APSA 37th Annual Meeting

May 21–24, 2006

HILTON HEAD
SOUTH CAROLINA

Marriott Beach & Golf Resort
Hilton Head, SC

American Pediatric
Surgical Association

Final Program

PLEASE BRING THIS PROGRAM WITH YOU

American Pediatric Surgical Association
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60 Revere Drive
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Final Program

Membership Directory

Directory begins on the flipside of the Final Program

Educational Objective

The APSA Annual Meeting is designed to provide four days of 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 program will begin with two half-day symposia: the first dealing with fetal therapy and the second addressing practice issues. Meeting attendees will also view and discuss videos and poster presentations on this day. The topics at these sessions have been selected jointly by the Program and Education committees and are based on member requests from recent surveys and on journal articles about what is relevant to their practices. The scientific sessions consist of basic research and practical clinical presentations. The poster sessions allow investigators an opportunity to share their research efforts on a more personal level.

This meeting covers a wide 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.

Accreditation Statement

APSA is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Credit Designation

APSA designates this educational activity for a maximum of 21.5 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.

Gail E. Besner, M.D. — Trillium Therapeutics, Inc.
Carroll "Mac" Harmon, M.D., Ph.D. — Stryker Endoscopy
Marion C.W. Henry, M.D., MPH — Intuitive Surgical
Robert E. Kelly Jr., M.D. — Walter Lorenz Surgical, Pfizer and Merck

No faculty members will discuss unlabeled/unapproved uses of drugs nor devices.

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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Exhibit Hours

Monday, May 22	6:30 a.m. – Noon
Tuesday, May 23	7:00 a.m. – Noon

APSA Planning Committees

APSA would like to thank the following people for their contribution to the APSA annual meeting program:

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Committee Meetings

The following is a listing of committee meetings during the APSA Annual Meeting:

Saturday, May 20

8:00 a.m. – 2:00 p.m.	Board of Governors	*Woodward*
2:00 p.m. – 3:30 p.m.	Education Committee	*Dunes*
2:00 p.m. – 6:00 p.m.	Pediatric Surgery Training Directors	*Sabal Palm*
6:00 p.m. – 8:00 p.m.	Committee on Trauma	*Dunes*
6:00 p.m. – 8:00 p.m.	Pectus Multicenter Investigator	*Indigo*
6:00 p.m. – 10:00 p.m.	Publications Committee	*Fairfield*
6:00 p.m. – 10:00 p.m.	Fetal Therapy Committee	*Woodward*

Sunday, May 21

6:30 a.m. – 8:00 a.m.	International Relations Committee	*Woodward*
6:30 a.m. – 8:00 a.m.	Outcomes Committee	*Fairfield*
4:00 p.m. – 6:30 p.m.	AAP Program Committee	*Woodward*

Monday, May 22

6:30 a.m. – 7:30 a.m.	APSA Foundation Board	*Woodward*
6:30 a.m. – 7:30 a.m.	Informatics Committee	*Fairfield*
6:30 a.m. – 7:30 a.m.	Practice Committee	*Dunes*
6:30 a.m. – 7:30 a.m.	Workforce Committee	*Lords Proprietors*
4:00 p.m. – 5:30 p.m.	ACS Advisory Council for Pediatric Surgery	*Woodward*
5:00 p.m. – 6:30 p.m.	*Journal of Pediatric Surgery* Reception	*Indigo*

Schedule at a Glance

Saturday, May 20

		Room
8:00 a.m. – 2:00 p.m.	APSA Board of Governors meeting	*Woodward*
2:00 p.m. – 5:00 p.m.	Committee meetings	
3:00 p.m. – 6:00 p.m.	Registration open	*Ballroom Foyer*
6:00 p.m. – 10:00 p.m.	Publications committee meeting/dinner	*Fairfield*
6:30 p.m. – 10:00 p.m.	APSA Board of Governors dinner	*Conroy's Restaurant*

Sunday, May 21

6:30 a.m. – 8:00 a.m.	Committee meetings	
7:00 a.m. – 5:30 p.m.	Registration open	*Ballroom Foyer*
8:00 a.m. – 11:30 a.m.	Symposium: Fetal Therapy	*Grand Ballroom*
11:30 a.m. – Noon	Refreshment break	*Ballroom Foyer*
Noon – 1:30 p.m.	Lunch with video session	*Grand Ballroom*
12:30 p.m. – 2:30 p.m.	Poster set-up	*Grand Ballroom A and B*
1:30 p.m. – 2:00 p.m.	Refreshment break	*Ballroom Foyer*
2:00 p.m. – 4:00 p.m.	Symposium: Practice Issues	*Grand Ballroom*
3:30 p.m. – 5:30 p.m.	Posters open for viewing	*Grand Ballroom A and B*
4:00 p.m. – 5:30 p.m.	Poster Presentations/Poster Viewing Area: Authors in Attendance	*Grand Ballroom A and B*
5:30 p.m. – 6:30 p.m.	Exhibit set-up	*Ballroom Foyer*
6:30 p.m. – 8:30 p.m.	Welcome Reception	*Basshead Deck*
9:00 p.m. – 10:00 p.m.	New Member Reception	*President's Suite*

Monday, May 22

6:00 a.m. – 7:30 a.m.	Annual Fun Run	*Pool—Beach Access Entrance*
6:30 a.m. – 7:30 a.m.	Committee meetings	
6:30 a.m. – 1:00 p.m.	Registration open	*Ballroom Foyer*
6:30 a.m. – Noon	Posters and Exhibits open for viewing	*Ballroom Foyer/ Grand Ballroom A and B*
6:45 a.m. – 7:30 a.m.	Continental breakfast in the exhibit area	*Ballroom Foyer*
7:30 a.m. – 9:15 a.m.	Welcome/Scientific Session 1	*Grand Ballroom*
9:15 a.m. – 10:15 a.m.	Robert E. Gross Lecture: Diana Bianchi, M.D.	*Grand Ballroom*
10:15 a.m. – 10:45 a.m.	Refreshment break	*Ballroom Foyer*
10:45 a.m. – Noon	Scientific Session 2	*Grand Ballroom*
Noon – 12:15 p.m.	Member Presentation: Robert K. Minkes, M.D., Ph.D.	*Grand Ballroom*
12:15 p.m. – 1:15 p.m.	Welcome New Members/Presidential Address: Judah Folkman, M.D.	*Grand Ballroom*

Monday, May 22 (Continued)

		Room
1:15 p.m. – 2:00 p.m.	Financial Planning for Doctors *Supported by Greenbook Financial Services*	*Grand Ballroom*
1:30 p.m. – 3:00 p.m.	Benji Brooks Luncheon	*Indigo*
2:00 p.m. – 7:00 p.m.	Golf Tournament	*Arthur Hills Golf Course*
2:00 p.m. – 6:00 p.m.	Tennis Tournament	*Palmetto Dunes Tennis Center*
3:30 p.m. – 7:00 p.m.	Kayaking Tour	*Hotel Entrance*
6:00 p.m. – 10:30 p.m.	Optional Dining Shuttle	*Hotel Entrance*

Tuesday, May 23

6:30 a.m. – 8:00 a.m.	Member business meeting and breakfast	*Grand Ballroom*
6:30 a.m. – 1:00 p.m.	Registration open	*Ballroom Foyer*
7:00 a.m. – Noon	Posters and Exhibits open for viewing	*Ballroom Foyer/ Grand Ballroom A and B*
7:00 a.m. – 8:00 a.m.	Continental breakfast (nonmembers)	*Ballroom Foyer*
8:00 a.m. – 10:00 a.m.	Scientific Session 3	*Grand Ballroom*
10:00 a.m. – 10:30 a.m.	Refreshment break	*Ballroom Foyer*
10:30 a.m. – Noon	Scientific Session 4	*Grand Ballroom*
Noon – 12:30 p.m.	Refreshment break	*Ballroom Foyer*
12:30 p.m. – 3:00 p.m.	Telesurgery demonstration with lunch	*Grand Ballroom*
2:00 p.m. – 5:00 p.m.	Exhibits dismantle	*Ballroom Foyer*
3:00 p.m. – 5:00 p.m.	Posters dismantle	*Grand Ballroom A and B*
3:00 p.m. – 4:30 p.m.	COG Surgeons' Meeting (open to all APSA meeting attendees)	*Sabal Palm Room*
6:30 p.m. – 10:30 p.m.	President's Banquet	*Grand Ballroom*

Wednesday, May 24

7:30 a.m. – 8:00 a.m.	Continental breakfast	*Ballroom Foyer*
7:30 a.m. – 11:30 a.m.	Registration open	*Ballroom Foyer*
8:00 a.m. – 8:30 a.m.	APSA Foundation Scholars: Elizabeth Beierle, M.D. and Kerilyn Nobuhara, M.D.	*Grand Ballroom*
8:30 a.m. – 9:30 a.m.	*Journal of Pediatric Surgery*/ International Guest Lecture: Pedro J. Roselló, M.D.	*Grand Ballroom*
9:30 a.m. – 11:30 a.m.	Scientific Session 5	*Grand Ballroom*
11:30 a.m.	Annual Meeting Adjourns	

General Information

Registration

Please note that all authors presenting a paper at the APSA 37th Annual Meeting are required to pay a registration fee.

The onsite registration fees for the annual meeting are:

APSA Member	$640
Physician Non-Member	$740
Student/Resident/Fellow*	$365
Nurse/Allied**	$365
Accompanying Person	$340

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

*** Registration for the APSA 37th Annual Meeting only; APSNA registration is by separate subscription.*

APSA Registration Desk

Registration will be located at the Ballroom Foyer during the following times:

Saturday, May 20	3:00 p.m. – 6:00 p.m.
Sunday, May 21	7:00 a.m. – 5:30 p.m.
Monday, May 22	6:30 a.m. – 1:00 p.m.
Tuesday, May 23	6:30 a.m. – 1:00 p.m.
Wednesday, May 24	7:30 a.m. – 11:30 a.m.

Scientific Sessions

All educational sessions will be held in the Grand Ballroom. The daily dress code is business or business casual attire.

Poster Viewing

Scientific posters will be located in the Grand Ballroom A and B and available for viewing during the following hours:

Sunday, May 21	3:30 p.m. – 5:30 p.m.
Monday, May 22	6:30 a.m. – Noon
Tuesday, May 23	7:00 a.m. – Noon

Authors are requested to be in attendance during continental breakfasts, the reception on Sunday evening and morning breaks to answer audience questions.

Speaker-Ready Room

The speaker-ready room will be available daily in the Grand Ballroom C. Computers will be provided for speakers to review their presentations.

The hours that the Speaker Ready Room will be open are:

Saturday, May 20	4:00 p.m. – 6:00 p.m.
Sunday, May 21	7:00 a.m. – 5:00 p.m.
Monday, May 22	6:30 a.m. – 1:00 p.m.
Tuesday, May 23	6:30 a.m. – 1:00 p.m.
Wednesday, May 24	7:00 a.m. – 10:30 a.m.

Presentation Check-In

Speakers must use Microsoft PowerPoint® slides during their presentations; 35mm slides will not be accepted. Refer to the Guide for Speakers distributed in January and available on the APSA Web site (www.eapsa.org) for information about preparing your presentation. Speakers must submit their computer presentations to the technician in the Speaker Ready Room (Grand Ballroom C) by 1 p.m. the day before they are scheduled to speak. Those speaking on Sunday may submit their materials between 4 and 6 p.m. on Saturday.

Exhibits

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

Monday, May 22	6:30 a.m. – Noon
Tuesday, May 23	7:00 a.m. – Noon

Continental breakfast and scheduled coffee breaks will be served in the exhibit area on Monday and Tuesday. For a list of exhibitors and booth assignments, see pages A168–A175.

APSA Business Meeting

The APSA Business Meeting will be held from 6:30 – 8:00 a.m. on Tuesday, May 23, in the Grand Ballroom. This is a breakfast meeting and is for APSA members only.

Welcome Reception

A Welcome Reception for all registrants will take place on the Basshead Deck from 6:30 – 8:30 p.m. on Sunday, May 21. 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 require a ticket to be admitted to the Welcome Reception. Casual attire is appropriate.

President's Banquet

The President's Banquet will be held in the Grand Ballroom on Tuesday, May 23. The reception will begin at 6:30 p.m. in the Grand Ballroom Foyer, and dinner will begin at 7:15 p.m. After dinner, you are invited to join us 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 require 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 Marriott Beach & Golf Resort's concierge at 843/686-8400. Additionally, a special program, the Children's Adventure Club, has been scheduled during the Tuesday evening President's Banquet, from 6 – 11 p.m. Participating children will have dinner and join in organized activities. The Children's Adventure Club is $45 U.S., per child, and the deadline to sign up is May 15. Contact the Marriott Recreation Department at 843/686-8424 for more information.

Companions' Hospitality Suite

The hospitality suite, Suite 276, will be open Monday from 9 – 11 a.m., Tuesday from 8 – 10:30 a.m. and Wednesday from 8 – 10:30 a.m. Continental breakfast will be served each morning for registered accompanying guests. Badges are requested for entry to the hospitality suite.

Dining Shuttle

APSA is pleased to offer a dining shuttle on Monday, May 22, between the Marriott Beach & Golf Resort and Harbour Town. Harbour Town stands proudly with its landmark lighthouse overlooking the Calibogue Sound. The quaint Mediterranean-style village surrounds Hilton Head's picturesque yacht-filled harbor. Along with many diverse dining establishments, Harbour Town boasts more than 20 independently owned and operated boutique clothing shops, gift and craft galleries, nature stores, jewelry stores and independent art galleries.

Dining shuttles between the Marriott Beach & Golf Resort and Harbour Town will depart the hotel beginning at 6 p.m., with the last return shuttle at 10:30 p.m. The cost is $25 U.S. per person.

Kayaking Tour

Let APSA introduce you to the fun-filled and exciting world of sea kayaking. This one-of-a-kind afternoon of adventure includes a guided tour through the calm waters of Broad Creek and its salt marshes. After preliminary instructions, paddlers will have an opportunity to closely observe dolphin, otter and other marine life and learn about the marsh's complex ecological system.

The APSA Kayaking Tour is scheduled for the afternoon of Monday, May 22. It promises to be an unforgettable experience for all ages. The cost is $73 U.S., per person, and includes all necessary equipment, plus bottled water. The tour will depart the hotel at 3:30 p.m. and return at approximately 7 p.m.

Benji Brooks Meeting and Luncheon

Join us for a luncheon meeting of the Benji Brooks Society. We will discuss issues that women are currently facing in the pediatric surgery arena and start planning for the society's future.

Optional Athletic Activities

The Annual 5K Fun Run will be on Monday, May 22, at 6 a.m. Sign-in will begin at 5:15 a.m. at the pool's beach access entrance with an organized warm up and stretch at 5:40 a.m. The run will be on the beach, so bring appropriate running shoes. The participation fee is $42 U.S., per runner, and will include a Fun Run T-shirt, water stations along the route, a light breakfast after the run and awards in a number of categories.

The 2006 APSA Golf Tournament will be a shotgun start at the Arthur Hills course at 2 p.m. on Monday, May 22. The tournament fee is $110 U.S., per golfer, and includes cart, greens fees, a boxed lunch and awards for the top players. Transportation to the course departs the hotel at 1:30 p.m.

The APSA Annual Tennis Tournament will be round-robin and begins at 2 p.m., Monday, May 22, at the Palmetto Dunes Tennis Center. The tournament fee is $45 U.S., per player, and includes light refreshments and awards for the top players.

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 the hotel operator or 843/686-8400 and request the APSA Registration Desk.

Guidelines for Authors and Discussants

1. Authors presenting papers are reminded that the presentations shall be limited to nine minutes, six minutes and three minutes (as indicated) for case presentations.

2. Computer disks and CD-ROMs must be turned in to the technician in the Speaker-Ready Room by 1 p.m. the day before they are to be presented. Those speaking on Sunday may submit materials between 4 and 6 p.m. on Saturday.

3. Posters: There will be two poster sessions, both of which will be presented in "walk rounds" format. Scientific posters should be set up Sunday afternoon from 12:30 – 2:30 p.m. and presenters must be available with their posters on Sunday between 4 and 5:30 p.m. to participate in the poster sessions. In addition, authors are asked to be in attendance during the morning refreshment breaks to discuss their presentations. All poster displays must be dismantled on Tuesday afternoon.

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 discussion 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.*

American Pediatric Surgical Foundation

The American Pediatric Surgical Association Foundation would like to thank the following APSA members who have contributed to the Foundation. The list is up-to-date as of February 9, 2006.

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Morgan, William W.
Moriarty, Kevin P.
Moulton, Steven L.
Muenchow, Sharon K.
Nikaidoh, Hisashi
Olsen, Margaret M.
Ortiz-Justiniano, Victor N.
Pendse, Prabhakar J.
Priebe, Cedric. J
Ranne, Richard D.
Ratner, Irving A.
Reddy, P. Prithvi
Reynolds, Marleta
Rosser, Samuel B.
Shafer, Alan D.
Shamberger, Robert C.
Shaw, Anthony
Slim, Michael S.
Sonnino, Roberta E.
Stolar, Charles J.
Teich, Steven
Teitelbaum, Daniel H.
Torres, Ascension M.
Tracy, Thomas F.
Valda, Victor
Velcek, Francisca T.
Waldhausen, John H. T.
Warner, Brad W
Webb, H. Warner
Weiss, Richard G.
Weissberg, Alan
Woolley, Morton M.
Wulkan, Mark
Yedlin, Steven
Zitsman, Jeffrey L.

Past APSA Annual Meeting Dates and Locations

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

Future Meetings

38th Annual Meeting
May 24–27, 2007
JW Marriott Orlando Grande Lakes
Orlando, Florida

39th Annual Meeting
May 29–June 1, 2008
JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona

Invited Speakers

Past Annual Meeting
Robert E. Gross Lecturers (1990 – 2005)

2005
W. Hardy Hendren, M.D.
"Looking Back 50 Years"

2004
Giulio (Dan) D'Angio
"The Role of the Surgeon in the Past, Present and Future of Pediatric Oncology"

2003
Lucien Leape, M.D.
"Safe Health Care — Are We Up to It?"

2002
Harold Shapiro, Ph.D.
"The Ethical Dimensions of Scientific Progress"

2001
Judah Folkman, M.D.
"Angiogenesis-Dependent Diseases"

2000
J. Bruce Beckwith, M.D.
"Pediatric Renal Tumors at the New Millennium: Myths, Misunderstandings, Controversies and Opportunities"

1999
Samuel A. Wells, Jr., M.D.
(Title not available)

1998
Richard M. Satava, M.D.
"Medicine in the 21st Century"

1997
Douglas W. Wilmore, M.D.
"Will Organ Growth Replace Transplantation? Lessons from Patients with Short Bowel Syndrome"

1996
Robert H. Bartlett, M.D.
"Surgery, Science and Respiratory Failure"

1995
David A. Williams, M.D.
"The Role of Interleukin-II on the Pathophysiology of the Small Intestine"

1994
W. French Anderson, Ph.D.
"Human Gene Therapy"

1993
Judah Folkman, M.D.
"Clinical Applications of Angiogenesis Research"

1992
Warren Zapol, M.D.
"Inhaled Nitric Oxide: A Selective Vaso-Dilator"

1991
Joel Cooper, M.D.
"History and Current Status of Lung Transplantation"

1990
Richard Simmons, M.D.
"Role of the Gut Flora in Surgery"

Past Annual Meeting
Overseas/International Guest Lecturers (1990 – 2005)

2005
Prof. Frans W.J. Hazebroek, M.D., Ph.D.
"Is Continuation of Life Support Always the Best Option for the Surgical Neonate?"

2004
David A. Lloyd, M.D., FRCS
"Tomorrow's Surgeons: Who Cares for the Patient?"

2003
Claire Nihoul-Fékété, M.D.
"Modern Surgical Management of Congenital Hyperinsulinemic Hypoglycemia"

Invited Speakers (Continued)

2002
Takeshi Miyano, M.D.
"Biliary Tree: A Gardener's 30-Year Experience"

2001
Pedro Rosselló, M.D.
"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, M.D.
"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 Hirschsprug's Disease"

1995
Sir Lewis Spitz, M.D., Ph.D., FRCS
"Esophageal Atresia — Past, Present and Future"

1994
Sean J. Corkery, M.D.h, FRCSI, FRCSEng
"In Pursuit of the Testis"

1993
Edward M. Kiely, FRCSI, FRCS
"The Surgical Challenge of Neuroblastoma"

1992
Yann Revillon, M.D.
"Intestinal Transplantation in France"

1991
Shemuel Nissan, M.D.
"The History of Surgery and Medicine in the Holy Land from the 19th Century"

1990
Jan C. Molenaar, M.D.
"Congenital Diaphragmatic Hernia — What Defect?"

Past Annual Meeting *Journal of Pediatric Surgery* Lecturers (2001 – 2005)

2005
Alberto Peña, M.D.
"Luck and Serendipity, the History of a Surgical Technique"

2004
R. Scott Jones, M.D.
"The American College of Surgeons Initiatives for Safety and Quality Improvement"

2003
Patricia Donahoe, M.D.
"Sustained Inquiry and Perseverance in the Clinic and at the Bench"

2002
Michael Harrison, M.D.
"Fetal Surgery: Trials, Tribulations and Territory"

2001
Joseph P. Vacanti, M.D.
"The History and Current Status of Tissue Engineering"

2006 Invited Speakers

Robert E. Gross Lecture: Diana Bianchi, M.D.
"Fetomaternal Cell Trafficking: A Story That Begins With Prenatal Diagnosis and May End With Stem Cell Therapy"

Dr. Diana W. Bianchi is the Natalie V. Zucker Professor of Pediatrics and of Obstetrics and Gynecology at Tufts University School of Medicine, and is Vice-Chair for Research in the Department of Pediatrics. A native of Manhattan, Dr. Bianchi graduated *magna cum laude* from the University of Pennsylvania. She received her medical degree from Stanford, and returned east to undertake residency training in pediatrics at The Children's Hospital, Boston. She then completed fellowships in the Harvard training programs in medical genetics and newborn medicine and is board-certified in all three specialties.

Dr. Bianchi has authored over 160 peer-reviewed clinical and research publications. Along with Timothy Crombleholme and Mary D'Alton she co-authored the textbook, Fetology: Diagnosis and Management of the Fetal Patient, which won the Association of American Publishers award for best textbook in clinical medicine in 2000, and has been translated into Japanese.

Dr. Bianchi is recognized locally, nationally and internationally for her leadership roles. She is a trustee of Tufts-New England Medical Center. Her international committee assignments and elected offices include Scientific Program Chair for the 2002 American Society of Human Genetics annual meeting, board member of the American Society of Human Genetics, Secretary of the International Society of Prenatal Diagnosis, and council member (Genetics) in the Society for Pediatric Research. She is a past president of the Perinatal Research Society and a member of the American Pediatric Society. She has been awarded the Distinguished Faculty Award from Tufts University and was named an honorary member of the Society for Maternal Fetal Medicine.

Journal of Pediatric Surgery/International Guest Lecture:
Pedro Rosselló, M.D.
"The Unfinished Business of American Healthcare"

Dr. Pedro Juan Rosselló was born in Puerto Rico's capital city of San Juan. Before following his father (a psychiatrist) into the practice of medicine, young Pedro would excel both as a student and as a sportsman. In tennis, he was team captain at the University of Notre Dame; won five Puerto Rico men's singles championships; and was nationally ranked by the United States Tennis Association. In 1966, he received Notre Dame's foremost scholar-athlete award and graduated *magna cum laude* with a Bachelor of Science degree. In 1970, at Yale Unicersity, he became a Doctor of Medicine cum laude.

Dr. Rosselló went on to specialize in general and pediatric surgery at Harvard University hospitals. In 1981, he received a Master's Degree in Public Health magna cum laude from the University of Puerto Rico. During his career as a pediatric surgeon and professor of medicine, Dr. Rosselló published dozens of research papers and made presentations at numerous professional conferences.

During two terms as Governor of Puerto Rico (1993–2001), Dr. Rosselló completely reformed the U.S. territory's public health system, establishing a system of universal insurance coverage. He was also elected to such prominent national posts as President of the Council of State Governments, Chair of the Democratic Governors' Association and Chair of the Southern Governors' Association. During those years, honorary LL.D. degrees were conferred upon him by the University of Notre Dame and the University of Massachusetts. Since 2005, he has held a seat in the Puerto Rico Senate.

PROGRAM IN DETAIL

Saturday, May 20

8:00 a.m. – 2:00 p.m.	APSA Board of Governors meeting	*Woodward*
2:00 p.m. – 5:00 p.m.	Committee meetings	
3:00 p.m. – 6:00 p.m.	Registration open	*Ballroom Foyer*
6:00 p.m. – 10:00 p.m.	Publications committee meeting/dinner	*Fairfield*
6:30 p.m. – 10:00 p.m.	APSA Board of Governors dinner	*Conroy's Restaurant*

Sunday, May 21

6:30 a.m. – 8:00 a.m.	Committee meetings	
7:00 a.m. – 5:30 p.m.	Registration open	*Ballroom Foyer*
8:00 a.m. – 11:30 a.m.	**Symposium: Fetal Diagnosis and Treatment — What Pediatric Surgeons Need to Know**	*Grand Ballroom*

{Moderator}
Alan W. Flake, M.D.
Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A.

{Educational Objectives:}
This symposium is designed to provide up-to-date, evidence-based information that will be useful to the pediatric surgeon who practices outside of a major Fetal Treatment Center, and wishes to participate more actively in counseling and management of the prenatal surgical patient. Specifically, the symposium will:

- Review successful models of integration of the pediatric surgeon into the multidisciplinary care of the prenatal surgical patient.
- Explain the essential components of the maternal-fetal patient interaction.
- Provide core information essential for accurate prenatal counseling for specific pediatric surgical anomalies.
- Update and review evidence-based data on the efficacy of prenatal interventions.
- Provide an overview of future directions in fetal diagnosis and treatment.

{Instructors}
N. Scott Adzick, M.D., *Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A.*

Alan W. Flake, M.D., *Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A.*

Michael R. Harrison, M.D., *University of California, San Francisco, San Francisco, CA, U.S.A.*

Russell W. Jennings, M.D., *Boston Children's Hospital, Boston, MA, U.S.A.*

Jean-Martin Laberge, M.D., *Montreal Children's Hospital, Montreal, Canada*

Hanmin Lee, M.D., *University of California, San Francisco, San Francisco, CA, U.S.A.*

Francois I. Luks, M.D., *Hasbro Children's Hospital, Providence, RI, U.S.A.*

Charles J. Stolar, M.D., *Morgan Stanley Children's Hospital of New York Presbyterian, New York, NY, U.S.A.*

Optimizing the Maternal-Fetal Patient Experience — Role of the Pediatric Surgeon
Establishing Your Role as a Pediatric Surgeon in Prenatal Diagnosis and Treatment
Francois I. Luks, M.D.

The Maternal-Fetal Patient Interaction
Alan W. Flake, M.D.

Common Pediatric Surgical Disorders —
Essential Knowledge for Prenatal Counseling
- Abdominal Wall Defects
- Congenital Diaphragmatic Hernia
- Congenital Lung Lesions
- Sacrococcygeal Teratoma
- Intestinal Anomalies
- Upper Airway Obstruction — (The EXIT Procedure)

Jean-Martin Laberge, M.D.
Hanmin Lee, M.D.

Fetal Intervention — What is Proven, What is Promising, What has Failed
- Open Fetal Surgery
  N. Scott Adzick, M.D.

- Fetoscopic intervention
  Francois I. Luks, M.D.

Controversies in Prenatal Diagnosis and Treatment: Point/Counterpoint
Session Moderator: Charles J. Stolar, M.D.
- CDH — Is Prenatal Treatment Ever Justified?
  Pro: Hanmin Lee, M.D.
  Con: Alan W. Flake, M.D.

- Prenatal Repair of Myelomeningocele —
  Is Fetal Surgery Justified for Non-Lethal Anomalies
  Pro: N. Scott Adzick, M.D.
  Con: Russell W. Jennings, M.D.

The Future of Fetal Intervention
Michael R. Harrison, M.D.

11:30 a.m. – Noon	Refreshment break	*Ballroom Foyer*
Noon – 1:30 p.m.	**Lunch with video session**	*Grand Ballroom*

{Moderators}:
John Gosche, M.D., Ph.D.
Fred Rescorla, M.D.

{Educational Objectives}
Session attendees will:
- Acquire knowledge of open and minimally invasive techniques for the treatment of pediatric surgical problems.

Underlining denotes the author scheduled to present at the meeting.

- Expand the various therapeutic options to approach common pediatric surgery problems.
- Develop an understanding of various new pediatric surgical techniques.

V1 THORACOSCOPIC LEFT LOWER LOBECTOMY FOR AN INTRALOBAR SEQUESTRATION

Casey M. Calkins, M.D.[1], Shawn D. St. Peter, M.D.[2], George W. Holcomb, III, M.D.[2]
[1]Children's Hospital of Wisconsin, Milwaukee, WI, U.S.A., [2]Children's Mercy Hospital, Kansas City, MO, U.S.A.

V2 LAPAROSCOPIC RIPSTEIN PROCEDURE FOR RECTAL PROLAPSE

Mark L. Wulkan, M.D.
Emory University School of Medicine, Atlanta, GA, U.S.A.

V3 TRANSANAL ENDORECTAL PULL-THROUGH IN THE PRONE POSITION

D. D. Potter, Penny L. Stavlo, CNP, Christopher Moir, M.D.
Mayo Clinic, Rochester, MN, U.S.A.

V4 THORACOSCOPIC REPAIR OF ESOPHAGEAL ATRESIA WITH TRACHEOESOPHAGEAL FISTULA

Denise E. Ventura, M.D., Hanmin Lee, M.D.
University of California, San Francisco, San Francisco, CA, U.S.A.

V5 LAPAROSCOPICALLY ASSISTED GASTRIC TRANSPOSITION FOR LONG-GAP ESOPHAGEAL ATRESIA

Philipp O. Szavay, M.D., Hans Joachim Kirschner, M.D., Hans-Walter Hacker, M.D., Joerg Fuchs, M.D.
University of Tuebingen, Tuebingen, Germany

V6 ADVANCED MINIMALLY INVASIVE SURGERY IN CHILDREN: ENDOSCOPIC EXCISION OF AN ORBITOFACIAL TUMOR

Farhad Sigari, M.D., MS, Adam M. Vogel, M.D., Dana L. Suskind, M.D., Donald C. Liu, M.D., Ph.D.
University of Chicago, Chicago, IL, U.S.A.

12:30 p.m. – 2:30 p.m.	Poster set-up	*Grand Ballroom A and B*
1:30 p.m. – 2:00 p.m.	Refreshment break	*Ballroom Foyer*
2:00 p.m. – 4:00 p.m.	**Symposium: Practice Issues**	*Grand Ballroom*

{Educational Objectives:}
At the end of this symposium, attendees will be able to:
- Summarize the key elements in the Maintenance of Certification for pediatric surgery.
- Explain the function of the RUC and CPT committees.
- Critically analyze the E&M coding structure and apply basic principles to enhance practice revenue.
- Evaluate the advances in technology which will impact pediatric surgical practice.

Underlining denotes the author scheduled to present at the meeting.

{Instructors}

John P. Crow, M.D., *Children's Hospital, Akron, OH, U.S.A., APSA representative on the ACS Coding and Reimbursement Committee*

Keith Georgeson, M.D., *University of Alabama Children's Hospital, Birmingham, AL, U.S.A, Chairperson, Pediatric Surgery Board of the American Board of Surgery*

Ronald B. Hirschl, M.D., *Mott Children's Hospital, Ann Arbor, MI, U.S.A., former Chair, APSA Informatics and Telemedicine Committee*

Michael H. Ratner, M.D., *University Hospital, Syracuse, NY, U.S.A., Chair, APSA Practice Committee*

Welcome and Introduction
Michael Ratner, M.D.

Maintenance of Certification: What does it mean to you?
Keith Georgeson, M.D.

Documentation

- RUC and CPT: Alphabet Soup for the Pediatric Surgeon
  John P. Crow, M.D.
- E&M Coding: Demystify and Discover
  Michael H. Ratner, M.D.
- Beyond "You've Got Mail": Using the Digital World to Enhance Your Practice
  Ronald B. Hirschl, M.D.

3:30 p.m. – 5:30 p.m.	**Posters open for viewing**	*Grand Ballroom A and B*
4:00 p.m. – 5:30 p.m.	**Poster Sessions (two concurrent sessions)**	
4:00 p.m. – 4:45 p.m.	**Poster Session IA: Gastrointestinal Surgery — Experimental and Clinical Perspectives**	*Grand Ballroom A*

{Moderators}:
Ai-Xuan Holterman, M.D.
Robert Kelly, Jr., M.D.

{Educational Objectives:}
Participants in this session will:

- Be aware of recent trends in and indications for antireflux surgery in the pediatric population.
- Gain further understanding of the mediators and mechanisms that contribute to adaptation after intestinal resection and injury.
- Define some of the cellular and morphologic alterations in and potential therapies for a variety of pediatric gastrointestinal diseases.

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P1 TRENDS IN FUNDOPLICATION FOR CHILDREN WITH GERD: ARE THERE REGIONAL AND DEMOGRAPHIC DIFFERENCES?

James T. McPhee, M.D., Maksim Zayaruzny, M.D., MPH, Pradeep P. Nazarey, M.D., Michael P. Hirsh, M.D., Paul D. Danielson, M.D.
University of Massachusetts Medical Center, Worcester, MA, U.S.A.

P2 RECENT CHANGES IN THE CHARACTERISTICS OF CHILDREN UNDERGOING ANTI-REFLUX SURGERY IN THE U.S.

Michael S. Lasser, BS[1], J.G. Liao, Ph.D.[2], Randall S. Burd, M.D., Ph.D.[1]
[1]UMDNJ-Robert Wood Johnson Medical School, New Brunswick, NJ, U.S.A., [2]UMDNJ-School of Public Health, Piscataway, NJ, U.S.A.

P3 PREDICTING THE NEED FOR FUNDOPLICATION IN PATIENTS WITH CONGENITAL DIAPHRAGMATIC HERNIA

Ivan R. Diamond, M.D., Kandice Mah, Peter C.W. Kim, M.D., Desmond Bohn, M.D., J. Ted Gerstle, M.D., Paul W. Wales, M.D.
Hospital for Sick Children, Toronto, ON, Canada

P4 ABNORMAL MUCOSAL INNERVATION OF THE GANGLIONIC BOWEL IN HIRSCHSPRUNG'S DISEASE

Thambipillai Sri Paran, FRCSI, Prem Puri, FRCS.
Children's Research Centre, Dublin, Ireland

P5 GH ADMINISTRATION ENHANCES LIVER FUNCTION AND SURVIVAL DURING BILIARY OBSTRUCTION IN MICE

Micheal Chen, M.D., Ph.D., Minhua Wang, Ph.D., GuoQiang Zheng, M.D., Ai-Xuan Holterman, M.D.
University of Illinois at Chicago, Chicago, IL, U.S.A.

P6 ILEAL PANETH CELLS EXPRESS FGF10 AFTER SMALL BOWEL RESECTION

Jennifer L. Curtis, M.D., Pierre M. Del Moral, Travis Chong, Frederic G. Sala, Lily Lee, M.D., Henri R. Ford, M.D., Saverio Bellusci, Ph.D., Kasper S. Wang, M.D.
Children's Hospital Los Angeles, Los Angeles, CA, U.S.A.

P7 THE USE OF PET SCAN TO IDENTIFY ECTOPIC PANCREATIC TISSUE RESPONSIBLE FOR CONGENITAL HYPERINSULINISM

William H. Peranteau[1], Olga Hardy[1], Abass Alavi[2], Bruce Pawel[1], Charles A. Stanley[1], N. Scott Adzick[1].
[1]The Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A., [2]The University of Pennsylvania, Philadelphia, PA, U.S.A.

P8 TIME-DEPENDENT EFFECTS OF EARLY STIMULATION WITH GLP-2 ON INTESTINAL ADAPTATION

Tatsuru Kaji, Laurie E. Wallace, Gary R. Martin, Hiroaki Tanaka, David L. Sigalet.
University of Calgary, Calgary, AB, Canada

Underlining denotes the author scheduled to present at the meeting.

P9 EPIDERMAL GROWTH FACTOR ENHANCES ENTEROCYTE MIGRATION BY DECREASING INTEGRIN EXPRESSION AND FUNCTION IN A PI3 KINASE-DEPENDENT MANNER

Cynthia L. Leaphart, M.D., Jaime A. Cavallo, BS, Selma Cetin, M.D., Jun Li, David J. Hackam, M.D., Ph.D.
Children's Hospital of Pittsburgh, Pittsburgh, PA, U.S.A.

P10 HB-EGF PROMOTES ENTEROCYTE PROLIFERATION AND MIGRATION IN NEONATAL RATS WITH NECROTIZING ENTEROCOLITIS

Jiexiong Feng, M.D., Ph.D., Gail E. Besner, M.D.
Children's Research Institute, Columbus, OH, U.S.A.

P11 THE SIGNIFICANCE OF FASTING AND POST-PRANDIAL GLP-2 LEVELS IN CORRELATION WITH INTESTINAL PERMEABILITY IN CROHN'S DISEASE — PRELIMINARY REPORT IN PEDIATRIC PATIENTS

Dragan Kravarusic, M.D.[1], David L. Sigalet, Professor[1], Jens J. Holst, Professor[2].
[1]Alberta Children's Hospital, Calgary, AB, Canada, [2]Panum Institute, University of Copenhagen, Copenhagen, Denmark

P12 RNA INTERFERENCE FOR TREATMENT OF ACUTE INFLAMMATORY COLITIS

Matthew S. Clifton, M.D., Julia Hoy, BS, Prema S. Idumalla, MS, Nigel W. Bunnett, Ph.D., Aditi Bhargava, Ph.D.
University of California, San Francisco, San Francisco, CA, U.S.A.

4:45 p.m. – 5:30 p.m. **Poster Session 2A:** *Grand Ballroom A*
New or Emerging Technologies and Therapies

{Moderators}:
John Gosche, M.D., Ph.D.
Daniel Teitelbaum, M.D.

{Educational Objectives:}
Participants in this session will:
- Be updated on recent advances in tissue engineering and their potential clinical applications.
- Be aware of how advanced diagnostic techniques and computer simulations are applicable to patient care and resident education.
- Become familiar with unique treatment options for difficult clinical problems.

P13 A COMPARISON OF CARDIAC MRI AND COMPUTED TOMOGRAPHY WITH ECHOCARDIOGRAM IN EVALUATION OF PATIENTS WITH PECTUS EXCAVATUM

N. Elizabeth Terry, M.D., William C. Boswell, M.D., Patrick McGraw, M.D., Robert Rollings, M.D.
Memorial Health University Medical Center, Savannah, GA, U.S.A.

Underlining denotes the author scheduled to present at the meeting.

P14 CONSISTENT AND DISTINCT BIOCHEMICAL SIGNATURES FOR NORMAL AND CANCEROUS TISSUE WITH NIR RAMAN SPECTROSCOPY

Gulay K. Serhatkulu[1], Houbei Dai[1], Rachel Weber[1], Alex Cao[1], Abhilash Pandya[1], Jagdish Thakur[1], Carl Freeman[1], Ratna Naik[1], Vaman Naik[2], Gregory Auner[1], Raja Rabah[3], Janet Poulik[3], Fazlul Sarkar[4], Michael Klein, M.D.[3]

[1]Wayne State University, Detroit, MI, U.S.A., [2]University of Michigan-Dearborn, Detroit, MI, U.S.A., [3]Children's Hospital of Michigan, Detroit, MI, U.S.A., [4]Karmanos Cancer Research Institute, Detroit, MI, U.S.A.

P15 SIMULATION MODELING OF NORMAL AND ABNORMAL DEVELOPMENT OF THE MAMMALIAN DIAPHRAGM

Jason C. Fisher, M.D., Lawrence Bodenstein, M.D.

Morgan Stanley Children's Hospital of New York Presbyterian, Columbia University Medical Center, Department of Pediatric Surgery, New York, NY, U.S.A.

P16 DEVELOPMENT OF A STANDARDIZED INTERACTIVE MULTIMEDIA-BASED PEDIATRIC SURGICAL REVIEW COURSE

Charles L. Snyder, M.D., Daniel J. Ostlie, M.D., George W. Holcomb, III, M.D.

Children's Mercy Hospital, Kansas City, MO, U.S.A.

P17 TISSUE ENGINEERED GAS EXCHANGE DEVICE USING MICROELECTRO MECHANICAL SYSTEMS TECHNOLOGY

Jennifer Anderson, M.D.[1], Irina Pomerantseva, M.D., Ph.D.[1], Katherine Kulig, B.A.[1], Jeffery T. Borenstein, Ph.D.[2], Eli Weinberg, MS[2], Mohammad R. Kaazempur-Mofrad, Ph.D.[3], Joseph P. Vacanti, M.D.[1]

[1]Massachusetts General Hospital, Boston, MA, U.S.A., [2]Draper Laboratory, Cambridge, MA, U.S.A., [3]University of California, Berkley, Berkley, CA, U.S.A.

P18 TISSUE ENGINEERING APPROACHES TOWARDS POTENTIAL LUNG AUGMENTATION IN PULMONARY HYPOPLASIA

Christine Finck[1], Mark Mondrinos[2], Honesto Poblete[1], Sirma Koutzaki[1], Peter Lelkes, Ph.D.[2]

[1]St. Christopher's Hospital for Children, Philadelphia, PA, U.S.A., [2]Drexel University Department of Bioengineering, Philadelphia, PA, U.S.A.

P19 ISOLATION AND SEEDING OF AUTOLOGOUS OVINE BONE MARROW DERIVED VASCULAR PROGENITOR CELLS ON A BIODEGRADABLE SCAFFOLD FOR USE AS A TISSUE ENGINEERED VASCULAR CONDUIT

Jason D. Roh, BA, Matthew P. Brennan, M.D., Peter M. Fong, Ph.D., Reynold Lopez-Soler, M.D., Ph.D., George Tellides, M.D., Ph.D., Alan Dardik, M.D., Ph.D., Christopher K. Breuer, M.D.

Yale University School of Medicine, New Haven, CT, U.S.A.

Underlining denotes the author scheduled to present at the meeting.

P20 A MULTILAYER TISSUE ENGINEERED LIVER DEVICE WITH AN INTRINSIC MICROFABRICATED VASCULAR NETWORK

Katayun Irani, M.D.[1], Katherine Kulig, BA[1], Eleanor Pritchard[2], Kimberly Bonner, BA[1], Brian Orrick, MBA[2], Kimberly Morgan[1], Mohammed Kaazempur-Mofrad, Ph.D.[3], Eli Weinberg, MS[3], Jeffrey Borenstein, Ph.D.[2], Joseph P. Vacanti, M.D.[4]

[1]Massachusetts General Hospital For Children, Boston, MA, U.S.A., [2]Charles Stark Draper Laboratories, Cambridge, MA, U.S.A., [3]Massachusetts Institute of Technology, Cambridge, MA, U.S.A., [4]Massachusetts General Hospital for Children, Boston, MA, U.S.A.

P21 MECHANICALLY-INDUCED ENTEROGENESIS: IDENTIFCATION OF MECHANISMS OF ACTION

Mohamed I. El-sawaf, M.D., Hua Yang, Ph.D., M.D., Ariel U. Spencer, M.D., Daniel H. Teitelbaum, M.D.

University of Michigan, Ann Arbor, MI, U.S.A.

P22 MESENCHYMAL STEM CELLS FROM TWO DISTINCT COMPARTMENTS HAVE ENHANCED OSTEOGENESIS IN RESPONSE TO A P53 TUMOR SUPPRESSOR MUTATION

Monika Tataria, M.D., Natalina Quarto, Ph.D., Karl G. Sylvester, M.D.

Stanford University, Stanford, CA, U.S.A.

P23 GASTRIC ELECTRICAL STIMULATION FOR ADOLESCENTS WITH INTRACTABLE NAUSEA AND GASTROPARESIS

Saleem Islam, M.D., John R. Gosche, M.D., Ph.D., Jo White-Ashmead, M.D., Laura R. Vick, M.D., Thomas R. Abel, M.D.

University of Mississippi Medical Center, Jackson, MS, U.S.A.

4:00 p.m. – 4:45 p.m **Poster Session 1B:** *Grand Ballroom B*
Cancer/Oncology — Basic Science/Clinical Outcomes

{Moderators}:
Stephen Shochat, M.D.
Daniel von Allmen, M.D.

{Educational Objectives:}
Participants in this session will:

- Be able to define how patient characteristics and treatment options impact outcome for pediatric cancer patients.
- Demonstrate a knowledge of unique therapeutic strategies for treating childhood malignancies.
- Gain further understanding of molecular and genetic signals that are associated with pediatric malignancies.

Underlining denotes the author scheduled to present at the meeting.

P24 OVARIAN SEROUS CYSTADENOCARCINOMA SIDE POPULATION EXHIBITS STEM CELL CHARACTERISTICS AND IS INHIBITED BY MULLERIAN INHIBITING SUBSTANCE *IN VITRO*

Paul Szotek, M.D.[1], Rafael Pieretti-Vanmarcke, M.D.[1], Yong Zhang, M.D.[1], Denise Connolly, Ph.D.[2], David Dombkowski[3], Frederic Preffer, M.D.[3], David T. MacLaughlin, Ph.D.[1], Peter T. Masiakos, M.D.[1], Patricia K. Donahoe, M.D.[1]

[1]Pediatric Surgical Research Laboratories, MassGeneral Hospital for Children, Harvard Medical School, Boston, MA, U.S.A., [2]Fox Chase Cancer Center, Philadelphia, PA, U.S.A., [3]Flow Cytometry Laboratory, Department of Pathlology, Massachusetts General Hospital, Boston, MA, U.S.A.

P25 WNT/β-CATENIN SIGNALING IN HEPATOBLASTOMA MICE MODEL

Masashi Kurobe, M.D.[1], Shelly Beer[2], Deen W. Felsher, M.D.[2], Karl G. Sylvester, M.D.[1]

[1]Stanford University, Department of Surgery, Stanford, CA, U.S.A., [2]Stanford University, Department of Medicine and Pathology, Stanford, CA, U.S.A.

P26 SUCCESSFUL LIVER TRANSPLANT FOR UNRESECTABLE HEPATOBLASTOMA, EARLY REFERRAL IS THE KEY

Adela T. Casas-Melley, M.D., Jeffrey Malatack, M.D., Deborah Consolini, M.D., Keith J. Mann, M.D., Christopher Rabb, M.D., Louise Flynn, APN, Pamela Woolfrey, MSN, Jerome Menendez, RN, Stephen Dunn, M.D.

AI duPont Hospital for Children, Wilmington, DE, U.S.A.

P27 WILMS' TUMOR PATHOGENESIS IS FACILITATED BY SERINE PHOSPHORYLATION OF STAT1

Olga Timofeeva, Ph.D.[1], Harold N. Lovvorn, III, M.D.[2], Shaun Opperman[2], Alan O. Perantoni, Ph.D.[1]

[1]National Cancer Institute, Frederick, MD, U.S.A., [2]Vanderbilt University School of Medicine, Children's Hospital, Nashville, TN, U.S.A.

P28 WILMS' TUMOR AND ALTERED EXPRESSION OF THE TRANSCRIPTIONAL REGULATOR, CITED1

Harold N. Lovvorn, III, M.D.[1], Scott Boyle[2], Shaun Opperman[1], Genbin Shi, Ph.D.[2], Marcia Wills, M.D.[3], Mark de Caestecker, Ph.D.[2]

[1]Vanderbilt University School of Medicine, Children's Hospital, Nashville, TN, U.S.A., [2]Vanderbilt University School of Medicine, Cell and Developmental Biology, Nashville, TN, U.S.A., [3]Vanderbilt University School of Medicine, Pediatric Pathology, Nashville, TN, U.S.A.

P29 PEDIATRIC FAMILIAL MEDULLARY THYROID CARCINOMA (FMTC) STAGE IS INDEPENDANT OF MUTATION SITE: BUT MUTATION AT C620 IS ASSOCIATED WITH HIRSCHSPRUNG'S DISEASE

Andreana Bütter, Julie Gagné, Ayman Al-Jazaeri, Mohammad Ali Emran, Cheri Deal, Dickens St-Vil.

Ste-Justine Hospital, Montreal, PQ, Canada

Underlining denotes the author scheduled to present at the meeting.

P30 THE IMPACT OF EXTENT OF THYROIDECTOMY ON NON-METASTATIC, ENCAPSULATED, AND COMPLETELY RESECTED DIFFERENTIATED THYROID CARCINOMA IN PATIENTS <21 YEARS OF AGE AT DIAGNOSIS

Daniel Rutigliano, M.D.[1], Charles Sklar, M.D.[1], Kurt Newman, M.D.[2], George Holcomb III, M.D.[3], Gerald Haase, M.D.[4], Michael P. La Quaglia, M.D.[1]

[1]Memorial Sloan-Kettering Cancer Center, New York, NY, U.S.A., [2]Children's National Medical Center, Washington, DC, U.S.A., [3]Children's Mercy Hospital, Kansas City, MO, U.S.A., [4]Denver Pediatric Surgeons, Denver, CO, U.S.A.

P31 ELUCIDATION OF THE PATHWAY OF NERVE GROWTH FACTOR INDUCED APOPTOSIS IN THE NEUROBLASTOMA CELL LINE SK-N-SH

Mary Beth Madonna, M.D., Yi Yong Qiu, M.D., Bill Chiu, M.D., Marleta Reynolds, M.D., Bernard Mirkin, Ph.D., M.D.

Children's Memorial Hospital, Northwestern University Feinberg School of Medicine, Chicago, IL, U.S.A.

P32 *SALMONELLA TYPHIMURIUM* IMPROVES SURVIVAL IN NEUROBLASTOMA BY NK CELL PROLIFERATION AND THE INCREASE IN CYTOKINES IL-6 AND INF-γ

Sean J. Barnett, MS, M.D., Brent S. Sorenson, BS, Brent W. Nelson, BS, Arnold S. Leonard, M.D., Ph.D., Daniel A. Saltzman, M.D., Ph.D.

University of Minnesota, Minneapolis, MN, U.S.A.

P33 NOVEL ACTION OF EPIDERMAL GROWTH FACTOR ON CASPASE-3 AND ITS POTENTIAL AS A CHEMOTHERAPEUTIC ADJUNCT FOR NEUROBLASTOMA

Bill Chiu, M.D., Bernard Mirkin, Ph.D., M.D., Marleta Reynolds, M.D., Mary Beth Madonna, M.D.

Children's Memorial Hospital, Northwestern University Feinberg School of Medicine, Chicago, IL, U.S.A.

P34 PHEOCHROMOCYTOMA AND PARAGANGLIOMA IN CHILDREN: OUTCOMES OF SURGICAL RESECTION

Tuan H. Pham, M.D., Ph.D., Christopher Moir, M.D., Geoffrey Thompson, M.D., Abdalla Zarroug, M.D., Chad Hamner, M.D., David Farley, M.D., Jon van Heerden, M.D., Aida Lteif, M.D., William F. Young, M.D.

Mayo Clinic Rochester, Rochester, MN, U.S.A.

P35 DOES AGE INFLUENCE OUTCOME AMONG PATIENTS WITH OSTEOSARCOMA?

Matthew T. Harting, M.D.[1], Martin L. Blakely, M.D.[2], Andrea Hayes-Jordan, M.D.[1], Norman A. Jaffe, M.D.[3], Richard J. Andrassy, M.D.[1], Kevin P. Lally, M.D.[1]

[1]University of Texas Medical School at Houston, Houston, TX, U.S.A., [2]University of Tennessee, Memphis, TN, U.S.A., [3]University of Texas MD Anderson Cancer Center, Houston, TX, U.S.A.

4:45 p.m. – 5:30 p.m. **Poster Session 2B: Trauma/Critical Care —** *Grand Ballroom B*
Congenital Diaphragmatic Hernia

{Moderators}:
Mary Brandt, M.D.
Alan Flake, M.D.

Underlining denotes the author scheduled to present at the meeting.

{Educational Objectives:}
Participants in this session will:
- Be able to report on how regional variations and patient characteristics impact trauma care in the pediatric age group.
- Be aware of altered gene expression associated with cystic adenomatoid malformation and congenital diaphragmatic hernia.
- Be able to cite new therapies that are in use and in development for burn wounds, reoxygenation injury and pulmonary failure.

P36 AGE-RELATED INJURY PREVENTION PRIORITIES IN THE UNITED STATES
Michael L. Nance, M.D.[1], Douglas J. Wiebe, Ph.D.[2]
[1]Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A., [2]Center for Clinical Epidemiology and Biostatistics, University of Pennsylvania, Philadelphia, PA, U.S.A.

P37 VARIATION IN THE MANAGEMENT OF PEDIATRIC BLUNT SPLENIC INJURY BASED ON TRAUMA CENTER STATUS
Sohail R. Shah, M.D., MHA[1], R. Scott Watson, M.D., MPH[2], Mary E. Hartman, M.D.[1], Walter T. Linde-Zwirble[3], Derek C. Angus, M.D., MPH[2], Jeffrey S. Upperman, M.D.[4]
[1]Children's Hospital of Pittsburgh, Pittsburgh, PA, U.S.A., [2]University of Pittsburgh, Pittsburgh, PA, U.S.A., [3]ZD Associates, LLC, Doylestown, PA, U.S.A., [4]Children's Hospital of Los Angeles, Los Angeles, CA, U.S.A.

P38 PEDIATRIC TRAUMA IN OPERATION IRAQI FREEDOM: 31ST COMBAT SUPPORT HOSPITAL, IRAQ
Rebecca McGuigan, M.D.[1], Philip Spinella, M.D.[2], Alec Beekley, M.D.[3], James Sebesta, M.D.[3], Jeremy Perkins, M.D.[4], Kurt Grathwohl, M.D.[2], Kenneth Azarow, M.D.[3]
[1]Martin Army Community Hospital, Fort Benning, GA, U.S.A., [2]Brooke Army Medical Center, Fort Sam, Houston, TX, U.S.A., [3]Madigan Army Medical Center, Fort Lewis, WA, U.S.A., [4]Walter Reed Army Medical Center, Washington, DC, U.S.A.

P39 A SILVER IMPREGNATED ANTIMICROBIAL DRESSING REDUCES HOSPITAL COST IN PEDIATRIC BURN PATIENTS
Heather Paddock, M.D., Renata Fabia, M.D., Shelia Giles, RN, John Hayes, Ph.D., Dawn Adams, MS, Gail Besner, M.D.
Columbus Children's Hospital, Columbus, OH, U.S.A.

P40 HB-EGF DECREASES NEUTROPHIL-ENDOTHELIAL CELL ADHESION AFTER ANOXIA/REOXYGENATION INJURY
Dorothy V. Rocourt, M.D., Veela Mehta, Ph.D., Gail E. Besner, M.D.
Children's Research Institute, Columbus, OH, U.S.A.

P41 COMPLICATIONS OF IMPLANTED CENTRAL VENOUS CATHETERS IN NEUTROPENIC CHILDREN
Arvand Elihu, M.D., Gerald Gollin, M.D., Kimberly Arledge, M.D.
Loma Linda University School of Medicine, Loma Linda, CA, U.S.A.

Underlining denotes the author scheduled to present at the meeting.

P42 GENE EXPRESSION IN CONGENITAL CYSTIC ADENOMATOID MALFORMATION: MOLECULAR EVIDENCE OF ARRESTED DEVELOPMENT

Amy J. Wagner, M.D., Amber N. Stambaugh, MS, Jess Edmondson, Matthew S. Clifton, M.D., Erich J. Grethel, M.D., Raul A. Cortes, M.D., Michael R. Harrison, M.D., Hanmin Lee, M.D., Kerilyn Nobuhara, M.D., Diana Farmer, M.D., Sam Hawgood, M.D.
University of California San Francisco, San Francisco, CA, U.S.A.

P43 MOLECULAR GENETIC PATHOPHYSIOLOGY IN HUMAN CONGENITAL DIAPHRAGMATIC HERNIA

Sibel Kantarci, Ph.D.[1], Barbara Pober, M.D.[2], Thomas B. Kinane, M.D.[3], Lihadh Al-Gazali, M.D.[4], David Casavant, M.D.[1], Dick Tibboel, M.D., Ph.D[5], Meaghan Russell, M.P.H.[1], Jay M. Wilson, M.D.[6], Charles Lee, Ph.D.[7], Patricia K. Donahoe, M.D.[1]
[1]Pediatric Surgical Research Laboratories, MassGeneral Hospital for Children, Harvard Medical School, Boston, MA, U.S.A., [2]Genetics and Teratology, MassGeneral Hospital for Children, Harvard Medical School, Boston, MA, U.S.A., [3]Massachusetts General Hospital, Harvard Medical School, Boston, MA, U.S.A., [4]United Arab Emirates University, United Arab Emirates, [5]Erasmus Medical Centre, Rotterdam, The Netherlands, [6]Children's Hospital Boston, Boston, MA, U.S.A., [7]Department of Pathology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, U.S.A.

P44 PERCENT PREDICTED LUNG VOLUMES AS MEASURED ON FETAL MAGNETIC RESONANCE IMAGING: A USEFUL BIOMETRIC PARAMETER FOR RISK STRATIFICATION IN CONGENITAL DIAPHRAGMATIC HERNIA

Carol E. Barnewolt, M.D., Shaun M. Kunisaki, M.D., Dario O. Fauza, M.D., Luanne P. Nemes, RN, MS, PNP, Judy A. Estroff, M.D., Russell W. Jennings, M.D.
Children's Hospital Boston, Boston, MA, U.S.A.

P45 PUMPLESS ARTERIOVENOUS EXTRACORPOREAL LIFE SUPPORT (A-V ECLS) IN A LUNG-INJURY MODEL: A NEW APPROACH TO ECMO?

J. Kristine Brown, M.D., Rupa Seetharamaiah, M.D., George B. Mychaliska, M.D., Robert H. Bartlett, M.D., Ronald B. Hirschl, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

5:30 p.m. – 6:30 p.m.	Exhibit set-up	*Ballroom Foyer*
6:30 p.m. – 8:30 p.m.	Welcome Reception	*Basshead Deck*
9:00 p.m. – 10:00 p.m.	New Member Reception	*Sabal Palm Room*

Monday, May 22

6:00 a.m. – 7:30 a.m.	Annual Fun Run	*Pool—Beach Access Entrance*
6:30 a.m. – 7:30 a.m.	Committee meetings	
6:30 a.m. – 1:00 p.m.	Registration open	*Ballroom Foyer*
6:30 a.m. – Noon	Posters and Exhibits open for viewing	*Ballroom Foyer/ Grand Ballroom A and B*
6:45 a.m. – 7:30 a.m.	Continental breakfast in the exhibit area	*Ballroom Foyer*

Underlining denotes the author scheduled to present at the meeting.

7:30 a.m. – 9:15 a.m. **Welcome/Scientific Session 1: Gastrointestinal Surgery** *Grand Ballroom*

{Moderators}:
Alan Flake, M.D.
Robert Kelly, Jr., M.D.

{Educational Objectives:}
Session attendees will:
- Be able to appraise alternative approaches to the management of common GI processes including appendicitis and gall bladder disease.
- Recognize various complications associated with the post-operative course of patients with Hirschsprung's disease and ulcerative colitis.
- Appreciate the complications associated with various common GI processes including splenic cysts, perianal abscesses and fecal incontinence.

1 MATCHED ANALYSIS OF NON-OPERATIVE MANAGEMENT AND IMMEDIATE APPENDECTOMY FOR PERFORATED APPENDICITIS (3 MINUTE)
Marion C.W. Henry, M.D., MPH[1], Gerald Gollin, M.D.[2], Saleem Islam, M.D.[3], Karl Sylvester, M.D.[4], Angela Walker, BS[5], Bonnie L. Silverman, Ph.D.[1], R. Lawrence Moss, M.D.[1].
[1]Yale University, New Haven, CT, U.S.A., [2]Loma Linda University, Loma Linda, CA, U.S.A., [3]University of Mississippi, Jackson, MS, U.S.A., [4]Stanford University, Stanford, CA, U.S.A., [5]University of Missouri, Columbia, MO, U.S.A.

2 SHOULD INTERVAL APPENDECTOMY BE PERFORMED IN PEDIATRIC PATIENTS INITIALLY TREATED NON-OPERATIVELY FOR APPENDICITIS? (3 MINUTE)
Devin Puapong, M.D., Anna Kaminski, M.D., Philip Haigh, M.D., Harry Applebaum, M.D.
Kaiser Permanente Los Angeles Medical Center, Los Angeles, CA, U.S.A.

3 LAPAROSCOPIC CHOLECYSTECTOMY AND CHOLANGIOSCOPIC COMMON BILE DUCT EXPLORATION IN THE MANAGEMENT OF CHOLEDOCHOLITHIASIS IN CHILDREN (3 MINUTE)
J. Kristine Brown, M.D., Eiichi Miyasaka, BA, Oliver S. Soldes, M.D., James D. Geiger, M.D., Ronald B. Hirschl, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

4 ABNORMAL GALLBLADDER EJECTION FRACTION DOES NOT PREDICT SYMPTOMATIC RELIEF FROM CHOLECYSTECTOMY IN CHILDREN WITH ACALCULOUS BILIARY PAIN (3 MINUTE)
Heather Paddock, M.D.[1], Vance Smith, M.D.[2], John Hayes, Ph.D.[1], Greg Bates, M.D.[1], Brian Kenney, M.D., MPH[1], Donna A. Caniano, M.D.[1], Benedict C. Nwomeh, M.D.[1]
[1]Columbus Children's Hospital, Columbus, OH, U.S.A., [2]The Ohio State University College of Medicine, Columbus, OH, U.S.A.

Underlining denotes the author scheduled to present at the meeting.

5 UTILIZATION OF ESOPHAGO-CRURAL SUTURES AND MINIMAL ESOPHAGEAL DISSECTION REDUCES THE INCIDENCE OF POSTOPERATIVE TRANSMIGRATION OF LAPAROSCOPIC NISSEN FUNDOPLICATION WRAP (3 MINUTE)

Shawn D. St. Peter, M.D.[1], Daniel J. Ostlie, M.D.[1], Patricia A. Valusek, M.D.[1], Casey M. Calkins, M.D.[2], Steven B. Shew, M.D.[3], George W. Holcomb, III, M.D.[1]
[1]Children's Mercy Hospital, Kansas City, MO, U.S.A., [2]Children's Hospital of Wisconsin, Milwaukee, WI, U.S.A., [3]University of California Los Angeles, Los Angeles, CA, U.S.A.

6 IMPACT OF BUTTON CECOSTOMY ON MANAGEMENT OF FECAL INCONTINENCE AND CONSTIPATION — NINE YEARS OF EXPERIENCE IN PEDIATRIC POPULATION (3 MINUTE)

Dragan Kravarusic, M.D.[1], Sarah Wong[2], Lanna Bottomly, RN[1], Andrew Wong, M.D.[1]
[1]Alberta Children's Hospital, Calgary, AB, Canada, [2]University of Calgary, Calgary, AB, Canada

7 POUCH OUTCOMES AMONG CHILDREN WITH ULCERATIVE COLITIS TREATED WITH CALCINEURIN INHIBITORS PRIOR TO ILEAL POUCH ANAL ANASTOMOSIS SURGERY (3 MINUTE)

Craig W. Lillehei, M.D., Elizabeth Hait, M.D., Melissa J. Shuman, BA, Athos Bousvaros, M.D., MPH, Robert C. Shamberger, M.D.
Boston Children's Hospital, Boston, MA, U.S.A.

8 LAPAROSCOPIC UNROOFING OF SPLENIC CYSTS RESULTS IN RECURRENCES (3 MINUTE)

Felix Schier[1], Karl-Ludwig Waag[2], Benno Ure[3].
[1]University Medical Centre, Mainz, Germany, [2]University Medical Centre, Mannheim, Germany, [3]University Medical Centre, Hannover, Germany

9 EVOLUTION OF TECHNIQUE IN THE TRANSANAL SOAVE PULL-THROUGH FOR HIRSCHSPRUNG DISEASE (3 MINUTE)

Ahmed Nasr, M.D., Jacob C. Langer, M.D.
Hospital for Sick Children, Toronto, ON, Canada

10 ARE THE LONG TERM RESULTS OF THE TRANSANAL PULL-THROUGH EQUAL TO THOSE OF THE TRANSABDOMINAL PULLTHROUGH? A COMPARISON OF TWO APPROACHES FOR HIRSCHSPRUNG DISEASE (3 MINUTE)

Mohamed I. El-sawaf, M.D., Robert A. Drongowski, Jennifer N. Chamberlain, Arnold G. Coran, M.D., Daniel H. Teitelbaum, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

11 SHOULD PERITONEAL DRAIN (PD) BE USED AS DEFINITIVE SURGICAL THERAPY IN COMPLICATED NECROTIZING ENTEROCOLITIS (NEC) IN INFANTS WEIGHTING <1500 G OR <1000 G? (3 MINUTE)

Maria Rusan, Jr., Ahmed Nasr, Aideen Moore, Peter Kim, M.D.
The Hospital for Sick Children, Toronto, ON, Canada

Underlining denotes the author scheduled to present at the meeting.

12 PROGRESSION OF PERIANAL ABSCESS TO FISTULA IN INFANTS: IMPLICATIONS FOR TREATMENT (3 MINUTE)

Emily R. Christison-Lagay, M.D.[1], Jason F. Hall, M.D.[1], Karen Bailey, M.D.[2], Andrew Terluk[2], Paul W. Wales, M.D.[2], Peter T. Masiakos, M.D.[1]

[1]Massachusetts General Hospital, Boston, MA, U.S.A., [2]The Hospital for Sick Children, Toronto, ON, Canada

9:15 a.m. – 10:15 a.m.	**Robert E. Gross Lecture: Diana Bianchi, M.D.**	*Grand Ballroom*
10:15 a.m. – 10:45 a.m.	Refreshment break	*Ballroom Foyer*
10:45 a.m. – Noon	**Scientific Session 2: Cancer/Oncology and Vascular Anomalies**	*Grand Ballroom*

{Moderators}:
Stephen Shochat, M.D.
Daniel von Allmen, M.D.

{Educational Objectives:}
Session attendees will:

- Better understand the staging and surgical approach to pediatric tumors.
- Develop a knowledge base for some of the more novel experimental modalities for pediatric neoplasms.
- Cite some of the various approaches to benign neoplasms of vascular or lymphatic origin.

13 AORTA AND INFERIOR VENA CAVA RECONSTRUCTION DURING ONCOLOGIC RESECTION IN CHILDREN (3 MINUTE)

Tuan H. Pham, M.D., Ph.D., Thomas Bower, M.D., Corey Iqbal, M.D., Abdalla Zarroug, M.D., Antony Joseph, MBBS, Gustavo Oderich, M.D., Marineh Yagubyan, M.D., Audra Noel, M.D., Christopher Moir, M.D.

Mayo Clinic Rochester, Rochester, MN, U.S.A.

14 PREDICTIVE VALUE OF THE PRETEXT STAGING SYSTEM IN CHILDREN WITH HEPATOBLASTOMA (3 MINUTE)

Rebecka L. Meyers, M.D.[1], Marcio H. Malogolowkin[2], Jon M. Rowland[3], Mark D. Krailo[4].

[1]University of Utah, Salt Lake City, UT, U.S.A., [2]Children's Hospital of Los Angeles, Los Angeles, CA, U.S.A., [3]Oakland Children's Hospital, Oakland, CA, U.S.A., [4]Cure Search, Children's Oncology Group, Arcadia, CA, U.S.A.

15 INTRAVASCULAR ADMINISTRATION OF TUMOR TROPIC NEUROPROGENITOR CELLS PERMITS TARGETED DELIVERY OF INTERFERON-β AND RESTRICTS TUMOR GROWTH IN A MURINE MODEL OF DISSEMINATED NEUROBLASTOMA (6 MINUTE)

Paxton V. Dickson, M.D.[1], John B. Hamner, M.D.[1], Seung U. Kim, M.D.[2], Cathy Y.C. Ng, MS[1], Karen S. Aboody, M.D.[3], Mary K. Danks, Ph.D.[1], Andrew M. Davidoff, M.D.[1]

[1]St. Jude Children's Research Hospital, Memphis, TN, U.S.A., [2]University of British Columbia, Vancouver, BC, Canada, [3]City of Hope National Medical Center, Duarte, CA, U.S.A.

Underlining denotes the author scheduled to present at the meeting.

Monday, May 22 (Continued)

16 RESECTION OF RESIDUAL MASSES AT THE END OF THERAPY FOR RHABDOMYOSARCOMA (RMS) (3 MINUTE)

David A. Rodeberg[1], Andrea Hayes-Jordan, M.D.[2], Julie Stoner, Ph.D.[3], Charles Paidas, M.D.[4], Kenneth Brown, M.D.[5], Lor Randall, M.D.[6], Eugene Wiener, M.D.[1], Doug Hawkins[7].

[1]Childrens Hospital of Pittsburgh, Pittsburgh, PA, U.S.A., [2]MD Anderson, Houston, TX, U.S.A., [3]University of Oklahoma, Tulsa, OK, U.S.A., [4]University of South Florida, Tampa, FL, U.S.A., [5]University of British Columbia, Vancouver, BC, Canada, [6]University of Utah, Salt Lake City, VA, U.S.A., [7]University of Washington, Seattle, WA, U.S.A.

17 INDUCTION OF CYTOLYTIC T LYMPHOCYTES (CTL) AGAINST PEDIATRIC SOLID TUMORS *IN VITRO* USING AUTOLOGOUS DCS PULSED WITH NECROTIC PRIMARY TUMOR (6 MINUTE)

Joel Shilyansky, M.D., Paulette Jacobs, MA, Kara Doffek, BS, Sonia Sugg, M.D.

Medical College of Wisconsin, Milwaukee, WI, U.S.A.

18 HEPATIC HEMANGIOMAS: SUB-TYPE CLASSIFICATION AND DEVELOPMENT OF A CLINICAL PRACTICE ALGORITHM AND REGISTRY (6 MINUTE)

Emily R. Christison-Lagay, M.D., Patricia E. Burrows, M.D., Steven J. Fishman, M.D.

Children's Hospital, Boston, Boston, MA, U.S.A.

19 ETHIBLOC SCLEROTHERAPY FOR TREATMENT OF LYMPHANGIOMAS DOES NOT COMPLICATE SURGICAL RESECTION (3 MINUTE)

Mohammad Ali Emran, Salam Yazbeck, M.D., Louise Caouette Laberge, M.D., Ayman Al-Jazaeri, Chad Allan Wiesenauer, Josée Dubois.

Ste-Justine Hospital, Montreal, PQ, Canada

Noon – 12:15 p.m.	**Member Presentation: Robert K. Minkes, M.D., Ph.D.**	*Grand Ballroom*
12:15 p.m. – 1:15 p.m.	**Welcome New Members/Presidential Address: Judah Folkman, M.D.**	*Grand Ballroom*
1:15 p.m. – 2:00 p.m.	**Financial Planning for Doctors** *Supported by Greenbook Financial Services*	*Grand Ballroom*
1:30 p.m. – 3:00 p.m.	Benji Brooks Luncheon	*Idigo*
2:00 p.m. – 7:00 p.m.	Golf Tournament	*Arthur Hills Golf Course*
2:00 p.m. – 6:00 p.m.	Tennis Tournament	*Palmetto Dunes Tennis Center*
3:30 p.m. – 7:00 p.m.	Kayaking Tour	*Hotel Entrance*
6:00 p.m. – 10:30 p.m.	Optional Dining Shuttle	*Hotel Entrance*

Tuesday, May 23

6:30 a.m. – 8:00 a.m.	Member business meeting and breakfast	*Grand Ballroom*
6:30 a.m. – 1:00 p.m.	Registration open	*Ballroom Foyer*
7:00 a.m. – Noon	Posters and Exhibits open for viewing	*Ballroom Foyer/ Grand Ballroom A and B*

Underlining denotes the author scheduled to present at the meeting.

7:00 a.m. – 8:00 a.m.	Continental breakfast (nonmembers)	*Ballroom Foyer*
8:00 a.m. – 10:00 a.m.	**Scientific Session 3:** **Thoracic Surgery/Fetal and Perinatal Issues/Ethical Issues**	*Grand Ballroom*

{Moderators}:
John Gosche, M.D., Ph.D.
Fred Rescorla, M.D.

{Educational Objectives:}
Session attendees will:
- Acquire knowledge of patent ductus arteriosus outcomes.
- Gain insight and knowledge into common pediatric thoracic conditions.
- Be able to integrate an approach to treat, as well as how to approach various problems with, pectus excavatum.
- Be able to recite and describe various approaches for assessing the risk of patients with congenital diaphragmatic hernia during the antenatal period.
- Develop an appreciation for the perinatal therapeutic approach to patients with prenatally diagnosed processes.
- Define the increased risk of inheritance of certain congenital anomalies.

20 PATENT DUCTUS ARTERIOSUS (PDA) LIGATION IN PREMATURE INFANTS: WHO REALLY BENEFITS, AND AT WHAT COST? (3 MINUTE)
Mehul V. Raval, M.D.[1], Matthew M. Laughon, M.D.[2], Carl L. Bose, M.D.[2], J. D. Phillips, M.D.[1]
[1]Department of Surgery, Division of Pediatric Surgery, University of North Carolina at Chapel Hill, Chapel Hill, NC, U.S.A., [2]Department of Pediatrics, Division of Neonatal-Perinatal Medicine, University of North Carolina at Chapel Hill, Chapel Hill, NC, U.S.A.

21 VERTICAL EXPANDABLE PROSTHETIC TITANIUM RIB (VEPTR) FOR THORACIC INSUFFICIENCY SYNDROME (TIS) (6 MINUTE)
John H.T. Waldhausen, M.D., Gregory J. Redding, M.D., Kit M. Song, M.D.
Children's Hospital, Seattle, WA, U.S.A.

22 MAGNETIC MINI-MOVER PRODCURE (3MP) FOR PECTUS EXCAVATUM:I. DEVELOPMENT, DESIGN, AND SIMULATIONS FOR FEASIBILITY (FORCE GENERATED) AND SAFETY (MAGNETIC FIELD STRENGTH NEAR THE HEART) (3 MINUTE)
Michael R. Harrison, M.D., Denise Estefan-Ventura, M.D., Richard Fechter, BS, Arthur M. Moran, Jr., BA, Darrell Christensen, BA, MA.
University of California, San Francisco, San Francisco, CA, U.S.A.

23 INFECTIOUS COMPLICATIONS AFTER THE NUSS REPAIR IN A SERIES OF 863 PATIENTS (3 MINUTE)
Susanna Shin, M.D.[1], Michael J. Goretsky, M.D., I-EVMS[2], Robert Kelly, M.D., I-EVMS[2], Tina Gustin, RN[1], Donald Nuss, M.D., I-EVMS.[2]
[1]Eastern Virginia Medical School, Norfolk, VA, U.S.A., [2]Children's Hospital of the Kings Daughters, Norfolk, VA, U.S.A.

Underlining denotes the author scheduled to present at the meeting.

24 WHEN IT'S NOT AN INFECTION: METAL ALLERGY AFTER THE NUSS PROCEDURE FOR REPAIR OF PECTUS EXCAVATUM (3 MINUTE)

Gregory Rushing, M.D.[1], Michael J. Goretsky, M.D., I-EVMS[2], Maripaz Morales, M.D.[2], Robert Kelly, M.D., I-EVMS[2], Tina Gustin, RN[2], Donald Nuss, M.D., I-EVMS.[2]

[1]Eastern Virginia Medical School, Norfolk, VA, U.S.A., [2]Children's Hospital of the Kings Daughters, Norfolk, VA, U.S.A.

25 *EX UTERO* INTRAPARTUM TREATMENT WITH PLACEMENT ON EXTRACORPOREAL MEMBRANE OXYGENATION (EXIT-TO-ECMO) FOR SEVERE CONGENITAL DIAPHRAGMATIC HERNIA (3 MINUTE)

Shaun M. Kunisaki, M.D.[1], Carol E. Barnewolt, M.D.[1], Judy A. Estroff, M.D.[1], Laura B. Myers, M.D.[1], Dario O. Fauza, M.D.[1], Louise E. Wilkins-Haug, M.D., Ph.D.[2], Ian A. Grable, M.D.[3], Luanne P. Nemes, RN, MS, PNP[1], Terry L. Buchmiller, M.D.[1], Jay M. Wilson, M.D.[1], Russell W. Jennings, M.D.[1]

[1]Children's Hospital Boston, Boston, MA, U.S.A., [2]Brigham and Women's Hospital, Boston, MA, U.S.A., [3]Beth Israel Deaconess Medical Center, Boston, MA, U.S.A.

26 FETAL LUNG-HEAD RATIO (LHR) IS NOT RELATED TO OUTCOME FOR ANTENATAL DIAGNOSED CONGENITAL DIAPHRAGMATIC HERNIA (CDH) (3 MINUTE)

Marc S. Arkovitz, Patricia Devine, M.D., Mark Russo, M.D., Nancy Budhorick, M.D., Charles J. Stolar, M.D.

Morgan Stanley Children's Hospital of New York-Presbyterian, New York, NY, U.S.A.

27 FETAL TRACHEAL OCCLUSION IN PATIENTS WITH SEVERE CONGENITAL DIAPHRAGMATIC HERNIA: A MORPHOLOGIC STUDY OF LUNG ARCHITECTURE AND ARTERIAL MUSCULARIZATION (3 MINUTE)

Enrico Danzer, M.D., Marcus G. Davey, Ph.D., Portia Kreiger, M.D., Eduardo D. Ruchelli, M.D., Mark P. Johnson, M.D., N. Scott Adzick, M.D., Alan W. Flake, M.D., Holly L. Hedrick, M.D.

The Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A.

28 PREDICTING INADEQUATE LONG-TERM LUNG DEVELOPMENT IN CHILDREN WITH CONGENITAL DIAPHRAGMATIC HERNIA: AN ANALYSIS OF LONGITUDINAL CHANGES IN VENTILATION AND PERFUSION (3 MINUTE)

Melissa J. Hayward, M.D., Virginia Kharasch, M.D., Catherine Sheils, M.D., Sandra Friedman, M.D., Mary-Jo Dunleavy, R.N., Sherri Utter, RND, David Zurakowski, Ph.D., Russell Jennings, M.D., Jay M. Wilson, M.D.

Children's Hospital Boston, Boston, MA, U.S.A.

29 MORTALITY IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH) ON EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO): A REPORT FROM THE CONGENITAL DIAPHRAGMATIC HERNIA STUDY GROUP (CDHSG) (3 MINUTE)

Rupa Seetharamaiah, M.D., John G. Younger, M.D., Robert H. Bartlett, M.D., Ronald B. Hirschl, M.D.

University of Michigan, Ann Arbor, MI, U.S.A.

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30 FETAL INTERVENTION FOR MASS LESIONS AND HYDROPS IMPROVES OUTCOME: A 15-YEAR EXPERIENCE (6 MINUTE)

Erich J. Grethel, Amy J. Wagner, M.D., Matthew S. Clifton, M.D., Raul A. Cortes, M.D., Diana L. Farmer, M.D., Michael R. Harrison, M.D., Robert H. Ball, M.D., Kerilyn K. Nobuhara, M.D., Hanmin Lee, M.D.

University of California, San Francisco, San Francisco, CA, U.S.A.

31 INCREASED HERITABILITY OF CERTAIN TYPES OF ANORECTAL MALFORMATIONS (3 MINUTE)

Richard A. Falcone, Jr., M.D., Marc A. Levitt, M.D., Alberto Peña, M.D., Michael Bates, M.D., Ph.D.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A.

32 ETHICS AND THE PEDIATRIC SURGEON (9 MINUTE)

Mary E. Fallat, M.D.[1], Donna A. Caniano, M.D.[2], Annie H. Fecteau, M.D.[3]

[1]University of Louisville, Louisville, KY, U.S.A., [2]Children's Hospital, Columbus, OH, U.S.A., [3]University of Toronto, Toronto, ON, Canada

10:00 a.m. – 10:30 a.m.	Refreshment break	*Ballroom Foyer*
10:30 a.m. – Noon	**Scientific Session 4: Nutrition and Intestinal Failure**	*Grand Ballroom*

{Moderators}:
Mary Brandt, M.D.
Daniel Teitelbaum, M.D.

{Educational Objectives:}
Session attendees will:

- Be able to define the complications associated with obesity and gain information regarding the surgical treatment of this condition in children.
- Recognize complications associated with intestinal failure, and be able to describe various approaches to treat and prevent these complications.
- Be able to define various approaches to feed patients post-operatively and in the newborn period.

33 REVERSAL OF TYPE 2 DIABETES AND IMPROVEMENT IN DYSLIPIDEMIA AND BLOOD PRESSURE FOLLOWING SURGICAL WEIGHT LOSS IN ADOLESCENTS: A MULTICENTER STUDY FROM THE PEDIATRIC BARIATRIC STUDY GROUP (3 MINUTE)

Thomas Inge, M.D., Ph.D.[1], Michael Chen, M.D.[2], Michael Helmrath, M.D.[3], Kimberly Wilson, MS[1], Rachel Akers, MPH[1], Judy Bean, Ph.D.[1], Victor Garcia, M.D.[1], Carroll "Mac" Harmon, M.D., Ph.D.[4], Stephen Daniels, M.D., Ph.D.[1], Lawrence Dolan, M.D.[1]

[1]Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A., [2]University of Florida College of Medicine, Gainesville, FL, U.S.A., [3]Baylor College of Medicine, Texas Children's Hospital, Houston, TX, U.S.A., [4]University of Alabama at Birmingham, Birmingham, AL, U.S.A.

34 SHORT-TERM RESULTS IN 41 OBESE U.S. CHILDREN TREATED WITH LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING (6 MINUTE)
Evan P. Nadler, M.D., Heekoung A. Youn, RN, Howard B. Ginsburg, M.D., Christine J. Ren, M.D., George A. Fielding, M.D.
New York University, New York, NY, U.S.A.

35 ETHANOL-LOCK TECHNIQUE FOR PERSISTENT BACTEREMIA OF LONG-TERM INTRAVASCULAR DEVICES IN PEDIATRIC PATIENTS (3 MINUTE)
Wes Onland, Cathy E. Shin, Stana Fustar, Theresa Rushing, Wing Yen Wong.
Los Angeles Children's Hospital, Los Angeles, CA, U.S.A.

36 ISOLATED LIVER AND MULTIVISCERAL TRANSPLANTATION FOR TPN-RELATED END-STAGE LIVER DISEASE (3 MINUTE)
Jaimie D. Nathan, Greg M. Tiao, M.D., Samuel A. Kocoshis, Maria H. Alonso, Frederick C. Ryckman, M.D.
Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A.

37 THE 2005 REPORT FROM THE INTERNATIONAL STEP DATA REGISTRY: INDICATIONS, EFFICACY, AND COMPLICATIONS — A GOOD FIRST STEP (3 MINUTE)
Biren P. Modi, M.D., Patrick J. Javid, M.D., Tom Jaksic, M.D., Ph.D., Hannah Piper, M.D., Monica Langer, M.D., Heung Bae Kim, M.D., on behalf of the International STEP Data Registry.
Children's Hospital Boston, Boston, MA, U.S.A.

38 EARLY FEEDING VS. 5-DAY FASTING AFTER ELECTIVE BOWEL ANASTOMOSES IN CHILDREN: A RANDOMIZED TRIAL (6 MINUTE)
Roberto Davila-Perez, M.D., Eduardo Bracho-Blanchet, M.D., Jose Manuel Tovilla-Mercado, M.D., Ricardo Reyes-Retana, M.D., Jaime Nieto-Zermeño, M.D.
Hospital Infantil de Mexico, México DF, Mexico

39 PROBIOTIC ACIDIFIED FORMULA IN AN ANIMAL MODEL REDUCES PULMONARY AND GASTRIC BACTERIAL LOAD (3 MINUTE)
Cristiano Boneti, M.D., Christine M. Habib, M.D., Jennifer E. Keller, M.D., Jose A. Diaz, M.D., Evan R. Kokoska, M.D., Richard J. Jackson, M.D., Samuel D. Smith, M.D.
Arkansas Children's Hospital, Little Rock, AR, U.S.A.

Noon – 12:30 p.m.	Refreshment break	*Ballroom Foyer*
12:30 p.m. – 3:00 p.m.	Telesurgery demonstration with lunch	*Grand Ballroom*
2:00 p.m. – 5:00 p.m.	Exhibits dismantle	*Ballroom Foyer*
3:00 p.m. – 5:00 p.m.	Posters dismantle	*Grand Ballroom A and B*
3:00 p.m. – 4:30 p.m.	COG Surgeons Meeting (open to all APSA meeting attendees)	*Sabal Palm Room*
6:30 p.m. – 10:30 p.m.	President's Banquet	*Grand Ballroom*

Underlining denotes the author scheduled to present at the meeting.

Wednesday, May 24

7:30 a.m. – 8:00 a.m.	Continental breakfast	*Ballroom Foyer*
7:30 a.m. – 11:30 a.m.	Registration open	*Ballroom Foyer*
8:00 a.m. – 8:30 a.m.	**APSA Foundation Scholars:** Elizabeth Beierle, M.D. and Kerilyn Nobuhara, M.D.	*Grand Ballroom*
8:30 a.m. – 9:30 a.m.	***Journal of Pediatric Surgery*/International Guest Lecture:** Pedro J. Roselló, M.D.	*Grand Ballroom*
9:30 a.m. – 11:30 a.m.	**Scientific Session 5: Basic Science/Trauma**	*Grand Ballroom*

{Moderators}:
Ai-Xuan Holterman, M.D.
Daniel Teitelbaum, M.D.

{Educational Objectives:}
Session attendees will:
- Improve their knowledge of embryonic growth, stem cells and fetal healing.
- Better understand the basic science approaches to pediatric neuroblastoma.
- Better understand the challenges of obtaining independent research funding and how to overcome those challenges.

40 A COMPARISON OF DIFFERENT PERINATAL SOURCES OF MESENCHYMAL PROGENITOR CELLS: IMPLICATIONS FOR TISSUE ENGINEERING (3 MINUTE)
Shaun M. Kunisaki, M.D., Julie R. Fuchs, M.D., Humberto Azpurua, M.D., David Zurakowski, Ph.D., Dario O. Fauza, M.D.
Children's Hospital Boston, Boston, MA, U.S.A.

41 NDSP: A NOVEL BIOMARKER AND GROWTH FACTOR IN NEUROBLASTOMA (6 MINUTE)
Sanjeev A. Vasudevan, M.D., Ningling Ge, M.D., Ph.D., Andrew D. Ludwig, B.A., Catherine L. Wesson, B.S., Kuan Wang, M.D., Ph.D., Xiao-Ying Shang, Ph.D., Susan M. Burlingame, B.S., M. Fatih Okcu, M.D., Heidi V. Russell, M.D., Jianhua Yang, Ph.D., Jed G. Nuchtern, M.D., FACS.
M.E. DeBakey Department of Surgery and Texas Children's Cancer Center, Department of Pediatrics, Baylor College of Medicine, Houston, TX, U.S.A.

42 PANCREATIC ALPHA-CELL DIFFERENTIATION BY MESENCHYME-TO-EPITHELIAL TRANSITION: IMPLICATIONS FOR STEM-CELL TREATMENTS? (6 MINUTE)
Warwick J. Teague, Autumn M. Rowan-Hull, Paul R.V. Johnson.
University of Oxford, Oxford, United Kingdom

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43 BLOCKADE OF VASCULAR ENDOTHELIAL GROWTH FACTOR RECEPTOR 2 (VEGFR2) SIGNALING ON TUMOR ENDOTHELIUM SUPPRESSES HUMAN NEUROBLASTOMA GROWTH IN MICE (3 MINUTE)

Paul Beaudry, M.D., M.Sc.[1], Matthew Rioth[2], David Poon[2], Daniela Prox, M.D.[2], Anderson Ryan, Ph.D.[3], John Heymach, M.D., Ph.D.[4], Judah Folkman, M.D.[2], Sandra Ryeom, Ph.D.[2]

[1]British Columbia's Children's Hospital, Vancouver, BC, Canada, [2]Boston Children's Hospital, Boston, MA, U.S.A., [3]Astra Zeneca, Macclesfield, United Kingdom, [4]MD Anderson, Houston, TX, U.S.A.

44 INTERFERON-β MEDIATED VESSEL STABILIZATION IMPROVES DELIVERY OF SYSTEMICALLY ADMINISTERED TOPOTECAN IN A MURINE MODEL OF RETROPERITONEAL NEUROBLASTOMA (6 MINUTE)

Paxton V. Dickson, M.D., John B. Hamner, M.D., Cathy Y.C. Ng, MS, Nikolaus L. Hagedorn, BS, Charles Fraga, MS, Clinton F. Stewart, Ph.D., Andrew M. Davidoff, M.D.

St. Jude Children's Research Hospital, Memphis, TN, U.S.A.

45 AGE-DEPENDENT RECRUITMENT OF NEUTROPHILS BY FETAL ENDOTHELIAL CELLS: IMPLICATIONS IN SCARLESS WOUND HEALING (6 MINUTE)

Bindi Naik-Mathuria, M.D., Andre Nicolas Gay, Ling Yu, Xi Zhu, Darrell Cass, M.D., C. Wayne Smith, M.D., Oluyinka O. Olutoye, M.D., Ph.D.

Baylor College of Medicine, Houston, TX, U.S.A.

46 NIH FUNDING AND THE PEDIATRIC SURGEON: CHARACTERIZING SUCCESSFUL APPLICANT PROFILES IN AN ERA OF INCREASING COMPETITION FOR EXTRAMURAL SUPPORT (3 MINUTE)

Shawn J. Rangel, M.D.[1], Jessica J. Kandel, M.D.[2], Charles J.H. Stolar, M.D.[2], R. L. Moss, M.D.[3], George K. Gittes, M.D.[4]

[1]Stanford University Medical Center, Stanford, CA, U.S.A., [2]Columbia University School of Medicine, New York, NY, U.S.A., [3]Yale University School of Medicine, New Haven, CT, U.S.A., [4]University of Pittsburgh School of Medicine, Pittsburgh, PA, U.S.A.

47 THE EPIDEMIOLOGY OF INFANT INJURIES AND ALARMING HEALTH DISPARITIES (3 MINUTE)

Richard A. Falcone, Jr., Rebeccah L. Brown, M.D., Victor F. Garcia, M.D.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A.

48 PEDIATRIC VASCULAR INJURIES: PATTERNS OF INJURY, MORBIDITY AND MORTALITY (3 MINUTE)

Denise B. Klinkner[1], Marjorie J. Arca, M.D.[2], Brian D. Lewis, M.D.[3], Keith T. Oldham, M.D.[2], Thomas T. Sato, M.D.[2]

[1]Children's Hospital of Wisconsin and Medical College of Wisconsin, Milwaukee, WI, U.S.A., [2]Children's Hospital of Wisconsin, Milwaukee, WI, U.S.A., [3]Medical College of Wisconsin, Milwaukee, WI, U.S.A.

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49 A GUIDELINE FOR EVALUATION OF THE CERVICAL SPINE IN THE PEDIATRIC TRAUMA PATIENT (3 MINUTE)

Eric R. Scaife, M.D.[1], Richard C.E. Anderson, M.D.[2], Kristine W. Hansen, R.N.[1], Stephen J. Fenton, M.D.[1], Douglas L. Brockmeyer, M.D.[1].

[1]Primary Children's Medical Center, Salt Lake City, UT, U.S.A., [2]Columbia University, New York, NY, U.S.A.

50 PEDIATRIC SNAKEBITES: LESSONS LEARNED FROM 114 CASES (3 MINUTE)

Brendan T. Campbell, M.D., M.P.H., John M. Corsi, MBA, Cristiano Boneti, M.D., Justin D. Phillips, Alisson L. Richards, Richard J. Jackson, M.D., Samuel D. Smith, M.D., Evan R. Kokoska, M.D.

Arkansas Children's Hospital, Little Rock, AR, U.S.A.

11:30 a.m. Annual Meeting Adjourns

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ABSTRACTS

V1 THORACOSCOPIC LEFT LOWER LOBECTOMY FOR AN INTRALOBAR SEQUESTRATION

Casey M. Calkins, M.D.[1], Shawn D. St. Peter, M.D.[2], George W. Holcomb, III, M.D.[2]
[1]Children's Hospital of Wisconsin, Milwaukee, WI, U.S.A., [2]Children's Mercy Hospital, Kansas City, MO, U.S.A.

A $2^1/_2$-year old male has been followed for a complex mass in the left lower lobe initially documented on a prenatal ultrasound. The lesion has remained stable, but has shown no evidence of regression. A left lower lobectomy was recommended by the Pulmonology Service. Preoperatively, a CT scan confirmed the complex cystic lesion in the left lower lobe with the presumptive diagnosis of a cystic adenomatoid malformation, but a Doppler ultrasound showed a possible aberrant arterial vessel from the aorta to the left lower lobe, raising the possibility of an intralobar sequestration.

This video will depict the salient features of the thoracoscopic left lower lobectomy in this infant. The port positions along with the thoracoscopic technique are highlighted. The patient was discharged on his first postoperative day and has not developed any postoperative problems in six months. Histologic examination of the specimen was consistent with an intralobar sequestration.

Notes:

V2 LAPAROSCOPIC RIPSTEIN PROCEDURE FOR RECTAL PROLAPSE

Mark L. Wulkan, M.D.
Emory University School of Medicine, Atlanta, GA, U.S.A.

Purpose:
This video will demonstrate a laparoscopic Ripstein rectopexy in an 11-year old girl with intractable rectal prolapse. The patient has had daily episodes of prolapse for years. She is usually able to self-reduce her prolapse; however, she has had multiple emergency room visits for assistance with reduction. She has been resistant to medical therapy, including aggressive laxative therapy and behavior therapy. Her mother and maternal grandmother have a similar history.

Methods:
The technique demonstrated is performed laparoscopically. A retrorectal window is formed at the level of the pre-sacral fascia. An 8-ply porcine small intestinal submucosa mesh was used for the rectopexy. The mesh is secured to the pre-sacral fascia and to the anterior aspect of the rectum.

Results:
The procedure was completed successfully. The patient is doing well with resolution of her rectal prolapse four months post-operatively.

Conclusions:
A Laparoscopic Ripstein rectopexy can be performed successfully.

Notes:

V3 TRANSANAL ENDORECTAL PULL-THROUGH IN THE PRONE POSITION

D. D. Potter, Penny L. Stavlo, CNP, Christopher Moir, M.D.
Mayo Clinic, Rochester, MN, U.S.A.

Purpose:
Transanal endorectal pull-through has been advocated for short- to medium-segment Hirschsprung's disease due to shorter hospital stay, lower costs, improved cosmesis, and similar outcomes when compared to traditional techniques. The prone jackknife position provides superior exposure to allow control of mesenteric vessels during transanal pull-through as compared to the lithotomy position. We report a case of transanal endorectal pull-through in the prone position in a 5-month old boy with medium segment Hirschsprung's disease.

Methods and Results:
Our patient is a 5-month old boy with a history of inability to pass meconium, feeding intolerance, and a rectal biopsy devoid of ganglion cells. Barium enema demonstrated a transition point in the mid-sigmoid colon. He was admitted two days prior to operation for colonic irrigation. On the day of operation, general anesthesia was initiated and the boy was placed in prone jackknife position. His buttocks were retracted laterally and a retractor was placed to expose the anal verge. Mucosectomy was begun 0.5 cm proximal to the dentate line and was carried out to 7 cm proximally. At this point, the abdomen was entered and full-thickness bowel was herniated through the anus. Mesenteric vessels were easily visualized and controlled with electrocautery. The dissection was carried 31 cm onto the colon, where ganglion cells were identified. The bowel was transected and a coloanal anastomosis was performed using absorbable suture. The wound was packed with petroleum impregnated gauze. One month postoperatively, our patient has had an excellent functional outcome with regular stools and no abdominal distention.

Conclusions:
Transanal endorectal pull-through in the prone position offers all the benefits of minimally invasive procedures for Hirschsprung's disease with the added advantage of excellent exposure of the mesenteric vessels without using laparoscopy. We favor this procedure for children with short to medium segment Hirschsprung's disease.

Notes:

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V4 THORACOSCOPIC REPAIR OF ESOPHAGEAL ATRESIA WITH TRACHEOESOPHAGEAL FISTULA

Denise E. Ventura, M.D., Hanmin Lee, M.D.
University of California, San Francisco, San Francisco, CA, U.S.A.

Purpose:
Esophageal atresia repair has been successfully performed via thoracotomy for many years. Since 1998, the thoracoscopic technique to correct esophageal atresia and tracheoesophageal fistula has emerged as an alternative approach to avoid morbidity related to thoracotomy. The authors present the technical steps of the procedure.

Methods:
All data and video presentation was done by IRB approval. A newborn underwent a thoracoscopic repair of esophageal atresia and tracheoesophageal fistula. Port placement was performed through two 5 mm and one 3 mm trocars on the right side using a 30 degree laparoscope. With the patient in near prone position the procedure was carried out transpleurally. The main steps for this procedure are identification of azygous vein, vagus nerve identification and preservtion, dissection of the tracheoesophagel fistula and the ligation with two titanium clips, identification of proximal pouch and primary anastomosis of the esophagus with interrupted sutures.

Results:
Operative time was 2.5 hours. There were no significant postoperative complications. Postoperative esophagram showed no leak and no significant stricture.

Conclusions:
The minimally invasive thoracoscopic technique is a safe and efficient approach to correct esophageal atresia and avoids the morbidity of a thoracotomy.

Notes:

V5 LAPAROSCOPICALLY ASSISTED GASTRIC TRANSPOSITION FOR LONG-GAP ESOPHAGEAL ATRESIA

Philipp O. Szavay, M.D., Hans Joachim Kirschner, M.D., Hans-Walter Hacker, M.D., Joerg Fuchs, M.D.
University of Tuebingen, Tuebingen, Germany

Purpose:
The most common indication for esophageal replacement in children is long-gap esophageal atresia. Gastric transpostition for this purpose has been carried out laparoscopically assisted in adults but in single cases also in the pediatric population and is a true option for these patients. Authors report with their video on the laparoscopically assisted procedure in an 5-month old boy with type Ib long-gap esophageal atresia

Methods:
Postnatally a cervical esophagostomy as well as a gastrostomy were performed. Surgery for gastric pull-up was carried including the closure of the gastrostomy site, exposure of the esophageal hiatus with transhiatal dissection, complete mobilization of the stomach, resection of the lower esophageal stump, the left-thoracic pull-up of the stomach with anastomosis to the upper esophageal stump and a laparoscopic feeding jejunostomy.

Results:
No intra- or perioperative complications occured. Operation time was 3 hours 50 minutes. The child could be weaned and extubated on day 2 after surgery, but had to be re-intubated due to respiratory problems. Further course was then unventful, postoperative upper GI studies showed no obtruction and a good gastric clearance six weeks after surgery.

Conclusions:
Gastric transposition seems to be the therapy of choice in children with need for esophageal replacement. As indicated in literature the surgical procedure can be done laparoscopically assisted. As in minimal invasive surgery in general also in patients with need for esophageal replacement, the morbidity of a large laparotomy and/or thoracotomy can be avoided. Careful patient selection and monitoring is mandatory. The laparoscopic approach is technically demanding but offers excellent view on site and exact preparation at the transhiatal dissection. Long-term results are pending.

Notes:

Underlining denotes the author scheduled to present at the meeting.

V6 ADVANCED MINIMALLY INVASIVE SURGERY IN CHILDREN: ENDOSCOPIC EXCISION OF AN ORBITOFACIAL TUMOR

Farhad Sigari, M.D., MS, Adam M. Vogel, M.D., Dana L. Suskind, M.D., Donald C. Liu, M.D., Ph.D.
University of Chicago, Chicago, IL, U.S.A.

Purpose:
Superficial extracranial orbitofacial tumors are readily amenable to standard surgical excision. However, this may result in cosmetically unacceptable. The developments of endoscopic techniques allow these scars to be hidden behind the hairline. We illustrate the technical considerations in the endoscopic excision of such tumors

Methods:
Retrospective case and operative video review.

Results:
A 12-year old girl presented with an asymptomatic slow growing midline forehead mass. A CT scan revealed the absence of intracranial extension. Because the mass was in the center of her forehead, the patient and her family had significant concerns regarding postoperative scarring. An endoscopic resection was performed via an incision placed 2 cm behind the hairline. A 30 degree, 6 mm scope was introduced through a single port and the mass was easily resected using a combination of blunt and sharp endoscopic instrumentation. The end cosmetic result was highly favorable.

Conclusions:
Endoscopic excision represents a viable option for the treatment of extracranial orbitofacial tumors in children. Important technical considerations include: preoperative imaging, port site, and appropriate endoscopic instrumentation.

Notes:

Poster Session IA: Gastrointestinal Surgery — Experimental and Clinical Perspectives

P1 TRENDS IN FUNDOPLICATION FOR CHILDREN WITH GERD: ARE THERE REGIONAL AND DEMOGRAPHIC DIFFERENCES?

James T. McPhee, M.D., Maksim Zayaruzny, M.D., MPH, Pradeep P. Nazarey, M.D., Michael P. Hirsh, M.D., Paul D. Danielson, M.D.
University of Massachusetts Medical Center, Worcester, MA, U.S.A.

Purpose:
Fundoplication represents the mainstay surgical management of medically refractory gastroesophageal reflux disease (GERD) in children. The goal of this study was to evaluate current national trends in fundoplication for children with GERD to determine which factors affect rates of surgery.

Methods:
This is a retrospective study of 4,059 children (19,952 nationally by weighted analysis) that underwent fundoplication for GERD based on the National Inpatient Sample (1998-2003). Chi-square and logistic regression analyses were performed to evaluate the influence of region, age, race, income, insurance, and hospital type on rates of fundoplication.

Results:
From 1998-2003 an estimated 93,332 children were discharged from U.S. hospitals with a primary diagnosis of GERD. Of those, 19,952 (21.4 %) underwent fundoplication during that hospitalization. Operative mortality was 0.39%. The highest rate of wraps were performed in the Western states (31.0%), followed by Midwest (23.0%), Northeast (18.0%), and the South (17.8%). Lower rates of surgery occurred in younger children (<1 year vs. 6-17 years), (8.98% vs. 63.4%, OR 16.9 [95% CI 12.7-22.4], p<.05); non-white vs. white children (17.7 % vs. 24.3%, OR 1.4 [95% CI 1.1-1.8], p <.05) and those treated at non-teaching vs. teaching hospitals (9.4% vs. 24.9%, OR 2.85 [95% CI 1.7-4.8], p <.05). While the univariate analysis demonstrated children with private insurance and greater family income had higher rates of fundoplication; this did not hold true in the multivariate regression analysis (p>.05). Post-operative length of stay averaged 4.7 days, no significant difference between regions and hospital types was found (p>.05).

Conclusions:
Based on a large population-based retrospective analysis, factors that significantly influence rates of fundoplication for children admitted with GERD include: admission to Western U.S. hospitals, admission to U.S. teaching hospitals, Caucasian race, and age > 6 years. The impact these higher rates of intervention have on long-term outcomes remains to be seen.

Notes:

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P2 RECENT CHANGES IN THE CHARACTERISTICS OF CHILDREN UNDERGOING ANTI-REFLUX SURGERY IN THE U.S.

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Purpose:
Over the past decade, a laparoscopic approach to anti-reflux surgery has been increasingly used in adults and children. Studies have suggested that laparoscopy has lowered thresholds for intervention and increased rates of anti-reflux surgery in adults. The degree to which laparoscopy may have affected use of these procedures in children is unknown. The purpose of this study was to analyze recent trends in utilization of antireflux surgery in children.

Methods:
Children (<18 yrs) undergoing antireflux surgery were identified in the Nationwide Inpatient Sample from 1996-2003. Census data was used to calculate the population-based rates of procedures stratifying by age and presence of neurological impairment (using ICD-9 codes). Survey statistics were used to account for the sampling design of the database.

Results:
From 1996-2003, 48,665 anti-reflux procedures were performed on children in the U.S. Overall, 45% of children were <1-year old, 39% were 1-9 years old and 16% were 10-17 years old. The population-based rate of procedures nearly doubled between 1996 and 2003 (6 vs. 11 procedures/100,000, p=0.03). A significant increase in the rate of procedures was observed in children <1-year old (p=0.04) and those 10-17 years old (p=0.008) but not among those 1-9 years old (p=0.13). Most (55%) children undergoing anti-reflux surgery were neurologically normal, with the percentage of neurologically normal children increasing from 47% in 1996 to 60% in 2003 (p<0.001). The largest increase in number of procedures was among neurologically normal children, most of whom (50%) were <1-year old. The most rapid increase in procedures was observed among neurologically normal children from 10-17 years old.

Conclusions:
The utilization of anti-reflux surgery has recently increased in children, mainly attributable to more procedures among neurologically normal children. These findings suggest that the indications for anti-reflux surgery in children have rapidly evolved in the laparoscopic era.

Notes:

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P3 PREDICTING THE NEED FOR FUNDOPLICATION IN PATIENTS WITH CONGENITAL DIAPHRAGMATIC HERNIA

Ivan R. Diamond, M.D., Kandice Mah, Peter C.W. Kim, M.D., Desmond Bohn, M.D., J. Ted Gerstle, M.D., Paul W. Wales, M.D.
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Purpose:
To examine pre-operative factors predictive of subsequent fundoplication for gastro-esophageal reflux in children with Congenital Diaphragmatic Hernia (CDH).

Method:
Retrospective cohort study of children who underwent repair of a CDH at a tertiary level pediatric hospital between 1 January 1995 and 31 December 2002. Excluded were children who died during their first admission (n=19), or who underwent fundoplication at the time of CDH repair (n=3). Follow-up continued to 1 September 2005 to capture those who underwent fundoplication. Univariate and multivariate logistic regressions were performed to examine pre-operative factors predictive of subsequent fundoplication.

Results:
Eighty-six patients were enrolled, and 10 of these underwent fundoplication at mean 1.5 years (range: 0.2 - 5.8 years) from CDH repair. Mean follow-up was 4.7 years (sd = 2.7 years). The following table presents the results of the regression analyses.
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Predictors of fundoplication						
	Univariate OR	**Univariate 95% CI**	**Univariate p-value**	**Multivariate OR**	**Multivariate 95% CI**	**Multivariate p-value**
Birth Weight	1.00	0.99 - 1.01	0.58			
Gestational Age	0.93	0.71 - 1.21	0.58			
Pre-ductal saturationV	0.97	0.78 - 1.21	0.78			
Use of Nitric oxide	4.49	1.07 - 18.81	0.04			
Use of HFOV	5.38	1.28 - 22.66	0.02			
Right-sided CDH	5.00	1.02 - 24.49	0.047			
Liver in chest	20.74	2.48 - 173.33	0.005	9.60	1.04 - 88.61	0.046
Stomach in chest	0.69	0.18 - 2.58	0.58			
Patch closure	13.88	2.69 - 71.62	0.002	6.03	1.05 - 34.61	0.044

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Birth weight, gestational age, pre-ductal saturation and the stomach within the chest were not predictive of subsequent fundoplication on univariate analyses. High Frequency Oscillatory Ventilation (HFOV), nitric oxide use, a right-sided hernia, liver within the chest and the need for patch closure predicted subsequent intervention. However on multivariate analysis only liver within the chest and patch closure of the hernia were significant in predicting subsequent fundoplication. Of the children who underwent fundoplication, 90% had liver within the chest and 80% underwent patch repair.

Conclusion:
Infants with CDH who have liver within the chest or require patch closure of their hernia are at increased risk for subsequent fundoplication for gastro-esophageal reflux. These children may represent a subpopulation who would benefit from fundoplication at the time of CDH repair.

Notes:

P4 ABNORMAL MUCOSAL INNERVATION OF THE GANGLIONIC BOWEL IN HIRSCHSPRUNG'S DISEASE

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Purpose:
Some patients continue to have persistent bowel symptoms following satisfactory pull-through operation for Hirschsprung's disease (HD). The postoperative bowel dysfunction includes enterocolitis, constipation and faecal incontinence. We designed this study to characterize innervation abnormalities in the proximal portion of the resected large bowel specimen after pull-through operation.

Methods:
Entire bowel specimens removed during transanal pull-through in 10 consecutive children with proven HD were examined using PGP 9.5 and Neurofilament immunohistochemistry. Comparison was made with normal colonic specimens taken from children with anorectal anomalies. The extent of staining was graded on the following scale: (-) = no fibers; (±) = occasional fibers; (+) = a few fibers; (++) = moderate number of fibers; (+++) = many fibers.

Results:
There was complete lack of PGP9.5 and Neurofilament staining in the mucosa of the aganglionic colon (Table). There were only a few positive fibers seen in the mucosa of the ganglionic bowel compared to many positive fibers in the control bowel.

	Aganglionic bowel	Transition Zone	Ganglionic bowel	Control bowel
Mucosal Innervation	-	±	+	+++
Submucosal (ganglia cells)	-	+	+++	+++
Myenteric (ganglia cells)	-	+	+++	+++

Conclusions:
The altered mucosal innervation in the ganglionic segment in HD may result in failure of transmission of neuronal signals to the epithelium from the integrated networks of the myenteric and submucous plexuses. This may adversely influence the secretory, absorptive and sensory functions of the pulled-through bowel.

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P5 GH ADMINISTRATION ENHANCES LIVER FUNCTION AND SURVIVAL DURING BILIARY OBSTRUCTION IN MICE

Micheal Chen, M.D., Ph.D., Minhua Wang, Ph.D., GuoQiang Zheng, M.D., Ai-Xuan Holterman, M.D.
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Growth hormone (GH) administration enhances hepatocyte proliferation and liver regeneration during partial hepatectomy.

Hypothesis:
GH would have similar sparing effects on liver injury during bile duct ligation (BDL).

Methods:
12 groups of CD1 mice underwent sham surgery or BDL along with 7, 14 or 28 days infusion of PBS or GH. BrdU labeling; serum albumin, cholesterol, LFT; body weight (wt) change ((sacrifice wt - wt_o)/ wt_o); % liver mass (liver wt/body wt at sacrifice) (n=8); and survival (7, 14 or 28 days BDL or GHBDL (n=40 each) and PBS/GH Sham (n=10 each) were measured. Results: GH treated sham mice have no liver mass change but 2-fold increases in % body wt change and hepatocyte proliferation (0.95+/-0.4 vs. 0% ($p<0.05$)) relative to PBS mice. All PBS or GH treated BDL mice have increased total bilirubin and serum ALT/AST (p=NS). Data are tabulated below as mean±SD with *p value<0.05 showing that GHBDL mice have lower serum Alkaline Phosphatase and cholesterol; higher albumin, % body wt change and survival by day 7 of surgery, and higher liver mass at day 28 compared to BDL mice. This is associated with higher hepatocyte proliferation.

Conclusion:
In biliary obstruction, early GH treatment diminishes cholestasis, improves liver function, prolongs survival, enhances hepatocyte proliferation and liver weight, suggesting that a mechanism by which GH treatment in obstructive biliary diseases attenuates injury and improves outcome is by stimulating hepatocyte proliferation.

	BDL Day 7	***GHBDL Day 7***	BDL Day 14	***GHBDL Day 14***	BDL Day 28	***GHBDL Day 28***
Alkaline Phosphatase (U/L)	1206 ± 88	****550 ± 248***	1500 ± 1315	****482 ± 223***	1239 ± 342	****423 ± 145***
Cholesterol (mg/dL)	231 ± 112	****113 ± 29***	191 ± 63	****135 ± 28***	222 ± 74	**** 129 ± 39***
Albumin (g/dL)	2.7 ± 0.7	****3.3 ± 0.5***	3 ± 0.4	****4 ± 0.6***	2.4 ± 0.6	**** 3.4 ± 0.2***
% survival	95	****98***	86	****97***	80	**** 89***
% body wt change	- 8 ± 2.7	**** 2 ± 0.9***	2 ± 1	****17 ± 15***	11 ± 13	**** 32 ± 11***
% liver mass	9 ± 2	***9 ± 2***	10 ± 2	***10 ± 4***	10 ± 3	**** 14 ± 2***
% hepatocyte proliferation	5 ± 1.7	***10.5 ± 3.2***	6 ± 5	***12 ± 7***	3.8 ± 2.8	****11.4 ± 3.7***

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P6 ILEAL PANETH CELLS EXPRESS FGF10 AFTER SMALL BOWEL RESECTION

Jennifer L. Curtis, M.D., Pierre M. Del Moral, Travis Chong, Frederic G. Sala, Lily Lee, M.D., Henri R. Ford, M.D., Saverio Bellusci, Ph.D., Kasper S. Wang, M.D.
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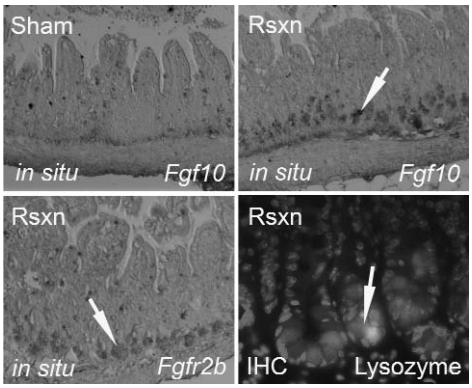
Purpose:
Fibroblast growth factor-10 (FGF10) is a key mitogen during gut organogenesis. We have previously shown that *Fgf10* expression, as demonstrated by a LacZ reporter in transgenic mice, increases in ileal crypt epithelial cells following 50% small bowel resection (SBR). We have also shown that immortalized intestinal progenitor cells, IEC-6, express *Fgfr2b* but not *Fgf10*. Exactly where *Fgf10* is expressed and what its target is following SBR remains unclear. Given the distribution of LacZ activity, we hypothesized that Paneth cells, which reside at the base of the intestinal crypts, express *Fgf10* following SBR. Furthermore, given the pro-mitogenic capacity of *FGF10,* we hypothesized that the adjacent epithelial progenitor cells express the receptor for *FGF10*, *Fgfr2b.*

Methods:
Adult transgenic mice (Mlc1v-nLacZ) with LacZ as a reporter gene for *Fgf10* expression underwent either 50% SBR or sham operation. Tissue sections were assayed for LacZ expression or for lysozyme (a Paneth cell marker) by immunofluorescence. *In situ* hybridization for *Fgf10* and *Fgfr2b* was performed for spatial localization of ligand and receptor. Real time RT-PCR was performed to quantitate the change in level of *Fgf10* expression.

Results:
Following SBR, *Fgf10* and *Fgfr2b* expression increases in the ileal crypts as determined by LacZ activity and by *in situ* hybridization. By real time RT-PCR, we found that *Fgf10* levels increase four-fold following SBR compared to sham. LacZ activity and *in situ* hybridization for *Fgf10* and *Fgfr2b* correspond spatially with lysozyme staining with immunohistochemistry.

Conclusions:
We conclude that *Fgf10* and *Fgfr2b* expression are upregulated in Paneth cells following SBR. We speculate that Paneth cells secrete *FGF10*, which acts in both a paracrine and an autocrine fashion to increase proliferation of intestinal progenitor cells and Paneth cells bearing the receptor FGFR2b. The increased expression of *Fgf10* may lead to enhanced adaptation following SBR.



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P7 THE USE OF PET SCAN TO IDENTIFY ECTOPIC PANCREATIC TISSUE RESPONSIBLE FOR CONGENITAL HYPERINSULINISM

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Purpose:
Congenital hyperinsulinism (HI) results from unregulated insulin secretion by pancreatic ?-islet cells. Rarely, this pancreatic tissue is of ectopic origin. Locating lesions responsible for HI is critical since excision of focal lesions can result in cure. Unfortunately, current diagnostic techniques have significant limitations. PET scans have been used to identify neuroendocrine tumors in the past. We evaluate the ability of PET to locate focal pancreatic tissue responsible for unregulated insulin secretion of ectopic origin.

Methods:
After obtaining IRB exemption, a retrospective case review was performed.

Results:
A two month old female diagnosed with HI was transferred to our facility after a 98% pancreatectomy failed to improve persistent hypoglycemia. A PET scan with ^{18}F-fluoro-l-dihydroxyphenylalanine (^{18}FDOPA) revealed four discrete foci of uptake, two believed in the pancreatic head and two inferior to the remnant pancreas. A completion pancreatectomy was performed at which time abdominal exploration identified four jejunal nodules thought responsible for the foci noted on PET. The distal three nodules were resected. Frozen section biopsy of the proximal nodule demonstrated pancreatic tissue of questionable normality. This tissue was not resected representing the only remaining pancreatic tissue to prevent iatrogenic diabetes. Postoperatively, the patient continued to require a high glucose infusion rate. A repeat PET scan demonstrated an area of increased ^{18}FDOPA uptake in the left upper abdomen believed to represent the remaining jejunal nodule. The patient returned to the operating room and the nodule was removed. Permanent sections of the remnant pancreatic head and four jejunal nodules confirmed the PET findings and demonstrated focal islet cell hyperplasia in all lesions. The patient was discharged without further hypoglycemic episodes.

Conclusions:
Unregulated insulin secretion from ectopic pancreatic tissue can result in HI. PET can successfully identify ectopic tissue responsible for hyperinsulinemic hypoglycemia highlighting its value in the diagnosis and management of HI.

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P8 TIME-DEPENDENT EFFECTS OF EARLY STIMULATION WITH GLP-2 ON INTESTINAL ADAPTATION

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Previous studies have shown that immediate treatment with Glucagon-like peptide-2 (GLP-2) will augment adaptation in short bowel syndrome (SBS).

Aim:
This study investigates the long-term effects in intestinal adaptation of short-term treatment with GLP-2 in a total parenteral nutrition(TPN)-supported model of SBS.

Methods:
Sprague-Dawley rats underwent a 90% resection of proximal small intestine followed by a jugular catheter insertion. Animals were divided into two groups: Control (TPN alone; n=8) and TPN+GLP-2 (10ug/kg/h; GLP-2 treatment started postresection for seven days, followed by a withdrawal of peptide in the second week; n=8). After 14 days, animals were sacrificed and the ileal remnant harvested. The intestinal tissue was processed for morphological analysis, crypt cell proliferation (CCP), apoptosis (Caspase-3) and glucose transporters (SGLT-1, GLUT-2 and GLUT-5).

Results:
Gross and microscopic intestinal morphology was significantly increased in TPN+GLP-2 compared to Control by GLP-2 treatment (length; TPN+GLP-2, 17.5 ± 0.9 cm vs. Control, 10.5 ± 0.3 cm, $p<0.01$, weight; TPN+GLP-2, 1.67 ± 0.11 g vs. Control, 1.19 ± 0.07 g, $p<0.01$, width; TPN+GLP-2, 1.26 ± 0.06 cm vs. Control, 0.98 ± 0.07 cm, $p<0.01$). Villus height was significantly increased in TPN+GLP-2 compared to Control (TPN+GLP-2, 467.8 ± 26.8 um vs. Control, 378.8 ± 25.5 um, $p<0.05$). The CCP was significantly increased in TPN+GLP-2 compared to Control (TPN+GLP-2, 19.2 ± 1.1 cells/crypt vs. Control, 13.2 ± 0.9 cell/crypt, $p<0.01$). No significant differences were seen in crypt depth, apoptosis and glucose transporters.

Conclusions:
Short-term post resection treatment with GLP-2 induces long-lasting increases in gross and microscopic intestinal morphology and crypt cell proliferation. However, the acute effects of increasing SGLT-1 expression are not seen. This suggests that shot-term treatment of GLP-2 following surgical resections may have long-term clinical benefits.

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P9 EPIDERMAL GROWTH FACTOR ENHANCES ENTEROCYTE MIGRATION BY DECREASING INTEGRIN EXPRESSION AND FUNCTION IN A PI3 KINASE-DEPENDENT MANNER

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Purpose:
NEC is characterized by impaired intestinal restitution and decreased enterocyte migration, in part through increased integrin expression. We and others have shown that epidermal growth factor (EGF) enhances enterocyte migration, although the mechanisms involved remain incompletely understood. We now hypothesize that EGF increases the rate of enterocyte migration *in vitro* and *in vivo* through a down-regulation of integrin expression and function, and sought to determine the mechanisms involved.

Methods:
Enterocyte migration was measured in a scrape-wounding assay of confluent IEC-6 cells using video-microscopy. Expression of β1 and α3 integrins in IEC-6 cells ± EGF(40ng/ml,0-12h) ± wortmannin (150nM) was measured by SDS-PAGE. Cell-matrix binding was measured by counting the number of fibronectin-coated beads bound to IEC-6 cells ±EGF. Ileal mucosal scrapings were obtained from newborn Swiss-Webster mice injected with saline or EGF (500ng/ml) either 4h or 12h previously.

Results:
EGF increases the rate of migration of IEC-6 enterocytes compared with control cells (μm/h:ctrl 7.5±2 vs. EGF:12.5±2,$p<0.05$), and decreased the expression of β1integrin in a time-dependent manner (β1/actin: ctrl: 2±.5, 4h: 1.5±0.5, 6h: 0.7±0.2, 12h: 0.1±0.1,$p<0.05$ ANOVA). EGF also decreased bead binding at 6h, demonstrating reduced integrin function (beads per cell: ctrl: 3±1 versus EGF 1±1, $p<0.05$). To determine the mechanisms involved, the decrease in integrin expression by EGF was partially blocked by pre-treatment with the phospho-inositol-3 kinase inhibitor wortmannin (150nM), (β1/actin: wortmannin at 6h, 1.8±.5 NS versus control). Strikingly, animals treated with EGF showed a time-dependent decrease in integrin expression on enterocytes compared with untreated animals, suggesting the *in vivo* significance of this effect.

Conclusions:
EGF decreases the expression of integrins in enterocytes, leading to decreased cell-matrix binding and enhanced enterocyte migration. These findings provide further mechanistic insights into a potential therapeutic role for EGF in diseases of intestinal inflammation such as NEC.

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P10 HB-EGF PROMOTES ENTEROCYTE PROLIFERATION AND MIGRATION IN NEONATAL RATS WITH NECROTIZING ENTEROCOLITIS

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Purpose:
We have previously shown that heparin-binding EGF-like growth factor (HB-EGF) decreases the incidence and severity of experimental necrotizing enterocolitis (NEC). Intestinal epithelial cell (IEC) proliferation and migration (restitution) are key elements in recovery of the intestine from injury. The aim of this study was to investigate whether the beneficial effect of HB-EGF in experimental NEC is mediated, in part, by its ability to affect IEC proliferation and migration.

Methods:
NEC was induced in newborn rat pups by exposing them to stress (repeated cycles of hypoxia and hypothermia with administration of hypertonic feedings plus LPS). To investigate the effect of HB-EGF on enterocyte proliferation and migration, bromodeoxyuridine (BrdU, 50 mg/kg) was administered intraperitoneally 18 h prior to sacrifice. Pups (n=30) were randomly divided into the following groups: 1) breast fed, 2) stressed, and 3) stressed but with HB-EGF (600 μ/kg) added to the feeds. Intestine was harvested and tissue sections immunostained for BrdU. Enterocyte migration rate was calculated as the distance from the bottom of the crypt to the foremost labeled enterocyte/18 h, and enterocyte proliferation was expressed as the number of BrdU positively stained cells/high power field (HPF). Results were analyzed by ANOVA with $p<0.05$ considered statistically significant.

Results:
The IEC migration rate in breast fed pups was 7.07 μm/h. This rate decreased significantly to 2.29 μm/h in stressed animals ($p<0.05$), and was significantly improved at 5.95 μm/h in animals subjected to stress but treated with HB-EGF ($p<0.05$ compared to stress alone). IEC proliferation was 208 cells/HPF in breast fed pups. Proliferation decreased significantly to 99 cells/HPF in stressed animals ($p<0.05$) and increased to 190 cells/HPF in stressed animals treated with HB-EGF ($p<0.05$ compared to stress alone).

Conclusions:
These results demonstrate that HB-EGF preserves enterocyte migration and proliferation in a neonatal rat model of NEC.

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P11 THE SIGNIFICANCE OF FASTING AND POST-PRANDIAL GLP-2 LEVELS IN CORRELATION WITH INTESTINAL PERMEABILITY IN CROHN'S DISEASE — PRELIMINARY REPORT IN PEDIATRIC PATIENTS

Dragan Kravarusic, M.D.[1], David L. Sigalet, Professor[1], Jens J. Holst, Professor[2].
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Purpose:
To describe the circulating fasting and post-prandial levels of bioactive GLP-2 in acute flare and remission of Crohn's disease in correlation with intestinal permeability and nutrient transport function.

In normal gut physiology the intestinotrophic hormone GLP-2 has been shown to promote mucosal growth, increase nutrient transport capacity and decreases permeability. It has been suggested that Crohn's disease may be caused by an abnormality in intestinal permeability; we hypothesized that this initial permeability defect may be related to abnormalities in GLP-2 release, or response.

Methods:
With ethics board approval, parents of patients (n=7) hospitalized for acute flare of ileal Crohn's disease (activity index>200) were approached for consent; age matched controls (n=10) were recruited from local schools. Fasting and post-prandial stimulated GLP-2 levels were measured; intestinal function was quantified using urinary recovery of inert sugar probes: 3-0 methylglucose (actively transported), and Lactulose/Mannitol (passive permeants). Crohn's patients were tested in the acute and remission phase and compared with healthy controls.

Results:
Patients with acute Crohn's had reduced post prandial levels of GLP-2: acute 24.1±10.9 vs. controls 30.9±6.6 and remission phase 38.9±10.3 pMol/L. Intestinal permeability was increased in acute Crohn's: Lactulose/Mannitol ratios: Acute 0.056±0.025 vs. controls 0.029±0.0079 and remission phase 0.0323±0.0099, while glucose absorption was decreased: Acute 43.3±5.6 vs. Controls 58.1±11.6 and remission phase 60.6±12.5 (% absorption). (All Data as Mean ± SD, all comparisons $p<0.05$).

Conclusions:
Our results suggest that patients with acute Chron's disease have decreased meal stimulated GLP-2 release, in association with increased permeability, and decreased nutrient absorption. These abnormalities appear to normalize in the remission phase. Further studies are indicated to determine if enteroendocrine abnormalities are causal, or associated with active Crohn's disease.

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P12 RNA INTERFERENCE FOR TREATMENT OF ACUTE INFLAMMATORY COLITIS

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Purpose:
Inflammatory bowel disease remains a difficult disease to treat. RNA interference (RNAi) is a powerful tool used for transient, site-specific gene silencing. Calcitonin receptor-like receptor (CRLR) is a mediator of intestinal blood flow and inflammation. The purpose of this study is to explore the potential of selective gene silencing of CRLR in the colon to alter the genesis of inflammation in a rat model of colitis.

Methods:
This study was approved by the IACUC. Male Sprague-Dawley rats were divided into four groups (n=4-6/group):
1. Saline injection, 50% ethanol vehicle enema;
2. Saline injection, trinitrobenzene sulfonic acid (TNBS) enema;
3. Control double-stranded RNA (dsRNA) injection, TNBS enema;
4. CRLR dsRNA injection, TNBS enema.The descending colon was harvested 72 hours after administration of dsRNA. Tissues were evaluated by hematoxylin/eosin, immunohistochemistry, and concomitant changes in inflammation measured using a myeloperoxidase (MPO) assay.

Results:
As expected, groups 2 and 3 displayed increased tissue edema, mucosal destruction, and increased submucosal infiltration of polymorphonuclear cells on histologic staining when compared to Control (group1). Interestingly, these symptoms were exacerbated in the group lacking CRLR (group 4). Myeloperoxidase assays revealed a significantly higher level of MPO in CRLR knock-down animals compared to group 2 ($P = 0.01$). Saline and control dsRNA injected animals exhibited similar histology and MPO activity. Immunohistochemical staining confirmed the knock-down of CRLR in rats receiving dsCRLR RNA. Rats receiving control dsRNA showed no difference in histology or MPO activity compared to saline injected animals.

Conclusions:
RNAi is successful in silencing CRLR, and can be used to create transient, tissue specific phenotypic knock-outs. Our data show that CRLR has an anti-inflammatory effect in the descending colon in the rat model of TNBS induced acute inflammatory colitis. RNAi has potential therapeutic applications in the treatment of acute inflammatory bowel disease.

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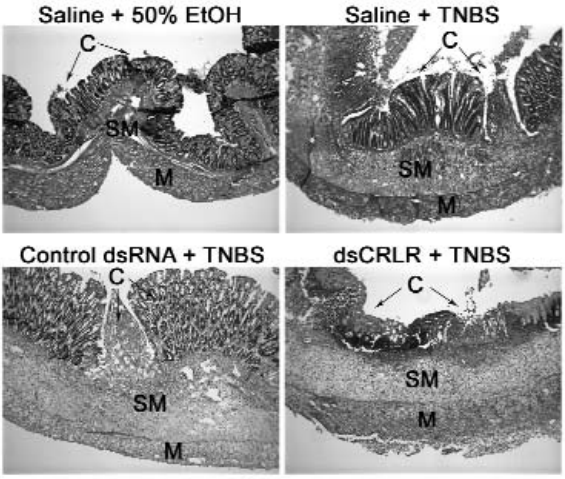

Figure 1. TNBS induces inflammation as evidenced by tissue edema, crypt (C) destruction, neutrophil infiltration and thickening of the submucosal (SM) and muscular (M) layers. Inhibition of CRLR exacerbates destruction, with absence of mucosal crypts, and altered submucosal and muscular architecture. Magnification: 5x.

Notes:

P13 A COMPARISON OF CARDIAC MRI AND COMPUTED TOMOGRAPHY WITH ECHOCARDIOGRAM IN EVALUATION OF PATIENTS WITH PECTUS EXCAVATUM

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Purpose:
A combination of computed tomography scanning and echocardiography have been used to grade the severity of the chest wall deformity and evaluate cardiac compression and valvular dysfunction in patients with Pectus Excavatum. Cardiac Magnetic Resonance Imaging (MRI) is being increasingly employed in the adult population and is the imaging modality of choice for evaluating central vasculature and paracardiac masses. Cardiac MRI has not been evaluated as an imaging modality in patients with Pectus Excavatum.

Methods:
Ten patients with pectus excavatum have undergone evaluation with cardiac MRI. The first three patients in our series underwent both cardiac MRI and Thoracic CT scan with Echocardiography as part of our routine work up. Due to superior results from cardiac MRI, we have not utilized CT Scan and echocardiography for the last seven patients. We compared functional and physical data obtained from these three studies in our first three patients.

Results:
Cardiac MRI is as reliable as Thoracic CT Scan in calculating the Pectus index. Cardiac MRI is superior to Echocardiography for assessing both right and left ventricular volumes throughout the cardiac cycle as well as assessing ventricular function. Cardiac MRI is superior to Echocardiography in assessing right ventricular compression secondary to the sternal depression. It also provides accurate measurement of ventricular stroke volume as well as volume of valvular regurgitation.

Conclusions:
Our experience shows cardiac MRI as being superior to CT scan combined with echocardiogram. This new imaging modality is able to quantitate mechanical cardiac dysfunction secondary to the Pectus Excavatum defect as well as provide dynamic images of improved quality compared to traditional cardiac echocardiography. Cardiac MRI has the advantage of subjecting the patient with Pectus Excavatum to one test, avoiding potentially harmful ionizing radiation, and provides more information with respect to global cardiac function.

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P14 CONSISTENT AND DISTINCT BIOCHEMICAL SIGNATURES FOR NORMAL AND CANCEROUS TISSUE WITH NIR RAMAN SPECTROSCOPY

Gulay K. Serhatkulu[1], Houbei Dai[1], Rachel Weber[1], Alex Cao[1], Abhilash Pandya[1], Jagdish Thakur[1], Carl Freeman[1], Ratna Naik1, Vaman Naik[2], Gregory Auner[1], Raja Rabah[3], Janet Poulik[3], Fazlul Sarkar[4], Michael Klein, M.D.[3]

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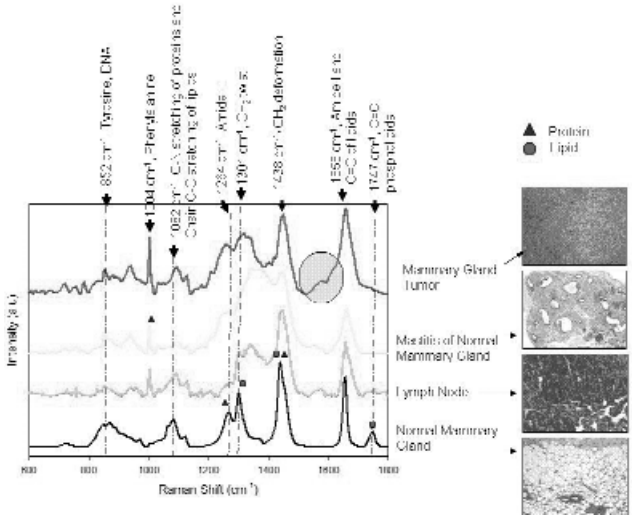
Raman Spectroscopy provides information about the molecular structures and has been widely used for the past several decades in non-destructive chemical analyses. In recent years there has been a remarkable increase in the application of Raman spectroscopy to the field of medicine. In Raman spectroscopy, a sample is irradiated with laser light, resulting in light scattering. The majority of scattered light has unchanged frequencies (Rayleigh line) whereas the rest is shifted in frequency (Raman Effect). The frequency shifts can be analyzed and presented as a Raman spectrum. Normal and neoplastic tissues have distinct biochemical features which are manifestations of unique biological processes. In this study, application of Raman spectroscopy in classification of normal and cancerous tissues was investigated. We will discuss three different models; i) mouse model for a mammary gland tumor differentiation ii) mouse model for pancreatic tumor differentiation and iii) children tumors such as lymphomas, neuroblastoma and kidney tumors. Each model will be discussed by comparing the normal tissue spectra and cancerous tissue spectra as well as statistical analysis methods such as one way analysis of variances, PCA and DFA to indicate the statistical significance. Our results revealed that NIR Raman spectroscopy can provide characteristic spectral signatures that can readily differentiate tumors from normal tissues.

In Figure 1 it showed that this technique is extremely sensitive to any pathologic alteration in the mammary gland tissue and was able to recognize specific spectral patterns for lymph nodes and mastitis to be differentiated from normal mammary glands as well as tumors. We will also discuss the future use of this technique as an *in vivo* surgical tool.



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P15 SIMULATION MODELING OF NORMAL AND ABNORMAL DEVELOPMENT OF THE MAMMALIAN DIAPHRAGM

Jason C. Fisher, M.D., Lawrence Bodenstein, M.D.
Morgan Stanley Children's Hospital of New York Presbyterian, Columbia University Medical Center, Department of Pediatric Surgery, New York, NY, U.S.A.

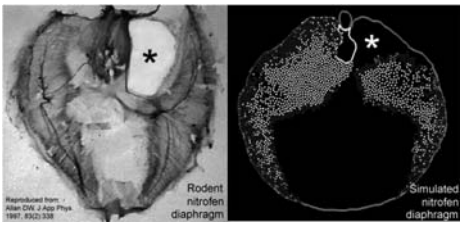
Purpose:
A true understanding of the etiology of posterolateral congenital diaphragmatic hernia (CDH) will require elucidation of the detailed embryology underlying normal and abnormal diaphragm development. We seek to investigate this morphogenesis using a novel cell-based computer model, and specifically interrogate historically accepted concepts and recent experimental results.

Background:
Traditional embryology describes contributions to the diaphragm from the septum transversum, pleuroperitoneal folds (PPF), esophageal mesentery, and body wall; however, recent animal studies suggest the folds are the major, if not sole, source of the muscular diaphragm. Posterior defects in the PPF, identified in a nitrofen CDH model, may be natural precursors to later and larger CDH defects.

Methods:
Diaphragmatic structures were digitized from transverse serial sections of paraffin embedded mouse embryos at E11.5 and E13. Using specially designed software (*Nudge*++™), structural boundaries and simulated cells were combined in a computer simulation. Cells were assigned putative behavioral programs that were progressively modified to produce a diaphragm consistent with anatomy observed in animal models.

Results:
Homology between the simulation and anatomic observations occurs when: (a) cell mitoses are restricted to the edge of growing tissue; (b) cells near the chest wall remain mitotically active; (c) mitotically active non-edge cells migrate toward the chest wall; and (d) anterior-posterior cell position within the PPF biases the direction of cell movement. With the PPF as the sole source of cells, a nitrofen rodent-like postero-*medial* CDH is formed (*Figure*, *); this contrasts the postero-*lateral* defect found in humans.

Conclusions:
(1) PPF-only morphogenesis may occur in rodents, but is unlikely in humans. (2) Features of human diaphragm development suggest additional cellular contributions from the esophageal mesentery and body wall, as envisioned by earlier workers. (3) Simultaneous development of a large posterior defect and a normal anterior diaphragm implies distinct developmental programs.



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P16 DEVELOPMENT OF A STANDARDIZED INTERACTIVE MULTIMEDIA-BASED PEDIATRIC SURGICAL REVIEW COURSE

Charles L. Snyder, M.D., Daniel J. Ostlie, M.D., George W. Holcomb, III, M.D.
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Purpose:
The evolution of surgery and its subspecialties has been marked by a gradual and inexorable increase in bureaucratic and governmental oversight and regulation. The process of speciality and subspecialty certification and recertification, the ABSITE exams, and CME and ethics credits have been part of trend. Currently, consideration is being given to increasing the frequency of the recertification cycle for general surgery. There are no currently available review texts/online materials for certification or recertification in pediatric surgery. We have developed such a course.

Methods:
Using Macromedia Flash MX, Dreamweaver MX, Authorware 6.5, and Robo Demo 5.0 software, we created a CD-based or online pediatric surgical review. There are 15 modules, each containing multiple topics; each topic has a variable number (usually ranging from 20 - 100) of questions, answers, and explanations. The program keeps score of the results (SCORM or AICC), and they can be sent to an email address. Pictures (JPEG, BMP), audiofiles, and videoclips can be used for questions or explanations as well as text.

Results:
We reviewed three of the recently published pediatric surgical textbooks, and created a series of questions, answers, and explanations. These were then transferred to XML format, and an interactive program created as described above. Over 3,000 questions have been entered into the program.

Conclusions:
This program allows a multimedia-based interactive scorable review which can be tailored to medical students, surgical residents/fellows, and pediatric surgical staff. This "P-Sesap" allows for long-needed standarized review and self-assessment in pediatric surgery.

Notes:

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P17 TISSUE ENGINEERED GAS EXCHANGE DEVICE USING MICROELECTRO MECHANICAL SYSTEMS TECHNOLOGY

Jennifer Anderson, M.D.[1], Irina Pomerantseva, M.D., Ph.D.[1], Katherine Kulig, B.A.[1], Jeffery T. Borenstein, Ph.D.[2], Eli Weinberg, MS[2], Mohammad R. Kaazempur-Mofrad, Ph.D.[3], Joseph P. Vacanti, M.D.[1]

[1]Massachusetts General Hospital, Boston, MA, U.S.A., [2]Draper Laboratory, Cambridge, MA, U.S.A., [3]University of California, Berkley, Berkley, CA, U.S.A.

Purpose:

Cystic adenomatoid malformation and diaphragmatic hernia are types of lung disease encountered in infants. Ventilation strategies for premature neonates can result in chronic lung disease. Tissue engineering offers a potential solution via live, functional engineered organ. A tissue engineered gas exchanger could provide oxygen and remove carbon dioxide in full-term infants with lung disease, and allow time for lung development in premature neonates. Fabricating large three-dimensional tissue has inherent challenges due to limited oxygen and nutrient diffusion. A novel approach has been implemented in our laboratory. A combination of MicroElectro Mechanical Systems technology and computational models of a fractal branching system enables construction of a scaffold of an intrinsic microvascular network from biocompatible polymer. This preformed vascular network can be seeded with cells and ultimately sustain growth and function of complex tissues.

Methods:

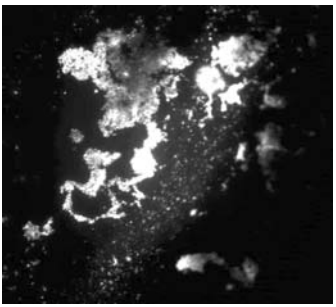
The prototype device consisted of vascular and parenchymal chambers, separated by a membrane. A network of vascular channels was etched onto a silicon wafer. These silicon wafers were used for replica molding of poly(dimethyl siloxane) (PDMS), which served as the polymer scaffold. The parenchymal chamber was seeded with a mouse alveolar type II cell line (MLE-12). Culture medium flowed through the vascular chamber at 0.5 ml/hour via syringe pump. The cells were assessed for viability using Live/Dead Assay Kit at 65 hours post-seeding.

Results:

After 65 hours, viability of the MLE-12 cells was tested within the devices (Figure 1). There were large areas of viable cells (green staining). The viability was similar to control cells seeded and incubated for 72 hours on a PDMS coated 12-well plate (data not shown).

Conclusions:

We have demonstrated viability of MLE-12 cells in a novel device designed to overcome oxygen and nutrient diffusion limitations. This is an important initial step in the development of a tissue-engineered gas exchange unit.



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P18 TISSUE ENGINEERING APPROACHES TOWARDS POTENTIAL LUNG AUGMENTATION IN PULMONARY HYPOPLASIA

Christine Finck[1], Mark Mondrinos[2], Honesto Poblete[1], Sirma Koutzaki[1], Peter Lelkes, Ph.D.[2]
[1]St. Christopher's Hospital for Children, Philadelphia, PA, U.S.A., [2]Drexel University Department of Bioengineering, Philadelphia, PA, U.S.A.

Pulmonary hypoplasia (PH) accounts for 15-20% of neonatal deaths annually. We have an established method of culturing murine fetal pulmonary cells (FPC) with the goal of encouraging 3-dimensional (3D) growth for lung augmentation in PH. Initially, FPC cultured in 2D utilizing different matrices demonstrated that Matrigel was superior in supporting epithelial and type II alveolar cell (AE2) growth. In addition, growth of FPC in 3D utilizing Matrigel hydrogel supported the formation of alveolar forming units (AFU). Presently, we investigated implantation of Matrigel plugs seeded with FPC and bFGF sponges *in vivo*. Our hypothesis was that AFU branching morphogenesis and vascular invasion will occur in parallel, reminiscent of embryonic development. We further hypothesized that enhanced vascularization would occur utilizing angiogenic growth factor bFGF.

Isolated C57/BL fetal lung cells (d18) were admixed with Matrigel and injected subcutaneously in mice (IACUC#30511). bFGF soaked sponges were implanted inside the Matrigel upon solidification. Control experiments consisted of Matrigel ± bFGF sponges. Tail vein injection with FITC-Dextran allowed visualization of vascular networks within the plugs. Results were evaluated utilizing H&E staining and IHC for keratin, lectin, von Willebrand Factor, and surfactant protein C. The total area of lectin staining provided a quantitative measure of vascularization. In addition, inflammation was characterized by IHC utilizing anti-CD3 and anti-macrophage antibodies.

No significant invasion of cells or blood vessels was observed in control plugs. By contrast, Matrigel plugs containing FPC exhibited numerous AFUs and ample vascularization. The addition of bFGF increased vascularization. Furthermore, the AFUs were seen in close proximity to tubular endothelial structures, reminiscent of a capillary-endothelial network.

These data suggest the feasibility of an *in vivo* model as a "natural bioreactor". The ability to generate 3D structures that morphologically resemble the alveolar-capillary network is a first step in developing functional distal pulmonary tissue constructs that can be used for pulmonary tissue augmentation.

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P19 ISOLATION AND SEEDING OF AUTOLOGOUS OVINE BONE MARROW DERIVED VASCULAR PROGENITOR CELLS ON A BIODEGRADABLE SCAFFOLD FOR USE AS A TISSUE ENGINEERED VASCULAR CONDUIT

Jason D. Roh, BA, Matthew P. Brennan, M.D., Peter M. Fong, Ph.D., Reynold Lopez-Soler, M.D., Ph.D., George Tellides, M.D., Ph.D., Alan Dardik, M.D., Ph.D., Christopher K. Breuer, M.D.

Yale University School of Medicine, New Haven, CT, U.S.A.

Purpose:

The repair of congenital cardiovascular and vascular defects is hindered by the inability of implanted grafts to grow. Tissue engineered vascular grafts (TEVG) may provide a more viable option given their potential for repair, remodeling, and growth. We have optimized techniques for creating a functional TEVG with a confluent internal cellular layer by isolating and seeding bone marrow derived vascular progenitor cells (BMVPC) on a biodegradable scaffold and implanting the resulting constructs as IVC interposition replacement grafts in a juvenile lamb model.

Methods:

BMVPCs were isolated from ovine bone marrow using a histopaque gradient and further selected by culture in endothelial growth media. Cell population included endothelial cells, evidenced by FACS after DiO-Ac-LDL labeling, and myofibroblasts, confirmed by smooth muscle actin (SMA) and calponin immunofluorescent staining. BMVPCs in varying concentrations ($2.5x10^5$, $5x10^5$, $1x10^6$, $2x10^6$, $5x10^6$ cells/cm^2) were statically seeded on PGA/P(CL/LA) [polyglycolic acid/ 50:50 L-lactide and e-caprolactone] tubular scaffolds either on day of isolation or after seven days of cell culture. *In vitro* cell growth on the scaffold was followed for 14 days by light microscopy and SEM. Both autologously seeded and unseeded scaffolds were implanted as intrathoracic IVC interposition grafts in a juvenile lamb model. Grafts were immediately harvested and analyzed using SEM.

Results:

BMVPCs were spindle-shaped and stained positive for SMA and calponin. Two-point-eight percent were positive for DiO-Ac-LDL. The scaffold was approximately 60% porous (pores: 20-200μm). Both seeded and unseeded scaffolds were impermeable to blood flow and remained patent *in vivo*. Concentration of $2x10^6$ cells/cm^2 and seven-day post-seeding incubation were optimal for creating a confluent cellular layer on the scaffold.

Conclusions:

We have optimized techniques necessary for constructing a functional TEVG possessing a confluent internal cellular layer using 14 days of *in vitro* culture. Current studies evaluating the growth potential of these TEVG are ongoing.

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P20 A MULTILAYER TISSUE ENGINEERED LIVER DEVICE WI TH AN INTRINSIC MICROFABRICATED VASCULAR NETWORK

Katayun Irani, M.D.[1], Katherine Kulig, BA[1], Eleanor Pritchard[2], Kimberly Bonner, BA[1], Brian Orrick, MBA[2], Kimberly Morgan[1], Mohammed Kaazempur-Mofrad, Ph.D.[3], Eli Weinberg, MS[3], Jeffrey Borenstein, Ph.D.[2], Joseph P. Vacanti, M.D.[4]

[1]Massachusetts General Hospital For Children, Boston, MA, U.S.A., [2]Charles Stark Draper Laboratories, Cambridge, MA, U.S.A., [3]Massachusetts Institute of Technology, Cambridge, MA, U.S.A., [4]Massachusetts General Hospital for Children, Boston, MA, U.S.A.

Purpose:
The large hepatic cell mass required to replace liver function, and the requirement of facile diffusion of nutrients and oxygen across this large mass makes hepatic tissue engineering challenging. We have enhanced diffusion of nutrients and oxygen to cells by fabricating a device with an intrinsic microvascular network adjacent to a cellular compartment, which has demonstrated cell survival and proliferation. We have expanded the device in three-dimensional space to house a larger number of hepatocytes. Our goal is to expand the device enough to house a large enough hepatic cell mass to eventually replace liver function.

Methods:
Using MicroElectroMechanical Systems technology, we fabricated a dual chamber device made of poly (dimethylsiloxane), with a poly(ethersulfone) membrane separating the chambers. The cellular capacity of the original design was expanded by increasing the cellular compartment size and by stacking multiple interconnected layers. Human and rat immortalized hepatic cell lines were seeded into the chambers. Stacked and single layer devices were perfused with serum-free medium, and compared to static well plates. The effluent was assayed daily for albumin and α-fetoprotein. Cell survival, morphology, and proliferation were assessed on day seven at experiment's termination.

Results:
Viability staining demonstrated similar survival and normal morphology in the stacked, single layer, and static well plates. DNA quantification studies demonstrated a 25% increased cell mass over seven days in the stacked devices. Enhanced albumin and α-fetoprotein concentrations were observed over seven days relative to the control well plates.

Conclusion:
By stacking our devices into layers, a larger hepatic cell mass was supported, a step towards a replacement for liver function in patients with end stage liver disease.

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P21 MECHANICALLY-INDUCED ENTEROGENESIS: IDENTIFCATION OF MECHANISMS OF ACTION

Mohamed I. El-sawaf, M.D., Hua Yang, Ph.D., M.D., Ariel U. Spencer, M.D., Daniel H. Teitelbaum, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

Purpose:
Distractive forces can induce growth of several organs. We have recently developed an implantable hydraulically-controlled device that can mechanically lengthen the small intestine of a pig. We have been able to induce length gain of 2.5-fold in as little as 14 days. This work aimed to investigate the potential mechanisms that might lead to this mechanically-induced enterogenesis.

Methods:
Our group targeted known mechanotransductive pathways which are responsible for distraction-induced growth in other organs, as well as known enterocyte proliferation factors. m-RNA expression using PCR was performed on mucosal scrapings from Lengthened and Control segments (n=8) of bowel, and results were standardized to the expression of GADPH. Differential protein analysis of factors expressed in the Lengthened versus Control group was performed with 2-D gel electrophoresis. A secondary mass spectrogram analysis was performed of 6 protein spots which were only expressed in the Lengthened group. Two tailed student's *t*-test was used to analyze the results at a significance level of 0.05.

Results:
PCR results are shown in the Table (*P<0.05).

Group	Proglucagon	Alpha-E integrin	C-Src	Wnt-5a	E-Cadherin	Hedgehog (Indian)
Control	0.42 ±0.18	0.58 ±0.07	0.28 ±0.01	0.08 ±0.02	0.27 ±0.02	0.28 ±0.07
Lengthened	0.55 ±0.22	0.58 ±0.20	0.43 ±0.10*	0.16 ±0.04*	0.32 ±0.04	0.29 ±0.14

Differential protein analysis showed expansion of desmin (a smooth muscle precursor) and beta-actin-profilin complex in the Lengthened group; interestingly both proteins are required for embryonic formation of crypt-villus structures.

Conclusions:
The PCR results suggest that both an integrin-mediated (C-Src) and Wnt-signaling (Wnt-5a) pathways may contribute to distraction-induced enterogenesis in this pig model. The finding of increased desmin and beta-actin-profilin complex also support a Wnt-mediated signaling pathway, and suggest such growth uses a similar mechanism as that utilized during embryonic formation of the intestine.

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P22 MESENCHYMAL STEM CELLS FROM TWO DISTINCT COMPARTMENTS HAVE ENHANCED OSTEOGENESIS IN RESPONSE TO A P53 TUMOR SUPPRESSOR MUTATION

Monika Tataria, M.D., Natalina Quarto, Ph.D., Karl G. Sylvester, M.D.
Stanford University, Stanford, CA, U.S.A.

Purpose:
Osteosarcomas occur in children during the growth spurt years, and are thought to arise from transformation of a mesenchymal stem cell (MSC) that resides in the growth plate of long bones. Absence of the p53 tumor suppressor gene is implicated in the origin and progression of osteosarcoma The objective of this study was to determine whether a p53 null mutation effects osteogenic differentiation in a cell autonomous manner by comparing bone marrow (BMSC) and adipose (AMSC) derived MSC differentiation. We hypothesized that a p53 null mutation would enhance osteogenic differentiation of MSCs from both compartments.

Methods:
Clonal BMSC and AMSC populations were derived from wild type (WT) and $p53^{-/-}$ mice, and were compared in osteogenic culture via alkaline phosphatase and alizarin red staining and quantification. The marker genes of osteogenic differentiation cbfa1, osteopontin, and osteocalcin were assessed by QRT-PCR during differentiation.

Results:
BMSC and AMSC from $p53^{-/-}$ mice under osteogenic conditions both showed earlier and increased alkaline phosphatase staining compared to WT cells. Bone nodule formation (alizarin red quantification) was accelerated and enhanced in both groups of $p53^{-/-}$ cells. The early and intermediate osteogenic markers, cbfa1 and osteopontin, were upregulated in both $p53^{-/-}$ MSC populations compared to WT cells during osteogenesis. The terminal osteogenic marker gene osteocalcin was paradoxically lower in both types of $p53^{-/-}$ MSCs indicating a lack of terminal differentiation.

Conclusions:
MSCs from two distinct compartments, bone marrow and adipose tissue, have an osteosarcoma phenotype as a result of the p53 tumor suppressor gene mutation. These findings suggest that the effect on osteogenesis of loss of the tumor suppressor gene p53 is a cell autonomous phenomenon, with the same effects observed on the biology of differentiation in distinct osteogenic capable cells. These findings have important implications for understanding the derivation of osteosarcoma from MSCs.

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P23 GASTRIC ELECTRICAL STIMULATION FOR ADOLESCENTS WITH INTRACTABLE NAUSEA AND GASTROPARESIS

Saleem Islam, M.D., John R. Gosche, M.D., Ph.D., Jo White-Ashmead, M.D., Laura R. Vick, M.D., Thomas R. Abel, M.D.
University of Mississippi Medical Center, Jackson, MS, U.S.A.

Purpose:
Gastric electrical stimulation (GES) has been performed in adults as a treatment for refractory nausea and vomiting, in patients who have failed medical treatment. It has not been systematically applied to individuals less than 18 years old with this problem.

Methods:
Six patients, one male, five female with chronic nausea and vomiting with a mean age of 15 years (range 13-18), were evaluated with gastric emptying studies and cutaneous electrogastrograms (EGG) for temporary GES. All patients had idiopathic gastroparesis. Five patients subsequently underwent placement of a permanent GES device - four were done laparoscopically and one was open. Each patient had an intra operative EGG and in three patients, seromuscular biopsies from the stomach or jejunum were obtained as well. Symptoms were recorded at baseline, after temporary pacing and then after permanent pacing using a Likert scale (0-4 for each symptom with a total of 5 symptoms). Statistical analysis was performed using a paired student's t test and a value of <0.05 was considered significant.

Results:
At baseline, all patients were symptomatic and most had delayed solid gastric emptying and abnormal EGG. As a group, there was a significant improvement in nausea (3.4 ± 0.4 to 1.7 ± 0.3, $p=0.005$), and combined symptoms score (11.3 ± 2.0 to 5.0 ± 1.5, $p=0.02$). Gastric emptying and EGG values also improved. Biopsy was abnormal in two of three patients, showing diminished and abnormal Cajal cells in the two patients who had less improvement long term. Follow up ranged from 1- 20 months, with an average of nine months.

Conclusions:
GES can be successfully applied to adolescents with intractable nausea and gastroparesis symptoms. The role of the cells of Cajal in enteric motility needs investigation. Long term efficacy of this therapy in children needs to be established.

Notes:

P24 OVARIAN SEROUS CYSTADENOCARCINOMA SIDE POPULATION EXHIBITS STEM CELL CHARACTERISTICS AND IS INHIBITED BY MULLERIAN INHIBITING SUBSTANCE *IN VITRO*

Paul Szotek, M.D.[1], Rafael Pieretti-Vanmarcke, M.D.[1], Yong Zhang, M.D.[1], Denise Connolly, Ph.D.[2], David Dombkowski[3], Frederic Preffer, M.D.[3], David T. MacLaughlin, Ph.D.[1], Peter T. Masiakos, M.D.[1], Patricia K. Donahoe, M.D.[1]

[1]Pediatric Surgical Research Laboratories, MassGeneral Hospital for Children, Harvard Medical School, Boston, MA, U.S.A., [2]Fox Chase Cancer Center, Philadelphia, PA, U.S.A., [3]Flow Cytometry Laboratory, Department of Pathlology, Massachusetts General Hospital, Boston, MA, U.S.A.

Purpose:

Ninety percent of ovarian neoplasms arise from the epithelial surface of the ovary. A dismal 30% 5-year survival may reflect progressive resistance of a stem cell-like tumor population to traditional chemotherapy. Mullerian Inhibiting Substance (MIS), a fetal glycoprotein that causes Mullerian Duct regression in male embryos, has been cloned, purified, and shown to cause *in vitro* and *in vivo* growth inhibition of mouse and human ovarian cancer cell lines. The growth characteristics of a mouse ovarian cancer stem cell-like side population (SP) were established and response of this population to MIS studied.

Methods:

Mouse serous cystadenocarcinoma (MOVCAR 7) cells expressing MIS type II receptor (MISRII) driven SV40 T-antigen, were sorted by flow cytometry for Hoechst 33342 efflux and recovered for subsequent MTT assays. The presence of MISRII receptor protein was confirmed with immunoblotting and immunohistochemistry, while MISRII, type I receptor, and Smad 1/5/8 mRNA were detected by RT-PCR. SP properties were confirmed by expression of ABCG2 transporter mRNA. Immunohistochemistry and flow cytometry were used to evaluate a panel of conventional stem cell markers such as c-Kit & Sca-1.

Results:

MOVCAR 7 cells express MISRII, MISRI, Smad 1/5/8, are c-Kit+/SCA-1- (n=3), and exhibit stem cell like Hoechst 33342 exclusion in a SP which enriches with serial sorting (2% to 26%) of the SP cells (Figure 1-A,B,C). MIS inhibited growth of both the SP and non-SP MOVCAR cells (n=3) and continued to inhibit the SP with each successive sort (Figure 1-D, E). Non-SP cells were recovered but died after one passage (n=3).

Conclusions:

MOVCAR cells have a side population with stem cell characteristics that sustain cancer cell growth, but continue to respond to MIS *in vitro* via a transduction pathway previously established in the embryonic Mullerian duct. Tumorcidal chemotherapy combined with stem cell modulators such as MIS should be considered in future treatment regimens.

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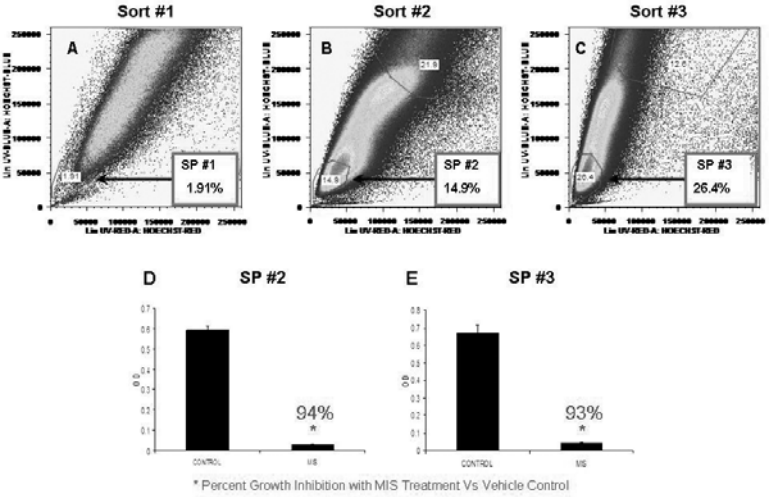
Figure 1



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P25 WNT/β-CATENIN SIGNALING IN HEPATOBLASTOMA MICE MODEL

Masashi Kurobe, M.D.[1], Shelly Beer[2], Deen W. Felsher, M.D.[2], Karl G. Sylvester, M.D.[1]
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Purpose:
The Wnt/β-catenin pathway regulates early hepatic development and has been implicated in human hepatocellular carcinoma (up to 40%) and hepatoblastoma (HB) (70%). A deeper understanding of the mechanisms by which dysregulated Wnt/β-catenin signaling promotes the embryonal tumor hepatoblastoma may provide insight into the derivation of this high mortality liver tumor and may suggest novel treatment strategies.

Methods:
We utilized a tetracycline regulatory system (Tet system) to generate transgenic mice in which the human proto-oncogene c-MYC could be conditionally over-expressed in murine hepatocytes. The MYC oncogene was activated during mouse embryonic liver development. After the onset of liver neoplasia in the mouse neonatal period, liver tumors were harvested for qualitative histological assessment and a quantitative assessment of the Wnt/β-catenin signal transduction pathway by Western blot and quantitative RT-PCR.

Results:
Mice in which MYC was activated in embryonic hepatoblasts succumbed to neoplasia within 10d of birth. Grossly, liver architecture was preserved, but marked hepatomegaly with HB histology was observed. Immunohistochemistry for β-catenin and glutamine synthetase, a known target gene of β-catenin in the liver, showed widespread and uniform expression in each of the tumors compared with control newborn mouse liver. The Wnt2b and Wnt16 genes were induced in liver tumors while the Wnt1, Wnt2, Wnt5a, Wnt5b, Wnt6, Wnt7a, Wnt7b, Wnt9a, Wnt9b, Wnt10a and Wnt11 genes had decreased expression in liver tumors compared to control newborn liver. A striking decrease was recognized in Wnt5a expression, which is thought to function as a tumor suppressor.

Conclusions:
The Wnt/β-catenin signal transduction pathway promotes hepatoblast proliferation in embryonic liver in response to MYC oncogene activation during HB oncogenesis. The Wnt/β-catenin signaling may be a target pathway for therapeutic intervention in the treatment of HB.

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P26 SUCCESSFUL LIVER TRANSPLANT FOR UNRESECTABLE HEPATOBLASTOMA, EARLY REFERRAL IS THE KEY

Adela T. Casas-Melley, M.D., Jeffrey Malatack, M.D., Deborah Consolini, M.D., Keith J. Mann, M.D., Christopher Rabb, M.D., Louise Flynn, APN, Pamela Woolfrey, MSN, Jerome Menendez, RN, Stephen Dunn, M.D.
AI duPont Hospital for Children, Wilmington, DE, U.S.A.

Purpose:
Treatment of children with stage III and IV hepatoblastoma has shown little improvement with 5-year survival rates of 64% and 25% respectively. A timely and organized treatment program including pre-operative chemotherapy combined with living donor liver transplant and post-operative chemotherapy has been employed seeking improved long term survival in stage III and IV cases.

Methods:
A retrospective review of eight patients treated with stage III and IV hepatoblastoma unresectable by conventional resection requiring complete hepatectomy and transplantation. Approval was obtained from our institutional review board.

Results:
Since August of 2001 we have treated six patients with unresectable stage III and two patients with initial stage IV hepatoblastoma. These patients (age 16 months-9 years) had all received extensive chemotherapy or prior resection. None had tumor documented outside of the liver at the time of transplant. All underwent hepatectomy including vena cava resection, in selected cases, with living donor orthotopic liver transplantation. All patients had at least two cycles of post-operative chemotherapy. Six of eight patients are alive and well with normalized alpha fetoprotein (AFP) levels. There were two late deaths from recurrent disease.

Conclusions:
Complete hepatectomy with living donor liver transplantation provides optimal surgical treatment in unresectable stage III and initial stage IV disease confined to the liver at resection. This small series indicates that children tolerate complete hepatectomy, transplantation and post-operative chemotherapy well. Referral to a transplant center during the first three cycles of chemotherapy is imperative for improved results from this approach.

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P27 WILMS' TUMOR PATHOGENESIS IS FACILITATED BY SERINE PHOSPHORYLATION OF STAT1

Olga Timofeeva, Ph.D.[1], Harold N. Lovvorn, III, M.D.[2], Shaun Opperman[2], Alan O. Perantoni, Ph.D.[1]

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Purpose:
Wilms' tumors arise from arrested differentiation of the metanephric mesenchyme. Normal metanephric development is dependent partly on Signal Transducers and Activators of Transcription (STAT) signaling. Since constitutively active tyrosine-phosphorylated STAT proteins also play a pivotal role in several human cancers, we studied STAT activity in the embryonal Wilms' tumor (WT).

Methods:
STAT1 tyrosine and serine phosphorylation patterns were characterized by immunoblotting in two WT cell lines and 21 primary WT's. To determine its effect upon tumorigenic behavior, we constitutively misexpressed two mutant STAT1 constructs (wild-type & dominant-negative STAT1) and an empty control vector by stable transfection of a human WT cell line. Effects on anchorage-independent and xenograft growth were determined for each altered cell line ($n \geq 10$ SCID mice per line). Transfected cells were assessed for resistance to apoptosis under conditions of growth stress using annexin-V and PARP cleavage. Finally, the implicated anti-apoptotic signaling factors, mcl-1 and bcl-2, were characterized in WT cells by immunoblotting and modulated by RNAi.

Results:
STAT1 was constitutively phosphorylated on serine 727, without tyrosine phosphorylation, in 2/2 WT cell lines and 19/21 primary WT's, but not in normal kidney tissue from eight WT-bearing patients. Transfection of human WT cells with a dominant-negative mutant, STAT1-S727A, reduced colony formation in soft agar by more than 90% and perturbed tumor development and volume in immunodeficient mice. In cell culture, the dominant-negative mutant induced apoptosis under conditions of growth stress, and siRNA for STAT1 specifically caused a loss of mcl-1 expression and induced apoptosis. Furthermore, siRNA for mcl-1 induced apoptosis, while siRNA for bcl-2 did not. Finally, all primary WT's expressed mcl-1.

Conclusion:
Our results highlight a novel role for serine-phosphorylated STAT1 in mediating Wilms' tumor cell survival and behavior through mcl-1 activation and indicate that either factor may function as a target for anticancer drug discovery.

Notes:

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P28 WILMS' TUMOR AND ALTERED EXPRESSION OF THE TRANSCRIPTIONAL REGULATOR, CITED1

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Purpose:

Wilms' tumors arise from alterations in the coordinated differentiation of renal progenitor cells. Cited1 is a transcriptional co-factor that has been shown to regulate in part the mesenchymal-to-epithelial conversion of the developing kidney. During early nephrogenesis, Cited1 is abundantly expressed in the cap mesenchyme overlying the ureteric bud, is down-regulated as these progenitor cells differentiate to form epithelial aggregates, and is absent in organized embryonic epithelia. Further, Cited1 is localized predominantly to the cytoplasm of mesenchyme during nephronic patterning. Cited1 may therefore coordinate early phases of cellular organization and differentiation associated with epithelial conversion. We hypothesized that Cited1 expression is altered in Wilms' tumorigenesis.

Methods:

To characterize expression of Cited1 in Wilms' tumor, we studied 20 favorable histology specimens (n=5 for each Stage, I-IV) provided by the Children's Oncology Group. Quantitative polymerase chain reaction, Western blot, and immunohistochemistry were used for detection of Cited1. Immunofluorescence was used to characterize its sub-cellular localization. Comparison was made to embryonic kidneys.

Results:

Cited1 was detected by each method in all 20 Wilms' tumors. By immunohistochemistry, Cited1 expression was intense in the blastemal, and absent in the stromal, compartments. Distinct from the normal developing kidney, Cited1 was expressed clearly in the nuclear compartment of blastema, and further, showed persistent intermediate expression restricted to the cytoplasm of disorganized epithelial structures.

Conclusions:

The transcriptional regulator, Cited1, is consistently detected in all stages of favorable histology Wilms' tumors. When compared with normal nephrogenesis, Cited1 expression abnormally persists in, and robustly shifts to the nucleus of, malignant blastema, and further, lingers in disorganized epithelial structures. These findings indicate that Cited1 is a marker of primitive blastemal elements in Wilms' tumor, and suggest that altered sub-cellular localization of Cited1 may regulate the persistence, differentiation or function of epithelial progenitor cells in Wilms' tumorigenesis.

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P29 PEDIATRIC FAMILIAL MEDULLARY THYROID CARCINOMA (FMTC) STAGE IS INDEPENDANT OF MUTATION SITE: BUT MUTATION AT C620 IS ASSOCIATED WITH HIRSCHSPRUNG'S DISEASE

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Ste-Justine Hospital, Montreal, PQ, Canada

Purpose:
Early genetic screening in children of parents with MEN2 syndrome has led to prophylactic total thyroidectomy. We reviewed our experience to determine the incidence of medullary carcinoma with respect to age at surgery and the location of the mutation.

Methods:
A retrospective chart review from 1996-2005 revealed 17 children with genetic screening of MEN syndrome or FMTC who underwent a prophylactic total thyroidectomy, central neck dissection with preservation of the parathyroid glands.

Results:
Seventeen patients, nine boys and eight girls, had a mean age of 9.1 years (range 3.5-15 years) at the time of surgery. Histologic findings, mutation level and age at surgery are summarized below. Four patients had previous diagnosis of Hirschsprung's disease.
Postoperatively, one child had transient hypocalcemia and there were no recurrent laryngeal nerve injuries. With a mean follow-up of 4.4 years (3 months-9 years). All patients are doing well and are free of MTC.

Conclusion:
There is no correlation between histologic findings and mean age at surgery. Hirschsprung's disease was found in 57% of patients with the Ret mutation at C620 level. Based on the age of the earliest cancer and the safety of total thyroidectomy, children should promptly undergo surgery after genetic screening or before five years of age. Patients with the C620 mutation should also have screening for Hirschsprung's disease.

Histologic findings according to mutation level and age at surgery

Histologic	#	Mean age (years)	Mutation Level	#	Associated Hirschsprung's disease
Normal thyroid tissue	3	6.3	C620	3	3
C-cell hyperplasia	11	9.7	C618 C620 C634 C768	1 3 4 3	- 1 - -
Medullary thyroid carcinoma (MTC)	3	7.3	C620 C634 C768	1 1 1	- - -
Total	17	9.1	C618 C620 C634 C768	1 7 5 4	- 4 - -

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P30 THE IMPACT OF EXTENT OF THYROIDECTOMY ON NON-METASTATIC, ENCAPSULATED, AND COMPLETELY RESECTED DIFFERENTIATED THYROID CARCINOMA IN PATIENTS <21 YEARS OF AGE AT DIAGNOSIS

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Purpose:
Differentiated thyroid carcinoma in young patients is associated with a good prognosis despite the presence of locally advanced disease and even distant parenchymal metastases at diagnosis. Previous analyses have shown that age at diagnosis and the presence of residual disease in the neck after thyroid resection are adverse prognostic factors for disease progression and the overall recurrence/progression rate in the literature is 35-40%. In this analysis we compared staging and treatment variables as well as outcome in patients undergoing either thyroid lobectomy, or subtotal or total thyroidectomy. This subset analysis included patients with encapsulated and completely resected primary tumors who did not have distant parenchymal metastases at diagnosis.

Methods:
These data were obtained from a previously published and anonymized thyroid cancer database compiled by the surgical committee of the former Children's Cancer Group. Kaplan Meier probability distributions were generated and compared using the log rank test.

Results:
Results are compiled and compared by the technique of thyroid resection in the table below (missing values eliminated): (SEE TABLE)
Also, the type of lymph node dissection, the distribution of histologic subtypes, and the frequency of recurrent nerve injury did not differ significantly between the techniques of thyroid resection.

Conclusions:
Patients with non-metastatic, encapsulated, completely resected differentiated thyroid carcinomas are a favorable prognosis subset. In this analysis subtotal or total thyroidectomy did not significantly improve progression-free survival compared to lobectomy and was associated with an increased risk of temporary or permanent hypocalcemia.

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	Lobectomy	Subtotal or Total Thyroidectomy	p <
N	36	40	
Median Age (range) (yrs.)	15.7 (0.5-20.8)	15.7 (5.0-20.3)	NS
Hx Cervical radiation	4/35	6/35	NS
Nodes +	19/36	29/40	NS
Size >2.5 cm	12/36	15/40	NS
I^{131} Treatment	4/29	10/38	NS
Thyroid suppression	26/36	36/40	NS
Post-operative Hypocalcemia	3/36	15/38	0.02
10-Year Progression-free survival (%)	91.0 ± 9.8	89.4 ± 11.6	NS

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P31 ELUCIDATION OF THE PATHWAY OF NERVE GROWTH FACTOR INDUCED APOPTOSIS IN THE NEUROBLASTOMA CELL LINE SK-N-SH

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Purpose:

We have previously reported the activation of p38 by nerve growth factor (NGF) in the NGF transfected neuroblastoma cell line (SK-N-SH/NGF). We hypothesize that this activation would lead to apoptosis in these cells through the downstream effectors of p38.

Methods:

Wild-type (SK-N-SH/WT) and NGF transfected (SK-N-SH/NGF) neuroblastoma cells were incubated with and without NGF (200ng/ml) for 0-24 hours. Whole cell lysates were then collected and Western immunoblots performed for ELK-1, STAT 1, ATF 2 and c-Jun, the known downstream targets of p38. Cells were also treated with or without NGF and DNA collected using the Suicide-Track DNA ladder isolation kit (Calbiochem) to assess for apoptosis.

Results:

SK-N-SH/NGF cells treated with NGF showed activation of ATF 2 and c-Jun but not STAT 1 or ELK-1 with the maximal effect after 1-hour incubation. NGF incubation of wild type cells did not activate any of the target proteins of p38. In addition, NGF induced apoptosis in the SK-N-SH/NGF cell line assessed by DNA laddering.

Conclusions:

Nerve growth factor can induced apoptosis in human neuroblastoma cells transfected with the NGF gene through the p38 pathway by activation of ATF 2 and c-Jun. We are currently testing if this effect may be utilized in treating neuroblastoma *in vivo*.

Notes:

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P32 *SALMONELLA TYPHIMURIUM* IMPROVES SURVIVAL IN NEUROBLASTOMA BY NK CELL PROLIFERATION AND THE INCREASE IN CYTOKINES IL-6 AND INF-γ

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Purpose:
Recent studies have demonstrated a reduction in neuroblastoma tumor burden in mice treated by oral gavage of *Salmonella typhimurium*. We sought to evaluate a survival benefit for mice treated with *Salmonella typhimurium* and to further characterize the cellular and cytokine response to its treatment.

Methods:
A murine model for retroperitoneal neuroblastoma was utilized in 6-8 week old female A/J mice. For survival studies, 14 days following injection mice (n=60) were orally gavaged with Saline (control), *Salmonella typhimurium* (Sal-NG), or *Salmonella typhimurium* with an IL-2 plasmid (Sal-pIL2). Mice were provided free access to food and water and the post-injection time (days) of their demise was recorded. In a separate set of experiments, neuroblastoma burdened mice were sacrificed 14 days post-treatment and peripheral blood analyzed by ELISA for the cytokines IL-2, IL-6, IL-12 and INF-γ. Lymphocytes where recovered from spleens of study mice and evaluated by flow cytometry. Statistical analysis utilizing Fisher's analysis and Student t-test was performed with $p<0.05$ designated as significant.

Results:
There is a statistically significant increase in survival of animals gavage fed Salmonella-IL2 when compared to animals fed Salmonella without the IL2 gene and controls. (Figure) In control, Sal-NG, and Sal-pIL2 mice, IL-6 (35.1, 130.25, and 478.71 pg/ml (control vs. Sal-pIL2 p=0.05)) and INF-γ (7.89, 98.2, 63.92 pg/ml (control vs. Sal-NG p=0.013)) were found to be statistically significant. There was no statistical difference found in IL-2 or IL-12 levels in any groups. Numbers of NK cells were found to be 1.846, 2.077 and 2.849 respectively (control vs. Sal-pIL2 p=0.0318). No statistical difference was noted in populations of CD4 or CD8 cells.

Conclusions:
Attenuated *Salmonella* species increase survival in mice injected with neuroblastoma. This process is mediated by an increase in natural killer cells and supported by an increase in the cytokines IL-6 and INF-γ.

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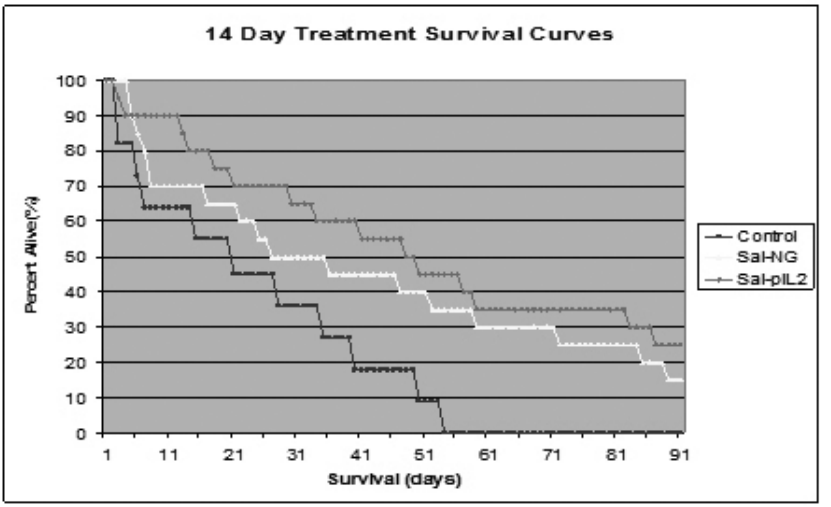
14 Day Treatment Survival Curves
Percent Alive(%)
100
90
80
70
60
50
40
30
20
10
0
1
11
21
31
41
51
61
71
81
91
Survival (days)
Control
Sal-NG
Sal-pIL2

Notes:

P33 NOVEL ACTION OF EPIDERMAL GROWTH FACTOR ON CASPASE-3 AND ITS POTENTIAL AS A CHEMOTHERAPEUTIC ADJUNCT FOR NEUROBLASTOMA

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Purpose:

We previously reported that epidermal growth factor (EGF) at 100ng/mL induced cleaved caspase-3 expression and apoptosis in neuroblastoma cells. Recent reports have described differential pro-caspase-3 expression in apoptosis. Based on these findings, we hypothesized that EGF induced increased pro-caspase-3 expression, thereby enhancing the cytotoxic potency of chemotherapeutic agents working via the apoptotic pathway.

Methods:

Wild-type (WT) and doxorubicin-resistant (Dox-R) SK-N-SH neuroblastoma cells were incubated with EGF at concentrations from 5-100ng/mL for 24 hours. Western immunoblots were performed with pro-caspase-3 antibody on whole-cell lysates collected to determine the EGF concentration that produced maximal pro-caspase-3 expression. Time course studies were performed after incubation with EGF (5ng/mL) for 1-3 days. RT-PCR, utilizing pro-caspase-3 primers was performed on both cell lines after EGF treatment to determine any transcriptional changes present. WT and Dox-R cells were also incubated with and without EGF (5ng/mL), and live cells were counted using trypan-blue dye after 1-3 days of incubation. Both cell lines were then incubated with and without EGF 5ng/mL for 24 hours before adding doxorubicin (10^{-8}M for WT, 10^{-7}M for Dox-R). Live cells were counted after 1-3 days of doxorubicin incubation.

Results:

Western immunoblots demonstrated that WT and Dox-R cells had maximal pro-caspase-3 expression following incubation with EGF 5ng/mL for three days. RT-PCR also showed increased DNA transcription. The growth rate of both cell lines was unaffected after EGF treatment alone. However, cell death was increased in EGF pre-treated cells compared to non-EGF-pretreated cells after incubating with doxorubicin for three days (53±8% increase in WT, $p<0.05$; 40±7% in Dox-R, $p<0.05$).

Conclusions:

EGF induces increased pro-caspase-3 protein expression and DNA transcription at 5ng/mL. The cytotoxic effect of doxorubicin, which induces apoptosis through the caspase-3 pathway, is augmented by EGF-pretreatment.

Notes:

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P34 PHEOCHROMOCYTOMA AND PARAGANGLIOMA IN CHILDREN: OUTCOMES OF SURGICAL RESECTION.

Tuan H. Pham, M.D., Ph.D., Christopher Moir, M.D., Geoffrey Thompson, M.D., Abdalla Zarroug, M.D., Chad Hamner, M.D., David Farley, M.D., Jon van Heerden, M.D., Aida Lteif, M.D., William F. Young, M.D.
Mayo Clinic Rochester, Rochester, MN, U.S.A.

Purpose:
Pheochromocytoma and paraganglioma (PP) are rare tumors that can present unique management challenges in children. This study aims to review our experience managing PP.

Methods:
An IRB-approved retrospective review from 1975-2005 identified 30 patients <18 years old with histologic confirmation of either pheochromocytoma or paraganglioma. Standard statistical methods were used for comparison, risk and survival analyses where a p value <0.05 was considered significant.

Results:
There were 12 pheochromocytomas and 18 paragangliomas with a male:female ratio of 1:1. The most common presentations were hypertension (64%), palpitation (53%), headache (47%) and mass-related effects (30%). Nine patients had familial pheochromocytoma or paraganglioma, while the remainder appeared to occur sporadically. Fourteen patients had malignant while 16 had benign disease. Malignant tumors were larger than benign tumors (8.6 vs. 4.5 cm, p=0.004). Statistically significant risk factors for malignancy were: 1) paraganglioma, (odds ratio [OR]=9.99, p=0.030); 2) sporadic as opposed to familial PP (OR=6.00, p=0.049), and 3) tumor size larger than 6-cm (OR=5.40, p=0.033). Surgical resection was performed in 28 patients with perioperative mortality and morbidity rates of 0 and 10%, respectively. Resection achieved symptomatic relief in 25 patients. Incidence of recurrence after initial negative microscopic resection was 16% at a mean time 24 ± 8 months. The 10-year disease-specific survival (DSS) of patients with benign disease was 100%. For patients with malignant disease, the 5- and 10-year DSS were 78% and 31%, respectively, and the mean survival was 157 ± 32 months.

Conclusions:
Patients diagnosed with paraganglioma, sporadic disease, or those with tumor diameters larger than 6-cm are at a significantly higher risk for having a malignant tumor. Surgical resection remains the treatment of choice for PP. Resection with negative microscopic margins is essential for cure; however, an incomplete resection has value in alleviating symptoms.

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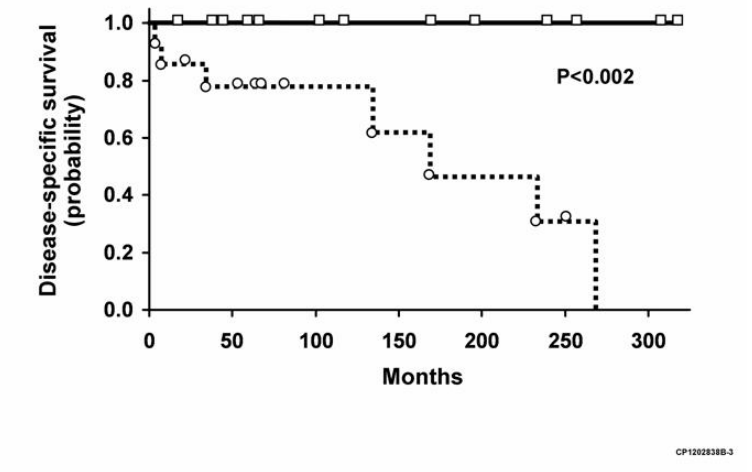

Figure 1. Kaplan-Meier analysis showing disease-specific survival for negative (square) versus positive (circle) microscopic resection margins at initial resection for pheochromocytoma and paraganglioma tumors.

Notes:

P35 DOES AGE INFLUENCE OUTCOME AMONG PATIENTS WITH OSTEOSARCOMA?

Matthew T. Harting, M.D.[1], Martin L. Blakely, M.D.[2], Andrea Hayes-Jordan, M.D.[1], Norman A. Jaffe, M.D.[3], Richard J. Andrassy, M.D.[1], Kevin P. Lally, M.D.[1]

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Purpose:

While osteosarcoma is more common among children and adolescents, it affects patients of all ages. Despite previous study, the importance of patient age as a prognostic factor remains unclear. Our objective was to determine if age at diagnosis is an independent prognostic indicator among patients with osteosarcoma.

Methods:

A retrospective cohort study of 438 patients with osteosarcoma of the extremities or trunk. Univariate and multivariate analyses of the data were conducted.

Results:

The median age at diagnosis was 18.1 years (0.1-78.8), 86% had primary extremity tumors, and median follow-up for survivors was 12.3 years (0.07-22.8). Children, adolescents, and young adults all had a 10-year survival around 50%, irrespective of exact age of dichotomization. However, the relative risk of death for age >40 was 1.5 (95% CI: 1.03-2.18, p=0.03) by univariate analysis. Younger patients ($\leq$40, n=382) were more likely to have extremity primary tumors (90.1 vs. 60.7%, p<0.01); no soft tissue extension of the primary bone tumor (71.2 vs. 55.6%, p=0.038); and a high degree of tumor necrosis after neoadjuvant chemotherapy (22.2 vs. 12.2%, p=0.05). In multivariate analysis, age was not an independent prognostic characteristic.

Conclusions:

Age does not appear to be a significant independent prognostic variable for overall survival or disease-free survival among patients with osteosarcoma. Although it appears that patients in the fifth decade and older fare worse than younger patients, our data indicate that other variables such as tumor necrosis, tumor extension, and tumor location are ultimately responsible for the observed decline in overall survival and disease-free survival.

Notes:

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P36 AGE-RELATED INJURY PREVENTION PRIORITIES IN THE UNITED STATES

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Purpose:
Injury is the leading cause of morbidity and mortality in the pediatric population in the United States. As the frequency of specific injury mechanisms varies by age group, it was suspected that injury prevention priorities would as well.

Methods:
We compared 14 injury mechanisms for children (age <20) entered into the National Trauma Data Bank (NTDB) for the period January 1994 to December 2003. We computed an injury prevention priority score (IPPS), which is a previously validated composite value reflecting the relative frequency of an injury mechanism and its relative median injury severity score (ISS), and similarly computed a hospital charge priority score (HCPS) based on median dollar amounts of billed hospital charges. The highest five scoring mechanisms overall and for each of four age groups are reported.

Results:
For the 10-year period, 211,482 children were entered into the NTDB from trauma centers across the United States. This pediatric trauma population was 67% male with a mean age of 12.3 years, a median ISS of 5 (mean=9), and a median charge of $7,668 (mean=$18,816). Motor vehicle-related injuries ranked first overall both by IPPS and HCPS and for three of the four age groups; other injury types ranked variously by IPPS and HCPS and across age groups (Table).

Conclusions:
Motor vehicle-related injuries ranked as the most severe and most costly prevention priority overall in this pediatric trauma population. However, prevention priorities varied for the different pediatric age groups emphasizing the importance of tailoring prevention initiatives to the target population.

Mechanism	Overall	0-4 years	5-9 years	10-14 years	15-19 years
	IPPS I HCPS	IPPS I HCPS	IPPS I HCPS	IPPS I HCPS	IPPS I HCPS
Motor vehicle	1 I 1	1 I 2	1 I 1	1 I 1	1 I 1
Transport, other	2 I 3	5 I .	2 I 5	2 I 3	3 I 4
Firearm	3 I 2	4 I 4	. I 4	5 I .	2 I 3
Pedestrian	4 I .	3 I 5	4 I .	3 I 4	5 I 2
Fall	5 I 5	1 I 2	3 I 3	4 I 5	. I .
Machinery	. I 4	. I 3	. I 2	. I .	. I .
Pedal cyclist	. I .	. I .	5 I .	. I .	. I .
Suffocation	. I .	. I .	. I .	. I 2	4 I 5

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P37 VARIATION IN THE MANAGEMENT OF PEDIATRIC BLUNT SPLENIC INJURY BASED ON TRAUMA CENTER STATUS

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[1]Children's Hospital of Pittsburgh, Pittsburgh, PA, U.S.A., [2]University of Pittsburgh, Pittsburgh, PA, U.S.A., [3]ZD Associates, LLC, Doylestown, PA, U.S.A., [4]Children's Hospital of Los Angeles, Los Angeles, CA, U.S.A.

Purpose:

The non-operative management of blunt splenic injury in children is so well established as a standard of care that the 1999 American Pediatric Surgical Association trauma committee called it "universally successful." We hypothesize that pediatric trauma care varies by hospital status. The objective of this study was to determine the frequency of operative management for pediatric splenic injury and explore whether the likelihood of total splenectomy varied as a function of hospital type.

Methods:

We examined all discharges in 2001 of children aged 1-19 years (n=705,998) in six states (FL, MA, NJ, NY, TX, and VA) comprising 27% of the U.S. population. We identified 1,761 patients with blunt splenic injury using *ICD-9* codes 865.00 - 865.09 and stratified them by trauma center certification. We calculated odds ratios (OR) for total splenectomy and death by trauma center status using multivariable logistic regression, adjusting for age, gender, and grade of splenic injury.

Results:

Dedicated pediatric trauma centers (PTCs) managed 106 (6%) children with blunt splenic injury. Total splenectomies were performed in 252 (14.3%); only one was performed at a PTC. Hospital mortality was 3.2% and was not related to splenectomy rates (adjusted OR [95% CI] of survival at PTC vs. all other centers = 0.93 [0.27, 3.13], p=0.9).

Comparison of Total Splenectomies for Trauma in Children

Type of Trauma Center	# of centers	Splenic injuries, n (% of subjects)	Splenic injuries per center, mean (median)	% receiving total splenectomies	Adjusted OR [95% CI] for total splenectomy, compared with PTC
Dedicated Pediatric (PTC)	9	106 (6.0%)	11.8 (11)	0.9%	1
Adult plus Pediatric	12	213 (12.1%)	17.8 (18)	11.3%	6.9 [0.9, 53.5], p=.063
Adult Level 1	47	610 (34.6%)	13.0 (8)	13.6%	8.3 [1.1, 61.7], p=.038
Adult Level 2	42	258 (14.7%)	6.1 (4)	16.3%	7.6 [1.0, 57.8], p=.049
Adult Level 3 or No Trauma Certification	270	574 (32.6%)	2.1 (1)	17.8%	8.6 [1.2, 63.7], p=.035
Total	380	1,761	4.6 (2)	14.3%	—

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Conclusions:
Consistent with standard of care, PTCs rarely perform total splenectomies for splenic injury. However, the majority of children with these injuries are managed elsewhere, frequently at hospitals with limited pediatric expertise or without high experience, and these centers are more than eight-times as likely to perform total splenectomy. Such huge, and likely unwanted, variation, demands further attention.

Notes:

P38 PEDIATRIC TRAUMA IN OPERATION IRAQI FREEDOM: 31ST COMBAT SUPPORT HOSPITAL, IRAQ

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[1]Martin Army Community Hospital, Fort Benning, GA, U.S.A., [2]Brooke Army Medical Center, Fort Sam, Houston, TX, U.S.A., [3]Madigan Army Medical Center, Fort Lewis, WA, U.S.A., [4]Walter Reed Army Medical Center, Washington, DC, U.S.A.

Purpose:

To review and report the experience of the 31st Combat Support Hospital (CSH) in Balad and Baghdad, Iraq with pediatric civilian trauma.

Methods:

Retrospective review from the Joint Theater Trauma Database of the pediatric patients seen at the 31st CSH. This has been approved by the Institutional Review Board of the Institute of Surgical Research at Brooke Army Medical Center.

Results:

From 1 January to 31 December 2004, 3,445 trauma patients were treated at the 31st CSH. One-hundred five were Iraqi children (age 17 and under). The average age of the children was 10.5 years, ranging from 1 year to 17 years. Ten died of their wounds. Forty sustained gunshot wounds, 13 had shrapnel wounds, and 22 were injured by improvised explosive devices (IEDs). None were wearing body armor. Fifteen arrived to our facility intubated. Seventy-three children required a total of 127 operations: 13 celiotomies, three thoracotomies, eight craniotomies, 20 skeletal fixations, and 49 wound debridements, among others. The mean injury severity score (ISS) in children was 14 (n = 52) and in adults was eight. This difference was statistically significant with a p value of less than 0.001. The mortality rate in children was 9.5% and in adults was 4.8% (p value 0.03).

Conclusions:

While traumatic injury is the leading cause of death in children over one year of age in the United States, penetrating injuries are uncommon. A high percentage of the injured Iraqi children that we treated sustained penetrating wounds. The injury severity scores in children were higher than those in adults and, thus, the mortality rate was higher. To our knowledge this will be the first reported series of wartime pediatric trauma patients comparing their outcome to that of an adult cohort.

Notes:

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P39 A SILVER IMPREGNATED ANTIMICROBIAL DRESSING REDUCES HOSPITAL COST IN PEDIATRIC BURN PATIENTS

Heather Paddock, M.D., Renata Fabia, M.D., Shelia Giles, RN, John Hayes, Ph.D., Dawn Adams, MS, Gail Besner, M.D.
Columbus Children's Hospital, Columbus, OH, U.S.A.

Purpose:
Since we began using novel silver impregnated antimicrobial dressing (AquacelAg®) in pediatric patients with partial-thickness burns, hospital length of stay (LOS) has been significantly reduced. AquacelAg® is a dressing that is simple to apply and which can be left in place for up to 14 days. Importantly, once adherent to the underlying burn (usually within 24 hours) pain is totally alleviated. Outer gauze dressings protect the underlying AquacelAg®, and can be easily changed by caregivers on an outpatient basis. In this study we investigated the cost-effectiveness of the use of this dressing.

Methods:
We retrospectively reviewed Burn Registry Data from a large Children's Hospital Burn Unit from January 2005 through August 2005 for inpatients with partial-thickness burns treated with AquacelAg®. A comparison group consisted of patients from the previous year treated with Silver sulfadiazine (SSD®) cream and matched for age and %TBSA burned. Patients with inhalation injury or full thickness burns were excluded. Intent to treat analysis was limited to patients with <22% TBSA burn. Direct costs (DC), total charges (TC), and net revenues were compared.

Results:
Direct costs were significantly less for AquacelAg® treated patients (n=38) compared to SSD® treated patients (n=39) (p=.01, t-test). Net margin (net revenue - total costs) was positive for patients treated with AquacelAg® (demonstrating profit) and negative for patients treated with SSD® (demonstrating revenue loss).

Discussion:
These data demonstrate that application of AquacelAg® reduces hospital LOS resulting in significant cost savings in the care of pediatric patients with partial-thickness burns. Since use of AquacelAg®: 1) shortens hospital stay allowing patients to be treated at home, 2) eliminates pain once adherent to the burn wound, and 3) is cost effective, use of this product is highly beneficial in the care of pediatric burn patients with partial-thickness burns.

	SSD	Aquacel	Per Patient	
Avg LOS	7.5	3.1	SSD	Aquacel
Gross Revenue	$601,600	$251,208	$15,426	$6,611
*Total Direct Cost**	*249,182*	*98,570*	*6,389*	*2,594*
Total Indirect Cost	*160,287*	*64,892*	*4,110*	*1,708*
Total Cost	409,469	163,462	10,499	4,302
Net Revenue	385,074	177,704	9,874	4,676
Net Margin (NR-TC)	(24,395)	14,242	(626)	375

**p=.01*

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P40 HB-EGF DECREASES NEUTROPHIL-ENDOTHELIAL CELL ADHESION AFTER ANOXIA/REOXYGENATION INJURY

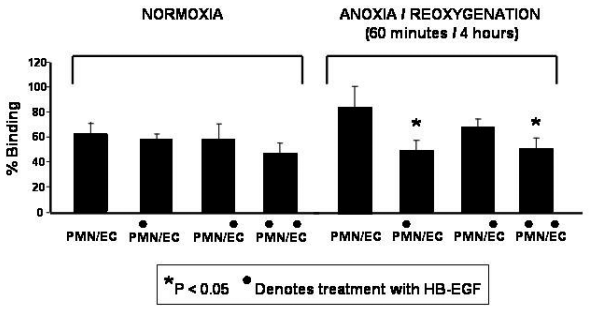
Dorothy V. Rocourt, M.D., Veela Mehta, Ph.D., Gail E. Besner, M.D.
Children's Research Institute, Columbus, OH, U.S.A.

Purpose:
Necrotizing enterocolitis (NEC) is a disease of uncertain etiology mediated, in part, by intestinal ischemia/reperfusion (I/R) injury. Hyperadhesiveness of neutrophils (PMN) to vascular endothelial cells (EC), followed by neutrophil transendothelial migration, play important roles in the initiation of I/R-mediated injury. We have shown that heparin-binding EGF-like growth factor (HB-EGF) decreases the incidence and severity of experimental NEC. Here we investigate whether the beneficial effects of HB-EGF are mediated, in part, by its ability to affect PMN-EC adhesion.

Methods:
Human PMN co-cultured with human umbilical vein endothelial cell (HUVEC) monolayers exposed to anoxia/reoxygenation (A/R) were used to mimic changes in the microvasculature that accompany I/R. Primary cultures of HUVEC were harvested from umbilical cords and PMN were isolate from blood of healthy volunteers. HUVEC monolayers were treated with either HB-EGF (100 ng/ml) or PBS for 60 min, exposed to either anoxia or normoxia for 60 min, and then reoxygenation for up to 4 h. Simultaneously, PMN were labeled with Calcein AM for 30 min and then treated with either HB-EGF or PBS for 30 min. Labeled PMN were then added to HUVEC monolayers for determination of PMN-EC adherence. In similar experiments where HUVEC monolaters were established on porous membranes, PMN transmigration through HUVEC monolayers was determined.

Results:
PMN-EC adhesion was significantly increased after exposure of EC to A/R compared to EC exposed to normoxia only (87 % vs. 64 % binding, $p<0.05$, Student t test). A/R-induced PMN-EC hyperadherence was significantly decreased by treatment of PMN with HB-EGF (51% vs. 87 % binding, $p<0.05$). HB-EGF did not affect PMN-EC adhesion under normoxic conditions. HB-EGF also decreased PMN transendothelial migration.

Conclusions:
These results indicate that HB-EGF significantly decreases A/R-induced PMN-EC adhesion. Thus, HB-EGF acts not only as a direct potent cytoprotective agent for the intestinal mucosa, but as an anti-infllammatory agent as well.



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P41 COMPLICATIONS OF IMPLANTED CENTRAL VENOUS CATHETERS IN NEUTROPENIC CHILDREN

Arvand Elihu, M.D., Gerald Gollin, M.D., Kimberly Arledge, M.D.
Loma Linda University School of Medicine, Loma Linda, CA, U.S.A.

Purpose:
Implanted central venous catheters (ICVC) are frequently placed in children with hematological malignancies associated with neutropenia. Although neutropenia is recognized as a risk factor for infection and compromised wound healing there is little data regarding the specific impact of neutropenia upon morbidity and mortality following placement of ports or cuffed central venous catheters.

Methods:
With Institutional Review Board approval, children with the diagnosis of acute lymphocytic leukemia (ALL) or aplastic anemia (AA) who had an ICVC placed between January 1999 and May 2005 were identified. The absolute neutrophil count (ANC) immediately prior to catheter placement was recorded. Subjects were divided into Group 1 (ANC $< 0.5 \times 10^3/\mu l$) and Group 2 (ANC $\geq 0.5 \times 10^3/\mu l$). Cases in which the catheter was removed within 100 days were identified and the reason for removal was determined. Differences in outcome were assessed using chi-square analysis.

Results:
One hundred ninety-five patients with ALL and 15 with AA were identified. The ANC was less than $0.5 \times 10^3/\mu l$ in 105 cases. The incidence of line removal within 100 days was 17.1% in Group 1 and 7.6% in Group 2 (p=0.01). In 11.4% of cases in Group 1 catheters were removed due to infection and/or wound dehiscence including two cases of mucormycosis at port sites that required extensive debridement and caused one death. In contrast, 1.4% of catheters in Group 2 were removed for infection. (p=0.005) The incidence of other causes of catheter failure, including thrombosis, leakage and migration, was similar between the groups.

Conclusions:
The placement of ICVC in neutropenic children was associated with substantially increased infectious morbidity and one death in our study population. When possible, ICVC should be avoided in the presence of neutropenia. If an implanted catheter is used there should be a low threshold for removal if signs of infection develop.

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P42 GENE EXPRESSION IN CONGENITAL CYSTIC ADENOMATOID MALFORMATION: MOLECULAR EVIDENCE OF ARRESTED DEVELOPMENT

Amy J. Wagner, M.D., Amber N. Stambaugh, MS, Jess Edmondson, Matthew S. Clifton, M.D., Erich J. Grethel, M.D., Raul A. Cortes, M.D., Michael R. Harrison, M.D., Hanmin Lee, M.D., Kerilyn Nobuhara, M.D., Diana Farmer, M.D., Sam Hawgood, M.D.
University of California San Francisco, San Francisco, CA, U.S.A.

Purpose:
Congenital cystic adenomatoid malformation (CCAM) is a hamartomatous lesion with unknown pathogenesis. It is hypothesized that CCAM results from an alteration in signaling pathways during lung development. Our purpose was to identify differentially expressed genes in CCAM compared to age and gender-matched control lung, and specifically to examine genes related to lung development.

Methods:
After obtaining IRB approval, we examined differential gene expression in CCAM versus normal lung from four groups: prenatally-resected CCAM, normal fetal lung, postnatally-resected CCAM, and normal lung. We obtained four CCAM samples in the prenatal period and two control fetal lung samples. We also compared five postnatally-resected CCAM specimens to two normal lung controls. We examined differential expression between each CCAM and the appropriate control lung, and also compared prenatal versus postnatal CCAM using microarray analysis. We further examined genes that had a minimum of a two-fold difference in expression (M value >2.0) and those with highest intensity (A value). Results were confirmed using quantitative RT-PCR.

Results:
CC-10 (or Clara Cell Specific protein, CCSP) was increased in CCAM compared to normal lung (M value=3.18, A value=13.35). Normal fetal lung had higher surfactant protein A (SP-A) expression compared to CCAM (M value=5.47, A value=13.43). There was also a statistically significant differential expression in hedgehog interacting protein (HHIP) in CCAM versus control (M value= -2.27, A value= 8.15). The quantitative RT-PCR results were consistent with microarray analysis.

Conclusions:
The increased CC-10 expression of and decreased SP-A expression in CCAM compared to normal lung is consistent with CCAM originating in the proximal tracheobronchial tree. The differential expression in HHIP could link the pathogenesis of CCAM to an aberration in lung development, as hedgehog is an important regulator of branching lung morphogenesis.

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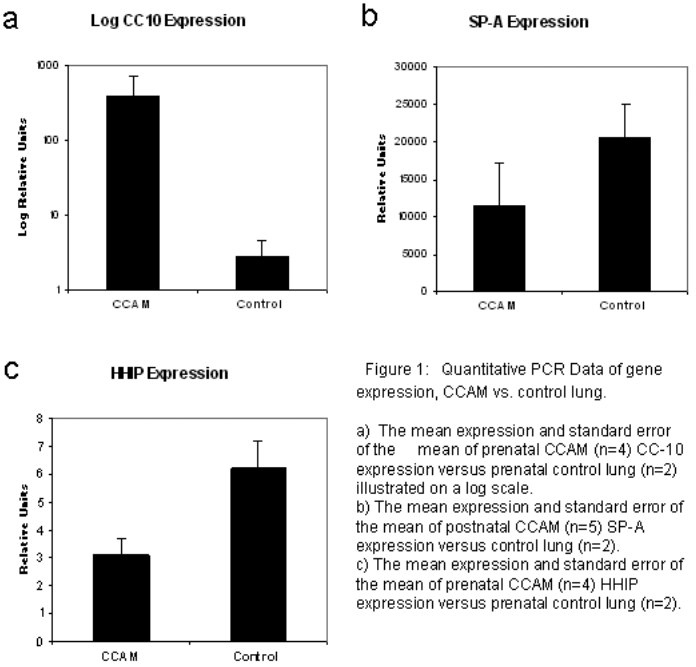

Figure 1: Quantitative PCR Data of gene expression, CCAM vs. control lung.

a) The mean expression and standard error of the mean of prenatal CCAM (n=4) CC-10 expression versus prenatal control lung (n=2) illustrated on a log scale.
b) The mean expression and standard error of the mean of postnatal CCAM (n=5) SP-A expression versus control lung (n=2).
c) The mean expression and standard error of the mean of prenatal CCAM (n=4) HHIP expression versus prenatal control lung (n=2).

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P43 MOLECULAR GENETIC PATHOPHYSIOLOGY IN HUMAN CONGENITAL DIAPHRAGMATIC HERNIA

Sibel Kantarci, Ph.D.[1], Barbara Pober, M.D.[2], Thomas B. Kinane, M.D.[3], Lihadh Al-Gazali, M.D.[4], David Casavant, M.D.[1], Dick Tibboel, M.D., Ph.D[5], Meaghan Russell, M.P.H.[1], Jay M. Wilson, M.D.[6], Charles Lee, Ph.D.[7], Patricia K. Donahoe, M.D.[1]

[1]Pediatric Surgical Research Laboratories, MassGeneral Hospital for Children, Harvard Medical School, Boston, MA, U.S.A., [2]Genetics and Teratology, MassGeneral Hospital for Children, Harvard Medical School, Boston, MA, U.S.A., [3]Massachusetts General Hospital, Harvard Medical School, Boston, MA, U.S.A., [4]United Arab Emirates University, United Arab Emirates, [5]Erasmus Medical Centre, Rotterdam, The Netherlands, [6]Children's Hospital Boston, Boston, MA, U.S.A., [7]Department of Pathology, Brigham and Women's Hospital, Harvard Medical School, Boston, MA, U.S.A.

Purpose:
Congenital Diaphragmatic Hernia (CDH) is a relatively common developmental anomaly with a high mortality primarily due to lung hypoplasia and pulmonary hypertension. Little is known about the etiology of CDH. About half of patients have isolated CDH, while the remainder have associated birth defects, chromosomal abnormalities or single gene disorders. We aim to identify genes involved in CDH in our carefully phenotyped cohort of patients by application of several complementary strategies.

Methods:
We have sequenced 66 of the 173 enrolled patients for 22 candidate genes identified from animal models, expression and functional patterns, or chromosomal hot spots. A web-based polymorphism phenotyping program, PolyPhen, was used on all nonsynonymous SNPS to predict the effect of amino acid changes on the structure and function of the protein.

We used 1Mb resolution array-based Comparative Genomic Hybridization (aCGH) to detect possible microdeletions or microduplications on 30 syndromic and nonsyndromic patients.

We are performing homozygosity mapping by application of the 10K Affymetrix chip on a large family containing several members affected with an autosomal recessive multiple anomaly condition with CDH as a cardinal feature.

Results:
We found several potentially damaging SNPs not previously reported in the dbSNP homepage, nor in 485 ethnically matched control group recruited as part of this study. However these SNPs were present in an unaffected parent. Functional studies of selected ROBO1 SNPs are underway and we also plan to expand the size of the control group.

We detected two abnormalities involving apparently de novo deletions of chromosomes 16q12.2 and 1q42 respectively. Prior cases with deletions of 1q42 suggest this is a CDH hotspot.

Conclusions:
These numerous strategies will identify genes, which when deleted or mutated, contribute to the development of CDH. These approaches provide a model for elucidating the genetic basis of common, but etiologically heterogeneous, birth defects.

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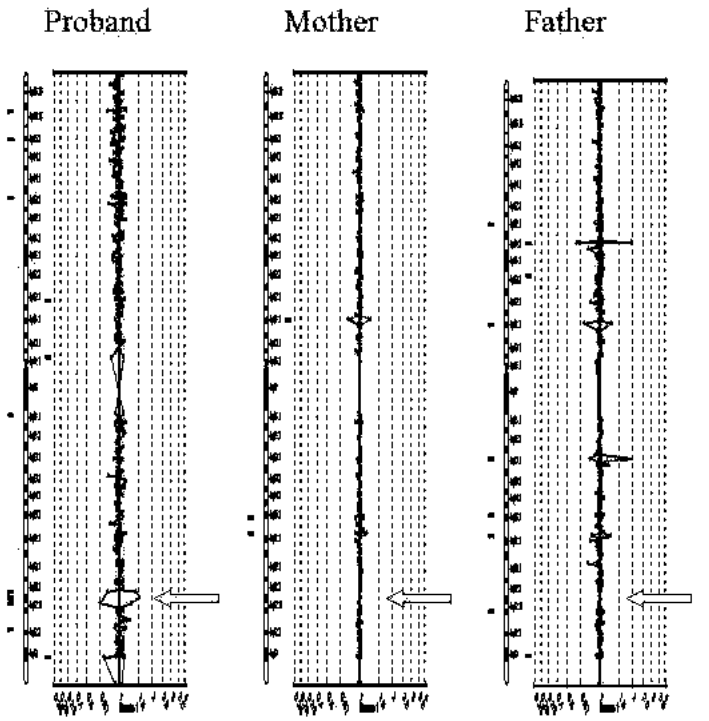

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P44 PERCENT PREDICTED LUNG VOLUMES AS MEASURED ON FETAL MAGNETIC RESONANCE IMAGING: A USEFUL BIOMETRIC PARAMETER FOR RISK STRATIFICATION IN CONGENITAL DIAPHRAGMATIC HERNIA

Carol E. Barnewolt, M.D., Shaun M. Kunisaki, M.D., Dario O. Fauza, M.D., Luanne P. Nemes, RN, MS, PNP, Judy A. Estroff, M.D., Russell W. Jennings, M.D.
Children's Hospital Boston, Boston, MA, U.S.A.

Purpose:
The lung-to-head ratio, as measured using fetal ultrasound, is the only currently used method to quantify risk in the fetus shown to have congenital diaphragmatic hernia (CDH). This measurement is difficult to reproduce and applies to a limited gestational age. We propose a new method of lung measurement in CDH using fetal magnetic resonance (MR) imaging as a way to stratify risk of pulmonary compromise.

Methods:
Seventeen fetuses with CDH were prospectively evaluated by MR. None had congenital heart disease. Lung volumes were measured in the coronal plane and expressed as a percent of the predicted lung volume (PPLV). Predicted lung volume was determined by subtracting mediastinal volume from total thoracic volume. The PPLV was correlated with use of extracorporeal membrane oxygenation (ECMO), hospital length of stay, and survival. Patient outcomes were also assessed after stratification of patients into a high-risk (PPLV less than 15) and low-risk (PPLV more than 15) category. Statistical analyses were performed using the Mann-Whitney, Spearman nonparametric correlation, and Fisher's exact tests, as appropriate ($p<0.05$).

Results:
Three pregnancies were terminated, resulting in outcomes data for 14 patients. The PPLV was 20.3±10.4 at a gestational age of 22.3±5.7 weeks. Overall survival was 78.6%. The PPLV was significantly associated with ECMO utilization, length of stay, and overall patient survival. All high-risk patients (n=5, PPLV 9.4±2.7) required prolonged ECMO support and had a 40% survival rate. In contrast, only one (11%) low-risk patient (n=9, PPLV 26.4±7.5) required ECMO, and survival was 100%.

Conclusions:
Early results suggest that percent predicted lung volumes measured by fetal magnetic resonance imaging can predict severity of disease in congenital diaphragmatic hernia. A value of less than 15 is associated with a need for prolonged respiratory support and/or death, despite aggressive postnatal management. A value of greater than 15 is associated with good outcomes.

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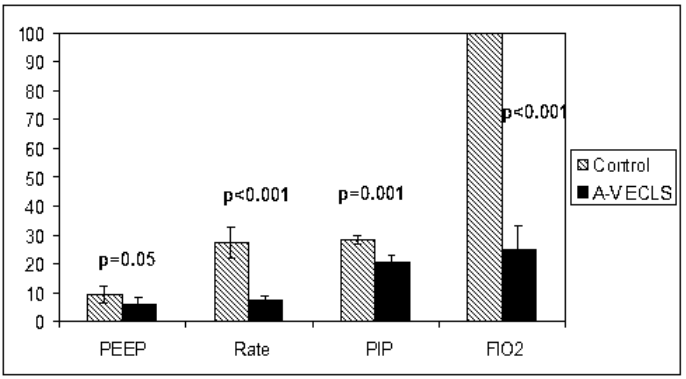
P45 PUMPLESS ARTERIOVENOUS EXTRACORPOREAL LIFE SUPPORT (A-V ECLS) IN A LUNG-INJURY MODEL: A NEW APPROACH TO ECMO?

J. Kristine Brown, M.D., Rupa Seetharamaiah, M.D., George B. Mychaliska, M.D., Robert H. Bartlett, M.D., Ronald B. Hirschl, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

Purpose:
Extracorporeal life support (ECLS, ECMO) is complex and expensive. We investigated the use of pumpless arteriovenous ECLS (A-V ECLS) in a newborn-sized, lung-injured rabbit model.

Methods:
Anesthetized, paralyzed, and heparinized adult New Zealand rabbits (n=11, weight 2.8±0.48kg) underwent tracheostomy and pulmonary saline lavage to achieve PO_2<60 mmHg and PCO_2>60 mmHg on baseline ventilator settings of rate=15 bpm, FiO_2=1.0, peak inspiratory pressure (PIP)=15 cm H_2O, and positive end-expiratory pressure (PEEP)=4 cm H_2O. In the A-V ECLS group, carotid-jugular cannulation followed by arteriovenous flow through a custom-made, low-resistance, 0.3m^2 hollow-fiber oxygenator was initiated. Oxygen sweep flow was set at 1 L/minute. Ventilator settings were adjusted in both the A-V ECLS (n=5) and control (n=6) groups to achieve $PO_2 \geq 60$ mmHg and $PCO_2 \leq 60$ mmHg. Statistical comparison utilized t-test analysis.

Results:
After saline lavage, baseline mean PCO_2 was 79±13 mmHg and mean PO_2 was 37±11 mmHg. During the experimental period of four hours, heart rate and mean arterial blood pressure did not significantly differ between the ECLS and control groups. Mean flow through the artificial lung was 47±14cc/kg/min. The A-V ECLS group required significantly less ventilatory support (rate, PIP, FiO_2, and PEEP) to achieve similar target PCO_2 and PO_2 levels (see graph) when compared to control animals.



Conclusions:
Pumpless A-V ECLS is a simple, effective means of supporting gas exchange and reducing ventilator requirements in this lung-injured, newborn-sized animal model. These data support further studies using pumpless A-V ECLS as an artificial placenta in neonates with respiratory failure.

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1 MATCHED ANALYSIS OF NON-OPERATIVE MANAGEMENT AND IMMEDIATE APPENDECTOMY FOR PERFORATED APPENDICITIS (3 MINUTE)

Marion C.W. Henry, M.D., MPH[1], Gerald Gollin, M.D.[2], Saleem Islam, M.D.[3], Karl Sylvester, M.D.[4], Angela Walker, BS[5], Bonnie L. Silverman, Ph.D.[1], R. Lawrence Moss, M.D.[1].

[1]Yale University, New Haven, CT, U.S.A., [2]Loma Linda University, Loma Linda, CA, U.S.A., [3]University of Mississippi, Jackson, MS, U.S.A., [4]Stanford University, Stanford, CA, U.S.A., [5]University of Missouri, Columbia, MO, U.S.A.

Purpose:
Attempts to determine the effectiveness of non-operative management for perforated appendicitis have not controlled for inherent differences in the clinical status of patients treated non-operatively versus immediate appendectomy. The objective of this study was to compare these management options in clinically similar patients.

Methods:
Multi-center cohort study from 1998-2003. First, we compared patients treated non-operatively versus those undergoing appendectomy to identify differences in 12 clinical parameters. Based upon these findings, we matched patients and compared the following outcomes in clinically similar groups: overall complications, abscess development, and length of stay (LOS). Analysis was performed according to intention to treat principles, using Fisher's exact and Student's t tests.

Results:
The only significant difference between patients treated non-operatively and those treated by appendectomy was the duration of pain on presentation (Table 1). The non-operative group had a mean duration of 6.8 days of pain on presentation versus 3.1 for appendectomy.

We then compared patients treated non-operatively to those undergoing appendectomy matched on duration of pain on presentation. These groups continued to be clinically comparable for the other 11 parameters. Compared to these matched controls the non-operative group had fewer complications (19 vs. 43%, $p<0.01$), fewer abscesses (4 vs. 24%, $p<0.01$) and a trend for shorter LOS (6.5 ± 5.7 vs. 8.8 ± 6.7 days, $p=0.08$).

Conclusions:
When non-operative management for perforated appendicitis was studied using appropriately matched clinical controls, we found that it resulted in a lower complication rate and shorter LOS. Our data suggest that non-operative management should be strongly considered in children with perforated appendicitis presenting with pain greater than five days.

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Clinical characteristics of non-operative versus immediate appendectomy patients			
Variable	**NO group N=48 Mean ± sd or n(%)**	**AP group N=192Mean ± sd or n(%)**	**p-value**
Mean Age	8.5 ± 3.8 yrs	9.8 ± 3.8 yrs	0.08
Mean pain duration	6.8 ± 5.2 days	3.1 ± 2.4 days	<0.001
Mean WBC at admit	17.7 ± 6.0	17.3 ± 6.2	0.73
Mean temp at admit	38.0 ± 0.9 C	38.1 ± 0.9 C	0.65
RLQ pain	19 (42%)	99 (56%)	0.11
Fecalith in OR	4 (9%)	27 (18%)	0.17
Insurance = medicaid	21 (44%)	64 (37%)	0.37

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2 SHOULD INTERVAL APPENDECTOMY BE PERFORMED IN PEDIATRIC PATIENTS INITIALLY TREATED NON-OPERATIVELY FOR APPENDICITIS? (3 MINUTE)

Devin Puapong, M.D., Anna Kaminski, M.D., Philip Haigh, M.D., Harry Applebaum, M.D.
Kaiser Permanente Los Angeles Medical Center, Los Angeles, CA, U.S.A.

Purpose: Interval appendectomy (IA) has been the traditional approach for patients initially treated non-operatively for appendicitis. The goal of this study was to evaluate outcomes for those patients treated without IA.

Methods: A retrospective cohort study of pediatric patients (age<18) with appendicitis was performed using data from 12 regional acute-care hospitals from 1992-2002 with mean length of follow-up of 7.5 years. Initial treatment was recorded as either immediate appendectomy, or non-operative treatment using intravenous antibiotics with or without percutaneous abscess drainage. Main outcomes measured were recurrent appendicitis and length of hospital stay. Cox proportional hazards regression modeling was used to evaluate factors associated with recurrence.

Results: Six thousand, four hundred forty-six patients were included in the study. Six thousand, three hundred fifty-two (98.5%) underwent initial appendectomy. Ninety-four patients were initially managed non-operatively; of these, 19 (20%) had perforated appendicitis, and 42 (44.7%) had evidence of an abscess. Ten of the 94 patients had IA, whereas the remaining 84 (89.4%) did not. Of these 84 patients, six (7%) developed recurrent appendicitis. Age (hazard ratio [HR] <5 yrs=2.38; 95%CI 0.43-13.02; p=0.31), gender (HR=0.19; 95%CI 0.02-1.65; p=0.13), type of appendicitis (HR perforated=0.77; 95%CI 0.09-6.59; p=0.81; HR abscess=6.57; 95%CI 0.76-56.24; p=0.08) and abscess drainage (HR=3.12; 95%CI 0.63-15.48; p=0.16) had no influence on recurrence. Patients treated without IA had a cumulative length of stay (CLOS) of 6.6 days compared to a CLOS of 8.5 days in the IA group (p=0.07). Patients with recurrent appendicitis had a CLOS of 15 days (p=0.001).

Conclusions:
Although patients with recurrent appendicitis have longer cumulative length of stay, recurrent appendicitis is rare after non-operative treatment. Routine interval appendectomy is not necessarily indicated in pediatric acute appendicitis treated non-operatively.

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3 LAPAROSCOPIC CHOLECYSTECTOMY AND CHOLANGIOSCOPIC COMMON BILE DUCT EXPLORATION IN THE MANAGEMENT OF CHOLEDOCHOLITHIASIS IN CHILDREN (3 MINUTE)

J. Kristine Brown, M.D., Eiichi Miyasaka, BA, Oliver S. Soldes, M.D., James D. Geiger, M.D., Ronald B. Hirschl, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

Purpose:
Our hypothesis is that cholangiography and cholangioscopic common bile duct (CBD) exploration during laparoscopic cholecystectomy (LC) is a superior strategy in the management of choledocholithiasis in children when compared to endoscopic retrograde cholangiopancreatography (ERCP) either preceding or following LC.

Methods:
Medical records of children undergoing LC over the past 10 years were reviewed. Indications for operation, operative findings, complications, and outcomes were reviewed. Statistical comparison utilized chi-square and t-test analyses.

Results:
Out of 114 children who underwent LC, six underwent preoperative ERCP for management of choledocholithiasis (5%, age=11.5±4.2years), with choledocholithiasis identified in 5/6 (83%), while 27 underwent intraoperative cholangiography (IOC, 24%, age=13.8±3.3years) with choledocholithiasis identified in 7/27 (27%). CBD stones were rarely observed without the presence of hyperbilirubinemia and/or CBD dilation on ultrasound (negative predictive value=98%, positive predictive value=52%, sensitivity=86%, specificity=89%). All patients with CBD stones identified by IOC were successfully treated by cholangioscopic CBD exploration (CCBDE), which consisted of laparoscopic-guided cannulation of the cystic duct with a cholangioscope, and basket or flush removal of stones. The total number of general anesthetics was significantly decreased in the IOC±CCBDE group (1.0±0.0) when compared to the LC+ERCP group (1.8±0.4, p=0.004). Mean length of stay (LOS) was significantly shorter in the IOC±CCBDE group when compared to the ERCP group (IOC±CCBDE=3.5±1.7days versus ERCP=12.6±8.2days, p=0.04). No complications occurred in the IOC±CCBDE group, but one heart transplant recipient developed sepsis after preoperative ERCP and LC. None of the IOC±CCBDE patients required subsequent ERCP for missed or recurrent choledocholithiasis.

Conclusions:
Cholangiography with cholangioscopic CBD exploration at the time of laparoscopic cholecystectomy is a superior and safe approach in the management of choledocholithiasis in children. Benefits of this approach over ERCP include shorter LOS and need for a single anesthetic, making it a useful addition to the pediatric surgeon's armamentarium.

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4 ABNORMAL GALLBLADDER EJECTION FRACTION DOES NOT PREDICT SYMPTOMATIC RELIEF FROM CHOLECYSTECTOMY IN CHILDREN WITH ACALCULOUS BILIARY PAIN (3 MINUTE)

Heather Paddock, M.D.[1], Vance Smith, M.D.[2], John Hayes, Ph.D.[1], Greg Bates, M.D.[1], Brian Kenney, M.D., MPH[1], Donna A. Caniano, M.D.[1], Benedict C. Nwomeh, M.D.[1]
[1]Columbus Children's Hospital, Columbus, OH, U.S.A., [2]The Ohio State University College of Medicine, Columbus, OH, U.S.A.

Purpose:
Children with atypical abdominal pain often undergo evaluation for biliary dyskinesia (BD). The purpose of this study was to determine if the current assessment of children with symptoms of biliary disease without gallstones is predictive of clinical improvement after cholecystectomy.

Methods:
We retrospectively reviewed charts of 51 children without cholelithiasis who underwent evaluation for BD followed by cholecystectomy between 1999 and 2005 at a tertiary children's hospital. We identified age, sex, clinical presentation, radiographic testing, and post-operative outcomes. Gallbladder ejection fraction (GBEF) was evaluated with either HIDA or ultrasound (U/S) using either fatty meal (FM) or cholecystokinin (CCK) stimulation. Abnormal GBEF was defined as <35%. Chi Squared analysis was performed between the rates at which each radiographic test predicted post-operative symptom resolution.

Results:
Patient ages were 3 months - 21 years, 37 were female, and the average time of follow-up was 136 days. Thirty-six of 51 children (71%) had abnormal GBEF by HIDA or U/S. Forty of 51 children (78%) reported relief of symptoms. Radiologic test sensitivity was 70% and specificity 27%. Chi Squared analysis revealed no statistically significant ability of GBEF to predict post-operative symptom relief.

Conclusions:
Selected children with clinical symptoms of biliary disease and normal GBEF benefit from cholecystectomy. Furthermore, these patients report relief at the same rate as children with abnormal GBEF. The clinical entity of pediatric acalculous biliary pain which responds to cholecystectomy has yet to be fully described, but should be investigated by prospective clinical trials.

		Symptomatic Relief	
		Yes	No
Gallbladder Ejection Fraction	<35%	28 (54%)	8 (16%)
	Normal	12 (24%)	3 (6%)

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5 UTILIZATION OF ESOPHAGO-CRURAL SUTURES AND MINIMAL ESOPHAGEAL DISSECTION REDUCES THE INCIDENCE OF POSTOPERATIVE TRANSMIGRATION OF LAPAROSCOPIC NISSEN FUNDOPLICATION WRAP (3 MINUTE)

Shawn D. St. Peter, M.D.[1], Daniel J. Ostlie, M.D.[1], Patricia A. Valusek, M.D.[1], Casey M. Calkins, M.D.[2], Steven B. Shew, M.D.[3], George W. Holcomb, III, M.D.[1]

[1]Children's Mercy Hospital, Kansas City, MO, U.S.A., [2]Children's Hospital of Wisconsin, Milwaukee, WI, U.S.A., [3]University of California Los Angeles, Los Angeles, CA, U.S.A.

Purpose:

Herniation of the fundoplication wrap through the esophageal hiatus is a common reason for surgical failure in children who have undergone laparoscopic Nissen fundoplication. Extensive mobilization of the gastroesophageal junction in combination with decreased adhesions after laparoscopy may contribute to the development of this complication. In an attempt to decrease the incidence of wrap migration, we changed our technique to minimal mobilization of the intra-abdominal esophagus and to placement of esophageal-crural sutures. In this study, we investigate the impact of these modifications on outcome.

Methods:

A retrospective analysis was performed of all patients undergoing laparoscopic fundoplication by the senior author from January 2000 through December 2004. Those undergoing operation with extensive esophageal mobilization and without esophago-crural sutures (Jan 00-Mar 02)(Group I) were compared to those in whom there was minimal esophageal dissection and had these sutures placed (Apr 02-Dec 04)(Group II).

Results:

Two hundred forty-nine patients underwent laparoscopic Nissen fundoplication during the study period. One hundred thirty patients were in Group I and 119 patients were in Group II. Comparative data is illustrated in Table 1. The rate of transmigration decreased from 12% in group I to 5% in Group II (p=.072). The relative risk of transmigration without esophago-crural sutures was 2.29 times the risk if these sutures were utilized.

Conclusions:

This retrospective study has shown that placement of esophago-crural sutures and minimization of the dissection around the esophagus results in a significant reduction in the development of wrap transmigration following laparoscopic Nissen fundoplication.

DATA POINT	GROUP I	GROUP II	Pvalue
	(130 PTS.)	(119 PTS.)	
Mean age (mo)	21.1	27.3	.236
Mean wt (kg)	10.0	11.6	.335
Mean op time (min)	93.4	102.4	.023
Mean length of fundoplication wrap (cm)	2.05	2.13	.074
No. pts. requiring gastrostomy	64	58	.999
No. pts. with esophago-crural sutures	0	ALL	
No. pts. with transmigration wrap	15 (12%)	6 (5%)	.072

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6 IMPACT OF BUTTON CECOSTOMY ON MANAGEMENT OF FECAL INCONTINENCE AND CONSTIPATION — NINE YEARS OF EXPERIENCE IN PEDIATRIC POPULATION (3 MINUTE)

Dragan Kravarusic, M.D.[1], Sarah Wong[2], Lanna Bottomly, RN[1], Andrew Wong, M.D.[1]
[1]Alberta Children's Hospital, Calgary, AB, Canada, [2]University of Calgary, Calgary, AB, Canada

Purpose:
To validate the long term impact of button cecostomy on management of fecal incontinence and constipation. In childhood and adolescents fecal soiling represents one of the most embarrassing and psychologically devastating problems. Physical and emotional distress associated with daily repeated rectal enemas, diet modifications/laxatives is largely minimized by the introduction of a cecostomy tube for colonic cleansing with antegrade colonic enemas (ACE).

Methods:
Over a period of nine years (1997-2005) we performed "button" cecostomy's in 68 patients, laparoscopic 39 (57.35%), open 29 (42.64%). Patient indications included: spina bifida (*n*=43), history of ano-rectal malformation repair (*n*=18), intractable functional constipation (*n*=4), miscellaneous causes (*n*=3). The mean patient age at the time of the surgical procedure was 10.8 SD ± 4.0 years. Mean postoperative follow-up was 4.03 SD ±1.76 years. Cleansing protocols, irrigation solutions differ among the patients.

Results:
For study purposes and with ethics board approval we adapted a standardized follow up questionnaire about management of incontinence/intractable constipation before and after button cecostomy insertion to validate the long term impact of ACE on symptom severity and quality of life scale. Complications included leakage of the irrigation solution (*n*=2), development of granulation tissue (*n*=11), and tube dislodgement (*n*=4). Patient/parents appraisal (scale 1-3) for improved symptoms (change+1.7±0.3) and quality of life (change+1.6±0.4) achieved statistical significance for both ($p<0.001$).

Conclusions:
In our opinion quality of life scale must be routinely included in all studies of clinical interventions aimed at improving aspects of fecal incontinence/soiling, and the length of intervention studies should be sufficiently long to adequately assess changes in quality of life associated with the intervention. Since button cecostomy and ACE are introduced in our institution as a management option, the treatment of fecal incontinence and intractable constipation significantly improved efficacy and patient compliance.

Notes:

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7 POUCH OUTCOMES AMONG CHILDREN WITH ULCERATIVE COLITIS TREATED WITH CALCINEURIN INHIBITORS PRIOR TO ILEAL POUCH ANAL ANASTOMOSIS SURGERY (3 MINUTE)

Craig W. Lillehei, M.D., Elizabeth Hait, M.D., Melissa J. Shuman, BA, Athos Bousvaros, M.D., MPH, Robert C. Shamberger, M.D.
Boston Children's Hospital, Boston, MA, U.S.A.

Purpose:
To describe the pouch outcomes of 13 children with ulcerative colitis (UC) who were pretreated with calcineurin inhibitors prior to ileal pouch anal anastomosis (IPAA) surgery.

Methods:
An IRB-approved retrospective chart review was performed of consecutive patients with UC treated with calcineurin inhibitors prior to IPAA surgery between 1998 and 2003. Primary endpoint was pouch outcome after at least two years of follow-up (healthy pouch, acute pouchitis, chronic refractory pouchitis or pouch failure). Secondary endpoints were early postoperative complications, number of stages and time between stages.

Results:
Thirteen (33%) of 39 consecutive patients who underwent IPAA for UC were treated with calcineurin inhibitors preoperatively (four with cyclosporine; nine with tacrolimus). All 13 patients were concomitantly treated with systemic steroids. 10/13 had been on 6-mercaptopurine and 8/13 were on mesalamine prior to calcineurin inhibitor therapy. All 13 IPAA surgeries were elective procedures performed by two surgeons with rectal mucosectomies and J pouches. 4/13 (31%) had healthy pouches, 7/13 (54%) had at least one episode of acute pouchitis (average: two episodes), 1/13 (8%) had chronic pouchitis and 1/13 (8%) was later determined to have Crohn's disease. There were no pouch failures. One of 13 (8%) had an early postoperative complication (intraabdominal abscess). Six of 13 (46%) had a two-staged procedure and Seven of 13 (54%) had a three-staged procedure. The mean time between the first and second stages was 3.8 months (range: 2-6 months) and 4.1 months (range: 2.5-6 months) between the second and third stages.

Conclusions:
In this series, chronic pouchitis was a rare complication among children who were pretreated with calcineurin inhibitors. Calcineurin inhibitor use did not lead to or portend increased early postoperative complications or affect the number or duration of surgical stages. Further studies are required to determine if preoperative calcineurin inhibitor use improves pouch outcomes or facilitates performance of two-staged procedures.

Notes:

8 LAPAROSCOPIC UNROOFING OF SPLENIC CYSTS RESULTS IN RECURRENCES (3 MINUTE)

Felix Schier[1], Karl-Ludwig Waag[2], Benno Ure[3].
[1]University Medical Centre, Mainz, Germany, [2]University Medical Centre, Mannheim, Germany, [3]University Medical Centre, Hannover, Germany

Purpose:
Laparoscopic management of non-parasitic splenic cysts by unroofing is reported as an appropriate treatment modality. In our experience, however, recurrences are frequent.

Methods:
Between 1995 and 2005, 14 children (aged 5 to 12 years, median 8.5 years) with primary and secondary, non-parasitic splenic cysts were treated laparoscopically. The cysts were unroofed either with the LigaSure or the Harmonic Scalpel. In three patients the inner surface was coagulated with the Argon beamer. In all children the cavity was packed with omentum. In four patients the omentum was additionally sutured to the splenic parenchyma. In none the cyst wall was removed.

Results:
No intraoperative complications occurred. No inadverted splenectomy or blood transfusion was necessary. In 10 children (71%) the cysts recurred at intervals ranging from two weeks to 15 months (median 5 months). Argon laser treatment of the surface also resulted in recurrence.

Conclusions:
We conclude that laparoscopic unroofing of true splenic cysts alone is inadequate. Regardless of the surgical approach, either complete excision or removal of the inner layer is necessary to prevent recurrences.

Notes:

9 EVOLUTION OF TECHNIQUE IN THE TRANSANAL SOAVE PULL-THROUGH FOR HIRSCHSPRUNG DISEASE (3 MINUTE)

Ahmed Nasr, M.D., Jacob C. Langer, M.D.
Hospital for Sick Children, Toronto, ON, Canada

Background:
The transanal Soave pull-through has become the standard operation for Hirschsprung disease in many pediatric surgical centres. Over the past eight years, we have modified our technique by decreasing the length of the rectal muscular cuff from approximately 10-15 cm to less than 2 cm, and by doing routine intraabdominal colonic biopsies through an umbilical incision prior to beginning the anal dissection. The aim of this study was to determine if these modifications have changed the early outcomes for these children.

Methods:
Retrospective cohort study of all patients undergoing transanal pull-through by a single surgeon between 1997 and 2005. The Hospital Research Ethics Board approved this study.

Results:
Comparison of patients by cuff length is shown below:

	Short rectal cuff N=(22)	**Long rectal cuff N=(23)**	**P value**
Gender (M:F)	*15:7*	*16:7*	
Age (days)	*25 ± 23*	*139 ± 167*	***0.002***
Weight at pull through (Kg)	*3.5 ± 0.7*	*6 ± 2.7*	***0.003***
Operating time (minutes)	*167 ± 34*	*186 ± 29*	***0.05***
Length of hospital stay (days)	*1.9 ± 0.6*	*2.7 ± 0.9*	***0.001***
Enterocolitis	*2*	*7*	0.1
# requiring daily dilatation	*1*	*7*	***0.047***

Preliminary colonic biopsy through an umbilical incision was done in 18 patients and not done in 27 patients. This had no significant effect on narcotic use (67% vs. 70%, P=0.8) and did not increase operating time (174 ± 31 vs. 179± 34 minutes, P=0.6). Hospital stay was shorter in the colonic biopsy group (1.9± 0.6 vs. 2.6± 0.9 days, P= 0.006).

Conclusion:
The transanal pull-through is being done earlier and on smaller patients. Results have improved, likely due to a combination of experience and use of a shorter rectal muscular cuff. The use of a preliminary colonic biopsy through an umbilical incision permits definitive identification of the histological transition zone without increasing postoperative pain or prolonging operative time or hospital stay.

Notes:

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10 ARE THE LONG TERM RESULTS OF THE TRANSANAL PULL-THROUGH EQUAL TO THOSE OF THE TRANSABDOMINAL PULLTHROUGH? A COMPARISON OF TWO APPROACHES FOR HIRSCHSPRUNG'S DISEASE (3 MINUTE)

Mohamed I. El-sawaf, M.D., Robert A. Drongowski, Jennifer N. Chamberlain, Arnold G. Coran, M.D., Daniel H. Teitelbaum, M.D.
University of Michigan, Ann Arbor, MI, U.S.A.

Purpose:
Transanal endorectal pull-through (TERPT) is becoming the most commonly performed procedure in the treatment of Hirschsprung's disease (HD), but overstretching of the anal sphincters remains a critical issue which may impact the long-term effect on continence. This study examined the long-term outcome of TERPT vs. a conventional trans-abdominal (ABD) pull-through for HD.

Methods:
Records of 41 patients >3 year old who underwent a pull-through for HD (TREPT, n=20; ABD, n=21) were reviewed, and their families were thoroughly interviewed and scored via a questionnaire which examined continence, stooling, and enterocolitis issues. Total scoring ranged from 0-40: 0-10 excellent; 11-20 good; 21-30 fair; 31-40 poor. Patients were operated upon between 1995 and 2003, during this time our group transitioned from the ABD to the TERPT technique. Two tailed student's t-test and ANCOVA were used to analyze data at a significance level of 0.05

Results:
Results (Table) are expressed as mean ± SD.

Groups	Total Score	Continence Score	Stool pattern Score	Enterocolitis Score	Age (months) at pullthrough	Age (years) at interview
ABD n=21	11.29 ± 7.76	4.38 ± 4.77	2.71 ± 2.24	4.19 ± 2.98	8.9 ± 12.4	8.5 ± 2.3*
TREPT	12.75	8.00 ± 6.07**	1.58 ± 1.26	3.18 ± 2.23	7.4 ± 13.4	5.8 ± 2.0
n=20	± 8.07					

**P<0.01 *P<0.05

A significant difference in age at interview between the two groups was noted. To control for the influence of different covariates, ANCOVA was used for multi-regression analysis. Age at interview, sex, age at pullthrough, Trisomy-21 and aganglionosis length did not influence scoring results.

Conclusion:
Our long-term study showed significantly better (two-fold) results regarding the continence score for the abdominal approach compared to a Trans-anal Endo-Rectal Pull-through. The stool pattern and enterocolitis scores were somewhat better for the Trans-anal Endo-Rectal Pull-through group, but not significantly. These findings raise an important issue about the current surgical management of Hirschsprung disease; however, more cases will need to be studied before a definitive conclusion can be made.

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11 SHOULD PERITONEAL DRAIN (PD) BE USED AS DEFINITIVE SURGICAL THERAPY IN COMPLICATED NECROTIZING ENTEROCOLITIS (NEC) IN INFANTS WEIGHTING <1500 G OR <1000 G? (3 MINUTE)

Maria Rusan, Jr., Ahmed Nasr, Aideen Moore, Peter Kim, M.D.
The Hospital for Sick Children, Toronto, ON, Canada

Purpose:
Surgical managements of NEC in low body weight infants (<1500 g or <1000 g) remain controversial. The original intent for PD was a temporary resuscitation prior to definitive surgery. However, recent trends suggest that PD may be used as definitively therapy.
Our purpose is to determine if the length of intervening time between the placement of PD and definitive surgery, patient stratification including separate analysis of patients with isolated perforation, and the severity of illness score, SNAP-II affect the outcomes.

Methods:
A retrospective analysis of all neonates requiring surgery for NEC (n=119) between 1995 and 2005 was performed. Patients were categorized according to types of procedures performed.

Results:

	G1: LAP Only	GII:PD + LAP	GIII: PD Only	p-value
N	67	27	25	
Age(days)	16.3 ± 15.5	11.2 ± 7.6	13.4 ± 8.2	0.1
Sex (F:M)	47 :20	15:12	9:16	
Gestational age (wks)	33 ± 4.2	28.5 ± 4.2	26.6 ± 3.09	0.004
Weight(Kg)	2100 ± 681.2	1116 ± 541.9	879.1 ± 230.4	0.04
SNAP-II	21.6 ± 15.7	33.25 ± 24.1	39.08 ± 18.232	0.002
SNAP-II for the survival group	*19.3 ± 17.8*	*25 ± 14.1*	*25.6 ± 13.2*	*0.001*
SNAP-II for the Death group	*21.4 ± 16.2*	*38.7 ± 27.3*	*45.8 ± 16.8*	*0.03*

Mortality rates were 45%, 52%, and 32% in respective Groups I, II, and III. The SNAP scores were predictive of mortality and survival. The mean length of intervening time between the placement of PD and definitive surgery was 15.8 ± 19 for the survival group and 4 ± 7.7 days for the death group (p=0.05). Stratification of isolated perforation did not significantly alter the mortality or intervening time interval between PD and surgery.

Conclusions:
The use of PD may be appropriate not only as initial treatment, but as definitive surgical therapy. SNAP-II is predictive of mortality and survival in these low body weight NEC infants. Better survival associated with longer intervening time between PD and surgery further supports the role of PD as definitive surgical modality in complicated NEC.

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12 PROGRESSION OF PERIANAL ABSCESS TO FISTULA IN INFANTS: IMPLICATIONS FOR TREATMENT (3 MINUTE)

Emily R. Christison-Lagay, M.D.[1], Jason F. Hall, M.D.[1], Karen Bailey, M.D.[2], Andrew Terluk[2], Paul W. Wales, M.D.[2], Peter T. Masiakos, M.D.[1]

[1]Massachusetts General Hospital, Boston, MA, U.S.A., [2]The Hospital for Sick Children, Toronto, ON, Canada

Purpose:
To define the frequency of progression of perianal abscess to fistula with and without procedural drainage.

Methods:
A retrospective review of all patients 12 months of age or younger presenting to two tertiary care institutions over an eight-year period (January 1997-February 2005) with perianal abscess.

Results:
Of 139 children initially identified, follow-up was available on 86. Ninety-seven percent of children were male. Mean age was 3.48 months (median age two months). Of the 86 patients, 46 (53.0%) were drained and 40 (47%) were not drained. Of those patients undergoing procedural drainage, 27 (58.7%) developed a fistula, whereas of those not undergoing drainage only 10 (25%) developed a fistula ($p<0.01$). Synchronous administration of antibiotics (IV or oral) employed in 34 of 35 patients from one institution was associated with an even greater decrease in fistula formation (9.5%) in the undrained population.

Conclusions:
Perianal abscess formation in infants less than 12 months of age is a separate entity from abscess formation in older age groups. Overall rate of progression to fistula in ano remains controversial and is estimated in the literature as low as 20% and as high as 80%. Some of the discrepancy in reporting the natural history and progression of perianal abscesses originates in a lack of consensus on the efficacies of various treatment paradigms either in terms of abscess recurrence or progression of the process to fistula in ano. In the largest study to date, a combined center series of patients presenting to two academic pediatric hospitals, we reviewed the frequency of progression of perianal abscess to fistula with and without procedural drainage.

Local hygiene and systemic antibiotics without drainage minimizes formation of fistula in ano in the setting of infantile perianal abscess.

Notes:

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13 AORTA AND INFERIOR VENA CAVA RECONSTRUCTION DURING ONCOLOGIC RESECTION IN CHILDREN (3 MINUTE)

Tuan H. Pham, M.D., Ph.D., Thomas Bower, M.D., Corey Iqbal, M.D., Abdalla Zarroug, M.D., Antony Joseph, MBBS, Gustavo Oderich, M.D., Marineh Yagubyan, M.D., Audra Noel, M.D., Christopher Moir, M.D.

Mayo Clinic Rochester, Rochester, MN, U.S.A.

Purpose:

To review the outcomes of pediatric oncologic operations that required repair or reconstruction of the IVC or aorta.

Methods:

An IRB-approved retrospective review from 1985-2005 identified 17 patients ≤18 years old who underwent abdominopelvic tumor resection and required repair or reconstruction of the IVC/aorta from tumor involvement or intraoperative injury. Standard statistical methods were used for comparison and survival analyses with a p value <0.05 considered significant.

Results:

Eighty-two percent underwent repair/reconstruction of the IVC while 18% had repair of the aorta. The type of IVC injuries were venorrhaphy/laceration (57%) and controlled transection (35%), while aortic injuries were from laceration. IVC venorrhaphy/laceration injuries were repaired either by primary closure or with a patch. One-half of IVC transections were reconstructed with a polytetrafluoroethylene graft, which remained patent. All aortic injuries required cross-clamping for primary or patch repairs. Overall morbidity and mortality rates were 29% and 0%, respectively. Patients requiring aortic repair had a significantly higher morbidity compared to those with IVC repair ($p = 0.002$). Radiation therapy did not adversely affect morbidity ($p = 0.70$). Complete gross resection of tumor was achieved in 59% of patients, providing a significantly improved five-year survival compared to incomplete resection (100% vs. 43%, $p = 0.018$). Overall five-year survival was 72% with a mean of 32 ± 2 months.

Conclusion:

Through a multidisciplinary approach, oncologic resection with repair/reconstruction of the IVC/aorta can be performed safely with no perioperative deaths and low morbidity. Replacement of the IVC with a polytetrafluoroethylene graft is feasible with excellent graft patency. Patients with aortic injury had higher postoperative morbidity; however, an aggressive surgical strategy may be justified to achieve significantly better survival.

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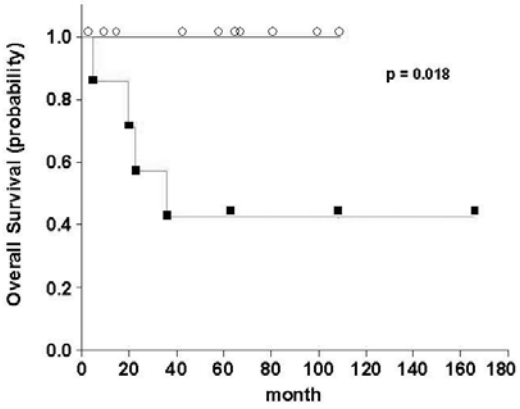

Figure 1. Kaplan-Meier plot showing overall survival of patients who underwent complete (circles) vs. incomplete (squares) gross resection of tumors with repair of IVC or aorta.

Notes:

14 PREDICTIVE VALUE OF THE PRETEXT STAGING SYSTEM IN CHILDREN WITH HEPATOBLASTOMA (3 MINUTE)

Rebecka L. Meyers, M.D.[1], Marcio H. Malogolowkin[2], Jon M. Rowland[3], Mark D. Krailo[4].
[1]University of Utah, Salt Lake City, UT, U.S.A., [2]Children's Hospital of Los Angeles, Los Angeles, CA, U.S.A., [3]Oakland Children's Hospital, Oakland, CA, U.S.A., [4]Cure Search, Children's Oncology Group, Arcadia, CA, U.S.A.

Purpose:
Recent analysis of the PRETEXT (pretreatment extent of disease) staging system developed by the International Society of Pediatric Oncology Liver Tumor Study Group (SIOPEL) found that PRETEXT is moderately accurate, has a slight tendency to overstage, is reproducible, and offers an excellent mechanism to monitor the effect of preoperative chemotherapy. This controversial recent report studied only a select subgroup of patients in SIOPEL-1, and yet it goes on to claim that PRETEXT is significantly superior to the CCSG/POG/COG staging system for predicting survival in children with hepatoblastoma. We wondered if the predictive survival advantage of PRETEXT staging could sustain a less selective analysis.

Methods:
Hepatoblastoma patients in the intergroup CCSG/POG study INT-0098 were retrospectively reviewed for determination of their PRETEXT stage at initial surgery (resection or biopsy). Detailed surgery and pathology reports enabling accurate PRETEXT staging were available for 155 of 182 patients enrolled in INT-0098. Pathologic subtype, tumor margins, repeat surgery, and AFP response were also noted.

Results:
Comparison of the staging categories for each of the two staging systems is shown in Table 1. Five-year overall survival by PRETEXT staging was I- 20/21(95%), II-41/49(84%), III-37/60(62%), and IV- 8/21(38%). Five-year overall survival by the CCSG/POG/COG staging system was Stage I, pure fetal histology- 9/9(100%), Stage I, unfavorable histology-43/45(95%), Stage II- 7/7(100%), Stage III- 37/57(65%), Stage IV- 14/37(38%). The predictive value of both systems was relatively good, and they were not significantly different from each other.

Conclusions:
Although the predictive value of PRETEXT staging remained robust, it was not superior to the staging system used by CCSG/POG/COG in predicting patient survival.

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	Resection without any Neoadjuvant ChemoTx			Resection after Neoadjuvant ChemoTx	
	Stage I Pure Fetal N=9	Stage I Unfavorable N=45	Stage II N=7	Stage III N=59	Stage IV N=37
PRETEXT I	3	12	3	1	2
PRETEXT II	4	26	4	16	5
PRETEXT III	2	7	-	29	22
PRETEXT IV	-	-	-	13	8

Notes:

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15 INTRAVASCULAR ADMINISTRATION OF TUMOR TROPIC NEUROPROGENITOR CELLS PERMITS TARGETED DELIVERY OF INTERFERON-β AND RESTRICTS TUMOR GROWTH IN A MURINE MODEL OF DISSEMINATED NEUROBLASTOMA (6 MINUTE)

Paxton V. Dickson, M.D.[1], John B. Hamner, M.D.[1], Seung U. Kim, M.D.[2], Cathy Y.C. Ng, MS[1], Karen S. Aboody, M.D.[3], Mary K. Danks, Ph.D.[1], Andrew M. Davidoff, M.D.[1]

[1]St. Jude Children's Research Hospital, Memphis, TN, U.S.A., [2]University of British Columbia, Vancouver, BC, Canada, [3]City of Hope National Medical Center, Duarte, CA, U.S.A.

Purpose:

Interferon-β (IFN-β) has potent anti-tumor activity; however, systemic toxicity has limited its clinical utility. We investigated the potential of targeted IFN-β delivery utilizing tumor-tropic neuroprogenitor cells (NPC) engineered to express IFN-β.

Methods:

Overexpression of IFN-β was established in the human NPC line F3.C1 (NPC-IFN) by adenovirus-mediated transduction. Disseminated neuroblastoma was established in SCID mice by tail vein injection of luciferase-expressing tumor cells. Fourteen days following tumor cell inoculation, systemic disease was confirmed with bioluminescence imaging (BLI). Mice were treated at this time and again 14 days later by intravenous injection of $2x10^6$ NPC-IFN or control (NPC-CTL) cells (n=9 mice/group). Progression of disease was monitored using BLI. At sacrifice, organ weights and histology further evaluated tumor burden.

Results:

Following initiation of therapy, serial BLI demonstrated a marked decrease in the rate of disease progression in mice receiving NPC-IFN. Bioluminescence, reflecting tumor burden, at day 40 was $1.67x10^9 \pm 7.91x10^8$photons/sec as compared to controls with $3.13x10^{10} \pm 4.7x10^9$photons/sec ($p<0.01$). Serum ELISA demonstrated undetectable systemic levels of IFN-β in both cohorts. At necropsy, control mice had bulky tumor replacing the liver and kidneys (liver weight 3.77±0.42grams; kidney weight 1.24±0.23grams, $p<0.01$ vs. both NPC-IFN treated and naïve mice) as well as extensive retroperitoneal and mediastinal adenopathy. Impressively, these sites within mice receiving NPC-IFN therapy appeared grossly normal with the exception of small nodules within the kidneys of some NPC-IFN treated mice (liver weight 1.61±0.09grams vs. 1.55±0.04grams in naïve mice, p=0.32; kidney weight 0.633±0.03grams vs. 0.480±0.03grams in naïve mice, p=0.02). The accumulation of NPC within sites of tumor growth was confirmed by immunofluorescence imaging.

Conclusions:

In this model of disseminated neuroblastoma, neuroprogenitor cells tropic for sites of tumor growth permitted targeted delivery of IFN-β, resulting in significant tumor growth restriction. This represents a novel approach for effective IFN-β therapy and may circumvent limitations associated with systemic toxicity.

Notes:

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16 RESECTION OF RESIDUAL MASSES AT THE END OF THERAPY FOR RHABDOMYOSARCOMA (RMS) (3 MINUTE)

David A. Rodeberg[1], Andrea Hayes-Jordan, M.D.[2], Julie Stoner, Ph.D.[3], Charles Paidas, M.D.[4], Kenneth Brown, M.D.[5], Lor Randall, M.D.[6], Eugene Wiener, M.D.[1], Doug Hawkins[7].

[1]Childrens Hospital of Pittsburgh, Pittsburgh, PA, U.S.A., [2]MD Anderson, Houston, TX, U.S.A., [3]University of Oklahoma, Tulsa, OK, U.S.A., [4]University of South Florida, Tampa, FL, U.S.A., [5]University of British Columbia, Vancouver, BC, Canada, [6]University of Utah, Salt Lake City, VA, U.S.A., [7]University of Washington, Seattle, WA, U.S.A.

Purpose:
RMS patients frequently have residual masses at the end of treatment despite receiving all planned therapy. For IRS IV children with Group III (non-metastatic, incompletely excised at presentation) RMS, we assessed resection of residual masses at the end of therapy.

Methods:
We evaluated 419 Group III children who completed all protocol therapy without developing progressive disease. Seventy-eight (19%) had a residual mass at the end of protocol therapy and 48 of these subsequently underwent resection. These patients were evaluated for their operative therapy and course.

Results:
The median size of the recurrent masses was 1.5cm (range <1cm to 15). The intent of the surgeon at the time of operation was biopsy in 27 patients and resection in the remaining 21. However the procedures actually performed were biopsy in 28, complete excision in eight, incomplete excision in 11 and one patient with no excision since no mass was found at time of exploration. During these operative procedures vital structures were removed in 1 of 28 biopsy patients (3%) also resulting in loss of function. In contrast, 10 of 19 resected patients (53%) had vital structures removed and four patients lost organ function. However, only four of 11 patients (36%) who had resection of vital structures had tumor completely resected. In addition, only nine of 48 (19%) pathologic specimens contained viable tumor. Only two of nine viable tumor patients were completely resected with negative margins. Those patients that had tumor resected had a worse survival (5-year survival 67%) compared to biopsy alone (5-year survival 96%) (p=0.003). However, these results are biased given that resected patients also had more extensive disease.

Conclusion:
Resection of masses at the end of therapy for RMS is associated with significant morbidity, rarely achieves excision of all viable tumor and may not improve survival.

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17 INDUCTION OF CYTOLYTIC T LYMPHOCYTES (CTL) AGAINST PEDIATRIC SOLID TUMORS *IN VITRO* USING AUTOLOGOUS DCS PULSED WITH NECROTIC PRIMARY TUMOR (6 MINUTE)

Joel Shilyansky, M.D., Paulette Jacobs, MA, Kara Doffek, BS, Sonia Sugg, M.D.
Medical College of Wisconsin, Milwaukee, WI, U.S.A.

Purpose:
Effective and generally applicable methods for generating cancer vaccines in children have not been defined. Dendritic cell (DC) are the most potent professional antigen presenting cells capable of activating primary cytolytic T cells. We tested the ability of DCs, generated from pediatric patient peripheral blood monocytes (PBMC) and pulsed with necrotic tumor, to activate autologous tumor-specific cytolytic T cells.

Methods:
Tumor and peripheral blood were obtained from pediatric patients undergoing biopsy or resection of advanced solid tumors according to an IRB approved protocol and after obtaining informed consent. To generate dendritic cells (DC), peripheral blood monocytes (PBMC) were treated with GM-CSF and IL-4. Maturation was induced with a cytokine cocktail containing TNFÎ±, IL-6, IL-1Î² and PGE_2 (CC). DC phenotype was assayed using flow cytometry. Tumor necrosis was induced by exposure to UV-B irradiation (1000 mJ). DCs, pulsed with UV-B treated primary tumor and matured with CC were used to stimulate autologous peripheral blood lymphocytes (PBL) weekly. Tumor-specific cytolytic activity was assayed using 4-hour ^{51}Cr release after three or four weeks.

Results:
PBMCs isolated from pediatric patients differentiated into immature DCs ($CD14^-$, $MHCII^+$, $CD80^{low}$, $CD86^{low}$) in the presence of GM-CSF and IL-4. CC induced maturation of DCs, characterized by increased expression of MHCII, CD83, CD80, and CD86. Patient PBL, stimulated *in vitro* with DCs loaded with necrotic primary tumor and matured with CC, specifically lysed autologous NB in seven of nine patients.

Conclusion:
DCs, generated from peripheral blood of children with advanced solid tumors and pulsed with necrotic primary tumor, undergo maturation and effectively stimulate autologous tumor-specific cytolytic T cells *in vitro*. We describe a simple method for generating a vaccine capable of activating CTL against pediatric solid tumors that does not require the genetic identification of tumor-associated antigens.

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18 HEPATIC HEMANGIOMAS: SUB-TYPE CLASSIFICATION AND DEVELOPMENT OF A CLINICAL PRACTICE ALGORITHM AND REGISTRY (6 MINUTE)

Emily R. Christison-Lagay, M.D., Patricia E. Burrows, M.D., Steven J. Fishman, M.D.
Children's Hospital, Boston, Boston, MA, U.S.A.

Purpose:
Hepatic hemangiomas, though histologically benign, may be associated with significant morbidity and mortality in afflicted infants. The literature presents much confusion regarding the natural history and treatment options for hepatic hemangiomas. Clinical manifestations range from asymptomatic, self-limiting lesions to congestive heart failure associated with high volume vascular shunting to fulminant hepatic failure with hypothyroidism, abdominal compartment syndrome, and death. There has been little rationale to choose among observation, corticosteroid, other pharmacologic agents, arterial embolization, hepatic artery ligation, resection, or liver transplantation for any given patient.

Methods:
We analyzed several recent retrospective radiologic analyses and pathologic studies to determine whether hepatic hemangiomas could be categorized, allowing prediction of their natural history and rational choice of therapies based upon their clinical presentation and radiographic appearance.

Results:
We propose that hepatic hemangiomas do not represent a single entity, but rather three principle categories of lesions: focal, multifocal, and diffuse. As these three categories represent different anatomic and physiologic variants so, too, may they respond differently to previously anecdotally applied treatment regimens. With input from international multidisciplinary authorities on hemangiomas, we developed and propose a clinical practice algorithm for the evaluation and management of hepatic hemangiomas (Figure 1). Toward that end we propose a plan to institute a web-based international hepatic hemangioma registry. Participants in the registry could obtain no-cost centralized review of imaging studies (and histology if available) and guidance regarding the management algorithm from an established multidisciplinary team. Regular follow-up clinical and imaging information will be detected.

Conclusion:
Longitudinal observation of response to more directed treatment protocols may contribute greatly to the understanding of these potentially fatal tumors.

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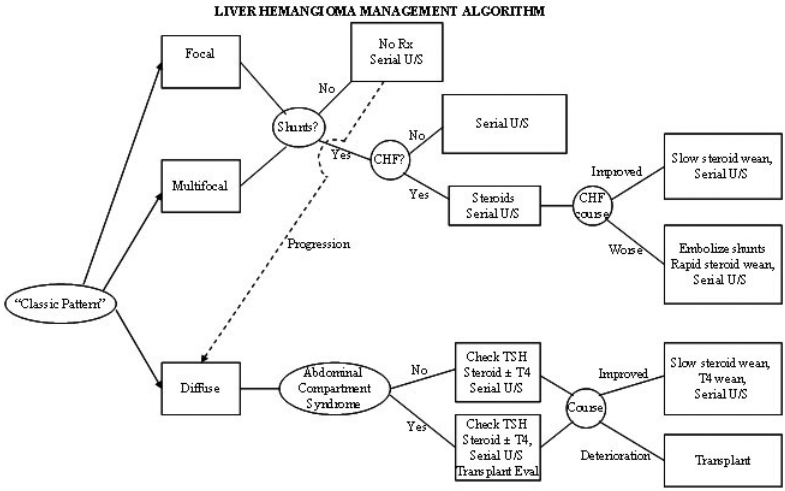

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19 ETHIBLOC SCLEROTHERAPY FOR TREATMENT OF LYMPHANGIOMAS DOES NOT COMPLICATE SURGICAL RESECTION (3 MINUTE)

Mohammad Ali Emran, Salam Yazbeck, M.D., Louise Caouette Laberge, M.D., Ayman Al-Jazaeri, Chad Allan Wiesenauer, Josée Dubois.
Ste-Justine Hospital, Montreal, PQ, Canada

Purpose:
To report the experience of excision of residual lymphangiomas after Ethibloc sclerotherapy.

Introduction:
Sclerotherapy for treatment of lymphangiomas has been slow to be accepted at many institutions despite low complication rate and excellent results. One major argument against acceptance of sclerotherapy is that failure of treatment will be associated with a more difficult operative resection because of the inflammatory reaction surrounding the lesion. The experience with surgical intervention following treatment has not been reported except anecdotally in the literature.

Methods:
Charts were reviewed of patients undergoing Ethibloc sclerotherapy and subsequent surgery. Factors including blood loss, operative time, complications and outcome were evaluated.

Results:
Eight patients underwent excision of residual lesions after Ethibloc sclerotherapy between 1992 and 2006. Three patients had cervical lymphangiomas, one axillary, one thoracic wall, one combined cervical/ intra-thoracic and one lower extremity. Lesions measured 35-3803 cm^3 (average 877 cm^3). Patients had two to four preoperative sclerotherapy attempts (average 2.6) and one to two surgical interventions (different sites) after sclerotherapy. Patient ages at the time of surgery ranged from 5 months to 11 years 8 months (average 3 years 11 months). Time between sclerotherapy and surgery ranged from 4 months to 3 years (average 1 year 5 months). Operative time averaged 4hrs 11min (range 1hr- 8hr 40min). Blood loss averaged 101ml (range 5-500cc). Dissection was described as extensive by surgeons, often involving major nervous or vascular structures, but easier than similar cases not having sclerotherapy. Three patients underwent sclerotherapy for postoperative seromas. No nerve injuries were noted.

Conclusion:
Surgery for residual lymphangiomas after Ethibloc sclerotherapie is not associated with increased morbidity or difficulty of surgery. Dissections were often made easier as reported by operating surgeons.

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20 PATENT DUCTUS ARTERIOSUS (PDA) LIGATION IN PREMATURE INFANTS: WHO REALLY BENEFITS, AND AT WHAT COST? (3 MINUTE)

Mehul V. Raval, M.D.[1], Matthew M. Laughon, M.D.[2], Carl L. Bose, M.D.[2], J. D. Phillips, M.D.[1]

[1]Department of Surgery, Division of Pediatric Surgery, University of North Carolina at Chapel Hill, Chapel Hill, NC, U.S.A., [2]Department of Pediatrics, Division of Neonatal-Perinatal Medicine, University of North Carolina at Chapel Hill, Chapel Hill, NC, U.S.A.

Purpose:
Patent ductus arteriosus (PDA) ligation in premature infants has been shown to have low surgical morbidity and mortality. Ligation goals include: prompt improvement in cardio-respiratory failure, with rapid wean from mechanical ventilation; less risk of prolonged mechanical ventilation and subsequent chronic lung disease (CLD); and survival to discharge. This study was designed to examine true morbidity after ligation and elucidate which preoperative factors might predict favorable outcomes.

Methods:
IRB-approved retrospective review of 197 infants, <38 weeks gestational age (GA), undergoing PDA ligation via thoracotomy between January 1, 1992 and January 1, 2004. CLD defined as need for supplemental oxygen at 36 weeks corrected GA. Student's t-test and chi-squared tests were employed.

Results:
Mean GA was 27 weeks (range: 23-35wks), birth weight (BW) was 957 grams (range: 440-3170 grams); infants underwent ligation at 16 days of life (range: 1-132 days). Duration of surgery was 50.5 minutes (range:13 to 150 min.). Mean postoperative times were: 27 days to extubation, 60 days to wean from supplemental oxygen, and 84 days to discharge. Early extubation (within 10 days of ligation) occurred in only 54 patients (30%). Only 44 (22%) survived to discharge without CLD. Forty patients (20%) died, with respiratory failure the most common cause (70%). In general, early extubation, survival without CLD, and survival to discharge were associated with: greater GA and BW, higher APGAR scores, greater age and weight at surgery, no preoperative IVH, lack of ventilator dependence, and lower ventilator settings ($p<0.05$). Amount/duration of indomethacin use, chest X-ray findings, and echocardiographic assessment of ductus size did not predict favorable outcomes (all $p>0.05$).

Conclusions:
Most premature infants currently undergoing PDA ligation at our institution do not experience the anticipated rapid improvements in cardiorespiratory status and go on to develop CLD. Few preoperative variables (including radiographic and echocardiographic assessment) definitively predict outcomes.

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21 VERTICAL EXPANDABLE PROSTHETIC TITANIUM RIB (VEPTR) FOR THORACIC INSUFFICIENCY SYNDROME (TIS) (6 MINUTE)

John H.T. Waldhausen, M.D., Gregory J. Redding, M.D., Kit M. Song, M.D.
Children's Hospital, Seattle, WA, U.S.A.

Purpose:
TIS is a group of congenital chest wall disorders that produce progressive respiratory restrictive disease. The VEPTR thoracoplasty is a new technique devised for the treatment of these children. This study describes our initial experience with this devise.

Methods:
This is a review of all patients undergoing VEPTR placement at a single institution between 2001-2005. This is an IRB approved study, number 00-0161-05.

Results:
Under an FDA study protocol all 21 patients had a total of 32 VEPTR devises placed. All were seen and approved for surgery by a pediatric surgeon, pulmonologist and orthopedic surgeon. Two patients had Jeunes syndrome and 19 had scoliosis, most with associated CO2 retention, pulmonary restrictive disease and respiratory failure. Associated conditions included ventilator dependence, arthrogryposis, VACTERL, CDH, flail chest, meningomyelocele, nutritional deficiency, and myopathy. Nine required gastrostomy tubes. Eleven patients had multiple fused ribs requiring opening thoracostomy to promote chest expansion. All but the two most recent patients have undergone sequential VEPTR expansion between one and seven times. There were no intraoperative complications and blood loss was minimal. No child required transfusion. All children had intraoperative spinal cord monitoring (SSEP). Four experienced intraoperative SSEP changes, but with lessened VEPTR expansion, all SSEP signals normalized. No neurologic deficits have resulted. Seven VEPTR devises have been revised for erosion through the bone or dislodgment, three have been removed. Five have been outgrown and either removed or replaced. One eroded through the skin. One became infected. Approximately 2/3 of children with CO2 retention pre-VEPTR had CO2 reduction post-VEPTR.

Conclusions:
1. TIS is often part of a broad constellation of congenital anomalies 2. VEPTR is a new method to treat children with TIS. 3. VEPTR can be placed safely with acceptable morbidity. 4. Preliminary results indicate that VEPTR may decrease CO2 retention in some patients.

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22 MAGNETIC MINI-MOVER PRODCURE (3MP) FOR PECTUS EXCAVATUM: I. DEVELOPMENT, DESIGN, AND SIMULATIONS FOR FEASIBILITY (FORCE GENERATED) AND SAFETY (MAGNETIC FIELD STRENGTH NEAR THE HEART) (3 MINUTE)

Michael R. Harrison, M.D., Denise Estefan-Ventura, M.D., Richard Fechter, BS, Arthur M. Moran, Jr., BA, Darrell Christensen, BA, MA.
University of California, San Francisco, San Francisco, CA, U.S.A.

Objectives:
Both the Nuss and modified Ravitch procedures can successfully correct pectus excavatum by forcing the sternum forward in one step and holding it in place under pressure with a metal strut. Although the abnormal costal cartilage will eventually remodel (1-2 years), a better way to achieve this change would be gradual correction using minimal force applied continuously over time (like remodeling teeth with orthodontic braces).

Methods:
We developed a device (magnimplant) in which a $1^1/_2$-inch diameter rare earth magnet with a ferromagnetic backing plate is encapsulated in a $1^3/_4$-inch diameter titanium case. The device can be attached to the sternum through a 2-inch substernal incision in a brief outpatient procedure. We tested the implantation and fixation in human cadavers.

We developed an external magnet (magnatract) that is fitted in a custom-molded orthotic device and held in place over the pectus defect by magnetic attraction to the implanted magnet. We measured the force applied to the sternum when the distance between the magnets is adjusted in small increments to produce gradual correction of the deformity. We also mapped the magnetic field in an anatomic simulation in order to measure the highest field strength that could reach the heart.

Results:
The outward force produced is plotted against the distance between the implanted and external magnets. The force at 2 cm is approximately 1.5 kg, adequate to move a pectus deformity approximately 1 cm. The magnetic field map measured in the two-magnet configuration is drawn as isobars. The maximum field strength reaching the surface of the heart is 0.02 T, well below the safety limit (4 T).

Conclusions:
Simulations suggest the 3MP is effective and safe, and can be developed for children with pectus excavatum.

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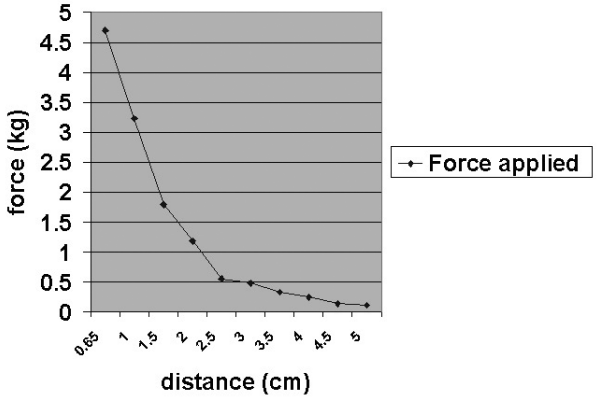

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23 INFECTIOUS COMPLICATIONS AFTER THE NUSS REPAIR IN A SERIES OF 863 PATIENTS (3 MINUTE)

Susanna Shin, M.D.[1], Michael J. Goretsky, M.D., I-EVMS[2], Robert Kelly, M.D., I-EVMS[2], Tina Gustin, RN[1], Donald Nuss, M.D., I-EVMS.[2]

[1]Eastern Virginia Medical School, Norfolk, VA, U.S.A., [2]Children's Hospital of the Kings Daughters, Norfolk, VA, U.S.A.

Purpose:

A nemesis of surgical implants is infection. We evaluated the various infectious complications after Nuss repair of pectus excavatum in 863 patients over 18 years.

Methods:

After IRB approval, a retrospective review of a prospectively gathered database was performed of patients who underwent minimally invasive repair of pectus excavatum and developed an infection. All patients received intravenous antibiotics prior to surgery continuing until discharge. Patients with a persistent fever after operation were discharged with oral antibiotics.

Results:

From January 1987 to September 2005, 863 patients underwent a minimally invasive pectus excavatum repair and 13 (1.5%) developed post-operative infections. These included six bar infections, four cases of cellulitis, and three stitch abscesses. Cellulitis was defined as erythema and warmth, which responded to a single course of antibiotics. Bar infections were defined as an abscess in contact with the bar. Surgical drainage and antibiotics resolved four of these abscesses (66.7%) while two patients (33.3%) required early bar removal (after 18 months). Cultures identified a single organism in each case and Staphylococcus aureus was the most common organism (83%) identified, and all being Methicillin sensitive (MSSA). All infections occurred on the side of the stabilizer if a stabilizer had been placed.

Conclusions:

Infectious complications after Nuss repair are uncommon and occurred in 1.5% of patients. Published rates of post-operative infection range from 1.0% to 6.8%. Superficial infections responded to antibiotics alone. Bar infection occurred in only 0.7% and required surgical drainage and long-term antibiotics. Only two of these patients (33.3% of bar infections) required early bar removal at 18 months due to recurring infections. Early bar removal should be a rare morbidity with the Nuss repair.

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24 WHEN IT'S NOT AN INFECTION: METAL ALLERGY AFTER THE NUSS PROCEDURE FOR REPAIR OF PECTUS EXCAVATUM (3 MINUTE)

Gregory Rushing, M.D.[1], Michael J. Goretsky, M.D., I-EVMS[2], Maripaz Morales, M.D.[2], Robert Kelly, M.D., I-EVMS[2], Tina Gustin, RN[2], Donald Nuss, M.D., I-EVMS.[2]

[1]Eastern Virginia Medical School, Norfolk, VA, U.S.A., [2]Children's Hospital of the Kings Daughters, Norfolk, VA, U.S.A.

Purpose:

Increasing use of implantable bars for minimally invasive pectus excavatum repair has introduced metal allergy (nickel and chromium) to pediatric surgeons. This is a well recognized entity in neurologic, orthopedic, and craniofacial surgery. This study was performed to evaluate metal allergy and its effects on treatment with the Nuss procedure in 862 patients.

Methods:

After IRB approval, we undertook a retrospective review of a prospectively gathered database of patients undergoing the Nuss procedure. Metal allergy was diagnosed with use of dermal patch; or clinically, based on rash, fever, elevated erythrocyte sedimentation rate, cultures, and pathology specimens. Data collection included demographics, an allergy to jewelry, or history of atopy. Clinical outcomes including need for re-operation, removal of stainless steel bar, and replacement with titanium bar were evaluated.

Results:

Over an 18-year period (1987-2005) 862 patients underwent the Nuss procedure. Nineteen (2.2%) were diagnosed with metal allergy, with an average age of 14.7 (9-23 years). Eighteen (95%) were males. A history of atopy was present in eight (42%) patients. Ten (63%) patients presented with a rash and erythema, one (6%) with granuloma, and five (32%) with effusion. Stainless steel bars were removed due to allergic skin breakdown in three patients. In two patients, replacement titanium bars were required. In all three of these patients, symptoms resolved after removal of stainless steel bars. Titanium bars were placed in three patients without event, after diagnosis with metal allergy on pre-operative screening.

Conclusions:

Allergy symptoms often are misdiagnosed as infection, but require different treatment. If a history of metal allergy or atopy is suggested preoperatively, the patient should be tested for metal allergy and if positive, a titanium bar used. Because the consequences of metal allergy may include the need to replace the bar, pediatric surgeons should be aware of this occurrence.

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25 *EX UTERO* INTRAPARTUM TREATMENT WITH PLACEMENT ON EXTRACORPOREAL MEMBRANE OXYGENATION (EXIT-TO-ECMO) FOR SEVERE CONGENITAL DIAPHRAGMATIC HERNIA (3 MINUTE)

Shaun M. Kunisaki, M.D.[1], Carol E. Barnewolt, M.D.[1], Judy A. Estroff, M.D.[1], Laura B. Myers, M.D.[1], Dario O. Fauza, M.D.[1], Louise E. Wilkins-Haug, M.D., Ph.D.[2], Ian A. Grable, M.D.[3], Luanne P. Nemes, RN, MS, PNP[1], Terry L. Buchmiller, M.D.[1], Jay M. Wilson, M.D.[1], Russell W. Jennings, M.D.[1]

[1]Children's Hospital Boston, Boston, MA, U.S.A., [2]Brigham and Women's Hospital, Boston, MA, U.S.A., [3]Beth Israel Deaconess Medical Center, Boston, MA, U.S.A.

Purpose:
Survival rates in severe congenital diaphragmatic hernia (CDH) remain below 50% in most major series. The purpose of this study was to determine whether *ex utero* intrapartum treatment with placement on extracorporeal membrane oxygenation (EXIT-to-ECMO) might lead to improved outcomes in this setting.

Methods:
A retrospective review of all prenatally diagnosed CDH patients evaluated between September 1999 and July 2005 was performed (n=90). Severe CDH was defined by liver herniation with a lung-to-head ratio (LHR) below 1.4, a percent predicted lung volume less than 15 by magnetic resonance imaging, and/or congenital heart disease by echocardiogram. Cases satisfying these criteria were offered *ex utero* intrapartum treatment (EXIT) with a trial of ventilation while on placental support. Fetuses with preductal oxygen saturations below 90% were placed on extracorporeal membrane oxygenation (ECMO) prior to delivery.

Results:
Fourteen fetuses with severe CDH underwent the EXIT procedure. The LHR was 1.09±0.45, and the percent predicted lung volume was 12.9±8.2. Four (28.6%) were diagnosed with congenital heart disease. The uteroplacental bypass time was 34.2±23.4 minutes, and there were no maternal complications. Three patients passed the ventilation trial and survived, but two of them required ECMO within 48 hours of life. The remaining 11 (78.6%) EXIT patients were placed on ECMO prior to delivery (ECMO time, 216.1±136.1 hours). Overall survival after EXIT-to-ECMO was 63.6%. Among survivors, time on mechanical ventilation and length of hospitalization were 27.1±10.8 and 98.3±32.5 days, respectively. At 1-year follow-up, all survivors had weaned off of supplemental oxygen, but 57.1% required diuretics and/or bronchodilators.

Conclusions:
This is the largest reported series utilizing *ex utero* intrapartum treatment with placement on extracorporeal membrane oxygenation for fetuses with severe congenital diaphragmatic hernia. Favorable survival rates can be achieved with acceptable pulmonary morbidity in a high-risk group of patients. Further analysis of this approach is warranted.

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26 FETAL LUNG-HEAD RATIO (LHR) IS NOT RELATED TO OUTCOME FOR ANTENATAL DIAGNOSED CONGENITAL DIAPHRAGMATIC HERNIA (CDH) (3 MINUTE)

Marc S. Arkovitz, Patricia Devine, M.D., Mark Russo, M.D., Nancy Budhorick, M.D., Charles J. Stolar, M.D.
Morgan Stanley Children's Hospital of New York-Presbyterian, New York, NY, U.S.A.

Introduction:
Pre-natal counseling for patients with diagnosed CDH is uncertain. LHR may identify poor prognosis fetuses and potential candidates for fetal intervention. LHR for prognosis is advocated primarily in fetal intervention centers, but not validated elsewhere.

Purpose:
We examined the prognostic significance of ante-natal LHR for post natal outcome. Specifically, we asked if LHR $\leq$ 1.0 had a different survival or need for ECMO compared to LHR >1.0.

Methods:
All antenatal patient records with a diagnosis of CDH from January of 2002 to June of 2005 were examined. Inclusion criteria were: isolated left sided CDH, no evidence of significant congenital heart disease or other anomalies/syndromes who were delivered and treated solely at this institution. LHR was determined with ultrasound by previously published methods. LHR values were compared based on the threshold currently proposed for fetal intervention: LHR$\leq$1.0 vs. LHR >1.0. Outcome was assessed as survival, defined as discharge to home, or need for ECMO. Data was analyzed by chi-square with $p<0.5$ statistically significant. The study was approved by the university Institutional Review Board.

Results:
Twenty-eight patients met inclusion criteria. Overall survival was 86% (24/28). Post-natal survival in fetuses with LHR $\leq$1.0 (8/11) was not statistically different from LHR >1.0 (16/17) [73% vs. 94%, p=0.114]. The need for ECMO in the group with LHR $\leq$1.0 (3/11) was greater but not significantly different than those with LHR >1.0 (1/17) [27% vs. 6%, p=0.114].

Conclusions:
In this experience, LHR does not predict survival, however, it may identify high risk fetuses that will require ECMO. While survival with isolated left sided CDH and LHR $\leq$1.0 may not identify a mortality rate high enough to warrant fetal intervention it may identify patients at risk of needing ECMO and antenatal referral to an ECMO center.

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27 FETAL TRACHEAL OCCLUSION IN PATIENTS WITH SEVERE CONGENITAL DIAPHRAGMATIC HERNIA: A MORPHOLOGIC STUDY OF LUNG ARCHITECTURE AND ARTERIAL MUSCULARIZATION (3 MINUTE)

Enrico Danzer, M.D., Marcus G. Davey, Ph.D., Portia Kreiger, M.D., Eduardo D. Ruchelli, M.D., Mark P. Johnson, M.D., N. Scott Adzick, M.D., Alan W. Flake, M.D., Holly L. Hedrick, M.D.

The Children's Hospital of Philadelphia, Philadelphia, PA, U.S.A.

Objective:

To determine the effect of fetal tracheal occlusion (TO) on lung architecture and arterial muscularization in non-survivors of severe congenital diaphragmatic hernia (CDH).

Material:

Fifteen fetuses underwent TO for severe CDH, with five survivors (AJOG 183:1059, 2000). Paraffin embedded lung specimens from seven of the 10 non-survivors (CDH-TO) and six age-matched fetuses (CDH) were available for morphometric analysis, which included measurements of point fraction of lung parenchyma (Pp) and surface density (Sv). Pulmonary arteries (PA) were categorized according to size ($<60\mu m$, $60\leq120\mu m$, $120\leq240\mu m$, and $>240\mu m$). External diameter and medial wall thickness were measured to calculate percent medial thickness (%MT).

Results:

Gestational age (GA) at TO was 27.6±0.9 weeks with a mean duration of TO of 32.6±6.8 days. GA at delivery (CDH-TO 31.9±0.9 vs. CDH 35.4±1.8 weeks, p=0.18) and postnatal survival time (CDH-TO 20.5±6.0 vs. CDH 18.6±7.8 days; p=0.85) were not different between groups. TO significantly increased lung-weight-to-body-ratio (CDH-TO 13.0±2.2 vs. CDH 6.6±0.9; p=0.02). TO tended to decrease right-lung Pp (CDH-TO 54.6±2.6 vs. CDH 65.7±5.9, p=0.05), whereas left-lung Pp remained similar between groups (CDH-TO 63.0±3.5 vs. CDH 66.7±4.1, p=0.51). Sv was not different between left (CDH-TO 171.3±16.1 vs. CDH 151.1±8.1, p=0.34) and right (CDH-TO 172.0±10.6 vs. CDH 160.8±3.6, p=0.33) lung. %MT in all left-lung PA and larger right-lung PA ($\geq120\mu m$) subgroups was significantly reduced following TO. %MT of the small right-lung PA ($\leq60\mu m$) was significantly increased (Table 1).

Conclusions:

Despite accelerated lung growth, non-survivors of fetal TO did not have differences in lung morphometry relative to non-survivors of severe CDH. However, TO appears to selectively improve PA medial thickness in the non-survivor group.

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Table 1.

%MT		CDH-TO	CDH	P value
≤60μm	left lung	43.8±2.0	50.6±1.7	p=0.02
	right lung	52.5±1.1	46.9±1.3	p<0.0001
60≤120μm	left lung	29.2±1.1	41.4±2.2	p<0.0001
	right lung	41.9±1.2	43.2±1.6	P=0.5
120≤240μm	left lung	21.5±1.4	43.4±1.8	p<0.0001
	right lung	36.1±2.9	48.0±2.9	p=0.006
>240μm	left lung	15.8±1.6	42.8±5.4	p<0.0001
	right lung	34.2±3.9	46.4±4.0	p=0.04

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28 PREDICTING INADEQUATE LONG-TERM LUNG DEVELOPMENT IN CHILDREN WITH CONGENITAL DIAPHRAGMATIC HERNIA: AN ANALYSIS OF LONGITUDINAL CHANGES IN VENTILATION AND PERFUSION (3 MINUTE)

Melissa J. Hayward, M.D., Virginia Kharasch, M.D., Catherine Sheils, M.D., Sandra Friedman, M.D., Mary-Jo Dunleavy, R.N., Sherri Utter, RND, David Zurakowski, Ph.D., Russell Jennings, M.D., Jay M. Wilson, M.D.
Children's Hospital Boston, Boston, MA, U.S.A.

Purpose:
Infants born with congenital diaphragmatic hernias (CDH) demonstrate a wide variability in post-natal "catch-up" lung growth, the least successful of whom have long term pulmonary morbidity. The purpose of this study was to assess pulmonary development utilizing sequential ventilation-perfusion (VQ) studies during childhood and to identify perinatal factors associated with a persistent VQ mismatch.

Methods:
The records of 137 patients seen in a multidisciplinary CDH clinic between 1990 and 2005 were reviewed. The 46 patients identified as having multiple VQ studies were used to compare changes in their ipsilateral ventilation-perfusion quotient (V/Q) over time with the variables: gender, laterality, ECMO, patch repair, birth weight, gestational age, duration of intubation, and length of stay. An abnormal V/Q (principally due to perfusion deficit) is defined as greater than 1.2, normal being 0.8 to 1.2. Data was analyzed utilizing logistic and linear regression models.

Results:
Several variables were found to significantly impact the likelihood and magnitude of prolonged VQ mismatch. The most influential of these factors were patch repair, initial V/Q, and ECMO. Patients with a higher (worse) initial V/Q continued to have a persistently high V/Q on subsequent scans ($p=0.02$). Patch repair was associated with a nearly seven times higher risk of persistent VQ mismatch ($p<0.001$). ECMO had a significant impact on long term VQ mismatch ($p=0.03$), but the effect was dependent of the side of the hernia.

Conclusions:
CDH infants with an abnormal V/Q tend to remain abnormal. While several other variables were predictive of long term VQ mismatch, the presence of a patch repair was by far the most significant. It is unclear whether this represents a greater degree of initial pulmonary hypoplasia, or subsequent impaired development driven by a non-functioning prosthetic patch. What is clear is that this group is at particular risk and requires long-term pulmonary follow-up.

Notes:

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29 MORTALITY IN INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA (CDH) ON EXTRACORPOREAL MEMBRANE OXYGENATION (ECMO): A REPORT FROM THE CONGENITAL DIAPHRAGMATIC HERNIA STUDY GROUP (CDHSG) (3 MINUTE)

Rupa Seetharamaiah, M.D., John G. Younger, M.D., Robert H. Bartlett, M.D., Ronald B. Hirschl, M.D.

University of Michigan, Ann Arbor, MI, U.S.A.

Purpose:

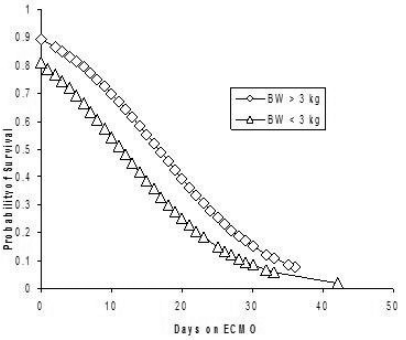
To identify factors determining mortality with CDH on ECMO and the time point where treatment is futile.

Methods:

We retrospectively analyzed the data on 3,100 CDH patients in the CDHSG from 82 participating pediatric surgical centers (1995-2004). Data regarding days on ECMO, survival, birthweight, APGAR, cardiac disease, second ECMO run, and prenatal diagnosis were analyzed. Survival analysis was done using logistic regression. Statistical significance was defined as $p<0.05$.

Results:

A total of 1,069 (35%) patients from the registry were identified as CDH patients managed with ECMO. Overall survival for the entire CDH group was 67% and for those on ECMO was 52% ($p<0.001$). When compared to survivors, nonsurvivors were more immature (37±2 vs. 38±2 weeks; $p<0.01$), had lower birthweights (2.9±0.5 vs. 3.2±0.5 kg; $p<0.001$), were more often prenatally diagnosed (63% vs. 53%; $p<0.01$), and were on ECMO longer (12.21±5 vs. 8.99±5 days; $p < 0.001$). We developed a logistic regression model which demonstrated that birthweight ($p<0.001$), APGAR at five minutes ($p<0.001$), days on ECMO ($p<0.001$), associated major cardiac abnormality ($p<0.014$), prenatal diagnosis ($p=0.008$) and second ECMO run ($p=0.002$) were found to independently predict mortality. Probability of survival graphs were created for birthweight, APGAR at five minutes, major cardiac abnormality, and second run by day on ECMO (See figure). A simplified model which allows clinicians to calculate the estimated survival based on day on ECMO, birthweight, and APGAR was also developed.



Conclusions:

Factors which determine mortality in infants with CDH on ECMO include birthweight, APGAR at five minutes, the number of days on ECMO, associated major cardiac anomalies, prenatal diagnosis, and a second ECMO run. This model can be used to predict estimated survival for individual patients thus facilitating decision-making with regard to futility in CDH patients on ECMO.

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30 FETAL INTERVENTION FOR MASS LESIONS AND HYDROPS IMPROVES OUTCOME: A 15-YEAR EXPERIENCE (6 MINUTE)

Erich J. Grethel, Amy J. Wagner, M.D., Matthew S. Clifton, M.D., Raul A. Cortes, M.D., Diana L. Farmer, M.D., Michael R. Harrison, M.D., Robert H. Ball, M.D., Kerilyn K. Nobuhara, M.D., Hanmin Lee, M.D.

University of California, San Francisco, San Francisco, CA, U.S.A.

Purpose:

The natural history of certain prenatally diagnosed masses is well known. In congenital cystic adenomatoid malformation of the lung (CCAM), the lesion can proceed one of two ways, either to regress and cause minimal morbidity, or to progress and enlarge, often resulting in hydropic changes in the fetus. Non-immune hydrops carries a dismal prognosis, with nearly all fetuses expiring before or shortly after birth. However, hydrops associated with fetal mass lesions can be halted and even reversed with fetal intervention and treatment of the underlying defect. We examined our patients with fetal mass lesions to evaluate survival after intervention.

Methods:

Institutional approval was obtained by the Committee on Human Research. A retrospective review was performed of 268 fetuses evaluated over 15 years with large mass lesions. All patients were evaluated for evidence of fetal hydrops using ultrasound criteria. Patients were divided according to type of intervention. Primary outcome measure was 30-day survival after birth.

Results:

1) Patients without fetal hydrops did not undergo fetal intervention and survived to 30 days after birth (152/158, 96%). 2) Patients with fetal mass lesions that developed hydrops fared poorly with no intervention (3/34 survival, 9%), whereas fetuses undergoing prenatal intervention fared much better (14/28 open, 50%, 3/9 percutaneous, 33%).

Conclusions:

Fetuses with prenatal diagnoses of masses not associated with hydrops have excellent prognosis with survival >95%. Non-immune hydrops associated with prenatal diagnosis of a fetal mass is a devastating complication with <10% survival. Open resection of a mass causing hydrops resulted in 50% survival, with reversal of hydrops in a group with near uniform fatality.

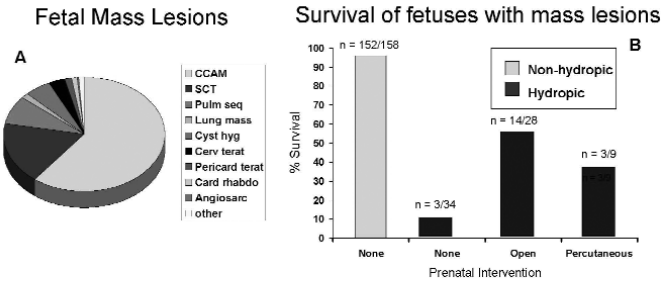

Figure 1: **A:** Breakdown of 250 mass lesions evaluated over 15 year period. **B:** Survival of non-hydropic and hydropic fetuses with mass lesions according to treatment strategy

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31 INCREASED HERITABILITY OF CERTAIN TYPES OF ANORECTAL MALFORMATIONS (3 MINUTE)

Richard A. Falcone, Jr., M.D., Marc A. Levitt, M.D., Alberto Peña, M.D., Michael Bates, M.D., Ph.D.
Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A.

Purpose:
Anorectal malformations (ARM) represent a diverse spectrum of abnormalities, and their etiology is unclear. Human and animal studies have pointed to genetic causes, so we studied patterns of heritability in a large case series.

Methods:
We searched our ARM database for all patients having family members with any congenital anomalies. This group of patients was analyzed to determine the type of ARM and the specific anomalies in affected family members.

Results:
Out of 1,606 patients with ARM, 39 (2.4%) had a family member with a congenital anomaly. The male-to-female distribution was 1:1.3, similar to the 1:1.1 ratio for the entire series. Six children (0.4%) were part of multigeneration families with anomalies. The associated non-ARM anomalies included four sacral masses, three gynecologic anomalies, two sacral anomalies, two hematologic anomalies, one esophageal and one duodenal atresia, one single kidney, one meningocele and one sibling with Down's syndrome. Twenty-four patients (1.4%) had one or more family members with an ARM. Familial ARMs were 2.4-fold more common in females. Among females with a positive family history, 73% of patients had either a vestibular or perineal fistula, compared to only 36% in patients without a family history (p=0.0004). Among males, 35% had perineal fistulas compared to only 10% of those without affected family members (p=0.0051).

Conclusions:
The incidence of ARM in the general population is 2-5 per 10,000 live births, so the presence of a positive family history in 1.4% is supportive of a strong genetic component to ARM. The risk of having an affected family member is significantly increased in the presence of a vestibular or perineal fistula (females) and perineal fistula (males). These new data allow for more informed counseling of families with an ARM and support the need for further genetic studies.

Notes:

32 ETHICS AND THE PEDIATRIC SURGEON (9 MINUTE)

Mary E. Fallat, M.D.[1], Donna A. Caniano, M.D.[2], Annie H. Fecteau, M.D.[3]
[1]University of Louisville, Louisville, KY, U.S.A., [2]Children's Hospital, Columbus, OH, U.S.A., [3]University of Toronto, Toronto, ON, Canada

Purpose:
Care of infants and children with life-impairing or life-threatening congenital and acquired disorders often raises ethical concerns for pediatric surgeons. The purpose of this survey was to determine the level of interest in clinical ethics and how respondents would manage ethical dilemmas within several clinical case scenarios.

Methods:
A 12-item validated questionnaire developed by the APSA Ethics and Advocacy Committee was provided for the APSA membership on the organizational Web site. General categories of questions included informed consent, patient privacy and what constitutes research.

Results:
The survey was completed by 235 of the 825 APSA members; a response rate of 28.4%. The majority (62%) were in academic practice, 22% had additional education or an advanced degree in ethics and 11% were members of a hospital ethics committee. There was a clear majority response for seven questions. Topics generating the most controversy included the impact of consent by minors, decision-making in the neurologically devastated child, what constitutes research in pediatric surgery, the use of interpreters for consent, and patient privacy. Respondents chose a well-referenced manuscript as the preferred modality for ethics education of the APSA membership.

Conclusions:
Pediatric surgeons have a general interest in clinical ethics as it relates to the care of their patients. An important mission of the APSA Ethics and Advocacy Committee can be to provide education that gives guidance and knowledge to the membership of APSA on timely topics in surgical ethics.

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33 REVERSAL OF TYPE 2 DIABETES AND IMPROVEMENT IN DYSLIPIDEMIA AND BLOOD PRESSURE FOLLOWING SURGICAL WEIGHT LOSS IN ADOLESCENTS: A MULTICENTER STUDY FROM THE PEDIATRIC BARIATRIC STUDY GROUP (3 MINUTE)

Thomas Inge, M.D., Ph.D.[1], Michael Chen, M.D.[2], Michael Helmrath, M.D.[3], Kimberly Wilson, MS[1], Rachel Akers, MPH[1], Judy Bean, Ph.D.[1], Victor Garcia, M.D.[1], Carroll "Mac" Harmon, M.D., Ph.D.[4], Stephen Daniels, M.D., Ph.D.[1], Lawrence Dolan, M.D.[1]

[1]Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A., [2]University of Florida College of Medicine, Gainesville, FL, U.S.A., [3]Baylor College of Medicine, Texas Children's Hospital, Houston, TX, U.S.A., [4]University of Alabama at Birmingham, Birmingham, AL, U.S.A.

Purpose:
Morbid obesity is associated with Type 2 diabetes mellitus (T2DM), dyslipidemia, and hypertension — all known risk factors for cardiovascular disease. Surgical weight loss has resulted in a marked reduction of these risk factors in adults. We hypothesized that surgical weight loss would have similar effects in adolescents.

Methods:
A retrospective review identified all patients with T2DM who were referred for weight loss surgery at three centers participating in the Pediatric Bariatric Study Group. Eight patients who were ≥1 year following Roux en Y gastric bypass (RYGBP) were included in this analysis of anthropometric measurements, blood pressure, and fasting biochemical data.

Results:
The study population was 63% female, all Caucasian, one of Hispanic ethnicity and had a mean age of 17.9 years (range 14-21) at the time of surgery. As shown in the Table, at one year post-surgery, subjects had significant weight loss, normalization of blood glucose, marked reversal of insulin resistance, and a reduction in triglycerides and blood pressure. Clinically significant improvements in HgbA1c levels were also seen (mean 6.7% pre-surgery to 5.6% at one year post-surgery; normal range 3.5-6.3%).

Conclusion:
As seen in adults, morbidly obese adolescents experience significant weight loss and remission of T2DM one year following RYGBP. Changes in blood pressure and triglycerides further suggest that future cardiovascular risks may be decreased as well. Although the long-term efficacy of RYGBP is not known in this age group, RYGBP should be considered a treatment option for morbidly obese adolescents who have T2DM.

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Changes in anthropometric measurements, blood pressure and fasting biochemical data after RYGBP					
	n	**Pre-surgery(M±SD)**	**n**	**1 year post-surgery(M±SD)**	**p**
BMI (kg/m^2)	8	49.6±4	8	32.3±6	0.001
# of Medications for T2DM	8	1.62±0.7	8	0.25±0.7	0.0004
Subjects on any T2DM Meds	8	7 of 8	7	1 of 7	0.05
Glucose (mg/dL)	7	141±46	6	85±17	0.04
Insulin (μU/ml)	6	51±35	5	9.3±4	0.03
HOMA Inulin Sensitivity	6	0.6±0.5	5	3.7±3	0.06
Triglycerides (mg/dL)	6	176±73	5	88.4±51	0.05
Systolic BP (mmHg)	7	139±15	7	117±15	0.05
Diastolic BP (mmHg)	7	80±10	7	63.5±7	0.008

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34 SHORT-TERM RESULTS IN 41 OBESE U.S. CHILDREN TREATED WITH LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING (6 MINUTE)

Evan P. Nadler, M.D., Heekoung A. Youn, RN, Howard B. Ginsburg, M.D., Christine J. Ren, M.D., George A. Fielding, M.D.
New York University, New York, NY, U.S.A.

Purpose:
Childhood obesity has reached epidemic proportions. Recently, gastric by-pass for morbidly obese adolescents has gained increasing support. However, this procedure carries at least a 1% mortality rate. Therefore, our center has been using laparoscopic adjustable gastric banding (LAGB) to treat adolescents with morbid obesity. This analysis is a report of short-term results in our first 41 patients.

Methods:
All children aged 13-17 who have undergone LAGB at our institution have been entered into our IRB-approved prospectively collected database. Data collected preoperatively included age, gender, race, and body-mass index (BMI). Post-operatively recorded data included length of stay, operative morbidity, need for re-operation, as well as BMI and percent excess weight loss (%EWL) at 3-month intervals.

Results:
Forty-one children aged 13-17 (mean 15.7) underwent LAGB at our institution since September 2001. Of these, 32 were female and nine were male. Mean pre-operative weight and BMI, as well as BMI and %EWL at six months, one year, and two years post-operatively are shown below (Table). There were no intra-operative complications. Two patients had band slips that required laparoscopic repositioning and one patient developed a hiatal hernia that required laparoscopic repair, all performed as outpatient procedures. A fourth patient developed perforated appendicitis 10 days after band placement requiring port removal.

Conclusions:
LAGB is not only a safe operation for morbidly obese pediatric patients, but represents an effective treatment strategy with a %EWL of at least 50% at both one and two year follow-up. Due to the minimal morbidity and absence of mortality, LABG is the optimal surgical option for adolescents with morbid obesity.

BMI and %EWL in 41 Obese US Adolescents

Time	Number	Weight	BMI	%EWL
Pre-op	41	295± 51 (221-415)	47± 7.2 (35-63)	NA
6 months	16	253± 60 (186-386)	40± 8.3 27-55)	37.7± 16.6 (11-72)
1 year	10	216± 64 (113-302)	34± 9.9 (20-47)	64.4± 28.9 (26-114)
2 years	2	18± 26 (199-236)	39± 5.0 (35-42)	51.5± 2.0 (50-53)

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35 ETHANOL-LOCK TECHNIQUE FOR PERSISTENT BACTEREMIA OF LONG-TERM INTRAVASCULAR DEVICES IN PEDIATRIC PATIENTS (3 MINUTE)

Wes Onland, Cathy E. Shin, Stana Fustar, Theresa Rushing, Wing Yen Wong.
Los Angeles Children's Hospital, Los Angeles, CA, U.S.A.

Purpose:
Long-term intravascular devices are increasingly vital for pediatric patients with chronic need of blood transfusions, intravenous medications or parenteral nutrition, but carry a considerable risk of catheter-related infection. We used the ethanol-lock technique, in conjunction with systemic antibiotics, to salvage central lines from removal and prevent persistence of infection.

Methods:
Over a period of 12 months, we evaluated the effect of the ethanol-lock technique in eradication of catheter-related infections and retention of 51 previously infected central lines in 40 patients with diverse underlying disorders. Children with suspected central line infections had a dose volume of 0.8-1.4 ml. 70% ethanol instilled into the catheter lumen over 12-24 hours and then withdrawn. Volume of ethanol was determined by type of central venous device. Clearance of infection and incidence of recurrence was evaluated in each treatment.

Results:
Of the 51 ethanol-lock treatments in 40 children with oncologic, hematologic, gastrointestinal, metabolic disorders, or post-BMT and small bowel transplants with different types of central venous devices, zero (0/51) catheters were removed due to persistent infection. Eighty-eight percent (45/51) of the treated episodes cleared with no recurrence, defined as a relapse within 30 days with the same pathogen. Twelve polymicrobial isolates and 33 monomicrobial isolates were successfully treated (75% and 94% respectively). There were no adverse reactions or side effects reported.

Conclusions:
This retrospective study supports the use of the ethanol-lock technique in conjunction with systemic antibiotics, as an effective and safe method in retaining the use of a previously infected central venous catheter, decreasing the need for line removal, and eradication of persistent pathogens in catheter-related infections. A randomized, double-blind controlled trial would help evaluate the effectiveness of this treatment prospectively in a larger population of children dependent on central venous access.

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36 ISOLATED LIVER AND MULTIVISCERAL TRANSPLANTATION FOR TPN-RELATED END-STAGE LIVER DISEASE (3 MINUTE)

Jaimie D. Nathan, Greg M. Tiao, M.D., Samuel A. Kocoshis, Maria H. Alonso, Frederick C. Ryckman, M.D.

Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A.

Purpose:

Total parenteral nutrition (TPN) has prolonged survival in children with intestinal failure; however, end stage liver disease due to TPN-induced cholestasis (ESLD-TPN) may preclude its value. ESLD-TPN is an indication for isolated liver transplantation (ILT) or multivisceral transplantation (MVT). ILT in patients with ESLD-TPN should only be considered in patients who have the potential for enteral autonomy. We examined our transplant experience in children with ESLD-TPN.

Methods:

We retrospectively reviewed ILT (n = 6) and MVT (n = 5) performed between 1994 - 2005 for ESLD-TPN. Mean age at transplant was 21 months (range 6 - 74). Etiologies of intestinal failure included necrotizing enterocolitis (n = 3), gastroschisis (n = 2), gastroschisis with volvulus (n = 3), gastroschisis with atresia (n = 1), malrotation (n = 1), and megacystis microcolon intestinal hypoperistalsis syndrome (n = 1).

Results:

Patients undergoing ILT had mean length of small bowel of 76 cm. Mean percent of enteral calories tolerated was 66%. Mean length of small bowel in patients undergoing MVT was 36 cm. None tolerated more than comfort feeds. Reduced-size (n = 5) and whole liver (n = 1) were used for ILT. MVT patients received liver-small bowel (n = 4) or liver-small bowel-colon (n = 1). Survival was 50% in ILT patients (mean follow-up 25 months). Two survivors are TPN-independent; the third patient requires TPN three days/week. Survival was 40% in MVT patients (mean follow-up 7 months). One MVT patient died of abuse and was TPN-independent. Both survivors are TPN-independent. Bilirubin levels were normal in 83% of survivors.

Conclusions:

ILT for ESLD-TPN due to intestinal failure is a viable option in patients with potential for enteral autonomy. MVT is the only alternative in patients without the potential for intestinal recovery. Survival can be achieved in patients with ESLD-TPN, but mortality remains high.

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37 THE 2005 REPORT FROM THE INTERNATIONAL STEP DATA REGISTRY: INDICATIONS, EFFICACY, AND COMPLICATIONS — A GOOD FIRST STEP (3 MINUTE)

Biren P. Modi, M.D., Patrick J. Javid, M.D., Tom Jaksic, M.D., Ph.D., Hannah Piper, M.D., Monica Langer, M.D., Heung Bae Kim, M.D., on behalf of the International STEP Data Registry.

Children's Hospital Boston, Boston, MA, U.S.A.

Purpose:

The International STEP Data Registry was created in 2003 for multi-center assessment of the serial transverse enteroplasty (STEP) operation. This first report of the International STEP Data Registry describes the diagnoses, indications, complications, and outcomes of the STEP operation.

Methods:

After IRB approval, surgeons enrolled their patients in the International STEP Data Registry via online data entry and telephone contact, with ongoing follow-up. Statistical analyses were performed using paired t-tests with significance set at $p<0.05$.

Results:

Sixteen centers enrolled 28 patients. The primary diagnoses were intestinal atresia (n=11), gastroschisis (n=8), necrotizing enterocolitis (n=4), volvulus (n=3), and other (n=2). Indications for STEP were short bowel syndrome (SBS) (n=20), bacterial overgrowth (n=5), and atresia with marginal bowel length in neonates (n=3). The STEP operation significantly increased mean small intestinal length in all groups (66±43 cm vs. 112±77 cm, $p<0.001$, n=18). Two patients required intraoperative repair for leak at the apex of a staple line. Early postoperative complications were bowel obstruction (n=1), abscess (n=1), and hematoma (n=1). Late complications included progression to transplantation (n=2) and mortality from preexisting liver failure (n=1). For the SBS cohort, excluding patients with incomplete data or progression to transplant or death, mean enteral tolerance was significantly increased from 35±34% to 74±35% of calories ($p<0.005$, n=14), with 13.5 month median follow-up. The STEP operation eliminated symptoms in all bacterial overgrowth patients. The three neonates each currently tolerate 100%, 80%, and 70% of calories enterally.

Conclusions:

Serial transverse enteroplasty has been performed at multiple centers with minimal complications and encouraging outcomes. Indications for the procedure have broadened beyond short bowel syndrome to include bacterial overgrowth and intestinal atresias with dilated proximal intestine. Continued accrual and follow-up of patients in the International STEP Data Registry will further elucidate the long term safety and efficacy of the serial transverse enteroplasty operation.

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38 EARLY FEEDING VS. 5-DAY FASTING AFTER ELECTIVE BOWEL ANASTOMOSES IN CHILDREN: A RANDOMIZED TRIAL (6 MINUTE)

Roberto Davila-Perez, M.D., Eduardo Bracho-Blanchet, M.D., Jose Manuel Tovilla-Mercado, M.D., Ricardo Reyes-Retana, M.D., Jaime Nieto-Zermeño, M.D.
Hospital Infantil de Mexico, México DF, Mexico

Purpose:
To determine the security and tolerance of early enteral feeding after elective bowel anastomoses in children

Methods:
Controlled randomized trial including all pediatric patients with elective intestinal anastomosis. Excluding newborns, bilious-digestive or rectal anastomoses, immunosuppressed, multiple anastomoses or close to Treitz, frozen abdomen. **VARIABLES:** Demografic, operative time, anastomosis placement, vomiting, abdominal distention, begin of peristaltic, gas and bowel movements, wound complications, reoperation. All patients were managed with antimicrobials, bowel preparation, general anesthesia. At the end of surgery were randomized into: **1. Experimental group (EG):** Early feeding group, after 24 hours fasting period, with good abdominal conditions the oral fluids and diet were started. **2. Control group (CG):** 5-day fasting. Once the regular diet was tolerated, the patients were discharged.

Statistics:
Central tendency, Student's t test, chi-squared, $p<0.05$ was significative.

Results:
From June 2003 to May 2004, 52 patients were included. Twenty-three patients from the EG (44%) and 29 from the CG (56%). Age 46±58 months, weight 16.1±15 kg, 20 malnutrition (38%). Stomal Ethiology: 23 anorectal-malformation (45%), six Hirschsprung (11%), 20 inflammatory (38%), three tumoral (6%). Anastomotic segment: ileon 11 (21%), ileo-colonic five (9%), colonic 36 (70%). Surgical time 143±45 min. Neither of patients had vomiting or required nasogastric-tube. Eight showed mild abdominal distension (15%), 10 had fever and 11 wound complications. Four intestinal fistula, no reoperations. Demographic variables showed no stadistical differences. Mean beggining of the oral intake was in second POP day in the EG vs. the 5-day in the CG ($p=0.02$). POP stay 6.9±2.8 vs. 10.1±4.1 days, peritalsis begining 1.43±0.5 vs. 1.86±0.63 days, flatus begining 1.7±0.5 vs. 2.14±0.5 days, bowel movements 2.4±0.5 vs. 2.6±0.5 days in the EG and CG respectively, without stadistical difference. The complication incidence was low and equally distributed.

Conclusions:
The early feeding after elective intestinal anastomosis in children is safe and well-tolerated.

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39 PROBIOTIC ACIDIFIED FORMULA IN AN ANIMAL MODEL REDUCES PULMONARY AND GASTRIC BACTERIAL LOAD (3 MINUTE)

Cristiano Boneti, M.D., Christine M. Habib, M.D., Jennifer E. Keller, M.D., Jose A. Diaz, M.D., Evan R. Kokoska, M.D., Richard J. Jackson, M.D., Samuel D. Smith, M.D.
Arkansas Children's Hospital, Little Rock, AR, U.S.A.

Purpose:
We previously reported diet acidified with citric acid effectively reinforces gastric acid protection against bacterial colonization and translocation. In the current study we examined use of a biologically acidified formula hypothesized to be more physiologic than formula acidified with free acid. This study was IACUC approved and designed to determine whether this diet is better tolerated and equally effective to acidification with citric acid against bacterial translocation and gut colonization in a premature infant rabbit model.

Methods:
One hundred twelve rabbit pups delivered via cesarean section one day preterm were randomly assigned to three feeding groups: Pelargon® Nestle at pH 4.55; NAN® Nestle, a control diet at pH 7.0 with similar composition; and NAN® Nestle acidified in the lab with citric acid at pH 4.55. Pups were gavage fed every 12 hours with *Enterobacter cloacae* challenges of 10 colony-forming units/ml of diet per feed and sacrificed on the third day of life. Lungs, liver, spleen, mesenteric lymph nodes (MLN), stomach and cecum were cultured and quantitatively analyzed for target organism growth and statistically analyzed using Chi square and Kruskal-Wallis tests.

Results:
Pelargon® compared to acidified NAN® and NAN®, significantly reduced the incidence of gastric colonization [15/33 (45%), 21/27 (78%), and 25/29 (86%) respectively, $P<0.01$] and pulmonary colonization [10/33 (30%), 19/27 (70%), 21/29 (72%), $P<0.01$]. Comparing the bacterial logs of colonized groups, the same benefit is observed in the lungs [0.77 ±1.22, 1.89 ±1.41, 2.12 ±1.47 $p < 0.01$]. Gut colonization and bacterial translocation were equivalent between treatment groups [MLN: 10/33 (30%), 11/27(40%), 8/29 (27%); Spleen: 10/33 (30%), 7/27 (26%), 8/29 (27%); Liver: 10/33 (30%), 6/27(22%), 9/29 (31%); Cecum: 33/33 (100%), 27/27(100%), 29/29 (100%)].

Conclusions:
Biologically acidified formula demonstrated superior protection against pulmonary and gastric colonization compared to normal pH and diets acidified with free acid. Its effects may potentially reduce clinical pulmonary infection.

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40 A COMPARISON OF DIFFERENT PERINATAL SOURCES OF MESENCHYMAL PROGENITOR CELLS: IMPLICATIONS FOR TISSUE ENGINEERING (3 MINUTE)

Shaun M. Kunisaki, M.D., Julie R. Fuchs, M.D., Humberto Azpurua, M.D., David Zurakowski, Ph.D., Dario O. Fauza, M.D.
Children's Hospital Boston, Boston, MA, U.S.A.

Purpose:
Engineered constructs derived from fetal and neonatal mesenchymal progenitor cells (MPCs) have been shown experimentally to be viable alternatives for the surgical treatment of a number of congenital anomalies. The optimal source of these cells for tissue engineering, however, remains to be defined. This study was aimed at comparing the immunophenotypical profiles and *in vitro* proliferation rates of MPCs derived from amniotic fluid, neonatal bone marrow, and cord blood.

Methods:
Cryopreserved ovine MPCs isolated from samples of amniotic fluid (n=4), neonatal bone marrow (n=4), and fetal umbilical cord blood (n=4) were expanded in culture under standard, adipogenic, and osteogenic conditions. Their multipotent potential was confirmed by osteogenic and adipogenic differentiation using the Von Kossa and Oil-O-Red stains, respectively. Early passaged cells cultured in standard media were characterized by both immunocytochemistry and flow cytometry using ovine-specific monoclonal antibodies. Cell proliferation was quantified over a 96-hour period by normalized DNA counts using Hoechst dye. Statistical analysis was by two-way repeated-measures analysis of covariance using the F-test for slope comparisons ($P<.05$).

Results:
All MPCs displayed indistinguishable morphological and immunophenotypical characteristics, consistent with that of mesenchymal progenitors, regardless of cell source. Cells were negative for CD31 and positive for vimentin, cytokeratins 8 and 18, CD29, CD44, CD90, and CD105 expressions. Proliferation rates for amniotic fluid-derived MPCs were significantly higher when compared to MPCs derived from neonatal bone marrow and cord blood (Figure).

Conclusions:
Mesenchymal progenitor cells derived from amniotic fluid, neonatal bone marrow, and cord blood are phenotypically similar, but display different cell expansion capabilities *in vitro.* Mesenchymal amniocytes proliferate significantly faster in culture than other mesenchymal cells. These findings, combined with its ease of access, further establish the amniotic fluid as the optimal source of mesenchymal progenitor cells for the surgical treatment of congenital anomalies by perinatal tissue engineering.

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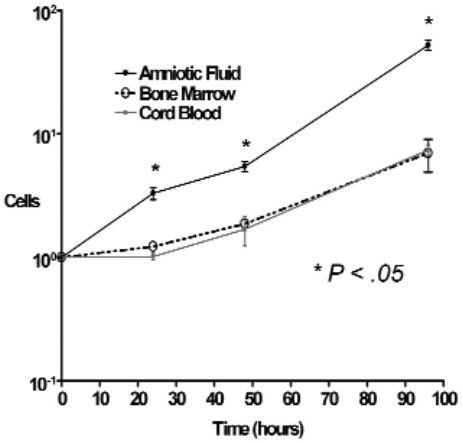

Notes:

41 NDSP: A NOVEL BIOMARKER AND GROWTH FACTOR IN NEUROBLASTOMA (6 MINUTE)

Sanjeev A. Vasudevan, M.D., Ningling Ge, M.D., Ph.D., Andrew D. Ludwig, B.A., Catherine L. Wesson, B.S., Kuan Wang, M.D., Ph.D., Xiao-Ying Shang, Ph.D., Susan M. Burlingame, B.S., M. Fatih Okcu, M.D., Heidi V. Russell, M.D., Jianhua Yang, Ph.D., Jed G. Nuchtern, M.D., FACS.

M.E. DeBakey Department of Surgery and Texas Children's Cancer Center, Department of Pediatrics, Baylor College of Medicine, Houston, TX, U.S.A.

Purpose:

Secreted proteins such as growth factors, cytokines and chemokines play important roles in tumor development. Having previously reported the initial cloning and characterization of neuroblastoma derived secretory protein (NDSP), we now hypothesize that NDSP expression correlates with prognosis in neuroblastoma and that this molecule is a paracrine growth factor for tumor cell proliferation.

Methods/Results:

Northern blot and real time PCR were used to test NDSP tissue specificity. NDSP was expressed at very low to insignificant levels in normal human tissues. Through a real time PCR analysis of many different types of adult and pediatric cancers, NDSP was found to have specific overexpression in neuroblastoma. After obtaining institutional approval for real time PCR analysis of patient tissue (IRB: H-9881), NDSP overexpression was found in 43 of 45 neuroblastoma tumor samples tested compared to control. This tissue analysis also revealed that high NDSP transcript levels correlated with high risk, poor prognosis patients (stage III and IV; $p=0.032$). Immunoblot of culture supernatant from neuroblastoma cell lines showed that NDSP is actively secreted. Further functional analysis was carried out in SK-N-AS cells with stable NDSP small-interfering RNA (siRNA) expression in order to knockdown endogenous NDSP expression. Greater than 80% NDSP protein knockdown was achieved, and these cells grew at a significantly slower rate ($p<0.03$) compared to controls. In soft agar, the NDSP knockdown cells grew significantly fewer colonies than the control cell lines ($p<0.02$). The proliferation defect seen in the NDSP knockdown cells was reversed when recombinant NDSP protein was introduced into the culture media.

Conclusions:

NDSP is a novel gene overexpressed in neuroblastoma correlating with poor prognosis. In addition, NDSP may serve as a novel paracrine growth factor for neuroblastoma cell proliferation and tumor formation.

Notes:

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42 PANCREATIC ALPHA-CELL DIFFERENTIATION BY MESENCHYME-TO-EPITHELIAL TRANSITION: IMPLICATIONS FOR STEM-CELL TREATMENTS? (6 MINUTE)

Warwick J. Teague, Autumn M. Rowan-Hull, Paul R.V. Johnson.
University of Oxford, Oxford, United Kingdom

Purpose:
The potential for curative stem-cell treatments of juvenile-onset diabetes mellitus has focussed research into normal pancreatic development. We are investigating the embryonic origin of pancreatic islets using an established avian model. The avian endocrine pancreas comprises two distinct islet types: B-islets (predominantly insulin-secreting beta-cells) and A-islets (predominantly glucagon-secreting alpha-cells). Islets were previously thought to originate solely from embryonic pancreatic epithelium, but we have previously shown that foregut mesenchyme can contribute cells to B-islets during development. This study tests the hypothesis that foregut mesenchyme can also differentiate into A-islets.

Methods:
In compliance with local ethical guidelines, dorsal pancreatic and stomach rudiments were micro-dissected from 4-day avian embryos. Chimeric recombinants were constructed using chick-pancreatic epithelium combined with quail-stomach mesenchyme (n=14), and cultured for seven days using minimal essential media. Foregut controls and recombinants were frozen-sectioned (7-10μm), and analysed using combined fluorescent and peroxidise immunocytochemistry against glucagon and quail-specific antigen respectively. A-islets (clusters of glucagon-positive cells) were determined to be of solely epithelial, solely mesenchymal or mixed embryonic origin. Chi-square tests were used to compare mesenchymal cellular contribution to A-islets with data previously obtained regarding the embryonic origin of B-islets ($p<0.05$ considered significant).

Results:
Fourteen recombinants yielded 48 A-islets. The majority (85%) of A-islets were solely epithelial in origin, but notably 5% were derived solely from mesenchyme, and a further 10% of mixed embryonic origin. When grouping all islets with a mesenchymal cellular contribution, A-islets differentiation from foregut mesenchyme was significantly reduced compared with B-islets (Figure 1, $p=0.03$).

Conclusion:
We demonstrate for the first time that avian pancreatic alpha-cells can differentiate from foregut mesenchyme. Clinical islet transplantation is reliant on the transplantation of intact islets containing both insulin- and glucagon-secreting cells. These findings therefore, may have important implications for the derivation of islet tissue from mesenchymal stem-cells to cure juvenile-onset diabetes.

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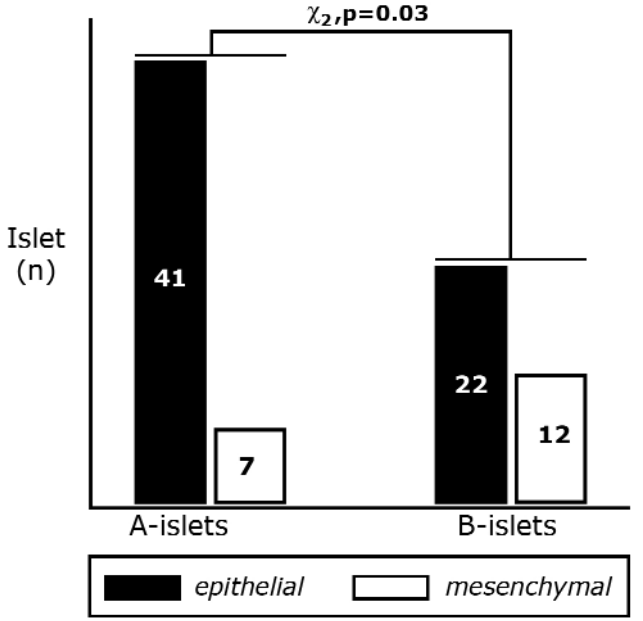

χ_2,p=0.03
Islet
(n)
41
7
22
12
A-islets
B-islets
epithelial
mesenchymal

Notes:

43 BLOCKADE OF VASCULAR ENDOTHELIAL GROWTH FACTOR RECEPTOR 2 (VEGFR2) SIGNALING ON TUMOR ENDOTHELIUM SUPPRESSES HUMAN NEUROBLASTOMA GROWTH IN MICE (3 MINUTE)

Paul Beaudry, M.D., M.Sc.[1], Matthew Rioth[2], David Poon[2], Daniela Prox, M.D.[2], Anderson Ryan, Ph.D.[3], John Heymach, M.D., Ph.D.[4], Judah Folkman, M.D.[2], Sandra Ryeom, Ph.D.[2]

[1]British Columbia's Children's Hospital, Vancouver, BC, Canada, [2]Boston Children's Hospital, Boston, MA, U.S.A., [3]Astra Zeneca, Macclesfield, United Kingdom, [4]MD Anderson, Houston, TX, U.S.A.

Purpose:

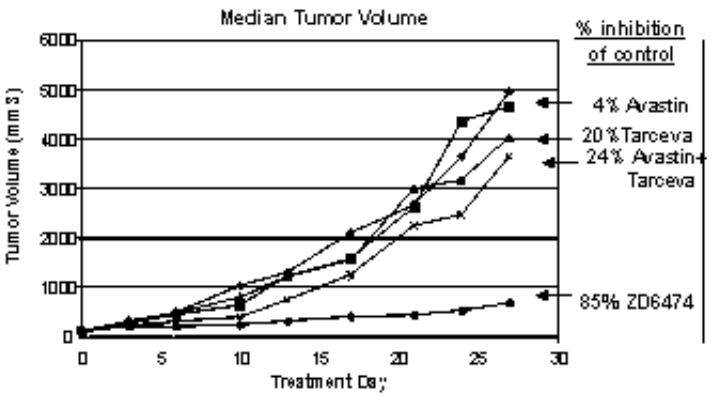
Antiangiogenic therapy for neuroblastoma may be an effective treatment. This work examines targeted inhibition of VEGF signaling in a neuroblastoma xenograft model.

Methods:

Neuroblastoma xenografts were established and mice were treated with the following: ZD6474, a small molecule receptor tyrosine kinase inhibitor of VEGFR2 and epidermal growth factor receptor (EGFR) signaling, Avastin, an anti-human VEGF monoclonal antibody, or Tarceva, an EGFR receptor tyrosine kinase inhibitor, alone or in combination with Avastin. After four weeks, tumor sections were examined for VEGFR2 and EGFR activation, endothelial cell apoptosis and microvessel density. Expression and function of VEGFR2 and EGFR on neuroblastoma cells and microvascular endothelial cells was analyzed *in vitro* by Western blot and proliferation assays.

Results:

Only ZD6474 treatment caused significant inhibition of tumor growth (Figure; T/C=0.15; $p<0.05$). Immunofluorescence analysis of ZD6474 treated tumors revealed significant endothelial cell apoptosis and inhibition of VEGFR2 and EGFR activation on tumor endothelium. Tarceva blocked EGFR signaling on tumor endothelial cells but did not cause increased apoptosis and did not inhibit tumor growth. Human avastin did not inhibit VEGFR2 phosphorylation on host-derived tumor endothelium, because it does not block mouse VEGF and thus had no effect on tumor growth. Absence of VEGFR2 and EGFR expression on several neuroblastoma cell lines was confirmed by Western.



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Conclusions:
Inhibition of VEGFR2 on endothelium in a neuroblastoma tumor bed by ZD6474 is a highly antiangiogenic and significantly inhibits growth of neuroblastoma in this model. Furthermore, we demonstrate EGFR inhibition is ineffective against neuroblastoma because neuroblastoma cells do not express EGFR. Finally, these experiments suggest that host VEGF can induce tumor angiogenesis independent of tumor cell derived-VEGF. This work suggests that antiangiogenic therapy may improve the survival of children with neuroblastoma.

Notes:

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44 NTERFERON-β MEDIATED VESSEL STABILIZATION IMPROVES DELIVERY OF SYSTEMICALLY ADMINISTERED TOPOTECAN IN A MURINE MODEL OF RETROPERITONEAL NEUROBLASTOMA (6 MINUTE)

Paxton V. Dickson, M.D., John B. Hamner, M.D., Cathy Y.C. Ng, MS, Nikolaus L. Hagedorn, BS, Charles Fraga, MS, Clinton F. Stewart, Ph.D., Andrew M. Davidoff, M.D.
St. Jude Children's Research Hospital, Memphis, TN, U.S.A.

Purpose:
We have recently demonstrated that continuous delivery of interferon-β stabilizes solid tumor vasculature and improves tumor perfusion. We have further investigated the functional consequences of this effect by assessing delivery and anti-tumor efficacy of conventional chemotherapy against neuroblastoma xenografts when used in combination with IFN-β

Methods:
Mice with established retroperitoneal tumors received adeno-associated virus vector encoding IFN-β (AAV IFN-β). One week later, at one hour prior to sacrifice, a 1mg/kg intravenous bolus of topotecan was given. Mice with size matched tumors, receiving control vector, served as controls. Intratumoral levels of topotecan were measured by high powered liquid chromatography and then standardized to plasma levels. Subsequent experiments evaluated the anti-tumor efficacy of topotecan alone or in combination with IFN-β.

Results:
The ratio of tumor to plasma levels of topotecan was increased by 58% (2.23 vs. 1.41, $p=0.128$) in mice receiving IFN-β therapy vs. size matched controls. In additional cohorts of mice, this resulted in an improved anti-tumor effect. Mice with established tumors ($310\pm29.5mm^3$) were treated with topotecan (1mg/kg daily for five days for two cycles) either alone or in combination with IFN-β. Topotecan monotherapy resulted in a reduction in mean tumor volume of 14% ($264\pm84.9mm^3$, $p=0.52$). However, when the same regimen was administered to mice receiving IFN-β therapy, a 61% ($120mm^3$, $p<0.01$) reduction in mean tumor volume was achieved. IFN-β monotherapy achieved only a restriction in further tumor growth ($712\pm115.7mm^3$, $p<0.05$) as compared to control vector treated tumors which had a mean volume of $2470\pm565.3mm^3$ at sacrifice.

Conclusions:
IFN-β mediated vessel stabilization and the resultant increase in tumor perfusion results in improved delivery of systemically administered topotecan, enhancing its anti-tumor efficacy. This approach of altering the tumor vasculature provides a strategy to help overcome solid tumor resistance to traditional cytotoxic agents.

Notes:

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45 AGE-DEPENDENT RECRUITMENT OF NEUTROPHILS BY FETAL ENDOTHELIAL CELLS: IMPLICATIONS IN SCARLESS WOUND HEALING (6 MINUTE)

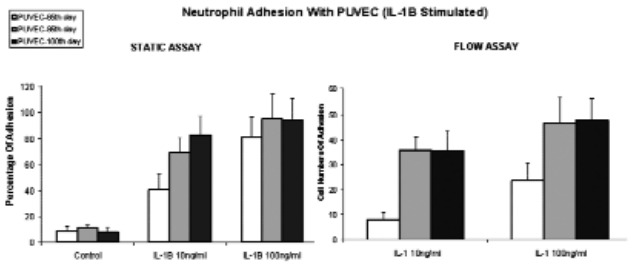
Bindi Naik-Mathuria, M.D., Andre Nicolas Gay, Ling Yu, Xi Zhu, Darrell Cass, M.D., C. Wayne Smith, M.D., Oluyinka O. Olutoye, M.D., Ph.D.
Baylor College of Medicine, Houston, TX, U.S.A.

Purpose:
Fetal dermal wound healing is characterized by the paucity of an inflammatory response and the absence of fibrosis. Later in gestation, a transition to adult-like healing with an acute inflammatory response and fibrosis is observed. Leukocyte-endothelial interaction is imperative for transmigration of leukocytes, a critical step in the inflammatory cascade. We embarked upon this study to determine if differences in endothelial cell function at different gestational stages modulate the inflammatory response, and thus correlate with the fetal dermal wound healing response.

Methods:
Endothelial cells (EC) were harvested from veins of fetal pigs at mid-gestation (day 60), early third trimester (day 85), and late third trimester (day 100); term = 115 days. Confluent primary endothelial monolayers were activated with porcine IL-1 beta at 10 and 100ng/ml and exposed to a suspension of adult neutrophils in a continuous flow system with a shear stress of 4 dynes/cm2 (n=6 per group), or under static conditions (n=4 per group). The adhesion of neutrophils to the ECs was quantified and compared. ANOVA was utilized for statistical analysis and *p<0.05 was considered significant.

Results:
In the flow studies, there was a dose-dependent increase in neutrophil recruitment by fetal endothelial cells at all gestational ages tested. Mid-trimester ECs had significantly less ability to recruit neutrophils compared to both third trimester groups at both cytokine concentrations tested. In the static studies, with increasing gestational age, the lower dose of cytokine was more likely to elicit the maximal neutrophil recruitment.

Conclusion:
There is a gestational age-dependent variation in EC neutrophil recruitment. Mid-gestation ECs require large doses of cytokine, while late third trimester ECs actively recruit neutrophils at lower cytokine concentrations. These findings correlate with the period of transition to adult-like healing and suggest an important role for fetal ECs in the inflammatory response related to fetal wound healing.



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46 NIH FUNDING AND THE PEDIATRIC SURGEON: CHARACTERIZING SUCCESSFUL APPLICANT PROFILES IN AN ERA OF INCREASING COMPETITION FOR EXTRAMURAL SUPPORT (3 MINUTE)

Shawn J. Rangel, M.D.[1], Jessica J. Kandel, M.D.[2], Charles J.H. Stolar, M.D.[2], R. L. Moss, M.D.[3], George K. Gittes, M.D.[4]

[1]Stanford University Medical Center, Stanford, CA, U.S.A., [2]Columbia University School of Medicine, New York, NY, U.S.A., [3]Yale University School of Medicine, New Haven, CT, U.S.A., [4]University of Pittsburgh School of Medicine, Pittsburgh, PA, U.S.A.

Purpose:
Continued advances in the field of pediatric surgery may hinge upon our ability to obtain funding from the NIH. However, future support may be threatened by ongoing budgetary constraints and increasing competition for extramural awards. The purpose of this study was to characterize the experience and academic profiles of pediatric surgeons currently holding NIH funding. Such insight may be used to develop strategies to improve the success of future investigators.

Methods:
Pediatric surgeons currently holding NIH awards were identified from the NIH CRISP database (n=33). A 19-item questionnaire was sent to these investigators to obtain details regarding research activity, mentorship experience, perceived obstacles to funding, and factors they considered essential for acquiring NIH support.

Results:
Thirty-one investigators responded to the questionnaire (awards: 16 RO1's, five non-RO1 R-awards, 10 KO8's, and two K24's). 'Dedicated' research time averaged 13.3 hours/week with a mean 'protected' time estimate of 33%. The majority (60%) of investigators required at least one re-submission for funding. Younger investigators (K08's) cited protected time and effective mentorship as the most critical elements for successful funding, while more experienced investigators (RO1's) felt that persistence following initial rejection was most important. The amount of start-up funding, participation in NIH peer-review, and 'luck' were considered less critical. Those who attended a grant-preparation workshop all felt this increased their chance of funding.

Conclusions:
Provision of adequate protected time, attendance at grant-preparation workshops, and intensive mentorship by a senior investigator experienced in NIH funding will be critical to maximize the chance of funding in an increasingly competitive environment. Resubmissions are the rule, and intensive feedback during the revision process by an experienced mentor may be the most critical step in this process. Division leaders should strive to provide this framework for success if we are to ensure adequate funding for pediatric surgical diseases in the future.

Notes:

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47 THE EPIDEMIOLOGY OF INFANT INJURIES AND ALARMING HEALTH DISPARITIES (3 MINUTE)

Richard A. Falcone, Jr., Rebeccah L. Brown, M.D., Victor F. Garcia, M.D.
Cincinnati Children's Hospital Medical Center, Cincinnati, OH, U.S.A.

Purpose:
Injury epidemiology is the underappreciated foundation of injury prevention and control strategies. Given the substantial disparity of infant injury-related mortality between African American (AA) and the U.S. rate (13.9 vs. 4.9/10,000) in our region, we sought to elucidate the epidemiology of infant injuries and mortality rates.

Methods:
Our trauma database was reviewed for all infant (28-364 days) injuries over a 10-year period. The mortality rates were analyzed based on race, mechanism and insurance type.

Results:
From 1995-2004, 1,270 infants were identified (127/yr). Sixty-nine percent were white, 26% AA, and 5% were other. The most common injury mechanisms were falls (37%) and abuse (18%). Overall mortality was 4.8%. The highest mortality rates were associated with suffocation (83%), drowning (30%), MVC (13%), and abuse (8%). There were significant disparities in outcomes based on race (Table 1). By mechanism, suffocation was the only injury that occurred with more frequency in AA (4.8 vs. 1.3%, p<.005).

Table 1. Infant injury mortality (%) by race

	AA	White	P value	RR [95% CI]
Overall	9.58	2.76	0.000001	3.48 [2.01,5.81]
Abuse	15.15	4.03	0.0051	3.76 [1.43,9.92]
Suffocation	100	54.55	0.0057	1.83 [1.07,9.92]
Drowning	42.86	26.67	0.2801	1.61 [0.48,5.33]
MVC	16.67	13.64	0.3280	1.22 [0.28,5.30]

Although 75% of AA versus 40% of whites were insured by Medicaid, there was no difference in mortality between those with commercial insurance or Medicaid. Separated by insurance type, the disparity in mortality rates between races remained significant.

Conclusions:
AA infants have 3.5 times increased risk of death from preventable injuries compared to white infants. This disparity persists despite controlling for type of insurance, a surrogate for socioeconomic status. Understanding these disparities and developing injury prevention programs targeting high-risk mechanisms of injury such as abuse and suffocation among AA is critical towards eventually eliminating these preventable deaths.

Notes:

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48 PEDIATRIC VASCULAR INJURIES: PATTERNS OF INJURY, MORBIDITY AND MORTALITY (3 MINUTE)

Denise B. Klinkner[1], Marjorie J. Arca, M.D.[2], Brian D. Lewis, M.D.[3], Keith T. Oldham, M.D.[2], Thomas T. Sato, M.D.[2]

[1]Children's Hospital of Wisconsin and Medical College of Wisconsin, Milwaukee, WI, U.S.A., [2]Children's Hospital of Wisconsin, Milwaukee, WI, U.S.A., [3]Medical College of Wisconsin, Milwaukee, WI, U.S.A.

Purpose:
Although rare, vascular trauma in the pediatric population results in significant morbidity and mortality. We sought to identify the patterns of injury and the associated morbidity and mortality from vascular trauma at our institution.

Setting:
A tertiary care pediatric ACS Level I trauma center.

Methods:
This is an IRB-approved, retrospective case series identified by ICD9 codes via an institutional pediatric trauma registry over the 12-year period from 1993 to April 2005.

Results:
One hundred twenty-seven cases of pediatric vascular injury were identified. Injuries isolated to the digits and unspecified vessels were excluded (24 cases). In the remaining 103 patients, the vast majority of vascular injuries occurred in males (74%). The average age was 11.3 years in males and 9.1 years in females (range: 1 - 18 years; overall mean age 10.7 years). Penetrating missile or knife wounds caused 67% of injuries, followed by blunt trauma (32%) and burn injury (0.97%). Anatomic location of injury included head/neck in 19.4%, torso in 13.5%, and extremities in 67%. Amputation or limb loss due to vascular insufficiency occurred in 12/107 (11.6%). In-hospital overall mortality was 9.7% (10/103), with eight deaths in males (10.5%) and two deaths in females (7.4%). Average length of stay was 12.1 days [10.9 d male, 15.2 d female]. Nonoperative management occurred in 9.7% (10/103), with one death due to stroke following carotid injury. Overall, 23.3% of patients underwent angiography, with one patient receiving interventional radiology treatment. Pediatric surgeons managed 29.1%, while extremity specialists treated 38.8%, vascular surgeons 17%, neurosurgeons 5.8%, and others the remaining 9.3%.

Conclusions:
Despite multidisciplinary diagnostic and treatment modalities available at tertiary care pediatric trauma centers, traumatic vascular injuries in infants and children carry significant morbidity and mortality.

Notes:

Underlining denotes the author scheduled to present at the meeting.

49 A GUIDELINE FOR EVALUATION OF THE CERVICAL SPINE IN THE PEDIATRIC TRAUMA PATIENT (3 MINUTE)

Eric R. Scaife, M.D.[1], Richard C.E. Anderson, M.D.[2], Kristine W. Hansen, R.N.[1], Stephen J. Fenton, M.D.[1], Douglas L. Brockmeyer, M.D.[1].

[1]Primary Children's Medical Center, Salt Lake City, UT, U.S.A., [2]Columbia University, New York, NY, U.S.A.

Purpose:

There are essentially no national guidelines for the management of pediatric trauma. A decision tool to guide the evaluation of a child's cervical spine is needed because of unique pediatric anatomy and injury patterns. We developed an instrument that incorporated a validated set of clinical criteria with an additional set of rules specific to the assessment of a pediatric patient.

Methods:

A guideline was developed by our institution's pediatric neurosurgeons in 2004. We review the experience at our Level I pediatric trauma center after the rule was implemented (2004-2005) and compare it to the experience two years before (2002-2003). Patients were excluded if they did not have their cervical spine evaluated.

Results:

The trauma service saw 693 activations in the first period (P1) and 902 in the second period (P2). The cervical spine was evaluated in 613 (88%) of the patients in P1 and 793 (88%) in P2. The average age of the children was 7.28 years (range 0.5-18) in P1, 7.91 years (0.3-20) in P2. The ISS was 13.6+/- 8.6 in P1 and 12+/- 8.2 in P2. Prior to our guideline, the neurosurgical service assessed the c-spine in the majority of the trauma patients. The guideline permitted the trauma service (general surgeons and nurse practitioners) to screen patients. During P1 neurosurgery evaluated 471 (77%) of the patients, in P2 the trauma service cleared 457 (58%) and triaged 277 (35%) of the patients to neurosurgery. There were 23 c-spine injuries in P1 (4% of patients) and 46 (5%) in P2. There was one missed injury in P1 and none in P2.

Conclusions:

Our guideline is an effective decision rule for evaluating the pediatric cervical spine. The tool permitted the trauma service to clear the majority of the patients with discretionary consultation of the neurosurgical service.

Notes:

Underlining denotes the author scheduled to present at the meeting.

50 PEDIATRIC SNAKEBITES: LESSONS LEARNED FROM 114 CASES (3 MINUTE)

Brendan T. Campbell, M.D., M.P.H., John M. Corsi, MBA, Cristiano Boneti, M.D., Justin D. Phillips, Alisson L. Richards, Richard J. Jackson, M.D., Samuel D. Smith, M.D., Evan R. Kokoska, M.D.

Arkansas Children's Hospital, Little Rock, AR, U.S.A.

Purpose:

Evidence-based guidelines for the treatment of pediatric snakebite injuries are lacking because they occur infrequently.

Methods:

We reviewed our experience treating snakebites from January 1995 through October 2005. Demographic (e.g., age, sex, geographic location) and clinical information (e.g., location of bite, species of snake, vital signs, labs, treatment, hospital length of stay) were obtained.

Results:

Over the last decade we have treated 114 children with confirmed snakebites. Mean age was 7.3±4.2 years (range 1-17 years), and snakebites were more common in males (n=68, 60%). All bites occurred on the extremities, and lower extremity bites were more common (n=71, 62%). Copperheads inflicted the most bite injuries (n=65, 57%), followed by rattlesnakes (n=9, 8%) and cottonmouths (n=7, 6%). The snake was not identified in 33 cases (29%). Seven children (6%) were treated with *Crotalidae* antivenin. Of the children treated with antivenin only four met criteria for treatment and one had an anaphylactic reaction. If compartment syndrome was suspected based on neurovascular exam compartment pressures were measured. Only two patients (1.8%) required fasciotomies. Over the last two years we have stopped empiric treatment with antibiotics and have not observed any infectious complications. Average hospital length of stay was 30±25 hours.

Conclusions:

Most children bitten by poisonous snakes can be managed conservatively with analgesics and elevation of the affected extremity. Treatment with *Crotalidae* antivenin and fasciotomy are rarely indicated.

Notes:

American College of Surgeons Commission on Cancer **Table Number: 15**

633 N. Saint Clair Street
Chicago, IL 60611
Phone: 312/202-5377
Fax: 312/202-5009
Email: lwatt@facs.org

The Commission on Cancer sets standards for quality multidisciplinary cancer care delivered primarily in hospital settings; surveys hospitals to assess compliance with those standards; collects standardized and quality data from approved hospitals to measure treatment patterns and outcomes; and uses the data to evaluate hospital provider performance and develop effective educational interventions.

American Pediatric Surgical Nurses Association **Table Number: 22**

600 Highland Avenue, K4/761
Madison, WI 53792
Phone: 608/438-7894
Fax: 608/261-1876
Email: helin@surgery.wisc.edu

APSNA membership spans the spectrum of the care of children requiring surgical care from trauma to burns to injury prevention; from inpatient to ambulatory surgery; inpatient and out patient care; neonatal through adolescent; NICO and PICU; the inpatient units: PACU; registered nurses in advanced practice.

Axiom® Medical, Inc. **Table Number: 16**

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Elsevier is proud to publish the Journal of Pediatric Surgery, the official journal of the American Pediatric Surgical Association. Please stop by our booth to view the latest issue of the journal and browse our other books and journals in the field of pediatric surgery.

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Greenbook Financial Services **Table Number: 10**

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Jerome Medical **Table Number: 12**

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LSI Solutions, Inc. **Table Number: 19**

7796 Victor-Mendor Road
Victor, NY 14564
Phone: 585/869-6614
Fax: 585/742-8086
Email: astringer@lsisolutions.com

LSI Solutions® proudly introduces The Running Device™ Surgery's Best Suturing Technology™. This new reloadable 5mm laparoscopic devise enables fast, easy and precise intracorporeal suture placement with almost no learning curve. The Running Device™ incorporates a unique protected safety needle to minimize exposed sharps to the patient and surgical team.

Memorial Health **Table Number: 21**

3535 Peachtree Street, Suite 520-273
Atlanta, GA 30144
Phone: 770/422-3367
Fax: 770/419-3367
Email: mdowns@williamlaine.com

Savannah's Memorial Health University Medical Center and Backus Children's Hospital are recruiting a surgeon to join our growing practice. Memorial Health is the only healthcare system in the country to be named a "Distinguished Hospital" by J.D. Power and Associates and a "100 Best Companies to Work For" by Fortune Magazine.

Moses Cone Health System **Table Number: 23**

1200 N. Elm Street
Greensboro, NC 27401
Phone: 336/832-7784
Fax: 336/832-7782
Email: cindy.bagwell@mosescone.com

Moses Cone Health System, located in Greensboro, NC, and UNC — Chapel Hill are jointly recruiting a pediatric surgeon. MCHS is a 1,000+ bed, five-hospital, not-for-profit, community system with a Level III, 36-bed NICU housed at the Women's Hospital (6,000 deliveries each year) and a four-bed PICU at Moses Cone Hospital.

Mountain States Health Alliance **Table Number: 2**

One Professional Park Drive, # 11
Johnson City, TN 37604
Phone: 800/876-5071
Fax: 423/431-2856
Email: physicianrecruitment@msha.com

Private, non-for-profit, nine-facility hospital system has exciting opportunities for pediatricians in picturesque mountains of northeast Tennessee to work with Children's Hospital at Johnson City Medical Center and local medical school. Area offers unequalled lifestyle, low cost of living, and excellent schools. Competitive salary/benefits packages. Call 800/876-5071 or email physicianrecruitment@msha.com

Olympus Surgical America, Inc. **Table Number: 4**

2 Corporate Center Drive
Melville, NY 11747
Phone: 845/398-9484
Fax: 845/398-9443
Email: beth.sweatt@olympussurgical.com

Olympus is focused on enhancing people's lives with a wide range of products and services. We are focused on being a useful resource to make your experience valuable and efficient. Olympus' goal is to always be on the cutting-edge and to provide you with the most current surgical optical equipment.

Omni-Tract Surgical **Table Number: 17**

4849 White Bear Parkway
St. Paul, MN 55110
Phone: 651/287-4340
Fax: 651/287-4488
Email: michelleb@omni-tract.com

Omni-Tract® Surgical designs, develops and manufactures table-mounted retractor systems offering unparalleled exposure for pediatric procedures. For over 30 years, surgeons world-wide have relied on us for innovation in meeting exposure requirements. Our new Omni-Flex Retractor System offers you the flexibility and strength for a variety of procedures and patient sizes.

Oxford University Press **Table Number: 13**

198 Madison Avenue
New York, NY 10016
Phone: 212/726-6079
Fax: 212/726-6449
Email: mary.ann.benner@oup.com

Please visit our booth featuring the titles from Hodder Arnold and Oxford University Press including A Paediatric Vade-mecum, Fourteenth Edition, Edited by Timothy G. Barrett, Anthony D. Lander and Vin Diwaker; Farquharson's Textbook of Operative General Surgery, Ninth Edition, Margaret Farquharson and Brendan Moran; and Practical Approach to Pediatric Intensive Care, Edited by Praveen Khilnani.

Specialty Surgical Products, Inc. **Table Number: 8**

1131 North U.S. Highway 93
Victor, MT 59875
Phone: 406/961-0102
Fax: 406/961-0103
Email: info@ssp-inc.com

Specialty Surgical Products' Clear Cast Silicone Silo Bags for staged repair of Gastroschisis and Omphalocele offer you the greatest content visualization during reduction and the sizer ring enclosed with each devise enables you to verify silo bag size selection before opening the devise. Pediatric anal dilators available in sizes 3mm - 19mm.

Stryker Endoscopy **Table Number: 5**

5900 Optical Court
San Jose, CA 95138
Phone: 408/754-2709
Fax: 408/754-2935
Email: megan.kundert@stryker.com

Stryker Corporation is one of the world's leading medical technology companies. The company's products include implants used in joint replacement, trauma, craniomaxillofacial and spinal surgeries; biologics; surgical, neurologic, ear, nose & throat and interventional pain equipment; endoscopic, surgical navigation, communications and digital imaging systems; as well as patient handling and emergency medical equipment.

Teddy Tech, Inc. **Table Number: 1**

2016 Yale Avenue
Camp Hill, PA 17011
Phone: 800/673-2407
Fax: 717/975-3566
Email: coughbuddy@teddy-tech.com

"Cough Buddy" appears to be just a teddy bear but is, in fact a specially designed pillow to help you cough and deep breathe after open heart, thoracic or abdominal surgery. "Cough Buddy" is designed to have a firm back so that he can serve as a splint for the patient's incision.

W. Lorenz Surgical, Inc. **Table Number: 18**

1520 Tradeport Drive
Jacksonville, FL 32218
Phone: 904/741-4400
Fax: 904/741-3912

W. Lorenz Surgical, Inc. is a leading developer, manufacturer and distributor of advanced craniomaxillofacial products. Featured innovative products include LactoSorb® an advanced resorbable fixation system and the Lorenz Pectus Bar used to aid in the correction of Pectus Excavatum.

West Virginia University — Charleston Division and Charleston Area Medical Center **Table Number: 20**

511 Brooks Street
Charleston, WV 25301
Phone: 304/338-3347
Fax: 304/388-6297
Email: carol.wamsley@camc.org

Women and Children's Hospital and West Virginia University - Charleston Division are recruiting a BC/BE Pediatric Surgeon to join a busy university practice affiliated with the only designated children's hospital in WV. Stop by for information and register for a WV products basket featuring specialty/gourmet foods, candles, CD's and other items.

Underlining denotes the author scheduled to present at the meeting.