

**American
Pediatric
Surgical
Association**

FINAL PROGRAM



May 28-30, 2009

**El Conquistador Golf
Resort & Golden Door Spa
Fajardo, Puerto Rico**



www.eapsa.org



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 making certain that it is sustained by economic health.

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Board of Governors 2009-2010



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Robert E. Gross	1970–1971
C. Everett Koop	1971–1972
H. William Clatworthy, Jr.	1972–1973
Orvar Swenson	1973–1974
Harvey E. Beardmore	1974–1975
Thomas M. Holder	1975–1976
Alexander H. Bill	1976–1977
E. Thomas Boles, Jr.	1977–1978
Morton M. Woolley	1978–1979
Robert G. Allen	1979–1980
Thomas V. Santulli	1980–1981
William B. Kiesewetter	1981
W. Hardy Hendren	1981–1983
Lester W. Martin	1983–1984
Judson G. Randolph	1984–1985
Dale G. Johnson	1985–1986
J. Alex Haller, Jr.	1986–1987
Robert J. Izant, Jr.	1987–1988
James A. O'Neill, Jr.	1988–1989
Eric W. Fonkalsrud	1989–1990
Robert M. Filler	1990–1991
Alfred A. deLorimier	1991–1992
Dick G. Ellis	1992–1993
Raymond A. Amoury	1993–1994
Jay L. Grosfeld	1994–1995
Arvin I. Philippart	1995–1996
Keith W. Ashcraft	1996–1997
H. Biemann Othersen, Jr.	1997–1998
Marc I. Rowe	1998–1999
Kathryn D. Anderson	1999–2000
David Tapper	2000–2001
Arnold G. Coran	2001–2002
R. Peter Altman	2002–2003
Brad M. Rodgers	2003–2004
Robert J. Touloukian	2004–2005
M. Judah Folkman	2005–2006
Patricia K. Donahoe	2006–2007
Moritz M. Ziegler	2007–2008
Michael R. Harrison	2008–2009

Secretary

Thomas M. Holder	1970–1973
Dale 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
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Lucian L. Leape.....	1972–1975
Robert G. Allen.....	1975–1978
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J. Alex Haller, Jr.....	1981–1984
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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
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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
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Robert J. Touloukian.....	1987–1990
Arvin I. Philippart.....	1988–1991
Albert W. Dibbins.....	1989–1992
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Eugene S. Wiener.....	1994–1997
Samuel H. Kim.....	1995–1998
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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

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Committee on Ethics

Thomas V. Whalen

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Henri R. Ford

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Committee on Video-Based Education

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Editors: ACS Portal for Pediatric Surgery

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Prem Puri, President

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Mark Wulkan, 2008-2011
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Terry Buchmiller, 2007-2010
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Ben Nwomeh, 2007-2010
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Philip Fryckman, 2007-2010
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Kevin P. Lally, 2007-2010
Joseph Lelli, 2007-2010
Dan Saltzman, 2007-2010
Joel Shilyansky, 2007-2010
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Steven Moulton, 2007-2010
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Peter Dillon, ex officio (NSQIP)

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Philip Glick, 2006-2009
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David Hitch, 2006-2009
Frederick Karrer, 2007-2010
Barry Newman, 2007-2010
David Notrica, 2008-2011
Charles Snyder, 2008-2011
Glaze Vaughn, 2008-2011
Charles Vinocur, 2006-2009
Tom Whalen, 2008-2011
Dennis Lund, ex officio (Board of Governors)
John Crow, ex officio (CPT Representative)

Program Committee

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Elizabeth A. Beierle, 2007-2010
R. Cartland Burns, 2008-2011
Peter F. Ehrlich, 2008-2011
Tom Jaksic, 2008-2011
Heung Bae Kim, 2008-2011
Kerilyn Nobuhara, 2006-2009
Stephen Shochat, 2003-2009
David Sigalet, 2006-2009
Karl Sylvester, 2008-2011
Dennis Vane, 2007-2010
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Henry E. Rice, ex officio (Education Committee)
George Gittes, ex officio (Publications Committee)

Publications Committee

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george.gittes@chp.edu
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Anne Fischer, 2008-2011
Nilda Garcia, 2008-2011
Allan Goldstein, 2006-2009
David Hackham, 2006-2009
Ai-Xuan Holterman, 2006-2009
Peter Kim, 2006-2009
David Rodeberg, 2006-2009
Stephen Shew, 2008-2011
Michael A. Skinner, 2007-2010
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John R. Gosche, ex officio (Program Committee)

**Pediatric Surgical Education and
Self Assessment Program Committee**

John Waldhausen, Chair
john.waldhausen@seattlechildrens.org
Kenneth S. Azarow
Kurt F. Heiss
David M. Powell
Henry E. Rice
Charles Snyder
Robert C. Shamberger, ex officio (Board of
Governors)
Tom Tracy, ex officio (American Board of
Surgery)

Surgical Quality and Safety Committee

Peter Dillon, Chair, 2007-2010
pdillon1@psu.edu
Kurt Newman, Vice Chair, 2007-2010
knewman@cnmc.org
Geoffrey Blair, 2007-2010
Michael Ishitani, 2007-2010
Donald C. Liu, 2007-2010
R. Lawrence Moss, 2007-2010
Frederick C. Ryckman, 2007-2010
Oliver Soldes, 2007-2010
James Stein, 2008-2011
Charles Vinocur, 2008-2011
Moritz Ziegler, ex officio (Board of
Governors)

Task Force on Family Issues

Diana L. Farmer, Chair, 2007-2009
diana.farmer@ucsfmedctr.org
James Gilbert, Vice Chair, 2007-2009
james.gilbertmd@hhsys.org
Colin Bethel, 2007-2010
Andrea Hayes-Jordan, 2007-2010
Aviva Katz, 2007-2010
Baird Mallory, 2006-2009
Jacqueline Saito, 2007-2010
Cathy Shin, 2007-2010
Sandra Tomita, 2007-2010
Daniel von Allmen, 2008-2011
Danielle Walsh, 2007-2010
John Wesley, 2008-2011
Michael R. Harrison, ex officio (Board of
Governors)

Trauma Committee

Randall S. Burd, Chair, 2008-2011
rburd@cnmc.org
Michael L. Nance, Vice Chair, 2008-2011
nance@email.chop.edu
William T. Adamson, 2008-2011
Kathryn Bass, 2008-2011
Charles S. Cox, 2007-2010
Richard Falcone, 2008-2011
Michael M. Fuenfer, 2008-2011
Barbara A. Gaines, 2007-2010
David Gourlay, 2008-2011
John Lawrence, 2006-2009
Robert Todd Maxson, 2008-2011
Steven Morrow, 2007-2010
David Notrica, 2008-2011
Nelson Rosen, 2006-2009
Daniel Ryan, 2006-2009
Mary E. Fallat, ex officio (Board of
Governors)

Workforce Committee

Julie Long, Chair, 2008-2011
julielong@earthlink.net
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jgeiger@med.umich.edu
Stanton Adkins, 2008-2011
Geoff Blair, 2008-2011
Phil Guzzeta, 2008-2011
Jessica Kandel, 2006-2009
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William Middleworth, 2008-2011
Allen Milewicz, 2007-2010
Don Nakayama, 2008-2011
Michael Ratner, 2007-2010
David Schmeling, 2007-2010
Jay Schnitzer, 2006-2009
Steven Stylianos, 2008-2011
Moritz Ziegler, ex officio (Board of
Governors)

American Pediatric Surgical Association Foundation

The American Pediatric Surgical Association Foundation (APSAF) was initiated to encourage the enrichment of members of APSA by providing support for projects that encompass the humanities, medical ethics, education, clinical epidemiology, biostatistics, health care delivery, computer sciences, as well as clinical or laboratory research as they relate to the surgical sciences or to the delivery of pediatric surgical care. Traveling fellowships and special scholarships may be considered.

Further information about the APSAF grant and applications for support of projects as well as information regarding contributions to the foundation may be obtained from the Foundation office: jgrosfel@iupui.edu. APSAF is an IRS approved charitable organization.

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San Francisco, CA
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Baldwin, Charles E.	Folkman, M. Judah	Ladd, Alan
Barlow, Barbara	Fonkalsrud, Eric W.	Lam, Vinh T.
Besner, Gail E.	Ford, Edward G.	Langer, Jacob C.
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Bower, Richard J.	Ginsburg, Howard B.	Lewis, J. Eugene
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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.

New Members 2009

Regular Members

Abramson, Lisa
Aspelund, Gudrun
Bickler, Stephen
Bourque, Michael
Campbell, Brendan
Collins, Joy
Dasgupta, Roshni
Downard, Cynthia
Foley, David
Frey, Ala
Fuchs, Julie
Goldin, Adam
Gourlay, David
Hackam, David
Hess, Donavan
Hirsch, Sarah
Huang, Eunice
Johnson, Sidney
Kim, Eugene
Kimmel, Stephen
Krishnaswami, Sanjay
Kuhn, Marcia
Lal, Dave
Lange, Patricia
Lewis, Fawn
Lim, Foong-Yen
Morowitz, Michael
Muratore, Christopher
Panayides, Kyriacos
Partrick, David
Patel, Haroon
Patel, Jateen
Pieretti, Rafael
Potoka, Doug
Purcell Jackson, Gretchen
Robertson, Daviel
Rollins, Michael
St. Peter, Shawn
Sullivan, Kerry
Wiesenaue, Chad

Candidate Members

Chandler, Nicole
Chong, Albert
Densmore, John
Egan, John
Emran, Mohammad
Escobar, Mauricio
Fitzpatrick, Colleen
Ignacio, Romeo
Leys, Charles
Minati, Douglas
Prince, Jose
Puapong, Devin
Rangel, Shawn
Rocourt, Dorothy
Rothenbach, Thomas
Safford, Shawn

Associate Members

Camps, Juan
Hulka, Frieda
Lugo-Vicente, Humberto

Articles of Incorporation of the American Pediatric Surgical Association

- 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.

Articles of Incorporation of the American Pediatric Surgical Association (continued)

- 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.

In Memoriam

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Schneider, Keith M.	1982
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Lozoya-Solis, Jesus	1984
Soave, Franco	1984
Rosenkrantz, Jens G.	1985
Cresson, Samuel L.	1986
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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
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Schisgall, Richard M.	1991
David, Ronald	1992
Kaufman, Bruce	1992
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Mcparland, Felix A.	1994
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Lilly, John R.	1995
Riker, William L.	1995
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Damis, Richard K.	1996
Goldstein, I. Richard	1996
Longino, Luther A.	1996
Welch, Kenneth J.	1996
Baffes, Thomas G.	1997
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Salzberg, Arnold M.	1997
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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

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Linkner, Laurance M.	2000
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Clatworthy, William	2001
Allen, James E.	2001
Lizarralde, A. Eduardo.....	2001
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Carcassone, Michel	2002
Cohn, Bertram D.	2002
Colodny, Arnold H.	2002
Eraklis, Angelo J.	2002
Smith, Willard D.	2002
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Tapper, David	2002
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Abrams, Martin W.	2003
Harberg, Franklin J. (Jim)	2003
Lynch, III, Frank P.	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, III, Arlvin I.	2004
McAlpin, Columbus D.	2004
Lloyd, James R.	2004
Moore, Thomas C.	2004
Rathouser, Frank	2005
Fitzpatrick, John	2005
Able, Luke W.	2006
Gibbs, Andrew	2006
Jewett, Theodore C.	2006
Rothmann, Bruce F.	2006
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Kliman, Murray
Klippel, Charles
Krasna, Irwin
Lafer, Dennis
Lewis, J. Eugene
Liebert, Peter
Lynn, Hugh
Marquez, Enrique
Martin, Lester
Mellish, R. W. Paul
Mestel, Ascher
Miller, Richard
Murphy, David
Othersen, H. Biemann
Priebe, Cedric
Putnam, Thomas
Randolph, Judson
Rittelli, Gianfranco
Sauvage, Lester
Schnauffer, Louise
Schullinger, John
Schultz, Lloyd
Schuster, Samuel
Shafer, Alan
Shandling, Barry
Shaw, Anthony
Shim, Walton
Somers, Laurence
Spencer, Bernard
Spencer, Rowena
Stahl, Nicholas
Steichen, Felicien
Stone, H. Harlan
Sukarochana, Kamthorn
Swenson, Orvar
Ternberg, Jessie
Touloukian, Robert
Trump, David
Tyson, Kenneth
Verhagen, Arie
Von Berg, Vollrad
Votteler, Theodore
Webb, H. Warner
White, John
Wilkinson, Albert
Woolley, Morton
Wrenn, Earle

Schedule at a Glance

Wednesday, May 27, 2009

7:00 a.m. – 5:00 p.m.	APSA/Kiwanis Registration	Atlantic Foyer
7:30 a.m. – 8:00 a.m.	Kiwanis Continental Breakfast	Grand Caribbean Terrace
8:00 a.m. – 5:00 p.m.	Kiwanis Pediatric Trauma Symposium	Grand Caribbean 6-8
10:00 a.m. – 3:00 p.m.	Board of Governors Meeting	Boardroom 1
11:15 a.m. – 12:00 p.m.	APSNA Bariatrics Session	Grand Atlantic 1
12:30 p.m. – 2:00 p.m.	Kiwanis Luncheon Program	Grand Caribbean 4
1:00 p.m. – 9:00 p.m.	Committee Meetings	Palmas B, Las Croabas A, Las Croabas B, Siete Mares A, Siete Mares B
2:00 p.m. – 6:00 p.m.	Training Program Directors Meeting	Grand Caribbean 1-3
7:30 p.m. – 10:00 p.m.	Board of Governors Dinner	The Strip House

Thursday, May 28, 2009

6:00 a.m. – 7:30 a.m.	Committee Meetings	Palmas B, Las Croabas A, Las Croabas B, Siete Mares A, Siete Mares B, Ceiba A, Cieba B, Grand Caribbean 7, Grand Caribbean 8
7:00 a.m. – 5:30 p.m.	Registration	Atlantic Foyer
7:00 a.m. – 7:30 a.m.	Continental Breakfast	Atlantic Foyer
7:30 a.m. – 7:45 a.m.	Welcome-Michael R. Harrison, MD	Grand Atlantic 2 & 3
7:45 a.m. – 11:00 a.m.	Education Session I: Pediatric Trauma/Critical Care	Grand Atlantic 2 & 3
11:00 a.m. – 11:15 a.m.	Refreshment Break	Atlantic Foyer
11:15 a.m. – 11:30 a.m.	Introduction of New Members	Grand Atlantic 2 & 3
11:30 a.m. – 12:30 p.m.	Presidential Address-Michael R. Harrison, MD	Grand Atlantic 2 & 3
12:30 p.m. – 12:45 p.m.	Refreshment Break and Box Lunch Pick Up	Atlantic Foyer
12:45 p.m. – 1:45 p.m.	Video Session with Lunch	Grand Atlantic 2 & 3
1:45 p.m. – 2:00 p.m.	Refreshment Break	Atlantic Foyer
2:00 p.m. – 4:00 p.m.	Concurrent Sessions: Education Session II: Interesting Neonatal Cases	Grand Caribbean 5
	Education Session III: Controversies in Contracting	Grand Atlantic 2 & 3
4:00 p.m. – 4:15 p.m.	Refreshment Break	Atlantic Foyer
4:15 p.m. – 5:45 p.m.	Concurrent Sessions: Poster Session I: Basic Science & Oncology Poster Session II: Clinical & Fetal Surgery	Icaco Vieques
6:30 p.m. – 8:30 p.m.	Welcome Reception	Main Pool

Friday, May 29, 2009

6:00 a.m. – 7:30 a.m.	Annual Fun Run	Atlantic Terrace
6:30 a.m. – 7:30 a.m.	Committee Meetings	Palmas B, Las Croabas A, Las Croabas B, Siete Mares A, Siete Mares B, Ceiba A, Cieba B, Grand Caribbean 7, Grand Caribbean 8
6:30 a.m. – 10:00 a.m.	Poster Set Up	Icaco & Vieques

Schedule at a Glance (continued)

Friday, May 29, 2009 (continued)

6:30 a.m. – 2:00 p.m.	Registration	Atlantic Foyer
6:45 a.m. – 7:30 a.m.	Continental Breakfast	Atlantic Foyer
6:45 a.m. – 1:15 p.m.	Exhibits Open	Atlantic Foyer
7:30 a.m. – 9:00 a.m.	<i>Scientific Session I: Common Problems</i>	Grand Atlantic 2 & 3
9:00 a.m. – 10:00 a.m.	Robert E. Gross Lecture: Stanley B. Prusiner, MD	Grand Atlantic 2 & 3
10:00 a.m. – 10:30 a.m.	Refreshment Break	Atlantic Foyer
10:00 a.m. – 2:00 p.m.	Poster Viewing	Icaco & Vieques
10:30 a.m. – Noon	<i>Scientific Session II: Congenital Anomalies</i>	Grand Atlantic 2 & 3
12:00 p.m. – 1:00 p.m.	Jay & Margie Grosfeld Lecture: Michael T. Longaker, MD	Grand Atlantic 2 & 3
1:00 p.m. – 1:30 p.m.	Refreshment Break and Box Lunch Pick Up	Atlantic Foyer
1:30 p.m. – 2:30 p.m.	<i>Luncheon Presentation</i> Surgical Simulators	Grand Atlantic 2 & 3
2:30 p.m. – 4:00 p.m.	Benjy Brooks Society Meeting	Flamboyant
3:00 p.m.	Optional Events: Golf, Tennis, Kayak/Snorkel Tour, Leisure time	
5:00 p.m. – 6:30 p.m.	<i>Journal of Pediatric Surgery</i> Reception	Las Croabas
6:30 p.m. – 8:00 p.m.	New Member Reception	Magnolia Ballroom

Saturday, May 30, 2009

6:30 a.m. – 8:00 a.m.	Member Business Meeting and Breakfast	Grand Atlantic 2 & 3
6:30 a.m. – 4:00 p.m.	Registration	Atlantic Foyer
7:00 a.m. – 8:00 a.m.	Continental Breakfast for Non-Members	Atlantic Foyer
7:00 a.m. – 11:00 a.m.	Exhibits Open	Atlantic Foyer
7:00 a.m. – 2:15 p.m.	Poster Viewing	Icaco & Vieques
8:00 a.m. – 9:30 a.m.	<i>Scientific Session III: Trauma, Transplant, Oncology & Critical Care</i>	Grand Atlantic 2 & 3
9:30 a.m. – 10:30 a.m.	<i>Journal of Pediatric Surgery</i> Lecture: Haile T. Debas, MD	Grand Atlantic 2 & 3
10:30 a.m. – 11:00 a.m.	Refreshment Break	Atlantic Foyer
11:00 a.m. – 12:15 p.m.	<i>Scientific Session IV: Practice Issues, Surgical Education & Outcomes</i>	Grand Atlantic 2 & 3
12:15 p.m. – 12:30 p.m.	Refreshment Break and Box Lunch Pick Up	Atlantic Foyer
12:30 p.m. – 1:15 p.m.	APSA Foundation Scholar: Douglas Miniati, MD	Grand Atlantic 2 & 3
1:15 p.m. – 2:15 p.m.	International Guest Lecture: Marcelo Martinez Ferro, MD	Grand Atlantic 2 & 3
2:15 p.m. – 2:30 p.m.	Refreshment Break	Atlantic Foyer
2:15 p.m. – 2:45 p.m.	Poster Dismantling	Icaco & Vieques
2:30 p.m. – 4:30 p.m.	<i>Session V: Surgical Innovation & Basic Science</i>	Grand Atlantic 2 & 3
6:45 p.m. – 7:30 p.m.	President's Reception	Atlantic Foyer
7:30 p.m. – 10:00 p.m.	President's Banquet	Grand Atlantic Ballroom

Educational Objectives

The APSA Annual Meeting is designed to provide comprehensive continuing education in the field of pediatric surgery. It is APSA's intent to bring together the world's leading authorities to present and discuss the most recent clinical and research efforts. The meeting will begin with the Education Day program on Thursday, May 28, with "Management Controversies and Future Directions in Pediatric Trauma/Critical Care." Afternoon sessions include "Interesting Neonatal Cases" and "Controversies in Contracting." Meeting attendees will view and discuss video and selected poster presentations as well. The topics at these sessions have been selected by the Program and Education Committees and are based on member requests from surveys and journal articles about what is relevant to their practices. The scientific sessions consist of basic research and practical clinical presentations. The poster sessions are intended to allow young investigators an opportunity to share preliminary research. This 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.

The Kiwanis Pediatric Trauma Institute at the Floating Hospital for Children at Tufts Medical Center presents the Kiwanis Pediatric Trauma Symposium in conjunction with the American Pediatric Surgical Association's 2009 Annual Meeting. This symposium is an opportunity for medical professionals to meet and discuss a variety of topics related to the treatment of pediatric trauma patients. The goal of the symposium is to stimulate dialog and intellectual synergy among leading experts in pediatric and adult fields relevant to the care of the injured child, focusing on resuscitation. CMEs will be offered.

Accreditation Statement

This activity has been approved for AMA PRA Category 1 Credit™. APSA is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

Credit Designation

APSA designates this educational activity for a maximum of 16.5 AMA PRA Category 1 Credits™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Policy on Faculty Disclosure

It is the policy of the ACCME and APSA that the 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. 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 of these faculty members have agreed not to mention products or services provided by the industry partner during their presentations. All other faculty indicated that they have no financial relationships to disclose.

- Chris Breuer, MD – Pall Corporation, Cytograft, Gunze Ltd. (grant/research support)
Leerink – Swann (consultant)
- Charles S. Cox, Jr, MD – CBR Inc, KCI, Athersys, Inc. (grant/research support)
Athersys, Inc. (consultant)
CBR, Inc. (speakers bureau)

Commercial Support

APSA would like to thank the *Journal of Pediatric Surgery* for its educational grant for the *Journal of Pediatric Surgery* Lecture and its educational grant for the transcription of the annual meeting technical sessions. APSA also thanks the current supporters and exhibitors for their unrestricted educational grants.

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2009 Exhibitors

American College of Surgeons
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Applied Medical Technology, Inc.
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Biomet Microfixation
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Diaphragmatic Hernia Research,
Awareness, and Support
The Children's Medical Center

Ethicon Endo-Surgery
Geisinger Health System
Greenbook Financial Services
IMDS
Karl Storz Endoscopy America, Inc.
Kimberly-Clark Health Care
LocumTenens.com
Mediflex Surgical Products
Ossur Americas
Providence Health & Services
Specialty Surgical Products, Inc.

Exhibit Hours

Friday, May 29 6:45 a.m. – 1:15 p.m.

Saturday, May 30 7:00 a.m. – 11:00 a.m.

APSA Planning Committees

APSA thanks the following people for their contribution of time and talents to the APSA annual meeting program:

Program Committee

John R. Gosche, MD, PhD, Chair
Daniel von Allmen, MD, Vice Chair
Elizabeth A. Beierle, MD
R. Cartland Burns, MD
Peter F. Ehrlich, MD
Tom Jaksic, MD
Heung Bae Kim, MD

Kerilyn K. Nobuhara, MD
Stephen J. Shochat, MD
David L. Sigalet, MD, PhD, FRSC(C), FACS
Dennis W. Vane, MD
Michael R. Harrison, MD, ex officio
Henry E. Rice, MD, ex officio
George K. Gittes, MD, ex officio

APSA Planning Committees (continued)

APSA Education Committee

Henry E. Rice, MD, Vice Chair
David M. Powell, MD, Vice Chair
William T. Adamson, MD
Kenneth S. Azarow, MD
Sheldon J. Bond, MD
Walter J. Chwals, MD
Robert A. Cusick, MD
Sanjeev Dutta, MD
Scott A. Engum, MD

Kurt F. Heiss, MD
Joseph A. Iocono, MD
Gene D. McGahren, MD, PhD
Andreas H. Meier, MD
Mary C. Santos, MD
Erik D. Skarsgard, MD
Shawn D. St. Peter, MD
Robert C. Shamberger, MD, ex officio
John R. Gosche, MD, PhD, ex officio

New Technology Committee

Michael K. Chen, MD, Chair
Timothy D. Kane, MD, Vice Chair
Chris K. Breuer, MD
Mark F. Brown, MD
Casey M. Calkins, MD
Andre V. Hebra, MD

Tamir H. Keshen, MD
Evan R. Kokoska, MD
David Lawlor, MD
Richard H. Pearl, MD
Henri R. Ford, MD, ex officio
Mac Harmon, MD, ex officio

General Information

Registration

All authors presenting a paper at the APSA 40th Annual Meeting are required to pay a registration fee.

The onsite registration fees for the annual meeting are:

Kiwanis Pediatric Trauma Symposium	\$300 USD
APSA 40th Annual Meeting	
APSA Member	\$640 USD
Physician Non-Member	\$740 USD
Student/Resident/Fellow*	\$365 USD
Nurse/Allied**	\$365 USD
Companion	\$340 USD

* Students, residents and fellows must have a letter from their chief of service to qualify for the reduced registration fee.

** Registration for the APSA 40th Annual Meeting only; APSNA registration is by separate subscription.

APSA Registration Desk

Registration will be located in the Atlantic Foyer during the following times:

Wednesday, May 27	7:00 a.m. – 5:00 p.m.
Thursday, May 28	7:00 a.m. – 5:30 p.m.
Friday, May 29	6:30 a.m. – 2:00 p.m.
Saturday, May 30	6:30 a.m. – 4:00 p.m.

Scientific Sessions

All educational sessions will be held in Grand Atlantic 2 & 3. Educational Session II on Thursday will be held in Grand Caribbean 5. The daily dress code is business or business casual attire.

General Information (continued)

Poster Set Up and Viewing

Scientific posters will be located in Icaico and Vieques and available for viewing during the following hours:

Thursday, May 28	4:15 p.m. – 5:45 p.m. Oral presentations only (no posters)
Friday, May 29	6:30 a.m. – 10:00 a.m. Poster Set Up 10:00 a.m. – 2:00 p.m. Poster Viewing
Saturday, May 30	7:00 a.m. – 2:15 p.m. Poster Viewing 2:15 p.m. – 2:45 p.m. Poster Dismantle

Authors present on Thursday evening and are requested to be in attendance during continental breakfasts and breaks to answer audience questions.

Speaker Ready Room

The Speaker Ready Room will be available daily in Culebra. Computers will be provided for speakers to review their presentations. The room will be open during the following times:

Wednesday, May 27	4:00 p.m. – 6:00 p.m.
Thursday, May 28	7:00 a.m. – 5:00 p.m.
Friday, May 29	6:30 a.m. – 2:00 p.m.
Saturday, May 30	6:30 a.m. – 2:30 p.m.

Presentation Check-In

Speakers must use Microsoft PowerPoint® slides during their presentations (Microsoft Office XP or earlier). Refer to the Guide for Speakers distributed prior to the meeting and available on the APSA Web site (www.eapsa.org) for information about preparing the presentation. Your presentation materials (ZIP disk, flash disk or CD-Rom) must be labeled and submitted to the A/V technician in the Speaker Ready Room **by 1 p.m. the day before your presentation.**

Exhibits

Commercial exhibits will be located in the Atlantic Foyer and will be open during the following hours:

Friday, May 29	6:45 a.m. – 1:15 p.m.
Saturday, May 30	7:00 a.m. – 11:00 a.m.

Continental breakfast and scheduled coffee breaks will be served in the exhibit area on Friday and Saturday. For a list of exhibitors and booth assignments, see pages 71-74.

APSA Business Meeting

The APSA Business Meeting will be held from 6:30 – 8 a.m. on Saturday, May 30, in Grand Atlantic 2 & 3. This is a breakfast meeting and is for APSA members only.

Welcome Reception

A Welcome Reception for all registrants will take place at the Main Pool from 6:30 – 8:30 p.m. on Thursday, May 28. Tickets for this reception will be included in your registration packet and will be required for admission to the reception. All guests 12 years and older will need a ticket to be admitted to the Welcome Reception. Casual attire is appropriate.

General Information (continued)

President's Banquet

The President's Banquet will be held in the Grand Atlantic Ballroom on Saturday, May 30. The reception will begin at 6:30 p.m. in the Atlantic Foyer and dinner will begin at 7:30 p.m. After dinner, you are invited to stay for dancing. Tickets for the reception and banquet are included in your registration packet and will be required for admission. All guests 12 years and older will need a ticket to be admitted to the banquet. Business or cocktail attire is requested.

Child Care Services

Babysitting services are available at any time by contacting the El Conquistador Golf Resort & Golden Door Spa's Concierge at +1-787-863-6040. Request for babysitting service must be made 48 hours in advance. El Conquistador Golf Resort & Golden Door Spa offers Camp Coqui: a full-service, on-property children's recreation program featuring fun-filled days of games and activities. Camp Coqui is for young guests ages 4 through 12. The cost of full day camp is \$75 with lunch; half day is \$45. Rate does not include 7 percent tax and is subject to change. Registration takes place every morning from 9 a.m. to 9:30 a.m. at Camp Coqui located at the Main North Pool. For additional information, call Camp Coqui at +1-787-863-1000, ext. 7616, or Guest Activities, ext. 7106.

Companion Hospitality Suite

The companion hospitality suite located in the Club Lounge will be open Thursday, Friday and Saturday from 8 – 10 a.m. Continental breakfast will be served each morning for registered accompanying guests. Badges are requested for entry to the hospitality suite.

Benjy Brooks Meeting and Luncheon

Friday, May 29, 2:30 p.m. – 4:00 p.m., Flamboyant

Join us for a meeting of the Benjy Brooks Society while enjoying desserts and coffee. Discuss issues that women are currently facing in the pediatric surgery arena and talk about the society's future. Plan to attend this informal session. Dessert and an agenda will be provided to those who register. The participation fee is \$38. After the session you can take advantage of the hotel's extensive spa facilities at the Golden Door Spa. Arrangements should be made in advance by calling +1-800-468-5228 or +1-787-863-1000 or visiting their Web site at www.elconresort.com for additional information.

Optional Activities

Golf Tournament

Friday, May 29, 3:00 p.m., Arthur Hills Golf Course

The 2009 APSA Golf Tournament will be held at The Arthur Hills Golf Course at the El Conquistador Golf Resort & Golden Door Spa. The Arthur Hills Golf Club offers a 72-par course that is challenging and player friendly.

The golf tournament will be a shotgun start at The Arthur Hills Golf Course.

Proper attire for men includes a shirt with collar, together with Bermuda-length shorts or slacks. Proper attire for women consists of a blouse and either a skirt, slacks or mid-thigh-length shorts. No denim-colored clothing of any kind may be worn. Spike shoes are not allowed.

The tournament fee is \$164 USD per golfer and includes cart, greens fees, scramble tournament, 2 bottles of water and awards for the top players.

General Information (continued)

Tennis Tournament

Friday, May 29, 3:00 p.m., Tennis Courts

The 2009 APSA Tennis Tournament will be held at the El Conquistador Golf Resort & Golden Door Spa, which features a tennis center and a full-service pro shop.

- The tournament will be round-robin.
- Light refreshment and awards for the top players will be included.
- Courts are hard courts.
- Collared shirts are required.
- Tournament fee is \$70 USD per person and includes snacks, a tennis pro to officiate the event and awards for the top players.

5K Fun Run

Friday, May 29, 6:00 a.m., Atlantic Terrace

This is an event you won't want to miss!

Sign-in will begin at 5:15 a.m. with an organized warm up and stretch at 5:40 a.m.

The run will be on pavement (not sand or gravel), so bring appropriate running shoes.

The participation fee is \$70 USD and includes a Fun Run T-shirt, water stations along the route, directional arrows placed along the course with a staff member on a bicycle leading runners through the course, a light breakfast after the run, and awards in a number of categories.

Hidden Beach Kayak/Snorkel Tour

Friday, May 29, 3:00 p.m. – 7:00 p.m., Departs from Main Entrance

Located on the northeast corner of Puerto Rico, Las Cabezas Bay is one of the island's most pristine beaches. You will have the opportunity to kayak in tropical blue waters as you paddle to a favorite hidden beach. Once on the beach you have the opportunity to snorkel on a protected reef with the master diver or walk the beach with an eco-guide. Beach chairs with canopies will be available.

- Duration is 4 hours, 3 p.m. to 7 p.m.
- The tour will leave the El Conquistador Golf Resort from the main entrance promptly at 3 p.m.
- Tour cost is \$100 USD per person and includes the kayak tour, snorkel or walk on the beach and ice-cold refreshments.
- Minimum of 18 people and maximum of 60 people.

Messages

A message board will be maintained in the registration area during registration hours.

Check the board frequently, as there will be NO PAGING during the meeting. To contact the message center, dial +1-787-863-1000 and request the APSA Registration Desk.

Guidelines for Authors and Discussants

1. Authors presenting papers are reminded that the presentations shall be limited to time previously indicated.
 2. Your presentation materials (ZIP disk, flash disk or CD-Rom) must be labeled and submitted to the A/V technician in the Speaker Ready Room, Culebra, by 1 p.m. the day before they are to be presented.
 3. Posters have been sorted into two sessions, scheduled on Thursday, May 28, between 4:15 and 5:45 p.m. Oral presentations for each poster will be presented at this time, but posters will not be on display until the following morning. Refreshments will be available during the Poster Sessions on Thursday.
- Scientific posters will be hung on Friday morning (following the oral poster presentations) between 6:30 and 10 a.m. and will be available for viewing after that. Authors are requested to be in attendance during the continental breakfasts and general session breaks each day to discuss their presentations.
4. Discussants from the floor should state their name and affiliation prior to their remarks. The discussions will be audio-recorded for transcription and printing in the *Journal of Pediatric Surgery*.
 5. Typed discussions should be limited to a maximum of 200 words. Typed discussions that exceed 200 words will be edited before they are submitted to the *Journal of Pediatric Surgery* for publication.
 6. Discussants will have the opportunity to edit a transcript of their remarks following the meeting. The Publications Committee reserves the right to edit the typed discussion before it is submitted to the *Journal of Pediatric Surgery*.

Future Meetings

41st Annual Meeting

May 16–19, 2010
Loews Portofino Bay Hotel
Orlando, Florida

42nd Annual Meeting

May 22–25, 2011
JW Marriott Desert
Springs Resort & Spa
Palm Desert, California

43rd Annual Meeting

May 20–23, 2012
JW Marriott San Antonio
Hill Country Resort & Spa
San Antonio, Texas

Past APSA Annual Meeting Dates and Locations

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

Invited Speakers

Past Annual Meeting Robert E. Gross Lectures

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) DiAngio, MD
"The Role of the Surgeon in the Past, Present and Future of Pediatric Oncology"

2003

Lucien Leape, MD
"Safe Health Care ó 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"

Invited Speakers (continued)

Past Annual Meeting International Guest Lectures

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

Prof. Sidney Cywes
"Some of the Little Things We Do – Something Old, Something New"

1997

Justin Kelly
"Bladder Exstrophy – Problems and Solutions"

1996

Prem Puri
"Variant Hirschsprung'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?"

Invited Speakers (continued)

Past Annual Meeting *Journal of Pediatric Surgery* Lectures

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 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"

Past Annual Meeting Jay & Margie Grosfeld Lectures

2008

Frederick J. Rescorla, MD
"What's New in Pediatric Surgery"

2009 Invited Speakers



**Robert E. Gross Lecture:
Stanley B. Prusiner, MD
"Designer Prions and a Quest for Therapy"**

Stanley B. Prusiner, MD, is Director of the Institute for Neurodegenerative Diseases and Professor of Neurology and Biochemistry at the University of California, San Francisco where he has worked since 1972. He received his undergraduate and medical training at the University of Pennsylvania and his postgraduate clinical training at UCSF. From 1969-72, he served in the U.S. Public Health Service at the National Institutes of Health. Editor of 12 books and author of over 350 research articles, Dr. Prusiner's contributions to scientific research have been internationally recognized.

Dr. Prusiner discovered an unprecedented class of pathogens that he named prions. Prions are infectious proteins that cause neurodegenerative diseases in animals and humans. Dr. Prusiner discovered a novel disease paradigm when he showed prions cause disorders in humans that can be manifest as (1) sporadic, (2) inherited and (3) infectious illnesses. Dr. Prusiner demonstrated that prions are formed when a normal, benign cellular protein acquires an altered shape. Dr. Prusiner's proposals of multiple shapes or conformations for a single protein as well as the concept of an infectious protein were considered heretical. Prior to Dr. Prusiner's discoveries, proteins were thought to possess only one biologically active conformation. Remarkably, the more common neurodegenerative diseases like Alzheimer's and Parkinson's diseases have been found over the past two decades to be, like the prion diseases, disorders of protein processing.

Dr. Prusiner is a member of the National Academy of Sciences, the Institute of Medicine, the American Academy of Arts and Sciences, the American Philosophical Society, and is a foreign member of the Royal Society, London. He is the recipient of numerous prizes, including the Potamkin Prize for Alzheimer's Disease Research from the American Academy of Neurology (1991); the Richard Lounsberry Award for Extraordinary Scientific Research in Biology and Medicine from the National Academy of Sciences (1993); the Gairdner Foundation International Award (1993); the Albert Lasker Award for Basic Medical Research (1994); the Paul Ehrlich Prize from the Federal Republic of Germany (1995); the Wolf Prize in Medicine from the State of Israel (1996); the Keio International Award for Medical Science (1996); the Louisa Gross Horwitz Prize from Columbia University (1997); and the Nobel Prize in Physiology or Medicine (1997).

Dr. Prusiner holds 50 issued or allowed United States patents all of which are assigned to the University of California.

2009 Invited Speakers (continued)



Journal of Pediatric Surgery Lecture:

Haile T. Debas, MD

"Global Health"

Haile T. Debas, MD, the Executive Director of UCSF Global Health Sciences, is recognized internationally for his contributions to academic medicine and is currently widely consulted on issues associated with global health.

A native of Eritrea, he received his MD from McGill University and completed his surgical training at the University of British Columbia.

At UCSF, he served as Dean (Medicine) (1993-2003), Vice Chancellor (Medical Affairs) for 6 years, and Chancellor for one year. A gastrointestinal surgeon by training, Dr. Debas is also the Maurice Galante Distinguished Professor of Surgery and chaired the UCSF Department of Surgery from 1986 to 2003.

Under Dr. Debas's stewardship, the UCSF School of Medicine became a national model for medical education, an achievement for which he was recognized with the 2004 Abraham Flexner Award of the AAMC. His prescient grasp of the implications of fundamental changes in science led him to create several interdisciplinary research centers that have been instrumental in reorganizing the scientific community at UCSF. He played a key role in developing UCSF's new campus at Mission Bay.

Dr. Debas has held leadership positions with numerous membership organizations and professional associations including serving as president of the American Surgical Association and chair of the Council of Deans of the AAMC. He has been a member of the Institute of Medicine since 1990, and is the current chair of the Membership Committee. He is a fellow of the American Academy of Arts and Sciences. He currently serves on the United Nations' Commission on HIV/AIDS and Governance in Africa, on the Committee on Science, Engineering, and Public Policy of the National Academy of Sciences, and as a member of the Board of Regents of the Uniformed Services University of the Health Sciences.

2009 Invited Speakers (continued)



Jay & Margie Grosfeld Lecture:

Michael T. Longaker, MD, MBA, FACS

“Regenerative Medicine: A Surgeon’s Perspective”

Michael T. Longaker earned his undergraduate degree at Michigan State University, (where he played varsity basketball and was a member of the 1979 NCAA Men’s Basketball Championship Team) and his medical degree at Harvard Medical School. He completed his surgical residency at the University of California, San Francisco, a residency in Plastic Surgery at NYU and a craniofacial fellowship at UCLA. The majority of his research training took place while he was a Post Doctoral Research Fellow in the Fetal Treatment Program under Dr. Michael R. Harrison and in the laboratory of Dr. Michael Banda in Radiobiology, both at UCSF. In December 2003, Dr. Longaker earned his M.B.A from University of California – Berkeley and Columbia University, in the inaugural class of their combined program. He was elected into Beta Gamma Sigma at Columbia Business School, which is the analog to Phi Beta Kappa for business programs.

Dr. Longaker joined the Stanford University School of Medicine on September 1, 2000, as Director of Children’s Surgical Research in the Department of Surgery, Division of Plastic and Reconstructive Surgery and the Lucile Salter Packard Children’s Hospital. In 2003, he was named the *Deane P. and Louise Mitchell Professor*. As Director of Children’s Surgical Research, Dr. Longaker has the responsibility to develop a children’s surgical research program in the broad areas of developmental biology, epithelial biology and tissue repair, and tissue engineering. Further, Dr. Longaker is the Deputy Director of the Stanford Institute of Stem Cell Biology & Regenerative Medicine, Director of the Program in Regenerative Medicine, Director of Research, Division of Plastic and Reconstructive Surgery, and has been name Professor, by Courtesy, in the Department of Bioengineering. He is also the Faculty Co-Chair for the Stanford University Initiative on Human Health.

Michael T. Longaker’s extensive research experience includes the cellular and molecular biology of extracellular matrix with specific applications to the differences between fetal and post-natal wound healing, the biology of keloids and hypertrophic scars and the cellular and molecular events that surround distraction osteogenesis with respect to craniofacial development. Most recently, his research has focused on multipotent mesenchymal cells derived from adipose tissue and their applications for tissue repair, replacement and regeneration. He brings to Stanford his unique understanding of wound healing, fetal wound healing research, developmental biology and tissue engineering.

Dr. Longaker is a member of all the major academic surgery societies and was president of the Society of University Surgeons (2007-08) and the Plastic Surgery Research Council (2006-07). He is one of a handful of surgeons elected into the American Society for Clinical Investigation, Association of American Physicians, and the prestigious Institute of Medicine of the National Academies. To date, he has over 925 publications and numerous federal grants to support his research.

2009 Invited Speakers (continued)



**International Guest Lecture:
Marcelo Martinez Ferro, MD
“New Approaches to Pectus and other MIS in Argentina”**

Dr. Marcelo Martinez Ferro was born in Buenos Aires and graduated from the Buenos Aires University School of Medicine in 1983. He completed his residency in pediatric surgery at the Ricardo Gutierrez Children’s Hospital and in 1988, joined the staff of Garrahan National Children’s Hospital. During his 15-year tenure at the hospital he dedicated himself to drastically improving the survival rate of newborn surgical patients whose mortality rate was historically very high in Argentina. In 1992 he completed a fellowship at the Fetal Treatment Center of the UCSF where he confirmed his interest in fetal treatment and video surgery.

Dr. Martinez Ferro developed numerous fetal, neonatal and pediatric surgical and MIS procedures, introducing them throughout South America. These unique procedures have earned him international recognition as a leader and pioneer in the field of pediatric MIS. Due to his broad experience in the treatment of malformations of the thoracic wall in particular, he is periodically invited to operate on patients and to train surgeons and around the globe.

Throughout his career, Dr. Martinez Ferro has maintained an active and busy academic and clinical practice. He is also an active member of many prominent surgical organizations and is the recent president elect of the International Pediatric Endosurgery Group. He has published nearly 100 articles on a multitude of pediatric surgery topics and remains a highly-requested lecturer and guest speaker for numerous surgical and medical societies around the world. In 2005, he published the first Spanish textbook on neonatal surgery “Neonatología Quirúrgica,” which remains a Latin American bestseller.

Dr. Martinez Ferro is currently the professor of surgery and pediatrics, Chief Division of Pediatric Surgery, at the Fundacion Hospitalaria Children’s Hospital and the Coordinator of Fetal Treatment Center at the CEMIC University Hospital in Buenos Aires. Dr. Martinez Ferro and his wife Valeria live in Buenos Aires with their three children.

Program in Detail

Wednesday, May 27

Kiwanis Pediatric Trauma Institute 7th International Conference on Pediatric Trauma

7:00 a.m. – 8:00 a.m. Registration *Atlantic Foyer*

7:30 a.m. – 8:00 a.m. Continental Breakfast *Grand Caribbean Terrace*

8:00 a.m. – 5:00 p.m. **Kiwanis Pediatric Trauma Symposium** *Grand Caribbean 6-8*

Educational Objectives:

The goal of this symposium is to stimulate dialog and intellectual synergy among leading experts in pediatric and adult fields relevant to care of the injured child, focusing on resuscitation.

8:00 a.m. – 8:15 a.m. **Welcome/Opening Remarks** *Grand Caribbean 6-8*

Brian F. Gilchrist, MD, Conference Co-Chair, KPTI

Randall S. Burd, MD, Conference Co-Chair, APSA

Arthur Cooper, MD, Conference Co-Chair, Program

Planning Committee

8:15 a.m. – 9:15 a.m. **Pediatric Injury Prevention** *Grand Caribbean 6-8*

Moderator:

Barbara A. Gaines, MD

How Safe Are Our Kids?

Martin R. Eichelberger, MD

Children's National Medical Center, Washington, DC, USA

How "Injury Free" Are Our Kids?

Peter P. Hirsch, MD

University of Massachusetts Memorial Center, Worcester, MA, USA

Injury Screening and Brief Intervention

Peter F. Ehrlich, MD

CS Mott Children's Hospital, Ann Arbor, MI, USA

Panel Discussion

9:15 a.m. – 10:15 a.m. **Recognition, Referral, Resuscitation** *Grand Caribbean 6-8*

Moderator:

Kenneth H. Sartorelli, MD

Early Indicators of Major Trauma in Children

Michael L. Nance, MD

Children's Hospital of Philadelphia, Philadelphia, USA

Field Triage Guidelines for Injured Children

Arthur Cooper, MD

Columbia University at Harlem Hospital, New York, NY, USA

Program in Detail

Wednesday, May 27 (continued)

Termination of Resuscitation: When to Stop?

Mary E. Fallat, MD

Kosair Children's Hospital, Louisville, KY, USA

Panel Discussion

10:15 a.m. – 10:30 a.m.	Refreshment Break	<i>Grand Caribbean Terrace</i>
10:30 a.m. – 11:30 a.m.	What We Do Know	<i>Grand Caribbean 6-8</i>

Moderator:

Jonathan I. Groner, MD

Who Gets Hurt?

Laura D. Cassidy, PhD

Children's Hospital of Wisconsin, Milwaukee, WI, USA

Who Gets Better?

Henri R. Ford, MD

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

Controlling the Damage

Steven Stylianos, MD

Miami Children's Hospital, Miami, FL, USA

Panel Discussion

11:30 a.m. – 12:30 p.m.	What We Don't Know	<i>Grand Caribbean 6-8</i>
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Moderator

Mindy B. Statter, MD

University of Texas Health Science Center at Houston, Houston, TX, USA

Cerebral Resuscitation

Charles S. Cox, Jr., MD

University of Texas Health Science Center at Houston, Houston, TX, USA

Volume Resuscitation

Denis S. Bensard, MD

Cincinnati CHMC St. Vincent Children's Hospital, Cincinnati, OH, USA

Ob(liv)ious Outcomes

Randall S. Burd, MD

Robert Wood Johnson Medical School, New Brunswick, NJ, USA

Panel Discussion

12:30 p.m. – 1:15 p.m.	Luncheon	<i>Grand Caribbean Salon 4</i>
1:15 p.m. – 2:00 p.m.	Presentation of Kiwanis Award for Service to Injured Children	<i>Grand Caribbean Salon 4</i>
	Brian F. Gilchrist, MD – Kiwanis Pediatric Trauma Institute	

Program in Detail

Wednesday, May 27 (continued)

GenRe Lecture: Mass Casualty Management

Madness in Madrid

Juan Vazquez, MD

Hospital Infantil La Paz, Madrid, Spain

TBD

Nicholas G. Guerina, MD, PhD

Floating Hospital for Children, Boston, MA, USA

2:00 p.m. – 3:00 p.m. **Children in War and Peace** *Grand Caribbean 6-8*

Moderator

Michael M. Fuenfer, MD

Gulf War I

Brian F. Gilchrist, MD

Floating Hospital for Children, Boston, MA, USA

Gulf War II

Kenneth S. Azarow, MD

Children's Hospital and Medical Center, Omaha, NE, USA

Argentina

Alberto E. Inon, MD, PhD

Asociacin Prevencion del Trauma Pediatrico

Panel Discussion

3:00 p.m. – 4:00 p.m. **Rescue, Relief, Rehabilitation** *Grand Caribbean 6-8*

Moderator

Guy F. Brisseau, MD

Diminishing Disasters

Jeffrey S. Upperman, MD

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

I Want My Mom!

Diana G. Fendya, MD

Children's National Medical Center, Washington, DC, USA

Once They Go Home

Andrea L. Winthrop, MD

Children's Hospital of Wisconsin, Milwaukee, WI, USA

Panel Discussion

4:00 p.m. – 4:15 p.m. Refreshment Break *Grand Caribbean Terrace*

4:15 p.m. – 4:45 p.m. **Open Discussion** *Grand Caribbean 6-8*
Pediatric Trauma Research Agenda For The Future

Moderator

Joseph J. Tepas, III, MD

Program in Detail

Wednesday, May 27 (continued)

4:45 p.m. – 5:00 p.m.	Wrap Up and Next Steps Conference Co-Chairs Brian F. Gilchrist, MD, Kiwanis Pediatric Trauma Institute Randall S. Burd, MD, American Pediatric Surgical Association Arthur Cooper, MD, Program Planning Committee	Grand Caribbean 6-8
APNSA Bariatrics Session	11:15 a.m. – Noon	Grand Atlantic 1
APSA Meetings		
7:00 a.m. – 5:00 p.m.	Registration Open	Atlantic Foyer
10:00 a.m. – 3:00 p.m.	Board of Governors Meeting	Boardroom I
1:00 p.m. – 9:00 p.m.	Committee Meetings	Palmas B, Las Croabas A, Las Croabas B, Siete Mares A, Siete Mares B
2:00 p.m. – 6:00 p.m.	Training Program Directors Meeting	Grand Caribbean 1-3
7:30 p.m. – 10:00 p.m.	Board of Governors Dinner	The Strip House

Thursday, May 28

6:00 a.m. – 7:30 a.m.	Committee Meetings	Palmas B, Las Croabas A, Las Croabas B, Siete Mares A, Siete Mares B, Ceiba A, Ceiba B, Grand Caribbean 7, Grand Caribbean 8
7:00 a.m. – 5:30 p.m.	Registration Open	Atlantic Foyer
7:00 a.m. – 7:30 a.m.	Continental Breakfast	Atlantic Foyer
7:30 a.m. – 7:45 a.m.	Welcome – Michael R. Harrison, MD	Grand Atlantic 2 & 3
7:45 a.m. – 11:00 a.m.	Education Session I: Pediatric Trauma/Critical Care	Grand Atlantic 2 & 3

Moderator

Walt Chwals, MD

Educational Objectives:

The first part of this symposium will focus on three topics of developing controversy in the management of pediatric trauma. A debate format will be used with two designated discussants (one pro versus one con) for each of these three debate topics (15 min presentation for each discussant with 10 min moderated question/answer period for each topic). Interactive electronic technology will allow for active audience participation during the debates. The second part of the symposium will include the presentation of novel therapeutic strategies in the treatment of traumatic brain injury (TBI) which hold remarkable promise for dramatic improvement in functional recovery. By the end of the symposium, the audience participant will be able to understand the contemporary merits and drawbacks of:

- 1) Standard 3-view radiographs versus computer tomography (CT) to initially evaluate traumatic C-spine injury in children.
- 2) Focused Abdominal Sonography for Trauma (FAST) versus CT to evaluate blunt abdominal trauma in children.
- 3) Embolization versus laparotomy to manage trauma-associated splenic hemorrhage in children.
- 4) The audience participant will be able to understand and discuss the mechanisms' rehabilitative potential of progenitor cell restoration strategies for treatment of TBI.

Program in Detail

Thursday, May 28 (continued)

C-SPINE CLEARANCE: RADIOGRAPHS VS CT

Jonathan I. Groner, MD
Columbus Children's Hospital, Columbus, OH, USA
Peter F. Ehrlich, MD
CS Mott Children's Hospital, Ann Arbor, MI, USA

ABDOMINAL IMAGING: FAST VS CT

Robert Letton, MD
Oklahoma University Health Sciences Center, Oklahoma City, OK, USA
Eric Scaife, MD
University of Utah School of Medicine, Salt Lake City, UT, USA

SPLENIC INJURY CARE: EMBOLIZATION VS LAPAROTOMY

Michael L. Nance, MD
Children's Hospital of Philadelphia, Philadelphia, PA, USA
Barbara A. Gaines, MD
Children's Hospital of Pittsburgh, Pittsburgh, PA USA

SPECIAL LECTURE: PROGENITOR CELL THERAPIES FOR BRAIN INJURY

Charles S. Cox, MD
University of Texas Health and Sciences Center at Houston, Houston, TX, USA

11:00 a.m. – 11:15 a.m.	Refreshment Break	Atlantic Foyer
11:15 a.m. – 11:30 a.m.	Introduction of New Members	Grand Atlantic 2 & 3
11:30 a.m. – 12:30 p.m.	Presidential Address: Michael R. Harrison, MD	Grand Atlantic 2 & 3
12:30 p.m. – 12:45 p.m.	Refreshment Break and Box Lunch Pick Up	Atlantic Foyer
12:45 p.m. – 1:45 p.m.	Video Session with Lunch	Grand Atlantic 2 & 3

Moderators:

John R. Gosche, MD; Peter F. Ehrlich, MD

Educational Objectives:

Participants in this session will learn about:

- Laparoscopic approaches for treating morbid obesity, pelvic organ prolapse, pancreatic pseudocyst and choledochal cyst.
- Thoracoscopic anatomic lingulectomy.
- Percutaneous laparoscopic assisted inguinal hernia repair.
- Redo transanal pull-through for Hirschsprung's disease.

V1 REDO TRANSANAL PULL-THROUGH FOR HIRSCHSPRUNG'S DISEASE

Belinda Dickie, MD, Marc A. Levitt, MD, Andrea Bischoff, Alberto Pena.
Cincinnati Children's Hospital, Cincinnati, OH, USA

V2 THORACOSCOPIC ANATOMIC LINGULECTOMY FOR CHRONIC SEGMENTAL BRONCHIAL OBSTRUCTION IN A CHILD

Steven S. Rothenberg¹, Marzena Krawiec²

¹The Rocky Mountain Hospital for Children, Denver, CO, USA, ²National Jewish Medical and Research Center, Denver, CO, USA

Program in Detail

Thursday, May 28 (continued)

- V3 PERCUTANEOUS ENDOSCOPICALLY ASSISTED REPAIR (PEAR) OF INGUINAL HERNIA**
Justin D. Klein, MD, Christopher G. B. Turner, MD, Dario O. Fauza, MD, Russell W. Jennings, MD
Children's Hospital Boston, Boston, MA, USA
- V4 LAPAROSCOPIC SLEEVE GASTRECTOMY FOR THE TREATMENT OF A MORBIDLY OBESE ADOLESCENT**
Evan P. Nadler, MD, Manish Parikh, MD
New York University School of Medicine, New York, NY, USA
- V5 LAPAROSCOPIC REPAIR OF PELVIC ORGAN PROLAPSE**
Christopher G. Turner, MD, Justin D. Klein, MD, Dario O. Fauza, MD, Russell W. Jennings, MD
Children's Hospital Boston, Boston, MA, USA
- V6 LAPAROSCOPIC TYPE 1 CHOLEDOCHAL CYST EXCISION IN A 6 MONTH OLD INFANT**
Grace Z. Mak, MD, Donald C. Liu, MD, PhD
University of Chicago, Chicago, IL, USA
- V7 HYBRID NOTES: INCISIONLESS INTRAGASTRIC STAPLED CYSTGASTROSTOMY OF A PANCREATIC PSEUDOCYST**
Kevin P. Moriarty, MD¹, Connie J. Rossini, MD², Anastasios G. Angelides, MD¹
¹*Baystate Children's Hospital, Springfield, MA, USA*, ²*Baystate Medical Center, Springfield, MA, USA*

1:45 p.m. – 2:00 p.m. Refreshment Break *Atlantic Foyer*

2:00 p.m. – 4:00 p.m. **Concurrent Education Sessions II & III**

Education Session II: Interesting Neonatal Cases *Grand Caribbean 5*

Mac Harmon, MD
Children's Hospital of Alabama, Birmingham, AL, USA

Education Session III: Controversies in Contracting *Grand Atlantic 2 & 3*

Why Consider a Contract

Donald Shaul, MD
Children's Hospital of Los Angeles, Sherman Oaks, CA, USA

Getting a Successful Contract

Donald Shaul, MD
Children's Hospital of Los Angeles, Sherman Oaks, CA, USA

Legal Matters

Erin Muellenberg, Esq
Reback, MacAndrews and Kjar, Manhattan Beach, CA, USA

Program in Detail

Thursday, May 28 (continued)

How To Negotiate The Deal

Henri R. Ford, MD

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

What To Do If The Hospital Fails To Honor The Contract

J. Duncan Phillips, MD

University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

Panel

4:00 p.m. – 4:15 p.m.	Refreshment Break	<i>Atlantic Foyer</i>
4:15 p.m. – 5:45 p.m.	Concurrent Poster Sessions I & II: Poster Session I: Basic Science & Oncology	<i>Icaco</i>

Moderators:

Karl Sylvester, MD; R. Cartland Burns, MD

Educational Objectives:

At the end of the session, participants will be able to:

- Describe the impact of stem cells on development and disease in lung and intestine.
- Explain mechanical issues associated with lung development.
- Compare inflammation and scar formation at various stages of development.
- Propose an etiologic mechanism for biliary atresia.
- List biologic modifiers affecting neuroblastoma cells.
- State factors involved in tumor cell growth and dissemination.

P1 OVER-EXPRESSION OF FGF10 INCREASES TRANSIT-AMPLIFYING CELLS BUT DOES NOT AFFECT INTESTINAL STEM CELLS

Cindy C. Tai, MD, Frederic G. Sala, PhD, Henri R. Ford, MD, Kasper S. Wang, MD, Tracy C. Grikscheit, MD, Saverio Bellusci, PhD
Children's Hospital Los Angeles, Los Angeles, CA, USA

P2 INTESTINAL STEM CELLS ARE IDENTIFIED IN MATURE NATIVE AND TISSUE-ENGINEERED SMALL INTESTINE

Frederic G. Sala, PhD, Tracy C. Grikscheit, MD
Children's Hospital Los Angeles, Los Angeles, CA, USA

P3 AMNIOTIC FLUID STEM CELLS ENHANCE SURVIVAL OF RATS WITH EXPERIMENTAL NEC RECEIVING HIGHER DOSE OF LIPOPOLYSACCHARIDE

Augusto Zani, MD, Simon Eaton, PhD, Mara Cananzi, MD, Giuseppe Lauriti, MD, Agostino Pierro, MD, Paolo De Coppi, MD, PhD
Institute of Child Health – University College London, London, United Kingdom

P4 STEM CELL THERAPY FOR PREMATURE LUNGS

Christine M. Finck, Blair R. Roszell, Ariel Seaton, BS
Connecticut Children's Medical Center, Hartford, CT, USA

Program in Detail

Thursday, May 28 (continued)

- P5 ENGINEERING AN ARTIFICIAL ALVEOLAR MEMBRANE: A NOVEL CONTINUOUSLY-PERFUSED MODEL WITHIN MICROCHANNELS**
Divya D. Nalayanda, Leilani M. Sharpe, William B. Fulton, Christopher M. Puleo, Tza-Huei Wang, PhD, Fizan Abdullah, MD, PhD
Johns Hopkins University, Baltimore, MD, USA
- P6 CONDITIONAL INACTIVATION OF EPITHELIAL C-MET SIGNALING CAUSES ABNORMAL LUNG GROWTH AND DEVELOPMENT**
Tim Jancelewicz, MD¹, Eun Jun Yun, Ph D¹, Walter Lorizio¹, Kerilyn K. Nobuhara, MD¹, Sam Hawgood, MD¹, Leland G. Dobbs, MD¹, Snorri S. Thorgeirsson, MD, PhD², Thiennu H. Vu, MD, PhD¹
¹*University of California, San Francisco, San Francisco, CA, USA*, ²*Laboratory of Experimental Carcinogenesis, Center for Cancer Research, National Cancer Institute, Bethesda, MD, USA*
- P7 INFLAMMATORY BOWEL DAMAGE IS AMELIORATED BY IN-UTERO REPAIR OF GASTROSCHISIS IN THE SHEEP MODEL**
Jacob T. Stephenson, MD¹, Kullada O. Pichakron, MD², Kevin Grayson, DVM, PhD², Lan Vu, MD³, Ramin Jamshidi, MD³, Tim Jancelewicz, MD³, Kerilyn K. Nobuhara, MD³
¹*University of California, Davis, Sacramento, CA, USA*, ²*David Grant Medical Center, Travis AFB, CA, USA*, ³*University of California, San Francisco, San Francisco, CA, USA*
- P8 INHIBITION OF INTRA-ABDOMINAL ADHESION FORMATION IN A RABBIT MODEL WITH THE ANGIOGENESIS INHIBITOR SUNITINIB**
Jonathan A. Meisel, MD, Hau D. Le, MD, Vincent E. de Meijer, MD, MSc, Mark Puder, MD, PhD
Children's Hospital Boston, Boston, MA, USA
- P9 WOUND SIZE MODULATES FETAL SCAR FORMATION THROUGH PROINFLAMMATORY CYTOKINE GENE EXPRESSION AND RECRUITMENT OF INFLAMMATORY CELLS**
Benjamin J. Herdrich, MD, Enrico Danzer, MD, Marcus G. Davey, PhD, Dustin M. Bermudez, MD, Antoneta Radu, Liping Zhang, Kenneth W. Liechty, MD
Children's Hospital of Philadelphia, Philadelphia, PA, USA
- P10 TEMPORAL SUSCEPTIBILITY OF PRIMARY MURINE CHOLANGIOCYTES MIMICS THE MURINE MODEL OF BILIARY ATRESIA.**
Alexander Bondoc, MD¹, Sujit Mohanty, PhD¹, Bryan Donnelly, BS¹, Mubeen Jafri, MD², Greg Tiao, MD¹
¹*Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA*, ²*University of Cincinnati College of Medicine, Cincinnati, OH, USA*
- P11 CHOLANGIOCYTE APOPTOSIS DURING LAMPREY METAMORPHOSIS**
Laura A. Boomer, MD¹, Seth A. Bellister, BS¹, Stanley Hillyard, PhD², John H. Youson, PhD³, John R. Gosche, MD, PhD¹
¹*University of Nevada School of Medicine, Las Vegas, NV, USA*, ²*University of Nevada Las Vegas, Las Vegas, NV, USA*, ³*University of Toronto, Toronto, ON, Canada*

Program in Detail

Thursday, May 28 (continued)

- P12 INHIBITION OF PLACENTA-LIKE GROWTH FACTOR INCREASES METASTASIS IN EXPERIMENTAL NEUROBLASTOMA**
Mary Jo Haley, MD, Jason C. Fisher, MD, Jeffrey W. Gander, MD, Sonia L. Hernandez, Yoon-Jung Boo, Jianzhong Huang, Darrell Yamashiro, MD, PhD, Jessica J. Kandel, MD
Morgan Stanley Children's Hospital of New York Presbyterian, Columbia University Medical Center, New York, NY, USA
- P13 HSP-70 INHIBITION WITH TRIPTOLIDE THERAPY CAUSES APOPTOSIS IN NEUROBLASTOMA**
Mara B. Antonoff, Daniel Borja-Cacho, MD, Rohit Chugh, MD, Brent Sorenson, Daniel A. Saltzman, MD, PhD, Ashok K. Saluja, PhD, Selwyn M. Vickers, MD, Thao Marquez, MD
University of Minnesota, Minneapolis, MN, USA
- P14 IMPROVED INHIBITION OF NEUROBLASTOMA WITH COMBINATION THERAPY OF ENRICHED OMEGA-3 DIET AND SUNITINIB OVER EITHER AGENT ALONE**
Emily R. Christison-Lagay, MD¹, Daniela Prox, MD², Deepak Panigraphy, MD², Hau Le, MD², Catherine E. Butterfield, BSc², Mark Puder, MD, PhD², Steven J. Fishman, MD², Carmen M. Barnes, PhD²
¹*Massachusetts General Hospital, Boston, MA, USA*, ²*Children's Hospital Boston, Boston, MA, USA*
- P15 IDENTIFYING BINDING PARTNERS OF THE TRANSCRIPTIONAL CO-ACTIVATOR, CITED¹, THAT REGULATE ITS NUCLEAR TRAFFICKING AND FUNCTION IN WILMS TUMOR**
Harold N. Lovvorn, III, MD, Amy Joan Ham, PhD, Jenifer Westrup, Mark deCaestecker, MD, PhD
Vanderbilt University Children's Hospital, Nashville, TN, USA
- P16 A NOVEL METHOD FOR DETECTION OF CIRCULATING TUMOR CELLS IN METASTASIZING EXPERIMENTAL PEDIATRIC SOLID TUMORS**
Jeffrey W. Gander, MD, Sonia Hernandez, MA, Jason C. Fisher, MD, Mary Jo Haley, MD, Jianzhong Huang, MD, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD
Children's Hospital of New York-Presbyterian, New York, NY, USA
- P17 TUMOR CELL LYSATE PREPARATION IS CRITICAL TO THE EFFICACY OF WHOLE TUMOR CELL VACCINE STRATEGIES**
Steven T. Elliott, MD, Suzanne A. Miles, PhD, Russell Williams, Alana Boyajian, Mark Kurzrok, Santiago Arciniegas, Wouter Kopp, Anthony D. Sandler, MD
Children's National Medical Center, Washington, DC, USA
- P18 MÜLLERIAN INHIBITING SUBSTANCE INHIBITS MIGRATION OF EPITHELIAL CANCER CELL LINES**
Henry L. Chang, MD, Rafael Pieretti-Vanmarcke, MD, Fotini Nicolaou, David T. MacLaughlin, PhD, Patricia K. Donahoe, MD
Massachusetts General Hospital, Boston, MA, USA

Program in Detail

Thursday, May 28 (continued)

P19 TOLL-LIKE RECEPTOR 8 AS A POTENTIAL THERAPEUTIC TARGET FOR SOFT TISSUE AND BONE SARCOMAS

Linda Adepoju, MD, Weiping Zou, MD, PhD, James D. Geiger, MD
University of Michigan, Ann Arbor, MI, USA

4:15 p.m. – 5:45 p.m.

**Poster Session II:
Clinical & Fetal Surgery**

Vieques

Moderators:

Dan von Allmen, MD; Moritz Zeigler, MD

Educational Objectives:

At the end of the session, participants will be able to:

- Identify factors impacting long term outcome for patients with congenital diaphragmatic hernia.
- State outcomes for pectus excavatum repair in reoperative and Marfan Syndrome patients.
- Describe management issues associated with complex liver diseases.
- List outcomes and treatment options for patients with anorectal malformations.
- Recognize risk of unnecessary radiation exposure in trauma patients.
- Identify techniques for assessing congenital anomalies in utero.

P20 THE EFFECT OF PRENATAL DIAGNOSIS ON THE CONTEMPORARY OUTCOME OF CONGENITAL DIAPHRAGMATIC HERNIA

Jeremy Grushka, MD¹, Jean-Martin Laberge, MD¹, Pramod Puligandla, MD, MSc¹, Erik D. Skarsgard, MD², The Canadian Pediatric Surgery Network
¹Department of Pediatric Surgery, Montreal Children's Hospital, McGill University, Montreal, QC, Canada, ²Division of Pediatric Surgery, BC, Children's Hospital, Vancouver, BC, Canada

P21 OUTCOMES FOLLOWING MUSCLE FLAP VERSUS PROSTHETIC PATCH REPAIR FOR LARGE DIAPHRAGMATIC HERNIAS

Ahmed Nasr, Marie-Chantal Struijs, Sigmund H. Ein, Jacob C. Langer, Priscilla P.L. Chiu
The Hospital for Sick Children, Toronto, Ontario, Canada

P22 MINIMALLY INVASIVE REPAIR OF PECTUS EXCAVATUM IN PATIENTS WITH MARFAN SYNDROME AND MARFANOID FEATURES

Richard E. Redlinger, Jr., MD¹, Gregory D. Rushing, MD¹, Alan B. Moskowitz, MS², Robert E. Kelly, Jr., MD², Donald Nuss, MB, ChB², M. Ann Kuhn, MD², Robert J. Obermeyer, MD², Michael J. Goretsky, MD²
¹Eastern Virginia Medical School, Norfolk, VA, USA, ²Children's Hospital of The King's Daughters, Norfolk, VA, USA

P23 USE OF AN ABSORBABLE STABILIZER FOR PECTUS EXCAVATUM REPAIR

Donald Nuss, MB, ChB¹, Robert E. Kelly, Jr., MD¹, Richard E. Redlinger, Jr., MD², Tina Haney, RN, MSN¹
¹Children's Hospital of The King's Daughters, Norfolk, VA, USA, ²Eastern Virginia Medical School, Norfolk, VA, USA

Program in Detail

Thursday, May 28 (continued)

- P24 RETROSPECTIVE REVIEW OF RE-OPERATIVE PECTUS EXCAVATUM REPAIRS**
Mara B. Antonoff, MD, Daniel A. Saltzman, MD, PhD, Donavon J. Hess, MD, PhD MBA, Robert D. Acton, MD
University of Minnesota, Minneapolis, MN, USA
- P25 13C-METHIONINE BREATH TEST TO ASSESS LIVER DISEASE IN CHILDREN WITH INTESTINAL FAILURE**
Debora Duro, MD¹, Shamae C. Fitzgibbons, MD², Clarissa Valim, MD, ScD³, Melissa Hull, MD², Lori Bechard⁴, Yong-Ming Yu, PhD⁵, Christopher Duggan, MD⁶, Tom Jaksic, MD, PhD²
¹Center for Advanced Intestinal Rehabilitation, Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA, ²Center for Advanced Intestinal Rehabilitation, Department of Surgery, Children's Hospital Boston, Boston, MA, USA, ³Department of Surgery, Children's Hospital Boston, Boston, MA, USA, ⁴Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA, ⁵Shriners Hospital for Children, Boston, MA, USA, ⁶Center for Advanced Intestinal, Rehabilitation, Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA
- P26 PORTOSYSTEMIC SHUNTS IN CHILDREN**
Joseph B. Lillegard, MD, PhD, Angela M. Hanna, MD, Travis J. McKenzie, MD, Christopher R. Moir, MD, David M. Nagorney, MD, Michael B. Ishitani, MD
Mayo Medical Center, Rochester, MN, USA
- P27 ALAGILLE SYNDROME: OUTCOME COMPARISON OF CONSERVATIVE TREATMENT VERSUS KASAI PROCEDURE**
Adam J. Kaye, MD¹, Elizabeth Rand, MD², Pedro Munoz, BA², Nancy B. Spinner, PhD², Alan W. Flake, MD², Binita M. Kamath, MBChir²
¹Hospital of the University of Pennsylvania, Philadelphia, PA, USA, ²Children's Hospital of Philadelphia, Philadelphia, PA, USA
- P28 REGIONALIZATION OF PEDIATRIC LIVER TRANSPLANTATION IN THE UNITED STATES**
Elisabeth T. Tracy, MD, Melissa E. Danko, MD, Tammy J. Westmoreland, MD, Debra L. Sudan, MD, Bradley H. Collins, MD, Janet E. Tuttle-Newhall, MD, Carlos E. Marroquin, MD, Henry E. Rice, MD, Paul C. Kuo, MD, Theodore N. Pappas, MD, John E. Scarborough, MD
Duke University, Durham, NC, USA
- P29 TRANSLATIONAL APPROACH TO IDENTIFY DOXORUBICIN RESISTANT GENES IN NEUROBLASTOMA**
Tamarah J. Westmoreland, MD, PhD, Gudrun Huper, MS, Melissa E. Danko, MD, Elisabeth T. Tracy, MD, Jeffrey C. Hoehner, MD, PhD, Henry E. Rice, MD, Jeffrey R. Marks, PhD, Craig B. Bennett, PhD
Duke University Medical Center, Durham, NC, USA

Program in Detail

Thursday, May 28 (continued)

- P30 HOW TO APPROACH THE OVARIAN MASS: CAN WE RISK STRATIFY FOR MALIGNANCY?**
Sarah C. Oltmann, MD¹, Nilda Garcia, MD¹, Robert Barber, RN², Rong Huang², Barry Hicks, MD¹, Anne Fischer, MD, PhD¹
¹University of Texas Southwestern Medical Center, Dallas, TX, USA, ²Children's Medical Center, Dallas, TX, USA
- P31 THE LONG-TERM FUNCTIONAL OUTCOMES AND QUALITY OF LIFE OF ADOLESCENTS AND ADULTS WITH HIRSCHSPRUNG'S DISEASE AND ANORECTAL MALFORMATIONS**
Thao T. Marquez, MD¹, Daniel A. Saltzman, MD, PhD¹, Samuel D. Smith, MD², Graham H. Cosper, MD², Robert D. Acton, MD¹, David A. Rothenberger, MD¹
¹University of Minnesota, Minneapolis, MN, USA, ²University of Arkansas for Medical Sciences, Little Rock, AR, USA
- P32 MRI GUIDED LAPAROSCOPIC ASSISTED ANORECTOPLASTY FOR IMPERFORATE ANUS**
George R. Raschbaum, MD, John C. Bleacher, MD, J. Damien Grattan-Smith, MD, Richard A. Jones, PhD
Children's Healthcare of Atlanta at Scottish Rite, Atlanta, GA, USA
- P33 TOPICAL MITOMYCIN-C FOR THE TREATMENT OF ANAL STENOSIS**
Claudia M. Mueller, PhD, MD¹, Mona Beaunoyer, MD², Arie Bensoussan, MD², Dickens St-Vil, MD², Sami Youssef, MD
¹Stanford University, Palo Alto, CA, USA, ²University of Montreal, Montreal, QC, Canada
- P34 DEVELOPMENT AND IMPLANTATION OF A BIOARTIFICIAL ANAL SPHINCTER**
Mohamed S. Hashish, Khalil N. Bittar, Robert R. Gilmont, Shreya Raghan, Eiichi Miyaska, Manabu Okawada, Daniel H. Teitelbaum
University of Michigan, Ann Arbor, MI, USA
- P35 REPEAT ABDOMINAL CT SCANS AFTER PEDIATRIC BLUNT TRAUMA: MISSED INJURIES, EXTRA COSTS, & UNNECESSARY RADIATION EXPOSURE**
Steven H. Cook, J. Duncan Phillips, Julia R. Fielding
University of North Carolina at Chapel Hill, Chapel Hill, NC, USA
- P36 LONG-TERM OUTCOMES OF PEDIATRIC SPLENIC INJURY IN CALIFORNIA**
Howard C. Jen, MD, Areti Tillou, MD MsEd, Stephen B. Shew, MD
David Geffen School of Medicine at UCLA, Los Angeles, CA, USA
- P37 REFLECTANCE SPECTROMETRY FOR REALTIME HEMOGLOBIN DETERMINATION OF PLACENTAL VESSELS DURING ENDOSCOPIC LASER SURGERY FOR TTTS**
Sean Curran, John McMurdy, PhD, John McMurdy, PhD, Stephen R. Carr, MD, Christopher S. Muratore, MD, Babara M. O'Brien, MD, Gregory P. Crawford, PhD, Francois I. Luks, MD, PhD
Brown Medical School, Providence, RI, USA

Program in Detail

Thursday, May 28 (continued)

P38	PRENATAL URINARY MATRIX METALLOPROTEINASE PROFILING AS A POTENTIAL DIAGNOSTIC TOOL IN FETAL OBSTRUCTIVE UROPATHY <i>Grace A. Nicksa, MD¹, David C. Yu, MD¹, Adam Curatolo², Brendan L. McNeish², Carol E. Barnewolt, MD³, Clarissa Valim, MD, ScD¹, Terry L. Buchmiller, MD¹, Marsha A. Moses, PhD², Dario O. Fauza, MD¹</i> ¹ <i>Children's Hospital Boston, Department of Surgery, Boston, MA, USA,</i> ² <i>Children's Hospital Boston, Vascular Biology Program, Boston, MA, USA,</i> ³ <i>Children's Hospital Boston, Department of Radiology, Boston, MA, USA</i>
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6:30 p.m. – 8:30 p.m.	Welcome Reception	<i>Main Pool</i>
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Friday, May 29

6:00 a.m. – 7:30 a.m.	Annual Fun Run	<i>Atlantic Terrace</i>
6:30 a.m. – 7:30 a.m.	Committee Meetings	<i>Palmas B, Las Croabas A, Las Croabas B, Siete Mares A, Siete Mares B, Ceiba A, Ceiba B, Grand Caribbean 7, Grand Caribbean 8</i>
6:30 a.m. – 10:00 a.m.	Poster Set Up	<i>Icaco & Vieques</i>
6:30 a.m. – 2:00 p.m.	Registration Open	<i>Atlantic Foyer</i>
6:45 a.m. – 7:30 a.m.	Continental Breakfast	<i>Atlantic Foyer</i>
6:45 a.m. – 1:15 p.m.	Exhibits Open	<i>Atlantic Foyer</i>
7:30 a.m. – 9:00 a.m.	Scientific Session I: Common Problems	<i>Grand Atlantic 2 & 3</i>

Moderators:

Tom Jaksic, MD; Peter F. Ehrlich, MD

Educational Objectives:

After this session, participants will be able to:

- Cite specific issues relating to CT diagnosis and management of perforated appendicitis.
- Discuss the value of partial splenectomy for hereditary spherocytosis and predictors of response to splenectomy in patients with ITP.
- Describe interim results of gastric banding for treatment of adolescents with morbid obesity.
- Identify potential options to limit liver injury in patients on TPN.

1 ACCURACY OF COMPUTED TOMOGRAPHY IN PREDICTING APPENDICEAL PERFORATION

Shawn D. St. Peter, MD, *Jason D. Fraser, MD*, Pablo Aguayo, MD, Susan W. Sharp, PhD, Charles J. Snyder, MD, Douglas Rivard, MD, Ronald J. Sharp, MD, Brent Cully, MD, Daniel J. Ostlie, MD
Children's Mercy Hospital, Kansas City, MO, USA

Program in Detail

Friday, May 29 (continued)

- 2 LAPAROSCOPIC APPENDECTOMY UPON PRESENTATION VERSUS INTERVAL APPENDECTOMY FOR PERFORATED APPENDICITIS WITH ABSCESS: A PROSPECTIVE, RANDOMIZED TRIAL**
Pablo Aguayo, MD, Jason D. Fraser, MD, Susan W. Sharp, PhD, Charles M. Leys, MD, Charles L. Snyder, MD, J Patrick Murphy, MD, Walter S. Andrews, MD, Ronald J. Sharp, MD, George W. Holcomb III, MD, MBA, Daniel J. Ostlie, MD, Shawn D. St. Peter
Children's Mercy Hospital, Kansas City, MO, USA
- 3 OPERATIVE MANAGEMENT OF INTRACTABLE CONSTIPATION IN CHILDREN**
Emily R. Christison-Lagay, MD, Michael P. Kurtz, MD, Daniel P. Doody, MD, Leonel Rodriguez, MD, Allan M. Goldstein, MD
Massachusetts General Hospital, Boston, MA, USA
- 4 SUBTOTAL SPLENECTOMY FOR HEREDITARY SPHEROCYTOSIS**
Jordan R. Gutweiler, MD, Meghna V. Misra, MD, Biren P. Modi, MD, Matthew Y. Suh, MD, Matthew M. Heeney, MD, Robert C. Shamberger, MD
Children's Hospital Boston, Boston, MA, USA
- 5 PREDICTING RESPONSES TO SPLENECTOMY IN CHILDREN WITH IMMUNE THROMBOCYTOPENIC PURPURA**
James H. Wood, David A. Partrick, Taru Hays, Moritz M. Ziegler
The Children's Hospital, University of Colorado Health Sciences Center, Denver, CO, USA
- 6 LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING (LAGB) FOR ADOLESCENTS IN THE FDA-IDE TRIAL: AN INTERIM REPORT OF WEIGHT, METABOLIC AND QUALITY OF LIFE OUTCOME**
Mark Holterman, MD, PhD¹, Allen Browne¹, Lydia Kruge², Amyar Pandya², Lisa Tussing¹, Sandra Gomez¹, Rui Liu, PhD², Amy Phipps², Nancy Browne¹, Ai-Xuan L. Holterman, MD²
¹*The New Hope Pediatric and Adolescent Weight Management Program, Chicago, IL, USA*, ²*RUSH University Medical Center, Chicago, IL, USA*
- 7 EFFECTS OF GLUTAMINE-SUPPLEMENTED PARENTERAL NUTRITION ON LIVER FUNCTION IN SURGICAL INFANTS: RESULTS OF A RANDOMISED CONTROLLED TRIAL**
Simon Eaton¹, Evelyn GP Ong¹, Venetia Horn², Nigel J. Klein³, Agostino Pierro³
¹*Institute of Child Health, London, United Kingdom*, ²*Great Ormond Street Hospital, London, United Kingdom*, ³*Institute of Child Health and Great Ormond Street Hospital, London, United Kingdom*
- 8 PARENTERAL FISH OIL AS MONOTHERAPY PREVENTS ESSENTIAL FATTY ACID DEFICIENCY IN PARENTERAL NUTRITION DEPENDENT PATIENTS**
Vincent E. de Meijer, MD, MSc, Hau D. Le, MD, Jonathan A. Meisel, MD, Kathleen M. Gura, PharmD, Mark Puder, MD, PhD
Children's Hospital Boston, Boston, MA, USA

Program in Detail

Friday, May 29 (continued)

- 9 REDUCTION IN STANDARD INTRAVENOUS FAT EMULSIONS FOR PATIENTS WITH PARENTERAL NUTRITION CHOLESTASIS: AN EFFECTIVE MODE OF TREATMENT**
Mary P. Cober, PharMD, Daniel H. Teitelbaum, MD, Ghassan Killu, PharMD Candidate, Allison Weber, PharMD Candidate, Shaun M. Kunisaki, MD, Kathleen B. Welch, MS, MPH
University of Michigan, Ann Arbor, MI, USA
- 10 THE RELATIONSHIP BETWEEN BIOPSY PROVEN PARENTERAL NUTRITION ASSOCIATED LIVER FIBROSIS AND BIOCHEMICAL CHOLESTASIS IN CHILDREN WITH SHORT BOWEL SYNDROME**
Shimae C. Fitzgibbons¹, Brian A. Jones, MD¹, Melissa Hull, MD¹, Debora Duro, MD², Christopher Duggan, MD², Dana Boctor, MD⁴, David L. Sigalet, MD³, Tom Jaksic, MD, PhD¹
¹*Center for Advanced Intestinal Rehabilitation, Department of Surgery, Children's Hospital Boston, Boston, MA, USA*, ²*Center for Advanced Intestinal Rehabilitation, Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA*, ³*Department of Gastroenterology, Alberta Children's Hospital, Calgary, AB, Canada*
- 11 REVERSAL OF INTESTINAL FAILURE ASSOCIATED LIVER DISEASE IN INFANTS AND CHILDREN ON PARENTERAL NUTRITION: EXPERIENCE WITH 88 PATIENTS AT A REFERRAL CENTER FOR INTESTINAL REHABILITATION**
Robert A. Cowles, MD, Kara A. Ventura, DNP, Mercedes Martinez, MD, Steven J. Lobritto, MD, Patricia A. Harren, DNP, Susan Brodliie, RD, Joanne Carroll, CPNP, Dominique M. Jan, MD
Morgan Stanley Children's Hospital of New York-Presbyterian and Columbia University Medical Center, New York, NY, USA

9:00 a.m. – 10:00 a.m.	Robert E. Gross Lecture Stanley B. Prusiner, MD	<i>Grand Atlantic 2 & 3</i>
10:00 a.m. – 10:30 a.m.	Refreshment Break	<i>Atlantic Foyer</i>
10:30 a.m. – Noon	Scientific Session II: Congenital Anomalies	<i>Grand Atlantic 2 & 3</i>

Moderators:

Kerilyn Nobuhara, MD; Karl Sylvester, MD

Educational Objectives:

Attendees will gain knowledge about:

- Management of infants with CDH.
- Prenatal treatments for CCAM and twin-twin transfusion syndrome.
- Long term outcomes for patients with cloacal exstrophy and anorectal malformations.
- Surgical options for laryngotracheal stenosis and long-gap esophageal atresia.

Program in Detail

Friday, May 29 (continued)

- 12 PATCH REPAIR IS ASSOCIATED WITH SIGNIFICANT MORBIDITY AND MORTALITY IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH)**
Mary E. Brindle¹, Elizabeth Oddone¹, Erik D. Skarsgard, MD, FRCSC, FACS, FAAP², The Canadian Perinatal Surgical Network¹
¹Alberta Children's Hospital, Calgary, AB, Canada, ²University of British Columbia, Vancouver, BC, Canada
- 13 ESTABLISHMENT OF PRE-TREATMENT BLOOD GAS TARGETS IMPROVES SURVIVAL IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH)**
Mary E. Brindle, MD, FRCPSC¹, Irene W. Ma, MD, MSc(Epi), FRCPC², Erik D. Skarsgard, MD, FRCSC, FACS, FAAP², The Canadian Perinatal Surgical Network¹
¹Alberta Children's Hospital, Calgary, AB, Canada, ²University of British Columbia, Vancouver, BC, Canada
- 14 PRENATAL PULMONARY HYPERTENSION INDEX (PPHI): NOVEL PRENATAL PREDICTOR OF SEVERE POSTNATAL PULMONARY ARTERY HYPERTENSION IN ANTENATALLY DIAGNOSED CONGENITAL DIAPHRAGMATIC HERNIA.**
Jose F. Vuletin, MD, James Cnota, MD, Beth Kline-Fath, MD, Foong-Yen Lim, MD, Beth Haberman, MD, Paul Kingman, MD, Jason Frischer, MD, Timothy Crombleholme, MD
Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA
- 15 PRENATAL STEROIDS FOR CONGENITAL CYSTIC ADENOMATOID MALFORMATIONS**
Patrick Curran, MS, Eric Jelin, MD, Larry Rand, MD, Shinjiro Hirose, MD, Vickie Feldstein, MD, Ruth Goldstein, MD, Hanmin Lee, MD
University of California, San Francisco, San Francisco, CA, USA
- 16 SURGICAL MANAGEMENT OF LARYNGOTRACHEAL STENOSIS IN CHILDREN**
Jaime Penchyna, MD, Hiram Alvarez -Neri, MD, Gerardo Blanco-Rodriguez, MD, Juan D. Porras-Hernandez, MD, Gustavo Teyssier-Morales, MD
Hospital Infantil de Mexico, Mexico
- 17 EXTRA-THORACIC ESOPHAGEAL ELONGATION (KIMURA'S TECHNIQUE): A FEASIBLE OPTION FOR THE TREATMENT OF PATIENTS WITH COMPLEX ESOPHAGEAL ATRESIA**
Natalia Tamburri, MD, Pablo Laje, MD, Mariano Bognione, MD, Marcelo Martinez Ferro, MD
National Pediatric Hospital JP Garrahan, Buenos Aires, Argentina.
- 18 PERINATAL PREDICTORS OF OUTCOME IN GASTROSCHISIS**
Jessica L.A. Mills, MD¹, Yi Lin, BSc², Ying MacNab, PhD², Erik D. Skarsgard, MD¹
¹Department of Surgery, BC Children's Hospital and University of British Columbia, Vancouver, BC, Canada, ²Department of Health Care and Epidemiology, University of British Columbia, Vancouver, BC, Canada

Program in Detail

Friday, May 29 (continued)

- 19 ANTIBIOTIC STRATEGIES AND INFECTIOUS COMPLICATIONS IN THE MANAGEMENT OF GASTROSCHISIS**
Robert J. Baird¹, Erik D. Skarsgard², Pramod Puligandla¹, Jean-Martin Laberge¹.
¹McGill University, Montreal, QC, Canada, ²University of British Columbia, Vancouver, BC, Canada
- 20 MEGARECTUM AFTER SURGERY FOR ANORECTAL MALFORMATIONS (ARM)**
Sathyaprasad C. Burjonrappa, MD, FRCS(Ed), Sarah Bouchard, Stéphanie Lapierre, Arié Bensoussan, MD
Sainte-Justine Hospital, Montreal, QC, Canada
- 21 GASTROINTESTINAL RAMIFICATIONS OF THE CLOACAL EXSTROPHY COMPLEX: A 24 YEAR EXPERIENCE**
David E. Sawaya, MD¹, John Gearhart, MD², Paul Colombani, MD², Seth Goldstein, MD², Rupa Seetharamaiah¹, Kristina Suson².
¹University Mississippi Medical Center, Jackson, MS, USA, ²Johns Hopkins Hospital, Baltimore, MD, USA
- 22 TWIN-TWIN TRANSFUSION SYNDROME: LIFE AFTER TRIALS**
Shinjiro Hirose, MD, Patrick Curran, MS, Vickie Feldstein, MD, Larry Rand, MD, Hanmin Lee, MD
UCSF, San Francisco, CA, USA

Noon – 1:00 p.m.	Jay & Margie Grosfeld Lecture	<i>Grand Atlantic 2 & 3</i>
	Michael T. Longaker, MD	
1:00 p.m. – 1:30 p.m.	Refreshment Break and Box Lunch Pick Up	<i>Atlantic Foyer</i>
1:30 p.m. – 2:30 p.m.	Surgical Simulators	<i>Grand Atlantic 2 & 3</i>
	Application of neonatal training boxes and virtual reality training (VRT) devices in education of pediatric endoscopic surgery	

Moderator:

Timothy Kane, MD

Educational Objectives:

- Have insight of the specific virtues of the neonatal training box.
- Have an overview of different VRT devices currently available.
- Understand the possibilities and limitations of the different training devices.
- Be able to determine if the neonatal box and VRT is an option for training endoscopic surgery in your institution.
- Be able to define the best choice for your specific purposes .

Invited Lecturers:

David C. van der Zee, MD, PhD¹, Milissa McKee, MD², Karen Diefenbach, MD²
¹Wilhelmina Children's Hospital, Utrecht, The Netherlands, ²Yale – New Haven Children's Hospital, New Haven, CT, USA

2:30 p.m. – 4:00 p.m.	Benjy Brooks Society	<i>Flamboyant</i>
3:00 p.m.	Golf Tournament	<i>Arthur Hills Golf Course</i>
3:00 p.m.	Tennis Tournament	<i>Tennis Courts</i>
3:00 p.m.	Kayak/Snorkel Tour	<i>Hotel Main Entrance</i>
5:00 p.m. – 6:30 p.m.	<i>Journal of Pediatric Surgery</i> Reception	<i>Las Croabas</i>
6:30 p.m. – 8:00 p.m.	New Member Reception	<i>Magnolia Ballroom</i>

Program in Detail

Saturday, May 30

6:30 a.m. – 8:00 a.m.	Member Business Meeting and Breakfast	<i>Grand Atlantic 2 & 3</i>
6:30 a.m. – 4:00 p.m.	Registration Open	<i>Atlantic Foyer</i>
7:00 a.m. – 8:00 a.m.	Continental Breakfast for Non-Members	<i>Atlantic Foyer</i>
7:00 a.m. – 11:00 a.m.	Exhibits Open	<i>Atlantic Foyer</i>
8:00 a.m. – 9:30 a.m.	Scientific Session III: Trauma, Transplant, Oncology and Critical Care	<i>Grand Atlantic 2 & 3</i>

Moderators:

Dennis Vane, MD; Stephen Shochat, MD

Educational Objectives:

This session will allow participants to:

- Identify management issues for children with solid organ and blunt intestinal injuries.
- Describe outcomes of patients with intestinal failure awaiting intestinal transplant.
- Cite the benefits of new therapies and predictors of response for patients with hepatoblastoma, neuroblastoma, Wilms tumor and rhabdomyosarcoma.
- Appreciate a possible drawback of ethanol lock treatment for central venous catheters.

23 A SINGLE INSTITUTION SERIES OF 800 CHILDREN WITH ABDOMINAL SOLID ORGAN INJURIES

Jordan R. Gutweiler, MD, David P. Mooney, MD, MPH
Children's Hospital Boston, Boston, MA, USA

24 DELAY IN DIAGNOSIS AND TREATMENT OF BLUNT INTESTINAL INJURY DOES NOT ADVERSELY AFFECT PROGNOSIS

Robert W. Letton¹, Veronica Worrell, PhD¹, Blunt Intestinal Injury Study Group
APSA Committee on Trauma
¹*Children's Hospital of Oklahoma, Oklahoma City, OK, USA*

25 OUTCOMES IN CHILDREN WITH INTESTINAL FAILURE FOLLOWING LISTING FOR INTESTINAL TRANSPLANT

Oliver B. Lao, MD¹, Patrick J. Healey, MD¹, James D. Perkins, MD², Jorge D. Reyes, MD¹, Adam B. Goldin, MD, MPH¹
¹*Seattle Childrens, Seattle, WA, USA*, ²*University of Washington, Seattle, WA, USA*

27 IMPROVED OUTCOME OF LIVER TRANSPLANTATION FOR UNRESECTABLE HEPATOBLASTOMA

Jaimie D. Nathan, Greg M. Tiao, James I. Geller, Lars M. Wagner, Mike A. Leonis, John C. Bucuvalas, Kathleen M. Campbell, Maria H. Alonso, Frederick C. Ryckman
Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA

Program in Detail

Saturday, May 30 (continued)

- 28** **DEFINING HEPATOBLASTOMA RESPONSIVENESS TO NEOADJUVANT THERAPY AS MEASURED BY TUMOR VOLUME AND SERUM ALPHA-FETOPROTEIN KINETICS**
Harold N. Lovvorn, III, MD, Melissa Hilmes, MD, Dan Ayers, Zhiguo Zhao, Pinki Prasad, MD, Myrick Shinall, Barry Berch, MD, James A. O'Neill, Jr., MD, Wallace W. Neblett, III, MD
Vanderbilt University Children's Hospital, Nashville, TN, USA
- 29** **TUMOR VOLUME AS A PREDICTOR OF ONCOLOGIC OUTCOME IN CHILDREN WITH INTERMEDIATE RISK RHABDOMYOSARCOMA**
Norbert Garcia-Henriquez, MD, David A. Rodeberg, MD
University of Pittsburgh, Pittsburgh, PA, USA
- 30** **IMPROVED SURVIVAL FOR CHILDREN WITH NEUROBLASTOMA AND WILMS TUMOR AT HIGH AND LOW VOLUME COG MEMBER HOSPITALS**
Juan C. Gutierrez, MD, Michael C. Cheung, MD, Ying Zhuge, MD, Leonidas G. Koniaris, MD, Juan E. Sola, MD
University of Miami, Miami, FL, USA
- 31** **MALIGNANCY AND OVARIAN TORSION: DO WE PRACTICE WHAT WE PREACH?**
Sarah C. Oltmann, MD¹, Anne Fischer, MD, PhD¹, Robert Barber, RN², Barry Hicks, MD¹, Nilda Garcia, MD¹
¹University of Texas Southwestern Medical Center, Dallas, TX, USA, ²Childrens Medical Center, Dallas, TX, USA
- 32** **ADVERSE CATHETER-RELATED EVENTS IN A PHASE I TRIAL OF PROPHYLACTIC ETHANOL ADMINISTRATION TO PREVENT CENTRAL VENOUS CATHETER INFECTIONS**
Mark L. Kayton, Edward G. Garmey, MD, Nicole M. Ishill, MS, Nai-Kong V. Cheung, MD, Brian H. Kushner, MD, Kim Kramer, MD, Shakeel Modak, MD, Carol Rossetto, RN, MSN, CPNP-AC, CPON, Courtney Hennelly, RN, MS, APN-BC, Melissa Parra, RN, CPNP-AC, Shoshana Rosenberg, MPH, Olga Santoro, BS, Glenn Heller, PhD, Michael P. La Quaglia, MD
Memorial Sloan-Kettering Cancer Center, New York, NY, USA
- 33** **PUSHING THE BOUNDARIES OF ECLS: OUTCOMES IN <34 WEEK EGA NEONATES**
Anne C. Kim, MD, MPH, Kimberly W. McCrudden, MD, Ankur Rana, MD, Robert Drongowski, MS, Robert H. Bartlett, MD, Ronald B. Hirschl, MD, George B. Mychaliska, MD
University of Michigan-Ann Arbor, Ann Arbor, MI, USA

9:30 a.m. – 10:30 a.m.	Journal of Pediatric Surgery Lecture	<i>Grand Atlantic 2 & 3</i>
	Haile T. Debas, MD	
10:30 a.m. – 11:00 a.m.	Refreshment Break	<i>Atlantic Foyer</i>
11:00 a.m. – 12:15 p.m.	Scientific Session IV:	<i>Grand Atlantic 2 & 3</i>
	Practice Issues, Surgical Education & Outcomes	

Moderators:

John R. Gosche, MD; Daniel von Allmen, MD

Program in Detail

Saturday, May 30 (continued)

Educational Objectives:

Participants in this session will be informed about:

- How insurance status affects the care of infants with congenital anomalies.
- Ambulatory wound care services for pediatric patients.
- New methods for pediatric surgical resident education.
- Long-term outcomes associated with CDH and high imperforate anus.

34 UNIVERSITY PEDIATRIC SURGERY: BENCHMARKING PERFORMANCE

Charles J. Stolar, MD¹, Aileen A. Alapan, MPH², Solomon A. Torres, MPA²

¹Morgan Stanley Children's Hospital of New York, Columbia University Medical Center, New York, NY, USA, ²Columbia University, College of Physicians and Surgeons, New York, NY, USA

35 A PEDIATRIC AMBULATORY WOUND SERVICE (PAWS): A NOVEL APPROACH IN WOUND MANAGEMENT

Brian T. Bucher, MD, Jennifer Seigel, Elyn Rosenblum, Charlene Nesslein, Sundeep Keswani, Robert Foglia, Patrick Dillon, MD, Brad Warner, MD, Martin S. Keller, MD

Washington University School of Medicine, St. Louis, MO, USA

36 THE PARADOXICAL EFFECT OF MEDICAL INSURANCE ON DELIVERY OF CARE FOR INFANTS WITH CONGENITAL ANOMALIES

Loren Berman, MD, Marjorie Rosenthal, MD, R. Lawrence Moss, MD

Yale School of Medicine, New Haven, CT, USA

37 LAPAROSCOPIC PYLOROMYOTOMY IS CHEAPER THAN OPEN PYLOROMYOTOMY: ECONOMIC ANALYSIS OF A RANDOMISED CONTROLLED TRIAL

Emma V. Carrington¹, Simon Eaton¹, Nigel J. Hall¹, Maurizio Pacilli¹, David P. Drake², Joe I. Curry², Edward M. Kiely², Paolo De Coppi², Agostino Pierro²

¹Institute of Child Health, London, United Kingdom, ²Institute of Child Health and Great Ormond Street Hospital, London, United Kingdom

38 DEVELOPMENT AND PRELIMINARY VALIDATION OF A NEW PEDIATRIC FUNDAMENTALS OF LAPAROSCOPIC SURGERY SIMULATOR

Georges Azzie¹, J. Ted Gerstle¹, David Lasko¹, Jessica Green², Oscar Henao², Monica Farcas², Allan Okrainec²

¹Hospital for Sick Children, Toronto, ON, Canada, ²Toronto Western Hospital-University Health Network, Toronto, ON, Canada

39 FACE AND CONTENT VALIDITIES OF A LOW FIDELITY LAPAROSCOPIC PYLOROMYOTOMY SIMULATION

Ana Ruzic, MD, James Hoskins, Margaret A. Plymale, RN MSN, Sean Skinner, Dan Davenport, PhD, Joseph A. Iocono, MD

University of Kentucky, Lexington, KY, USA

Program in Detail

Saturday, May 30 (continued)

40 LONG-TERM SURGICAL OUTCOMES IN 93 SURVIVORS OF CONGENITAL DIAPHRAGMATIC HERNIA (CDH)
Tim Jancelewicz, MD, Lan T. Vu, MD, Barbara J. Bratton, PNP, Hanmin Lee, MD, Diana L. Farmer, MD, Michael R. Harrison, MD, Doug N. Miniati, MD, Tippi C. Mackenzie, MD, Shinjiro Hirose, MD, Kerilyn K. Nobuhara, MD
University of California, San Francisco, San Francisco, CA, USA

41 LONG TERM FUNCTIONAL OUTCOME AND QUALITY OF LIFE IN PATIENTS WITH HIGH IMPERFORATE ANUS
Mohamed Hashish¹, Hamada H. Dawoud², Roland B. Hirschl¹, Steven W. Bruch¹, Akram M. ElBatarny², George B. Mychaliska¹, Robert Drongowski¹, Peter F. Ehrlich¹, Sayed Z. Hassaballa², Nagi I. El-Dosuky², Daniel H. Teitalbaum¹
¹University of Michigan, Ann Arbor, MI, USA, ²Tanta University, Tanta, Egypt

12:15 p.m. – 12:30 p.m.	Refreshment Break and Box Lunch Pick Up	<i>Atlantic Foyer</i>
12:30 p.m. – 1:15 p.m.	APSA Foundation Scholar Douglas Miniati, MD	<i>Grand Atlantic 2 & 3</i>
1:15 p.m. – 2:15 p.m.	International Guest Lecture Marcelo Martinez Ferro, MD	<i>Grand Atlantic 2 & 3</i>
2:15 p.m. – 2:30 p.m.	Refreshment Break	<i>Atlantic Foyer</i>
2:30 p.m. – 4:30 p.m.	Scientific Session V: Surgical Innovation & Basic Science	<i>Grand Atlantic 2 & 3</i>

Moderators:

Elizabeth A. Beierle, MD; Keith E. Georgeson, MD

Educational Objectives:

Participants will be updated on:

- Approaches in development for management of pectus excavatum and short bowel syndrome.
- The application of proteomics to identify markers of progressive NEC.
- Investigations into the effects of exogenous factors on survival in models of NEC and intestinal ischemia.
- The effects of lipid emulsions and growth factor in an animal model of TPN induced liver disease.
- Efficacy of secreted midkine and oncolytic viruses as possible adjuncts to therapy for neuroblastoma.

42 MAGNETIC MINI-MOVER PROCEDURE FOR PECTUS EXCAVATUM II: AN FDA SPONSORED TRIAL (IDE #G050196)
Michael R. Harrison, MD, Patrick F. Curran, MS, Richard J. Fechter, BS, Darrell Christensen, MA, Shinjiro Hirose, MD
University of California, San Francisco, San Francisco, CA, USA

43 A NEW VIDEOSCOPIIC DEVICE TO AVOID CARDIAC INJURY IN MINIMALLY INVASIVE PECTUS EXCAVATUM REPAIR: THE PECTOSCOPE
Hyung Joo Park, Jongho Cho, In Sung Lee, Kwang Taik Kim, Young Ho Choi
Korea University Medical Center, Ansan, Republic of Korea

Program in Detail

Saturday, May 30 (continued)

- 44** **INTESTINAL LENGTHENING USING AN IMPLANTABLE SPRING: A NOVEL APPROACH FOR THE TREATMENT OF SHORT BOWEL SYNDROME**
Shant Shekherdian, MD, Mohanchandra K. Panduranga, PhD, Gregory P. Carman, PhD, James C.Y. Dunn, MD, PhD
UCLA Medical Center, Los Angeles, CA, USA
- 45** **EVALUATION OF A NOVEL SMALL DIAMETER TISSUE ENGINEERED ARTERIAL GRAFT**
Tamar L. Mirensky, MD, Corey Fein, Gerard Nguyen, BSE, Tai Yi, MD, Narutoshi Hibino, MD, PhD, Gustavo Villalona, MD, Edward Mcgillicuddy, MD, Toshiharu Shinoka, MD, PhD, Christopher Breuer, MD
Yale University School of Medicine, New Haven, CT, USA
- 46** **TWO-STAGE BASILIC VEIN TRANSPOSITION A NEW APPROACH FOR PEDIATRIC DIALYSIS ACCESS**
Anne C. Kim, MD/MPH¹, Sean McLean, MD¹, Alissa M. Swearingen, MD², Kathleen D. Graziano, MD³, Ronald B. Hirschl, MD¹
¹*University of Michigan-Ann Arbor, Ann Arbor, MI, USA*, ²*Phoenix Integrated Surgical Residency, Phoenix, AZ, USA*, ³*Phoenix Children's Hospital, Phoenix, AZ, USA*
- 47** **PROTEOMICS FOR THE IDENTIFICATION OF CANDIDATE BIOMARKERS OF PROGRESSIVE NEC IN HUMAN INFANTS**
Karl G. Sylvester¹, John Whitin, PhD¹, Tom Yu, BS¹, Gigi Liu, BS¹, Joyce Simpson, RN², Mary Brandt, MD³, Fizan Abdulla, MD, PhD⁴, Chris Duggan, MD⁵, Tom Jaksic, MD, PhD⁵, James Dunn, MD, PhD⁶, Mary Cay Harris, MD⁷, Michael Posencheg, MD⁸, R. Larry Moss, MD², Harvey Cohen, MD, PhD¹
¹*Stanford University, Stanford, CA, USA*, ²*Yale University, New Haven, CT, USA*, ³*Baylor University, Houston, TX, USA*, ⁴*Johns Hopkins University, Baltimore, MD, USA*, ⁵*Boston Children's Hospital, Boston, MA, USA*, ⁶*UCLA and Mattel Children's Hospital, Los Angeles, CA, USA*, ⁷*Children's Hospital of Philadelphia, Philadelphia, PA, USA*, ⁸*Hospital of the University of Pennsylvania, Philadelphia, PA, USA*
- 48** **HB-EGF PRESERVES INTESTINAL MICROVASCULATURE AND STRUCTURAL ARCHITECTURE IN RAT PUPS WITH EXPERIMENTAL NECROTIZING**
Xiaoyi Yu, MD, MS, Andrei Radulescu, MD, PhD, Gail E. Besner, MD
Nationwide Children's Hospital, Columbus, OH, USA
- 49** **EXOGENOUS TREFOIL FACTOR 2 MARKEDLY IMPROVES SURVIVAL AFTER INTESTINAL ISCHEMIA.**
Bruce Sprague, BS, Cynthia A. Gingalewski, MD
Children's National Medical Center, Washington, DC, USA

Program in Detail

Saturday, May 30 (continued)

- 50** **FOUR INTRAVENOUS LIPID EMULSIONS AND THEIR EFFECTS ON HEPATIC STEATOSIS IN A MURINE MODEL**
Jonathan A. Meisel, MD¹, Hau D. Le, MD¹, Vincent E. de Meijer, MD, MSc¹, Vania Nose, MD, PhD², Kathleen Gura, PharmD¹, Mark Puder, MD, PhD¹
¹Children's Hospital Boston, Boston, MA, USA, ²Brigham and Women's Hospital, Boston, MA, USA
- 51** **DOCOSAHEXAENOIC ACID AND ARACHIDONIC ACID PREVENT ESSENTIAL FATTY ACID DEFICIENCY AND HEPATIC STEATOSIS IN A MURINE MODEL OF PARENTERAL NUTRITION-ASSOCIATED LIVER DISEASE.**
Hau D. Le, MD¹, Jonathan A. Meisel, MD¹, Vincent E. de Meijer, MD, MSc¹, Kathleen Gura, PharmD¹, Katherine Novak, BS¹, Vania Nose, MD, PhD², Bruce R. Bistrrian, MD, PhD³, Mark Puder, MD, PhD¹
¹Children's Hospital Boston, Boston, MA, USA, ²Brigham and Women's Hospital, Boston, MA, USA, ³Beth Israel Deaconess Medical Center, Boston, MA, USA
- 52** **GROWTH FACTOR MODULATION OF HEPATIC INFLAMMATION: A NOVEL APPROACH TO THE MANAGEMENT OF TPN ASSOCIATED LIVER DISEASE**
Keith A. Thatch, MD¹, Michael S. Katz, MD¹, Marian M. Haber, MD², Marshall Z. Schwartz, MD¹
¹St. Christopher's Hospital for Children/Drexel University College of Medicine, Philadelphia, PA, USA, ²Drexel University College of Medicine, Philadelphia, PA, USA
- 53** **A MOUSE MODEL OF POST-PULLTHROUGH HIRSCHSPRUNG ASSOCIATED ENTEROCOLITIS**
Philip K. Frykman, MD, PhD, Zhi Cheng, MD, Lifu Zhao, MD, MSc, Deepti Dhall, MB, BS
Cedars-Sinai Medical Center, Los Angeles, CA, USA
- 54** **EFFECTS OF NOTCH4 ON LUNG VASCULAR REMODELING**
Eric Jelin, MD, Jennifer Ng, BS, Rong Wang, PhD, Douglas Miniati, MD
University of California, San Francisco, San Francisco, CA, USA
- 55** **SECRETED MIDKINE CONFERS DOXORUBICIN RESISTANCE IN HUMAN SKN-SH NEUROBLASTOMA CELLS BOTH IN VITRO AND IN VIVO**
Mary Beth Madonna, Sandra Clark, MS, Baojun Chang, MD, PhD, Xin Zheng, MD, PhD, Janette L. Holub, MD, MPH, Fei Chu, MD, PhD
Children's Memorial Hospital, Chicago, IL, USA
- 56** **THE UTILITY OF ONCOLYTIC VIRUSES AGAINST NEUROBLASTOMA**
Nicole Redding¹, Steven Robbins, PhD², Peter Forsyth, MD², Grant McFadden, PhD³, John Bell, PhD⁴, Paul Beaudry, MD, MSc¹
¹Alberta Childrens Hospital, Calgary, AB, Canada, ²Southern Alberta Cancer Research Institute, Calgary, AB, Canada, ³University of Florida, Gainesville, FL, USA, ⁴Ottawa Health Research Gainesville, FL, USA, ⁴Ottawa Health Research Institute, Ottawa, ON, Canada

6:45 p.m. – 7:30 p.m.

President's Reception

Atlantic Foyer

7:30 p.m. – 10:00 p.m.

President's Banquet

Grand Atlantic Ballroom

10:00 p.m.

Annual Meeting Adjourns

2009 Exhibitor and Supporter Directory

American College of Surgeons

Booth Number: 4

633 N. Saint Clair Street
Chicago, IL 60611
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Fax: +1-312-202-5061
E-mail: cfisher@facs.org
Web site: www.facs.org

The American College of Surgeons National Surgical Quality Improvement Program (ACS NSQIP) introduces ACS NSQIP Pediatric, the first multispecialty outcomes-based program in pediatric surgery. ACS NSQIP Pediatric, currently in its pilot phase, has been designed to collect surgical data and 30-day outcomes on pediatric patients undergoing major surgical procedures. Please visit our booth to learn about the program including hospital eligibility, pilot project results, and the application process.

American Pediatric Surgical Association Foundation

Booth Number: 5

702 Barnhill Drive, Suite 2500
Indianapolis, IN 46202
Phone: +1-317-274-5710
Fax: +1-317-274-5777
E-mail: jgrosfel@iupui.edu
Web site: www.eapsa.org

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American Pediatric Surgical Nurses Association

Booth Number: 8

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Columbus, OH 43235
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Fax: +1-614-722-3903
E-mail: jackie.cronau@
nationwidechildrens.org
Web site: www.apsna.org.

APNSA membership spans the spectrum of the care of children requiring surgical care from trauma to burns to injury prevention; from inpatient to ambulatory surgery;

www.eapsa.org

inpatient and out patient care; neonatal through adolescent; NICO and PICU; the inpatient units: PACU; registered nurses in advanced practice.

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Booth Number: 1

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Jacksonville, FL 32218
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E-mail: kelly.duke@biomet.com
Web site: www.biometmicrofixation.com

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CHERUBS - The Association of Congenital Diaphragmatic Hernia Research, Awareness, and Support

Booth Number: 19

3650 Rogers Road, #290
Wake Forest, NC 27587
Phone: +1-815-610-0128

E-mail: info@cherubs-cdh.org
Web site: www.cherubs-cdh.org

CHERUBS was founded in 1995 to support families of children born with Congenital Diaphragmatic Hernia. CHERUBS is the world's largest CDH organization, with over 2800 members in 38 countries. Membership for parents is free and our organization is run solely by volunteers and funded through donations from members and the public. CHERUBS advocates for research of CDH by promoting awareness and searching for the cause, prevention and best treatment of Congenital Diaphragmatic Hernia.

The Children's Medical Center

Booth Number: 7

One Children's Plaza
Dayton, OH 45404
Phone: +1-937-641-3450
Fax: +1-937-641-5400

Email: graybillm@childrensdayton.org
Web site: www.childrensdayton.org

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Ethicon Endo-Surgery

Booth Number: 17

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Cincinnati, OH 45242
Phone: +1-513-337-3498
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E-mail: daltman1@its.jnj.com
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Danville, PA 17822-2428
Phone: +1-570-214-9725
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Supporter: Traditional

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2009 Exhibitor and Supporter Directory (continued)

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Booth Number: 18

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Booth Number: 11

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Booth Number: 25

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Booth Number: 22

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V1

REDO TRANSANAL PULL-THROUGH FOR HIRSCHSPRUNG'S DISEASE

Belinda Dickie, MD, Marc A. Levitt, MD, Andrea Bischoff, Alberto Pena
Cincinnati Children's Hospital, Cincinnati, OH, USA

Purpose:

Reoperative surgery after a prior pull-through for Hirschsprung's disease may be indicated for recurrent obstructive symptoms, persistent enterocolitis, chronic distention and failure to thrive. These symptoms can be caused by mechanical factors such as a stricture, stenotic Soave cuff, a mega rectal Duhamel pouch, or a dilated distal pull-through segment. Aganglionic or transition zone bowel at the distal pull-through may also be causative factors. A transanal resection, with a Swenson-like approach is a useful option in such cases.

Methods:

We present a case of a three year old boy with Hirschsprung's disease. He had a previous laparoscopic assisted Soave pull-through and presented one year after surgery with severe constipation and recurrent enterocolitis. A contrast enema revealed a dilated distal pull-through segment. Examination under anesthesia demonstrated a stricture at the anastomosis and repeat biopsy showed transition zone bowel. A redo transanal Swenson-like pullthrough was performed in the prone position. No laparotomy or laparoscopy was required. A coloanal anastomosis was done with nondilated, ganglionated bowel. The patient recovered uneventfully. He was tolerating a normal diet and stooling normally on discharge.

Conclusions:

Patients with Hirschsprung's disease presenting with obstructive symptoms following their operation need to be carefully evaluated to determine the etiology of their complication. In certain cases requiring reoperation, a redo transanal pull-through is a feasible and effective approach to surgically manage these patients.

Notes:

THORACOSCOPIC ANATOMIC LINGULECTOMY FOR CHRONIC SEGMENTAL BRONCHIAL OBSTRUCTION IN A CHILD

Steven S. Rothenberg¹, Marzena Krawiec²

¹The Rocky Mountain Hospital for Children, Denver, CO, USA, ²National Jewish Medical and Research Center, Denver, CO, USA

Purpose:

To demonstrate the surgical techniques for performing an anatomic segmental lung resection in a child

Methods:

A 5 year old male with a two year history of chronic cough, reactive airway disease, and recurrent pulmonary infections of the left upper lobe was evaluated by high resolution CT scan and bronchoscopy. Both studies showed an inflammatory obstruction of the lingular segmental bronchus which was unresponsive to medical therapy and attempted dilations. CT scan showed bronchiectasis distal to the obstruction. The patient underwent a thoracoscopic lingulectomy

Results:

The surgery was accomplished successfully thoracoscopically. The lingular segmental bronchus, artery, and vein were each individually identified, sealed, and divided. A clear line of ischemic demarcation then allowed division of the parenchyma to the lingular from the rest of the upper lobe. The surgery was performed through 3 ports and took 2 hours. A chest tube was left in place for 36 hours and the patient was discharged on the second post-operative day. At 3 months follow-up the patient's cough is resolved and there have been no post-operative complications.

Conclusion:

While the need is rare, this video demonstrates that formal thoracoscopic segmentectomy is feasible and a viable option in patients with appropriate pulmonary pathology.

Notes:

PERCUTANEOUS ENDOSCOPICALLY ASSISTED REPAIR (PEAR) OF INGUINAL HERNIA

Justin D. Klein, MD, Christopher G. B. Turner, MD, Dario O. Fauza, MD, Russell W. Jennings, MD
Children's Hospital Boston, Boston, MA, USA

Conventional laparoscopic repair of inguinal hernias in children include 2 or 3 trocars and intraperitoneal suturing. Various large series have reported recurrence and complication rates of this form of repair to be no different from that of open repair. In addition, three previous studies have described different variations of a single-trocar method, with varying outcomes. This video illustrates our approach to single-trocar-based inguinal hernia repair, which employs a concurrent percutaneous groin puncture and high ligation of the inguinal ring by an extraperitoneal knot/suture performed with a commercially available wrist arthroscopy kit. Advantages of the technique demonstrated herein include direct bilateral visualization of the internal inguinal rings, negligible tissue trauma, minimal risk of injury to cord structures, very short operating time, and excellent cosmetic outcome. Our method is demonstrated on an otherwise healthy, former 35-week premature, 8-year-old boy. Percutaneous endoscopically assisted repair (PEAR) is a practical option for the treatment of pediatric inguinal hernias.

Notes:

LAPAROSCOPIC SLEEVE GASTRECTOMY FOR THE TREATMENT OF A MORBIDLY OBESE ADOLESCENT

Evan P. Nadler, MD, Manish Parikh, MD
New York University School of Medicine, New York, NY, USA

The patient is a now 15.5 year old female who was originally seen in October 2007 for evaluation for weight loss surgery. At the time of initial evaluation, she weighed 302 pounds at a height of 5'3" with a calculated BMI of 51. She has an extensive psychiatric history including schizoaffective disorder, pervasive developmental disorder, and mild mental retardation. Her cognitive functioning was determined to be similar to that of a second grade student. She had been obese since age 8 and her mother believes that the obesity was initiated by her psychiatric medications. Her obesity-related comorbid illnesses included polycystic ovarian syndrome only. She had been placed on metformin 6 months prior to her initial visit and her weight gain had plateaued. She was not able to lose any weight over the 6 months of metformin treatment. After 6 months of a failed medically-supervised weight loss attempt with our nutritionist, the decision was made to pursue weight loss surgery. Due to her mental capacity, she was deemed a poor candidate for laparoscopic adjustable gastric banding. After a lengthy discussion with the family regarding alternative weight loss procedures, the decision was made to perform a laparoscopic sleeve gastrectomy. The technical aspects of this procedure are detailed in the accompanying video. The procedure took approximately 2 hours and the patient was discharged on post-operative day 2. At her 6 week post-operative visit, she had lost 32 pounds from baseline.

Notes:

LAPAROSCOPIC REPAIR OF PELVIC ORGAN PROLAPSE

Christopher G. Turner, MD, Justin D. Klein, MD, Dario O. Fauza, MD, Russell W. Jennings, MD

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Pelvic organ prolapse may involve any combination of bladder, uterus, and/or rectum caudal displacement. It is very uncommon in children and has been associated with activities involving chronic pelvic floor strain, such as gymnastics and parachuting. This video illustrates the diagnostic evaluation and principles of laparoscopic repair of this disease in a 14-year-old girl, former gymnast, who presented with prolapse of the uterus, bladder, and rectum. The goal of this repair is to suspend the vagina, uterus, and rectum through the intrapelvic implantation of a bioprosthesis, which is anchored under moderate tension to three points, namely the perineal body, the posterior fornix-cervix junction and the sacral promontory. As a result of this fixation, the two mobile lower anchoring points (the perineal body and the posterior fornix-cervix junction) are suspended from the fixed upper anchoring point (the sacral promontory), thus preventing the prolapse. The laparoscopic, bioprosthesis-based repair is a viable option for the treatment of pelvic organ prolapse in children.

Notes:

LAPAROSCOPIC TYPE 1 CHOLEDOCHAL CYST EXCISION IN A 6 MONTH OLD INFANT

Donald C. Liu, MD, PhD, Grace Z. Mak, MD
University of Chicago, Chicago, IL, USA

The use of minimally-invasive techniques to treat choledochal cyst disease has been much reported in the adult literature. Laparoscopic choledochal cyst excision with the attending complex biliary reconstruction, however has not been well-described in children, particularly infants. We report of a six/month old female with a Type 1 choledochal cyst who underwent successful laparoscopic cyst excision and biliary reconstruction. Video documentation of the surgery with attention to the critical steps are included.

Notes:

HYBRID NOTES: INCISIONLESS INTRAGASTRIC STAPLED CYSTGASTROSTOMY OF A PANCREATIC PSEUDOCYST

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Purpose:

We present a case report of a novel hybrid natural orifice transluminal endoscopic surgery (NOTES). The operation performed was a trans-gastric cystgastrostomy with endoscopic guidance for a pancreatic pseudocyst. This operation was completed entirely through an existing gastrostomy site with no incisions avoiding the peritoneal cavity.

Methods:

This is a case of a 7-year-old boy with neurologic impairment from congenital HSV encephalitis who is tube fed. He had acute pancreatitis and developed a 9 cm pancreatic pseudocyst. The pseudocyst failed to resolve after 6 weeks and developed a mature wall. Due to a history of multiple abdominal surgeries and known abdominal adhesions, a minimally invasive approach that would avoid entering the peritoneal cavity was the desired approach. The technique involved a trans-oral endoscope for visualization and the use of the gastrostomy as access to the gastric lumen and pseudocyst. The pancreatic pseudocyst was stabilized with two T-fasteners and confirmed with needle aspiration under endoscopic visualization. The pseudocyst was then opened with the LigaSure™, Valleylab. The cystgastrostomy anastomosis was completed with an Endopath® ETS-Flex Articulating Linear Stapler/Cutter, Ethicon Endo-Surgery, Inc. The operation took less than 2 hours and was completed without an incision. Under the policies of the Human Research Protection Program, review of a single case is outside the scope of the definition of human subjects research and does not require IRB review and approval.

Results:

The patient did well post-operatively and had a dramatic reduction in size of the pancreatic pseudocyst to 3.5 cm by two weeks.

Conclusions:

We conclude that hybrid Notes:cystgastrostomy performed through an existing gastrostomy is an excellent approach for minimally invasive drainage of pancreatic pseudocysts.

Notes:

P1

OVER-EXPRESSION OF FGF10 INCREASES TRANSIT-AMPLIFYING CELLS BUT DOES NOT AFFECT INTESTINAL STEM CELLS

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Background:

We have previously shown that Fibroblast Growth Factor 10 (FGF10) plays a crucial role in colonic epithelial survival and proliferation during embryogenesis; and that it is a positive regulator of gut adaptation after massive small bowel resection. Over-expression of *Fgf10* in the gut epithelium using an inducible transgenic mouse model has shown an increase of proliferating cells and goblet cells in adult small intestine. Thus, we hypothesized that over-expression of *Fgf10* in intestinal epithelium would lead to an increase in intestinal stem cells.

Methods:

Villin-Cre;rtTA^{lox} driver mice were crossed with *Tet(O)Fgf10* responder mice to generate mice capable of inducible expression of *Fgf10* in the gut epithelium after exposure to doxycycline. Controls were age-matched wild type (WT) mice also given doxycycline. Immunohistochemistry was performed using doublecortin and CaM kinase-like-1 (DCAMKL-1) as a putative stem cell marker and proliferating cell nuclear antigen (PCNA) for identification of proliferating cells.

Results:

Over-expression of *Fgf10* in the intestinal epithelium does not change the number of intestinal stem cells as identified by DCAMKL-1; however, over-expression of *Fgf10* induces an increase of PCNA-positive cells. The location of DCAMKL-1 positive cells is consistent with published results, and double staining of DCAMKL-1 and PCNA demonstrates that DCAMKL-1 positive cells do not stain for PCNA.

Conclusions:

Our data show that FGF10 induces proliferation of the transit amplifying cells but does not alter the number of intestinal stem cells. Control of intestinal stem cell number is likely via other pathways such as Wnt signaling. FGF10 may have a therapeutic benefit in disease processes involving intestinal failure by increasing the number of transit amplifying progenitor cells. The fact that FGF10 does not alter the number of intestinal stem cells may be of clinical importance because of concern of tumorigenesis.

Notes:

INTESTINAL STEM CELLS ARE IDENTIFIED IN MATURE NATIVE AND TISSUE-ENGINEERED SMALL INTESTINE

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Objective:

Determine the temporal expression pattern of the newly described intestinal stem cell marker Doublecortin and CaM kinase-like-1 (DCAMKL-1) in developing native and tissue-engineered small intestine.

Background:

DCAMKL-1 is reported as an intestinal stem cell marker in PCNA negative cells, but its temporal expression in development is unknown. We now report a murine model of tissue-engineered small intestine (TESI), and the temporal expression pattern of DCAMKL-1 in native intestine and TESI.

Methods:

Organoid units, mesenchymal cell cores surrounded by epithelium, were derived from small intestines of 2-day-old mice and implanted on a biodegradable scaffold into the omentum of NOD/SCID mice. After 4 weeks, the resulting TESI was harvested and analyzed by histological techniques. In parallel, small intestines were harvested from mice at comprehensive time points and analyzed for the expression of DCAMKL-1/PCNA as well as markers for differentiated epithelial cells.

Results:

DCAMKL-1 staining is not identified until mice are 14 days old. Stem cells positive for DCAMKL-, and negative for PCNA were identified in mature TESI, even when derived from tissue that did not have DCAMKL-1 positive cells at harvest, but were not identified in the early formation of TESI. Mature TESI was positive for all other differentiated epithelial cell types including enteroendocrine, enterocytes, paneth, and goblet cells.

Conclusion:

The putative stem cell marker, DCAMKL-1 is a marker for the stem cells of the mature intestine, and therefore may not be useful in identifying progenitors in disease processes of immature intestine such as NEC. The presence of DCAMKL-1 positive cells along with every other intestinal cell types in tissue-engineered small intestine generated from 2 day-old pups indicates that TESI is identical to a mature native small intestine. Moreover, the presence of stem cells in tissue-engineered small intestine may indicate ongoing regenerative and therapeutic potential for pediatric disease.

Notes:

AMNIOTIC FLUID STEM CELLS ENHANCE SURVIVAL OF RATS WITH EXPERIMENTAL NEC RECEIVING HIGHER DOSE OF LIPOPOLYSACCHARIDE

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Purpose:

It is reported that bacterial lipopolysaccharide (LPS) in vitro is a strong activator of mesenchymal stem cells. It has previously been demonstrated that amniotic fluid stem (AFS) cells injected in a neonatal rat model of NEC integrate in the bowel wall and ameliorate morbidity and mortality. The aim of the present study was to evaluate whether the effect of AFS cells is modulated by the dose of LPS delivered in rats with experimental NEC.

Methods:

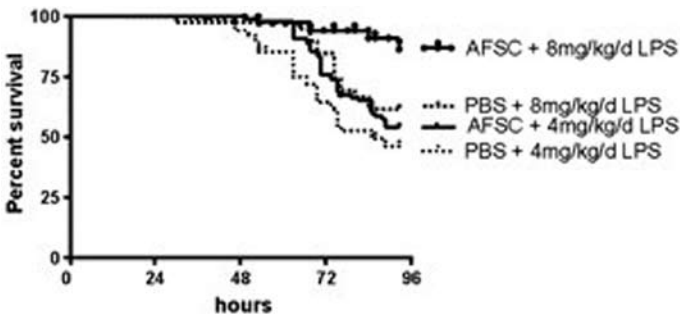
The study received both Ethics and Home Office approvals. AFS cells were obtained from GFP+ pregnant rats at E16 and expanded in culture. NEC was induced by gavage feeding of formula + hypoxia + oral LPS at two doses (4mg/kg/day or 8mg/kg/day). At 24 and 48h of life neonatal rats were intraperitoneally injected with either phosphate buffered saline (PBS) or AFS cells, and were randomised to 4 groups: A) NEC (LPS 4mg/kg/day) + PBS, B) NEC (LPS 8mg/kg/day) + PBS, C) NEC (LPS 4mg/kg/day) + AFS cells and D) NEC (LPS 8mg/kg/day) + AFS cells. At 96 hours of life, all survivors were sacrificed and survival rates were compared by logrank test for Kaplan-Meier curves.

Results:

Rats receiving AFSC and the higher dose of LPS, had a significantly higher survival than all other groups ($p < 0.0005$ vs. each other group; Figure). There were no differences in survival among the other 3 groups.

Conclusions:

AFS cells significantly increase survival (83%) in NEC rats given higher dose of LPS. Although this appears paradoxical, LPS may stimulate AFSC and enhance their effectiveness in preventing mortality in this model. Stem cell therapy may represent a new therapeutic option for children with NEC.



Notes:

STEM CELL THERAPY FOR PREMATURE LUNGS

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Purpose:

In prematurity, pulmonary hypoplasia accounts for 70% of neonatal mortality. This process is characterized by disrupted epithelial and vascular development. The ability to encourage stem cells to progress to alveolar epithelial/endothelial lineage may help treat this disease.

Methods:

Murine embryonic stem cells were subjected to ActivinA(20ng/ml) and FGF2(50ng/ml) to derive distal airway cells. Lung explants, harvested at E18, were cultured at an air-liquid interface and subjected to sFlt-1, a VEGF inhibitor to disrupt vascular development, reminiscent of pulmonary hypoplasia. Explants were separated into: I: explants +media; II: explant + sFlt-1; III: explants + sFlt-1 + undifferentiated embryonic stem cells; IV: explants + sFlt-1 + differentiated distal airway cells. Explants and stem cells were evaluated after 3 days. Morphologic assessment was performed by measuring mean cord length and branching morphogenesis. Phenotypic evaluation of the explant and stem cells was performed by immunohistochemistry of markers for epithelial differentiation (SpC), and endothelial differentiation (PECAM). Explant apoptosis was measured with the TUNEL assay. The supernatant was evaluated for VEGF by ELISA.

Results:

Treatment of the explant with sFlt-1 resulted in decreased mean cord length and increased apoptosis. These changes were ameliorated by culture with differentiated but not undifferentiated stem cells. Explants treated with differentiated distal airway cells displayed preservation of endothelial and epithelial cells. In addition, these differentiated stem cells maintained their differentiation in culture. ELISA data suggest increased VEGF in supernatant of explants treated with differentiated stem cells.

Conclusions:

Treatment with embryonic stem cells differentiated to distal airway cells ameliorated apoptosis and preserved morphology and differentiation in lung explants subjected to vascular disruption. Differentiated distal airway cells themselves also maintained phenotypic differentiation suggesting paracrine signaling perhaps, VEGF, between the explant and cells. These results indicate that cellular therapy with differentiated stem cells may represent a viable treatment option in pulmonary hypoplasia.

Notes:

ENGINEERING AN ARTIFICIAL ALVEOLAR MEMBRANE: A NOVEL CONTINUOUSLY-PERFUSED MODEL WITHIN MICROCHANNELS

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Purpose:

Pulmonary hypoplasia is a condition of the newborn that is characterized by underdeveloped lungs and adverse outcomes. One strategy in the treatment of patients with hypoplasia is to augment underdeveloped lungs using biocompatible artificial lung tissue. However, one central challenge in current pulmonary tissue engineering efforts remains development of a stable biomimetic alveolar-capillary membrane. The objective of our laboratory is to build a series of biomimetic microfluidic devices that specifically model the alveolar membrane. Current designs include a single-layer microchip that exposes alveolar cell types to controlled fluidic stimuli. A more advanced multi-layered device allows for alveolar cells to be cultured at an air-liquid interface while allowing constant media nourishment and waste removal, thus better mimicking the physiologic milieu of the alveolar-capillary interface. Both devices possess the benefit of parallel testing.

Methods:

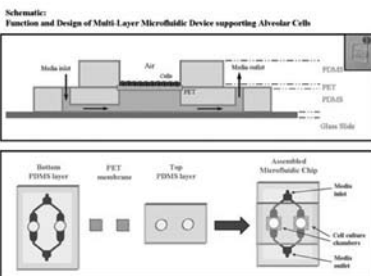
Microdevices were fabricated using soft lithography in a biocompatible transparent polymeric material, PDMS (polydimethylsiloxane), sealed covalently to glass. The multi-stage microdevice also integrated a suspended polyethylene terephthalate (PET) membrane connected via microfluidic channels to constant media and air access. Pulmonary endothelial (HMEC-1) and alveolar epithelial (A549) cell lines, along with fetal pulmonary cells harvested from Swiss Webster mice at day-18 gestational age, were studied under multiple hydrodynamic shear conditions and liquid-to-cell ratio regimes. Cultures were examined for cell viability, proliferation and lung surfactant production using the surfactant droplet test.

Results:

The single layer differential-flow microdevice allowed for a systematic determination of the optimal growth conditions of various lung-specific cell types in a microfluidic environment. Our devices showed a greater surfactant based decrease in surface tension of the alveolar hypophase compared to existing traditional air-interface transwell cultures.

Conclusions:

We have successfully developed biomimetic microfluidic devices that specifically allow stable alveolar cell growth at the air-liquid interface. This work may serve as the basis for an implantable artificial alveolar membrane.



Notes:

CONDITIONAL INACTIVATION OF EPITHELIAL C-MET SIGNALING CAUSES ABNORMAL LUNG GROWTH AND DEVELOPMENT

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Purpose:

The tyrosine kinase receptor c-Met is required for organogenesis, but the prenatal lethality of the null mutant limits the study of the role of c-Met in embryonic and postnatal lung development. Using transgenic mice, we inactivated the c-Met gene in lung epithelium in order to examine the function of this receptor during lung growth and development.

Methods:

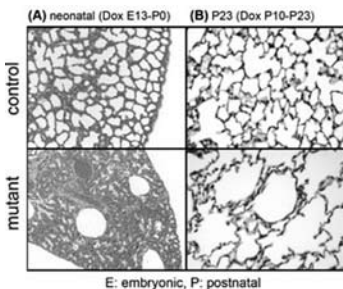
With IACUC approval, we generated triple transgenic mice in which rtTA is expressed in respiratory epithelial cells under the control of the human SP-C promoter. In the presence of doxycycline (Dox), rtTA binds to the (tetO)7-CMV promoter, which activates expression of Cre recombinase, resulting in deletion of a critical region of the floxed c-Met gene in lung epithelium. Pregnant dams were administered Dox from E13 to P0 (birth) to study the effects of c-Met inactivation on canalicular lung development. Postnatally, dams and pups were administered Dox during various time points to assess loss of function effects on alveolar lung development. Lung sections were stained and analyzed for morphology. Gene expression was measured using RT-PCR, and protein expression was assessed with immunohistochemistry. BrdU immunostaining was used to measure cell proliferation.

Results:

Inactivation of c-Met from E13-P0 was perinatal lethal due to respiratory failure, and expression of c-Met was 60% less than controls by RT-PCR. Grossly, lungs had visible air-filled cysts (**Figure 1A**), and histologically the cysts were lined by bronchiolar epithelium. The lungs were hypoproliferative as measured by a reduction in BrdU-positive cells. When Dox was administered from P10-P23, a patchy sub-pleural emphysematous phenotype was observed in mutant lungs and not in littermate controls (**Figure 1B**). Reduced phosphorylated c-Met protein expression was observed by immunohistochemistry in these lungs.

Conclusions:

Abnormal canalicular and alveolar lung development occurs with inactivation of the c-Met gene; further experiments are underway to determine the mechanisms underlying these phenotypes.



Notes:

INFLAMMATORY BOWEL DAMAGE IS AMELIORATED BY IN-UTERO REPAIR OF GASTROSCHISIS IN THE SHEEP MODEL

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Purpose:

Gastroschisis is associated with inflammatory changes in the exposed bowel which leads to intestinal dysmotility after postnatal repair. The insult is a combined effect of amniotic fluid exposure and mechanical constriction. We hypothesized that in-utero anatomic repair is possible in a sheep model, and that it may halt the inflammatory damage caused by both mechanisms

Methods:

Gastroschisis was surgically created in mid-gestation (day 75) in 8 sheep fetuses. On gestational day 100, 2 fetuses underwent open fetal gastroschisis repair, where the eviscerated bowel was returned to the peritoneal cavity and the abdominal wall was primarily closed. All fetuses were harvested at 135 days gestation. Amniotic fluid was sampled at each intervention, and small intestine was removed at final harvest for histology and immunohistochemistry.

Results:

Six fetuses survived the initial operation, and both fetuses that underwent gastroschisis repair survived to term. At 100 and 135 days gestation, the eviscerated bowel showed progressive signs of inflammation and peel development. The amniotic fluid exhibited an increase in digestive enzymes and a decrease in urinary markers, with a significant increase in lactic acid (see table). The gross and microscopic inflammatory changes in the gastroschisis bowel at 100 days gestation were completely resolved at term following in-utero repair. The amniotic fluid of the repair group exhibited intermediate values in digestive enzymes, urinary markers, and lactic acid. IHC for c-kit showed no difference between groups.

Conclusion:

In-utero anatomic repair of gastroschisis is possible in mid-gestation in the fetal lamb model, and it appears to ameliorate the inflammatory process. Lactic acid may be an amniotic fluid marker of severity of gastroschisis.

Biochemical Analysis of Amniotic Fluid

	75 Day Control n=8	100 Day Control n=2	100 Day Gastro n=2	135 Day Control n=6	135 Day Gastro Repaired n=2	135 Day Gastro n=4
Glucose (g/dL)	14.0 (6-25)	11.0 (11)	4.0* (3-5)	4.0 (2-5)	3.0 (2-4)	3.0 (2-7)
Creatinine	0.5 (0.2-3.7)	2.0 (1.9-2.0)	1.2* (1.1-1.3)	6.2 (3.8-11.7)	4.5 (4.2-4.7)	1.8** (1.2-2.6)
Amylase	14.0 (1-23)	10.0 (7-13)	15.0 (8-22)	3.0 (2-5)	8.0 (4-12)	19.5* (6-73)
Lactic Acid	1.6 (0.6-2.5)	1.0 (0.8-1.2)	3.5** (3.2-3.9)	1.6 (0.9-4.6)	1.8 (1.5-4.0)	5.4** (3.9-7.0)

Concentrations expressed as median and range

* P < 0.05

** P < 0.01

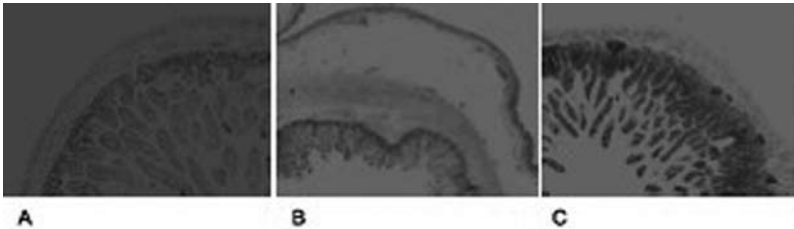


Figure 5: H&E of small intestine at 135 day gestation, 40x magnification.

A – control. B – Gastrochisis. C – Gastrochisis repaired.

Notes:

INHIBITION OF INTRA-ABDOMINAL ADHESION FORMATION IN A RABBIT MODEL WITH THE ANGIOGENESIS INHIBITOR SUNITINIB

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Purpose:

To determine the effects of sunitinib, a vascular endothelial growth factor receptor (VEGFR) antagonist, on intra-abdominal adhesion formation.

Methods:

Twenty female New Zealand white rabbits underwent a standard adhesion procedure. The uterus was eviscerated and abraded with a scalpel until punctate hemorrhages appeared, and then returned to anatomical position. One day prior to surgery, the rabbits were randomly assigned to be treated with sunitinib (10 mg/kg) or saline control via orogastric gavage. Rabbits were treated daily for a total of ten days. On postoperative day ten, the rabbits were sacrificed and their adhesions were scored. Adhesions were graded from 0-4 in two categories: tenacity of the adhesions to the uterus and the percentage of the uterus involved. Comparison of medians between groups was made using the Mann-Whitney Rank Sum Test, and differences deemed significant at $p < 0.05$.

Results:

Nine out of the ten control rabbits developed adhesions. Two rabbits in the sunitinib treated group died from esophageal injury during the gavaging process. Three of the eight surviving rabbits in the sunitinib treated group were completely adhesion free. The sunitinib treated rabbits had a median tenacity score of 1.0 [inter-quartile range (IQR) 0-1.75; range 0-2] compared to a score of 3.25 (IQR 3-3.5; range 0-4) in the control animals ($p=0.004$). Median percent involvement score for the sunitinib treated rabbits vs. controls was 1.0 (IQR 0-1.0; range 0-1) and 4.0 (IQR 4.0-4.0; range 0-4) respectively ($p=0.003$). Collectively, the sunitinib treated rabbits had a median total adhesion score of 2 (IQR 0-2.75; range 0-3) compared to 7 (IQR 6-7.5; range 0-8) in the control group ($p=0.003$).

Conclusion:

Adhesion formation is angiogenesis-dependent and is in part mediated through the vascular endothelial growth factor receptor. Sunitinib, a VEGFR 1 and 2, and platelet derived growth factor antagonist, significantly reduces adhesion formation in a rabbit model.

Notes:

WOUND SIZE MODULATES FETAL SCAR FORMATION THROUGH PROINFLAMMATORY CYTOKINE GENE EXPRESSION AND RECRUITMENT OF INFLAMMATORY CELLS

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Purpose:

The fetus has the ability to heal dermal wounds without scar. While increasing wound size is known to promote fetal scar formation, the mechanism by which wound size modulates scar formation is unknown. Scarless fetal wounds are known to be associated with decreased production of the proinflammatory cytokines IL-6 and IL-8 and a decreased inflammatory cell infiltrate compared to adult wounds. We hypothesized that increasing fetal wound size would cause increased gene expression of IL-6 and IL-8, an increased cellular inflammatory response, and scar formation.

Methods:

Small (2mm) or large (8mm) excisional dermal wounds were created in early gestation fetal sheep (65-77 days gestation). Wounds were harvested at 3 days, 7 days, or 1 month and analyzed using histology for scar formation, real-time RT-PCR for gene expression of IL-6 and IL-8, and immunohistochemistry for the presence of CD45+ inflammatory cells. RQ values were analyzed with an unpaired t-test using $p < 0.05$ to denote significance.

Results:

At 1 month, small wounds demonstrated normal dermal architecture containing hair follicles, whereas scar formation was observed in larger wounds ($n=4$). Large wounds had 2.9-fold greater IL-6 gene expression ($RQ=10.6 \pm 2.5$, $n=3$, vs. 3.6 ± 2.3 , $n=4$, $p \leq 0.01$) and 2.4-fold greater IL-8 gene expression ($RQ=3.6 \pm 1.4$, $n=3$, vs. 1.5 ± 0.5 , $n=4$, $p \leq 0.03$) than small wounds at 3 days. The density of CD45+ inflammatory cells in the wound base was greater in large wounds compared to small wounds at 3 days ($n=3$) and 7 days ($n=4$). By 1 month, the cellular inflammatory response had subsided in all wounds ($n=4$).

Conclusions:

Increased fetal wound size results in elevated gene expression of the proinflammatory cytokines IL-6 and IL-8, increased inflammatory cell infiltrate, and corresponds to a change in wound phenotype from scarless regeneration to scar formation. Targeting these components of the inflammatory response is a potential strategy for altering postnatal fibrotic wound healing.

Notes:

TEMPORAL SUSCEPTIBILITY OF PRIMARY MURINE CHOLANGIOCYTES MIMICS THE MURINE MODEL OF BILIARY ATRESIA

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Purpose:

Biliary atresia is a disease of the neonate which results in obliteration of the biliary tree. A murine model of biliary atresia has been established in which infection of newborn mice with rhesus rotavirus (RRV) leads to an obstructive cholangiopathy. Using this model, we have shown that RRV infection must take place before day of life (DOL) 3 or biliary obstruction does not occur. The basis of the temporal dependence of both the human disease, insofar as it is limited to the neonatal period, and the murine model has not been established. *In vivo* studies performed in our lab revealed that infection by DOL 3 results in higher amounts of live virus in extrahepatic biliary samples with marked cholangiocyte destruction by histology. We hypothesized that the basis for this temporal dependence was differential susceptibility of the biliary epithelium to RRV infection.

Methods:

Neonatal BALB/c mice were sacrificed on day of life (DOL) 2 and 9, and their livers were harvested, homogenized and primary cholangiocytes were purified by Percoll gradient. Cholangiocytes were cultured and infected with RRV at a multiplicity of infection (MOI) of 300 which would ensure that all cells would be infected, and live viral yield was assayed.

Results:

At an MOI of 300, live rotaviral yield from infected cholangiocytes harvested on DOL 2 was seven-fold higher than that obtained from cholangiocytes harvested on DOL 9 (353.34 vs. 49.71 virus particles/cell respectively, $p < 0.05$).

Conclusions:

RRV infection in the early perinatal period is necessary to induce the murine model of biliary atresia. *In vitro* studies demonstrate that more mature biliary epithelial cells are less susceptible to viral infection. These findings suggest that the temporal dependence of this disease model may be due, in part, to differential susceptibility of primary murine cholangiocytes, recapitulating *in vivo* findings.

Notes:

CHOLANGIOCYTE APOPTOSIS DURING LAMPREY METAMORPHOSIS

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¹University of Nevada School of Medicine, Las Vegas, NV, USA, ²University of Nevada Las Vegas, Las Vegas, NV, USA, ³University of Toronto, Toronto, ON, Canada

Background:

Progress towards understanding the etiology of biliary atresia has been hindered by lack of a suitable animal model. Lampreys are primitive vertebrates that have developmental stages. During metamorphosis to the adult lamprey, the biliary system of the larval lamprey disappears. Bile duct loss in the lamprey has been proposed as a model for human biliary atresia. We have begun exploring the molecular events associated with bile duct loss during lamprey metamorphosis by determining when apoptosis first affects cholangiocytes.

Materials and Methods:

Wild caught larval sea lampreys were purchased through a commercial supplier and were housed in aquaria under controlled environmental conditions. Animals of adequate size and length (120 cm in length and 3 grams in weight) were induced to undergo metamorphosis by exposure to 0.01% KClO₄ at water temperatures of between 18-22°C. During induction animals were observed weekly and the stage of metamorphosis was determined based upon external features. The stage of metamorphosis was confirmed by the person who described the staging system (Dr. John Youson). After euthanasia, lamprey livers were harvested, immediately frozen and cut for immunohistochemical analyses. Apoptosis was detected using a fluorescent TUNEL assay (Promega) and cholangiocytes were identified by fluorescent staining for CK-19 (Sigma). Multiple sections from livers of lampreys at various stages of metamorphosis were evaluated using confocal microscopy. Percentage of apoptotic cholangiocytes were determined by 2 independent blinded investigators.

Results:

Percent apoptosis was 40% in premetamorphic (stage 0) lamprey (n=2), 59% at stage 1 (n=3), 50% at stage 2 (n=2), and 12% at stage 3 (n=2). These apoptotic indices are being confirmed using other markers of apoptosis.

Conclusions:

Our preliminary results suggest that cholangiocyte apoptosis occurs during the earliest stages of lamprey metamorphosis. This animal model may provide clues to a genetic etiology for bile duct loss in humans.

Notes:

INHIBITION OF PLACENTA-LIKE GROWTH FACTOR INCREASES METASTASIS IN EXPERIMENTAL NEUROBLASTOMA

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Purpose:

New tumor vessels are thought to result primarily from activity of vascular endothelial growth factor (VEGF). VEGF blockade has been validated as a therapy for human cancer, although even responsive tumors ultimately progress. The VEGF family member placenta-like growth factor (PLGF) forms both heterodimers with VEGF and homodimers, contributing to pathologic angiogenesis, although the relative importance of these forms is unknown. We have previously reported that experimental neuroblastoma can be partially inhibited by VEGF blockade but ultimately progresses, suggesting recruitment of alternative pathways. We hypothesized that targeting both VEGF and PLGF might increase effective inhibition of tumor growth.

Methods:

Lentivirally-delivered shRNA was used to stably knock-down PLGF in cultured SY5Y human neuroblastoma cells engineered to express luciferase. Xenografts were induced by implantation of 10^6 sh-PLGF or control-shRNA transfected cells into athymic mice (PLGF-knockdown, N=36; controls, N=42). Cohorts were further randomized at 1 week to receive bevacizumab (anti-huVEGF antibody, 250 mcg/dose intraperitoneally) or vehicle for 5 weeks. Tumor growth was monitored using bioluminescence imaging, and mice sacrificed when flux reached 3.0×10^9 photons/second. Metastasis was assessed by bioluminescence and culture of bone marrow, and differential tumor growth measured by Kaplan-Meier survival analysis.

Results:

VEGF blockade slowed control xenograft growth ($P=0.001$) as previously observed, but did not restrict growth in PLGF knockdown tumors ($P=0.89$). Unexpectedly, metastasis was significantly increased in PLGF knockdown xenografts compared to untransfected controls (76% vs. 16%; $P=0.03$; Figure 1). VEGF blockade significantly reduced the bone marrow metastasis burden in these animals (33% vs. 17% of total metastatic photon-flux; $P<0.001$).

Conclusions:

Loss of PLGF markedly increases metastasis in this neuroblastoma model. This effect is partially reversed by VEGF blockade, through a reduction in bone marrow metastasis. These data suggest that unbalanced VEGF/PLGF signaling may play a role in the spread of neuroblastoma to distant sites, and warrants further investigation.

SY5Y Xenograft Metastasis as Measured with Bioluminescence



Primary intraneural xenografts are represented by the intense large signal. Note that multiple smaller signals are present throughout the mouse in the left, indicating multiple metastases. The control mouse (no knockdown) demonstrates no metastatic signal.

Notes:

HSP-70 INHIBITION WITH TRIPTOLIDE THERAPY CAUSES APOPTOSIS IN NEUROBLASTOMA

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Purpose:

Advanced-stage cases of neuroblastoma, the most common pediatric extracranial tumor, are highly resistant to conventional chemotherapy. Our recent work demonstrated that Hsp-70 inhibition with triptolide, from the plant *Tripterygium wilfordii*, kills neuroblastoma cells in vitro. Decreased expression of Hsp-70 mRNA and protein, as well as corresponding elevations in caspase activity and Annexin-V staining have suggested that cells undergo apoptosis via Hsp-70 inhibition. The primary objective of this study was to evaluate the effect of triptolide therapy on neuroblastoma tumors in vivo. In particular, we aimed to look at markers of apoptosis and Hsp-70 expression in residual tumors of treated and control mice.

Methods:

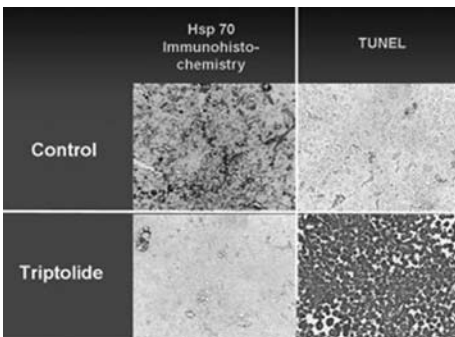
With IACUC approval, an orthotopic tumor model was developed by retroperitoneal injection of 1×10^6 N2a cells into A/J mice. Following randomization, treatment and control groups received daily injections of triptolide 0.4 mg/kg and vehicle, respectively. At 21 days, remaining mice were sacrificed, and tumors were measured. Immunohistochemistry was used to characterize levels of Hsp-70 in residual tumors. Terminal deoxynucleotidyl transferase dUTP nick end labeling (TUNEL) was performed on tumor tissue to identify cells undergoing apoptosis.

Results:

Mice receiving triptolide therapy had smaller tumors than control: average tumor volume $0.33 \pm 0.11 \text{ cm}^3$ (vs 1.99 ± 0.54), average mass $0.35 \pm 0.12 \text{ g}$ (vs 1.84 ± 0.53), $p < 0.01$, $N=2$. Hsp-70 immunohistochemistry showed significant staining in tumors from control mice, and minimal staining in tumors from treated mice. Tumors from mice receiving triptolide had significant TUNEL staining, while those receiving vehicle did not show evidence of apoptosis.

Conclusions:

Residual tumors from triptolide-treated mice expressed decreased levels of Hsp-70 and demonstrated a significant extent of apoptosis. We conclude that triptolide therapy triggers apoptosis of neuroblastoma tumors in vivo by inhibition of Hsp-70. Triptolide may have a future role in targeted therapy for neuroblastoma, and further studies are warranted.



Notes:

IMPROVED INHIBITION OF NEUROBLASTOMA WITH COMBINATION THERAPY OF ENRICHED OMEGA-3 DIET AND SUNITINIB OVER EITHER AGENT ALONE

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Background:

Omega-3 (n-3) polyunsaturated fatty acids (PUFAs) inhibit the growth of neuroblastoma cell lines in vitro but no studies have demonstrated a beneficial effect of a diet enriched in n-3 on reduction of neuroblastoma tumor growth in vivo. Because preclinical studies with n-3 in other neoplasms have demonstrated efficacy of combination therapies, we investigated the effect of simultaneous treatment with dietary enrichment of n-3 and the VEGF, PDGF and Kit inhibitor sunitinib, alone and in combination on orthotopic and subcutaneous neuroblastoma tumor models.

Methods:

Cells from the human neuroblastoma SK-NSH line were implanted in an orthotopic (left adrenal) or subcutaneous position in SCID mice. Mice were fed either standard chow or a diet enriched in n-3 (10% Menhaden oil). For each diet a group of animals was administered no drug (methylcellulose carrier) or sunitinib (20 mg/kg/day) by oral gastic gavage.

Results:

At 21 days, n-3 diet and sunitinib administration as single agent therapies were associated with a significant reduction of tumor volume over control diet in both tumor models ($p < 0.001$). Sunitinib was more efficacious than n-3 enrichment in the subcutaneous model ($p = 0.003$), but the two treatments were comparable in the orthotopic model. Combination treatment improved tumor inhibition (T/C = 0.19 vs 0.32). Mice fed n-3 for 3 weeks before tumor implantation had further inhibition of tumor growth. Tumor inhibition appeared unrelated to cellular proliferation by Ki67 staining. Combination therapy decreased tumor microvessel density ($p = 0.033$). There was a significant reduction CD45+ inflammatory cells in all treated groups compared to control diet.

Conclusion:

Combination therapy using an enriched n-3 diet and sunitinib improved inhibition of neuroblastoma tumors over single agent therapy. Early initiation of a diet high in n-3 PUFAs may further limit tumor growth. These effects may be mediated through pro-apoptotic or anti-inflammatory mechanisms and warrant further investigation.

Notes:

IDENTIFYING BINDING PARTNERS OF THE TRANSCRIPTIONAL CO-ACTIVATOR, CITED1, THAT REGULATE ITS NUCLEAR TRAFFICKING AND FUNCTION IN WILMS' TUMOR

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Background:

Wilms' tumors (WT) are thought to arise from the abnormal retention and failed maturation of epithelial progenitor cells in the developing kidney. We have shown previously that CITED1, a CBP/p300-interacting transcriptional co-activator, is expressed specifically in early nephronic progenitor cells, is down-regulated as epithelial conversion proceeds, yet its expression persists in WT and correlates with tumor severity. We have shown further that CITED1 enriches the nucleus of WT blastema relative to its predominantly cytosolic expression in nephronic progenitor cells.

Methods:

To identify potential CITED1 binding partners that might regulate its context-dependent nuclear trafficking and function, endogenous CITED1 was immunoprecipitated from subcellular fractions of breast carcinoma MCF7 cells (which richly express CITED1 in the cytosol and nucleus) and a human WT cell line, WiT49, manipulated to express predominantly cytosolic or nuclear-enriched CITED1. Proteins were separated by gel-electrophoresis, and in-gel digest was performed for mass spectrometry (MS) to identify candidate CITED1 binding partners. Proteins detected from control IgG immunoprecipitates were excluded as non-specific. Validation of MS results was performed using co-immunoprecipitation strategies.

Results:

MS detected spectra of multiple candidate CITED1 binding partners, including importantly the tumor suppressor p53, in both wild type MCF7 and experimental WiT49 cell lines. Interaction with p53 was validated after immunoprecipitation of CITED1 and detection of p53 on Western blot using both MCF7 and experimental WiT49 cell lines. Immunoprecipitation of p53 from the WiT49 cell lines was also performed and detected CITED1. Numerous other proteins showed differential spectral hits between the cytosol and nucleus.

Conclusions:

These findings suggest potentially important regulatory functions of CITED1 in WT pathogenic responses. The interaction with p53 is particularly intriguing, as this tumor suppressor has been shown to accumulate in unfavorable histology and aggressive variants of WT. Future directions will explore the clinical relevance and functional significance of this interaction.

Notes:

A NOVEL METHOD FOR DETECTION OF CIRCULATING TUMOR CELLS IN METASTASIZING EXPERIMENTAL PEDIATRIC SOLID TUMORS

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Purpose:

Pulmonary metastasis is a leading cause of death in childhood cancer. Circulating tumor cells, which contribute to hematogenous metastasis, are known to independently predict survival in some cancers. Currently, reverse transcriptase PCR is used to detect circulating tumor cells (CTCs) clinically and in tumor modeling. However, this is only an indirect measure of tumor cell number, and may therefore not accurately reflect CTC quantity. We hypothesized that bioluminescence could sensitively and accurately be used to detect CTCs in a mouse model of Ewing's sarcoma.

Methods:

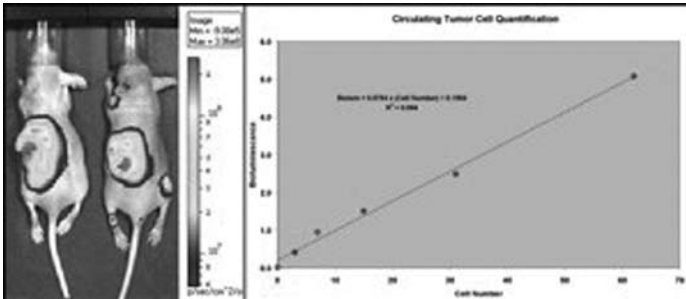
All studies were approved by the Animal Care Committee. Cultured SKNEP Ewing's sarcoma cells were stably transfected with firefly luciferase. Aliquots of cells in decreasing amounts were resuspended in 300 uL of mouse blood, erythrocytes lysed with Cell Lysis Solution, and centrifuged. The remaining pellet was treated with Passive Lysis Buffer, added to 100 uL of Luciferase Assay Reagent II, and bioluminescence recorded for each sample via a luminometer. To test our assay *in vivo*, 5 mice were implanted with 1×10^6 SKNEP-luc cells. Primary tumor growth was monitored via bioluminescence, 300 uL blood was obtained when this reached 1×10^9 photons, and assayed as above.

Results:

In vitro studies, CTC quantity was highly correlated with bioluminescence reading ($R^2=0.99$). *In vivo*, three of five mice had tumors that reached our criteria for the assay. We were able to detect 1 to 2 CTCs per 300 uL in each mouse, indicating relative scarcity of hematogenously disseminated cells in this model. Metastasis could also be detected using *in vivo* bioluminescence (Figure).

Conclusions:

Bioluminescence reading reliably quantitates CTCs in our metastasizing tumor model. These data may prove useful in studying mechanisms which influence tumor cell entry and survival in the circulation. Such studies will be critical to understanding the effects of anti-angiogenesis on tumor metastasis.



Notes:

TUMOR CELL LYSATE PREPARATION IS CRITICAL TO THE EFFICACY OF WHOLE TUMOR CELL VACCINE STRATEGIES

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Purpose:

Tumor vaccine strategies have as yet unmet potential in the treatment of solid tumors. Vaccine strategies frequently use tumor cell lysate as the source of tumor antigens for induction of tumor specific immunity. This study evaluates the effect of tumor cell lysate preparation on immunity as a prelude to tumor vaccine formulation.

Methods:

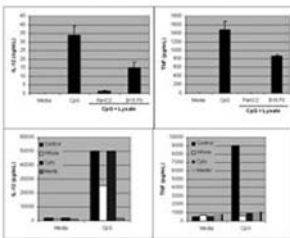
Murine primary bone marrow derived macrophages were used as a model of innate immunity and stimulated with the Toll-like receptor agonist CpG oligonucleotide (2µg/mL), with and without various preparations of tumor lysate, and cytokine (IL-12 and TNF) production were determined. Lysates from various solid tumor types including Neuroblastoma were tested. Tumor cell lysates were further fractionated into plasma membrane and cytoplasmic components by high-speed centrifugation and the effect of each component on innate immunity was examined. To determine the effect on acquired immunity, fresh splenocytes were cultured with tumor lysates and the effect on T-cell IFN- secretion was examined.

Results:

Tumor lysate prepared by either freeze thaw cycling or chemotherapy blocked macrophage activation and subsequent T-cell immunity. PanO2 lysate decreased IL-12 production by 96% and TNF was completely suppressed. B16.F0 tumor lysate decreased IL-12 production by 55.6% and TNF production by 41.8%. The cell membrane was responsible for this suppressive effect. Heat shock preparation or fixation of lysate did not suppress APC function or T-cell activation.

Conclusions:

Macrophage and T-cell function is profoundly suppressed when presented with freeze-thawed tumor lysate, a common method of antigen preparation in whole tumor cell vaccine strategies. The cell membrane is responsible for the suppressive effect on APCs that is avoided by heat-shock preparation of the lysate. If vaccine strategies are to meet their therapeutic potential, lysate preparation is critical and heat-shocked lysate is a good option for the antigen source.



Notes:

MÜLLERIAN INHIBITING SUBSTANCE INHIBITS MIGRATION OF EPITHELIAL CANCER CELL LINES

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Purpose:

For most cancers, recurrence and metastasis is largely responsible for patient mortality. Chemotherapeutics capable of both inhibiting tumor growth and preventing metastasis could offer significant benefits over agents which only target one aspect of cancer biology. Our laboratory has established Müllerian Inhibiting Substance (MIS) as an anti-cancer agent *in vitro* and *in vivo* both as a single agent and in combination with traditional chemotherapeutics. Given the complex remodeling necessary for ductal regression during development, we examined, in addition to its effect on cancer cell proliferation, whether MIS could affect cell migration and invasion.

Materials and Methods:

Using 6 doses each with 10 replicates, an MIS dose response curve and an IC50 for inhibition of cell growth were established using a methylthiazolotetrazolium inhibition assay. To evaluate the effect of MIS on migration, the Hep3 (head and neck), MDA-MB-231 (breast), and IGROV-1 (ovarian) cancer cell lines shown to be invasive in chick chorioallantoic membrane assays were plated in a matrigel invasion assay with MIS in the attractant media at concentrations below the IC50 established for cell growth inhibition.

Results:

We established an IC50 of 15 µg/ml of MIS below which inhibition of cell proliferation was marginal. Despite this, we found that at concentrations of 7 µg/ml, MIS inhibited migration in Hep3 (138 vs. 313, $p < 0.002$, $n = 4$) and MDA-231 (54 vs. 554, $p < 0.04$, $n = 4$) with IGROV-1 not reaching statistical significance (130 vs. 249, $p = 0.16$, $n = 4$), indicating MIS affects migration independent of inhibiting cell proliferation.

Conclusions:

Our results show that at concentrations below those required for inhibiting cell growth, MIS has a significant effect on cancer cell invasion *in vitro*. We will confirm the anti-invasive effects of MIS on cancer cells *in vivo*, and use these assays to elucidate the mechanisms responsible for this phenomena with an eye on its impending clinical application.

Notes:

TOLL-LIKE RECEPTOR 8 AS A POTENTIAL THERAPEUTIC TARGET FOR SOFT TISSUE AND BONE SARCOMAS.

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Purpose:

There are limited treatment options for patients with advanced or recurrent soft tissue sarcomas (STS) and bone sarcomas (BS). Combination chemotherapy, which is most often used, has limited efficacy because of high rates of chemoresistance. The aim of this study was to discover a novel therapeutic target molecule for STS.

Methods:

We investigated Toll-like receptor 8 (TLR 8) mRNA and protein expression in STS and BS cell lines derived from patients. TLR 8 mRNA expression in sarcoma cell lines compared to normal tissues was validated by quantitative PCR. WST-1 cytotoxicity assay was used to measure the effect of Polyuridylic acid (PolyU), a TLR 8 ligand, on cell viability. RNA interference was used to knock down TLR 8 expression and study the involvement of TLR 8 in polyU induced cell death. Apoptosis was assessed by Annexin V staining, rate of proliferation by BrdU incorporation, and apoptotic proteins were visualized by western blotting.

Results:

Sarcoma cells express TLR 8 mRNA and protein. TLR 8 mRNA is over-expressed in sarcoma cell lines compared to normal tissues. Stimulation of TLR 8 with its ligand, polyU, results in significant decrease in cell viability in both chemoresistant and chemosensitive sarcoma cell lines. PolyU reduces sarcoma cell number by inducing apoptosis and inhibition of cell proliferation. PolyU can cause apoptosis in sarcoma cell lines by two independent death pathways: it can activate caspases or other pro-apoptotic proteins that function through a caspase independent pathway.

Conclusion:

TLR 8 is expressed in sarcoma cell lines. Ligation of TLR8 in sarcoma cell lines can inhibit proliferation and induce apoptosis through two independent death pathways. These observations suggest that TLR 8 may represent a potential therapeutic target for STS and BS.

Notes:

THE EFFECT OF PRENATAL DIAGNOSIS ON THE CONTEMPORARY OUTCOME OF CONGENITAL DIAPHRAMATIC HERNIA

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Background:

Despite advances in neonatal care of CDH, significant variations exist in the mortality rates reported by individual centers. Some clinicians are relatively pessimistic and recommend termination of pregnancy when a prenatal diagnosis is made. We report the outcome of CDH in a contemporary prospective multicenter study.

Methods:

CDH cases were abstracted from a disease-specific, 16 hospital, national network which began patient accrual in May 2005. 13 hospitals contributed to this study. All patients were admitted with CDH within 24 hours of life.

Results:

175 patients have data completed until hospital discharge or death. There were 20 terminations (11.4%), 2 stillbirths (1%) and 6 died in transport. Lung-to-Head ratio was available in only 12% of cases (11% \geq 1.0, 1% $<$ 1.0). 147 patients were admitted alive to a participating tertiary care hospital. 139 (95%) had prenatal ultrasound. Survival was 120/147 (81.6%). Prenatal diagnosis was made in 94 patients (64%) and their survival was 74%. 53 patients without prenatal diagnosis had a survival rate of 96%. ECMO was used in 7.5% of patients with 4/11 (36%) survival. In 15% of patients, no repair of CDH was attempted.

Conclusion:

The overall survival rate in CDH in this study is better than reported by other group, however prenatal diagnosis is associated with a 26% mortality. When the diagnosis is missed on prenatal ultrasound, the survival is generally favorable, possibly related to an intraabdominal position of the stomach.

Notes:

OUTCOMES FOLLOWING MUSCLE FLAP VERSUS PROSTHETIC PATCH REPAIR FOR LARGE DIAPHRAGMATIC HERNIAS

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Purpose:

Repair of large congenital diaphragmatic hernias (CDH) still poses a significant challenge, and many patients have large defects that cannot be repaired primarily. Two techniques have been widely used: autologous anterior abdominal wall muscle flap and synthetic patch. Our goal was to compare the short-term and long-term outcomes of these two approaches.

Methods:

Retrospective review of all neonates undergoing CDH repair at our institution from 1969 to 2006. Research ethics board approval was obtained

Results:

Of 188 children undergoing surgery for CDH, primary diaphragmatic repair could not be accomplished in 51 infants (27%). Nineteen had a muscle flap and 32 had a prosthetic patch repair (Gore-tex® n=15, Marlex n=9, Surgisis® n=5, Silastic n=3). There was no significant difference in gestational age or birth weight between groups. Three patients developed an abdominal wall defect at the muscle flap donor site, but none required surgical intervention. Musculoskeletal deformities were found in 9 patients, 3 after a muscle flap and 6 after a prosthetic patch (p=0.7). Post-operative bowel obstruction occurred in 3 muscle flap patients and 1 patch patient (p=0.2). There were 10 recurrences among survivors: 2 after a muscle flap and 8 after a prosthetic patch (p=0.3) There were two deaths among the muscle flap patients (10%) and 3 deaths among the prosthetic patch repair patients (9%) (p=0.1). Lack of difference between groups was confirmed after controlling for age and co-morbidities in a multivariate logistic regression

Conclusions:

These results suggest that autologous anterior abdominal wall muscle flap and prosthetic patch repairs provide similar short-term and long-term outcome.

Notes:

MINIMALLY INVASIVE REPAIR OF PECTUS EXCAVATUM IN PATIENTS WITH MARFAN SYNDROME AND MARFANOID

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Purpose:

The presence of a pectus excavatum (PE) requiring surgical repair is a major skeletal feature of Marfan syndrome. Marfanoid patients have phenotypic findings but do not meet all diagnostic criteria. We sought to examine the clinical and management differences between Marfan syndrome patients and those who are marfanoid compared to all other patients undergoing minimally invasive PE repair.

Methods:

A retrospective IRB-approved review was conducted of a prospectively gathered database of all patients who underwent minimally invasive repair of PE. Patients were grouped according to diagnosis of Marfan syndrome (MAR), Marfanoid appearance (OID) and all others (ALL). Patient demographics, preoperative testing, operative strategy, complications and postoperative surveys were evaluated. Fisher's exact test and Chi-square were applied for statistical analysis.

Results:

From 06/1987 to 09/2008, 1192 patients underwent minimally invasive PE repair (MAR=33, OID=212, ALL= 947). There was a significantly higher proportion of females with either MAR or OID who underwent repair (21.5%vs 15.5%, p=0.04). MAR patients had significantly more severe PE determined by CT index (MAR=8.75, OID=5.82, ALL=4.94, p<0.0001) and required multiple pectus bars (≥2) to be placed during operation (MAR=58%, OID=36%, ALL=29%, p=0.001). There was a trend toward higher wound infection rates in MAR patients (MAR=6%, OID=1.4, ALL=1.3%, p=0.07). The recurrence rate was similar among all groups (MAR=0%, OID=2% ALL=0.7%, p=0.12). Successful outcome from surgeon's perspective in either MAR or OID patients was similar to ALL (98%vs 98%, p=0.88) and correlated well with patient satisfaction after repair (96%vs 95%, p=0.43).

Conclusions:

Minimally invasive pectus excavatum repair is safe in patients with Marfan syndrome or marfanoid features with equally good results. Marfan syndrome patients have clinically more severe pectus excavatum requiring multiple bars for chest repair and may have slightly higher wound infections rates. Patients are satisfied with minimally invasive repair despite a phenotypically more severe chest wall defect.

Notes:

USE OF AN ABSORBABLE STABILIZER FOR PECTUS EXCAVATUM REPAIR

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Purpose:

To evaluate the safety of an absorbable stabilizer for the minimally invasive repair of pectus excavatum. In 2004 it was decided to manufacture pectus excavatum stabilizers from Lactosorb®, poly L-lactic/polyglycolic acid polymer. Lactosorb® plates, meshes and screws (Biometmicrofixation, Jacksonville) are widely and successfully used in plastic and craniofacial reconstructive surgery. However, because pectus stabilizers are much thicker than previous craniofacial plates, and there was concern that they would not re-absorb adequately or act as a nidus for seroma and abscess formation.

Materials Methods:

Institutional Review Board permission and subsequent informed consent were obtained for the use of lactosorb stabilizers in ten (10) patients whose ages ranged from 14 to 29 years. Four patients had only one pectus bar and six had two bars placed for a total of 16 bars. The lactosorb stabilizers were attached to 11 pectus bars with PDS sutures. Eight bars did not require a metal stabilizer and therefore only a single lactosorb stabilizer was applied to each. Three bars had both a metallic and lactosorb stabilizer. After periodic follow up, the bars were removed 2 ½ to 4 years post insertion.

Results:

No patients experienced problems related to the absorbable stabilizers. No seromas or abscesses developed. There were no vertical bar displacements; in one patient the bar had shifted slightly to his right side at time of removal with no change in chest configuration. Bar removal was facilitated in all patients as there was minimal tissue reaction at the site of absorbable stabilizer placement as compared to metal stabilizers.

Conclusion:

Prototype, solid Lactosorb® pectus excavatum stabilizers were completely re-absorbed at the time of bar removal, 2 ½ to 4 years after insertion. The lack of tissue reaction greatly facilitated bar removal.

Notes:

RETROSPECTIVE REVIEW OF RE-OPERATIVE PECTUS EXCAVATUM REPAIRS

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Purpose:

Despite success of several techniques described for Pectus Excavatum repair, a minority of patients require multiple re-operations for recurrence or other complications. We aimed to review our experience in re-operative Pectus Excavatum repairs and to identify features correlating with need for additional re-operations.

Methods:

With IRB approval, charts were reviewed of all re-operative Pectus Excavatum repairs over three years at a university-based children's hospital. Number and type of previous repairs, time between operations, lengths of stay, analgesia, and complications were recorded.

Results:

From 02/2004-12/2007, 170 Pectus Excavatum repairs were performed. Among these, 27 were re-operative (22 patients). Average re-operative age was 24.5 years (range 12-52), and 63% were male. The average number of previous repairs was 1.4 (range 1-4), and average time since the last repair was 5.8 years (range 1 month-38 years). Two-thirds of patients had previous repairs performed at outside institutions. Reasons for re-operation included: recurrence (59.3%), pseudoarthrosis (29.6%), bar displacement (11.1%), and rib disarticulation (3.7%). Re-operative repair types included 6 Nuss (22.2%), 13 Ravitch (48.2%), 4 suture-thoracoplasty (14.8%), 3 bone thoracoplasty (11.1%), and 1 thoracoplasty with absorbable plate (3.7%). Overall, 14.8% of re-operative patients required subsequent further re-operations. 15.8% of patients undergoing repeat open repairs and 33.3% of patients undergoing repeat minimally invasive repairs required additional operative interventions. There was no need for additional repairs among patients who had open repairs following minimally invasive repairs, nor for any patients who had minimally invasive repairs following open repairs.

Conclusions:

We conclude that patients with failed open repairs will have better success with minimally invasive repairs, while patients with failed minimally invasive repairs will have better success with open repairs. This may be attributed to patient selection versus scar tissue/adhesions. When faced with re-operative Pectus Excavatum, we recommend consideration of an alternative operative approach from the initial procedure.

Notes:

13C-METHIONINE BREATH TEST TO ASSESS LIVER DISEASE IN CHILDREN WITH INTESTINAL FAILURE

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¹Center for Advanced Intestinal Rehabilitation, Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA, ²Center for Advanced Intestinal Rehabilitation, Department of Surgery, Children's Hospital Boston, Boston, MA, USA, ³Department of Surgery, Children's Hospital Boston, Boston, MA, USA, ⁴Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA, ⁵Shriners Hospital for Children, Boston, MA, USA, ⁶Center for Advanced Intestinal, Rehabilitation, Division of Gastroenterology and Nutrition, Children's Hospital Boston, Boston, MA, USA

Objective:

Children with short bowel syndrome suffer from Intestinal Failure Associated Liver Disease (IFALD). Current biochemical tests are static measures of liver function. L[1-¹³C]-methionine is a stable (non-radioactive) isotope exclusively metabolized in liver mitochondria and can be quantified by measuring expired ¹³CO₂. We hypothesized that the ¹³C-methionine breath test (¹³C-MBT) would be a feasible, non-invasive measure of hepatic function in children with IFALD.

Methods:

Nineteen patients with intestinal failure were studied. Eight patients had documented liver biopsies, four of which underwent repeat studies after clinically suspected changes in liver function. After collection of baseline breath samples, 2 mg/kg of sterile, pyrogen-free ¹³C-Methionine was given intravenously followed by 6 paired breath samples obtained every 20 minutes. Samples were analyzed for ¹³CO₂ enrichment using isotope ratio mass spectrometry. Serial biochemical liver tests and PELD scores were recorded. Total ¹³CO₂ production was measured by indirect calorimetry. The cumulative % recovery of administered ¹³CO₂ from the injected ¹³C-Methionine (% Recovery) and the area under the curve (AUC) were calculated.

Results:

All 19 patients (median age = 5.1 months, IQR = 4.4-8.0 months) tolerated the ¹³C-MBT without any adverse events. Among 8 patients with liver biopsies, 3 had cirrhosis and 5 had cholestasis or fibrosis. The mean % Recovery of patients with and without cirrhosis was 3.3 and 9.4 % respectively (p=.07). The AUC of patients with and without cirrhosis was 179 and 462 respectively (p<.05). 3 of the 4 patients with repeated measurements showed increased ¹³C-Methioine excretion with decreased PELD scores.

Conclusion:

The intravenous administration of the stable isotope ¹³C-Methionine and serial breath collection is a feasible, safe and potentially clinically relevant approach for evaluation of hepatic function in children with intestinal failure. The ¹³C-Methioine breath test may be a reliable measure to assess progression or improvement of liver disease in this patient population.

Notes:

P26

PORTOSYSTEMIC SHUNTS IN CHILDREN

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Purpose:

Review the safety and long-term success at a single institution with portosystemic shunts in children.

Methods:

IRB-approved, retrospective chart review of all children 19 years and younger undergoing surgical portosystemic shunting from January 1988- September 2007.

Results:

Ten patients were identified, 8 females and 2 males, mean age 15 years (range 5-19 years). Primary diagnoses were congenital hepatic fibrosis (5), Budd Chiari (2), portal vein thrombosis (2), and cystic fibrosis (1). Indications for shunt were portal hypertension (10), varices (9) and ascites (3). Shunts performed were distal splenorenal (4), side-to-side portocaval shunt (3), proximal splenorenal (2), and a portocaval H graft (1). No perioperative mortalities occurred. Morbidity was 30% and included a urinary infection, central line infection, worsening ascites, and one negative reexploration. Seventy-percent of patients had good outcomes and symptom improvement. Ninety-percent of shunts remained patent; with one occluded at a mean follow-up of 5.2 years (range 0.5-13.16 years). Two patients underwent subsequent liver transplant. Two patients died at 0.5 and 12.8 years postoperatively; the first from multisystem failure in cystic fibrosis and the latter from posttransplant complications. Finally, no patients with low MELD or PELD scores preoperatively went on to liver transplantation, whereas, 67% of patients with high MELD or PELD scores (>18) required future liver transplantation.

Conclusions:

The need for portosystemic shunts in children is rare. However, in the liver transplant era their utility in patients with preserved liver function remains important. We conclude that they can be performed safely, with low morbidity, and with good long-term success. At distant follow-up, most shunts remain patent with improvement of patient symptoms and with a diminished need for liver transplantation. High preoperative MELD and PELD scoring seem to be linked to a subsequent need for liver transplantation suggesting that these patients may not benefit long-term from portosystemic shunts.

Notes:

ALAGILLE SYNDROME: OUTCOME COMPARISON OF CONSERVATIVE TREATMENT VERSUS KASAI PROCEDURE

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Purpose:

Alagille Syndrome (AGS) is an autosomal dominant disorder, caused by mutations in *Jagged1*, in which multiple organ systems are involved with variable penetrance. AGS frequently presents with neonatal cholestasis, and can be confused with other causes of infantile conjugated hyperbilirubinemia, most notably biliary atresia. We present the largest multi-institutional experience of surgical outcomes for biliary disease in AGS. Our specific aim was to assess outcomes of children with AGS who underwent the Kasai procedure.

Methods:

A retrospective review of the Alagille Syndrome Diagnostic Center database at our institution was performed looking for clinically-defined AGS patients who underwent a Kasai. A cohort of control subjects was selected with equivalent symptoms of neonatal jaundice and matched for age and presence of cardiac anomaly. *Jagged1*-mutation analysis was performed on 16/19 participants. Clinical courses were reviewed. Fisher exact and t tests were used for analysis.

Results:

Of 430 AGS patients, 19 underwent a Kasai procedure (K). The control cohort consisted of 41 patients (C). Total Bilirubin measured between 6 to 10 weeks of age was equivalent [K:9.6 C:9.5], GGT levels were higher in the control group [K:493.4 C:626.4]. Of note, the Kasai cohort had a significantly larger number of Liver transplants [K:9 (47.3%), C:7 (17.1%), $P < 0.03$] and sustained higher mortality [K:5 (26.3%), C:1 (2.4%), $P = 0.01$]. There was no genotype-phenotype correlation between the mutations identified and patients who underwent Kasai.

Conclusions:

Our data confirms that the Kasai procedure, although appropriate for children with biliary atresia, does not benefit children with AGS and actually has worse outcomes. The current data suggest that the Kasai is not a marker for underlying severe liver disease, and the procedure itself has a detrimental effect on outcome. In cases of neonatal jaundice, the surgeon should rule out AGS by screening for syndromic findings before operating.

Notes:

P28

REGIONALIZATION OF PEDIATRIC LIVER TRANSPLANTATION IN THE UNITED STATES

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Purpose:

Regionalization of complex surgical procedures to high-volume centers may improve patient outcomes. However, temporal trends in pediatric liver transplantation have not been previously described.

Methods:

We performed a retrospective analysis of 3546 pediatric orthotopic liver transplant procedures included in the Scientific Registry of Transplant Recipients (SRTR) over three consecutive 30-month time periods between 1999 and 2006. Transplant centers not considered freestanding children’s hospitals were divided into low-volume (LVC; annual volume \leq 11 procedures) and high-volume centers (HVC; annual volume $>$ 12), while freestanding children’s hospitals (FCH) were analyzed separately. One-year observed-to-expected patient death ratios provided by the SRTR were calculated for each group in each time period. Statistical analysis was performed using chi square test (significant at $p < 0.05$).

Results:

From Period 1 to Period 3, an increasing percentage of transplants were performed in high-volume centers, while the percentage performed in freestanding children’s hospitals did not change significantly (Table 1). In Period 1, recipient outcomes at freestanding children’s hospitals were superior to those at high-volume non-freestanding centers (O:E ratios: HVC 1.17, FCH 0.64, $p = 0.01$) but there was not a significant difference between high-volume and low-volume non-freestanding centers (O:E ratios: HVC 1.17, LVC 1.12, $p = NS$). By Period 3, outcomes at high-volume non-freestanding centers were better than outcomes at low-volume centers (O:E ratios: HVC 0.79, LVC 1.64, $p < 0.01$) and equivalent to outcomes at freestanding children’s hospitals (O:E ratios: HVC 0.79, FCH 0.92, $p = NS$).

Conclusions:

Regionalization of pediatric liver transplantation to high-volume centers is associated with the emergence of a volume-outcomes relationship. Recipient outcomes at high-volume centers are now comparable to those achieved at freestanding children’s hospitals.

Table 1. Regionalization of Pediatric Liver Transplantation Procedures in the United States

Center Volume Category	Period 1 (% of all procedures)	Period 3 (% of all procedures)	p value (P3 vs P1)
LVC	29%	22%	$< .01$
HVC	30%	36%	$< .01$
FCH	41%	42%	NS

Notes:

TRANSLATIONAL APPROACH TO IDENTIFY DOXORUBICIN RESISTANT GENES IN NEUROBLASTOMA

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Purpose:

Neuroblastoma tumors are susceptible to doxorubicin therapy, and drug resistance portends a poor prognosis. To better understand the mechanisms of doxorubicin resistance, we performed a functional genomics screen in yeast to identify genes that mediate doxorubicin resistance. We focused on the yeast doxorubicin resistant gene *DHH1* and its human ortholog *DDX6* because *DDX6* is highly conserved and located within a chromosomal region frequently deleted in neuroblastoma tumors.

Methods:

We performed a genome-wide screen in the *Saccharomyces cerevisiae* diploid deletion collection to identify mutants that confer doxorubicin sensitivity. We then examined the mutants for cell cycle progression defects. Lastly, we examined cell cycle progression following siRNA ablation of *DDX6* in human cells using flow cytometry analysis.

Results:

We identified 376 genes that conferred doxorubicin resistance and found that most were conserved (76%) with human orthologs. This diploid screen found 5-fold more doxorubicin resistant genes, including *DHH1*, than detected in a prior screen in haploid yeast. Since most doxorubicin damage is repaired by recombination using a chromosomal homolog, this suggests that many doxorubicin resistance genes, including *DHH1*, may function as G1-specific repair genes. In support of this mechanism, *DHH1* exhibits severe G1-S phase transition defects following doxorubicin exposure in yeast. Moreover, *DDX6* arrests in G1 after siRNA ablation in human cells, which correlates with the findings in yeast.

Conclusions:

We have found that many yeast genes, including *DHH1*, appear to mediate doxorubicin resistance. One mechanism of doxorubicin resistance is appropriate G1 checkpoint arrest after exposure. Loss of the orthologs of the doxorubicin resistant genes identified in this screen, including *DDX6*, may be used to predict tumor susceptibility. Understanding of these mechanisms may allow the development of tumor drug screening assays and improve the therapy of this childhood tumor.

Notes:

HOW TO APPROACH THE OVARIAN MASS: CAN WE RISK STRATIFY FOR MALIGNANCY?

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Purpose:

With a 10% malignancy rate in pediatric ovarian masses, what factors are helpful in distinguishing those at higher risk and do we risk stratify accordingly?

Methods:

After IRB approval (IRB#022008-095) a 15½ year retrospective review of operative ovarian cases was performed.

Results:

424 patients were identified; mean age 12.5y (1d to 19y). No difference existed between benign (12.5y) and malignant (11.8y) cases. The 1-8 yr age group had highest incidence of malignancies (22%, OR 3.02, 95%CI 1.33-6.86). A chief complaint of mass or previous puberty vs. one of pain had an odds ratio for malignancy of 4.84 and 5.67 respectively (95%CI 2.48-9.45 and 1.60-20.30). Imaging of benign neoplasms had a median size of 8cm (0.9-36cm) compared to malignancies at 14.25cm (6.2-50cm, $p < 0.05$). An ovarian mass size of ≥ 6 cm on preoperative imaging had an odds ratio of 31.4 for malignancy (95%CI 1.90-520.27), compared to using ≥ 5 cm (OR 18.9, 95%CI 1.14-314.6). 89% of masses were benign: normal histology 13% (n=59), benign cysts 42% (n=184), benign neoplasms 34% (n=149). Of 11% malignancies (n=46), the pathologies included: germ cell (52%, n=24), stromal (26%, n=12), epithelial (17%, n=8) and other (4%, n=2). Tumor markers obtained in 75% of malignancies, but were elevated in only 58%. 13% of those sent in benign cases were elevated. Elevated BHCG, AFP and CA 125 were significantly associated with malignancy ($p < 0.02$), but elevated CEA not ($p = 1880$).

Conclusion:

This is the largest reported series of pediatric ovarian masses at a single academic center, and confirms 10% malignancy rate. A mass measurement of ≥ 6 cm on imaging has the greatest odds ratio for malignancy. Tumor markers were not conclusive in all cases but useful for postoperative surveillance.

Notes:

THE LONG-TERM FUNCTIONAL OUTCOMES AND QUALITY OF LIFE OF ADOLESCENTS AND ADULTS WITH HIRSCHSPRUNG'S DISEASE AND ANORECTAL MALFORMATIONS

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Background:

The long-term functional outcomes and quality of life (QOL) of older adolescents and adults with Hirschsprung's disease and anorectal malformations remains largely unknown.

Methods:

A multi-institutional observational study of all patients (current age >12) who underwent surgery for either Hirschsprung's disease (HD) or an anorectal malformation (ARM) was performed. Eligible subjects were mailed questionnaires to assess current bowel function and quality of life. Bowel dysfunction was assessed with fecal incontinence severity index (FISI) and patient assessment of constipation (PAC-SYM) questionnaires. General and disease specific quality of life were measured with the SF-36 v.2, fecal incontinence quality of life (FIQOL) and constipation quality of life (PAC-QOL).

Results:

Seventeen patients completed questionnaires. Mean age was 17.0 (range 12-25). HD patients had mean FISI scores of 3.00, 1.10, 1.89, and 1.90 for gas, mucous, liquid and stool incontinence respectively (range 0-5 reflecting increasing severity). ARM patients had FISI scores of 3.17, 0.71, 1.17, 2.50, respectively. Constipation was less frequent with mean PAC-SYM global scores of 0.42 for HD subjects and 0.49 for ARM patients (range of severity 0-4). General QOL as measured by the SF-36 v.2 suggested impaired functioning for ARM patients with physical component (PC) norm-based score of 47.5 and mental component (MC) score of 45.8 when compared to general population norms HD patients had PC score of 50.0 and MC of 50.2 scores that were comparable to general population norms (Figure 1). Disease specific quality of life measures (FIQOL & PAC-QOL) were not found to be significantly different between patients with HD and ARM.

Conclusion:

Global QOL appears to be more diminished for ARM patients than HD patients when compared to the general population. This difference, however, is not present when considering disease specific QOL which may reflect life-long adaptive strategies in the management of incontinence and constipation.

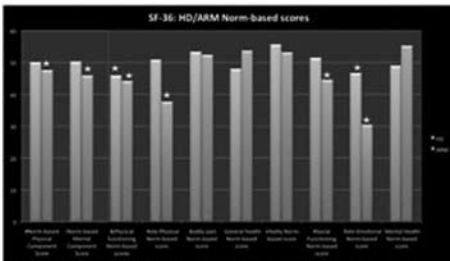


Figure 1. SF-36 Norm-based scores for HD & ARM patients. Stars signify functional impairment when compared to general population norms.

Notes:

MRI GUIDED LAPAROSCOPIC ASSISTED ANORECTOPLASTY FOR IMPERFORATE ANUS

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Purpose:

MRI has been well described as a modality for evaluation of a failed anorectal pull-through procedure for imperforate anus. To the authors' knowledge, intraoperative MRI has not been previously used to guide a laparoscopic assisted anorectoplasty (LAARP). We propose that such a procedure would assure anatomically correct placement of the pulled-through rectum.

Methods:

Three male patients with imperforate anus and a prostatic urethral fistula underwent an MRI guided LAARP in an operative MRI suite. The patient's ages ranged from five to six months of age at the time of their pull-through procedure. Preoperative MRIs with mineral oil within the distal colostomy were performed on all patients to document the anatomy of the rectourethral fistula and its relationship to the parasagittal and vertical muscle complex. The perineum was pierced with an MRI compatible needle at the central portion of the parasagittal muscle complex as determined by a direct muscle stimulator. Further incremental advancement of the needle within the muscle complex was guided by serial MRIs in axial, coronal and sagittal planes until the levator floor was penetrated and the peritoneal cavity was entered. LAARP was then completed.

Results:

Completion MRI demonstrated placement of the pulled through segment in a central location thru the length of the muscle complex. Serial MRIs performed intraoperatively during advancement of the localization needle demonstrated a curved path of the vertical fibers. Attempts to non-incrementally advance the needle in a straight plane resulted in a breach of the vertical muscle complex or eccentric placement of the needle.

Conclusions:

MRI guided LAARP results in anatomically correct placement of the rectum within the vertical muscle complex. Straight needle advancement techniques in LAARP could result in a deviation of the pulled through rectum from the central muscular path. Further follow up will be required to demonstrate functional advantage.

Notes:

TOPICAL MITOMYCIN-C FOR THE TREATMENT OF ANAL STENOSIS

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Purpose:

Anal stenosis is a well-known and feared consequence of anorectal surgery. Daily dilations are often prescribed in the immediate postoperative period in order to avoid stricture of the anus. Nonetheless, stenosis may still occur and, particularly in older children, may require serial dilations under anesthesia. Topical mitomycin-C has been found to be effective in the treatment of esophageal strictures. In this paper, we review our experience with topical mitomycin-C as an adjunct to anal dilation for children with anal stenosis.

Methods:

Cases of children with anal stenosis who were treated with both dilation and the application of topical mitomycin-C between 2000 and 2008 were analyzed retrospectively. Anal diameter was measured with Hegar bougies both before and after treatment with mitomycin-C. For each treatment cottonoid swabs soaked in mitomycin-C (concentration 1mg/ml) were placed on the anal mucosa for two to six minutes. Treatment success was defined by clinician satisfaction, improvement in anal size and need for repeat intervention.

Results:

Ten children with anal stenosis who underwent anal dilation with mitomycin-c were identified. Children's mean age was 30 months. Seven children underwent initial surgery for anorectal malformations, two for Hirschsprung's disease, and one for familial polyposis. All children underwent only one application of mitomycin-C. Average increase in anal size after application was 5.78 mm (+ 3.4 mm) of diameter. Clinicians expressed overall satisfaction with their use of mitomycin-C. Only one child required repeat surgical intervention for stenosis after treatment with mitomycin-C. No complications were associated with the use of mitomycin-C.

Conclusions:

All children treated with mitomycin-C showed satisfactory improvement in their anal size after a single application. Further, the application of mitomycin-C in our population was straightforward and safe. Therefore, we advocate its use as an adjunct to bougie dilation in the treatment of severe anal stenosis.

Notes:

DEVELOPMENT AND IMPLANTATION OF A BIOARTIFICIAL ANAL SPHINCTER

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Purpose:

Current treatment of imperforate anus is fraught with numerous problems, and there are no long-term ideal solutions for such patients. Although loss of continence is multifactorial, the integrity of the internal anal sphincter (IAS) has particular significance to continence. The aim of this study was to develop a bioengineered 3-dimensional IAS from sphincteric smooth muscle cells. We hypothesized that: 1-Bioengineered IAS constructs undergo vascularization following implantation into a mouse. 2-Bioengineered IAS constructs will retain cellular integrity and normal functionality post-implantation.

Methods:

Bioengineered 3-dimensional physiologic IAS rings were created from isolated mouse IAS smooth muscle cells (C57 BL/6J). After 3 weeks of *in vitro* growth, IAS constructs were surgically implanted into the subcutaneous tissue of syngeneic mice, and treated with either FGF-2 (0.25 ug/daily) (N=3), or saline (N=2) using a micro-osmotic pump. IAS were harvested between 15 and 21 days, and tested *in vitro* for functional contraction/relaxation graded from poor(0) to excellent(4). Tissues were processed for viability assessment (H&E; scored:0-6); neovascularization (anti-Von Willebrand Factor (VWF); scored:0-4 according to the intensity and vessel number); and evidence of neuro in growth (anti-NeuN; present or absent)

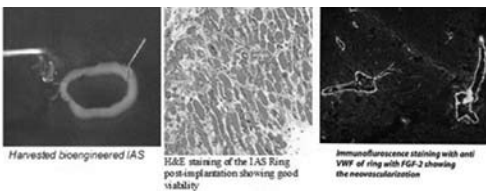
Results:

Results of implanatation are shown in Table and Figure. No rejection was noted in any of the rings. Use of FGF-2 resulted in normal viability, neovascularization, functionality. Interestingly, neuro in-growth was found in all FGF-2 treated IAS constructs.

Conclusions:

The use of a bioengineered IAS can be successfully implanted, and with growth factor treatment undergoes successful neovascularization, retains cellular integrity and function. This approach may become a viable option for patients suffering from fecal incontinence

Group	Neovascularization (VWF)	Viability (H&E) Mean (range)	Funtion: Mean (range)	Neuro in-growth
FGF-2	Grade 4 intensity with vessels number ≥ 4	5 (4-6)	Relaxation: 3.5 (3-4) Contraction: 3.9 (3.5-4)	Present (3/3)
Saline control	Grade 2 intensity with vessels number ≤ 3	3 (2-4)	Relaxation: 2.6 (2-3) Contraction: 2.9 (2.5-3.5)	Absent (2/2)



Notes:

REPEAT ABDOMINAL CT SCANS AFTER PEDIATRIC BLUNT TRAUMA: MISSED INJURIES, EXTRA COSTS, & UNNECESSARY RADIATION EXPOSURE

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Purpose:

We hypothesized that pediatric blunt trauma patients, initially evaluated at non-trauma centers with abdominal computed tomography scans (CT), often receive repeat scans after transfer. This study was designed to quantify this phenomenon, assess consequences, and elucidate possible causes.

Methods:

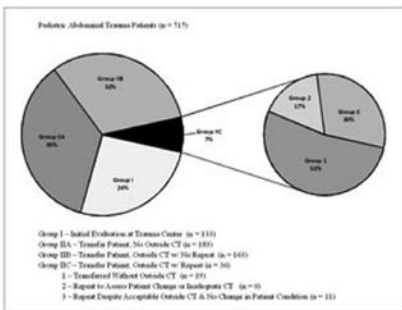
IRB-approved, retrospective chart review of pediatric blunt abdominal trauma patients (age < 16 yrs) treated at a level I trauma center from March 2002 through December 2007, who were evaluated with abdominal CT at the trauma center or a referring facility.

Results:

521 patients met study criteria, with 6 patients excluded due to inability to verify outside records. For the remaining 515 patients, 382 (74%) were initially seen at outside emergency rooms before transfer. 199 of those 382 (52%) underwent abdominal CT prior to transfer. 36 of those 199 (18%) underwent repeat CT scanning at our level I trauma center. Of these 36 patients, 19 (53%) were transferred without their outside CT's. 9 of these 19 (47%) had significant abdominal injuries. Of the remaining 17, 6 (17%) had repeat scans to assess changes in vital signs/patient condition or due to inadequate outside imaging. The remaining 11 (30%) were repeated despite an acceptable outside CT and no change in patient condition. Only 1 of 11 resulted in changed management. Additional radiation delivered from these repeat scans totaled 180 mSv (milliSieverts) and additional charges totaled over \$110,000. There was a clear trend toward increased repeat scanning (6.7% in 2002, to 16.7% in 2007).

Conclusions:

Abdominal CT scans, for evaluation of pediatric blunt trauma, are frequently repeated after transfer from outside hospitals. Yet without diagnostic information gained from these repeat scans, almost half may have had missed injuries, with resultant morbidity. Over 80% of repeat scanning is potentially preventable, with better education of transport personnel (paramedics and EMTs) and emergency department physicians.



Notes:

LONG-TERM OUTCOMES OF PEDIATRIC SPLENIC INJURY IN CALIFORNIA

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Introduction:

Long-term outcomes of nonoperative and operative managements for pediatric splenic injury are lacking. The purpose of this study was to investigate trends and outcomes in the management of pediatric splenic injury at a state level.

Methods:

From 1999-2006, patients ≤ 18 y/o with splenic injuries were extracted from California's Patient Discharge Database after IRB approval (n=5,089). Patient demographics, injury grade, immediate and delayed operations, readmissions, and complications were analyzed. Complications consisted of delayed splenic rupture, post-traumatic splenic pseudocyst, intestinal obstruction, and post-splenectomy sepsis.

Results:

A total of 5,089 splenic injuries were identified, and 2,705 patients (53%) were followed for one-year after injury. The severity of injuries did not change over time. There was a steady decrease in the rate of immediate operations from 23% in 1999 to 15% in 2006 (p $<$ 24 hours after admission, readmissions for operations and complications remained low and unchanged. Four patients (0.2%) suffered delayed splenic rupture from grade 3-4 splenic injuries within 3 weeks of discharge after nonoperative management and were treated with readmission splenectomies. In contrast, 6 were readmitted for bowel obstruction after operative management, where 2 required bowel resections. There were no differences when comparing the readmission (1 vs. 0.4%, p=0.08) and readmit operation (0.3 vs. 0.3%, p=0.82) rates between patients who had splenic operation versus those who did not. Of the patients who were readmitted, 29% did not present to the hospital that managed their initial injury.

Conclusion:

The steady increase in the utilization of non-operative management for pediatric splenic injuries in California has occurred without a concurrent increase in delayed operations or readmissions. Long-term complications from contemporary management of pediatric splenic injuries are rare. The rate of delayed splenic rupture was 0.2% and occurred only in grade 3-4 injuries within 3 weeks of discharge.



Notes:

REFLECTANCE SPECTROMETRY FOR REAL-TIME HEMOGLOBIN DETERMINATION OF PLACENTAL VESSELS DURING ENDOSCOPIC LASER SURGERY FOR TTTS

Sean Curran, John McMurdy, PhD, John McMurdy, PhD, Stephen R. Carr, MD, Christopher S. Muratore, MD, Babara M. O'Brien, MD, Gregory P. Crawford, PhD, Francois I. Luks, MD, PhD
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Purpose:

In endoscopic fetal surgery for twin-to-twin transfusion syndrome (TTTS), mapping out placental vessels is of utmost importance. We have developed a non-invasive technique to determine hemoglobin content through spectral analysis of diffusely reflected broadband visible illumination from individual blood vessels.

Methods:

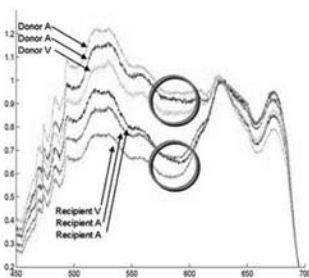
The reflection of an incoming xenon endoscopic light source was captured through a 500 μm fiber-optic, coupled to a fixed grating spectrometer (2 nm resolution). A 450-700 nm wavelength range was used for analysis. The concept of noninvasive hemoglobin determination was based on the knowledge that hemoglobin and oxyhemoglobin reflectance curves are highly similar at 400-650 nm, and that the ratio of reflectance intensity from 630 and 590 nm correlates with total hemoglobin concentration. Three data capturing methods were studied: 1) fixed-image spectrum capture with fiber aimed at (but not touching) center of a vessel, 2) no-touch scanning perpendicular to the vessel and dynamic spectra capture, and 3) dynamic spectra capture and analysis of the intensity peak during brief vessel touch. The study was IRB-approved.

Results:

Seven controls (laparoscopic operations in otherwise healthy children) were enrolled. Four vessels were analyzed in each case. The brief-touch technique with intensity peak analysis yielded the most reproducible results between multiple vessels in the same patient while the other two collection methods showed significantly higher variability. Spectrometry was also applied to three TTTS patients: the (anemic) donor and (polycytemic) recipient twin fetuses could be differentiated, with excellent correlation between vessels (arteries and vein) of the same fetus (figure).

Conclusions:

It is possible to differentiate donor- from recipient placental vessels by spectral analysis of the reflected light through the endoscope, using a non-invasive and real-time method. This may improve the accuracy of endoscopic laser ablation of placental vessels in TTTS, and may allow instant endoscopic hemoglobin determination for laparoscopic procedures as well.



Notes:

PRENATAL URINARY MATRIX METALLOPROTEINASE PROFILING AS A POTENTIAL DIAGNOSTIC TOOL IN FETAL OBSTRUCTIVE UROPATHY

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¹Children's Hospital Boston, Department of Surgery, Boston, MA, USA, ²Children's Hospital Boston, Vascular Biology Program, Boston, MA, USA, ³Children's Hospital Boston, Department of Radiology, Boston, MA, USA

Purpose:

The diagnostic evaluation, patient stratification, and prenatal counseling for congenital obstructive uropathy remain sub-optimal. Matrix metalloproteinase (MMP) expression profiles are emerging as a valuable diagnostic tool in assorted disease processes. We sought to determine whether congenital obstructive uropathy impacts MMP expression in fetal urine.

Methods:

After IACUC approval, fetal lambs (n=25) were divided in two groups at 94-100 days gestation (term=145 days): group I (n=12) underwent a sham operation and group II (n=13) underwent creation of a complete urinary tract obstruction via ligation of the urachus and either the urethra (in males), or the bladder outlet (in females). Animals were followed by serial post-operative ultrasounds. Survivors from both groups were sacrificed at comparable times post-operatively and submitted to multiple analyses. Gelatin zymography panels were performed on fetal urine for 4 MMP species with 10 different molecular weights. Statistical analysis was by the Fisher's Exact Test, with significance set at $p < 0.05$.

Results:

Overall fetal survival was 80% (20/25); 2 survivors were excluded from analysis due to infection. Post-operative serial ultrasounds confirmed obstructive uropathy in all survivors in group II. At necropsy, all obstructed animals showed bilateral hydronephrosis and bladder dilation on gross inspection and evident renal dysplasia on histology. Various statistically significant differences in MMP expression between the two groups were identified. The following profiles were present only in obstructed animals: any MMP other than 63 kDa and 65 kDa ($P=0.009$); any MMP other than MMP-2 ($P=0.029$); two or more MMPs excluding MMP-2s (0.029); and three or more MMPs ($P=0.029$).

Conclusions:

In an ovine model, limited matrix metalloproteinase expression is present in the urine of normal fetuses. Fetal obstructive uropathy impacts urinary matrix metalloproteinase expression in various distinguishable patterns. Prenatal urinary matrix metalloproteinase profiling may become a practical and valuable diagnostic tool in the evaluation of congenital obstructive uropathy.

Notes:

ACCURACY OF COMPUTED TOMOGRAPHY IN PREDICTING APPENDICEAL PERFORATION

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

Introduction:

Some surgeons use initial non-operative management for patients who present with perforated appendicitis. These strategies depend on accurately delineating perforation by computed tomography (CT). Since 2005, we have employed an evidence based definition for perforation as a hole in the appendix or fecalith in the abdomen which has been shown to clearly separate those with a high risk of abscess from those without. To clarify the consistency of CT in the diagnosis of perforation, 6 surgeons and 2 radiologists were given a test of 200 blinded CT scans to identify which patients would have an intraoperative diagnosis of perforation.

Methods:

A junior (resident1) and senior (resident2) surgical resident, a junior (radiologist1) and senior (radiologist2) staff radiologist, and four attending pediatric surgeons with 3-30 years experience (surgeons1-4, respectively) reviewed 200 CT scans of pediatric patients who had undergone a laparoscopic appendectomy. They were blinded to the results and outcome. The reviewers were asked to decide on perforated or non-perforated according to our intraoperative definition for each study.

Results:

The results of each reviewers interpretation of the CTs is listed below in Table 1. In total the reviewers were correct 72% of the time. The specificity varied on how many patients the examiners were willing to call perforated, but as can be seen from the specificity, there was a substantial risk of calling a patient perforated when there was no perforation at operation.

Conclusions:

This study shows that triage of patient care based on a preoperative diagnosis of perforation may be an imprudent practice that will subject many patients to an unnecessary prolonged course of care.

Table 1. Outcomes of CT interpretation

	Percent Correct	Sensitivity	Specificity
Resident 1	71	52	89
Resident 2	74	66	83
Surgeon 1	76	75	77
Surgeon 2	70	63	76
Surgeon 3	70	64	75
Surgeon 4	66	53	62
Radiologist 1	81	78	83
Radiologist 2	68	45	88

Notes:

LAPAROSCOPIC APPENDECTOMY UPON PRESENTATION VERSUS INTERVAL APPENDECTOMY FOR PERFORATED APPENDICITIS WITH ABSCESS: A PROSPECTIVE, RANDOMIZED TRIAL

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Purpose:

Perforated appendicitis is a common condition in children which may be complicated by a well-formed abscess. Conservative management with percutaneous drainage of the abscess followed by intravenous antibiotics usually allows for an uneventful interval appendectomy. While this strategy has become well accepted, there have been no published data comparing drainage/interval appendectomy to appendectomy upon presentation. Therefore, we conducted a randomized trial between these 2 management strategies.

Methods:

After IRB approval (IRB# 06 11-164), children found to have a well-defined abdominal abscess by CT imaging were randomized to laparoscopic appendectomy upon admission or intravenous antibiotics with percutaneous drainage of the abscess (when possible) and subsequent interval laparoscopic appendectomy 10 weeks later. This was a pilot study by design with a sample size of 30 chosen based on our recent volume.

Results:

On presentation, there were no differences between groups in age, weight, body mass index, gender distribution, temperature, leukocyte count, number of abscesses, or greatest 2-dimensional area of abscess in axial view. There were no conversions to open in either group. Outcome variables are listed in Table 1.

Conclusions:

While initial laparoscopic appendectomy trends toward requiring longer operative times, there appears to be no advantages between these strategies in terms of total hospitalization, recurrent abscess rate or total charges.

Table 1: Laparoscopic Operation vs Non-operative Management and Interval Appendectomy

	Early Operation	Interval Appendectomy	P Value
Operation Time (Minutes)	63.0 +/- 32.5	43.5 +/- 26.0	0.08
Total Length of Stay (Days)	6.7 +/- 4.1	7.1 +/- 7.5	0.86
Recurrent Abscess	20.0%	26.7%	0.99

Notes:

OPERATIVE MANAGEMENT OF INTRACTABLE CONSTIPATION IN CHILDREN

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Purpose:

Intractable constipation in children is an uncommon but debilitating condition. When medical therapy fails, the optimal surgical approach has not been clearly defined. We reviewed our experience with operative management of intractable constipation to identify predictors of success and to compare outcomes following 3 surgical approaches – antegrade continence enema (ACE), enteral diversion, and primary resection.

Methods:

Retrospective review of pediatric patients undergoing ACE, diversion, or resection for intractable, idiopathic constipation from 1994-2007. Satisfactory outcome was defined as minimal fecal soiling and passage of stool at least every other day (ACE, resection) or functional enterostomy without abdominal distention (diversion).

Results:

Forty-five patients (range=4 months to 26 years, mean=9 years) were identified. Sixteen patients underwent ACE, 20 underwent primary diversion (4 ileostomy, 16 colostomy), and 9 had primary colonic resections with a satisfactory outcome in 63%, 90%, and 22%, respectively. Of the 20 patients diverted, 16 had intestinal continuity reestablished at a mean of 27 months post-diversion, with 15 of these having a satisfactory outcome at an average follow-up of 49 months. Four patients underwent closure of the enterostomy without resection, while the remainder underwent resection of dysmotile colon based on preoperative colonic manometry. Of patients undergoing ACE, age <12 years was a predictor of success, while preoperative colonic manometry was not. Repeat manometry 1 year post-ACE showed improvement in all patients tested. On retrospective review, patient compliance emerged as contributing to ACE failure.

Conclusions:

ACE and enteral diversion are effective initial procedures in the management of refractory constipation. Greater than 90% of diverted patients have an excellent outcome with the reestablishment of intestinal continuity. Colon resection should not be offered as an initial therapy as it is associated with nearly 80% failure rate and the need for additional surgery.

Notes:

SUBTOTAL SPLENECTOMY FOR HEREDITARY SPHEROCYTOSIS

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Background:

The standard of care for the surgical treatment of hereditary spherocytosis (HS) is shifting from total splenectomy to subtotal splenectomy in the hope of retaining phagocytic function.

Methods:

After IRB approval was obtained we retrospectively reviewed the records of 21 consecutive children with a mean age of 11 years who underwent subtotal splenectomy for the treatment of HS.

Results:

Indications for subtotal splenectomy included anemia, fatigue, pain, growth failure, and sequestration crises. In 19 cases the lower pole was preserved, and in two the upper pole was preserved. Approximately 15-20% of the spleen was preserved in all cases. Only one complication occurred, a hemodynamically stable post-operative hemorrhage that required the transfusion of 2 units of blood. As a result of the subtotal splenectomy, the mean absolute hematocrit increased by 7%, the mean hemoglobin increased by 2.5 g/dL, and the mean absolute reticulocyte percentage decreased by 6% in the short term (median of 4 months). In the long term (median of 3 years) the mean absolute hematocrit increased by 7.3%, the mean hemoglobin increased by 2.8 g/dL, and the mean absolute reticulocyte percentage decreased by 3.2%. No patients required subsequent transfusion for anemia or experienced sequestration crisis. Some splenic regrowth was observed, but without recrudescence of pre-operative anemia. No patients required a subsequent total splenectomy. Howell-Jolly bodies increased in the short term and remained stable in the long term for those with available smears to review. Of the 13 patients who did not undergo simultaneous cholecystectomy, none required a subsequent cholecystectomy for biliary disease.

Conclusion:

Subtotal splenectomy is an effective surgical treatment for HS that results in the relief of anemia, yet potentially preserves some filtering capacity of the spleen.

Notes:

PREDICTING RESPONSES TO SPLENECTOMY IN CHILDREN WITH IMMUNE THROMBOCYTOPENIC PURPURA

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Purpose:

Predicting the response to splenectomy in children with immune thrombocytopenic purpura (ITP) remains elusive as clear prognosticators have not been identified. The purpose of this study is to evaluate quantifiable preoperative predictors of splenectomy outcome in children with ITP.

Methods:

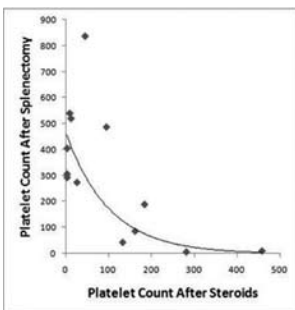
The charts of 19 children who underwent splenectomy for ITP were retrospectively reviewed. Platelet responses to treatment are reported as best response, and they are categorized as follows: non-response (NR) $<50,000$; partial-response (PR) $50,000-150,000$; or complete-response (CR) $>150,000$. Data are recorded as mean \pm standard deviation.

Results:

After splenectomy, thirteen patients had CR (platelets= $406,000 \pm 174,000$), three patients had PR (platelets= $64,000 \pm 19,000$), and three patients had NR (platelets= $20,000 \pm 20,000$). No correlation existed between CR to splenectomy and any of the following: age, platelet count at diagnosis, last platelet count prior to splenectomy, platelet count on post-operative day one, or responses to pre-operative IVIG, WinRho, or Rituximab. However, NR or PR to steroid therapy was correlated with CR to splenectomy ($p=0.04$), and platelet response to steroids was inversely related to platelet response to splenectomy (see graph). Patients with platelets $<100,000$ after steroids were more likely to have CR after splenectomy (OR= 57, $p=0.02$). Additionally, there was a shorter interval from diagnosis to splenectomy for patients with CR as compared to PR or NR to splenectomy (20 ± 10 months v. 53 ± 46 months, $p<0.05$).

Conclusions:

Pre-operative non-responsiveness to steroids predicts complete response to splenectomy for ITP in children. Conversely, children whose platelets rise above $100,000$ after steroid treatment are at high-risk for failure of splenectomy, thus challenging the widely-held notion that steroid-responsiveness portends a favorable outcome after splenectomy. Furthermore, children with complete response to splenectomy had shorter intervals from diagnosis to operation, suggesting that steroid non-responsive and partially-responsive children should be considered for early splenectomy.



Relationship of platelet counts after splenectomy (y-axis) to best platelet counts after steroid therapy (x-axis). Solid line represents the linear regression of the data points.

Notes:

LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING (LAGB) FOR ADOLESCENTS IN THE FDA-IDE TRIAL: AN INTERIM REPORT OF WEIGHT, METABOLIC AND QUALITY OF LIFE OUTCOME.

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A prospective longitudinal trial of the safety/efficacy of LAGB for morbidly obese adolescents aged 14-17 using a LAP-BAND® IDE from the FDA was initiated in 2005. The intermediate outcome for patients with complete 12 months of follow up was analyzed. Our prospective database collection was IRB-approved. Baseline characteristics and outcome were analyzed in patients enrolled between 3/2005 and 6/2007 using descriptive and comparative statistics (Chi square and Wilcoxon signed rank tests). Follow up at >24 months was excluded from the analysis because of the small group size.

Methods:

Male:Female and Hispanic/African American/ Caucasian patient ratios were 1:3 and 1/1.3/4.7 respectively. Baseline (mean+/-SD) BMI was 50+/-10 kg/m² (range 39-74) and excess weight was 178+/-53 IBS Comorbidities included clinical depression (18% of the patients); hypertension (35%); dyslipidemia (80%); insulin resistance (90%) and metabolic syndrome (95%) as predictors for cardiovascular diseases and diabetes; and histologic non alcoholic steatohepatitis (88% of 17 liver biopsies), a risk factor for chronic liver disease. Mean follow up was 26+/-9 months. At 6, 12 and 18 months, % excess weight loss (%EWL) was 26+/-17% (n=20), 34+/-22% (n=18) and 41+/-27% (n=12) respectively. At 12 and 18 months of follow up, hypertension was resolved in all patients (p = 0.01), metabolic syndrome in 61% and 80% respectively (p< 0.001), along with significant improvement in dyslipidemia (p=0.03) and Pediatric Quality of Life scores (p≤0.05 and 0.003 respectively). At 12 months, 5/18 patients had <20%EWL, yet dyslipidemia and metabolic syndrome were resolved in 2/5 patients. At 18 months, 2/12 patients had <20%EWL.

Results:

Morbidly obese adolescents exhibit early baseline metabolic abnormalities. At intermediate follow up, weight loss led to resolution or improvement of major obesity-related comorbidities in the majority of the patients. **(continued)**evaluation of the long term efficacy of LAGB as a surgical adjunct to a comprehensive obesity treatment program is ongoing.

Notes:

EFFECTS OF GLUTAMINE-SUPPLEMENTED PARENTERAL NUTRITION ON LIVER FUNCTION IN SURGICAL INFANTS: RESULTS OF A RANDOMISED CONTROLLED TRIAL.

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Purpose:

To determine whether glutamine supplementation of parenteral nutrition (PN) affects liver function.

Methods:

An ethically-approved double-blind multi-centre randomised controlled trial was performed in surgical infants <3 months old. Treatment group received 0.4g/kg/day alanyl-glutamine PN supplementation until full enteral feeding was reached. The placebo group received isonitrogenous isocaloric PN. Incidence of liver dysfunction was recorded prospectively as a secondary outcome: the number of patients with one or more episodes of cholestasis, hypoalbuminemia, hyperammonemia and impaired liver function was considered (see Table for definitions). Data were analysed by binary logistic regression analysis, adjusting for minimisation criteria used in the randomisation.

Results:

174 patients were randomised (87 glutamine, 87 placebo). There was no difference between glutamine and placebo group in incidence of cholestasis, hyperammonemia or impaired liver function (Table).

Table 1: Effects of glutamine and placebo on incidence of liver dysfunction

	Number of patients n/N (%)	Number of patients n/N (%)	Odds ratio glutamine vs. placebo [95% confidence interval]	p-value
	Glutamine	Placebo		
Cholestasis (conjugated bilirubin ≥ 2 mg/dl)	34/84 (40.5)	25/82 (34.1)	1.6 [0.8, 3.4]	0.18
Hyperammonemia (≥ 100 μ mol/l)	17/68 (25.0)	17/69 (24.6)	0.9 [0.4, 2.1]	0.73
Impaired liver function (AST ≥ 60 OR ALT ≥ 45 U/l)	29/85 (34.1)	26/79 (32.9)	1.1 [0.5, 2.2]	0.90
Hypoalbuminemia (≤ 2 g/dl)	21/83 (25.3)	8/79 (10.1)	4.1 [1.4, 11.6]	0.009
*not all patients had each test performed				

There was, however, a significantly increased risk of hypoalbuminemia in patients receiving glutamine. As decreased serum albumin is associated with infection, we repeated the regression analysis, adjusting for patients who had one or more episodes of sepsis. Sepsis was associated with increased incidence of hypoalbuminemia (odds ratio 8.2 [2.3, 28.7], $p=0.001$), but the effect of glutamine remained significant in this model (odds ratio 4.4 [1.4, 13.4], $p=0.01$). Six patients receiving glutamine had hypoalbuminemia without infection, compared with none in the placebo group ($p=0.011$, Chi-squared test).

Conclusions:

Glutamine neither impairs nor improves liver function during PN in surgical infants, and does not cause hyperammonemia. However, hypoalbuminemia occurs in 25% of patients receiving glutamine. This could be due to impaired albumin synthesis during infection, possibly because intake of other amino acids is decreased in order to make the infusions isonitrogenous.

Notes:

PARENTERAL FISH OIL AS MONOTHERAPY PREVENTS ESSENTIAL FATTY ACID DEFICIENCY IN PARENTERAL NUTRITION DEPENDENT PATIENTS

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Purpose:

Due to the low levels of linoleic acid and alpha-linolenic acid, the use of fish oil-based lipid emulsions as the sole source of fat for patients receiving parenteral nutrition has raised concerns for the development of essential fatty acid deficiency, hindering its adoption into clinical practice. The purpose of this chart review was to examine the fatty acid profiles of patients without any enteral caloric intake and who were completely dependent on parenteral nutrition and a fish oil-based lipid emulsion for onset of essential fatty acid deficiency.

Methods:

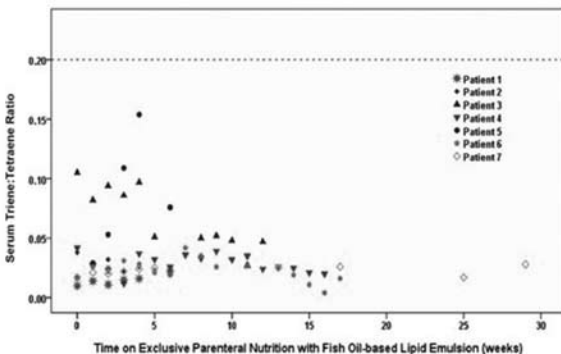
After IRB approval, prospectively collected data from seven patients was reviewed looking for evidence of essential fatty acid deficiency, defined as an elevated triene:tetraene ratio (mead acid/arachidonic acid >0.2). All patients received parenteral nutrition with a fish oil-based lipid emulsion at $1\text{g}\cdot\text{kg}^{-1}\cdot\text{day}^{-1}$ of as the sole source of fat calories for at least 1 month. The fish oil-based lipid emulsion as monotherapy was used under a compassionate use protocol approved by the FDA.

Results:

Median gestational age at the time of birth was 35 weeks (range 24 to 36 weeks), and median age at the start of treatment was 1.9 months (range 0.8 to 37 months). After a median time of 2.8 months (range 1.1 to 6.7 months) on exclusive parenteral nutrition and fish oil-based lipid emulsion none of the patients developed biochemical evidence of essential fatty acid deficiency (see figure). Median direct bilirubin levels at base line were 4.2 mg/dL (range 2.5 to 9.6 mg/dL), and normalized in 6 patients (median 0.5 mg/dL; range 0.1 to 8.9 mg/dL) before advancement to enteral feeds.

Conclusions:

We conclude that, when dosed appropriately, fish oil-based lipid emulsions contain sufficient amounts of essential fatty acids to prevent essential fatty acid deficiency in patients completely dependent on parenteral nutrition.



Notes:

REDUCTION IN STANDARD INTRAVENOUS FAT EMULSIONS FOR PATIENTS WITH PARENTERAL NUTRITION CHOLESTASIS: AN EFFECTIVE MODE OF TREATMENT

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Purpose:

Long-term parenteral nutrition (PN) is associated with PN-associated cholestasis (PNAC). Standard soybean-based intravenous fat emulsion (IFE) has been hypothesized to be a causative factor for PNAC. This study evaluated the use, efficacy, and adverse effects of IFE reduction (IFER) in the management of PNAC.

Methods:

Between August 2005-September 2007, we prospectively treated all neonates who developed a direct bilirubin > 2.5mg/dL in our NICU by decreasing IFE delivery to 1g/kg/dose, given twice weekly; n=17). The control arm consisted of patients matched for diagnosis, gestational age, and birth weight between August 2003-July 2005 (3g/kg/day; n=17). Weekly bilirubin levels were compared for progression of PNAC using linear mixed modeling analyses. Models were fitted using Proc Mixed in SAS. We carefully controlled for patient age at initiation of IFER between treatment and control groups. Patient demographics and adverse effects (e.g., essential fatty acid deficiency, EFA) were assessed monthly. Significance was set at $P < 0.05$.

Results:

The figure shows modeling curves of total bilirubin levels in IFER and control groups. The estimated slope for the control group was slightly positive but not significantly different from zero (estimated slope=0.011, $p=0.802$); whereas the estimated slope for the IFER group was significantly negative (estimated slope= -0.097, $p=0.041$). Therefore, despite the IFER group starting with higher total bilirubin levels, a progressive reduction was seen, falling below control levels by 3 to 4 weeks. EFA deficiency became abnormal in 5 of 17 IFER patients (triene:tetraene ratio > 0.05). Resolution of EFA deficiency was achieved with addition of a third day of IFE per week. No observed difference in the rate of sepsis or mortality was observed between the two groups.

Conclusions:

We conclude reduction of IFER for patients with PNAC is beneficial in the treatment of PNAC. Monitoring of EFA levels is critical to ensure patient safety.

Notes:

THE RELATIONSHIP BETWEEN BIOPSY PROVEN PARENTERAL NUTRITION ASSOCIATED LIVER FIBROSIS AND BIOCHEMICAL CHOLESTASIS IN CHILDREN WITH SHORT BOWEL SYNDROME

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Purpose:

To determine the frequency of biochemical cholestasis (direct bilirubin (DB) \geq 2mg/dl) in children with short bowel syndrome and biopsy proven parenteral nutrition (PN) associated liver disease, and to define predictive factors for the degree of hepatic fibrosis.

Methods:

Following IRB approval, a retrospective review was conducted of patients followed by two multidisciplinary intestinal rehabilitation clinics between January 1st 2000 and September 30th 2008. Inclusion criteria were exposure to parenteral nutrition (>30 days) and having undergone a liver biopsy. Liver biopsies were graded from 0-3 based upon degree of fibrosis in the pathology report. The most recent DB within 10 days prior to biopsy was recorded.

Results:

A total of 64 children underwent 83 liver biopsies. The most common diagnoses included necrotizing enterocolitis (34%), gastroschisis (23%) and intestinal atresia (12.5%). Median age at biopsy was 5.3 months with a median duration of PN of 4.6 months (range:1-104 months). 70.3% of patients had a history of exposure to Omega-3 fats. 89% (74/83) of liver biopsies demonstrated some degree of fibrosis (fibrosis scale 1-3) while 9.6% (8/83) had evidence of cirrhosis. 83% of biopsies without fibrosis and 55% of biopsies with fibrosis were obtained in patients without evidence of biochemical cholestasis (p=0.20). 3 of the 8 patients with cirrhosis on liver biopsy (37%) had no evidence of biochemical cholestasis. Univariate analysis identified gestational age (p=0.05), history of exposure to IV omega-3 fats (p=0.007), and length of PN treatment (p=0.03) as variables significantly associated with the degree of liver fibrosis.

Conclusions:

In children with short bowel syndrome, biochemical cholestasis does not reflect the presence or degree of histologically confirmed parenteral nutrition-associated liver fibrosis. Careful follow-up, combined with further refinement of hepatoprotective strategies, may be warranted in this patient population.

Notes:

REVERSAL OF INTESTINAL FAILURE-ASSOCIATED LIVER DISEASE IN INFANTS AND CHILDREN ON PARENTERAL NUTRITION: EXPERIENCE WITH 88 PATIENTS AT A REFERRAL CENTER FOR INTESTINAL REHABILITATION

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Purpose:

Intestinal Failure-Associated Liver Disease (IFALD) complicates the treatment of children with intestinal failure (IF) receiving parenteral nutrition (PN). We hypothesized that prevention or resolution of IFALD was possible in most children and that this would result in improved outcomes.

Methods:

We reviewed prospectively gathered data on all children referred to the intestinal rehabilitation and transplantation center at our institution. Total bilirubin level (TB) was used as the marker for IFALD. Patients were grouped based on TB at referral and at subsequent visits. Standard treatment consisted of cycling of PN, limiting lipid infusion, enteral stimulation, use of ursodeoxycholic acid and surgical intervention when necessary. Outcomes such as mortality, dependence on PN, and need for transplantation were assessed. Statistical analysis was performed using Fisher's exact test.

Results:

Eighty-eight patients with intestinal failure and on PN were treated at our center from 2003-2008. Median age at referral was 5 months (1 wk – 22 yrs). Prematurity was a complicating factor in 58 patients and necrotizing enterocolitis was the most common diagnosis. Seventy-seven children had short bowel syndrome while the remaining 11 had extensive motility disorders. Ninety-seven percent of children required significant alteration of their PN administration. At referral, 75 of 88 children had $TB \geq 2.0$ mg/dL and 13 had $TB < 2.0$ mg/dL. TB normalized in 65 of 75 children with elevated TB at referral and TB remained elevated in 10. Normalization of TB was associated with a mortality of 4.6% and transplantation was needed in 6%. Conversely, when TB remained elevated, mortality was 60% ($p=0.001$ vs. TB normalized) and transplantation occurred in 90% due to failure of surgical and medical rehabilitation.

Conclusions:

A majority of children referred for treatment of IF have IFALD. A dedicated IF rehabilitation program can reverse IFALD in many children and this is associated with improved outcome.

Notes:

PATCH REPAIR IS ASSOCIATED WITH SIGNIFICANT MORBIDITY AND MORTALITY IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH)

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Purpose:

Infants with CDH who have diaphragmatic defects requiring patch repair have historically had higher morbidity and greater mortality than those amenable to primary repair. In an era of gentle ventilatory strategies and delayed surgery, we undertook to examine the current association between patch requirement and mortality and morbidity in a national pediatric surgical database.

Methods:

Our cohort is comprised of all patients with CDH from a 16 hospital pediatric surgical Network. Of the 147 infants in the database, 22 did not survive to surgical repair. The remaining patients were either treated with a patch or primary repair of their defect. These two groups were analyzed both for their baseline variables including gender, gestational age and SNAP-II score (a validated predictor of CDH mortality). Primary outcome was mortality. Secondary outcomes included the need for ECMO, the need for supplemental oxygen, tube feeding or anti-reflux therapy at discharge, and LOS

Results:

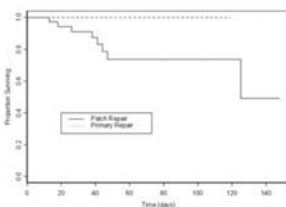
35 patients underwent patch repair, while 84 underwent primary repair (6 undergoing thoracoscopic repair were excluded). Infants requiring patch repair had significantly higher SNAP-II scores. There was no mortality in the patients undergoing primary repair compared with 23% of those undergoing patch repair (See Figure 1). Infants with patch repairs had higher need for ECMO, oxygen at discharge, longer length of stay and increased frequency of GERD (Table 1)

Conclusions:

Infants with CDH undergoing primary repair appear to have excellent survival. Despite significant improvements in the management of infants with CDH, infants requiring patch repair still experience comparably higher morbidity and mortality.

Outcome Patch vs. Primary Repair

Variable	Patch (N=35)	Primary (N=84)	P value
Median age at Surgery (days)	5 (IQR 2-4)	5 (IQR 2-11)	0.005
Median SNAP	16 (0-53)IQR 12-28	9 (0-41)IQR 0-16	P<0.005
Died	8 (23%)	0 (0%)	P=0.003
Need for ECMO	5 (14%)	1 (1%)	P<0.05
GERD documented	16 (46%)	20 (24%)	P<0.05
Oxygen at discharge	14 (40%)	11 (13%)	P<0.05
Tube feeding required at discharge	14 (40%)	24 (29%)	P>0.05



Notes:

ESTABLISHMENT OF PRE-TREATMENT BLOOD GAS TARGETS IMPROVES SURVIVAL IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH)

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Purpose:

Immediate deployment of neonatal intensive care unit (NICU) stabilization strategies which seek to normalize physiology may be a determinant in the observed trend towards improved survival in CDH. We sought to compare outcomes of CDH infants managed with or without blood gas targets within a national pediatric surgical network database.

Methods:

Our cohort is comprised of all patients with CDH from a 16 hospital CDH Network. For each CDH admission, the treating NICU team was asked to complete a pre-printed sheet outlining target high and low parameters for any or all of: pH, pCO₂, pO₂, and pre/post ductal O₂ sat. The primary outcome in this study is all cause mortality. Secondary outcomes include: need for ECMO, days on mechanical ventilation, days on supplemental oxygen, and length of stay. Outcomes between infants with and without blood gas targets were compared, adjusted for illness severity (SNAPII), gender and gestational age.

Results:

Of 147 CDH infants, 63 had blood gas targets while 84 did not. The two groups were comparable with respect to baseline characteristics. Infants with blood gas targets had a significantly lower mortality than those without (Hazard ratio 0.27, P= 0.006). There were no significant differences in secondary outcomes between these two groups (table 1).

Conclusion:

Blood gas targets for management of infants with CDH are associated with improved survival. Although the willingness to create and use stabilization targets to guide early NICU care may be a surrogate for other NICU factors (including experience, staffing, lack of time/interest to fill out form), it is clearly associated with improved survival in CDH.

Outcomes adjusted for gender, gestational age and SNAP

Outcome	HR/LR	95% CI	P value
All cause mortality	0.27	0.11-0.69	0.006
Need for ECMO	1.30	0.32-5.18	0.71
Ventilation >20 days	2.16	0.83-5.64	0.12
Supplemental oxygen >10 days	1.20	0.46-3.11	0.70
Length of Stay >40 days	1.17	0.55-2.52	0.69

Notes:

PRENATAL PULMONARY HYPERTENSION INDEX (PPHI): NOVEL PRENATAL PREDICTOR OF SEVERE POSTNATAL PULMONARY ARTERY HYPERTENSION IN ANTENATALLY DIAGNOSED CONGENITAL DIAPHRAGMATIC HERNIA

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Purpose:

To assess the potential of prenatal predictors of postnatal severe pulmonary artery hypertension (PAH) in patients with prenatal diagnosis of left congenital diaphragmatic hernia (CDH) and define a new prenatal pulmonary hypertension index (PPHI)

Methods:

The IRB approved this retrospective chart review of a cohort of patients prenatally diagnosed with CDH between July 2006 and May 2008. Two groups were defined by the presence of systemic/suprasystemic (SSPAH, n=9) or subsystemic (SuPAH, n=9) pulmonary arterial pressure, estimated by postnatal echocardiogram at 3 weeks of age. Prenatal MRI was used to measure the diameters of the right pulmonary artery (RPA_d), left pulmonary artery (LPA_d), the aorta at the diaphragmatic level (Ao_d) and the length of cerebellum (C_c) to standardize for gestation age. These measurements were used to obtain the PPHI (LPA_d/C_c) and the modified McGoon index [$MGI=(RPA_d+LPA_d)/Ao_d$]. We compared the predictive value of the PPHI and MGI to lung/head ratio (LHR), percent predicted lung volumes (PPLV) and total lung volume (TLV) for pulmonary hypertension and survival.

Result:

The PPHI and MGI had a significant, negative correlation with pulmonary artery pressure in patients with isolated left CDH ($r=-0.61$, $p=0.005$ and $r=-0.72$, $p<0.005$, respectively). The PPHI and MGI are significantly lower in subjects with SSPAH compared to those with SuPAH (1.15 ± 0.48 v/s 1.63 ± 0.28 , $p=0.004$ and 0.71 ± 0.15 v/s 1.05 ± 0.11 , $p<0.001$). The PPHI and MGI are not significantly different between the survivors and non-survivors. There were no significant differences between the groups comparing the LHR, PPLV and TLV. PPHI and MGI are superior to LHR, PPLV and TLV in predicting PAH.

Conclusion:

PPHI and MGI accurately predict the postnatal severity of PAH and are better than the LHR, PPLV and TLV in predicting PAH. These are the first prenatal indices found to predict postnatal PAH in CDH.

Notes:

PRENATAL STEROIDS FOR CONGENITAL CYSTIC ADENOMATOID MALFORMATIONS

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Purpose:

Administration of antenatal steroids in fetuses with congenital cystic adenomatoid malformations (CCAM) of the lung may improve outcomes. The purpose of this study was to evaluate the effect of prenatal steroid treatment in fetuses treated at our center with CCAM.

Methods:

This was a retrospective chart review of all patients (n=372) with CCAM lesions referred to our institution. Inclusion criteria were 1) sonographically diagnosed CCAM lesion at our institution; 2) maternal administration of a single course antenatal corticosteroids (betamethasone); and 3) no fetal intervention within 14 days of steroid administration. The primary endpoints were survival to birth and neonatal discharge. Presence of hydrops fetalis, CCAM volume, CVR, and mediastinal shift were analyzed pre- and post-administration of betamethasone.

Results:

Thirteen patients with predominantly microcystic CCAM lesions were treated with prenatal steroids. All fetuses (100%) survived to delivery and 10/13 (77%) survived to neonatal discharge. Mean gestational age at delivery was 34.7 weeks. Seven (53%) fetuses had nonimmune hydrops fetalis and nine (69%) had a CCAM volume ratio (CVR) > 1.6 at the time of steroid administration. After a course of steroids, hydrops resolved in 71% (5/7). Two fetuses with non-resolved hydrops fetalis were delivered secondary to PPRM shortly following treatment (9, 13 days). Survival in fetuses to neonatal discharge with hydrops or CVR>1.6 was 57% (4/7) and 67% (6/9), respectively.

Conclusion:

In high-risk fetal patients with predominantly microcystic CCAM lesions, betamethasone may be an effective treatment modality. This series is a pilot study for a prospective randomized trial comparing treatment of CCAM with betamethasone to placebo.

	At steroid administration						Follow-up		
Patients	Predominantly microcystic lesion	Left lung lesion	Right lung lesion	Mean GA (wks)	Hydrops	Mediastinal shift	Mean CVR (cm ²)	Hydrops Resolved	Negative CVR Trend
n=13	13/13 (100%)	5/13 (38.5%)	8/13 (62.5%)	25 2/7	7/13 (53.8%)	13/13 (100%)	2.7	5/7 (71.4%)	6/9 (66.7%)

Notes:

SURGICAL MANAGEMENT OF LARYNGOTRACHEAL STENOSIS IN CHILDREN

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Purpose:

To present the results of a pediatric laryngotracheal reconstruction program, making evident that laryngotracheal reconstruction is an excellent choice for the treatment of children with laryngotracheal stenosis.

Methods:

We included 91 patients between 11 months to 17 years age, attended in our hospital with the diagnosis of airway stenosis from January 2000 to March 2008. Patients underwent surgical management that consisted in partial cricoid resection and anastomosis, tracheal resection and end to end anastomosis or slide tracheoplasty.

Results:

We performed 77 partial cricoid resection and tyrotacheal anastomosis in 75 patients, including 9 patients with combined glottic and subglottic stenosis in which we performed extended cricotracheal resection (CTR + posterior costal graft). Sex distribution was 44 male and 31 female with a mean age of 4.6 years. 61 patients were classified as Cotton III and 14 were Cotton IV. At the present time 68 patients are decanulated (90.6%). There were three cases of dehiscence of the anastomosis in the PO of CTR needed emergency reoperation with good final result.

Sixteen patients (11 males and 5 females, with a mean age of 3.9 years) with diagnosis of tracheal stenosis beyond the subglottic space underwent surgery. Congenital tracheal stenosis was diagnosed in 3 cases, and 13 were secondary to prolonged intubation. Tracheal resection and end to end anastomosis were performed in 14 cases and a slide tracheoplasty in 2. Nine patients were operated with extracorporeal circulation support.

Conclusion:

Severe laryngotracheal stenosis is the most challenging pediatric airway surgical problem. It can involve all its segments, from subglottis to carina, representing a life threatening condition. Subglottic stenosis is the part of airway more frequently involved and CTR in severe cases has been a good surgical option with high decanulation rates. Tracheal stenosis can be surgical managed with resection and anastomosis in acquired cases.

Notes:

EXTRA-THORACIC ESOPHAGEAL ELONGATION (KIMURA'S TECHNIQUE): A FEASIBLE OPTION FOR THE TREATMENT OF PATIENTS WITH COMPLEX ESOPHAGEAL ATRESIA

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Purpose:

The aim of this study was to evaluate the outcome of all patients who underwent extra-thoracic esophageal elongation (EEE), and determine its role, among other surgical options, for the treatment of patients with complex esophageal atresia (EA).

Methods:

We performed a retrospective chart review of all patients who underwent EEE between March 1997 and September 2008.

Results:

We performed 20 EEE within the analyzed period. The diagnoses were type C EA (n=12), type A EA (n=5), type B EA (n=2) and type D EA (n=1). Mean age at the initiation of the EEE was 10 months. Twelve patients had a primary esophagostomy, and 8 patients had a secondary esophagostomy. At the time of the study, 15/20 patients had finished the treatment, 4/20 patients were still being elongated, and one patient had died from prematurity-associated complications before the final reconstruction. Of the fifteen patients who finished the treatment, 12 (80%) were reconstructed satisfactorily, and 3 (20%) had to be prematurely interrupted. Of the 12 patients who were reconstructed satisfactorily, 10 (83%) are asymptomatic with a good life quality, whereas 2 (17%) have a pseudodiverticulum and esophageal dysmotility (although both have fairly good PO intake). Finally, 5/12 patients present gastroesophageal reflux (2 required a fundoplication and 3 are being treated medically).

Conclusions:

We believe that the EEE is a helpful option for selected cases of long-gap EA, and for those patients with a short-gap EA that required a primary esophagostomy (due to a clinical condition that precludes a primary repair) or a secondary esophagostomy (due to a complication of a previous primary repair). With this technique we avoided an esophageal replacement in 80% of cases, and because the EEE does not invalidate a later esophageal replacement, we believe that EEE is a feasible initial option for this selected group of patients.

Notes:

PERINATAL PREDICTORS OF OUTCOME IN GASTROSCHISIS

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Purpose:

Optimal postnatal treatment of gastroschisis is unknown. Validated outcome predictors may enable "risk-guided" therapy leading to improved outcomes. We sought to identify perinatal risk/treatment predictors of outcome from a national gastroschisis database.

Methods:

249 cases of gastroschisis ascertained between May 2005 and August 2008 were analyzed for mortality, morbidity and treatment outcomes. Risk variables evaluated included gestational age (GA), birthweight, a validated neonatal illness severity (SNAP-II) score, time of birth and intended and actual surgical management. Biostatistical analysis included logistic regression for binary outcomes and linear regression for continuous outcomes.

Results:

(Table 1): 239 infants survived to hospital discharge (96%). The only predictor of mortality was SNAP-II score (RR = 1.066, 95% CI = 1.02 – 1.114). Higher SNAP-II scores also predicted prolonged ventilation (p=0.025) and length of stay (LOS) (p=0.01). Abdominal closure within 6 hours of birth predicted shorter LOS (p=0.01) and shorter duration of TPN (p=0.028). GA inversely predicted prolonged ventilation (p=0.0007) and development of cholestasis (conjugated bilirubin >10 mg/dl) (RR = 0.767, 95% CI = 0.605 – 0.972). Treatment intent of infants with higher SNAP-II scores favored primary over silo closure (RR = 1.043, 95% CI = 1.013-1.075), yet also predicted a higher likelihood of closure "failure" (RR = 1.035, 95% CI = 1.006-1.065).

Conclusions:

SNAP -II scores significantly predict mortality and non-mortality outcomes in gastroschisis. Although urgent abdominal closure shows an association with reduced LOS, it may be undesirable in the more severely ill infant. Our data suggest that gestational immaturity is a significant adverse outcome predictor and refute the purported value of routine preterm delivery.

Summary of Selected Perinatal Risk Variables and Outcomes for Gastroschisis Cohort

Gestational Age (weeks)	36.2 (+/- 2.1)
Birthweight (grams)	2566.3 (+/- 564.7)
Mean Day 1 SNAP-II Score	9.2 (+/- 12.3)
Urgent Abdominal Closure (<6h)	118 (50%)
Intended Closure Successful	210 (85%)
Survival to Hospital Discharge	239 (96%)
Mean LOS (days)	49.7 (+/- 46.6)
Mean Days Ventilation	5.0 (+/- 6.0)
Mean Days TPN	37.4 (+/- 32.4)
Cholestasis (conjugated bilirubin >10mg/dl at discharge)	45 (18%)

Notes:

ANTIBIOTIC STRATEGIES AND INFECTIOUS COMPLICATIONS IN THE MANAGEMENT OF GASTROSCHISIS

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Purpose:

There is no evidence to direct antibiotic therapy in gastroschisis management, and it is unclear whether infectious complications vary according to abdominal closure method. We used a gastroschisis-specific database to examine antibiotic administration and surgical closure practices, and their relationship to infectious complications.

Methods:

A national, prospective gastroschisis database was interrogated for episodes of wound infection and bacteremia-confirmed line sepsis. Antibiotic use, type of venous access, abdominal closure method, timing and location were studied. Groups were compared using the Fisher's Exact test.

Results:

Of 249 patients, 237 were placed on a course of antibiotics at birth for an average 6.0 days (range: 1-36 days), and the mean time to abdominal closure was 2.9 days (range 0-25 days). The most common regimen was ampicillin and gentamicin (n=144, 60.8%). There were 79 episodes of bacteremia, with coagulase-negative staphylococcus being the most frequent isolate (67.1%). Thirty patients (12.0%) had a documented abdominal closure site infection, and 35 patients (14.1%) had at least one episode of line sepsis. Abdominal closure was performed in the NICU (n=43, 17.3%) or operating room (OR) (n=206, 82.7%), with an observed trend (independent of line type) towards higher line sepsis rates in the NICU closure group (23.3% vs.13.0%, p=0.09), and no difference in wound infection rates (11.6% vs.13.0%, p=1.0). One hundred and eighteen (47.4%) abdominal closures took place within 6 hours of NICU admission, while 88 (35.3%) were delayed greater than 24 hours. The wound infection rate was greater in the delayed group (22.7% vs.5.9%, p=0.0006), with no difference in the incidence of line sepsis (18.2% vs.13.6%, p=0.437).

Conclusions:

Infectious complications remain an important consideration in gastroschisis management, and the frequency of staphylococcal isolates should guide empiric antibiotic choice. Early closure reduces wound infection rates while OR closure reduces the risk of line sepsis.

Notes:

MEGARECTUM AFTER SURGERY FOR ANORECTAL MALFORMATIONS (ARM)

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Purpose:

Megarectum complicating surgery for ARM has implications for longterm continence. Factors influencing continence and defecation include intact rectal reservoir, innervation/proprioception of the anorectal muscle complex, functioning anorectal inhibitory reflex (AIR), and intact perception at the anal margin. We studied outcomes after surgery for ARM with emphasis on megarectum; particularly as to whether altered rectal proprioception from anatomic sacro-coccygeal anomalies affect incidence. We also assessed whether an abnormal AIR could trigger passive rectal dilatation without mechanical obstruction.

Methods:

Eighty six infants (53 male) with ARM over 20 years were included. Demographics, surgical history, pathology, defecation patterns, imaging, manometry, and morbidity were analyzed. Incidence of sacrococcygeal malformations in children with and without megarectum were compared using Fisher test. Manometry results were evaluated for integrity of AIR and correlated to megarectum occurrence.

Results:

There were 22 high/intermediate and 64 low ARM's. Fourteen (16%) developed a megarectum: 5/22 in high and 9/64 in low anomalies ($p=0.33$). Twelve patients underwent megarectum resection at a median of 2.6 yrs (7mo-10yrs); 2 received bowel management protocols. Fifty-seven percent (8/14) of children with and 7% (5/72) without megarectum had sacro-vertebral anomalies ($p=0.0001$). Patients with pre-op manometry ($N=5$) demonstrated an intact AIR. Colonic manometry demonstrated hyperactive colons ($N=2$). Constipation was the predominant pre-op symptom; 3 patients suffered from incontinence after resection. All the specimens showed normal innervation and thickened muscularis on pathology.

Conclusions:

Sacral anomalies, which are more prevalent in children who developed megarectum may result in abnormal rectal proprioception contributing to this pathology. Innervation anomalies may coexist, although pre-op manometries showed normal AIRs. Rectal dysmotility may lead to stool retention with subsequent dilatation, and patients who underwent colonic manometry had diffuse colonic hypermotility. Further physiologic and cellular studies are needed to elucidate the causes of this significant complication after surgical ARM repair in the absence of obstruction.

Notes:

GASTROINTESTINAL RAMIFICATIONS OF THE CLOACAL EXSTROPHY COMPLEX: A 24 YEAR EXPERIENCE

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Purpose:

Cloacal exstrophy is a rare and complex congenital anomaly requiring coordination among multiple pediatric subspecialties. There is currently no consensus regarding the fate and function of the hindgut, which plays an integral role in their long-term gastrointestinal health and genitourinary reconstruction.

Methods: A retrospective chart review was performed evaluating 77 patients with cloacal exstrophy treated during the previous 24 years at our institution.

Results:

77 patients with cloacal exstrophy were treated between 1985 and 2008. 65 were White, 6 were African-American, 3 were Asian, and 3 were Hispanic. Genotypes included 44 XY, 32 XX, and 1 XYY. 51 were reared as females and 26 as males. The hindgut length was 2 to 5 centimeters in 11 patients, 6 to 10 centimeters in 13 patients, 11 to 15 centimeters in 9 patients, and 16 to 20 centimeters in 7 patients. The hindgut length was unknown in 37 patients. 45 patients had tubularization of the cecal plate with an end colostomy and 30 patients had an ileostomy placed for bowel diversion purposes. 3 patients suffered from short gut syndrome. 28 patients had genitourinary reconstruction, 9 using small bowel and 19 using colon.

Conclusion:

This cohort demonstrates a higher incidence of cloacal exstrophy in genetic males, although patients were more likely to be raised as phenotypic females. With modern reconstructive techniques, gender conversion has been required far less frequently. Associated anomalies include spinal deformities, intestinal duplications, and renal agenesis. The length of hindgut found at initial exploration was typically less than half that of normal newborns. Infants are currently more likely to have cecal plate tubularization with end colostomy rather than an ileostomy. Few patients were candidates for abdominoperineal pull-through procedures due to spinal anomalies resulting in poorly developed pelvic musculature. All patients with short gut syndrome had an end ileostomy.

Notes:

TWIN-TWIN TRANSFUSION SYNDROME: LIFE AFTER TRIALS

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Purpose:

Twin-twin transfusion syndrome (TTTS) is the most serious complication affecting monochorionic pregnancies with an incidence of about 10%. If untreated, TTTS can lead to 80% mortality and significant neurologic morbidity.

Methods:

We retrospectively examined all patients treated for TTTS from 2005 to 2008. We identified 56 mothers carrying 112 fetuses. Each patient underwent single port, percutaneous, endoscopic laser ablation of chorangiopagus vessels. Variables examined included maternal age, placental location, premature rupture of membranes, survival, gestational age at presentation, gestational age at birth, birth weight, and maternal and fetal complications.

Results:

Forty-eight mothers underwent laser therapy. Mean maternal age was 28 years. Forty-nine percent of placentas were anterior. Premature rupture of membranes occurred in 14% of patients. There were 53 survivors. There was at least 1 survivor in 80 percent of patients. There were 2 survivors in 46 percent of patients. Mean gestational age at delivery was 31 weeks. Mean birth weight of the donor twins was 1714 graMS Mean birth weight of the recipient twins was 2108 graMS There was one maternal complication in which one patient required a postoperative blood transfusion. We identified three major perinatal complications in surviving infants. One patient developed a spontaneous ileal perforation and underwent successful operative repair. Two additional patients developed necrotizing enterocolitis. One of those patients died and the other was managed medically.

Conclusions:

These data are consistent with those previously reported in the literature. The major source of morbidity and mortality in the fetuses is premature rupture of membranes leading to preterm labor and early delivery. There were three major perinatal complications in survivors. There were no major maternal complications.

Notes:

A SINGLE INSTITUTION SERIES OF 800 CHILDREN WITH ABDOMINAL SOLID ORGAN INJURIES

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Purpose:

We sought to refine our understanding of the non-operative management of abdominal solid organ injuries in children and examine the effect of a clinical pathway guideline (CPG) on their care.

Methods:

Children suffering an abdominal solid organ injury from July, 1993 to 2008 were identified in the trauma registry of a pediatric trauma center. Mean length of stay (LOS), injury severity score (ISS), transfusion, need for operation and deaths were extracted. Records were then examined in 5 year periods: pre-CPG (1993-1997), implementation-CPG (1998-2002), post-CPG (2003-2008).

Results:

800 children suffered 841 abdominal solid organ injuries: 454 splenic, 213 hepatic, 141 renal, and 33 pancreatic. Patients' mean age was 11 years, ISS 12, GCS 14 and LOS 7 days. 58 patients were transfused: 16 en route, 11 emergency department, 7 operating room and 34 intensive care unit. There were 5 deaths, 1 arrived in extremis and 4 from overwhelming brain injury. Nine abdominal operations were performed: 2 splenorrhaphies (0.4%), one nephrectomy (0.7%), and 4 pancreatectomies or drainage procedures (12%). The rate of CT-diagnosed concomitant bowel injury was 3%, and 1.1% of patients underwent bowel operation. No liver or spleen injured patient initially managed non-operatively subsequently required an operation. Six patients later underwent an interventional radiology procedure. When analyzed by CPG period, the mean injury grade did not vary significantly over time (2.39, 2.37 and 2.41), yet the ICU admission percentage dropped (71%, 40%, 32%) as did the LOS (8, 6, 6 days).

Conclusion:

This review represents the largest single institution series of pediatric abdominal solid organ injuries. Operative intervention is unusual and no splenectomies were performed in 454 patients. Operative bowel injuries occurred in 1.1%, and mortality was secondary to brain injury. Standardized care guidelines effectively reduce ICU admissions and LOS.

Notes:

DELAY IN DIAGNOSIS AND TREATMENT OF BLUNT INTESTINAL INJURY DOES NOT ADVERSELY AFFECT PROGNOSIS

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Purpose:

Blunt intestinal injury (BII) requiring surgical intervention in the pediatric trauma population remains difficult to diagnose. We sought to determine whether delay in treatment had an adverse effect on patient outcome.

Methods:

A multi-institutional retrospective chart review utilizing the APSA Committee on Trauma was initiated after IRB approval was obtained at each of the 18 institutions. All children ≤ 15 years of age diagnosed with a BII were identified and only those with BII noted during surgery or autopsy from January, 2002 through December, 2007 were included. The data form was designed and approved prior to chart review and all data was combined into one database.

Results:

358 patients were accrued into the study. 214 patients had sufficient data to determine the interval between injury and operation. These were divided into 4 groups (<6 hours, 6-12 hours, 12-24 hours, > 24 hours) based on time from injury to intervention. Early and late complications as well as hospital days were compared in each group. There were 3 deaths from an abdominal source in the < 6 hour group and 2 in the 6-12 hour group. ISS was significantly greater in the < 6 hour intervention group. There was no correlation between time to surgery and complication rate, nor was there a significant increase in hospital days. The morbidity data is presented in Table 1.

Table 1

HRS	PTS	AGE	ISS	EARLY COMP	LATE COMP	HOSP DAYS
0-6	82	7.7 ±3.8	21.0 ±14.6*	14(17.1%)	5(6.1%)	11.1 ±8.1
6-12	65	8.5 ±3.7	14.2 ±9.9	12(18.5%)	7(10.7%)	11.3 ±12.0
12-24	53	8.6 ±4.1	13.4 ±8.8	11(20.8%)	3(5.7%)	15.1 ±28.0
> 24	14	8.3 ±3.4	16.2 ±8.2	3(21.4%)	2(14.3%)	20.1 ±25.2

* Significantly greater than 6-12 and 12-24 hr ISS, p<0.005

Conclusions:

These data suggest that delay in operative intervention does not have a significant effect on prognosis after pediatric blunt intestinal injury. Appropriate observation and serial examination rather than urgent exploration would appear adequate when the diagnosis is in question.

Notes:

OUTCOMES IN CHILDREN WITH INTESTINAL FAILURE FOLLOWING LISTING FOR INTESTINAL TRANSPLANT

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Purpose:

The interval of time from listing to intestinal transplant for specific conditions is unknown. The purpose of this study was to describe the population of pediatric patients waiting for intestinal transplant and to evaluate the risk of death or transplant by specific disease states.

Methods:

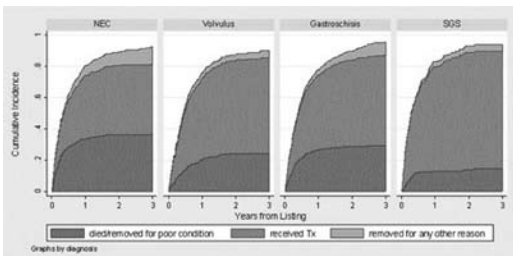
We studied the United Network for Organ Sharing (UNOS) database (1/1/1991-5/16/08), identified all patients ≤ 21 years-old at first listing for intestinal transplant and examined their age, sex, weight, and diagnoses leading to listing. Time to list removal was summarized with cumulative incidence curves. We performed a multinomial logistic regression analysis to compare relative risk ratios for removal from the list for transplant, death or other reasons.

Results:

We identified 1,712 children listed for intestinal transplant (57% male, 51% < 1 yr at listing). Median weight at listing was 8.1kg (IQR 6.1-14.1). The most common conditions for listing were gastroschisis (23%), necrotizing enterocolitis (NEC) (19%), short gut syndrome (13%), and volvulus (12%). Of the 852 patients transplanted, median time to transplant was 114 days (IQR 40-227), with 69% also receiving a liver. Median age and weight at intestinal transplant were 1yr (IQR 1-5) and 10kg (IQR 6.5-16.3). There were 694 children removed from the list. The regression analysis comparing relative risk of removal from the list for transplant versus death demonstrated significant differences among disease conditions ($p < 0.01$). Compared to the gastroschisis group, the relative risk ratio for death versus transplant was higher in the NEC group ($p = 0.017$), lower in the short gut syndrome group ($p = 0.002$) and not significantly different in the volvulus group ($p = 0.96$).

Conclusions:

We have described the population of pediatric patients listed for and receiving an intestinal transplant. We conclude that the relative risk of transplant versus death in this population varies significantly by the disease state of the patient.



Notes:

IMPROVED OUTCOME OF LIVER TRANSPLANTATION FOR UNRESECTABLE HEPATOBLASTOMA

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Purpose:

Orthotopic liver transplantation (OLT) continues to evolve as a viable option for patients with unresectable hepatoblastoma, and 5-year post-transplantation survival rates of 80% can be achieved. The purpose of this study was to evaluate our institutional experience in the management of patients with Stage III and Stage IV hepatoblastoma treated by complete hepatectomy and liver transplantation.

Methods:

We retrospectively reviewed the clinical courses of 16 patients (aged 6 months to 5 years) with unresectable hepatoblastoma managed by OLT at our institution from March 1998 through August 2008. Approval was obtained from our institutional review board.

Results:

All 16 patients underwent extensive pre-transplant chemotherapy. Of 14 patients with Stage III hepatoblastoma, 11 underwent primary OLT for unresectable tumors, and 3 underwent salvage OLT due to tumor recurrence following initial tumor resection. Two patients with Stage IV hepatoblastoma presented with lung metastases, which were excised prior to transplantation. No patient had evidence of extrahepatic disease at the time of OLT. Whole organ grafts (n = 9), cadaveric left lobe grafts (n = 5), and living-donor left lateral segment grafts (n = 2) were utilized. Post-transplant chemotherapy was administered to 88% of patients. Graft survival and recurrence-free patient survival were 100% in this cohort of 16 patients with median post-transplant follow-up of 37.9 months (range, 2.3 – 127.1 months).

Conclusions:

Primary OLT with neoadjuvant chemotherapy results in superior long-term outcomes in patients with unresectable Stage III hepatoblastoma. Liver transplantation provides a viable therapeutic approach in patients with recurrent hepatoblastoma after initial attempts at resection, and in those with pulmonary metastases at diagnosis if their extrahepatic disease can be eradicated prior to OLT. Optimal outcomes in patients with advanced hepatoblastoma can be achieved with early referral to transplant centers with expertise and a multidisciplinary approach to this patient population.

Notes:

DEFINING HEPATOBLASTOMA RESPONSIVENESS TO NEOADJUVANT THERAPY AS MEASURED BY TUMOR VOLUME AND SERUM ALPHA-FETOPROTEIN KINETICS

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Purpose:

Hepatoblastoma is commonly inoperable at presentation, necessitating multiple cycles of toxic neoadjuvant chemotherapy before definitive resection. To refine the paradigm for timing of resection, we questioned whether a plateau in hepatoblastoma responsiveness to neoadjuvant therapy could be detected by calculating tumor volume (TV) and serum -fetoprotein (sAFP) kinetics.

Methods:

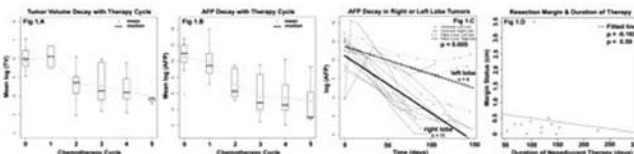
After IRB approval, we retrospectively reviewed our institutional experience managing hepatoblastoma between January 1996 and September 2008. Included for analysis were all babies diagnosed with an epithelial-type hepatoblastoma. Those having initially unresectable hepatoblastomas were identified to calculate TV and sAFP as measures of treatment responsiveness over time. TV was calculated ($L \times W \times H \times 0.52$) in babies having complete serial and digitalized CT scans. Effects of therapy type, therapy duration, and lobe of liver involvement on TV, sAFP, margin status, and toxicity were analyzed using reduced monotonic regression, mixed model analysis of variance, the Wilcoxon rank sum test, and Spearman correlations.

Results:

Of 23 children treated for hepatoblastoma during this era, 4 were resected primarily. Fifteen children had complete digital films for kinetics analysis. Both TV and sAFP decreased dramatically over time (Fig. 1A,B; $p < 0.0001$). No statistically significant difference in mean TV or sAFP was detected after chemotherapy cycle 2. Left lobe tumors had significantly greater presenting levels of, and slower decay in, sAFP compared to right lobe tumors (Fig 1C; $p = 0.005$), although no statistically significant differences in TV existed between liver lobes. Resection margins did not change with therapy duration (Fig 1D). No statistically significant differences were observed in TV or sAFP responses between 3 or 4 agent neoadjuvant regimens, yet toxicity incidence was high.

Conclusions:

Hepatoblastoma responsiveness to therapy may plateau over time, and is accurately predicted by measuring TV and sAFP kinetics. As a result, treatment toxicities may be reduced by resection earlier on the plateau and tailoring of chemotherapeutic regimens.



Notes:

TUMOR VOLUME AS A PREDICTOR OF ONCOLOGIC OUTCOME IN CHILDREN WITH INTERMEDIATE RISK RHABDOMYOSARCOMA

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Purpose:

Refinement of established prognostic factors among intermediate risk RMS patients may improve the prediction of oncologic outcomes. We explored the prognostic value of tumor volume and patient weight versus tumor diameter and patient age (traditional prognostic factors).

Methods:

Complete patient information for non-metastatic RMS patients enrolled on the COG intermediate risk study (D9803, 1999-2005) was available for 362 patients. Age, stage, IRS surgical group, T-stage, nodal status, histology, primary site, maximum tumor diameter, tumor volume, height, weight, and treatment regimen were considered. The Kaplan-Meier method was used to estimate survival. A recursive partitioning method was used to identify cut-points for prognostic factors associated with event-free survival. Cox-proportional hazards regression models were used to estimate the association between patient characteristics and the risk of failure or death. Covariates were deleted from the regression model until all covariates were significant at the 0.05 level.

Results:

A recursive partitioning algorithm for Event Free Survival (EFS) suggests that prognostic groups should be defined by tumor volume (using a cut-point of 22.35 cm³), weight (using a cut-point of 52.2 lb), and embryonal histology as summarized in the figure. Similar results were obtained utilizing the traditional prognostic factors of tumor diameter and patient age. However, when utilizing tumor volume and patient weight, more patients were classified in the middle risk category. Interestingly, tumor volume and patient age added significant outcome information to the standard prognostic factors including tumor diameter and patient age (p=0.002).

Conclusions:

The recursive partitioning models showed that the factors most strongly associated with the risk of failure were tumor volume, patient weight, and histology. Based on regression modeling, volume and weight are superior predictors of oncologic outcome compared to the standard prognostic factors, tumor diameter and patient age, in children with intermediate risk RMS

Notes:

IMPROVED SURVIVAL FOR CHILDREN WITH NEUROBLASTOMA AND WILMS TUMOR AT HIGH AND LOW VOLUME COG MEMBER HOSPITALS

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Purpose:

To determine the prognostic significance of hospital surgical volume and COG membership on outcomes for neuroblastoma and Wilms tumor.

Methods:

The Florida Cancer Data System was queried from 1981-2004. A cumulative surgical volume of greater than 50% defined high-volume center (HVC) and below that a low-volume center (LVC).

Results:

Of the 869 neuroblastoma patients identified, 463 were treated at 5 COG/HVC, 246 at 11 COG/LVC and 160 at 50 non-COG/LVC. COG centers treated more adrenal and mediastinal tumors ($p=0.005$). Radiotherapy was more frequently given at non-COG centers ($p<0.001$). Chemotherapy was more frequently administered at COG/HVC ($p<0.001$). While there was no statistical difference between survival rates at COG/HVC and COG/LVC ($p=0.347$), five and ten-year survival rates were significantly higher than at non-COG/LVC ($p=0.001$).

Of the 790 Wilms tumor patients identified, 395 were treated at 5 COG/HVC, 210 at 11 COG/LVC and 185 at 39 non-COG/LVC. COG hospitals treated younger patients with lower-staged tumors ($p<0.05$). Chemotherapy was more frequently administered at COG centers ($p<0.001$). There was no difference in radiotherapy use. Although there was a difference between survival rates at COG/HVC and COG/LVC ($p=0.020$), five and ten-year survival rates were significantly higher than at non-COG/LVC ($p<0.05$).

Multivariate analysis confirmed patients with either neuroblastoma or Wilms tumor did significantly better when treated at a COG hospital.

Conclusions:

Children treated at COG hospitals had significantly improved survival for neuroblastoma and Wilms tumor, independent of surgical volume. Non-COG hospitals must identify and address differences in care resulting in inferior outcomes.

Notes:

MALIGNANCY AND OVARIAN TORSION: DO WE PRACTICE WHAT WE PREACH?

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Purpose:

With ovarian torsion, concern for underlying malignancy has previously driven surgeons to resect the ovary. Detorsion alone has been recommended with follow-up ultrasound surveillance, yet is not routine practice. The incidence of malignancy presenting with ovarian torsion is not documented. Does the risk of malignancy justify salpingoophorectomy and decreased fertility?

Method:

After IRB exemption (IRB#-022008-095), a 15½ year retrospective review was conducted to identify cases of operative ovarian torsion in our medical center. Tumors with neoplastic pathology (malignant and benign) were analyzed and compared to all reported cases in the literature.

Results:

114 patients (mean age 10y, 2d to 19y, SEM±0.52) with operatively proven ovarian torsion were identified. Four malignancies (3.5%) and 18 benign neoplasms (15.8%) were present in this age group. Malignancies consisted of serous borderline tumors (2), juvenile granulosa cell tumor (1) and dysgerminoma (1). All were stage IA and cured with resection, except a stage IB dysgerminoma which required chemotherapy. The literature yielded 12 articles with 9 (1.6%) malignancies and 186 (33%) benign neoplasms in a total of 568 patients with operative ovarian torsion. Those malignancies were juvenile granulosa cell tumor (n=4), dysgerminoma (n=2), carcinoma (n=1) and serous borderline tumors (n=2).

Conclusion:

By combining our series with those in the literature, a 1.9% malignancy rate occurred in 682 patients with ovarian torsion, markedly less than the reported malignancy rate of 10% in children with ovarian masses. In our series, which represents the largest reported series of torsion in the pediatric literature, all malignancies presented as stage I. These data support a role for detorsion and post-operative ovarian surveillance, with reoperation for persistent masses. Further study is needed to determine if delayed resection of those malignancies presenting with torsion would result in tumor progression within the time interval to definitive resection, and thus change prognosis.

Notes:

ADVERSE CATHETER-RELATED EVENTS IN A PHASE I TRIAL OF PROPHYLACTIC ETHANOL ADMINISTRATION TO PREVENT CENTRAL VENOUS CATHETER INFECTIONS

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Purpose:

Catheter-related bloodstream infections remain a costly problem for which there is no simple preventative strategy. We report preliminary results of a prospective, phase I clinical trial of prophylactic ethanol-lock administration for the prevention of central venous catheter infections.

Methods:

Institutional Review Board approval was received. Patients on an intravenous antibody protocol for neuroblastoma, with surgically-implanted central lines, were enrolled for up to six months. During four consecutive days on prespecified treatment weeks, 70% ethanol was administered to dwell overnight in each patient's catheter instead of heparin. Symptoms were assessed by monitoring and questionnaires; blood ethanol levels were measured each treatment week. Positive blood cultures were tracked. Accrual was targeted at 50 patients. Comparisons to historical controls (2002-2005) were made using Fisher's exact test for six-month infection rates, and log rank test for time-to-infection.

Notes:

PUSHING THE BOUNDARIES OF ECLS: OUTCOMES IN <34 WEEK EGA NEONATES

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Purpose:

Extracorporeal life support (ECLS) is usually reserved for infants ≥ 34 weeks estimated gestational age (EGA) due to concerns about incidence of mortality and intracranial hemorrhage (ICH). We sought to characterize survival, rates of ICH, and complications in <34 week EGA neonates placed on ECLS.

Methods:

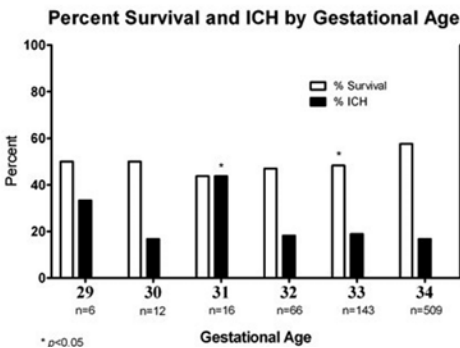
756 neonates of EGA < 34 weeks were identified in the Extracorporeal Life Support Organization (ELSO) Registry (1976-2008). Data analyzed included birthweight, survival, pre-ECLS conditions, ventilatory parameters and complications (including ICH and other neurological outcomes). Chi-square and logistic regression analyses were performed using SPSS (significance $p < 0.05$).

Results:

Survival rates for neonates 29-33 weeks EGA was similar (range 44-50%). When compared to survival rates of 34 week EGA neonates (58%), survival was statistically different for 33 week EGA (48%, $p=0.048$, see graph). ICH incidence was statistically higher for 31 week EGA neonates (44% or 7/17, $p=0.002$), but not statistically different for others [29 (33% or 2/6), 30 (17% or 2/12), 32(18% or 12/66), 33(16% or 8/50) week EGA neonates] when compared to 34 week EGA. ICH and survival did not correlate with EGA during logistic regression analysis.

Conclusion:

Survival appeared to be similar for 29-32 week EGA when compared to 34 week EGA neonates. However, there was a statistically significant difference between 33 week and 34 week EGA infants. In addition, ICH rates were only statistically different for 31 week EGA when compared to 34 week EGA neonates. These data suggest that ECLS in the modern era can be used at EGA as low as 29 weeks with reasonable survival and acceptable rates of ICH.



Notes:

UNIVERSITY PEDIATRIC SURGERY: BENCHMARKING PERFORMANCE

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Objectives:

University pediatric surgery sections (UPS) are supported by diverse resources whose nature/depth/fate are unclear. We queried UPS revenue/expense/hospital performance variables and asked if an "industry" performance overview could be an instrument for benchmarking UPS performance?

Methods:

Comprehensive performance data – 2005, 2006, and 2007(annualized on 10 months) -were requested from all major UPS (ACGME fellowship training). Results were collected anonymously by SurveyMonkey® (<http://www.surveymonkey.com/s.asp?u=867533901754>). Descriptive statistics are reported as mean with 95% CI. American Medical Group Association (AMGA) Survey benchmarked compensation.

Results:

19/30 UPS data on 130 surgeons: 11/19 complete, Hospital data denied in 8.

Conclusions Regarding Industry Overview/Benchmarking:

1. Most revenue is generated by clinical work, least by research/education
2. Most expenses are incurred by compensation, least by research/education
3. Hospital revenues are often obfuscated
4. Hospital return on investment/clinical FTE is substantial

Notes:

Section Effort (%)	2007	2006	2005
<ul style="list-style-type: none"> Clinical Admin/Academic Research 	70 CI: 63.64 – 74.73 16% CI: 13.04 – 20.12 14% CI: 10.12 – 18.34	72 CI:66.77 – 77.84 16% CI: 12.69 – 19.90 12% CI: 8.39 – 15.93	72 66.65 – 78.07 16% CI: 12.27 – 19.84 12% CI: 8.34 – 16.59
Mean Compensation (all ranks)	\$388,387 CI: 325,475 – 451,2999	\$378,524 CI: 313,034 – 444,014	\$358,906 CI: 293,400 – 424,411
RVU/surgeon/year	7,233 CI: 5809 – 8659	7,689 CI: 6310 – 9068	7,343 CI: 6092 – 8595
Gross Professional Charges/Surgeon/Year	\$1,188,601 CI: 922,394-1,454,808	\$1,318,887 CI: 1,136,097-1,501,677	\$1,270,719 CI: 1,081,690-1,459,748
Professional Collections/Surgeon/Year	\$424,581 CI: 297,765-551-398	\$494,424 CI: 367,725-621,574	\$470,037 CI: 364,058-576,017
Revenue Sources (%) <ul style="list-style-type: none"> Practice income Hospital support External support 	60 CI: 50 – 70 29 CI: 15.7 – 42.4 12 CI: 6.06 – 17.91	60 CI: 49.9 – 69.9 29 CI: 15.5 – 42.5 13 CI: 5.8 – 19.2	60 CI: 49.3 – 71.6 26 CI: 12.1 – 39.8 15 CI: 7.3 – 21.9
Salary Expense (% total) <ul style="list-style-type: none"> Physician Administrative RN/PA Research staff 	2007 1.8 CI: 72.4 – 81.5 10.88 CI: 7.4 – 14 7.05 CI: 3.5 – 10.6 5.65 CI: 1.3 – 10	2006 73.5 CI:62.7 – 84.4 10.4 CI: 7.2 – 13.5 5.9 CI: 2.5 – 9.2 4.2 CI: 0.4 – 7.9	2005 0.3 CI: 63.4 – 85.0 0.1 CI: 7.1 – 13.1 0.5 CI: 1.9 – 8.9 0.6 CI: 0.4 – 6.9
Average Length of Stay <ul style="list-style-type: none"> inpatient NICU 	2007 1.8 CI: 5.4 – 8.2 39.2 CI: 25.1 – 53.4	2006 1.9 CI: 5.6 – 8.3 36.9 CI: 27.9 – 46.1	2005 0.97 CI: 5.8-8.2 36.3 28-48.5
Mean Inpatient Hospital Charges (\$) <ul style="list-style-type: none"> Gross Collections Collection Rate 	87,459,907 CI: 11935265-162984549 35,348,433 CI: 2691147-68005718 40.4%	84,643,727 CI: 12542511-156744942 38,761,142 CI: 7896991-69625292 45.8%	75,399,637 CI: 10929606 -139869667 36,077264 CI: 55971524-66183003 47.8%
Mean Hospital Collections/Clinical FTE	\$7,728,664 CI: 2500604-12956723	\$10,949,415 CI: 3167188-18731642	\$10,162,236 CI: 2489420-17835052
Mean C.M.I. <ul style="list-style-type: none"> Inpatient NICU 	1.72 CI: 1.4-2.0 4.8 CI: 1.7-7.8	1.7 CI: 1.4-2.1 4.5 CI: 2.2-6.8	1.7 CI: 1.4-2.0 1.8 3.8 CI: 2.1-5.6

A PEDIATRIC AMBULATORY WOUND SERVICE (PAWS): A NOVEL APPROACH IN WOUND MANAGEMENT

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Purpose:

In 2001, in response to an overwhelming increase in patient visits for various pediatric abscesses, burns, and other wounds, an ambulatory burn and procedural sedation program (PAWS) was developed to minimize Operating Room utilization. This study sought to determine the effect of this program on patient outcome and resource utilization.

Methods:

The hospital records of all children managed through PAWS from 2001-2007 were reviewed. Outcomes measured include patient demographics, number and location of visits per patient, cause of wounds, and reimbursement. Chi-Square test was performed using GraphPad Prism.

Results:

Overall, 7620 children (age 0-18 years) received wound care through PAWS from 2001-2007. There were no differences in patient age, race, and gender during this time period. Between 2001 and 2007, the percentage of patients seen as outpatients increased from 51% to 68% ($p < 0.0001$), and the average number of visits per patient decreased from 3.9 to 2.4 ($p = 0.008$). In 2007, forty-six percent of the children required only one visit. In 2007, 74% of the visits were for management of wound and soft tissue infections, compared to only 9% in 2001 ($p < 0.0001$). The contribution margin of a PAWS visit and total contribution margin in 2007 was \$1052 and \$4.0 million, respectively.

Conclusions:

The creation of PAWS has allowed for the transition in management of most pediatric skin and soft tissue wounds and infections to an independent ambulatory setting, alleviating the need for Operating Room resources, while functioning at a profitable cost margin for the hospital.



Notes:

THE PARADOXICAL EFFECT OF MEDICAL INSURANCE ON DELIVERY OF CARE FOR INFANTS WITH CONGENITAL ANOMALIES

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Purpose:

Neonates with congenital anomalies account for some of the most complex and costly care of any patient population. When these infants are insured, caring for them generates a large financial margin, and when they are uninsured the cost of their care is borne by the treating institution. The purpose of this study was to determine whether insurance status is associated with the tendency to transfer neonates with congenital anomalies born in non-children's hospitals for surgical care.

Methods:

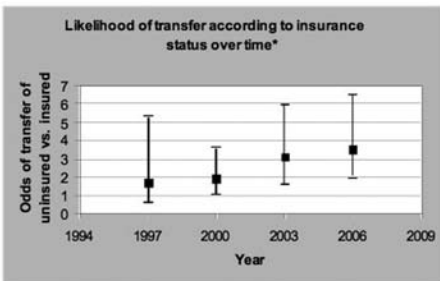
We used the KID (Kids' Inpatient Database) to study neonates with abdominal wall defects (gastroschisis or omphalocele), esophageal atresia, or intestinal atresia who were born in United States non-children's hospitals in 1997, 2000, 2003 and 2006. We tested associations between payer status and transfer using the chi square test, and used multiple logistic regression to adjust for covariates. Our main outcome measure was whether patients were treated in the non-children's hospital in which they were born or transferred for surgical care.

Results:

Neonates without insurance were significantly more likely to be transferred (76.9%) than those with private insurance (60.4%) or Medicaid (60.4%); ($p < 0.0001$). After adjusting for year, diagnosis, race/ethnicity, gender, socioeconomic status, and hospital characteristics, uninsured patients were still 2.57 (95% confidence interval 1.83, 3.62) times more likely to be transferred compared to patients with private insurance or Medicaid. This trend increased over time (see figure).

Conclusions:

The current reimbursement structure in the United States provides an incentive for non-children's hospitals to retain insured patients with congenital anomalies and transfer uninsured patients with these same anomalies. This places a disproportionate financial burden on children's hospitals, while paradoxically causing insured infants to be cared for at hospitals that may not be best equipped to provide complex care.



Notes:

LAPAROSCOPIC PYLOROMYOTOMY IS CHEAPER THAN OPEN PYLOROMYOTOMY: ECONOMIC ANALYSIS OF A RANDOMISED CONTROLLED TRIAL

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Purpose:

Infantile Hypertrophic Pyloric Stenosis (IHPS) can be corrected by either Open (OP) or Laparoscopic Pyloromyotomy (LP). LP may provide clinical benefits of reduced time to post-operative full feeds and reduced post-operative inpatient stay. The aim of this study was to compare the cost effectiveness of LP vs. OP.

Methods:

OP and LP were compared in an IRB-approved multi-centre randomised double blind controlled trial, for which the primary outcomes were time to full feeds and time to discharge. In order to undertake a detailed cost analysis, we assigned costs to: pathology, imaging, medical staff, medication, ward, operative, and outpatient appointments for 73 patients recruited from one of the participating centres. Laparoscopic equipment costs were based on a 5-year expected lifetime and use for 4 operations per day. Costs were derived from the hospital finance department and national reference costs, and were converted to US dollars at an exchange rate of 1 GBP = 1.75 USD. Data were compared using linear regression analysis, adjusting for the minimization criteria used in the trial.

Results:

Operation costs were similar between the two groups (Table 1). A shorter time to full feeds and shorter hospital stay in LP vs. OP patients resulted in a highly significant difference in ward costs, and a small difference in other costs. Overall, LP patients were \$1182 less expensive to treat than OP patients.

Conclusions:

This study indicates that LP is a cost effective alternative to OP as it delivers improved clinical outcome at a reduced price.

Table 1: Economic analysis of OP vs. LP

	OP \$ mean±SEM	LP \$ mean±SEM	adjusted mean difference \$ [95% confidence interval]	p value
operative cost	3308±142	3065±228	-201 [-793, +391]	0.5
ward cost	3180±248	2480±118	-872 [-1386, -359]	0.001
other cost	732±38	645±22	-108 [-193, -24]	0.012
Grand Total cost	7220±324	6191±252	-1182 [-1994, -370]	0.005

Notes:

DEVELOPMENT AND PRELIMINARY VALIDATION OF A NEW PEDIATRIC FUNDAMENTALS OF LAPAROSCOPIC SURGERY SIMULATOR

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Purpose:

To develop a simulator for training and assessment of pediatric laparoscopic surgical skills and complete preliminary validation of the model.

Methods:

A pediatric fundamentals of laparoscopic surgery (PFLS) simulator based on the existing adult Fundamentals of Laparoscopic Surgery (FLS) model was developed to reflect differences in size, ergonomics and unique tissue characteristics in children. Participants were stratified according to level of expertise: novice (<10 adult or pediatric laparoscopic procedures/year), intermediate (10-50 pediatric laparoscopic procedures/year) and expert (>50 pediatric laparoscopic procedures/year). All participants were tested on the previously validated adult FLS simulator as well as the newly developed PFLS simulator. Standard FLS metrics and scoring were used to assess the performance on the five tasks in each simulator. Mean FLS and PFLS scores were compared using paired Student's t-test. ANOVA was used to compare PFLS performance among the 3 experience groups. Statistical significance was assessed at $p \leq 0.05$.

Results:

Although mean FLS and PFLS total scores were not significantly different, the PFLS intracorporeal suturing score was lower than that in its adult counterpart ($p= 0.001$). Among all participants, the pattern cutting score was higher in the PFLS than in the FLS simulator ($p< 0.001$). The differences in the other three tasks were not significant. Total PFLS scores differentiated novice from intermediate ($p<0.001$) and from expert ($p<0.001$), but not intermediate from expert ($p= 0.66$).

Conclusions:

Our preliminary data suggests that this new PFLS simulator allows discrimination between the novice and the more experienced surgeon providing evidence for construct validity (intermediate and expert levels). Of the five PFLS tasks assessed, intracorporeal suturing seemed to reflect limitations of pediatric laparoscopic surgery most effectively. Further development of the PFLS simulator and its five tasks are required to establish its role in teaching and validating laparoscopic skills in the pediatric population.

Notes:

FACE AND CONTENT VALIDITIES OF A LOW FIDELITY LAPAROSCOPIC PYLOROMYOTOMY SIMULATION

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Purpose:

A low fidelity model for laparoscopic pyloromyotomy was developed as a component of a teaching module for surgical residents. To evaluate the face and content validities of the model, pediatric surgeons used the simulation and completed questionnaires about its realism and accuracy.

Methods:

After using the simulation, surgeons completed a 12-item questionnaire defining their level of training, number of pyloromyotomies completed in the previous year, and perceived skill level for laparoscopic pyloromyotomy. Surgeons also rated level of agreement on a four-point scale (1=strongly disagree, 4 = strongly agree) with statements concerning the accuracy with which the model simulated essential components of pyloromyotomy. Surgeons rated the quality of the model as a teaching tool. Descriptive statistics were used to analyze responses. Chi square tests were performed to determine if level of experience influenced responses.

Results:

29 pediatric surgeons used the model and completed the questionnaire. A majority had performed from 26 to more than 50 pyloromyotomies during the previous year. Nearly two-thirds (59%) of the surgeons' self-perceived skill at performing laparoscopic pyloromyotomy was advanced or expert. The surgeons agreed that the model accurately simulated essential components of the pyloromyotomy (mean = 3.2, SD = .4). They indicated that the model would be an excellent tool to introduce surgeons with only basic laparoscopic skills (mean = 3.4, SD = .5), surgeons with expert skills (mean = 3.3, SD = .5) and residents/fellows (mean = 3.5, SD = .6) to laparoscopic pyloromyotomy. No influence on item responses was found based on level of experience with pyloromyotomy.

Conclusion:

A low fidelity model can accurately simulate essential components of laparoscopic pyloromyotomy. Face and content validities of the simulation are demonstrated. Construct validity and efficacy of the model is underway and will be discussed.

Notes:

LONG-TERM SURGICAL OUTCOMES IN 93 SURVIVORS OF CONGENITAL DIAPHRAGMATIC HERNIA (CDH)

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Purpose:

Operative management of CDH has improved substantially, but data regarding long-term outcomes is necessary in order to further refine repair techniques and enhance patient care. We retrospectively analyzed data from a cohort of CDH survivors in order to characterize surgical outcomes and compare the durability of different types of patch repair.

Methods:

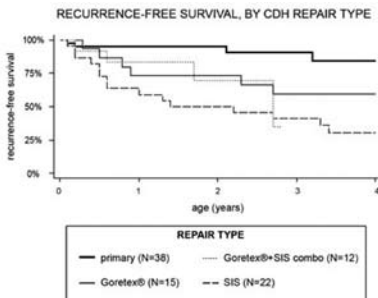
Data was prospectively gathered from a cohort of CDH patients who were followed in a multidisciplinary clinic. The incidence of CDH recurrence and other postoperative complications was documented. Logistic regression analysis of perinatal and operative history was performed to determine predictors of adverse surgical outcomes. Univariate statistical techniques used included chi-squared, Fisher's exact, and the Wilcoxon rank sum tests. Hazard ratios (HR) and Kaplan-Meier analysis were used to estimate relative risk. A P value of less than 0.05 was considered significant.

Results:

Ninety-three patients underwent CDH repair with a median current age of 4.5 years, with one death in the group (1%). A total of 30 patients (32%) have had a CDH recurrence, 11 patients (12%) have required laparotomy for bowel obstruction, and 36 (39%) have developed a significant chest deformity. An initial CDH patch repair using SIS alone generated the highest estimated risk of recurrence out of all repair types (HR 7.0, $P=0.001$, **Figure 1**), though Goretex®+SIS composite repairs approached equivalent recurrence-free survival by three years of age. Bowel obstruction was significantly more common in patients with a history of patch repair, hernia recurrence, and low birth weight ($P<0.01$). A patch repair was independently predictive of subsequent chest deformity (odds ratio 9, $P=0.02$).

Conclusions:

Long-term operative complications are common amongst CDH survivors, particularly in those with an initial patch repair; therefore, the participation of a pediatric surgeon is vital in the follow-up of these patients.



Notes:

LONG TERM FUNCTIONAL OUTCOME AND QUALITY OF LIFE IN PATIENTS WITH HIGH IMPERFORATE ANUS

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Purpose:

Anorectal malformations (ARMs) are associated with a large number of functional sequelae which may affect a child's long-term quality-of-life (QOL). The purpose of this study was to better quantify patient's functional stooling outcome, and then attempted to identify how these outcomes related to the QOL in patients with high imperforate anus.

Methods:

48 patients from two children's hospitals underwent repair of high imperforate anus, and were >4 y.o. when their family's were interviewed. Clinical functional scoring consisted of a 13-item questionnaire to assess long-term stooling habits (score range: 0-30); and were classified into 3 groups: A:30-21(good); B:20-11(fair); and C:10-0(poor). The QOL scoring was based on a 7 item questionnaire, and ranged from 0-16; Good(16-11),Fair(10-6), and poor(5-0). Statistical analysis included ANOVA and linear regression analysis.

Result:

All were surgically cared for between 1998-2003. Comparison of the QOL and clinical scoring showed no significant difference between the 2 institutions ($P>0.05$). Mean(\pm SD) age at survey was 6.5 ± 1.6 years. There was direct correlation between the QOL and Clinical score (Pearson $r^2=0.827$; beta-coefficient=24.7, $P<0.001$). A significant correlation was identified between age at survey and both the functional stooling score (Pearson $r^2=0.334$, $P=0.02$) and QOL (Pearson $r^2=0.265$, $P=.068$); in each case lower(poorer) scores increased with increasing age. Patients with associated congenital anomalies had a high rate of poor QOL (8, 42.1%; $P=.001$). Severe soiling >5/day (5 patients,10.4%) and those with severe constipation (6 patients,12.5%) showed significant correlation with low(poor) stooling scores ($P=.001$). Stooling scores also decreased significantly with increasing severity/complexity of the ARM ($P=.001$).

Conclusion:

A significant number of children suffered from functional problems and these were directly associated with poor QOL. In contrast to previous perceptions, our study showed that QOL and stooling patterns are perceived to worsen with age. This suggests that children with ARMs need long-term follow-up and counseling.

Notes:

MAGNETIC MINI-MOVER PROCEDURE FOR PECTUS EXCAVATUM II: AN FDA-SPONSORED TRIAL (IDE #G050196)

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Purpose:

The Magnetic Mini-Mover Procedure uses magnetic force to pull the sternum forward and gradually remodel deformed costal cartilage in patients with pectus excavatum. Under IRB approval, we studied the safety and efficacy of this procedure in 10 otherwise healthy patients aged 8-14 with Pectus Severity Index > 3.5 (normal = 2.56).

Methods:

In an outpatient procedure, a titanium-encased 1½" x 3/16" neodymium-iron-boron disc magnet was attached to the sternum through a subxiphoid incision. A second magnet was housed in a low-profile brace that was custom-fitted to the patient and held in place solely by magnetic attraction. Force level was patient-adjustable and was measured and logged every 10 minutes. Patients were evaluated monthly.

Results:

1. *Implant Procedure:* Operating time decreased from 90 to 30 minutes as implant techniques evolved. Nine were outpatient procedures; one patient was admitted for observation. Three patients required evacuation of retained pleural air postoperatively and two required a second outpatient procedure (one to loosen the implant and one to replace an uncoupled implant).
2. *Brace Wear Compliance:* Mean daily brace wear-time ranged from 10.4 to 21.4 hours (mean = 16.8 h/day). Wear-time increased as brace design evolved.
3. *Pectus Severity Improvement:* All methods of assessing severity (imaging, volume, photo, depth gauge) showed gradual improvement in all ten patients. Mean time to satisfactory correction varied with age: in patients aged 8-12, correction took < 10 months; in patients aged 13-14, correction took up to 18 months.

Conclusions:

1. The Magnetic Mini-Mover Procedure is a safe, outpatient, minimally-invasive, and cost-effective treatment for pectus excavatum.
2. Patients were surprisingly compliant in wearing the external magnet throughout the day and night.
3. Pectus deformity improved more rapidly in pre-pubertal patients, but all were satisfied with the correction achieved. The Magnetic Mini-Mover Procedure will be further assessed in a multicenter trial.

Notes:

A NEW VIDEOSCOPIIC DEVICE TO AVOID CARDIAC INJURY IN MINIMALLY INVASIVE PECTUS EXCAVATUM REPAIR: THE PECTOSCOPE

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Purpose:

The most devastating complication during the minimally invasive repair of pectus excavatum is a cardiac perforation. Conventional thoracoscopy, however, does not provide visualization to the most critical portion of the dissection, i.e. the contact-point between the depressed chest wall and the heart. To grant complete safety from cardiac injury, we developed a new videoscopic device, the pectoscope, which provides continuous visualization-guided steering through the entire path.

Methods:

Among a single surgeon's experience with 1,170 pectus excavatum repairs, 117 patients were repaired with the aid of the pectoscope between April 2008 and September 2008. The pectoscope consists of three units: a fiberscope; a curved hollow sheath; and a transparent observation-dissection module. It combines three functions in a single system: visualization; a dissection-introducer; and a guide for bar passage. (Figure 1) The pectoscope is introduced into the thoracic cavity directly through the hinge point. With a continuous view, the plane between the chest wall and the heart can be dissected through the hinge point, and then; the sheath utilized as a guide.

Results:

The mean age of the patients was 10.3 years (range 2-33 years). The male to female ratio was 3. Adult patients (age= \geq 15 years) were 28.2% (n=33). 70(60%) patients were symmetric and 47 (40%) were the asymmetric type. There were 10 minor complications (8.5%): pneumothorax (n=2, 1.7%), pleural effusion (n=5, 4.3%), and wound seroma (n=3, 2.6%). There were no problems related to the pectoscopy. Outcomes were excellent in 116/117 (99.1%) and; good in 1/117(0.9%).

Conclusions:

The novel videoscopic device, the Pectoscope, seems to be effective in preventing cardiac injury and make the procedure simple and trouble-free. A continuous view to a critical plane throughout the whole pathway guarantees safety, and triple functions-visualization, dissection, and a guide- may replace the hassle of a conventional introducer and thoracoscopy.

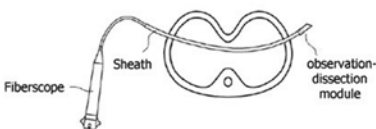


Figure 1. The pectoscope consists of three units: a fiberscope, a curved hollow sheath for introduction of the fiberscope, and a transparent observation-dissection module at the tip of the sheath. It combines three functions in a single system: visualization; a dissection-introducer (tunnel); and a guide for bar passage.

Notes:

INTESTINAL LENGTHENING USING AN IMPLANTABLE SPRING: A NOVEL APPROACH FOR THE TREATMENT OF SHORT BOWEL SYNDROME

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Purpose:

Previously, intestinal segments were lengthened with an external device that applied gradual mechanical tension. The feasibility of in-vivo intestinal lengthening using an implantable spring was evaluated in this study.

Methods:

Biocompatible Nitinol springs capable of five-fold expansions were compressed using absorbable sutures and were implanted into isolated segments of proximal jejunum in rats. Springs compressed with non-absorbable sutures served as controls. The animals were followed with serial abdominal x-rays until the springs became fully expanded. Intestinal segments were then retrieved for histological analyses. Student's t-test was employed for statistical analyses.

Results:

Intestinal segments implanted with springs compressed with non-absorbable sutures demonstrated no significant lengthening. Intestinal segments implanted with springs compressed with fast-absorbing plain gut sutures led to the full expansion of the implanted springs within one week, but the springs eroded through the ends of the intestinal segments. Intestinal segments implanted with springs compressed with slower-absorbing Chromic sutures led to the full expansion of the implanted springs after eight weeks. These intestinal segments were lengthened 4.9-fold compared to 1.2-fold lengthening in the controls ($p < 0.05$). After the removal of the springs, the intestinal segments recoiled but still demonstrated a greater than 4-fold increase in length ($p < 0.05$). Histological analyses revealed smooth muscular thickening but otherwise normal architecture.

Conclusions:

Continuous mechanical force with an implantable spring successfully lengthened isolated segments of small bowel in an animal model. While similar results have been demonstrated using other devices, the current device is totally implantable and offers the potential of use in non-isolated intestinal segments.

Notes:

EVALUATION OF A NOVEL SMALL DIAMETER TISSUE ENGINEERED ARTERIAL GRAFT

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Purpose:

The implantation of tissue-engineered venous interposition grafts for the repair of congenital anomalies is already a clinical reality in pediatric patients, however such work using similar arterial grafts is limited because of the concern for the development of aneurysm formation and rupture. To this end, our group developed a novel tissue-engineered vascular graft with an improved biomechanical profile suitable for implantation into the arterial system. This pilot study demonstrates the feasibility of constructing such grafts and the clinical utility of these constructs as arterial interposition grafts.

Methods:

Biodegradable tissue-engineered vascular conduits (0.6 mm internal diameter) were fabricated by electrospinning poly-L-lactic acid onto a rotating mandril and applying a copolymer poly-caprolactone-lactide sealant. Following biomechanical characterization, the grafts were implanted as infrarenal aortic interposition grafts in immunocompromised mice (n=4) in accordance with IACUC guidelines and followed for up to 4 weeks post-implantation without the use of anticoagulation or antiplatelet therapy. Serial ultrasounds of the grafts were conducted in vivo to evaluate for graft stenosis, thromboembolic complications or aneurysm formation. Histologic and immunohistochemical techniques were employed to further evaluate graft morphometry and characterize cellular composition.

Results:

Electrospun tissue-engineered vascular grafts can withstand pressures of up to 3100 mmHg. All grafts implanted in this study remained patent without evidence of thromboembolic complications. Histologic analysis revealed collagen deposition with cellular infiltration throughout the grafts. There was no evidence of occlusion, dilation or aneurysm formation. Immunohistochemical evaluation revealed the development of a circumferential smooth muscle cell layer and a monolayer of endothelial cells lining the lumen.

Conclusion:

We conclude that electrospun tissue-engineered arterial conduits can be constructed such that the grafts maintain the strength necessary to withstand high arterial pressures and can undergo successful implantation as aortic interposition grafts with remodeling leading to the development of neotissue resembling that of the native aorta.

Notes:

TWO-STAGE BASILIC VEIN TRANSPOSITION-A NEW APPROACH FOR PEDIATRIC DIALYSIS ACCESS

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Purpose:

With the Fistula First program, there has been increased effort to create arteriovenous fistulas (AVF) as primary dialysis access. Two-Stage basilic vein transposition (Two-Stage BVT) allows maturation of smaller veins, often a limiting factor in the pediatric population, prior to elevation and use. We sought to determine whether using Two-Stage BVT improves maturation, use and patency compared to Other AVF, including AV grafts (AVG).

Methods:

Thirty-one patients underwent AV access creation between 2002-2008. Data were collected on types of access, maturation (by ultrasound criteria or successful access for dialysis), complications and patency. SPSS was used for descriptive, chi-square and t-test analyses (significance $p < 0.05$).

Results:

Forty-two AV access procedures were performed: 15(36%) two-stage BVT, 13(31%) one-stage BVT, 6(14%) radiocephalic, 4(10%) brachiocephalic, and 4(10%) AVG. Follow-up averaged 22 ± 3 months for Two-Stage BVT and 31 ± 5 months for Other AVF ($p = 0.03$). All Two-Stage BVT matured, compared to 48%(13/27) of Other AVF ($p = 0.001$). More Two-Stage BVT fistulas (87% or 13/15) were used for dialysis than Other AVF (52% or 14/27, $p = 0.024$). Fistula failure occurred in 7% Two-Stage BVT compared to 67% Other AVF ($p < 0.001$). Primary patency (requiring no interventions to maintain patency) rates were higher among Two-Stage BVT fistulas (67%) compared with Other AVF (15%) and assisted primary patency (requiring intervention to maintain patency) rates were lower among Two-Stage BVT fistulas (20%) compared to Other AVF (37%, $p = 0.003$).

Conclusions:

There is an increased rate of fistula maturation and use for dialysis as well as lower rates of fistula failure with Two-Stage BVT. Thrombosis-free patency was higher with Two-Stage BVT, reflected in significantly higher primary patency rates and lower assisted primary patency rates. The two-stage BVT shows great promise as the preferred approach to creation of AVF in pediatric patients with smaller veins.

Notes:

PROTEOMICS FOR THE IDENTIFICATION OF CANDIDATE BIOMARKERS OF PROGRESSIVE NEC IN HUMAN INFANTS

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¹Stanford University, Stanford, CA, USA, ²Yale University, New Haven, CT, USA, ³Baylor University, Houston, TX, USA, ⁴Johns Hopkins University, Baltimore, MD, USA, ⁵Boston Children's Hospital, Boston, MA, USA, ⁶UCLA and Mattel Children's Hospital, Los Angeles, CA, USA, ⁷Children's Hospital of Philadelphia, Philadelphia, PA, USA, ⁸Hospital of the University of Pennsylvania, Philadelphia, PA, USA

Purpose:

Necrotizing Enterocolitis (NEC) is among the leading causes of morbidity and mortality in premature infants. Despite decades of research, attempts to identify clinical parameters that reliably identify infants with severe NEC that are likely to progress to irreversible disease have been unsuccessful. We hypothesized that protein biomarkers of progressive disease could be identified in the peripheral blood of infants with early stage NEC using proteomic and bioinformatic techniques.

Methods:

Premature infants with early stage NEC as defined by Bell's criteria were enrolled in the NEC Consortium prospective national database. Plasma from infants with progressive NEC (defined as those infants requiring surgery or drain placement) (n=19) and non-progressive NEC (defined as those infants whose disease could be managed medically) (n=26) were subjected to anion exchange chromatography followed by surface-enhanced laser desorption/ionization time of flight mass spectrometry (SELDI-TOF-MS). Both instrument software and simultaneous spectrum analysis techniques were utilized to extract reproducible protein peak information. Mann-Whitney U-Tests and False Discovery Rates (FDR) were applied to identify likely candidate biomarkers. MS peaks with an FDR less than 5% were identified.

Results:

313 MS peaks out of 1191 total peaks were found with a $p < 0.011$ and an $FDR < 5\%$. Of these, 40 peaks were found to have $p < 1 \times 10^{-5}$ with an FDR of 0. The ROC curves, as a measure of the sensitivity and specificity, identified 8 peaks (as distinguishing progressive or non-progressive disease) with an ROC of = to or > 0.85 . Previous results using only clinical and demographic data were not as useful in distinguishing the two groups.

Conclusions:

Using proteomic and bioinformatic techniques we were able to identify several protein MS peaks that are candidate biomarkers of NEC. The powerful combination of a national prospective NEC database combining epidemiologic data and biologic studies may lead to new diagnostic and prognostic parameters for NEC.

Notes:

HB-EGF PRESERVES INTESTINAL MICROVASCULATURE AND STRUCTURAL ARCHITECTURE IN RAT PUPS WITH EXPERIMENTAL NECROTIZING ENTEROCOLITIS

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 Nationwide Children's Hospital, Columbus, OH, USA

Purpose:

Necrotizing enterocolitis (NEC) is the most common gastrointestinal emergency in neonates. Although the exact etiology of NEC remains unknown, decreased intestinal blood flow may play a critical role. The goal of this study was to assess the effect of heparin-binding EGF-like growth factor (HB-EGF) on mesenteric microcirculatory blood flow and intestinal injury in preterm rat pups subjected to experimental NEC.

Methods:

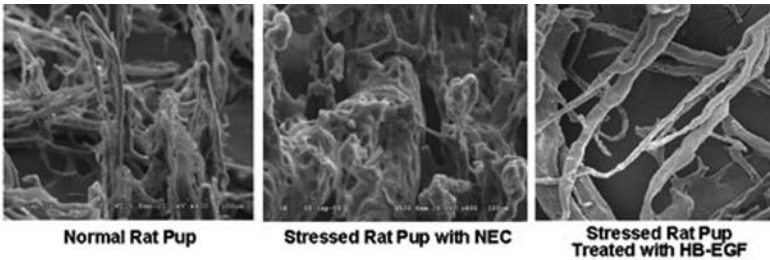
Newborn rat pups were stressed for 3 days by hypertonic formula feeding, hypoxia, hypothermia, and administration of lipopolysaccharide, with some pups receiving HB-EGF (800 µg/kg/dose) enterally. Control animals received breast milk for 3 days. Villous microcirculation was assessed by FITC-dextran angiography, with intestinal villous and submucosal microcirculatory blood flow evaluated by confocal microscopy. Villous microvasculature was additionally studied by vascular corrosion casting and scanning electron microscopy (SEM). Intestinal injury was graded using a histologic injury scoring system.

Results:

Microcirculatory blood flow to the intestine was significantly decreased by 37% in the villi and by 36% in the submucosa in pups subjected to stress compared to breast fed pups, as determined by FITC-dextran angiography ($p < 0.05$). Stressed pups treated with HB-EGF had a 65% increase in villous blood flow and a 40% increase in submucosal blood flow compared to stressed pups that did not receive HB-EGF ($p < 0.05$). These results were confirmed by SEM of the microvasculature, which showed intact microvasculature in breast fed pups, degraded microvasculature in stressed pups, and preserved microvasculature in stressed pups treated with HB-EGF (see Figure). The changes in villous microvasculature correlated with histologic injury scores, with stressed pups treated with HB-EGF showing decreased histologic injury ($p < 0.05$).

Conclusions:

HB-EGF greatly promoted intestinal microcirculatory blood flow in newborn rat pups subjected to experimental NEC, indicating that HB-EGF may play a key role in the therapy of various diseases manifested by decreased intestinal blood flow, including NEC.



Notes:

EXOGENOUS TREFOIL FACTOR 2 MARKEDLY IMPROVES SURVIVAL AFTER INTESTINAL ISCHEMIA.

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Introduction:

Necrotizing enterocolitis (NEC), a syndrome affecting premature infants involves a breach in the mucosal barrier. Trefoil factor 2 (TFF2) plays a critical role in mucosal defense and repair. In a murine model of NEC, we have found that mice lacking TFF2 had more severe intestinal injury a prolonged cytokine response and higher mortality than wild type mice. We now examine the effect of exogenous TFF2, in combination with the probiotic bacteria *Lactococcus lactis*, upon overall survival after intestinal ischemia.

Methods:

Lactococcus lactis was genetically modified to secrete TFF2. Paired TFF2 knockout mice were separated into 2 groups; those that received TFF2 (n=38) and those that received the control *L lactis* (n=38). Animals underwent 45 minutes of midgut ischemia as previously described. Mice received a daily intragastric administration of 1×10^9 of TFF2-secreting and non-secreting *L. lactis* on days -1, 0, 1, 2, and 3 post-ischemia. Animal survival was monitored for 30 days.

Results:

The 30-day survival rate of TFF2 knockout mice after midgut ischemia is <10%. TFF2 knockout mice receiving the probiotic *L lactis* alone, had an improvement in survival from 10 to 43%, while TFF2 knockout mice that received the combination of TFF2 and *L. lactis* had an even greater improvement in survival from 10 to 66%. .

Conclusions:

NEC remains a difficult disease to prevent and to halt its progression once initiated. Use of probiotic bacteria is now being intensely studied as one possible therapeutic candidate. We have now demonstrated that the probiotic bacteria, *Lactococcus lactis* alone and genetically modified to produce TFF2 markedly improves survival after intestinal ischemia/reperfusion. Exogenous administration of bioactive TFF2 in combination with the probiotic *L. lactis* bacteria may be a useful therapeutic approach for managing NEC. The exact mechanisms of this protection still need to be addressed.

Notes:

FOUR INTRAVENOUS LIPID EMULSIONS AND THEIR EFFECTS ON HEPATIC STEATOSIS IN A MURINE MODEL

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

Purpose:

To determine what type of intravenous lipid emulsion is best at preventing hepatic steatosis in a murine model of parenteral nutrition-associated liver disease.

Methods:

Twenty-five C57BL/6J mice were placed on a fat free, high carbohydrate diet (HCD) and randomized into 5 equal groups. Each group received one of four commercially available lipid emulsions (soybean oil, soybean/safflower oil, soybean/olive oil, or fish oil) or saline control via tail vein injection every other day (2.4 grams/kg/day) for 19 days. Control mice received standard rodent chow (n=5). Mice were sacrificed after 19 days. Serum was analyzed for liver function tests and livers were analyzed for degree of steatosis by hematoxylin and eosin, Oil Red O staining, and Magnetic Resonance Imaging (MRI). Comparison of medians between groups was made using the One Way Analysis of Variance, and differences deemed significant at $p < 0.05$.

Results:

Histology revealed severe steatosis in both the soybean and saline alone groups. Livers in the fish oil group were normal. Livers in the soybean/safflower oil and soybean/olive oil groups had moderate microvesicular steatosis. Median alanine aminotransferase (ALT) values were significantly higher in the mice receiving the HCD + saline [97 IU/L (IQR 82.3-114.0)] when compared to control mice [35 (IQR 32.3-40.5) ($p=0.008$)]. Similarly, the mice receiving fish oil had significantly lower median ALT levels [37 IU/L (IQR 32.3-43.8)] than the ones receiving the HCD + saline [97 IU/L (IQR 82.3-114.0) ($p=0.008$)]. Finally, ALT values in the fish oil group were not statistically different than chow controls ($p=0.84$). Quantitative analysis by MRI revealed significantly less steatosis in the fish oil group when compared to the mice receiving HCD + saline alone ($p < 0.001$).

Conclusion:

Intravenous lipid emulsions containing fish oil are superior at preventing hepatic steatosis in a murine model of parenteral nutrition-associated liver disease.

Notes:

DOCOSAHEXAENOIC ACID AND ARACHIDONIC ACID PREVENT ESSENTIAL FATTY ACID DEFICIENCY AND HEPATIC STEATOSIS IN A MURINE MODEL OF PARENTERAL NUTRITION-ASSOCIATED LIVER DISEASE

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¹Children's Hospital Boston, Boston, MA, USA, ²Brigham and Women's Hospital, Boston, MA, USA, ³Beth Israel Deaconess Medical Center, Boston, MA, USA

Purpose:

To determine whether docosahexaenoic acid (DHA) and arachidonic acid (AA) are true essential fatty acids and if their supplementation alone can prevent essential fatty acid deficiency (EFAD) and attenuate fatty liver changes in a murine model of parenteral nutrition-associated liver disease (PNALD).

Methods:

Thirty 6 week-old C57Bl/6J mice were divided into groups of five. Treatment groups were placed on a fat-free, high carbohydrate diet (HCD) *ad libitum* and supplemented via orogastric gavage with one of four different lipid emulsions containing DHA, AA and hydrogenated coconut oil (HCO) in varying amounts. The supplemented DHA+AA (DHA: AA ratio of 20:1) provided 0%, 0.21%, 2.1% and 4.2% of daily caloric intake, and HCO was added to total 5%. Control mice received a standard rodent diet. After 19 days, mice were sacrificed and livers were graded for degree of steatosis on hematoxyline and eosin stain. Serum alanine transaminase was measured and fatty acid profiles were analyzed for EFAD as determined by serum triene:tetraene ratio >0.2.

Results:

Mice on HCD only or ones supplemented with HCO alone developed severe hepatic steatosis (median steatosis scores 3) and severe EFAD (serum triene:tetraene ratios 0.84 ± 0.12 and 0.84 ± 0.15 , respectively). Mice on 0.21% DHA+AA developed mild steatosis (median steatosis score 1) and EFAD (serum triene:tetraene ratio 0.42 ± 0.04). Mice on 2.1% or 4.2% DHA+AA had normal livers (median steatosis scores 0) and no EFAD (serum triene:tetraene ratios 0.02 ± 0.01 and 0.03 ± 0.01 , respectively). Finally, the 4.2% group had significantly lower alanine transaminase compared to mice on HCD only (28.3 ± 1.1 U/L vs. 77.8 ± 16.0 U/L, $p < 0.05$).

Conclusions:

Supplementation of DHA and AA alone, at appropriate amounts, can prevent both EFAD and hepatic steatosis in a murine model of PNALD. This indicates that DHA and AA are the true essential fatty acids.

Notes:

GROWTH FACTOR MODULATION OF HEPATIC INFLAMMATION: A NOVEL APPROACH TO THE MANAGEMENT OF TPN ASSOCIATED LIVER DISEASE

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Purpose:

Dependence on total parenteral nutrition (TPN) in intestinal failure or short bowel syndrome (SBS) patients can lead to many complications. The most significant complication is chronic liver injury leading to liver failure that requires transplantation. This study was designed to assess hepatocyte growth factor's potential in modulating the hepatic response in a cholestatic liver injury model.

Methods:

Female Sprague-Dawley rats were divided into three groups: control(N=5); chronic liver injury(ANIT every 3.5 days at 75mg/kg, N=5); and chronic liver injury and hepatocyte growth factor(ANIT + HGF at 250µg/kg/day, N=5). The rats initially underwent massive small bowel resections. Seven days later, they were given an initial intraperitoneal(IP) injections of saline(control) or ANIT, and implantation of a fourteen day osmotic mini-pump for continuous intravenous saline(control and chronic liver-injury groups) or HGF. IP Saline or ANIT injections were subsequently administered every 3.5 days to create a chronic cholestatic model. After 14 days of bi-weekly IP injections, the animals were euthanized and liver biopsies were obtained. The liver biopsies were evaluated by histology, immunofluorescence(IF) staining for IL-6 and TNF- expression, and assessment of apoptosis by TUNEL technique. Analysis of variance(ANOVA) was used to determine statistical significance.

Results:

In this chronic liver injury model HGF did not effect the grade of inflammation, however, HGF did induce retention of the ductal structures, and avoided ductal proliferation, damage and evidence of primary sclerosing cholangitis ($p<0.05$). HGF induced less IL-6 ($p<0.01$) and TNF- expression($p<0.01$). In addition, apoptotic activity was also significantly less in the HGF group($p<0.01$).

Conclusions:

HGF preserved the hepatic ductal system, modulated the hepatic inflammatory response and reduced the apoptotic index in this chronic cholestatic liver injury model. HGF may diminish or prevent liver damage in patients with TPN-induced liver injury.

Notes:

A MOUSE MODEL OF POST-PULLTHROUGH HIRSCHSPRUNG ASSOCIATED ENTEROCOLITIS

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Purpose:

To develop an animal model of post-pullthrough Hirschsprung's associated enterocolitis (HAEC).

Methods:

After obtaining IACUC approval, we employed the targeted endothelin receptor B (Ednrb)-null mouse, a model of HD with short-segment aganglionosis known to develop enterocolitis. First, we characterized the prevalence and severity of enterocolitis in non-operated Ednrb^{-/-} mice (n=18) compared to nonoperated littermate wildtype controls (n=5). At a median of 24 days of life, Ednrb^{-/-} mice became clinically ill, at which point we harvested colon and ileum, and cultured blood, peritoneal lavage, spleen, kidney and liver. Enterocolitis was semi-quantitatively evaluated by two blinded pathologists using a new murine scoring system and then compared with level of bacteremia using Wilcoxon rank-sum test and Spearman correlations. Next, we performed a novel, single-stage pullthrough operation (JPS *in press*) on Ednrb^{-/-} (n=8) and wildtype (n=6) mice. Six weeks after surgery we harvested and cultured tissues, and analyzed them as described above. Enterocolitis scores between groups (nonoperated vs. pullthrough) for each genotype were compared with t-test.

Results:

We found 62% of Ednrb^{-/-} mice developed bacteremia with a mean histologic score of 5.1 ± 1.7 (\pm SD), which was significantly worse than the mean histologic score of 2.0 ± 2.0 in non-bacteremic mice ($P=0.010$). Enterocolitis scores positively correlated with bacterial colony forming units in peritoneal lavage, kidney and liver ($r=0.41$, $P=0.04$). Wildtype controls had essentially no inflammation and no bacteremia. The second experiment found that Ednrb^{-/-} mice that underwent the pullthrough operation had lower enterocolitis scores than nonoperated Ednrb^{-/-} mice (4.2 ± 0.5 vs. 3.3 ± 0.5) N.S., although the pullthrough procedure itself introduced significant inflammation in the wildtype mice. No bacteremia was found in the operated group.

Conclusions:

The pullthrough operation rescues the lethality of the Ednrb^{-/-} mice. Furthermore, pullthrough reduces, but does not eliminate enterocolitis in the Ednrb^{-/-} mice, making this a valuable model to study mechanisms of HAEC.

Notes:

EFFECTS OF NOTCH4 ON LUNG VASCULAR REMODELING

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Background:

Many pediatric lung vascular diseases are incompletely understood at the molecular level. Some of these include arteriovenous malformations, pulmonary hypertension, alveolar capillary dysplasia, and bronchopulmonary dysplasia. Notch signaling, which is critical to developing tissues and arteriovenous specification, may play a role in pediatric lung vascular disease.

Objective: To test the hypothesis that constitutive expression of Notch4 in the endothelium causes vascular lesions in lungs.

Methods:

With IACUC approval, we used a transgenic mouse that, under temporal control of a tetracycline response element, constitutively expresses the active domain of Notch4 specifically in the endothelium (Tie2-tTA:TRE-int3, "mutants"). Notch4 expression began at weaning and the mice were studied 4, 6 and 8 weeks thereafter. Mutants were compared to littermate controls. Routine histology, immunohistochemistry and angiograms were used to characterize lung pathology. Functional tests included arterial blood gas measurements and Evans Blue permeability assays.

Results:

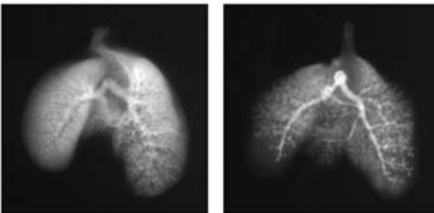
Mutants exhibited impaired lung function 6 weeks after gene expression. Mean pO₂ decreased by 22%±5 (p=0.004) and mean pCO₂ increased by 58%±16 (p=0.004) compared to controls. Gross examination of mutant lungs revealed hemorrhages (10.6±3.1 hemorrhages in mutants vs. 0.3±0.1 in controls, p=0.005). Vascular permeability was similar between mutant and control even after ventilator induced injury. Angiograms showed poor filling of distal arterioles and pruning of the blood vessels in the mutants versus controls. Immunohistochemistry for smooth muscle cell alpha actin (SMA) demonstrated that mutants trended towards fewer blood vessels with no overall difference in muscularization (SMA intensity).

Conclusions:

Constitutively expressed endothelial Notch4 is associated with lung hemorrhages and impaired lung function in mice. Hemorrhages are not due to increased vascular permeability and may be related to anatomic structural vascular defects such as arteriovenous malformations and improperly remodeled blood vessels.

Control (Tie2-tTA)

Tie2-tTA : TRE-int3



Six weeks after Notch4 expression, barium sulfate-gelatin pulmonary angiograms show poor filling of distal arterioles and pruning of the blood vessels in the mutants (right) versus controls (left).

Notes:

SECRETED MIDKINE CONFERS DOXORUBICIN RESISTANCE IN HUMAN SK-N-SH NEUROBLASTOMA CELLS BOTH *IN VITRO* AND *IN VIVO*

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Purpose:

Previously, we investigated the hypothesis that a secreted factor in neuroblastoma cells confers drug resistance to other tumor cells in the same mass. To study this hypothesis, a human neuroblastoma cell line resistant to doxorubicin was established (SK-N-SH/Dox6). A cDNA array was performed on both wild and SK-N-SH/Dox6 cells and found that midkine was expressed only in drug resistant cells. We then confirmed that midkine was markedly upregulated in SK-N-SH/Dox6 cells via Western blot. Media from resistant cells conferred doxorubicin resistance to SK-N-SH/W cells. The purpose of this study is to confirm these results with co-culture experiments and *in vivo*.

Methods:

Measurement of intracellular and secreted midkine in SK-N-SH/Dox6 and SK-N-SH/W cells was determined by Human Midkine ELISA kit. Co-culture experiments were performed using 6 well plates with inserts. The plates contained SK-N-SH/W, SK-N-SH/Dox6 or SK-N-SH/Dox6 cells treated with siRNA to midkine. All inserts contained SK-N-SH/W cells. After incubation with doxorubicin, viable cells in the inserts were counted. For the *in vivo* experiments, SCID mice were injected with SK-N-SH/W/GFP cells (labeled with GFP to delineate cells from SK-N-SH/Dox6 cells) and SK-N-SH/Dox6 cells in various ratios. Once the tumors were palpable, the mice were treated with three injections of Doxorubicin (2.5mg/kg) intraperitoneally every 3 days. Tumor volume was measured.

Results:

ELISA showed that both SK-N-SH/W and SK-N-SH/Dox6 cells produce midkine but only SK-N-SH/Dox6 cells secrete midkine. Co-culture experiments determined that SK-N-SH/W cells have increased survival after doxorubicin exposure when cultured with SK-N-SH/Dox6 cells but not those pretreated with siRNA to midkine. The *in vivo* experiments illustrated that resistance is conferred from the SK-N-SH/Dox6 cells to the SK-N-SH/W/GFP cells.

Conclusions:

This study confirms that drug resistant cells confer factors to drug sensitive cells both *in vitro* and *in vivo*. We also determined the likely protein for this effect is midkine.

Notes:

THE UTILITY OF ONCOLYTIC VIRUSES AGAINST NEUROBLASTOMA

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Background:

Neuroblastoma is the second most common pediatric extracranial malignant tumor. High-risk patients do poorly and little progress has been made in improving their outcome. A subset of tumor cells called Tumor Initiating Cells (TICs) may drive aggressive tumor behavior and treatment resistance. A neuroblastoma TIC has been identified, but their role in tumor behavior is not yet characterized and their susceptibility to existing and novel cancer treatments is unknown. Myxoma and Vesicular Stomatitis Virus (VSV) are two oncolytic viruses that have been shown to effectively destroy brain tumor cells, which share a common ancestry with neuroblastoma in that both are derived from neural crest cells. It is unknown if these oncolytic viruses can effectively destroy neuroblastoma cells or neuroblastoma TICs.

Objectives:

Characterize the ability of VSV and Myxoma to target and destroy 1) neuroblastoma cells; and 2) neuroblastoma TICs.

Methods:

In vitro cellular viability assays on infected neuroblastoma and neuroblastoma TIC lines were performed. Infection and viral protein production was assessed by intergenic fluorescent protein expression, Western blot detection of viral proteins from infected cell lysates, and measurement of infected cell lysate viral activity by plaque assay. Following IACUC approval, *in vivo* viral activity was measured using intratumoral injection in an established human neuroblastoma mouse xenograft model.

Results:

Both myxoma and VSV infect and kill neuroblastoma cells *in vitro*. Cytopathic effects and green fluorescent protein (Myxoma) or rhodamine (VSV) expression were both seen after infection. Infection of neuroblastoma cells was further confirmed by Western blot detection of myxoma and VSV viral protein expression. Further, both myxoma and VSV were found to effectively inhibit the growth of neuroblastoma in subcutaneous xenografts.

Conclusions:

Myxoma and VSV effectively destroy several neuroblastoma cell lines and may offer a novel approach to treatment of high-risk neuroblastoma.

Notes:



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