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

We do this by:

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

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Go to the APSA website at [www.eapsa.org](http://www.eapsa.org) and:
- Join the discussions on the All-Member Group
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1972-1973

Thomas M. Holder
1975-1976
Alexander H. Bill
1976-1977

William B. Kiesewetter
1981

E. Thomas Boles, Jr.
1977-1978

W. Hardy Hendren
1981-1983

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1978-1979

Lester W. Martin
1983-1984

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1979-1980

Judson G. Randolph
1984-1985

Thomas V. Santulli
1680-1981

Dale G. Johnson
1985-1986
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J. Alex Haller, Jr.
1986-1987

Alfred A. deLorimier
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1987-1988

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1988-1989

Raymond A. Amoury
1993-1994

Eric W. Fonkalsrud
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Jay L. Grosfeld
1994-1995

Robert M. Filler
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Arvin I. Philippart
1995-1996
APSA PAST PRESIDENTS (CONT.)

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1996-1997

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2001-2002

H. Biemann Othersen, Jr.  
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Marc I. Rowe  
1998-1999

Bradley M. Rodgers  
2003-2004

Kathryn D. Anderson  
1999-2000

Robert J. Touloukian  
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David Tapper  
2000-2001

M. Judah Folkman  
2005-2006
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2006-2007

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PAST OFFICERS

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Federico A. Arcari ........................................... 1970–1971
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Dale G. Johnson .............................................. 1977–1979
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PAST OFFICERS (CONT.)

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Albert W. Dibbins .......................................................... 1989–1992
Patricia K. Donahoe ........................................................ 1990–1993
Moritz M. Ziegler ........................................................... 1992–1995
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Thomas M. Krummel ....................................................... 1999–2002
Keith E. Georgeson ......................................................... 2000–2003
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<table>
<thead>
<tr>
<th><strong>APSA REPRESENTATIVES</strong></th>
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**Pediatric Surgery Board**  
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### Committee on Ethics
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### Emergency Service – Hospital - Trauma
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### International Relations Committee
Roberta E. Sonnino

### Membership Services Liaison Committee – B/R
Henri R. Ford

### Membership – Trauma
David W. Tuggle
<table>
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<tr>
<th>Committee/Program</th>
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<tr>
<td><strong>National Surgical Quality Improvement Program</strong></td>
<td>Peter W. Dillon, Chair</td>
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<td>Keith T. Oldham</td>
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<td><strong>Steering Committee</strong></td>
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<td>Marshall Z. Schwartz</td>
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<td><strong>Publications – Trauma</strong></td>
<td><strong>Verification/Consultation – Trauma</strong></td>
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<td>Henri R. Ford</td>
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<td>Perry W. Stafford</td>
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<td><strong>Quality Assurance – Trauma</strong></td>
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<td>Joseph J. Tepas, III</td>
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</table>
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ARTICLES OF INCORPORATION OF THE
AMERICAN PEDIATRIC SURGICAL ASSOCIATION

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

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

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

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

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

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

Seventh: Upon dissolution of the Corporation, or any partial or entire liquidation of its property or assets, all of the Corporation’s property of every nature and description shall, after making provision for discharge of all of the liabilities of the Corporation, be paid over and transferred to such one or more organizations or institutions which are then exempt from federal income tax under Section 501(a) of the Code by reason of being described in either Section 501(c)(3) or Section 501(c)(6) of the Code, as shall be selected by a majority of persons who
Eighth: No member of the Board of Governors, officer, or employee of the Corporation, or any other person, shall receive any profit from the operations or liquidation of the Corporation, except as reasonable compensation for services actually rendered to the Corporation.

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

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

PRINCIPLES OF MEDICAL ETHICS

Members:

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

Article I: MEMBERSHIP

SECTION 1. REGULAR MEMBERSHIP

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

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

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

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

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

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

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

Section 2. Candidate Members
2.1. A candidate member must be currently licensed to practice surgery in the United States or Canada.
2.2. Candidate members must have successfully completed the examination in general surgery given by the American Board of Surgery or by the Royal College of Surgeons of Canada or, they must be eligible for examination by those respective boards.
2.3. Only residents in ACGME-approved pediatric surgical residency programs are eligible for candidate membership.
2.4. An individual may remain a candidate member for five years following completion of an approved pediatric surgical residency program at which time the candidate membership will expire. This five-year period is in addition to the time spent as a candidate member during pediatric surgery residency.
If candidate membership expires, one may still apply for regular membership at any time in the future. Candidate membership is not mandatory in order to qualify for regular membership.
2.5. A candidate member who has completed his/her training in an ACGME-approved pediatric surgery residency position or equivalent Royal College of Surgeons of Canada approved program must practice pediatric surgery exclusively as stipulated in section 1.4. for regular membership.
2.6. Candidate members are not eligible for appointment with voting privileges on standing or ad hoc committees, but may be appointed by the president as consultant members for a period not to exceed two years.
2.7. Candidate members will have the same meeting attendance requirements as regular members, but will not have voting privileges. Candidate members are not eligible to hold office. Candidate members will be subject to 20% of the current regular membership dues and will be governed by all other bylaws applicable to regular membership.
2.8. A candidate member will require sponsorship by a regular member for abstracts submitted for presentation to the annual APSA scientific meeting.

Section 3. Charter Membership
3.1. A charter membership shall be extended to a person actively engaged in the practice of pediatric surgery, who has already amply demonstrated excellence and fitness as a trained specialist in pediatric surgery, who has devoted his practice to pediatric surgery and who is certified by the American Board of Surgery or by the Royal College of Surgeons of Canada.
3.2. A list of charter membership was established and then closed on April 15, 1970.

Section 4. Honorary Membership
4.1. Honorary membership may be conferred upon a physician for outstanding contributions to pediatric surgery by unanimous vote of the board of governors and an affirmative vote by two-thirds of the voting membership attending the annual meeting business meeting.
4.2. Honorary members will be governed by the bylaws as regular members but will not be subject to dues or the meeting attendance requirement and will not be eligible to hold office.
Section 5. International Membership

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

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

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

Section 6. Associate Members

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

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

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

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

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

Section 7. Resident Members

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

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

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

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

Section 8. Application Procedures

8.1. New applications for regular, associate or international membership will be initiated by the prospective member. For regular membership, the
procedure may begin prior to the completion of the required one year of pediatric surgery practice. See Article 1, Section 1.5. The application will need supporting letters from three members in good standing. One of these three letters must be from the training director of the prospective member. At least one sponsor must attest that the applicant exemplifies a high standard of ethical behavior as set forth in the principles of medical ethics in the preamble to the bylaws.

Applicants for international membership will require one additional letter of recommendation from a physician who is acquainted with the individual's professional competence and ethics in his/her own practice community.

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

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

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

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

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

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

Section 9. Application Form
9.1. The application shall include:
9.1.1. Curriculum vitae
9.1.2. Bibliography
9.1.3. Applicants for regular or international membership must submit a tabulation, by case, of the operative experience of the applicant during the 12-month period immediately preceding his/her application. Applicants for associate membership must submit a tabulated operative experience covering the 24-month period immediately preceding his/her application. All operative reports must be signed by the chief(s) of surgery where the applicant works. The report should indicate whether the applicant was surgeon, first or teaching assistant.

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

Section 10. Resignation
10.1. Any member may submit his/her resignation at any time in writing to the president to be effective on the date of submission.

Section 11. Fiscal Year
11.1. The fiscal year shall be from January 1 to December 31.

Section 12. Dues
12.1. Dues shall be set by the board of governors and approved by the membership at the annual meeting. Dues will be announced by letter by the first day of October and must be paid by the first day of December.
12.2. No annual dues shall be required of a member following his/her 65th birthday or upon retirement from active practice whichever is sooner. (Member will be termed a “senior member.”) No annual dues shall be required of any member during any year that person is disabled and unable to practice for six months or more.
12.3. Under special circumstances and by approval of the board of governors, dues may be waived for any member for one calendar year.
12.4. An initiation fee equal to one-half of the annual dues will be levied on all new members at the time of their induction into membership in the organization. This fee must be paid prior to issuing a certificate of membership.

Section 13. Certificate of Membership
13.1. A certificate of membership will be designed and issued to each member, signed by the president and the secretary.

Section 14. Loss of Membership
14.1. A member may be dropped from membership for:
14.1.1. Missing three consecutive meetings without written excuse, submitted
to the secretary and considered justifiable by the board of governors. Members over 60 years of age, honorary, international and senior members will be excused from this requirement.

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

14.1.3. Failure to remit dues within six months of the announced date will result in loss of membership in the Association. Members in arrears will receive a registered letter at least one month prior to the date of loss of membership outlining this action. Reinstatement of membership may be obtained by petitioning the board of governors. Payment of past dues owed as well as a reinstatement fee equal to the initiation fee for the organization will be required to resume membership.

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

14.3. Discipline.

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

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

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

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

14.3.2.3. Grossly immoral, dishonorable or unprofessional conduct.

14.3.3. Due process.

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

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

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

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

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

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

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

14.3.3.2.2. The member shall be informed that he/she may appear at the meeting in person and with counsel, if he/she so elects, so as to state his/her response to the charges.
14.3.3.3. The board of governors shall consider the recommendation(s) of the ad hoc committee at its next regular meeting or upon extraordinary session, but no earlier than thirty days from time of the member’s notification.

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

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

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

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

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

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

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

### Article II OFFICERS

#### Section 1. The Officers

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

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

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

#### Section 2. Term of Office

2.1. The terms of each above office shall be:

<table>
<thead>
<tr>
<th>Office</th>
<th>Term</th>
</tr>
</thead>
<tbody>
<tr>
<td>President</td>
<td>1 year</td>
</tr>
<tr>
<td>President-Elect</td>
<td>1 year</td>
</tr>
<tr>
<td>Secretary</td>
<td>3 years</td>
</tr>
<tr>
<td>Treasurer</td>
<td>3 years</td>
</tr>
</tbody>
</table>
Article III  BOARD OF GOVERNORS

Section 1. Membership of the Board of Governors

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

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

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

Section 2. Chair of the Board of Governors

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

Section 3. Functions of the Board of Governors

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

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

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

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

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

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

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

Article IV  DUTIES OF OFFICERS

Section 1. The President

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

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

1.3. Shall sign all official documents.

1.4. Shall make appropriate committee appointments.

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

Section 2. The President-Elect

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

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

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

2.4. Shall become president the next year.
### Section 3. The Secretary

<p>| | |</p>
<table>
<thead>
<tr>
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</tr>
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<tbody>
<tr>
<td>3.1.</td>
<td>Shall record the proceedings at all meetings.</td>
</tr>
<tr>
<td>3.2.</td>
<td>Shall notify the membership of all meetings and publish and distribute the agenda of the annual meeting business meeting.</td>
</tr>
<tr>
<td>3.3.</td>
<td>Shall maintain a registry of membership.</td>
</tr>
<tr>
<td>3.4.</td>
<td>Shall conduct appropriate correspondence and maintain a file of such.</td>
</tr>
<tr>
<td>3.5.</td>
<td>Shall submit a report of the minutes of the previous annual business meeting.</td>
</tr>
<tr>
<td>3.6.</td>
<td>Upon the disability of the president and then the president-elect, shall assume the office of the president automatically—to serve only until the next annual meeting.</td>
</tr>
</tbody>
</table>

### Section 4. The Treasurer

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

### Article V MEETINGS

#### Section 1. Annual Meeting

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

#### Section 2. Guests and the Annual Meeting

<p>| | |</p>
<table>
<thead>
<tr>
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</thead>
<tbody>
<tr>
<td>2.1.</td>
<td>The scientific sessions of the annual meeting shall be open to all interested physicians who register for the meeting.</td>
</tr>
<tr>
<td>2.2.</td>
<td>Interested paramedical professionals may be invited by any member in good standing.</td>
</tr>
<tr>
<td>2.3.</td>
<td>A registration fee may be required of non-members and guests at the discretion of the program committee.</td>
</tr>
<tr>
<td>2.4.</td>
<td>The privilege of the floor at the scientific sessions will be restricted to the membership and to others who have been given official designation by letter from the secretary.</td>
</tr>
</tbody>
</table>
Section 3. Quorum

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

Article VI BYLAWS

Section 1. Time of Effect

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

Section 2. Amendments of the Bylaws

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

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

Article VII PERMANENT COMMITTEES

Section 1. Permanent Committees

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

Article VIII AD HOC COMMITTEES

Section 1. Membership

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

Article IX REPRESENTATION TO OTHER SOCIETIES

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

Article X HISTORIAN

An historian shall be appointed by the president.
Article XI    OFFICIAL SEAL

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

Article XII    INDEMNIFICATION AND INSURANCE

Section 1.     Indemnification

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

Section 2.     Insurance

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

Approved May 30, 2009
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APSA
FOUNDATION
In 1991, a small group of APSA members discussed establishing a foundation for APSA which would foster support for scientific investigation in the field of children’s surgery by providing an Annual Enrichment Grant to qualified applicants.

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

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

By its third year, contributors to the APSAF had increased to 7% of the total APSA membership. In 1998, efforts to enhance donor participation were increased by further formalization of the donation process. Dr. Grosfeld was asked to continue as Chairman of the Board of Directors, serving indefinitely at the discretion of the Board. In 2003, Dr. Wilkinson stepped down as Foundation Agent and was replaced by Dr. John Noseworthy. Gift level categories were established including Donor (up to $1,000), Gold Donor ($1,000 or more), Robert E. Gross benefactor ($5,000) and more recently, the William A. Ladd Society level of giving ($10,000) was added. The latter three levels can be achieved by cumulative annual gifts. The active APSA president was considered an ex officio member of the Foundation Board, and in 2007, a second at-large member was added to the Board.
The corpus of the Foundation has at times exceeded $500,000 despite the fact that less than 20% of the membership have as yet contributed to the Fund. The stipend for each grant has gradually increased, at first to $10,000, and in the past five years to $25,000. Currently, two grants are awarded annually. Since its inception, the APSA Foundation has provided more than $385,000 in grant support for our young pediatric surgeon-scientists (see list below). The return on investment has been extraordinary!

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

**APSAF BOARD OF DIRECTORS**

**Chairman**  
Jay L. Grosfeld  
Indianapolis, IN

**Secretary**  
Diana L. Farmer  
San Francisco, CA

**Treasurer**  
Dennis P. Lund  
Madison, WI

**Members**  
Keith E. Georgeson  
Spokane, WA  
Michael R. Harrison  
San Francisco, CA  
Jean-Martin Laberge  
Montreal, Canada  
Thomas F. Tracy, Jr.  
Providence, RI  
Moritz M. Ziegler  
Cincinnati, OH

**Foundation Agent**  
John Noseworthy  
Jacksonville, FL

**Ex Officio Member**  
Marshall Z. Schwartz  
Philadelphia, PA  
APSA President
APSA FOUNDATION GRANT RECIPIENTS

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

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

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

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

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

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

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

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

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

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

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

Christopher K. Breuer, MD
Do Tissue Engineered Venous Conduits Grow? Investigating the Growth Potential of Tissue Engineered Venous Conduits in a Juvenile Lamb Model
APSA FOUNDATION GRANT RECIPIENTS (CONT.)

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

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

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

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

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

APSA FOUNDATION CONTRIBUTORS

The American Pediatric Surgical Association Foundation thanks the following individuals who have contributed to the Foundation.

WILLIAM E. LADD SOCIETY BENEFAC'TORS - $10,000
Grosfeld, Jay L.
Hays, Daniel M.
Knowles, Joan
Noseworthy, John
West, Karen W.
ROBERT E. GROSS BENEFACTORS - $5,000
Altman, R. Peter
Ashcraft, Keith W.
Bleacher, John C.
Caniano, Donna A.
Coran, Arnold G.
Donahoe, Patricia K.
Dunn, James
Engum, Scott A.
Filston, Howard C.
Ford, Henri R.
Gilchrist, Brian F.

Harrison, Michael R.
Hendren, W. Hardy
Klein, Michael D.
Lankau, Charles Jr.
LaQuaglia, Michael P.
Oldham, Keith T.
Puranik, Subhash, R.
Rescorla, Frederick J.
Rodgers, Bradley, M.
Rouse, Thomas M.
Scherer, L.R. (Tres)

Schmeling, David J.
Schwartz, Marshall Z.
Shamberger, Robert C.
Sherman, Neil J.
Toyama, William M.
Tunell, William P.
Weinberger, Malvin
Wesley, John R.
Ziegler, Moritz M.

APSA GOLD SUPPORTER LEVEL BENEFACTORS - $1,000
Acton, Robert D.
Alaish, Samuel M.
Anderson, Glen F.
Anderson, Kathryn D.
Andrews, David A.
Arensman, Robert M.
Ashcraft, Keith W.
Azizkhan, Richard G.
Baldwin, Charles E.
Barlow, Barbara A.
Beaver, Bonnie L.
Beierle, Elizabeth A.
Besner, Gail E.
Billmire, Deborah F.
Bliss, David W.
Bower, Richard J.
Brand, Theodore
Breaux, Charles W. Jr.
Brennnom, William S.
Browne, Allen F.
Burnweit, Cathy A.
Campbell, John R.
Carr, Michael G.
Cavett, Clinton
Chahine, A. Alfred
Christian, Jeffrey S.
Coln, C. Dale
Cosentino, Catherine
Coughlin, John P.
Curci, Michael R.
DeLorimier, Alfred A.
Dibbins, Albert W.

Dimler, Michael W.
Dolgin, Stephen E.
Doody, Daniel P.
Doolin, Edward J.
Dorman, George W.
Downey, Earl C. Jr.
Drucker, David E. M.
Ein, Sigmund H.
Ellis, Dick G.
Emmens, Robert W.
Fallat, Mary E.
Farmer, Diana L.
Feins, Neil
Filler, Robert M.
Folkman, M. Judah
Fonkalsrud, Eric W.
Ford, Edward G.
Gandhi, Rajinder P.
Gauderer, Michael W. L.
Gingalewski, Cynthia A.
Ginsburg, Howard B.
Glick, Philil L.
Goodwin, Charles B.
Gropp, Diller B.
Grone, Jonathan I.
Gutman, Frank M.
Guzzetta, Philip C.
Harris, Burton H. &
Kathleen
Healey, Patrick J.
Hicks, Barry A.
Hill, J. Laurance

Hirschl, Ronald B.
Hoelzer, Dennis J
Holcomb, George III
Holder, Thomas M.
Holterman, Mark J &
Ai-Xuan L.
Hulka, Frieda M.
Jan, Dominque M.
Johnson, Dale G.
Jolley, Stephen G.
Kays, David W.
Kessler, Edmund
Kim, Peter C. W.
Kim, Samuel H.
King, Denis R.
Klotz, Donald Jr.
Krummel, Thomas M.
Laberge, Jean-Martin
Ladd, Alan P.
Lam, Vinh T.
Langenburg, Scott E.
Langer, Jacob C.
Latchaw, Laurie A.
Ledbetter, Daniel J.
Lewis, J. Eugene
Lobe, Thom E.
Loeff, Deborah S.
Long, Julie A.
Luks, Francois I.
Lund, Dennis P.
Lynch, James M.
Maddox, John R.
APSA FOUNDATION CONTRIBUTORS (CONT.)

Madonna, Mary B.            Priebe, Cedric J.            Stylianos, Steven
Magnusun, David K.              Radhakrishnan, Jayant            Superina, Riccardo A.
Marr, Clifford C.                  Randolph, Judson            Talbert, James L.
McGahren, Eugene D.             Raynor, Stephen C.            Taylor, Lesli A.
McGill, Leigh C.                Reyes, Hernan M.            Templeton, John M.
Meier, Donald E.                Rice, Henry E.              Tepas, Joseph J.
Morgan, William W.              Ricketts, Richard R.            Touloukian, Robert. J.
Moriarty, Kevin P.              Rouse, Thomas M.            Tracy, Thomas F.
Moss, R. Lawrence               Rowe, Marc I.                Turner, Charles S.
Murphy, John P.                Ryckman, Frederick C.            Upperman, Jeffrey S.
Newman, Barry M.               Sawin, Robert            Vane, Dennis W.
O’Neill, James A.              Saxton, Mark L.            Vegunta, Ravindra K.
Ortiz-Justiniano, Victor N.     Schullinger, John N.            Wagner, Charles W.
Ostlie, Daniel J.            Shaul, Donald B.              Waldhausen, John H.T.
Othersen, H. Biemann           Silen, Mark L.                Weber, Thomas R.
Pearl, Richard H.               Skinner, Michael A.         Whalen, Thomas V.
Pinch, Lewis W.                  Slim, Michael S.            Wiener, Eugene S.
Pittinger, Timothy P.              Smith, C.D.              Wilkinson, Albert H.
Powell, Randall W.             Snyder, Charles J.
Powell, David M.               Statter, Mindy B.

DONORS
Adamson, William T.              Martin, Abigail E.
Barnhart, Douglas C.            Miniati, Douglas N.
Bourque, Michael D.            Moore-Olufemi, Stacey
Breuer, Christopher K.            Nuss, Donald
Brown, Rebeccah L.              Pendse, Prabhakar D.
Casas-Melley, Adela              Ramenofsky, Max L.
Cywes, Robert                Ratner, Michael H.
DeRoss, Anthony              Reynolds, Marleta
Ehrlich, Peter F.                Rosser, Samuel B.
Falcone, Richard A. Jr.               Shaw, Anthony
Fiore, Nicholas F. Jr.          Stringel, Gustavo, L.
Fischer, Anne C.               Teitelbaum, Daniel H.
Fishman, Steven J.            Trump, David S.
Friedman, David L.            Valda, Victor
Fryckman, Philip K.              Wesson, David C.
Gibbs, David L.                Yamataka, Atsuyuki
Glynn, Loretto A.            Zitsman, Jeffrey S.
MEMBERSHIP
AWARD RECIPIENTS

APSA Distinguished Service Award Recipients
Stephen L. Gans
Marc I. Rowe
Thomas M. Holder
Lucian L. Leape
Harvey E. Beardmore
W. Hardy Hendren

ACS/APSA Executive Leadership Program in Health Policy and Management
Patrick V. Bailey - 2011
Aviva L. Katz - 2010
Dennis P. Lund - 2009
George W. Holcomb, III - 2008

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

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

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

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

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

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

2008
Emily T. Durkin, MD
The Ontogeny of Human Fetal NK Cell Allorecognition: A Potential Barrier to in Utero Transplantation
AWARD RECIPIENTS (CONT.)

APSA Posters of Distinction

Basic Science

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

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

Clinical

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

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

NEW MEMBERS 2010-2011

APSA Board of Governors and Membership Congratulates Our Newest Members

Regular Members

Aidlen, Jeremy T. Hirose, Shinjiro Qureshi, Faisal G.
Aldrink, Jennifer H. Hoover, John D. Rangel, Shawn J.
Avansino, Jeffrey R. Hui, Thomas T.L. Renaud, Elizabeth J.
Beanever, Gregory T. Javid, Patrick J. Rothenbach, Thomas A.
Barnett, Sean S. Ignacio, Romeo C. Rothstein, David H.
Berch, Barry R. Kayton, Mark L. Ryckman, Jon G.
Browne, Marybeth Koontz, Curt S. Safford, Shawn D.
Burjonrappa, Lau, Stanley T. Sawaya, David E.
Sathyaprasad C. Leys, Charles M. Schulman, Andrew M.
Chong, Albert J. Little, Danny C. Somme, Stig
Cina, Robert A. Lopez, Monica E. Stewart, F. Dylan
Deans, Katherine J. Mak, Grace Z. Streck, Christian J.
Densmore, John C. Martin, Abigail E. Timapuri, Shaheen J.
Durham, Megan M. McKee, Jason Q. Tsao, KuoJen
Egan, J. Craig Miniati, Douglas N. Weinsheimer, Robert L.
Escobar, Mauricio A. Moore-Olufemi, Stacey D. Weldon, Christopher B.
Eubanks, James W. Mueller, Claudia M. Woo, Russell K.
Frischer, Jason S. Nathan, Jaimie D. Zallen, Garret S.
Gates, Robert L. Newton, Christopher R. Zarroug, Abdalla E.
Halter, Jeffrey M. Pham, Tuan H.
Hernandez, Ambrosio III
NEW MEMBERS 2010-2011 (CONT.)

Candidate Members
Alder, Adam C.               Henry, Marion C.W.
Austin, Mary T.              Hunter, Catherine J.
Badillo, Andrea T.           Jamshidi, Ramin
Bernabe, Kathryn Q.          Jarboe, Marcus D.
Bruzoni, Matias              Juang, David
Chun, Jeannie Y.             Keswani, Sundee G.
Clifton, Matthew S.          Kreykes, Nathaniel S.
Donoghue, Lydia J.           Larson, Shawn D.
Draus, John M.               Lee, Timothy C.
Duke, Duane S.               Lombardo, Michele L.
Dzakovic, Alexander          Mattix, Kelly D.
Gonzalez, Raquel             Mills, Jessica L.A.
Gosain, Ankush               Newman, Erika L.
Greenspon, Yosef J.          Norelius, Rona L.
Grethel, Eric J.             Pandya, Samir R.
Hansen, Erik N.              Perger, Lena
Radhakrishnan, Ravi S.
Rana, Ankur R.
Rasmussen, Sara K.
Rauth, Thomas P.
Riehle, Kimberly J.
Rodriguez, Jose Ruben
Scholz, Stefan
Skarda, David E.
Stafford, Shawn J.
Stehr, Wolfgang
Stephenson, Jacob T.
Uffman, John K.
Wagner, Amy J.
Yoo, Edward Y.
Zeller, Kristen A.

Associate Members
Kumar, Tarun
Moores, Donald C.

International Members
Muhssein, Haidar M.

Resident Members (General Surgery Residents)
Aarabi, Shahram               Honeyman, Joshua N.
Baregami, Naira               Iqbal, Corey W.
Berdan, Elizabeth A.          Lazar, David A.
Berman, Loren                Le, Louis D.
Biller, Christina             Lesher, Aaron P.
Bogert, James M.              Mace, Adam G.
Bozeman, Andrew P.            McEvoy, Maureen P.
Buesing, Keely L.             Murphy, Andrew J.
Diesen, Diana L.              Nicksa, Grace A.
Falk, Gavin A.                Oyetunji, Tolulope A.
Fialkowski, Elizabeth A.      Raval, Mehul V.
Fraser, Jason D.              Redlinger, Richard E.
Fuller, Megan K.              Rich, Barrie S.
Gendy, Amir S.                Rivas, Erick F.
Gill, Richdeep                Russell, Robert T.
Gray, Brian W.                Saadai, Payam
Saites, Constantine G.
Santore, Matthew T.
Schwartz, Jennifer A.
Seims, Aaron D.
Shirah, Gina R.
Short, Scott S.
Snyder, Christopher W.
Tracy, Elisabeth R.
Van Arendonk, Kyle J.
Westmoreland,
Tamarah J.
Williams, Regan, F.
Wood, James H.
Zequeira, Jorge J.
PLEDGE FOR NEW MEMBERS

Pledge for New Members of the American Pediatric Surgical Association

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

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

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

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

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

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

<table>
<thead>
<tr>
<th>Name</th>
<th>Year</th>
</tr>
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<tbody>
<tr>
<td>Gilbert, Michel</td>
<td>1972</td>
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<tr>
<td>Gamion, Robers S. Jr.</td>
<td>1973</td>
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<tr>
<td>Chamberlain, John W.</td>
<td>1974</td>
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<td>Snyder, William H. Jr.</td>
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<td>Bracey, Altamount</td>
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<td>Erwin, James H.</td>
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<td>White, Robert F.</td>
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<td>Karn, Gordon M.</td>
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<td>Hawes, Ernest B.</td>
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<td>Lozoya-Solis, Jesus</td>
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<td>Soave, Franco</td>
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<td>Rosenkrantz, Jens G.</td>
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<td>Cresson, Samuel L.</td>
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<td>Owings, Richard S.</td>
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<td>Pilling, George P. IV</td>
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<td>Stewart, David R</td>
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<td>Simpson, James Stanley</td>
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<td>Gross, Robert E.</td>
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<td>Ravitch, Mark M.</td>
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<td>Ballantine, Thomas V.N</td>
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<td>Ferguson, Colin C.</td>
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<td>Mishalany, Henry</td>
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<td>Kaufman, Bruce</td>
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<td>Sakaguchi, Shimpei</td>
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<td>Segnitz, Richard H.</td>
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<td>Gans, Stephen L.</td>
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<td>Kumar, A.P. Mahesh</td>
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<td>McParland, Felix A.</td>
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<td>Pokorny, William J.</td>
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<td>Richardson, William R.</td>
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<tr>
<td>Benson, Clifford D.</td>
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<td>Lilly, John R.</td>
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<td>Riker, William L.</td>
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<td>Bill, Alexander H. (Sandy)</td>
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<td>Cheu, Henry W.</td>
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<td>Danis, Richard K.</td>
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<td>Goldstein, I. Richard</td>
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<td>Longino, Luther A.</td>
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<td>Welch, Kenneth J.</td>
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<td>Bettex, Marcel</td>
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<td>Brennan, L. Patrick</td>
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<td>Hamilton, James P.</td>
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<td>Stanley-Brown, Edward G.</td>
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<td>Knutrud, Ola</td>
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<td>Warden, M. James</td>
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<td>Winslow, Paul</td>
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<td>Zachary, R.B.</td>
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<td>Linkner, Laurence M.</td>
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<td>Colodny, Arnold H.</td>
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<td>So, Henry B.</td>
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<td>Zwiren, Gerald T.</td>
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<td>Abrams, Martin W.</td>
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<td>Harberg, Franklin J. (Jim)</td>
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<td>Lynch, Frank P. III</td>
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<td>Smith, E. Ide</td>
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<td>Rickham, Peter P.</td>
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<td>Huseby, Thomas L.</td>
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<td>Pickett, Lawrence K.</td>
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<td>Bronsther, Burton</td>
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<td>Stahl, Nicholas M.</td>
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<td>Phillipart, Arlvin I. III</td>
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<td>McAlpin, Columbus D.</td>
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<td>Lloyd, James R.</td>
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<td>Rathouser, Frank</td>
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<td>Fitzpatrick, John</td>
<td>2005</td>
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<td>Able, Luke W.</td>
<td>2006</td>
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</tbody>
</table>
IN MEMORIAM

Andrews, Gibb .............. 2006
Jewett, Theodore C. ......... 2006
Rothmann, Bruce F. ......... 2006
Wiener, Eugene S. .......... 2006
Beardmore, Harvey E. ....... 2007
Black, Preston R. ............ 2007
Cox, Joseph A. .............. 2007
Exelby, Philip R. ............. 2007
Mollitt, Daniel L. ............. 2007
Ratner, Irving A. ............ 2007
McClennathan, James E. .... 2007
Wolfson, Philip J. ......... 2007
Folkman, M. Judah .......... 2007
Smith, Melvin D. .......... 2007
McGovern, Bruce .......... 2008
MacDonald, James S. ....... 2008
Campbell, Timothy J. ....... 2008
Votteler, Theodore P. ....... 2008
Cooney, Donald R. .......... 2008
Cooke, Ronald W. .......... 2009
Anderson, Alan E. .......... 2009
de Lorimier, Alfred A. .... 2009
Fisher, John H. .......... 2009
Mercer, Stanley ........... 2010
Schultz, Lloyd R. .......... 2010
Hayes, Lawrence E. ....... 2010
Besser, Arthur S. ........ 2010
Arie D. Verhagan .......... 2010

FOUNDING MEMBERS

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

CHARTER MEMBERS

Raymond A. Amoury, Kansas City, MO
H. Paulsen Armstrong, Baton Rouge, LA
A. Robert Beck, New York, NY
Jerrold M. Becker, New Hyde Park, NY
Clifford R. Boeckman, Salem, SC
Scott J. Boley, Bronx, NY
William E. Bomar, Gray Court, SC
John D. Burrington, Colorado Springs, CO
John L. Cahill, Indian Wells, CA
Walter S. Cain, Birmingham, AL
Gordon S. Cameron, Dunas, ON, Canada
Daniel T. Cloud, Phoenix, AZ
David L. Collins, San Diego, CA
Elizabeth Coryllos, Mineola, NY
C. Peter Crowe, Tucson, AZ
Joseph S. David, Eagle, ID
Jean G. DesJardins, Saint-Laurent, QC, Canada
Pieter A. deVries, Larkspur, CA
George W. Dorman, Prescott, AZ
Jacques C. Ducharme, Mont Royal, QC, Canada
Dick G. Ellis, Fort Worth, TX
John H. Fisher, Marshfield, MA
Eric W. Fonkalsrud, Santa Monica, CA
Eugene Garrow, Jersey City, NJ
Marvin Glicklich, Fox Point, WI
Leonard Graivier, Dallas, TX
Jacob A. Haller, Glencoe, MD
Daniel M. Hays, Riverside, CA
Bruce M. Henderson, Corpus Christi, TX
W. Hardy Hendren, Duxbury, MA
Jack H. Hertzler, Franklin, MI
George W. Holcomb, Nashville, TX
Thomas M. Holder, Prairie Village, KS
James W. Hopkins, Windsor Heights, IA
George A. Hyde, Horare, Avondale, Zimbabwe
Patrick F. Jewell, Lincoln, CA
Frank R. Johnson, Woodstock, IL
Kenneth Kenigsberg, Glen Cove, NY
William N. Kincannon, Santa Barbara, CA
Murray R. Kliman, Vancouver, BC, Canada
Charles H. Klippel, Paxton, MA
Irwin H. Krasna, Forest Hills, NY
Dennis J. Lafer, Jacksonville, FL
J. Eugene Lewis, St. Louis, MO
Peter S. Liebert, White Plains, NY
Hugh B. Lynn, Winchester, VA
Enrique Marquez, San Juan, PR
Lester W. Martin, Bellbrook, OH
R. W. Paul Mellish, Dhahran, Saudi Arabia
Ascher L. Mestel, Brooklyn, NY
Richard C. Miller, Jackson, MS
David R. Murphy, Kingston, ON Canada

H. Biemann Othersen, Charleston, SC
Cedric J.Priewe, Stony Brook, NY
Thomas C. Putnam, Rockland, ME
Judson Randolph, Nashville, TN
Lester R. Sauvage, Seattle, WA
Louise Schnaufer, Philadelphia, PA
John N. Schullinger, Woodstock, VT
Lloyd Schultz, Omaha, NE
Samuel R. Schuster, Westboro, MA
Alan D. Shafer, Dayton, OH
Barry Shandling, Toronto, ON, Canada
Anthony Shaw, Pasadena, CA
Walton K.T. Shim, Honolulu, HI
Laurence A. Somers, Lafayette Hill, PA
Bernard J. Spencer, Sanibel Island, FL
Rowena Spencer, New Orleans, LA
Nicholas M. Stahl, Charlestown, RI
Felicien M. Steichen, Mamaroneck, NY
H. Harlan Stone, Glenville, NC
Kamthorn Sukarochana, Pittsburgh, PA
Orvar Swenson, Charleston, SC
Jessie L. Ternberg, St. Louis, MO
Robert J. Touloukian, New Haven, CT
David S. Trump, Grants Pass, OR
Kenneth R. Tyson, Burnet, TX
Arie D. Verhagen, Hamilton, OH
Vollrad J. Von Berg, Hot Springs, AR
Theodore P. Votteler, Dallas, TX
H. Warner Webb, Jacksonville, FL
John J. White, Seattle, WA
Albert H. Wilkinson, Jacksonville, FL
Morton M. Woolley, Rancho Mirage, CA
Earle L. Wrenn, Greensboro, NC
SCHEDULE AND PROGRAM
Back of Tab Page
Remove before Printing
# SCHEDULE AT A GLANCE

## Saturday, May 21

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>8:00 a.m. – 2:00 p.m.</td>
<td>APSA Board of Governors Meeting</td>
<td>Director’s Suite 3</td>
</tr>
<tr>
<td>2:00 – 8:00 p.m.</td>
<td>Pediatric Surgery Training Program Directors Meeting</td>
<td>Desert Salons 9-11</td>
</tr>
<tr>
<td>3:00 – 6:00 p.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
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<tr>
<td>3:00 – 6:00 p.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
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<tr>
<td>3:00 – 6:00 p.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
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<tr>
<td>6:00 – 10:00 p.m.</td>
<td>APSA Board of Governors Dinner</td>
<td>Mikado Restaurant</td>
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<tr>
<td>6:30 – 10:00 p.m.</td>
<td>Publications Committee Meeting</td>
<td>Director’s Suite 1</td>
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## Sunday, May 22

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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</thead>
<tbody>
<tr>
<td>6:00 – 8:00 a.m.</td>
<td>Committee Meetings</td>
<td>See page 69 for ancillary meeting schedule</td>
</tr>
<tr>
<td>7:00 a.m. – 5:00 p.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
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<tr>
<td>7:00 a.m. – 5:00 p.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
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<tr>
<td>7:00 a.m. – 5:00 p.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
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<tr>
<td>7:00 – 8:00 a.m.</td>
<td>Continental Breakfast</td>
<td>Desert West Foyer</td>
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<tr>
<td>7:45 – 8:00 a.m.</td>
<td>President’s Welcome</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>8:00 – 10:00 a.m.</td>
<td>Companion Hospitality Room Open for Registered Companions</td>
<td>Sea Grille, 1st Floor</td>
</tr>
<tr>
<td>8:00 – 11:00 a.m.</td>
<td><strong>Education Session I</strong> APSA-APSNA Combined Symposium: Intestinal Failure</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>10:00 – 11:30 am</td>
<td>Companions Meeting</td>
<td>Director’s Suite 1</td>
</tr>
<tr>
<td>10:45 – 11:00 a.m.</td>
<td><strong>APSA</strong> Lynne D. Farber, RN, MSN, CPNP-PC, AC</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>11:00 – 11:30 a.m.</td>
<td>Refreshment Break</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>11:30 a.m. – 12:30 p.m.</td>
<td><strong>Journal of Pediatric Surgery Lecture</strong> Professor Lewis Spitz The History of Paediatric Surgery in the United Kingdom and the National Health Service</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>12:30 – 12:45 p.m.</td>
<td>Box Lunch Pick-Up</td>
<td>Desert Salons Foyer</td>
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<td>12:45 – 1:45 p.m.</td>
<td><strong>Video Session with Lunch</strong></td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>2:00 – 4:00 p.m.</td>
<td><strong>Concurrent Sessions:</strong> Education Session II Quality Improvement and Reducing Variation</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td></td>
<td><strong>Education Session III</strong> Surgical Care of the Obese Child</td>
<td>Springs Salon F</td>
</tr>
<tr>
<td>4:00 – 5:00 p.m.</td>
<td>Wine and Cheese Reception</td>
<td>Desert West Foyer</td>
</tr>
</tbody>
</table>
**SCHEDULE AT A GLANCE (CONT.)**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
</table>
| 4:15 – 6:00 p.m. | **Concurrent Poster Sessions:**  
**Poster Session I**  
Clinical  
**Poster Session II**  
Basic Science and Fetal Surgery | Desert Salons 9-11  
Desert Salons 12-14 |
| 6:30 – 8:30 p.m. | Welcome Reception                                                                        | Springs Pool and Grove |

**Monday, May 23**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>6:30 – 7:30 a.m.</td>
<td>Committee Meetings</td>
<td>See page 69 for ancillary meeting schedule</td>
</tr>
<tr>
<td>6:30 – 10:00 a.m.</td>
<td>Poster Set Up</td>
<td>Desert Salons 9-14</td>
</tr>
<tr>
<td>6:30 a.m. – 1:00 p.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>6:30 a.m. – 1:00 p.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
</tr>
<tr>
<td>6:30 a.m. – 1:00 p.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>6:45 – 7:30 a.m.</td>
<td>Continental Breakfast</td>
<td>Springs Salons A-F</td>
</tr>
<tr>
<td>6:45 a.m. – 1:00 p.m.</td>
<td>Open</td>
<td>Springs Salons A-F</td>
</tr>
</tbody>
</table>
| 7:30 – 9:00 a.m. | **Scientific Session I**  
Clinical Surgery | Desert Salons 7-8 |
| 8:00 – 10:00 a.m. | Companion Hospitality Room  
Open for Registered Companions only                                   | Sea Grille, 1st Floor |
| 9:00 – 10:00 a.m. | **Robert E. Gross Lecture**  
Judson G. Randolph, MD  
*Notes on the Early Development of Pediatric Surgery in the United States* | Desert Salons 7-8 |
| 9:30 a.m. – 12:30 p.m. | Companion Tour – Living Desert Reserve:Tour Lobby  
“Animal Attraction” Tour  
Pre-Registration Required | Desert Salons 7-8 |
| 10:00 – 10:30 a.m. | Refreshment Break                                                                        | Springs Salons A-F |
| 10:00 a.m. – Noon | Posters open for viewing                                                                  | Desert Salons 9-14 |
| 10:30 – 11:45 a.m. | **Scientific Session II**  
Oncology and Critical Care                                                              | Desert Salons 7-8 |
| 11:45 a.m. – Noon | Introduction of New Members                                                               | Desert Salons 7-8 |
| Noon – 1:00 p.m. | **Presidential Address**  
Marshall Z. Schwartz, MD  
*Healthcare Quality, Access, Cost, Workforce and Surgical Education: The Ultimate Perfect Storm* | Desert Salons 7-8 |
| 1:00 – 2:30 p.m. | Benjy Brooks Society Meeting and Luncheon  
Pre-Registration Required                                                             | Director’s Suite 4 |
| 2:00 – 6:00 p.m. | Golf Tournament  
Pre-Registration Required                                                        | JW Marriott Valley Course |
<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>3:00 – 5:00 p.m.</td>
<td>Tennis Tournament Pre-Registration Required</td>
<td>JW Marriott Lawn &amp; Tennis Club</td>
</tr>
<tr>
<td>5:00 – 6:30 p.m.</td>
<td><em>Journal of Pediatric Surgery Reception</em></td>
<td>Director’s Suite 4</td>
</tr>
<tr>
<td>6:00 – 7:30 p.m.</td>
<td>Residents, International and New Members Reception (By Invitation)</td>
<td>The Pointe</td>
</tr>
<tr>
<td>6:00 – 10:00 p.m.</td>
<td>Complimentary shuttle transportation provided to/from the El Paseo area for registered APSA guests to dine off site – Optional</td>
<td>Tour Lobby</td>
</tr>
</tbody>
</table>

**Tuesday, May 24**

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>6:30 – 8:00 a.m.</td>
<td>Member Business Meeting with Breakfast</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>6:30 a.m. – 3:30 p.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>6:30 a.m. – 3:30 p.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
</tr>
<tr>
<td>6:30 a.m. – 3:30 p.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>7:00 – 8:00 a.m.</td>
<td>Continental Breakfast for Non-Members</td>
<td>Springs Salons A-F</td>
</tr>
<tr>
<td>7:00 a.m. – 3:30 p.m.</td>
<td>Posters open for viewing</td>
<td>Desert Salons 9-14</td>
</tr>
<tr>
<td>7:00 a.m. – 3:30 p.m.</td>
<td>Exhibits open</td>
<td>Springs Salons A-F</td>
</tr>
<tr>
<td>8:00 – 9:00 a.m.</td>
<td>Jay and Margie Grosfeld Lecture Anthony Atala, MD Regenerative Medicine: New Approaches to Healthcare</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>8:00 – 10:00 a.m.</td>
<td>Companion Hospitality Room Open for Registered Companions only</td>
<td>Sea Grille, 1st floor</td>
</tr>
<tr>
<td>9:00 – 10:30 a.m.</td>
<td><em>Scientific Session III</em> Fetal/Neonatal</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>10:30 – 11:00 a.m.</td>
<td>Refreshment Break</td>
<td>Springs Salons A-F</td>
</tr>
<tr>
<td>11:00 a.m. – 12:15 p.m.</td>
<td><em>Scientific Session IV</em> Basic Science</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>12:15 – 12:30 p.m.</td>
<td>Box Lunch Pick-Up</td>
<td>Desert Salons Foyer</td>
</tr>
<tr>
<td>12:30 – 1:30 p.m.</td>
<td>Innovation Session with Lunch The Sheikh Zayed Institute Award for Innovation in Pediatric Surgery will be announced during the President’s Banquet</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>1:30 – 1:45 p.m.</td>
<td>APSA Foundation Scholar Cynthia D. Downard, MD Control of Intestinal Microcirculation in NEC</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>1:45 – 2:30 p.m.</td>
<td>APSA Updates</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>Time</td>
<td>Event</td>
<td>Location</td>
</tr>
<tr>
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</tbody>
</table>
| 2:30 – 3:30 p.m. | **International Guest Lecture**  
Professor Takeshi Miyano  
*A Brief History of Pediatric Surgery and Healthcare Systems in Japan* | Desert Salons 7-8 |
| 3:30 – 5:30 p.m. | Poster Dismantle                  | Desert Salons 9-14 |
| 3:30 – 6:45 p.m. | Leisure time                       |                   |
| 6:45 – 7:30 p.m. | President’s Reception              | Springs Patio     |
| 7:30 – 10:00 p.m. | President’s Banquet                | Desert Salons 7-8 |

**Wednesday, May 25**

<table>
<thead>
<tr>
<th>Time</th>
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</tr>
</thead>
<tbody>
<tr>
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<td>Committee Meetings</td>
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<tr>
<td>7:00 – 10:30 a.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
</tr>
<tr>
<td>7:00 – 11:30 a.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>7:00 – 11:30 a.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>7:30 – 8:00 a.m.</td>
<td>Continental Breakfast</td>
<td>Desert West Foyer</td>
</tr>
</tbody>
</table>
| 8:00 – 9:00 a.m. | **Scientific Session V**  
Quality Improvement/Clinical Care | Desert Salons 7-8 |
| 9:00 – 10:00 a.m. | **COG Session**  
Germ Cell Tumors; Neuroblastoma | Desert Salons 7-8 |
| 10:00 – 10:15 a.m. | Refreshment Break                | Desert West Foyer |
| 10:15 – 11:30 a.m. | **Pediatric Surgery Case Debates and Controversies** | Desert Salons 7-8 |
| 11:30 a.m. | Annual Meeting Concludes          |                   |
### ANCILLARY MEETINGS—BY DAY

<table>
<thead>
<tr>
<th>Day</th>
<th>Group</th>
<th>Time</th>
<th>Room</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saturday</td>
<td>APSA Board of Governors Annual Meeting</td>
<td>8 am – 2 pm</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td></td>
<td>Association of Pediatric Surgery Training Program Directors</td>
<td>2 – 9 pm</td>
<td>Desert Salons 9-11</td>
</tr>
<tr>
<td></td>
<td>Publications Committee</td>
<td>6:30 – 10 pm</td>
<td>Director's Suite 1</td>
</tr>
<tr>
<td>Sunday</td>
<td>Program Committee</td>
<td>6 – 7 am</td>
<td>Desert Salon 1</td>
</tr>
<tr>
<td></td>
<td>New Technology Committee</td>
<td>7 – 8 am</td>
<td>Desert Salon 1</td>
</tr>
<tr>
<td></td>
<td>Quality &amp; Safety Committee</td>
<td>6 – 7 am</td>
<td>Desert Salon 2</td>
</tr>
<tr>
<td></td>
<td>Pediatric NSQIP Champions</td>
<td>7 – 8 am</td>
<td>Desert Salon 2</td>
</tr>
<tr>
<td></td>
<td>Education/PSSAP/CME Committees</td>
<td>6 – 8 am</td>
<td>Desert Salon 3</td>
</tr>
<tr>
<td></td>
<td>Trauma Committee</td>
<td>6 – 7 am</td>
<td>Desert Salon 4</td>
</tr>
<tr>
<td></td>
<td>Pediatric Trauma Society/APSNA/STN</td>
<td>7 – 8 am</td>
<td>Desert Salon 4</td>
</tr>
<tr>
<td></td>
<td>Workforce Committee</td>
<td>6 – 7 am</td>
<td>Director's Suite 1</td>
</tr>
<tr>
<td></td>
<td>Practice Committee</td>
<td>7 – 8 am</td>
<td>Director's Suite 1</td>
</tr>
<tr>
<td></td>
<td>Companions’ Meeting</td>
<td>10 – 11:30 am</td>
<td>Director's Suite 1</td>
</tr>
<tr>
<td></td>
<td>Finance Committee</td>
<td>6 – 7 am</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td></td>
<td>Childhood Obesity Committee</td>
<td>7 – 8 am</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td></td>
<td>SICHA Update Meeting</td>
<td>6 – 8 am</td>
<td>Director's Suite 4</td>
</tr>
<tr>
<td></td>
<td>International Relations Committee</td>
<td>6 – 8 am</td>
<td>Chairman's Boardroom</td>
</tr>
<tr>
<td></td>
<td>Fetal Diagnosis &amp; Treatment Committee</td>
<td>7 – 8 am</td>
<td>President’s Boardroom</td>
</tr>
<tr>
<td>Monday</td>
<td>Informatics &amp; Telemedicine Committee</td>
<td>6:30 – 7:30 am</td>
<td>Desert Salon 1</td>
</tr>
<tr>
<td></td>
<td>Chest Deformities Center</td>
<td>1 – 3 pm</td>
<td>Desert Salon 1</td>
</tr>
<tr>
<td></td>
<td>ACS Advisory Council for Pediatric Surgery</td>
<td>4 – 5:30 pm</td>
<td>Desert Salon 1</td>
</tr>
<tr>
<td></td>
<td>APSA Foundation Board Meeting</td>
<td>6:15 – 7:30 am</td>
<td>Desert Salon 2</td>
</tr>
<tr>
<td></td>
<td>Pectus Multicenter Study</td>
<td>6:30 – 7:30 am</td>
<td>Desert Salon 3</td>
</tr>
<tr>
<td></td>
<td>BCM Fellowship Reunion</td>
<td>4:00 – 6:00 pm</td>
<td>Desert Salon 3</td>
</tr>
<tr>
<td></td>
<td>DHREAMS Annual NIH Meeting</td>
<td>6:30 – 7:30 am</td>
<td>Desert Salon 4</td>
</tr>
<tr>
<td></td>
<td>Children’s Steering Committee</td>
<td>6:30 – 7:30 am</td>
<td>Director's Suite 1</td>
</tr>
<tr>
<td></td>
<td>Outcomes and Clinical Trials Committee</td>
<td>6:30 – 7:30 am</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td></td>
<td>Family &amp; Community Relations</td>
<td>6:30 – 7:30 am</td>
<td>Director's Suite 4</td>
</tr>
<tr>
<td></td>
<td>OCHSIC</td>
<td>6 – 8 am</td>
<td>Chairman’s Boardroom</td>
</tr>
<tr>
<td></td>
<td>Membership &amp; Credentials Committee</td>
<td>6:30 – 7:30 am</td>
<td>President’s Boardroom</td>
</tr>
<tr>
<td>Tuesday</td>
<td>Neonatal Research Network</td>
<td>4 – 6 pm</td>
<td>Desert Salon 1</td>
</tr>
<tr>
<td></td>
<td>Surgical Critical Care</td>
<td>3:30 – 5 pm</td>
<td>Desert Salon 2</td>
</tr>
<tr>
<td>Wednesday</td>
<td>Cancer Committee</td>
<td>6:30 – 7:30 am</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td></td>
<td>Ethics &amp; Advocacy Committee</td>
<td>6 – 7:30 am</td>
<td>Director's Suite 4</td>
</tr>
</tbody>
</table>
## ANCILLARY MEETINGS—BY GROUP

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<td>8 am – 2 pm</td>
<td>Director's Suite 3</td>
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<tr>
<td>APSA Foundation Board Meeting</td>
<td>Monday</td>
<td>6:15 – 7:30 am</td>
<td>Desert Salon 2</td>
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<td>Association of Pediatric Surgery Training Program Directors</td>
<td>Saturday</td>
<td>2 – 9 pm</td>
<td>Desert Salon 9-11</td>
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<td>Monday</td>
<td>4 – 6 pm</td>
<td>Desert Salon 3</td>
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<td>Cancer</td>
<td>Wednesday</td>
<td>6:30 – 7:30 am</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td>Chest Deformities Center</td>
<td>Monday</td>
<td>1 – 3 pm</td>
<td>Desert Salon 1</td>
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<tr>
<td>Childhood Obesity</td>
<td>Sunday</td>
<td>7 – 8 am</td>
<td>Director's Suite 3</td>
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<tr>
<td>Children's Steering Committee</td>
<td>Monday</td>
<td>6:30 – 7:30 am</td>
<td>Director's Suite 3</td>
</tr>
<tr>
<td>CME, Education and PSSAP Committees</td>
<td>Sunday</td>
<td>6 – 8 am</td>
<td>Desert Salon 3</td>
</tr>
<tr>
<td>Companion Meeting</td>
<td>Sunday</td>
<td>10 – 11:30 am</td>
<td>Director's Suite 1</td>
</tr>
<tr>
<td>DHREAMS Annual NIH Meeting</td>
<td>Monday</td>
<td>6:30 – 7:30 am</td>
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<td>7 – 8 am</td>
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<td>Monday</td>
<td>6:00 – 8:00 am</td>
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<tr>
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<td>Monday</td>
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<td>Sunday</td>
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<tr>
<td>Program</td>
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<td>6 – 7 am</td>
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<tr>
<td>PSSAP, CME and Education Committees</td>
<td>Sunday</td>
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<td>Sunday</td>
<td>6 – 7 am</td>
<td>Desert Salon 2</td>
</tr>
<tr>
<td>Trauma</td>
<td>Sunday</td>
<td>6 – 7 am</td>
<td>Desert Salon 4</td>
</tr>
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<td>Workforce</td>
<td>Sunday</td>
<td>6 – 7 am</td>
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</tr>
</tbody>
</table>
EDUCATIONAL OVERVIEW

The APSA Annual Meeting is designed to provide comprehensive continuing education in the field of pediatric surgery. APSA strives to bring together the world’s leading pediatric surgery authorities to present and discuss the most recent clinical and research efforts. This meeting covers the breadth of pediatric surgery and is intended to acquaint attendees with the latest research findings, clinical discoveries and trends that influence the day-to-day practice of pediatric surgery. Specific sessions relating to educating members on new developments in medical technology have been added to supplement the traditional sessions on clinical practice and basic science research chosen by the Program and Education Committees. The scientific sessions consist of basic research and practical clinical presentations. The poster sessions are intended to provide young investigators an opportunity to share preliminary clinical research, basic science work and novel ideas.

The meeting will begin with the Education Day program on Sunday, May 22, with the APSA/APSNA joint symposium on intestinal failure. Afternoon sessions include surgical care of the obese child and basic science and fetal therapy. Meeting attendees will also view and discuss video and selected poster presentations. In addition to five scientific sessions with abstract presentations, a cancer session, Case Debates and Controversies, this year’s meeting also debuts the Innovation Session. Attendees will view 7 abstracts in the innovation arena of pediatric surgical care.

Learning Objectives

At the conclusion of the meeting, attendees will be able to:
• Identify the components of clinical research that support improvements in clinical practice and how those concepts are applied to specific conditions
• Analyze the general concepts of basic science research efforts impacting pathologic conditions treated by pediatric surgeons
• Apply new knowledge regarding the development of new and emerging technologies that impact the field of pediatric surgery

Accreditation Statement

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

APSA designates this educational activity for a maximum of 21.5 AMA PRA Category 1 Credit(s)™. Physicians should only claim credit commensurate with the extent of their participation in the activity.
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Policy on Faculty Disclosure

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Disclosures

Disclosure forms were provided to and signed by all APSA 2010-2011 committee members.

Committees Disclosures

Edward M. Barksdale Consultant/Speakers Bureau: Nestle.

Christopher K. Breuer Grant/Research Support: Gunze Ltd, Pall Corp, ATRM. Other Financial/Material Interest: IP Yale Univ., Pall Corporation.

Peter W. Dillon Salary: Royalty - Syntheses; Consultant: Syntheses

Gerald M. Haase Ownership Interest (i.e., stockholder): Premier Micronutrient Corporation

Mary L. Hilfiker Consultant: Johnson & Johnson


Thomas H. Inge Grant/Research Support: Ethicon Endosurgery
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<th>Name</th>
<th>Ownership Interest (i.e., stockholder):</th>
<th>Role</th>
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<tr>
<td>Thomas M. Krummel</td>
<td>Visible Production, PEAK Surgical, WingTec, Miret Surgical</td>
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<tr>
<td>Don K. Nakayama</td>
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<td>Speakers Bureau: Pfizer Corporation</td>
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<tr>
<td>J. Duncan Phillips</td>
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<td>Consultant: Kimberly-clark Global Sales; Speakers Bureau: Fresenius USA Manufacturing</td>
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<td>Todd A. Ponsky</td>
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<td>Speakers Bureau: Styker, Storz, Covidien</td>
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<tr>
<td>Mark Puder</td>
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<td>Grant/Research Support: License agreement between CHB and Fresenius Kubi. Consultant: Martek. Patent for Omegaven has been submitted by Children’s Hospital Boston.</td>
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<td>Steven S. Rothenberg</td>
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<td>Consultant: Storz, Covidien</td>
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<tr>
<td>Daniel A. Saltzman</td>
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<tr>
<td>Mary C. Santos</td>
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<td>Ownership Interest (i.e., stockholder): Covidien</td>
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<tr>
<td>David L. Sigalet</td>
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<td>Grant/Research Support: Ferring Corporation; Consultant: Nycomed Corporation</td>
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<tr>
<td>John R. Wesley</td>
<td></td>
<td>Consultant: Excelsior Medical Corporation (pre-filled, pre-sterilized, unit dose IV syringe devices)</td>
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<tr>
<td>Jeffrey L. Zitsman</td>
<td></td>
<td>Allergan system: LAP band.</td>
</tr>
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Disclosures
Disclosure forms were provided to and signed by all invited speakers.

Invited Speaker Disclosures
Allen F. Browne Consultant: Allergan Corporation

Disclosures
Disclosures were collected from all abstract authors at the time of abstract submissions.

Abstract Author Disclosures
V1 Steven S. Rothenberg Consulting - StorzCovidien
V7 J. Geiger Intellectual Property; University of Michigan and Storz have a licensing agreement as James Geiger is the inventor.
17 J.B. Zwischenberger Consulting - Ikaria, Inc.. Grant/Research Support; NIH; Ikaria, Inc.. Royalty; Avalon
Journal of Pediatric Surgery Lecture

Professor Lewis Spitz, PhD, MD (Hon)
“The History of Paediatric Surgery in the United Kingdom and the National Health Service”

Graduated in medicine at the University of Pretoria and specialized in general surgery at the University of the Witwatersrand, South Africa. Trained in paediatric surgery in Liverpool with P.P. Rickham and at Great Ormond Street Hospital, London with H.H Nixon. Consultant Paediatric Surgeon at Baragwanath Hospital and Transvaal Memorial Hospital, Johannesburg; The Children’s Hospital, Sheffield 1974 - 1979; Nuffield Professor of Paediatric Surgery, Institute of Child Health, University College, London and Consultant Paediatric surgeon, Great Ormond Street Hospital, London 1979-2004.

President of the British Association of Paediatric Surgeons 1996-8, Chairman Specialist Advisory Committee Royal Colleges of Surgeons 1994-6, President, Paediatric Section, Royal Society of Medicine 2007-8.

Degrees: MB ChB, Ph.D, FRCS (Edin), FRCS(Eng), FRCS(Irel)(Hon), FAAP(Hon), FRCPCH(Hon), FCS(SA)(Hon), MD (Hon- Sheffield, Witwatersrand).


Publications:
Peer review articles – 407
Invited papers – 23
Chapters – 85
Books – 9 including:
Great Ormond Street Handbook of Paediatrics and Child Health

Learning Objectives:
• Provide an insite into paediatric surgery in the United Kingdom
• Show how the National Health Service has affected the development of paediatric surgery
• Illustrate how training in paediatric surgery has changed
Robert E. Gross Lecture

Judson G. Randolph, MD

“Notes on the Early Development of Pediatric Surgery in the United States”

Raised in Nashville, Tennessee, Dr. Randolph is a graduate from Vanderbilt University and Vanderbilt Medical School. He trained in general surgery at the MGH in Boston, and in Pediatric Surgery at Boston Children’s Hospital. In 1963 he accepted the newly created position of Surgeon-in-Chief at the Children’s Hospital in Washington, DC, a post he held until retirement in 1992. Dr. Randolph served as Chairman of the Surgical Section of the American Academy of Pediatrics and was elected President of the American Pediatric Surgical Association in 1984. In 1998 he was chosen the Distinguished Graduate of Vanderbilt Medical School, and in 1999 was awarded the Ladd Gold Medial of the American Academy of Pediatrics. He is very proud of the many accomplishments of the graduates of the training program in Washington, and of the 5 children, which he and his wife, Comfort, raised.

Learning Objectives:

• The involvement of William E. Ladd on the beginnings of pediatric surgery in the United States
• The work and development of Robert E. Gross at the Boston Children’s Hospital
• Orvar Swenson, H.W. Clatworthy and others who took the specialty of pediatric surgery forward
Anthony Atala, MD, is the Director of the Wake Forest Institute for Regenerative Medicine, and the W.H. Boyce Professor and Chair of the Department of Urology at Wake Forest University. Dr. Atala is a practicing surgeon and a researcher in the area of regenerative medicine. His current work focuses on growing new human cells, tissues and organs.

Dr. Atala works with several journals and serves in various roles, including Editor-in-Chief of Current Stem Cell Research and Therapy, and Therapeutic Advances in Urology; as Associate Editor of Tissue Engineering and Regenerative Medicine, The Journal of Rejuvenation Research, Nanotechnology in Engineering and Medicine, Gene Therapy and Regulation, and Current Reviews in Urology; and as Executive Board Member or Section Editor of the journal Tissue Engineering and International Journal of Artificial Organs.

Dr. Atala is a recipient of many awards, including the U.S. Congress funded Christopher Columbus Foundation Award, bestowed on a living American who is currently working on a discovery that will significantly affect society, and the Gold Cystoscope and Samuel Gross Awards for advances in his field. Dr. Atala was named by Scientific American as a Medical Treatments Leader of the Year for his contributions to the fields of cell, tissue and organ regeneration. Dr. Atala's work was listed as Time Magazine’s top 10 medical breakthroughs of the year, and as Discover Magazine’s Number 1 Top Science Story of the Year in the field of medicine in 2007.

Dr. Atala has led or served several national professional and government committees, including the National Institutes of Health working group on Cells and Developmental Biology, and the National Institutes of Health Bioengineering Consortium. He is currently a NIH “Quantum Grant” awardee. He is the editor of nine books, has published more than 300 journal articles and has applied for or received over 200 national and international patents.

Learning Objectives:
- Describe the field of tissue engineering and regenerative medicine
- Appraise the current state of stem cell research
- Describe some of the current research and clinical applications
International Guest Lecture

Professor Takeshi Miyano, MD
“A Brief History of Pediatric Surgery and Healthcare Delivery Systems in Japan”

Professor Takeshi Miyano is currently the managing director of Juntendo University Nerima Hospital and Professor Emeritus of Juntendo University. He trained at the Juntendo School of Medicine, as well as the University of Liverpool, Royal Liverpool Children’s Hospital (United Kingdom).

He has served in many roles at Juntendo University, including head of pediatric surgery department, vice director and managing director of the university hospital.

Professor Miyano has served as president of the Japanese Society of Pediatric Surgeons and the Japanese Society for Small Bowel Transplantation.

Professor Miyano has also led international pediatric surgical societies, having served as president of the Asian Association of Pediatric Surgeons, International Pediatric Endosurgery Group and the World Federation of Associations of Pediatric Surgeons.

Learning Objectives:
• Summarize the history of pediatric surgery in Japan
• Summarize the history of the health care delivery system of Japan
• Reflect on the difference between the delivery of pediatric surgery in Japan and their own experience
APSA 42ND ANNUAL MEETING
PROGRAM IN DETAIL
### Saturday, May 21

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>8:00 a.m. – 2:00 p.m.</td>
<td>APSA Board of Governors Meeting</td>
<td>Director’s Suite 3</td>
</tr>
<tr>
<td>2:00 – 8:00 p.m.</td>
<td>Pediatric Surgery Training Program Directors Meeting</td>
<td>Desert Salons 9-11</td>
</tr>
<tr>
<td>3:00 – 6:00 p.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>3:00 – 6:00 p.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
</tr>
<tr>
<td>3:00 – 6:00 p.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>6:00 – 10:00 p.m.</td>
<td>APSA Board of Governors Dinner</td>
<td>Mikado Restaurant</td>
</tr>
<tr>
<td>6:30 – 10:00 p.m.</td>
<td>Publications Committee Meeting</td>
<td>Director’s Suite 1</td>
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### Sunday, May 22

<table>
<thead>
<tr>
<th>Time</th>
<th>Event</th>
<th>Location</th>
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<tbody>
<tr>
<td>6:00 – 8:00 a.m.</td>
<td>Committee Meetings</td>
<td>See page 69 for ancillary meeting schedule</td>
</tr>
<tr>
<td>7:00 a.m. – 5:00 p.m.</td>
<td>Registration Open</td>
<td>Desert West Foyer</td>
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<tr>
<td>7:00 a.m. – 5:00 p.m.</td>
<td>Speaker Ready Room Open</td>
<td>Desert Salons 5-6</td>
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<td>7:00 a.m. – 5:00 p.m.</td>
<td>Internet Café Open</td>
<td>Desert West Foyer</td>
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<tr>
<td>7:00 – 8:00 a.m.</td>
<td>Continental Breakfast</td>
<td>Desert West Foyer</td>
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</table>
| 7:45 – 8:00 a.m.  | President’s Welcome
Marshall Z. Schwartz, MD                                          | Desert Salons 7-8         |
| 8:00 – 11:00 a.m. | Education Session I
APSA/APSNA Symposium: Intestinal Failure                             | Desert Salons 7-8         |

**Moderator:**
Daniel H. Teitelbaum, MD

**Catheter-Related Sepsis**

Eunice Huang, MD

**Educational Objectives:**
Attendees will be able to:
- Discuss pathophysiology and treatment and prophylaxis of infections
- Identify recently published literature on strategies for the prevention of central catheter infection during central catheter insertion
- Discuss recently published literature addressing strategies for central catheter care for the prevention of central catheter infection
- Compare strategies used by some institutions to minimize risk of recurrent central line infections in their intestinal failure patients
Enteral Access  
*Marcus Jarboe, MD; Lynne D. Farber, RN, MSN, CPNP-PC, AC*

**Educational Objectives:**  
Attendees will be able to:  
- Describe non-conventional approaches to placement of enteric feeding tubes  
- Compare how imaging, endoscopy and other modalities are useful in placing non-conventional and/or difficult enteral feeding tubes  
- Identify feeding strategies (drip vs. bolus), and how best to initiate specific types of feedings (human milk, semi-elemental vs. elemental) for children with gastroschisis and malabsorption issue  
- Discuss strategies used by some institutions to troubleshoot gastrostomy issues such as leaking and granulation tissue.

Hepatoprotective Therapies for TPN-Associated Cholestatis  
*Margaret Helin, MS, RN, CPNP; Robert Cowles, MD*

**Educational Objectives:**  
Attendees will be able to:  
- Discuss pathophysiology and treatment with enteral fish oil  
- Review the literature on modalities to prevent and/or treat TPN-cholestasis  
- Discuss status of parenteral fish oils (Omegaven)

Transitioning the Child to Homecare  
*Neil Ead, RN*

**Educational Objectives:**  
Attendees will be able to:  
- Discuss the obstacles for setting up homecare  
- Discuss creation of a safety net and care plan for continuity of care once the child is discharged

Intestinal Lengthening  
*Tom Jaksic, MD*

**Educational Objectives:**  
Attendees will be able to:  
- Identify the alternatives, indications and contraindications to bowel lengthening surgery  
- Describe the technique and mechanism of action of the Serial Transverse Enteroplasty (STEP)  
- State the mortality and likelihood for attaining full enteral nutrition associated with the STEP operation (based upon the data of the International STEP Registry)

NEC Child with Extreme Loss of Intestine/Ethical Care  
*Abigail E. Martin, MD*

**Educational Objective:**  
Attendees will be able to:  
- Discuss when and if it is appropriate to refer a patient for an intestinal transplant
Dysmotility
Steven Teich, MD

Educational Objectives:
Attendees will be able to:
• Describe the etiology and nonsurgical therapies for treatment of gastroparesis in children and adolescents
• Explain the workup for patients with gastroparesis
• Define the principles of gastric stimulation for gastroparesis

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<tr>
<th>Time</th>
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<tr>
<td>10:00 – 11:30 a.m.</td>
<td>Companion Meeting</td>
<td>Director’s Suite 1</td>
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<tr>
<td>10:45 – 11:00 a.m.</td>
<td>APSNA, Lynne D. Farber, President</td>
<td>Desert Salons 7-8</td>
</tr>
<tr>
<td>11:00 – 11:30 a.m.</td>
<td>Refreshment Break</td>
<td>Desert West Foyer</td>
</tr>
<tr>
<td>11:30 a.m. – 12:30 p.m.</td>
<td>Journal of Pediatric Surgery Lecture:</td>
<td>Desert Salons 7-8</td>
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<td>Professor Lewis Spitz</td>
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<td>The History of Paediatric Surgery in the United Kingdom and the National Healthcare Service</td>
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<tr>
<td>12:30 – 12:45 p.m.</td>
<td>Box Lunch Pick-Up</td>
<td>Desert Salons Foyer</td>
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<td>12:45 – 1:45 p.m.</td>
<td>Video Session with Lunch</td>
<td>Desert Salons 7-8</td>
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Moderators:
Daniel J. Ostlie, MD; Ai-Xuan Holterman, MD

Educational Objectives:
At the completion of this session the learner will be able to describe:
• Minimally invasive approaches to duodenal atresia and liver masses
• Single site surgery for spherocytosis
• Immediate post-natal treatment of sacrococcygeal teratoma
• Thoracoscopic treatment for myasthenia gravis

V1 LAPAROSCOPIC REPAIR OF A DUODENAL ATRESIA AND LADD’S PROCEDURE IN A NEONATE WITH MALROTATION
Steven S. Rothenberg, MD
The Rocky Mountain Hospital For Children, Denver, CO, USA.

V2 LAPAROSCOPIC NEPHRECTOMY FOR WILMS TUMOR IN A ONE YEAR OLD GIRL
Guido Seitz, MD; Steven W. Warmann, MD, Martin Ebinger, MD, Falko Fend, MD, Jörg Fuchs, MD.
1University Children’s Hospital, Tuebingen, Germany, 2University Hospital, Department of Pathology, Tuebingen, Germany.

V3 SINGLE SITE UMILIBICAL LAPAROSCOPIC SPLENECTOMY AND CHOLECYSTECTOMY IN A PATIENT WITH HEREDITARY SPHEROCYTOSIS AND CHOLELITHIASIS
Patricia A. Valusek, MD; George W. Holcomb III, MD, MBA.
1Childrens Minneapolis, Minneapolis, MN, USA, 2Children’s Mercy Hospital, Kansas City, MO, USA.
### V4  THORACOSCOPIC THYMECTOMY FOR MYASTHENIA GRAVIS
Jill Zalieckas, MD, Charles J. Stolar, Steven S. Rothenberg.
1Columbia University, New York, NY, USA, 2Columbia University, New York, NY, USA.

### V5  LAPAROSCOPIC LEFT LATERAL SEGMENTECTOMY IN A CHILD
Karen Diefenbach, MD, Milissa McKee, MD, MPH.
Yale University School of Medicine, New Haven, CT, USA.

### V6  EXIT TO RESECTION OF A FETAL SACROCOCCYGEAL TERATOMA
Payam Saadai, MD, Diana L. Farmer, MD, Tippi MacKenzie, MD, Danny Wu, MD, Ruth Goldstein, MD, Charles Cauldwell, MD, Thomas Shimotake, MD, Michael Harrison, MD, Hanmin Lee, MD, Doug Miniati, MD.
University of California San Francisco, San Francisco, CA, USA.

### V7  INNOVATIVE INSTRUMENT FOR LAPAROSCOPIC PYLOROMYOTOMY
Marcus Jarboe, MD, James Geiger, MD.
University of Michigan, Ann Arbor, MI, USA.

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**2:00 – 4:00 p.m.**  
**Concurrent Education Sessions**  
**Desert Salons 7-8**  
**Education Session II:**  
**Quality Improvement and Reducing Variation**

**Moderator:**  
Keith E. Georgeson, MD

**Patient Safety and Surgery: More than Just Work Hour Restrictions**  
Shawn J. Rangel, MD

**Educational Objectives:**  
Attendees will be able to:  
- Describe the problems of patient safety in surgery  
- Describe the role of Time Out, how it reduces injury  
- Describe the role of checklists and how their use reduces unwanted VIC

**Variation in Care: Why is it a Problem? What does it Really Mean to a Surgeon?**  
Kurt F. Heiss, MD

**Educational Objectives:**  
Attendees will be able to:  
- Identify lessons learned from adult NSQIP experience  
- Be able to explain how Pediatric NSQIP will differ from adult program  
- Describe what NSQIP will contribute to improving outcomes in pediatric surgery  
- Describe how VIC influences quality through underuse, overuse and misuse  
- Describe examples from surgery where reducing VIC improved quality  
- Identify examples of how reducing unwanted VIC will improve quality in pediatric surgery
Team Work: The Next Horizon on Patient Safety
Thomas V. Whalen, MD

Educational Objectives:
Attendees will be able to:
• Describe how development of team functions can improve performance in the OR
• Describe how the development of crew resource management has improved safety in aviation
• Describe the limitations of checklists and time outs in patient safety
• Describe how leveling of hierarchy and clarification of roles can improve communication and safety in the OR

After NSQIP: The Role of Process Improvement, When the Real Work Begins
Frederick C. Ryckman, MD

Educational Objectives:
Attendees will be able to:
• Recognize that tools like NSQIP will point out system flaws in care that couldn’t be seen before
• Describe how process improvement will be necessary to reduce unwanted variation in care and to use the information from NSQIP to improve care
• Cite examples of how this tool has helped reduce VIC and improve safety

2:00 – 4:00 p.m. Education Session III: Springs Salon F
Surgical Care of the Obese Child

Moderator:
Thomas H. Inge, MD

Patient Related Issues
Jeffrey L. Zitsman, MD

Educational Objectives:
Attendees will be able to:
• Describe and evaluate the following comorbid conditions related to obese patients: cardiac/blood pressure, endocrine, PCOS, pseudotumor, hepatic

Facility Related Issues
Mark P. Michalsky, MD

Educational Objectives:
Attendees will be able to:
• Describe the facility limitations to caring for obese patients: weight limits, transportation, sensitivity

How the Pathophysiology of Obesity May Impact Care
Kirk W. Reichard, MD

Educational Objectives:
Attendees will be able to:
• Describe how obesity affects: anesthesia, drug dosing, trauma concerns, DVT risk
Clinical Vignettes of Common Conditions
Allen F. Browne, MD

Educational Objectives:
Attendees will be able to:
- Identify issues related to the care of obese patients with abdominal pain related to appendicitis and ovarian torsion, cholecystitis, trauma, soft tissue infection

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<td>4:00 – 5:00 p.m.</td>
<td>Wine and Cheese Reception</td>
<td>Desert West Foyer</td>
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<tr>
<td>4:15 – 6:00 p.m.</td>
<td>Concurrent Poster Sessions</td>
<td>Desert Salons 9-11</td>
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</table>

**Poster Session I:**
Clinical

**Moderators:**
David J. Schmeling, MD; Heung Bae Kim, MD

Educational Objectives:
At the completion of this session, the participants will be able to:
- Describe the use of registries to assess surgical complications
- Cite the workup and outcome for thyroid nodules in pediatric patients
- Review outcome factors for infants with gastroschisis
- Describe prognostic factors for CDH and Melanoma

**P1**
AHRQ PEDIATRIC INDICATORS AS A QUALITY METRIC FOR SURGERY IN CHILDREN: DO THEY PREDICT ADVERSE OUTCOMES?
Daniel S. Rhee, MD, MPH, Yiyi Zhang, MHS, Dominic Papandria, MD, Gezzer Ortega, MD, Fizan Abdullah, MD, PhD.
Johns Hopkins, Baltimore, MD, USA.

**P2**
INCIDENCE AND NATURE OF POSTOPERATIVE COMPLICATIONS IN PATIENTS ADMITTED TO THE NEONATAL INTENSIVE CARE UNIT
Alexander P. Stoffan, MD, Anne Hansen, MD, J W. Sparks, MD, Elizabeth K. Norton, RN, Keri Kucharski, RN, BSN, Jessica Baxter, Shawn J. Rangel, MD, MSCE.
Children’s Hospital Boston, Boston, MA, USA.

**P3**
LESSONS FROM A LIVER HEMANGIOMA REGISTRY: SUBTYPE CLASSIFICATION
Ann M. Kulungowski¹, Ahmad I. Alomari, MD², Aditya Chawla, BA¹, Emily R. Christison-Lagay, MD¹, Steven J. Fishman, MD¹.
¹Children’s Hospital Boston, Vascular Anomalies Center, Department of Surgery, Harvard Medical School, Boston, MA, USA, ²Children’s Hospital Boston, Vascular Anomalies Center, Department of Radiology, Harvard Medical School, Boston, MA, USA.

**P4**
OPTO-ELECTRONIC PLETHYSMOGRAPHY (OEP) DEMONSTRATES ABRUPTION OF REGIONAL CHEST WALL MOTION DYSFUNCTION IN PECTUS EXCAVATUM PATIENTS FOLLOWING NUSS REPAIR
Richard E. Redlinger, Jr., MD¹, Ashley Wootton, BS², Robert E. Kelly, Jr., MD², Donald Nuss, MB, ChB³, Michael J. Goretsky, MD², M. Ann Kuhn, MD², Robert J. Obermeyer, MD².
¹Eastern Virginia Medical School, Norfolk, VA, USA, ²Children’s Hospital of the King’s Daughters, Norfolk, VA, USA.
P5  CENTRAL LIVER RESECTION, A FEASIBLE SUBSTITUTE FOR TRANSPLANTATION IN CASES OF HEPATOBLASTOMA
Claudia N. Emami, MD MPH, Michael Petrosyan, MD, Marcio Malogolowkin, MD, James Stein, MD FACS FAAP.
Children’s Hospital Los Angeles, Los Angeles, CA, USA.

P6  CONGENITAL LUNG ANOMALIES: CAN WE POSTPONE RESECTION?
Nadja C. Colon, MD, Cameron Schlegel, BS, Dai H. Chung, MD, Gretchen Jackson, MD, PhD.
Vanderbilt Children’s Hospital, Nashville, TN, USA.

P7  IS DAILY DILATATION NECESSARY AFTER SURGERY FOR HIRSCHSPRUNG DISEASE AND ANORECTAL MALFORMATIONS?
Sara Temple, MD, Jacob C. Langer, MD.
Hospital for Sick Children, Toronto, ON, Canada.

P8  CIRCULATING THYROTROPIN RECEPTOR MRNA FOR EVALUATION OF THYROID NODULES AND SURVEILLANCE OF THYROID CANCER
Jesse Gutnick, MD, Oliver S.Soldes, MD, Manjula Gupta, PhD, Mira Milas, MD.
Cleveland Clinic Foundation, Cleveland, OH, USA.

P9  PROGRESSION-FREE SURVIVAL IN WELLDIFFERENTIATED THYROID CANCER IN PEDIATRIC AND ADOLESCENT PATIENTS
Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

P10  PREDICTORS OF SURVIVAL IN CHILDHOOD AND ADOLESCENT CUTANEOUS MELANOMA
Memorial Sloan-Kettering Cancer Center, New York, NY, USA.

P11  AGE PREDICTS MONOTHERAPY IMMUNOSUPPRESSION IN PEDIATRIC LIVER TRANSPLANT RECIPIENTS AT ONE YEAR POST TRANSPLANT
David W. Rittenhouse, MD1, Leslie J. Krueger, PhD2, Jobayer Hossain, PhD3, Louise Flynn, MSN3, Dana S. Mannino, APN3, Shylah Haldeman, BSN3, Stephen P. Dunn, MD3.
1Thomas Jefferson University Hospital, Philadelphia, PA, USA, 2TARC Inc., Freehold, NJ, USA, 3Alfred I. duPont Hospital for Children, Wilmington, DE, USA.

P12  THE ROLE OF SURGICAL MANAGEMENT FOR CHRONIC ITP: A COST ANALYSIS OF SPLENECTOMY VERSUS MEDICAL MANAGEMENT
Natalie R. Gwilliam, BA1, David A. Lazar, MD2, Mary L. Brandt, MD2, Donald H. Mahoney, MD3, David E. Wesson, MD2, Timothy C. Lee, MD2.
1Baylor College of Medicine, Houston, TX, USA, 2Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX, USA, 3Department of Pediatrics, Baylor College of Medicine, Houston, TX, USA.
P13  LONG-TERM NEURODEVELOPMENTAL OUTCOMES IN CHILDREN BORN WITH GASTROCHISIS
Adam S. Gorra, MD1, Holly J. Roberts, PhD2, Barbara J. Jackson, PhD2, Kenneth S. Azarow, MD1, Howard Needleman, MD1, Robert A. Cusick, MD1.
1University of Nebraska/Children’s Hospital and Medical Center, Omaha, NE, USA, 2University of Nebraska - Department of Education and Child Development, Munroe-Meyer Institute, Omaha, NE, USA.

P14  TAKING THE GOOD WITH THE BAD: CHANGES IN GASTROCHISIS MANAGEMENT OVER THE LAST DECADE ARE ASSOCIATED WITH BOTH BENEFITS AND SHORTCOMINGS
Brent R. Weil, MD, Charles M. Leys, MD, Frederick J. Rescorla, MD.
Riley Children’s Hospital, Indianapolis, IN, USA.

P15  COMPLICATIONS OF LAPAROSCOPIC PANCREATECTOMY FOR CONGENITAL HYPERINSULINISM OF INFANCY
Shireen A. Nah, MBBS, MRCS, MS, Paolo De Coppi, MD, PhD, Dunia Ismail, Virpi V. Smith, PhD, FRCPath, CSci, FIBMS, Michael Ashworth, FRCPath, Khalid Hussain, MB.ChB, MRCP, MSc, MRCPCH, MD, Simon Eaton, PhD, Agostino Pierro, MD, FRCS, FAAP.
Great Ormond Street Hospital for Children & UCL Institute of Child Health, London, United Kingdom.

P16  THE CONGENITAL DIAPHRAGMATIC HERNIA COMPOSITE PROGNOSTIC INDEX (CDH-CPI) ACCURATELY PREDICTS SURVIVAL IN LEFT-SIDED CDH
Louis D. Le, MD, Foong Y. Lim, MD, Sundeep G. Keswani, MD, Mounira Habli, MD, Jason S. Frischer, MD, Beth E. Haberman, MD, Paul S. Kingma, MD, Timothy M. Crombleholme, MD.
Cincinnati Children’s Hospital, Cincinnati, OH, USA.

4:15 – 6:00 p.m.  Poster Session II:  Desert Salons 12-14
Basic Science and Fetal Surgery

Moderators:
Peter F. Nichol, MD; Ai-Xuan Holterman, MD

Educational Objectives:
Participants in this session will be able to:
• Describe progress in the understanding of the biology of various pediatric tumors
• Explain management of fetal anomalies and twin-twin transfusion
• Identify cytokine mediators in cell systems of fetal and pediatric disorders

P17  HEPATOCYTE GROWTH FACTOR AND OMEGA-3 ENRICHED FEEDS HAVE A SYNERGISTIC EFFECT ON MUCOSAL MASS IN AN ANIMAL MODEL OF INFLAMMATORY BOWEL DISEASE
Michael S. Katz, Keith A. Thatch, MD, Marshall Z. Schwartz, MD.
St. Christopher’s Hospital for Children, Philadelphia, PA, USA.
P18 TIE2 INHIBITION RESTRICTS VASCULAR BASEMENT MEMBRANE REMODELING
Ari Reichstein, MD1, Jeffrey W. Gander, MD1, Yan Jun Chang Chang2, Sonia L. Hernandez, PhD2, Jianzhong Huang, MD1, Darrell Yamashiro, MD, PhD1, Jessica Kandel, MD1.

1Morgan Stanley Children’s Hospital, Columbia University Medical Center, New York, NY, USA, 2Columbia University Medical Center, New York, NY, USA.

P19 THE PRO-INFLAMMATORY EFFECTS OF TOLL-LIKE RECEPTOR AGONISTS ARE COUNTERED BY INTERLEUKIN 10 PRODUCTION IN A DOSE-RESPONSE FASHION.
Ryan M. Walk, MD, Steven T. Elliott, MD, Jason A. Snyder, MD, Felix C. Blanco, MD, Stanislaw Vukmanovic, MD, Anthony D. Sandler, MD.

Children’s National Medical Center, Washington, DC, USA.

P20 INHIBITION OF FOCAL ADHESION KINASE IN HEPATOBLASTOMA CELLS LEADS TO DECREASED CELL SURVIVAL
Lauren A. Gillory, MD1, Jerry Stewart, Jr., BS1, Vita M. Golubovskaya, PhD2, Elizabeth A. Beierle, MD1.

1University of Alabama at Birmingham, Birmingham, AL, USA, 2Roswell Park Cancer Institute, Buffalo, NY, USA.

P21 POTENT ALLOGENEIC T-CELL RESPONSES TO MURINE NEUROBLASTOMA
Felix C. Blanco, MD, Ryan Walk, MD, Jason Snyder, MD, Stanislaw Vukmanovic, MD, Anthony D. Sandler, MD.

CNMC, Washington, DC, USA.

P22 HYPOXIA INDUCIBLE FACTOR-1α; AT THE INTERFACE BETWEEN DRUG RESISTANCE AND TUMOR INVASIVENESS IN NEUROBLASTOMA
Timothy B. Lautz, MD, Fei Chu, MD, PhD, Mary Beth Madonna, MD.

Children’s Memorial Hospital, Northwestern University Chicago, IL, USA.

P23 INHIBITION OF VASCULAR ENDOTHELIAL GROWTH FACTOR DIRECTS ABSORPTIVE LINEAGE DIFFERENTIATION OF THE INTESTINAL EPITHELIUM VIA NOTCH ACTIVATION
Jamil A. Matthews, MD, Frederic G. Sala, PhD, Allison L. Speer, MD, Erik R. Barthel, MD, Tracy C. Grikscheit, MD.

Saban Institute-Children’s Hospital Los Angeles, Los Angeles, CA, USA.

P24 NOVEL ZEBRAFISH MODEL REVEALS CRITICAL ROLE FOR MAPK IN LYMPHANGIOGENESIS
R. Dawn Fevurly, MD, Sean Hasso, PhD, Steven J. Fishman, MD, Joanne Chan, PhD.

Children’s Hospital Boston, Boston, MA, USA.
P25  AMNIOTIC FLUID STEM CELLS CAN FUNCTIONALLY DIFFERENTIATE ALONG SMOOTH MUSCLE LINEAGE - POTENTIAL FOR REGENERATIVE MEDICINE
Marco Ghionzoli1, Giulia Costanzi1, Steven W. Shaw1, Giorgia Totonelli1, Massimo Garrilobi1, Andrea Repele1, Stelios T. Andreadis2, Antonio Messineo3, Agostino Pierro1, Simon Eaton1, Paolo De Coppi1.
1UCL Institute of Child Health and Great Ormond Street Hospital for Children, London, United Kingdom, 2Department of Chemical and Biological Engineering, University at Buffalo, Amherst, New York, NY, USA, 3Dept. of Paediatric Surgery, University of Firenze, Firenze, Italy.

P26  EXPRESSION OF NONHOMOLOGOUS ENDJOINING PROTEINS IN NEURAL CREST CELLS AND NEUROBLASTOMA
Li Wang, PhD, Allyson Lieberman, BS, Valerie Castle, MD, Anthony Opipari, MD, PhD, Roland Kwok, PhD, Erika Newman, MD.
Mott Children’s Hospital The University of Michigan Medical School, Ann Arbor, MI, USA.

P27  NOVEL MECHANISMS OF THE ANTIINFLAMMATORY CYTOKINE IL-10 IN REGENERATIVE FETAL WOUND REPAIR
Louis D. Le, MD, Foong Y. Lim, MD, Helen N. Jones, PhD, Nabil Ghobril, MD, Mounira Habli, MD, Timothy M. Crombleholme, MD, Sundeep G. Keswani, MD.
Cincinnati Children’s Hospital, Cincinnati, OH, USA.

P28  TRACHEO-ESOPHAGEAL DISPLACEMENT INDEX (TEDI) AND PREDICTORS OF AIRWAY OBSTRUCTION FOR FETUSES WITH GIANT NECK MASSES
Darrell L. Cass, MD, Christopher I. Cassady, MD, David A. Lazar, MD, Kenneth J. Moise, MD, Anthony Johnson, DO, Timothy C. Lee, MD, Oluwinka O. Olutoye, MD, PhD.
Texas Children’s Fetal Center, Houston, TX, USA.

P29  LONGTERM MORBIDITY AFTER FETAL ENDOSCOPIC SURGERY FOR SEVERE TWIN-TO-TWIN TRANSFUSION SYNDROME
Benjamin Kowitt, Debra Watson-Smith, RN, Richard Tucker, PhD, Christopher S. Muratore, MD, Barbara M. O’Brien, MD, Betty Vohr, MD, Stephen R. Carr, MD, Francois I. Luks, MD, PhD.
Alpert Medical School of Brown University, Providence, RI, USA.

P30  FETAL ECHOCARDIOGRAPHY GUIDES INDICATIONS FOR SURGERY IN FETUSES WITH HYDROPS
Darrell L. Cass, MD, Oluwinka O. Olutoye, MD, PhD, Nancy Ayres, MD, Kenneth J. Moise, MD, Carolyn A. Altman, MD, Anthony Johnson, DO, Christopher I. Cassady, MD, David A. Lazar, MD, Timothy C. Lee, MD, Regina M. Lantin, MD.
Texas Children’s Fetal Center, Houston, TX, USA.

P31  METAGENOMIC ANALYSIS OF INTESTINAL BACTERIA FROM INFANTS WITH COMPLICATED NECROTIZING ENTEROCOLITIS
Valeriy Poroyko, PhD1, Michael Morowitz, MD2.
1University of Chicago Pritzker School of Medicine, Chicago, IL, USA, 2University of Pittsburgh School of Medicine, Pittsburgh, PA, USA.
P32  HEPATIC INFLAMMATION AND ALTERED HEPATOBILIARY TRANSPORTER EXPRESSION IN A MOUSE MODEL OF SMALL BOWEL BACTERIAL OVERGROWTH
Jaimie D. Nathan, MD, Bin Wang, MD, Lili Miles, MD, Joel E. Mortensen, PhD, Jorge A. Bezerra, MD.
Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA.

6:30–8:30 p.m.  Welcome Reception  Springs Pool and Grove

Monday, May 23
6:30 – 7:30 a.m.  Committee Meetings  See page 69 for ancillary meeting schedule
6:30 a.m. – 10:00 a.m.  Poster Set-up  Desert Salons 9-14
6:30 a.m. – 1:00 p.m.  Registration Open  Desert West Foyer
6:30 a.m. – 1:00 p.m.  Speaker Ready Room Open  Desert Salons 5-6
6:30 a.m. – 1:00 p.m.  Internet Café Open  Desert West Foyer
6:45 – 7:30 a.m.  Continental Breakfast  Springs Salons A-F
6:45 a.m. – 1:00 p.m.  Exhibits Open  Springs Salons A-F
7:30 – 9:00 a.m.  Scientific Session I  Desert Salons 7-8

Clinical Surgery

Moderators:
Peter F. Nichol, MD; Michael A. Helmrath, MD

Educational Objectives:
At the completion of this session, attendees will be able to:
• Describe opportunities for international surgical outreach programs
• Cite the need criteria for observation of premature infants after surgery
• Describe the impact of medical therapies on complications in surgery for ulcerative colitis
• Compare surgical approaches to long gap esophageal atresia

1  PEDIATRIC SURGERY IN HAITI: A ONE YEAR EXPERIENCE
Shahram Aarabi, MD, MPH1, David Mooney, MD, MPH2, Jason Smithers, MD2.
1University of Washington, Seattle, WA, USA, 2Children’s Hospital Boston, Boston, MA, USA.

2  A NOVEL SURGICAL RISK SCORE FOR CHILDREN
Fizan Abdullah, MD, PhD, Daniel S. Rhee, MD, MPH, Yiyi Zhang, MHS, Jessica Yang, BS, Jose Salazar, MD, Kristin Chrouser, MD MPH, Paul M. Colombani, MD, MBA, David C. Chang, PhD, MBA, MPH.
Johns Hopkins, Baltimore, MD, USA.

3  OVERNIGHT OBSERVATION FOR PREMATURITY
Carrie A. Laituri, MD, Carissa L. Garey, MD, Benjamin J. Pieters, MD, Peter Mestad, MD, Eric E. Weissand, MD, Shawn D. St. Peter, MD.
Children’s Mercy Hospital & Clinics, Kansas City, MO, USA.
4 PANCREATIC HEAD RESECTION AND ROUX-EN-Y PANCREATICOJEJUNOSTOMY FOR THE TREATMENT OF THE FOCAL FORM OF CONGENITAL HYPERINSULINISM.
Pablo Laje, MD, Susan A. Becker, Charles A. Stanley, MD, N. Scott Adzick, MD.
The Children’s Hospital of Philadelphia, Philadelphia, PA, USA.

5 ESOPHAGEAL GROWTH INDUCTION (FOKER PROCESS) FOR THE TREATMENT OF LONG GAP ESOPHAGEAL ATRESIA: INITIAL EXPERIENCE AT A SINGLE INSTITUTION
Russell W. Jennings, MD1, David Clendenin, MD1, Michael Manfredi, MD1, Bradley Linden, MD1, John Foker, MD, PhD2.
1Children’s Hospital, Boston, MA, USA, 2University of Minnesota, Minneapolis, MN, USA.

6 PYLOROMYOTOMY: RANDOMIZED CONTROL TRIAL OF LAPAROSCOPIC VERSUS OPEN TECHNIQUE WITH LONG-TERM FOLLOW-UP.
Sabina Siddiqui, MD, Alfred P. Kennedy, MD.
University of Tennessee, Knoxville, TN, USA.

7 PEDIATRIC CHRONIC ULCERATIVE COLITIS: DOES INFLIXIMAB INFLUENCE POST-IPAA COMPLICATIONS?
Raeelene Kennedy, MD, D. Dean Potter, MD, Christopher Moir, MD, Abdalla Zarroug, MD, William Faubion, MD, Jeanne Tung, MD.
Mayo Clinic, Rochester, MN, USA.

8 RESTORATIVE PROCTOCOLECTOMY WITHOUT DIVERTING ILEOSTOMY IN CHILDREN WITH ULCERATIVE COLITIS
Brian W. Gray, MD, James Geiger, MD, Ronald Hirschl, MD.
University of Michigan, Ann Arbor, MI, USA.

9 MESENTERICO-LEFT PORTAL VEIN BYPASS IS SUPERIOR TO PORTOSYSTEMIC SHUNT IN THE MANAGEMENT OF EXTRAHEPATIC PORTAL VEIN OBSTRUCTION
Timothy B. Lautz, MD, Joseph C. Melvin, BS, Lisa Keys, MSN, Riccardo A. Superina, MD.
Children’s Memorial Hospital, Northwestern University, Chicago, IL, USA.

10 MAGNETIC MINI-MOVER PROCEDURE FOR PECTUS EXCAVATUM III: SAFETY AND EFFICACY IN AN FDA SPONSORED CLINICAL TRIAL
Michael R. Harrison, MD, Kelly D. Gonzales, MD, Barbara J. Bratton, MSN, PNP, Darrell Christensen, CO, Patrick F. Curran, BS, Richard Fechter, BS, Shinjiro Hirose, MD, UCSF, San Francisco, CA, USA.

11 SINGLE INCISION LAPAROSCOPIC SURGERY: A RANDOMIZED CONTROL TRIAL IN ACUTE APPENDICITIS
Eduardo A. Perez, MD, Robert Barber, Anne C. Fischer, MD PhD.
Childrens Medical Center, Dallas, TX, USA.
12  **TRANSLUMINAL ENDOSCOPIC FUNDOPPLICATION FOR THE TREATMENT OF REFLUX DISEASE: COMPARISON TO LAPAROSCOPIC NISSEN**
Stephanie Chen, BS, Marcus D. Jarboe, MD, Robert D. Drongowski, MS, Daniel H. Teitelbaum, MD.
*University of Michigan, Ann Arbor, MI, USA.*

8:00 – 10:00 a.m.  Companion Hospitality Room Open for Registered Companions Only  
Sea Grille, 1st Floor

9:00 – 10:00 a.m.  **Robert E. Gross Lecture**  
Judson G. Randolph, MD  
*Notes on the Early Development of Pediatric Surgery in the United States*

9:30 a.m. – 12:30 p.m.  Companion Tour – Living Desert Reserve: “Animal Attraction” Tour  
Tour Lobby

10:00 – 10:30 a.m.  Refreshment Break  
Springs Salons A-F

10:00 a.m. – Noon  Posters Open for Viewing  
Desert Salons 9-14

10:30 – 11:45 a.m.  **Scientific Session II:**  
Oncology and Critical Care

**Moderators:**  
Joel Shilyansky, MD; Daniel von Allmen, MD

**Educational Objectives:**  
Attendees will be able to:  
- Cite the evidence for lymph node sampling in Wilms' tumor  
- Describe the use of ECMO for pediatric respiratory failure  
- Review alterations in coagulation factors in trauma patients

13  **CANCER STEM/PROGENITOR CELLS RESPOND POORLY TO CHEMOTHERAPEUTIC AGENTS**  
Katia Meirelles, MD, Leo Andrew O. Benedict, MD, David Dombkowski, Frederic I. Preffer, Jose Teixeira, PhD, David T. MacLaughlin, PhD, Patricia K. Donahoe, MD, Xiaolong Wei, MD, PhD.
*Massachusetts General Hospital, Cambridge, MA, USA.*

14  **OMEGA-3 FATTY ACIDS INHIBIT THE GROWTH OF NEUROBLASTOMA TUMORS AND INDUCE A TUMOR SPECIFIC ESSENTIAL FATTY ACID DEFICIENCY IN A MURINE XENOGRAFT TUMOR MODEL**  
Deepika Nehra, MD¹, Hau D. Le, MD², Erica M. Fallon, MD², Vincent E. de Meijer, MD, MSc³, Amy H. Pan, BA³, Paul Mitchell, MS³, Mark Puder, MD, PhD³.

¹Children's Hospital Boston; Massachusetts General Hospital, Boston, MA, USA, ²Children's Hospital Boston; Beth Israel Deaconess Medical Center, Boston, MA, USA, ³Children's Hospital Boston, Boston, MA, USA.
15 LYMPH NODE SAMPLING IN WILMS TUMOR: WHAT IS “ENOUGH”? 
Kathleen Kieran, MD1, Jeffrey S. Dome, MD2, Peter F. Ehrlich, MD3, Michael L. 
Ritchey, MD3, Robert C. Shamberger, MD3, Daniel M. Green, MD6, Andrew M. 
Davidoff, MD6.
1University of Iowa, Iowa City, IA, USA, 2Children’s National Medical Center, 
Washington, DC, USA, 3University of Michigan, Ann Arbor, MI, USA, 4Pediatric 
Urology Associates, Phoenix, AZ, USA, 5Children's Hospital Boston, Boston, 
MA, USA, 6St Jude Children’s Research Hospital, Memphis, TN, USA.

16 ELIMINATION OF THE MATERNALLY INDUCED ADAPTIVE IMMUNE 
RESPONSE ALLOWS UNIFORM ACHIEVEMENT OF HIGH LEVEL 
ALLOGENEIC CHIMERISM BY IN UTERO HEMATOPOIETIC CELL 
TRANSPLANTATION AND POSTNATAL MINIMAL CONDITIONING BMT 
Matthew T. Santore, MD, Demetri J. Merianos, MD, Carlyn A. Todorow, Alan 
W. Flake, MD.
The Children’s Hospital of Philadelphia, Philadelphia, PA, USA.

17 SURVIVAL IS IMPROVED IN VENOVENOUS VERSUS VENOARTERIAL 
ECMO FOR PEDIATRIC NON-CARDIAC SEPSIS: A STUDY OF THE 
ELSO REGISTRY 
Sean C. Skinner, MD, Joseph A. Iocono, MD, Hubert O. Ballard, MD, Marion 
D. Turner, MD, Daniel L. Davenport, PhD, Joseph B. Zwischenberger, MD.
University of Kentucky, Lexington, KY, USA.

18 ECMO CANNULATION TRENDS FOR PEDIATRIC RESPIRATORY FAILURE 
AND CENTRAL NERVOUS SYSTEM INJURY 
Michael D. Rollins, MD1, Ania Hubbard, MD1, Luke Zabrocki, MD2, Douglas C. 
Barnhart, MD, MPH1, Susan L. Bratton, MD, MPH1.
1Primary Children’s Medical Center, University of Utah, Salt Lake City, UT, 
USA, 2Naval Medical Center San Diego, San Diego, CA, USA.

19 UTILITY OF NEURORADIOGRAPHIC IMAGING IN PREDICTING 
OUTCOMES FOLLOWING NEONATAL EXTRACORPOREAL MEMBRANE 
OXYGENATION 
Michael D. Rollins, MD1, Bradley A. Yoder, MD1, Kevin R. Moore, MD2, Douglas 
C. Barnhart, MD, MPH1, Chris Jones, RN2, Donald M. Null, MD1, Robert J. 
DiGeronimo, MD1.
1Primary Children’s Medical Center, University of Utah, Salt Lake City, UT, 
USA, 2Primary Children’s Medical Center San Diego, San Diego, CA, USA.

20 COAGULATION CHANGES IN PEDIATRIC TRAUMA PATIENTS 
Mark Leo Ryan, MD, Chad M. Thorson, MD, Christian A. Otero, MD, Edgar J. 
Pierre, MD, David M. Andrews, MD, Holly L. Neville, MD, Juan E. Sola, MD, 
Kenneth G. Proctor, PhD.
University of Miami Miller School of Medicine, Miami, FL, USA.

11:45 a.m. – Noon Introduction of New Members Desert Salons 7-8
### Monday/Tuesday

**Noon – 1:00 p.m.**  
**Presidential Address**  
Marshall Z. Schwartz, MD  
*Healthcare Quality, Access, Cost, Workforce and Surgical Education: The Ultimate Perfect Storm*  
*Desert Salons 7-8*

**1:00 – 2:30 p.m.**  
Benjy Brooks Society Meeting and Luncheon  
Pre-Registration Required  
*Director’s Suite 4*

**2:00 – 6:00 p.m.**  
Golf Tournament -  
Pre-Registration Required  
*JW Marriott Valley Course*

**3:00 – 5:00 p.m.**  
Tennis Tournament -  
Pre-Registration Required  
*JW Marriott Lawn & Tennis Club*

**5:00 – 6:30 p.m.**  
Journal of Pediatric Surgery Reception  
*Director’s Suite 4*

**6:00 – 7:30 p.m.**  
Residents, International and New Member Reception  
(By Invitation Only)  
*The Pointe*

**6:00 – 10:00 p.m.**  
Complimentary Shuttle Transportation  
Provided to/from the El Paseo Area for Registered APSA guests to dine off site—Optional  
*Tour Lobby*

**Tuesday, May 24**

**6:30 – 8:00 a.m.**  
Member Business Meeting with Breakfast  
*Desert Salons 7-8*

**6:30 a.m. – 3:30 p.m.**  
Registration Open  
*Desert West Foyer*

**6:30 a.m. – 3:30 p.m.**  
Speaker Ready Room Open  
*Desert Salons 5-6*

**6:30 a.m. – 3:30 p.m.**  
Internet Café Open  
*Desert West Foyer*

**7:00 – 8:00 a.m.**  
Continental Breakfast for Non-members  
*Springs Salons A-F*

**7:00 a.m. – 3:00 p.m.**  
Posters Open for Viewing  
*Desert Salons 9-14*

**7:00 a.m. – 3:30 p.m.**  
Exhibits Open  
*Springs Salons A-F*

**8:00 – 9:00 a.m.**  
**Jay and Margie Grosfeld Lecture**  
Anthony Atala, MD  
*Regenerative Medicine: New Approaches to Healthcare*  
*Desert Salons 7-8*

**8:00 – 10:00 a.m.**  
Companion/Hospitality Room Open for Registered Companions only  
*Sea Grille, 1st Floor*

**9:00 – 10:30 a.m.**  
**Scientific Session III**  
Fetal/Neonatal  
*Desert Salons 7-8*

**Moderators:**  
R. Cartland Burns, MD; Carroll M. Harmon, MD
Educational Objectives:
At the completion of this session, the learner will be able to:
• Describe fetal inflammatory responses to various conditions of stress
• Cite the relationship between symptoms and outcome in biliary atresia
• Describe the use of ultrasound in NEC
• Review surgical options for several surgical congenital anomalies

21 A PRACTICAL PRENATAL SOURCE OF AUTOLOGOUS NEURAL PROGENITOR CELLS FOR THE TREATMENT OF SPINA BIFIDA
Christopher G. Turner, MD1, Justin D. Klein, MD1, Junmei Wang, PhD2, Devang Thakor, PhD2, Darcy Benedict, BA2, Azra Ahmed, BS1, Yang D. Teng, MD PhD2, Dario O. Fauza, MD.
1Children’s Hospital Boston, Boston, MA, USA, 2Brigham and Women’s Hospital, Boston, MA, USA.

22 CRANIOFACIAL REPAIR WITH FETAL BONE GRAFTS ENGINEERED FROM AMNIOTIC MESENCHYMAL STEM CELLS
Christopher G. Turner, MD, Justin D. Klein, MD, Fabienne Gray, MD, Azra Ahmed, BS, David Zurakowski, PhD, Dario O. Fauza, MD.
Children’s Hospital Boston, Boston, MA, USA.

23 MICROCYSTIC CONGENITAL CYSTIC ADENOMATOID MALFORMATION WITH HYDROPS FETALIS: STEROIDS VERSUS OPEN FETAL RESECTION
Kenneth Loh, MD, Eric Jelin, MD, Shinjiro Hirose, MD, Vickie Feldstein, MD, Ruth Goldstein, MD, Hanmin Lee, MD.
University of California, San Francisco, San Francisco, CA, USA.

24 CARDIAC REGENERATION FOLLOWING FETAL MYOCARDIAL INFARCTION IS ASSOCIATED WITH DECREASED PROINFLAMMATORY CYTOKINES, DECREASED INFLAMMATION, AND INCREASED REACTIVE OXYGEN SPECIES SCAVENGERS
Myron Allukian, MD1, Benjamin J. Herdrich, MD1, Dustin M. Bermudez, MD1, Liping Zhang, MS2, Junwang Xu, PhD2, Wenda Wu, MS2, Bianca C. Chin, MD1, Wanda Dorsett-Martin, DVM2, Robert C. Caskey, MD2, Marc E. Mitchell, MD2, Joseph H. Gorman, MD1, Robert C. Gorman, MD1, Kenneth W. Liechty, MD.
1Hospital Of University of Pennsylvania, Philadelphia, PA, USA, 2University of Mississippi Medical Center, Jackson, MS, USA.

25 MATERNAL–FETAL CELLULAR SIGNALING MECHANISMS IN SEVERE CONGENITAL DIAPHRAGMATIC HERNIA
Geonna Marie Bautista, MD1, Kelly D. Gonzales, MD1, Amar Nijagal, MD1, Clara Ward, MD1, Doug N. Miniati, MD1, Hanmin Lee, MD1, Sheila Keating, MD2, Philip Norris, MD2, Tzong-Hae Lee, MD2, Michael Busch, MD2, Tippi C. MacKenzie, MD1.
1University of California, San Francisco, San Francisco, CA, USA, 2Blood Systems Research Institute, San Francisco, CA, USA.
26 NITRIC OXIDE USE IN NEONATES WITH CONGENITAL DIAPHRAGMATIC HERNIA: A TIME-TREND ANALYSIS
Kimberly A. Ruscher, MD MPH1, Tina Thomas, MBBS1, Katherine Herbst2, Stephen Neff2, Kelleigh Briden2, Brendan T. Campbell, MD MPH.
1University of Connecticut, Hartford, CT, USA, 2Connecticut Children’s Medical Center, Hartford, CT, USA.

27 PROSTHETIC MESH PLUG FOR NEONATAL THORACOSCOPIC CONGENITAL DIAPHRAGMATIC HERNIA REPAIR: OUTCOMES OF A NEW TECHNIQUE
Katrine Lofberg, MD1, David Bliss, MD2, Julie Mckee, MD2, Garret Zallen, MD2, Mark Silen, MD1, Sanjay Krishnaswami, MD2.
1Oregon Health and Science University, Portland, OR, USA, 2Oregon Health and Science University and Legacy Emanuel Children’s Hospital, Portland, OR, USA.

28 ABDOMINAL WALL MUSCLE FLAP IS SUPERIOR TO PATCH REPAIR OF LARGE CONGENITAL DIAPHRAGMATIC HERNIAS
Michael D. Rollins, MD1, Elisabeth Jacques2, Eric R. Scaife, MD1, Bradley A. Yoder, MD1, Rebekka L. Meyers, MD1, Annette Harman, NP3, Earl C. Downey, MD1, Douglas C. Barnhart, MD, MPH1.
1Primary Children’s Medical Center, University of Utah, Salt Lake City, UT, USA, 2University of Utah School of Medicine, Salt Lake City, UT, USA, 3Primary Children’s Medical Center, Salt Lake City, UT, USA.

29 INFLUENCE OF LOCATION OF DELIVERY ON OUTCOME IN NEONATES WITH GASTROSCHISIS
Ahmed Nasr, MD, MS, Jacob C. Langer, MD, The Canadian Pediatric Surgical Network.
The Hospital for Sick Children Toronto, Toronto, ON, Canada.

30 EFFECTS OF GLUTAMINE SUPPLEMENTATION ON PLASMA AMINO ACIDS IN SURGICAL INFANTS RECEIVING PARENTERAL NUTRITION
Simon Eaton1, Evelyn GP Ong1, Anahita Dehbozorgi1, Steve Krywawych2, Venetia Horn2, Nigel J. Klein3, Agostino Pierro1.
1UCL Institute of Child Health, London, United Kingdom, 2Great Ormond Street Hospital for Children, London, United Kingdom, 3UCL Institute of Child Health and Great Ormond Street Hospital for Children, London, United Kingdom.

31 THE ROLE OF ULTRASOUND IN THE DIAGNOSIS AND MANAGEMENT OF NECROTIZING ENTEROCOLITIS
Csilla Balassy, MD1, Aideen Moore, MD2, J. Ted Gerstle, MD3, Alan Daneman, MD2.
1Medical University of Vienna, Vienna, Austria, 2Hospital for Sick Children, Toronto, ON, Canada.
32 DURATION OF SYMPTOMS PRE-KASAI PORTOENTEROSTOMY, NOT AGE AT KASAI, IS PROGNOSTIC IN BILIARY ATRESIA.
Momoko Wada, MD, Hiroti Nakamura, MD, Hiroyuki Koga, MD, Go Miyano, MD, Rafael H. Dizon, MD, Geoffrey J. Lane, MD, Yoshifumi Kato, MD, Tadaharu Okazaki, MD, Atsuyuki Yamataka, MD.
Juntendo University School of Medicine, Tokyo, Japan.

10:30 – 11:00 a.m. Refreshment Break Springs Salons A-F
11:00 a.m. – 12:15 p.m. Scientific Session IV Desert Salons 7-8

Moderators:
Gail E. Besner, MD; Karl G. Sylvester, MD

Educational Objectives:
Participants in this session will be able to:
• Describe progress in the understanding of the pathophysiology of NEC
• Discuss advances in translational approaches to intestinal failure
• Identify stem cell applications to pediatric surgery

33 CYCLOSPORINE IMPROVES MESENTERIC PERFUSION AND ATTENUATES NEC-LIKE INTESTINAL INJURY IN ASPHYXIATED NEWBORN PIGLETS DURING REOXYGENATION
Richdeep S. Gill, MD, Namdar Manouchehri, MD, Qin Liu, MD, PhD, Tze-Fun Lee, MD, PhD, David Bigam, MD, MSc, FRCSC, Po-Yin Cheung, MBBS, PhD, FRCP (Can&Edin).
University of Alberta, Edmonton, AB, Canada.

34 HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) IMPROVES INTESTINAL BARRIER FUNCTION AND REDUCES MORTALITY IN A MURINE MODEL OF PERITONITIS
Jixin Yang, MD, Andrei Radulescu, MD, PhD, Chun-Liang Chen, PhD, Hong-Yi Zhang, MD, Iyore James, MD, Gail Besner, MD.
Nationwide Children’s Hospital, Columbus, OH, USA.

35 TGF β ACTIVITY IS REQUIRED FOR ENTEROBACTER SAKAZAKII INDUCED NECROTIZING ENTEROCOLITIS
Claudia N. Emami, MD MPH, Rahul Mittal, PhD, Henri R. Ford, MD MHA FACS, Nemani V. Prasadaro, PhD.
Children’s Hospital Los Angeles, Los Angeles, CA, USA.

36 OVER-EXPRESSION OF HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) PROTECTS INTESTINAL MYENTERIC PLEXUS NEURONS FROM ISCHEMIA/REPERFUSION INJURY
Yu Zhou, MD, PhD, Kyle Markmann, Gail E. Besner, MD.
Nationwide Children’s Hospital, Columbus, OH, USA.
37 RAPAMYCIN-INDUCED TUMOR VASCULATURE REMODELING IN RHABDOMYOSARCOMA XENOGRAFTS INCREASES THE EFFECTIVENESS OF ADJUVANT IONIZING RADIATION
Adrienne L. Myers, MD, Wayne S. Orr, MD, Jason Denbo, MD, Cathrine Ng, Andrew Davidoff, MD.
St. Jude Children’s Research Hospital, Memphis, TN, USA.

38 THE MATERNAL ADAPTIVE IMMUNE RESPONSE AGAINST PATERNAL ANTIGENS INCITES FETAL DEMISE AFTER FETAL INTERVENTION
Amar Nijagal, MD, Marta Wegorzewska, Tom Le, Julissa Gonzalez, Tippi C. MacKenzie, MD.
UCSF, San Francisco, CA, USA.

39 EXPRESSION PATTERNS OF THE ENTERIC HORMONE GLP-2 AND ITS RECEPTOR IN INFANTS WITH INTESTINAL FAILURE
Jennifer Stanger, MD1, Dana Doctor, MD2, Laurie Wallace, MD3, Esmaeel Taqi, MD2, Viona Lam, MD1, Jens Holst, MD4, David Sigalet, MD1.
1Division of Pediatric General Surgery, Department of Surgery, University of Calgary, Calgary, AB, Canada, 2Division of Pediatric Gastroenterology, University of Calgary, Calgary, AB, Canada, 3Inflammation, Infection, Immunology Institute, Faculty of Medicine, University of Calgary, Calgary, AB, Canada, 4Panum Institute, Copenhagen, Denmark.

40 KEY MESENCHYMAL COMPONENTS OF TISSUEENGINEERED SMALL INTESTINE DO NOT DERIVE FROM BONE MARROW STEM CELLS
Frederic G. Sala, PhD, Jamil A. Matthews, MD, Allison L. Speer, MD, Erik R. Barthel, MD, PhD, Tracy C. Grikscheit, MD.
Childrens Hospital Los Angeles, Los Angeles, CA, USA.

41 A SIMPLIFIED PROTOCOL FOR HUMAN MESENCHYMAL AMNIOCYTE-INDUCED PLURIPOTENT STEM CELL MAINTENANCE: IMPLICATIONS FOR DISEASE MODELS AND CLINICAL APPLICABILITY
Azra Ahmed, BS, Christopher G. Turner, MD, Justin D. Klein, MD, Yuin-Han Loh, PhD, Odelya Hartung, BS, Philip Manos, BA, Fabienne Gray, MD, George Q. Daley, MD PhD, Dario O. Fauza, MD.
Children’s Hospital Boston, Boston, MA, USA.

12:15 – 12:30 p.m.  Box Lunch Pick-Up  Desert Salons Foyer
12:30 – 1:30 p.m.  Innovation Session and Lunch  Desert Salons 7-8
Abstracts on New and Innovative Techniques and Procedures
The Sheikh Zayed Institute Award for Innovation in Pediatric Surgery will be announced during the President’s Banquet

Moderators:
Timothy D. Kane, MD; Milissa A. McKee, MD
**Educational Objectives:**
Participants will be able to:

- Discuss new and innovative techniques or procedures which are being pursued both in the laboratory and clinically in our field, that are not yet mainstream
- Discuss the barriers and difficulties in research and clinical application of innovative techniques and technology
- Explain the advancement of innovative technology in pediatric surgery in the future

i1 **INNOVATIVE PEDIATRIC SURGERY: DO WE NEED INSTITUTIONAL REVIEW BOARD APPROVAL?**
Erica M. Carlisle, MD, Josh Hemmerich, PhD, Rashed Hasan, BS, Peter Angelos, MD, PhD, Michael J. Morowitz, MD, Kevin K. Roggin, MD. 
*University of Chicago Medical Center, Chicago, IL, USA.*

i2 **A NOVEL ANTI-REFLUX PROCEDURE: GASTROPLASTY WITH RESTRICTED ANTRUM TO CONTROL EMESIS (GRACE)**
Lucas P. Neff, MD1, Robert D. Becher, MD1, Aaron U. Blackham, MD1, Natalie E. Banks, BS2, Erin L. Mitchell, DVM2, John K. Petty, MD1.
1'Wake Forest University Department of General Surgery, Section of Pediatric Surgery, Winston-Salem, NC, USA, 2'Wake Forest University School of Medicine, Winston-Salem, NC, USA.

i3 **EVALUATION OF INTESTINAL VIABILITY USING 3-CCD (CHARGE COUPLED DEVICE) IN CHILDREN UNDERGOING APPENDECTOMY**
Maridelle B. Millendez1,2, MD, Nicole J. Crane, PhD2, Eric A. Elster, MD2,3,4, Shawn D. Safford, MD3.
1'Walter Reed Army Medical Center, Washington, DC, USA, 2'Naval Medical Research Center, Silver Spring, MD, USA, 3'National Naval Medical Center, Bethesda, MD, USA, 4'Uniformed Services University of the Health Sciences, Bethesda, MD, USA

i4 **DEVELOPMENT OF AN ENDOLUMINAL INTESTINAL LENGTHENING CAPSULE**
Rebecca Stark, MD, Tatiana Zupekan, MD, Mohanchandra Panduranga, PhD, Gregory Carman, PhD, James C.Y. Dunn, MD PhD 
*UCLA Medical Center, Los Angeles, CA, USA.*

i5 **A NEW ANIMAL MODEL OF EXTRACORPOREAL FETAL SUPPORT WITH PRESERVATION OF THE PLACENTA**
Jose H. Salazar, MD1, Alodia Gabre-Kidan, MD2, Gezzer Ortega, MD1, Shelly S. Choo, BA1, Daniel S. Rhee, MD, MPH1, Dominic Papandria, MD1, Diana Scorpio, DVM, MPH1, Dawn Ruben, DVM1, Gary Oldenburg, RRT-NPS1, Haven Custis, RT1, Melanie Albano1, Caitlin Harris1, William B. Fulton, MS1, Jude Crino, MD1, Fizan Abdullah, MD, PhD1.
1'Johns Hopkins University, Baltimore, MD, USA, 2'Columbia University, New York, NY, USA.
AN ENSEMBLE ALGORITHM FOR IMPROVED NEC RISK STRATIFICATION
Gigi Liu, MD1, Bruce X. Ling, PhD1, Fizan Abdulla, MD, PhD2, Mary Brandt, MD3, Mary Cay Harris, MD4, Larry Moss, MD5, Karl G. Sylvester, MD1.
1Stanford University, Stanford, CA, USA, 2Johns Hopkins University, Baltimore, MD, USA, 3Texas Children’s Hospital, Houston, TX, USA, 4Children’s Hospital of Philadelphia, Philadelphia, PA, USA, 5Nationwide Children’s Hospital, Columbus, OH, USA.

USING ROBOTIC TELECOMMUNICATIONS TO TRIAGE PEDIATRIC DISASTER VICTIMS
Rita V. Burke, PhD, MPH1, Bridget M. Berg, MPH1, Paul Vee1, Inge Morton1, Alan Nager, MD1, Robert Neches, PhD2, Randall Wetzel, MD1, Jeffrey S. Upperman, MD1.
1Children’s Hospital Los Angeles, Los Angeles, CA, USA, 2University of Southern California, Los Angeles, CA, USA.

1:30 – 1:45 p.m. APSA Foundation Scholar Desert Salons 7-8
Cynthia D. Downard, MD
Kosair Children’s Hospital, Louisville, KY
Control of Intestinal Microcirculation in NEC

1:45 – 2:30 p.m. APSA Updates Desert Salons 7-8

2:30 – 3:30 p.m. International Guest Lecture Desert Salons 7-8
Professor Takeshi Miyano
A Brief History of Pediatric Surgery and Healthcare Delivery Systems in Japan

3:00 – 5:00 p.m. Poster Dismantle Desert Salons 9-14

3:30 – 6:45 p.m. Leisure Time

6:45 – 7:30 p.m. President’s Reception Springs Patio

7:30 – 10:00 p.m. President’s Banquet Desert Salons 7-8

Wednesday, May 25

6:30 – 7:30 a.m. Committee Meetings See page 69 for ancillary meeting schedule

7:00 – 10:30 a.m. Speaker Ready Room Open Desert Salons 5-6

7:00 – 11:30 a.m. Internet Café Open Desert West Foyer

7:00 – 11:30 a.m. Registration Open Desert West Foyer

7:30 – 8:00 a.m. Continental Breakfast Desert West Foyer

8:00 – 9:00 a.m. Scientific Session V Desert Salons 7-8
Quality Improvement/Clinical Care

Moderators:
Daniel J. Ostlie, MD; Daniel von Allmen, MD

Educational Objectives:
Participants will be able to:
• Describe the quality of pediatric surgical research publications
• Cite the impact of legislation on trauma
• Understand programs that can improve patient experience and care

42  A CRITICAL APPRAISAL OF PUBLISHED RANDOMIZED TRIALS WITHIN PEDIATRIC GENERAL SURGERY DURING THE TIME PERIOD 2000 – 2009
Martin L. Blakely, MD, MS¹, Lillian Kao, MD, MS², Rupa Seetharamaiah, MD¹, KuoJen Tsao, MD², Eunice Y. Huang, MD, MS¹, Kevin P. Lally, MD, MS².
¹University of Tennessee Health Science Center, Memphis, TN, USA, ²University of Texas Health Science Center, Houston, TX, USA.

43  QUALITY IMPROVEMENT AND PATIENT CARE CHECKLISTS IN INTRA-HOSPITAL TRANSFERS INVOLVING PEDIATRIC SURGERY PATIENTS
Don K. Nakayama, MD, MBA¹, Sally S. Lester, RN², Darla R. Rich, RN, FNP², Bryan C. Weidner, MD¹, Joshua B. Glenn, MD¹, Issam J. Shaker, MD².
¹Medical Center of Central Georgia, Mercer University School of Medicine, Macon, GA, USA, ²Medical Center of Central Georgia, Macon, GA, USA.

44  PEDIATRIC HERNIA REPAIR: ONE STOP SHOPPING
Sean J. Barnett, MD, MS, Jason S. Frischer, MD, John A. Gaskey, MHA, Frederick C. Ryckman, MD, Daniel von Allmen, MD.
Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA.

45  VARIATION IN RESOURCE UTILIZATION ASSOCIATED WITH THE MANAGEMENT OF APPENDICITIS IN CHILDREN: IMPLICATIONS FOR QUALITY IMPROVEMENT THROUGH COMPARATIVE ANALYSIS AND COLLABORATIVE NETWORKING
Shawn J. Rangel, MD, MSCE¹, Jessica Baxter, BS¹, Jeff Barnes².
¹Children’s Hospital Boston, Boston, MA, USA, ²Child Health Corporation of America, Shawnee Mission, KS, USA.

46  EPIDURAL VERSUS PATIENT CONTROLLED ANALGESIA FOR POST-OPERATIVE PAIN FOLLOWING PECTUS EXCAVATUM REPAIR: A PROSPECTIVE, RANDOMIZED TRIAL
Shawn D. St. Peter, MD, Kathryn A. Weesner, MD, Eric E. Weissend, MD, Susan W. Sharp, PhD, Patricia A. Valusek, MD, Charles L. Snyder, MD, Ronald J. Sharp, MD, George W. Holcomb III, MD, MBA, Daniel J. Ostlie, MD.
Children’s Mercy Hospital, Kansas City, MO, USA.

47  LEGISLATING ALL-TERRAIN VEHICLE ACCESS BY CHILDREN: EFFECTS ON INJURY RATES AND SEVERITY
Natalie L. Yanchar, MD, MSc, Nadia Murphy, MD.
IWK Health Centre, Dalhousie University, Halifax, NS, Canada.

48  MEDIAN ARCULATE LIGAMENT SYNDROME IN CHILDREN: LAST STOP BEFORE FUNCTIONAL GASTROINTESTINAL DISORDERS?
Christopher L. Skelly, MD, Aviram Assidon, Grace Z. Mak, MD, Kathleen Romanowski, MD, Melissa Ruiz, MD, Donald C. Liu, MD, PhD.
University of Chicago, Chicago, IL, USA.
49  A MULTI-CENTER EVALUATION OF THE ROLE OF MECHANICAL BOWEL PREPARATION IN PEDIATRIC COLOSTOMY TAKEDOWN
Katherine Serrurier, BA1, Jie Liu, MPH1, Francine Breckler, PharmD2, Nini Khozeimeh, MD3, Deborah Billmire, MD2, Cynthia Gingalewski, MD3, Gerald Gollin, MD1.

1Loma Linda University School of Medicine, Loma Linda, CA, USA, 2Riley Hospital for Children, Indianapolis, IN, USA, 3Children’s National Medical Center, Washington, DC, USA.

9:00 – 10:00 a.m.  COG Session
Germ Cell Tumors; Neuroblastoma

Moderator:
Andrew M. Davidoff, MD

Educational Objectives:
Attendees will be able to:
• Identify current therapy of neuroblastoma and pediatric germ cell tumors
• Debate surgical guidelines, clinical outcomes and new initiatives for the treatment of these tumors

Germ Cell Tumor Update
Elizabeth A. Beierle, MD

Neuroblastoma: Surgical Guidelines
Peter Mattei, MD

10:00 – 10:15 a.m.  Refreshment Break

Moderators:
Carroll M. Harmon, MD

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

11:30 a.m.  Annual Meeting Adjourns
VIDEO SESSION

Sunday, May 22, 12:45 – 1:45 p.m.

V1
LAPAROSCOPIC REPAIR OF A DUODENAL ATRESIA AND LADD’S PROCEDURE IN A NEONATE WITH MALROTATION

Steven S. Rothenberg
The Rocky Mountain Hospital For Children, Denver, CO, USA

Purpose
To demonstrate current refinements of technique in performing a duodenal anastomosis in a neonate with duodenal atresia. This work is IRB exempt.

Methods
A 33 week premature infant with a prenatal diagnosis of Duodenal atresia was explored laparoscopically on day two of life for repair. The patients weight was 2 Kg. Two 3mm ports and one 4mm port were used for the procedure. The patient was also found to have malrotation at the time of surgery. The procedure consisted of a Ladd’s procedure and duodenoduodenostomy. Techniques of abdominal wall retraction sutures are demonstrated.

Results
The procedure was completed successfully laparoscopically. The procedure took 60 minutes. An NG tube was used for 5 days and feeds were started on post-op day 6.

Conclusions
This video demonstrates that a laparoscopic duodenoduodenostomy and Ladd's procedure is efficacious and safe even in a small premature.

Notes:
VIDEO SESSION

V2
LAPAROSCOPIC NEPHRECTOMY FOR WILMS TUMOR IN A ONE YEAR OLD GIRL

Guido Seitz, MD¹, Steven W. Warmann, MD¹, Martin Ebinger, MD¹, Falko Fend, MD², Jörg Fuchs, MD¹

¹University Children’s Hospital, Tuebingen, Germany, ²University Hospital, Department of Pathology, Tuebingen, Germany

Purpose
To demonstrate the technique of a simultaneous laparoscopic nephrectomy of the left kidney and tru-cut biopsy on the right kidney for suspected bilateral Wilms tumor in a one year old girl.

Methods
Preoperative work-up revealed a large left sided Wilms tumor. In the contralateral kidney MRI revealed a suspicious alteration of the upper pole. Preoperative chemotherapy was administered according to the SIOP2001/GPOH protocol. Decision was taken to perform a laparoscopic nephrectomy on the left side and a laparoscopic biopsy of the right kidney.

The patient was placed in supine position. One 5 mm and two 3 mm ports were placed. The tumor was completely mobilized using the harmonic knife. The renal artery and vein were ligated and transected with the harmonic scalpel. The tumor was removed via a Pfannenstiel’s incision because of its large size. A laparoscopically guided tru-cut biopsy of the upper pole was performed on the right kidney. Lymph node sampling was performed from all relevant levels.

Results
A complete tumor resection without microscopic residuals was achieved. The post-operative course was uneventful. Histological work up revealed nephroblastoma of intermediate risk (stromal subtype without anaplasia) on the left side and nephroblastomatosis on the right side. All lymph nodes were tumor free. Postoperative chemotherapy was continued.

Conclusions
Laparoscopic tumor nephrectomy is feasible even in young children suffering from nephroblastoma; however, a cautious selection of patients is essential. Intraoperative tumor spillage should be avoided in any case.

Notes:
SINGLE SITE UMBILICAL LAPAROSCOPIC SPLENECTOMY AND
CHOLECYSTECTOMY IN A PATIENT WITH HEREDITARY
SPHEROCYTOSIS AND CHOLELITHIASIS

Patricia A. Valusek, MD¹, George W. Holcomb III, MD, MBA²
¹Childrens Minneapolis, Minneapolis, MN, USA, ²Children’s Mercy Hospital, Kansas City, MO, USA

Purpose
Single site umbilical laparoscopic surgery (SSULS) is being increasingly employed by pediatric surgeons for cholecystectomy, splenectomy, and appendectomy. It is also being used by some surgeons for pyloromyotomy. This patient is a 6-year-old whose older sister had undergone a laparoscopic splenectomy utilizing the traditional four port laparoscopic approach. She required splenectomy for symptoms related to her hereditary spherocytosis and also was found to have cholelithiasis on ultrasound.

The salient features of a single site laparoscopic splenectomy and cholecystectomy will be shown. The principles of the SSULS approach are similar with the traditional four port technique including complete mobilization of the spleen and ligation and division of the splenic vessels with the endoscopic stapler, placement of the spleen in an endoscopic retrieval bag and intracorporeal morcellation of the spleen. For the cholecystectomy, identification of the cystic duct and artery is depicted along with retrograde mobilization from its liver attachment.

This patient recovered uneventfully and was discharged on the first postoperative day. He has not developed any postoperative complications with a follow-up of one year.

Notes:
Thoracoscopic thymectomy is a safe, effective method of thymus removal. This technique offers decreased morbidity compared to the traditional trans-cervical or trans-sternal approaches.
VIDEO SESSION

V5
LAPAROSCOPIC LEFT LATERAL SEGMENTECTOMY IN A CHILD
Karen Diefenbach, MD, Milissa McKee, MD, MPH
Yale University School of Medicine, New Haven, CT, USA

Purpose
To demonstrate the technique used to perform a left lateral segmentectomy of the liver in a pediatric patient.

Methods
We report a case of a child with a solitary nodule in the left lobe of the liver which was successfully resected laparoscopically. An 8 y/o female previously treated with chemotherapy and radiation for astrocytoma in the first year of life subsequently presented with a left renal cell carcinoma. She underwent a left nephrectomy and post-operative surveillance imaging revealed a growing nodule in the left lateral segment of the liver. Because of concern for malignancy (metastatic disease or new primary), she underwent laparoscopic left lateral segmentectomy for diagnostic and therapeutic purposes. This video demonstrates the technique.

Results
We successfully completed an anatomic left lateral segmentectomy laparoscopically. The patient did well and was subsequently discharged home on post-operative day six.

Conclusion
Laparoscopic anatomic liver resections are feasible for selected lesions in pediatric patients.

Notes:
VIDEO SESSION

V6
EXIT TO RESECTION OF A FETAL SACROCCYGEAL TERATOMA
Payam Saadai, MD, Diana L. Farmer, MD, Tippi MacKenzie, MD, Danny Wu, MD, Ruth Goldstein, MD, Charles Cauldwell, MD, Thomas Shimotake, MD, Michael Harrison, MD, Hanmin Lee, MD, Doug Miniati, MD
University of California San Francisco, San Francisco, CA, USA

Purpose
Sacrococcygeal Teratoma (SCT) is the most common congenital tumor diagnosed in the newborn. When diagnosed postnatally, the reported mortality is less than 10%. However, when diagnosed in the fetus, mortality is greater than 50% suggesting a high rate of in-utero demise. In the presence of hydrops, the disease is almost universally fatal. We report the case of a fetus with SCT and severe hydrops who was successfully managed with attempted radiofrequency ablation followed by Ex-Utero Intrapartum Treatment (EXIT) procedure and immediate postnatal resection.

Methods
A 33 year-old gravida 4 para 2 female was diagnosed with fetal SCT at gestational age 15 weeks. Over 8 weeks, the SCT grew from 2cm to 12cm, and she was referred to our institution for further management. By 26 weeks placentomegaly and fetal hydrops had developed. Percutaneous radiofrequency ablation was performed in an effort to halt tumor growth. Although hydrops and tumor growth initially improved, serial ultrasounds subsequently demonstrated worsening hydrops, chorioamniotic separation, and impending fetal demise. EXIT to near-total SCT resection was performed urgently at 26 5/7 weeks. On day of life (DOL) #2, diverting colostomy was created. On DOL #9, re-exploration, completion resection, coccygectomy, and wound closure was performed.

Results
Initially the patient required high frequency oscillatory ventilation and inhaled nitric oxide for premature lung disease. This was subsequently weaned. She is currently at DOL #50 (gestational age 34 weeks) and weighs 1520 grams. She is tolerating full feeds and is on 2L high flow nasal cannula. The tumor weighed 1600g while the estimated birth weight was 1000g. Pathology revealed a 90% mature teratoma with no malignant elements.

Conclusions
Ex-Utero Intrapartum Treatment to resection is a viable surgical strategy for a fetus with sacrococcygeal teratoma and severe hydrops. This case demonstrates the need for close surveillance of these patients as well as potential treatment options.

Notes:
Purpose
Pyloric stenosis is a common pediatric surgery problem. Pyloromyotomies are often performed with laparoscopy. Three difficulties that are often encountered are: damage to the duodenum secondary to retraction with left hand, inadequate securement during the myotomy and inadequate mobilization past the liver.

Methods
The laparoscopic pyloric immobilizer was created to cradle the pyloris during a laparoscopic pyloromyotomy.

Results
The laparoscopic pyloric immobilizer provides safe, and adequate retraction with great stability during the operation.

Conclusions
The pyloric immobilizer is an instrument that gives benefits of safety, manipulation and stabilization when performing a laparoscopic pyloromyotomy.

Notes:
P1

AHRQ PEDIATRIC INDICATORS AS A QUALITY METRIC FOR SURGERY IN CHILDREN: DO THEY PREDICT ADVERSE OUTCOMES?

Daniel S. Rhee, MD, MPH, Yiyi Zhang, MHS, Dominic Papandria, MD, Gezzer Ortega, MD, Fizan Abdullah, MD, PhD
Johns Hopkins, Baltimore, MD, USA

Purpose
The Pediatric Quality Indicators (PDI) were developed by the Agency for Healthcare Research and Quality (AHRQ) as metrics to compare patient safety and quality of pediatric care. They are being considered for mandatory reporting as well as to be associated with pay-for-performance efforts. The present study evaluates the PDIs predictive value for mortality, length of stay, and hospital charges for surgery in children.

Methods
A cross-sectional study was performed using State Inpatient data from 1988-2007. Patients under 18 years of age who underwent an inpatient surgical procedure were included. Patients were evaluated for rates of selected PDIs. Odds of mortality were calculated using multiple logistic regression adjusting for age, gender, race, region, hospital type, and co-morbidities. Multiple linear regression was used for length of stay and total charges.

Results
A total of 1,964,456 patients were included. The unadjusted mortality rate was 5.4% for all patients with at least one PDI and 0.6% for those with none. Multivariate analysis showed a significant increase in risk for mortality for accidental puncture/ laceration, postoperative hemorrhage, postoperative respiratory failure, postoperative sepsis, wound dehiscence, selected infections from medical care, and transfusion reactions. Occurrence of any PDI was associated with a 1.7 times increased risk of mortality. PDIs were associated with an increased length of stay and total hospital charges.

Conclusions
The present study shows that the presence of any Pediatric Quality Indicator is associated with increased mortality risk as well as increased hospital stay and total hospital charges. This provides positive evidence for the utility of these indicators as metrics for quality and patient safety.
## Patient Outcomes by Selected Pediatric Quality Indicators

<table>
<thead>
<tr>
<th>Pediatric Quality Indicator (PDI)</th>
<th>Mortality (%)</th>
<th>p-value</th>
<th>Length of Stay (days)</th>
<th>p-value</th>
<th>Total charges ($)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PDI 1 - Accidental Puncture/Laceration</td>
<td>1.8</td>
<td>0.00</td>
<td>5.0</td>
<td>0.00</td>
<td>43,167</td>
<td>0.00</td>
</tr>
<tr>
<td>PDI 5 Iatrogenic Pneumothorax in Non-neonates</td>
<td>1.7</td>
<td>0.07</td>
<td>8.0</td>
<td>0.00</td>
<td>81,681</td>
<td>0.00</td>
</tr>
<tr>
<td>PDI 8 Postoperative Hemorrhage or Hematoma</td>
<td>2.3</td>
<td>0.01</td>
<td>3.9</td>
<td>0.00</td>
<td>53,338</td>
<td>0.00</td>
</tr>
<tr>
<td>PDI 9 Postoperative Respiratory Failure</td>
<td>5.1</td>
<td>0.00</td>
<td>17.2</td>
<td>0.00</td>
<td>141,215</td>
<td>0.00</td>
</tr>
<tr>
<td>PDI 10 Postoperative Sepsis</td>
<td>2.5</td>
<td>0.00</td>
<td>26.5</td>
<td>0.00</td>
<td>172,150</td>
<td>0.00</td>
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<tr>
<td>PDI 11 Postoperative Wound Dehiscence</td>
<td>3.4</td>
<td>0.01</td>
<td>15.0</td>
<td>0.00</td>
<td>77,413</td>
<td>0.00</td>
</tr>
<tr>
<td>PDI 12 Selected Infections Due to Medical Care</td>
<td>1.2</td>
<td>0.00</td>
<td>34.0</td>
<td>0.00</td>
<td>210,551</td>
<td>0.00</td>
</tr>
<tr>
<td>PDI 13 Transfusion Reaction</td>
<td>8.9</td>
<td>0.01</td>
<td>25.0</td>
<td>0.00</td>
<td>237,901</td>
<td>0.00</td>
</tr>
<tr>
<td>Any PDI</td>
<td>1.7</td>
<td>0.00</td>
<td>5.8</td>
<td>0.00</td>
<td>70,064</td>
<td>0.00</td>
</tr>
</tbody>
</table>

**Notes:**
P2
INCIDENCE AND NATURE OF POSTOPERATIVE COMPLICATIONS IN PATIENTS ADMITTED TO THE NEONATAL INTENSIVE CARE UNIT
Alexander P. Stoffan, MD, Anne Hansen, MD, J W. Sparks, MD, Elizabeth K. Norton, RN, Keri Kucharski, RN, BSN, Jessica Baxter, Shawn J. Rangel, MD, MSCE
Children’s Hospital Boston, Boston, MA, USA

Introduction
Little is known regarding the epidemiology of postoperative complications in patients admitted to the neonatal intensive care unit. The purpose of this study was to characterize the incidence & nature of adverse events in this patient population.

Methods
As part of a pilot effort to identify opportunities for quality improvement surrounding neonatal surgical care, 24 potentially preventable events relevant to neonates were identified through a multi-disciplinary steering committee represented by neonatology, anesthesiology, surgery & nursing. A six-month retrospective analysis of these events was then conducted for all neonates on the general surgery service through a standardized 30-day postoperative audit of progress notes, laboratory & imaging data, and review of our institution’s Serious Event Reporting System Database.

Results
73 neonates underwent a total of 131 procedures, resulting in a collective 2,922 NICU-patient days of follow-up. The most common preoperative diagnosis included NEC, CDH, TEF and duodenal atresia. A total of 210 events were identified, reflecting 2.9 complications per patient and an overall patient-event rate of 73%. Dislodgement of tubes and indwelling devices (e.g. endotracheal tubes, vascular access & epidural catheters) accounted for 24% of all events, followed by wound infections (13%), pressure-related soft-tissue complications (8%), device malfunction (e.g. PICC lines, 7%), pneumonia (7%), and iatrogenic pneumothorax (5%). Premature infants were at a significantly higher risk for complications when compared to term infants (82.9% vs. 61.5%; OR 3.01, 95% CI: 1.01-8.99).
Conclusions
The rate of postoperative complications in the neonatal population is alarmingly high. The majority of neonates will experience at least one adverse event during the postoperative period, many of which may be avoidable. These data can be used as a basis for future investigation to explore the relative morbidity, cost & preventability of specific events in order to prioritize the development of perioperative checklists and other NICU-based safety interventions.

Notes:
POSTER SESSION I

P3
LESSONS FROM A LIVER HEMANGIOMA REGISTRY: SUBTYPE CLASSIFICATION
Ann M. Kulungowski1, Ahmad I. Alomari, MD2, Aditya Chawla, BA1, Emily R. Christison-Lagay, MD1, Steven J. Fishman, MD1
1Children’s Hospital Boston, Vascular Anomalies Center, Department of Surgery, Harvard Medical School, Boston, MA, USA, 2Children’s Hospital Boston, Vascular Anomalies Center, Department of Radiology, Harvard Medical School, Boston, MA, USA

Purpose
A previously proposed classification of hepatic hemangioma (HH) postulated three types of lesions: focal, multifocal, and diffuse. A registry www.liverhemangioma.org was created to longitudinally track patients to validate this classification and learn more about these poorly studied lesions.

Methods
Registry records entered prospectively or retrospectively were reviewed. Fisher’s exact test was used for comparisons.

Results
Of 121 patients, 119 fit into previously described categories of focal (n=33), multifocal (n=68), and diffuse (n=18) based on imaging characteristics (Figure 1). Two patients with features of multifocal and diffuse may represent the theorized “missing link” between these types. The focal group had a balanced gender distribution (female=48.5%); whereas, multifocal and diffuse had a predilection towards females (66.2% and 70.0%, respectively) similar to cutaneous infantile hemangioma (IH). In those with antenatal imaging, the mass was detected prenatally in 30% (9/30) of patients with focal HH; detection was not possible in multifocal (0/55) nor diffuse (0/15) HH (p<0.001) indicating postnatal proliferation typical of common cutaneous IH. Cutaneous hemangiomas accompanied 77.4% (48/62) of multifocal HH, 53.3% (8/15) of diffuse HH, and 15.3% (4/26) of focal HH (p<0.001). Hypothyroidism was documented in 100% (16/16) of patients with diffuse HH, 21.4% (9/42) with multifocal HH, but in no patients with focal HH (0/17) (p<0.001); profound hypothyroidism (TSH>50 mU/ml) occurred only in diffuse HH (11/15). Neither “missing link” patient had detectable antenatal lesions and both had profound hypothyroidism.
**POSTER SESSION I**

**Conclusions**
Analysis of the Liver Hemangioma Registry confirms that focal lesions are biologically distinct from multifocal and diffuse HH. Elucidation of the natural history of distinct subtypes will facilitate refinement of treatment algorithms. Detection of intermediate “missing link” lesions suggests feared diffuse HH may evolve from previously undetected multifocal HH. Screening ultrasonography in infants with multiple cutaneous IH may allow for earlier detection, facilitating therapy to prevent profound hypothyroidism and fatal abdominal compartment syndrome.

![Axial images of hepatic hemangiomas (HH).](image)

*Left:* Focal HH appears as a solitary lesion with peripheral enhancement. (*Left, middle*)

*Middle:* Multifocal HH has scattered lesions throughout the liver. (*Right, middle*)

*Right:* The proposed “missing link” has increasing number of hemangiomas replacing a large portion of the hepatic parenchyma. (*Right*)

*Diffuse HH shows nearly total hepatic parenchymal replacement.*

**Notes:**
P4
OPTO-ELECTRONIC PLETHYSMOGRAPHY (OEP) DEMONSTRATES ABROGATION OF REGIONAL CHEST WALL MOTION DYSFUNCTION IN PECTUS EXCAVATUM PATIENTS FOLLOWING NUSS REPAIR
Richard E. Redlinger, Jr., MD\(^1\), Ashley Wootton, BS\(^2\), Robert E. Kelly, Jr., MD\(^2\), Donald Nuss, MB, ChB\(^2\), Michael J. Goretsky, MD\(^2\), M. Ann Kuhn, MD\(^2\), Robert J. Obermeyer, MD\(^2\)
\(^1\)Eastern Virginia Medical School, Norfolk, VA, USA, \(^2\)Children’s Hospital of the King’s Daughters, Norfolk, VA, USA

Purpose
We previously utilized motion capture technology to demonstrate that pectus excavatum (PE) patients have significantly decreased chest wall motion at the pectus defect compared to the rest of the chest versus unaffected individuals and utilize abdominal respiratory contributions to compensate for decreased upper chest wall motion. We hypothesize that PE repair will reverse chest wall motion dysfunction.

Methods
A prospective, IRB-approved study compared PE patients before and after Nuss repair. Each consenting subject had 89 hemispherical reflective markers (42 anterior, 47 posterior) attached to anatomic thoracic landmarks and performed deep breathing maneuvers captured by infrared cameras. Chest wall reconstruction allows thoracic volume and marker excursion calculation using proprietary software. 65 patients with uncorrected PE ages 10-21 underwent Opto-Electronic Plethysmography (OEP) analysis. Repeat analysis was performed in 42 patients six months after repair (PO).

Results
Volume of the chest wall and its subdivisions (pulmonary rib cage, abdominal rib cage and abdomen) were calculated. Total chest wall volume at rest was significantly increased after repair (PE=13.48L, PO=14.87L, \(p<0.01\)) and in each thoracic compartment. Chest wall motion during respiration was measured at specific marker points along the anterior surface. Marker excursion at the midline was compared to more lateral aspects of the chest wall at multiple horizontal anatomic levels of the thorax and abdomen. PO patients had increased midline marker excursion at the pectus defect compared to PE patients with significant decrease in excursion at level of the umbilicus (Figure 1).
Conclusions
OEP kinematic analysis demonstrates that chest wall remodeling during Nuss repair results in increased thoracic volume. Chest wall motion dysfunction at the pectus defect is reversed following Nuss repair. Abdominal respiratory contributions are also markedly decreased. These findings may help to explain improvement in exertional symptoms, including easy fatigability and shortness of breath, in PE patients following minimally invasive PE repair.

Notes:
POSTER SESSION I

P5
CENTRAL LIVER RESECTION, A FEASIBLE SUBSTITUTE FOR TRANSPLANTATION IN CASES OF HEPATOBLASTOMA
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Children’s Hospital Los Angeles, Los Angeles, CA, USA

Purpose
The purpose of our study was to review our experience with central hepatic resection in the setting of hepatoblastoma. These resections are uncommon and involve resection of segments 4A and 5 of the liver. New COG guidelines recommend consideration of transplantation for these lesions. We reviewed our patients who have undergone resection to examine peri-operative events and outcomes.

Method
After the approval of the Institutional Review Board, we retrospectively evaluated patients with the diagnosis of hepatoblastoma who underwent central hepatic resection by the same surgeon from 1995 to present. Pertinent data was obtained from the charts and radiologic studies.

Results
6 patients, (3F & 3M) were identified. All patients had diagnosis of hepatoblastoma made by CT scan and biopsy. Ages ranged from 16 months to 4 1/2 years. All patients received a minimum of 4 and a maximum of 7 cycles of neo-adjuvent chemotherapy. There were no intra-operative complications and all of the patients tolerated the operation well. One patient had tumor rupture prior to the operation. 6 of 7 patients are currently alive 1-7 years post resection. One patient had recurrence in the caudate a year later which was resected but subsequently died from metastatic disease. This particular patient had extensive disease with lung nodules prior to primary resection. She underwent 4 cycles of neo-adjuvent chemotherapy with a good response and therefore underwent resection. The AFP levels in all patients decreased to normal levels except for the one case of mortality. The surviving patients are in good health; two require hearing aids due to the side effects of chemotherapy.

Conclusion
Central hepatic resections are technically challenging but feasible and safe. Clear margins can be achieved with adequate resections and outcomes are good. This approach offers a reasonable option to transplantation in appropriately selected patients.

Notes:
P6
CONGENITAL LUNG ANOMALIES: CAN WE POSTPONE RESECTION?
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Vanderbilt Children’s Hospital, Nashville, TN, USA

Background/Purpose
Congenital lung anomalies encompass a spectrum of rare lesions that may be asymptomatic or regress, but can result in respiratory distress, recurrent infection, and an increased risk of malignancy. Resection is generally recommended, but this decision must consider the likelihood of adverse events with and without operation. The purpose of this study was to review a 10-year experience with the management and outcomes of congenital lung anomalies at a single institution.

Methods
With IRB approval, the Pediatric Surgery database was screened using ICD-9 and CPT codes corresponding to lung anomalies and operative resection from January 2000 to December 2009. Medical records were reviewed for patient demographics, type of lung lesion, symptoms, and complications of surgery or the disease.

Results
Of ninety-one patients, 56% were male and 44% were female with a mean gestational age of 36.9 weeks. The initial diagnosis included congenital cystic adenomatoid malformation (59.3%), pulmonary sequestration (13.2%), congenital lobar emphysema (13.2%), and bronchogenic cyst (12.1%). When symptomatic, patients presented with shortness of breath (3.3%), cough (4.4%), and recurrent respiratory infection/pneumonia (14.3%). Nineteen patients were observed, three became symptomatic with constitutional respiratory symptoms, and resection was performed in fourteen. Only one patient had complete regression. Eighty-three patients (91.2%) underwent resection at an average of 17.4 months. Sixty-seven procedures were performed open, 12 thoracoscopic, and 3 were converted to open. Pathology confirmed the diagnosis in all but 22 cases (26.5%), for which imaging proved to be incorrect or mischaracterized hybrid lesions. Postoperative complications included pneumothorax (4.8%), chylothorax (1.2%), empyema (1.2%), and pneumonia (1.2%). There was no incidence of malignancy or death.

Conclusion
Our data showed that no serious pulmonary complications occurred during the observational period, and thus, this raises the possibility of further postponing surgical treatment for asymptomatic patients.

Notes:
P7
IS DAILY DILATATION NECESSARY AFTER SURGERY FOR HIRSCHSPRUNG DISEASE AND ANORECTAL MALFORMATIONS?
Sara Temple, MD, Jacob C. Langer, MD
Hospital for Sick Children, Toronto, ON, Canada

Purpose
Most surgeons recommend daily dilatation after surgery for Hirschsprung disease (HD) and anorectal malformations (ARM). However, this may cause significant distress for parents and children, and there is also a theoretical risk of inexperienced parents disrupting the anastomosis or causing perforation. We aimed to determine the efficacy or potential harm of this practice.

Methods
Retrospective chart review of all children undergoing HD or ARM repair over 5 years. Patients with long-segment HD or low anorectal malformations, and those with inadequate followup data were excluded. Groups were compared using chi-square and t-tests, with a p value < 0.05 considered significant.

Results
There were 95 patients; 34 had HD and 61 had ARM. After an initial postoperative period of 1-2 weeks, 65 underwent routine dilatation by parents, and 30 underwent weekly calibration by the surgeon, with daily dilatation by the parents only if the anastomosis was felt to be narrow. Groups were similar with respect to age and weight at operation, gender, incidence of associated chromosomal and structural anomalies, and use of a preliminary stoma or laparoscopic approach. Of the 30 children undergoing weekly calibration, only 3 (10%) developed narrowing which required conversion to the daily parental dilatation approach. These children were kept in the weekly calibration group for analysis of outcomes. There were no significant differences between the two approaches, as shown below:
## Conclusions
Weekly calibration by the surgeon is associated with similar outcomes to daily dilatation by the parents. Because this approach is kinder to the parents and the child, it should be seriously considered for the postoperative management of children with HD or ARM.

## Notes:
CIRCULATING THYROTROPIN RECEPTOR MRNA FOR EVALUATION OF THYROID NODULES AND SURVEILLANCE OF THYROID CANCER

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Purpose

Thyrotropin receptor (TSHR) mRNA is a novel blood test for evaluation of thyroid nodules and follow-up of thyroid cancer. It has not been evaluated in children.

Methods

21 patients (range 12-17 years, median 16 years; 20 female), identified from a prospective IRB-approved registry, had 39 TSHR mRNA measurements by quantitative RT-PCR to evaluate thyroid nodules and/or thyroid cancer recurrence. Thyroidectomy was performed in 17 patients. Fine needle aspiration (FNA) cytology was performed on 10 patients.

Results

TSHR mRNA was measured pre-operatively in 9 patients (4 cancer with 1 false negative; 5 benign with 2 false positives). Of 4 non-operative patients, 3 had FNA (2 negative and 1 elevated THSR mRNA) and 1 was followed clinically (negative TSHR mRNA). Post-thyroidectomy surveillance of 12 patients with thyroid cancer showed that TSHR mRNA:

(1) Confirmed clinical assessment of disease after initial or re-operative surgery in all 12 patients (100%; 11 patients cured and TSHR mRNA undetectable; 1 not cured, confirmed by nuclear scan, and TSHR mRNA remains elevated consistent with residual disease).

(2) Was concordant with thyroglobulin in 14 of 19 (73%) measurements. In 3 of 19 (16%), TSHR mRNA was the only blood test useful for disease assessment because of elevated anti-thyroglobulin antibodies.

(3) Identified residual disease in 4 patients (confirmed by surgery in 3 and nuclear scan in 1).

(4) Was falsely negative in 2 and was the only sign of recurrence in 1.

Overall, for 39 determinations, TSHR mRNA as a measurement for thyroid cancer demonstrated the following characteristics, comparable to its performance in adults: sensitivity 73%, specificity 82%, positive predictive value 62%, negative predictive value 88%, and accuracy 79%.
Conclusion
Thyrotropin receptor mRNA provides a complementary evaluation to thyroglobulin and FNA for thyroid cancer. It may also assist in pre-operative management of thyroid nodules in pediatric patients.

Notes:
P9
PROGRESSION-FREE SURVIVAL IN WELL-DIFFERENTIATED THYROID CANCER IN PEDIATRIC AND ADOLESCENT PATIENTS
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Purpose
We previously reported our experience, from 1949-1986, with well-differentiated thyroid cancer in pediatric patients. This study reviews our experience from the past 2 decades in order to identify changes in predictors of outcome.

Methods
With IRB approval, we retrospectively identified 145 consecutive pediatric patients with well-differentiated thyroid cancer at our institution between 1990 and 2009. Our primary outcome was 5-year progression-free survival. We also examined possible risk factors, including age, neck radiation, histology, stage, size, nodal involvement, metastases, isthmus involvement, type of thyroid surgery, extent of lymph node dissection, and 131Iodine therapy, for progression or recurrence of disease.

Results
The median age at diagnosis was 18 years (range, 3.6-22.0), and 75% of patients were female. A family history was positive in 14%, and 5% received prior neck radiation. Fine needle aspiration was performed in 79% of patients; 81% of which were definitive. Papillary carcinoma made up 97% of tumors, while 3% were follicular. Regional lymph nodes were involved in 60% at diagnosis, and 8% had lung metastases. Extrathyroidal invasion was present in 44% of patients, and 31% had bi-lobar disease. There were no mortalities. The 5-year progression-free survival (±SE) was 78.7% (±4.0). Extrathyroidal invasion, multifocality, and positive lymph nodes were associated with a higher rate of progression (p=0.003, p=0.013, p=0.023). Age at diagnosis, radiation, size, histology, stage, metastases, and extent of lymph node dissection were not significant predictors of outcome. Isthmus involvement, 131Iodine therapy, and total thyroidectomy were strongly associated with more extensive disease, confounding analysis of their impact on progression-free survival.

Conclusions
Our data, once again, suggest an excellent prognosis of well-differentiated, pediatric thyroid cancer. Extrathyroidal extension seems to be the most important predictor of prognosis. There is a strong correlation between extent of disease and therapeutic intervention.

Notes:
P10
PREDICTORS OF SURVIVAL IN CHILDHOOD AND ADOLESCENT CUTANEOUS MELANOMA
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Memorial Sloan-Kettering Cancer Center, New York, NY, USA

Purpose
Because of the rarity of reports of pediatric and adolescent cutaneous melanoma, we analyzed our institutional experience to assess survival, prognostic variables, and effect of regional lymphadenectomy.

Methods
With IRB approval, we retrospectively reviewed 161 consecutive pediatric patients with cutaneous melanoma treated at our institution between 1980-2010. We evaluated the effect of age at diagnosis, American Joint Committee on Cancer stage, depth of invasion, nodal involvement, and distant metastases on overall survival. Outcome was also determined in the subgroups of patients with melanoma in-situ and spitzoid melanoma. Finally, we analyzed the prognostic effect of regional lymphadenectomy.

Results
Median age at diagnosis was 19 years (range, 5.3-22.0), and 59% of patients were female. Patients presented with growth of a mole (41%), a new mole (39%), change in color of a mole (30%), bleeding (17%), ulceration (17%), and pruritus (12 %). Stage distribution was: 51% (stage 1); 27% (stage 2); 16% (stage 3); and 6% (stage 4). Sentinel lymph node biopsy was performed in 62 patients, 14 of whom had positive lymph nodes identified. Median follow-up time was 4.5 years (range, 0.04-28.8 years), and overall 5-year survival (±SE) was 75.9% (±4.1). Predictors of survival using univariate analysis included stage (p=0.002), depth of invasion (p=0.002), regional nodal involvement (p=0.004), and presence of metastatic disease (p<0.0001) at diagnosis. Age at diagnosis did not affect outcome (p=0.4). There were no deaths in patients with melanoma in-situ (n=11) and spitzoid melanoma (n=22). The use of regional lymphadenectomy was strongly correlated with nodal involvement and depth, which confounded analysis as a prognostic variable.

Conclusions
American Joint Committee on Cancer stage and its components were predictors of outcome. Melanoma in-situ and spitzoid melanoma were associated with 100% survival in our analysis. Our data suggest that the role of regional lymphadenectomy should be prospectively evaluated in this cohort.

Notes:
POSTER SESSION 1

P11
AGE PREDICTS MONOTHERAPY IMMUNOSUPPRESSION IN PEDIATRIC LIVER TRANSPLANT RECIPIENTS AT ONE YEAR POST TRANSPLANT
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Purpose
The goal in managing pediatric liver transplant recipients is to minimize immunosuppression as soon and as safely as possible. There are no standard guidelines for immunosuppression minimization although more rapid steroid tapering in the first year post transplant has been accepted by most centers. We chose to retrospectively characterize those children who have been able to achieve stable steroid free monotherapy immunosuppression at 1 year post liver transplant. We hypothesized that analysis of these patients might suggest patient groups or characteristics which would correlate with less immunosuppressive needs.

Methods
After obtaining IRB approval we performed a retrospective review of liver transplant recipients at our institution from 2000-2010. We defined monotherapy immunosuppression as a child being on either tacrolimus or sirolimus and off of steroids. We then analyzed clinical parameters for statistical significance in predicting steroid free monotherapy immunosuppression at 1 year post transplant. Statistical tests were two tailed with significance set at p<0.05.

Results
We identified 81 patients that underwent liver transplantation at our institution with available follow up data. Among the many studied variables, age at transplant was statistically significant for predicting successful steroid free monotherapy immunosuppression at 1 year post transplant (p<0.03). The median age of patients on monotherapy (n=51) at 1 year post transplant was 2 years and of patients on more than one drug (n=30) was 8 years. The younger the age at liver transplant, the greater the likelihood of achieving monotherapy immunosuppression with 68% of children < 2 years old and 82% of children ≤ 6 months old on monotherapy.
Conclusions
Younger liver transplant recipients have a statistically significant higher chance of achieving steroid free monotherapy immunosuppression at 1 year post transplant. This subset of patients should be further studied as candidates for immunosuppression reduction.

Notes:
P12
THE ROLE OF SURGICAL MANAGEMENT FOR CHRONIC ITP: A COST ANALYSIS OF SPLENECTOMY VERSUS MEDICAL MANAGEMENT

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1Baylor College of Medicine, Houston, TX, USA, 2Michael E. DeBakey Department of Surgery, Baylor College of Medicine, Houston, TX, USA, 3Department of Pediatrics, Baylor College of Medicine, Houston, TX, USA

Purpose
Indications and timing for splenectomy in pediatric chronic idiopathic thrombocytopenic purpura (cITP) are controversial due to high spontaneous remission rates and concern for overwhelming post-splenectomy infection (OPSI). The objective of this study was to assess the risks/costs and benefits of medical versus surgical intervention for children with cITP.

Methods
After IRB approval, medical records for all children with cITP who underwent splenectomy from 2002 through 2009 at our institution were retrospectively reviewed (n=24). Two children did not meet our inclusion criteria. Pre- and postoperative data was collected. Medical and surgical costs were calculated based on pharmacy charges per dose and hospital charges, respectively. Data were analyzed with D’Agostino-Pearson test for normality and a Wilcoxon test performed to determine significance between groups. A p-value < 0.05 was considered significant.

Results
The median age at diagnosis was 11 years (3-16). Medical management included steroids (n=21), IVIG (n=19), Anti-D antibody (n=19), or a combination of these therapies (n=22). Nineteen patients (86%) reported side effects from medical therapy. Median age at splenectomy was 13 years (6-18) and time to surgery was 23 months from diagnosis (6-104). Splenectomy significantly increased platelet counts from 27K to 380K post-operatively (p<0.0001). Five children (23%) experienced post-operative complications, including OPSI following a dog bite (n=1) and undiagnosed fever (n=4). Following splenectomy, 7 patients (35%) temporarily relapsed after minor viral infections necessitating additional medical therapy. At last follow-up (15 months, 1-79), 19 patients (86%) were asymptomatic with platelet counts >50K. Two of the three symptomatic children were diagnosed with distinct auto-immune syndromes. Cost of splenectomy was significantly less than the cost of IVIG/Anti-D antibody therapy ($20,803 vs. $146,284, p<0.0002).

Conclusions
Earlier surgical consultation for children with cITP may be justified given the high success rate, low morbidity and cost differential of splenectomy.
POSTER SESSION I

Notes:
P13
LONG-TERM NEURODEVELOPMENTAL OUTCOMES IN CHILDREN BORN WITH GASTROSCHISIS
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\textsuperscript{1}University of Nebraska/Children’s Hospital and Medical Center, Omaha, NE, USA, \textsuperscript{2}University of Nebraska - Department of Education and Child Development, Munroe-Meyer Institute, Omaha, NE, USA

Purpose
We evaluated 2-year neurodevelopmental outcomes in children with gastroschisis.

Methods
We reviewed the records of children with gastroschisis treated by one surgical practice between August 2001 and July 2008. Children discharged from the NICU were referred to the state-sponsored Developmental Tracking Infant Progress Statewide (TIPS) program. We reviewed TIPS screening assessments performed before age 2. Local school districts evaluated children referred by TIPS and determined their eligibility for early intervention services. We defined poor outcomes as a score of “failure” or “moderate/high risk” on the screening assessment or enrollment in early intervention services by 2 years. Children born with gastroschisis were compared to case-matched nonsurgical, nonsyndromic TIPS children of similar gestational age and birthweight. To rule-out selection bias, we also compared characteristics of gastroschisis children in the TIPS database with gastroschisis children that did not follow-up with TIPS. Chi-square and t-tests were used for statistical analysis.

Results
105 children were born with gastroschisis. 46 followed up with TIPS. Characteristics were similar between children that followed up with TIPS and children that did not. There was no statistically significant difference in performance on screening assessments or in the rate of enrollment in early intervention services between the gastroschisis children and matched controls.

Conclusions
Children born with gastroschisis have similar 2-year neurodevelopmental outcomes as nonsurgical, nonsyndromic NICU children of similar gestational age and birthweight. Both groups of children have a higher rate of enrollment in early intervention than their healthy peers, whose enrollment rate is historically below 2 percent. These data suggest neurodevelopmental outcomes in gastroschisis children are delayed secondary to prematurity rather than the presence of the surgical disease.
POSTER SESSION I

Comparison of neurodevelopmental outcomes of gastroschisis children and matched controls

<table>
<thead>
<tr>
<th></th>
<th>Gastrochisis Patients (n=46)</th>
<th>Matched Controls (n=46)</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor Performance on Screening</td>
<td>23.9%</td>
<td>39.1%</td>
<td>NS</td>
</tr>
<tr>
<td>Enrollment in Early Intervention Services</td>
<td>15.2%</td>
<td>13.0%</td>
<td>NS</td>
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</table>

Notes:
P14
TAKING THE GOOD WITH THE BAD: CHANGES IN GASTROSCISIS MANAGEMENT OVER THE LAST DECADE ARE ASSOCIATED WITH BOTH BENEFITS AND SHORTCOMINGS
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Riley Children’s Hospital, Indianapolis, IN, USA

Purpose
Techniques employed for closure of gastroschisis have largely shifted from early primary closure to placement of a preformed silo and delayed closure. The purpose of this study was to identify how closure techniques have changed over time and how this has affected outcomes.

Methods
Following IRB approval, records of patients undergoing gastroschisis closure at a tertiary referral children’s hospital from 2000-2009 were reviewed. Patient characteristics and clinical outcome data were collected and compared among patients undergoing primary closure versus preformed silo placement. Outcomes were also compared in an era when primary closure was the dominant technique (2000-2002) versus an era when application of a primary silo predominated (2003-2009).

Results
From 2000-2009, 203 patients underwent gastroschisis closure (Table 1). Primary closure was performed in 50% of patients from 2000-2002 versus 12.3% from 2003-3009 (P<0.0001). Pre-formed silos were placed in 34.7% of patients from 2000-2002 versus 84.4% of patients from 2003-2009 (P<0.0001). Rates of perinatal intestinal ischemia and/or atresias were similar between both time periods and both closure techniques. Patients treated from 2000-2002 experienced shorter ICU stay, reduced ventilator days, and shorter time to achievement of full enteral nutrition (P<0.05). Patients treated from 2003-2009 experienced fewer post-operative ventral hernias and wound infections (P<0.05). Patients undergoing early primary closure developed ventral hernias at higher rates compared to those treated with a preformed silo (P=0.0006). ICU stay was longer for patients receiving preformed silos (P=0.002).

Conclusion
We conclude that change in our management strategy over the last decade has resulted in slightly prolonged ICU stay, ventilator days, and time to full feeds, but a reduction in post-operative hernias and wound infections.
# POSTER SESSION I

Table 1. Comparison of outcomes by time period and method of closure

<table>
<thead>
<tr>
<th>Method of Closure</th>
<th>2000-2002 (n = 49)</th>
<th>2003-2009 (n = 154)</th>
<th>P</th>
<th>Primary Closure (n = 43)</th>
<th>P</th>
<th>Preformed Silo (n = 147)</th>
<th>P</th>
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</thead>
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<tr>
<td>Primary Closure</td>
<td>24 (50%)</td>
<td>19 (12.3%)</td>
<td>&lt; 0.0001</td>
<td>18 (41.9%)</td>
<td>0.0006</td>
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<tr>
<td>Preformed Silo</td>
<td>17 (34.7%)</td>
<td>130 (84.4%)</td>
<td>&lt; 0.0001</td>
<td>25 (17%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hand-sewn Silo</td>
<td>8 (16.3%)</td>
<td>5 (3.3%)</td>
<td>&lt; 0.0001</td>
<td>3 (7%)</td>
<td>0.10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post-operative Ventr.</td>
<td>18 (36.7%)</td>
<td>30 (19.5%)</td>
<td>0.01</td>
<td>18 (41.9%)</td>
<td>0.0006</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wound Infection</td>
<td>14 (28.6%)</td>
<td>18 (11.7%)</td>
<td>0.005</td>
<td>3 (7%)</td>
<td>0.10</td>
<td></td>
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<tr>
<td>ICU Stay (days)</td>
<td>Median 12</td>
<td>Median 14</td>
<td>0.05</td>
<td>Median 9</td>
<td>0.002</td>
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<tr>
<td></td>
<td>(Range 2 - 92)</td>
<td>(Range 3 - 200)</td>
<td></td>
<td>(Range 2 - 128)</td>
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<td></td>
</tr>
<tr>
<td></td>
<td>Median 3</td>
<td>Median 14</td>
<td></td>
<td>Median 3</td>
<td></td>
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<td></td>
</tr>
<tr>
<td></td>
<td>(Range 0 - 128)</td>
<td>(Range 3 - 200)</td>
<td></td>
<td>(Range 0 - 100)</td>
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<tr>
<td>Ventilator Days</td>
<td>Median 6</td>
<td>Median 3</td>
<td>0.02</td>
<td>Median 4.5</td>
<td>0.06</td>
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<tr>
<td></td>
<td>(Range 0 - 22)</td>
<td>(Range 0 - 128)</td>
<td></td>
<td>(Range 0 - 128)</td>
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<tr>
<td>Days From Birth to Full Nutrition</td>
<td>Median 25</td>
<td>Median 31</td>
<td>0.04</td>
<td>Median 29</td>
<td>0.07</td>
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<td>(Range 12 - 365)</td>
<td>(Range 11 - 365)</td>
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<td>(Range 12 - 365)</td>
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Notes:
P15
COMPLICATIONS OF LAPAROSCOPIC PANCREATECTOMY FOR CONGENITAL HYPERINSULINISM OF INFANCY
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Purpose
Surgery for congenital hyperinsulinism of infancy (CHI) is required to treat patients with diffuse CHI unresponsive to medical treatment and for all children with focal CHI. The use of laparoscopy for pancreatic resection in CHI has recently been described. We aim to report the complications associated with laparoscopic pancreatectomy.

Methods
Ethical approval was obtained (09SG02). Data was extracted from a prospective database of all children treated with CHI in our Institution between 2004 and 2010. Data are reported as median and range. Statistical analysis was done using chi-squared test as appropriate. P-value<0.05 was taken as significant.

Results
Forty consecutive children had laparoscopic pancreatectomy for CHI; 27 diffuse, 12 focal and 1 mosaic type. Median age was 3 months (1-120). **Diffuse and mosaic CHI**: laparoscopic near-total pancreatectomy was attempted; 8 were converted to open, 1 was aborted due to ongoing pancreatitis and completion near-total pancreatectomy done 2 months later. **Focal CHI**: limited laparoscopic pancreatectomy was attempted with 1 conversion to open; there were 10 distal pancreatectomies (1 neck, 3 body, 6 tail lesions) and 2 subtotal pancreatectomies (1 neck lesion, 1 extensive focal lesion in body and tail). Drains were not used. Three patients with diffuse CHI sustained common bile duct injury (2 strictures and one division) requiring open choledocho-duodenostomy. One child with focal CHI had a postoperative pancreatic collection that resolved spontaneously. There were no splenic injuries, bowel injuries or deaths. At follow-up all children are well, tolerating bolus enteral feeding. Three children are diabetic and 5 require medical treatment for hypoglycaemia, all with diffuse CHI.
**Conclusion**
Laparoscopic pancreatectomy for focal CHI is successful with low risk of post-operative complications. Bile duct injury can occur after near-total pancreatectomy for diffuse CHI reflecting the extent of dissection required. Laparoscopic pancreatectomy should be performed in centres of advanced laparoscopic expertise.

**Notes:**
**Objective**

CDH is associated with significant morbidity and mortality. Although a number of prenatal predictors of severity, mortality, morbidity, and ECMO have been proposed no single parameter has been proven uniformly accurate. We developed the CDH-CPI to incorporate all known prognostics variables into a single composite index in order to improve prognostic accuracy. The purpose of this study is to examine the ability of the CDH-CPI to predict survival in left-sided CDH patients.

**Study Design**

A retrospective review of left-sided CDH patients at our institution between 2004-2010 was conducted. Ten prenatal parameters of the CDH-CPI were collected: 1) karyotype, 2) presence of syndromes, 3) congenital heart disease (CHD), 4) left ventricle-to-right ventricle ratio discordance (LV/RV), 5) Modified McGoon Index (MMI), 6) presence of a sac, 7) liver position, 8) lung-to-head ratio (LHR), 9) percent predicted lung volume (PPLV) and 10) total lung volume (TLV). Each parameter was scored as either +1 for a positive predictor or 0 for a negative predictor. Patients with significant congenital heart disease (CHD) such as double outlet right ventricle received a score of -1 in the CHD parameter. Study exclusions include lethal anomalies/syndromes and hypoplastic left heart. A total score was tabulated and patients stratified according to total score and survival. Data expressed as mean±SEM.

**Results**

Out of a total of 73 cases of CDH, 64 patients with a prenatal diagnosis of left CDH were identified with all CDH-CPI parameters. There were 3 intrauterine fetal demises (4.6%). Patients with a left-sided CDH and a CDH-CPI score of 8 had a significantly higher percent survival than patients with a CDH-CPI score 8 (chi-square, 86% vs. 39%, p=0.0001). Statistical analysis was performed on the CDH-CPI scores of survivors and non-survivors and was found to be significantly different (7.95±0.201 vs. 6.36±0.339, p=0.000065).
POSTER SESSION I

Conclusion
The CDH-CPI accurately stratifies survival in left-sided CDH. The amalgamation of 10 prenatal parameters of the CDH-CPI may be a better prenatal predictor than any single currently used predictor.

Notes:
POSTER SESSION II

Basic Science and Fetal Surgery
Sunday, May 22, 4:15 – 6:00 p.m.

P17
HEPATOCYTE GROWTH FACTOR AND OMEGA-3 ENRICHED FEEDS
HAVE A SYNERGISTIC EFFECT ON MUCOSAL MASS IN AN ANIMAL
MODEL OF INFLAMMATORY BOWEL DISEASE
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Purpose
Hepatocyte growth factor (HGF) has been shown to increase mucosal mass
and microvascular density and decrease inflammatory markers and apopto-
sis in an animal model of inflammatory bowel disease (IBD). Luminal Omega-3
(OM-3) has been shown to be anti-angiogenic, reduce inflammation and may
be protective against Crohn’s disease. This study was designed to evaluate the
potential synergistic effect of HGF and OM-3.

Methods
This study was performed under IACUC protocol #17714. Twenty adult female
transgenic HLA-B27 rats were divided into four groups based on the type of rat
chow (with or without OM-3) and the content of their subcutaneous osmotic mi-
nipump (with or without HGF). Group 1: Regular rat chow, pump-saline. Group
2: OM-3 enriched feeds, pump-saline. Group 3: Regular rat chow, pump-HGF
150 ug/kg/day. Group 4: OM-3 enriched feeds, pump-HGF 150 ug/kg/day. All
rats were sacrificed at 14 days after pump placement. Small and large bowel
mucosa was harvested and DNA and protein were extracted. Mucosal DNA
content (ug/mg mucosa) was measured at 260 nm with purity measured by ab-
sorbance ratio 260nm/280nm and protein content (ug/mg mucosa) was mea-
sured using the Bradford Assay (Bio-Rad, Hercules, CA). Statistical analysis
was done by ANOVA with post-hoc Tukey’s HSD test (Instat 3, GraphPad, San
Diego, CA).

Results
Content of protein and DNA in the ileum were significantly increased by sup-
plementation of HGF (p<0.001, p<0.01, respectively) alone. OM-3 significantly
increased protein content but not DNA (p=0.02, p=0.3, respectively). However,
combined they had a synergistic effect greater than either supplement alone
(p=0.0001, p=0.002, respectively) in comparison to controls. In the colon HGF
and OM-3 did not significantly increase protein or DNA content individually or
together.
Conclusions
This is the first demonstration of the synergistic effect of the combination of a growth factor (HGF) and a dietary supplement (OM-3) in an immunologic model of IBD.

Notes:
P18
TIE2 INHIBITION RESTRICTS VASCULAR BASEMENT MEMBRANE REMODELING
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Purpose
Antagonism of vascular endothelial growth factor (VEGF) is a clinically validated cancer strategy, yet most patients eventually develop progressive disease. Experimental models and clinical observation suggest that VEGF blockade leads to activation of alternative proangiogenic pathways, including angiopoietin/Tie2 signaling. Acquisition of perivascular stroma (including vascular basement membrane, VBM) is a prominent feature of both recurrent tumors and those exposed to Tie2 agonists. We hypothesized that blockade of Tie2 would disrupt VBM remodeling in experimental tumor vasculature.

Methods
All animal experimentation was conducted in accordance with our IACUC-approved protocol. Human Ewing’s sarcoma (SKNEP1) cells were transfected with a luciferase reporter gene and either soluble Tie2 (sTie2) or control plasmids. Tumor xenografts were intrarenally implanted in nude mice (controls: n=6; sTie-expressing: n=12). Primary tumor growth was weekly quantified in vivo using bioluminescence, and animals killed at a threshold of 109 photons/second. Tumors were harvested and tissues analyzed by immunohistochemistry.

Results
Final tumor mass was equivalent between sTie2 (2.04±0.4 g) and control (2.32±0.4 g) groups (p=NS). Secreted sTie2 resulted in a 47% decrease in endogenous Tie2 activation by quantitative western blot, yet sTie2-tumors demonstrated no difference in endothelial density (p=0.26). Similarly, vascular mural cell and macrophage recruitment did not alter. However, remodeling of basement membrane was strikingly perturbed in sTie2-tumors, with reduced collagen-IV-enveloped vessels. In particular, sTie2 appeared to reduce branching, with consequent limitation of small vessel development.
Conclusions
Inhibition of Tie2 impairs VBM patterning in our model. This feature of tumor angiogenesis functions to stabilize perfusion, and is prominent in tumors that acquire resistance to VEGF blockade. Thus, our data suggest a novel function for angiopoietin/Tie2 signaling in patterning of perivascular matrix, and are consistent with prior studies implicating Tie2 activation in the development of resistance to VEGF inhibitors. Further investigation of combined VEGF/Tie2 targeting effects may be warranted.

Notes:
P19
THE PRO-INFLAMMATORY EFFECTS OF TOLL-LIKE RECEPTOR AGONISTS ARE COUNTERED BY INTERLEUKIN 10 PRODUCTION IN A DOSE-RESPONSE FASHION.
Ryan M. Walk, MD, Steven T. Elliott, MD, Jason A. Snyder, MD, Felix C. Blanco, MD, Stanislav Vukmanovic, MD, Anthony D. Sandler, MD
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Purpose
Toll-like receptor (TLR) agonists have potential utility as cancer vaccine adjuvants through stimulation of antigen-presenting cells (APCs) and subsequent amplification of the cytotoxic T-cell response. Prior in-vitro studies in our laboratory, however, have demonstrated macrophage interleukin-10 (IL-10) release upon exposure to TLR agonists, with consequent inhibition of this cytotoxic response. The purpose of this study was to evaluate markers of the pro- and anti-inflammatory cytokine response in TLR stimulated macrophages.

Methods
Murine primary bone marrow derived macrophages were harvested and exposed for 24 hours to varying concentrations of known TLR agonists: OK-432 (TLR 2/4), polyinosinic:polycytidylic acid (Poly I:C) (TLR3) and CpG oligonucleotide (CpG) (TLR9). Macrophage cytokine secretion was measured for the pro-inflammatory cytokine, Tumor Necrosis Factor Alpha (TNF-α), and the inhibitory cytokine, IL-10, via ELISA. Mice were handled according to Institutional Animal Care and Use Committee (IACUC) protocol and IACUC approval for the study was obtained.

Results
Exposure of Macrophages to varying concentrations or combinations of TLR agonists led to secretion of both TNF-α and IL-10 in a consistent dose-response curve. Irrespective of the TLR agonist used, dose-response kinetics were characterized by increasing production and eventual plateau of the cytokine measured. Notably, production of IL-10 lagged behind the production of TNF-α, and an optimal pro-inflammatory dose was identified for each TLR agonist.
Conclusions
At lower concentrations, Toll-like receptor agonists stimulate macrophages to induce a pro-inflammatory response, while at higher concentration this response is countered by interleukin-10 secretion. The transition from stimulation to inhibition occurs over a relatively narrow range of Toll-Like receptor agonist concentration. This observation is critical as inappropriate concentrations or combinations of the Toll-like receptor agonists will suppress the desired immune response for vaccine therapy. Strategies designed to either ablate or constrain macrophage interleukin-10 production are essential for Toll-Like receptor agonists to have clinical utility as vaccine adjuvants.

Notes:
P20
INHIBITION OF FOCAL ADHESION KINASE IN HEPATOBLASTOMA CELLS LEADS TO DECREASED CELL SURVIVAL
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Introduction
Hepatoblastoma is the most common liver cancer in children. It continues to have a poor prognosis with disease free survival rates less than 50% for advanced stage, metastatic and relapsed disease. Focal adhesion kinase (FAK) is a cell signaling molecule that is upregulated in a number of human tumors, including hepatocellular carcinoma. FAK modifies numerous cell signaling pathways to increase the survival, proliferation and invasion of tumor cells. Its inhibition leads to decreased tumor cell survival. Currently, there are no studies examining FAK in hepatoblastoma. We hypothesize that FAK will be expressed by hepatoblastoma tumor cells and that FAK inhibition will decrease cellular viability, invasion and migration in hepatoblastoma cells.

Methods
HuH6 hepatoblastoma cells are cultured under standard conditions. Total FAK expression and FAK (Y397) phosphorylation is detected with immunoblotting. Inhibitory molecule, 1,2,4,5-benzenetetraamine tetrahydrochloride (Y15), is used to block FAK at varying concentrations and time points. Cell viability is measured using alamar blue assays. Cellular invasion and migration are evaluated with standard assays. Data are compared with Student’s t-test or ANOVA as appropriate, reported as mean ± SEM, and statistical significance determined at P ≤ 0.05.

Results
HuH6 cells exhibit strong FAK expression with significant phosphorylation. FAK inhibition with Y15 (2.5 μM) results in a significant decrease in cell viability (Graph). In addition, treatment with Y15 at 2.5 μM leads to decreased cellular invasion by 20%. Finally, migration of HuH6 cells is significantly decreased after Y15 (2.5 μM) treatment (100% ± 3.3% vs. 76.6% ± 2.3%, p = 0.03, control vs. Y15).

Conclusions
The hepatoblastoma cell line, HuH6, expresses FAK. FAK inhibition in these cells results in a significant decrease in cell survival, invasion and migration. These novel findings suggest that targeting FAK may be a potential therapeutic strategy for hepatoblastoma and is being further investigated.
Notes:
P21
POTENT ALLOGENEIC T-CELL RESPONSES TO MURINE NEUROBLASTOMA
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Purpose
Current immunotherapy strategies fail to elicit effective immunity against established neuroblastoma (NB). The lack of effective immunity is most likely a result of tumor adaptation and immune tolerance. Immune tolerance to syngeneic T-cells led us to hypothesize that, under appropriate circumstances, allogeneic recognition of tumor antigens will induce a more effective T-cell response that is not subject to tumor tolerance. This study was designed to determine whether allogeneic cytotoxic T-cells could be induced against NB in vitro for ultimately treating murine NB.

Methods
After IACUC approval, primary mouse bone marrow macrophages were harvested and loaded with three different tumor antigen preparations: heat shocked NB lysate, proteinase digested NB lysate and Survivin peptide (a tumor specific peptide). Antigen loaded macrophages were subsequently cultured with syngeneic (H-2Kd) or allogeneic (H-2b) T-cells. The T-cells were re-stimulated with antigen and the effector response to tumor and antigen was measured by IFN-γ production.

Results
Syngeneic T-cells failed to respond to tumor antigen, while allogeneic effector T-cells induced a robust immune response to tumor associated antigens. Re-stimulation of primed allogeneic T-cells went on to induce a remarkable response to live tumor cells. Surprisingly, non-specific immunity was not observed in this model when allogeneic T-cells were cultured with unprimed macrophages.

Conclusions
We conclude that potent immune responses can be induced by priming allogeneic T-cells against NB. The lack of non-specific immunity observed in this model, portends well to the adoptive transfer of these T-cells into allogeneic hosts for the treatment of NB. These findings may pave the way to using unrelated donor T-cells for the treatment of NB.

Notes:
P22
HYPOXIA INDUCIBLE FACTOR-1α AT THE INTERFACE BETWEEN DRUG RESISTANCE AND TUMOR INVASIVENESS IN NEUROBLASTOMA
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Purpose
Neuroblastoma drug resistance is frequently associated with increased invasiveness and angiogenesis, but the mechanism underlying this association is poorly understood. The purpose of this study was to (1) assess the correlation between drug resistance and invasiveness and (2) investigate potential mediators of this effect.

Methods
Wild type (WT) cells from an n-myc amplified (Be(2)-C) and a non-amplified (SK-N-SH) line were exposed to progressively increasing concentrations of doxorubicin to generate drug-resistant (DR) cells. Invasiveness was compared between the WT and DR cells by clonogenic survival and in vitro invasion assay (BD Biocoat MatrigelTM Matrix). Potential molecular pathways mediating this effect were assessed by western blot analysis and RT-PCR.

Results
Compared to their WT counterparts, the in vitro invasiveness was increased 2-6 fold in the DR cell lines. Likewise, clonogenic survival was 3-fold higher. The DR cells had increased expression of hypoxia-inducible factor-1α (HIF-1α) and matrix metalloproteinases 2 and 9 (MMP-2 and MMP-9). Inhibition of all class II histone deacetylases (HDACs) by suberoylanilide hydroxamic acid (SAHA) or trichostatin-A treatment, or specific siRNA knockdown of HDAC6 (a member of the class II HDAC family) resulted in decreased HIF-1α expression and decreased in vitro invasiveness.

Conclusion
Neuroblastoma cells which develop resistance to doxorubicin also exhibit a more invasive phenotype. Hypoxia inducible factor-1α may be a key mediator of this overlap. Histone deacetylase inhibitors, which are known to reduce HIF-1α activity and enhance drug sensitivity, may also reduce tumor invasiveness.

Notes:
P23  
INHIBITION OF VASCULAR ENDOTHELIAL GROWTH FACTOR DIRECTS ABSORPTIVE LINEAGE DIFFERENTIATION OF THE INTESTINAL EPITHELIUM VIA NOTCH ACTIVATION

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Purpose

Notch is a key mediator in the terminal differentiation of the intestinal epithelium toward its absorptive lineage. Vascular endothelial growth factor, primarily known as an endothelial cell mitogen activates Notch via Dll4 ligand expression. We sought to determine the effect of Notch signaling on intestinal epithelial cell differentiation following mesenchymal specific reduction of vascular endothelial growth factor (VEGF).

Methods

Transgenic mice designed to express the soluble VEGF receptor, sFlt-1 ((Der-mo-1/rtTA/sFlt-1; loss of function) specifically in the mesenchyme were generated (N=15). Small intestine from the mutant and littermate control mice was resected. Serum VEGF levels were quantified with ELISA using the Bradford protocol. Paraffin embedded, formalin fixed sections were immunostained with Notch specific antibody using the standard dab protocol. Immunofluorescence was performed with primary antibodies specific for terminally differentiated epithelial cell types: villin, a marker for enterocytes (absorptive lineage) and Muc-2 a marker for goblet cells (secretory lineage). Each cell type per villus axis was quantified.

Results

There was a significant reduction in tissue VEGF levels in the mutant small intestine compared to littermate controls. Increased Notch positive nuclei were seen along the villus axis in the mutant small intestine while in the littermate control intestine, cell surface Notch staining with few areas of nuclei staining were noted. Quantification of the differentiated intestinal epithelium demonstrated a significant increase in the percentage of villin positive cells per villus axis compared to controls (85.2+/-4.9% vs. 68.0+/-4.0%; P<0.001). Conversely, we observed a significant decrease in the percentage of muc-2 positive cells per villus axis in our mutant small intestine compared to controls (14.8+/-4.9 vs. 32+/-4.0%; P<0.001).
**Conclusion**

Mesenchymal specific inhibition of VEGF in the intestine results in activation of notch signaling and terminal differentiation of the intestinal epithelium towards the absorptive lineage.

**Notes:**
P24
NOVEL ZEBRAFISH MODEL REVEALS CRITICAL ROLE FOR MAPK IN LYMPHANGIOGENESIS
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Purpose
Lymphatic disorders are poorly understood with few available animal models. Taking advantage of the transparent zebrafish embryo, we designed a novel assay to measure lymphatic development using transgenic zebrafish with fluorescently labeled endothelial cells. We focused on the two major branches of the vascular endothelial growth factor receptor (VEGFR) signaling pathway, the MAPK and PI3K pathways.

Methods
Direct visualization of lymphatic development was performed in control untreated embryos or under chemical inhibition. After a number of trials, we determined that a 6-hour pulse of inhibitor 3 days post fertilization (dpf) was ideal to measure lymphangioblast migration and thoracic duct formation. Fish were analyzed for the presence of the thoracic duct at 4 dpf (n > 30 specimens). Statistical analysis was performed using Chi squared and Wald tests.

Results
Thoracic duct formation was prevented using selective inhibitors against kinases (MAPK, PI3K/TOR, or VEGFR; p≤ 0.002 using Wald test, compared with controls). These targeted kinases were important for thoracic duct formation, as the duct failed to form in a majority of treated animals (Figure 1A). Remarkably, MAPK pathway inhibition most robustly reduced lymphangiogenesis (Figure 1A), demonstrated by the lack of lymphatic endothelial cells in the area of the forming duct (Figure 1B, C).

Conclusion
We conclude that MAPK pathway function downstream of the VEGFRs is crucial at the early stages of lymphatic vessel development. This study provides a novel animal model and a potential target pathway for further investigation. Owing to the conservation of vascular biology across all vertebrates, we suggest further examination of MAPK pathway deregulation as a potential mechanism underlying lymphatic disease in humans.

Notes:
POSTER SESSION II

P25
AMNIOTIC FLUID STEM CELLS CAN FUNCTIONALLY DIFFERENTIATE ALONG SMOOTH MUSCLE LINEAGE - POTENTIAL FOR REGENERATIVE MEDICINE

Marco Ghionzoli¹, Giulia Costanzi¹, Steven W. Shaw¹, Giorgia Totonelli¹, Massimo Garriboli¹, Andrea Repelle¹, Stelios T. Andreadis², Antonio Messineo³, Agostino Pierro¹, Simon Eaton¹, Paolo De Coppi¹

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Purpose
In order to create a biocompatible tissue to replace absent or damaged organ, the generation of a functional smooth muscle (SM) layer is essential. In our study we aimed to derive functional SM cells from human Amniotic Fluid Stem (hAFS) cells.

Methods
Amniotic cells obtained by amniocentesis (15 to 35 weeks of gestation) were immunoselected for c-kit. Clonal hAFS lines were generated by limiting dilution. Controls were cultured in optimal conditions (a-MEM containing 15%FBS 1%Glu, 1% Pen/Strep Chang supplement) whereas differentiation was obtained in cells maintained for 21 days in DMEM 15% FBS supplemented with 5ng/ml PDGF-BB and 2.5ng/ml TGF-b1. Quantitative PCR was used to evaluate aSMA, desmin and calponin expression. Immunofluorescence on cells was performed for a-SMA, desmin and smoothelin. A transmission electron microscopy (TEM) analysis was carried out on both cultures. Moreover, functionality was assessed using a validated collagen lattice functional assay. Lastly, two lentiviral vectors encoding ZsGreen and DsRed respectively under the aSMA and MHC promoter were used to transduce untreated hAFSC (T-hAFSC). T-hAFSC were analysed by fluorescence activated sorting in control and differentiating conditions.

Results
hAFSC driven toward SM lineage SMhAFSC expressed significantly higher level of aSMA, desmin and calponin transcripts after d7 of selective culture condition. These results were confirmed at immunofluorescence. Ultrastructural cell features of SMhAFSC seen at TEM confirmed increased intermediate filaments, dense bodies and glycogen deposits in comparison to controls. Functional assay evaluations demonstrated that SMhAFSC retain a higher contractility potential compared to controls. Genetically engineered cells confirmed up to 48% of SM commitment when submitted to differentiation conditions.
Conclusion
hAFSC under selective cultural conditions are able to give rise to a functional
SM cells. hAFSC might be an amenable to tissue engineer muscular layer of
hollow structures.

Notes:
P26
EXPRESSION OF NONHOMOLOGOUS END-JOINING PROTEINS IN NEURAL CREST CELLS AND NEUROBLASTOMA
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Purpose
Neuroblastoma originates from neural crest cells and acquires genomic aberrations that often correlate with clinical outcomes. Understanding the mechanisms of genomic instability can provide insights into tumor development. Nonhomologous end joining (NHEJ) of DNA double strand breaks generate chromosomal abnormalities through inaccurate repair. The finding that neuroblastoma cell survival requires Ku70, a protein of central importance in NHEJ, provided initial support for the model that NHEJ is tumorigenic in neuroblastoma. We then hypothesized that gene products required for NHEJ are expressed in neuroblastoma cell lines and in undifferentiated neural crest cells from which these tumors develop.

Methods
Neuroblastoma cell lines (neuroblastic and Schwannian) and normal fibroblasts were cultured. Human embryonic stem cells were induced to differentiate into neural crest cells and further into neural crest derived cells. Differentiated cells were sorted based on surface markers. Lysates were prepared and analyzed for RNA and protein expression of multiple genes with defined roles in NHEJ. Public data sets of RNA expression in neuroblastoma tumors were accessed. Expression data on DNA repair genes was correlated to survival using Kaplan-Meier survival analysis.

Results
Compared to fibroblasts and Schwannian-type cells, tumorigenic neuroblastic neuroblastoma tumor cell lines uniformly expressed higher levels of DNA-dependent protein kinase catalytic subunit (DNA-PK, ca. 4-fold elevation). Levels of DNA-PK RNA were higher in neural crest stem cells compared to adult tissues. DNA-PK expression decreased as neural crest cells differentiated in culture. Higher expression of DNA-PK was associated with poor overall survival (p<0.01).
Conclusion
DNA-PK, an important mediator in NHEJ repair of DNA double strand breaks, is found at high levels in neuroblastoma cells and in undifferentiated neural crest cells, the precursor of neuroblastoma. These data support a novel model in which NHEJ mediates neuroblastoma tumorigenesis. Since DNA-PK expression correlates with poor survival, ongoing work is evaluating the therapeutic potential of this target in neuroblastoma.

Notes:
P27
NOVEL MECHANISMS OF THE ANTI-INFLAMMATORY CYTOKINE IL-10 IN REGENERATIVE FETAL WOUND REPAIR

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Aims
Mid-gestation fetal skin heals scarlessly with high levels of hyaluronan (HA) in the extracellular matrix (ECM) and an attenuated inflammatory response. We previously demonstrated 1) elevated IL-10 levels in fetal skin, 2) the necessity of IL-10 in the formation of HA-rich pericellular matrices (PCM) by fetal fibroblasts (FFb), 3) IL-10-/- fetal wounds heal with scar, and 4) IL-10 induces postnatal wounds to heal scarlessly. Combining these data with accepted IL-10 signaling pathways, we hypothesize that IL-10’s regenerative effects are partially mediated by HA-rich ECM formation and regulation through induction of HA synthases (HAS1-3) via a STAT3-dependent mechanism.

Methods
Adult murine fibroblasts (AFb) were cultured with IL-10 (200 ng/ml) and assessed for 1) STAT3-phosphorylation by Western blot 2) HAS1-3 gene expression by qRT-PCR and 3) PCM production by particle exclusion assay. To identify the roles of STAT3 and HA synthases in PCM formation, AFb were transduced with either lentiviral STAT3-targeted shRNA or cultured with an HA synthase inhibitor (4-methylumbilliferone [4-MU]) and IL-10; HA formation was assessed. Control cells were not treated with IL-10. Data expressed as mean±SEM. Statistical analysis by t-Test or ANOVA.

Results
IL-10 significantly induced STAT3 phosphorylation at 1 hour (control 0.65±0.13 vs. treatment 1.25±0.126, p=0.03), HAS1 gene expression at 3hrs (control 0.95±0.065 vs. treatment 1.29±0.05; p=0.01), and HAS2 at 1,2,3 hours (control 0.98±.04 vs. treatment 1.19±0.4; p=0.02, control 0.96±.07 vs. treatment 1.35±0.4; p=0.007, control 0.88±.02 vs. treatment 1.22±0.06; p=0.007) but not HAS3 expression. AFb PCM is reduced compared to FFb (PCM area/cell area 1.84±0.08 vs. 2.78±0.14; p<0.001), however IL-10 treatment recapitulates the fetal HA-rich PCM phenotype in AFb (AFb+IL-10 2.74±0.24 vs. FFb2.79±0.35; p=NS). HA-rich PCM induction by IL-10 was attenuated in STAT3 knockdown AFb versus control (1.215±0.0283 vs. 2.781±0.138; p<0.001). Co-treatment of AFb with 4-MU+IL-10 significantly reduced PCM (1.33±0.04 vs. 2.78±0.14; p<0.001).
Conclusion
IL-10 has direct effects on formation of an HA-rich PCM via STAT3 signaling and HAS gene expression. These data may in part account for the function of IL-10 in the fetal regenerative response and suggest that IL-10 plays a unique role in the fetus, not as an immunomodulatory agent, but as a central regulator of the ECM.

Notes:
POSTER SESSION II

P28
TRACHEO-ESOPHAGEAL DISPLACEMENT INDEX (TEDI) AND PREDICTORS OF AIRWAY OBSTRUCTION FOR FETUSES WITH GIANT NECK MASSES

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Purpose
There are no established patient selection guidelines to determine which fetuses with giant neck masses may benefit from an EXIT procedure as the mode of delivery. The purpose of this study was to describe the tracheo-esophageal displacement index (TEDI), a novel, reproducible measurement of fetal airway displacement, and to correlate this measurement and other prenatal findings to the extent of airway obstruction at birth.

Methods
The prenatal course, fetal imaging, EXIT procedures, and postnatal outcomes of all fetuses with giant neck masses evaluated between 2001 and 2009 were reviewed. Each infant’s airway was categorized as uncomplicated (spontaneous respirations or easy intubation) or complicated (need for airway instrumentation or tracheostomy) based on clinical findings at birth. Prenatal variables such as tumor size, diagnosis, presence of polyhydramnios, and MRI-based TEDI were correlated with airway difficulty. Results were analyzed with Fisher’s exact test and ROC curves.

Results
There were 22 fetuses with a giant neck mass (10 lymphatic malformations; 9 teratomas; 2 hemangioendotheliomas; 1 cervical thymic cyst), diagnosed at 24.7±6.4 weeks’ gestational age, of which 21 had 27 fetal MRIs. One fetus with a teratoma developed hydrops and died in-utero, and 2 fetuses underwent pregnancy termination. Prenatal variables associated with a complicated airway at birth included TEDI (p= 0.01), teratoma diagnosis (p=0.04), tumor volume (p=0.03), and polyhydramnios (p=0.01). By ROC curve analysis, TEDI > 12 correlated strongly with complicated airway with an area under the curve of 0.85, 100% specificity and 72% sensitivity (p<.003). All fetuses classified with an uncomplicated airway (n=7) had a diagnosis other than teratoma and normal amniotic fluid volume.

Conclusion
In fetuses with giant neck masses, the presence of polyhydramnios, teratoma diagnosis, or TEDI > 12 are predictive of a complicated airway at birth. Our data suggest that fetuses without these findings may be delivered safely without an EXIT approach.

Notes:
P29
LONGTERM MORBIDITY AFTER FETAL ENDOSCOPIC SURGERY FOR SEVERE TWIN-TO-TWIN TRANSFUSION SYNDROME

Benjamin Kowitt, Debra Watson-Smith, RN, Richard Tucker, PhD, Christopher S. Muratore, MD, Barbara M. O’Brien, MD, Betty Vohr, MD, Stephen R. Carr, MD, Francois I. Luks, MD, PhD

Alpert Medical School of Brown University, Providence, RI, USA

Purpose
Twin-to-twin transfusion syndrome (TTTS) affects 10-15% of identical twin gestations and, if severe, leads to 80-100% dual mortality. The optimal treatment for severe disease is endoscopic laser coagulations, which improves outcome to 80% survival of at least one twin. Limiting surgery to the most severe forms has not increased mortality, while avoiding maternal and fetal risks of surgery in lower stages. The aim of this study is to analyze gestational age-stratified long-term morbidity in these patients.

Methods
Fetal laser ablation was performed on 60 mothers between 2000 and 2009. Fifty-one children survived beyond the neonatal period. We obtained long-term follow-up on 36 patients (a follow-up rate of 71%, including 100% of dual survivors). Full perinatal and updated pediatric records were obtained on all patients, and were compared with gestational age (GA)-matched twins and singletons in this institution’s neonatal follow-up database and recently published reviews. Sequelae were categorized by body system as major permanent, major resolved, minor, and perinatal.

Results
Forty percent of TTTS survivors had at least one major sequela, all but 13% of which were fully resolved at a median follow-up of 52 months (4.4 years). These included neurological (10%), infectious and muscular/skeletal (3%) morbidities. There were no permanent cardiac, gastrointestinal, genitourinary, renal or respiratory sequelae. All major complications were seen in patients born before 29 weeks GA. There were no significant differences in the incidence of temporary or permanent complications between this cohort of patients and GA-matched control patients.

Conclusions
At a median follow-up of more than 4 years, preliminary findings suggest that these children do not differ from non-operated twins and singletons. The only predictor of temporary or permanent sequelae is the degree of prematurity at birth.

Notes:
POSTER SESSION II

P30
FETAL ECHOCARDIOGRAPHY GUIDES INDICATIONS FOR SURGERY IN FETUSES WITH HYDROPS

Darrell L. Cass, MD, Oluyinka O. Olutoye, MD, PhD, Nancy Ayres, MD, Kenneth J. Moise, MD, Carolyn A. Altman, MD, Anthony Johnson, DO, Christopher I. Cassady, MD, David A. Lazar, MD, Timothy C. Lee, MD, Regina M. Lantin, MD

Texas Children’s Fetal Center, Houston, TX, USA

Purpose

Hydrops has been considered the primary indication for surgery in fetuses with vascular tumors (VT) or lung masses (LM), with the assumption that hydrops reflects underlying heart failure and impending fetal demise. The role of echocardiography in defining fetal heart function and heart failure in these patients has not been defined.

Methods

The prenatal course, outcomes, and imaging of all fetuses with VT and high-risk LM (CCAM-volume ratio>1.6 or findings of hydrops) evaluated since 2001 were reviewed retrospectively. Hydrops was defined as accumulation of fluid in ≥2 compartments. Abnormal echocardiography findings included increased/decreased cardiac output, elevated right heart pressure, chamber dilation and decreased contractility. Data were analyzed with Fisher’s exact test.

Results

Hydrops was identified in 48% (11/23) of fetuses with high-risk LM. Fetuses with hydrops and abnormal echocardiography (n=9) demonstrated poor survival without fetal surgery (11%) compared to 100% survival in fetuses with hydrops and a normal echocardiogram (n=3; p=0.02). Compared to hydrops, an abnormal echocardiogram had equal sensitivity (88%), but superior specificity (87%), positive (78%) and negative (93%) predictive value for mortality or need for fetal surgery. Of 23 fetuses with VT (11 sacrococcygeal and 9 cervical teratomas; 2 hemangioendothelioma; 1 lymphovenous malformation), hydrops was identified in 26% and abnormal echocardiogram in 61%. All fetuses with hydrops had an abnormal echocardiogram and either died or required fetal surgery. However, 84% of fetuses with abnormal echocardiograms alone survived without fetal surgery.

Conclusions

In some fetuses with LM hydrops may result from mass-related impaired lymphatic drainage rather than primary cardiac causes, and may not unilaterally predict mortality and the need for fetal intervention. Echocardiographic changes in these fetuses are the best predictors of mortality and need for fetal intervention. For fetuses with VT hydrops results from high-output cardiac failure and best predicts demise or need for fetal intervention.

Notes:
P31
METAGENOMIC ANALYSIS OF INTESTINAL BACTERIA FROM INFANTS WITH COMPLICATED NECROTIZING ENTEROCOLITIS
Valeriy Poroyko, PhD¹, Michael Morowitz, MD²
¹University of Chicago Pritzker School of Medicine, Chicago, IL, USA, ²University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

Purpose
The contribution of gut bacteria to the pathogenesis of NEC is poorly understood. New approaches to study bacteria in resected intestinal tissue have not yet been applied to study NEC. The goal of this study was to perform metagenomic analysis of mucosa-associated bacteria isolated from infants with NEC.

Methods
Diseased small intestine was collected from 3 infants undergoing bowel resection for NEC. Flow cytometry was used to isolate individual microbial cells from human lymphocytes and enterocytes. Bacterial DNA from each sample was amplified and sequenced on the Roche 454 pyrosequencing platform. Taxonomic assignments of the bacteria were made by analysis of DNA sequences encoding the 16S ribosomal gene. Assembled metagenomic sequences were annotated by performing a BLAST search against the NCBI non-redundant protein database, and the functional potential of the microbiome was evaluated with the MG-RAST tool for genome annotation.

Results
Pyrosequencing of bacterial DNA yielded over 412 Mb of genomic sequence data, with minimal contamination from eukaryotic DNA. The observed microbial communities were extremely simple (mean no. organisms per sample = 4) and were dominated by gram-negative members of the Enterobacteriaceae family. Sequences from Enterobacter, Klebsiella, and E. coli were most abundant. No clostridial sequences were identified. Annotation of gene sequences demonstrated that the gut microbiome in all NEC samples (see figure) was enriched with genes encoding for carbohydrate metabolism (e.g. fructose utilization) and bacterial virulence (e.g. antibiotic transporters and the type IV secretion apparatus).

Conclusion
This is the first reported culture-independent study of mucosa-associated bacteria in NEC. These data illustrate that intestinal tissue in NEC is colonized by a limited set of microorganisms, and that the genomes of these organisms are marked by high virulence potential. Future animal and clinical studies will be needed to elucidate how the specific organisms and genes identified contribute to NEC pathogenesis.
POSTER SESSION II

Notes:
P32
HEPATIC INFLAMMATION AND ALTERED HEPATOBILIARY TRANSPORTER EXPRESSION IN A MOUSE MODEL OF SMALL BOWEL BACTERIAL OVERGROWTH
Jaimie D. Nathan, MD, Bin Wang, MD, Lili Miles, MD, Joel E. Mortensen, PhD, Jorge A. Bezerra, MD
Cincinnati Children’s Hospital Medical Center, Cincinnati, OH, USA

Purpose
The pathogenesis of parenteral nutrition-associated liver disease in children with intestinal failure is poorly understood; however, small bowel bacterial overgrowth (SBBO) is believed to play an important role. Furthermore, there is evidence that gut-derived endotoxins may alter expression of hepatobiliary transporters, and altered transporter expression is associated with cholestatic liver injury. Aims of this study were to: (1) develop and characterize a novel mouse model of SBBO; and, (2) determine the impact of SBBO on expression of hepatobiliary transporter Mdr2.

Methods
We created a jejunal self-filling blind loop (SFBL) in C57BL/6 mice to induce small bowel bacterial overgrowth. Control mice underwent laparotomy. Three weeks later, at sacrifice, intraluminal bacteria and mesenteric lymph nodes were cultured. Serum transaminases, alkaline phosphatase, GGT, and bilirubin were measured. Liver was harvested for histological grading of injury and for quantification of hepatic Mdr2 gene expression by RT-PCR. Statistical analyses utilized Student’s t-test, χ²2 method, and Mann-Whitney test (significance at p<0.05).

Results
Creation of a jejunal SFBL induced a dramatic increase in aerobic and anaerobic intraluminal bacterial counts (4.63±1.66x10⁸ cfu/ml and 9.16±1.59x10⁸ cfu/ml, respectively), compared to controls (6.23±1.96x10² cfu/ml and 1.05±0.60x10⁴ cfu/ml, respectively; p<0.001). Mesenteric lymph nodes from SFBL mice grew bacteria in 10/10, compared to 1/11 from controls (χ²=17.4; p<0.0001). SFBL mice had significantly elevated serum alkaline phosphatase (163.9±14.4 IU/L vs. 102.5±8.0 IU/L; p=0.005), as well as significantly higher histological scores for cholangitis, vasculitis, and hepatocellular injury parameters. Consistent with a gut-liver axis, hepatic expression of Mdr2 in SFBL mice was suppressed, displaying 41% lower levels, compared to controls (p<0.05).
Conclusions
Small bowel bacterial overgrowth and bacterial translocation in this model results in biochemical and histological evidence of liver injury, and reduced hepatic expression of hepatobiliary transporter gene Mdr2. This mouse model may help to elucidate the mechanism of liver injury induced by small bowel bacterial overgrowth in patients with parenteral nutrition-associated liver disease and other cholangiopathies.

Notes:
Clinical

Monday, May 23, 7:30 – 9:00 a.m.

1

PEDiatric SURGERY IN hAI: A ONE YEAR EXPERIENCE
Shahram Aarabi, MD, MPH¹, David Mooney, MD, MPH², Jason Smithers, MD²

¹University of Washington, Seattle, WA, USA, ²Children’s Hospital Boston, Boston, MA, USA

Purpose
The global burden of surgical disease has recently become a subject of international attention. There is growing evidence quality care can be provided in even the most resource-poor settings. We sought to deliver pediatric surgical care and training to local providers in rural Haiti over the course of one year.

Methods
The author served as a visiting pediatric and adult general surgeon at an established public-private partnership hospital in rural Haiti. For two weeks every two months between June 2009 and June 2010 he worked to develop the surgical program at the facility, fulfill clinical duties, collaborate with Haitian surgeons, and train Haitian and American residents and medical students. All operative patients (n=131) during this time period are presented.

Results
A total of 147 operative procedures were performed on 131 patients over the course of 12 weeks. 55 of the 65 days in Haiti were operative, with a mean of 2.6 performed per day. 78% of procedures performed were elective and 22% were emergent. Non-operative time was spent seeing patients in clinic, taking care of inpatients, and seeing new inpatient or emergency room consults. 32.1% of patients were adult, 67.9% were pediatric, including 15 children < 1 year old and 5 children < 2 months old. 28.9% of our patients lived locally while 71.1% came - usually by foot - from various locations across the country. In 91.2% of cases the author was assisted by an American medical student, American resident, Haitian resident, or Haitian staff surgeon. Only three major cases were not training cases. The total complication rate was 9.7%; 7.5% of cases lead to major complications.

Conclusion
We conclude that it is possible to provide quality pediatric surgical care in resource poor settings. Further, we believe that it is possible to provide such care while training local surgeons and residents.

Notes:
2

A NOVEL SURGICAL RISK SCORE FOR CHILDREN

Fizan Abdullah, MD, PhD, Daniel S. Rhee, MD, MPH, Yiyi Zhang, MHS, Jessica Yang, B.S., Jose Osuna-Salazar, MD, Kristin Chrouser, MD MPH, Paul M. Colombani, MD, M.B.A., David C. Chang, PhD, M.B.A., MPH

Johns Hopkins, Baltimore, MD, USA

Purpose

There is a lack of broadly applicable measures for risk adjustment in pediatric surgical patients. This study develops a risk stratification model that predicts mortality after surgical operations in children.

Methods

The model was created using nationally representative state inpatient databases from 1988 to 2006. Patients <18 years of age who received an inpatient surgical procedure were included. The multivariate training model used age and 69 different co-morbidities as independent variables to predict inpatient mortality. Point values were assigned based on coefficients and summed to give a risk score from 0-10. These point scores were validated in the 2006 dataset. Models were evaluated with Receiver Operating Characteristics (ROC) analysis. The index was compared to the Charlson Comorbidity Index using the validation dataset.

Results

A total of 2,087,915 surgical patients were identified in the training dataset, and 82,074 patients in the validation dataset. Generated risk scores positively correlated with inpatient mortality. A risk score of 0 was associated with <0.01% mortality rate in both datasets, and a score >=10 with 63% (training) and 45% (validation) mortality. In the training dataset, the ROC was 0.9553 for the original model, and 0.949 (95% Confidence interval 0.947-0.950) after conversion to an 11-point scale. In the validation dataset, this point scale’s ROC was 0.960 (95% CI 0.952-0.967) compared to the Charlson Co-morbidity Index’s ROC of 0.596 (95% CI 0.575-0.616).

Conclusions

This study depicts creation of a broadly applicable model for risk adjustment that predicts inpatient mortality with more reliability than current risk indices in pediatric surgical patients. This risk index will allow co-morbidity-adjusted outcomes in all areas of pediatric surgery.
## ROC for Risk Indices in Training and Validation Datasets

<table>
<thead>
<tr>
<th>Dataset</th>
<th>Model</th>
<th>ROC (95% Confidence Interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Training Dataset (1988-2005)</td>
<td>Original</td>
<td>0.9553</td>
</tr>
<tr>
<td></td>
<td>11-Category Point Scale</td>
<td>0.949 (0.947-0.950)</td>
</tr>
<tr>
<td>Validation Dataset (2006)</td>
<td>11-Category Point Scale</td>
<td>0.960 (0.952-0.967)</td>
</tr>
<tr>
<td></td>
<td>Charlson Comorbidity Index</td>
<td>0.596 (0.575-0.616)</td>
</tr>
</tbody>
</table>

**Notes:**
OVERNIGHT OBSERVATION FOR PREMATURITY
Carrie A. Laituri, MD, Carissa L. Garey, MD, Benjamin J. Pieters, MD, Peter Mestad, MD, Eric E. Weissand, MD, Shawn D. St. Peter, MD
Children’s Mercy Hospital & Clinics, Kansas City, MO, USA

Purpose
Overnight observation for apneic events is standard practice in former preterm infants, however, the literature supporting current protocols is limited. Therefore, we reviewed our experience with overnight observation to assess post-anesthetic risks in former premature patients.

Methods
A retrospective review was conducted on all former preterm patients admitted for observation following inguinal herniorrhaphy from January, 2000 to October, 2009. Our protocol for overnight admission included patients born prior to 37 weeks gestation who are less than 60 weeks post-conceptional age (PCA).

Results
There were 363 patients, of which, 23 were less than 40 weeks PCA (Group 1), 244 were 40-49.9 weeks PCA (Group 2), and 96 were 50-60 weeks PCA (Group 3). The demographics and recovery room courses are listed in Table 1.

Events registered by cardiorespiratory alarms occurred in 4 patients (1.1%), 2 from Group 1 and 2 from Group 2. In Group 1, one event occurred during nasogastric tube placement requiring intubation and the other was apneic desaturation during feeding which resolved spontaneously. In Group 2, one event was apnea-induced bradycardia that resolved spontaneously without stimulation and another was in a patient on home monitors with an event similar to home reports. There were no events in Group 3. The oldest gestational age for any of the 4 infants was 33 weeks.

Conclusions
These data suggest that apneic events in the recovery room do not relate well to events during overnight observation. Conservative guidelines for overnight observation after inguinal hernia repair could be set for patients born prior to 36 weeks gestation who are under 50 weeks PCA.
Table 1. Overnight stay for prematurity outcomes based on age group.

<table>
<thead>
<tr>
<th></th>
<th>&lt;40 weeks</th>
<th>40-49.99 weeks</th>
<th>50-60 weeks</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean PCA (wks)</td>
<td>38.4</td>
<td>45.0</td>
<td>54.1</td>
<td>N/A</td>
</tr>
<tr>
<td>Mean weight (kg)</td>
<td>3.5</td>
<td>4.2</td>
<td>6.0</td>
<td>N/A</td>
</tr>
<tr>
<td>Desaturation in PACU</td>
<td>56.5%</td>
<td>43.4%</td>
<td>28.6%</td>
<td>0.02</td>
</tr>
<tr>
<td>Apnea in PACU</td>
<td>43.5%</td>
<td>20.9%</td>
<td>15.3%</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Assisted Ventilation</td>
<td>17.4%</td>
<td>10.7%</td>
<td>10.2%</td>
<td>NS</td>
</tr>
<tr>
<td>Bradycardia in PACU</td>
<td>0</td>
<td>4.9%</td>
<td>2%</td>
<td>NS</td>
</tr>
<tr>
<td>Total A/B/D in PACU</td>
<td>56.5%</td>
<td>45.1%</td>
<td>28.6%</td>
<td>0.02</td>
</tr>
<tr>
<td>Mean OP time (min)</td>
<td>34.3 ± 12.9</td>
<td>35.5 ± 17.2</td>
<td>40.5 ± 23.4</td>
<td>NS</td>
</tr>
</tbody>
</table>

Notes:
4

PANCREATIC HEAD RESECTION AND ROUX-EN-Y PANCREATICOJEJUNOSTOMY FOR THE TREATMENT OF THE FOCAL FORM OF CONGENITAL HYPERINSULINISM.

Pablo Laje, MD, Susan A. Becker, Charles A. Stanley, MD, N. Scott Adzick, MD
The Children’s Hospital of Philadelphia, Philadelphia, PA, USA

Purpose
Determine the outcome of patients who underwent pancreatic head resection and Roux-en-Y pancreaticojejunostomy to the remaining normal pancreatic body and tail for the treatment of a focal lesion in the pancreatic head causing congenital hyperinsulinism (HI).

Methods
138 patients underwent pancreatic resection for focal HI between 1999 and 2010; 22 patients in that group underwent pancreatic head resection and reconstruction.

Results
There were 12 females and 10 males. Preoperative focal lesion localization was done by ASVS in the first 5 cases, and a [18-F]-DOPA-PET scan in the following 17. PET scans correlated 100% with the intraoperative lesion location. Median age at surgery was 8 weeks (range: 3 to 56). Median weight at surgery was 5.8 kg (range: 4.3 to 9.8). Seven cases had a focal lesion that extended beyond the pancreatic head into the pancreatic neck (5), proximal body (1), or distal body (1). All patients had a total or near-total pancreatic head resection and two patients had a pylorus-preserving Whipple procedure. Three patients had previously undergone incomplete resection of the pancreatic head focal lesion. All pancreaticojejunostomies were performed with interrupted monofilament sutures between the end of the jejunal limb and the pancreatic surface so that the cut end of the pancreatic body was tucked within the bowel lumen. Median operative time from incision to closure was 251 minutes (numerous biopsies for frozen section analysis are invariably required). Mean blood loss was 25 ml (range: 5 to 75). There were no intraoperative complications. There was one postoperative pancreatic leak that sealed after drainage. All patients are cured of HI.

Conclusions
We conclude that pancreatic head resection with Roux-en-Y pancreaticojejunostomy is a safe and effective procedure for the treatment of the HI baby with a large focal lesion in the pancreatic head that is not amenable to local resection alone.

Notes:
5
ESOPHAGEAL GROWTH INDUCTION (FOKER PROCESS) FOR THE TREATMENT OF LONG GAP ESOPHAGEAL ATRESIA: INITIAL EXPERIENCE AT A SINGLE INSTITUTION
Russell W. Jennings, MD1, David Clendenin, MD1, Michael Manfredi, MD1, Bradley Linden, MD1, John Foker, MD, PhD2
1Children’s Hospital, Boston, MA, USA, 2University of Minnesota, Minneapolis, MN, USA

Purpose
Long-gap esophageal atresia (LGEA) poses numerous challenges for pediatric surgeons. A promising approach to LGEA repair is linear esophageal growth induction (Foker Process). Our aim was to provide follow-up data on the first 12 patients treated using this approach by this team over the last 6 years.

Methods
Retrospective review of twelve patients with LGEA who underwent growth induction of their esophageal segments using the Foker Process. Patient data including fistulas, esophageal remnants, gap length, number of thoracotomies required, stricture treatment, length of follow-up and patient outcome data were obtained. IRB Protocol M10-10-052.

Results
All patients had a true primary esophageal repair. Eleven of 12 patients had esophageal gaps over 4 cm, 5 had proximal tracheo-esophageal fistulas, and 2 patients had rudimentary lower esophageal segments below the diaphragm, and 1 patient had an esophageal primordium. Patients required 2-4 thoracotomies. After anastomosis all underwent at least one esophageal dilation (range 1-17 dilations), while 2 patients also required temporary esophageal stent placement for the treatment of difficult esophageal strictures. All patients underwent Nissen fundoplication. Ten patients are now eating by mouth, 1 is undergoing treatment for oral aversion, and 1 is starting oral feeds. Follow-up is from 1 month to 6 years. See Table 1.

Conclusions
These results suggest that in patients with LGEA even the most rudimentary esophageal segments and ultra-long gaps have potential for adequate growth response using the Foker Process. This approach allows a true primary esophageal anastomosis with excellent results in follow-up to 6 years.
### Table 1. Selected LGEA results using the Foker Process. VB=Vertebral bodies.

<table>
<thead>
<tr>
<th>Age at Operation</th>
<th>Gap</th>
<th>Time for Growth</th>
<th>Dilations</th>
<th>G-tube</th>
<th>Result</th>
<th>Follow-up</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 mo</td>
<td>5 cm</td>
<td>21 days</td>
<td>Yes, n/a</td>
<td>No</td>
<td>Normal</td>
<td>6 years</td>
</tr>
<tr>
<td>5 mo</td>
<td>8 VB</td>
<td>16 days</td>
<td>2</td>
<td>Yes</td>
<td>Thickened feeds</td>
<td>5 years</td>
</tr>
<tr>
<td>2 mo</td>
<td>n/a</td>
<td>6 days</td>
<td>7</td>
<td>No</td>
<td>Normal</td>
<td>4 years</td>
</tr>
<tr>
<td>2 days</td>
<td>5 cm</td>
<td>7 days</td>
<td>1</td>
<td>No</td>
<td>Normal</td>
<td>4 years</td>
</tr>
<tr>
<td>4 mo</td>
<td>5 cm</td>
<td>21 days</td>
<td>Yes, n/a</td>
<td>No</td>
<td>Normal</td>
<td>4 years</td>
</tr>
<tr>
<td>4 mo</td>
<td>5 VB</td>
<td>15 days</td>
<td>5</td>
<td>No</td>
<td>Normal</td>
<td>2.5 years</td>
</tr>
<tr>
<td>2 mo</td>
<td>4 VB</td>
<td>10 days</td>
<td>5</td>
<td>No</td>
<td>Normal</td>
<td>1.5 years</td>
</tr>
<tr>
<td>3 mo</td>
<td>4 cm</td>
<td>20 days</td>
<td>2</td>
<td>No</td>
<td>Normal</td>
<td>1 year</td>
</tr>
<tr>
<td>3 mo</td>
<td>4 cm</td>
<td>23 days</td>
<td>1</td>
<td>No</td>
<td>Normal</td>
<td>1 year</td>
</tr>
</tbody>
</table>

**Notes:**
Purpose

Pyloric stenosis is the most common surgical disease of infants. Open pyloromyotomy has been the gold standard in treatment, with the laparoscopic approach rapidly gaining adoption. We present a prospective, randomized trial between the two approaches with long-term follow-up.

Methods

After obtaining IRB approval, 98 patients with hypertrophic pyloric stenosis were prospectively enrolled and randomized to either open or laparoscopic pyloromyotomy. Post-operative and hospital course were evaluated by review of hospital records and long-term follow-up with scripted telephone survey. Primary outcomes were length of operating room time, length of surgical procedure, length of postoperative stay, time to re-feeding and complications. Secondary outcomes evaluated were cosmetic results and parental satisfaction using Likert scales. Results were evaluated with Fisher exact and Student t tests.

Results

From July 2005 through November 2009, 98 patients were enrolled in the study. There were no significant differences in length of operating room time (mean for laparoscopic- 1:07 minutes, mean for open- 0:54 minutes), length of surgical procedure (mean for laparoscopic- 0:24 minutes, mean for open- 0:24 minutes), length of postoperative stay (mean for laparoscopic- 1 day, mean for open- 1 day), and time to re-feeding (mean for laparoscopic- 16 hours, mean for open- 16 hours). There were two complications in the open group - a wound dehiscence and a surgical site infection; and two complications in the laparoscopic group - a conversion to open pyloromyotomy for mucosal perforation and a suture granuloma requiring wound exploration (p = 1.00). In long-term follow-up (mean of 44 months), parents described statistically significant cosmetic results with laparoscopic approach across all dimensions queried (p = 0.036).

Conclusions

There is no difference in operating time, hospital stay, or re-feeding patterns between open and laparoscopic pyloromyotomy. The complication rates were similar between the two methods. However, long-term cosmetic results were significantly superior in the laparoscopic group.

Notes:
7

PEDIATRIC CHRONIC ULCERATIVE COLITIS: DOES INFLEXIMAB INCREASE POST-IPAA COMPLICATIONS?

Raelene Kennedy, MD, D.D. Potter, MD, C. Moir, MD, A. Zarroug, MD, W. Faubion, MD, J. Tung, MD

Mayo Clinic, Rochester, MN, USA.

Purpose

Ileal pouch anal anastomosis (IPAA) is a common surgical approach to chronic ulcerative colitis (CUC). Pre-operative use of Infliximab (IFX) has raised concern of increased postoperative complications. We sought to compare outcomes of pediatric patients (< 18 years) who were treated with IFX prior to IPAA to those who did not.

Methods

Patients who underwent IPAA from 2003 to 2008 for CUC, and were < 18 years of age were included. Patient records were retrospectively reviewed for preoperative medication usage, operative technique, and 1 year postoperative complications (leak, wound infection, small bowel obstruction, pouchitis). Subjects were divided into two groups; those who received IFX within 8 weeks preoperatively and those who did not. T-tests and Chi square analysis were employed, where p<0.05 was considered significant.

Results

Eleven patients received IFX preoperatively and 27 children did not during the study period. Steroid use was similar (IFX: 91% vs Control: 78%); however, thiopurines were used more commonly in the IFX group (91% vs 41%, p<0.01). Preoperative albumin was lower in the IFX group (3.4 vs 4, p=0.03). All complications following IPAA were more frequent in the IFX group as compared to controls (55% vs 26%) but this was not statistically significant. Postoperative leaks occurred in 9% of patients in the IFX group and 7% of controls. Small bowel obstruction was significantly higher in the IFX group (55% vs 7.4%, p=0.01). Long-term complications (1 year) occurred in 64% of the IFX group and 62% of the controls. Pouchitis accounted for nearly all the late complications.

Conclusion

Children that were treated with IFX prior to IPAA suffered nearly twice as many post-operative complications as compared to controls. Long-term outcomes are similar. Currently, we recommend colectomy with end ileostomy for patients that have received IFX within 8 weeks of surgical intervention for CUC.

Notes:
RESTORATIVE PROCTOCOLECTOMY WITHOUT DIVERTING ILEOSTOMY IN CHILDREN WITH ULCERATIVE COLITIS

Brian W. Gray, MD, James Geiger, MD, Ronald Hirschl, MD
University of Michigan, Ann Arbor, MI, USA

Purpose
Restorative proctocolectomy (RP) and J-pouch ileoanal anastomosis (IPAA) is routinely performed as a two-stage procedure with a diverting ileostomy for children with ulcerative colitis (UC). Whether this procedure can be safely performed without diverting ileostomy has not been extensively examined. Our goal was to examine outcomes of children who underwent RP and IPAA without ileostomy.

Methods
We performed a single-institution retrospective review of UC patients who had RP and IPAA with (+Ostomy) or without (-Ostomy) diverting ileostomy from 2002-2010. Decision for or against ileostomy was based on surgeon and patient preference. 50 patients were studied (28 +Ostomy, 22 -Ostomy). Demographics, early complications, and functional outcomes were examined. Comparisons were made using the t-test for equality of means.

Results
Preoperative demographics were similar in mean+S.D. age (13.6+3.5yrs -Ostomy, 14.3+3.0yrs +Ostomy), serum albumin (3.64+0.71 -Ostomy, 3.62+0.72 +Ostomy), BMI (20.8+6.9 -Ostomy, 21.3+8.6 +Ostomy), and daily corticosteroid dose (23.66+15.68mg -Ostomy, 28.5+15.74mg +Ostomy). Total hospital length of stay was less in -Ostomy (14+8.9 days vs. 17+8.6 days), but not statistically significant (p 0.24). There were two episodes of pouch leak in each group, and 2 patients had anastomotic leak on ostomy takedown. 8 experienced complications in -Ostomy and 9 in +Ostomy. The -Ostomy group required less anastomotic dilations in the OR (0.4+0.8 vs 1.4+1.9, p<0.05). Functional outcomes were not significantly different regarding pouchitis episodes per patient (0.6+1.1 -Ostomy, 0.6+1.0 +Ostomy), bowel movements per day (5.5+1.9 -Ostomy, 6.7+4.0 +Ostomy), and daily postop loperamide dose (8.44+4.31mg -Ostomy, 6.82+4.0mg +Ostomy).

Conclusion
Short and long term outcomes can be equivalent in patients with and without diverting ileostomy, but questions remain regarding patient selection and quality of life impact. This is the first report to explore the risks and benefits of performing RP and IPAA without diverting ileostomy in children, which appears to be a reasonable surgical option in those with UC.

Notes:
MESENTERICO-LEFT PORTAL VEIN BYPASS IS SUPERIOR TO PORTOSYSTEMIC SHUNT IN THE MANAGEMENT OF EXTRAHEPATIC PORTAL VEIN OBSTRUCTION

Timothy B. Lautz, MD, Joseph C. Melvin, BS, Lisa Keys, MSN, Riccardo A. Superina, MD
Children’s Memorial Hospital, Northwestern University, Chicago, IL, USA

Purpose
Extrahepatic portal vein obstruction (EHPVO) causes significant morbidity in affected children. Symptoms including variceal hemorrhage and hypersplenism are due to portal hypertension, while metabolic complications including growth impairment, neurocognitive dysfunction and coagulopathy are caused by abnormal portal circulation. The purpose of this study was to compare the effectiveness of mesenterico-left portal vein bypass (MLPVB) and portosystemic shunt for relieving the symptoms of EHPVO.

Methods
Consecutive patients who underwent an operation for EHPVO by a single surgeon from 1999-2009 were compared. MLPVB was considered first for all patients, but portosystemic shunts were performed when MLPVB was not feasible. All outcome parameters were obtained at least one year after surgery.

Results
Seventy-two children underwent successful MLPVB (Group 1, 79% of total) and were compared to 19 patients who underwent portosystemic shunts (Group 2, 14 splenorenal and 5 mesocaval). Variceal bleeding resolved in all 59 Group 1 patients and in 13 of 14 Group 2 patients who manifested this symptom preoperatively. Before surgery, patients with EHPVO suffered from growth retardation (weight z-score -0.39 ± 1.09), thrombocytopenia (platelets 119 ± 101 thou/ml), hyperammonemia (ammonia 50 ± 30 μmol/L), and impaired synthetic function (INR 1.27 ± 0.28), with no significant differences between operative groups (all p>0.1). Following surgery, Group 1 had significantly better improvement compared to Group 2 in weight z-score (p=0.048), platelet count (p=0.006), INR (p=0.002) and ammonia (p=0.002) (Table 1).

Conclusion
Both mesenterico-left portal vein bypass and portosystemic shunting provide equivalent long-term relief from variceal hemorrhage due to portal hypertension. MLPVB restores portal circulation and achieves superior relief of both thrombocytopenia and metabolic symptoms, including growth failure, synthetic dysfunction and hyperammonemia.
## Outcome of MLPVB versus portosystemic shunt

<table>
<thead>
<tr>
<th></th>
<th>MLPVB</th>
<th>Portosystemic shunt</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight z-score</td>
<td>+0.76 ± 1.02</td>
<td>+0.13 ± 0.59</td>
<td>0.048</td>
</tr>
<tr>
<td>Platelet count (thou/ml)</td>
<td>+77.7 ± 64.0</td>
<td>+29.0 ± 56.1</td>
<td>0.006</td>
</tr>
<tr>
<td>INR</td>
<td>-0.23 ± 0.27</td>
<td>+0.22 ± 0.63</td>
<td>0.002</td>
</tr>
<tr>
<td>Serum ammonia (umol/L)</td>
<td>-25.5 ± 36.7</td>
<td>+18.0 ± 33.3</td>
<td>0.002</td>
</tr>
</tbody>
</table>

**Notes:**
10
MAGNETIC MINI-MOVER PROCEDURE FOR PECTUS EXCAVATUM III: SAFETY AND EFFICACY IN AN FDA SPONSORED CLINICAL TRIAL
Michael R. Harrison, MD, Kelly D. Gonzales, MD, Barbara J. Bratton, MSN, PNP, Darrell Christensen, CO, Patrick F. Curran, BS, Richard Fechter, BS, Shinjiro Hirose, MD
UCSF, San Francisco, CA, USA

Purpose
The Magnetic Mini Mover Procedure (3MP) uses magnetic force to gradually remodel pectus excavatum deformity using an implanted titanium enclosed magnet (Magnimplant) coupled with an external magnetic brace (Magnatract). After obtaining an Investigational Device Exemption (IDE), IRB approval, and FDA funding through the Office of Orphan Products Development (OOPD), we performed a trial to determine safety and probable efficacy.

Methods
Ten otherwise healthy patients with severe pectus excavatum deformities (age 8-14 years, Pectus Severity Index (PSI) > 3.5) underwent 3MP for an average treatment duration of 18 months. Safety was assessed by monthly office visits with chest x-ray and perioperative EKG. PSI was assessed by computed tomography (CT) scan. Cost of 3MP was compared to Nuss and Ravitch procedures in our institution during the same time period.

Results
Safety: The 3MP device had no detectable ill-effect on any physiologic parameter including subjective exercise tolerance or EKG. There were no significant operative complications or wound infections following insertion of the Magnimplant or during therapy Magnatract. Retained pleural air had to be evacuated in 3 patients. Device weld failure or malpositioning required revision in 5 patients. PSI as measured by CT or chest x-ray proved surprisingly unreliable. PSI improved >50% in the 5 youngest (pre pubertal or pubertal) patients. Post pubertal patients with non compliant chest walls did not show significant improvement. However, subjective satisfactory correction was seen in most patients. Cost: The overall average cost for the 3MP was $46,859, compared to the Nuss ($81,206) or Ravitch ($81,022).

Conclusion
The 3MP is a safe, cost-effective, minimally invasive outpatient alternative treatment for pectus excavatum, which achieves good results for pre-pubertal and pubertal patients but not post-pubertal patients. The FDA has funded a second multicenter trial focused on pre- pubertal and pubertal patients as determined by bone age.

Notes:
11
SINGLE INCISION LAPAROSCOPIC SURGERY: A RANDOMIZED CONTROL TRIAL IN ACUTE APPENDICITIS

Eduardo A. Perez, MD, Robert Barber, Anne C. Fischer, MD PhD
Childrens Medical Center, Dallas, TX, USA

Purpose
Single Incision Laparoscopic Surgery (SILS) is a novel area of minimally invasive surgery using a single incision to minimize all ports to one site. The end result is an incision that can be strategically placed in the umbilicus for a perceived scarless abdomen. We rationalized that a randomized control trial was important given the rapid popularization of this approach.

Methods
An IRB-approved prospective randomized trial was performed comparing patients undergoing SILS (SILS-A) or conventional (LAP-A) appendectomy at a free-standing children’s hospital with a 1 yr follow up.

Results
A total of 46 patients were randomized equally to SILS-A and LAP-A. Fifty percent were males, and 67% were Hispanics. Ages spanned from 3 -15 years of age without a difference between groups: 50% were <8 years. The technique for SILS-A involved a single supraumbilical curvilinear incision with 3 fascial incisions. Ports were inserted to varying depths to minimize restriction of instrument movement. Coaxial visualization was improved by use of a 5mm 30° scope. To be technically comparable to the LAP-A, a stapler device was used which required upsizing a 5mm to 12mm port. Mean duration of operation was 46m ± 4 (22 - 129) compared to 32m ± 1.8 (18 - 59) for standard LAP-A (p=0.003). There were no conversions nor difference in LOS. Postoperative complications consisted of two wound infections in the SILS-A group (N.S.), no hernias were seen. No difference in readmissions, diet tolerance, fever or postoperative pain was noted between the two groups.

Conclusions
SILS approach has been shown to be feasible in the pediatric population despite a limited abdominal domain in the younger ages. While OR times are currently longer than LAP-A, they are comparable and no other outcomes appreciably differed between both techniques at the time of hospitalization or in follow up.

Notes:
12
TRANSLUMINAL ENDOSCOPIC FUNDOPLICATION FOR THE
TREATMENT OF REFLUX DISEASE: COMPARISON TO
LAPAROSCOPIC NISSEN

Stephanie Chen, BS, Marcus D. Jarboe, MD, Robert D. Drongowski, MS,
Daniel H. Teitelbaum, MD
University of Michigan, Ann Arbor, MI, USA

Purpose
Although laparoscopic Nissen fundoplication (LNF) is the standard surgical
treatment for gastroesophageal reflux disease (GERD), surgical complications
and post-operative pain are common, especially for those who for those under-
going re-operative LNF. To address this challenge, we utilized an endoscopic
approach, or transoral, incisionless fundoplication (TIF) procedure (using the
EsophyX, EndGastric Solutions, Inc.); and hypothesized that a TIF would lead
to comparable outcomes to those undergoing a LNF.

Methods
Patients who underwent a TIF were matched to LNF control patients according
to age (mean±SD: TIF:16.0±5.4 vs.LNF:13.8±3.6years), presence of neurologi-
cal impairment (TIF:9/11,82%; LNF11/14,79%), and if this was a redo procedure
(TIF:5/11,45%; LNF:5/14,36%). A retrospective chart review was conducted to
compare patient outcomes and post-operative complications. Fisher exact test
and independent samples t-test were utilized to compare groups.

Results
Of 11 TIF procedures, 10 had sufficient follow-up (>2 months) for evalua-
tion. TIF was associated with a significantly (P=0.001) shorter procedure time
(112.6±35.0 mins) compared to LNF (204.2±66.9 mins), with comparable blood
loss, length of stay, and morphine usage. One TIF patient was re-admitted and
underwent endoscopic clipping of a GI bleeder. Although follow-up in the TIF
group was much shorter than the LNF group (Table), at last follow-up TIF was
found to have effectively resolved GERD symptoms in nine, mild GERD in one,
and none required a subsequent LNF. Adverse complaints were not uncom-
mon; gagging, dysphagia, and feeding difficulties for TIF patients seem to oc-
cur at rate similar to LNF patients (Table).

Conclusions
TIF appears to be as safe and effective as laparoscopic LNF for treating GERD.
However, further studies and a longer follow-up must be conducted to better
assess efficacy.
## SCIENTIFIC SESSION I

### Outcome Data TIF vs. LNF

<table>
<thead>
<tr>
<th>Group</th>
<th>TIF</th>
<th>LNF</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean Age (years)</td>
<td>16.5±5.1</td>
<td>13.8±3.5</td>
<td>0.146</td>
</tr>
<tr>
<td>Mean body weight (Kg)</td>
<td>45.7±13.3</td>
<td>33.7±15.3</td>
<td>0.067</td>
</tr>
<tr>
<td>Length of hospital stay (days)</td>
<td>2.4±2.5</td>
<td>3.1±1.3</td>
<td>0.308</td>
</tr>
<tr>
<td>Morphine usage (mg/hosp. stay)</td>
<td>9.2±16.5</td>
<td>7.4±10.2</td>
<td>0.881</td>
</tr>
<tr>
<td>Post-procedure gagging/dysphagia (%)</td>
<td>3/10 (30)</td>
<td>5/14 (36)</td>
<td>1.000</td>
</tr>
<tr>
<td>Post-procedure recurrent reflux</td>
<td>1/10</td>
<td>4/11</td>
<td>0.358</td>
</tr>
<tr>
<td>Duration of follow-up (months)</td>
<td>7.6 ± 8.1</td>
<td>43 ± 33.8</td>
<td>0.01</td>
</tr>
</tbody>
</table>

**Notes:**
Oncology and Critical Care

Monday, May 23, 10:30 – 11:45 a.m.

13
CANCER STEM/PROGENITOR CELLS RESPOND POORLY TO CHEMOTHERAPEUTIC AGENTS.

Katia Meirelles, MD, Leo Andrew O. Benedict, MD, David Dombkowski, Frederic I. Preffer, Jose Teixeira, PhD, David T. MacLaughlin, PhD, Patricia K. Donahoe, MD, Xiaolong Wei, MD, PhD

Massachusetts General Hospital, Cambridge, MA, USA

Purpose
Tumor initiating cells are defined by tumorigenecity, recurrence, metastasis, and drug resistance. These cells are thought to be capable of unlimited self-renewal, proliferation, and differentiation. We hypothesized that these cells can be identified by surface markers and have different responses to chemotherapeutic agents.

Methods
Flow cytometry was used to screen 130 markers to define a panel of three cell surface markers consisting of CD44, CD24, and Epcam (3+) from human fimbria, ascites from ovarian cancer patients, and human ovarian cancer cell lines (OVCAR-5, SKOV-3, IGROV-1). We tested these cells for migration using a matrigel assay and colony formation in vitro. Furthermore tumor free intervals were determined after flank injection of 100 and 1000 separated cells following limiting dilution in mice. Chemotherapeutic agents, such as doxorubicin, cisplatin, and paclitaxel, were compared to Mullerian Inhibiting Substance (MIS) for effect on separated 3+/3- stem/progenitor cells.

Results
We examined 3+ cells that also displayed a loss of E-cadherin. These 3+E-cad cells showed shorter tumor free intervals \textit{in vivo} after limiting dilution (FIGURE 1) and increased colony formation \textit{in vitro} when compared to 3-Ecad- cells. Loss of E-cadherin is associated with epithelial to mesenchymal transformation, which is thought to contribute to cancer progression by increasing proliferation, invasion, and/or metastasis. Whereas the 3+ cells were found in greater than 3% of unseparated cells, the 3+Ecad- cells were identified in less than 1% of unseparated cells. In addition, doxorubicin treated cells showed resistance and selection for 3+Ecad- colonies in comparison to MIS, which decreased the absolute number of 3+E- colonies.
Conclusion
We conclude that the 3+Ecad- cells may be a highly enriched “stem/progenitor cell” population from human ovarian cancer cell lines. These findings provide us with an experimental model in which to identify mechanisms contributing to stem/progenitor characteristics and to elucidate therapeutic targets for ovarian cancer.

Notes:
OMEGA-3 FATTY ACIDS INHIBIT THE GROWTH OF NEUROBLASTOMA TUMORS AND INDUCE A TUMOR SPECIFIC ESSENTIAL FATTY ACID DEFICIENCY IN A MURINE XENOGRAFT TUMOR MODEL

Deepika Nehra, MD1, Hau D. Le, MD2, Erica M. Fallon, MD2, Vincent E. de Meijer, MD, MSc3, Amy H. Pan, BA3, Paul Mitchell, MS3, Mark Puder, MD, PhD3

1Children’s Hospital Boston; Massachusetts General Hospital, Boston, MA, USA, 2Children’s Hospital Boston; Beth Israel Deaconess Medical Center, Boston, MA, USA, 3Children’s Hospital Boston, Boston, MA, USA

Purpose
To determine the effect of dietary omega-3 fatty acids on neuroblastoma tumor growth and tumor fatty acid profiles in a murine subcutaneous xenograft model of neuroblastoma.

Methods
After IACUC approval, five-week old SCID mice were randomized into one of three dietary groups (n=five in each group). In each group 10% of total calories were provided in the form of fat: soybean oil (omega-6 rich), hydrogenated coconut oil (essential fatty acid deficient) or a 20:1 ratio of DHA:AA (omega-3 rich). After three weeks of dietary pre-treatment, human neuroblastoma cells (SK-NSH) were subcutaneously implanted in the left flank. All animals were euthanized after three weeks and tumor volume and weight measured. Tumors, muscle surrounding tumor, liver and serum were collected for fatty acid analysis.

Results
The omega-3 diet was associated with a smaller median tumor volume and weight compared to the omega-6 diet. The median tumor volume among mice fed the omega-3 diet was less than half (48%) that observed in mice fed the omega-6 diet (p=0.07). The median tumor weight in mice fed the omega-3 diet was one-third (33%) that observed among mice fed the omega-6 diet (p=0.05). Tumors from animals fed the omega-3 diet developed a biochemical essential fatty acid deficiency (EFAD) based on the triene:tetraene (T:T) ratio (EFAD = T:T ratio>0.2) while the serum and other organs did not demonstrate EFAD. The mean±SD tumor T:T ratio was nearly four times higher in the omega-3 group compared to the omega-6 group (0.337±0.045 vs. 0.088±0.024, p=<0.01). This dramatic change occurred in tumors while the liver T:T ratios were significantly less than 0.2 in both the omega-3 and omega-6 groups (0.007±0.007 and 0.016±0.006, respectively). EFAD deficiency was seen in all tissues harvested from animals on the hydrogenated coconut oil diet.

Conclusion
Dietary omega-3 fatty acids have an inhibitory effect on in vivo neuroblastoma tumor growth and induce a tumor specific fatty acid deficiency.

Notes:
LYMPH NODE SAMPLING IN WILMS TUMOR: WHAT IS “ENOUGH”?  

Kathleen Kieran, MD¹, Jeffrey S. Dome, MD², Peter F. Ehrlich, MD³, Michael L. Ritchey, MD⁴, Robert C. Shamberger, MD⁵, Daniel M. Green, MD⁶, Andrew M. Davidoff, MD⁶  
¹University of Iowa, Iowa City, IA, USA, ²Children’s National Medical Center, Washington, DC, USA, ³University of Michigan, Ann Arbor, MI, USA, ⁴Pediatric Urology Associates, Phoenix, AZ, USA, ⁵Children’s Hospital Boston, Boston, MA, USA, ⁶St Jude Children’s Research Hospital, Memphis, TN, USA  

Purpose  
Disease stage provides prognostic information and dictates treatment therapy for patients with unilateral Wilms tumor (WT). Lymph node (LN) metastases confer an increased disease stage, independent of the extent of the local primary tumor. Previous research suggests that surgeon assessment of LN status correlate poorly with pathologic findings. We therefore sought to determine if a minimum number of LNs are needed for an adequate LN dissection.  

Methods  
We reviewed the records of all patients enrolled in National Wilms Tumor Study (NWTS) 4 and 5. Patients with metastatic or bilateral disease, neoadjuvant chemotherapy, or anatomic variants were excluded. Data were abstracted on patient demographics, primary tumor characteristics, clinical and pathologic disease stages, number of LNs sampled (overall and positive), and disease-specific and overall patient outcomes. Our primary endpoint was the number of LNs needed to obtain a single positive LN; our secondary endpoint was the impact of proportion of positive LNs on event-free survival.  

Results  
3216 patients with favorable (n=2958), focal anaplastic (n=52) or diffuse anaplastic histology (n=206) underwent LN dissection and had complete evaluable information available. 587 (18%) had positive LNs. Figure 1 depicts the proportion of patients with positive LNs, stratified by the number of LNs sampled; the likelihood of obtaining at least 1 positive LN increases when at least 7 LNs were sampled. Among patients with 7 or more LNs sampled, those with ≥45% of LNs positive fared more poorly with regard to event-free survival (RR=2.29, p=0.04), particularly if anaplasia was present on histologic assessment.  

Conclusions  
LN sampling for WT provides valuable staging and prognostic information. Proper staging is more likely in patients in whom at least 7 nodes are obtained during LN sampling; in patients with an adequate number of LNs, the proportion of LNs positive may predict response to therapy and overall outcome for some patient subsets.  

Notes:
SCIENTIFIC SESSION II

16
ELIMINATION OF THE MATERNALLY INDUCED ADAPTIVE IMMUNE RESPONSE ALLOWS UNIFORM ACHIEVEMENT OF HIGH LEVEL ALLOGENEIC CHIMERISM BY IN UTERO HEMATOPOIETIC CELL TRANSPLANTATION AND POSTNATAL MINIMAL CONDITIONING BMT
Matthew T. Santore, MD, Demetri J. Merianos, MD, Carlyn A. Todorow, Alan W. Flake, MD
The Children’s Hospital of Philadelphia, Philadelphia, PA, USA

Purpose
We have previously demonstrated that donor hematopoietic chimerism after in utero hematopoietic cell transplantation (IUHCT) is limited by an adaptive alloimmune response induced by maternal antibodies transferred in breast milk. If this transfer is eliminated by foster nursing recipients with naïve mothers, 100% of recipients remain chimeric. We hypothesized that fostered mice would be tolerant to the donor and that high levels of donor chimerism could be uniformly achieved using a strategy of postnatal enhancement of chimerism by a minimal conditioning same donor BMT.

Methods
E14 Balb/c fetuses received 20 million B6-GFP BM cells/fetus by intravascular injection. The pups were fostered at P1. At P28 a minimally ablative dose of busulfan(15mg/kg) was administered followed by transplantation of T-cell depleted B6-GFP BM cells. Chimerism was assessed by flow cytometry as the percentage of CD45+ cells that were GFP+. Skin grafts were placed from donor and third party mice to assess donor specific tolerance.

Results
At one, three and five months after the postnatal transplant an increasing level of chimerism was observed relative to fostered controls that did not receive the postnatal BMT (2M-66.7% vs. 31.7%, 3M-78.2% vs. 26.4%, 5M-90.1% vs. 24.4%, p<0.001). Enhancement of chimerism was associated with donor specific tolerance as demonstrated by acceptance of the B6-GFP skin graft and rejection of third party grafts.

Conclusions
This study demonstrates for the first time that high level allogeneic chimerism can be achieved in 100% of recipients by the potential clinical strategy of IUHCT followed by a minimal conditioning same donor postnatal BMT.

Notes:
SCIENTIFIC SESSION II

17 SURVIVAL IS IMPROVED IN VENOVENOUS VERSUS VENOARTERIAL ECMO FOR PEDIATRIC NON-CARDIAC SEPSIS: A STUDY OF THE ELSO REGISTRY

Sean C. Skinner, MD, Joseph A. Iocono, MD, Hubert O. Ballard, MD, Marion D. Turner, MD, Daniel L. Davenport, PhD, Joseph B. Zwischenberger, MD

University of Kentucky, Lexington, KY, USA

Purpose
To compare survival in venoarterial (VA) versus venovenous (VV) ECMO for septic, non-cardiac, pediatric patients.

Method
Following approval, we reviewed the ESLO registry data from 1990-2008 in patients less than 18 years old, with a primary, secondary or discharge diagnosis of sepsis. Patients with congenital heart diagnoses and with both or unknown modalities were excluded. Survival between VA and VV ECMO was compared using chi squared analysis and with multivariable logistic regression adjusting for age, use of vasoactive drugs and advanced respiratory maneuvers.

Results
4556 ECMO runs were reviewed of which 3256 (75.1%) were VA and 1076 (24.9%) were VV. Overall survival was 67.7% and was higher in VV (78.9%) than VA (64.0%) ECMO (p < .001). Survival decreased with age from 73.2% of neonates to 40.1% of children and 31.5% of adolescents (P<0.001) with improved survival in the VV group for all age groups (Figure). This was despite the VV group having greater severity of illness as measured by vasoactive drug support and advanced respiratory maneuvers (e.g. HFOV, Surfactant, iNO, use of paralytics) (Table). After multivariable adjustment VA ECMO carried a higher mortality risk than VV (odds ratio 2.06, 95% CI 1.74-2.44, P<0.001).

Conclusion
The data show that VV ECMO has a survival benefit in select non-cardiac pediatric patients with sepsis after adjustment for age, vasoactive drug use and advanced respiratory maneuvers. Despite the obvious selection bias (3 to 1 in favor of VA) in this registry, evaluable endpoints favored VV ECMO. Venovenous ECMO should be the first therapy chosen when technically feasible.
Survival and Risk Factors by ECMO Modality

<table>
<thead>
<tr>
<th>Variable</th>
<th>VA (n=3256)</th>
<th>VV (n=1076)</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Survival</td>
<td>64.0 %</td>
<td>78.9 %</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Vasoactive Drug Support</td>
<td>83.4 %</td>
<td>88.2 %</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Advanced Respiratory Maneuvers</td>
<td>73.1 %</td>
<td>85.0 %</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Notes:
SCIENTIFIC SESSION II

18
ECMO CANNULATION TRENDS FOR PEDIATRIC RESPIRATORY FAILURE AND CENTRAL NERVOUS SYSTEM INJURY
Michael D. Rollins, MD1, Ania Hubbard, MD1, Luke Zabrocki, MD2, Douglas C. Barnhart, MD, MPH1, Susan L. Bratton, MD, MPH1
1Primary Children’s Medical Center, University of Utah, Salt Lake City, UT, USA, 2Naval Medical Center San Diego, San Diego, CA, USA

Background
Guidelines regarding arterial cannula site for pediatric patients requiring veno-arterial (VA) ECMO support are lacking and the risks of central nervous system (CNS) injury related to cannula site are not well quanitated. We sought to: 1) review cannulation information for pediatric respiratory failure 2) to evaluate CNS complication rates by cannulation site and 3) to determine adjusted risk of CNS injury associated with support mode

Methods
The ELSO registry was queried for all patients <18 years treated from January 1, 1993 to December 31, 2007 for respiratory failure. Exclusion criteria included missing cannulation site information, and patients with complex arterial cannulation. The primary outcome was radiographic evidence of CNS injury. Statistical analyses were performed using non parametric tests and a logistic regression model was developed to evaluate patient demographics, pre- ECMO and initial ECMO features associated with radiographic evidence of CNS injury.

Results
2617 ECMO runs for available for analysis. VA support was used in 62% of patients. The carotid artery was used in 93%. The femoral artery was increasingly used in patients > 10 years (1-5 years 3%, 5.1-10 years 12%, 10.1-15 years 31%, 15.1-18 years 45%) and in patients > 20 kg. Venovenous (VV) ECMO was used in >50% of children > 10 years old. No significant difference was identified in CNS injury between carotid and femoral cannulation in any age group. CNS injury in patients > 20 kg was 10% for femoral vs. 17% among carotid cannulation (p=ns). VV support was independently associated with decreased odds of CNS injury compared to VA cannulation (OR 0.57; 95% Confidence Interval 0.41-0.79)

Conclusion
VA ECMO is the most common mode of support in pediatric respiratory failure patients. No significant difference in CNS injury was noted between carotid and femoral artery cannulation; however, the adjusted risk was significantly lower in the VV group.

Notes:
UTILITY OF NEURORADIOGRAPHIC IMAGING IN PREDICTING OUTCOMES FOLLOWING NEONATAL EXTRACORPOREAL MEMBRANE OXYGENATION

Michael D. Rollins, MD¹, Bradley A. Yoder, MD¹, Kevin R. Moore, MD², Douglas C. Barnhart, MD, MPH¹, Chris Jones, RN², Donald M. Null, MD¹, Robert J. DiGeronimo, MD¹

¹Primary Children’s Medical Center, University of Utah, Salt Lake City, UT, USA, ²Primary Children’s Medical Center, Salt Lake City, UT, USA

Background
Head ultrasound (HUS) is routine while patients are on extracorporeal membrane oxygenation (ECMO). The need for follow up neuroimaging once off ECMO and the optimal radiographic study remains unclear. We sought to evaluate the correlation between findings on HUS and magnetic resonance imaging (MRI) and determine the significance of these findings to neurodevelopmental outcome.

Methods
A retrospective review was performed (2003-2010) to identify neonates who had a MRI following ECMO. Each MRI was reviewed by a single pediatric neuroradiologist. Neurodevelopmental follow up data was collected from clinic visits. Statistical analyses were performed using non parametric tests and a logistic regression model was developed to evaluate patient demographics, ECMO features and radiographic findings associated with abnormal neurodevelopment.

Results
48 neonates had a MRI (24 congenital diaphragmatic hernia (CDH), 24 non-CDH) following ECMO. Venoarterial ECMO with ligation of the carotid artery was performed in 35 patients while 13 were managed with venovenous ECMO. CDH patients had a mean ECMO time of 9.5 days and non-CDH patients 6.6 days. HUS was abnormal in 25% following ECMO whereas MRI was abnormal in 65%. Correlation between an abnormal HUS and an abnormal MRI was 100% but an additional 53% of patients with a normal HUS had an abnormal MRI. Venoarterial ECMO was significantly associated with an abnormal MRI (p=0.02). Follow up data was available for 26 neonates (18 patients > 12 months). Neither diagnosis, ECMO duration, HUS nor MRI findings were predictive of neurodevelopmental outcome. Neonates that required a feeding tube at discharge were more likely to have abnormal neurodevelopment compared to those who did not (p=0.002).
SCIENTIFIC SESSION II

Conclusions
MRI identified significantly more radiographic abnormalities compared to routine HUS following neonatal ECMO, however neither MRI nor HUS findings correlated with neurodevelopmental outcome. Feeding ability at discharge was the overall best predictor of neurologic impairment in survivors.

Notes:
COAGULATION CHANGES IN PEDIATRIC TRAUMA PATIENTS

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Purpose
In adults, alterations in the coagulation cascade are a homeostatic compensation to severe injury. It is unknown whether similar changes occur in pediatric patients. The purpose of this study was to test the hypothesis that children and infants exhibit a hypercoagulable response to trauma.

Methods
This protocol was IRB approved and conducted with informed consent and assent (where applicable). The study population was comprised of 16 patients (15:1 blunt:penetrating, 11 males, 5 females, age 8 months - 14 years) who were transported to a level one trauma center by ambulance or air rescue services. Within one hour of arrival, approximately 3 mL of venous blood was drawn into a tube containing sodium citrate. That sample was analyzed at exactly 60 min using a TEG 5000 Thromboelastograph (Haemoscope Corporation, Niles, IL). Parameters included the time to clot initiation (R time), rapidity of clot strengthening (K, α), and maximum clot strength (MA). Routine coagulation data was measured in parallel, including prothrombin time (PT), international normalized ratio (INR), partial thromboplastin time (PTT) and as well as platelet count.

Results
Both clotting time (R time=6.4±0.6 min (M±SE), reference range (RR) =9-27 min) and fibrin cross-linking were markedly accelerated (K=1.4±0.2 min, RR=2-9; α=69.4±2.3 degrees, RR=22-58). Overall clot strength was within normal range (mean MA=63.5±1.4 mm, RR=44-64). Conventional measurements of coagulation did not deviate from normal (PT, INR and PTT=12.4± 0.2 s, 1.1±0.0, and 28.3±1.2 s, respectively), despite a pronounced thrombocytosis (441,000±24,000/μL, RR=150-400K).

Conclusions
Traumatic injury induces a hypercoagulable state in pediatric patients, as measured by thromboelastography. Four of five markers associated with a prothrombotic state (R, K, α, and platelets) were greater than the reference range values. This is the first demonstration of altered clotting kinetics in this patient population using this technology. Further study is required to assess the functional significance and mechanistic explanation for these changes.

Notes:
A PRACTICAL PRENATAL SOURCE OF AUTOLOGOUS NEURAL PROGENITOR CELLS FOR THE TREATMENT OF SPINA BIFIDA

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Purpose

It has been shown experimentally that donor neural stem cells (NSCs) can lead to some degree of fetal spinal cord repair, in a xenologous animal model. Neural cells can be found in the amniotic fluid in the setting of neural tube defects (NTDs). We sought to determine whether a progenitor population of neural cells could also be found therein in this setting, as a prerequisite for preclinical developments.

Methods

After IACUC approval, 62 pregnant Sprague-Dawley dams were divided into experimental (n=42) and control (n=20) groups, depending on prenatal exposure to retinoic acid for the induction of fetal NTDs. All animals were killed before term for analysis (n=685 fetuses). Amniotic fluid samples from both groups underwent epigenetic selection for NSC, followed by exposure to neural differentiation media. Representative cell samples underwent multiple morphological and phenotypical analyses at different time points, with rat hippocampal NSCs and rat mesenchymal stem cells serving as positive and negative controls, respectively.

Results

None of the 267 control fetuses had a structural abnormality, whereas at least one NTD was present in 52\% (217/418) of the experimental fetuses, namely either an isolated spina bifida (n=144), an isolated encephalocele (n=24), or a combination of the two (n=49). Only amniotic samples from fetuses with a NTD yielded cells with typical neural progenitor morphology and robust expression of both Nestin and Sox-2, key markers of NSCs. These cells responded to differentiation media displaying elaborate morphological changes, along with expression of Glial Fibrillary Acidic Protein (GFAP), Beta-Tubulin, and/or O4, concomitantly with Nestin downregulation, pointing to phenotypic differentiation into different neural lineages.
Conclusions
The amniotic fluid can harbor neural progenitor cells in the setting of experimental neural tube defects. The amniotic fluid may be a practical source of autologous neural progenitor cells applicable to novel forms of therapy for spina bifida.

Notes:
Purpose
The ethically sensible realm of fetal tissue engineering as a perinatal therapy can be expanded beyond life-threatening anomalies by amniotic fluid cell-based methods, in which cell procurement poses no additional risk to the mother. Congenital craniofacial defects typically lead to substantial, lifelong psychosocial consequences and meaningful morbidity. We sought to determine whether osseous grafts engineered from amniotic mesenchymal stem cells (aMSCs) could be an alternative to craniofacial repair.

Methods
After IACUC approval, New Zealand rabbits (n=12) underwent creation of a 44-56 mm² full thickness diploic nasal bone defect. Animals were then equally divided in 2 groups based on how the defect was repaired, namely size-matched implants of electrospun poly (L-lactic) acid nanofibers with or without allogeneic aMSCs. Cell processing included phenotyping by flow cytometry, labeling with green fluorescent protein (GFP), nanoscaffold seeding at identical densities, and construct maintenance in osteogenic medium for 19-20 weeks. Animals were killed 8 weeks post-implantation, for multiple analyses. Statistical analysis included ANOVA, post-hoc Bonferroni adjusted comparisons and Levene’s F-test, as appropriate (p<0.05).

Results
Closure of the defect was noted on gross inspection in all animals. Micro-CT scanning (2D and 3D) showed no significant differences in radiodensity between the groups. However, extracellular calcium levels were significantly higher in engineered grafts than in acellular implants (p=0.003), with no significant differences in alkaline phosphatase activity. Levene’s test indicated significantly greater variability in mineralization in acellular implants than in engineered grafts by both direct calcium (P=0.008) and micro-CT measurements (p=0.032). Variances were not significantly different for alkaline phosphatase activity. GFP-positive cells were documented in the engineered grafts.

Conclusions
Craniofacial repair with osseous grafts engineered from amniotic mesenchymal stem cells lead to enhanced and more consistent mineralization when compared with an equivalent acellular prosthetic repair. Amniotic fluid-derived engineered bone may become a practical alternative for perinatal craniofacial reconstruction.

Notes:
MICROCYSTIC CONGENITAL CYSTIC ADENOMATOID MALFORMATION WITH HYDROPS FETALIS: STEROIDS VERSUS OPEN FETAL RESECTION

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Background/Purpose
Congenital Cystic Adenomatoid Malformations (CCAM) are rare lesions often diagnosed during routine prenatal ultrasound. The presence of hydrops fetalis is an indicator of poor prognosis. Open fetal surgical intervention and steroids have been used in fetuses with combined CCAM and hydrops fetalis. Here we present a retrospective review of fetuses undergoing either open fetal surgery or steroids for microcystic CCAM with hydrops fetalis.

Method
A retrospective review of patients undergoing open fetal surgery or steroids for CCAM at our institution was reviewed. The primary outcome was survival to birth.

Results
A retrospective review of all patients referred to our institution with the diagnosis of CCAM was reviewed. Of this cohort, only those with the combination of microcystic CCAM and the presence of hydrops fetalis were included. A total of 13 patients were treated with a single course of steroids and 11 patients underwent open fetal surgery. There were 10/13 (77%) fetuses in the steroid group who survived to birth versus 5/11 (45%) in the open fetal surgery group. Among the patients who received steroids, 10/13 (77%) had resolution of hydrops with a mean time to resolution of 28 days. Of those who had open fetal surgery, 2/11 (18%) had resolution of hydrops with a mean time to resolution of 26 days. The mean gestational age at birth was 36.5 weeks for the steroid group and 28.9 weeks for the open fetal surgery group. Patients needing ventilator support at birth were 1/11 (0.9%) in the steroid group and 8/13 (62%) in the surgery group.

Conclusions
The present study confirms that fetuses with microcystic CCAM and hydrops fetalis benefit more from steroid treatment than open fetal surgery. Further investigation is warranted in order to evaluate the spectrum of CCAM fetuses who would benefit from steroid treatment.

Notes:
Purpose
The fetal response to injury in several tissues has been shown to be regenerative. We have recently published the first mammalian model of cardiac regeneration where the fetal response to MI results in restoration of functional myocardium. We hypothesized that following fetal MI there is decreased production of proinflammatory cytokines, decreased inflammation, and decreased oxidative stress.

Methods
After obtaining IACUC approval, anteroapical MI’s encompassing 20% of the left ventricle were created in mid-gestation fetal and adult sheep. Echocardiography was performed pre-, post-, 3 days, and 1 month after infarction. Hearts were harvested at 3 and 30 days after infarction (n=4 per group). Gene expression of IL6, IL8, SOD1, SOD2, and Catalase was assessed by microarray and real time PCR. Inflammation was assessed by immunohistochemistry for CD45.

Results
Three days post-MI, both fetal and adult infarcts demonstrated increased IL-6 and IL-8 gene expression, but adult expression was 20 and 40 fold higher than fetal, respectively. By 30 days post-MI, fetal IL-6 and IL-8 gene expression had returned to baseline whereas adult remained elevated (p<0.003). Fetal infarcts also had increased catalase gene expression at 3 and 30 days (p<0.02) and increased SOD2 expression at 30 days. Inflammatory cells were also decreased in fetal infarcts. By 30 days post-MI, fetal hearts demonstrated no akinetic myocardium and normal EF, whereas adult hearts demonstrated infarct expansion and decreased EF.
Conclusions
The regenerative fetal response to MI is associated with decreased proinflammatory cytokines, decreased inflammation, and increased oxygen radical scavengers. Understanding the mechanisms by which the fetus is able to create an environment that promotes regenerative healing and restoration of cardiac function has significant potential in the development of therapeutic strategies to promote cardiac regeneration following adult MI.

Notes:
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MATERNAL-FETAL CELLULAR SIGNALING MECHANISMS IN SEVERE CONGENITAL DIAPHRAGMATIC HERNIA

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Purpose
Congenital diaphragmatic hernia (CDH) represents a wide disease spectrum with some patients suffering neonatal demise and others showing a milder course. We studied maternal-fetal cellular trafficking and other signaling mechanisms at the maternal-fetal interface to understand factors leading to fetal/neonatal distress.

Methods
We obtained matched maternal and cord blood samples from 8 CDH patients (lung-to-head ratio (LHR) 0.65-2.0; n=2 underwent prenatal tracheal occlusion, TO) and 7 term controls. Levels of maternal cells in fetal blood (maternal microchimerism, MMc) were determined using quantitative real-time PCR for non-shared HLA-DR alleles and expressed as % maternal cells/fetal cells. Serum cytokines were analyzed in 5 CDH patients (n=3 high MMc; n=2 low MMc) and 3 normal controls using a human 39plex immunoassay kit (Millipore).

Results
MMc was low but detectable in normal controls (0.006 ± 0.004). MMc in 5 CDH patients was similarly low (0.004-0.06) but 3 patients with LHR<0.9 and neonatal demise had unusually high levels (0.1-6.5). Prenatal TO did not lead to increased MMc. Both maternal and fetal serum in patients with high MMc demonstrated increased levels of pro-inflammatory cytokines IL-1α, IL-1β, TNF-β, IL-6 (a marker of fetal inflammatory response syndrome), and IL-13. Maternal serum in high MMc patients had increased IL-10, a possible compensatory anti-inflammatory cytokine, and IL-7, a cytokine important for B and T-cell development. The chemokines MIP-1α and MCP-1 were increased in fetal serum while MIP-1α and IL-8 were increased in maternal serum, suggesting a mechanism for recruitment of maternal cells.

Conclusions
Patients with severe CDH demonstrate pro-inflammatory signals in both fetal and maternal serum at the time of birth, even before respiratory effort has begun. A careful analysis of such signals may identify prognostic indicators detectable in maternal blood or amniotic fluid to stratify fetuses who may benefit from TO as well as define developmental pathways leading to pulmonary hypertension.
Notes:
NITRIC OXIDE USE IN NEONATES WITH CONGENITAL DIAPHRAGMATIC HERNIA: A TIME-TREND ANALYSIS

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Purpose

Prospective, randomized trials have demonstrated that nitric oxide (NO) fails to improve outcomes in neonates with congenital diaphragmatic hernia (CDH). The objective of this study is to describe national trends, interhospital variability in use, and costs associated with NO utilization in neonates with CDH.

Methods

The Pediatric Health Information System (PHIS) database was queried over a seven year period (2003 - 2010) to identify all patients with a diagnosis code of CDH and a procedure code for CDH repair. Query terms included gestational age, birth weight, sex, race, insurance type, diagnoses/procedures, mortality, total hospital charges, total drug and NO charges. Patients with congenital cardiac anomalies and inaccurate NO charge data were excluded. Statistical analysis was performed with SPSS 19.0.

Results

During the study period, 3651 infants with CDH were identified at 41 PHIS hospitals. 284 with cardiac anomalies and missing /inaccurate data were excluded. Overall, mortality rate was 15% (469/3367), range 14.5-16.9%. Mortality for patients treated with NO was 47% (359/805). Mean total charge for all infants treated for CDH was $233,063,775±43,829 (median $61,775). Nearly one quarter (n=805, 24%) of neonates with CDH were treated with NO, with mean NO charges of $101,519±160,634 (median $45,924). Percentage of patients treated with NO varied significantly between hospitals (0.0-48.7%). Hospitals using the drug billed NO charges ranging from 0.1-31.2% of total hospital charges. Mortality, NO and ECMO rates over time are illustrated in the graph.

Conclusions

In the United States nitric oxide continues to be used to treat pulmonary hypertension in patients with congenital diaphragmatic hernia even though its efficacy remains to be proven. Limiting the use of nitric oxide in patients with congenital diaphragmatic hernia to clinical trials would lead to a substantial reduction in healthcare costs, and help determine if any CDH patients benefit significantly from it.
Mortality, NO, and ECMO Utilization Rates Over Time, 2003 - 2009

Rate (%)

2003 2004 2005 2006 2007 2008

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PROSTHETIC MESH PLUG FOR NEONATAL THORACOSCOPIC CONGENITAL DIAPHRAGMATIC HERNIA REPAIR: OUTCOMES OF A NEW TECHNIQUE
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Purpose
Neonatal thoracoscopic repair of Congenital Diaphragmatic Hernia (T-CDH) is being more frequently performed. While several studies demonstrate thoracoscopic placement of a conventional prosthetic patch as a viable option, concerns persist over technical difficulty and applicability. Alternate types and methods of prosthetic placement may alleviate this. We describe our outcomes when utilizing a pre-formed, cone-shaped prosthetic mesh plug and compare it to our own experience using a flat patch for T-CDH.

Methods
From 2004 to 2010 we performed 53 consecutive T-CDH repairs, 25 of which were repaired with a prosthetic plug or patch. Comparative demographics examined included sex, gestational age, birth weight, percent outborn, side of CDH, age at repair, liver or stomach in chest, and ECMO utilization. Outcome measures evaluated were operative time, blood loss, minor and major complications, and recurrence.

Results
A plug was utilized in 15 (60%) neonates and a flat patch in 10 (40%). Demographic characteristics were similar between the two groups. When compared to those who received a patch, neonates who had a plug placed had significantly decreased operative times (216 vs. 159 minutes, p=.02), blood loss (37 vs. 10 milliliters, p=.04), and minor complications (78 vs. 33%, p=.04). There was no significant difference in major complications including post-op mortality (30 vs. 13%, p=.35) or recurrence rates (20 vs. 13%, p= 1.0) over a median follow-up of 3.2 years. All recurrences were repaired using minimal access techniques.

Conclusions
This is the largest series of prosthetic T-CDH repairs yet reported and demonstrates that outcomes for T-CDH with a plug compare favorably to those performed with a patch. Potential benefits of the plug may be related to the configuration of the plug, which obviates the need for prosthetic sizing, requires fewer sutures for securing, and decreases the need for unfurling of the diaphragm as compared to the patch.

Notes:
ABDOMINAL WALL MUSCLE FLAP IS SUPERIOR TO PATCH REPAIR OF LARGE CONGENITAL DIAPHRAGMATIC HERNIAS

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**Purpose**

Large congenital diaphragmatic hernias (CDH) require a patch or an abdominal wall muscle flap to close the defect. We sought to compare the diaphragmatic hernia recurrence rate among patients repaired with a synthetic patch, acellular dermal matrix, and abdominal wall muscle flap.

**Methods**

A retrospective review of neonates with CDH at a tertiary care children’s hospital was performed (1999-2010). Patients in whom primary repair was not possible were selected for review. Defect sizes in survivors requiring muscle flap or patch repair were classified according to the congenital diaphragmatic hernia registry (A-D). Statistical analyses were performed to compare groups who survived to discharge.

**Results**

158 neonates were identified with an overall survival of 85%. The recurrence rate in those repaired primarily was 2% (median follow-up 5.8 years). Primary repair was not accomplished in 46 patients (29%). Twenty-eight patients were repaired with a muscle flap and 18 with a patch (13 synthetic; 5 dermal matrix). There was no statistical difference in survival between the muscle flap and patch group (82% vs. 55%, \(p=0.09\)). The defect size in the muscle flap group were B (1, 4.4%), C (17, 74%), and D (5, 22%) whereas the patch group were C (9, 90%) and D (1, 10%). Hernia recurrence (median follow-up 3.6 years) occurred in 4.4% of the muscle flap group (1) and 50% (5) in the patch group (\(p=0.005\)). No difference was demonstrated in hernia recurrence between the synthetic and dermal matrix groups (\(p=1.0\)). The single muscle flap recurrence occurred 3.8 years post operatively, and the median time to recurrence in the patch group was 185 days.

**Conclusions**

Abdominal wall muscle flap is an effective and durable method of repairing large congenital diaphragmatic hernias. The recurrence rate following muscle flap repair is similar to primary repair and superior to repairs using synthetic or dermal matrix products.
SCIENTIFIC SESSION III

Kaplan-Meier: Time to Recurrent Hernia

Muscle flap repair

Patch repair

p < 0.0009 by Log-Rank Test

Time in Weeks

Notes:
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INFLUENCE OF LOCATION OF DELIVERY ON OUTCOME IN NEONATES WITH GASTROSCISIS
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Purpose
It is not clear in the literature whether infants with a prenatal diagnosis of gastroschisis should be delivered in a perinatal centre with level 3 NICU and surgical facilities (“inborn”), or if they could be safely delivered in a more local hospital and then transferred to a perinatal centre (“outborn”). Our goal was to determine the impact of delivery site on outcomes for neonates diagnosed with gastroschisis.

Methods
Data were obtained from The Canadian Pediatric Surgery Network, covering the years (2005-2008) over 18 pediatric surgical centres. “Inborn” was defined as birth in a hospital with a NICU or connected to a NICU by a bridge or tunnel. “Outborn” was defined as requiring transfer by ambulance or flight. A p value of < 0.05 was considered to be significant.

Results
Of 395 infants with gastroschisis, 237 were inborn and 158 were outborn. Univariate analysis demonstrated no significant difference between groups with respect to gestational age, birth weight, days on TPN or length of hospital stay. There was a significant difference with regards to SNAP II score, complication rates, comorbidities and age at final closure. Logistic regression showed that location of delivery was a significant independent predictor for incidence of complications, as were SNAP II, comorbidities, and presence of bowel atresia or necrosis. The odds ratio of developing a complication when outborn was 1.6 (p=0.05).

Conclusions
Delivery outside of a perinatal centre is a significant predictor of complications for infants born with gastroschisis.

Notes:
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EFFECTS OF GLUTAMINE SUPPLEMENTATION ON PLASMA AMINO ACIDS IN SURGICAL INFANTS RECEIVING PARENTERAL NUTRITION

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Purpose

Parenteral nutrition (PN) is necessary for many surgical infants. Recently, there has been interest in glutamine supplementation of PN for prevention of sepsis and decreasing time to full enteral feeds. However, the effects of glutamine on plasma amino acids levels is unknown. Our aim was to determine the effects of glutamine supplementation on plasma amino acids.

Methods

An ethically-approved double-blind multi-centre randomised controlled trial was performed in surgical infants <3 months old. Glutamine group received 0.4g/kg/day alanyl-glutamine PN supplementation until full enteral feeding was reached, up to a maximum of 3 months. Placebo received isonitrogenous isocaloric PN. Plasma samples were taken for amino acid analysis in centres which guaranteed appropriate sample storage and transport. Amino acid trends over time were modelled by multi-level regression modelling (MLWin) and adjusted for diagnosis, gestational age, length of bowel resected and presence/absence of the ileocecal valve. Estimates are given as mean±SEM.

Results

174 infants were randomised (87 glutamine, 87 placebo). Amino acid data were available on 81 patients (40 glutamine, 41 placebo). There was a significant increase in levels of essential amino acids (increase of 67.8± 31.4 micromolar per week of PN, p=0.03) and branched chain amino acids (increase of 53.1± 16.8 micromolar per week of PN, p<0.001) over time. Glutamine supplementation, however, was associated with a relative decrease in both essential amino acids (-123.3± 55.4 micromolar per week of PN, p=0.026) and branched chain amino acids (-76.0±9.8 micromolar per week of PN, p=0.01).

Conclusions

Glutamine supplementation has significant negative effects on plasma amino acid concentrations. This is probably because, in order to make the PN solutions isonitrogenous, the concentration of other amino acids in PN has to be decreased. This finding has important implications for growth and muscle function, with the potential benefits of glutamine supplementation balanced against the potential negative effects of sub-optimal amino acid levels.

Notes:
THE ROLE OF ULTRASOUND IN THE DIAGNOSIS AND MANAGEMENT OF NECROTIZING ENTEROCOLITIS

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Purpose
Ultrasound (US) has been previously shown to detect abnormalities related to necrotizing enterocolitis (NEC); these findings may influence clinical care. The purpose of this study was to assess the role of US in the diagnosis and clinical management of NEC.

Methods
We retrospectively evaluated the clinical and imaging findings in 39 consecutive neonates with NEC, who were admitted to a tertiary level neonatal intensive care unit. Radiographs were analyzed for bowel dilatation, elongation, separation, intramural gas, portal venous gas and pneumoperitoneum. US were evaluated for free fluid, intramural gas, portal venous gas, pneumoperitoneum, bowel wall thickness and perfusion, and peristalsis. Patients were assessed based on how the US findings helped to establish the diagnosis and/or influenced the clinical management.

Results
Seven (18%) of the 39 patients only required radiographs, as this was sufficient for diagnosis and clinical management (intramural gas in 4, pneumoperitoneum in 3). In the remaining 32 cases, US were performed in addition to radiographs (within 26 hours of the radiograph study) to assist with the diagnosis of NEC in 17/32 (53%) and assist with clinical management in 15/32 (46%). US findings were able to establish the diagnosis of NEC in 13/17 (76%) and changed clinical management of NEC in 10/15 (67%), including 7/15 (47%) who were directed to surgical interventions (laparotomy and peritoneal drainage). Thus, US provided additional information for diagnosis and/or clinical management in 23/32 (72%) of those neonates who had US and 23/39 (59%) of all neonates in this study.

Conclusions
US plays an important role as a valuable adjunct to plain radiography in the diagnosis and/or clinical management of a significant number of patients with NEC, particularly when radiographic findings are non-specific, or discordant with the clinical picture. In the future, US may become a fundamental component of the care of neonates with NEC.

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DURATION OF SYMPTOMS PRE-KASAI PORTOENTEROSTOMY, NOT AGE AT KASAI, IS PROGNOSTIC IN BILIARY ATRESIA.

Momoko Wada, MD, Hiroki Nakamura, MD, Hiroyuki Koga, MD, Go Miyano, MD, Rafael H. Dizon, MD, Geoffrey J. Lane, MD, Yoshifumi Kato, MD, Tadaharu Okazaki, MD, Atsuyuki Yamataka, MD

Juntendo University School of Medicine, Tokyo, Japan

Purpose

The prognostic importance of the age at Kasai portoenterostomy (KPE) in biliary atresia (BA) is now being challenged. We examine the age at onset, age at KPE and duration of symptoms as more relevant prognostic factors.

Methods

We treated 74 consecutive BA patients with KPE between 1989 and 2010. 1989 was chosen since that was when LTx became available in Japan, and so that we could focus only on cases in the LTx era in order to minimize bias in the findings. Medical records were reviewed to evaluate: age at onset of symptoms (<31 days: n=40, 31-60 days: n=24, >60 days: n=10), age at KPE (<31 days: n=6, 31-60 days: n=23, >60 days: n=45), and duration of symptoms pre-KPE (<31 days: n=38, 31-60 days: n=24, >60 days: n=12). Age at onset was defined as the age when the first acholic stool was recorded. For each factor, the ratio of patients becoming jaundice-free (total serum bilirubin < 1.5mg/dL) and the ratio of survivors without LTx were compared statistically using the Chi-squared test.

Results

Data are shown in Table 1. We found a significant relationship between duration of symptoms and the ratio of patients who survived without LTx (p=0.03). The ratio was reduced when duration was over 60 days. Age at onset, age at KPE, and duration of symptoms pre-KPE did not affect the jaundice-free ratio.

Conclusions

We are the first to show that duration of symptoms pre-KPE may be more prognostic than age at KPE. Our data did not support KPE at an early age, but suggest that KPE should not be delayed after diagnosis.
## Table 1

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<td><strong>Age at KPE</strong></td>
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<td>Jaundice-free</td>
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**Notes:**
Basic Science
Tuesday, May 24, 11:00 a.m. – 12:15 p.m.

33 CYCLOSPORINE IMPROVES MESENTERIC PERFUSION AND ATTENUATES NEC-LIKE INTESTINAL INJURY IN ASPHYXIATED NEWBORN PIGLETS DURING REOXYGENATION
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Purpose
Asphyxia-related intestinal injury in newborns may present similar to necrotizing enterocolitis (NEC). The intestinal injury in both asphyxiated neonates and NEC is partially associated with hypoxia-reoxygenation injury, resulting in a final common pathway ending with cell death. Cyclosporine has been shown to reduce myocardial cell death following ischemia-reperfusion. We hypothesize that cyclosporine treatment may attenuate NEC-like intestinal injury in asphyxiated newborn piglets during reoxygenation.

Methods
Thirty-six newborn piglets (1-4 days-old) were acutely anesthetized and instrumented for continuous monitoring of cardiac output, mean arterial pressure, and superior mesenteric arterial flow. After stabilization, normocapnic alveolar hypoxia (10-15% oxygen) was instituted for 2h followed by reoxygenation with 100% oxygen for 0.5h, then 21% for 3.5h. The piglets were blindly block-randomized to receive one of three cyclosporine boluses intravenously (2.5, 10 or 25 mg/kg) or normal saline solution as a placebo (control) after 5 minutes of 100% reoxygenation (n=8 each). A non-asphyxiated, sham-operated group was included (n=4) to control for effects of the surgical model. Intestinal samples were collected for lactate concentration and histological assessment (Park’s criteria).

Results
At 2h of hypoxia, piglets had cardiogenic shock (cardiac output 45% of baseline), hypotension (mean arterial pressure of 30mmHg), acidosis (pH=7.04) and decreased superior mesenteric perfusion (all p<0.05 compared to sham group, ANOVA). Cyclosporine (2.5 and 10 mg/kg) treatment significantly improved SMA flow (106% and 97% of baseline vs. 60% of baseline in controls, respectively). SMA oxygen delivery improved with cyclosporine-treatment and small-bowel lactate was decreased compared to controls (P<0.05). NEC-like injuries were found in some control piglets, including pneumatosis and hemorrhagic...
infarct. Histological injury was attenuated in all cyclosporine-treated groups compared to controls (P<0.05).

Conclusion
We are first to demonstrate that post-resuscitation administration of cyclosporine causes improvement in mesenteric perfusion and attenuates NEC-like intestinal injury in newborn piglets following asphyxia-reoxygenation.

Notes:
HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) IMPROVES INTESTINAL BARRIER FUNCTION AND REDUCES MORTALITY IN A MURINE MODEL OF PERITONITIS

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Purpose

The morbidity and mortality associated with bacterial peritonitis remain high. Heparin-binding EGF-like growth factor (HB-EGF) is a potent intestinal cytoprotective agent. The aim of this study was to evaluate the protective effects of HB-EGF in a model of murine peritonitis.

Methods

HB-EGF(-/-) knockout (KO) mice and their HB-EGF(+/-) wild type (WT) counterparts underwent either sham surgery or cecal ligation and puncture (CLP). A cohort of HB-EGF KO mice underwent CLP with administration of exogenous HB-EGF (800μg/kg IP daily). Villus length, intestinal permeability, intestinal epithelial cell (IEC) apoptosis and survival were determined. Bacterial cultures from peritoneal fluid and mesenteric lymph nodes (MLN) were obtained.

Results

After exposure to CLP, HB-EGF KO mice had significantly shorter villi (1.37±0.13 vs. 1.96±0.4 relative units, p<0.03) and increased IEC apoptosis indices (0.0093±0.0033 vs. 0.0016±0.0014, p<0.01) than those of WT mice. Intestinal permeability in HB-EGF KO mice undergoing CLP was significantly increased compared to that of WT mice (17.01 ± 5.18 vs. 11.50 ± 4.67 nl/min/cm2, p<.03). After CLP, HB-EGF KO mice had increased bacterial counts compared to WT mice in both peritoneal fluid (25,313 ± 17,558 vs. 11,955 ± 6,653 CFU/ml) and MLN (19,009 ± 11,200 vs. 5,948 ± 2,988 CFU/ml/g). Administration of exogenous HB-EGF led to significantly decreased bacterial counts in MLN in both HB-EGF KO and WT mice exposed to CLP (p<0.01). Upon exposure to CLP, HB-EGF KO mice had significantly decreased survival compared to WT mice (p<0.01). Survival of HB-EGF KO mice subjected to CLP was significantly improved upon treatment with exogenous HB-EGF (p<0.01).

Conclusions

HB-EGF gene knockout increases susceptibility to peritonitis-induced intestinal injury, with decreased villous length, increased IEC apoptosis, increased intestinal permeability, and increased mortality. Administration of exogenous HB-EGF to HB-EGF KO mice undergoing CLP decreases bacterial translocation and increases survival. These results support a protective role of HB-EGF in peritonitis-induced sepsis.

Notes:
Introduction
Necrotizing enterocolitis (NEC) is the leading gastrointestinal cause of morbidity and mortality in the neonatal population. Enterobacter sakazakii (ES) is a gram-negative opportunistic pathogen that has been implicated in outbreaks of NEC in neonatal ICUs around the world. We have previously demonstrated that ES administration to 3-day old breast-fed mouse pups results in up-regulation of TGFβ in the intestine, and morphological changes characteristic of NEC. We hypothesize that ES mediates intestinal injury through a TGFβ-dependent pathway.

Methods
3-day-old, breast-fed C57Bl6 mouse pups were administered 10^3 CFUs of ES. The pups’ intestines and serum were collected post mortem. Cytokine levels were measured using ELISA. Intestinal epithelial cells were isolated by Percoll gradient and separated by FACS analysis for E-cadherin expression using primary antibody. TGFβ receptor expression was determined in a similar fashion. The experiments were repeated in vitro using CaCo2 cells. siRNA to TGF β protein and its receptors were used to knock down TGFβ/TGFβ receptor expression. Enterocytes were subsequently infected with ES. Apoptosis was measured using Propidium Iodine staining. Tight junction integrity was assessed in CaCo2 monolayers in the absence of TGFβ activity post ES infection using Trans-epithelial electrical resistance (TEER) and HRP permeability.

Results
ES infection significantly increased TGFβ levels in the serum and intestinal mucosa of infected pups. (Figure 1A) Similarly, TGFβ receptor levels are also increased in the intestinal mucosa both in vivo (Figure 1B) and in vitro. Knocking down TGFβ or TGFβ receptor expression abrogated ES-induced apoptosis and epithelial barrier injury.

Conclusion
ES infection up regulates TGFβ activity. TGFβ protein appears to be required for ES-induced apoptosis and gut barrier injury. We conclude that TGFβ plays a key role in ES-induced NEC.
A: TGFβ protein expression is up-regulated following OmpA+ ES infection in these animals in a time dependent fashion.
B: TGFβ receptor expression is up regulated in the intestinal mucosa of OmpA+ infected animals.

Notes:
OVER-EXPRESSION OF HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) PROTECTS INTESTINAL MYENTERIC PLEXUS NEURONS FROM ISCHEMIA/REPERFUSION INJURY

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Purpose

Impaired intestinal motility is an important cause of significant morbidity after necrotizing enterocolitis (NEC). The intestinal dysfunction that is present after successful treatment of NEC suggests that the compromised enteric nervous system (ENS) is not fully recovered from the intestinal insult. Heparin-binding EGF-like growth factor (HB-EGF) is a neurotrophic factor for injured neuronal cells. The goal of this study was to assess the neuroprotective effects of HB-EGF over-expression on myenteric plexus neurons after ischemia/reperfusion (I/R) injury.

Methods

HB-EGF transgenic (TG) and wild type (WT) mice were subjected to 1h of superior mesenteric artery occlusion followed by reperfusion. PGP9.5 whole mount immunohistochemistry (IHC) of the distal ileum was performed to study morphological changes of myenteric plexus ganglia at 1 and 7 days postoperatively. Apoptotic neurons were identified by caspase-3 IHC and the apoptotic rate was calculated as caspase-3 positive neurons/total neurons. Western blotting was used to evaluate the expression levels of neuronal nitric oxide synthase (nNOS) and acetylcholinesterase, two major neural regulators of gastrointestinal motility, and postsynaptic density protein 95 (PSD95), a scaffolding protein involved in synapse formation.

Results

WT mice subjected to intestinal I/R injury had significant neuronal degeneration in the myenteric plexus ganglia, characterized by neuronal debris, distorted neurites and apoptotic neurons. HB-EGF TG mice had preserved structural integrity of the myenteric plexus, with significantly decreased apoptotic indices after I/R injury compared to WT mice (3.4±1.2% vs. 12.7±2.3%, p<0.05). WT mice had significantly decreased expression of nNOS and PSD95, but not acetylcholinesterase, after I/R injury. However, HB-EGF TG mice had preserved nNOS and PSD95 protein production after injury.

Conclusion

HB-EGF over-expression promotes nNOS and PSD95 expression, and protects myenteric plexus neurons, after intestinal I/R-induced neuronal injury. These findings support the possibility that HB-EGF may preserve neurogenesis in the ENS, and improve gastrointestinal motility, in patients with intestinal I/R injury, including NEC.

Notes:
RAPAMYCIN-INDUCED TUMOR VASCULATURE REMODELING IN RHABDOMYOSARCOMA XENOGRAFTS INCREASES THE EFFECTIVENESS OF ADJUVANT IONIZING RADIATION

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Purpose

Rapamycin is a macrolide antibiotic that affects the mTOR signaling pathway. It has pleotropic antitumor effects including the inhibition of VEGF production. Inhibition of VEGF may cause transient “normalization” of tumor vasculature by pruning away the most immature vessels, resulting in transient improvement in tumor perfusion and oxygenation. These effects should potentiate the antitumoral effects of adjuvant ionizing radiation because its activity is critically dependent upon target tissue oxygenation.

Methods

Mice bearing orthotopic Rh30 alveolar rhabdomyosarcomas were initially treated with rapamycin (5 mg/kg IP daily x5) or vehicle control. Tumors were then evaluated for changes in intratumoral oxygenation, perfusion, vessel permeability and microvessel density. Additional mice were treated with 5 daily doses of rapamycin, 1 dose of irradiation (4Gy), or 5 doses of rapamycin with irradiation administered on the 6th day, and evaluated for changes in tumor growth 14 days after the completion of therapy.

Results

Tumor vessel permeability changed only minimally (6.6±0.97vs.7.2±1.3mcg, p=0.75) but microvessel density (3,153±932vs.20,477±3,717.9pixels/HPF, p=0.0003) decreased and intratumoral oxygenation increased significantly after 5 doses of rapamycin (0.0385±0.0141vs.0.0043±0.0023mmHg/mm3, p=0.007). This effect abated 5 days after the end of treatment, however. Contrast-enhanced ultrasound demonstrated increased signal intensity (113.4±14.9vs. 94.0±6.6dB, p=0.27), a measure of tumor perfusion, and a significantly increased rate of change of signal intensity after 5 days of rapamycin (6.6±0.8vs.4.2±0.4dB/sec, p=0.03). Average increase in tumor volume 14 days after treatment was smallest in mice treated with the combination of rapamycin and irradiation (423±89mm3) as compared to rapamycin alone (648±139mm3), irradiation alone (660±90mm3) and control (1,864±145mm3).

Conclusion

Combination therapy with rapamycin given prior to irradiation appears to normalize tumor vasculature, improving tumor perfusion and oxygenation at the time of irradiation and increasing tumor sensitivity to adjuvant irradiation in alveolar rhabdomyosarcoma.

Notes:
38
THE MATERNAL ADAPTIVE IMMUNE RESPONSE AGAINST PATERNAL ANTIGENS INCITES FETAL DEMISE AFTER FETAL INTERVENTION
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Purpose
Activation of the innate immune system has been described as the primary mechanism of preterm labor (PTL) after fetal intervention. We have previously demonstrated that maternal T cells limit engraftment after in utero hematopoietic cell transplantation (IUHCTx) in mice. We hypothesized that the maternal adaptive immune system may also lead to PTL and the demise of allogeneic fetuses after fetal intervention.

Methods
BALB/c (H-2\textsuperscript{d/d}) females were bred to BALB/cxB6 (H-2\textsuperscript{d/b}) males, such that the fetuses were either H-2\textsuperscript{d/d} (syngeneic to the mother) or H-2\textsuperscript{d/b} (semi-allogeneic to the mother). The survival of fetuses after IUHCTx of B6 (H-2\textsuperscript{b/b}, paternal antigen) or C3H (H-2\textsuperscript{k/k}, third-party antigen) donor cells was determined. As a control, H-2\textsuperscript{d/b} mothers were bred to H-2d/d fathers such that all of the pups were syngeneic to the mother.

Results
Among uninjected fetuses (n=35), 43% of the surviving mice were H-2\textsuperscript{d/b} and 57% were H-2\textsuperscript{d/d}. After IUHCTx of H-2b/b (n=20), there was a striking decrease in the percentage of surviving H-2\textsuperscript{d/b} pups (25%) compared to H-2\textsuperscript{d/d} pups (75%, p <0.05). This selective loss of semi-allogeneic fetuses was not seen when T cells were selectively removed from the mother (n=10), indicating a crucial role for maternal T cells in inciting fetal demise. When third party antigen was transplanted (n=15), there was equivalent survival of H-2\textsuperscript{d/b} (47%) and H-2\textsuperscript{d/d} (53%) offspring, indicating the antigen specificity of the maternal immune response in PTL. Furthermore, when the mother was H-2\textsuperscript{d/b}, there was no survival disadvantage of H-2\textsuperscript{d/b} pups after IUHCTx of H-2\textsuperscript{b/b}.

Conclusions
In utero transplantation of allogeneic cells triggers an antigen-specific maternal adaptive immune response, resulting in PTL and selective loss of semi-allogeneic fetuses. This is the first description of the role of the adaptive immune system in PTL and suggests that treatment strategies directed towards suppressing this response may be beneficial after fetal intervention.
Notes:
Purpose
The enteroendocrine hormone Glucagon-like 2 peptide (GLP-2) is an important regulator of intestinal function, and may control adaptation following resection. However, little is known about the expression of GLP-2 and the GLP-2 receptor in infants with intestinal failure (IF).

Methods
With ethics approval from Sept 06-Sept 10, IF infants were prospectively enrolled and underwent monitoring of nutritional status, measurement of GLP-2 levels, and tissue sampling for GLP-2 receptor analysis (if operation was undertaken). Controls were solicited from inpatients admitted for non-intestinal illness.

Results
11 controls and 28 IF patients were enrolled. All infants required significant feeding to stimulate GLP-2 production. Infants with short bowel (<40 cm) or gastroschisis (GSC) had reduced fasting levels of GLP-2 (14±0.7 vs. controls 28±6 pmol/L). In GSC over time GLP-2 levels increased (peak 92.6 ± 12.1) and paralleled discontinuation of PN. Long term (3-12 months), the post prandial GLP-2 levels decreased significantly (52 ±6, p <0.001). In infants with SBS if peak GLP-2 levels exceeded 30 pmol/l, those infants were able to be weaned from PN. GLP-2 receptor (GLP-2r) expression increased with age in the ileum and colon; in intestine out of the enteric stream (distal stomas), the GLP-2r expression was decreased. In gastroschisis infants, receptor expression was remarkably increased vs. age matched controls (41±31 vs. controls 0.91±0.74 by quantitative RT-PCR). (All data mean ±SD).

Conclusions
Infants with IF have an altered pattern of GLP-2 production. There is an initial low GLP-2 output, followed by hyperresponsive output and then normalization as tolerance of enteral nutrition increases. In all infants, expression of the GLP-2 receptor increases over time. The receptor expression is increased in gastroschisis patients. These findings require further study, but suggest a feeding regimen to increase endogenous GLP-2 production and that exogenous GLP-2 may be useful therapeutically in infants with IF.

Notes:
Purpose
Short bowel syndrome in children leads to severe morbidity and mortality. Current therapies do not offer adequate palliation. In previous work, we used tissue-engineered small intestine (TESI) to rescue Lewis rats that underwent massive small bowel resection. In the mouse model, we showed that the epithelium of TESI derived from the donor implanted cells while the mesenchyme was from both donor and host origins. The purpose of this work is to identify the bone marrow stem cell contributions to the mesenchyme of TESI.

Methods
C57BL/6J wild type mice underwent Green Fluorescence Protein (GFP) bone marrow transplantation. After confirmation of engraftment, the mice were implanted with TESI by implanting polymers seeded with organoid units, multicellular clusters of epithelial and mesenchymal cells, derived from full-thickness small intestine of two-week-old C57BL/6J wildtype mice. Organoid units were isolated using a variation of the previous protocol reported in the Lewis rat. TESI was harvested after 4 weeks. Immunohistochemistry was performed to identify the origin of each mesenchymal cell type.

Results
Tissue-engineered small intestine was successfully generated. As expected, the epithelium was GFP negative while a major part of the mesenchyme was GFP positive (figure A). However, co-immunofluorescence staining with smooth muscle actin (SMA) to identify the intestinal subepithelial myofibroblasts (mesenchymal cells of the stem cell niche), or with SMA and Desmin to identify the muscularis demonstrated no contribution of the bone marrow stem cells to those cell types (figure B).

Conclusion
Although previous lineage tracing demonstrated a host contribution to the mesenchyme of TESI, these data suggest that all the important cell types necessary for a functional engineered tissue may grow only from the donor implanted cells. Further evaluation of the cellular and molecular mechanisms involved during the formation of tissue-engineered intestine will direct the most efficient conditions to establish future human therapies.
SCIENTIFIC SESSION IV

Notes:
A SIMPLIFIED PROTOCOL FOR HUMAN MESENCHYMAL AMNIOCYTE-INDUCED PLURIPOTENT STEM CELL MAINTENANCE: IMPLICATIONS FOR DISEASE MODELS AND CLINICAL APPLICABILITY
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Purpose
Amniotic mesenchymal stem cells (aMSCs) have emerged experimentally as a valuable resource for cell and tissue replacement strategies in the perinatal treatment of surgical disease. These cells have also been shown to be an accessible target for induced pluripotent stem cell (iPS) reprogramming, albeit through labor-intensive, costly and clinically unwieldy methodologies. We aimed at determining whether iPS-aMSCs could be maintained and expanded in the absence of a mouse embryonic fibroblast (MEF) feeder layer, so as to facilitate their use as platforms for models of human development and disease, as well as eventual therapeutic applications.

Methods
After IRB approval, expanded human aMSCs, characterized by comprehensive flow cytometry, were reprogrammed by ectopic expression of six transcription factors previously shown to enhance iPS generation efficiency, namely OCT4, SOX2, MYC, KLF4, hTERT and SV40 Large T. During reprogramming, all cells were cultured on a MEF layer, and then divided into two groups. In one group, no additional manipulations were performed. In another, cells were transitioned to a MEF-free environment, on plates pre-coated with a solubilized basement membrane preparation containing numerous extracellular matrix proteins, cytokines and growth factors (Matrigel). At passage 7, viable cells from both groups were characterized by immunohistochemistry for multiple markers of a primitive pluripotent state shared with human embryonic stem cells.

Results
Feeder-free iPS-aMSCs showed similar morphology and expansion kinetics to those maintained under traditional culture conditions. Both Matrigel- and MEF-grown cells stained for all the markers of an embryonic stem cell-like state, namely Tra-1-81, Tra-1-60, SSEA3, SSEA4, OCT4, and NANOG.

Conclusions
Reprogrammed amniotic mesenchymal stem cells can be successfully expanded in a feeder-free environment, while maintaining an undifferentiated, embryonic-like pluripotent state. These findings will contribute to the use of these cells in models of human development and disease, as well as lend further support to their eventual clinical viability in regenerative therapies.

Notes:
INNOVATION SESSION

Abstracts on New and Innovative Techniques and Procedures
Tuesday, May 24, 12:30 – 1:30 p.m.

i1
INNOVATIVE PEDIATRIC SURGERY: DO WE NEED INSTITUTIONAL REVIEW BOARD APPROVAL?
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Purpose
Regulation of surgical innovation is controversial, especially regarding vulnerable populations like children. Prior to 2000, Institutional Review Board (IRB) approval of pediatric biomedical research was documented in a minority of manuscripts. We hypothesize that recent manuscripts regarding innovative pediatric surgery still fail to document approval.

Methods
Medline search for innovative pediatric surgery manuscripts was conducted. Articles not related to innovative pediatric surgery, not written in English, published before 2005, and reviews were excluded. Relevant articles were classified by: study focus (new device, innovative technique, modification of existing procedure), study location (US, international), number of subjects, retrospective/prospective design, children’s hospital, author’s sub-specialty, academic/private institution, and documented approval. “Instructions to Authors” section of all journals was surveyed for IRB approval requirement for publication. Independent variables were analyzed via single predictor binary logistic regression analyses. Multivariate binary logistic regression analysis was used to identify variables that predict IRB approval.

Results
1290 articles were identified. 262 met inclusion criteria. 85% of journals required approval in “Instructions to Authors,” however, only 33% of manuscripts documented approval. Positive univariate predictors of documented approval included number of subjects: n>159 (OR=1.006, 95% CI:1.002-1.010, p<0.01), international study (OR 2.8, 95% CI:1.6-4.8, p<0.001), study focus: new device (OR 2.7, 95% CI:1.1-6.8, p<0.05), and surgical sub-specialty: ENT (OR 17.9, 95% CI:3.8-8, p<0.001). Retrospective/prospective design, children’s hospital, academic/private, and required IRB approval were not significant predictors of documented approval. Multivariate binary logistic regression analysis indicated number of subjects: n>159 (OR=1.008, 95% CI:1.002-1.013, p< 0.01), US-based study (OR 3.6, 95% CI:1.9-7.1, p<0.001), and surgical sub-specialty: ENT (OR 4.8, 95% CI:1.6-14.6, p<0.01) were significant predictors of documented approval.
INNOVATION SESSION

Conclusions
Despite requirement of IRB approval for publication, only a minority of articles on innovative pediatric surgery document approval. Number of subjects (n>159), US-based study, and author’s sub-specialty (ENT) independently predicted documented approval. We speculate that lack of documentation of approval reflects limited guidelines on what constitutes surgical research and possibly a belief among surgeons that IRBs are not an appropriate forum to review innovative surgical procedures.

Notes:
INNOVATION SESSION

i2

A NOVEL ANTI-REFLUX PROCEDURE: GASTROPLASTY WITH RESTRICTED ANTRUM TO CONTROL EMESIS (GRACE)

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Purpose

Nissen Fundoplication (NF) is the most commonly performed operation to treat severe gastroesophageal reflux disease (GERD) and vomiting in neurologically impaired (NI) children. However, failure rates of NF in this population are higher, and alternatives to NF have technical and functional drawbacks. We hypothesize that a novel anti-reflux procedure, GRACE, would be superior to NF at reducing emetic reflux.

Methods

With IACUC approval, a prospective randomized study was designed to test GRACE versus NF in a canine model. Subjects underwent initial gastrostomy tube placement. Baseline gastric emptying and induced vomiting studies were performed. Subjects were then randomized to GRACE or NF. GRACE tubularizes the stomach along the lesser curvature with a divided staple line, creating a barrier to emesis and a gastric reservoir for feeding (figure). After GRACE or NF, gastric emptying and vomiting studies were repeated to assess physiologic function and reduction of emetic reflux. Statistical comparisons were made using t-test and multivariable regression; p < 0.05 was considered significant.

Results

Fifteen dogs were randomized to GRACE or NF. Gastric emptying before and after anti-reflux procedures was not significantly different between groups (p=0.4), indicating preservation of gastric function. Post-operatively, both NF and GRACE procedures significantly prevented reflux compared to baseline: NF decreased reflux amounts on average by 38% (p=0.04) and GRACE by 69% (p=0.0005). However, when compared to each another, the GRACE procedure reduced emetic reflux significantly more than the NF did (p=0.03).

Conclusions

GRACE outperforms NF in reducing emetic reflux in a canine model. This novel procedure preserves gastric function and is well-tolerated. Given the long-term failure rates of NF, we believe that the GRACE procedure may provide an innovative, safe, and superior alternative to NF as a primary or repeat anti-reflux procedure for GERD in NI children.
Notes:
EVALUATION OF INTESTINAL VIABILITY USING 3-CCD (CHARGE COUPLED DEVICE) IN CHILDREN UNDERGOING APPENDECTOMY

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Introduction
The surgeon’s ability to determine intestinal viability is limited to subjective measures of visual clues of perfusion. This inability to accurately determine viability presents a conundrum for the surgeon. In our laboratory, we use 3-CCD technology to separate visible light into its three primary wavelengths; by using an easily implemented mathematical algorithm, the amount of light detected by the CCDs can be directly correlated with tissue oxygenation. In this study, we report the use of 3-CCD technology to determine intestinal perfusion by using an appendectomy model for ischemia.

Methods
In this study, we sequentially recorded 10 laparoscopic appendectomies for appendicitis. In brief, the recorded images are analyzed by selecting three regions of interest (ROIs) and evaluating the intensity levels (a.u.) at various locations along the appendix. Figure 1A demonstrates the enhanced image of an ischemic appendix during an appendectomy. The black box indicates the fat ROI, the white boxes indicate the appendix ROIs, and the dashed black boxes indicate the colon ROIs. The colon was used as a control for normal perfusion and a ratio to fat was performed to normalize the data.

Results
As an indication of decreased perfusion, the appendix demonstrated a significant reduction in mean ROI values over time. Figure 1B represents fat normalized ROI intensity values calculated for the colon (R² = 0.02), appendix (R² = 0.92), and distal appendix (R² = 0.89).

Conclusions
In this primary study, we have demonstrated proof of principle for 3-CCD technology to determine bowel ischemia. We have conclusively demonstrated reduced intensity levels in areas of known ischemia. Given the ability of this technology to identify areas of ischemia, this technique has the potential to significantly change the management of malrotation with volvulus, necrotizing enterocolitis and intestinal reconstruction in the future.
INNOVATION SESSION

Notes:
i4
DEVELOPMENT OF AN ENDOLUMINAL INTESTINAL LENGTHENING CAPSULE
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Purpose
Prior studies demonstrated the ability to lengthen intestinal segments with mechanical force using a spring-loaded device implanted in an isolated segment of small bowel. This required the use of a dissolvable suture to deploy the spring; however, this occurred in an unpredictable manner. We make two innovations to this technique. First, we implemented the use of a gelatin capsule coated with a cellulose polymer to control delivery of the device. Second, the force of the spring was decreased to allow for slower expansion of the intestine.

Methods
Nickel titanium springs with varying forces were compressed and placed in gelatin capsules. These capsules were coated with a cellulose polymer and placed in an isolated segment of rat jejunum. Serial X-rays were used to determine the time course of capsule disintegration, spring deployment, and the rate of spring expansion. Retrieved jejunal segments were measured, and histological analyses were performed. Student’s t tests were used for statistical analysis.

Results
Intestinal segments were successfully lengthened from 1.0 cm to 4.0 cm ± 0.2 cm (p < 0.001). The optimal force for the gradual expansion of jejunal segment was 0.04 N, based on force versus rate of expansion analysis. Maximal length was achieved on day 10 (range 8-14 days). With the polymer-coated capsule, the spring was reliably deployed between 24-48 hours post implantation. Lengthened bowel retained normal small bowel architecture.

Conclusion
Use of a low-force nickel titanium spring resulted in four-fold lengthening of a segment of the small intestine. The use of a polymer-coated capsule provided a reliable way to control the timing of spring deployment. Patients with short-gut syndrome may be able to ingest the capsule orally with delivery and deployment of the spring within the intestinal lumen. The location of spring deployment can be varied by changing the solubility of the capsule.

Notes:
INNOVATION SESSION

i5
A NEW ANIMAL MODEL OF EXTRACORPOREAL FETAL SUPPORT WITH PRESERVATION OF THE PLACENTA

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Purpose
Previous efforts to develop a system of extraterine support for premature fetuses have utilized cesarean delivery followed by umbilical vessel cannulation to maintain extracorporeal life support. The present paper describes a novel technique involving gravid hysterectomy with extracorporeal membrane oxygenation (ECMO) support of the uterus via the uterine vessels. Use of such a technique also mitigates the effects of heparinization on the fetus as heparin does not significantly cross the placenta in mammals.

Methods
After IACUC approval, mixed-breed pregnant ewes underwent a gravid hysterectomy between 135-145 days gestational age. The uterine vessels were cannulated bilaterally and circulatory support was provided using ECMO. The cannulated uterus was immersed in euthermic saline to maintain temperature. Fetal heart rate was recorded via ultrasound. Serial metabolic and hematologic panels were closely followed for both ewe and uterus. The experiments were ended at fetal demise.

Results
Thirteen sheep were included in the study, 11 twin and 2 singleton. Eleven sheep had one live fetus successfully initiated on ECMO. Analysis of the surgical approach demonstrated that uterine vessel cannulation prior to hysterectomy was associated with longer viability. The average survival time from full ECMO support to death was 1 hour and 34 minutes (range 0:04-6:14).

Conclusions
This study presents a new model to maintain fetal support that preserves the maternal-placental junction through cannulation of the uterine vessels of a gravid uterus, a method that helps mitigate the adverse effects of direct heparin exposure on the fetus. This novel approach may provide insight for future studies of fetal physiology and mechanisms of support.

Notes:
i6

AN ENSEMBLE ALGORITHM FOR IMPROVED NEC RISK STRATIFICATION

Gigi Liu, MD1, Bruce X. Ling, PhD1, Fizan Abdulla, MD, PhD2, Mary Brandt, MD3, Mary Cay Harris, MD4, Larry Moss, MD5, Karl G. Sylvester, MD1

1Stanford University, Stanford, CA, USA, 2Johns Hopkins University, Baltimore, MD, USA, 3Texas Children’s Hospital, Houston, TX, USA, 4Children’s Hospital of Philadelphia, Philadelphia, PA, USA, 5Nationwide Children’s Hospital, Columbus, OH, USA

Purpose

Necrotizing Enterocolitis (NEC) is a leading cause of neonatal morbidity and mortality. Bell’s diagnostic criteria and numerous epidemiologic studies fail to identify infants at risk for disease progression to surgical NEC. Prior attempts to risk stratify infants with NEC did not utilize integrated algorithm methods to achieve powerful disease classifiers. We hypothesized that an integrative analysis of clinical parameters along with peptide biomarkers would result in a superior predictive algorithm of NEC progression.

Methods

Clinical parameters and biologic specimens at initial diagnosis were collected from 24 patients in a multi-institutional prospective study under IRB approval. Subject’s clinical course was post-hoc identified as either progressive (surgery) (n=11) or non-progressive (medical) NEC (n=13). A NEC score was calculated from the predictive probabilities from a linear regression model (LDA) of eleven clinical parameters whose p-values were less than 0.01 in Mann-Whitney U tests. Based on this NEC score, patients were then divided into low, intermediate, and high risk for developing progressive NEC at the time of initial diagnosis. A discrete biomarker panel that was selected using unbiased proteomic techniques including statistical learning and feature selection, was subsequently applied to further resolve the NEC score calculated from the clinical parameters.

Results

Both the NEC clinical scoring system and the urine peptide biomarker panel independently categorized low (n=6) and high (n=7) risk patients as non-progressors and progressors respectively. The peptide biomarker panel further resolves the ambiguity in the progression of NEC among the intermediate-risk patients (7 non-progressors and 4 progressors) with 100% accuracy and a p-value of 0.003 by Fisher exact test.

Conclusion

Ensemble data mining methods utilizing clinical and biomarker classifiers produces an effective integrated algorithm for predicting NEC progression. Thus, this ensemble methodology has significant potential as a superior risk stratification tool to target suitable therapies at earlier stages of NEC.

Notes:
Purpose
Telemedicine has been proposed as a possible alternative means of bringing expertise to hospitals that may lack certain specialists. Particularly during a disaster, hospitals may be overwhelmed and have an insufficient number of pediatric specialists available to care for injured children. We hypothesize that staff will support the application of telemedicine to triage and treat victims of a disaster. The aim of this study is to demonstrate the proof of concept of remotely providing pediatric expertise via a robot in order to treat pediatric victims.

Methods
Recently, three Los Angeles County hospitals held a joint disaster drill. One of the participating hospitals is a tertiary pediatric hospital. The disaster scenario involved a crash of a Metrolink train, resulting in a large surge of traumatic injuries. The equipment consisted of the InTouch Health Inc. robotic telecommunications system, the RP-7 Remote Presence System. The robot operates on a Wi-Fi network under the control of the physician at the control station at the remote site. After IRB approval, we conducted qualitative analysis based on observations of providers and victims (N=76 and N=131, respectively) participating in the drill.

Results
Pediatric specialists successfully provided remote triage and treatment consults of victims via the robot. The robot proved to be a useful means to extend resources and provide expert consult if pediatric specialists were unable to physically be at the site. This technology also allowed providers to remain at the incident command post to make key decisions while also remotely triaging victims.

Conclusions
We have successfully demonstrated that this technology can be used for triage and treatment of pediatric victims. For greatest cost efficiency, the use of robotic telecommunications may be expanded to trauma, critical care and other disease entities in children requiring acute medical consults.
A CRITICAL APPRAISAL OF PUBLISHED RANDOMIZED TRIALS WITHIN PEDIATRIC GENERAL SURGERY DURING THE TIME PERIOD 2000 - 2009

Martin L. Blakely, MD, MS1, Lillian Kao, MD, MS2, Rupa Seetharamaiah, MD1, KuoJen Tsao, MD2, Eunice Y. Huang, MD, MS1, Kevin P. Lally, MD, MS2

1University of Tennessee Health Science Center, Memphis, TN, USA,
2University of Texas Health Science Center, Houston, TX, USA

Purpose

A premise underlying evidence-based medicine is that studies exist that provide valid comparisons of treatments. Randomized trials produce the highest level of evidence if designed correctly. Poor quality trials may lead to poor treatment decisions. This study evaluates two hypotheses: 1) the number of RCTs within the field of pediatric surgery is increasing (compared to prior publications), and 2) the quality of RCTs is relatively low according to well-accepted guidelines.

Methods

We conducted a search for randomized trials within pediatric general surgery between 1/1/2000 - 12/31/2009 using PubMed and other electronic databases. The search and 1584 studies were reviewed by 2 independent authors. Trials found to be relevant were critically examined. The quality of each trial was judged according to the Jadad score (0-5 points; >/= 3 high quality) and by adherence to 7 components of the CONSORT statement. If there was disagreement between 2 initial reviewers, a third reviewer was utilized and a consensus reached.

Results

73 trials met inclusion criteria and were critically reviewed. A 3rd reviewer was utilized for 18 trials (25%). The majority of trials were conducted outside the US (n=49, 67%). Only 6 trials satisfied all 7 components of the CONSORT statement utilized. Specific quality measures are shown in the table:
### Conclusions

The number of published RCTs within pediatric general surgery (approximately 7 / year) is increasing, but the majority do not conform to published guidelines and are of relatively low quality. By focusing on the areas found in this study, the quality of future trials can improve dramatically.

### Notes:
QUALITY IMPROVEMENT AND PATIENT CARE CHECKLISTS IN INTRAHOSPITAL TRANSFERS INVOLVING PEDIATRIC SURGERY PATIENTS

Don K. Nakayama, MD, MBA1, Sally S. Lester, RN2, Darla R. Rich, RN, FNP2, Bryan C. Weidner, MD1, Joshua B. Glenn, MD1, Issam J. Shaker, MD2

1Medical Center of Central Georgia, Mercer University School of Medicine, Macon, GA, USA, 2Medical Center of Central Georgia, Macon, GA, USA

Purpose
Intrahospital transfers from the emergency department (ED), patient wards, and critical care areas to the operating room (OR), post-anesthesia care unit, and radiology suite are necessary but hazardous aspects of pediatric surgical care. We used a Plan-Do-Study-Act (PDSA) quality improvement (QI) process to determine risks during intrahospital transfer and to improve patient safety.

Methods
A multidisciplinary QI team developed a checklist that documented patient data and face-to-face nurse-to-nurse handoffs for all intrahospital transfers involving pediatric surgical inpatients. The checklist summarized major clinical events, including any problems that arose, providing concurrent summaries that were collected by three-month quarters (Q1, Q2, etc.) over twelve months.

Results
There were 903 intrahospital transfers involving 583 operations. Identified care areas in 444 (49% of 903) included NICU in 103 (23% of 444); PICU, 21 (5%); ED, 1 (1%); and pediatric ward, 318 (72%). Total hand-offs were documented in 436 (48% of 903): 1 hand off in 234 (54% of 436); 2, 143 (33%); 3, 49 (11%); >4, 10 (2%). Documented problems occurred in 31 transfers (3.4% of 903), the most during Q1 (19/191, 9.9%). Incidence fell to 3.5% (9/260) in Q2; 0.4% (1/243), Q3; and 1.0% (2/209), Q4 (p < 0.001, r x c contingency table, Chi-square). Patient care issues (14/31, 45%) were the most common problem, followed by with documentation (10, 32%) and process problems (7, 23%). The QI team addressed these issues and was able to resolve such important problems as patient instability during transport (5 in Q1, none in Q3 and Q4) and poor pain control (3 in Q2, 1 in Q3 and Q4).

Conclusions
QI based on a systems approach, such as the cascade of care concept, emphasizes ongoing process analysis by multidisciplinary teams. Checklists reinforce communication and provide feedback on whether system goals are being achieved.

Notes:
Purpose
Multiple visits to the hospital for the evaluation, treatment and follow-up of straight forward surgical problems are inconvenient, can result in lost work for the parents, and missed school for the child. Skilled primary care providers uncommonly misdiagnose the most routine surgical problems, with the majority being scheduled as elective procedures on a later date. We hypothesized that with proper previsit screening, patients with select diagnoses can be evaluated in an out patient clinic setting and if the diagnosis is confirmed, undergo operation the same day.

Methods
Criteria were developed to identify straightforward referrals to our surgical practice for umbilical, epigastric, or inguinal hernias. Scripting was created to offer families the option of consultation and, if indicated, surgical treatment on the same day. Details regarding the consultation process as well as preoperative instructions were given to those interested in participating. Data collected included number of patients, number of cases performed, insurance status, and reimbursement for professional and hospital consultation and surgical fees. Families were then surveyed postoperatively to assess their satisfaction with the process. (IRB#2010-1470).

Results
Twenty-five patient candidates agreed to participate in this pilot program. Of the 25 patients, the diagnosis and indication for surgery was confirmed in 21 (84%) who underwent repair the day of their consultation. 53% of patients had commercial insurance while 47% had Medicaid. The preoperative consultation fee was reimbursed in 12 of 15 (80%) encounters (71% Medicaid, 88% commercial). All surgical cases were reimbursed. Patient and family satisfaction was high (table).

Conclusions
From this pilot study we conclude that it is feasible to provide same day evaluation and service for straightforward pediatric hernias with acceptable financial reimbursement and extremely high parent satisfaction. One stop shopping reduces missed work and school, potentially reducing the economic impact in the family.
Notes:
VARIATION IN RESOURCE UTILIZATION ASSOCIATED WITH THE
MANAGEMENT OF APPENDICITIS IN CHILDREN: IMPLICATIONS FOR
QUALITY IMPROVEMENT THROUGH COMPARATIVE ANALYSIS AND
COLLABORATIVE NETWORKING

Shawn J. Rangel, MD, MSc1, Jessica Baxter, BS1, Jeff Barnes2

1Children’s Hospital Boston, Boston, MA, USA, 2Child Health Corporation of America, Shawnee Mission, KS, USA

Purpose
The purpose of this study was to characterize variation in hospital-specific resource utilization associated with the management of acute appendicitis in children.

Methods
We conducted a retrospective one-year audit of all patients (n=11,831, mean: 329 cases/center) admitted with appendicitis at 36 hospitals participating in the Pediatric Health Information System database (6/2009-6/2010). Cases were divided into uncomplicated (non-perforated with length of stay (LOS) ≤2 days) and complicated (perforated with LOS≥3 days) groups and analyzed separately for institutional variation in utilization of imaging and laboratory tests, inpatient readmission rates, and total hospital cost and charges. The complicated group was further analyzed for variation in LOS, utilization of TPN and PICC lines, and transition to oral antibiotics prior to discharge.

Results
Significant differences between hospitals were found for all measures in the uncomplicated group (range in use of CT/US imaging in the ED: 0.12-0.92 studies/encounter [p<0.001]; laboratory tests: 0.4-1.9 tests/admission [p<0.001]; readmission rates: 1.8-14.1% [p<0.001], mean hospital cost: $5,522-$10,459 [p<0.001]; mean hospital charges: $10,577-$32,379 [p<0.001]), and for all measures in the complicated group (total imaging tests: 0.4-2.5 studies/admission [p<0.001]; laboratory tests: 1.6-7.2 tests/admission [p<0.001]; readmission rates: 3.8-30.2% [p<0.001]; mean hospital cost: $6,030-$27,547 [p<0.001]; mean hospital charges: $26,151-$64,978 [p<0.001]). For the complicated group, significant variation was also found for LOS (4.8-7.1 days, p<0.001), utilization of PICC lines (0.0-87.2%, p<0.001) and TPN (1.2-39.5%, p<0.001), and transition to oral antibiotics (1.4-51.0%, p<0.001). High and low performing outliers (based on 95% confidence intervals) were identified for all measures examined.

Conclusions
Significant variation exists in the resource utilization associated with the management of appendicitis in children. These outcomes should be correlated with clinically meaningful endpoints to derive novel value-based quality measures for comparative analysis. Creation of a collaborative network to explore differences...
in “outlier” practice patterns may provide an effective strategy for streamlining quality improvement relating to the management of appendicitis in children.
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EPIDURAL VERSUS PATIENT CONTROLLED ANALGESIA FOR POST-OPERATIVE PAIN FOLLOWING PECTUS EXCAVATUM REPAIR: A PROSPECTIVE, RANDOMIZED TRIAL
Shawn D. St. Peter, Kathryn A. Weesner, MD, Eric E. Weissend, MD, Susan W. Sharp, PhD, Patricia A. Valusek, MD, Charles L. Snyder, MD, Ronald J. Sharp, MD, George W. Holcomb III, MD, MBA, Daniel J. Ostlie, MD
Children's Mercy Hospital, Kansas City, MO, USA

Purpose
Management of post-operative pain is a challenge after the minimally invasive repair of pectus excavatum. Pain is managed by either a thoracic epidural (EPI) or patient controlled analgesia (PCA) with intravenous narcotics, and currently, prospective comparative data are lacking. Therefore, we conducted a prospective, randomized trial to evaluate the relative merits of these 2 pain management strategies.

Methods
After obtaining permission/assent (IRB # 06 08 128), patients were randomized to either EPI or PCA with fixed protocols for each. Primary outcome variable was length of stay with a power of 0.8 and $\alpha$ of 0.05. Visual analog pain scores were recorded twice daily. The epidural catheter was removed on post-operative day 3 if it was still present.

Results
From September, 2006 to May, 2010, 110 patients were enrolled. Outcome data are listed in Table 1, and pain scores are listed in Table 2. The epidural was could not be placed or was removed within 24 hours in 12 patients (23%). In the remaining patients, a PCA was required after transition in 7 patients (16%). Conclusions: There is longer operating room time, longer time with a urinary catheter, more calls to anesthesia and greater hospital charges with and thoracic epidural after the repair of pectus excavatum compared to patient-controlled analgesia. Pain scores favor epidural early in post-operative course and patient-controlled analgesia later.
### SCIENTIFIC SESSION V

#### Table 1 - Outcomes

<table>
<thead>
<tr>
<th>Parameter</th>
<th>EPI (N = 55)</th>
<th>PCA (N = 55)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>OR Time (Hrs:Mins)</td>
<td>1:58 +/- 0:21</td>
<td>1:35 +/- 0:20</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Calls to Anesthesia</td>
<td>1.7 +/- 2.0</td>
<td>0.4 +/- 0.7</td>
<td>&lt;0.001</td>
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<tr>
<td>Hrs to Foley Removal</td>
<td>64.6 +/- 14.5</td>
<td>52.3 +/- 27.1</td>
<td>0.004</td>
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<tr>
<td>Hrs to Oral Pain Meds</td>
<td>67.1 +/- 21.2</td>
<td>60.8 +/- 20.7</td>
<td>0.12</td>
</tr>
<tr>
<td>Length of Stay (Days)</td>
<td>4.54 +/- 1.01</td>
<td>4.25 +/- 1.0</td>
<td>0.13</td>
</tr>
<tr>
<td>Hospital Charges</td>
<td>$45.4K +/- 7.3K</td>
<td>$38.6K +/- 6.7K</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

#### Table 2 - Mean Daily Pain Scores

<table>
<thead>
<tr>
<th>Group</th>
<th>Recovery Room</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Day 3</th>
<th>Day 4</th>
<th>Day 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>EPI</td>
<td>4.2 +/- 2.5</td>
<td>3.4 +/- 2.4</td>
<td>4.0 +/- 2.0</td>
<td>4.2 +/- 1.9</td>
<td>4.3 +/- 1.7</td>
<td>3.9 +/- 1.8</td>
</tr>
<tr>
<td>PCA</td>
<td>5.7 +/- 2.2</td>
<td>4.6 +/- 1.9</td>
<td>4.8 +/- 2.0</td>
<td>4.1 +/- 1.8</td>
<td>3.8 +/- 2.2</td>
<td>3.7 +/- 2.4</td>
</tr>
</tbody>
</table>

Notes:
47

LEGISLATING ALL-TERRAIN VEHICLE ACCESS BY CHILDREN: EFFECTS ON INJURY RATES AND SEVERITY

Natalie L. Yanchar, MD, MSc, Nadia Murphy, MD

IWK Health Centre, Dalhousie University, Halifax, NS, Canada

Purpose
In 2004-2005, intense public debate led to implementation of tough legislation restricting all-terrain vehicle (ATV) use by children <16, strictest for those <14. We compared rates and severity of pediatric ATV-related trauma across the province before and after implementation of legislation.

Methods
Rates of hospitalizations and death across the province (n=162), as well as emergency department (ED) visits, hospitalizations and measures of injury severity of those admitted to the regional pediatric trauma centre (n=98) were compared between January 1999 to December 2004 and January 2005 to December 2008. Chart-review and population-based-datasets were analyzed. Rates were compared by chi-square. Associations with outcomes of interest were determined by logistic regression, with 95% confidence intervals not spanning 1.0 considered significant.

Results
Although the number of deaths (~1 per year) did not change, rates of ATV-related ED visits to the trauma centre decreased by 36%, trauma centre hospitalizations by 75%, and all provincial hospitalizations by 38%. Conversely, the proportion of older (≥14) patients admitted to the trauma centre rose to 61% from 41% and the proportion with an ISS≥12 increased from 17.5% to 33.3%. Neither change, however, reached statistical significance (p=0.14 and p=0.13, respectively). Helmet use increased from 76% to 86% and was significantly associated with a reduced risk of head injury (OR=0.11, 95%CI:0.04-0.32 ). Over the same period, provincial ATV sales decreased by 54% while national sales stayed relatively stable.

Conclusions
Implementation of legislation restricting access of children to operating ATVs, coupled with intense public debate, was associated with decreased hospitalizations but a seemingly modest increase in injury severity secondary to ATV-related trauma. Less effect was seen on older youth to whom regulations were more lenient. Ultimately, despite these measures, ATV-related Injuries and deaths still occur, suggesting legislation focused on restricting access has some, but limited, efficacy in reducing pediatric ATV-related trauma.
Provincial Pediatric All-Terrain Vehicle-Related Trauma (age<16)

Notes:
MEDIAN ARCUATE LIGAMENT SYNDROME IN CHILDREN: LAST STOP BEFORE FUNCTIONAL GASTROINTESTINAL DISORDERS?

Christopher L. Skelly, MD, Aviram Assidon, Grace Z. Mak, MD, Kathleen Romanowski, MD, Melissa Ruiz, MD, Donald C. Liu, MD, PhD
University of Chicago, Chicago, IL, USA

Purpose
Given the overlapping symptoms of functional gastrointestinal disorders (FGID) and median arcuate ligament syndrome (MALS) in patients diagnosed with chronic abdominal pain (CAP), we prospectively screened 59 pediatric patients with a diagnosis of CAP for MALS.

Methods
We screened 59 consecutive patients (51 females, 8 males; ages 8-21 yrs; median-17 yrs) with diagnosis of CAP within the domain of FGID with duplex ultrasound. A positive screen was defined as celiac artery peak systolic velocity (PSV) over 200 cm/sec and end diastolic velocity (EDV) over 55 cm/sec correlating to flow-reducing stenosis >70%. Positive duplex findings were confirmed by standard CT angiography (CTA). 22 patients underwent laparoscopic release with PSV normalization in all patients based on intraoperative duplex. Assessment of surgical treatment outcome was determined by pre- and post-surgical administration of PedsQLtm questionnaires.

Results
32 (54%) had velocities suggestive of MALS; 30 (94%) were female (p<0.00001). The average PSV of the positive group was 321 cm/s (213-800 cm/s) versus 154.2 cm/s (90-196 cm/s) in the negative group (p<0.00001). MALS patients had celiac to aortic ratio of 2.1 (1.1-6.1) compared to the negative group 1.1 (0.8-1.6) (p<0.0001). CTA findings correlated 64% to duplex. Post-operative duplex revealed decreased PSV to 215 cm/s (91-373 cm/s) (p<0.00001 compared to pre-operatively). Of the 22 patients, 17 are symptom free with a mean follow-up of 6 months. Overall scores from the PedsQLtm questionnaire improved from 56 (30-85) pre-operatively to 92 (72-100) post-operatively (p<0.0001).

Conclusion
This unexpectedly high proportion of females screened positively for MALS and early success in QOL outcome measures after surgery lead us to hypothesize that MALS might be earlier diagnosed and possibly treated in pediatric patients with CAP previously thought to be functional in origin. The significance of celiac to aortic ratio may prove to be useful criteria in the early diagnosis of MALS.

Notes:
A MULTI-CENTER EVALUATION OF THE ROLE OF MECHANICAL BOWEL PREPARATION IN PEDIATRIC COLOSTOMY TAKEDOWN

Katherine Serrurier, BA1, Jie Liu, MPH1, Francine Breckler, PharmD2, Nini Khozeimeh, MD3, Deborah Billmire, MD2, Cynthia Gingalewski, MD3, Gerald Gollin, MD1

1Loma Linda University School of Medicine, Loma Linda, CA, USA, 2Riley Hospital for Children, Indianapolis, IN, USA, 3Children’s National Medical Center, Washington, DC, USA

Purpose

The necessity for mechanical bowel preparation in colonic surgery has been challenged by studies in adults, yet 96% of pediatric surgeons still utilize bowel preparation for colorectal procedures. We sought to evaluate the utility of mechanical bowel preparation in a multi-center, retrospective study of children undergoing colostomy takedown.

Methods

The records of 272 children who underwent colostomy takedown at three large children’s hospitals were reviewed under a protocol approved by the IRB at each institution. The administration of mechanical bowel preparation and perioperative antibiotics was documented. Length of stay and the incidences of wound, anastomotic, and other complications were compared.

Results

A polyethylene glycol bowel prep was administered to 187 subjects and 122 of these were admitted preoperatively for that purpose. No bowel prep was used in 85. All subjects received perioperative, intravenous antibiotics and 52% of those with bowel preps received preoperative, oral antibiotics. There was no difference between the groups in terms of gender or the indication for colostomy. Outcomes are presented in the table. The subset that received oral antibiotics had no differences in outcomes from subjects who did not.

Conclusions

In a large, multi-center study we found that the use of a mechanical bowel preparation in children prior to colostomy takedown was associated with a greater risk for wound infection, no protection from other complications, and a longer length of stay. This corroborates adult studies and suggests that bowel preparation may be safely omitted in many children who undergo colonic surgery, thereby reducing cost and discomfort.
### SCIENTIFIC SESSION V

Outcomes with and without mechanical bowel preparation

<table>
<thead>
<tr>
<th></th>
<th>Bowel Prep (n=187)</th>
<th>No Bowel Prep (n=85)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (mean, months)</td>
<td>19.4</td>
<td>20.9</td>
<td>0.35</td>
</tr>
<tr>
<td>Wound infection (%)</td>
<td>14.4</td>
<td>5.8</td>
<td>0.04</td>
</tr>
<tr>
<td>Abdominal abscess (%)</td>
<td>1.1</td>
<td>1.2</td>
<td>0.96</td>
</tr>
<tr>
<td>Anastomotic leak (%)</td>
<td>1.6</td>
<td>1.2</td>
<td>0.79</td>
</tr>
<tr>
<td>C. Diff. infection (%)</td>
<td>0.5</td>
<td>2.3</td>
<td>0.18</td>
</tr>
<tr>
<td>Length of stay (mean, days)</td>
<td>5.6</td>
<td>4.4</td>
<td>0.05</td>
</tr>
</tbody>
</table>

Notes:
APSA PAST MEETING LECTURES

Journal of Pediatric Surgery Lectures

2010
Robert H. Bartlett, MD
“ECMO: Gross, Beethoven, Krummel and Georgeson”

2008
Thomas M. Krummel, MD
“Inventing Our Future: Training the Next Generation of Surgeon Innovators”

2007
Alan W. Flake, MD
“Stem Cell Biology and Pediatric Surgery – Deciphering the Venn Diagram”

2006
Pedro Rosselló, MD
“The Unfinished Business of American Healthcare”

2005
Alberto Peña, MD
“Luck and Serendipity, the History of a Surgical Technique”

2004
R. Scott Jones, MD
“The American College of Surgeons Initiatives for Safety and Quality Improvement”

2003
Patricia K. Donahoe, MD
“Sustained Inquiry and Perseverance in the Clinic and at the Bench”

2002
Michael R. Harrison, MD
“Fetal Surgery: Trials, Tribulations and Territory”

2001
Joseph P. Vacanti, MD
“The History and Current Status of Tissue Engineering”

Robert E. Gross Lectures

2010
John D. Birkmeyer, MD
“Measuring and Improvement the Quality of Pediatric Surgery”

2009
Stanley B. Prusiner, MD
“Designer Prions and a Quest for Therapy”

2008
Michael W.L. Gauderer, MD
“Creativity and the Surgeon”

2007
Francisco G. Cigarroa, MD
“Leading an Academic Health Center in the 21st Century: A Pediatric Surgeon’s Perspective”

2006
Diana Bianchi, MD
“Fetomaternal Cell Trafficking: A Story that Begins with Prenatal Diagnosis and May End with Stem Cell Therapy”

2005
W. Hardy Hendren, MD
“Looking Back 50 Years”

2004
Giulio (Dan) D’Angio, MD
“The Role of the Surgeon in the Past, Present and Future of Pediatric Oncology”
2003
Lucien Leape, MD
“Safe Health Care — Are We Up to It?”

1994
W. French Anderson, PhD
“Human Gene Therapy”

2002
Harold Shapiro, PhD
“The Ethical Dimensions of Scientific Progress”

1993
Judah Folkman, MD
“Clinical Applications of Angiogenesis Research”

2001
Judah Folkman, MD
“Angiogenesis-Dependent Diseases”

1992
Warren Zapol, MD
“Inhaled Nitric Oxide: A Selective Vaso-Dilator”

2000
J. Bruce Beckwith, MD
“Pediatric Renal Tumors at the New Millennium: Myths, Misunderstandings, Controversies and Opportunities”

1991
Joel Cooper, MD
“History and Current Status of Lung Transplantation”

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

1990
Richard Simmons, MD
“Role of the Gut Flora in Surgery”

1998
Richard M. Satava, MD
“Medicine in the 21st Century”

2010
Christopher K. Breuer, MD
“The Development and Translation of the Tissue Engineered Vascular Grafts”

1997
Douglas W. Wilmore, MD
“Will Organ Growth Replace Transplantation? Lessons from Patients with Short Bowel Syndrome”

2009
Michael T. Longaker, MD, MBA, FACS
“Regenerative Medicine: A Surgeon’s Perspective”

1996
Robert H. Bartlett, MD
“Surgery, Science and Respiratory Failure”

2008
Frederick J. Rescorla, MD
“What’s New in Pediatric Surgery”

1995
David A. Williams, MD
“The Role of Interleukin-II on the Pathophysiology of the Small Intestine”

Jay & Margie Grosfeld Lectures

2010
Christopher K. Breuer, MD
“The Development and Translation of the Tissue Engineered Vascular Grafts”

Final Program
# APSA Past Meeting Lectures (Cont.)

**International Guest Lecturers**

<table>
<thead>
<tr>
<th>Year</th>
<th>Lecturer</th>
<th>Title</th>
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<tbody>
<tr>
<td>2010</td>
<td>Jan Alice Marcel Deprest, MD</td>
<td>“Prenatal Management of the Fetus with Isolated CDH”</td>
</tr>
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<td>2009</td>
<td>Marcelo Martinez Ferro, MD</td>
<td>“New Approaches to Pectus and Other MIS in Argentina”</td>
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<td>2008</td>
<td>Tadashi Iwanaka, MD</td>
<td>“Technical Innovation, Standardization and Skill Qualification of Pediatric Minimally Invasive Surgery in Japan”</td>
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<td>2007</td>
<td>Claire Nihoul-Fékété, MD</td>
<td>“Is Regionalism of Complex Pediatric Malformations Desirable and Feasible? The Example of Disorders of Sexual Development”</td>
</tr>
<tr>
<td>2005</td>
<td>Prof. Frans W.J. Hazebroek, MD, PhD</td>
<td>“Is Continuation of Life Support Always the Best Option for the Surgical Neonate?”</td>
</tr>
<tr>
<td>2004</td>
<td>David A. Lloyd, MD, FRCS</td>
<td>“Tomorrow’s Surgeons: Who Cares for the Patient?”</td>
</tr>
<tr>
<td>2003</td>
<td>Claire Nihoul-Fékété, MD</td>
<td>“Modern Surgical Management of Congenital Hyperinsulinemic Hypoglycemia”</td>
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<tr>
<td>2002</td>
<td>Takeshi Miyano, MD</td>
<td>“Biliary Tree: A Gardener’s 30-Year Experience”</td>
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<tr>
<td>2001</td>
<td>Pedro Rosselló, MD</td>
<td>“One Nation, with Liberty and Justice…and Healthcare for All”</td>
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<tr>
<td>2000</td>
<td>Leela Kapila, FRCS</td>
<td>“Are These the Children of a Lesser God?”</td>
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<tr>
<td>1999</td>
<td>Bernardo Ochoa, MD</td>
<td>“Pediatric Surgery in Latin America”</td>
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<tr>
<td>1998</td>
<td>Sidney Cywes, MD</td>
<td>“Some of the Little Things We Do — Something Old, Something New”</td>
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<tr>
<td>1997</td>
<td>Justin Kelly</td>
<td>“Bladder Exstrophy — Problems and Solutions”</td>
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<tr>
<td>1996</td>
<td>Prem Puri, MD</td>
<td>“Variant Hirschsprung’s Disease”</td>
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<tr>
<td>1995</td>
<td>Sir Lewis Spitz, MD, PhD, FRCS</td>
<td>“Esophageal Atresia — Past, Present and Future”</td>
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<tr>
<td>1994</td>
<td>Sean J. Corkery, MCh, FRCSI, FRCSeng</td>
<td>“In Pursuit of the Testis”</td>
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</table>
1993
Edward M. Kiely, FRCSI, FRCS
“The Surgical Challenge of Neuroblastoma”

1992
Yann Revillon, MD
“Intestinal Transplantation in France”

1991
Shemuel Nissan, MD
“The History of Surgery and Medicine in the Holy Land from the 19th Century”

1990
Jan C. Molenaar, MD
“Congenital Diaphragmatic Hernia - What Defect”
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The American Pediatric Surgical Association Foundation (APSAF) was founded to provide support for young pediatric surgical investigators and promote pediatric surgical research and education. The year 2011 marks the 15th Anniversary of distribution of grants to investigators from the Foundation. The vital support provided by APSA members energizes the productivity of our young pediatric surgeon-scientists and results in important new discoveries that benefit our patients and profession. This would be impossible to accomplish without your generous financial support. Help us celebrate this milestone by donating to the APSA Foundation.

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Web site: www.childrenscentralcal.org

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The SOCS (Pediatric Surgical Outcomes Collection System) Project is designed to automate and ease the data collection process required for the ACS NSQIP program. Automatically selects/organizes cases in cycles based on the specific guidelines provided by NSQIP. Auto-populates 47% of the data fields required by NSQIP with future plans to add at least 20% more automation. Provides reporting functionality acting as an internal Quality Improvement database allowing research of possible trends in the surgical population.

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May 28 - 30, 2009
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Fajardo, Puerto Rico

39th Annual Meeting
May 27 - 31, 2008
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Phoenix, Arizona

38th Annual Meeting
May 24 - 27, 2007
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Orlando, Florida

37th Annual Meeting
May 21 - 24, 2006
Marriott Beach & Golf Resort
Hilton Head, South Carolina

36th Annual Meeting
May 29 - June 1, 2005
JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona

35th Annual Meeting
May 27 - 30, 2004
Sawgrass Marriott Resort
Ponte Vedra Beach, Florida

34th Annual Meeting
May 25 - 28, 2003
Marriott Harbor Beach Resort & Spa
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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
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44th Annual Meeting
May 2 - 5, 2013
Marco Island Marriott Beach Resort,
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45th Annual Meeting
May 29 - June 1, 2014
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46th Annual Meeting
April 30 - May 3, 2015
Harbor Beach Marriott Resort & Spa
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