ludwig-Maximilians-University, Munich, Germany, [4]Hiroshima University Hospital, Hiroshima, Japan, [5]University of Southern California Keck School of Medicine, Los Angeles, CA, USA, [6]International Breast Cancer Study Group, Bern, Switzerland, [7]Children's Healthcare of Atlanta, Emory University, Atlanta, GA, USA, [8]University Hospital of Padua, Padua, Italy, [9]Altona Children's Hospital/University Medical Center Hamburg-Eppendorf, Hamburg, Germany, [10]University of Utah, Salt Lake City, UT, USA.

7 **UPFRONT BIOPSY FOR UNILATERAL PEDIATRIC RENAL TUMORS: AN INCREASING PROBLEM?**

Peter F. Ehrlich[1], Douglas C. Barnhart, MD[2], Ken Gow, MD[3], Tom Hamilton, MD[4], Jessica Kandel, MD[5], Mike Chen, MD[6], Elizabeth Mullen[7], Paul Grundy[8], Jeff Dome, MD[9], Paul Grundy[8].

[1]University of Michigan, Ann Arbor, MI, USA, [2]Primary Children's Hospital, Primary Children's Hospital Salt Lake City, UT, USA, [3]Seattle Children's Hospital, University of Washington Seattle, WA, USA, [4]Boston Children's Hospital, Boston, MA, USA, [5]Columbia University, New York City, NY, USA, [6]University of Alabama Birmingham, Birmingham, AL, USA, [7]Boston Children Hospital, Boston, MA, USA, [8]University of Alberta, Edmonton, AB, Canada, [9]Childrens National Medical Center, Washington, DC, USA.

8 **NEPHRON SPARING SURGERY (NSS) FOR UNILATERAL WILMS' TUMOR (uWT) THE SIOP 2001 EXPERIENCE**

J.C.H. Wilde, MD[1], Daniel C. Aronson, MD, PhD[1], Beate Sznajder[2], Harm van Tinteren[2], MarkPowis[3], Bruce Okoye[4], Giovanni Cecchetto[5], Georges Audry[6], Jörg Fuchs[7], Dietrich von Schweinitz[8], Hugo A. Heij[1], Norbert Graf[9], Christophe Bergeron[10], Kathy Pritchard-Jones[11], Marry van den Heuvel-Eibrink[12], Modesto Carli[5], Foppe Oldenburger[13], Bengt Sandstedt[14], Jan de Kraker[1], Jan Godzinski[15].

[1]Emma Children's Hospital AMC, Amsterdam, Netherlands, [2]Netherlands Cancer Institute – AVL, Amsterdam, Netherlands, [3]Leeds Teaching Hospitals Trust, Leeds, United Kingdom, [4]St. George's Healthcare NHS Trust, London, United Kingdom, [5]University-Hospital of Padua, Padua, Italy, [6]Hôpital Trousseau, Paris, France, [7]Children's Hospital, University of Tübingen, Tübingen, Germany, [8]Dr von Hauner Children's Hospital, Ludwig-Maximilians-University, Munich, Germany, [9]University of Homburg, Saar, Germany, [10]Institut d'Hémato-Oncologie Pédiatrique, Lyon, France, [11]Institute of Child Health & Great Ormond Street Hospital, London, United Kingdom, [12]Sophia Children's Hospital, ErasmusMC, Rotterdam, Netherlands, [13]Academic Medical Center, Amsterdam, Netherlands, [14]Childhood Cancer Research Unit, Karolinska Institutet, Astrid Lindgren Children´s Hospital, Stockholm, Sweden, [15]Marciniak Hospital, Wroclaw and Medical University, Chair of Emergency, Wroclaw, Poland.

9 **SIGNIFICANCE OF IMAGE-DEFINED RISK FACTORS (IDRFs) FOR SURGICAL TREATMENT OF NEUROBLASTOMAS**

Maximilian Stehr, MD, PhD, Moritz Erichsen, Florian Bergmann, MD, Dietrich von Schweinitz, MD, PhD.

Department of Pediatric Surgery, Ludwig-Maximilians-Universität, Munich, Germany.

10 **MARGIN STATUS DOES NOT IMPACT TUMOR RECURRENCE AFTER NEPHRON-SPARING SURGERY FOR BILATERAL WILMS' TUMOR**

Kathleen Kieran, MD[1], Mark A. Williams, MD[2], Jeffrey S. Dome, MD[3], Matthew J. Krasin, MD[4], Andrew M. Davidoff, MD[4].

[1]University of Iowa, Iowa City, IA, USA, [2]University of Tennessee/LeBonheur Children's Hospital, Memphis, TN, USA, [3]Children's National Medical Center, Washington, DC, USA, [4]St Jude Children's Research Hospital, Memphis, TN, USA.

11 **SYSTEMIC INHIBITION OF TIE2 SIGNALING ENHANCES SURVIVAL IN A EWING'S SARCOMA TUMOR MODEL**

Ari Reichstein, MD, Alejandro Garcia, MD, Yan Jun Chang, BS, Matthew Leskowitz, BA, Sonia L. Hernandez, PhD, Jianzhong Huang, MD, Darrell Yamashiro, MD, PhD, Jessica Kandel, MD.

Morgan Stanley Children's Hospital, Columbia University Medical Center, New York, NY, USA.

Noon – 1:00 p.m.	Lunch (individual arrangements)	
1:00 – 3:00 p.m.	**Liver Tumors Session** **Focus on Hepatoblastoma**	*Exhibit Ballroom* *Salon A*

Moderators:
Daniel C. Aronson, MD; Rebecka L. Meyers, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:

- Discuss the risk stratification, PRETEXT, treatment strategies and outcomes from current hepatoblastoma multicenter clinical trials around the world (SIOPEL, COG, GPOH, and JPLT)
- Describe recent advances in chemotherapy, and new findings in genetic and histologic predictors of poor prognosis
- Contrast risk and benefits of aggressive resection vs. liver transplantation for children with locally advanced tumors

State of the Art: Pretext, Multicenter Studies and Collaboration
Dolores Lopez-Terrand, MD; Piotr Czauderna, MD

Predicting Prognosis and Biological Markers
Erso Hiyama, MD; Beate Haeberle, MD

Surgical Management
Gregory M. Tiao, MD; Riccardo A. Superina, MD; Jean de Ville de Goyet, MD

High Risk Tumors
Marcio Malogowkin, MD

3:00 – 3:30 p.m.	Refreshment Break	*Exhibit Ballroom Foyer*
3:30 – 5:00 p.m.	**Spectacular/Challenging Cases Session**	*Exhibit Ballroom Salon A*

Moderators:
Daniel C. Aronson, MD; Andrew M. Davidoff, MD

Sunday, May 20, 2012

ASPA-IPSO Symposium in Conjunction with APSA Education Day

8:00 – 11:00 a.m.	**Education Session I** **Management of Metastatic Disease**	*Cibolo Canyon Salons 5-11*

Moderators:
Jan Godzinski, MD; Peter Mattei, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:

- Discuss the various diagnostic and therapeutic options available in the care of children with metastases
- Describe the accepted indications for sentinel lymph node biopsy in children and the important technical aspects of performing the procedure
- Identify the basic principles of hyperthermic intraperitoneal chemotherapy and discuss the potential usefulness of this novel technique in children with cancer

Tactics of Surgical Treatment in Metastatic Patients
Giovanni Cecchetto, MD

Sentinel Lymph Node Biopsy
Kenneth W. Gow, MD

Surgery of Distant Metastases-Standard and Innovative Techniques
Joerg Fuchs, MD

Hyperthermic Intraperitoneal Chemotherapy
Andrea A. Hayes-Jordan, MD

11:00 – 11:15 a.m.	Refreshment Break	*Cibolo Canyon Foyer*
11:15 a.m. – 12:15 p.m.	**Jay and Margie Grosfeld Lecture** M. James Kaufman, PhD **Healthcare Reform —** **The Impact on Children**	*Cibolo Canyon* *Salons 5-11*

Learning Objectives:
At the conclusion of this lecture, participants will be able to:
- Discuss the latest on health care reform, state exchanges and other new programs and their impact on children's health care
- Discern Medicaid and CHIP's roles as part of health care reform
- Cite the opportunity and challenges health care faces and how the 2012 election may impact children

12:30 – 2:00 p.m.	Lunch Session — TBA	*Cibolo Canyon* *Salons 5-11*
2:00 – 4:00 p.m.	**Concurrent Sessions** **Education Session II** **Joint Session with APSMA:** **Billing and Coding Update**	*Cibolo Canyon* *Salons 5-11*

Moderator:
J. Duncan Phillips, MD

Coding, Billing, and Other "Hot Topics" in Pediatric Surgery Practice: Everything You Think You Already Know (but really could know better)
Donald B. Shaul, MD; Phyllis Blackman, MBA; Joseph Newsome; Mustafa H. Kabeer, MD; J. Duncan Phillips, MD; James C. Gilbert, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:
- State the differences between CPT, ICD-9, RVUs, RBRVS and the RUC
- Understand and discuss the substance of and relationship between the National Correct Coding Initiative and the Patient Protection and Affordable Care Act
- Identify what role Pediatric Surgeons play in Accountable Care Organizations (ACOs) and what it means for health care to be "Vertically Integrated"
- Cite the start date for ICD-10 and what the major changes will be
- Identify some of the most common coding issues and concerns facing pediatric surgeons

2:00 – 4:00 p.m.	**Education Session III** **Basic Science Update**	*Cibolo Canyon* *Salons 1-4*

Moderator:
Christopher K. Breuer, MD

Translational Research
Jay P. Vacanti, MD; Christopher K. Breuer, MD; Gail E. Besner, MD; Anthony D. Sandler, MD; Michael R. Harrison, MD; N. Scott Adzick, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:
- Define translational research and its relevance to practice of pediatric surgery
- Develop an appreciation for basic science research as the foundation for rational design and the basis for advancing our discipline
- Understand and discuss the regulatory process and the importance of quality control and quality assurance measures to insure patient safety
- Describe some of the more common challenges that slow down the process of translation
- Apply some of the strategies for accelerating translational research
- Cite examples of how translational research is changing the practice of pediatric surgery

---

4:15 – 6:00 p.m.	**Concurrent Poster Sessions** **Poster Session I** **Basic Science**	*Cibolo Canyon* *Salons 5-11*

Moderators:
Ai-Xuan Holterman, MD; J. Ted Gerstle, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:
- Understand and explain the mediating and protective factors for intestinal disease in the newborn
- Describe the potential roles of stems cells in modulating disease processes
- Describe a feature(s) of the electronic health record that improves perioperative performance
- Cite the risk benefits of neonatal hemodialysis

P1 **THE ROLE OF NOTCH INHIBITION IN A NOVEL HEPATOBLASTOMA ORTHOTOPIC MODEL**
Alejandro Garcia, MD, Roderick Alfonso, BS, Angela Kadenhe-Chiweshe, MD, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD.
Columbia University, New York, NY, USA.

P2 **LACTOCOCCUS LACTIS ATTENUATES INTESTINAL DAMAGE AND IMPROVES SURVIVAL IN A PRETERM RABBIT MODEL OF INTESTINAL INJURY**

Andrew P. Bozeman, MD, Melvin S. Dassinger, MD, Rhea J. Birusingh, MD, Samuel D. Smith, MD.

University of Arkansas for Medical Sciences, Arkansas Children's Hospital, Little Rock, AR, USA.

P3 **NEUREGULIN-4 IS PROTECTIVE AGAINST EXPERIMENTAL NECROTIZING ENTEROCOLITIS IN A RAT MODEL**

Shannon L. Castle, MD, Anatoly V. Grishin, PhD, Henri R. Ford, MD, MHA, Mark R. Frey, PhD.

Children's Hospital Los Angeles, Los Angeles, CA, USA.

P4 **INTESTINAL ALKALINE PHOSPHATASE ADMINISTRATION IN NEWBORNS DECREASES INOS EXPRESSION IN A NEONATAL NECROTIZING ENTEROCOLITIS RAT MODEL**

Rebecca M. Rentea, MD, Scott R. Welak, MD, Katherine M. Friedrich, MD, Kirkwood A. Pritchard, PhD, Keith T. Oldham, MD, David M. Gourlay, MD.

Medical College of Wisconsin, Milwaukee, WI, USA.

P5 **AN *IN VIVO* MODEL OF HUMAN-DERIVED FIBROUS HAMARTOMA OF INFANCY**

Fabienne L. Gray, MD, Azra Ahmed, BS, Christopher G. Turner, MD, MPH, Yuin-Han Loh, PhD, Alexander Devine, BS, Odelya Hartung, BS, David Zurakowski, PhD, George Q. Daley, MD, PhD, Dario O. Fauza, MD.

Children's Hospital Boston, Boston, MA, USA.

P6 **OSTEOPONTIN BLOCKADE WITH AN RNA APTAMER RESULTS IN REDUCTION OF EPITHELIAL-MESENCHYMAL TRANSITION AND PULMONARY METASTATIC BURDEN IN A XENOGRAFT MODEL OF OSTEOSARCOMA**

Lindsay J. Talbot, MD[1], Zhiyong Mi, PhD[2], Syamal D. Bhattacharya, MD[1], Hongtao Guo, MD, PhD[1], Henry E. Rice, MD[1], Paul C. Kuo, MD, MBA[2].

[1]Duke University, Durham, NC, USA, [2]Loyola University, Chicago, IL, USA.

P7 **THE FETAL ORIGIN OF INFLAMMATORY MEDIATORS IN GASTROSCHISIS**

Cerine Jeanty, MD[1], Sheila Keating, PhD[2], Geoanna Bautista, BS, MA[1], Amar Nijagal, MD[1], Shinjiro Hirose, MD[1], CJ Kim, MD, PhD[3], Roberto Romero, MD[3], Philip Norris, MD[1], Mike Busch, MD, PhD[2], Tippi MacKenzie, MD[1].

[1]University of California, San Francisco, San Francisco, CA, USA, [2]The Blood Systems Research Institute, San Francisco, CA, USA, [3]Perinatology Research Branch, NICHD/NIH/DHHS, Detroit, MI, USA.

P8 **GENERATION OF PERINATAL INDUCED PLURIPOTENT STEM (iPS) CELLS ON A DEFINED, NON-XENOGENIC POLYMER SUBSTRATE**

Guihua Jiang, MS, Luis G. Villa-Diaz, PhD, Cynthia DeLong, PhD, K. Sue O'Shea, PhD, Paul H. Krebsbach, DDS, PhD, Shaun M. Kunisaki, MD, MSc.

University of Michigan, Ann Arbor, MI, USA.

P9 **HUMAN INDUCED PLURIPOTENT STEM CELL-DERIVED NEURAL CREST STEM CELLS INTEGRATE INTO THE INJURED SPINAL CORD IN THE FETAL LAMB MODEL OF MYELOMENINGOCELE**

Payam Saadai, MD[1], Aijun Wang, PhD[2], Yvette Nout, DVM, PhD[1], Timothy L. Downing[2], Katrine Lofberg, MD[1], Michael S. Beattie, PhD[1], Jacqueline C. Bresnahan, PhD[1], Song Li, PhD[2], Diana L. Farmer, MD[1].

[1]University of California, San Francisco, CA, USA, [2]University of California, Berkeley, CA, USA.

P10 **HAEMATOPOIETIC STEM CELLS DERIVED BOTH FROM SHEEP AND HUMAN AMNIOTIC FLUID ENGRAFT AFTER TRANSPLANTATION: POTENTIAL FOR *IN UTERO* AUTOLOGOUS GENE/CELL THERAPY**

Steven Shaw[1], Anna L. David[2], Mike Blundell[3], Kuang-Han Lee[2], Steven Howe[3], Caterina Pipino[1], Panagiotis Maghsoudlou[1], Jane Lin[1], Anthony Atala[4], Simon Eaton[1], Agostino Pierro[1], Christopher D. Porada[4], Adrian Thrasher[3], Paolo De Coppi[1].

[1]Surgery Unit, ICH, University College London, London, United Kingdom, [2]Institute for Women's Health, University College London, London, United Kingdom, [3]Molecular Immunology Unit, ICH, University College London, London, United Kingdom, [4]Wake Forest Institute for Regenerative Medicine, North Carolina, NC, USA.

P11 ***IN UTERO* TRANSPLANTATION FOLLOWED BY POSTNATAL ENGRAFTMENT CAN BE ACHIEVED WITH PRIMARY ADULT HUMAN HEPATOCYTES IN PIGS**

James Fisher, MD[1], Joseph Lillegard, MD, PhD[1], Travis Mckenzie, MD[1], Peter Wettstein, PhD[1], Markus Grompe, MD[2], Brad Feltis, MD, PhD[3], Scott Nyberg, MD, PhD[1].

[1]Mayo Clinic - Rochester, Rochester, MN, USA, [2]Oregon Health and Sciences University, Portland, OR, USA, [3]Childrens Hospitals and Clinics of Minnesota, Minneapolis, MN, USA.

P12 **INTRA-AMNIOTIC DELIVERY OF EXPANDED AMNIOTIC-DERIVED NEURAL PROGENITOR CELLS IN A SYNGENEIC MODEL OF SPINA BIFIDA**

Christopher G. Turner, MD, MPH[1], Elliot C. Pennington, MD[1], Fabienne L. Gray, MD[1], Azra Ahmed, BS[1], Yang D. Teng, MD, PhD[2], Dario O. Fauza, MD[1].

[1]Children's Hospital Boston, Boston, MA, USA, [2]Brigham and Women's Hospital, Boston, MA, USA.

P13 **NONINVASIVE MEASUREMENTS OF CARDIAC HEMODYNAMIC AND TISSUE PERFUSION INDICES IN NORMAL INFANTS**

Eric L. Long, MD[1], Barbara Weaver, RN[1], Joshua Glenn, MD[1], Robert Vogel, PhD[2], Andrew Bozeman, MD[1], Brandon Lerner, BA[1], Renee Kleris, BA[1], Joseph Van de Water, MD[1], Don K. Nakayama, MD[1], Misael Rodriguez, MD[1].

[1]Medical Center of Central Georgia, Macon, GA, USA, [2]Georgia Southern University, Statesboro, GA, USA.

P14 **RENAL REPLACEMENT THERAPY IN NEWBORNS: IS THE HEROIC EFFORT WARRANTED?**

Lena Perger, MD[1], Reed Dimmit, MD[2], Carroll M. Harmon, MD, PhD[3].

[1]Department of Surgery, Scott & White Hospital, Texas A&M College of Medicine, Temple, TX, USA, [2]Department of Pediatrics, University of Alabama at Birmingham, Childrens Hospital of Alabama, Birmingham, AL, USA, [3]Department of Surgery, University of Alabama at Birmingham, Children's Hospital of Alabama, Birmingham, AL, USA.

P15 **TEMPORARY STIMULATION ALLOWS EXPANSION OF INDICATIONS FOR GASTRIC ELECTRICAL STIMULATION**

Jillian McLaughlin, BS[1], Archana Kedar, MD[2], Christopher D. Jolley, MD[1], Tom Abell, MD[2], Saleem Islam, MD, MPH[1].

[1]University of Florida, Gainesville, FL, USA, [2]University of Mississippi, Jackson, MS, USA.

P16 **IMPROVING PERIOPERATIVE PERFORMANCE: THE USE OF THE ELECTRONIC HEALTH RECORD AND OPERATIONS MANAGEMENT**

Robert P. Foglia, MD[1], Adam C. Alder, MD FACS[1], Gardito Ruiz, MHA[2].

[1]UT Southwestern, Dallas, TX, USA, [2]Children's Medical Center Dallas, Dallas, TX, USA.

4:15 – 6:00 p.m. **Poster Session II** *Cibolo Canyon*
Clinical *Salons 1-4*

Moderator:
David A. Rodeberg, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:
- Describe the prognostic factors and management of rare pediatric solid tumors
- Describe an innovative technique for abdominal wall closure
- Explain the importance of ethanol in preventing central line infections
- Describe the epidemiology of firearm fatalities in children and adolescents

P17 **REDUCTION IN FREQUENCY OF ETHANOL LOCK THERAPY RESULTS IN A MARKED INCREASE IN CENTRAL VENOUS ACCESS DEVICE(CVAD) SEPSIS: FDA-INITIATED MANDATES OR A TRAGEDY IN THE MAKING?**

Matthew W. Ralls, MD, R. Alex Blackwood, MD, Meghan A. Arnold, MD, Pamela I. Brown, MD, Luisa Partipilo, Pharm D BCNSP, James Dimond, Daniel H. Teitelbaum, MD.

University of Michigan, Ann Arbor, MI, USA.

P18 **INCREASING AGE AT TIME OF PECTUS EXCAVATUM REPAIR IN CHILDREN: EMERGING CONSENSUS?**

Dominic J. Papandria, MD, Gezzer Ortega, MD, Jose H. Salazar-Osuna, MD, Jeffrey Lukish, MD, Paul Colombani, MD, MBA, Fizan Abdullah, MD, PhD.

Johns Hopkins University School of Medicine, Baltimore, MD, USA.

P19 **VENTRICULOPERITONEAL SHUNT FAILURE AND ABDOMINAL SURGERY: EFFECT OF ABDOMINAL SURGERY ON OUTCOME**

Angela Li Ching Ng[1], Gillian Humphrey, MD, MBChB, FRCS (paedsurg)[2], Ian Kamaly, MD, MBChB, FRCS (SN)[2], Muhammad Baath, MD, MBChB, FRCS[2].

[1]University Hospital South Manchester, Manchester, United Kingdom, [2]Royal Manchester Children's Hospital, Manchester, United Kingdom.

P20 **RESULTS OF SURGICAL PORTAL SYSTEMIC SHUNT FOR SEVERE PORTAL HYPERTENSION IN CHIDREN WITH BILIARY ATRESIA AFTER A SUCCESSFUL KASAI PROCEDURE**

Florent Guerin, MD[1], Stephanie Jaszienski, MD[1], Lionel Charre, MD[2], Frédéric Gauthier, PhD[1], Virginie Fouquet, MD[1], Hélène Martelli, PhD[1], Sophie Brachereau, MD[1].

[1]Bicêtre Hospital, Le Kremlin Bicêtre, France, [2]René Dubos Hospital, Pontoise, France.

P21 **DESMOID FIBROMATOSIS IN CHILDREN AND ADOLESCENTS: A CONSERVATIVE APPROACH TO MANAGEMENT**

Joshua N. Honeyman, Till-Martin Theilen, Molly Knowles, Margaret M. McGlynn, Eric J. Stanelle, Emily R. Christison-Lagay, Paul A. Meyers, Michael P. La Quaglia.

Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

P22 **A FALL IN TRANSITIONAL ZONE PULLTHROUGHS IN HIRSCHSPRUNG'S DISEASE FOLLOWING A CHANGE IN PRACTICE - COMPLETION OF AN AUDIT CYCLE**

Amiria Catherine Lynch, MBChB, DCH, FRACS, Victoria A. Lane, MBChB, MRCS, Ian D. Sugarman, MB ChB, FRCS (Ed), FRCS (Paed).

Leeds Teaching Hospital NHS Trust, Leeds, United Kingdom.

P23 **THE EFFECTIVENESS OF LAPAROSCOPIC NEEDLE-ASSISTED INGUINAL HERNIA REPAIR (LNAR) IN YOUNG CHILDREN**

Christian J. Streck, MD, Aaron P. Lesher, MD, Robert A. Cina, MD, Andre Hebra, MD.

Medical University of South Carolina, Charleston, SC, USA.

P24 **TEMPORAL RELATIONSHIPS BETWEEN POSITIVE URINE CULTURE AND ONSET OF NECROTIZING ENTEROCOLITIS**

Syamal D. Bhattacharya[1], Christoph P. Hornik, MD[1], Abigail Martin, MD[1], Clark Reese, MD[2], C.M. Cotten, MD, MHS[1], Margarita Bidegain, MD[1], Phillip B. Smith, MD, MHS[1].

[1]Duke University Medical Center, Durham, NC, USA, [2]Duke University Medical Center, Sunrise, FL, USA.

P25 **INNOVATIVE TECHNIQUE FOR COVERAGE OF NEONATAL GIANT ABDOMINAL WALL DEFECTS**

Alan Coleman, MD[1], Thomas McGill, MD[1], Ruth Gard, RN, BSN, CCRC[1], James Holmes, MD[2], John Griswold, MD[1].

[1]Texas Tech University HSC, Lubbock, TX, USA, [2]Wake Forest University, Salem, NC, USA.

P26 **EPIDEMIOLOGY OF 577 PEDIATRIC FIREARM FATALITIES: A 2-YEAR REVIEW OF THE NATIONAL TRAUMA DATA BANK (NTDB)**

Tolulope A. Oyetunji, MD, MPH[1], Adil H. Haider, MD, MPH[2], Augustine C. Obirieze, MD, MPH[1], Michael Fisher, BS[1], Aderon'e O. Oyetunji, MD[3], Edward E. Cornwell III, MD[1], Benedict C. Nwomeh, MD, MPH[4].

[1]Howard University College of Medicine, Washington, DC, USA, [2]Johns Hopkins University School of Medicine, Baltimore, MD, USA, [3]University of Baltimore Merrick School Of Business, Baltimore, MD, USA, [4]Ohio State University College of Medicine, Columbus, OH, USA.

P27 **PRIMARY TUMOR LOCATION AND GRADE DETERMINE SURVIVAL IN PEDIATRIC LIPOSARCOMA**

Eric J. Stanelle, MD, Emily R. Christison-Lagay, MD, Samuel Singer, MD, Paul A. Meyers, MD, Michael P. La Quaglia, MD.

Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

P28 **PEDIATRIC SYNOVIAL SARCOMA: PROGNOSTIC FACTORS, MANAGEMENT OF PULMONARY METASTASIS, AND SURVIVAL OUTCOMES**

Eric J. Stanelle, MD, Emily R. Christison-Lagay, MD, Sara J. Abramson, MD, Anita P. Price, MD, John H. Healey, MD, Samuel Singer, MD, Paul A. Meyers, MD, Michael P. La Quaglia, MD.

Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

P29 **OUTCOME AND RISK FACTORS FOR PATIENTS WITH PERINEAL AND PERIANAL RHABDOMYOSARCOMA–A REPORT FROM THE CHILDREN'S ONCOLOGY GROUP SOFT TISSUE SARCOMA COMMITTEE**

Roshni Dasgupta, MD, MPH[1], James Anderson, PhD[2], Andrea Hayes-Jordan, MD[3], Torun Yock, MD[4], Sheri Spunt, MD[5], Doug Hawkins, MD[6], David Rodeberg, MD[7].

[1]Cincinnati Childrens Hospital Medical Center, Cincinnati, OH, USA, [2]University of Nebraska Medical Center, Omaha, NE, USA, [3]MD Anderson Hospital, Houston, TX, USA, [4]Massachusetts General Hospital, Boston, MA, USA, [5]St. Jude's, Memphis, TN, USA, [6]Seattle Childrens Hospital, Seattle, WA, USA, [7]Eastern Carolina University, Greenville, NC, USA.

P30 **FIBROUS SOFT TISSUE TUMORS: INCREASED RECURRENCE RISK FOR DIGITAL AND EXTREMITY LESIONS**

Jennifer H. Aldrink, MD, Kathleen Nicol, MD, Steven Teich, MD.

Nationwide Children's Hospital, Columbus, OH, USA.

P31 **SURGICAL TREATMENT OF PULMONARY METASTASES OF OSTEOSARCOMA: LONG-TERM RESULTS**

Pablo A. Lobos, MD, Julia E. Udaquiola, MD, Germán L. Farfalli, MD, Patricia D. Streitenberger, MD, Gastón R. Elmo, MD, Mauricio M. Urquizo Lino, MD, Luzía Toselli, MD, David Smith, MD.

Hospital Italiano de Buenos Aires, Buenos Aires, Argentina.

P32 **LUNG TO HEAD RATIO IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIAS DOES NOT PREDICT LONG-TERM PULMONARY HYPERTENSION**

Alejandro Garcia, MD, Abbey Fingeret, MD, Eunice Hahn, MD, Matthew Leskowitz, Gudrun Aspelund, MD, MS, Usha Krishnan, MD, Charles JH Stolar, MD.

Columbia University, New York, NY, USA.

P33 **One Horsepower Versus Multiple Horsepower In Pediatric Trauma**

David J. Hobbs, Diana Ropele, RN, James M. DeCou, MD, Helen DeVos.

Children's Hospital, Grand Rapids, MI, USA.

Monday, May 21, 2012

7:30 – 9:00 a.m. **Scientific Session II Clinical: Clinical Trials and Quality Improvement** *Cibolo Canyon Salons 5-11*

Moderators:
Daniel J. Ostlie, MD; Peter F. Ehrlich, MD

12 **AMERICAN COLLEGE OF SURGEONS NATIONAL QUALITY IMPROVEMENT PROGRAM PEDIATRIC: A BETA PHASE REPORT**

Jennifer L. Bruny, MD[1], Bruce L. Hall, MD, PhD, MBA, FACS[2], Douglas C. Barnhart, MD, MSPH[3], Deborah F. Billmire, MD[4], Mark S. Dias, MD[5], Peter W. Dillon, MD, FACS[6], Charles Fisher, BS[7], Kurt F. Heiss, MD, FAAP, FACS[8], William L. Hennrikus, MD[9], Clifford Y. Ko, MD, MS, MSHS, FACS[10], R Lawrence Moss, MD, FACS[11], Keith T. Oldham, MD, FACS[12], Karen E. Richards, BS[7], Rahul K. Shah, MD[13], Charles D. Vinocur, MD, FACS[14], Moritz M. Ziegler, MD, FACS[1].

[1]Division of Pediatric Surgery, Department of Surgery, University of Colorado, Children's Hospital Colorado, Aurora, CO, USA, [2]Division of Research and Optimal Patient Care, ACS, Chicago, IL. Department of Surgery, Washington Univ in St Louis, Barnes Jewish Hospital, Center for Health Policy, The Olin Business School, Washington Univ in St Louis, St Louis Veterans Affairs Med Cen, Saint Louis, IL, USA, [3]Division of Pediatric Surgery, University of Utah, Salt Lake City, UT, USA, [4]Division of Pediatric Surgery, Riley Hospital for Children, Indianapolis, IN, USA, [5]Departments of Neurosurgery and Pediatrics, Pennsylvanian State University School of Medicine, Hershey, PA, USA, [6]Division of Pediatric Surgery, Department of Surgery, Pennsylvania State University School of Medicine, Hershey, PA, USA, [7]Division of Research and Optimal Patient Care, American College of Surgeons, Chicago, IL, USA, [8]Division of Pediatric Surgery, Department of Surgery, Emory University, Atlanta, GA, USA, [9]Department of Orthopedic Surgery, Pennsylvania State University School of Medicine, Hershey, PA, USA, [10]Division of Research and Optimal Patient Care, ACS, Chicago, IL. Department of Surgery, University of California Los Angeles David Geffen School of Medicine and the VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA, [11]Nationwide Children's Hospital The Ohio State University, College of Medicine, Columbus, OH, USA, [12]Department of Surgery, Medical College of Wisconsin, Milwaukee, WI, USA, [13]Division of Otolaryngology, Children's National Medical Center, Washington, DC, USA, [14]Division of Pediatric Surgery, Thomas Jefferson University, A.I. DuPont Hospital for Children, Wilmington, DE, USA.

13 **HOSPITAL QUALITY REPORTING MAY NOT ACCURATELY MEASURE HOSPITAL QUALITY**

Shauna M. Levy, MD[1], Galit Holzmann-Pazgal, MD[1], Kevin P. Lally, MD[1], Koya Davis, MPH[2], Lillian S. Kao, MD[1], KuoJen Tsao, MD[1].

[1]Children's Memorial Hermann Hospital & University of Texas Medical School at Houston, Houston, TX, USA, [2]Children's Memorial Hermann Hospital, Houston, TX, USA.

14 **DISCREPANCIES BETWEEN A PEDIATRIC HEALTH INFORMATION SYSTEM COHORT AND AN INSTITUTIONAL COHORT FOR ESOPHAGEAL ATRESIA**

Jason P. Sulkowski, MD[2], Peter C. Minneci, MD, MHSc[1], Lindsey Asti[1], Katherine J. Deans, MD, MHSc[1].

[1]Nationwide Children's Hospital, Columbus, OH, USA, [2]Children's Hospital of Philadelphia, Philadelphia, PA, USA.

15 **PRIMARY PAYER STATUS IS SIGNIFICANTLY ASSOCIATED WITH POSTOPERATIVE MORTALITY, MORBIDITY, AND HOSPITAL RESOURCE UTILIZATION IN PEDIATRIC SURGICAL PATIENTS WITHIN THE UNITED STATES**

Matthew L. Stone, MD, Damien J. LaPar, MD, MSc, Daniel P. Mulloy, MD, Sara K. Rasmussen, MD, PhD, Bartholomew J. Kane, MD, PhD, Eugene D. McGahren, III, MD, Bradley M. Rodgers, MD.

The University of Virginia, Charlottesville, VA, USA.

16 **TRENDS IN OPERATIVE EXPERIENCE OF NORTH AMERICAN PEDIATRIC SURGICAL RESIDENTS**

Abbey L. Fingeret, MD, Charles J.H. Stolar, MD, Robert A. Cowles, MD.

Division of Pediatric Surgery, Morgan Stanley Children's Hospital of New York-Presbyterian Hospital, Columbia University Medical Center, New York, NY, USA.

17 **FIRST EMPLOYMENT CHARACTERISTICS FOR THE 2011 PEDIATRIC SURGERY FELLOWSHIP GRADUATES**

Charles J.H. Stolar, MD, Gudrun Aspelund, MD, MS.

Morgan Stanley Children's Hospital/Columbia University Medical Center, New York, NY, USA.

18 **VARIATION IN PRACTICE AND RESOURCE UTILIZATION ASSOCIATED WITH THE MANAGEMENT OF INTUSSUSCEPTION AT FREESTANDING CHILDREN'S HOSPITALS**

Samuel E. Rice-Townsend, MD[1], C. Jason Smithers, MD[1], Catherine Chen, MD, MPH[1], Jeff N. Barnes, BS[2], Shawn J. Rangel, MD, MSCE[1].

[1]Children's Hospital Boston, Boston, MA, USA, [2]Child Health Corporation of America, Shawnee Mission, KS, USA.

19 **A PROSPECTIVE STUDY TO DEFINE PEDIATRIC PRESSURE ULCERS AND TO ASSESS A QUALITY IMPROVEMENT BUNDLE IN REDUCING THEIR PREVALENCE**

Alice Leung, MD, Marty O. Visscher, PhD, Ann Marie Nie, RN, WOCN, Sean J. Barnett, MD, Jason S. Frischer, MD, Timothy M. Crombleholme, MD, Sundeep G. Keswani, MD.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA.

20 **PROSPECTIVE COMPARISON OF NON-NARCOTIC VERSUS NARCOTIC OUT-PATIENT ORAL ANALGESIC USE AFTER LAPAROSCOPIC APPENDECTOMY AND EARLY DISCHARGE**

Fuad Alkhoury, MD, Cathy Burnweit, MD, Colin Knight, MD, Steven Stylianos, MD, Jeannette Zerpa, Raquel Pasaron, JoAnne Mora, Alexandra Aserlind, Leopoldo Malvezzi, MD.

Miami Children's Hospital, Miami, FL, USA.

21 **CLINICAL PRACTICE GUIDELINES (CPGS) REDUCE COSTS IN THE MANAGEMENT OF ISOLATED SPLENIC INJURIES AT PEDIATRIC TRAUMA CENTERS**

Ivan M. Gutierrez, MD, David Zurakowski, PhD, Qiaoli Chen, MS, David P. Mooney, MD, MPH.

Children's Hospital Boston, Boston, MA, USA.

22 **LAPAROSCOPIC COMMON BILE DUCT EXPLORATION IN CHILDREN IS ASSOCIATED WITH DECREASED COST, AND LENGTH OF STAY: RESULTS OF A TWO-CENTER ANALYSIS**

Scott S. Short, MD[1], Philip K. Frykman, MD, PhD[1], Nam Nguyen, MD[2], Quin Liu, MD[2], Dror Berel, MS[1], Kasper Wang, MD[2].

[1]Cedars Sinai Medical Center, Los Angeles, CA, USA, [2]Children's Hospital Los Angeles, Los Angeles, CA, USA.

9:00 – 10:00 a.m.	**Robert E. Gross Lecture** Daniel M. Green, MD **The Evolution for Treatment of Wilms' Tumor**	*Cibolo Canyon Salons 5-11*

Learning Objectives:

At the conclusion of this lecture, participants will be able to:

- Discuss the historical background for the National Wilms' Tumor Study Group
- Review the results of the National Wilms' Tumor Studies
- Discuss the advantages and disadvantages of pre-nephrectomy chemotherapy for Wilms' Tumor

10:00 – 10:30 a.m.	Refreshment Break	*Cibolo Canyon Foyer*

10:30 – 11:45 a.m. **Scientific Session III** *Cibolo Canyon*
Fetal/Neonatal and Trauma *Salons 5-11*

Moderators:
Michael A. Helmrath, MD; Terry L. Buchmiller, MD

23 **RECURRENCE AFTER THORACOSCOPIC CONGENITAL DIAPHRAGMATIC REPAIR IN NEONATES AND INFANTS**
Henry Chang, MD, Thomas T. Sato, MD, David M. Gourlay, MD, Casey M. Calkins, MD, Dave R. Lal, MD, John J. Aiken, MD, Keith T. Oldham, MD, Jessica Enters, BSN, Marjorie J. Arca, MD.
Children's Hospital of Wisconsin, Milwaukee, WI, USA.

24 **HEART RATE CHARACTERISTICS INDEX (HERO SCORE) IDENTIFIES NICU PATIENTS WITH SEVERE NECROTIZING ENTEROCOLITIS**
Matthew L. Stone, MD, Bartholomew J. Kane, MD, PhD, Douglas E. Lake, PhD, Joseph R. Moorman, MD, Eugene D. McGahren, III, MD, Bradley M. Rodgers, MD, Karen D. Fairchild, MD.
The University of Virginia, Charlottesville, VA, USA.

25 **EXIT-TO-RESECTION FOR FETUSES WITH LARGE LUNG MASSES AND PERSISTENT MEDIASTINAL COMPRESSION NEAR BIRTH**
Darrell L. Cass, MD, Oluyinka O. Olutoye, MD, PhD, Christopher I. Cassady, MD, Nancy A. Ayres, MD, R. Todd Ivey, MD, Timothy C. Lee, MD, Irving J. Zamora, MD.
Texas Children's Fetal Center and Baylor College of Medicine, Houston, TX, USA.

26 **PULMONARY RADIAL ALVEOLAR COUNT AND VASCULAR MORPHOMETRY AFTER PRENATAL TEMPORARY GEL PLUG OCCLUSION OF THE FETAL TRACHEA IN A RABBIT MODEL OF CONGENITAL DIAPHRAGMATIC HERNIA**
Ramy Elattal, BS, Barrie S. Rich, MD, Oliver J. Muensterer, MD, PhD.
Weill Cornell Medical College, New York, NY, USA.

27 ***EX UTERO* INTRAPARTUM TREATMENT TO EXTRACORPOREAL MEMBRANE OXYGENATION (EXIT-TO-ECMO) STRATEGY FOR SEVERE CONGENITAL DIAPHRAGMATIC HERNIA (CDH)**
Naira Baregamian, Foong-Yen Lim, Sundeep G. Keswani, Jason S. Frischer, Beth Haberman, Paul Kingma, Mounira Habli, Ronald Jaekle, James Van Hook, William J. Polzin, Timothy M.Crombleholme.
Cincinnati Childrens Hospital Medical Center, Cincinnati, OH, USA.

28 **VENOUS THROMBOEMBOLISM AFTER TRAUMA: ARE ADOLESCENTS MORE LIKE CHILDREN OR ADULTS?**
Kyle J. Van Arendonk, MD, Eric B. Schneider, PhD, F. Dylan Stewart, MD, Paul M. Colombani, MD, Elliott R. Haut, MD.
Johns Hopkins University School of Medicine, Baltimore, MD, USA.

29 **FATE OF THE COMBINED ADULT & PEDIATRIC TRAUMA CENTERS: IMPACT OF INCREASED PEDIATRIC TRAUMA REQUIREMENTS**

Kevin N. Johnson, MD[1], Pamela Garcia-Filion, PhD, MPH[2], Melissa Twomey, MS, RN[2], David Notrica, MD[2].

[1]Mayo Clinic, Phoenix, AZ, USA, [2]Phoenix Children's Hospital, Phoenix, AZ, USA.

30 **PROBIOTIC PROPHYLAXIS AFTER PULLTHROUGH FOR HIRSCHSPRUNG'S DISEASE TO REDUCE INCIDENCE OF ENTEROCOLITIS: A PROSPECTIVE, RANDOMIZED, DOUBLE BLIND, PLACEBO CONTROLLED, MULTICENTER TRIAL**

Sabina Siddiqui, MD[1], Mohamed El-Sawaf, MD[2], Moustafa Mahmoud, MD[2], Robert Drongowski, MS[1], Daniel H. Teitelbaum, MD[1].

[1]University of Michigan, Department of Pediatric Surgery, Ann Arbor, MI, USA, [2]Tanta University, Department of Pediatric Surgery, Tanta, Egypt.

31 **CONGENITAL HEART DISEASE AND HETEROTAXY: UPPER GASTROINTESTINAL FLUOROSCOPY IS MISLEADING AND SURGERY IN AN ASYMPTOMATIC PATIENT IS NOT BENEFICIAL**

Stephanie C. Papillon, MD[1], Osnat Zmora, MD[1], Catherine J. Goodhue, MN[1], Shalini S. Sharma, MD, MPH[2], Winfield J. Wells, MD[1], Henri R. Ford, MD[1], Jeffrey S. Upperman, MD[1], Gerald A. Bushman, MD[1], Kasper Wang, MD[1], Richard Kim, MD[1], James R. Pierce, MD[1].

[1]Children's Hospital Los Angeles, Los Angeles, CA, USA, [2]USC Keck School Of Medicine, Los Angeles, CA, USA.

11:45 a.m. – 12:45 p.m. **International Guest Lecture** *Cibolo Canyon Salons 5-11*

Benno M. Ure, MD

Enthusiasm, Evidence and Ethics: The Triple E of Minimally Invasive Pediatric Surgery

Learning Objectives:

At the conclusion of this lecture, participants will be able to:

- Review the introduction and distribution of minimally invasive pediatric surgery: from enthusiastic reports to evidence-based medicine
- Discuss the advantages and disadvantages of minimally invasive pediatric surgery today and make recommendations for appropriate indications and future developments will be based on data from clinical trials and experimental research
- Identify the ethical aspects of performing, teaching and reporting on minimally invasive pediatric surgery.

Tuesday May 22, 2012

8:00 – 9:00 a.m.	***Journal of Pediatric Surgery* Lecture** Brad W. Warner, MD **Adaptation: Paradigm for an Academic Career and the Gut**	*Cibolo Canyon Salons 5-11*

Learning Objectives:
At the conclusion of this lecture, participants will be able to:
- Describe intestinal adaptation responses
- Identify the various cell types contributing to adaptation
- Discuss pitfalls associated with basic science research

9:00 – 10:15 a.m.	**Scientific Session IV** **Basic Science**	*Cibolo Canyon Salons 5-11*

Moderators:
Gail E. Besner, MD; J. Ted Gerstle, MD

32 **RELAXIN: A POTENTIAL VASODILATOR ROLE IN NECROTIZING ENTEROCOLITIS**
Alexandra C. Maki, MD, Laura A. Galganski, BA, Jessica A. Shepherd, BA, Paul J. Matheson, PhD, Richard N. Garrison, MD, Cynthia D. Downard, MD, MMSc.
University of Louisville, Louisville, KY, USA.

33 **DOES EXOGENOUS GLUCAGON-LIKE PEPTIDE-2 IMPROVE CLINICAL OUTCOMES IN NEONATAL PIGLET MODELS OF SURGICAL SHORT BOWEL SYNDROME?**
Megha Suri[1], Justine M. Turner[2], Patrick N. Nation[3], Pamela Wizzard[3], David L. Sigalet[4], Paul W. Wales[1].
[1]The Hospital for Sick Children, Toronto, ON, Canada, [2]Stollery Children's Hospital, Edmonton, AB, Canada, [3] University of Alberta, Edmonton, AB, Canada, [4]Alberta Children's Hospital, Calgary, AB, Canada.

34 **FETAL INTERVENTION TRIGGERS THE ACTIVATION OF PATERNAL ANTIGEN-SPECIFIC MATERNAL T CELLS**
Amar Nijagal, Marta Wegorzewska, Tom Le, Tippi C. MacKenzie, MD.
UCSF, San Francisco, CA, USA.

35 **MESENCHYMAL STEM CELLS (MSC) AND HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) ACT SYNERGISTICALLY TO PROTECT THE INTESTINES FROM EXPERIMENTAL NECROTIZING ENTEROCOLITIS**
Jixin Yang, MD, Daniel Watkins, MD, Chun-Liang Chen, PhD, Hong-Yi Zhang, MD, Yu Zhou, MD, PhD, Markus Velten, MD, Gail E. Besner, MD.
Nationwide Children's Hospital, Columbus, OH, USA.

36 **HUMAN TISSUE-ENGINEERED SMALL INTESTINE FORMS FROM POSTNATAL TISSUE AS A MOUSE XENOGRAFT**
Erik R. Barthel, MD, PhD, Daniel E. Levin, MD, Xiaogang Hou, PhD, Allison L. Speer, MD, Frédéric G. Sala, PhD, Yasuhiro Torashima, MD, PhD, Jamil A. Matthews, MD, Tracy C. Grikscheit, MD.
Children's Hospital Los Angeles, Los Angeles, CA, USA.

37 **METASTATIC POTENTIAL IN NEUROBLASTOMA AFTER DUAL INHIBITION OF VEGF AND NOTCH**
Alejandro Garcia, MD, Debarshi Banerjee, PhD, Sonia Hernadez, PhD, Angela Kadenhe- Chiweshe, MD, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD.
Columbia University, New York, NY, USA.

38 **THE BACTERIAL RECEPTOR TOLL LIKE RECEPTOR 4 (TLR4) REGULATES THE NORMAL RECRUITMENT OF ADAPTIVE IMMUNE CELLS IN THE NEWBORN GUT IN THE PATHOGENESIS OFNECROTIZING ENTEROCOLITIS**
Joyce Y. Lin, MD, Charlotte Egan, PhD, Matthew D. Neal, MD, Chhinder Sodhi, PhD, Ibrahim Yazji, MD, Misty Good, MD, Sapana J. Shah, MD, Amin Afrazi, Hongpeng Jia, MD, Maria Branca, Tom Prindle, Zachary Grant, David J. Hackam, MD, PhD, FACS.
Children's Hospital of Pittsburgh of UPMC, Pittsburgh, PA, USA.

39 **EGENERATION OF ENTERIC GANGLIA IN MECHANICALLY LENGTHENED JEJUNUM**
Rebecca Stark, MD, Elvin Chiang, Ziyad Jabaji, MD, James CY Dunn, MD, PhD.
UCLA, Los Angeles, CA, USA.

40 **AN ALTERNATE DNA REPAIR MECHANISM IN NEUROBLASTOMA**
Erika A. Newman, MD, Daniela Bashilari, BS, Anthony Opipari, MD, PhD, Roland Kwok, PhD, Valerie Castle, MD.
The University of Michigan, Ann Arbor, MI, USA.

10:15 – 10:45 a.m.	Refreshment Break	*Cibolo Canyon Foyer*
10:45 – 11:15 a.m.	**APSA Foundation Scholars** Shaun M. Kunisaki, MD University of Michigan, Ann Arbor, Michigan, USA *Mesenchymal Stem Cell Regulation of Fetal Lung Development in Diaphragmatic Hernia* Cassandra M. Kelleher, MD Massachusetts General Hospital for Children, Massachusetts, USA *Extracellular Components Critical to Alveolarization: Contributions of Elastin*	*Cibolo Canyon Salons 5-11*

11:15 – 11:30 a.m.	Introduction of New Members	*Cibolo Canyon Salons 5-11*
11:30 a.m. – 12:30 p.m.	**Presidential Address** Robert C. Shamberger, MD **Cooperative Group Trials in Pediatric Oncology: The Surgeon's Role**	*Cibolo Canyon Salons 5-11*
12:30 – 12:45 p.m.	Lunch Box Pick-Up	*Cibolo Canyon Foyer*
12:45 – 1:45 p.m.	**Innovation Session** *Abstracts on New and Innovative Techniques and Procedures*	*Cibolo Canyon Salons 5-11*

Learning Objectives:
At the conclusion of this session, participants will be able to:
- Discuss new and innovative mechanical monitoring devices for pediatric surgical problems
- Discuss developing techniques for treatment of congenital disorders
- Describe potential new devices for pediatric surgical care

Moderators:
Peter F. Ehrlich, MD; Christopher K. Breuer, MD

i1 **NON-THERMAL PLASMA: AN INNOVATIVE TECHNOLOGY FOR TUMOR ABLATION**
Ryan M. Walk, MD[1], Jacob Kirsch, BA[2], Priya Srinivasan, PhD[1], Lina Chakrabarti, PhD[1], Jason A. Snyder, MD[1], Felix C. Blanco, MD[1], Michael Keidar, PhD[2], Anthony D. Sandler, MD[1].
[1]Children's National Medical Center, Washington, DC, USA, [2]George Washington University, Washington, DC, USA.

i2 **DEVELOPMENT OF AN ARTIFICIAL PLACENTA V: 70 HOUR VENO-VENOUS EXTRACORPOREAL LIFE SUPPORT AFTER VENTILATORY FAILURE IN PREMATURE LAMBS**
Brian W. Gray, MD, Ahmed El-Sabbagh, MD, Kelly L. Koch, Sara J. Zakem, Alvaro Rojas-Pena, MD, Raja Rabah, MD, Robert H. Bartlett, MD, George B. Mychaliska, MD.
University of Michigan, Ann Arbor, MI, USA.

i3 **TISSUE EXPANDER STIMULATED LENGTHENING OF ARTERIES (TESLA): A NOVEL APPROACH TO THE TREATMENT OF MIDDLE AORTIC SYNDROME**
Heung Bae Kim, MD, Khashayar Vakili, MD, Biren P. Modi, MD, Michael A. Ferguson, MD, Kristina M. Potanos, MD, Steven J. Fishman, MD.
Children's Hospital Boston, Boston, MA, USA.

i4 **PERCUTANEOUS OBLITERATION OF PATENT PROCESSUS VAGINALIS: A RAT MODEL FOR FUTURE INGUINAL HERNIA REPAIR IN CHILDREN**
Saad Al-Qahtani, Yassir Asiri, Ammar Al-Rikabi, Abdulrahman Al-Zahem, Ayman Al-Jazaeri.
College of Medicine, King Saud University, Riyadh, Saudi Arabia.

i5 **DEVELOPMENT OF A NOVEL, CLOSED-SYSTEM, IMPLANTABLE SENSOR TO ASSESS THE MECHANOBIOLOGY OF FETAL LUNG MATURATION**

Mozziyar Etemadi, MS, Samuel C. Schecter, MBBS, Eveline H. Shue, MD, James A. Heller, BS, Shuvo Roy, PhD, Douglas Miniati, MD.

UCSF, San Francisco, CA, USA.

i6 **ROBOIMPLANT (REMOTELY OPERATED BIONIC ORTHO IMPLANT) II: DEVELOPMENT, DESIGN, AND TESTING OF A CONTROLLER FOR NONINVASIVE ACTUATION OF AN IMPLANTED TELESCOPIC ROD USED TO CORRECT STRUCTURAL DEFORMITIES**

Jonathan A. Liu, MS, James A. Heller, BS, Mozziyar Etemadi, MS, Dillon A. Kwiat, BS, Richard Fechter, BS, Shuvo Roy, PhD, Michael R. Harrison, MD.

University of California, San Francisco, CA, USA.

i7 **WIRELESS MONITOR FOR DATA-DRIVEN TREATMENT OF PECTUS CARINATUM**

Jonathan A. Liu, MS[1], Mozziyar Etemadi, MS[1], Marcelo Martinez Ferro, MD[2], Asis Lopez, BS[1], Dillon A. Kwiat, BS[1], Shuvo Roy, PhD[1], Michael R. Harrison, MD[1].

[1]University of California, San Francisco, CA, USA, [2]Fundación Hospitalaria Children's Hospital, Buenos Aires, Argentina.

i8 **DEVELOPMENT OF AN ISOLATION BED FOR PATIENTS UNDERGOING MIBG TREATMENT FOR NEUROBLASTOMA**

Sabina Siddiqui, MD[1], Sean Vance, AIA[2], Laura L. McCormick, PhD[3], Douglas Mullen, PhD[3], Hannah J. Hensel, MBA[3], James D. Geiger, MD[1].

[1]University of Michigan, Department of Pediatric Surgery, Ann Arbor, MI, USA, [2]University of Michigan, School of Art and Architecture, Ann Arbor, MI, USA, [3]University of Michigan, Medical Innovation Center, Ann Arbor, MI, USA.

1:45 – 2:45 p.m. **Video Session** *Cibolo Canyon Salons 5-11*

Moderators:
Joel Shilyansky, MD; Wallace W. Neblett, MD

Learning Objectives:
At the conclusion of this session, participants will be able to:
- Describe minimally invasive approach to diaphragmatic eventration
- Define principles of operative management of cloacal exstrophy in the newborn
- Identify laparoscopic treatment for celiac artery compression
- Cite thoracoscopic treatment for congenital esophageal stenosis

V1 **LAPAROSCOPIC RESECTION OF A TYPE IV CHOLEDOCHAL CYST COMBINED WITH PANCREATIC HEAD RESECTION IN A 5-YEAR-OLD GIRL**

Hans Joachim Kirschner, MD[1], Guido Seitz, MD[1], Dietmar Stueker, MD[2], Juergen Schaefer, MD[3], Joerg Fuchs[1].

[1]Department of Pediatric Surgery and Pediatric Urology, University Children`s Hospital, Tuebingen, Germany, [2]University Department of General, Visceral and Transplant Surgery –Surgical Endoscopy, University of Tuebingen, Tuebingen, Germany, [3]Department of Diagnostic and Interventional Radiology – Pediatric Radiology, University of Tuebingen, Tuebingen, Germany.

V2 **CLOACAL EXSTROPHY — OPERATIVE MANAGEMENT OF THE NEWBORN**

Marc A. Levitt, MD, Andrea Bischoff, MD, Alberto Peña, MD.

Cincinnati Children's Hospital, Cincinnati, OH, USA.

V3 **LAPAROSCOPIC ASSISTED PSARP FOR RECTO-BLADDERNECK AND HIGH PROSTATIC FISTULA**

Marc A. Levitt, MD, Andrea Bischoff, MD, Alberto Peña, MD.

Cincinnati Children's Hospital, Cincinnati, OH, USA.

V4 **THORACOSCOPIC DISTAL ESOPHAGECTOMY FOR CONGENITAL ESOPHAGEAL STENOSIS FROM TRACHEOBRONCHIAL REMNANTS**

Eveline H. Shue, MD, Hanmin Lee, MD, Shinjiro Hirose, MD.

University of California San Francisco, San Francisco, CA, USA.

V5 **LAPAROSCOPIC MEDIAN ARCUATE LIGAMENT RELEASE FOR SYMPTOMATIC CELIAC ARTERY COMPRESSION: 2 CASES WITH VIDEO**

James Wall, MD, Matias Bruzoni, MD, Sanjeev Dutta, MD.

Lucile Packard Children's Hospital at Stanford, Palo Alto, CA, USA.

V6 **LAPAROSCOPIC REPAIR OF DIAPHRAGM EVENTRATION**

Oliver B. Lao, MD, MPH, Shahab F. Abdessalam, MD.

Children's Omaha, Omaha, NE, USA.

Wednesday May 23, 2012

8:00 – 9:15 a.m. **Scientific Session V** *Cibolo Canyon*
Clinical Pediatric Surgery *Salons 5-11*

Moderators:
David J. Schmeling, MD; Todd A. Ponsky, MD

Learning Objectives:
At the conclusion of the session, participants will be able to:
- Discuss the impact of new technology on common pediatric surgical conditions
- Describe the efficacy of gastric pacing
- Cite the evidence for bracing therapy for pectus carinatum

41 **INFLUENCE OF NEW TECHNOLOGY ON PRACTICE PATTERNS AND OUTCOME: THE CASE OF THE PREFORMED SILO**

Edmund Yang, MD, PhD[1], Derek Banyard, MD[2], Catherine Dale, MD[3].

[1]St. Louis Fetal Care Institute, Cardinal Glennon Children's Medical Center, St. Louis, MO, USA, [2]Department of Surgery, Charleston Area Medical Center, Charleston, WV, USA, [3]Department of Surgical Sciences, Vanderbilt University, Nashville, TN, USA.

42 **SINGLE INCISION VERSUS STANDARD 4-PORT LAPAROSCOPIC CHOLCYSTECTOMY: A PROSPECTIVE RANDOMIZED TRIAL**

Daniel J. Ostlie, MD, Obinna O. Adibe, MD, David Juang, MD, Corey W. Iqbal, MD, Susan W. Sharp, PhD, Charles L. Snyder, MD, Walter S. Andrews, MD, Ronald J. Sharp, MD, George W. Holcomb, III, MD, Shawn D. St. Peter, MD.

Children's Mercy Hospital, Kansas City, MO, USA.

43 **CARDIOVASCULAR RECOVERY FOLLOWING BARIATRIC SURGERY IN EXTREMELY OBESE ADOLESCENTS: PRELIMINARY RESULTS USING CARDIAC MAGNETIC RESONANCE (CMR) IMAGING**

Marc P. Michalsky[1], John A. Bauer, PhD[1], Steven Teich, MD[1], Dara P. Schuster, MD[2], Subha V. Raman, MD[2].

[1]Nationwide Children's Hospital, Columbus, OH, USA, [2]The Ohio State University Medical Center, Columbus, OH, USA.

44 **FACTORS INFLUENCING THE OUTCOME OF PORTAL REPERFUSION FOR EXTRA HEPATIC/PORTAL VENOUS OBSTRUCTION (EHPVO) – A 15-YEAR EXPERIENCE**

Florent Guerin, MD[1], Valeska Bidault, MD[1], Sabine Irtan, MD[2], Frédéric Gauthier, PhD[1], Virginie Fouquet, MD[1], Hélène Martelli, PhD[1], Sophie Branchereau, MD[1].

[1]Bicêtre Hospital, Le Kremlin Bicêtre, France, [2]Necker-Enfants Malades, Paris, France.

45 **EFFICACY OF PERMANENT GASTRIC ELECTRICAL STIMULATION FOR THE TREATMENT OF GASTROPARESIS AND REFRACTORY NAUSEA AND VOMITING IN CHILDREN AND ADOLESCENTS**

Steven Teich, Hayat M. Mousa, MD, Carlo DiLorenzo, MD.

Nationwide Children's Hospital, Columbus, OH, USA.

46 **BRACING IS AN EFFECTIVE NON-OPERATIVE THERAPY IN PATIENTS WITH *PECTUS CARINATUM*: AN INTERIM REPORT OF THE CALGARY PROTOCOL**

Richy T. Lee, MD[1], Scott Moorman[2], Marc Schneider, BSc[3], David L. Sigalet, MD, PhD[1].

[1]Alberta Children's Hospital, Calgary, AB, Canada, [2]University of Alberta, Edmonton, AB, Canada, [3]Braceworks, Calgary, AB, Canada.

47 **RISK FACTORS FOR REOPERATION AND MORTALITY IN PEDIATRIC PATIENTS UNDERGOING PERITONEAL DIALYSIS**
Jennifer Phan, Steve Stanford, Joshua Zaritsky, MD, Daniel DeUgarte, MD.
UCLA, Los Angeles, CA, USA.

48 **A PROSPECTIVE RANDOMIZED TRIAL OF ULTRASOUND VERSUS LANDMARK GUIDED CENTRAL VENOUS ACCESS IN THE PEDIATRIC POPULATION**
Matias Bruzoni[1], Bethany J. Slater, MD[1], Karl G. Sylvester, MD[1], Craig T. Albanese, MD, MBA[1], Claudia M. Mueller, MD, PhD[1], Shawn D. St. Peter, MD[2], Susan W. Sharp, PhD[2], Daniel J. Ostlie, MD[2], Sanjeev Dutta, MD[1].
[1]Stanford University, Palo Alto, CA, USA, [2]Children's Mercy Hospital, Kansas City, MO, USA.

49 **INCISION AND DRAINAGE OF SUBCUTANEOUS ABSCESSES WITHOUT THE USE OF PACKING**
Michael J. Leinwand, MD[1], Marc T. Downing, MD[1], Dwight Slater, MD[2], Marci Beck, RN[1], Karen Burton, RN[1], Donna Moyer, RN[1].
[1]Bronson Children's Hospital, Kalamazoo, MI, USA, [2]Michigan State University / Kalamazoo Center for Medical Studies, Kalamazoo, MI, USA.

9:15 – 10:15 a.m. **Outcomes and Clinical Trials Update** *Cibolo Canyon*
NEC; VATS/Empyema *Salons 5-11*

Moderator:
Fizan Abdullah, MD

Learning Objectives:
At the conclusion of the session, participants will be able to:
- Discuss the current literature regarding necrotizing enterocolitis in infants
- Discuss the current literature regarding empyema in children
- Implement the above into their practice

Management of Necrotizing Enterocolitis in Infants
Cynthia D. Downard, MD; Gudrun Aspelund, MD

Diagnosis and Management of Empyema in Children
Shawn D. St. Peter, MD; Saleem Islam, MD

10:15 – 10:30 a.m. Refreshment Break *Cibolo Canyon Foyer*

10:30 a.m. – Noon	**Pediatric Surgery Case Debates and Controversies**	*Cibolo Canyon Salons 5-11*

Moderator:
Carroll M. Harmon, MD

Learning Objective:
Participants in this session will debate treatment options for difficult pediatric surgical cases

Noon	Annual Meeting Concludes

APSA-IPSO Symposium
Oncology Research

Saturday, May 19, 10:30 a.m. – Noon

1

PRIMARY NEPHRECTOMY AND INTRA-OPERATIVE TUMOR RUPTURE: REPORT FROM THE CHILDREN'S ONCOLOGY GROUP (COG) RENAL TUMORS COMMITTEE

Peter F. Ehrlich[1], Ken Gow, MD[2], Doug Barnhardt, MD[3], Jessica Kandel, MD[4], Tom Hamilton, Md[5], Mike Chen, MD[6], Mitchell Price[7], Arlene Naranjo, Md[8], Elizabeth Perlman, MD[9], Jeff Dome, MD[10], Elizabeth Mullen[11].

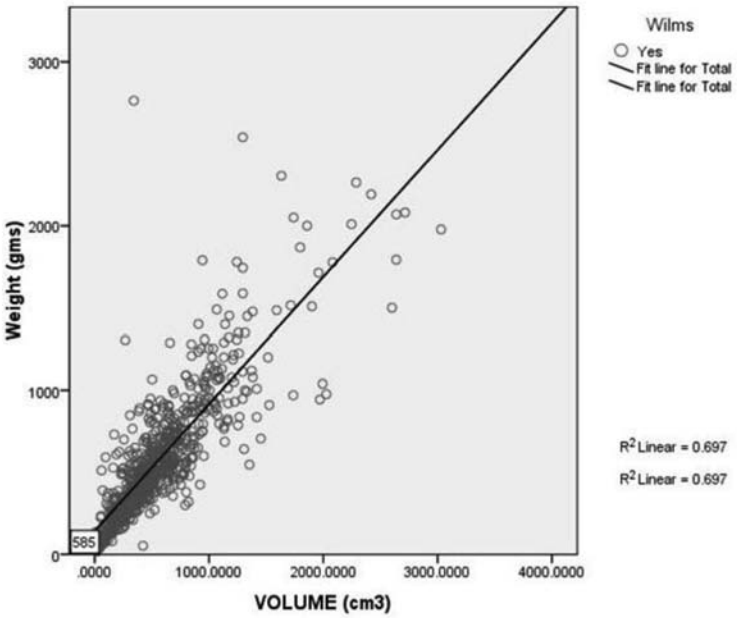
[1]University of Michigan, Ann Arbor, MI, USA, [2]University of Washington, Washington, WA, USA, [3]Primary Childrens Hospital, Salt Lake City, UT, USA, [4]Columbia University, New York City, NY, USA, [5]Boston Childrens Hospital, Boston, MA, USA, [6]University of Alabama, Birmingham, AL, USA, [7]University of Illinois, Chicago, IL, USA, [8]University of Florida, Ganesville, FL, USA, [9]Childrens Memorial, Chicago, IL, USA, [10]Childrens National Medical Center, Washington, DC, USA, [11]Boston Chgildrens, Boston, MA, USA.

Purpose:
Intra-operative rupture (IOR) is associated with increased recurrence and more intensive therapy. In prior studies the rate of IOR was 15.1-19.0%. This study was performed to determine current rates and identify factors associated with IOR.

Methods:
Patients enrolled on study AREN03B2 (2006-present) undergo central review of radiology, pathology and surgery to assign tumor stage. Adherence to protocol, tumor laterality, weight and size, preoperative and IOR are recorded. Analyses were performed using chi square and logistic regression. Odd ratios(OR) are shown with 95% confidence intervals.

Results:
There were 1,131 primary nephrectomies for unilateral WT with an IOR rate of 9.7% with an additional 1.8% having tumor spill during renal vein thrombectomy. IOR correlated with diameter (>12cm, P< 0.0001) and laterality (right, p=0.0414). Simple logistic regression indicated that IOR increased 2.7% [p=0.0240, OR 1.024(1.004, 1.052)]with each 1 cm increase in diameter(3-21cm)and 4.7% [p= 0.0147 1.047 (1.009, 1.086)]with each 100g increase in weight (80-1800g). Multiple logistic regression indicated that laterality [right p =0.04 OR 1.45 (1.004,2.110)] and weight (p=0.03, OR 1.039 (1.003,1.075) were predictive of IOR when diameter was included as a continuous variable. However, diameter as a binary variable was highly prognostic of IOR(p>0.0002), while laterality and weight were not. The OR for IOR peaked at 15 cm 2.606 (1.737,3.910). There is a linear correlation between volume and weight p=0.01.

Conclusions:
IOR occurs in less than 10% of primary nephroureterectomies for WT. IOR correlates with large and right-sided tumors



NOTES:

2

CLINICAL AND MOLECULAR CHARACTERISTICS OF INVASIVE NEUROBLASTOMA

Kappler Roland, PhD, Nathalie Kremer, MD, Josef Mueller-Hoecker, MD, PhD, Maximilian Stehr, MD, PhD, Dietrich G. von Schweinitz, MD, PhD.

University of Munich, Munich, Germany.

Purpose:
To investigate clinical and molecular genetic characteristics of neuroblastomas (NB) with invasive growth into vessel walls and solid organs.

Methods:
After IRB approval and informed consent of all patients clinical data of 63 NB consecutively resected between January 2009 and June 2011 were analysed for intraoperative finding of invasive growth, surgical radicality, INSS stage, histology, MYCN status, and patient's outcome. Material of 4 NB with and 6 NB without invasive growth was analysed with a multi-gene PCR array for the expression of 94 genes associated with migration, invasion, signal transduction and inflammation.

Results:
Invasion of vessel walls and/or solid organs was intraoperatively found in 17 (27%) NB. The resection was complete (>95%) in 57 (11 of these invasive NB), incomplete (<95%) and a biopsy only each in 3 patients with invasive NB. Three tumors were in stage 1, 5 in stage 2, 24 in stage 3 (7 with invasion) and 31 in stage 4. Histologically 4 tumors were ganglioneuromas, 26 ganglioneuroblastomas, 26 differentiating NB and 7 undifferentiated NB. MYCN was amplified in 11/63 tumors. After the short follow-up 49 (78%) patients were tumor-free, 6 (9%) alive with tumor, 7 (11%) died of tumor and one after surgery. Invasive growth did not correlate with stage or histology but significantly with MYCN amplification (p=.0026) and poor outcome (p=.0001). The array analysis identified 10 genes (FOXF1, CCND2, LEF1, NOTCH2, BMP2, TGFB1, TGFBR3, STAT1, NF-kappa B and G-CSF receptor) with overexpression only in the 6 invasive compared to the 4 non-invasive analysed NB.

Conclusions:
Approximately one quarter of advanced NB grow invasively into vessel walls and/or solid organs which is associated with MYCN amplification and impaired prognosis. Invasive NB seem to be characterized by a specific gene expression pattern.

NOTES:

3

TREATMENT, OUTCOME AND SURGICAL TREATMENT FAILURES IN PATIENTS SUFFERING FROM PARATESTICULAR RHABDOMYOSARCOMA TREATED WITHIN THE COOPERATIVE SOFT TISSUE SARCOMA TRIALS CWS-86, -91-, -96, AND -2002P

Guido Seitz, MD[1], Joerg Fuchs, MD[1], Tobias Dantonello, MD[2], Daniel Kosztyla[2], Thomas Klingebiel, MD[3], Ewa Koscielniak, MD.[2]

[1]University Children`s Hospital, Tuebingen, Germany, [2]Olgahospital, Stuttgart, Germany, [3]University Children`s Hospital, Frankfurt/Main, Germany.

Purpose:
Paratesticular rhabdomyosarcoma (RMS) accounts for 7% of all rhabdomyosarcomas. The tumor localization seems to be favourable as the outcome is better than in other tumor localizations even in alveolar RMS. Complete surgical resections are often possible due to early diagnosis of the tumors. The aim of this study was to analyze the primary and secondary surgical approach as well as outcome of patients suffering from paratesticular RMS treated within the CWS Trials.

Methods:
One-hundred-seventy-three patients with diagnosis of paratesticular RMS were included into the trials between 1986 and 2008. Nine patients were excluded due to an incomplete data set. Finally, one-hundred-sixty-four patients were analyzed. All patients underwent primary tumor resection or biopsies followed by secondary tumor resection and were treated with multiagent chemotherapy according to the treatment protocols. All trials were approved by the IRB.

Results:
Eight patients had alveolar RMS, one-hundred-fifty-six patients had embryonal histology. The five year overall survival rate was 91.9% ± 2.2. The 5 year event free survival was 86.4% ± 2.8. Nineteen patients had positive lymph nodes at initial diagnosis. Local relapse was observed in four patients. Lymph node recurrence was found in eleven patients. Metastatic relapse occurred in thirteen patients. Radiotherapy was used in nineteen patients. Inadequate primary surgical approaches were observed in fourty-six patients (incomplete primary resection without unilateral orchiectomy or transscrotal approaches). Nevertheless, only two of these patients are not in complete remission. Hemiscrotectomy was carried out in twentysix patients (primary n=1, secondary n=25).

Conclusions:
We conclude that patients suffering from paratesticular RMS treated within the CWS trials have an excellent prognosis. A high number of inadequate primary surgical approaches were observed. An inadequate primary surgical approach should be followed by a complete tumor resection. Radiotherapy seems to be indicated for lymph node metastases.

NOTES:

4

HIF-1α ACTIVATION MEDIATES RESISTANCE TO ANTIANGIOGENIC THERAPY IN NEUROBLASTOMA XENOGRAFTS

Joseph E. Hartwich, MD[1], W. Shannon Orr, MD[2], Catherine Y. Ng, MS[3], Yunyu Spence, PhD[3], Christopher Morton, BS[3], Andrew M. Davidoff, MD[3].

[1]Virginia Commonwealth University, Richmond, VA, USA, [2]University of Tennessee Health Science Center, Memphis, TN, USA, [3]St Jude Children's Research Hospital, Memphis, TN, USA.

Introduction:
The anti-tumor activity of angiogenesis inhibitors is often limited by the development of resistance to these drugs. Here we establish HIF-1α as a major factor in the development of this resistance in neuroblastoma xenografts and test whether this can be overcome by the co-administration of low dose topotecan to achieve HIF-1α inhibition.

Methods:
Neuroblastoma xenografts were established by injecting unmodified SKNAS or NB-1691 cells (2x10^{6}cells), or cells in which HIF-1α expression had been knocked down with siRNA, into the retroperitoneal space of SCID mice. Treatment of established tumors included bevacizumab (5mg/kg q2wk), sunitinib (40mg/kg qd) or topotecan (0.5mg/kg qd) alone, or a combination in which topotecan was added to either sunitinib or bevacizumab, for a total of two weeks. Tumors were sized by ultrasound before and after treatment. All studies were approved by institutional IRB and IACUC prior to initiation.

Results:
SKNAS xenografts showed no difference in relative growth in HIF-1α knockdowns compared to unmodified tumors (55.83+/-3.39 vs. 63.24+/-2.62 p=0.174). However, HIF-1α knockdown xenografts demonstrated relative final volumes that were significantly lower than unmodified tumors when both were treated with bevacizumab (30.21+/-6.49 vs. 40.64+/-3.16 p=0.041) or sunitinib (17.13+/-2.21 vs. 40.89+/-2.13 p=0.001). Monotherapy of unmodified xenografts with bevacizumab, sunitinib or topotecan was largely ineffective. However, relative final volumes of SKNAS xenografts were significantly less in cohorts treated with sunitinib+topotecan (14.6+/-1.65 vs.32.36+/- 5.069[sunitinib alone], p= 0.011) and bevacizumab+topotecan (19.88 +/-4.07 vs. 44.06+/-6.059[bevacizumab alone], p=0.016). Results were similar when performed with NB1691 xenografts. Tumor samples from mice treated with low dose topotecan demonstrated a decrease in expression of severeal HIF-responsive genes as confirmed by qPCR.

Conclusion:
Upregulation of HIF-1α appears to be a significant mechanism of resistance to antiangiogenic therapies in human neuroblastoma. Supressing HIF-1α potentiates the effects of the antiangiogenic drugs bevacizumab and sunitinib in a mouse orthotopic xenograft model.

5

mTOR INHIBITION PROMOTES OSTEOPROTEGERIN PRODUCTION, DELAYING TIME TO PATHOLOGIC FRACTURE IN AN ORTHOTOPIC XENOGRAFT MODEL OF NEUROBLASTOMA BONE METASTASIS

Joseph E. Hartwich, MD[1], W. Shannon Orr, MD[2], Catherine Y. Ng, MS[3], Yunyu Spence, PhD[3], Wayne Furman, MD[3], Lisa M. McGregor, MD, PhD[3], Andrew M. Davidoff, MD[3].

[1]Virginia Commonwealth University, Richmond, VA, USA, [2]University of Tennessee Health Science Center, Memphis, TN, USA, [3]St Jude Children's Research Hospital, Memphis, TN, USA.

Introduction:
Osteoprotegerin (OPG) is a decoy receptor for RANK ligand that can inhibit osteoclastogenesis and slow the progression of osteolytic bone lesions. Previous studies have suggested that mTOR inhibition upregulates OPG production. We tested the hypothesis that the mTOR inhibitor rapamycin could inhibit neuroblastoma bone metastases through its action on OPG.

Methods:
An orthotopic model of bone metastasis was established by injecting CHLA-20 or NB-1691 neuroblastoma cells into the femora of SCID mice. Mice with established disease were subsequently treated with rapamycin (5mg/kg IP daily) or vehicle control (DMSO1:1000). X-rays were obtained twice a week to detect pathologic fractures. Serum OPG levels were measured by ELISA after two weeks of treatment. In addition, OPG levels in serum samples from a cohort of patients treated with temsirolimus, a rapamycin analogue, were compared to sera from patients who did not receive temsirolimus. Institutional IRB and IACUC approval was obtained prior to initiation of all studies.

Results:
Both cell lines were shown to be resistant to direct cytotoxic effects of rapamycin treatment in vitro and in vivo. Mice with bone disease receiving rapamycin had increased serum levels of OPG in the CHLA-20 (36.89pg/mL+/-3.90 vs.18.4pg/mL+/-1.67, p=0.004) and NB-1691 tumor-bearing groups (46.03pg/mL+/-2.67 vs. 17.96pg/mL+/-1.84, p=0.001), and a significantly longer median time to pathologic fracture compared to control with CHLA-20 (103 days vs. 74.5 days, p=0.0139) and NB1691 (93 days vs. 62 days p=0.0086) xenografts. In human patients treated with temsirolimus, OPG levels were significantly increased compared to a control group that did not receive the drug (1299pg/mL+/-29.98 vs. 702.7pg/mL+/-37.10, p=0.0251).

Conclusion:
Treatment with mTOR inhibitors increased OPG expression in both a mouse xenograft model and human patients. In the xenograft model, increased OPG expression correlated with a delay to pathologic fracture, implicating a potential role for mTOR inhibitors in the treatment of neuroblastoma bone metastases.

NOTES:

6

HEPATOCELLULAR CARCINOMA IN CHILDREN AND ADOLESCENTS: THE INTERNATIONAL THERAPEUTIC EXPERIENCE

Marcio H. Malogolowkin[1], Piotr Czauderna[2], Beate Haberle[3], Eiso Hiyama[4], Irene Schmid[3], Mark Krailo[5], Rudolf Maibach[6], Howard Katzenstein[7], Giorgio Perilongo[8], Daniel C. Aronson[9], Dietrich VonSchweinitz[3], Rebecka L. Meyers[10].

[1]Childrens Hospital Los Angeles, Los Angeles, CA, USA, [2]Medical University of Gdansk, Gdansk, Poland, [3]Children`s Hospital of the Ludwig-Maximilians-University, Munich, Germany, [4]Hiroshima University Hospital, Hiroshima, Japan, [5]University of Southern California Keck School of Medicine, Los Angeles, CA, USA, [6]International Breast Cancer Study Group, Bern, Switzerland, [7]Children's Healthcare of Atlanta, Emory University, Atlanta, GA, USA, [8]University Hospital of Padua, Padua, Italy, [9]Altona Children's Hospital/University Medical Center Hamburg-Eppendorf, Hamburg, Germany, [10]University of Utah, Salt Lake City, UT, USA.

Purpose:
In comparison to adults, hepatocellular carcinoma (HCC) in children and adolescents is less often associated with inflammatory liver diseases. The large de-novo tumors arising in non-cirrhotic livers suggest unique pathogenesis in the pediatric population.

Methods:
An international HCC workshop was organized so members of the four major pediatric liver tumor study groups (COG,SIOPEL,GPOH, and JPLT) could review and compare results of their HCC studies in children. These pediatric studies were primarily developed for children with hepatoblastoma (HB), with enrollment of children with HCC as a subgroup.

Results:
A total of 243 children with HCC were registered onto these studies. Chemotherapy included a platinum agent, cisplatin or carboplatin, in combination with one or more drugs. 60% of children were more than 10 years old; 140(58%) presented with advanced disease; 74(30%) had metastatic disease at diagnosis. Complete surgical resection was performed at diagnosis in 45(19%). Of the 162 patients evaluable for tumor response to neo-adjuvant chemotherapy, 2(1%) had a complete response, and 66(41%) had a partial response. Post-chemotherapy surgical resection was attempted in 134 of the patients, and complete resection achieved in 46(34%); partial resection in 29(22%); and liver transplant in 14(10%). Overall 91 of the 243 children entered onto these studies (37%) had a complete resection at some point during therapy. 3-year and 5-year EFS and OS ranged between 10-36% and 16-46%, respectively.

Conclusions:
Compared to HB, HCC has a relatively poor outcome in children. More importantly, this unique group of children with HCC respond to therapy quite different than adults with inflammatory liver disease. Given the rarity of this childhood tumor, international collaboration is essential to evaluate novel therapeutic approaches, to establish the role of liver transplantation, and to continue to improve our understanding of the biology of HCC in this pediatric patient population.

NOTES:

7

UPFRONT BIOPSY FOR UNILATERAL PEDIATRIC RENAL TUMORS: AN INCREASING PROBLEM?

Peter F. Ehrlich[1], Douglas C. Barnhart, MD[2], Ken Gow, MD[3], Tom Hamilton, MD[4], Jessica Kandel, MD[5], Mike Chen, MD[6], Elizabeth Mullen[7], Paul Grundy[8], Jeff Dome, MD[9], Paul Grundy[8].

[1]University of Michigan, Ann Arbor, MI, USA, [2]Primary Children's Hospital, Primary Children's Hospital Salt Lake City, UT, USA, [3]Seattle Children's Hospital, University of Washington Seattle, WA, USA, [4]Boston Children's Hospital, Boston, MA, USA, [5]Columbia University, New York City, NY, USA, [6]University of Alabama Birmingham, Birmingham, AL, USA, [7]Boston Children Hospital, Boston, MA, USA, [8]University of Alberta, Edmonton, AB, Canada, [9]Childrens National Medical Center, Washington, DC, USA.

Purpose:

Primary nephrectomy is the suggested initial treatment for children treated on Children's Oncology Group(COG)/National Wilms Tumors Study (NWTS) protocols. Preoperative chemotherapy is recommended only when renal sparing surgery is desired, such as bilateral Wilms Tumor (WT) or when the tumor is inoperable based on published guidelines. On NWTS -3 the rate of preoperative chemotherapy was 3%, and for NWTS-5 it had risen to 9.3%. A biopsy results in Stage III therapy with abdominal radiation and more intense chemotherapy. The purpose of this study was to investigate the current rates of preoperative chemotherapy.

Methods:

A cohort of stage III unilateral favorable histology patients were identified from the current COG ARENO3B2 study. Those with complete data and stage III by biopsy only were evaluated. Accepted indications for preoperative chemotherapy on COG protocols are: (1)tumor thrombus above the level of the hepatic veins(2) pulmonary compromise from a massive tumor (3) resection requires removal of contiguous structures(4) surgeons' judgment- a nephrectomy would result in significant morbidity, tumor spill, or residual tumor. Operative and radiological reports were reviewed and divided into three groups: (A) criteria met for biopsy; (B)data indeterminate; (C) criteria not met. Patients with bilateral WT or a single kidney were excluded. Descriptive statistics are presented.

Results:

From a sample of unilateral favorable histology WT, 309 had stage III, of these 115 were stage III due to biopsy only. Following review, 71 (61%) were categorized as Group A (large tumor accounted>60%); 13 (11%) were group B and 31 (26%) were group C. The most common reason for classification into group C was biopsy of a small tumor in a child with stage IV disease.

Conclusion:
Pre-resection chemotherapy rates are higher for the current COG WT protocols. It is possible that a significant proportion could have undergone primary nephrectomy potentially avoiding treatment toxicity and late effects.

NOTES:

8

NEPHRON SPARING SURGERY (NSS) FOR UNILATERAL WILMS' TUMOR (uWT) THE SIOP 2001 EXPERIENCE

J.C.H. Wilde, MD[1], Daniel C. Aronson, MD, PhD[1], Beate Sznajder[2], Harm van Tinteren[2], Mark Powis[3], Bruce Okoye[4], Giovanni Cecchetto[5], Georges Audry[6], Jörg Fuchs[7], Dietrich von Schweinitz[8], Hugo A. Heij[1], Norbert Graf[9], Christophe Bergeron[10], Kathy Pritchard-Jones[11], Marry van den Heuvel-Eibrink[12], Modesto Carli[5], Foppe Oldenburger[13], Bengt Sandstedt[14], Jan de Kraker[1], Jan Godzinski[15].

[1]Emma Children's Hospital AMC, Amsterdam, Netherlands, [2]Netherlands Cancer Institute – AVL, Amsterdam, Netherlands, [3]Leeds Teaching Hospitals Trust, Leeds, United Kingdom, [4]St. George's Healthcare NHS Trust, London, United Kingdom, [5]University-Hospital of Padua, Padua, Italy, [6]Hôpital Trousseau, Paris, France, [7]Children's Hospital, University of Tübingen, Tübingen, Germany, [8]Dr von Hauner Children's Hospital, Ludwig-Maximilians-University, Munich, Germany, [9]University of Homburg, Saar, Germany, [10]Institut d'Hémato-Oncologie Pédiatrique, Lyon, France, [11]Institute of Child Health & Great Ormond Street Hospital, London, United Kingdom, [12]Sophia Children's Hospital, ErasmusMC, Rotterdam, Netherlands, [13]Academic Medical Center, Amsterdam, Netherlands, [14]Childhood Cancer Research Unit, Karolinska Institutet, Astrid Lindgren Children´s Hospital, Stockholm, Sweden, [15]Marciniak Hospital, Wroclaw and Medical University, Chair of Emergency, Wroclaw, Poland.

Purpose:
Although total nephrectomy remains the standard approach to uWT, the SIOP WT-2001 protocol allowed NSS for polar or peripherally non- infiltrating tumors. Aim: Inventory of the current SIOP NSS-experience.

Methods:
Patients in SIOP WT-2001 (stage 1-4) with an unequivocal surgical resection technique recorded, were included. All had neo-adjuvant chemotherapy and delayed surgery. In 98 (3%) NSS was performed and in 2527 total nephrectomy (TN) - 453 (15%) were excluded. Results regarding complications, local recurrence and 5 year-survival were compared and stratified for abdominal stage (1-3) and histopathology.

Results:
Abdominal staging in the NSS group was stage I in 57, stage II in 13, stage III in 20 (Lymph nodes positive [LN+] in 12, margin positive in 14 among which 7 were LN negative [LN-]), and unknown in 8. Histopathology showed low risk in 8, intermediate in 76, high risk in 11 (11.2%), missing in 3. NSS had a rupture rate of 4% (equal to TN) and other complications in 11% (more than TN, p=0.02). Treatment of positive margins consisted of irradiation in 4 (3 with LN-), kidney remnant (KR) removal in 4, and so far unknown in 6. Six KR's were irradiated, 3 with LN+. One margin was positive for nephroblastomatosis and was not irradiated nor re-resected. After NSS, 5 relapses occurred with no deaths,

OS and EFS after NSS were 100.0 (95%CI: 100.0-100.0) and 93.8 (95%CI: 88.7–99.3), respectively (log rank NSS versus TN: OS p=0.03, EFS p=0.06).

Conclusions:
NSS was performed in a small minority (3%) of uWT patients. Their complication rate was significantly higher but rupture rates equaled those of TN. Despite excellent long term survival with few relapses, the gain in nephrons needs to be weighed against the risk of causing a stage III tumour due to microscopic residue, with the consequent need for intensified therapy.

NOTES:

9

SIGNIFICANCE OF IMAGE-DEFINED RISK FACTORS (IDRFs) FOR SURGICAL TREATMENT OF NEUROBLASTOMAS

Maximilian Stehr, MD, PhD, Moritz Erichsen, Florian Bergmann, MD, Dietrich von Schweinitz, MD, PhD.

Department of Pediatric Surgery, Ludwig-Maximilians-Universität, Munich, Germany.

Purpose:
To evaluate the correlation and significance of Image-Defined Risk Factors (IDRFs) with radicality, complications and outcome of surgical treated neuroblastomas.

Methods:
Patients were treated according to different trials with ethical approval (e.g. NB97, No. 9764; NB2004, No. 04049). Data from all patients were collected retrospectively and anonymized. From 2003 to 2010 we operated on 125 children with neuroblastomas. Due to missing data in the follow-up only 104 children were included in this study. 18 patients had no IDRF (IDRF-) while 84 had one or more (IDRF+). In 2 patients IDRF-status could not be evaluated properly. In 9 patients a biopsy (PE) only was taken. In the other patients tumor resection was incomplete (IC=50-90% removal of the tumor mass) in 17, near complete (NC=>90%) in 42 and complete (C=100%) in 36.

Results:
In 21 patients intra- or postoperative complications occured (intraoperative:1 death, massive bleeding, loss of one kidney; postoperative: ileus, chylascos, lymphocele, sepsis, RDS, necrosis of one ureter, renal failure, pancreatitis, bile leakage). Of these 21 patients 17 were IDRF+ and 2 were IDRF-, respectively. The 2 patients with no defined IDRF-status exhibited also complications. IDRF+ were 9/9 patients with PE, 17/17 patients with IC-resection, 39/42 patients with NC-resection, and 19/36 patients with C-resection. Statistics showed significance for the groups PE vs. C ($p=0.005$), IC vs. NC ($p=0.001$), IC vs. C ($p=<0.001$), and NC vs. C ($p=0.005$). No significance could be shown for the groups PE vs. IC and PE vs. NC. In 18 patients local relapses occured during the follow-up. 15 patients were IDRF+, and only 1 patients was IDRF-. The 2 patients with no defined IDRF-status developped also relapses. However, this remarkable trend was statistically not significant.

Conclusion:
The IDRF-system is a usefull additional tool predicting surgical risk in the treatment of neuroblastomas.

NOTES:

10

MARGIN STATUS DOES NOT IMPACT TUMOR RECURRENCE AFTER NEPHRON-SPARING SURGERY FOR BILATERAL WILMS' TUMOR

Kathleen Kieran, MD[1], Mark A. Williams, MD[2], Jeffrey S. Dome, MD[3], Matthew J. Krasin, MD[4], Andrew M. Davidoff, MD[4].

[1]University of Iowa, Iowa City, IA, USA, [2]University of Tennessee/LeBonheur Children's Hospital, Memphis, TN, USA, [3]Children's National Medical Center, Washington, DC, USA, [4]St Jude Children's Research Hospital, Memphis, TN, USA.

Purpose:
Nephron-sparing surgery (NSS) has been advocated for patients with favorable histology (FH) bilateral Wilms tumor (BWT); however, positive surgical margins may be a consequence. We reviewed our experience to determine whether the presence of positive margins impacted local tumor recurrence.

Methods:
We identified all patients who underwent NSS for BWT at our institution from November 1999 - March 2009. Data abstracted included patient demographics, tumor histopathology including margin status, the use of adjuvant flank ionizing radiation (XRT) and the development of tumor recurrence.

Results:
Twenty-one patients were included in our analysis. Eighteen patients underwent bilateral NSS and three patients underwent unilateral nephrectomy and contralateral NSS. Five of 21 patients (23.8%) had at least one positive margin and so received ipsilateral flank XRT; an additional 2 patients (9.5%) received flank irradiation because of focal anaplasia. Seven (33%) patients developed recurrent disease (RD), a mean of 18.0 (range 1.3-39.9) months after NSS. Five (23.8%) patients recurred in the kidney. One patient had a retroperitoneal recurrence and one patient recurred in the kidney and liver with anaplastic WT (original histology was blastemal predominant, favorable histology) and ultimately died of disease. The rate of recurrence for patients with positive margins was similar to those with negative margins (1/5 [20%] vs 6/16 [37.5%]; p=0.47); however, all patients with positive margins received XRT, whereas only 2/16 (12.5%; p<0.001) patients with negative margins did. At a mean follow-up of 55.3 months (range 5.2-142.3), 20/21 patients are alive, without disease.

Conclusions:
These results support the aggressive use of NSS for patients with BT, since in our experience, the surgical margin status after NSS did not impact the rate of local recurrence. However, the use of adjuvant flank irradiation is likely to have contributed to this outcome.

NOTES:

11
SYSTEMIC INHIBITION OF TIE2 SIGNALING ENHANCES SURVIVAL IN A EWING'S SARCOMA TUMOR MODEL

Ari Reichstein, MD, Alejandro Garcia, MD, Yan Jun Chang, BS, Matthew Leskowitz, BA, Sonia L. Hernandez, PhD, Jianzhong Huang, MD, Darrell Yamashiro, MD, PhD, Jessica Kandel, MD.

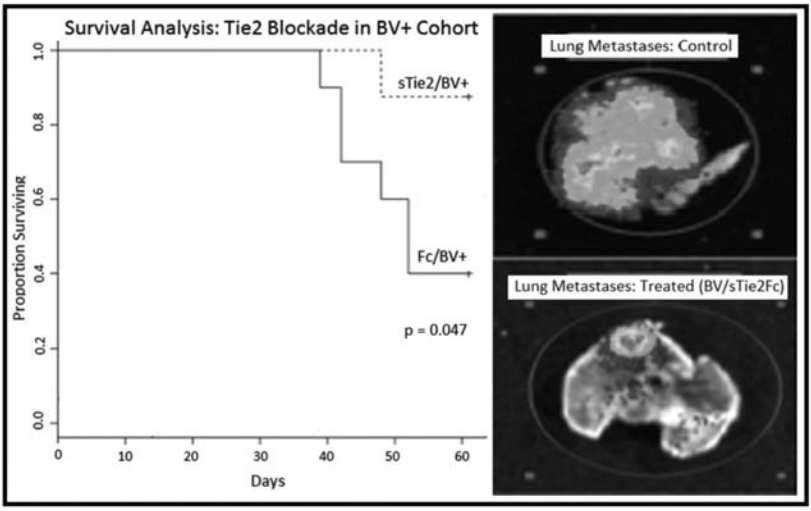
Morgan Stanley Children's Hospital, Columbia University Medical Center, New York, NY, USA.

Purpose:
Vascular endothelial growth factor (VEGF) antagonism is a clinically validated cancer treatment, yet most patients eventually develop progressive disease. Activation of Tie2, which mediates an alternative proangiogenic pathway that can rescue vasculature from VEGF blockade, may function in such resistance. We hypothesized that combining Tie2 and VEGF inhibition would more effectively limit tumor growth than VEGF inhibition alone.

Methods:
All animal experimentation was conducted according to our IACUC-approved protocol. Xenografts were implanted in nude mice by intrarenal injection of 10^6 SKNEP1 cultured Ewing's sarcoma cells. We engineered adenoviral vectors to induce expression of either a soluble Tie2 construct (sTie2Fc) or Fc. Mice were randomized to sTie2Fc or Fc groups, and injected with adenovirus at week 1 post-implantation. To study Tie2 blockade in the context of VEGF antagonism, groups were further subdivided to anti-VEGF antibody (bevacizumab, BV) or vehicle treatment (N=10,10,10,13 respectively: control+Fc, control +sTie2-Fc, BV+Fc, BV+sTie2-Fc). In vivo tumor growth was measured using serial bioluminescence imaging, with animals sacrificed at 10^9 photons/second. Harvested organs were analyzed, with metastatic burden quantified using bioluminescence.

Results:
BV+sTie2Fc-treated animals displayed significantly delayed tumor growth as compared to the BV+Fc treated group (51 vs 41 days; p=.047, Kaplan-Meier analysis). Lung metastasis was reduced in animals treated with BV+sTie2Fc in comparison to animals treated with control/Fc (10% vs 56% incidence, p= 0.033). We observed a trend toward reduced metastasis in BV+sTie2Fc versus BV+Fc, although this did not reach significance (10% vs 40%, p=NS).

Conclusions:
The addition of Tie2 inhibition to VEGF blockade significantly delayed tumor growth and limited metastasis in our model. These data are consistent with prior studies indicating that Tie2 activation can stabilize vasculature and recruit stromal support cells in tumors exposed to anti-VEGF agents. Further investigation of Tie2 blockade during VEGF inhibition may enhance the efficacy and durability of this treatment strategy.


Survival Analysis: Tie2 Blockade in BV+ Cohort
Proportion Surviving
1.0
0.8
0.6
0.4
0.2
0.0
0
10
20
30
40
50
60
Days
sTie2/BV+
Fc/BV+
p = 0.047
Lung Metastases: Control
Lung Metastases: Treated (BV/sTie2Fc)

NOTES:

Poster Session I
Basic Science

Sunday, May 20, 4:15 – 6:00 p.m.

P1
THE ROLE OF NOTCH INHIBITION IN A NOVEL HEPATOBLASTOMA ORTHOTOPIC MODEL

Alejandro Garcia, MD, Roderick Alfonso, BS, Angela Kadenhe-Chiweshe, MD, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD.

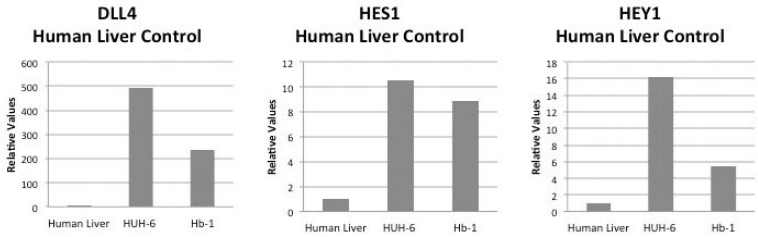
Columbia University, New York, NY, USA.

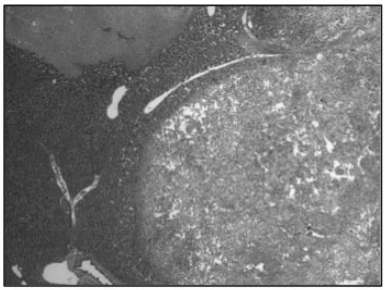
Purpose:
Hepatoblastoma is the most common childhood primary tumor of the liver and approximately half the patients with hepatoblastoma present with advanced disease at diagnosis. Notch signaling is critical to proper liver fetal development. The Notch pathway also plays an important role in angiogenesis. We hypothesize that inhibition of Notch will lead to disruption of the tumor vasculature and consequently decreased tumor growth.

Methods:
Notch expression and pathway activity was compared across hepatoblastoma cells lines, CU-HB1 and HuH6, to control normal human liver using RT-PCR. We successfully established and characterized a novel HuH6 hepatoblastoma orthotopic xenograft model. Next, Huh6 cells were engineered to express soluble Notch1 receptor decoy or empty vector, confirmed by Western blot. Proliferation was assessed by BRD-U assay. HuH-6 cells were then implanted intrahepatically in NCR nude mice. Tumor growth was monitored with bioluminescence. Perfusion was assed by immunohistochemistry. IACUC approval was obtained for all experiments.

Results:
Huh6 and CU-Hb1 hepatoblastoma cell lines exhibited increased significantly higher activity of the Notch pathway (Dll4- Notch ligand, HES1 and HEY1- downstream Notch pathway effectors) compared to normal liver (Figure). Established xenografts were validated by histological investigation (Figure). Notch inhibition in xenografts showed decreased staining for vascular markers and evidence of vascular disruption.

Conclusion:
Our results suggest that Notch expression is important for angiogenesis in experimental hepatoblastoma. We demonstrate the first use of a reproducible orthotopic xenograft model that can be used for further study.





Huh6- wild type (H+E 4X)

NOTES:

P2

LACTOCOCCUS LACTIS ATTENUATES INTESTINAL DAMAGE AND IMPROVES SURVIVAL IN A PRETERM RABBIT MODEL OF INTESTINAL INJURY

Andrew P. Bozeman, MD, Melvin S. Dassinger, MD, Rhea J. Birusingh, MD, Samuel D. Smith, MD.

University of Arkansas for Medical Sciences, Arkansas Children's Hospital, Little Rock, AR, USA.

Purpose:
There is an emerging interest in probiotics to benefit neonatal intestinal dysfunction, especially necrotizing enterocolitis. Our lab has focused on Lactococcus lactis as a unique probiotic. Less studied than others, L. Lactis does not demonstrate virulence factors and supports decreased pathogenic bacterial translocation and colonization in a preterm rabbit model of enteral sepsis. Presently, we investigate if intestinal damage severity and survivorship are impacted by probiotic use in a neonatal rabbit model of intestinal injury.

Methods:
Following cesarean section, rabbit pups (n=129) were assigned to two groups: CONTROLS (n=69) and PROBIOTIC (n=60). Pups were gavage fed milk replacer, ranitidine, indomethacin and Enterobacter cloacae ($1x10^5$ cfu/ml diet). In addition, the PROBIOTIC group received L. lactis ($1x10^7$ cfu/ml diet). The pups' anal orifices were blocked using tissue adhesive. Anal blocks were removed on day 2. Dying pups were euthanized and their intestines processed for histology; surviving pups were euthanized on day 7. A blinded pathologist examined specimens for histological changes of intestinal mucosal injury using a de novo grading scale 0-4, where grades 1-2 represented mild intestinal injury, and grades 3-4 denoted severe injury. Survival results were analyzed using Fisher's exact test.

Results:
There were significant differences in survival between the two groups beginning on day 5 and continuing through day 7. When comparing intestinal injury, the CONTROL group, 21/57 (37%) exhibits a greater incidence of severe injury than their PROBIOTIC counterparts, 6/52 (12%), p=0.003.

Conclusions:
As a dietary supplement, *Lactococcus lactis* significantly improves survival and diminishes severity of intestinal damage in a preterm rabbit model of intestinal injury.

Survival					
	Day 3	Day 4	Day 5	Day 6	Day 7
Probiotic	52/60 (87%)	48/60 (80%)	36/60 (58%)	29/60 (48%)	28/60 (47%)
Control	57/69 (83%)	47/69 (68%)	24/69 (35%)	16/69 (23%)	16/69 (23%)
P=	0.63	0.016	0.005*	0.003*	0.006*

NOTES:

P3

NEUREGULIN-4 IS PROTECTIVE AGAINST EXPERIMENTAL NECROTIZING ENTEROCOLITIS IN A RAT MODEL

Shannon L. Castle, MD, Anatoly V. Grishin, PhD, Henri R. Ford, MD, MHA, Mark R. Frey, PhD.

Children's Hospital Los Angeles, Los Angeles, CA, USA.

Purpose:
In human and murine colitis models, expression of the ErbB4 receptor tyrosine kinase is elevated. Furthermore, ErbB4 expression inhibits cytokine-induced apoptosis in cultured colon epithelial cells, suggesting a protective role in the intestine. Activation of the related kinase Epidermal Growth Factor Receptor reduces the severity of necrotizing enterocolitis (NEC) in rats. However, the role of ErbB4 in NEC is unknown. In this study we used in vitro and in vivo models to test the hypothesis that ErbB4 activation protects against experimental NEC.

Methods:
Newborn rat pups were formula fed (FF), FF with the ErbB4-specific ligand neuregulin-4 (NRG4), or breastfed (BF). FF and NRG4 groups were gavage-fed and stressed with hypoxia and hypothermia, with or without 100ng/pup NRG4 per feed. Animals were euthanized upon the development of severe disease and ileum collected. IEC-6 rat ileal epithelial cells were treated with 108 cfu/mL Cronobacter sakazakii, and 100 ng/ml of ErbB4 ligands heregulin-1 or NRG4, with or without treatment with the Src-kinase inhibitor PP2 (2.5 μM). Apoptosis was assessed by luminescent caspase activity assay.

Results:
Immunofluorescence analysis showed increased ErbB4 expression with experimental NEC in rats. Development of NEC was delayed in pups treated with NRG4, with 89% surviving to 36 hours, compared to 55% of the FF group. Kaplan-Meier analysis showed a significant survival advantage ($p=0.039$) for NRG4-treated animals. In vitro, the ErbB4 ligands heregulin and NRG4 reduced bacteria-induced apoptosis by 31+/-1.0% and 45+/-5.6%, respectively ($p=<0.001$). Src inhibition by PP2 reduced NRG4 protection ($p=0.03$).

Conclusions:
NRG4, a ligand specific for ErbB4, is protective against early development of necrotizing enterocolitis in a neonatal rat model and against bacteria-induced apoptosis in vitro. Src kinase inhibition reverses the protective effect of NRG4 in vitro. ErbB4 may represent a highly selective therapeutic target to prevent or attenuate human necrotizing enterocolitis.

NOTES:

P4

INTESTINAL ALKALINE PHOSPHATASE ADMINISTRATION IN NEWBORNS DECREASES INOS EXPRESSION IN A NEONATAL NECROTIZING ENTEROCOLITIS RAT MODEL

Rebecca M. Rentea, MD, Scott R. Welak, MD, Katherine M. Friedrich, MD, Kirkwood A. Pritchard, PhD, Keith T. Oldham, MD, David M. Gourlay, MD.

Medical College of Wisconsin, Milwaukee, WI, USA.

Purpose:
Our previous research has shown supplementation of Intestinal Alkaline Phosphatase (IAP), an endogenous protein expressed in the intestines, decreased the severity of Necrotizing Enterocolitis (NEC) associated intestinal injury and increased in enteral activity in a dose dependent manner. We hypothesized that IAP administration prevents the development of NEC related intestinal inflammation.

Methods:
Full-term newborn Sprague-Dawley rat pups were sacrificed on day of life four. Control pups were vaginally delivered and breast-fed; cesarean delivered rat pups were exposed to intermittent hypoxia in addition to feeds containing LPS (NEC). Select NEC pups received either 40, 4 or 0.4 units/kg of bovine IAP (NEC+ IAP40u, IAP4u or IAP0.4u) once daily in formula. Real-time PCR (qRT-PCR) was used to analyze IAP and iNOS mRNA from the rat pups terminal ileum. Data were analyzed by paired two-tail t-test and expressed as mean +/-SEM and $p \leq 0.01$ (0.05/5) considered significant after correcting for multiple comparisons using the Bonferroni method.

Results:
Supplemental, enteral IAP was associated with a decrease in NEC related intestinal injury in a dose dependent fashion. NEC resulted in a 7 fold increase of iNOS mRNA compared to control ($p \leq 0.002$). Supplemental enteral IAP administration in all groups resulted in a 3 fold decrease in iNOS mRNA expression ($p \leq 0.01$). IAP expression decreased 3 fold in control vs NEC pups ($p \leq 0.0003$). Interestingly,while we have previously reported that IAP administration increases enzyme activity, IAP mRNA expression did not differ between NEC and IAP treated rat pups.

Conclusions:
We conclude NEC is associated with increased intestinal and systemic inflammation, and that enteral IAP administered can prevent NEC related injury through mitigation of intestinal inflammation as evidenced by iNOS mRNA expression. IAP administration does not increase IAP mRNA expression in pups sustaining NEC stresses. Prophylactic enteral IAP may prove to be a useful strategy to prevent NEC.

NOTES:

P5
AN *IN VIVO* MODEL OF HUMAN-DERIVED FIBROUS HAMARTOMA OF INFANCY

Fabienne L. Gray, MD, Azra Ahmed, BS, Christopher G. Turner, MD, MPH, Yuin-Han Loh, PhD, Alexander Devine, BS, Odelya Hartung, BS, David Zurakowski, PhD, George Q. Daley, MD, PhD, Dario O. Fauza, MD.

Children's Hospital Boston, Boston, MA, USA.

Purpose:
The pathophysiology and natural history of hamartomas remain poorly understood. To date, only transgenic models of two specific hamartomas have been described, having done little to enhance our understanding of these tumors. In this study, we describe a human-derived, cell-based animal model of a hamartoma, namely the fibrous hamartoma of infancy.

Methods:
Human amniotic mesenchymal stem cells (aMSCs), characterized by comprehensive flow cytometry, underwent induced pluripotent stem cell (iPS) reprogramming by ectopic expression of six transcription factors previously shown to enhance iPS generation efficiency, namely OCT4, SOX2, MYC, KLF4, hTERT and SV40 Large T. After reprogramming, cells were cultured either on a mouse embryonic fibroblast (MEF) feeder layer, or on a MEF-free environment. Reprogramming was confirmed by immunohistochemistry for markers of a primitive pluripotent state shared with human embryonic stem cells, specifically: Tra-1-81, Tra-1-60, SSEA3, SSEA4, OCT4, and NANOG. After IACUC approval, 16 severe combined immunodeficient mice received intra-muscular injections of 100 µL of a suspension of the iPS-aMSCs in the thigh. Animals were killed 6 months thereafter for analysis.

Results:
Gross tumors were found in 5/16 (31.25%) of the animals, all of which had pathognomonic findings of fibrous hamartoma of infancy, including foci of collagen-poor myxoid mesenchymal tissue, islands of mature adipocytes and fibrotic bundles - there are no specific markers for these tumors. There were no significant differences in the proportions of tumor-bearing animals depending on cell maintenance protocol (MEF vs. MEF-free; P=0.99, Fisher's exact test). Positive expression of E-cadherin confirmed human ontogeny of the tumor cells in all specimens.

Conclusions:
Reprogrammed human amniotic mesenchymal stem cells can generate the fibrous hamartoma of infancy reproducibly in a murine model. This capacity is independent of maintenance in an embryonic feeder layer. This platform may be instrumental to advances in the understanding and treatment of this disease

NOTES:

P6

OSTEOPONTIN BLOCKADE WITH AN RNA APTAMER RESULTS IN REDUCTION OF EPITHELIAL-MESENCHYMAL TRANSITION AND PULMONARY METASTATIC BURDEN IN A XENOGRAFT MODEL OF OSTEOSARCOMA

Lindsay J. Talbot, MD[1], Zhiyong Mi, PhD[2], Syamal D. Bhattacharya, MD[1], Hongtao Guo, MD, PhD[1], Henry E. Rice, MD[1], Paul C. Kuo, MD, MBA[2].

[1]Duke University, Durham, NC, USA, [2]Loyola University, Chicago, IL, USA.

Purpose:
Osteosarcoma (OS) is the most common bone tumor in childhood. Metastatic disease can require multiple pulmonary resections and has a poor prognosis. Targeted therapy to reduce pulmonary metastases would significantly improve outcomes in OS. Epithelial-mesenchymal transition (EMT) is a cell-biology program that promotes metastasis by increasing mobility, plasticity, dedifferentiation, and anti-apoptotic effects in tumors. Osteopontin (OPN) is known to induce EMT in multiple cancers. Osteopontin blockade may be useful to prevent metastasis in OS.

Methods:
Xenograft mice were generated via intratibial injection of LM7, an aggressive OS cell line. Mice were treated every other day with an aptamer against OPN, a nonfunctional aptamer, or saline for five weeks. Tumor growth was measured by bioluminescence. After necropsy, flow cytometry was performed on lungs to recover metastatic cells, which were analyzed by real-time reverse PCR for markers of EMT. H&E staining and IHC were performed on primary and metastatic tumors to evaluate for tumor characteristics and EMT markers.

Results:
Primary tumor growth between groups was not significantly different, although aptamer-treated mouse tumor size began to separate from controls in week four. Aptamer treatment resulted in a 50% reduction in pulmonary metastatic burden in treated mice compared to controls ($p = < 0.001$). Expression of alpha-SMA, tenascin-C, and FSP-1, markers of EMT, were increased over baseline in all groups, but to a lesser degree in aptamer-treated tumors (tenascin C $p = 0.02$). OPN expression in metastatic cells was similar across groups. Histologic examination revealed more extensive primary tumor necrosis in aptamer-treated mice while no necrosis was seen in pulmonary lesions. IHC revealed nuclear localization of tenascin C in metastatic but not primary lesions.

Conclusions:
OPN blockade resulted in reduced pulmonary metastasis in a xenograft model of OS. This may have been due to inhibition of EMT in cells at the primary site prior to metastasis initiation.

NOTES:

P7

THE FETAL ORIGIN OF INFLAMMATORY MEDIATORS IN GASTROSCHISIS

Cerine Jeanty, MD[1], Sheila Keating, PhD[2], Geoanna Bautista, BS, MA[1], Amar Nijagal, MD[1], Shinjiro Hirose, MD[1], CJ Kim, MD, PhD[3], Roberto Romero, MD[3], Philip Norris, MD[1], Mike Busch, MD, PhD[2], Tippi MacKenzie, MD[1].

[1]University of California, San Francisco, San Francisco, CA, USA, [2]The Blood Systems Research Institute, San Francisco Francisco, CA, USA, [3]Perinatology Research Branch, NICHD/NIH/DHHS, Detroit, MI, USA.

Purpose:
Intestinal inflammation is a key contributor to the morbidity from gastroschisis. While inflammatory mediators in amniotic fluid have been examined, those in cord blood (CB) may be a more accurate reflection of neonatal intestinal health. We examined maternal and CB cytokine profiles of patients with gastroschisis to determine the inflammatory modulators associated with this condition.

Methods:
We obtained maternal and CB samples from 13 gastroschisis patients and 15 term controls with IRB approval. Levels of 39 cytokines and chemokines were analyzed using a human 39plex immunoassay kit (Millipore). Gastroschisis patients were stratified into two groups based on time to full feeds (≤21 days (n=8); >21 days (n=5)). Groups were compared using the Mann-Whitney test; p value <0.05 was considered significant.

Results:
The mean gestational age at delivery was 36 5/7 for gastroschisis and 39 4/7 for controls ($p<0.005$). Levels of the chemokines eotaxin, GCSF, CXCL-2, CCL-2, and the inflammatory mediators IL-1alpha, IL-6 and soluble IL-2 receptor alpha were significantly increased in CB of gastroschisis patients compared to controls (Figure 1). In maternal blood, the levels of these mediators were unchanged and those of other chemokines were significantly decreased (fractalkine, GM-CSF, macrophage inflammatory protein), suggesting a compensatory mechanism to limit inflammation. Patients who were slow to feed had significantly decreased levels of IFN-alpha and IL-12(p40).

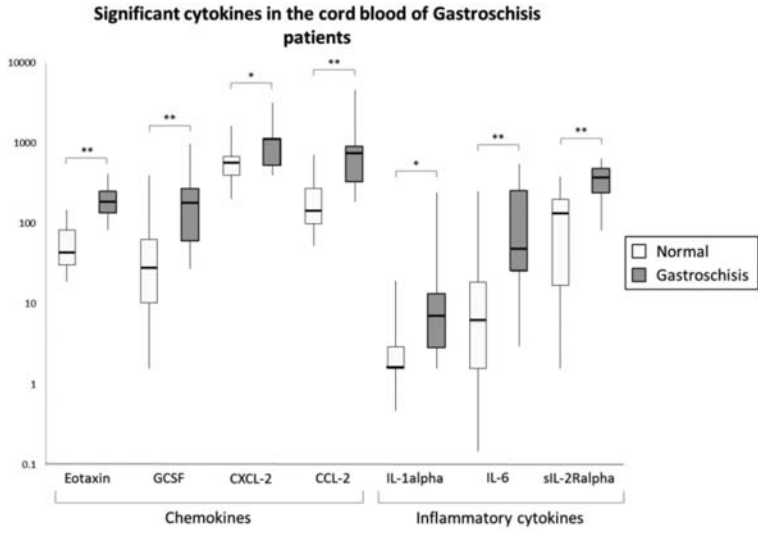


Figure 1: Cytokine levels in the cord blood of normal (n=15) and gastroschisis (n=13) patients. Groups were compared using the Mann-Whitney test (* = p<0.05, ** = p<0.005).

Conclusions:
Gastroschisis fetuses express high systemic levels of cytokines and chemokines which may contribute to bowel inflammation and injury. Our data indicate that the origin of these inflammatory mediators is fetal (with possible maternal compensation) and suggests that their ongoing production may explain the lack of success with amnioexchange for this disease. Elevated levels of IL-6, IL-1, and CCL-2 have been described in other models ischemia/reperfusion injury. Further studies are needed to identify and regulate particular molecular pathways that lead to bowel damage in gastroschisis.

NOTES:

P8

GENERATION OF PERINATAL INDUCED PLURIPOTENT STEM (iPS) CELLS ON A DEFINED, NON-XENOGENIC POLYMER SUBSTRATE

Guihua Jiang, MS, Luis G. Villa-Diaz, PhD, Cynthia DeLong, PhD, K. Sue O'Shea, PhD, Paul H. Krebsbach, DDS, PhD, Shaun M. Kunisaki, MD, MSc.

University of Michigan, Ann Arbor, MI, USA.

Purpose:
Induced pluripotent stem (iPS) cells are differentiated somatic cells that have been genetically reprogrammed into an embryonic stem (ES) cell-like state. Since iPS cells are capable of differentiating into cells derived from any of the three germ layers, these cells have the potential to provide a limitless source of tissue for regenerative replacement therapies. Unfortunately, a major barrier towards clinical application relates to safety concerns associated with xenoculture. The purpose of this study was to determine whether a clinical-grade, methacrylate-based synthetic polymer (PMEDSAH) could be used to support the generation and maintenance of human iPS cells derived from perinatal tissues.

Methods:
After IRB approval, human amniocytes cells underwent lentiviral reprogramming via ectopic expression of Oct4, Sox2, c-Myc, and Klf4 in a defined media on either PMEDSAH or a control xenogenic substrate (Matrigel) derived from Engelberth-Holm-Swarm tumor basement membranes. After three weeks, the cells were evaluated in multiple assays with human ES cells as positive controls.

Results:
Prior to iPS induction, human amniocytes had a mesenchymal morphology and demonstrated variable expression of several pluripotent stem cells markers, including Oct4, Sox2, SSEA3, SSEA4, and Tra-1-60. Three weeks after reprogramming, iPS colonies were successfully generated on both PMEDSAH and control substrate based on morphology and alkaline phosphatase expression. There was strong expression of all pluripotency markers, including NANOG, Oct4, Sox3, SSEA3, SSEA4, Tra-1-60, and Tra-1-81, by immunocytochemical staining. Furthermore, amniocyte-derived iPS cells cultured on PMEDSAH were capable of embryoid body formation and showed neural and muscle progenitor cell differentiation as demonstrated by tubulin beta-III and SMA expression, respectively.

Conclusions:
To our knowledge, this is the first study to show that a defined, non-xenogenic polymer can support the generation and maintenance of iPS cells derived from perinatal somatic tissues. Further analysis of PMEDSAH in the development of clinical-grade iPS cell technologies for pediatric surgical applications is warranted.

NOTES:

P9

HUMAN INDUCED PLURIPOTENT STEM CELL-DERIVED NEURAL CREST STEM CELLS INTEGRATE INTO THE INJURED SPINAL CORD IN THE FETAL LAMB MODEL OF MYELOMENINGOCELE

Payam Saadai, MD[1], Aijun Wang, PhD[2], Yvette Nout, DVM, PhD[1], Timothy L. Downing[2], Katrine Lofberg, MD[1], Michael S. Beattie, PhD[1], Jacqueline C. Bresnahan, PhD[1], Song Li, PhD[2], Diana L. Farmer, MD[1].

[1]University of California, San Francisco, CA, USA, [2]University of California, Berkeley, CA, USA.

Purpose:

Neurological damage in myelomeningocele (MMC) results from abnormal spinal cord development and subsequent in utero trauma. Despite improvements in hindbrain herniation after fetal closure, distal neurological function (e.g. paralysis) remains limited due to damage incurred prior to repair. Neural crest stem cells (NCSCs) can improve locomotor and sensory function in rodent models of spinal cord injury by enhancing regeneration, providing cell replacement, neuroprotection, inducing angiogenesis, and modulating scar formation. This study evaluated the viability of using NCSCs derived from human induced pluripotent stem cells (iPSCs) in the fetal lamb model of MMC.

Methods:

Fetal lambs underwent a surgically induced lumbosacral MMC defect at gestational days 75-76. IPSCs derived from human skin fibroblasts were differentiated into neural crest lineage identified by expression of early migratory NCSC markers. NCSCs were suspended in Matrigel, seeded on a biodegradable nanofibrous scaffold, and implanted at the time of in utero repair (gestational days 100-105, n=2). Lambs were delivered and perfusion fixed at term (gestational days 135-136). Gross necropsy was performed and spinal cords were harvested for histopathological analysis.

Results:

IPSC-derived NCSCs demonstrated expression of early NCSC markers (e.g. nestin, vimentin) before implantation. Immunohistological staining for human-specific antibodies (NuMA) was strongly positive in repaired lambs at the MMC lesion site, confirming the local retention and survival of the transplanted human cells. Concurrent staining for neurofilaments (NFM) to identify spinal cord axonal elements, demonstrated integration of human stem cells within spinal cord tissue [Figure 1]. No tumors were grossly identified.

Conclusions:

Grafted human iPSC-derived NCSCs survive and integrate with host neural tissue in the injured spinal cord in the fetal lamb model of MMC. This study is the first description of human stem cell engraftment in a model of fetal MMC and supports the feasibility of using NCSCs to address spinal cord damage in MMC.

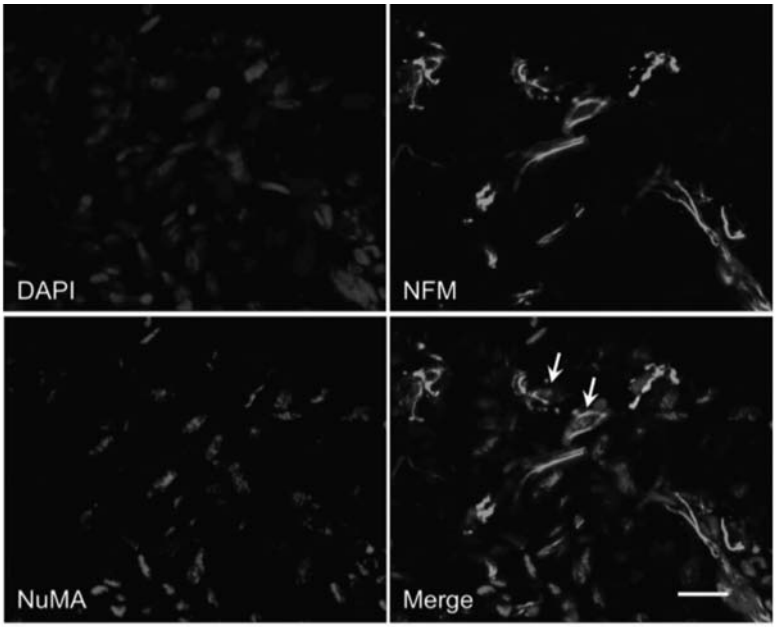


Figure 1. Transplantation of human iPSC derived NCSCs in the injured spinal cord in the fetal lamb model of MMC. Immunofluorescence staining of axon marker NFM (green) and human nuclei antigen NuMA (red) in a cross section of the repaired spinal cord tissue. Arrows indicate the co-localization of NFM and NuMA. Scale bar = 10 μm.

NOTES:

P10

HAEMATOPOIETIC STEM CELLS DERIVED BOTH FROM SHEEP AND HUMAN AMNIOTIC FLUID ENGRAFT AFTER TRANSPLANTATION: POTENTIAL FOR *IN UTERO* AUTOLOGOUS GENE/CELL THERAPY

Steven Shaw[1], Anna L. David[2], Mike Blundell[3], Kuang-Han Lee[2], Steven Howe[3], Caterina Pipino[1], Panagiotis Maghsoudlou[1], Jane Lin[1], Anthony Atala[4], Simon Eaton[1], Agostino Pierro[1], Christopher D. Porada[4], Adrian Thrasher[3], Paolo De Coppi[1].

[1]Surgery Unit, ICH, University College London, London, United Kingdom, [2]Institute for Women's Health, University College London, London, United Kingdom, [3]Molecular Immunology Unit, ICH, University College London, London, United Kingdom, [4]Wake Forest Institute for Regenerative Medicine, North Carolina, NC, USA.

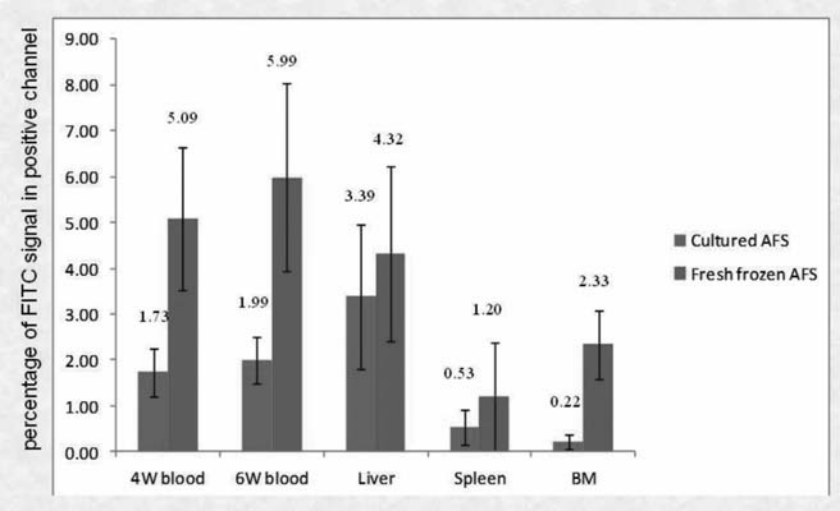
Purpose:
Mouse amniotic fluid c-Kit(+)/Lin(-) stem (AFS) cells display hematopoietic potential. Using recently developed sheep-specific CD34 antibody and established isolation system we explored haematopoietic function of both sheep/human haematopoietic potential after in utero stem cell translantation.

Methods:
Human AFS cells (hAFSC) were isolated as previously described (1,2) while sheep CD34+ sheep AFS cells (sAFSC) were isolated from AF collected at 59.5±4.5 days. hAFSC were transplanted into peritoneal cavity of every fetal mouse from 6 mothers. The peripheral blood of recipient mice was analysed at 4-weeks after birth for engraftment by flow-cytometry using anti-human beta2-microglobin antibody. PCR and immuno-staining was performed on neonatal tissues collected at 6-weeks. The bone marrow was assayed for colony-forming cells (CFC). Differently, sAFSC were transduced overnight (HIV-SFFV-eGFP, MOI=50) and injected either intravenously into NOD-SCID-gamma (NSG) mice (3x10^5, N=4 per group) or by peritoneal injection back into donor fetuses (n=7; 2x10^4 sAFSC) by ultrasound-guided.

Results:
Engraftment of fresh hAFS cells was significantly higher than cultured hAFS cells according to flow-cytometry analysis of peripheral blood and bone marrow ($p<0.05$), also slightly higher in liver and spleen (Figure and Table). Bone marrow harvested from transplanted animals could generate colonies of human origins. Similarly, NSG mice transplanted with sAFSC showed 3-months after transplantation cells of donor origini in peripheral blood, spleen, liver, and BM. Moreover, five lambs injected with autologous CD34+ cells survived to birth (71.4%); peripheral blood of all lambs contained GFP+ cells (2.3-3.8%), maintained at 4-weeks of age.

Conclusions:
hAFSC and sAFSC have haematopoietic potential. There is a potential for establishing autologous in utero gene/cell therapy approach.

Reference:
1. Nat Biotechnol, 2007.
2. Blood, 2009.



NOTES:

P11

IN UTERO TRANSPLANTATION FOLLOWED BY POSTNATAL ENGRAFTMENT CAN BE ACHIEVED WITH PRIMARY ADULT HUMAN HEPATOCYTES IN PIGS

James Fisher, MD[1], Joseph Lillegard, MD, PhD[1], Travis Mckenzie, MD[1], Peter Wettstein, PhD[1], Markus Grompe, MD[2], Brad Feltis, MD, PhD[3], Scott Nyberg, MD, PhD[1].

[1]Mayo Clinic - Rochester, Rochester, MN, USA, [2]Oregon Health and Sciences University, Portland, OR, USA, [3]Childrens Hospitals and Clinics of Minnesota, Minneapolis, MN, USA.

Purpose:
Primary human hepatocytes can be e pan ed in tyrosinemic mice. This occurs because native hepatocytes are metabolically defective allowing selective growth of transplanted human hepatocytes, and their immune system is altered preventing rejection of transplanted human hepatocytes. The gene defect causing tyrosinemia has been engineered into pigs. However, these pigs have unaltered immune systems. Therefore, we hypothesize that *in utero* transplantation of human hepatocytes will avoid the need for postnatal immunosuppression and improved engraftment.

Methods:
Fetal pigs received liver injections of $1x10^7$ human hepat cytes at gestational day 40. Piglets were delivered by cesarean-section at gestational day 115. *In utero* injected piglets received $4x10^7$ human hepatocytes by direct liver injection 3 days postnatally. Using ELISA, serum was analyzed for human albumin at 2, 4, and 6 weeks postnatally. Piglets were euthanized at 6 weeks, and their livers harvested and examined by immunohistochemistry (IHC), PCR and fluorescence *in situ* hybridization (FISH) for human specific sequences.

Results:
In utero injected and postnatally engrafted piglets using human hepatocytes showed significant serum levels of human albumin at 2, 4, and 6 weeks of life. RT-PCR from liver tissue was strongly positive for human albumin. The presence of human hepatocytes was confirmed by IHC and FISH analysis. PCR amplification of human beta-2 microglobulin was obtained.

Conclusions:
Exposure of fetal pigs to human hepatocytes early in gestation allows for engraftment of human hepatocytes after birth. These engrafted hepatocytes persist and continue to function postnatally. *In utero* transplantation followed by postnatal engraftment may provide a future means of expansion of human hepatocytes in tyrosinemic pigs.

NOTES:

P12
INTRA-AMNIOTIC DELIVERY OF EXPANDED AMNIOTIC-DERIVED NEURAL PROGENITOR CELLS IN A SYNGENEIC MODEL OF SPINA BIFIDA

Christopher G. Turner, MD, MPH[1], Elliot C. Pennington, MD[1], Fabienne L. Gray, MD[1], Azra Ahmed, BS[1], Yang D. Teng, MD, PhD[2], Dario O. Fauza, MD[1].

[1]Children's Hospital Boston, Boston, MA, USA, [2]Brigham and Women's Hospital, Boston, MA, USA.

Purpose:
Neural stem cells (NSCs) can promote variable fetal spinal cord repair after surgical local delivery in a xenologous model of spina bifida. The amniotic fluid can be a source of NSCs in the setting of experimental neural tube defects. Using a syngeneic rodent model, we sought to determine the fate of expanded amniotic-derived NSCs after simple intra-amniotic injection in fetuses with spina bifida

Methods:
After IACUC approval, 20 pregnant Lewis dams received retinoic acid on gestational day 10 (E10; term=E21-22) for induction of fetal neural tube defects. Ten dams served solely as amniotic fluid donors on E20, when epigenetic isolation of amniotic-derived NSCs was performed and confirmed by the formation of neurospheres and expression of Nestin and Sox-2. NSCs were expanded in culture and labeled with 5-bromo-2'-deoxyuridine (BrdU). The remaining 10 dams received intra-amniotic injections of the processed NSCs ($1.5x10^4$ cells in 50 μL), blindly in all their viable fetuses (n=37) on E17. Animals were killed before term. Fetuses with spina bifida underwent histological screening for the presence of donor NSCs via immunohistochemistry for BrdU.

Results:
Among the 20 fetuses that received intra-amniotic injections and were still viable at euthanasia, 16 (80%) had either isolated spina bifida, or spina bifida associated with encephalocele. Donor cells were identified in 93.3% (14/15) of the fetuses that could be analyzed, selectively populating the neural placode and/or adjacent nerve roots, typically in clusters and retaining an undifferentiated morphology. They were present predominantly on exposed neural surfaces, though a few were detected deeper in neighboring neural tissue.

Conclusions:
The amniotic cavity can serve as a route of administration of neural stem cells in the setting of experimental spina bifida. Simple intra-amniotic delivery of neural stem cells may be a practical adjuvant to novel strategies for the treatment of spina bifida.

NOTES:

P13

NONINVASIVE MEASUREMENTS OF CARDIAC HEMODYNAMIC AND TISSUE PERFUSION INDICES IN NORMAL INFANTS

Eric L. Long, MD[1], Barbara Weaver, RN[1], Joshua Glenn, MD[1], Robert Vogel, PhD[2], Andrew Bozeman, MD[1], Brandon Lerner, BA[1], Renee Kleris, BA[1], J seph Van de Water, MD[1], Don K. Nakayama, MD[1], Misael Rodriguez, MD[1].

[1]Medical Center of Central Georgia, Macon, GA, USA, [2]Georgia Southern University, Statesboro, GA, USA.

Purpose:
Neonatal care lacks reproducible and clinically relevant measurements of hemodynamic indices (stroke volume, SV; cardiac output, CO; cardiac index, CI) and end-organ oxygenation (cerebral, renal, splanchnic). Two technological innovations have allowed non-invasive, real-time measurement of both: Electrical cardiometry (EC) detects ascending aorta erythrocyte orientation changes to provide hemodynamics; near-infrared spectroscopy (NIRS) assesses regional oxygen delivery as regional oxygen saturation (rSO2). Despite their promise EC and NIRS have not been studied thoroughly in surgical infants. A first step is to verify that EC and NIRS provide reproducible measurements that correlate under steady-state conditions in healthy infants.

Methods:
An IRB-approved observational study with parental consent of EC (ICON, Cardiotronic Inc., La Jolla, CA) and NIRS (INVOS, Covidien/Somanetics, Boulder, CO) in 3 healthy asymptomatic infants (1.9-2.5 kg, 34-41 wk gestation, 2-15 wk of age) over 5 hours of observation. Coefficients of variation (CoV) estimated reproducibility. Linear regression gave correlations between hemodynamic indices and rSO2, p significant at < 0.05.

Results:
EC gave measurements of CO and CI with CoV that ranged from 11.2%-15.6%, SV having more variability, 16.4%-18.0%. Cerebral rSO2 were most consistent (CoV 5.2%-9.4%), followed by renal (8.1%-14.8%) and splanchnic rSO2 (18.4%-20.6%). Strongest correlations between EC and NIRS were with SV and renal perfusion ($r=0.58$, $p<0.0001$), but significant correlations also were present between hemodynamic indices and cerebral (with CO and CI; $r=0.39$, $p<0.0001$) and splanchic rSO2 (with CI; $r=0.38$ $p<0.0001$).

Conclusions:
Data variation limits clinical decisions based on single EC and NIRS measurements. Research is needed to determine the value of EC and NIRS in detecting trends over longer periods, especially during the therapeutic administration of cardiotonic and vasoactive medications. Of interest is whether the technology can characterize the changes in hemodynamics and perfusion associated with surgical conditions such as necrotizing enterocolitis, gastroschisis, and patent ductus arteriosus.

NOTES:

P14
RENAL REPLACEMENT THERAPY IN NEWBORNS: IS THE HEROIC EFFORT WARRANTED?

Lena Perger, MD[1], Reed Dimmit, MD[2], Carroll M. Harmon, MD, PhD[3].

[1]Department of Surgery, Scott & White Hospital, Texas A&M College of Medicine, Temple, TX, USA, [2]Department of Pediatrics, University of Alabama at Birmingham, Childrens Hospital of Alabama, Birmingham, AL, USA, [3]Department of Surgery, University of Alabama at Birmingham, Children's Hospital of Alabama, Birmingham, AL, USA.

Purpose:
The aim of this study was to review surgical aspects of care and outcomes in newborns requiring renal replacement therapy (RRT).

Methods:
After the study protocol was approved by the IRB, a retrospective chart review was performed on all patients who received RRT in the first 30 days of life between 2006 to 2010.

Results:
23 patients were identified, 18 boys and 5 girls. Median gestational age and birth weights were 36 (range 30-39) weeks and 2.74 (range 1.98-3.69) kg respectively. 16 patients had end-stage renal disease (ESRD) and 7 patients had inborn errors of metabolism. Initial therapy was continuous veno-venous hemofiltration (CVVH) in all patients. A total of 101 surgical procedures were performed, with a median of three operations per patient (range 1-13). 36 hemodialysis lines were placed with a maximum of five catheters in one patient. Two patients died within the first week and complications occurred in 100% of infants who survived beyond one week of life. Most common complications were dialysis circuit clotting (78%), bleeding (52%), bacteremia (30%), cardiopulmonary resuscitation (30%), multisystem organ failure (26%), pneumothorax (26%), catheter occlusion (17%), and venous thrombosis (17%). Mortality in ESRD patients was 69%, and 57% in infants with metabolic disease. 60% of deaths occurred by 21 days of age (range 5-992). Out of eight survivors, five are severely and two are moderately developmentally delayed. Only the infant with transient hyperammoniemia survived without long-term sequelae.

Conclusion:
Aggressive treatment of critically ill newborn infants with renal and metabolic disease using CVVH is becoming increasingly attempted. Though at times lifesaving, this approach appears to have an extraordinarily high mortality and morbidity and warrants careful clinical investigation in the future. Knowledge of potential risks and benefits is crucial for pediatric surgeons when asked to provide dialysis access for this group of patients.

NOTES:

P15

TEMPORARY STIMULATION ALLOWS EXPANSION OF INDICATIONS FOR GASTRIC ELECTRICAL STIMULATION

Jillian McLaughlin, BS[1], Archana Kedar, MD[2], Christopher D. Jolley, MD[1], Tom Abell, MD[2], Saleem Islam, MD, MPH[1].

[1]University of Florida, Gainesville, FL, USA, [2]University of Mississippi, Jackson, MS, USA.

Purpose:
Gastric and intestinal motility disorders remain difficult to diagnose and treat in children and adolescents, often leading to poor quality of life for the patients and families. We have previously described the successful use of gastric electrical stimulation (GES) for gastroparesis (GP). This study details our experience using temporary stimulation (tGES) in a variety of motility disorders including gastroparesis.

Methods:
After IRB approval, we performed a retrospective review of all children who underwent tGES via endoscopic or trans gastrostomy routes. Data regarding demographics, primary and comorbid conditions, hospital course and effects of tGES were recorded as well as outcomes.

Results:
74 patients underwent tGES. 78% were female, and 75% were Caucasian. Endoscopic approach was employed in 69% while 36% underwent trans gastrostomy tGES; 34% underwent more than one attempt at tGES. The most common indication was GP in 87%, while cyclical vomiting, and generalized motility disorders were seen in 13%. We noted that there were a greater number of responders to tGES in the GP patients with regards to symptom scores (nausea, vomiting and total symptom score) (63%) vs. non GP cases (25%). A majority (70%) of the responders in both groups have gone on to receive permanent implants, with insurance issues preventing the remaining 30%. In the non GP group, the non responders have had persistent poor quality of life with requirements for TPN, GJ tube feeds, and prolonged hospitalization.

Conclusions:
tGES is a minimally invasive means of selecting and screening patients who will respond to stimulation. It has allowed us to apply the therapy to patients who would have traditionally not been considered as candidates. We recommend the use of tGES screening in all cases prior to considering a permanent GES implantation to improve response rates.

NOTES:

P16
IMPROVING PERIOPERATIVE PERFORMANCE: THE USE OF THE ELECTRONIC HEALTH RECORD AND OPERATIONS MANAGEMENT

Robert P. Foglia, MD[1], Adam C. Alder, MD, FACS[1]; Gardito Ruiz, MHA[2].

[1]UT Southwestern, Dallas, TX, USA, [2]Children's Medical Center Dallas, Dallas, TX, USA.

Purpose:
Perioperative (PO) services require the orchestration of multiple staff, space and equipment. Our aim was to identify whether implementation of an electronic health record (EHR) and operations management (OM) improved PO performance .

Methods:
We compared 2006, pre EHR and OM implementation, to 2010, post implementation. The EHR allows : identification of delays and the accountable service or person; and collection and collation of data for analysis in multiple venues, including operational, financial and quality.. OM consisted of: communication to staff of PO vision and metrics , obtaining credible data and analysis, implementation of performance improvement processes. Metrics assessed included: operative cases; first case on time starts (FCOTS) ; reason for delay; and operating revenue.

Results:
In 2006, 19,148 operations were performed (13,545 in the Main OR(MOR) area, and 5,603 at satellite locations), FCOTS was 12%, reasons for first case delay were not identifiable, and operating revenue was $115.8 M overall, with $78.1 M in the MOR. In 2010, cases increased to 25,856 (+35%), MOR increased to 13,986 (3%); FCOTS improved to 46%; operations outside the MOR increased to 11,870 (112%); case delays were ascribed to: nurses 7%, anesthesiologists 22%, surgeons 33%, and other (patient, hospital) 38%. 5 surgeons (7%) accounted for 29% of surgical delays and 4 anesthesiologists (8%) for 45% of anesthesiology delays; operating revenue increased to $177.3 M (+53%) overall, and for the MOR rose to $101.5 M (+30%).

Conclusions:
The EHR and OM resulted in credible data, promptly sharing the metrics, and pinpointing individual provider performance. Implementation of these strategies allowed us to shift cases between facilities, reallocate OR blocks, increase FCOTS four fold, and operative cases by 35%, and was associated with a 53% increase in operating revenue. The fact that revenue increase was greater than case volume (53% vs. 35%) speaks for optimized performance.

NOTES:

Poster Session II
Clinical

Sunday, May 20, 4:15 – 6:00 p.m.

P17
REDUCTION IN FREQUENCY OF ETHANOL LOCK THERAPY RESULTS IN A MARKED INCREASE IN CENTRAL VENOUS ACCESS DEVICE(CVAD) SEPSIS: FDA-INITIATED MANDATES OR A TRAGEDY IN THE MAKING?

Matthew W. Ralls, MD, R. Alex Blackwood, MD, Meghan A. Arnold, MD, Pamela I. Brown, MD, Luisa Partipilo, Pharm D BCNSP, James Dimond, Daniel H. Teitelbaum, MD.

University of Michigan, Ann Arbor, MI, USA.

Background:
Ethanol lock therapy(ELT) is increasingly used for pediatric intestinal failure(IF) patients to prophylax against blood stream infections(BSI). The dosing and frequency of ELT remains undefined. Recent scrutiny of pharmaceutical production facilities by the FDA led to voluntary shutdown of the sole supplier of ethanol, resulting in a nationwide shortage. To conserve our ethanol supply, we reduced ELT frequency from daily to once or twice weekly. We hypothesized that reduced ELT frequency would be associated with a significant increase in BSI incidence.

Methods:
We retrospectively reviewed our TPN dependent IF children. Primary outcome measure was CVAD infections per 1,000 catheter days after ELT frequency reduction. Data were compared(via paired t-test) to the same group over the 1 year prior to ethanol shortage, as well as historical controls with and without daily ethanol use. Secondary measures included organisms causing BSI.

Results:
During the shortage, 13 outpatients received ELT. Ethanol shortage affected eight patients. Mean ±SD age was 9.09±7.77 years. Mean BSI rate per 1,000 catheter days was 0.68±1.27 before ELT shortage. This significantly increased to 6.16±2.49 after frequency reduction ($p<0.001$). This rate was also significantly elevated compared to the entire group of IF children on ELT (1.3±3.0), and were not significantly different than historical IF controls not on ELT (8.0±5.4). Seven of eight children developed BSI within 142 days of frequency reduction, resulting in 6/7 hospitalizations and 2 ICU admissions for septic shock. Mean length of stay (15.5 days) averaged $104,783.59(±111,034.87) in charges. Six of seven infected catheters were removed and required replacement, another developed subglottic stenosis from prolonged intubation and required tracheostomy. Causative organisms included gram negatives(6); MRSA(1) and Candida sp(1).

Conclusions:
ELT frequency reduction resulted in complete failure in BSI prophylaxing. Nationwide shortage of this one drug has been costly both financially and in patient morbidity.

NOTES:

P18

INCREASING AGE AT TIME OF *PECTUS EXCAVATUM* REPAIR IN CHILDREN: EMERGING CONSENSUS?

Dominic J. Papandria, MD, Gezzer Ortega, MD, Jose H. Salazar-Osuna, MD, Jeffrey Lukish, MD, Paul Colombani, MD, MBA, Fizan Abdullah, MD, PhD.

Johns Hopkins University School of Medicine, Baltimore, MD, USA.

Purpose:
Advances in surgical technique and understanding of pectus excavatum repair continue to change practice patterns over the last decade. The present study examines trends in operative age in a nationwide administrative database.

Methods:
A cross-sectional analysis was performed using State Inpatient Databases data from 1998-2009. Elective admissions were selected if they contained both a diagnosis code for pectus excavatum and a procedure code for operative repair. Descriptive statistics were computed and patients were subdivided by operative year for further analysis. Groups were compared using Pearson's chi-square (categorical variables) and the Kruskal-Wallis test (non-normally distributed continuous variables).

Results:
A total of 4,858 cases were identified. The mean age at operation was 13.5 (SD 3.3) years, and length of stay averaged 4.6 (SD 1.7) days. Mean patient age increased steadily from 11.8 to 14.4 years ($p<0.001$) and ranges have narrowed with patients most frequently undergoing repair at age 16 by 2009 (Figure 1). The proportion of patients under 9 years of age undergoing repair has fallen steadily from 24.2% in 1998 to 3.5% in 2009 ($p<0.001$).

Conclusion:
Over the twelve-year period examined, the age at repair of pectus excavatum has continued to trend higher. This is consistent with previous findings and with overall trends in patient selection reported in the literature. This selection pattern may reflect evolving consensus regarding optimal management of pectus excavatum and provide clinical guidance regarding appropriate referral and intervention.

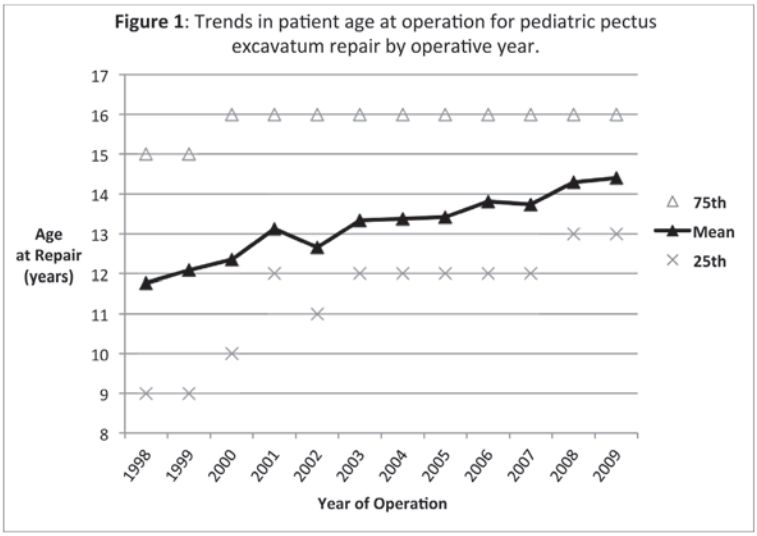


Figure 1: Trends in patient age at operation for pediatric pectus excavatum repair by operative year.
Age at Repair (years)
17
16
15
14
13
12
11
10
9
8
1998
1999
2000
2001
2002
2003
2004
2005
2006
2007
2008
2009
Year of Operation
75th
Mean
25th

NOTES:

P19

VENTRICULOPERITONEAL SHUNT FAILURE AND ABDOMINAL SURGERY: EFFECT OF ABDOMINAL SURGERY ON OUTCOME

Angela Li Ching Ng[1], Gillian Humphrey, MD, MBChB, FRCS (paedsurg)[2], Ian Kamaly, MD, MBChB, FRCS (SN)[2], Muhammad Baath, MD, MBChB, FRCS[2].

[1]University Hospital South Manchester, Manchester, United Kingdom, [2]Royal Manchester Children's Hospital, Manchester, United Kingdom.

Purpose:
To investigate the impact and effect of timing of abdominal surgery on ventriculoperitoneal (VP) shunt survival in children.

Methods:
We conducted a retrospective data collection on patients aged 0-18 years undergoing primary shunt insertion or revision between the 1st January 2008 and 31st December 2010. We categorized subjects into "shunt-only" (SO) and "shunt and abdominal surgery" (SA) groups. We calculated and compared shunt survival times in the two groups. In the SA cohort, we analyzed shunt survival times for shunts inserted at 6 months, 12 months and anytime beyond 12 months of abdominal surgery. All shunts were followed up until the 1st June 2011. Kaplan-Meier survival curves and log rank were used for statistical analyses.

Results:
342 shunts in 109 patients were included in this study. 118 of the shunts were from the SA group. The median shunt survival time was 3.75 months (95% CI = 1.01-6.47) and 22.6 months (95% CI = 8.76-36.4) in the SA and SO groups respectively. Abdominal surgery increased risk of shunt failure by 54.7% per abdominal procedure (log rank = 16.6, $P < 0.01$). In the SA group, 35, 29 and 54 shunts were inserted within 6 months, 12 months and at any time beyond 12 months of abdominal surgery, respectively. The median shunt survival was 1.48 months if abdominal surgery occurred within 6 months (95% CI = 0.57-2.65) and 12 months (95% CI = 0.00-3.09) from a shunt procedure. Beyond 12 months, there was a five-fold increase in median shunt survival time to 7.39 months (95% CI = 0.00-20.1, log rank = 23.2, $p<0.01$).

Conclusion:
Abdominal surgery increases the risk of VP shunt failure. Delaying abdominal surgery by one year from shunt insertion may prolong shunt survival.

NOTES:

P20

RESULTS OF SURGICAL PORTAL SYSTEMIC SHUNT FOR SEVERE PORTAL HYPERTENSION IN CHIDREN WITH BILIARY ATRESIA AFTER A SUCCESSFUL KASAI PROCEDURE

Florent Guerin, MD[1], Stephanie Jaszienski, MD[1], Lionel Charre, MD[2], Frédéric Gauthier, PhD[1], Virginie Fouquet, MD[1], Hélène Martelli, PhD[1], Sophie Brachereau, MD[1].

[1]Bicêtre Hospital, Le Kremlin Bicêtre, France, [2]René Dubos Hospital, Pontoise, France.

Purpose:
To assess our experience with surgical portal systemic shunt (SPSS) for portal hypertension (PH) in biliary atresia (BA), and the transplant free survival (TFS) after the procedure.

Methods:
From 1975 to 2008, 34 patients with BA were submitted to SPSS for severe PH following an initial successful Kasai procedure. We assessed the long term survival without liver transplantation (LT). Results were expressed in median [range]; survivals were compared with the Gehan-Breslow-Wilcoxon test. $P<0.05$ was significant. No need for IRB approval.

Results:
Age at onset of PH was 24 months [1-96], 29 patients (85%) had gastrointestinal bleeding (GIB) prior to SPSS. Surgery was performed at 5.5 years [2-13.5]old. Serum conjugated bilirubin level (cBL) was ≤ 20 µmol/l in 23 patients (68%) and was 34umol/l [26-85] in 11 (32 %). Two patients had mild pulmonary shunting, and none had pulmonary hypertension. Surgery consisted in 19 spleno-caval /renal and 15 mesenteric/portal-caval anastomosis. Median follow-up was 121 months [1-264]. PH recurred in 5 (14%) patients with spleno-renal shunts. Long term complications consisted in episodic hepatic encephalopathy in 5 patients, pulmonary hypertension in 5; and 2 de novo pulmonary shunts. Five patients died without LT, 25 months [1-285] following SPSS, 4 of them before the setting of the pediatric LT program (1986), and the last in 1992, from fulminant liver failure. Six patients had a LT at 67 months [19-228] following SPSS, and 5/6 survived. Global TFS at 2, 5, 10 years after SPSS were respectively: 88%, 81%, and 70%. TFS was better if cBL before SPSS was<20umol/l (95%, 90% and 80% vs. 72%, 62% and 52%) (p=0.04).

Conclusions:
Surgical portal systemic shunt for severe portal hypertension in biliary atresia offers a long life-span without liver transplantation, provided that it is limited to anicteric patients without cardiovascular complications.

NOTES:

P21

DESMOID FIBROMATOSIS IN CHILDREN AND ADOLESCENTS: A CONSERVATIVE APPROACH TO MANAGEMENT

Joshua N. Honeyman, Till-Martin Theilen, Molly Knowles, Margaret M. McGlynn, Eric J. Stanelle, Emily R. Christison-Lagay, Paul A. Meyers, Michael P. La Quaglia.

Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

Purpose:
Although desmoid fibromatosis is a biologically benign fibrous tumor associated with good overall survival, it is also associated with high rates of recurrence and significant morbidity. To determine whether a period of non-operative management had any impact on overall survival (OS) or progression-free survival (PFS), we reviewed our institutional experience with this tumor.

Methods:
With IRB waiver, we retrospectively reviewed 81 consecutive cases of desmoid fibromatosis diagnosed between 1970 and 2010. Medical records were reviewed for data on demographics, diagnosis, treatment, and outcomes. Survival data were analyzed using the Kaplan-Meier method. Significance was determined using the log-rank test.

Results:
The median age at diagnosis was 15.8 years; the male-to-female ratio was 1:1. Ten patients had familial adenomatous polyposis (FAP). Tumors were located in the extremities in 48 patients (59%), abdominal/chest wall in 15 (19%), head/neck in 6 (6%), and the abdomen in 12 (15%). Tumor size was > 5 cm in 41 patients and < 5 cm in 20 patients. Fourteen patients received neoadjuvant chemotherapy; 1 patient received neoadjuvant radiotherapy. Twenty-two patients underwent initial non-operative management; 58 patients underwent primary resection. Twelve patients who were initially observed eventually underwent surgical resection. Five-year OS was 100%. There were 3 late deaths from disease occurring at 6, 11, and 26 years; all deaths occurred in patients with intra-abdominal tumors associated with FAP. PFS was 31.7% at 5 years. There was no significant difference in survival between patients who underwent initial surgical resection compared to those who underwent initial observation (27.6 versus 36.4 years, $p = 0.066$). Resection status, tumor size, tumor location, and FAP were not correlated with PFS.

Conclusion:
Our findings support a conservative approach to the management of desmoid fibromatosis in children and adolescents. Resection should be reserved for tumors that are growing or are causing serious symptoms.

NOTES:

P22
A FALL IN TRANSITIONAL ZONE PULLTHROUGHS IN HIRSCHSPRUNG'S DISEASE FOLLOWING A CHANGE IN PRACTICE - COMPLETION OF AN AUDIT CYCLE

Amiria Catherine Lynch, MBChB, DCH, FRACS, Victoria A. Lane, MBChB, MRCS, Ian D. Sugarman, MB ChB, FRCS (Ed), FRCS (Paed).

Leeds Teaching Hospital NHS Trust, Leeds, United Kingdom.

Purpose:
This institute previously reported a transitional zone pullthrough rate of 15% (13 out of 88 cases). Following this a change of practice was instigated. This paper is to report the results of this change in practice and thus close the audit cycle.

Methods:
Previously, at this institute, standard practice was to send a single point intra-operative biopsy. Following an audit, this changed to that of sending a full thickness donught, with the pathologist reporting on ganglion cell presence and absence of hypertrophied nerve trunks in four quadrants. We report the results of this change from December 1999-June2011 in patients undergoing either a Duhamel or transanal pullthrough.

Results:
Since the senior author joined the institute all pullthroughs performed under his care have had intra-operative donught biopsies. Of the 49 cases performed 3 (6%) had transitional zone pullthroughs. One was because of misinterpretation by an adult pathologist. Of the other two cases, of whom both children were syndromic, they both had macroscopic/microscopic transitional zones greater than 10cm, with the bowel being ganglionic for almost all of the 10cm but one and two hypertrophied nerve trunks respectively, being reported intra-operatively. In both of these cases the pathologists expressed doubt regarding the importance of these nerve trunks in view of the length of normal ganglionation.

Conclusions:
It has been shown that the transitional zone in patients with Hirschsprung's disease is not straight but waved, with peaks and troughs. Many papers report transitional zone pullthoughs and one major reason is that a single point biopsy may not be circumferentially representative. We have shown, by a change of intra-operative practice, that this problem can be reduced as a complication of surgery. Further work is required into the significance, or not, of hypertrophied nerves that appear to persist over great distance, in otherwise normal ganglionic bowel.

NOTES:

P23

THE EFFECTIVENESS OF LAPAROSCOPIC NEEDLE-ASSISTED INGUINAL HERNIA REPAIR (LNAR) IN YOUNG CHILDREN

Christian J. Streck, MD, Aaron P. Lesher, MD, Robert A. Cina, MD, Andre Hebra, MD.

Medical University of South Carolina, Charleston, SC, USA.

Purpose:
Previous studies of laparoscopic pediatric inguinal hernia repair were limited by not only the lack of prospective outcomes monitoring, but also higher recurrence rates. In this study, we prospectively evaluate a single port needle-directed extraperitoneal approach to high ligation of the hernia sac in children.

Methods:
Between January 2009 and September 2011, demographic data, operative data, and complications were prospectively recorded in a clinical outcomes database for all children under age 13 who underwent LNAR at a tertiary care children's hospital. Follow-up occurred at two weeks and one year. A retrospective review of this database was performed following IRB approval.

Results:
371 patients underwent 498 laparoscopic inguinal hernia repairs during the 31-month study period. Patients ages ranged between 13 days-12.8 years (mean±SD, 2.4±2.7 years) and weighed between 1.5 and 62 kg (12.0± 8.8 kg). Right-sided hernias were repaired in 38.0%, left in 20.9%, bilateral in 37.5%. Operative time (mean±SD [range]) was 19.5±6.8 [8-45] minutes for single-sided repair and 26.8±8.4 [12-44] minutes for bilateral repair and was similar between term and pre-term infants. There were no intraoperative complications. Early complications occurred in 6 patients (1.2%): 4 (0.8%) with cellulitis, 1 (0.2%) suture granuloma, and 1 (0.2%) missed contralateral hernia. Late complications occurred in 9 patients (1.8%): two (0.4%) had a hernia recurrence, six (1.2%) had suture granulomas, and one (0.2%) missed contralateral hernia. The overall complication rate was 3.0%, and was slightly higher in pre-term infants (3.7%).

Conclusions:
We conclude that LNAR is safe, efficient, and effective for inguinal herniorraphy in children under age 13 and particularly useful in pre-term infants where dissection of the hernia sac can be avoided. The overall recurrence rate of 0.4% is low. A recurrence rate of 0.7% in pre-term infants is significantly lower than reported in recent retrospective studies of laparoscopic and open hernia repairs.

NOTES:

P24
TEMPORAL RELATIONSHIPS BETWEEN POSITIVE URINE CULTURE AND ONSET OF NECROTIZING ENTEROCOLITIS

Syamal D. Bhattacharya[1], Christoph P. Hornik, MD[1], Abigail Martin, MD[1], Clark Reese, MD[2], C M. Cotten, MD, MHS[1], Margarita Bidegain, MD[1], Phillip B. Smith, MD, MHS[1].

[1]Duke University Medical Center, Durham, NC, USA, [2]Duke University Medical Center, Sunrise, FL, USA.

Purpose:
Although an association between necrotizing enterocolitis (NEC) and endotoxemia has been described, no studies have evaluated the relationship between urinary tract infections (UTI) and NEC. In our practice, we have noted several cases of NEC to be temporally related to the diagnosis of UTI, specifically with Gram negative species. Our objective was to assess the association between prior UTIs and NEC.

Methods:
Using the Pediatrix Medical Group® database of admissions to 305 NICUs from 1997-2011, we identified all infants born <32 weeks gestation and <1500 g birth weight with urine cultures (UCx). Infants with congenital gastrointestinal or urogenital defects were excluded. Primary outcome was diagnosis of NEC within 7 days of a UCx. We used univariable and multivariable logistic regression controlling for gestational age, inotropic support on the day of culture and age on the day of culture to evaluate the association between UCx result and development of NEC.

Results:
We identified 23,799 infants with 43,125 UCx; 2317 (9.7%) infants had a UCx within 7 days of a diagnosis of NEC. Of these, 364 (15.7%) were culture positive. On univariable analysis we observed increased odds of NEC for infants with positive vs. sterile UCx (OR=1.12 [95% CI; 1.07, 1.35]). Gram negative UTIs also increased the odds of NEC (OR=1.26 [1.09, 1.45]). On multivariable analysis, we observed increased odds of NEC following positive UCx (OR=1.39 [1.23, 1.56]). The odds were also higher when the multivariable regression was limited to Gram negative organisms (OR=1.53 [1.31, 1.77]).

Conclusions:
The temporal association between NEC and UTI in premature infants with positive UCx was validated in this cohort. Examination of clinical approaches to UTI, including antimicrobial use and feeding, both of which influence NEC risk, are needed to inform clinicians on the best approach to infants with UTI.

NOTES:

P25

INNOVATIVE TECHNIQUE FOR COVERAGE OF NEONATAL GIANT ABDOMINAL WALL DEFECTS

Alan Coleman, MD[1], Thomas McGill, MD[1], Ruth Gard, RN, BSN, CCRC[1], James Holmes, MD[2], John Griswold, MD[1].

[1]Texas Tech University HSC, Lubbock, TX, USA, [2]Wake Forest University, Salem, NC, USA.

Purpose:
Giant abdominal wall defects (omphalocele and gastroschisis) in neonates present several clinical challenges, including pulmonary hypoplasia, physiologic disturbances (fluid and electrolyte imbalance, nutritional deficiencies) and coverage of the defect. In omphalocele, no membrane in giant defects can be devastating. We present a new method of closure using a biologic wound dressing (Integra) and an autologous non-cultured cells technique (ReCell) in a premature infant, who was prenatally diagnosed with a giant abdominal wall defect with no membrane. This is the first report of this technique in a human subject.

Methods:
A 1975-gram 35-week gestational age infant was born with a giant omphalocele and no membrane. Placement of a silo failed to increase abdominal domain or reduce externalized organs. Continued oncotic losses, hemodynamic instability, concerns for sepsis and increasing respiratory requirements necessitated aggressive planning for abdominal coverage. Integra provided initial coverage without need for donor harvest, allowing for some physiologic recovery almost immediately. ReCell was then selected for the reported ability to generate fully functional and physiologically identical skin without need to harvest large areas or add to operative/preparation time. Skin grafting and cultured epithelial autograft were not considered due to cost, reliability and a lack of necessary surface area. FDA and IRB approval were obtained for off label and compassionate use.

Results:
By postoperative day seven of ReCell application, the patient tolerated feeds and made substantial gains in respiratory requirements. By day 24 most of the defect was covered with early epithelialization and subsequent keratinized skin.

Conclusions:
Use of Integra as a temporizing scaffold followed by application of ReCell has thus far proven to be an effective method of covering a large abdominal wall defect in a neonate. We hope to close the created ventral hernia using component separation or tissue expansion. Image keratinizing skin POD 32.

NOTES:

P26

EPIDEMIOLOGY OF 577 PEDIATRIC FIREARM FATALITIES: A 2-YEAR REVIEW OF THE NATIONAL TRAUMA DATA BANK (NTDB)

Tolulope A. Oyetunji, MD, MPH[1], Adil H. Haider, MD, MPH[2], Augustine C. Obirieze, MD, MPH[1], Michael Fisher, BS[1], Aderon'e O. Oyetunji, MD[3], Edward E. Cornwell III, MD[1], Benedict C. Nwomeh, MD, MPH[4].

[1]Howard University College of Medicine, Washington, DC, USA, [2]Johns Hopkins University School of Medicine, Baltimore, MD, USA, [3]University of Baltimore Merrick School Of Business, Baltimore, MD, USA, [4]Ohio State University College of Medicine, Columbus, OH, USA.

Purpose:
To delineate the epidemiology of pediatric firearm injuries, including ethno-demographic patterns with impact on years of potential life lost (YPLL).

Methods:
2-year review of the National Trauma Data Bank (2007-08) was conducted. Firearm fatalities were identified by mechanism and hospital disposition. Records with age <18 years were included. The data was analyzed by demographic and injury characteristics and YPLL was calculated by ethnicity, using an estimated life expectancy of 75 years.

Results:
A total of 6,255 firearm fatalities were documented, with 577 deaths occurring in the pediatric group. African-Americans accounted for 49.7% of the fatalities, Hispanics, 19.2%; Whites 17.7% and other ethnicity 13.4%. Age range was from 0-17 years (median=16 years). Median injury severity score was 25 (Interquartile range (IQR) 18-32) with a median Glasgow coma scale score of 3 (IQR 3-3). Traumatic brain injury was present in 84.2% of the records. By intent, assault accounted for 72.8%, self inflicted injury 12.7% and unintentional injuries were 8.2%. Most firearm fatalities occurred at home (33.6%), more than any other location. By emergency department (ED) disposition, 29.3% died in the ED, 32.9% were admitted to the intensive care unit and 30.0% were taken to the operating room. By insurance, 26.5% were uninsured (highest proportion), followed by Medicaid at 19.9%. The youngest pediatric group involved was in African-Americans, with 1 out of 10 children <6 years. African-Americans had a total of 17,446 YPLL, Hispanics 6776 YPLL and Whites 6718 YPLL.

Conclusion:
Firearm fatalities still remain a violent and ethnic issue, with African-American bearing the lion share of the burden. Centuries of productive life are lost as a result of gun violence, with little or no impact from secondary prevention. Focused primary prevention in terms of gun control and education is required to stem the wave of violence, most especially with the involvement of pre-teenage children.

NOTES:

P27

PRIMARY TUMOR LOCATION AND GRADE DETERMINE SURVIVAL IN PEDIATRIC LIPOSARCOMA

Eric J. Stanelle, MD, Emily R. Christison-Lagay, MD, Samuel Singer, MD, Paul A. Meyers, MD, Michael P. La Quaglia, MD.

Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

Purpose:
Liposarcoma is extremely rare in the pediatric population, so few reports on pediatric liposarcoma have been published. To identify prognostic factors and determine treatment outcomes (in terms of overall survival), we reviewed our institutional experience treating pediatric patients with liposarcoma.

Methods:
With IRB approval, we retrospectively reviewed all pediatric patients (age ≤ 22 years) with pathologically confirmed liposarcoma treated between 1960 and 2011. Demographic data and tumor characteristics were evaluated. Histologic subtype, tumor location, margin status, recurrence, and adjuvant therapy (radiotherapy or chemotherapy) were analyzed for any correlation with overall survival.

Results:
We identified 35 patients, 20 males and 15 females, with a mean age of 17.26 years. Twenty-two (63%) patients had peripheral tumors and 13 (37%) had centrally located tumors. Histologically, 30 (86%) tumors were low-grade (25 myxoid and 5 well-differentiated) and 5 (14%) were high-grade pleomorphic. No tumors were dedifferentiated. Eleven patients experienced a recurrence; of these patients, 9 (82%) had central tumors and 2 (18%) had peripheral lesions. All nonsurvivors (n = 8) had central disease. Overall 5-year survival was 78%, with a median survival time of 5.31 years (range, 0.26-30.30 years). Tumor grade ($p = 0.003$), histologic subtype ($p = 0.012$), and primary location ($p = 0.001$) all correlated with survival, as did stage ($p < 0.001$) and margin status ($p = 0.001$). Adjuvant therapy did not improve survival.

Conclusions:
Primary tumor location, tumor grade, and surgical margin status are strongly correlated with poor survival in pediatric patients with liposarcoma. Peripheral lesions are associated with excellent prognosis, and surgery alone with negative margins appears to be adequate treatment in these cases. Low-grade myxoid liposarcoma is more common. High-grade, centrally located tumors are associated with poor prognosis. The role of adjuvant therapy needs further elucidation.

NOTES:

P28
PEDIATRIC SYNOVIAL SARCOMA: PROGNOSTIC FACTORS, MANAGEMENT OF PULMONARY METASTASIS, AND SURVIVAL OUTCOMES

Eric J. Stanelle, MD, Emily R. Christison-Lagay, MD, Sara J. Abramson, MD, Anita P. Price, MD, John H. Healey, MD, Samuel Singer, MD, Paul A. Meyers, MD, Michael P. La Quaglia, MD.

Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

Purpose:
Treatment of synovial sarcoma (SS) is challenging because of its unpredictable clinical behavior compared to other sarcomas. We reviewed our institutional experience with pediatric SS patients, including those with pulmonary metastasis, to identify prognostic indicators and survival outcomes.

Methods:
With IRB approval, we retrospectively reviewed all pediatric patients (age <22 years) with pathologically confirmed SS treated over a 40-year period (1970–2010). Patient and clinical characteristics were evaluated for prognostic significance and number and type of surgical metastasectomy. Survival data were stratified based on treatment modality, tumor characteristics, and recurrence.

Results:
A total of 111 patients were identified. The mean age was 14.9 years (range, 4-22 years). Sixty-seven tumors (60%) were monophasic, 42 (38%) were biphasic, and 2 (2%) were of unknown histology. Median follow-up was 5.1 years (range, 0.74-36.8 years). The 5-year overall survival (OS) rate was 73%. Local and metastatic recurrence decreased 5-year OS rates to 61% and 46%, respectively. Tumor size >5 cm ($p=0.001$) and invasiveness ($p=0.002$) correlated with OS. Histology, tumor location, and margin status did not correlate with OS. Radiotherapy improved 5-year survival in patients with monophasic tumors ($p=0.041$), large tumors ($p=0.001$), and recurrent disease ($p<0.001$). Chemotherapy did not improve 5-year survival in any category.

Forty-one patients with SS had pulmonary metastasis. Thirty-one (76%) underwent pulmonary metastasectomy, for a total of 72 surgical resections (range, 1-8/patient). Two- and 5-year OS rates for patients who underwent metastasectomy were 65% and 24%, respectively. Unresected pulmonary disease correlated with shorter OS ($p<0.001$). Neither palliative debulking (incomplete metastasectomy) nor postoperative radiotherapy improved survival.

Conclusions:
We found that pulmonary metastasectomy correlates with improved survival, and radiotherapy of the primary site is effective in select patients. The role of chemotherapy in this group of patients warrants future study.

NOTES:

P29

OUTCOME AND RISK FACTORS FOR PATIENTS WITH PERINEAL AND PERIANAL RHABDOMYOSARCOMA–A REPORT FROM THE CHILDREN'S ONCOLOGY GROUP SOFT TISSUE SARCOMA COMMITTEE

Roshni Dasgupta, MD, MPH[1], James Anderson, PhD[2], Andrea Hayes-Jordan, MD[3], Torun Yock, MD[4], Sheri Spunt, MD[5], Doug Hawkins, MD[6], David Rodeberg, MD[7].

[1]Cincinnati Childrens Hospital Medical Center, Cincinnati, OH, USA, [2]University of Nebraska Medical Center,Omaha, NE, USA, [3]MD Anderson Hospital, Houston, TX, USA, [4]Massachusetts General Hospital, Boston, MA,USA, [5]St. Jude's, Memphis, TN, USA, [6]Seattle Childrens Hospital, Seattle, WA, USA, [7]Eastern Carolina University,Greenville, NC, USA.

Purpose:
To describe patient demographics, determine risk factors and outcomes for patients with perineal and perianal rhabdomyosarcoma**.**

Methods:
The records of 40 patients (30 perineal and 10 perianal) enrolled on IRS-III, IRS-IV, D9602 and D9803 (1984-2005) were reviewed. Median follow up of all patients was 6.9 years**.**

Results:
Twenty (50%) of patients were male and 23 (57%) were between 1-9 years of age. Twenty-five (66%) patients had tumors >5cm, 28 (70%) were alveolar histology, 24 (60%)were stage 3 and 17 (43%)were group 3. Estimated 5 year event free survival (EFS) was 57% [95% CI; 40%,71%] and overall survival was 59% [41%, 73%]. Multi-variable regression models noted that age > 10 years ($p<0.008$), stage ≥3 ($p<0.03$), group ≥3 ($p<0.005$) and T2 ($p<0.02$) invasive tumors were poor prognostic factors affecting 5 year EFS. Histology of tumor did not have an affect on prognosis. A Cox proportional hazards model identified that age > 10 years at presentation was the most important prognostic factor.

Conclusion:
Patients with perineal rhabdomyosarcoma have a poor overall prognosis, probably related to age, large invasive tumors that are difficult to resect, and metastatic disease. Patients with smaller tumors that can be completely resected have significantly better outcomes.

NOTES:

P30

FIBROUS SOFT TISSUE TUMORS: INCREASED RECURRENCE RISK FOR DIGITAL AND EXTREMITY LESIONS

Jennifer H. Aldrink, MD, Kathleen Nicol, MD, Steven Teich, MD.

Nationwide Children's Hospital, Columbus, OH, USA.

Purpose:
Fibrous soft tissue tumors (FSTT) of infancy and childhood present a challenge to surgeons due to ambiguous histological interpretation, propensity for local recurrence, and uncertainty regarding appropriate therapy. The purpose of this study was to identify factors contributing to an increase risk of recurrence.

Methods:
Records were reviewed for patients diagnosed and treated for FSTT at our institution from 1992-2010. Data included demographics, tumor location, operative treatment, histology, and recurrence. Statistical analysis was performed using the student t-test.

Results:
86 patients with a mean age of 5.6 years (range 3 days to 20 years) underwent treatment of FSTT. Location of tumors included 26 trunk, 16 head and neck, 16 lower extremities, 15 digits, 12 upper extremities, and one unknown location. Histopathology included fibromatosis (45), infantile digital fibromatosis (14), plantar fibromatosis (8), fibromatosis colli (6), aggressive fibromatosis (5), fibrous hamartoma of infancy (3), and other (5). Mean follow up was 64 months +/-55 months (range 1-201 months). There were 11 recurrences (13%), with a mean time to recurrence of 23 months (range 1 to 44 months). Extremity (5) and digital lesions (4) were most likely to recur; 21% of such lesions recurred (mean 25 months) compared to 7% of lesions located elsewhere (mean 17 months). Histopathology and age in those that recurred was not significantly different compared to those that did not recur. Positive or unknown margins were present in 9/11 recurrences. Therapy consisted of wide local excision in all cases. One patient was treated with amputation, and two patients received postoperative chemotherapy.

Conclusions:
Digital and extremity fibrous soft tissue tumors have an increased risk for local recurrence, and tend to recur later compared to other locations. The surgical goal is to obtain complete local resection with clear margins. This may be challenging in these particular locations, leading to increased recurrence rates.

NOTES:

P31
SURGICAL TREATMENT OF PULMONARY METASTASES OF OSTEOSARCOMA: LONG-TERM RESULTS

Pablo A. Lobos, MD, Julia E. Udaquiola, MD, Germán L. Farfalli, MD, Patricia D. Streitenberger, MD, Gastón R. Elmo, MD, Mauricio M. Urquizo Lino, MD, Luzía Toselli, MD, David Smith, MD.

Hospital Italiano de Buenos Aires, Buenos Aires, Argentina.

Purpose:
Almost 20% of patients with osteosarcoma present with detectable metastases, being most of them (85%) localized at the lung parenchyma. Five-year survival in non-metastatic osteosarcoma is 60-70%, and 10-30% in patients with metastatic disease. The purpose of this study is to determine the survival and prognostic factors in a group of patients surgically treated for osteosarcoma lung metastases with more than 5 yearfollow up.

Methods:
Retrospective analysis of a series of 249 patients treated for osteosarcoma between 1992 and 2006. Only patients who presented with osteosarcoma and pulmonary metastases and treated with surgical excision of the primary tumor and all palpable lung nodules were included (n=38). All cases received the same chemotherapy regimen (methotrexate, ifosfamide, adryamicine). Kaplan-Meier curves were used for survival analysis. Various prognostic factors (number of nodules, timing of appearance, etc.) were analysed using the Log-Rank test.

Results:
Overall 5 and 10 yr survival in our series was 31% (CI95%:46%-16%) and 26% (CI95%:40%-12%). respectively. A significantly better survival ($p=0.001$) was observed for the group of patients who had good response to chemotherapy (53% 5 yr. OS). No statistical significance was observed for the other proposed prognostic factors.

Conclusions:
Long term survival for patients with metastatic osteosarcoma treated with conventional chemotherapy and surgery remains continues to be poor. Patients who respond well to neoadjuvant chemotherapy have better prognosis. Perhaps, this group of patients should be stratified in a different prognosis group for adequate selection of treatment.

NOTES:

P32
LUNG TO HEAD RATIO IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIAS DOES NOT PREDICT LONG-TERM PULMONARY HYPERTENSION

Alejandro Garcia, MD, Abbey Fingeret, MD, Eunice Hahn, MD, Matthew Leskowitz, Gudrun Aspelund, MD, MS, Usha Krishnan, MD, Charles JH Stolar, MD.

Columbia University, New York, NY, USA.

Purpose:
Prenatal lung to head ratio (LHR) has been used for antenatal evaluation of infants with congenital diaphragmatic hernias (CDH). We report a single institution experience with CDH and LHR, and we asked if LHR is associated with acute and chronic pulmonary hypertension.

Methods:
Echocardiograms on all inborn infants with CDH (December 2001-March 2011) were reviewed. LHR values were stratified (Table). Echocardiograms at one and three months post-repair and most recent were reviewed to assess for presence of pulmonary hypertension (tricuspid regurgitation, septal bowing, ductal shunting, right ventricular dysfunction). Estimated gestational age, birth weight, hernia side, extracorporeal membrane oxygenation (ECMO) need, age at surgery, and death rate were obtained. Bivariate and multivariate analyses were performed with ANOVA, chi-square, and logistic regression. IRB approval was obtained.

Results:
106/140 evaluable infants with CDH had LHR obtained at median 28 weeks gestation (median LHR=1.25 (range 0.4-5.3)). Median follow-up was 26.6 months (range 4.6-97.5). LHR was significantly associated with pulmonary hypertension at one month but not at 3 months or long term. LHR was also predictive of death ($p<0.03$).

Conclusions:
Prenatal lung to head ratio predicts pulmonary hypertension at one month but not long term in infants with congenital diaphragmatic hernias. The use of lung to head ratio can be a useful adjunct for prenatal counseling; however, its use at predicting long term cardiopulmonary status is limited. That lung to head ratio predicts pulmonary hypertension at one month but not long term suggests significant remodeling of the pulmonary vasculature can occur over time.

Bivariate analysis of clinical characteristics of infants with CDH stratified by LHR						
	All (n=106)	LHR< 0.85 (n=19)	LHR 0.85-1.0 (n=8)	LHR 1.01-1.4 (n=37)	LHR> 1.4 (n=42)	p-value
Pulmonary hypertension one month, %	38.2	80.0	80.0	39.2	14.8	0.0001
Pulmonary hypertension three months, %	25.6	33.3	66.7	23.8	15.4	0.22
Pulmonary hypertension long term, %	16.0	0.0	50.0	20.8	5.9	0.54
LHR week, mean	28.2	26.4	26.5	27.4	29.9	0.01
Death, %	24.5	57.9	37.5	18.9	11.9	0.001
Estimated gestational age (weeks), mean	37.8	37.1	37.8	38.1	38	0.07
Weight (grams), mean	2946	2638	2983	3033	3002	0.21
Left sided hernia, %	89.6	78.9	87.5	89.1	95.2	0.22
ECMO, %	25.5	36.8	37.5	35.1	9.5	0.01

NOTES:

P33
ONE HORSEPOWER VERSUS MULTIPLE HORSEPOWER IN PEDIATRIC TRAUMA

David J. Hobbs, Diana Ropele, RN, James M. DeCou, MD, Helen DeVos.
Children's Hospital, Grand Rapids, MI, USA.

Purpose:
Horse and Off-Road Vehicle (ORV) riding are popular recreational activities among children, but both can result in serious injuries and even death. This study compares injury patterns among children from equestrian activities (1 horsepower (hp)) and from riding ORV's, including all-terrain vehicles, dune buggies, golf carts, and snowmobiles (some > 100 hp).

Methods:
From the trauma registry of a level I Pediatric Trauma Center, patients 0-17 years old with horse-related and ORV injuries over a 10-year period (2001-2010) were identified. Demographic and clinical data were obtained. The Mann-Whitney test was utilized for statistical comparisons between Horse and ORV groups.

Results:
Of 6125 patients entered into the registry over the study period, there were 94 (1.5%) with Horse-related injuries and 231 (3.8%) with ORV injuries. The Horse group was younger (9.5±0.5 vs. 11.4±0.3 years, $p=0.001$) and included more females (78% vs. 29%, $p<0.0001$). Helmet usage was reported at 18% and 39% for Horse and ORV groups, respectively. Injury severity scores were slightly higher for the ORV group (11.8±0.6 vs. 10.0±0.8, $p=0.044$), but there were no statistical differences in ICU admission rates, ICU lengths of stay, or numbers requiring immediate surgical intervention. There was 1 mortality (1.1%) in the horse group and 3 (1.3%) in the ORV group ($p=0.86$).

Conclusions:
Despite the significantly higher horsepower of ORV's, injuries and outcomes are quite similar for horse and ORV pediatric trauma patients. Those with horse-related injuries tend to be younger and mostly female. Increased helmet usage, appropriate training, and supervision are needed in both groups to minimize serious injuries.

NOTES:

Scientific Session II
Clinical Trials and Quality Improvement

Monday, May 21, 7:30 – 9:00 a.m.

12
AMERICAN COLLEGE OF SURGEONS NATIONAL QUALITY IMPROVEMENT PROGRAM PEDIATRIC: A BETA PHASE REPORT

Jennifer L. Bruny, MD[1], Bruce L. Hall, MD, PhD, MBA, FACS[2], Douglas C. Barnhart, MD, MSPH[3], Deborah F. Billmire, MD[4], Mark S. Dias, MD[5], Peter W. Dillon, MD, FACS[6], Charles Fisher, BS[7], Kurt F. Heiss, MD, FAAP, FACS[8], William L. Hennrikus, MD[9], Clifford Y. Ko, MD, MS, MSHS, FACS[10], R Lawrence Moss, MD, FACS[11], Keith T. Oldham, MD, FACS[12], Karen E. Richards, BS[7], Rahul K. Shah, MD[13], Charles D. Vinocur, MD, FACS[14], Moritz M. Ziegler, MD, FACS[1].

[1]Division of Pediatric Surgery, Department of Surgery, University of Colorado, Children's Hospital Colorado, Aurora, CO, USA, [2]Division of Research and Optimal Patient Care, ACS, Chicago, IL. Department of Surgery, Washington Univ in St Louis, Barnes Jewish Hospital, Center for Health Policy, The Olin Business School, Washington Univ in St Louis, St Louis Veterans Affairs Med Cen, Saint Louis, IL, USA, [3]Division of Pediatric Surgery, University of Utah, Salt Lake City, UT, USA, [4]Division of Pediatric Surgery, Riley Hospital for Children, Indianapolis, IN, USA, [5]Departments of Neurosurgery and Pediatrics, Pennsylvanian State University School of Medicine, Hershey, PA, USA, [6]Division of Pediatric Surgery, Department of Surgery, Pennsylvania State University School of Medicine, Hershey, PA, USA, [7]Division of Research and Optimal Patient Care, American College of Surgeons, Chicago, IL, USA, [8]Division of Pediatric Surgery, Department of Surgery, Emory University, Atlanta, GA, USA, [9]Department of Orthopedic Surgery, Pennsylvania State University School of Medicine, Hershey, PA, USA, [10]Division of Research and Optimal Patient Care, ACS, Chicago, IL. Department of Surgery, University of California Los Angeles David Geffen School of Medicine and the VA Greater Los Angeles Healthcare System, Los Angeles, CA, USA, [11]Nationwide Children's Hospital The Ohio State University, College of Medicine, Columbus, OH, USA, [12]Department of Surgery, Medical College of Wisconsin, Milwaukee, WI, USA, [13]Division of Otolaryngology, Children's National Medical Center, Washington, DC, USA, [14]Division of Pediatric Surgery, Thomas Jefferson University, A.I. DuPont Hospital for Children, Wilmington, DE, USA.

Purpose:
The American College of Surgeons (ACS) National Surgical Quality Improvement Program Pediatric (NSQIP-P) expanded to beta phase testing with the enrollment of 30 institutions for the calendar year 2010 . Data collection and analysis were aimed at program refinement and development of risk-adjusted models for inter-institutional comparisons.

Methods:
Data from the first full year of beta-phase NSQIP-P was analyzed. Patient accrual used ACS-NSQIP methodology tailored to pediatric specialties. Preliminary risk adjusted modeling for *all pediatric and neonatal operations* and *pediatric (excluding neonatal) abdominal operations* was performed for *all cause morbidity* and *surgical site infections* (SSI), using hierarchical logistic regression methodology and eight predictor variables. Results were expressed as odds ratios with 95% confidence intervals.

Results:
During calendar year 2010, 30 institutions enrolled 37,141 patients. 1644 total CPT codes were entered of which 456 accounted for 90% of the cases. 450 codes were entered less than twice (1.2% of cases). For all cases, overall mortality was 0.26%, overall morbidity 7.90%, and the SSI rate 1.84%. For neonatal cases, mortality was 2.06%, morbidity 21.72%, and the SSI rate 3.61%. For the all operations model: risk-adjusted morbidity institutional odds ratios ranged 0.48-2.63, with 9/30 hospitals categorized as low outliers and 9/30 high outliers; while risk-adjusted SSI institutional odds ratios ranged 0.36-2.04, with 2/30 hospitals low outliers and 7/30 high outliers.

Conclusion:
This report represents the first risk-adjusted hospital-level comparison of surgical outcomes in infants and children using NSQIP-P data. Programmatic and analytic modifications will improve the impact of this program as it moves into full implementation. These results indicate that NSQIP-P has the potential to serve as a model for determining risk-adjusted outcomes in the neonatal and pediatric population with the goal of developing quality improvement initiatives for the surgical care of children.

NOTES:

13

HOSPITAL QUALITY REPORTING MAY NOT ACCURATELY MEASURE HOSPITAL QUALITY

Shauna M. Levy, MD[1], Galit Holzmann-Pazgal, MD[1], Kevin P. Lally, MD[1], Koya Davis, MPH[2], Lillian S. Kao, MD[1], KuoJen Tsao, MD[1].

[1]Children's Memorial Hermann Hospital & University of Texas Medical School at Houston, Houston, TX, USA, [2]Children's Memorial Hermann Hospital, Houston, TX, USA.

Purpose:
Surgical site infections (SSI) are utilized as a measure of hospital quality. Typically, hospital infection control programs assign surgical wound classification (SWC) based on data from the medical record. This SWC is frequently recorded by the operating room support staff and may not be confirmed by the operative surgeon. The hospital assigned SWC is used to risk-stratify operations for the likelihood of SSI development. The purpose of this report is to assess the accuracy of hospital-documented compared to surgeon-based SWC in pediatric appendectomies.

Methods:
After IRB exemption, we performed an analysis of pediatric patients (n=312) undergoing appendectomy for acute appendicitis a large bed children's hospital (October 1, 2010 - August 31, 2011). The surgeon and hospital assigned SWC were collected. SWCs based on surgeon diagnosis included clean contaminated (acute non-perforated, non-gangrenous), contaminated (gangrenous), and dirty (perforated with and without an abscess). SSI data were obtained from a 30 post-operative day prospective continuous surveillance program, including inpatient and outpatient data. χ^2 analysis was utilized to evaluate outcomes.

Results:
The surgeon based and hospital assigned SWCs differed in 71% of cases. The classifications were discordant by more than 1 class in 45% of those cases. Of the evaluated patients, 12.4% developed an SSI (Table). SSI rates stratified by surgeon diagnosis were 3.4% in uncomplicated and 21.1% in complicated cases. Based on hospital SWC, SSI rates were 11.1% in uncomplicated and 10.2% in complicated cases.

Conclusions:
We conclude that hospital reported SSI rates may be an unreliable measure of quality and require validation. Processes of reporting quality data should be carefully evaluated.

Table					
Surgical Wound Classification (n=312) Case breakdown	Surgeon Based		Hospital Documented		χ^2
	SSI rate based on SWC	Case breakdown	SSI rate based on SWC		
Uncomplicated	57.4%	3.4%	83.7%	11.1%	$p<0.05$
Clean	0.0%	0.0%	42.9%	9.7%	
Clean-Contaminated	57.4%	3.4%	40.7%	12.6%	
Complicated	42.6%	21.1%	15.7%	10.2%	$p=0.09$
Contaminated	6.1%	10.5%	11.9%	10.8%	
Dirty	36.5%	22.8%	4.5%	7.1%	

NOTES:

14

DISCREPANCIES BETWEEN A PEDIATRIC HEALTH INFORMATION SYSTEM COHORT AND AN INSTITUTIONAL COHORT FOR ESOPHAGEAL ATRESIA

Jason P. Sulkowski, MD[2], Peter C. Minneci, MD, MHSc[1], Lindsey Asti[1], Katherine J. Deans, MD, MHSc[1].

[1]Nationwide Children's Hospital, Columbus, OH, USA, [2]Children's Hospital of Philadelphia, Philadelphia, PA, USA.

Purpose:
Definitive clinical studies for congenital pediatric surgical diseases are difficult to perform. Multi-institutional administrative databases may provide large enough datasets to perform clinical studies in these populations. The purpose of this study was determine the feasibility of studying esophageal atresia (EA) using the Pediatric Health Information System (PHIS) database.

Methods:
We performed a PHIS data abstraction to identify neonates diagnosed with EA with or without tracheoesophageal fistula (TEF) who underwent initial repair between January 1, 2000 and December 31, 2010. The PHIS cohort was developed using the following criteria: ICD-9 750.3 (EA with or without TEF), first admission, and one or more EA/TEF related ICD-9 procedure codes. Validation of this cohort was performed by reviewing the experience at a single participating PHIS institution and comparing it to its PHIS generated dataset.

Results:
There were 1711 neonates in the overall PHIS cohort, 72 in the institutional PHIS cohort and 140 in the institutional dataset. There was substantial variability between the institutional data and its PHIS cohort especially with regard to the clinical diagnoses and length of stay (Table).

Conclusions:
This analysis identifies substantial discrepancies between institutional clinical data and clinical data extracted from the PHIS database. Possible explanations for these discrepancies include a PHIS search strategy that was too simplistic or inappropriate to identify the full cohort of patients, inaccurate institutional coding of clinical diagnoses or inaccurate data extraction during institutional chart review. Administrative database analysis has the potential to be a powerful tool for studying clinical outcomes; however caution and rigor will be necessary to validate the data to ensure the legitimacy of the findings.

Data comparison of PHIS to Institutional Data			
Variable	Total PHIS Cohort (n=1711)	Institutional PHIS cohort (n=72)	Institutional data (n=140)
Female (%)	746 (43.6%)	30 (41.7%)	61 (43.6%)
Hispanic (%)	235 (13.7%)	13 (18.1%)	17 (12.1%)
Prematurity (%)	525 (30.7%)	14 (19.4%)	50 (36.2%)
PDA (%)	696 (40.7%)	21 (29.2%)	22 (15.7%)
ASD (%)	639 (37.4%)	19 (26.4%)	12 (8.6%)
VSD (%)	338 (19.8%)	18 (25%)	21 (15%)
TOF (%)	60 (3.5%)	2 (2.8%)	3 (2.1%)
LOS (Median, IQR)	29 days (16-71 days)	36 days (15-76 days)	26 days (13-57)
Age at Repair (Median, IQR)	2 days (1-5 days)	1 day (1-3 days)	1 day (1-4 days)

NOTES:

15

PRIMARY PAYER STATUS IS SIGNIFICANTLY ASSOCIATED WITH POSTOPERATIVE MORTALITY, MORBIDITY, AND HOSPITAL RESOURCE UTILIZATION IN PEDIATRIC SURGICAL PATIENTS WITHIN THE UNITED STATES

Matthew L. Stone, MD, Damien J. LaPar, MD, MSc, Daniel P. Mulloy, MD, Sara K. Rasmussen, MD, PhD, Bartholomew J. Kane, MD, PhD, Eugene D. McGahren, III, MD, Bradley M. Rodgers, MD.

The University of Virginia, Charlottesville, VA, USA.

Purpose:
Current healthcare reform efforts have highlighted the potential impact of insurance status on patient outcomes. The influence of primary payer status (PPS) within the pediatric surgical population remains unknown. The purpose of this study was to examine risk-adjusted associations between PPS and postoperative morbidity, mortality, and resource utilization in pediatric surgical patients within the United States.

Methods:
A weighted total of 153,333 pediatric surgical patients were evaluated using the national Kid's Inpatient Database (KID 2003 and 2006): appendectomy, intussusception, decortication, pyloroplasty, congenital diaphragmatic hernia repair, and colonic resection for Hirschsprung's disease. Patients were stratified according to PPS: Medicare (n=180), Medicaid (n=51,862), uninsured (n=12,539), and private insurance (n=88,753). Multivariable hierarchical regression modeling was utilized to evaluate risk-adjusted associations between PPS and outcomes.

Results:
Overall median patient age was 12 years, operations were primarily non-elective (92.4%), and appendectomies accounted for the highest proportion of cases (81.3%). After adjustment for patient, hospital, and operation-related factors, PPS was independently associated with in-hospital death ($P<0.0001$) and postoperative complications ($P<0.02$), with increased risk for Medicaid and uninsured populations (**Table**). Moreover, Medicaid PPS was also associated with greater adjusted lengths of stay and total hospital charges ($P<0.001$). Importantly, these results were dependent on operation type.

Conclusions:
Primary payer status is associated with risk-adjusted postoperative mortality, morbidity, and resource utilization among pediatric surgical patients. Uninsured patients are at increased risk for postoperative mortality while Medicaid patients accrue greater morbidity, hospital lengths of stay, and total charges. These results highlight a complex interaction between socioeconomic and patient-related factors, and primary payer status should be considered in the preoperative risk stratification of pediatric patients.

Primary payer status effect on postoperative morbidity and mortality in pediatric surgical patients				
Primary Payer Type	Mortality Model: Odds Ratio (95% C.I.)	Mortality Model: P-value	Morbidity Model: Odds Ratio (95% C.I.)	Morbidity Model: P-value
Medicare	4.68 (0.74-29.4)	<0.0001	1.08 (0.40-2.91)	0.02
Medicaid	1.24 (0.93-1.67)	<0.0001	1.14 (1.05-1.24)	0.02
Uninsured	3.72 (2.21-6.27)	<0.0001	1.04 (0.91-1.18)	0.02
Private Insurance	1.00	<0.0001	1.00	0.02

NOTES:

16

TRENDS IN OPERATIVE EXPERIENCE OF NORTH AMERICAN PEDIATRIC SURGICAL RESIDENTS

Abbey L. Fingeret, MD, Charles J.H. Stolar, MD, Robert A. Cowles, MD.

Division of Pediatric Surgery, Morgan Stanley Children's Hospital of New York-Presbyterian Hospital, Columbia University Medical Center, New York, NY, USA.

Purpose:
Expansion of the number of training programs in pediatric surgery occurred from 2003 through 2010. We sought to determine the effect of program expansion on case volume and distribution of operative experience.

Methods:
Public domain data on pediatric surgery resident summary statistics available from the Accreditation Council for Graduate Medical Education (ACGME) from July 2003 through July 2010 were analyzed. Total case volume as primary surgeon or teaching assistant, average case volume per resident, standard deviation, mode, minimum and maximum number of cases per resident was evaluated. Average total cases per resident, minimally invasive laparoscopic and thoracoscopic cases, and requisite cases as defined by the ACGME categories of: tumor, important pediatric surgical, and neonatal cases were analyzed by a Cuzick Wilcoxon-type nonparametric trend statistic using a significance level of 0.05. Skewness was assessed by Pearson coefficient with levels of -0.5 to 0.5 defining a parametric distribution.

Results:
The number of pediatric surgical training residents increased by 42% during the years reported, from 24 to 34. No statistically significant difference was found in the average number of total cases or requisite cases per resident. The average volume of minimally invasive procedures increased significantly (Figure 1). Case volume per resident was non-parametrically distributed with increasing positive skewness over time (Table 1).

Conclusion:
The increase in number of pediatric surgical resident training positions has not adversely affected overall operative experience or exposure to highly specialized requisite cases. The increasing skewedness of total and index cases suggests shifting variability in case distribution over time.

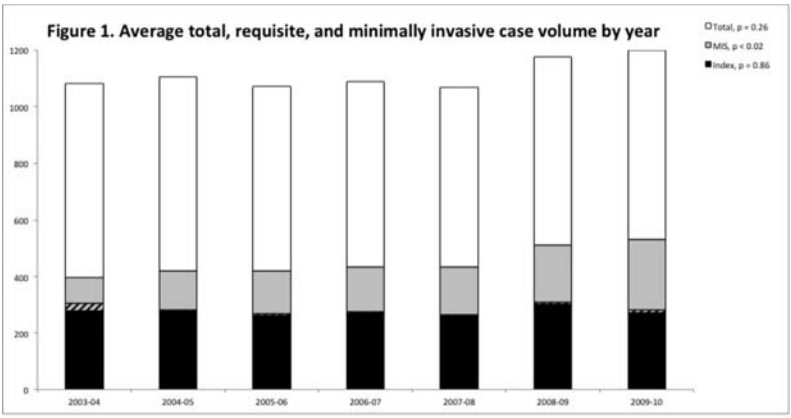


Figure 1. Average total, requisite, and minimally invasive case volume by year
Total, p = 0.26
MIS, p < 0.02
Index, p = 0.86
1200
1000
800
600
400
200
0
2003-04
2004-05
2005-06
2006-07
2007-08
2008-09
2009-10

NOTES:

17

FIRST EMPLOYMENT CHARACTERISTICS FOR THE 2011 PEDIATRIC SURGERY FELLOWSHIP GRADUATES

Charles J.H. Stolar, MD, Gudrun Aspelund, MD, MS.

Morgan Stanley Children's Hospital/Columbia University Medical Center, New York, NY, USA.

Purpose:
Information regarding initial employment of graduating pediatric surgery fellows is limited and unreliable. A more complete observational data set could yield benchmarks of initial career environment.

Methods:
An anonymous survey was distributed in July 2011 to 41 graduating pediatric surgery fellows from all ACGME training programs. We interrogated details of initial positions including financial/research support, fringe benefits, debt load, and demographics. Analysis used SPSS; $p<0.05$ was considered significant.

Results:
35/41 (85%) responded. Male: female ratio - 17:17 (one unknown). Ethnicity - Caucasian 63%, Asian 23%, Hispanic 6%, African-American 0%, other/unknown 9%. Graduates carried median debt of $200,000 (range: 0-$850,000); 71% were married; 60% had median 2 children. 71% were university or hospital employees. 74% were unaware of a business plan. Median starting compensation was $354,500 (range: $140,000-$506,000). Awareness of resources committed was very limited, including malpractice type and premium, and investigational support. Starting salary was greatest for >90% clinical obligation appointments (median $427,500 vs. $310,000; $p=0.002$), *independent* of geographic location. Compensation had no relationship to private practice/HMO vs. hospital/university/military position, coastal vs. non-coastal location, and practice sites number. Median clinical time was 75%; research time 10%. Only 6 graduates were able to completely detail resource commitments. 49% identified a formal mentor. Graduates covered 1-3 different offices (median 1) and 1-5 surgery sites (median 2) *independent* of geographic location. 60% were satisfied with their compensation.

Conclusion:
Recently graduated pediatric surgery fellows are engaged mainly in clinical care. Investigational work is not incentivized. Compensation was driven by clinical obligations and not geography, employer, or offsite obligations. Graduates have limited understanding and are unschooled regarding how their compensation is generated, the supporting business plan, nature of malpractice coverage, and commitments to mentoring and research support. Graduates generally enter practice with important fiscal and parenting obligations. Training programs and employers inconsistently educate new graduates in these regards.

NOTES:

18

VARIATION IN PRACTICE AND RESOURCE UTILIZATION ASSOCIATED WITH THE MANAGEMENT OF INTUSSUSCEPTION AT FREESTANDING CHILDREN'S HOSPITALS

Samuel E. Rice-Townsend, MD[1], C. Jason Smithers, MD[1], Catherine Chen, MD, MPH[1], Jeff N. Barnes, BS[2], Shawn J. Rangel, MD, MSCE[1].

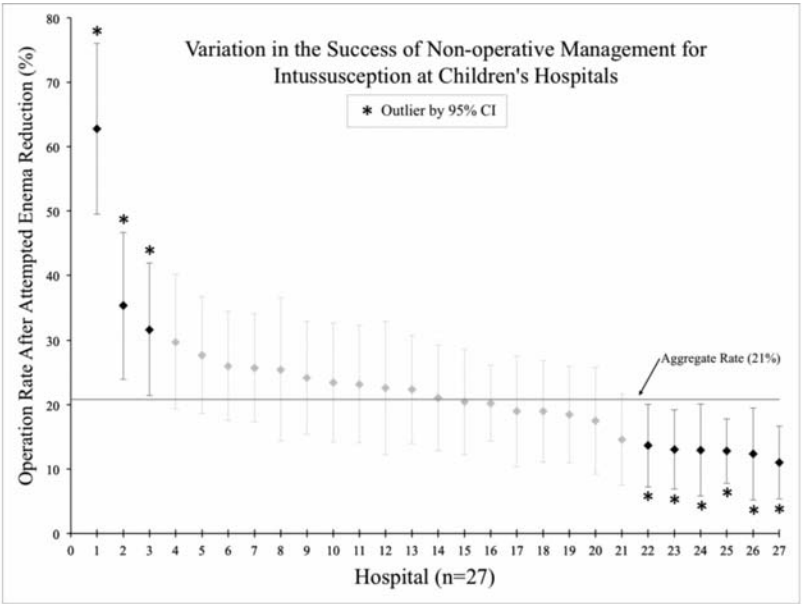
[1]Children's Hospital Boston, Boston, MA, USA, [2]Child Health Corporation of America, Shawnee Mission, KS, USA.

Purpose:
The purpose of this study was to characterize variation in care, resource utilization and outcomes associated with the management of intussusception at freestanding children's hospitals.

Methods:
We conducted a retrospective cohort study (1/1/09-6/30/11) of all patients admitted with intussusception at 27 freestanding children's hospitals participating in the Pediatric Health Information System database. Patients managed operatively without an initial attempt at enema reduction were excluded. Cases from each center were analyzed for the rate of successful non-operative management, and admission-related hospital cost and charges. Cases successfully managed non-operatively were further analyzed for prophylactic antibiotic utilization and rates of same-day discharge.

Results:
2,544 patients were identified (mean: 94 cases/center, range: 51-178). The median patient age was 17 months [IQR: 8-31] and 66% were male. The aggregate rate of operative management following attempted enema reduction was 21% and this was significantly different between centers (range by center: 11-62.8%; Chi^2 $p<0.0001$) (Figure). Among patients successfully managed non-operatively, significant differences were observed between centers with regard to antibiotic utilization (aggregate rate: 23.3%; range: 1.2-73.7%; $p<0.0001$) and same-day discharge (aggregate rate: 15.2%; range: 0-57%; $p<0.0001$). Significant differences were also observed for all cases of intussusception in case-related charges (aggregate median: $7,110; range $3,544-$22,097; Wilcoxon non-parametric ANOVA $p<0.0001$) and case-related costs (aggregate median: $2,865; range: $1,574-$6,763; $p<0.0001$), as well as for cost and charges associated with successful non-operative management (aggregate median charges: $6,350, range: $2,497-$10,306, $p<0.0001$; aggregate median cost: $2,490, range: $829-$5,905, $p<0.0001$).

Conclusion:
Significant variation in practice and resource utilization exists between children's hospitals in the management of intussusception. Prospective analysis of variation through a collaborative quality improvement platform could accelerate the dissemination of best-practice guidelines for optimizing cost-effective treatment and improving the success rate of non-operative management.


Variation in the Success of Non-operative Management for Intussusception at Children's Hospitals
* Outlier by 95% CI
Operation Rate After Attempted Enema Reduction (%)
Aggregate Rate (21%)
Hospital (n=27)
0
10
20
30
40
50
60
70
80
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27

NOTES:

19

A PROSPECTIVE STUDY TO DEFINE PEDIATRIC PRESSURE ULCERS AND TO ASSESS A QUALITY IMPROVEMENT BUNDLE IN REDUCING THEIR PREVALENCE

Alice Leung, MD, Marty O. Visscher, PhD, Ann Marie Nie, RN, WOCN, Sean J. Barnett, MD, Jason S. Frischer, MD, Timothy M. Crombleholme, MD, Sundeep G. Keswani, MD.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA.

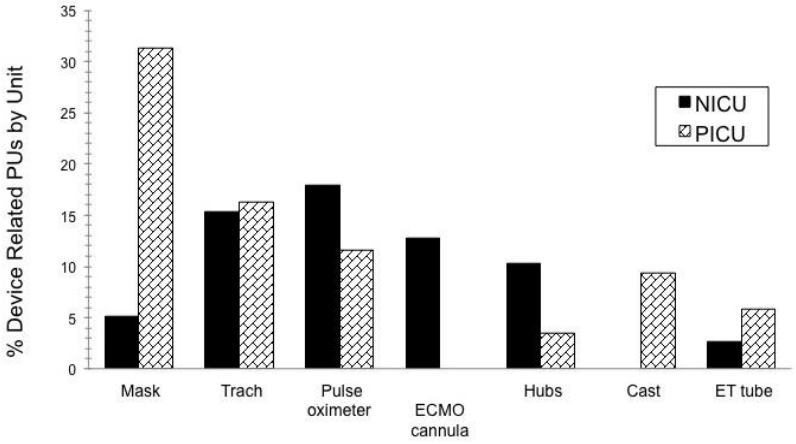
Purpose:
Pressure ulcers (PUs) accounts for over $11 billion of US health care costs with hospital acquired Stage III and IV PUs classified as 'never events' by CMS. The incidence and pathogenesis in the pediatric population is unknown. Therefore, we conducted a prospective study to evaluate the incidence and etiology of PUs in a pediatric population.

Methods:
With IRB approval, a prospective study of all patients in the neonatal and pediatric intensive care unit (NICU and PICU) between 2007 and 2009 was conducted. Skin assessments were conducted every two weeks by designated staff. Stage, location and suspected cause (traditional defined as pressure over bony prominences versus device related) were determined based on the National Pressure Ulcer Advisory Panel staging system. At 12 months, a quality improvement point of care bundle (QIPOC) was initiated in the PICU. Data was analyzed using Chi^2-analysis.

Results:
2898 patient evaluations were conducted (NICU 1719;PICU 1179) with a greater prevalence of PUs in the PICU (NICU 3.2% vs. PICU 12.6%,$p<0.001$). PUs were predominantly stage II in both ICUs. The majority of PUs in both groups were device related (Table 1,Figure 1). PICU patients were more susceptible to traditional PUs than NICU patients. After implementation of QIPOC in the PICU, we noted a significant decrease in overall PUs ($Chi^2=15.8$,$p<0.001$) and traditional PUs ($Chi^2 15.3$,$p\leq 0.001$) with a trend towards significance in device related PUs ($Chi^2 3.4$,$p=0.07$).

Conclusion:
We have identified an underappreciated prevalence of PUs in the pediatric population which, unlike in adults, are primarily caused by device related pressure. While our QIPOC intervention is efficacious, better surveillance, novel metrics and further interventions are needed to further decrease PU prevalence.

Incidence of pressure ulcers caused by traditional vs device related in NICU and PICU				
	NICU (n=1719)		PICU (n=1179)	
	Traditional	Device Related	Traditional	Device Related
Total	17	39	64	86
Stage I	1	5	20	34
Stage II	5	27	35	44
Stage III/IV	10	6	7	8



NOTES:

20

PROSPECTIVE COMPARISON OF NON-NARCOTIC VERSUS NARCOTIC OUT-PATIENT ORAL ANALGESIC USE AFTER LAPAROSCOPIC APPENDECTOMY AND EARLY DISCHARGE

Fuad Alkhoury, MD, Cathy Burnweit, MD, Colin Knight, MD, Steven Stylianos, MD, Jeannette Zerpa, Raquel Pasaron, JoAnne Mora, Alexandra Aserlind, Leopoldo Malvezzi, MD.

Miami Children's Hospital, Miami, FL, USA.

Purpose:
To compare narcotic versus non-narcotic outpatient oral pain management after pediatric laparoscopic appendectomy and early discharge.

Methods:
In a prospective study from 7/1/2010 to 3/30/2011, children undergoing laparoscopic appendectomy on our rapid discharge protocol were treated with either acetaminophen/ibuprofen (non-narcotic) or acetaminophen plus codeine or oxycodone (narcotic) post-operative oral analgesia. The groups were effectively divided based on clinical practice patterns, as two surgeons in a four-person faculty group employed the non-narcotic regimen, while the other two routinely used narcotics. Parents were asked to document days of medication use and time needed for return to normal activity. In addition, parents rated their satisfaction with the pain control method. Student *t*-test was used for statistical analysis.

Results:
During the study period, 207 consecutive children underwent appendectomy for acute, non-perforated appendicitis (n=186) or planned interval appendectomy (n=21). A single port, single instrument transumbilical approach was used in 198 patients (96%). There was no difference in age or mean time to discharge between the non-narcotic (n=104) and narcotic (n=103) groups. The cohorts had equivalent number of medication days and similar mean times to normal activity. Ninety-seven percent of the parents of children in the non-narcotic group stated that the pain was controlled by the prescribed medication, compared to 90 percent in the narcotic group.

	Non-narcotic (n=104)	Narcotic (n=103)	p value
Age	11.2 y	11.6 y	0.94
Time to discharge (hours)	6 h	7 h	0.16
Number of medication days	1.9 d	1.8 d	0.95
Time to normal activity (days)	4.5 d	5.0 d	0.92
Parental satisfaction	97%	90%	0.049

Conclusion:
This study indicates that after pediatric laparoscopic appendectomy and early discharge, non-narcotic medication is equivalent to narcotic-based therapy for outpatient oral analgesia, with higher parental satisfaction.

NOTES:

21

CLINICAL PRACTICE GUIDELINES (CPGS) REDUCE COSTS IN THE MANAGEMENT OF ISOLATED SPLENIC INJURIES AT PEDIATRIC TRAUMA CENTERS

Ivan M. Gutierrez, MD, David Zurakowski, PhD, Qiaoli Chen, MS, David P. Mooney, MD, MPH.

Children's Hospital Boston, Boston, MA, USA.

Purpose:
The APSA Trauma Committee proposed the non-operative management of splenic injuries using CPGs in 1998. An analysis was conducted to determine the financial impact of CPGs on the management of these injuries.

Methods:
The Pediatric Health Information System (PHIS) database was used to identify children who sustained an isolated splenic injury between June 2005 and June 2010. Demographics, length of stay (LOS), readmission rates, laboratory, imaging, procedural and total cost data were extracted for all hospitals verified as a pediatric trauma center by the American College of Surgeons and/or designated by their local authority. Comparisons were made between facilities self-identifying as having a splenic injury management CPG and those without. This study was exempted from IRB review.

Results:
1154 children with isolated splenic injuries were managed in 26 pediatric trauma centers: 20 with a CPG and 6 without (non-CPG). Children treated at centers with CPG compared to non-CPG were comparable in male gender (79% vs. 77%, $P = 0.50$), % high grade of 3 or 4 (55% vs. 47%, $P = 0.07$), although patients were somewhat older among centers with a CPG in place (median 12 vs. 9 years, $P < 0.01$). Median costs were significantly lower at CPG than non-CPG centers for imaging ($163 vs. $641, $P < 0.001$), laboratory ($629 vs. $1044, $P< 0.001$) and total hospital stay ($9,868 vs. $10,830, $P < 0.001$). Comparable median LOS for CPG and non-CPG (3 vs. 2 days, $P = 0.38$) and readmission rates <90 days (2.9% vs. 4.4%, $P = 0.15$) were observed. Multiple linear regression indicated that LOS ($P < 0.001$) and utilization of a CPG ($P = 0.007$) are significant independent predictors of total cost.

Conclusion:
CPG for management of isolated splenic injuries at a pediatric trauma center results in significantly reduced imaging, laboratory, and total hospital costs.

NOTES:

22

LAPAROSCOPIC COMMON BILE DUCT EXPLORATION IN CHILDREN IS ASSOCIATED WITH DECREASED COST, AND LENGTH OF STAY: RESULTS OF A TWO-CENTER ANALYSIS

Scott S. Short, MD[1], Philip K. Frykman, MD, PhD[1], Nam Nguyen, MD[2], Quin Liu, MD[2], Dror Berel, MS[1], Kasper Wang, MD[2].

[1]Cedars Sinai Medical Center, Los Angeles, CA, USA, [2]Children's Hospital Los Angeles, Los Angeles, CA, USA.

Purpose:
Our aim was to compare outcomes of children undergoing laparoscopic cholecystectomy with laparoscopic common bile duct exploration (LC+CBDE) to those undergoing laparoscopic cholecystectomy with adjunctive endoscopic retrograde cholangiopancreatography (LC+ERCP). Primary outcomes included rate of complications, total operative time, length of stay, and hospital cost.

Methods:
We performed a two-center retrospective review of all children (<18 years) undergoing LC+CBDE or LC+ERCP between January 2000 and July 2011. IRB approval was obtained at both institutions (# 11-0051 and # 25387). Patients were identified by CPT code 47564 or any combination of codes 47562-47564 and 43260-43273. Wilcoxon test was performed on continuous variables and logistic regression modeling on categorical data. A p-value < 0.05 was considered significant. Outcomes with a p-value < 0.2 were selected for multivariable analysis.

Results:
42 patients were identified. 24 (57%) underwent LC+ERCP and 18 (43%) underwent LC+CBDE. Demographic and clinical factors were well matched between groups. Total operative was similar between groups (157 min vs. 152 min, p=0.26). LC+CBDE patients had zero major complications and five minor complications (retained stone-3, pancreatitis-1, late recurrence-1). LC+ERCP patients experienced two major (duodenal perforation-1, bleeding requiring transfusion-1) and four minor complications (pancreatitis-2, retained stone-2), (p = 0.57). Length of stay was significantly longer (15.7 days vs. 6.6 days, p = 0.02) and average hospital cost was significantly higher ($38285 vs. $14266, p < 0.01) in the LC+ERCP group. Multivariable analysis revealed that cost was significantly lower in patients undergoing LC+CBDE (p=0.05, OR= 0.71; 95% CI: (0.51 - 0.97)).

Conclusion:
Laparoscopic common bile duct exploration at time of cholecystectomy is associated with decreased length of stay, decreased cost, and has similar or improved morbidity compared to endoscopic retrograde cholangiopancreatography.

NOTES:

Scientific Session III
Fetal/Neonatal and Trauma

Monday, May 21, 10:30 – 11:45 a.m.

23
RECURRENCE AFTER THORACOSCOPIC CONGENITAL DIAPHRAGMATIC REPAIR IN NEONATES AND INFANTS

Henry Chang, MD, Thomas T. Sato, MD, David M. Gourlay, MD, Casey M. Calkins, MD, Dave R. Lal, MD, John J. Aiken, MD, Keith T. Oldham, MD, Jessica Enters, BSN, Marjorie J. Arca, MD.

Children's Hospital of Wisconsin, Milwaukee, WI, USA.

Purpose:
Previous studies have demonstrated improved short term outcomes with thoracoscopic repair of congenital diaphragmatic hernia (CDH), including reduced time to full enteral nutrition, shorter duration of narcotic use, and shorter length of stay. Recent case series suggest that thoracoscopic repair of CDH may have a high recurrence rate. We examined our institutional experience with this technique to identify factors that may predispose to recurrence.

Methods:
Institutional Review Board approval (CHW 11/118 GC 1340) was obtained to analyze data from our surgical registry for neonates and infants under six months of age who underwent thoracoscopic CDH repair from January 2003 to July 2011. We compared the population of patients with and without recurrences with respect to estimated gestational age at birth, weight at surgery, age at surgery, need for high frequency oscillatory ventilation, highest postoperative peak inspiratory pressure on pressure control ventilation, postoperative days on the ventilator, steroid administration, peri-operative bacteremia, patch repair, FiO2 immediately postoperatively, and type of sutures utilized.

Results:
Thirty three patients underwent thoracoscopic CDH repair during the study period. Within an average follow-up of 2.5±2.1 years, there were 6 (18%) recurrences. Median time to recurrence was 177 days. While there were 7 patch repairs in the non-recurrence group and no patches used in the recurrence group, this difference did not reach statistical significance. We found no statistically significant difference in the parameters we analyzed between the two populations.

Conclusions:
Our thoracoscopic recurrence rate of 18% is consistent with recent published reports. Recurrences occur early, often within 6 months of initial repair. In comparing those who recurred and those who did not, we found no differences in post-operative ventilatory management, steroid administration, patch placement, or suture type These data emphasize the need for close follow-up with clinical examination and imaging in patients undergoing neonatal thoracoscopic CDH repair.

NOTES:

24

HEART RATE CHARACTERISTICS INDEX (HERO SCORE) IDENTIFIES NICU PATIENTS WITH SEVERE NECROTIZING ENTEROCOLITIS

Matthew L. Stone, MD, Bartholomew J. Kane, MD, PhD, Douglas E. Lake, PhD, Joseph R. Moorman, MD, Eugene D. McGahren, III, MD, Bradley M. Rodgers, MD, Karen D. Fairchild, MD.

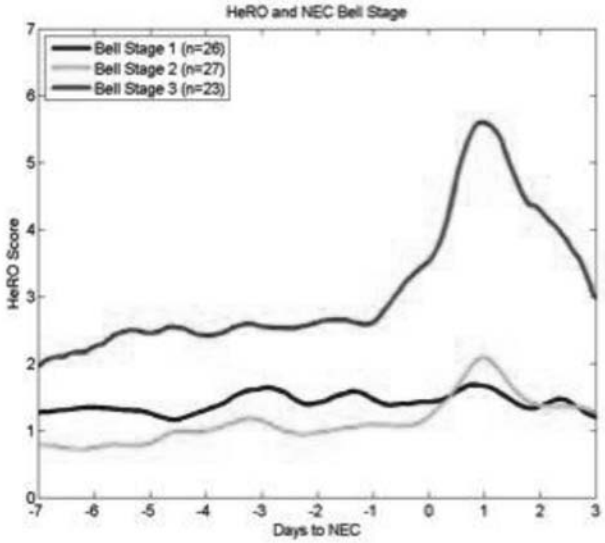
The University of Virginia, Charlottesville, VA, USA.

Purpose:
Abnormal heart rate characteristics of transient decelerations and reduced variability signal illness in Neonatal Intensive Care Unit (NICU) patients. The Heart Rate Observation (HeRO) monitor continuously displays a score representing the chance of sepsis developing in the next 24 hours. HeRO monitoring was recently shown to reduce mortality in NICU patients but its role in identifying patients with intestinal pathology has not been established. The purpose of this study was to evaluate the association between HeRO score and necrotizing enterocolitis (NEC).

Methods:
We retrospectively reviewed clinical factors and HeRO scores of all infants with suspected or proven NEC within a single NICU from 2007-2011. Infants were stratified as Bell's Stage 1, 2, or 3 based on clinical presentation, laboratory and radiographic findings, and need for surgical intervention. Cases of spontaneous intestinal perforation were excluded.

Results:
Of 89 cases of suspected or proven NEC, 76 cases in 68 patients had HeRO scores available at the time of diagnosis: Bell's Stage 1 (n=26), Stage 2 (n=27), Stage 3 (n=23). Median age at diagnosis was 26 days (25-75th%: 13-38 days). Surgical intervention within 1 week of NEC diagnosis occurred in 34 patients (median 21 hours from diagnosis): 29 laparotomies (7 following initial drain placement) and 4 peritoneal drains alone. HeRO score was significantly higher in patients with Stage 3 NEC compared to other stages in the week prior to diagnosis (Figure). For this group, HeRO score increased significantly 12-24 hours prior to diagnosis, continued to increase for 24 hours after diagnosis, and increased significantly immediately after surgical intervention (all $p<0.05$).

Conclusion:
Abnormal heart rate characteristics occur in NICU patients with Bell's Stage 3 NEC. The significant rise in HeRO score 12-24 hours before diagnosis of severe NEC demonstrates that continuous HeRO monitoring of NICU patients offers the possibility of earlier intervention and improved outcomes.


HeRO and NEC Bell Stage
Bell Stage 1 (n=26)
Bell Stage 2 (n=27)
Bell Stage 3 (n=23)
HeRO Score
Days to NEC
0
1
2
3
4
5
6
7
-7
-6
-5
-4
-3
-2
-1
0
1
2
3

NOTES:

25

EXIT-TO-RESECTION FOR FETUSES WITH LARGE LUNG MASSES AND PERSISTENT MEDIASTINAL COMPRESSION NEAR BIRTH

Darrell L. Cass, MD, Oluyinka O. Olutoye, MD, PhD, Christopher I. Cassady, MD, Nancy A. Ayres, MD, R. Todd Ivey, MD, Olutoyin A. Olutoye, MD, Timothy C. Lee, MD, Irving J. Zamora, MD.

Texas Children's Fetal Center and Baylor College of Medicine, Houston, TX, USA.

Purpose:
The purpose of this study was to identify prenatal diagnostic features that might be helpful in selecting fetuses with lung masses (LM) who may benefit from the ex-utero intrapartum treatment (EXIT) procedure as the optimal mode of delivery.

Methods:
The fetal imaging and CCAM-volume-ratio (CVR), prenatal course, fetal treatment, operative and pathologic findings and postnatal outcomes of all fetuses with lung masses evaluated at a comprehensive fetal treatment center between July 2001 and September 2011 were reviewed retrospectively. Fetuses with hydrops and/or CVR ≥ 2 were classified as high risk. Indications for early fetal intervention included hydrops and heart failure and indications for an EXIT-to-resection strategy were the finding of a LM with persistent mediastinal compression (PMC, defined by fetal MRI) near the time of delivery.

Results:
Of 103 fetuses evaluated for LM, 79 were classified as low-risk. No fetus in this group showed PMC near birth and none required perinatal treatment. Of the 24 fetuses classified as high-risk, 8 developed cardiac failure early in gestation (<26 weeks), of which 4 survived (3 following fetal surgery), and 4 died in-utero. Four high-risk fetuses with resolution of the mass-effect and lack of PMC near birth were asymptomatic postnatally and treated electively. Twelve high-risk fetuses (CVR 2.1-3.6 at > 32 weeks' gestation) had PMC near birth: 7 underwent EXIT-to-resection (37 to 38 4/7 weeks' gestation) and 5 were treated with standard perinatal care. All babies treated with EXIT-to-resection did well with discharge at 5, 7, 8, 9, 22, 25 and 71 days post-operatively. In contrast, all 5 fetuses with PMC at delivery who did not have an EXIT developed respiratory distress following birth requiring an urgent, difficult operation; 2 died.

Conclusion:
The EXIT-to-resection procedure is the optimal delivery approach for those fetuses with very large lung masses and persistent mediastinal compression near birth.

NOTES:

26

PULMONARY RADIAL ALVEOLAR COUNT AND VASCULAR MORPHOMETRY AFTER PRENATAL TEMPORARY GEL PLUG OCCLUSION OF THE FETAL TRACHEA IN A RABBIT MODEL OF CONGENITAL DIAPHRAGMATIC HERNIA

Ramy Elattal, BS, Barrie S. Rich, MD, Oliver J. Muensterer, MD, PhD.

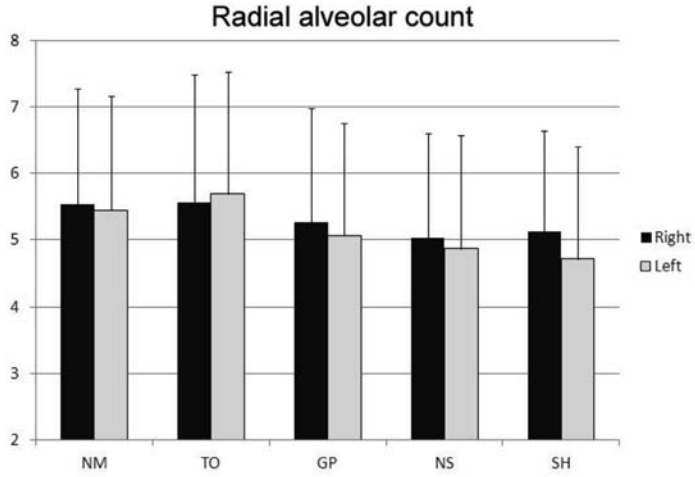
Weill Cornell Medical College, New York, NY, USA.

Purpose:
Temporary tracheal occlusion (TO) induces lung growth in congenital diaphragmatic hernia (CDH) but is also associated with significant drawbacks. We devised a temporary gel plug that induced lung growth when placed in the fetal trachea during the saccular phase of lung development in previous experiments. This study evaluates the effects of temporary versus permanent tracheal occlusion on histologic radial alveolar count and vascular morphometrics.

Methods:
After obtaining IACUC approval, experimental CDH was created in a total of 64 NZ white rabbit fetuses. These were randomized to intratracheal instillation of a fibrin gel plug (GP) , tracheal suture ligation (TO), intratracheal instillation of normal saline (NS), or sham amniotomy (SH). Non-manipulated fetuses of the litter without CDH served as controls (NM). Fetuses were harvested at gestational day 29. The lungs were inflation-fixed with 10% formalin, and hematoxylin/eosin-stained histologic sections of the lungs were assessed blindly for radial alveolar count (RAC) and arterial proportional adventitial thickness (%AT). Results were statistically compared.

Results:
RAC was significantly lower in the ipsilateral (left) lung of SH CDH-fetuses than in the contralateral (right) lung ($p=0.011$). Mean RAC was higher after TO ($p<0.001$) and GP ($p=0.03$) compared to SH fetuses (figure). Furthermore, relative adventitial thickness %AT was higher in GP (50+/-28, $p<0.001$) and TO (45+/-26, $p=0.003$) fetuses than in healthy, non-CDH NM controls (36+/-19).

Conclusions:
Tracheal occlusion by a temporary gel plug or a permanent suture led to increased RAC, although the effect was more pronounced with permanent TO. Interestingly, both interventions were associated with an increased relative adventitial thickness. These findings may help explain potential adverse effects of tracheal occlusion in the clinical setting, despite causing accelerated lung growth and an increased number of alveoli. Future fetal surgical approaches should include the pharmacologic modulation of vascular development in addition to mechanical tracheal occlusion.



NOTES:

27

EX UTERO INTRAPARTUM TREATMENT TO EXTRACORPOREAL MEMBRANE OXYGENATION (EXIT-TO-ECMO) STRATEGY FOR SEVERE CONGENITAL DIAPHRAGMATIC HERNIA (CDH)

Naira Baregamian, Foong-Yen Lim, Sundeep G. Keswani, Jason S. Frischer, Beth Haberman, Paul Kingma, Mounira Habli, Ronald Jaekle, James Van Hook, William J. Polzin, Timothy M. Crombleholme.

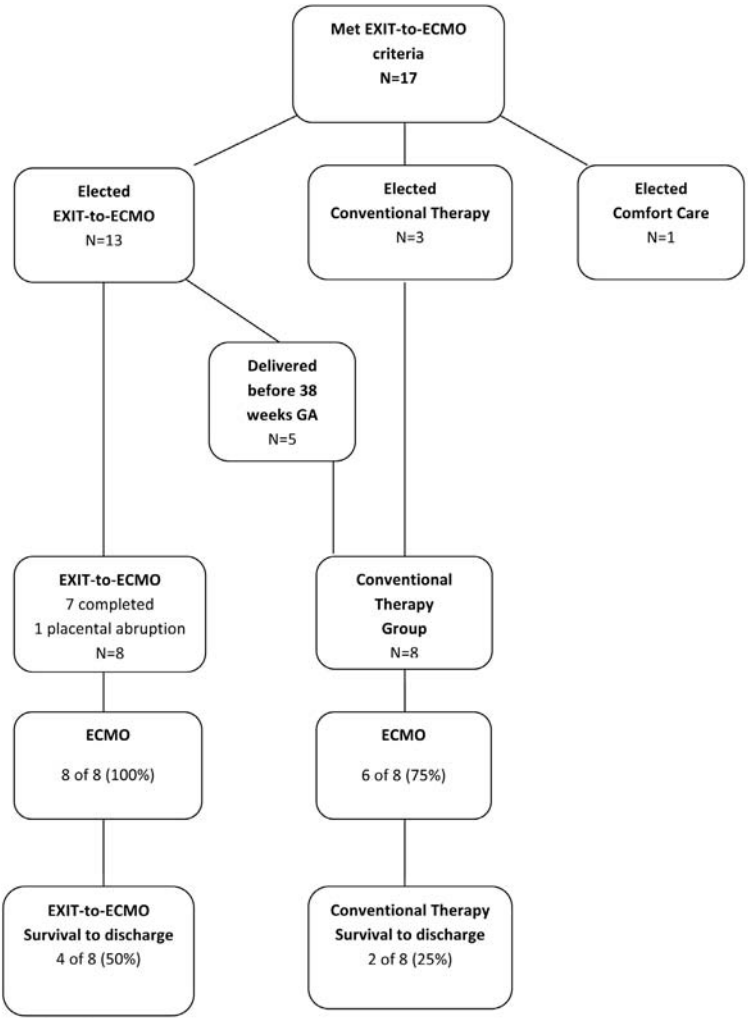
Cincinnati Childrens Hospital Medical Center, Cincinnati, OH, USA.

Purpose:
Dismal survival (~20%) with severe fetal congenital diaphragmatic hernia (observed/expected lung to head ratio (O/E LHR)<25% and percent predicted lung volumes (PPLV)<15) with conventional postnatal approaches prompted use of a therapeutic innovation, the EXIT-to-ECMO strategy. The purpose of this study is to determine outcomes of EXIT-to-ECMO in severe CDH compared to conventional postnatal management.

Methods:
This is a 6-year (2005-2010) prospective non-randomized IRB-approved study of fetuses with severe CDH defined by significant liver herniation, O/E LHR<25%, PPLV<15, with EXIT-to-ECMO at 38 weeks vs. conventional therapy. Nineteen patients were identified, 4 had right CDH with PPLV<15 and significant liver herniation, 15 had left CDH with O/E LHR<25%, PPLV<15, and significant liver. 17 patients met criteria for EXIT-to-ECMO, 2 were excluded due to chromosomal abnormality (n=1) and abnormal fetal posturing (n=1); 13 (76.5%) elected EXIT-to-ECMO, 2 (11.7%) elected Cesarean with ECMO standby, 1 (5.88%) opted for vaginal delivery, 1 (5.88%) chose comfort care and was excluded from analysis. Maternal-fetal outcomes were analyzed.

Results:
Of the 13 patients elected EXIT-to-ECMO, 5 (38.4%) delivered before 38 weeks and had conventional treatment, 8/13 (61.6%) underwent EXIT-to-ECMO with 1 patient abrupting and having ECMO cannulation completed after delivery. Survival-to-discharge for EXIT-to-ECMO procedure was 50% (4/8). In the conventional treatment group (n=8 total; n=3 by choice, n=5 delivered early), 6/8 required ECMO (75%) and survival was 2/8 (25%). The difference between survivals in two groups was not statistically significant, and was attributed partly due to small sample size. There was one maternal complication of placental abruption (12.5%) during EXIT-to-ECMO.

Conclusions:
EXIT-to-ECMO strategy in high-risk patients with severe CDH achieves better survival than conventional therapy, is comparable to reported **survival** with fetal tracheal occlusion, EXIT-to-ECMO is a therapeutic innovation deserving further study and evaluation, perhaps in a multicenter randomized clinical trial.


Met EXIT-to-ECMO criteria N=17
Elected EXIT-to-ECMO N=13
Elected Conventional Therapy N=3
Elected Comfort Care N=1
Delivered before 38 weeks GA N=5
EXIT-to-ECMO 7 completed 1 placental abruption N=8
Conventional Therapy Group N=8
ECMO 8 of 8 (100%)
ECMO 6 of 8 (75%)
EXIT-to-ECMO Survival to discharge 4 of 8 (50%)
Conventional Therapy Survival to discharge 2 of 8 (25%)

NOTES:

28

VENOUS THROMBOEMBOLISM AFTER TRAUMA: ARE ADOLESCENTS MORE LIKE CHILDREN OR ADULTS?

Kyle J. Van Arendonk, MD, Eric B. Schneider, PhD, F. Dylan Stewart, MD, Paul M. Colombani, MD, Elliott R. Haut, MD.

Johns Hopkins University School of Medicine, Baltimore, MD, USA.

Purpose:
The risk of venous thromboembolism (VTE) after trauma is lower for children than for adults, in whom increasing patient age is a known risk factor for VTE. The aim of this study was to identify a precise age at which VTE risk after trauma increases in order to guide the use of VTE prophylaxis, in particular within the adolescent population in whom prophylaxis practices vary considerably.

Methods:
After IRB approval, the records of all injured patients aged 0-21 years admitted in 2007 were analyzed using the National Trauma Data Bank (version 8.0), a registry of U.S. trauma data maintained by the American College of Surgeons. The risk of VTE was quantified across patient age, and multivariable logistic regression was used to estimate the impact of age on the odds of VTE while adjusting for other VTE risk factors, including Injury Severity Score, Glasgow Coma Scale, and mechanism of injury (blunt/penetrating).

Results:
Of the 131,326 patients studied, 428 (0.33%) were diagnosed with VTE: 316 patients (74%) with deep venous thrombosis, 90 (21%) with pulmonary embolism, and 22 (5%) with both. Patients with VTE were significantly older than those without (median age 17.6 years vs. 13.0 years; $p<0.001$). The risk of VTE increased dramatically at age 15 (Figure 1A), and 90% of the VTE events identified were found in patients aged ≥15. After adjusting for other risk factors, patients aged ≥15 had a 6-fold higher odds of VTE compared to younger patients (OR: 6.42, 95% CI: 4.35-9.45; $p<0.001$) (Figure 1B).

Conclusion:
The risk of VTE after trauma varies by age and increases dramatically at age 15. While VTE appears to be an uncommon complication in younger trauma patients, patients aged ≥15 may best be treated as adults with mechanical and pharmacologic VTE prophylaxis based on appropriate risk factors.

NOTES:

29

FATE OF THE COMBINED ADULT & PEDIATRIC TRAUMA CENTERS: IMPACT OF INCREASED PEDIATRIC TRAUMA REQUIREMENTS

Kevin N. Johnson, MD[1], Pamela Garcia-Filion, PhD, MPH[2], Melissa Twomey, MS, RN[2], David Notrica, MD[2].

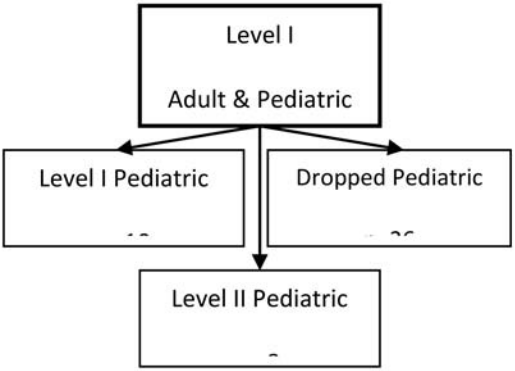
[1]Mayo Clinic, Phoenix, AZ, USA, [2]Phoenix Children's Hospital, Phoenix, AZ, USA.

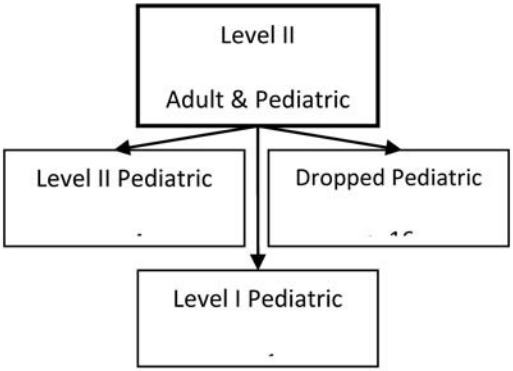
Purpose:
The American College of Surgeons (ACS) verifies trauma centers meeting the criteria for optimal management of the injured patient. ACS verification has been associated with better outcomes and lower statewide pediatric mortality. Prior to 2007, the verification requirements as a combined pediatric and adult trauma center were minimal. In 2007, the ACS raised the verification requirements. The new pediatric trauma centers also began receiving a verification level (Level I or II pediatric) independent of the adult center's verification level.

Methods:
Data on ACS verification level of all trauma centers from December 2006 was obtained and compared to September 2011. Data on re-verified and new pediatric trauma centers under the new requirements were collected.

Results:
Prior to 2007, the ACS listed 58 verified Level I and 24 Level II pediatric trauma centers (44 were Level I and 21 Level II combined adult and pediatric centers). Of the Level I combined centers, 26 (59%) dropped pediatric verification completely, 8 (18%) became verified Level II pediatric trauma centers, and 10 (23%) of the combined Level I centers went on to achieve Level I pediatric trauma center verification. Of the 21 combined Level II centers, 16 (76%) dropped their pediatric verification, 4 (19%) re-verified, and 1 (4.7%) became a Level I pediatric center. All 14 stand-alone pediatric Level I pediatric trauma centers and all 3 Level II centers achieved re-verification. Since 2007, 10 new pediatric Level I trauma centers were verified for a total of 35 centers.

Conclusion:
Higher verification standards for pediatric trauma centers resulted in 65% of the combined centers dropping pediatric verification status. During the same time period, the number of children's hospitals verifying as Level I pediatric trauma centers increased 79%. The total number of centers committed to pediatric trauma may have been over-represented during the early time period.


Level I
Adult & Pediatric
Level I Pediatric
Dropped Pediatric
Level II Pediatric


Level II
Adult & Pediatric
Level II Pediatric
Dropped Pediatric
Level I Pediatric

NOTES:

30

PROBIOTIC PROPHYLAXIS AFTER PULLTHROUGH FOR HIRSCHSPRUNG's DISEASE TO REDUCE INCIDENCE OF ENTEROCOLITIS: A PROSPECTIVE, RANDOMIZED, DOUBLE BLIND, PLACEBO CONTROLLED, MULTICENTER TRIAL

Sabina Siddiqui, MD[1], Mohamed El-Sawaf, MD[2], Moustafa Mahmoud, MD[2], Robert Drongowski, MS[1], Daniel H. Teitelbaum, MD[1].

[1]University of Michigan, Department of Pediatric Surgery, Ann Arbor, MI, USA, [2]Tanta University, Department of Pediatric Surgery, Tanta, Egypt.

Purpose:
Hirschsprung-associated enterocolitis (HAEC) is one of the most troublesome problems encountered after a pullthrough. Although the etiology is unknown, it has been suspected that altered microbiome or loss of intestinal epithelial barrier may play a role. As probiotics enhance intestinal barrier function and normalize the microbiome, we hypothesized that prophylactic administration of probiotics after a pullthrough procedure would decrease the incidence of HAEC.

Methods:
A prospective, multicenter, double-blind, placebo-controlled, randomized trial was conducted at 2 separate children's hospitals from 3/1/2006 to 12/31/2009. Infants undergoing pullthrough were randomized into a treatment arm receiving orally administered commercially available probiotic(VSL#3, VSL Pharmaceuticals) or placebo receiving maize starch for a period of 3 months post-operatively once tolerating an enteral diet. Primary outcome was the incidence of post-operative HAEC. Other outcomes included severity of HAEC by clinical grade and extent of aganglionosis. Pearson Chi Square analysis as well as logistic regression was used to determine clinical significance.

Results:
Sixty-two patients were recruited (Site: A=22; B=40). Two were lost to follow up and not included in final analysis. Probiotics were administered to 32 patients. The overall incidence of HAEC was 28.3%. Distribution of placebo/probiotics were equal between sites(P=0.858). Mean age at pullthrough was 6.5±8.1(±SD) months. Incidence of HAEC was not statistically different between study group(Table). Stratification based on study center, severity grade of HAEC and extent of aganglionosis also failed to show significant differences(Table). Other demographic, clinical and management characteristics were similar.

Conclusions:
Incidence and severity of HAEC was not reduced with prophylactic administration of probiotics. Future studies are needed to better determine the etiology and possible ways of preventing this complex condition.

Incidence of HAEC and Probiotic Administration				
		Placebo	Probiotic	p-value
HAEC	Yes	7	10	0.775
	No	21	22	
Severity of HAEC	Grade I	6	3	0.779
	Grade II	0	6	
	Grade III	7	10	
Length of Aganglionic Segment - Standard (RectoSigmoid)	HAEC Yes	4	6	0.144
	HAEC No	19	19	
Length of Aganglionic Segment - Long Segment	HAEC Yes	3	4	0.155
	HAEC No	5	4	

NOTES:

31

CONGENITAL HEART DISEASE AND HETEROTAXY: UPPER GASTROINTESTINAL FLUOROSCOPY IS MISLEADING AND SURGERY IN AN ASYMPTOMATIC PATIENT IS NOT BENEFICIAL

Stephanie C. Papillon, MD[1], Osnat Zmora, MD[1], Catherine J. Goodhue, MN[1], Shalini S. Sharma, MD, MPH[2], Winfield J. Wells, MD[1], Henri R. Ford, MD[1], Jeffrey S. Upperman, MD[1], Gerald A. Bushman, MD[1], Kasper Wang, MD[1], Richard Kim, MD[1], James R. Pierce, MD[1].

[1]Children's Hospital Los Angeles, Los Angeles, CA, USA, [2]USC Keck School Of Medicine, Los Angeles, CA, USA.

Purpose:
In the setting of congenital heart disease, visceral heterotaxy is associated with digestive tract abnormalities. We sought to define the gastrointestinal anatomy, to determine the risk of volvulus, benefit of screening UGI, and outcomes associated with Ladd's Procedure in these patients.

Methods:
After IRB approval, medical records at Children's Hospital Los Angeles between 2003 and 2010 were searched for heterotaxy, asplenia, polysplenia, and situs ambiguous. Variables included demographics, cardiovascular diagnosis, gastrointestinal symptoms, imaging and surgical arrangement of abdominal organs, operations, morbidities, and mortality.

Results:
107 patients were identified: 46 with polysplenia, 43 with asplenia, 5 with inversus, and 13 with uncharacterized splenic morphology. Upper GI was performed in 2 patients for suspected volvulus, in 31 for obstructive symptoms, and in 31 as "screening." No imaging was performed in the remaining 43 asymptomatic patients, none of whom developed volvulus. Of 35 patients with duodenojejunal malposition (DJM) on imaging, 31 received operation and none had malrotation or narrow mesentery. 8 developed postoperative complications (2 wound infections, 6 bowel obstructions). All obstructions required reoperation and bowel resection. Of 7 patients with radiographic malrotation, all received a Ladd's procedure. The single patient with volvulus (which was clinically suspected) had viable bowel. There was one bowel obstruction among this group which required reoperation and resection.

Conclusions:
Our analysis suggests that in patients with heterotaxy, while abnormalities of rotation are common, complete malrotation with risk of volvulus is not. Radiographic DJM is not associated with volvulus risk. Postoperative bowel obstruction and need for reoperation is higher. With poor tolerance of complication, we advocate avoiding operation in the asymptomatic patient and patients with DJM.

		Symptoms			
		None (Screening)	Obstructive (non-emergent)	Bilious emesis (emergent)	Total
Rotation	Normal	16	3	0	19
	Duojejunal malposition	9	25	1	35
	Malrotation	3	3	0	6
	Volvulus	0	0	1	1
	Inversus	3	0	0	3
	Total	31	31	2	64

NOTES:

Scientific Session IV
Basic Science

Tuesday, May 22, 9:00 – 10:15 a.m.

32
RELAXIN: A POTENTIAL VASODILATOR ROLE IN NECROTIZING ENTEROCOLITIS

Alexandra C. Maki, MD, Laura A. Galganski, BA, Jessica A. Shepherd, BA, Paul J. Matheson, PhD, Richard N. Garrison, MD, Cynthia D. Downard, MD, MMSc.

University of Louisville, Louisville, KY, USA.

Purpose:
Necrotizing enterocolitis (NEC) is a gastrointestinal emergency in premature infants with a complex, multifactorial etiology. A critical event in the pathophysiology of NEC appears to be microvascular vasoconstriction and hypoperfusion leading to tissue ischemia. Relaxin is a ubiquitous vasodilatory hormone that has reproductive (RLXN-1), cardiovascular (RLXN-2) and neural (RLXN-3) isoforms. We hypothesized that tissue and serum relaxin levels would be decreased in NEC and might partially account for the vasoconstriction of NEC.

Methods:
After IACUC approval, Sprague-Dawley rat pups were separated by litter. NEC (n=79) was induced by intermittent exposure to cold, hypoxia, lipopolysaccharide and formula feeding in premature rat pups (C-section). Control pups delivered vaginally and were dam fed (CONTROLS, n=42). At 12-, 24-, 48-, 72- and 96-hours post-birth, ileum and serum samples were obtained from euthanized pups. RLXN-1 levels in ileum samples (western blot) and RLXN-2 levels in serum samples (ELISA) were measured. Levels were compared between groups and time points by 2-way analysis of variance (ANOVA).

Results:
RLXN-1 levels were significantly decreased in NEC pups compared to age-matched CONTROLS ($p<0.05$) and NEC levels decreased over time (i.e., 12-hr > 24-hr levels and 24-hr > 48-hr levels, $p<0.05$). In the serum, RLXN-2 levels were decreased in NEC pups compared to CONTROL pups at every time point (12-, 24-, 72- and 96-hrs post-birth). RLXN-2 levels also decreased with time ($p<0.05$).

Conclusions:
Relative levels of vasodilators relaxin-1 and relaxin-2 decrease over time in neonatal rat pups with experimental NEC. Relaxin is present at higher concentrations in maternal milk than formula. The loss of a potent vasodilator in the small intestinal microvasculature could contribute to the worsening NEC vasoconstriction and hypoperfusion over time that leads to tissue ischemia, injury and death. These findings suggest that relaxin might be a potential therapeutic target for this disease process.

33

DOES EXOGENOUS GLUCAGON-LIKE PEPTIDE-2 IMPROVE CLINICAL OUTCOMES IN NEONATAL PIGLET MODELS OF SURGICAL SHORT BOWEL SYNDROME?

Megha Suri[1], Justine M. Turner[2], Patrick N. Nation[3], Pamela Wizzard[3], David L. Sigalet[4], Paul W. Wales[1].

[1]The Hospital for Sick Children, Toronto, ON, Canada, [2]Stollery Children's Hospital, Edmonton, AB, Canada, [3] University of Alberta, Edmonton, AB, Canada, [4]Alberta Children's Hospital, Calgary, AB, Canada.

Purpose:
Short bowel syndrome (SBS) is a significant cause of neonatal morbidity and mortality. Early adaptation and growth of the remnant intestine is desired in order to improve outcomes. Glucagon-like peptide-2 (GLP-2) is an intestinotrophic hormone, derived principally from the ileum, and has been shown to augment the intestinal adaptive response in several animal models of SBS and adult patients with the syndrome. We hypothesize that exogenous GLP-2 treatment will improve clinical outcomes in neonatal piglet models of short bowel syndrome that include the presence or absence of remnant ileum.

Methods:
Thirty neonatal piglets (mean age 3.0±0.2d; mean weight 2.2±0.0kg) were randomized to three surgical and two treatment groups. Surgical groups included: operative control (sham), 75% mid-intestinal resection with a jejunoileal anastomosis (JI), and 75% distal resection with a jejunocolic anastomosis (JC). Parenteral nutrition (PN) via a jugular-catheter commenced on day 1 and was weaned as enteral nutrition (EN) via a gastric-catheter was advanced to maintain the caloric intake. Treatment with GLP-2 (11nmol/kg/day) or saline was initiated by continuous infusion on day 2. Piglets were maintained for 14 days. Clinical, morphological and microscopic outcomes were obtained. Comparisons were made within surgical groups by one-way ANOVA for two independent means (alpha <0.05).

Results:
Clinical, morphological, & microscopic outcomes in sham, JI, JC pigs treated with GLP2 or saline

	Sham +Saline (n=4)	Sham +GLP2 (n=4)	JI +Saline (n=5)	JI +GLP2 (n=6)	JC +Saline (n=6)	JC +GLP2 (n=5)
Presence of diarrhea, days	1.8±1.0	1.8±1.1	8.4±0.8	8.8±0.8	11.8±0.3*	9.0±1.1
EN as sole nutrition, days	7.5±0.3	7.3±0.3	2.4±1.2	4.0±1.0	0.3±0.2	3.0±1.0*
Bowel length increase, %	36±9	25±1	32±2	70±9*	-1±3	13±5*
Jejunum - Villus height, um	609±22	776±35*	725±31	955±41*	688±23	710±33
Jejunum - Crypt depth, um	185±6	198±10	204±9	233±7	165±6	218±6*
Ileum - Villus height, um	926±62	631±39*	766±49	999±43*	--	--
Ileum - Crypt depth, um	172±7	164±8	194±9	193±6	--	--

Conclusion:
GLP-2 treatment improves morphological and microscopic parameters of intestinal adaptation in JI and JC neonatal piglets, compared to saline controls. Furthermore, improved clinical outcomes are observed in GLP-2 treated JC animals. Since the JC anatomical subtype (no remnant ileum) represents the majority of clinical cases of neonatal SBS, these results support the potential role of GLP-2 as a treatment for pediatric SBS.

NOTES:

34

FETAL INTERVENTION TRIGGERS THE ACTIVATION OF PATERNAL ANTIGEN-SPECIFIC MATERNAL T CELLS

Amar Nijagal, Marta Wegorzewska, Tom Le, Tippi C. MacKenzie, MD.

UCSF, San Francisco, CA, USA.

Purpose:
Preterm labor is a devastating consequence of fetal intervention. We have previously shown that maternal T cells mediate demise of the semi-allogeneic fetus after in utero transplantation (IUT). Based on these findings, we hypothesized that IUT triggers the activation of maternal T cells that specifically recognize the paternal alloantigen.

Methods:
We bred B6 (H-$2^{b/b}$) females to BALB/cxB6 (H-$2^{d/b}$) males such that half of the fetuses carried the foreign paternal antigen H-2^{d}. T cells that recognize this paternal antigen were obtained from T cell receptor transgenic mice, labeled with the membrane dye CFSE to detect proliferation, and transferred into pregnant B6 females at E13.5, followed by in utero transplantation (IUT) of B6 or BALB/c hematopoietic cells at E14.5. At E19.5, we measured the proliferation of paternal antigen-specific T cells in the uterine draining lymph nodes (udLN), spleen, and non-draining lymph nodes (ndLN).

Results:
IUT of both B6 and BALB/c donor cells led to a significant increase in the percentage of proliferating H-2^{d}-specific T cells in udLN compared to uninjected controls (uninjected: 14.3 ± 4.9%; B6 IUT: 71.4 ± 8.8%, $p<0.005$; BALB/c IUT 62.0 ± 8.6%, $p<0.005$). There was a similar increase in maternal T cell proliferation in the spleen (uninjected: 18.5 ± 3.8%; B6 IUT: 51.1 ± 10.0%, $p<0.05$; BALB/c IUT 45.1 ± 4.8%, $p<0.005$) but not in ndLN. The donor cell source had no impact on the degree of proliferation, demonstrating that fetal intervention itself is the primary factor responsible for maternal T cell activation.

Conclusions:
We show that fetal intervention results in the activation of maternal T cells which specifically recognize the paternal alloantigen. These findings suggest that fetal loss after intervention is similar to T cell-mediated graft rejection and that immunomodulatory strategies to suppress maternal T cell responses following fetal surgery may have clinical benefit.

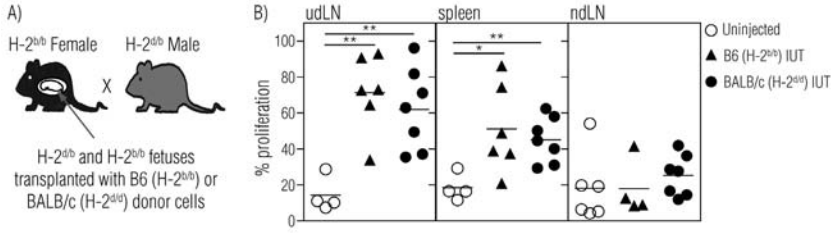


Figure: A) Schematic of breeding strategy used to evaluate the proliferation of T cells that recognize paternal antigen (H-2^d). B6 (H-$2^{b/b}$) females were bred to BALB/cxB6 (H-$2^{d/b}$) males such that half of the fetuses carried the foreign paternal antigen H-2^d. Fetuses underwent in utero transplantation (IUT) with either B6 or BALB/c donor hematopoietic cells. **B)** Proliferation of paternal antigen specific T cells in the uterine draining lymph nodes (udLN), spleen and non draining lymph nodes (ndLN) 5 days after IUT. *p-value <0.05, **p-value <0.005 by Student's t-test.

NOTES:

35

MESENCHYMAL STEM CELLS (MSC) AND HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) ACT SYNERGISTICALLY TO PROTECT THE INTESTINES FROM EXPERIMENTAL NECROTIZING ENTEROCOLITIS

Jixin Yang, MD, Daniel Watkins, MD, Chun-Liang Chen, PhD, Hong-Yi Zhang, MD, Yu Zhou, MD, PhD, Markus Velten, MD, Gail E. Besner, MD.

Nationwide Children's Hospital, Columbus, OH, USA.

Purpose:
We have previously shown that HB-EGF protects the intestines from experimental NEC. MSC are known to engraft into injured tissues to promote healing. The current study investigated whether MSC protect the intestines from NEC, whether HB-EGF promotes MSC engraftment, and whether MSC and HB-EGF can act synergistically to prevent NEC.

Methods:
NEC was induced in neonatal rats by exposure to hypoxia/hypothermia/hypertonic formula/LPS. MSC were delivered either intraperitoneally (IP) or intravenously (IV) via the umbilical vein. HB-EGF was administered enterally by addition to each feeding. Pups were randomly assigned to: 1) breast-feeding, 2) NEC, 3) NEC + HB-EGF, 4) NEC + MSC IP, 5) NEC + HB-EGF + MSC IP, 6) NEC + MSC IV, or 7) NEC + HB-EGF + MSC IV. MSC engraftment, histologic injury, intestinal permeability and mortality were determined.

Results:
IV-administered MSC had increased intestinal engraftment compared to IP-administered MSC (30.2±9.5 *vs.* 21.1±7.7 cells/section, p=0.0332). HB-EGF increased engraftment of IP-administered MSC (36.5±9.2 *vs.* 21.1±7.7 cells/section, p=0.0003) and IV-administered MSC (39.9±8.4 *vs.* 30.2±9.5 cells/section, p=0.0428). Pups in all experimental groups (Groups 3-7) had a decreased incidence of NEC and decreased intestinal permeability compared to non-treated pups (Group 2), with the lowest incidence of NEC (30.4% *vs.* 65.8%, p=0.0093) and lowest permeability (0.87±0.31 *vs.* 19.97±7.94 µg/ml, p<0.0001) in pups treated with HB-EGF+MSC IV. Pups exposed to experimental NEC had significantly decreased survival compared to breast-fed pups (23.7% *vs.* 100%, p=0.0021). Pups in all experimental groups (Groups 3-7) had significantly improved survival compared to non-treated pups (Group 2), with the best survival in pups treated with HB-EGF+MSC IV (73.9% *vs.* 23.7%, p=0.0401).

Conclusions:
HB-EGF and MSC act synergistically to reduce injury and improve survival in experimental NEC. This may represent a novel strategy for the prevention of NEC in the future.

NOTES:

36

HUMAN TISSUE-ENGINEERED SMALL INTESTINE FORMS FROM POSTNATAL TISSUE AS A MOUSE XENOGRAFT

Erik R. Barthel, MD, PhD, Daniel E. Levin, MD, Xiaogang Hou, PhD, Allison L. Speer, MD, Frédéric G. Sala, PhD, Yasuhiro Torashima, MD, PhD, Jamil A. Matthews, MD, Tracy C. Grikscheit, MD.

Children's Hospital Los Angeles, Los Angeles, CA, USA.

Purpose:
Tissue-engineered small intestine (TESI) represents a potentially durable cure for short bowel syndrome. In our technique, rodent and porcine organoid units (OU) prepared from resected bowel are grown on biodegradable scaffolds in vivo into fully differentiated intestine. Others have demonstrated the survival of fetal human intestinal xenografts following subcutaneous implantation in mice. However, for human therapy, OU would have to be derived from postnatal tissue. We hypothesized that our approach would effectively produce human TESI. Here, we give the first report of full-thickness human intestinal tissue derived from postnatal human small bowel OU.

Methods:
OU were prepared from deidentified human small bowel resection specimens obtained per an IRB-approved protocol, loaded onto biodegradable scaffolds and implanted into omenta of NOD/SCID gamma chain-deficient mice. TESI was harvested after 6 weeks, formalin-fixed and immunostained for β2 microglobulin to identify human tissue, villin for enterocytes, lysozyme for Paneth cells, chromogranin-A for enteroendocrine cells, mucin-2 for goblet cells, and smooth muscle actin and desmin to demonstrate muscularis.

Results:
Immunofluorescence of human TESI grown in NOD/SCID murine hosts reveals mucosal differentiation, muscularis and human origin (Figure 1). All four differentiated cell types of mature human small intestine are present, shown by villin (A), lysozyme (B), chromogranin-A (D), and mucin-2 (E) staining. Smooth muscle actin and desmin staining in the surrounding mesenchyme demonstrates the presence of a muscularis and supporting intestinal subepithelial myofibroblasts (C). Human β2-microglobulin staining establishes donor origin (F).

Conclusions:
Application of our technique to human tissue resulted in the survival, growth, and differentiation of postnatally derived human small intestinal organoid units into full thickness tissue-engineered small intestine in murine hosts. This is proof-of-concept for the reliable tissue engineering of human small intestine using a postnatal organoid units-on-scaffold approach. This regenerative medicine strategy may hold great promise for the treatment of short bowel syndrome in pediatric surgical patients.

37

METASTATIC POTENTIAL IN NEUROBLASTOMA AFTER DUAL INHIBITION OF VEGF AND NOTCH

Alejandro Garcia, MD, Debarshi Banerjee, PhD, Sonia Hernadez, PhD, Angela Kadenhe- Chiweshe, MD, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD.

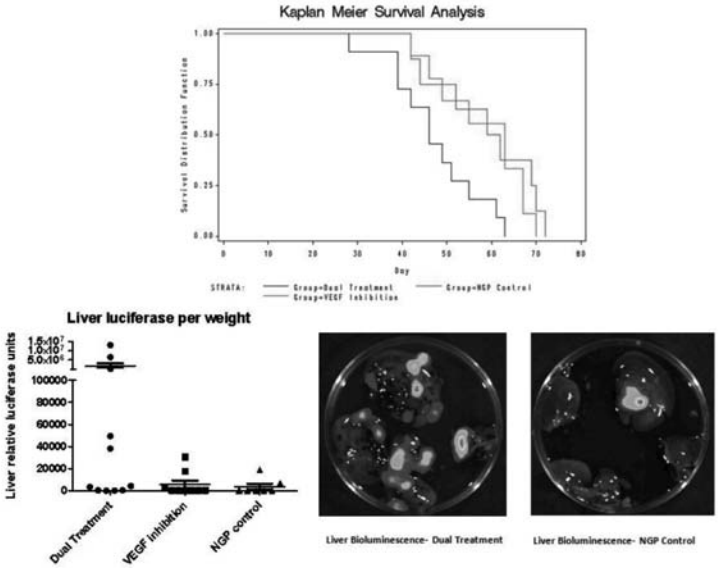
Columbia University, New York, NY, USA.

Purpose:
Vascular endothelial growth factor (VEGF) inhibition is a validated cancer treatment. However, by imposing hypoxia, VEGF blockade may paradoxically enhance metastasis. Notch proteins also function as key angiogenic effectors, and cross-regulate VEGF expression, raising the question of whether combined treatment would alter metastasis. We hypothesized that dual VEGF/Notch blockade would enhance hypoxia and metastatic propensity in experimental neuroblastoma.

Methods:
10[6] NGP neuroblastoma cells were xenografted intrarenally in nude mice. Animals were randomized to anti-VEGF antibody bevacizumab, Notch1-decoy, dual treatment, and vehicle control groups (N=30 each). Metastatic burden in target organs was quantified by bioluminescence. To examine inherent metastatic propensity, we cultured cells harvested from liver metastases of dual-treated and bevacizumab-treated mice, injecting 10[6] cells intravenously in nude mice (N=11, N=9 respectively), with parental NGP cells as controls (N=8). Metastatic growth was monitored via bioluminescence. IACUC approval was obtained for all experiments.

Results:
Combined treatment did not reduce NGP tumor growth compared to either agent alone. Versus controls, hypoxia increased in Notch and VEGF-inhibited groups ($p=0.05$, Kruskal-Wallis), with an additive effect of dual treatment versus either treatment alone ($p=0.005$, Kruskal-Wallis). Dual-treated mice exhibited increased liver metastatic burden by histology and bioluminescence compared to either treatment or controls ($p<0.001$, $p<0.005$ respectively, Kruskal-Wallis). Cells harvested from dual-treated animals' metastases resulted in greater liver metastasis after intravenous injection than cells harvested from bevacizumab-only metastases or controls ($p=0.043$, Kruskal-Wallis). Additionally, dual-treated metastasis-derived tumors progressed more quickly than bevacizumab-treated metastasis-derived ($p=0.017$, chi-square) and NGP tumors ($p=0.0306$, chi-square).

Conclusions:
Dual VEGF/Notch targeting of NGP tumors resulted in increased hypoxia and metastatic burden. Recent data indicates that tumoral hypoperfusion can promote progression, potentially by selecting for biologically aggressive behaviors. Our results suggest that the increased hypoxia imposed by dual Notch/VEGF blockade causes dissemination of cells with an enhanced propensity to metastasize. These data warrant further preclinical investigation.


Kaplan Meier Survival Analysis
Survival Distribution Function
1.00
0.75
0.50
0.25
0.00
0
10
20
30
40
50
60
70
80
Day
STRATA:
Group=Dual Treatment
Group=NGP Control
Group=VEGF Inhibition
Liver luciferase per weight
Liver relative luciferase units
1.5x10^7
1.0x10^7
5.0x10^6
100000
80000
60000
40000
20000
0
Dual Treatment
VEGF inhibition
NGP control
Liver Bioluminescence- Dual Treatment
Liver Bioluminescence- NGP Control

NOTES:

38

THE BACTERIAL RECEPTOR TOLL LIKE RECEPTOR 4 (TLR4) REGULATES THE NORMAL RECRUITMENT OF ADAPTIVE IMMUNE CELLS IN THE NEWBORN GUT IN THE PATHOGENESIS OF NECROTIZING ENTEROCOLITIS

Joyce Y. Lin, MD, Charlotte Egan, PhD, Matthew D. Neal, MD, Chhinder Sodhi, PhD, Ibrahim Yazji, MD, Misty Good, MD, Sapana J. Shah, MD, Amin Afrazi, Hongpeng Jia, MD, Maria Branca, Tom Prindle, Zachary Grant, David J. Hackam, MD, PhD, FACS.

Children's Hospital of Pittsburgh of UPMC, Pittsburgh, PA, USA.

Purpose:
Necrotizing enterocolitis (NEC) is thought to develop after an exaggerated inflammatory response to bacteria within the newborn intestine. Normal bacterial recognition in the newborn gut requires the regulated recruitment of adaptive immune cells (i.e. CD3+Tcells and CD45R+Bcells) through mechanisms that remain largely unexplored. A critical role for the bacterial receptor TLR4 in NEC pathogenesis has recently been identified, although the mechanisms involved remain incompletely understood. We now hypothesize that TLR4 within the newborn intestinal epithelium regulates normal recruitment of adaptive immune cells in the newborn intestine and thus plays a key role in NEC pathogenesis.

Methods:
The recruitment of adaptive immune cells of newborn and adult mice was determined by confocal microscopy on ileal whole-mounts using cell-specific markers that were subjected to 3-Dimensional rendering by IMARIS software. TLR4 was specifically deleted from the intestinal epithelium by breeding TLR4-loxp mice with villin-cre to yield epithelial-TLR4-knockout mice (TLR4-villin-CKO). NEC was induced in newborn mice by 4-days of formula gavage/hypoxia, and disease severity was assessed by cytokine expression and histology.

Results:
In wild-type mice, there was a significant increase in the recruitment of each adaptive immune cell subset over time, which was reflective of the development of an intact mucosal immune system. By contrast, in TLR4-villin-CKO mice, there was a striking decrease in the recruitment of each adaptive immune cell subset compared to wild-type mice ($p<0.05$), revealing a surprising and necessary effect of epithelial TLR4 on normal intestinal immune development. Strikingly, the loss of adaptive immune cells in TLR4-villin-CKO mice was associated with a marked reduction in NEC severity compared to wild-type mice, reflecting the physiological relevance of these findings.

Conclusions:
The bacterial receptor TLR4 plays a novel and important role in the recruitment of adaptive immune cells within the newborn intestine which plays an important role in the pathogenesis of NEC.

39

REGENERATION OF ENTERIC GANGLIA IN MECHANICALLY LENGTHENED JEJUNUM

Rebecca Stark, MD, Elvin Chiang, Ziyad Jabaji, MD, James CY Dunn, MD, PhD.

UCLA, Los Angeles, CA, USA.

Purpose:
In prior studies we demonstrated the ability to lengthen intestinal segments with mechanical force and the feasibility of their restoration back into intestinal continuity. The enteric nervous system represents an important component of the lengthened intestinal segment. We therefore analyzed the histological appearance of the intestinal ganglia in the lengthened and restored segments of the jejunum.

Methods:
A 1-cm segment of rat jejunum was isolated from continuity and was lengthened using a spring in vivo (n=10). After six weeks, the lengthened jejunum was either retrieved for analysis or restored back into intestinal continuity. Rats with restored, lengthened jejunal segments were euthanized at 2 and 5 weeks later for specimen retrieval. Immunostaining for S100 was performed to identigy ganglia. Ganglia were analyzed by the number per high-power field (pHPF) and were compared to those in the normal jejunum. Student's t-test was used for statistical comparison.

Results:
The normal jejunum had 10 ± 4.0 myenteric ganglia and 8.8 ± 1.0 submucosal ganglia pHPF. Lengthened jejunal segments had significantly fewer ganglia (3.8 ± 2.5 ganglia pHPF in the myenteric plexus and none in submucosal plexus, $p<0.05$). Jejunal segments that were restored back into intestinal continuity had a gradual return in the number of ganglia (3.3 ± 1.3 myenteric and 0.13 ± 0.18 submucosal ganglia pHPF after 2 weeks, $p<0.05$ compared to normal jejunum, and 9.2 ± 2.9 myenteric and 5.8 ± 2.2 submucosal ganglia pHPF after 5 weeks, $p>0.05$ compared to normal jejunm).

Conclusions:
Mechanical lengthening of the jejunum led to an absence of ganglia in the submucosal plexus and a paucity of ganglia in the myenteric plexus. After restoration of the lengthened jejunum back into intestinal continuity, the number of ganglia returned to a near normal state. The function of these regenerated ganglia needs to be assessed in the future.

NOTES:

40

AN ALTERNATE DNA REPAIR MECHANISM IN NEUROBLASTOMA

Erika A. Newman, MD, Daniela Bashilari, BS, Anthony Opipari, MD, PhD, Roland Kwok, PhD, Valerie Castle, MD.

The University of Michigan, Ann Arbor, MI, USA.

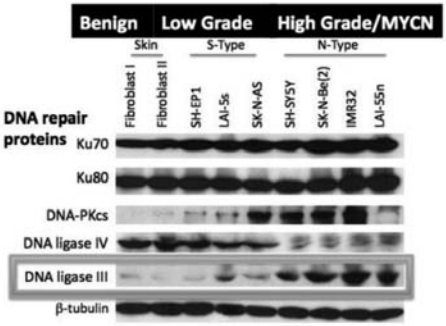
Purpose:
Neuroblastoma is an embryonic cancer of neural crest lineage and acquires genomic aberrations that correlate with clinical outcomes. The DNA damage machinery is emerging as a mechanism of genetic instability in cancer. Nonhomologous end-joining (NHEJ) plays a role in physiologic and defective DNA repair. Recent evidence has suggested that in contrast to canonical NHEJ, an alternate NHEJ (aNHEJ) pathway is functional and more prone to deletions and translocations. *We hypothesized* that aNHEJ is functional in neuroblastoma and is a novel mechanism of pathogenicity.

Methods:
Neuroblastoma (neuroblastic and Schwanninan), Ewing's sarcoma (TC 71 and AC73), ovarian cancer (CaOV3 and CaOv4), and skin fibroblasts were cultured. Lysates were analyzed for RNA and protein expression of DNA ligase III. Plasmid end-joining assays were used to determine end-joining activity and efficiency. We examined whether down-regulation of aNHEJ would decrease cellular proliferation using DNA Ligase III siRNA knockdown, growth curves, and BrdU incorporation. Using RNA expression data from public data sets (Oncomine.com and Oncogenomic.com), Kaplan-Meier survival curves were generated.

Results:
Neuroblastic neuroblastoma cells uniformly express higher levels of DNA Ligase III (DNA ligase III, ca. 4-fold elevation) and is highest in those tumors with MYCN overexpression, a pattern not observed in other cancers. Neuroblastoma tumors display increased end-joining activity that is highly inaccurate. DNA ligase III knockdown results in decreased cellular growth and proliferation in neuroblastoma. Higher expression of DNA Ligase III is associated with poor overall survival ($p<0.01$).

Conclusion:
DNA Ligase III, an important mediator of aNHEJ is highly expressed in neuroblastoma cell lines and contributes to overall cell survival. These data support a model in which, for the first time, neuroblastoma tumors display inaccurate DNA repair that contributes to pathogenesis and may drive genetic instability. Since DNA Ligase III expression correlates with poor patient survival, ongoing work is evaluating the therapeutic potential of this target in neuroblastoma.

DNA Ligase III expression is high in High-risk Neuroblastoma



NOTES:

INNOVATION SESSION
Abstracts on New and Innovative Techniques and Procedures

Tuesday, May 22, 12:45 – 1:45 p.m.

i1
NON-THERMAL PLASMA: AN INNOVATIVE TECHNOLOGY FOR TUMOR ABLATION

Ryan M. Walk, MD[1], Jacob Kirsch, BA[2], Priya Srinivasan, PhD[1], Lina Chakrabarti, PhD[1], Jason A. Snyder, MD[1], Felix C. Blanco, MD[1], Michael Keidar, PhD[2], Anthony D. Sandler, MD[1].

[1]Children's National Medical Center, Washington, DC, USA, [2]George Washington University, Washington, DC, USA.

Purpose:
Plasma is an ionized gas that is typically generated at high temperature, but recent breakthroughs have allowed for production of plasma at room temperature and atmospheric pressure. This non-thermal plasma (NTP) may offer the capability of delivering reactive oxygen species directly into tissues, potentially providing a novel modality for targeted cancer therapy. We sought to evaluate NTP's effect on neuroblastoma, both *in-vitro* and in an *in-vivo* murine model.

Methods:
NTP was generated from Helium gas. Neuroblastoma cells, both mouse (Neuro2a) and human (SK-N-SH and IMR32), were treated for 0, 30, 60, and 120 seconds and assayed for apoptotic and metabolic effects immediately, at 24 hours, and at 48 hours post-treatment. *In vivo,* mice were subcutaneously injected with neuroblastoma. Once tumors reached 5-millimeters in diameter, a single transdermal treatment was administered. Measured endpoints were tumor volume and survival. All mice were handled in accordance with Institutional Animal Care and Use Committee (IACUC) guidelines.

Results:
NTP decreased metabolic activity, induced apoptosis, and dramatically reduced viability of cancer cells in direct proportion to both duration of NTP exposure and time post-treatment. Accordingly, in the 120-second treatment group, 73% of cells were apoptotic and only 4% remained viable at 48 hours (both $p < 0.001$ *vs.* control). More importantly, *in-vivo,* a single treatment ablated tumors and eventual tumor growth velocity was slowed. Furthermore, survival nearly doubled, with a median of 15 *vs.* 28 days (Mantel-Cox p-value 0.002).

Conclusions:
Our findings demonstrate the remarkable sensitivity of neuroblastoma to NTP treatment, both *in-vitro* and in an *in-vivo* mouse model of established tumor. While further investigation is necessary to both definitively establish the mechanism and optimize the treatment protocol, these initial observations establish Non-Thermal Plasma as a potentially potent novel ablative therapy.

i2

DEVELOPMENT OF AN ARTIFICIAL PLACENTA V: 70 HOUR VENO-VENOUS EXTRACORPOREAL LIFE SUPPORT AFTER VENTILATORY FAILURE IN PREMATURE LAMBS

Brian W. Gray, MD, Ahmed El-Sabbagh, MD, Kelly L. Koch, Sara J. Zakem, Alvaro Rojas-Pena, MD, Raja Rabah, MD, Robert H. Bartlett, MD, George B. Mychaliska, MD.

University of Michigan, Ann Arbor, MI, USA.

Purpose:
An extracorporeal artificial placenta would change the paradigm of treating extremely premature infants who suffer substantial mortality and morbidity. We hypothesized that using a veno-venous extracorporeal life support (VV-ECLS) artificial placenta after the onset of ventilatory failure would stabilize hemodynamics, gas exchange, cerebral perfusion, and maintain fetal circulation for 70 hours.

Methods:
A near-term neonatal lamb model (130 days; term=145 days) was used (n=7). A hysterotomy was performed, and the right jugular vein (drainage) and umbilical vein (reinfusion) were cannulated with 10-12Fr cannulas. An endotracheal tube (ETT) was placed, and the lamb was transitioned to an infant ventilator. After respiratory failure (persistent pO2<60, pCO2>100, or hemodynamic instability), the ETT was filled with amniotic fluid and occluded, and VV-ECLS was initiated. Lambs were maintained on VV-ECLS for 70 hours.

Results:
Six of 7 lambs survived for 70 hours. Mean (±S.D.) ventilation time was 54±22 minutes. Hemodynamics and gas exchange during ventilation and VV-ECLS are displayed in Table 1. Mean VV-ECLS flow was 318±69mL/min. During VV-ECLS, mean carotid blood flow was 107±27mL/min, and cerebral near-infrared spectroscopy showed mean saturation of 52%±11%. Echocardiography was attempted during VV-ECLS in one animal and revealed right to left foramen ovale and ductus arteriosus flow. Necropsy showed a patent ductus arteriosus and no intracranial hemorrhage in all animals. Lung histology was normal in 4 lambs, with pulmonary hemorrhage in 1 and meconium aspiration in another.

Conclusions:
The VV-ECLS artificial placenta stabilized premature infants with ventilatory failure and maintained fetal circulation, hemodynamic stability, adequate gas exchange, and cerebral perfusion for 70 hours. Future studies will address the factors necessary for long-term support.

VV- ECLS = veno-venous extracorporeal life support; values shown in mean ± standard deviation						
Mode	MAP (mmHg)	HR (BPM)	pO2 (mmHg)	pCO2 (mmHg)	pH	Lactate (mmol/L)
Ventilation	51±14	157±38	115±136	89±30	7.06±0.15	5.7±2.3
VV-ECLS	44±14	191±29	38±7	48±8	7.33±0.07	2.0±1.8

NOTES:

i3

TISSUE EXPANDER STIMULATED LENGTHENING OF ARTERIES (TESLA): A NOVEL APPROACH TO THE TREATMENT OF MIDDLE AORTIC SYNDROME

Heung Bae Kim, MD, Khashayar Vakili, MD, Biren P. Modi, MD, Michael A. Ferguson, MD, Kristina M. Potanos, MD, Steven J. Fishman, MD.

Children's Hospital Boston, Boston, MA, USA.

Purpose:
To describe a novel method to induce longitudinal growth of the aorta in a child with middle aortic syndrome (MAS).

Methods:
This is a case report of the first patient to undergo Tissue Expander Stimulated Lengthening of Arteries (TESLA).

Results:
The patient is a 3 year old girl who presented with severe renovascular hypertension, lower extremity claudication and left kidney atrophy secondary to MAS and bilateral renal artery stenoses. The aorta had a hemodynamically signficant 3.5 cm long stenosis beginning just inferior to the SMA. Due to her small size and need for future growth, we sought an alternative to the conventional therapy of bypass grafting. We hypothesized that a retroaortic tissue expander could mimic the stretching of arteries observed with large retroperitoneal tumors and this might allow for a resection of the stenosis with primary anastomosis. During laparotomy for left nephrectomy, we placed a tissue expander posterior to the aortic bifurcation. This was slowly inflated over a period of 9 months by which time CT angiogram confirmed adequate aortic lengthening. At the final operation, we easily resected 4 cm of aorta, performed a primary aortic anastomosis, and reimplanted the right renal artery and inferior mesenteric artery. The child is doing well 5 months following her operation with equalization of upper and lower extremity blood pressures. Her hypertension has significantly improved and she is gradually weaning from medical therapy. Her renal function has normalized.

Conclusion:
We have demonstrated that the principle of stretch induced growth may be safely applied to lengthen the aorta to treat middle aortic syndrome. This novel approach to the lengthening of blood vessels may have broad implications in the treatment of vascular disorders in children and adults.

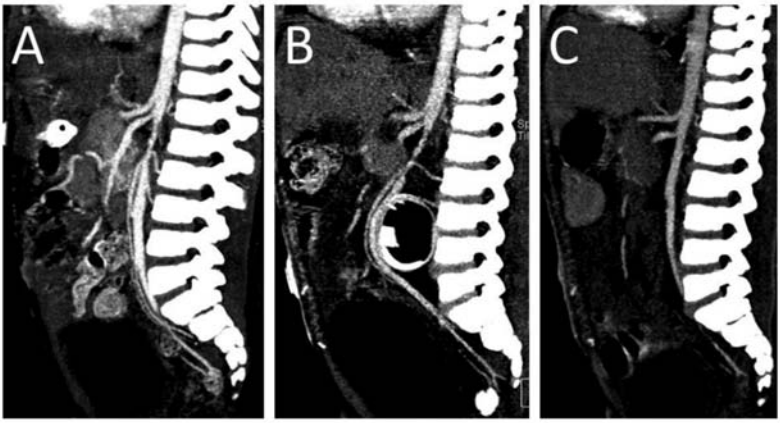


Figure 1: A) Preoperative CTA showing aortic stenosis
B) Tissue expander in retroaortic position stretching aorta
C) CTA after resection of stenosis with primary aortic anastomosis

NOTES:

i4

PERCUTANEOUS OBLITERATION OF PATENT PROCESSUS VAGINALIS: A RAT MODEL FOR FUTURE INGUINAL HERNIA REPAIR IN CHILDREN

Saad Al-Qahtani, Yassir Asiri, Ammar Al-Rikabi, Abdulrahman Al-Zahem, Ayman Al-Jazaeri.

College of Medicine, King Saud University, Riyadh, Saudi Arabia.

Purpose:
Inguinal hernia repair is one of the commonest procedures performed by pediatric surgeons. After Percutaneous intervention could be the next advancement in inguinal hernia repair in children. We are proposing a rat model for percutaneous inguinal hernia repair (PHR) using 2-octyl-cyanoacrylate (2OC).

Methods:
4-weeks old Lewis rats were randomly divided into 3 groups; bilateral PHR (BH) (n=15), unilateral PHR (UH) (n=11) and a sham group (n=14). After inducing a pneumoperitoneum via 24-guage cannula another 24-guage cannula attached to a saline filled syringe is introduced in the inflated patent processus vaginalis (PPV) while aspirating. Once air bubbles are seen in the syringe the cannula is advanced into the PPV. The canal is then obliterated by injecting 0.1 ml of 2-octyl-cyanoacrylate while saline is used in the sham group. Herniography and mating were performed at postoperative week 4 and 10, respectively. All rats were sacrificed at week 12 and their inguino-testicualr areas were harvested for histological examination.

Results:
All rates survived the procedures. Herniography revealed complete closure of PPV in 25/30 of BH, 11/11 of UH and 0/28 of the sham group. All 2OC treated sides were found obliterated at the post-mortem gross examination. Histological analysis revealed patent vase in all rats however, peri-sac mild to moderate foreign body reactions and fat necrosis were noticed in the injected sides. All rats demonstrated fertility after mating.

Conclusions:
Our rat model demonstrates that percutaneous obliteration of PPV can be safe and feasible. The procedure is potentially less invasive alternative to open or laparoscopic techniques in pediatric group however, the need for pneumoperitoneum and the risk of adhesive material intraperitoneal spillage remain the main challenges to overcome.

NOTES:

i5

DEVELOPMENT OF A NOVEL, CLOSED-SYSTEM, IMPLANTABLE SENSOR TO ASSESS THE MECHANOBIOLOGY OF FETAL LUNG MATURATION

Mozziyar Etemadi, MS, Samuel C. Schecter, MBBS, Eveline H. Shue, MD, James A. Heller, BS, Shuvo Roy, PhD, Douglas Miniati, MD.

UCSF, San Francisco, CA, USA.

Purpose:
Proper lung growth and development depend on fluid mechanical forces that occur during gestation. These forces are determined in part by pressure within the tracheobronchial tree and modulated by lung fluid secretion rate, glottic activity, and fetal diaphragmatic contractions. The purpose of the study was to develop a high fidelity, completely implantable, closed monitoring system to characterize pressures sensed by the lungs throughout gestation.

Methods:
We custom manufactured devices using an Omega PX-26 differential pressure sensor, analog amplifier/filter, data acquisition unit, and microSD card for data storage (Figure 1A). At 110 days gestation, sensors were implanted into the trachea and oropharynx of fetal lambs (n=6). Fetuses then continued gestation until devices were retrieved for data collection.

Results:
Benchtop ex vivo validation demonstrated accurate recording with 0.2cm water pressure resolution at a rate of 256Hz with near zero drift. Six devices were implanted into 6 lambs/ewes. Average operative time for implantation was 3 hours. Of the first four operations, there were two spontaneous abortions, one maternal death, and one preterm operative delivery. The final two procedures successfully maintained pregnancy until scheduled operative delivery. Mean recording time was 8 days ± 3.6 days. Tracheal pressure has a wide dynamic range—over 35 cm H2O—and also a unique set of "breath" morphologies that can be quantified using this system. Comparison of oropharyngeal and tracheal pressures indicated periods of oropharynx/glottis opening and closing. An example tracing is shown in Figure 1B.

Conclusion:
We successfully created a highly sensitive, completely implantable pressure-sensing device and used it to take longitudinal recordings of intratracheal and oropharyngeal pressures for up to 13 days. Future work will further characterize fetal breathing patterns and correlate these patterns to biological changes in the developing lungs, in both normal and pathologic states, such as congenital diaphragmatic

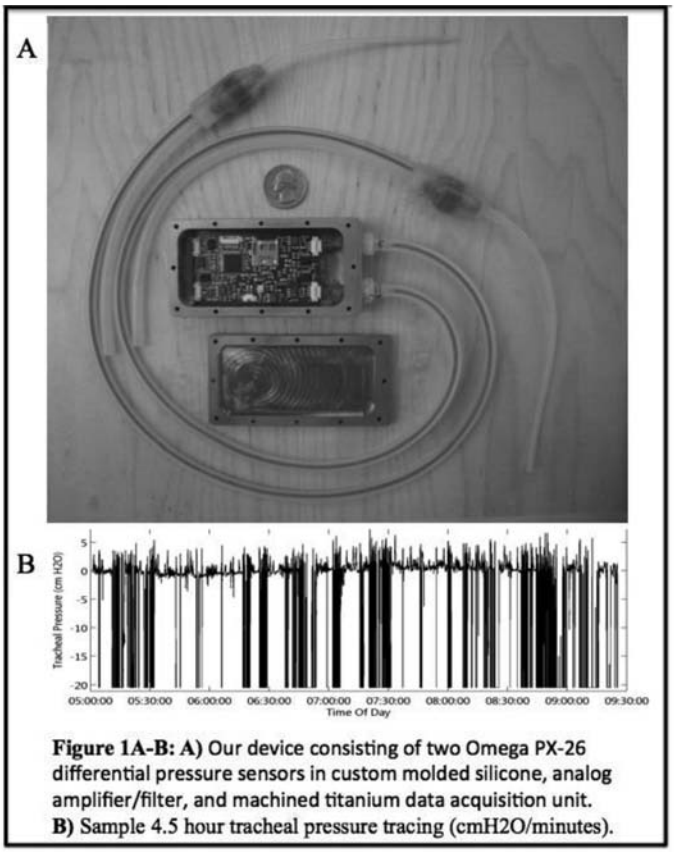


Figure 1A-B: A) Our device consisting of two Omega PX-26 differential pressure sensors in custom molded silicone, analog amplifier/filter, and machined titanium data acquisition unit. **B)** Sample 4.5 hour tracheal pressure tracing (cmH2O/minutes).

NOTES:

i6

ROBOIMPLANT (REMOTELY OPERATED BIONIC ORTHO IMPLANT) II: DEVELOPMENT, DESIGN, AND TESTING OF A CONTROLLER FOR NONINVASIVE ACTUATION OF AN IMPLANTED TELESCOPIC ROD USED TO CORRECT STRUCTURAL DEFORMITIES

Jonathan A. Liu, MS, James A. Heller, BS, Mozziyar Etemadi, MS, Dillon A. Kwiat, BS, RichardFechter, BS, Shuvo Roy, PhD, Michael R. Harrison, MD.

University of California, San Francisco, CA, USA.

Purpose:
An implantable rod system capable of being lengthened and shortened noninvasively would greatly improve the treatment of skeletal deformities like early onset scoliosis and limb length discrepancies. We have developed and tested an expandable rod system which is driven externally using magnetic coupling. We have developed and tested an external controller that: 1) senses when the controller and implant are coupled, 2) accurately records the progress of lengthening or shortening, and 3) measures and records the torque produced and calculates the force exerted on the internal structures by the expanding rod.

Methods:
The system is built using off-the-shelf components to actuate noninvasively an implantable telescopic rod using wireless, magnetic coupling. The designed system monitors the progress of treatment, measures the extension or retraction in the form of turns delivered, and measures the torque required to actuate the telescopic rod.

Results:
After characterization, the system was shown to reliably produce implant displacement within a 1.35 mil (34 μm) accuracy and to measure torque deviations on the order of 20 mNm. This extension sensitivity is much smaller than the typical daily lengthening dosage of 1 mm. The entire system - driver and implant - can deliver up to 300 N of force.

Conclusion:
The construction of a low-cost, noninvasive actuator presents a significant improvement over the current standard. This system allows extension or shortening with micron-scale accuracy along with measurement of the force required to do so. Precise control over the distraction process along with knowledge of the forces applied during treatment allows physicians for the first time to monitor progress using real-time data and thus gain insight into the factors that influence the biologic process of bone and soft tissue remodeling under stress. This new data-driven treatment will facilitate new approaches correcting to structural anomalies.

NOTES:

i7

WIRELESS MONITOR FOR DATA-DRIVEN TREATMENT OF *PECTUS CARINATUM*

Jonathan A. Liu, MS[1], Mozziyar Etemadi, MS[1], Marcelo Martinez Ferro, MD[2], Asis Lopez, BS[1], Dillon A. Kwiat, BS[1], Shuvo Roy, PhD[1], Michael R. Harrison, MD[1].

[1]University of California, San Francisco, CA, USA, [2]Fundación Hospitalaria Children's Hospital, Buenos Aires, Argentina.

Purpose:
Pectus carinatum can be successfully treated using the Dynamic Compression System, a noninvasive bracing system which applies force to the chest wall deformity over a period of months to years in order to gradually reshape the chest cartilage. Although the initial pressure required to correct the deformity can be measured, it would be useful to measure, record, and share information on the applied forces over the entire compression and remodeling process and gauge patient compliance. This work describes a wireless monitoring system capable of use in conjunction with current bracing systems to provide real-time, accurate monitoring of the corrective process.

Methods:
A wireless data logger is built using Bluetooth 2.0 technology and low-cost, off-the-shelf microcontrollers to form a device capable of monitoring the pressure applied to the pectus carinatum defect over time. The monitoring system is designed for integration with the current braces used for pectus carinatum.

Results:
Benchtop testing of the wireless data logger shows that pressure data can be streamed to a nearby Bluetooth-enabled device, which can send that data to a centralized location (the "cloud") for remote monitoring by physicians, family, and regulatory bodies, such as the FDA. On-board temperature and pressure sensors are capable of measuring patient compliance. Furthermore, the data logger's small size allows it to be easily integrated into existing braces, making it an instant upgrade to current bracing equipment.

Conclusion:
We demonstrate that a low-cost solution is available and capable of monitoring the forces required to treat pectus carinatum. Using off-the-shelf components, we are able to wirelessly transmit pressure and temperature data to any nearby Bluetooth-enabled device for remote monitoring and collection. This data is useful not only to assess treatment progression and compliance, but also to enable physicians to adjust the bracing pressure or protocol over time to best treat patients.

NOTES:

i8

DEVELOPMENT OF AN ISOLATION BED FOR PATIENTS UNDERGOING MIBG TREATMENT FOR NEUROBLASTOMA

Sabina Siddiqui, MD[1], Sean Vance, AIA[2], Laura L. McCormick, PhD[3], Douglas Mullen, PhD[3], Hannah J. Hensel, MBA[3], James D. Geiger, MD[1].

[1]University of Michigan, Department of Pediatric Surgery, Ann Arbor, MI, USA, [2]University of Michigan, School of Art and Architecture, Ann Arbor, MI, USA, [3]University of Michigan, Medical Innovation Center, Ann Arbor, MI, USA.

Purpose:
Neuroblastoma carries a grim prognosis with cure rates of only 25-30% in children >18 months of age with metastatic disease. Treatment with targeted radioactive metaiodobenzyguanidine (MIBG) shows a decrease in tumor burden and remission; however, the children become 'radioactive' for a three to five-day period and requiring strict isolation. The radiation poses a risk for health care workers, family members, and the patient.

Methods:
Focused observations of children being cared for during treatment with MIBG identified four key problem areas. These were researched and validated through further observations and engagements with patient, families and clinical staff. A multi-disciplinary team of nuclear physicists, safety experts, architects, engineers, physicians and nurses participated in a brainstorming event. Participants were educated about the key issues then generated solutions for treatment. These solutions were evaluated for feasibility and impact then advanced by the design team.

Results:
Over a hundred solutions were generated in the key areas of safe urine disposal, hands-free delivery of the radioactive drug, comprehensive room design and the 'isolation bed'. The final design includes a protected urine collection system, Geiger counter, IV pump, and retractable "awning" shield to allow transport. These features are combined into a thematic bed equipped with audiovisual capabilities to facilitate patient-family interaction.

Conclusions:
The designed bed specifically allows a patient to remain in isolation while allowing the medical staff and family to safely interact with the child. In addition, the bed allows a safe method of egress for the patient should the patient require evacuation from the hospital or transfer to an area of higher acuity of care. The principles of the isolation bed can be applied to other disease processes - including isolation for immunosuppressed patients as well as patients requiring respiratory isolation (i.e. H1N1 patients).

NOTES:

VIDEO SESSION
Tuesday, May 22, 1:45 – 2:45 p.m.

V1
LAPAROSCOPIC RESECTION OF A TYPE IV CHOLEDOCHAL CYST COMBINED WITH PANCREATIC HEAD RESECTION IN A 5-YEAR-OLD GIRL

Hans Joachim Kirschner, MD[1], Guido Seitz, MD[1], Dietmar Stueker, MD[2], Juergen Schaefer, MD[3], Joerg Fuchs[1].

[1]Department of Pediatric Surgery and Pediatric Urology, University Children`s Hospital, Tuebingen, Germany ,[2]University Department of General, Visceral and Transplant Surgery –Surgical Endoscopy, University of Tuebingen, Tuebingen, Germany, [3]Department of Diagnostic and Interventional Radiology – Pediatric Radiology, University of Tuebingen, Tuebingen, Germany.

Purpose:
We present a case of a five-year-old girl suffering from severe recurrent pancreatitis. Diagnostic work up including MRCP and ultrasound revealed a type IV choledochal cyst with multiple concretions in the pancreatic duct. ERCP was carried out in order to remove the concretions, which failed twice. Therefore, a decision was taken to perform a laparoscopic choledochal cyst resection combined with duodenum-preserving pancreatic head resection.

Methods:
A four trocar technique was used for the minimal invasive approach. After resection of the choledochal cyst, a duodenum-preserving pancreatic head resection was carried out. Multiple concretions were removed from the pancreatic duct. A pancreatico-jejunostomy and a hepatico-duodenostomy were carried out.

Results:
We performed a successful laparoscopic choledochal cyst resection combined with pancreatic head resection, without compromising the duodenum. Postoperatively, the child had a normal bile flow and only a mild dilatation of the pancreatic duct without stones. At follow up after 12 months, no symptoms of pancreatitis or cholangitis occurred.

Conclusion:
Combined minimal invasive resection of a choledochal cyst with duodenum-preserving pancreatic head resection can be carried out safely in smaller children. This video highlights the essential steps of the operation.

NOTES:

V2

CLOACAL EXSTROPHY — OPERATIVE MANAGEMENT OF THE NEWBORN

Marc A. Levitt, MD, Andrea Bischoff, MD, Alberto Peña, MD.

Cincinnati Children's Hospital, Cincinnati, OH, USA.

Purpose:
Management of the newborn with cloacal exstrophy represents a significant challenge, requiring correct surgical interventions and multi-disciplinary collaboration, which dramatically impact the ultimate functional result, realized many years later.

Methods:
A video was recorded highlighting the important technical details of the surgical management of the newborn with cloacal exstrophy.

Results:
The principles of the newborn surgical management are the omphalocele repair, bladder closure (primarily or staged), and creation of a true end colostomy, having the ultimate goals of achieving dryness for urine, cleanliness for stool, and sexual function. The cecal plate is removed from within the two hemibladders. The area behind the hemibladders must be carefully inspected looking for the hindgut. The mesentery of the hindgut is carefully preserved. The cecal plate is closed and an end stoma is made. Mullerian structures should be inspected to rule out atresia in female patients. The omphalocele is incised and a primary or delayed closure is performed. The ureteral orifices are canulated and the two hemibladders are approximated for a delayed closure or rarely, a primary bladder closure.

Conclusions:
The neonatal surgical repair of cloacal exstrophy should be performed with the future possibility of an intestinal pullthrough in mind, incorporating all colon in the intestinal tract. The decision regarding which tissues to use for genitourinary reconstruction should be coordinated with the ultimate colonic pull-through plan.

NOTES:

V3

LAPAROSCOPIC ASSISTED PSARP FOR RECTO-BLADDERNECK AND HIGH PROSTATIC FISTULA

Marc A. Levitt, MD, Andrea Bischoff, MD, Alberto Peña, MD.

Cincinnati Children's Hospital, Cincinnati, OH, USA.

Purpose:
10% of male patients with anorectal malformation are born with the highest type that is called recto-bladderneck fistula which is unreachable posterior sagitally only and represents an ideal indication for a laparoscopic assisted repair.

Methods:
A video was recorded highlighting the important technical details of a laparoscopic assisted posterior sagittal anorectoplasty for recto-bladderneck fistula.

Results:
The distal rectum is identified near the peritoneal reflexion, and the peritoneum around it is divided, remaining as close as possible to the rectal wall in order to avoid injuries to vas deferens, ureters, and nerves. The dissection continues circumferentially and distally to the point where it narrows down and meets the bladderneck. The fistula is divided and an endoloop is used to ligate it. Cauterization and division of avascular attachments of the rectum allows gaining of rectal length. The center of the sphincter is determined with the use of an electric stimulator and a minimal posterior sagittal incision is made with the legs elevated. A plane of dissection and a space in front of the sacrum is created, immediately behind the urethra, up to the peritoneal cavity. A laparoscopic dissection is carried out behind the bladder to meet the perineal dissection. The distal rectum is pulled down, assuring the correct orientation. When further rectal dissection is required, selective ligation of the peripheral branches of the inferior mesenteric vessels is performed. The bowel wall should be kept intact to preserve its intramural blood supply. The posterior sagittal incision is closed in layers. The posterior edge of the muscle complex is tacked to the posterior rectal wall which helps to avoid prolapse and the anoplasty is performed.

Conclusions:
The combination of laparoscopy and PSARP allows for a safe reconstruction in cases of recto-bladderneck and in selected high prostatic fistulas.

NOTES:

V4

THORACOSCOPIC DISTAL ESOPHAGECTOMY FOR CONGENITAL ESOPHAGEAL STENOSIS FROM TRACHEOBRONCHIAL REMNANTS

Eveline H. Shue, MD, Hanmin Lee, MD, Shinjiro Hirose, MD.

University of California San Francisco, San Francisco, CA, USA.

Purpose:
Congenital esophageal stenosis is a rare diagnosis with an incidence of 1 in 25,000 to 50,000 live births, and can be due to membranous webs, fibromuscular hypertrophy, or tracheobronchial remnants. Tracheobronchial remnants in the esophagus are thought to be caused by anomalous division of the respiratory and gastrointestinal tracts during gestation. We present a successful vagus-sparing thoracoscopic distal esophageal resection with esophago-esophagostomy to treat congenital esophageal stenosis.

Methods:
A 15 month old healthy girl was referred to our institution for congenital esophageal stenosis. She tolerated liquids, but would regurgitate undigested food. She failed multiple attempts at endoscopic balloon dilation. Esophageal manometry showed normal peristalsis, and endoscopic ultrasound showed a short segment distal esophageal stricture. An esophagram showed a distal esophageal stenosis with proximal dilation. She was referred for surgical management and offered a thoracoscopic distal esophagectomy.

Results:
Using a standard 3 port left-sided thoracoscopy, the distal esophagus was exposed, and the left and right vagus nerves were carefully identified and preserved. To delineate the extent of the esophageal stenosis, the anesthesiologists passed a Foley catheter into the stomach. The Foley balloon was inflated and the catheter was retracted until there was resistance, which defined the distal margin of the esophageal stenosis. The balloon was deflated and reinflated proximal to the esophageal stenosis to delineate the proximal margin. This was also confirmed using intraoperative endoscopy. The stenosis was resected, and a primary hand-sewn esophago-esophagostomy was performed. The patient's postoperative course was uneventful. A postoperative esophagram showed no anastamotic leak, and no residual esophageal stenosis. She tolerated solid food, and her chest tube was removed on postoperative 6. She was discharged on postoperative day 7. Pathology showed congenital esophageal stenosis due to tracheobronchial remnants.

Conclusions:
Distal esophagectomy for congenital esophageal stenosis can be accomplished through minimally invasive techniques without increased morbidity.

NOTES:

V5

LAPAROSCOPIC MEDIAN ARCUATE LIGAMENT RELEASE FOR SYMPTOMATIC CELIAC ARTERY COMPRESSION: 2 CASES WITH VIDEO

James Wall, MD, Matias Bruzoni, MD, Sanjeev Dutta, MD.

Lucile Packard Children's Hospital at Stanford, Palo Alto, CA, USA.

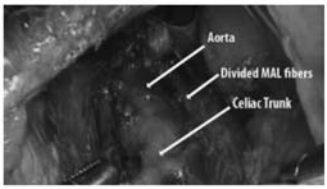
Purpose:
Median arcuate ligament (MAL) syndrome is characterized by postprandial abdominal pain and weight loss resulting from narrowing of the celiac artery by the insertion of the diaphragmatic crural muscle fibers. The celiac artery can also be partially obstructed by excessive nerves in the local celiac ganglia. The syndrome is rare and often considered a diagnosis of exclusion requiring a battery of imaging and endoscopic exams. Evolving MRI sequences offer promise in diagnosis of the syndrome. Treatment by MAL release has been widely reported and the first laparoscopic approach was reported in 2000. To date, only one case of laparoscopic MAL release has been reported in an adolescent.

Methods:
One 12-year-old male patient and one 17-year-old female were referred for post prandial pain and weight loss. Both patients underwent GI workup including endoscopy that did not discover an underlying cause. MRI revealed significant dynamic celiac axis obstruction with respiration and gastric distention. Additionally, a fixed post stenotic dilatation of the celiac trunk was seen in one patient. Laparoscopic MAL release was performed in both cases.

Results:
MAL release and complete skeletonization of the celiac trunk was performed successfully in both cases via a laparoscopic transperitoneal approach with five 5 mm trocars. Operating times were 127 minutes and 138 minutes with hospital stays of 1 and 2 days respectively. Laparoscopic ultrasound confirmed elevated peak flows in the celiac artery in one case. On short-term follow-up both patients reported improvement in postprandial abdominal pain.

Conclusions:
Laparoscopic MAL release is feasible in the adolescent population. Dynamic MRI imaging and laparoscopic ultrasound are emerging modalities in the diagnosis of MAL.

NOTES:



V6

LAPAROSCOPIC REPAIR OF DIAPHRAGM EVENTRATION

Oliver B. Lao, MD, MPH, Shahab F. Abdessalam, MD.

Children's Omaha, Omaha, NE, USA.

Surgical repair of diaphragmatic eventration, in the form of plication or tightening of the diaphragm, was first described in 1923. Since then, a variety of open and minimally invasive techniques have been described, with approaches from the abdomen and the chest, and reported advantages and disadvantages for both.

We demonstrate the use of an endostapler in a minimally invasive eventration repair in a pediatric patient. In contradiction to most other reported repairs, we approach the repair in a minimally invasive fashion through the abdomen. We invert the redundant diaphragm downward for our plication given this approach. We feel that this allows for better visualization of the intra-abdominal organs, avoids the pain and thoracostomy tube associated with a thoracoscopic procedure and gives a much more reliable and reproducible result. In addition the procedure can be done, on average, in less than 30 minutes, and it can be done as an outpatient procedure.

NOTES:

SCIENTIFIC SESSION V
Clinical Pediatric Surgery

Wednesday, May 23, 8:00 – 9:15 a.m.

41
INFLUENCE OF NEW TECHNOLOGY ON PRACTICE PATTERNS AND OUTCOME: THE CASE OF THE PREFORMED SILO

Edmund Yang, MD PhD[1], Derek Banyard, MD[2], Catherine Dale, MD[3].

[1]St. Louis Fetal Care Institute, Cardinal Glennon Children's Medical Center, St. Louis, MO, USA, [2]Department of Surgery, Charleston Area Medical Center, Charleston, WV, USA, [3]Department of Surgical Sciences, Vanderbilt University, Nashville, TN, USA.

Purpose:
New surgical technology can lead to new clinical uses yet, improved outcome is not always evident. We reviewed historical trends in the use of preformed silos for gastroschisis to determine if practice patterns for use altered over time and if improved outcome was evident.

Methods:
Clinical records of gastroschisis patients between 1990 and 2008 were reviewed for multiple variables (IRB #040701, Vanderbilt University). The first preformed silo was used in June 2001, so the eras before and after were defined as pre and post-silo. Patients were scored into groups of primary repair (PR), failed attempted primary repair (FA), or routine silo (RS) if no attempted intestinal reduction was documented. Patients who required a mandatory silo for physiologic or anatomic reasons were excluded.

Results:
There were 238 gastroschisis patients. In the pre-silo era, 75% (59 of 79) had PR and 9% (7) had RS. Yet in the post-silo era, only 52% of patients (82 of 159) had PR and 26% had RS (41, $p<0.0001$, Fisher's exact). Furthermore, 22% of the post-silo operations (35 of 159) were performed in the NICU, resulting in 91% RS placement (32). Different surgeons (11) had widely different rates of RS use, ranging from 0% to 59%. Encounter between 12 and 4AM also resulted in the highest RS use, equaling 29% and 50% in the pre and post-silo eras. Mean LOS increased from 36 to 44 days between eras ($p=0.17$, t test).

Conclusions:
Incorporation of the preformed silo into gastroschisis care was associated with more frequent RS use, often in the NICU, and a lower rate of PR. Surgeon-specific practice and time of encounter also appeared to influence RS use. These changes in practice were associated with a trend towards increasing LOS. New technology can change clinical practice; however outcome can be imperceptibly altered and must be periodically monitored.

NOTES:

42

SINGLE INCISION VERSUS STANDARD 4-PORT LAPAROSCOPIC CHOLCYSTECTOMY: A PROSPECTIVE RANDOMIZED TRIAL

Daniel J. Ostlie, MD, Obinna O. Adibe, MD, David Juang, MD, Corey W. Iqbal, MD, Susan W. Sharp, PhD, Charles L. Snyder, MD, Walter S. Andrews, MD, Ronald J. Sharp, MD, George W. Holcomb, III, MD, Shawn D. St. Peter, MD.

Children's Mercy Hospital, Kansas City, MO, USA.

Purpose:
Laparoscopy through a single umbilical incision is an emerging technique supported by several case series, but prospective comparative data are lacking. Therefore, we conducted a prospective, randomized trial comparing single site umbilical laparoscopic cholecystectomy to 4-port laparoscopic cholecystectomy.

Methods:
After IRB approval (09 07-132), patients were randomized to laparoscopic cholecystectomy via a single umbilical incision or standard 4-port approach. The primary outcome variable was operative time. Using historical operative times from our institution and utilizing a power of 0.8 and an alpha of 0.05, a sample size of 30 patients were calculated for each arm. Patients with signs of inflammation, complicated disease or weight over 100 kg were excluded. Single site cases were performed using the Covidien SILS® port with additional retracting instrument alongside the port, but within the umbilical incision. Post-operative management was controlled. Surgeons subjectively scored degree of technical difficultly from 1 = easy to 5 = difficult.

Results:
From 8/2009 through 7/2011, 60 patients were enrolled. There were no differences in patient characteristics (Table). Operative time and degree of difficulty were greater with the single site approach (Table). There were more doses of analgesics used and greater hospital charges in the single site group which trended toward significance.

Conclusions:
The single umbilical incision approach to laparoscopic cholecystectomy requires longer operative times and are associated with a greater degree difficulty as assessed the surgeon. There was a trend toward more doses of post-operative analgesics and greater hospital charges with the single site approach single.

Table			
	Single Incision(N=30)	4-Port (N=30)	P-Value
Age (yrs)	14.0 +/- 3.2	13.3 +/- 3.3	0.39
Weight (kg)	55.0 +/- 19.4	59.7 +/- 24.0	0.40
Gender (% female)	80%	80%	0.99
Gallstones (% present)	50%	56.7%	0.7
Operative Time (mins)	68.6 +/- 22.1	56.1 +/- 22.1	0.03
Difficulty Rating (1-5)	2.7 +/- 1.0	1.9 +/- 0.8	0.005
Total Analgesic Doses	16.4 +/- 17.8	10.1 +/- 4.3	0.06
Postoperative Length of Stay (hours)	22.7 +/- 6.2	22.2 +/- 6.8	0.44
Hospital Charges ($)	29.7K +/- 27.3K	20.6K +/- 6.9K	0.08

NOTES:

43

CARDIOVASCULAR RECOVERY FOLLOWING BARIATRIC SURGERY IN EXTREMELY OBESE ADOLESCENTS: PRELIMINARY RESULTS USING CARDIAC MAGNETIC RESONANCE (CMR) IMAGING

Marc P. Michalsky[1], John A. Bauer, PhD[1], Steven Teich, MD[1], Dara P. Schuster, MD[2], Subha V. Raman, MD[2].

[1]Nationwide Children's Hospital, Columbus, OH, USA, [2]The Ohio State University Medical Center, Columbus, OH, USA.

Purpose:
Although the prevalence of extreme childhood obesity and associated comorbidities has led to increased utilization of weight loss surgery (WLS) in adolescents, baseline cardiovascular characteristics and longitudinal changes following WLS in this population are not well defined. Our experience suggests that the use of transthoracic echocardiography (TTE) for preoperative cardiac assessment may provide suboptimal imaging (due to increased upper body adiposity) and therefore limit perioperative cardiovascular risk assessment. CMR, noted for high fidelity imaging in obese subjects, has emerged as an extremely useful diagnostic tool. We report the use of CMR imaging in a small cohort of extremely obese adolescents undergoing WLS.

Methods:
A retrospective analysis of adolescent WLS patients at a single institution was conducted. Data collection included mean age, sex, baseline and longitudinal body mass indices (BMI) and routine CMR measures (left ventricular mass (LVM), LV end-diastolic volume (LVEDV), ejection fraction (EF) and myocardial perfusion reserve indices (MPRI)). Comparison of CMR results to normative non-obese control data ("expected normal values") was conducted using Student's t-tests.

Results:
Ten subjects (9 female) with a mean age and BMI of 15 years and 51.75 ± 0.21kg/m^2 respectively, were studied. Comparison of obese adolescents (OB) vs. normal weight (NW) controls (Figure 1A-1D) shows evidence of increased LVM (122±25gm vs. 101±10gm), and LVEDV (156±25mL vs. 109±9mL), with an average EF of 61.5±5% (range 52% to 67%) vs. 71% and 74% expected EF for males and females, respectively and reduced MPRI (1.83±0.4 vs. 4.2±1.1). Five subjects demonstrated adenosine-induced subendocardial ischemia (SEI) pre-WLS with complete normalization (3/5) or marked improvement (2/5) following WLS. LVM was reduced an average of 8gm (range 2 to 12gm) in four subjects.

Conclusion:
Extreme adolescent obesity is associated with significant structural and functional cardiovascular pathophysiology. Reversal of cardiac perfusion deficits and structural abnormalities is associated with surgical weight reduction.

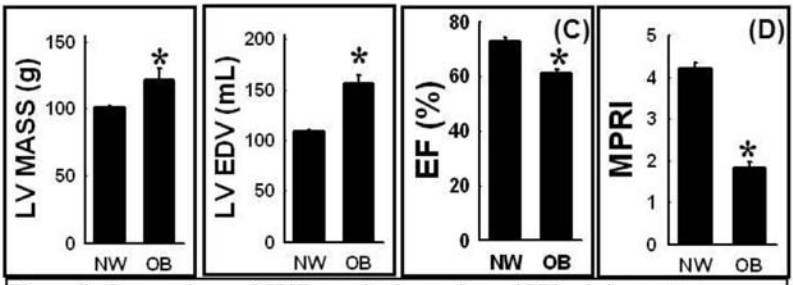


Figure 1: Comparison of CMR results from obese (OB) adolescents to published normal weight (NW) normative reference values. (A) Left ventricular (LV) mass, **(B)** LV end diastolic volume, **(C)** LV ejection fraction, **(D)** Myocardial Perfusion Reserve Index (MPRI). * p<0.01

NOTES:

44

FACTORS INFLUENCING THE OUTCOME OF PORTAL REPERFUSION FOR EXTRA HEPATIC/PORTAL VENOUS OBSTRUCTION (EHPVO) – A 15-YEAR EXPERIENCE

Florent Guerin, MD[1], Valeska Bidault, MD[1], Sabine Irtan, MD[2], Frédéric Gauthier, PhD[1], Virginie Fouquet, MD[1], Hélène Martelli, PhD[1], Sophie Branchereau, MD[1].

[1]Bicêtre Hospital, Le Kremlin Bicêtre, France, [2]Necker-Enfants Malades, Paris, France.

Purpose:
To analyze the factors predicting a successful mesenteric to left portal branch venous bypass (portal reperfusion =PR) for EHPVO.

Methods:
A monocentric retrospective study of 69 patients who underwent surgery for EHPVO from 1996 to 2010. Results were in median and range.Statistics with Fischer's exact test and logistic regression. P<0.05 is significant. No need for IRB approval.

Results:
Patients were aged 35 months [0-162] at EHPVO diagnosis, 24(35%) had an umbilical catheter (UC) history, 46 (67%) had an episode of variceal bleeding. The patency of intra-hepatic portal vessels was assessed by blocked hepatic phlebography, percutaneous portography and CT scan in 29, 17 and 23 cases. Of these, respectively 23 (79%), 8(47%), and 8 (34%) were deemed favorable for PR. It was significantly higher for phlebography (p=0.01). Out of 69 patients, 42 PR were attempted, 10 (23%) failed during surgery and were converted to portal systemic shunt (PSS). Out of the 32 remaining, 10 had post operative thrombosis, 4 were successfully re-permeabilized, 2 converted to PSS. The median follow-up was 58 months [6-160] (5 patients excluded because of short FU). In intention to treat analysis, the overall success rate for PR was 60% (22/37). Late complications consisted in 4 persistent thromboses. The success rate of PR was significantly impaired by UC: 2/9 vs. 20/28 without (P=0.01) and the unfavorable imaging assessment: 0/5 vs. 22/32 if favorable (P<0.01). In a multivariate analysis, only a previous history of UC was significantly predicting a failure (p=0.02).

Conclusions:
PR for EHPVO is optimal but with a 60% success rate. Preoperative assessment by hepatic phlebography is reliable. An attempt at PR after a history of UC bears a high risk of failure.

NOTES:

45

EFFICACY OF PERMANENT GASTRIC ELECTRICAL STIMULATION FOR THE TREATMENT OF GASTROPARESIS AND REFRACTORY NAUSEA AND VOMITING IN CHILDREN AND ADOLESCENTS

Steven Teich, Hayat M. Mousa, MD, Carlo DiLorenzo, MD.

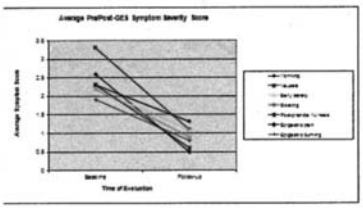
Nationwide Children's Hospital, Columbus, OH, USA.

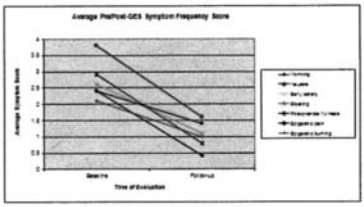
Purpose:
Permanent gastric electrical stimulation (GES) has been performed in adults as a treatment for gastroparesis and refractory nausea and vomiting in patients who have failed medical therapy. We assess the feasibility and clinical outcomes of permanent GES in children.

Methods:
Permanent GES was performed in sixteen children (10 females/ 6 males), median age 15 years (range 4-19 years). All patients had chronic nausea and vomiting refractory to medical therapy for median duration of 2 years (range 3 months - 14 years). All patients met ROME III criteria for functional dyspepsia. At referral, three patients were on total parenteral nutrition exclusively, three on jejunal feeds, and ten on oral feeds. Symptoms, route for nutrition, and satisfaction with procedure were recorded before and after permanent GES. Overall global health and complications after permanent GES were also recorded. Statistical analysis was performed using paired Student's t test.

Results:
At baseline, all patients were symptomatic with nausea, and 50% had delayed gastric emptying (<40% at 2 hours). After permanent GES(n=16), there was significant improvement in severity of vomiting ($p=0.0001$), frequency of vomiting ($p=0.0003$), severity of nausea ($p<0.0001$), and frequency of nausea ($p<0.0001$). Follow-up ranged from 0.5 to 23 months, with 13 of the 16 patients reporting sustained improvement in symptoms. After GES, 13/16 were on oral feeds exclusively, two patients on oral plus G-tube, and one patient on TPN plus oral plus G-tube plus intermittent TPN. Complications of permanent GES included incision separation in one patient and pocket tenderness in two patients.

Conclusions:
1). Permanent GES can be successfully applied to children with functional dyspepsia and gastroparesis who fail medical therapy. 2). Significant improvement in combined symptoms up to 2 year period, 3). No serious adverse effects of permanent GES. 4). Long-term efficacy and dependency of GES therapy in children needs to be established.





NOTES:

46

BRACING IS AN EFFECTIVE NON-OPERATIVE THERAPY IN PATIENTS WITH *PECTUS CARINATUM*: AN INTERIM REPORT OF THE CALGARY PROTOCOL

Richy T. Lee, MD[1], Scott Moorman[2], Marc Schneider, BSc[3], David L. Sigalet, MD, PhD[1].

[1]Alberta Children's Hospital, Calgary, AB, Canada, [2]University of Alberta, Edmonton, AB, Canada, [3]Braceworks, Calgary, AB, Canada.

Purpose:
Pectus carinatum is a common congenital chest wall malformation. The mainstay of treatment has been surgery. Preliminary results suggest that non-operative bracing may be an effective alternative, but the length and intensity of optimal therapy are unknown. We report the interim results of a self-adjustable low profile bracing system (the Calgary Protocol): all pectus carinatum patients evaluated at our chest wall clinic were prospectively asked to join an IRB approved monitoring study.

Methods:
122 patients were evaluated from October 2003 to April 2011, 97 patients were prescribed external bracing, of those 89 were male. In August 2011, patients were evaluated: 32 patients are in correction phase (bracing 23 h/day), 44 patients have completed the corrective phase and are in maintenance (8 h/day) while 21 patients have finished the complete protocol. Correction required 195 ± 41 days and maintenance 328 ± 330 days. All patients received a questionnaire and clinic review.

Results:
The following table represents a summary of the results:

Summary of the interim results of the Calgary Protocol			
	Beginning of bracing	In Maintenance	Completed protocol
Number of patients	97	44	21
Age (years)	14.44 ± 1.93	14.86 ± 1.88	16.34 ± 1.85
Tanner stage	3.61± 0.52	3.81 ± 0.30	3.93 ± 0.16
Protrusion (cm)	2.11 ±0.98	0.51± 0.63*	0.53± 0.70*
Chest appearance (self rating/5)	2.89± 1.07	4.28± 0.80**	4.27±0.87**
Exercise tolerance (self rating/5)	4.40 ± 1.08	4.61 ± 1.00	4.83 ± 0.44
Compliance (parent rated/5)		4.36 ± 1.10	4.32 ±1.42

Data Mean ±SD * Student's t-test, p<0.05 ** Mann-Whitney U-test, p<0.02

Conclusions:
These results show that a self adjusting brace system can give effective correction of pectus carinatum protrusion, with excellent patient satisfaction. Preliminary evidence of durability suggests that continued bracing until skeletal maturity is required to give long term stability to the correction.

NOTES:

47

RISK FACTORS FOR REOPERATION AND MORTALITY IN PEDIATRIC PATIENTS UNDERGOING PERITONEAL DIALYSIS

Jennifer Phan, Steve Stanford, Joshua Zaritsky, MD, Daniel DeUgarte, MD.

UCLA, Los Angeles, CA, USA.

Purpose:
As peritoneal dialysis (PD) is the preferred long-term dialysis modality in the pediatric population, we sought to identify variables that increase the risk of PD catheter reoperation and mortality.

Methods:
An IRB-approved retrospective review of patients undergoing PD catheter insertions at a single center from 1994-2009 was performed. The following variables were evaluated: age (<1 year), omentectomy, concomitant gastrostomy, laparoscopic technique, and comorbidities (prematurity, congenital heart disease, and pulmonary hypoplasia). Multivariable Cox regressions analyses were used to evaluate patient survival as well as reoperation-free survival of PD catheters.

Results:
210 patients with a median age of 10 years underwent PD insertion. Mortality for the cohort was 8% with a median follow up of 72 months. Reoperation for malfunction and infection was required in 47% of patients with a median PD survival of 16 months. Post-operative hernias requiring surgical repair occurred in 14% of patients. Multivariate Cox regressions analyses identified age <1 year, lack of omentectomy, and prematurity as variables significantly associated with complications (see table). In addition, the adjusted hazard ratio of creating a gastrostomy at the time of PD insertion on PD survival free of reoperation for infection was 3.85 (p=0.041; 95% CI 1.05-14.1).

Conclusions:
In this large study of pediatric patients undergoing peritoneal dialysis, higher complications rates were noted in infants (age < 1 year). Omentectomy is associated with a lower risk of reoperation. Placement of a gastrostomy tube at the time of PD insertion was associated with a higher risk of reoperation for infection.

Variable	Adjusted Hazard Ratio (95% Confidence Interval)	p value
Outcome 1: Overall Patient Survival		
Age	7.68 (2.09 - 28.1)	0.002
Omentectomy	0.35 (0.124 - 0.99)	0.048
Outcome 2: PD Survival Free of Reoperation for Malfunction or Infection		
Age < 1 year	1.80 (1.04 - 3.13)	0.035
Omentectomy	0.627 (0.42 - 0.95)	0.027
Outcome 3: PD Survival Free of Hernias Requiring Surgical Repair		
Age < 1 year	4.65 (1.56 - 13.84)	0.006
Prematurity	2.78 (1.04 - 7.39)	0.040

NOTES:

48

A PROSPECTIVE RANDOMIZED TRIAL OF ULTRASOUND VERSUS LANDMARK GUIDED CENTRAL VENOUS ACCESS IN THE PEDIATRIC POPULATION

Matias Bruzoni[1], Bethany J. Slater, MD[1], Karl G. Sylvester, MD[1], Craig T. Albanese, MD, MBA[1], Claudia M. Mueller, MD, PhD[1], Shawn D. St. Peter, MD[2], Susan W. Sharp, PhD[2], Daniel J. Ostlie, MD[2], Sanjeev Dutta, MD[1].

[1]Stanford University, Palo Alto, CA, USA, [2]Children's Mercy Hospital, Kansas City, MO, USA.

Purpose:
The American College of Surgeons advocates for use of ultrasound in central venous catheter placement, however this is not universally embraced by pediatric surgeons. Complication risk correlates positively with number of venous cannulation attempts. We hypothesize that ultrasound-guided central venous cannulation leads to an increase in successful venous cannulation at first attempt, suggesting a potential reduction in complication rates.

Methods:
With IRB approval, a randomized prospective study of 141 children undergoing central venous catheter placement at two tertiary pediatric centers was performed. Patient accrual was based on power analysis. Exclusion criteria included known non-patency of a central vein or coagulopathy. The internal jugular vein was accessed in the ultrasound group. The internal jugular vein or subclavian vein was accessed, based on surgeon preference, in the landmark group. The primary outcome measure was success of venous cannulation on first attempt. Secondary outcome measures included: number of total attempts, access times, number of arterial punctures, and other complications. Continuous variables were compared using 2-tailed Student's t test. Discrete variables were analyzed with Chi square test. Significance was defined as P value <0.05.

Results:
141 patients were enrolled between April 2008 and September 2011. There was no difference when comparing demographic data. Success at first attempt was achieved in 66% of patients in the ultrasound group versus 42% in the landmark group ($p=0.004$). Success within three attempts was achieved in 94% of ultrasound group versus 73% of landmark group ($p=0.001$). The complication rate was 6% in the ultrasound group and 11% in the landmark group.

Conclusion:
Ultrasound reduced the number of cannulation attempts necessary for venous access. This indicates a potential to reduce complications when ultrasound is used by pediatric surgeons, suggesting that it should be considered during central venous access in children.

NOTES:

49

INCISION AND DRAINAGE OF SUBCUTANEOUS ABSCESSES WITHOUT THE USE OF PACKING

Michael J. Leinwand, MD[1], Marc T. Downing, MD[1], Dwight Slater, MD[2], Marci Beck, RN[1], Karen Burton, RN[1], Donna Moyer, RN[1].

[1]Bronson Children's Hospital, Kalamazoo, MI, USA, [2]Michigan State University / Kalamazoo Center for Medical Studies, Kalamazoo, MI, USA.

Purpose:
The current standard of care for the treatment of subcutaneous abscesses is incision and drainage followed by packing with gauze. Removal of the gauze is often quite painful, so we sought to determine whether incision and drainage without packing was equally efficacious.

Methods:
A prospective, randomized trial was performed after institutional review board approval at our community-based teaching hospital. One hundred pediatric patients with subcutaneous abscesses were enrolled between May, 2008 and December, 2010. All underwent incision and drainage followed by 7 days of oral antibiotics and warm soaks. Patients were randomized to either the packing or non-packing groups. The packing was removed 12 to 24 hours after the procedure. Patients were excluded if diabetic or immunosuppressed, if the abscess was perirectal or perianal, secondary to a previous operative procedure, or if it was not subcutaneous. Data collected on the operative day included: age, gender, abscess location and size. Swabs were sent for culture and sensitivities. Follow-up phone calls on postoperative days 7 and 30 were performed. If recurrence was suspected, patients were evaluated in clinic. Data was analyzed using Fisher's exact test, a z-test for two proportions for categorical variables, and a two-sample t-test for continuous variables.

Results:
Eighty-five patients completed the study (43 in the packing group, 42 in the non-packing group). The two groups were not statistically different with respect to initial parameters. There was one recurrent abscess in each of the groups (p=1). Another patient in the non-packing group developed a second abscess 7 cm from the original abscess. There was no difference in Methicillin-resistant Staphylococcus aureus rates (81.4% in the packing group, 83.3% in the non-packing group, p=1).

Conclusions:
Subcutaneous abscesses treated by incision and drainage without packing have the same low rate of recurrence as those treated with packing while omitting a potentially painful component of therapy.

NOTES:

Journal of Pediatric Surgery Lectures

2011
Professor Lewis Spitz
"The History of Paediatric Surgery in the United Kingdom and the National Health Service"

2010
Robert H. Bartlett, MD
"ECMO: Gross, Beethoven, Krummel and Georgeson"

2008
Thomas M. Krummel, MD
"Inventing Our Future: Training the Next Generation of Surgeon Innovators"

2007
Alan W. Flake, MD
"Stem Cell Biology and Pediatric Surgery – Deciphering the Venn Diagram"

2006
Pedro Rosselló, MD
"The Unfinished Business of American Healthcare"

2005
Alberto Peña, MD
"Luck and Serendipity, the History of a Surgical Technique"

2004
R. Scott Jones, MD
"The American College of Surgeons Initiatives for Safety and Quality Improvement"

2003
Patricia K. Donahoe, MD
"Sustained Inquiry and Perseverance in the Clinic and at the Bench"

2002
Michael R. Harrison, MD
"Fetal Surgery: Trials, Tribulations and Territory"

2001
Joseph P. Vacanti, MD
"The History and Current Status of Tissue Engineering"

Robert E. Gross Lectures

2011
Judson G. Randolph, MD
"Notes on the Early Development of Pediatric Surgery in the United States"

2010
John D. Birkmeyer, MD
"Measuring and Improvement the Quality of Pediatric Surgery"

2009
Stanley B. Prusiner, MD
"Designer Prions and a Quest for Therapy"

2008
Michael W.L. Gauderer, MD
"Creativity and the Surgeon"

2007
Francisco G. Cigarroa, MD
"Leading an Academic Health Center in the 21st Century: A Pediatric Surgeon's Perspective"

2006
Diana Bianchi, MD
"Fetomaternal Cell Trafficking: A Story that Begins with Prenatal Diagnosis and May End with Stem Cell Therapy"

2005
W. Hardy Hendren, MD
"Looking Back 50 Years"

2004
Giulio (Dan) D'Angio, MD
"The Role of the Surgeon in the Past, Present and Future of Pediatric Oncology"

2003
Lucien Leape, MD
"Safe Health Care — Are We Up to It?"

2002
Harold Shapiro, PhD
"The Ethical Dimensions of Scientific Progress"

2001
Judah Folkman, MD
"Angiogenesis-Dependent Diseases"

2000
J. Bruce Beckwith, MD
"Pediatric Renal Tumors at the New Millennium: Myths, Misunderstandings, Controversies and Opportunities"

1999
Samuel A . Wells, Jr., MD
(Title not available)

1998
Richard M. Satava, MD
"Medicine in the 21st Century"

1997
Douglas W. Wilmore, MD
"Will Organ Growth Replace Transplantation? Lessons from Patients with Short Bowel Syndrome"

1996
Robert H . Bartlett, MD
"Surgery, Science and Respiratory Failure"

1995
David A . Williams, MD
"The Role of Interleukin-II on the Pathophysiology of the Small Intestine"

1994
W. French Anderson, PhD
"Human Gene Therapy"

1993
Judah Folkman, MD
"Clinical Applications of Angiogenesis Research"

1992
Warren Zapol, MD
"Inhaled Nitric Oxide: A Selective Vaso-Dilator"

1991
Joel Cooper, MD
"History and Current Status of Lung Transplantation"

1990
Richard Simmons, MD
"Role of the Gut Flora in Surgery"

Jay & Margie Grosfeld Lectures

2011
Anthony Atala, MD
"Regenerative Medicine: New Approaches to Healthcare "

2010
Christopher K. Breuer, MD
"The Development and Translation of the Tissue Engineered Vascular Grafts"

2009
Michael T. Longaker, MD, MBA, FACS
"Regenerative Medicine: A Surgeon's Perspective"

2008
Frederick J. Rescorla, MD
"What's New in Pediatric Surgery"

International Guest Lecturers

2011
Professor Takeshi Miyano, MD
"A Brief History of Pediatric Surgery and Healthcare Delivery Systems in Japan"

2010
Jan Alice Marcel Deprest, MD
"Prenatal Management of the Fetus with Isolated CDH"

2009
Marcelo Martinez Ferro, MD
"New Approaches to Pectus and Other MIS in Argentina"

2008
Tadashi Iwanaka, MD
"Technical Innovation, Standardization and Skill Qualification of Pediatric Minimally Invasive Surgery in Japan"

2007
Claire Nihoul-Fékété, MD
"Is Regionalism of Complex Pediatric Malformations Desirable and Feasible? The Example of Disorders of Sexual Development"

2005
Prof. Frans W.J. Hazebroek, MD, PhD
"Is Continuation of Life Support Always the Best Option for the Surgical Neonate?"

2004
David A. Lloyd, MD, FRCS
"Tomorrow's Surgeons: Who Cares for the Patient?"

2003
Claire Nihoul-Fékété, MD
"Modern Surgical Management of Congenital Hyperinsulinemic Hypoglycemia"

2002
Takeshi Miyano, MD
"Biliary Tree: A Gardener's 30-Year Experience"

2001
Pedro Rosselló, MD
"One Nation, with Liberty and Justice…and Healthcare for All"

2000
Leela Kapila, FRCS
"Are These the Children of a Lesser God?"

1999
Bernardo Ochoa, MD
"Pediatric Surgery in Latin America"

1998
Sidney Cywes, MD
"Some of the Little Things We Do — Something Old, Something New"

1997
Justin Kelly, MD
"Bladder Exstrophy — Problems and Solutions"

1996
Prem Puri, MD
"Variant Hirschsprug's Disease"

1995
Sir Lewis Spitz, MD, PhD, FRCS
"Esophageal Atresia — Past, Present and Future"

1994
Sean J. Corkery, MCh, FRCSI, FRCSEng
"In Pursuit of the Testis"

1993
Edward M. Kiely, FRCSI, FRCS
"The Surgical Challenge of Neuroblastoma"

1992
Yann Revillon, MD
"Intestinal Transplantation in France"

1991
Shemuel Nissan, MD
"The History of Surgery and Medicine in the Holy Land from the 19th Century"

1990
Jan C. Molenaar, MD
"Congenital Diaphragmatic Hernia - What Defect"

Tab Page
Remove before Printing

EXHIBITS & SUPPORT

Back of Tab Page
Remove before Printing

APSA THANKS THE FOLLOWING COMPANIES FOR THEIR UNRESTRICTED EDUCATIONAL GRANTS

APSA-IPSO Symposium

Cookies for Kids' Cancer
Covidien
The Kingsbury Firm, LLC

APSA 43rd Annual Meeting

Elsevier
Ethicon Endo-Surgery
Sheikh Zayed Institute for Pediatric Surgical Innovation
at Children's National Medical Center

Exhibitors

American College of Surgeons
American Pediatric Surgical Association Foundation (APSAF)
American Pediatric Surgical Nurses Association (APSNA)
Applied Medical Technology, Inc. (AMT)
Baptist Health System
Bentec Medical
Blank Children's Hospital
CHERUBS — The Association of Congenital Diaphragmatic Hernia Research, Awareness and Support
Community Health Network
CORPAK MedSystems
Elliot Health System (EHS)
ELSEVIER
HCA Kids
Kaiser Permanente, The Permanente Medical Group, Inc. (TPMG, Inc.)
Kimberly-Clark Healthcare – GOLD EXHIBITOR
LocumTenens.com
Lurie Children's Hospital-Thoracoscopic TEF Simulation Trainer
Medical Foundation for Children
Mediflex Surgical Products
Pediatric Search Partners
Presbyterian Healthcare Services (PHS)
Sacred Heart Health System
Saint Francis Health System
Specialty Surgical Products, Inc.
Stryker
Thompson Surgical Instruments
Weatherby Healthcare

EXHIBITORS

American College of Surgeons

633 N Saint Clair Street Chicago, IL 60611-3211
Phone: 312-202-5000
Toll free: 800-621-4111
Fax: 312-202-5001
www.facs.org

The American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP®) is the first nationally validated risk adjusted program to measure and improve the quality of surgical care. The program's database compares 30 day surgical outcomes among participating hospitals. The ACS has developed a pediatric version (ACS NSQIP Pediatric) in collaboration with APSA.

American Pediatric Surgical Association Foundation (APSAF)

The American Pediatric Surgical Association Foundation (APSAF) was founded in 1995 to provide support for young pediatric surgical investigators and promote pediatric surgical research and education. The vital support provided by APSA members and others energizes the productivity of our young pediatric surgeon-scientists and results in important new discoveries that benefit our patients and profession. This would be impossible to accomplish without your generous financial support. Please consider contributing to the APSA Foundation in 2012.

Applied Medical Technology, Inc. (AMT)

8000 Katherine Boulevard
Brecksville, OH 44141
Phone: +1-440-717-4000
Fax: +1-440-717-4200
E-mail: lszpak@appliedmedical.net
Web site: www.amtinnovation.com

AMT product line offers better outcomes! **The AMT Balloon Mini ONE®** is the lowest profile Button on the market. The apple shaped balloon covers more surface area to minimize leaks. The all **NEW AMT G-JET™** Button's unique internal tube geometry addresses clogging issues and provides a more than 60% increase in cross sectional area to provide better flow. **The AMT Bridle™ Nasal Tube Retaining System** optimizes nutrition while reducing the cost of nasal tubes.

APSNA – American Pediatric Surgical Nurses Association

4405 Herrick Lane
Madison, WI 53711
Phone: +1-608-262-2146
Fax: +1-608-261-1876
E-mail: resch@surgery.wisc.edu
Web site: www.apsna.org

Encourage your nurses, nurse practitioners and physician assistants to join the American Pediatric Surgical Nurses Association. APSNA is an organization of more than 550 pediatric surgical nursing colleagues who care for children in peri-operative, inpatient and outpatient settings. APSNA membership spans the spectrum of the care of children, from neonates through adolescents, requiring surgical treatment. As registered nurses, advanced practice nurses, physician assistants and non-nurse affiliates, we are as diverse as the children we serve.

Baptist Health System

8711 Village Drive, Suite 114
San Antonio, TX 78217
Phone: +1-210-297-2243
Fax: +1-210-297-2257
E-mail: kxbuster@baptisthealthsystem.com
Web site: www.baptisthealthsystem.com

North Central Baptist Hospital has served San Antonio for over 20 years. As the only provider of pediatric services in northern San Antonio, North Central is the home of the Baptist Regional Children's Center and offers a broad spectrum of specialized pediatric care including emergency medicine, inpatient and intensive care, surgery, sedation services and outpatient testing and treatment. Pediatric services at North Central and across Baptist Health System are continually expanding to meet the community's needs.

Bentec Medical

1380 East Bedmer Street
Woodland, CA 95776
Phone: +1-530-406-3333
Fax: +1-530-406-3306
E-mail: sales@bentecmed.com
Web site: www.bentecmed.com

Bentec Medical specializes in the manufacturing of medical device products including latex free Silicone Penrose Drains, Sheeting, Tubing, Biliary Catheters, Ultra Clean Glove Cleaner and Surgical Wipes, and Sterile Ventral Wall Defect Silo Bags. The Silo Bag is designed for staged silo closure of congenital ventral wall defects in infants. Our medical device products are successfully used in Oncology, Cardiology, Urology, Pediatrics, ENT and Interventional Radiology. Bentec Medical is ISO 13485:2003, and FDA & CE Registered.

Blank Children's Hospital

1200 Pleasant Street
Des Moines, IA 50309
Phone: +1-515-241-5911
Fax: +1-515-241-6044
E-mail: clarkml@ihs.org
Web site: www.ihs.org

Blank Children's Hospital is the only Level 2 Trauma Center in Central Iowa. Blank is a full tertiary care hospital. Blank has an ACGME Pediatrics Residency Program which is academically affiliated with the University of Iowa Medical School. Teaching pediatric and family practice residents and medical students is an integral part of the practice. Research opportunities are available.

CHERUBS – The Association of Congenital Diaphragmatic Hernia Research, Awareness and Support

6350 Rogers Road #290
Wake Forest, NC 21587
Phone: +1-919-610-0129
Fax: +1-815-425-9155
E-mail: dawn.williamson@cdhsupport.org
Web site: www.cdhsupport.org

CHERUBS is an international charity created to help families affected by Congenital Diaphragmatic Hernia by providing free support services, promoting and funding CDH research and raising awareness of this devastating birth defect through dozens of volunteer-supported projects. Founded in 1995, CHERUBS has helped more than 3,800 families in 38 countries.

Community Health Network

7240 Shadeland Station, Suite 300
Indianapolis, IN 46256
Phone: +1-317-621-2141
Fax: +1-317-355-7590
E-mail: especkman@ecommunity.com
Web site: www.ecommunity.com/physicianrecruitment/

Community Health Network is a leading not-for profit, six hospital system nationally recognized for our successful physician partnerships. Ranked among the nation's most integrated healthcare systems, Community Health Network is Central Indiana's leader in access to innovative and compassionate healthcare services. Community's full continuum of care integrates hundreds of physicians, acute care and specialty hospitals, surgery centers, physician offices, home care services, walk-in care centers and employer health services.

CORPAK MedSystems

1001 Asbury Drive
Buffalo Grove, IL 60089
Phone: +1-800-323-6305
Fax: +1-847-403-3777
E-mail: questions@corpak.com
Web site: www.corpakmedsystems.com

Do you have enterally fed patients who suffer from gastric distention or feeding intolerance due to delayed gastric emptying? The Farrell Valve Gastric Decompression System offers relief from gastric pressure. This closed system connects in-line with the feeding tube and set and becomes a reservoir overflow for gas and gastric contents. The Farrell Valve allows the release of air, prevents exposure to caregivers, prevents loss of calories and increases the patient's comfort. Please come see us in the Exhibit Hall.

Elliot Health System (EHS)

One Elliot Way
1st Floor, Medical Staff Recruitment
Manchester, NH, 03103
Phone: +1-603- 663-4926
E-mail: mcragon1@elliot-hs.org
Web site: www.elliotphysicians.org

Elliot Health System (EHS) is the largest provider of comprehensive healthcare services in Southern New Hampshire. The cornerstone of EHS is Elliot Hospital, a 296-bed acute care facility located in Manchester (New Hampshire's largest city). Established in 1890, Elliot Hospital offers Southern New Hampshire communities caring, compassionate and professional patient service regardless of race, religion, national origin, gender, age, disability, marital status, sexual preference or ability to pay.

ELSEVIER

1600 JFK Blvd.
Suite 1800
Philadelphia, PA 19103
Phone: +1-215-239-3490
Fax: +1-215-239-3494
E-mail: c.isard@elsevier.com
Web site: www.us.elsevierhealth.com

ELSEVIER is a leading publisher of health science publications, advancing medicine by delivering superior reference information and decision support tools to doctors, nurses, health practitioners and students. With an extensive media spectrum – print, online and handheld, we are able to supply the information you need to the most convenient format.

HCA Kids

3 Maryland Farms
Suite 250
Brentwood, TN 37027
Phone: +1-615-372-5196
Fax: +1-866-789-2288
E-mail: tyler.browning@hcahealthcare.com
Web site: www.hcahealthcare.com

HCA Kids is a centralized recruitment office where you can make just one call to find out about opportunities in excellent facilities across the nation. HCA owns and manages 164 hospitals in 20 states. We offer opportunities in big cities and rural areas, near sandy beaches and skyward reaching mountains. Excellent opportunities are available in private practices and two of the top academic medical centers in the world.

Kaiser Permanente, The Permanente Medical Group, Inc. (TPMG, Inc.)

1600 Eureka Road
Roseville, California, 95661
Phone: +1-800-777-4912
Fax: +1-510-625-5487
E-mail: Christine.K.Stough@kp.org
Web site: www.physiciancareers.kp.org

Kaiser Permanente is seeking a Pediatric Surgeon to join an established practice of senior pediatric surgeons at our 174-bed, state of the art, Women and Children's Center in Roseville, CA just outside of Sacramento. The W&CC in Roseville is the regional referral center for pediatric tertiary care and includes over 100 primary care Pediatricians, 8 Pediatric Hospitalists and over 25 Pediatric Subspecialists. Pediatric Surgeons who join TPMG, Inc. enjoy competitive salaries and an unparalleled benefit package.

Kimberly-Clark Healthcare

1400 Holcomb Bridge Rd
Roswell, GA 30076
Phone: +1-770-587-8578
Fax:+1- 920-225-4531
E-mail: abwillner@kcc.com
Web site: www.kcdigestivehealth.com

Kimberly-Clark's priority is to deliver easy and reliable digestive health solutions that help you improve patient outcomes and quality of life. Our trusted clinical solutions are utilized more often than any other around the world including our pioneering MIC-KEY* balloon-retained gastrostomy feeding tube and twin gold-standard for diagnosing H. pylori CLOtest* & PYtest*. The Kimberly-Clark* Advantage* is our drive to provide you everyday essentials and help foster one less worry for you. Visit us at www.kcdigestivehealth.com.

LocumTenens.com

2655 Northwinds Parkway
Alpharetta, GA 30009
Phone: +1-800-562-8663
Fax: +1-770-643-5797
E-mail: customerservice@locumtenens.com
Web site: www.LocumTenens.com

Founded in 1995, LocumTenens.com is a full-service locum tenens staffing agency specializing in emergency medicine, primary care, anesthesiology, psychiatry, radiology, surgery and CRNA jobs with U.S. hospitals, medical groups and community health centers. LocumTenens.com operates the largest internet job board for the industry with direct access to thousands of jobs – in all specialties – posted directly by facilities and agencies nationwide. LocumTenens.com is part of the Jackson Healthcare family of companies. To learn more, visit www.locumtenens.com.

Lurie Children's Hospital – Thoracoscopic TEF Simulation Trainer

2300 Children's Plaza, Box 63
Chicago, IL 60614
Phone: +1-773-880-4340
Fax: +1-773-880-4588
E-mail: kbarsness@luriechidrens.org
Web site: www.luriechildrens.org

We have developed two different simulation models for thoracoscopic esophageal atresia/tracheoesophageal fistula repair. The simulation models are scaled to a 3.2 kg infant, with a full reproduction of the chest and relevant anatomic defects. We are collecting validation data and welcome all practicing pediatric surgeons and pediatric surgical trainees to come to our station to try their hand on the models. Validation data will be collected only with your permission.

Medical Foundation for Children

10305 James Ryan Way
Austin, TX 78730
Phone: +1-512-680-8885
E-mail: mpatton@txmda.com

Despite constant innovation in the medical device field, some children continue to suffer because they are treated with devices that were designed for adults. Even routine medical discoveries rarely are made available to children because the innovators are often inexperienced in the commercialization process. Furthermore, medical device companies actually have a financial disincentive to produce products for the relatively small pediatric device market. The Medical Foundation for Children was established to commercialize medical devices that are designed specifically for babies and children.

Mediflex Surgical Products

250 Gibbs Road
Islandia, NY 11749
Phone: +1-800-764-0474
Fax: +1-631-582-4520
E-mail: sales@mediflex.com
Web site: www.mediflex.com

Four years ago Mediflex introduced the first Table Mounted Retractor System ever developed specifically for the pediatric surgical patient. Its acceptance by the Pediatric Surgical Community has been virtually universal. If you have not yet had a chance to see the Mini-Bookler® System, please stop by Mediflex table during the 2012 APSA Meeting in San Antonio. You'll also see the best selling Surgical Arms in the world and our new US-made Pediatric Laparoscopic Instruments.

Pediatric Search Partners

100 Congress Avenue, Suite 2000
Austin, TX 78701
Phone: +1-877-440-3832
Fax: +1-512-672-7038
E-mail: Glenda@pediatricsearchpartners.com
Web site: www.pediatricsearchpartners.com

Pediatric Search Partners is a boutique search firm focused solely within pediatrics and pediatric subspecialties.

With over 75 years' combined experience, including creating and directing the physician recruitment department for a top academic children's hospital, our leaders have the knowledge, track record of success and expertise to handle your search or assist you in finding the practice you've envisioned.

Our mission is to match the physicians and surgeons who care for children with opportunities they truly care about.

Presbyterian Healthcare Services (PHS)

1101 Central SE
Albuquerque, NM 87125
Phone: +1-866-757-5263
Fax: +1-505-823-8734
E-mail: kkernagh@phs.org
Web site: www.phs.org

Presbyterian Healthcare Services (PHS) is New Mexico's
- only private, not-for-profit healthcare system,
- largest provider of health care,
- largest managed care organization,
- fastest growing physician group,
- largest provider of birthing services.

Through a multi-million-dollar investment in facilities, technology and equipment, The Children's Center at Presbyterian is New Mexico's premier provider of hospital services for children.

PHS operates eight hospitals throughout New Mexico. We seek board certified/board eligible pediatric surgeons for The Children's Center on the Presbyterian Hospital campus.

Enjoy a lifestyle practice with your clinic attached to the hospital, no trauma designation and potential for a four-day workweek if desired.

Sacred Heart Health System

5151 North 9th Ave
Pensacola, FL 32504
Phone: +1-850-416-762
Fax: +1-850-416-1147
E-mail: vbaker@shhpens.org
website: www.sacred-heart.org

Sacred Heart Health System, in Pensacola, Florida, is seeking quality Pediatric Surgeons to join their team. BC in Pediatric Surgery preferred. Sacred Heart Children's Hospital is partnered with Nemours Children's Clinic to provide highly specialized care to children along the Gulf Coast. This is an excellent opportunity for exceptional Pediatric Surgeons to work with an elite medical staff and practice quality medicine in paradise! Position offers many perks including: very competitive salary with bonus, rich benefits package and more. Contact Valarie Baker, vrbaker@shhpens.org or 850-416-7623 for more information.

Saint Francis Health System/The Children's Hospital at Saint Francis

6161 S. Yale Ave.
Tulsa, OK 74136
Phone: 1-918-494-8463
E-mail: lalandwerlin@saintfrancis.com
Web site: www.saintfrancis.com

As part of the Saint Francis Health System, The Children's Hospital at Saint Francis is Eastern Oklahoma's only children's hospital. We provide sophisticated, state of the art medical care to children in a child-friendly environment. Our new facility opened in 2008 and has 20 PICU beds, 58 level IIId NICU private rooms and 84 general pediatric beds with an additional 14 bed day hospital and 10 bed pediatric emergency center.

Specialty Surgical Products, Inc.

1131 N. US Hwy 93
Victor, MT 59875
Phone: +1-406-961-0102
Fax: +1-406-961-0103
E-mail: info@ssp-inc.com
Web site: www.ssp-inc.com

SSP, your trusted source for the Aus Systems rbi2 suction rectal biopsy system, clear cast silicone silo bags for staged Gastroschisis repair and single patient use pediatric anal dilators, is proud to introduce our new silicone silo bag with wire ring! Stop by our booth to see our new product line and view how all of our products can simplify your practice.

Stryker

5900 Optical Court
San Jose, CA 95138
Phone: +1-408-754-2000
E-mail: min.choi@stryker.com
Web site: www.stryker.com/endoscopy

Stryker is one of the world's leading medical technology companies and is dedicated to helping healthcare professionals perform their jobs more efficiently while enhancing patient care. The Company offers a diverse array of innovative medical technologies, including reconstructive, medical and surgical, and neurotechnology and spine products to help people lead more active and more satisfying lives.

Thompson Surgical Instruments

10170 E. Cherry Bend Road
Traverse City, MI 49684
Phone: +1-231-922-5178
Fax: +1-231-922-0174
E-mail: kim.murray@thompsonsurgical.com
Web site: www.thompsonsurgical.com

Weatherby Healthcare

6451 North Federal highway
Suite 800
Ft. Lauderdale, FL 33308
Phone: +1-800-586-5022
Fax: +1-800-463-2817
E-mail: info@weatherbyhealthcare.com
Web site: www.weatherbyhealthcare.com

Weatherby Healthcare, a premier staffing company known since 1995 for its Red Ribbon Service, provides each pediatric surgeon with a personal consultant who locates the best opportunity from hundreds of jobs nationwide. Meeting the needs of pediatric surgeons and patients alike is our top priority.

APSA Past Meetings

41st Annual Meeting
May 16 - 19, 2010
Loews Portofino Bay Hotel at Universal Orlando
Orlando, FL

40th Annual Meeting
May 28 - 30, 2009
El Conquistador Golf Resort & Golden Door Spa
Fajardo, Puerto Rico

39th Annual Meeting
May 27 - 31, 2008
JW Marriott Desert Ridge
Phoenix, Arizona

38th Annual Meeting
May 24 - 27, 2007
JW Marriott Orlando Grande Lakes
Orlando, Florida

37th Annual Meeting
May 21 - 24, 2006
Marriott Beach & Golf Resort
Hilton Head, South Carolina

36th Annual Meeting
May 29 - June 1, 2005
JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona

35th Annual Meeting
May 27 - 30, 2004
Sawgrass Marriott Resort
Ponte Vedra Beach, Florida

34th Annual Meeting
May 25 - 28, 2003
Marriott Harbor Beach Resort & Spa
Ft. Lauderdale, Florida

33rd Annual Meeting
May 19 - 22, 2002
The Arizona Biltmore Resort and Spa
Phoenix, Arizona

32nd Annual Meeting
May 20 - 23, 2001
The Registry Resort
Naples, Florida

31st Annual Meeting
May 25 - 28, 2000
Walt Disney World Swan
Lake Buena Vista, Florida

30th Annual Meeting
May 16 - 19, 1999
Westin Mission Hills
Rancho Mirage, California

29th Annual Meeting
May 10 - 13, 1998
The Hyatt Regency
Hilton Head, South Carolina

28th Annual Meeting
May 18 - 21, 1997
The Registry Resort
Naples, Florida

27th Annual Meeting
May 19 - 22, 1996
The Hyatt Regency
San Diego, California

26th Annual Meeting
May 20 - 23, 1995
The Boca Raton Resort and Club
Boca Raton, Florida

25th Annual Meeting
May 14 - 17, 1994
Loews Ventana Canyon Resort
Tucson, Arizona

24th Annual Meeting
May 15 - 18, 1993
The Hyatt Regency
Hilton Head, South Carolina

23rd Annual Meeting
May 12 - 16, 1992
The Broadmoor
Colorado Springs, Colorado

I have enclosed a special donation for the
American Pediatric Surgical Association Foundation:

❑ $1,000—Gold Level Supporter ❑ $5,000—Robert E. Gross Benefactor ❑ $10,000—William E. Ladd Society

❑ $100 ❑ $200 ❑ $500 ❑ other _____

❑ Check ❑ MasterCard ❑ Visa ❑ American Express ❑ Discover Card

Credit Card#_____ Exp. Date_____

Signature_____

Name as it appears on credit card _____

Street Address _____

City_____State_____Zip Code_____

Telephone # (_____)_____ E-mail _____

This gift is in honor of_____
Please provide address of individual who is to receive notification of gift

APSAF is an IRS approved charitable foundation for whom gifts and donations are tax deductible to the extent permitted by law. You will receive documentation confirming your charitable gift to the Foundation.

EARN VALUABLE PEDIATRIC SURGERY CMES

You already earn pediatric surgery CME credits by attending the APSA annual meeting. Now APSA brings you additional ways to earn those important CMEs.

➢ **APSA/Journal of Pediatric Surgery CME Program**

The *Journal of Pediatric Surgery* includes CME exams designated for potential CME credit. Each exam is based on articles published in *Journal of Pediatric Surgery* and consists of a test with short questions followed by a brief evaluation. The CME activity is free to APSA members.

➢ **Pediatric Surgery Self-Assessment Program (PSSAP) - Part I, II, III, IV and V**

The PSSAP allows APSA members to earn CME credits by taking this online exam to earn 10 credits per Part with each Part featuring 60 questions in a four-section assessment.

Go online to the APSA website at www.eapsa.org and take the assessment, enhance your knowledge and earn 10 CMEs (per Part) today!

Available now; Eligible for CME credits soon

➢ **Using the Evidence in Pediatric Surgery**

Although not yet available for CME credit, this new evidence-based learning program is now available to APSA members.

Using the article-based educational program, Evidence in Pediatric Surgery will help practicing pediatric surgeons incorporate the available medical evidence into the care of their patients. This program is designed to provide a concise, clinical problem-based review of the evidence for a selected topic. The reviews are generated by searching the medical literature for peer-reviewed articles published within the past five years pertaining to human pediatric subjects. Evidence meeting these criteria is then organized into sections based on clinical problems. The first article in the series is now available: GERD.

See the APSA website to start earning more pediatric surgery CMEs!
www.eapsa.org

American Pediatric Surgical Association
Future Meetings

44th Annual Meeting
May 2 - 5, 2013
Marco Island Marriott Beach Resort,
Golf Club & Spa
Marco Island, Florida, USA

45th Annual Meeting
May 29 - June 1, 2014
JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona, USA

46th Annual Meeting
April 30 - May 3, 2015
Harbor Beach Marriott Resort & Spa
Fort Lauderdale, Florida, USA

APSA 43
FORTY-THIRD
ANNUAL MEETING

www.eapsa.org