American Pediatric Surgical Association

APSA 41st Annual Meeting

Final Program

May 16 – 19, 2010

Loews Portofino Bay Hotel at Universal Orlando

Orlando, FL USA

www.eapsa.org

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

We do this by:

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

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Laura A. Boomer
Cholangiocyte Apoptosis During Lamprey Metamorphosis

Best Podium Presentation

Eric Jelin
Effects of Notch4 On Lung Vascular Remodeling

Best Poster Presentation (2008)

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

Best Podium Presentation

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

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

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

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Focal Adhesion Kinase and Vascular Endothelial Growth Factor Receptor-3 in Human Neuroblastoma

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Intestinal Dysmotility in Fetal Repair of Gastroschisis

2004

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

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

2003

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

2002

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

2001

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

2000

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

1999

Gail E. Besner
Heparin-Binding EGF-Like Growth Factor (HB-EGF) and Intestinal Ischemia Reperfusion Injury

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Pledge For New Members of the American Pediatric Surgical Association

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

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

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

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

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

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

New Members 2009-2010

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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 are then members of the Board of Governors of the Corporation.

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

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

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

Bylaws of the American Pediatric Surgical Association

PREAMBLE

PRINCIPLES OF MEDICAL ETHICS

Members:

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

Article I MEMBERSHIP

Section 1. Regular Membership

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

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

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

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

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

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

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

Section 2. Candidate Members

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

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

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

2.4. An individual may remain a candidate member for five years following completion of an approved pediatric surgical residency program at which time the candidate membership will expire. This five-year period is in addition to the time spent as a candidate member during pediatric surgery residency.

If candidate membership expires, one may still apply for regular membership at any time in the future. Candidate membership is not mandatory in order to qualify for regular membership.

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

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

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

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

Section 3. Charter Membership

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

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

Section 4. Honorary Membership

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

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

Section 5. International Membership

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

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

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

Section 6. Associate Members

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

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

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

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

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

Section 7. Resident Members

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

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

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

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

Section 8. Application Procedures

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

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

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

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

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

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

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

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

Section 9. Application Form

9.1. The application shall include:

9.1.1. Curriculum vitae

9.1.2. Bibliography

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

9.2. The candidate membership application shall include:

9.2.1. Curriculum vitae

9.2.2. Bibliography

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

Section 10. Resignation

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

Section 11. Fiscal Year

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

Section 12. Dues

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

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

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

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

Section 13. Certificate of Membership

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

Section 14. Loss of Membership

14.1. A member may be dropped from membership for:

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

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

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

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

14.3. Discipline.

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

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

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

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

14.3.2.3. Grossly immoral, dishonorable or unprofessional conduct.

14.3.3. Due process.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Article II OFFICERS

Section 1. The Officers

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

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

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

Section 2. Term of Office

2.1. The terms of each above office shall be:

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

Article III BOARD OF GOVERNORS

Section 1. Membership of the Board of Governors

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

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

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

Section 2. Chair of the Board of Governors

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

Section 3. Functions of the Board of Governors

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

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

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

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

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

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

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

Article IV DUTIES OF OFFICERS

Section 1. The President

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

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

1.3. Shall sign all official documents.

1.4. Shall make appropriate committee appointments.

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

Section 2. The President-Elect

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

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

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

2.4. Shall become president the next year.

Section 3. The Secretary

3.1. Shall record the proceedings at all meetings.

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

3.3. Shall maintain a registry of membership.

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

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

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

Section 4. The Treasurer

4.1. Shall bill to and collect from members all dues and fees pertaining to the Association.

4.2. Shall render disbursements for authorized official expenses.

4.3. Shall maintain a financial ledger.

4.4. Shall maintain records, which shall be available for an annual audit by an appropriate auditing committee of members appointed by the president or by an outside accounting firm.

4.5. Shall present a report to the membership at the business session of the annual meeting.

4.6. Shall maintain at the expense of the Association a surety bond for the treasurer and all others handling Association funds.

4.8. The first treasurer shall be elected for a two-year term.

Article V MEETINGS

Section 1. Annual Meeting

1.1. There shall be an annual meeting, the time and place of which shall be established by the board of governors at least a year in advance.

1.2. There shall be a scientific meeting incorporated into the annual meeting.

1.3. There shall be a business meeting incorporated into the annual meeting, which will be open only to members in good standing and at which official business shall be transacted.

1.4. All meetings shall be guided by the current edition of Robert's Rules of Order.

Section 2. Guests and the Annual Meeting

2.1. The scientific sessions of the annual meeting shall be open to all interested physicians who register for the meeting.

2.2. Interested paramedical professionals may be invited by any member in good standing.

2.3. A registration fee may be required of non-members and guests at the discretion of the program committee.

2.4. The privilege of the floor at the scientific sessions will be restricted to the membership and to others who have been given official designation by letter from the secretary.

Section 3. Quorum

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

Article VI BYLAWS

Section 1. Time of Effect

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

Section 2. Amendments of the Bylaws

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

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

Article VII PERMANENT COMMITTEES

Section 1. Permanent Committees

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

Article VIII AD HOC COMMITTEES

Section 1. Membership

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

Article IX REPRESENTATION TO OTHER SOCIETIES

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

Article X HISTORIAN

An historian shall be appointed by the president.

Article XI OFFICIAL SEAL

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

Article XII INDEMNIFICATION AND INSURANCE

Section 1. Indemnification

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

Section 2. Insurance

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

Approved May 30, 2009

In Memoriam

Gilbert, Michel 1972
Gamion, Robers S., Jr. 1973
Chamberlain, John W. 1974
Snyder, William H., Jr. 1974
Bracey, Altamount 1978
Erwin, James H. 1979
White, Robert F. 1980
Allen, Robert G. 1981
Karn, Gordon M. 1981
Kiesewetter, William B. 1981
Schneider, Keith M. 1982
Hawes, Ernest B. 1984
Lozoya-Solis, Jesus 1984
Soave, Franco 1984
Rosenkrantz, Jens G. 1985
Cresson, Samuel L. 1986
Owings, Richard S. 1986
Pilling, George P., IV 1986
Stewart, David R. 1986
Simpson, James Stanley 1988
Gross, Robert E. 1988
Ravitch, Mark M. 1989
Ballantine, Thomas V.N. 1990
Ferguson, Colin C. 1991
Mishalany, Henry 1991
Schisgall, Richard M. 1991
David, Ronald 1992
Kaufman, Bruce 1992
Harkins, George A. 1993
Sakaguchi, Shimpei 1993
Segnitz, Richard H. 1993
Gans, Stephen L. 1994
Kumar, A.P. Mahesh 1994
McParland, Felix A. 1994
Pokorny, William J. 1994
Richardson, William R. 1994
Benson, Clifford D. 1995
Lilly, John R. 1995
Riker, William L. 1995
Bill, Alexander H. (Sandy) 1996
Cheu, Henry W. 1996
Danis, Richard K. 1996
Goldstein, I. Richard 1996
Longino, Luther A. 1996
Welch, Kenneth J. 1996
Baffes, Thomas G. 1997
Bettex, Marcel 1997
Salzberg, Arnold M. 1997
Santulli, Thomas V. 1997
Brennan, L. Patrick 1998
Brooks, Benjy F. 1998
Carson, James A. 1998
Hamilton, James P. 1998
Stanley-Brown, Edward G. 1998
Knutrud, Ola 1999
Warden, M. James 1999
Winslow, Paul 1999
Zachary, R.B. 1999
Linkner, Laurance M. 2000
Meeker, Irving A., Jr. 2000

Chisholm, Tague C. 2000
McAteer, Jerry 2001
Clatworthy, William 2001
Allen, James E. 2001
Lizarralde, A. Eduardo 2001
Weitzman, Jordan J. 2001
Campbell, David P. 2001
Carcassone, Michel 2002
Cohn, Bertram D. 2002
Colodny, Arnold H. 2002
Eraklis, Angelo J. 2002
Smith, Willard D. 2002
So, Henry B. 2002
Tapper, David 2002
Zwiren, Gerald T. 2002
Abrams, Martin W. 2003
Harberg, Franklin J. (Jim) 2003
Lynch, III, Frank P. 2003
Smith, E. Ide 2003
Rickham, Peter P. 2003
Huseby, Thomas L. 2004
Izant, Robert 2004
Pickett, Lawrence K. 2004
Bronsther, Burton 2004
Stahl, Nicholas M. 2004
Phillipart, III, Arlvin I. 2004
McAlpin, Columbus D. 2004
Lloyd, James R. 2004
Moore, Thomas C. 2004
Rathauser, Frank 2005
Fitzpatrick, John 2005
Able, Luke W. 2006
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
McClenathan, James E. 2007
Pitts, R. Marshall 2007
Wolfson, Philip J. 2007
Folkman, M. Judah 2007
Smith, Melvin D. 2007
McGovern, Bruce 2008
MacDonald, James S. 2008
Campbell, Timothy J. 2008
Votteler, Theodore P. 2008
Cooney, Donald R. 2008
Cooke, Ronald W. 2009
Anderson, Alan E. 2009
de Lorimier, Alfred A. 2009
Fisher, John H. 2009

Founding Members

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

Charter Members

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

Schedule at a Glance

Saturday, May 15, 2010

8:00 a.m. – 2:00 p.m.	APSA Board of Governors Meeting	*Donatello*
2:00 – 9:00 p.m.	Committee Meetings	*Bernini, DaVinci, Michelangelo 1, Michelangelo 2*
2:00 – 6:00 p.m.	Training Program Directors Meeting	*Tuscan 1*
4:00 – 6:00 p.m.	Registration Open	*Tuscan Foyer*
6:30 – 10:00 p.m.	Publications Committee Meeting	*Del Mare*
7:00 – 10:00 p.m.	APSA Board of Governors Dinner	*Bice Restaurant (Venezia Room)*

Sunday, May 16, 2010

6:00 – 8:00 a.m.	Committee Meetings	*Bernini 1, Bernini 2, Michelangelo 1, Michelangelo 2, DaVinci 1, DaVinci 2, Donatello, Vincenza*
7:00 a.m. – 5:00 p.m.	Registration Open	*Tuscan Foyer*
7:30 – 8:00 a.m.	Continental Breakfast	*Tuscan Foyer*
8:00 – 11:00 a.m.	**Education Session 1:** Evidence-Based Medicine in Pediatric Surgery	*Tuscan Ballroom*
11:00 – 11:30 a.m.	Refreshment Break	*Tuscan Foyer*
11:30 a.m. – 12:30 p.m.	***Journal of Pediatric Surgery*** **Lecture:** Robert H. Bartlett, MD ECMO: Gross, Beethoven, Krummel and Georgeson	*Tuscan Ballroom*
12:30 – 12:45 p.m.	Box Lunch Pick-Up	*Tuscan Ballroom*
12:45 – 1:45 p.m.	**Video Session with Lunch**	*Tuscan Ballroom*
1:45 – 2:00 p.m.	Refreshment Break	*Tuscan Foyer*
2:00 – 4:00 p.m.	**Concurrent Sessions:**	
	Education Session 2: Evidence-Based Clinical Practice Guidelines and Outcomes Research: Road to the Future	*Tuscan Ballroom*
	Education Session 3: International Collaborations in Pediatric Surgery: Assessing and Meeting Needs. Presented by the International Relations Committee **The Global View,** *Steven W. Bickler, MD* **The Need,** *Lohfa Chirdan, MBBS* **The Assessment,** *Donald E. Meier, MD* **The Challenge of Meeting Needs,** *J. Ted Gerstle, MD* **The Role of Trainees,** *Diana L. Farmer, MD* **The Nuts and Bolts,** *Marilyn W. Butler, MD*	*Venetian 4, 5*

Sunday, May 16, 2010 continued

4:00 – 5:45 p.m.	**Concurrent Sessions:** **Poster Session I:** Basic Science & Oncology	 *Venetian 1, 2, 3*
	Poster Session II: Clinical & Fetal Surgery	*Venetian 4, 5*
5:45 – 6:30 p.m.	International Reception	*Michelangelo*
6:30 – 8:30 p.m.	Welcome Reception	*Harbor Piazza*

Monday, May 17, 2010

6:00 – 7:30 a.m.	Annual Fun Run	*Thirsty Fish*
6:30 – 7:30 a.m.	Committee Meetings	*Bernini 1, Bernini 2, Michelangelo 1, Michelangelo 2, DaVinci 1, DaVinci 2, Verona, Vincenza*
6:30 a.m. – 1:00 p.m.	Registration Open	*Tuscan Foyer*
6:45 a.m. – 1:15 p.m.	Exhibits Open	*Tuscan Foyer*
6:45 – 7:30 a.m.	Continental Breakfast	*Tuscan Foyer*
7:30 – 9:00 a.m.	**Scientific Session I:** Common Problems and Trauma	*Tuscan Ballroom*
9:00 – 10:00 a.m.	**Robert E. Gross Lecture:** *John D. Birkmeyer, MD* Measuring and Improving the Quality of Pediatric Surgery	*Tuscan Ballroom*
10:00 – 10:30 a.m.	Refreshment Break	*Tuscan Foyer*
10:30 – 11:45 a.m.	**Scientific Session II:** Neonatal	*Tuscan Ballroom*
11:45 a.m. – Noon	Introduction of New Members	*Tuscan Ballroom*
Noon – 1:00 p.m.	**Presidential Address:** *Keith Georgeson, MD* Pioneers, Cowboys and Desperados: A Brief History of Pediatric Surgical Innovation	*Tuscan Ballroom*
1:00 – 2:30 p.m.	Benjy Brooks Society Meeting	*Verona*
1:30 p.m.	Depart for Optional Events: Cooking Class Golf	 *Hotel Main Entrance* *Hotel Main Entrance*
5:00 – 6:30 p.m.	*Journal of Pediatric Surgery* Reception	*Verona*
5:30 – 6:30 p.m.	Residents' Reception	*Casa Dolce*
6:30 – 8:00 p.m.	New Members' Reception	*Casa Dolce*

Tuesday May 18, 2010

6:30 – 8:00 a.m.	Member Business Meeting and Breakfast	*Tuscan Ballroom*
6:30 a.m. – 3:30 p.m.	Registration Open	*Tuscan Foyer*
7:00 – 8:00 a.m.	Continental Breakfast for Non-Members	*Tuscan Foyer*
7:00 a.m. – 12:30 p.m.	Exhibits Open	*Tuscan Foyer*

Tuesday May 18, 2010 continued

8:00 – 9:30 a.m.	**Scientific Session III:** Practice Issues, Surgical Education, Outcomes	*Tuscan Ballroom*
9:30 – 10:30 a.m.	**Jay and Margie Grosfeld Lecture:** *Christopher K. Breuer, MD* The Development and Translation of the Tissue Engineered Vascular Grafts	*Tuscan Ballroom*
10:30 – 11:00 a.m.	Refreshment Break	*Tuscan Foyer*
11:00 a.m. – 12:15 p.m.	**Scientific Session IV:** Oncology and Critical Care	*Tuscan Foyer*
12:15 – 12:30 p.m.	Box Lunch Pick-Up	*Tuscan Ballroom*
12:30 – 1:30 p.m.	**New Technology Session wth Lunch**	*Tuscan Ballroom*
1:30 – 2:00 p.m.	**APSA Foundation Scholars** *Tippi C. MacKenzie, MD* The Maternal Immune Response to in Utero Hematopoietic Stem Cell Transplantation *Kelly A. Miller, MD* The Pathogenic Role of Enteric Glia in Hirschsprung's Enterocolitis	*Tuscan Ballroom*
2:00 – 2:30 p.m.	APSA Updates	*Tuscan Ballroom*
2:30 – 3:30 p.m.	**International Guest Lecture:** *Jan Deprest, MD* Prenatal Management of the Fetus with Isolated CDH	*Tuscan Ballroom*
6:45 – 7:30 p.m.	President's Reception	*Tuscan Foyer*
7:30 – 10:00 p.m.	President's Banquet	*Tuscan Ballroom*

Wednesday May 19, 2010

6:30 – 7:30 a.m.	Committee Meetings	*Verona, Vincenza*
7:00 – 11:30 a.m.	Registration Open	*Tuscan Foyer*
7:00 – 8:00 a.m.	Continental Breakfast	*Tuscan Foyer*
8:00 – 9:00 a.m.	**Scientific Session V:** Basic Science	*Tuscan Ballroom*
9:00 – 10:00 a.m.	**COG Session**	*Tuscan Ballroom*
10:00 – 10:15 a.m.	Refreshment Break	*Tuscan Foyer*
10:15 – 11:30 a.m.	**Scientific Session VI:** Pediatric Surgery Case Debates and Controversies	*Tuscan Ballroom*
11:30 a.m.	Annual Meeting Ends	
Noon – 6:00 p.m.	**APSA/IPEG Workshop** Advanced Neonatal Endoscopic Techniques *Separate registration required*	*Off Site*

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 16, with "Evidence-Based Medicine." Afternoon sessions include "Clinical Practice Guidelines and Outcomes Research" and "International Collaborations in Pediatric Surgery: Assessing and Meeting Needs." Meeting attendees will also view and discuss video and selected poster presentations.

Learning Objectives

At the conclusion of the meeting, attendees will:

- 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.

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

Policy on Faculty Disclosure

It is the policy of the ACCME and APSA that the faculty disclose and resolve real or apparent conflicts of interest relating to the content of the educational activity, and also disclose discussions of unlabeled/unapproved uses of drugs or devices during their presentations. The following faculty members have disclosed a financial relationship with an industry partner. The relationship was proven not to have an impact on the science presented at this annual meeting. All of these faculty members have agreed not to mention products or services provided by the industry partner during their presentations. All other faculty indicated that they have no financial relationships to disclose.

Disclosures

Disclosure forms were provided to and signed by all APSA 2009-2010 committee members.

Committees and Invited Speaker Disclosures

Charles W. Breaux, Jr.	Ownership Interest (i.e. stockholder): Covidien; Exelixis; General Electric; Intuitive Surgical; Johnson & Johnson; Medtronic; Mindray Medical.
Christopher K. Breuer	Grant/Research Support: Gunze Corporation; Pall Corporation; ATRM LLC.
Tory Meyer	Ownership Interest (i.e. stockholder): Strictly Pediatrics Surgery Center; Streth Pediatrics Office Building.
David J. Hackman	Grant/Research Support: Abbott Nutritionals
Steven L. Moulton	Grant/Research Support: 10 Blade Inc.; Flashback Technologies LLC. Ownership Interest (i.e. stockholder): 10 Blade Inc.; Flashback Technologies LLC.
J. Duncan Phillips	Consultant: Kimberly-Clark Corporation; Fresenius Corporation
Mark Puder	Other Financial/Material Interest: Patent submitted by Children's Hospital Boston for use of Omegaven.
Kirk W. Reichard	Ownership Interest (i.e. stockholder): Wellstat Biologics.
Timothy M. Crombleholme	Grant/Research Support: NIH grant support.
John R. Wesley	Consultant: Excelsior Healthcare. Ownership Intererest (i.e. stockholder): Baxter Healthcare.
W. Glaze Vaughan	Owndership Interest (i.e. stockholder): Ambulatory Surgery Center.
Charles S. Cox	Grant/Research Support: CBR Inc; ICCI Inc.; Atherys Inc. Ownership Interest (i.e. stockholder): Emit Corp. Speakers Bureau: CBR, Inc.
Allen F. Browne	Consultant: Allergan Medical Corporation.
Barbara A. Gaines	Grant/Research Support: Kohl's Department Stores; Allstate Corporation. Ownership Interest (i.e. stockholder): Phizer.
David L. Sigalet	Grant/Research Support: Crohn's & White Foundation of Canada Consultant: Ferring Pharmaceuticals; Wycond.

Abstract Presentations

Abstract 53, New Technology:

G.A. Villalona	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support
R. Sawh-Martinez	U.S. National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support
T. Mirensky	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support
N. Hibino	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/

	Research Support
T. Yi	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support
A. Shoffner	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support
E. McGillicuddy	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support
T. Shinoka	National Institutes of Health (NIH; K08HL83980–2), Grant/Research Support; Doris Duke Charitable Foundation Clinical Scientist Development Award, Grant/Research Support; Cytograft and Pall Corporation, Grant/Research Support.

Abstract 62, Scientific Session V:

T. Shinoka	Pall Corporation, Grant/Research Support; Cytograft Corporation, Grant/Research Support
C.K. Breuer	Pall Corporation, Grant/Research Support; Cytograft Corporation, Grant/Research Support.

Abstract 50, New Technology:

J.A. Deprest	European Commission euroSTEC grant, Instituut Wetenschap & Technologie (Flanders, Belgium), Grant/Research Support.

Abstract 55, New Technology:

T.A. Ponsky	Covidien, Stryker, Storz, Speaker's Bureau
E. Barksdale	Nestle, Speaker's Bureau
R. Onders	Synapse Biomedical, Consulting; Synapse Biomedical, Stockholder/Ownership.

Abstract P31, Poster Session II:

M. Puder	Patent pending for use of Omegaven as treatment for PN liver disease, Intellectual Property.

Commercial Support

APSA would like to thank the *Journal of Pediatric Surgery* for its educational grant for the 2010 *Journal of Pediatric Surgery* Lecture and its educational grant for the transcription of the annual meeting technical sessions. APSA also thanks the current supporters and exhibitors for their unrestricted educational grants.

Supporters

ELSEVIER

Exhibitors

American College of Surgeons
American Pediatric Surgical Nurses Association
American Pediatric Surgical Association Foundation
AMT- Applied Medical Technology, Inc
Bentec Medical
Biomet Microfixation
CHERUBS- The Association of Congenital Diaphragmatic Hernia Research, Awareness and Support
Community Health Network
Elsevier
Endo Pharmaceuticals
Hospital Corporation of America
Integra Surgical
IPSAC-VN International Pediatric Specialists Alliance for the Children of Vietnam
Kaiser Permanente
Karl Storz Endoscopy America, Inc
LocumTenens.com
Mediflex Surgical Products
Meridian Health
Ossur Americas
Pediatric Search Partners
Providence Health & Services
Raymond Blank Children's Hospital
Specialty Surgical Products, Inc
Stryker Endoscopy
The Children's Medical Center of Dayton
Ultimate Escape Luxury Destination Clubs
Weatherby Locums

Registration

All authors presenting a paper at the APSA 41st Annual Meeting are required to pay a registration fee.

The onsite registration fees for the annual meeting are:

APSA 41st Annual Meeting

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

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

*** Registration for the APSA 41st Annual Meeting only; APSNA registration is by separate subscription.*

APSA Registration Desk

Registration will be located Tuscan Foyer during the following times:

Saturday, May 15	4:00 – 6:00 p.m.
Sunday, May 16	7:00 a.m. – 5:00 p.m.
Monday, May 17	6:30 a.m. – 1:00 p.m.
Tuesday, May 18	6:30 a.m. – 3:30 p.m.
Wednesday, May 19	7:00 – 11:30 a.m.

Scientific Sessions

All educational sessions will be held in the Tuscan Ballroom. Education Session III on Sunday will be held in Venetian 4 and 5. The daily dress code is business or business casual attire.

Poster Set Up and Viewing

Scientific posters will be located in Venetian 1, 2, 3 for viewing during the following hours:

Sunday, May 16	4:00 – 5:45 p.m. Oral presentations only (no posters)
Monday, May 17	6:30 – 10:00 a.m. Poster Set Up
	10:00 a.m – Noon Poster Viewing
Tuesday, May 18	7:00 a.m. – 3:00 p.m. Poster Viewing
	3:30 p.m. – 5:30 p.m. Poster Dismantle

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

Speaker Ready Room

The Speaker Ready Room will be available daily in Ligurian 1. Computers will be provided for speakers to review their presentations. The room will be open during the following times:

Saturday, May 15	4:00 – 6:00 p.m.
Sunday, May 16	7:00 a.m. – 5:00 p.m.
Monday, May 17	6:30 a.m. – 1:00 p.m.
Tuesday, May 18	6:30 a.m. – 3:30 p.m.
Wednesday, May 19	7:00 – 10:00 a.m.

Presentation Check-In

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

Exhibits

Commercial exhibits will be located in the Tuscan Foyer and will be open during the following hours:
Monday, May 17 .....6:45 a.m. – 1:15 p.m.
Tuesday, May 18 .....7:00 a.m. – 12:30 p.m.

Continental breakfast and scheduled coffee breaks will be served in the exhibit area on Monday and Tuesday. For a list of exhibitors and booth assignments, see page 80.

APSA Business Meeting

The APSA Business Meeting will be held from 6:30 – 8 a.m. on Tuesday, May 18, in the Tuscan Ballroom. This is a breakfast meeting and is for APSA members only.

Welcome Reception

A Welcome Reception for all registrants will take place on Monday, May 17 in the Harbor Piazza 6:30 – 8:30 p.m. Tickets for this reception will be included in your registration packet and will be required for admission to the reception. All guests 12 years and older will need a ticket to be admitted to the Welcome Reception. Casual attire is appropriate.

President's Banquet

The President's Banquet will be held on Tuesday, May 18 in the Tuscan Ballroom. The reception will begin at 6:45 p.m. in the Tuscan Foyer and dinner will begin at 7:30 p.m. After dinner, you are invited to stay for dancing. Tickets for the reception and banquet are included in your registration packet and will be required for admission. All guests 12 years and older will need a ticket to be admitted to the banquet. Business or cocktail attire is requested.

Child Care Services

Childcare facility, Campo Portofino®, is located next to the Beach Pool on the west side of the hotel. Children 4-14 years of age are welcome. Children must be potty trained. Campo Portofino is open from 5:00 – 11:30 p.m. Sunday through Thursday and 5:00 p.m. – 12:00 a.m. Friday and Saturday nights. Hours of operation do vary seasonally. Cost per child is $15 per hour. Children using this facility are supervised at all times by certified childcare professionals. Reservations must be made at least 24 hours in advance with the concierge by calling +1-407-503-1200. Other babysitting services may be arranged through the concierge. Please note: the agencies and babysitters referred by the concierge are not affiliated with the hotel and consequently, the hotel is not responsible for the services rendered by the agencies or babysitter.

Companion Hospitality Suite

The hospitality suite, #2325, will be open Sunday, Monday and Tuesday from 8 – 10 a.m. Continental breakfast will be served each morning for registered accompanying guests. Badges are requested for entry to the hospitality suite.

Benjy Brooks Meeting and Luncheon

Monday, May 17, 1 – 2:30 p.m., Verona
Join us for a meeting of the Benjy Brooks Society while enjoying lunch. Discuss issues that women are currently facing in the pediatric surgery arena and talk about the society's future. Plan to attend this informal session. Lunch and an agenda will be provided to those who register. The participation fee is $38.

Optional Activities

Golf Tournament

Monday, May 17, 2:15 p.m., Shingle Creek Golf Course

The 2010 APSA Golf Tournament will be held at the Shingle Creek Golf Club located just a few miles from the Loews Portofino Bay Hotel. The par 72 course offers a layout that is challenging and flexible enough to test your skills without frustrating the less experienced players. Lush tropical landscaping, a lazy creek that meanders through the course, undulated fairways and hard, fast greens make this a truly enjoyable Florida style golf experience.

- Players are required to register by April 5.
- On the registration form, indicate your handicap and if you will be renting golf clubs. You may pay for the rental clubs at the golf course.
- The golf tournament will take place at 2:15 p.m. on Monday, May 17. It will be a shotgun start at the Shingle Creek Golf Club.
- Transportation will depart promptly at 1:30 p.m. from the main entrance.
- Proper attire for men includes a collared shirt, together with Bermuda-length shorts or slacks. Proper attire for women consists of a blouse and either a skirt, slacks or mid-thigh-length shorts. No denim-colored clothing of any kind may be worn. Soft spikes are required.
- The tournament fee is $104 per golfer and includes transportation to/from the course, greens fee, cart, scramble tournament, box lunch, a bottle of water and awards for the top players.
- For more information about the course, visit http://www.ShingleCreekGolf.com.

5K Fun Run

Monday, May 17, 6:00 a.m. Meet at the Thirsty Fish on the Harbor Piazza.

This is a popular event you won't want to miss — APSA's annual 5K Fun Run will be held on Monday, May 17, at 6 a.m.

- Sign-in will begin at 5:30 a.m. with an organized warm up and stretch at 5:40 a.m.
- The run will be on pavement (not sand or gravel), so bring appropriate running shoes.
- The participation fee is $70 and includes a Fun Run T-shirt, water stations along the route, directional arrows placed along the course, staff to facilitate the event, a light breakfast after the run, and awards in a number of categories.

Cooking Demo/Class

Monday, May 17, 1:30 p.m.

The Westye Group Southeast, Inc. houses an exciting appliance showroom featuring a live demonstration kitchen, wine storage galleries, laundry rooms, outdoor barbecue areas and more. Upon arrival you will be escorted to the demonstration kitchen to delight in an interactive gourmet cooking class. A talented chef will welcome you and show you how to prepare a delectable meal while using the state-of-the-art appliances featured in the showroom. Using cooking stations arranged throughout the room, you will create the different items which will be served to the group afterwards for your enjoyment.

- The tour will leave the Loews Portofino Bay Hotel main entrance promptly at 1:30 p.m. and return at approximately 5:30 p.m.
- Tour cost is $100 per person and includes transportation, hands-on cooking demo, appetizers, meal and beverages.
- Minimum of 20 people and maximum of 25 people.

Messages

A message board will be maintained in the registration area during registration hours. Check the board frequently, as there will be NO PAGING during the meeting. To contact the message center, dial the hotel operator and request the APSA Registration Desk.

Guidelines for Authors and Discussants

1. Authors presenting papers are reminded that the presentations shall be limited to time previously indicated.
2. Your presentation materials (ZIP disk, flash disk or CD-Rom) must be labeled and submitted to the A/V technician Room by 1 p.m. the day before they are to be presented.
3. Posters have been sorted into two sessions, scheduled on Sunday, May 16, between 4:00 and 5:45 p.m. Oral presentations for each poster will be presented at this time, but posters will not be on display until the following morning. Refreshments will be available during the Poster Sessions on Sunday.
4. Scientific posters will be hung on Monday morning (following the oral poster presentations) between 6:30 and 10 a.m. and will be available for viewing after that. Authors are requested to be in attendance during the continental breakfasts and general session breaks each day to discuss their presentations.
5. Discussants from the floor should state their name and affiliation prior to their remarks. The discussions will be audio-recorded for transcription and printing in the *Journal of Pediatric Surgery*.
6. Typed discussion should be limited to a maximum of 200 words. Typed discussions that exceed 200 words will be edited before they are submitted to the *Journal of Pediatric Surgery* for publication.
7. Discussants will have the opportunity to edit a transcript of their remarks following the meeting. The Publications Committee reserves the right to edit the typed discussion before it is submitted to the *Journal of Pediatric Surgery*.

Past APSA Annual Meeting Dates and Locations

40th Annual Meeting
May 28-30, 2009
El Conquistador Golf Resort & Golden Door Spa
Fajardo, Puerto Rico

39th Annual Meeting
May 27-31, 2008
JW Marriott Desert Ridge
Phoenix, Arizona

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

37th Annual Meeting
May 21–24, 2006
Marriott Beach & Golf Resort
Hilton Head, South Carolina

36th Annual Meeting
May 29–June 1, 2005
JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona

35th Annual Meeting
May 27–30, 2004
Sawgrass Marriott Resort
Ponte Vedra Beach, Florida

34th Annual Meeting
May 25–28, 2003
Marriott Harbor Beach Resort & Spa
Ft. Lauderdale, Florida

33rd Annual Meeting
May 19–22, 2002
The Arizona Biltmore Resort and Spa
Phoenix, Arizona

32nd Annual Meeting
May 20–23, 2001
The Registry Resort
Naples, Florida

31st Annual Meeting
May 25–28, 2000
Walt Disney World Swan
Lake Buena Vista, Florida

30th Annual Meeting
May 16–19, 1999
Westin Mission Hills
Rancho Mirage, California

29th Annual Meeting
May 10–13, 1998
The Hyatt Regency
Hilton Head, South Carolina

Past APSA Annual Meeting Dates and Locations cont.

28th Annual Meeting
May 18–21, 1997
The Registry Resort
Naples, Florida

27th Annual Meeting
May 19–22, 1996
The Hyatt Regency
San Diego, California

26th Annual Meeting
May 20–23, 1995
The Boca Raton Resort and Club
Boca Raton, Florida

25th Annual Meeting
May 14–17, 1994
Loews Ventana Canyon Resort
Tucson, Arizona

24th Annual Meeting
May 15–18, 1993
The Hyatt Regency
Hilton Head, South Carolina

23rd Annual Meeting
May 12–16, 1992
The Broadmoor
Colorado Springs, Colorado

Future Meetings

42nd Annual Meeting
May 22–25, 2011
JW Marriott Desert Springs Resort & Spa
Palm Desert, California

43rd Annual Meeting
May 20-23, 2012
JW Marriott San Antonio Hill Country
Resort & Spa
San Antonio, Texas

44th Annual Meeting
May 2-5, 2013
Marco Island Marriott Beach Resort,
Golf Club & Spa
Marco Island, Florida

APSA 45th Annual Meeting
May 29-June 1, 2014
JW Marriott Desert Ridge Resort & Spa
Phoenix, Arizona

APSA 46th Annual Meeting
April 30-May 3, 2015
Harbor Beach Marriott Resort & Spa
Fort Lauderdale, Florida

Past Annual Meeting Robert E. Gross Lectures

2009
Stanley B. Prusiner, MD
"Designer Prions and a Quest for Therapy"

2008
Michael W.L. Gauderer, MD
"Creativity and the Surgeon"

2007
Francisco G. Cigarroa, MD
"Leading an Academic Health Center in the 21st Century: A Pediatric Surgeon's Perspective"

2006
Diana Bianchi, MD
"Fetomaternal Cell Trafficking: A Story that Begins with Prenatal Diagnosis and May End with Stem Cell Therapy"

2005
W. Hardy Hendren, MD
"Looking Back 50 Years"

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

2003
Lucien Leape, MD
"Safe Health Care — Are We Up to It?"

2002
Harold Shapiro, PhD
"The Ethical Dimensions of Scientific Progress"

2001
Judah Folkman, MD
"Angiogenesis-Dependent Diseases"

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

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

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

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

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

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

1994
W. French Anderson, PhD
"Human Gene Therapy"

1993
Judah Folkman, MD
"Clinical Applications of Angiogenesis Research"

1992
Warren Zapol, MD
"Inhaled Nitric Oxide: A Selective Vaso-Dilator"

1991
Joel Cooper, MD
"History and Current Status of Lung Transplantation"

1990
Richard Simmons, MD
"Role of the Gut Flora in Surgery"

Past Annual Meeting Jay & Margie Grosfeld Lectures

2009
Michael T. Longaker, MD, MBA, FACS
"Regenerative Medicine: A Surgeon's Perspective"

2008
Frederick J. Rescorla, MD
"What's New in Pediatric Surgery"

Past Annual Meeting Overseas/International Guest Lecturers

2009
Marcelo Martinez Ferro, MD
"New Approaches to Pectus and Other MIS in Argentina"

2008
Tadashi Iwanaka, MD
"Technical Innovation, Standardization and Skill Qualification of Pediatric Minimally Invasive Surgery in Japan"

2007
Claire Nihoul-Fékété, MD
"Is Regionalism of Complex Pediatric Malformations Desirable and Feasible? The Example of Disorders of Sexual Development"

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

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

2003
Claire Nihoul-Fékété, MD
"Modern Surgical Management of Congenital Hyperinsulinemic Hypoglycemia"

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

2001
Pedro Rosselló, MD
"One Nation, with Liberty and Justice…and Healthcare for All"

2000
Leela Kapila, FRCS
"Are These the Children of a Lesser God?"

1999
Bernardo Ochoa, MD
"Pediatric Surgery in Latin America"

1998
Sidney Cywes, MD
"Some of the Little Things We Do — Something Old, Something New"

1997
Justin Kelly
"Bladder Exstrophy — Problems and Solutions"

1996
Prem Puri, MD
"Variant Hirschsprug's Disease"

1995
Sir Lewis Spitz, MD, PhD, FRCS
"Esophageal Atresia — Past, Present and Future"

1994
Sean J. Corkery, MCh, FRCSI, FRCSEng
"In Pursuit of the Testis"

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

1992
Yann Revillon, MD
"Intestinal Transplantation in France"

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

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

Past Annual Meeting *Journal of Pediatric Surgery* Lectures

2008

Thomas M. Krummel, MD
"Inventing Our Future: Training the Next Generation of Surgeon Innovators"

2007

Alan W. Flake, MD
"Stem Cell Biology and Pediatric Surgery – Deciphering the Venn Diagram"

2006

Pedro Rosselló, MD
"The Unfinished Business of American Healthcare"

2005

Alberto Peña, MD
"Luck and Serendipity, the History of a Surgical Technique"

2004

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

2003

Patricia 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 Lecture:
John D. Birkmeyer, MD
"Measuring and Improving the Quality of Pediatric Surgery"

John D. Birkmeyer, MD, a graduate of Boston College and Harvard Medical School, completed his General Surgery residency and a fellowship in Outcomes Research at Dartmouth. He then joined the Dartmouth faculty in 1996, later serving as Chief of the Section of General Surgery. He moved to the University of Michigan in 2004, where he is the George D. Zuidema Professor of Surgery and Director of M-SCORE. Dr. Birkmeyer has broad interests in outcomes research, claims data analysis, and value-based purchasing strategies in surgery. With funding from the National Cancer Institute, his current research is exploring why some hospitals and surgeons have better outcomes than others, with ultimate goal of improving care in all settings. Research funded by the National Institute on Aging is aimed at developing hospital-level measures of costs and quality with administrative data. He chairs the expert panel on evidence-based hospital referral for the Leapfrog Group, a large coalition of public and private health care purchasers. He was elected to the Institute of Medicine of the National Academy of Sciences in 2006.

Learning Objectives:

- To give surgeons a framework for understanding different measures of quality in pediatric surgery
- To review the trade-offs associated with different strategies for quality improvement
- To make surgeons aware of basic principles of collaborative quality improvement

***Journal of Pediatric Surgery* Lecture:**
Robert H. Bartlett, MD
"ECMO: Gross, Beethoven, Krummel and Georgeson"

Dr. Robert Bartlett developed extracorporeal life support (ECLS) from the laboratory through the first successful clinical trials to routine practice worldwide. ECLS has led to new understanding of the pathophysiology of renal, cardiac, and pulmonary failure, which provides the basis for much of modern critical care. Dr. Bartlett continues laboratory and clinical research at the University of Michigan where he is Professor of Surgery, Emeritus.

Learning Objectives:

- Who was Gross?
- Indications for ECMO
- Results of ECMO in Children

Jay & Margie Grosfeld Lecture:
Christopher K. Breuer, MD
"The Development and Translation of the Tissue Engineered Vascular Grafts"

Dr. Christopher Breuer is an Associate Professor of Surgery at Yale University School of Medicine. He received his undergraduate degree from the College of the Holy Cross and is a graduate of the Dartmouth-Brown Program in Medicine. Dr. Breuer completed his general surgical training at Brown University and his pediatric surgical fellowship at Hasbro Children's Hospital under the guidance of Dr. Thomas Tracy. Dr. Breuer is a surgeon-scientist with a research interest in translational vascular tissue engineering. He received his training in tissue engineering while working as a surgical research fellow under the mentorship of Dr. Joseph Vacanti at Boston Children's Hospital. Dr. Breuer runs a NIH-funded laboratory focusing on the cellular and molecular mechanisms of neovessel formation. Along with his collaborator Dr. Toshiharu Shinoka, he recently received approval from the FDA supporting the first clinical trial evaluating the use of tissue engineered vascular grafts in children. Dr. Breuer is past recipient of the APSA Foundation Enrichment Award, ASA Research Fellowship, the Doris Duke Charitable Foundation Research Award, and ACS Jacobson Award. He is married to his wife Julie and has three sons, Kane (10), Thomas (9), and Christopher (7).

Learning Objectives:

- Define the classic tissue engineering paradigm
- Describe cell trafficking with regards to neotissue formation
- Explain the role of the host in neotissue formation

International Guest Lecture:
Jan Alice Marcel Deprest, MD
"Prenatal Management of the Fetus with Isolated CDH"

Jan Alice Marcel Deprest was born in Bruges, Belgium and received his degree in medicine in 1985 from KU Leuven, Belgium. He became a Specialist in Gynaecologist and Obstetrics in 1991. He obtained his doctorate degree (PhD) in the use of endoscopy in fetal medicine at de Medische Wetenschappen, KU Leuven, Belgium in 1995.

Dr. Deprest is currently a Professor of Obstetrics and Gynaecology at KU Leuven. He is head of the programme of Fetal Therapy University Hospitals Leuven — the largest referral centres for fetal diagnosis and therapy in Belgium, offering all standard non-invasive and invasive diagnostic tests and procedures. He is the director of the Centre for Surgical Technologies, a research and training centre in new surgical technologies, mainly endoscopic surgery. He is a co-director of the Pelvic Floor Programme, with Professor D. De Ridder (Urology), that runs the mono- as well as multidisciplinary clinics, the urogynaecological ultrasound unit and a research programme; editor of Prenatal Diagnosis; and is a clinical researcher (2000-2010) at the Fonds Wetenschappelijk Onderzoek Vlaanderen.

Dr. Deprest has published over 250 peer-reviewed papers in scientific journals, over 50 book chapters, supervises over 14 doctoral students in research on fetal medicine or urogynaecology, and has developed new hardware and methods for fetal therapy, such as fetoscopic instrumentation, cord occlusion and endoscopic tracheal occlusion for diaphragmatic hernia.

Learning Objectives:

- To describe methods for 2D-ultrasound prediction of the condition, as a method that is widely available
- To report on current experience with volumetric assessment of lung hypoplasia and liver herniation in terms of outcome
- To describe (scanthy) experience with prenatal assessment of lung vascularisation and resistance in the pulmonary circulation

Given that prenatal therapy is part of the objectives:

- To describe currently experience with Fetoscopic Endoluminal Tracheal Occlusion (FETO) in Europe
- To report on current status of trials of prenatal treatment of CDH in EUROPE

Program in Detail

Saturday, May 15

8:00 a.m. – 2:00 p.m.	APSA Board of Governors Meeting	*Donatello*
2:00 – 9:00 p.m.	Committee Meetings	*Bernini, Michelangelo 1, Michelangelo 2, DaVinci*
2:00 – 6:00 p.m.	Training Program Directors Meeting	*Tuscan 1*
4:00 – 6:00 p.m.	Registration Open	*Tuscan Foyer*
6:30 – 10:00 p.m.	Publications Committee Meeting	Del Mare
7:00 – 10:00 p.m.	APSA Board of Governors Dinner	*Bice Restaurant (Venezia Room)*

Sunday, May 16

6:00 – 7:30 a.m.	Committee Meetings	*Bernini 1, Bernini 2, Michelangelo 1, Michelangelo 2, DaVinci 1, DaVinci 2 Donatello, Vincenza*
7:00 a.m. – 5:00 p.m.	Registration Open	*Tuscan Foyer*
7:00 – 7:45 a.m.	Continental Breakfast	*Tuscan Foyer*
7:45 – 8:00 a.m.	**President's Welcome – Keith E. Georgeson, MD**	*Tuscan Ballroom*
8:00 – 11:00 a.m.	*Education Session I* **Evidence-Based Medicine in Pediatric Surgery**	*Tuscan Ballroom*

Moderator
George W. Holcolmb, III, MD, MBA

Educational Objectives:

- To describe why medical care is seemingly grounded based on anecdotal evidence and a physician's personal experience.
- To describe the current landscape of evidence-based medicine within pediatric surgery.
- To describe health services research within academic surgery.
- To describe how prospective clinical research can be performed at one's institution.

Human Nature and Personal Experience: The Pediatric Surgeon's Worst Enemies
R. Lawrence Moss, MD
Hasbro Children's Hospital, Providence, RI, USA

Evidence-Based Medicine within Pediatric Surgery
Daniel J. Ostlie, MD
Children's Mercy Hospital, Kansas City, MO, USA

Health Services Research and Changing Paradigms in Academic Surgery
John D. Birkmeyer, MD
University of Michigan, Ann Arbor, MI, USA

How to Develop a Center for Prospective Clinical Trials at Your Institution
Shawn D. St. Peter, MD
Children's Mercy Hospital, Kansas City, MO, USA

11:00 a.m. – 11:30 a.m.	Refreshment Break	*Tuscan Foyer*
11:30 – 12:30 p.m.	***Journal of Pediatric Surgery* Lecture:** **Robert H. Bartlett, MD** **ECMO: Gross, Beethoven, Krummel and Georgeson**	*Tuscan Ballroom*
12:30 – 12:45 p.m.	Box Lunch Pick-Up	*Tuscan Ballroom*
12:45 – 1:45 p.m.	**Video Session with Lunch**	*Tuscan Ballroom*

Moderators:
R. Cartland Burns, MD; Peter F. Nichol, MD

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

- Describe technical aspects of anorectal and perineal surgery
- Cite the technical aspects of a transoral approach to reflux disease
- Describe the minimally invasive treatment options for several congenital lesions

V1 **ENDOSCOPIC-GUIDED EXCISION OF A MIDLINE FOREHEAD DERMOID**
Sherif Emil, MD,CM[1], Nabil Fanous, MD[2], Valerie Cote, MD,CM[2].
[1]Montreal Children's Hospital, Montreal, QC, Canada, [2]McGill University Health Centre, Montreal, QC, Canada

V2 **RIGHT THORACOSCOPY FOR BILATERAL CONGENITAL CYSTIC LUNG LESIONS**
Abigail E. Martin, MD, Francois I. Luks, MD
Hasbro Children's Hospital, Providence, RI, USA

V3 **TRANSANAL RESECTION, HOW TO AVOID FECAL INCONTINENCE**
Alberto Pena, MD, Andrea Bischoff, MD, Marc A. Levitt, MD
Cincinnati Children Hospital, Cincinnati, OH, USA

V4 **THE PRONE POSITION FOR COLORECTAL PROBLEMS: NOT JUST FOR ANORECTAL MALFORMATIONS**
Kaveer Chatoorgoon, MD, Belinda Dickie, MD PhD, Alberto Pena, MD, Andrea Bischoff, MD, Richard A. Falcone, MD, MPH, Jason S. Frischer, MD, Marc A. Levitt, MD
Cincinnati Children's Hospital, Cincinnati, OH, USA

V5 **TRANSORAL FUNDOPLICATION IN A CHILD WITH RECURRENT GERD AFTER FUNDOPLICATION**
Shaun Kunisaki, MD, Marcus S. Jarboe, MD, Daniel H. Teitelbaum, MD
University of Michigan, Ann Arbor, MI, USA

V6 **LAPAROSCOPIC RESECTION OF CHOLEDOCHAL CYST: RECONSTRUCTION BY HEPATICODUODENOSTOMY**
Katherine J. Deans, MD, Thane A. Blinman, MD, Alan W. Flake, MD
Children's Hospital of Philadelphia, Philadelphia, PA, USA

V7 **SINGLE PORT, PERCUTANEOUS LYSIS OF AMNIOTIC BANDS**
Shinjiro Hirose, MD, Hanmin Lee, MD
University of California San Francisco, San Francisco, CA, USA

2:00 – 4:00 p.m.	**Concurrent Sessions II & III:** **Education Session II:** **Outcomes and Clinical Trials Committee** **Evidence-Based Clinical Practice Guidelines and Outcomes Research: Road to the Future**	*Tuscan Ballroom*

Moderators:
Marjorie J. Arca, MD; Fizan Abdullah, MD, PhD

Educational Objectives:
- Understand the current, evidence-based data about the use of antibiotics in appendicitis patients
- Understand the current, evidence-based data about outcomes regarding closure techniques in neonates with gastroschisis and omphalocele
- Learn about the results of the Research Funding Survey conducted by APSA Outcomes Committee in December 2009
- Understand the basic definitions of outcomes research
- Learn about available clinical databases and their applicability by subspecialty interest
- Participate in a step-by-step example of a hypothesis-driven research plan development utilizing patient databases

Introduction and Summary of Research Funding Survey Results
Marjorie J. Arca, MD
Children's Hospital of Wisconsin, Milwaukee, WI, USA

Systematic Literature Reviews of Clinical Problems Use of Antibiotics in Appendicitis
Steven L. Lee, MD
Harbor-UCLA Medical Center, Torrance, CA, USA

Abdominal Wall Defects: Methods of Closure
Saleem Islam, MD
University of Florida, Gainesville, FL, USA

Outcomes Research Primer, Tutorial & Panel Discussion
Outcomes Research in Pediatric Surgery: Resources, Methods & Controversies
Fizan Abdullah, MD, PhD
Johns Hopkins University School of Medicine, Baltimore, MD, USA

David Chang, PhD, MPH, MBA
University of California San Diego, San Diego, CA, USA

Questions to Expert Panel
Fizan Abdullah, MD, PhD
Gudrun Aspelund, MD, MS
Randall S. Burd, MD
David Chang, PhD, MPH, MBA
Adam M. Goldin, MD, MPH
Laura Cassidy, PhD

2:00 – 4:00 p.m. **Education Session III: International Relations Committee** *Venetian 4, 5*

Moderators:
Marilyn W. Butler, MD; Georges Azzie, MD

Educational Objectives:
- Learn about the global burden of surgical diseases and the consideration of pediatric surgery as a public health issue in low- and middle-income countries
- Become aware of the challenges individuals and organizations face in terms of needs assessment, whether the volunteer work be short-term relief work or long-term capacity-building collaborations
- Learn about practical considerations of international volunteerism in pediatric surgery, including the role of trainees

International Collaborations in Pediatric Surgery: Assessing and Meeting Needs
The Global View
Steven D. Bickler, MD
Rady Children's Hospital, San Diego, CA, USA

The Need
Lohfa Chirdan, MBBS
University of Jos, Jos, Plateau, Nigeria

The Assessment
Donald E. Meier, MD
Paul L. Foster School of Medicine, Texas Tech University HSC, El Paso, TX, USA

The Challenge of Meeting Needs
J. Ted Gerstle, MD
Hospital for Sick Children, Toronto, Ontario, Canada

The Role of Trainees
Diana L. Farmer, MD
University of California San Francisco, San Francisco, CA, USA

The Nuts and Bolts
Marilyn W. Butler, MD
Lucile Packard Children's Hospital, Stanford, CA, USA

4:00 – 5:45 p.m. **Concurrent Popster Sessions I & II:**
Poster Session I: Basic Science & Oncology *Venetian 1, 2, 3*

Moderators:
C. Mac Harmon, MD; Karl G. Sylvester, MD

Educational Objectives:
At the completion of this session, participants will be able to:

- Describe ongoing research into the biochemical characteristics and control of neuroblastoma
- Cite advances is the understanding and development of repair for fetal tracheal defects
- Describe developments in the understanding of neonatal sepsis and necrotizing enterocolitis
- Review recent contributions to the sources and utility of stem cells

P1 **GROWTH IN MICROGRAVITY CULTURE MIMICS BIOLOGICAL BEHAVIOR OF NEUROBLASTOMA CELL LINES**
Edward J. Doolin, MD, Robert E. Redden, MSc, Jane E. Minturn, MD, Garrett M. Brodeur, MD
Children's Hospital of Philadelphia, Philadelphia, PA, USA

P2 **NERVE GROWTH FACTOR ACTIVATION OF CASPASE-INDUCED APOPTOSIS IN HUMAN SK-N-SH NEUROBLASTOMA IN VITRO**
Janette L. Holub, MD, MPH, Yi-Yong Qiu, MD, Mary Beth Madonna, MD
Children's Memorial Research Center, Chicago, IL, USA

P3 **NOTCH 3 ACTIVATION INCREASES PROLIFERATION AND HAS NO EFFECT ON MIGRATION IN A NEUROBLASTOMA TUMOR MODEL**
Jeffrey W. Gander, MD, Sonia L. Hernandez, Sae J. Kim, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD
Children's Hospital of New York-Presbyterian, New York, NY, USA

P4 **CHARACTERIZATION OF TUMOR-STEM LIKE CELLS AND RESISTANCE TO STANDARD THERAPIES IN MOUSE NEUROBLASTOMA**
Thamara Abouantoun, PhD, Steven T. Elliott, MD, Felix C. Blanco, MD, Stanislav Vukmanovic, PhD, Suzanne A. Miles, PhD, Anthony D. Sandler, MD
Children's National Medical Center, Washington, DC, USA

P5 **THE ROLE OF DENDRITIC CELLS IN NECROTIZING ENTEROCOLITIS INDUCED BY ENTEROBACTER SAKAZAKII**
Claudia N. Emami, MD, Rahul Mittal, PhD, Prasadarao V. Nemani, PhD, Henri R. Ford, MD, FACS
Children's Hospital Los Angeles, Los Angeles, CA, USA

P6 **HEPATIC MITOCHONDRIAL METABOLISM, MEASURED USING [1-13C]METHIONINE BREATH TEST, IN SEPTIC AND NON-SEPTIC INFANTS**
Haitham Dagash, MD, Augusto Zani, MD, Mark J. Peters, MD, Allan Goldman, MD, Agostino Pierro, MD, Simon Eaton, MD
UCL Institute of Child Health, London, United Kingdom

P7 **SUBMUCOSAL GLAND DEVELOPMENT IN A FETAL TRACHEA HUMAN-XENOGRAFT MODEL: IMPLICATIONS FOR FETAL GENE THERAPY**
Louis D. Le, MD, Sundeep G. Keswani, MD, Foong Y. Lim, MD, Mounira A. Habli, MD, Timothy M. Crombleholme, MD
Cincinnati Children's Hospital, Cincinnati, OH, USA

P8 **AMNIOTIC CELLS AUGMENT FETAL WOUND HEALING**
Justin D. Klein[1], Christopher G. Turner[1], Grace A. Nicksa[1], Azra Ahmed[1], Taro Koyama[2], David Zurakowski[1], Elof Eriksson[2], Dario O. Fauza[1]
[1]Children's Hospital Boston, Boston, MA, USA, 2Brigham and Women's Hospital, Boston, MA, USA

P9 **BILE DUCT EPITHELIAL CELLS; A NOVEL SOURCE OF NEUTROPHIL COLLAGENASE (MMP8)**
Christopher S. Muratore, MD, Mark W. Harty, PhD, Yonghong Zhou, David Mills, PhD, Thomas F. Tracy, MD
Hasbro Children's Hospital/Rhode Island Hospital, Providence, RI, USA

P10 **ENDOTHELIN CONVERTING ENZYME-1 PROMOTES RECYCLING OF THE NEUROKININ-1 RECEPTOR IN ENTERIC NEURONS**
Juan C. Pelayo, MD, Daniel P. Poole, PhD, Nigel W. Bunnett, PhD
University of California San Francisco, San Francisco, CA, USA

P11 **MATRIX METALLOPROTEINASE-9 INDUCES VASCULAR HYPERPERMEABILITY FOLLOWING TRAUMATIC BURN INJURY**
Hayden W. Stagg, MD, J. Greg Whaley, MD, Binu Tharakan, PhD, Felicia A. Hunter, Danny C. Little, MD, Ed W. Childs, MD
Texas A&M-Scott & White, Temple, TX, USA

P12 **EXPRESSION OF IROQUOIS GENES IS UPREGULATED DURING EARLY LUNG DEVELOPMENT IN THE NITROFEN INDUCED PULMONARY HYPOPLASIA**
Takashi Doi, MD, Aušra Lukoši, Jens Dingemann, MD, Elke Ruttenstock, MD, Prem Puri, MS, FRCS, FRCS (ED), FACS
The Children's Research Centre, Dublin, Ireland.

P13 **TRACHEAL DEFECT REPAIR USING A PLGA-COLLAGEN HYBRID SCAFFOLD REINFORCED BY A COPOLYMER STENT WITH BASIC FIBROBLAST GROWTH FACTOR-IMPREGNATED GELATIN HYDROGEL**
Yukihiro Tatekawa, MD, Guoping Chen, PhD, Hiroaki Komuro, MD, Michio Kaneko, MD
Tsukuba University, Tsukuba, Japan.

P14 **CELL LINEAGE TRACING OF TISSUE-ENGINEERED SMALL INTESTINE IN THE MOUSE MODEL DEMONSTRATES CONTRIBUTIONS TO THE STEM CELL NICHE AND THE ENTIRE EPITHELIUM**
Frederic G. Sala, PhD, Jamil A. Matthews, MD, Allison L. Speer, MD, Dianne C. Skelton, Tracy C. Grikscheit, MD
Childrens Hospital Los Angeles, Los Angeles, CA, USA

P15 **THE DEVELOPMENT OF NECROTIZING ENTEROCOLITIS RESULTS IN UPREGULATION OF THE WNT TARGET AND INTESTINAL STEM CELL SIGNATURE GENE, ASCL2: A NOVEL PATHWAY LEADING TO INTESTINAL STEM CELL IMPAIRMENT AFTER BARRIER INJURY**
Matthew D. Neal, MD, Chinder Sodhi, PhD, Anthony Russo, BS, David J. Hackam, MD, PhD
Division of Pediatric Surgery, Pittsburgh, PA, USA

P16 **A NOVEL MODEL FOR INVESTIGATING THE EFFECTS OF VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) IN THE MOUSE MESENCHYME**
Jamil A. Matthews, MD, Frederic G. Sala, PhD, Tracy C. Grikscheit, MD
Saban Institute-Children's Hospital Los Angeles, Los Angeles, CA, USA

P17 **COMPARISON OF SMALL MOLECULES AND ACTIVIN/WNT3A IN DERIVING DEFINITIVE ENDODERM FROM EMBRYONIC STEM CELLS**
Blair Roszell, MS[1], Ariel Seaton, BSc[1], Christine M. Finck, MD[2]
[1]University of Connecticut Health Center, Farmington, CT, USA, [2]Connecticut Children's Medical Center, Hartford, CT, USA

4:00 – 5:45 p.m. **Poster Session II: Clinical & Fetal Surgery** *Venetian 4, 5*

Moderator:
Daniel von Allmen, MD

Educational Objectives:
Participants in this session will acquire knowledge of:
- Technical approaches to common congenital and clinical problems in children
- Diagnostic approaches to appendicitis and chest wall abnormalities
- Laparoscopic treatment of undescended testicle
- Treatment and outcomes in patients with liver disease

P18 **DO PATIENTS WITH DOWN SYNDROME DEVELOP APPENDICITIS?**
Mary E. Nabers, BA[1], Michael W. L. Gauderer, MD[1], Dawn W. Blackhurst, PhD[1], R. Curtis Rogers, MD[1], Wendy R. Cornett, MD[2]
[1]Children`s Hospital, Greenville Hospital System, Greenville, SC, USA, [2]Greenville Hospital System, Greenville, SC, USA

P19 **AN EVIDENCE-BASED CLINICAL PROTOCOL FOR DIAGNOSIS OF ACUTE APPENDICITIS DECREASED THE USE OF COMPUTED TOMOGRAPHY IN CHILDREN: A PROSPECTIVE STUDY**
Obinna O. Adibe, MD, Erik N. Hansen, MD, MPH, Albert J. Chong, MD, MPH, Lena Perger, MD, Richard Keijzer, MD, PhD, Oliver J. Muensterer, MD, PhD, Keith E. Georgeson, MD, Carroll M. Harmon, MD, PhD
University of Alabama at Birmingham, Birmingham, AL, USA

P20 **EXPERIENCE PERFORMING 64 CONSECUTIVE STAPLED INTESTINAL ANASTOMOSES IN SMALL CHILDREN AND INFANTS**
Ian C.S. Mitchell, MD, Robert Barber, RN, Anne C. Fischer, MD, PhD, FACS, David T. Schindel, MD, FACS
University of Texas Southwestern Medical Center, Dallas, TX, USA

P22 **3-DIMENSIONAL COMPUTED TOMOGRAPHY (3-D CT) FOR EVALUATION AND MANAGEMENT OF CHILDREN WITH COMPLEX CHEST WALL ANOMALIES: USEFUL INFORMATION OR JUST PRETTY PICTURES?**
Elizabeth H. Calloway, BA[1], Ali N. Chhotani, BS[1], Yueh Z. Lee, MD, PhD[2], J. Duncan Phillips, MD[3]
[1]UNC Chapel Hill School of Medicine, Chapel Hill, NC, USA, [2]UNC Chapel Hill Department of Radiology, Chapel Hill, NC, USA, [3]UNC Chapel Hill Department of Surgery, Chapel Hill, NC, USA

P23 **PRE-CLOSURE FLUID RESUSCITATION INFLUENCES OUTCOME IN GASTROSCHISIS**
Leigh A. Jansen, MD[1], Yi Lin, MSc[2], Ying MacNab, PhD[2], Erik D. Skarsgard, MD[1], Pramod S. Puligandla, MD[3] and the Canadian Pediatric Surgery Network.
[1]BC Children's Hospital, Vancouver, BC, Canada, [2]University of BC School of Population and Public Health, Vancouver, BC, Canada, [3]The Montreal Children's Hospital of the McGill University Health Centre, Montreal, QC, Canada

P24 **A CRITICAL REVIEW OF PREMATURE INFANTS WITH INGUINAL HERNIAS: OPTIMAL TIMING OF REPAIR, INCARCERATION RISK, AND POSTOPERATIVE APNEA**
Steven L. Lee, MD, Joseph M. Gleason, MD, Roman M. Sydorak, MD
Kaiser Permanente, Los Angeles Medical Center, Los Angeles, CA, USA

P25 **TEMPORAL ASSOCIATION BETWEEN BLOOD TRANSFUSION AND NECROTIZING ENTEROCOLITIS IN PREMATURE INFANTS**
Diana L. Diesen, MD, Elizabeth T. Tracy, MD, Melissa E. Danko, MD, Michael Cotten, MD, David T. Tanaka, MD, Henry E. Rice, MD
Duke University, Durham, NC, USA

P26 **OUTCOME OF PATCH REPAIR IN CONGENITAL DIAPHRAGMATIC HERNIA IN FETO ERA**
Chandrasen K. Sinha, FRCS, MCH, Shailesh Patel, FRCS, N Ade-Ajayi, FRCS, Prof Mark Davenport, FRCS
King's College Hospital, London, United Kingdom

P27 **LAPAROSCOPIC TWO-STAGE FOWLER-STEPHENS ORCHIDOPEXY FOR ABDOMINAL TESTIS**
Dacia Di Renzo, MD, Michele Lombardo, MD, Anthony A. Caldamone, MD, Francois I. Luks, MD
Brown Medical School, Providence, RI, USA

P28 **TISSUE-ENGINEERED ESOPHAGUS IS A VERSATILE IN VIVO MOUSE MODEL WITH INTACT ARCHITECTURE**
Allison L. Speer, MD, Frederic G. Sala, PhD, Jamil A. Matthews, MD, Dianne C. Skelton, BSc, Tracy C. Grikscheit, MD
Childrens Hospital Los Angeles, Los Angeles, CA, USA

P29 **ARGON BEAM COAGULATION NEGATES THE NEED FOR DRAINAGE IN THE EXCISION OF CYSTIC HYGROMAS**
Helena M. Crowley, MDPhD[1], Sarah McPartland, MD[1], Brian F. Gilchrist[2], Carl-Christian Jackson, MD[1]
[1]Tufts Medical Center, Boston, MA, USA, [2]Elliot Hospital, Manchester, NH, USA

P30 **EXTRAHEPATIC PORTAL VEIN THROMBOSIS AFTER UMBILICAL CATHETERIZATION: IS IT A GOOD CHOICE FOR "REX SHUNT"?**
Nelson E M Gibelli, MD, Ana Cristina Aoun Tannuri, MD, Maria Lúcia Pinho-Apezzato, MD, João Gilberto Maksoud-Filho, MD, Manoel Carlos Prieto Velhote, MD, Uenis Tannuri, MD
Instituto da Criança - Hospital das Clínicas – Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

P31 **SURGICAL INTERVENTION IN THE SETTING OF PARENTERAL NUTRITION ASSOCIATED CHOLESTASIS MAY EXACERBATE LIVER INJURY**
Danielle A. Arsenault, RN NP, Alexis K. Potemkin, RN BSN, Elizabeth Robinson, BA, Vincent E. de Meijer, MD MSc, Mark Puder, MD PhD
Childrens Hospital Boston, Boston, MA, USA

P32 **EFFECTS OF THE ADMINISTRATION OF PENTOXIFYLLINE AND PREDNISOLONE ON THE EVOLUTION OF PORTAL FIBROGENESIS SECONDARY TO BILIARY OBSTRUCTION — AN EXPERIMENTAL STUDY IN GROWING ANIMALS**

Wagner de Castro Andrade, MD[1], Nelson E.M. Gibelli, MD[1], Luiz Fernando Ferraz da Silva, MD[2], Venâncio
Avancini Ferreira Alves, MD[2], Uenis Tannuri, MD[1]
[1]Instituto da Criança - Hospital das Clínicas – Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil, [2]Hospital das Clínicas - Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil

P33 **LONG-TERM OUTCOME IN CHILDREN AFTER RESECTION OF CONGENITAL CYSTIC ADENOMATOID MALFORMATION (CCAM)**

Salvador Guevara-Gallardo, MD, Eric B. Jelin, MD, Timothy Jancelewicz, MD, Barbara J. Bratton, MSN, PNP, Roberta L. Keller, MD, Tippi MacKenzie, MD, Shinjiro Hirose, MD, Doug Miniati, MD, Diana L. Farmer, MD, Hanmin Lee, MD
University of California, San Francisco, San Francisco, CA, USA

5:45 – 6:30 p.m.	International Guest Reception	*Michelangelo*
6:30 – 8:30 p.m.	Welcome Reception	*Harbor Piazza*

Monday, May 17

6:00 – 7:30 a.m.	Annual Fun Run	*Thisty Fish*
6:30 – 7:30 a.m.	Committee Meetings	*Bernini 1, Bernini 2, Michelangelo 1, Michelangelo 2, DaVinci 1, DaVinci 2, Verona, Vincenza*
6:30 a.m. – 1:00 p.m.	Registration Open	*Tuscan Foyer*
6:45 – 7:30 a.m.	Continental Breakfast	*Tuscan Foyer*
6:45 a.m. – 1:15 p.m.	Exhibits Open	*Tuscan Foyer*
7:30 – 9:00 a.m.	**Scientific Session I: Common Problems and Trauma**	

Moderators:
Dennis W. Vane, MD; Daniel J. Ostlie, MD

Educational Objectives:
At the end of the session, the learner will be able to:

- Describe outcomes in appendicitis
- Differentiate current and experimental therapies for Hirschprung's disease
- Cite the significance of first rib fractures in pediatric trauma
- Describe management issues in pediatric inflammatory bowel disease

1 **A MULTI-INSTITUTIONAL COMPARISON OF PEDIATRIC APPENDICITIS OUTCOMES BETWEEN TEACHING AND NONTEACHING HOSPITALS – 3 minutes**

Arezou Yaghoubian, MD[2], Steven L. Lee, MD[1], Christian de Virgilio, MD[2]
[1]Kaiser Permanente, Los Angeles Medical Center, Los Angeles, CA, USA, Harbor-UCLA Medical Center, Torrance, CA, USA

2 **PROSPECTIVE, RANDOMIZED TRIAL ASSESSING QUALITY OF LIFE IN PATIENTS WITH RUPTURED APPENDICITIS – 3 minutes**

Regan F. Williams, MD[1], Allison Hester, MSN[2], Barbara Culbreath, BSN[3], Elizabeth Paton, NP[3], James W. Eubanks, III, MD[1], S. Douglas Hixson, MD[1], Eunice Y. Huang, MD[1], Max R. Langham, MD[1], Martin L. Blakely, MD, MS[1]

[1]University of Tennessee Health Science Center, Memphis, TN, USA, [2]University of Arkansas for Medical Sciences, Little Rock, AR, USA, [3]LeBonheur Children's Medical Center, Memphis, TN, USA

3 **GLOWING IN THE DARK: TIME OF DAY AS A DETERMINANT OF RADIOGRAPHIC IMAGING IN THE EVALUATION OFABDOMINAL PAIN IN CHILDREN – 3 minutes**

Elizabeth J. Renaud, MD, Andrew Burr, MD, Erin Cooley, MD, Joseph Makris, MD, Mariann Manno, MD, Anthony DeRoss, MD, Michael Hirsh, MD

UMass Memorial Medical Center, Worcester, MA, USA

4 **PREOPERATIVE MECHANICAL BOWEL PREPARATIONS FOR PEDIATRIC GASTROINTESTINAL SURGERY: IS IT NECESSARY? – 3 minutes**

Kim McIltrot, CRNP, David C. Chang, PhD MBA MPH, Sharon Dudley-Brown, MD, Daniel S. Rhee, MD, Jose H. Salazar-Osuna, MD, Fizan Abdullah, MD PhD, Paul M. Colombani, MD

Johns Hopkins, Baltimore, MD, USA

5 **RESTORATIVE PROCTOCOLECTOMY WITH AND WITHOUT PROTECTIVE ILEOSTOMY IN A PEDIATRIC POPULATION – 3 minutes**

Daniel P. Doody, MD, Daniel P. Ryan, MD

Massachusetts General Hospital, Boston, MA, USA

6 **OMEGA-3 FATTY ACID AND GROWTH FACTOR MANIPULATION OF INTESTINAL ANGIOGENESIS: A NOVEL APPROACH TO THE MANAGEMENT OF INFLAMMATORY BOWEL DISEASE – 3 minutes**

Keith A. Thatch, MD[1], Michael S. Katz, MD[1], Marian M. Haber, MD[2], Marshall Z. Schwartz, MD[1]

[1]St. Christopher's Hospital for Children, Philadelphia, PA, USA, [2]Drexel University College of Medicine, Philadelphia, PA, USA

7 **RE-DILATION OF BOWEL AFTER INTESTINAL LENGTHENING PROCEDURES – 3 minutes**

Eiichi A. Miyasaka, MD, Pamela I. Brown, MD, Daniel H. Teitelbaum, MD

University of Michigan, Ann Arbor, MI, USA

8 **TRANSPLANTATION OF NEURAL CREST PROGENITOR CELLS INTO MURINE AGANGLIONIC RECTUM – 3 minutes**

Shant Shekherdimian, MD, MPH, Nan Ye Lei, James C.Y. Dunn, MD, PhD

UCLA Medical Center, Los Angeles, CA, USA

9 **INTRASPHINCTERIC BOTULINUM TOXIN DECREASES RATE OF HOSPITALIZATION FOR POST-OPERATIVE OBSTRUCTIVE SYMPTOMS IN CHILDREN WITH HIRSCHSPRUNG'S DISEASE – 3 minutes**

Bashar Patrus, MD, Ahmed Nasr, MD, Jacob C. Langer, MD, J. Ted Gerstle, MD

The Hospital for Sick Children, Toronto, ON, Canada

10 **HEPATICODUODENOSTOMY VERSUS HEPATICOJEJUNOSTOMY FOR RECONSTRUCTION AFTER RESECTION OF CHOLEDOCHAL CYST – 3 minutes**

Matthew T. Santore, MD, Brittany J. Behar, BA, Thane A. Blinman, MD, Edward J. Doolin, MD, Holly L. Hedrick, Peter Mattei, MD, Michael L. Nance, MD, N. Scott Adzick, MD, Alan W. Flake, MD

The Children's Hospital of Philadelphia, Philadelphia, PA, USA

11 **LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING IN ADOLESCENTS: SHORT-TERM RESULTS – 3 minutes**

Jeffrey L. Zitsman, MD[1], Ilene Fennoy, MD, MPH[1], Mary Ann Witt, DNSc[1], Janet Schauben, RD[1], Michael Devlin, MD[2], Marc Bessler, MD[1]
[1]Columbia University Medical Center, New York, NY, USA,[2]NY Psychiatric Institute, New York, NY, USA

12 **THE PREDICTIVE VALUE OF PREOPERATIVE PET-CT SCANS IN CHILDREN WITH CONGENITAL HYPERINSULINISM OF INFANCY – 3 minutes**

Augusto Zani, MD[1], Shireen Nah, MD[1], Ori Ron, MD[1], Virpi Smith, MD[1], Michael Ashworth[1], MD, Oliver Blankenstein, MD[2], Wolfgang Mohnike, MD[2], Simon Eaton, PhD[1], Paolo De Coppi, PhD[1], Khalid Hussain, MD[1], Agostino Pierro, MD[1]
[1]Great Ormond Street Hospital for Children – University College London Institute of Child Health, London, United Kingdom; [2]Department of Endocrinology, Charité-University Medicine, Berlin, Germany.

13 **SUCCESSFUL OUTCOME WITH ORGAN DONATION AND TRANSPLANTION FOLLOWING CARDIAC ARREST IN PEDIATRIC TRAUMA – 3 minutes**

Rachel Adams Greenup[1], Casey M. Calkins, MD[2], Brian D. Shames, MD[1], Laura C. Cassidy, PhD[2], Thomas T. Sato, MD[2]
[1]Medical College of Wisconsin, Milwaukee, WI, USA, [2]Children's Hospital of Wisconsin, Milwaukee, WI, USA

14 **SIGNIFICANCE OF FIRST RIB FRACTURES IN BLUNT PEDIATRIC THORACIC TRAUMA – 3 minutes**

Nicholas A. Hamilton, MD, Martin S. Keller, MD
Washington University, Saint Louis, MO, USA

15 **PARTIAL SPLENECTOMY FOR HEREDITARY SPHEROCYTOSIS: A MULTI-INSTITUTIONAL REVIEW – 3 minutes**

Keely L. Buesing, MD[1], Elisabeth T. Tracy, MD[2], Colleen Kiernan[3], Aimee Pastor, RN[4], Laura Cassidy, PhD, MS[1], J. Paul Scott, MD[1], Russell E. Ware, MD, PhD[5], Andrew M. Davidoff, MD[5], Frederick J. Rescorla, MD[3], Jacob C.
Langer, MD[4], Henry E. Rice, MD[2], Keith T. Oldham, MD[1]
[1]Children's Hospital of Wisconsin/Medical College of Wisconsin, Milwaukee, WI, USA,[2]Duke University Medical Center, Durham, NC, USA,[3]Riley Hospital for Children/Indiana University School of Medicine, Indianapolis, IN, USA, [4]The Hospital for Sick Children/University of Toronto, Toronto, ON, Canada, St. Jude Children's Research Hospital, Memphis, TN, USA

9:00 – 10:00 a.m.	**Robert E. Gross Lecture:** **John D. Birkmeyer, MD** **Measuring and Improving the Quality of Pediatric Surgery**	*Tuscan Ballroom*
10:00 – 10:30 a.m.	Refreshment Break	*Tuscan Foyer*
10:30 – 11:45 a.m.	**Scientific Session II: Neonatal**	*Tuscan Ballroom*

Moderators:
Karl G. Sylvester, MD; Michael A. Helmrath, MD

Educational Objectives:
This session will enable participants to:

- Review the evidence for alterations in cerebral blood flow during CDH repair
- Cite current treatment options for treatment of diaphragmatic hernia
- Describe the biochemical and clinical characteristics of necrotizing enterocolitis and spontaneous intestinal perforation
- Describe morbidity from repair of tracheal esophageal fistula

16 **PROTOCOLIZED MANAGEMENT OF INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA: EFFECT ON SURVIVAL – 3 minutes**

Mara B. Antonoff, MD[1], Virginia A. Hustead, MD[2], Shawn S. Groth, MD[1], David J. Schmeling, MD[2]
[1]University of Minnesota, Minneapolis, MN, USA, [2]Children's Hospitals and Clinics of Minnesota, Minneapolis, MN, USA

17 **MID TERM FOLLOW-UP IN HIGH RISK CDH SURVIVORS: PATCHING THE DIAPHRAGM AFFECTS THE OUTCOME – 3 minutes**

Laura Valfrè, MD, Annabella Braguglia, MD, Francesco Morini, Andrea Conforti, Natalia Chukhlantseva, MD, Andrea Dotta, MD, Carlo Corchia, Pietro Bagolan, MD
Bambino Gesù Children's Hospital Rome, Rome, Italy

18 **PREDICTING THE DEVELOPMENT OF CHRONIC PULMONARY HYPERTENSION IN PATIENTS WITH CDH ON ECMO: WHAT CAN WE DO AND WHEN – 3 minutes**

Jashodeep Datta, BS[1], Sharon E. Phillips[1], Edmund Y. Yang, MD, PhD[2]
[1]Vanderbilt University School of Medicine, Nashville, TN, USA, [2]Cardinal Glennon Children's Medical Center, St. Louis, MO, USA

19 **NATIONAL TRENDS IN NITRIC OXIDE USE IN NEONATES WITH CONGENITAL DIAPHRAGMATIC HERNIA – 3 minutes**

Kimberly A. Ruscher, MD MPH[1], Kevin A. Modeste, MBBS[1], Stephen Neff[2], Brendan T. Campbell, MD MPH[2]
[1]University of Connecticut, Hartford, CT, USA,[2]Connecticut Children's Medical Center, Hartford, CT, USA

20 **FETAL CEREBRAL BLOOD FLOW IN CONGENITAL DIAPHRAGMATIC HERNIA – 3 minutes**

Tim Van Mieghem, MD, Leonardo Gucciardo, MD, Elisa Done', Luc De Catte, MD PhD, Dominique Van Schoubroeck, MD, Roland Devlieger, MD PhD, Jan A. Deprest, MD PhD
University Hospitals Leuven, Leuven, Belgium.

21 **DECREASED CEREBRAL OXYGEN SATURATION DURING THORACOSCOPIC REPAIR OF CONGENITAL DIAPHRAGMATIC HERNIA AND OESOPHAGEAL ATRESIA IN INFANTS – 6 minutes**

Luca Giacomello, MD[1],Mark Bishay, MB ChB, MRCS[1], Merrill McHoney, MD, PhD[1], Paolo De Coppi, MD, PhD[1], Joe Brierley, MRCP, MRCPCH[1], Louise Harding, FRCA[2], Edward M. Kiely, FRCSI, FRCS[2], Joseph I. Curry, MBBS, FRCS[2], David P. Drake, FRCS[2], Kate Cross, FRCS[2], Simon Eaton, PhD[3], Agostino Pierro, MD, FRCS, FAAP[1]
[1]Great Ormond Street Hospital for Children and UCL Institute of Child Health, London, United Kingdom, [2]Great Ormond Street Hospital for Children, London, United Kingdom, [3]UCL Institute of Child Health, London, United Kingdom

22 **SYMPTOMATIC VOCAL CORD PARALYSIS IN INFANTS OPERATED ON FOR ESOPHAGEAL ATRESIA AND/OR TRACHEO-ESOPHAGEAL FISTULA – 3 minutes**

Francesco Morini, MD, Barbara D. Iacobelli, MD, Alessandro Crocoli, MD, Sergio Bottero, MD, Marilena Trozzi, MD, Andrea Conforti, MD, Pietro Bagolan, MD
Bambino Gesù Children's Research Hospital, Rome, Italy

23 **NECROTIZING ENTEROCOLITIS IS ASSOCIATED WITH NEONATAL INTESTINAL INJURY – 3 minutes**

Kristyn Mannoia, BA, Danilo Boskovic, PhD, Laurel Slater, BSN, RNC, Megan Plank, BS, Danilyn Angelis, PhD, Gerald Gollin, MD
Loma Linda University School of Medicine, Loma Linda, CA, USA

24 **URINE BIOMARKERS OF PROGRESSIVE NECROTIZING ENTEROCOLITIS – 3 minutes**
Xuefeng B. Ling, PhD[1], Gigi Liu, BS[1], James Schilling, PhD[1], Fizan Abdullah, MD, PhD[3], Mary Brandt, MD[4], Mary Cay Harris, MD[5], Harvey Cohen, MD, PhD[1], R. Larry Moss, MD[2], Karl Sylvester, MD[1]
[1]Stanford University, Stanford, CA, USA, [2]Yale University, New Haven, CT, USA, [3]Johns Hopkins University, Baltimore, MD, USA, [4]Texas Children's Hospital, Baylor University, Houston, TX, [5]The Children's Hospital of Philadelphia, University of Pennsylvania, Philadelphia, PA, USA

25 **SURGERY FOR NECROTIZING ENTEROCOLITIS IN THE UNITED STATES: CURRENT PRACTICE AND MORTALITY – 3 minutes**
Melissa A. Hull, MD[1], Brian A. Jones, MD[1], Joseph H. Carpenter, MS[2], Michael Kenney, MS[2], Jennifer C. Michelle, MPH[2], David Zurakowski, PhD[1], Jeffrey D. Horbar, MD[2], Tom Jaksic, MD, PhD[1]
[1]Children's Hospital Boston, Boston, MA, USA,[2]Vermont Oxford Network, Burlington, VT, USA

26 **MORTALITY OF NEONATAL SPONTANEOUS INTESTINAL PERFORATION: A MULTIINSTITUTIONAL ASSESSMENT – 3 minutes**
Brian A. Jones, MD[1], Melissa A. Hull, MD[1], Joseph H. Carpenter, MS[2], Michael Kenny, MS[2], Jennifer C. Michelle, MPH[2], David Zurakowski, PhD[1], Jeffrey D. Horbar, MD[2], Tom Jaksic, MD, PhD[1]
[1]Children's Hospital Boston, Boston, MA, USA, [2]Vermont Oxford Network, Burlington, VT, USA

27 **DOES MULTIDISCIPLINARY PRENATAL COUNSELING OR DELIVERY METHOD AFFECT OUTCOME IN GASTROSCHISIS? – 3 minutes**
Christopher W. Snyder, MD, MSPH, Joseph R. Biggio, MD, Donna T. Bartle, RN, BSN, Phillip R. Brinson, BS, Leandra A. Barnes, Keith E. Georgeson, MD, Oliver J. Muensterer, MD, PhD
University of Alabama at Birmingham, Birmingham, AL, USA

11:45 a.m. – Noon	**Introduction of New Members**	*Tuscan Ballroom*
Noon – 1:00 p.m.	**Presidential Address: Keith E. Georgeson, MD Pioneers, Cowboys and Desperados: A Brief History of Pediatric Surgical Innovation**	*Tuscan Ballroom*
1:00 – 2:30 p.m.	Benjy Brooks Society Meeting	*Verona*
1:30 p.m.	Depart for Optional Events: Golf Tournament, Cooking Class (Off Site), Leisure Time	
5:00 – 6:30 p.m.	***Journal of Pediatric Surgery* Reception**	*Verona*
5:30 – 6:30 p.m.	**Residents' Reception**	*Casa Dolce*
6:30 – 8:00 p.m.	**New Member Reception**	*Casa Dolce*

Tuesday, May 18

6:30 – 8:00 a.m.	Member Business Meeting and Breakfast	*Tuscan Ballroom*
6:30 a.m. – 3:30 p.m.	Registration Open	*Tuscan Foyer*
7:00 – 8:00 a.m.	Continental Breakfast for Non-members	*Tuscan Foyer*
7:00 a.m. – 12:30 p.m.	Exhibits Open	*Tuscan Foyer*
8:00 – 9:30 a.m.	**Scientific Session III: Practice Issues, Education and Outcomes**	*Tuscan Ballroom*

Moderators:
Peter F. Nichol, MD; R. Cartland Burns, MD

Educational Objectives:
Participants in this session will acquire knowledge of:

- The treatment and outcomes of intestinal failure
- The impact of manuscript submission guidelines on manuscript quality
- Prospective trials on management of pediatric surgical problems
- Efficacy of new approaches to common problems

28 **VERTICAL EXPANDABLE PROSTHETIC TITANIUM RIB (VEPTR) DEVICE INSERTION: DOES IT IMPROVE PULMONARY FUNCTION? – 3 minutes**
Samir K. Gadepalli, MD, Frances A. Farley, MD, Wan C. Tsai, MD, Peter J. Strouse, MD, Robert A. Drongowski, MS, Ronald B. Hirschl, MD
University of Michigan, Ann Arbor, MI, USA

29 **PEDIATRIC ACS NSQIP: FEASIBILITY OF A NOVEL, PROSPECTIVE ASSESSMENT OF SURGICAL OUTCOMES — A PHASE I REPORT – 6 minutes**
Mehul V. Raval, MD[1], Peter W. Dillon, MD[2], ACS NSQIP Pediatric Steering Committee[3].
[1]Division of Optimal Patient Care, American College of Surgeons; Division of Pediatric Surgery, Department of Surgery, Feinberg School of Medicine, Northwestern University, Children's Memorial Hospital, Chicago, IL, USA, [2]Division of Pediatric Surgery, Department of Surgery, Penn State College of Medicine, Hershey, PA, USA, [3]American College of Surgeons and American Pediatric Surgical Association, Chicago, IL, USA

30 **ELEMENTS OF SUCCESSFUL INTESTINAL REHABILITATION – 3 minutes**
David Sigalet, MD, Viona Lam, RN, BSN, Dana Boctor, MD, MSc, Marli Robertson, MD
University of Calgary, Calgary, AB, Canada

31 **OUTCOMES FROM GASTRIC ELECTRICAL STIMULATION IN CHILDREN – 3 minutes**
Saleem Islam, MD[1], Shamaila Waseem, MD[1], Christopher Jolley, MD[1], Thomas Abell, MD[2]
[1]University of Florida, Gainesville, FL, USA, [2]University of Mississippi, Jackson, MS, USA

32 **RESULTS OF A LONGITUDINAL STUDY OF RIGOROUS MANUSCRIPT SUBMISSION GUIDELINES DESIGNED TO IMPROVE THE QUALITY OF CLINICAL RESEARCH REPORTING IN A PEER-REVIEWED SURGICAL JOURNAL – 3 minutes**
Kathryne E. Wynne, BA[1], B. Joyce Simpson, RN, MPh[1], Loren Berman, MD[1], Shawn J. Rangel, MD[2], Jay L. Grosfeld, MD[3], R. Lawrence Moss, MD[1]
[1]Yale University, New Haven, CT, USA, [2]Boston Children's Hospital, Boston, MA, USA, Indiana University School of Medicine, Indianapolis, IN, USA

33 **MINIMAL VERSUS EXTENSIVE ESOPHAGEAL MOBILIZATION DURING LAPAROSCOPIC FUNDOPLICATION: A PROSPECTIVE RANDOMIZED TRIAL – 6 minutes**
Shawn D. St. Peter, MD[1], Douglas C. Barnhart, MD[2], Daniel J. Ostlie, MD[1], KuoJen Tsao, MD[3], Charles M. Leys, MD[4], Susan W. Sharp, PhD[1], Donna Bartle, RN[4], Tracie Morgan, RN[4], Carroll M. Harmon, MD[4], Keith E. Georgeson, MD[4], George W. Holcomb III, MD, MBA[1]
[1]Children's Mercy Hospital, Kansas City, MO, USA, [2]University of Utah/Primary Children's Medical Center, Salt Lake City, UT, USA, [3]University of Texas Medical Center, Houston, TX, USA, [4]University of Alabama Birmingham, Birmingham, AL, USA, [4]Riley Hospital for Children, Indianapolis, IN, USA

34 **IMPACT OF OMEGA 3 FATTY ACIDS ON LIVER FUNCTION TESTS AND LIVER HISTOLOGY IN CHILDREN WITH PARENTERAL NUTRITION DEPENDENT INTESTINAL FAILURE – 6 minutes**
Carroll M. Harmon, MD, PhD[1], Donna T. Bartle[1], Traci Morgan, RN, BSN[1], Lindsay Q. Toole, RD, LD, CNSD[2], Cheralynn D. Hopkins, LGSW[2], David R. Kelly, MD[1], Reed A. Dimmitt, MD[1]
[1]University of Alabama at Birmingham, Birmingham, AL, USA, [2]The Children's Hospital of Alabama, Birmingham, AL, USA

35 **MORE OR LESS? — LESSONS LEARNED FROM LAPAROENDOSCOPIC SINGLE SITE SURGERY IN CHILDREN – 6 minutes**
Erik N. Hansen, MD, Carroll M. Harmon, MD, PhD, Oliver Muensterer, MD, PhD, Keith Georgeson, MD
Children's Hospital of Alabama, Birmingham, AL, USA

36 **ON BOARD THE USNS COMFORT WITH CONTINUING PROMISE 2009 — A MODEL FOR HUMANITARIAN ASSISTANCE – 6 minutes**
Shawn D. Safford, MD[1], Jacob Glaser, MD[1], Timothy Donahue, MD[1], John Bastien, MD[2]
[1]National Naval Medical Center, Bethesda, MD, USA, [2]Portsmouth Naval Medical Center, Portsmouth, VA, USA

37 **THE CONTRIBUTION OF GASTROESOPHAGEAL REFLUX DISEASE TO WEIGHT LOSS IN NICU INFANTS – 3 minutes**
Joanne E. Baerg, MD, Douglas Deming, MD
Loma Linda University Children's Hospital, Loma Linda, CA, USA

38 **PROSPECTIVE VALIDATION OF AN ABBREVIATED BEDREST PROTOCOL IN THE MANAGEMENT OF BLUNT SPLEEN AND LIVER INJURY IN CHILDREN– 6 minutes**
Shawn D. St. Peter, MD[1], Susan W. Sharp, PhD[1], Saleem Islam, MD[2], Charles L. Snyder, MD[1], Ronald J. Sharp, MD[1], Walter S. Andrews, MD[1], J Patrick Murphy, MD[1], George W. Holcomb, III, MD[1], Daniel J. Ostlie, MD[1]
[1]Children's Mercy Hospital, Kansas City, MO, USA, [2]University of Florida, Gainesville, FL, USA

39 **HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) PROMOTES ENTERIC NEURAL CREST CELL MIGRATION – 3 minutes**
Yu Zhou, MD, PhD, Iyore A. Otabor, MD, Amanda Wetzl, Gail E. Besner, MD
Nationwide Children's Hospital, Columbus, OH, USA

9:30 – 10:30 a.m.	**Jay & Margie Grosfeld Lecture: Christopher K. Breuer, MD The Development and Translation of the Tissue Engineered Vascular Grafts**	*Tuscan Ballroom*
10:30 – 11:00 a.m.	Refreshment Break	*Tuscan Foyer*
11:00 a.m. – 12:15 p.m.	**Scientific Session IV: Oncology and Critical Care**	*Tuscan Ballroom*

Moderators:
Elizabeth A. Beierle, MD; Daniel von Allmen, MD

Educational Objectives:
At the conclusion of this session the learner will be able to:
- Describe new understandings of the characteristics of primary and metastatic sarcoma
- Review the efficacy of liver resection in hepatoblastoma
- Describe surgical options for various pediatric tumors

40 **THE MDM2 INHIBITOR NUTLIN-3A POTENTLY SUPPRESSES BOTH TUMOR ANGIOGENESIS AND TUMOR GROWTH IN NEUROBLASTOMA – 3 minutes**
Eugene S. Kim, MD, Danielle Patterson, MD, Dongbing Gao, Nikki Sikorski, Zaowen Chen, Jason S. Shohet, MD, PhD
Baylor College of Medicine, Houston, TX, USA

41 **RESECTABILITY AND OPERATIVE MORBIDITY AFTER NEOADJUVANT CHEMOTHERAPY IN NEUROBLASTOMA PATIENTS WITH ENCASEMENT OF MAJOR VISCERAL ARTERIES – 3 minutes**
Barrie S. Rich, MD, Maureen P. McEvoy, MD, Natasha E. Kelly, MD, Edwin Oh, BS, Sara J. Abramson, FACR, Anita P. Price, FACR, Nai-Kong V. Cheung, MD, Brian H. Kushner, MD, Michael P. La Quaglia.
Memorial Sloan-Kettering Cancer Center, New York, NY, USA

42 **LOCAL CONTROL, SURVIVAL, AND OPERATIVE MORBIDITY AND MORTALITY AFTER RE-RESECTION AND INTRA-OPERATIVE RADIATION THERAPY FOR RECURRENT OR PERSISTENT PRIMARY HIGH-RISK NEUROBLASTOMA – 6 minutes**
Barrie S. Rich, MD, Maureen P. McEvoy, MD, Michael P. La Quaglia, Suzanne L. Wolden, MD
Memorial Sloan-Kettering Cancer Center, New York, NY, USA

43 **INHIBITION OF CYCLO-OXYGENASE 2 REDUCES EWING'S SARCOMA LUNG METASTASIS DURING VEGF BLOCKADE BY ALTERING EXPRESSION OF INFLAMMATORY PATHWAY GENE SETS – 3 minutes**
Jeffrey W. Gander, MD, Jason C. Fisher, MD, Sonia L. Hernandez, MA, Jianzhong Huang, MD, Darrell J. Yamashiro, MD, PhD, Jessica J. Kandel, MD
Children's Hospital of New York-Presbyterian, New York, NY, USA

44 **MESENCHYMAL STEM CELLS (MSCs) DEPENDENT REGRESSION OF PULMONARY METASTASIS IS MEDIATED BY INHIBITION OF PLATELET DERIVED GROWTH FACTOR RECEPTOR-BETA (PDGFR-B) – 3 minutes**
Peter Walker, MD[2], Yong Xin Wang, PhD[1], Charles S. Cox, MD[2], Andrea A. Hayes-Jordan[1]
[1]MD Anderson Cancer Center, Houston, TX, USA, [2]University of Texas Houston Health Sciences Center, Houston, TX, USA

45 **SELECTIVE INHIBITION OF CYCLOOXYGENASE-2 (COX-2) SUPPRESSES METASTATIC DISEASE WITHOUT AFFECTING PRIMARY TUMOR GROWTH IN A MURINE MODEL OF EWING'S SARCOMA – 3 minutes**
Amir S. Gendy, MD[1], Aaron Lipskar, MD[1], Edward Chen, MD[1], Richard D. Glick, MD[1], Bettie Steinberg, PhD[2], Morris Edelman, MD[1], Samuel Z. Soffer, MD[1]
[1]Schneider Childrens Hospital, New Hyde Park, NY, USA, [2]Feinstein Institute for Medical Research, Manhasset, NY, USA

46 **TREATMENT AND OUTCOME OF PEDIATRIC PATIENTS SUFFERING FROM PERINEAL RHABDOMYOSARCOMA- RESULTS FROM THE COOPERATIVE SOFT TISSUE SARCOMA STUDIES CWS-86, -91, -96 AND -2002P – 6 minutes**
Guido Seitz, MD[1], Tobias Dantonello, MD[2], Thomas Klingebiel, MD[3], Ewa Koscielniak, MD[2], Jörg Fuchs, MD[1]
[1]University Children`s Hospital, Tuebingen, Germany, [2]CWS Study Group, Olgahospital, Stuttgart, Germany, [3]University Children`s Hospital, Frankfurt / Main, Germany.

47 **THYROID CARCINOMA IN CHILDREN IS BEST DIAGNOSED BY SURGICAL EXCISION – 3 minutes**
Nini Kozeimeh, MD, Cynthia A. Gingalewski.
Children's National Medical Center, Washington, DC, USA

48 **IMPROVED SURVIVAL WITH LYMPH NODE SAMPLING IN WILMS TUMOR – 3 minutes**
Michael C. Cheung, MD, Relin Yang, MD, Ying Zhuge, MD, Leonidas G. Koniaris, MD, Holly L. Neville, MD, Juan E. Sola, MD
University of Miami, Miami, FL, USA

49 **OUTCOME OF RESECTION FOR PRETEXT III AND IV HEPATOBLASTOMA – 3 minutes**
Timothy Lautz, MD, Tamar Ben-Ami, MD, Niramol Tantemsapya, MD, Riccardo Superina, MD
Children's Memorial Hospital, Chicago, IL, USA

12:15 – 12:30 p.m. Box Lunch Pick-Up *Tuscan Foyer*

12:30 – 1:30 p.m. **New Technology Session: Abstracts on New and Innovative Techniques and Procedures** *Tuscan Ballroom*

Moderators:
Milissa A. McKee, MD; Tamir A. Keshan, MD

Educational Objectives:
- Participants will be exposed to new techniques or device utilization not yet 'mainstream' in pediatric surgery practice
- Participants may be able to identify potential application of these approaches and question presenters about potential applications in practice

50 **PERCUTANEOUS FETAL ENDOSCOPIC TRACHEAL OCCLUSION FOR ISOLATED CONGENITAL DIAPHRAGMATIC HERNIA WITH UNFAVOURABLE PROGNOSIS – 6 minutes**
Jan A. Deprest, MD PhD[1], Tim Van Mieghem, MD[1], Leonardo Gucciardo, MD[1], Philip De Koninck, MD[1], Luc De Catte, MD PhD[1], Roland Devlieger, MD PhD[1], Karel Allegaert, MD PhD[1], Jacques Jani, MD PhD[2], Eduard Gratacos, MD PhD[3], Kypros Nicolaides, MD PhD[2]
[1]University Hospitals Leuven, Leuven, Belgium, [2]King's College Hospital, London, United Kingdom, [3]Hospital Clinic, Barcelona, Spain.

51 **PRE-CLINICAL REGULATORY VALIDATION OF AN ENGINEERED DIAPHRAGMATIC TENDON MADE WITH AMNIOTIC MESENCHYMAL STEM CELLS – [6] minutes**
Christopher G. Turner[1], Justin D. Klein[1], Shaun A. Steigman[1], Myriam Armant[2], Grace A. Nicksa[1], David Zurakowski[1], Jerome Ritz[2], Dario O. Fauza[1]
[1]Children's Hospital Boston, Boston, MA, USA, [2]CBR Institute for Biomedical Research, Boston, MA, USA

52 **CARDIAC OUTPUT MEASUREMENTS IN NEONATES, ONE-HALF KILOGRAM TO FOUR KILOGRAMS – 6 minutes**
Andrew P. Bozeman, MD, Misael Rodriguez, MD, Joseph M. Van De Water, MD, Barbara Weaver, RN, Bao Ngoc T. Ho, BS, Robert L. Vogel, PhD, Don K. Nakayama, MD
Mercer University School of Medicine/ Medical Center of Central Georgia, Macon, GA, USA

53 **DETERMINATION OF THE CELLULAR ORIGIN OF VASCULAR NEOTISSUE IN TISSUE ENGINEERED VASCULAR GRAFTS (TEVG) – 6 minutes**
Gustavo A. Villalona, MD[1], Rajendra Sawh-Martinez, BS[2], Tamar Mirensky, MD[3], Narutoshi Hibino, MD, PhD[2], Tai Yi, BS[2], Adam Shoffner, BS[2], Edward McGillicuddy, MD[3], Toshiharu Shinoka, MD, PhD[1], Christopher Breuer, MD[1]
[1]Yale New Haven Children's Hospital, New Haven, CT, USA, [2]Yale School of Medicine, New Haven, CT, USA, [3]Yale New Haven Hospital, New Haven, CT, USA

54 **TOWARDS EFFECTIVE PEDIATRIC MINIMALLYINVASIVE SURGICAL SIMULATION – 6 minutes**
Joshua M. Hamilton, MD[1], Kanav Kahol, PhD[2], Aaron Ashby, MS[3], Leigh C. McGill, MD[4], David M. Notrica, MD[4], John J. Ferrara, MD[1]
[1]Phoenix Integrated Surgical Residency, Phoenix, AZ, USA, [2]Banner Good Samaritan Simulation Education and Training Center, Phoenix, AZ, USA, [3]Arizona State University Department of Biomedical Informatics, Tempe, AZ, USA, [4]Phoenix Children's Hospital, Phoenix, AZ, USA

55 **EXPERIENCE WITH DIAPHRAGM PACING IN REPLACING MECHANICAL VENTILATORS IN CHILDREN – 6 minutes**
Todd A. Ponsky, MD[1], Edward Barksdale, MD[1], Mary Jo Elmo, NP[1], Raymond Onders[2].
[1]Rainbow Babies and Children's Hospital, Cleveland, OH, USA, [2]University Hospitals of Cleveland, Cleveland, OH, USA

1:30 – 2:00 p.m. **APSA Foundation Scholars** *Tuscan Ballroom*

	Tippi C. MacKenzie, MD *University of California San Francisco, San Francisco, California, USA* **The Maternal Immune Response to in Utero Hematopoietic Stem Cell Transplantation**	
	Kelly A. Miller, MD *University of Pittsburgh, Pittsburgh, Pennsylvania, USA* **The Pathogenic Role of Enteric Glia in Hirschsprung's Enterocolitis**	
2:00 – 2:30 p.m.	APSA Updates	*Tuscan Ballroom*
2:30 – 3:30 p.m.	**International Guest Lecture: Jan Deprest, MD** *Leuven, Belgium* **Prenatal Management of the Fetus with an Isolated CDH**	*Tuscan Ballroom*
3:30 – 6:45 p.m.	Leisure Time	
6:45 – 7:30 p.m.	**President's Reception**	*Tuscan Foyer*
7:30 – 10:00 p.m.	**President's Banquet**	*Tuscan Ballroom*

Wednesday, May 19

6:30 – 7:30 a.m.	Committee Meetings	*Verona, Vincenza*
7:00 – 11:30 a.m.	Registration Open	*Tuscan Foyer*
7:00 – 8:00 a.m.	Continental Breakfast	*Tuscan Foyer*
8:00 – 9:00 a.m.	**Scientific Session V: Basic Science**	**Tuscan Ballroom**

Moderators:
Ai-Xuan L. Holterman, MD; Gail E. Besner, MD

Educational Objectives:
Participants in this session will acquire knowledge of:

- Advances in the understanding of developmental biology associated with surgical disorders
- Progress in research into tissue engineering
- Factors associated with the pre- and post-natal tissue development

56 **MATERNAL SILDENAFIL DECREASES PULMONARY ARTERIAL WALL THICKNESS IN A FETAL OVINE DIAPHRAGMATIC HERNIA MODEL – 3 minutes**
Eric Jelin, MD[1], Timothy Jancelewicz, MD[1], Kullada Pichakron, MD[2], Roberta L. Keller, MD[1], Jared Clay, MD[2], Jacob Stephenson, MD[2], Kevin Grayson, DVM[2], Farrah Eaton, BSc[3], Christina Luong[3], Bernard Thebaud, MD
PhD[3], Kerilyn Nobuhara, MD[1], Doug Miniati, MD[1]
[1]University of California, San Francisco, San Francisco, CA, USA, [2]David Grant USAF Medical Center, Travis AFB, CA, USA, [3]University of Alberta, Edmonton, AB, Canada

57 **SERIAL AMNIOINFUSIONS PREVENT FETAL PULMONARY HYPOPLASIA IN A LARGE ANIMAL MODEL OF OLIGOHYDRAMNIOS – 3 minutes**
Grace A. Nicksa, MD[1], David C. Yu, MD[1], Brian T. Kalish1, David Zurakowski, PhD[1], Justin D. Klein, MD[1], Christopher G.B. Turner, MD[1], Carol E. Barnewolt, MD[2], Dario O. Fauza, MD[1], Terry L. Buchmiller, MD[1]
[1]Children's Hospital Boston, Department of Surgery, Boston, MA, USA, [2]Children's Hospital Boston, Department of Radiology, Boston, MA, USA

58 **ENDOTHELIAL-DERIVED EPOXYEICOSATRIENOIC ACIDS PROMOTE COMPENSATORY LUNG GROWTH AFTER UNILATERAL PNEUMONECTOMY IN TRANSGENIC MICE – 3 minutes**

Hau D. Le, MD[1], Darryl C. Zeldin, MD[2], Vincent E. de Meijer, MD, MSc[1], Matthew L. Edin, PhD[2], Craig R. Lee, PhD[3], Jo-Ann Vergilio, MD[1], Bruce D. Hammock, PhD[4], Mark W. Kieran, MD, PhD[1], Mark Puder, MD, PhD[1], Dipak Panigrahy, MD[1]
[1]Children's Hospital Boston, Boston, MA, USA, [2]National Institute of Environmental Health Science, Research Triangle Park, NC, USA, [3]University of North Carolina, Chapel Hill, NC, USA, [4]University of California, Davis, CA, USA

59 **SPHINGOSINE-1-PHOSPHATE ENHANCES INTESTINAL BARRIER FUNCTION BY MODULATING PARACELLULAR JUNCTIONAL PROTEINS – 3 minutes**

Jose Greenspon, MD[1], Jennifer A. Timmons, MD[1], Alexis D. Smith, MD[1], Riuyun Li, BS[2], Jian-Ying Wang, MD, PhD[2], Eric D. Strauch, MD[1], Douglas J. Turner, MD[1]
[1]University of Maryland School of Medicine, Baltimore, MD, USA, [2]Baltimore Veterans Affairs Medical Center, Baltimore, MD, USA

60 **THE INFLUENCE OF INTRA-LUMINAL CONTENTS ON INTESTINAL ADAPTATION UNDER THE SAMESYSTEMIC HORMONAL CONDITIONS - 3 minutes**

Esmaeel Taqi, MD, Laurie Wallace, MD, Elaine de Heuvel, MD, David Sigalet, MD
Alberta Childrens Hospital, Calgary, AB, Canada

61 **EGFR INHIBITION IS ANTIANGIOGENIC IN EXPERIMENTAL NEUROBLASTOMA – 3 minutes**

Jason S. Frischer, MD[1], Kelly Crawford, BS[1], Caroline Berglund[1], Jason C. Fisher, MD[2], Timothy P. Cripe, MD, PhD[1]
[1]Cincinnati Children's Hospital Medical Center, Cincinnati, OH, USA, [2]Columbia University, New York, NY, USA

62 **CONSTRUCTING TRANSLATABLE TISSUEENGINEERED ARTERIAL GRAFTS: A NOVEL APPROACH – 3 minutes**

Tamar L. Mirensky, MD, Rajendra F. Sawh-Martinez, BS, Bernard S. Salameh, MD, Narutoshi Hibino, MD, PhD, Tai Yi, MD, PhD, Toshiharu Shinoka, MD, PhD, Christopher K. Breuer, MD
Yale University School of Medicine, New Haven, CT, USA

63 **CONDITIONAL MUTATION OF FIBROBLAST GROWTH FACTOR RECEPTORS 1 AND 2 RESULTS IN AN OMPHALOCELE IN MICE ASSOCIATED WITH DISRUPTIONS ON VENTRAL BODY WALL MUSCLE FORMATION – 3 minutes**

Peter F. Nichol, MD, PhD[1], Yukio Saijoh, PhD[2]
[1]University of Wisconsin School of Medicine and Public Health, Madison, WI, USA, [2]University of Utah, Salt Lake City, UT, USA

64 **FUNCTIONAL ANERGY OF NON-DELETED DONOR-REACTIVE HOST NK CELLS STABILIZES PRENATAL CHIMERISM – 3 minutes**

Amir M. Alhajjat, MD, Aimen F. Shaaban, MD
University of Iowa Carver College of Medicine, Iowa City, IA, USA

9:00 – 10:00 a.m.	**COG Session** **Current Therapy for Pediatric Solid Tumors: Renal Tumors and Liver Tumors**	*Tuscan Ballroom*

Moderator:
Andrew Davidoff, MD

Educational Objectives:

- Provide an update on the current therapy of specific pediatric solid tumors
- An emphasis on surgical guidelines, outcomes and new initiatives

Harold N. Lovvorn, MD
The New Hepatoblastoma Trial, AHEP0731: What's in it For Surgeons
Kenneth W. Gow, MD
Renal Tumor Studies: Surgical COGitation & COGnition

10:00 – 10:15 a.m.	Refreshment Break	*Tuscan Foyer*
10:15 – 11:30 a.m.	**Scientific Session VI: Pediatric Surgery Case Debates and Controversies**	*Tuscan Ballroom*

Moderators:
C. Mac Harmon, MD

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

11:30 a.m.	Annual Meeting Adjourns	
Noon – 6:00 p.m.	**APSA/IPEG Workshop: Advanced Neonatal Endoscopic Techniques**	Off site, transportation provided

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Abstracts
Underlining denotes the author scheduled to present at the meeting

V1

ENDOSCOPIC-GUIDED EXCISION OF A MIDLINE FOREHEAD DERMOID

Sherif Emil, MD,CM[1], Nabil Fanous, MD[2], Valerie Cote, MD, CM[2]
[1]Montreal Children's Hospital, Montreal, QC, Canada, [2]McGill University Health Centre, Montreal, QC, Canada

Purpose:

Endoscopic excision of benign forehead masses in children aims to avoid conspicuous scarring and disfigurement. We present a video of an endoscopic-guided excision of an intra-osseous dermoid cyst, located in the mid forehead.

Methods:

A 10 month old, dark-skinned, boy was seen for an asymptomatic mid-forehead mass that was noted soon after birth. The mass was firm and fixed. CT scan demonstrated a 1-cm intra-osseous mass in the mid forehead consistent with a dermoid cyst. The mass did not penetrate the inner table of the cranium. The child was operated on at age 18 months by a team comprised of a pediatric general surgeon and a head and neck surgeon with extensive experience in facial plastics. The mass was removed successfully through a single, vertical scar 2 cm above the hairline, using a combination of endoscopic and classic instruments. The procedure resulted in excellent cosmesis without any visible scar, and high parent satisfaction.

Conclusions:

Endoscopic excision of benign forehead masses results in excellent cosmesis, and can eliminate potential facial disfigurement in children.

Notes:

V2

RIGHT THORACOSCOPY FOR BILATERAL CONGENITAL CYSTIC LUNG LESIONS

Abigail E. Martin, MD, Francois I. Luks, MD
Hasbro Children's Hospital, Providence, RI, USA

Congenital cystic lung lesions are often identified during routine antenatal ultrasonography. Bilateral lesions are rare, but can be managed using a similar algorithm as unilateral lesions. We present the case of a baby with prenatally diagnosed bilateral cystic lesions located at the inferomedial aspect of the right and left lungs who was asymptomatic at birth. Despite preoperative imaging, it was unclear whether these were separate bilateral lesions. The patient underwent a right thoracoscopy. Intraoperatively we found a parenchymal lesion extending across the midline into the left hemithorax, and we were able to safely resect it from the right. Our experience shows that intraoperative examination of bilateral congenital lung lesions provides valuable information that may allow a unilateral approach to resection in selected cases.

Notes:

V3

TRANSANAL RESECTION, HOW TO AVOID FECAL INCONTINENCE

Alberto Pena, MD, Andrea Bischoff, MD, Marc A. Levitt, MD
Cincinnati Children Hospital, Cincinnati, OH, USA

Purpose:

Transanal resection of the rectosigmoid is a valuable technique applicable for Hirschsprung's disease, non-manageable idiopathic constipation, and idiopathic rectal prolapse. However, it represents a risk of producing damage to the continence mechanism. A series of important technical steps are crucial to avoid damage to the anal canal and sphincters. These are shown in a short video.

Methods:

In operations designed to remove the rectosigmoid and pull-through a new portion of colon it is mandatory to preserve the patient's continence mechanism. This is achieved by avoiding damage to the sphincter and preserving the anal canal for up to 2 centimeters above the pectinate line. Damage to the continent mechanism can result from inadvertently resecting part, or the entire anal canal, leaving the patient without sensation. In addition, the striated sphincter mechanism may be resected or overstretched.

Results:

Over a period of ten years, 13 patients from other institutions were referred suffering from fecal incontinence following a transanal rectosigmoid resection. An examination under anesthesia demonstrated that the anal canal was non-existent or seriously damaged. During the same period of time we have done 125 transanal resections of the rectosigmoid and have made every effort to preserve intact the continence mechanism. As a result, we developed a series of technical recommendations that include: a) use of a Lone-Star retractor, b) placing and then replacing the eight hooks deeper so that the pectinate line is protected and hidden, c) placing multiple fine sutures on the rectal wall to apply uniform traction, d) starting the resection two centimeters above the pectinate line, e) avoiding overstretching of the anus using a three point exposure technique (one narrow malleable, a forceps or suction tip, and rectum; forming a triangle).

Conclusions:

With these technical maneuvers a transanal rectal and rectosigmoid resection can be performed preserving the continence mechanism.

Notes:

V4

THE PRONE POSITION FOR COLORECTAL PROBLEMS: NOT JUST FOR ANORECTAL MALFORMATIONS

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Purpose:

Management of colorectal problems in the area of the pelvis is challenging - problems are often too low to comfortably reach from above or too high to reach from below. We have found that the prone position affords excellent exposure to solve this problem. With this case, we show that dissection of the rectum in the prone position can be applied to a variety of colorectal problems, in combination with a laparotomy or laparoscopy.

Methods:

We present an 18 year old male with Crohn's disease and a rectal stricture. His disease was limited to the rectum, with normal proximal colon. A diverting ileostomy was performed to control proctitis and recurrent perirectal abscesses. Despite diversion and maximal medical management, he developed a rectovesical fistula. The stricture was not passable endoscopically, and on exam, it was palpable at 3 cm from the anal verge. Given the good condition of his more proximal colon, our plan was to do a pullthrough of the left colon with resection of the rectum, and a coloanal anastamosis, rather than leaving him with a permanent stoma. A transanal dissection was performed in the prone position, and once the transanal dissection was complete, the perineum was packed with sterile gauze. The patient was then flipped supine and placed in lithotomy position. The intra-abdominal mobilization then proceeded laparoscopically, and the non-diseased colon was pulled down and anastomosed just above the dentate line.

Results:

This video highlights the advantages of performing the transanal rectal dissection in the prone position, rather than in lithotomy. These advantages include: better visualization, better ergonomics, better lighting and easier assisting.

Conclusions:

The prone position is useful in many rectal procedures, such as Hirschsprung's disease (both primary and redo cases), ulcerative colitis, severe idiopathic constipation, rectal prolapse and presacral and pelvic masses.

Notes:

V5

TRANSORAL FUNDOPLICATION IN A CHILD WITH RECURRENT GERD AFTER FUNDOPLICATION

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Purpose:

To present a video which demonstrates a fairly new and novel approach to treating recurrent gastroesophageal reflux disease (GERD) following a laparoscopic fundoplication. The authors demonstrate the use of an EsophyX device (Endogastric Solutions, Inc.), which performs a fundoplication intragastrically via the transoral route.

Methods:

The approach shown demonstrates the key steps in an EsophyX technique. The procedure beings with an initial placement of a pediatric endoscope without the device to measure the Z-line, rule out hiatal hernia and determine the extent of a residual fundoplication. The EsophyX device is then inserted, opened, and the endoscope placed outside the device, to allow for inspection of the suturing. A helical retractor is screwed into the esophago-gastric junction and then pulls gastric tissue into the tissue molds of the device. The molds are closed around the tissue, 2 stylets are passed through the gastric tissue, and polypropylene T-fasteners are released which holds the tissue together. Additional firings of the device are performed achieving a 2 cm long, 180 to 270 degree fundoplication. The device is then opened and removed.

Results:

The patient tolerated the procedure without difficulty.

Conclusions:

The video demonstrates a new, and potentially advantageous, approach to performing a fundoplication after recurrence.

Notes:

V6

LAPAROSCOPIC RESECTION OF CHOLEDOCHAL CYST: RECONSTRUCTION BY HEPATICODUODENOSTOMY

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Purpose:

Laparoscopic resection of choledochal cysts (CDC) is being increasingly applied. The most commonly described biliary reconstruction is a hepaticojejunostomy, with the roux-en-y limb often constructed extracorporally. Over the past 10 years, we have utilized hepaticoduodenostomy (HD) after open resection of CDC because it reduces operative time, entails only a single anastomosis, and is equally safe and effective. The purpose of this video is to demonstrate the use of HD as a simple reconstructive technique after laparoscopic resection of CDC, and to provide an overview of our first year of experience with this procedure.

Methods:

A retrospective chart review was performed over a 12 month period from October, 2008 to October, 2009. Peri-operative data were collected and analyzed.

Results:

Six laparoscopic choledochal cyst excisions were performed in our institution between October, 2008 and October 2009. Five of the cysts were fusiform lesions of the common bile duct (Type 1); one of the cysts was a multicystic lesion of the extra-hepatic bile ducts (Type 4). One procedure was converted to an open procedure. The average patient age was 3.1 yrs. The average operative time was 303 minutes. The median length of stay was 4 days and the median time to complete enteral intake was 3 days. There were no anastomotic leaks or peri-operative infections.

Conclusions:

The use of HD for biliary reconstruction after laparoscopic resection of CDC offers the advantages of a single anastomosis, total intracorporeal technique, and shorter operative times. Our initial experience suggests equal safety and efficacy with the open procedure.

Notes:

V7

SINGLE PORT, PERCUTANEOUS LYSIS OF AMNIOTIC BANDS

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Purpose:

Amniotic bands are a rare complication of pregnancy. The reported incidence ranges from 1:1,200 to 1:15,000 gestations. Left untreated, amniotic bands can result in amputations or severe anatomic abnormalities. We present a case of fetoscopic lysis of amniotic bands.

Methods:

The patient was a G1P0 27 year old woman at 20 weeks gestation. The pregnancy was complicated by amniotic bands constricting the left upper extremity. Diagnosis was made at 19 weeks gestation. Ultrasound examination demonstrated arterial inflow to the extremity but no venous outflow. Percutaneous, ultrasound assisted, fetoscopic lysis of amniotic bands was performed at 20 weeks. Intra-operative findings included amniotic bands, two areas of constriction, massive upper extremity edema, and a potentially viable distal extremity.

Results:

Successful fetoscopic lysis of amniotic bands was performed. Follow up ultrasound examination demonstrated improved arterial inflow and presence of venous outflow with improvement in distal edema.

Conclusion:

We conclude that percutaneous, fetoscopic lysis of amniotic bands is a potential strategy for limb salvage in pregnancies complicated by amniotic bands.

Notes:

P1

GROWTH IN MICROGRAVITY CULTURE MIMICS BIOLOGICAL BEHAVIOR OF NEUROBLASTOMA CELL LINES

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Purpose:

Factors which predict outcomes in neuroblastoma include: patient age, stage, histology and genetic profile. There is uncertainty determining the biological behavior and prognosis for intermediate-risk patients. A microgravity culture, which allows growth in three dimensions, may be useful for studying cell behavior.

Methods:

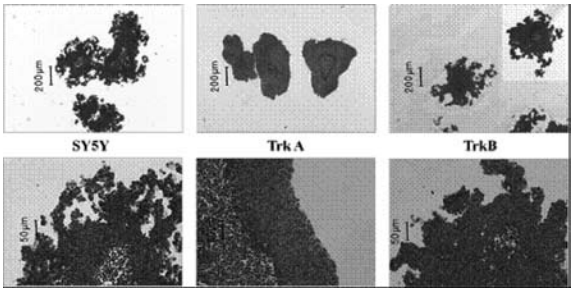
We studied: 1) *MYCN* amplification comparing three cell lines with varied numbers() of copies of the oncogene [CHP-212 (2); SK-N-AS (1); IMR-32 (20)]. 2) The impact of Trk receptor expression comparing a Trk-null cell line (SY5Y) to subclones transfected with either TrkA or TrkB. 3) The effect of tyrosine kinase inhibitor Lestaurtinib (CEP701) on the Trk-null parental SY5Y line. Single cell line suspensions were seeded into the STLV (Synthecon) at 5 x 10^5 cells/ml and cultured for eight days. The resulting organoids were evaluated for size and shape. The single suspended cells were counted over time in the lines comparing *MYCN*.

Results:

Organoid size correlated well with malignant potential. *MYCN* amplified cells were larger than the non-amplified counterparts (>1600 microns in diameter vs. ~250 microns). The single cell counts fell much more rapidly in the *MYCN* amplified lines (10% remained at 2 hr) compared to the nonamplified lines (56% at 2 hr), suggesting more rapid incorporation into the organoid. SY5Y cells created a stellate organoid. This growth pattern persisted in the TrkB subclone, but the TrkA subclone grew as spheres (fig). Lestaurtinib eliminated the cellular projections when inoculated on day 4. When exposure began on day 0, lestaurtinib also reduced the ultimate size of the organoid.

Conclusions:

Biological characteristics such as growth, cohesion and morphology can be measured in a reproducible way using a microgravity culture system. This may represent a way to characterize the biological and potentially clinical behavior of a tumor in a more refined way than the histological appearance or traditional cell culture alone.



Notes:

P2

NERVE GROWTH FACTOR ACTIVATION OF CASPASE-INDUCED APOPOTOSIS IN HUMAN SK-N-SH NEUROBLASTOMA IN VITRO

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Purpose:

Nerve growth factor (NGF) is a member of a family of neurotrophins whose effects on neuronal cell survival and differentiation are mediated through two distinct receptors: a high-affinity tyrosine kinase receptor (TrkA), and a low-affinity p75 receptor. We previously reported that SK-N-SH neuroblastoma cells transduced with NGF cDNA demonstrated characteristics of differentiation and more readily underwent cell death. The purpose of the current study is to determine if NGF stimulation induces apoptosis in neuroblastoma in vitro, and the mediators by which NGF exerts this effect.

Methods:

The LPCX retroviral vector was used to achieve stable transduction of cDNA for NGF and the p75 receptor into SK-N-SH neuroblastoma cells. Wild type, NGF transfected, and p75 transfected cells were then incubated with varying concentrations of NGF (100-400ng/mL) for varying time periods up to 24 hours. A laddering assay was performed to determine the presence of DNA fragments characteristic of apoptotic cell death. The expression of cleaved and total caspase was determined by Western immunoblotting of whole cell lysates.

Results:

Western blotting confirmed stable cDNA transfection. TrkA expression in both the NGF and p75 transfected cell lines was equivalent to the wild type cell, but p75 expression was significantly higher in both. DNA laddering assay demonstrated that both NGF transfected and p75 transfected cell lines underwent apoptosis after stimulation with NGF. Cleaved caspase-3 increased significantly with NGF stimulation of both the NGF transfected and p75 transfected cell lines, with maximal activation occurring at 1 hour, regardless of NGF dose. Levels of cleaved caspase-3 return to the wild type baseline at 24 hours. Total caspase-3 expression was equivalent in the wild type and transfected cell lines.

Conclusion:

This study confirms that NGF stimulation of SK-N-SH neuroblastoma cells induces apoptosis through activation of the caspase cascade in vitro, and that p75 is the likely receptor mediating this effect.

Notes:

P3

NOTCH 3 ACTIVATION INCREASES PROLIFERATION AND HAS NO EFFECT ON MIGRATION IN A NEUROBLASTOMA TUMOR MODEL

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Purpose:

Pediatric neuroblastoma (NB) is frequently diagnosed when already metastatic, and carries a poor prognosis despite the most intensive available treatments. The Notch family is a newly described family of proteins that includes cell surface receptors (Notch 1, 2, 3 and 4). Notch proteins are critically involved in cell fate regulation, with dysregulation a common feature in human cancers. Notch3, in particular, has been shown to increase cell proliferation in certain cancer cell lines, and correlates with stage and prognosis in neuroblastoma. Conversely, Notch3 represses migration in non-malignant cell types. We hypothesized that over-expression of Notch3 in NB cells would increase behaviors linked to clinical aggressiveness in tumors.

Methods:

Cultured SY5Y NB cells were infected with a plasmid containing the Notch3 intercellular domain (N3IC) gene or control GFP. RNA was extracted and activity assessed by real-time RT-PCR for canonical downstream N3IC targets Hes1, Hey1, and Notch3 itself. To assess proliferation, SY5Y GFP- and N3IC-overexpressing viability and proliferation was quantified at 48 and 72 hours via MTT assay. To assess invasion of extracellular matrix (ECM), serum-starved fluorescently-labeled SY5Y control and N3IC cells were loaded into the inner chamber of a Matrigel™ coated polyethylene-terephthalate porous membrane. Transmembrane migration through a serum gradient was quantified at 24 hours.

Results:

As compared to GFP controls, N3IC expression induced significant increases in Hes1,Hey1, and Notch3. N3IC expression resulted in increased SY5Y viability by 100% and 54% at 48 and 72 hours, by MTT. Intriguingly, migration through Matrigel was slightly increased although the study was not powered to detect significance.

Conclusion:

Notch3 signaling selectively stimulates tumor cell behaviors associated with aggressive growth (expression of Notch target genes and proliferation) in SY5Y NB cells. Migration was not repressed, differentiating this response from those of normal cells. Targeting Notch3 signaling may provide a novel therapeutic approach to NB.

Notes:

P4

CHARACTERIZATION OF TUMOR-STEM LIKE CELLS AND RESISTANCE TO STANDARD THERAPIES IN MOUSE NEUROBLASTOMA.

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Purpose:

There is valid data supporting the cancer stem cell theory for solid tumors and it appears that tumor cells responsible for failure after successful initial therapy exhibit stem cell properties. Such properties include self-renewal, potent tumorigenic activity, an undifferentiated state and resistance to chemo or radiation therapy. We investigated the existence and characteristics of such cells in mouse neuroblastoma.

Methods:

Mouse neuroblastoma was harvested and cultured in DMEM + 10% FBS or NeuroCult complete media specific for the growth and enrichment of neural stem cells. Cell staining was performed to assess stem cell phenotype and differentiation and compared to adult neural stem cells. Tumorigenicity was assessed in vivo with inoculation of neuroblastoma cell types. Stem-like and non stem-like cells were subjected to radiation (2.5Gy) followed by viability and apoptosis assays. Similarly, cell types were also treated with varying doses of Doxorubicin to assess sensitivity. IACUC approval was obtained.

Results:

We describe a subset of neuroblasoma cells harvested from established tumors and cell lines with features and characteristics of neural stem cells. These cells self-renew, grow in neurospheres and under appropriate conditions undergo multi-lineage differentiation. These stem-like cells are remarkably tumorigenic and the tumors produced are pathologically similar to those with features of high risk disease observed in patients. These tumor stem cells are very resistant to radiation and chemotherapy treatment when compared to non stem-like tumor cells.

Conclusions:

We have shown that a subpopulation of tumor-stem cells are present in the mouse neuroblastoma model. This subset of resistant cancer stem-like cells present in mouse neuroblastoma might represent the resistant cell type responsible for treatment failures in patients. Neuroblastoma stem cells appear to be a necessary target for the development of novel therapeutics in this disease.

Notes:

P5

THE ROLE OF DENDRITIC CELLS IN NECROTIZING ENTEROCOLITIS INDUCED BY ENTEROBACTER SAKAZAKII

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Purpose:

Enterobacter Sakazakii (ES) is a rare and virulent pathogen that has been associated with outbreaks of necrotizing enterocolitis (NEC) in neonates. Our previous studies have shown that ES expressing outer membrane protein A (OmpA), induces NEC in neonatal rats by triggering enterocyte apoptosis. Intestinal dendritic cells (DCs) play a key role in luminal antigen sampling. However their role in NEC has not been explored. In this study, we investigate the role of DCs in NEC.

Methods:

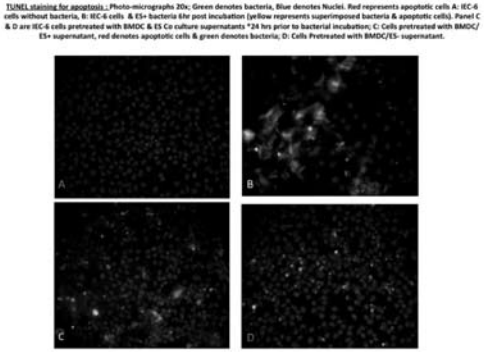
Phagocytosis of rat bone marrow-derived DCs (BMDCs) was measured after infection with OmpA+ (ES+) or OmpA- (ES-) using gentamicin protection assays. Supernatant from DC-ES co-cultures were used to measure cytokines via ELISA and to pre-treat IEC-6 cells for 24 hrs. ES-induced apoptosis in naive and pre-treated IEC-6 cells was measured using TUNEL staining. CaCo2 monolayers were co-cultured with DCs and barrier permeability (trans-epithelial electrical resistance or TEER) was measured after exposure to ES. DCs isolated from the intestine of ES-infected mice were tested for maturation using flow cytometry.

Results:

ES+ invades and survives within BMDCs, while ES- does not. IEC-6 cells pre-treated with DC-ES co-culture supernatant show increased apoptosis when challenged with ES+ but not ES- (Fig. 1C vs. 1D). DCs infected with ES+ produce higher levels of IL-10 and TGF-β and lower levels of IL-6 and TNFα than ES-. Addition of ES-infected DCs to CaCo2 monolayers increases the permeability of the monolayers as measured by TEER. Intestinal DCs isolated from infected mouse pups fail to express maturation markers.

Conclusion:

These results suggest that ES+ invades BMDCs and dampens their immune signaling by preventing pro-inflammatory cytokine production and expression of maturation markers. The secreted factors resulting from the interaction of ES+ with DCs may play a role in increasing apoptosis in the intestinal epithelium leading to NEC.



TUNEL staining for apoptosis : Photo-micrographs 20x; Green denotes bacteria, Blue denotes Nuclei. Red represents apoptotic cells A: IEC-6 cells without bacteria, B: IEC-6 cells & ES+ bacteria 6hr post incubation (yellow represents superimposed bacteria & apoptotic cells). Panel C & D are IEC-6 cells pretreated with BMDC & ES Co culture supernatants *24 hrs prior to bacterial incubation; C: Cells pretreated with BMDC/ES+ supernatant, red denotes apoptotic cells & green denotes bacteria; D: Cells Pretreated with BMDC/ES- supernatant.

Notes:

P6

HEPATIC MITOCHONDRIAL METABOLISM, MEASURED USING [1-13C]METHIONINE BREATH TEST, IN SEPTIC AND NON-SEPTIC INFANTS

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Purpose:

Animal studies have found liver mitochondrial function to be impaired by sepsis, but it is not known whether this is also the case in septic infants. Recently, the [1-13C]methionine breath test (13C-MBT) has been introduced as a non-invasive test of hepatic mitochondrial function. Our aim was to use the 13C-MBT to determine whether mitochondrial function is impaired in septic infants.

Methods:

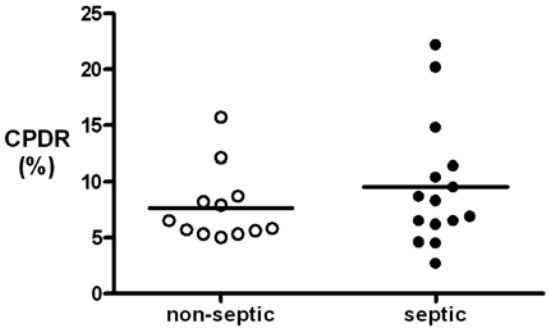
After institutional ethical approval (99SG41), and parental informed consent, we studied two groups of infants <1 year of age, in ICU or in the surgical ward. Group 1 consisted of infants (n=15) with clinical sepsis, according to established consensus criteria. Group 2 consisted of stable infants (n=12) with no evidence of sepsis or systemic inflammatory response syndrome. Each group received a 13C-MBT, consisting of intravenous injection of 3mg/kg [1-13C]methionine. Breath samples were taken at baseline and every 15 minutes up to 2 hours, and the cumulative percent dose recovered (CPDR) calculated. Data are presented as mean±SEM and compared by t-test and linear regression.

Results:

Groups were comparable for age (non-septic 1.4±0.5 months, septic 1.9±0.4) and weight (non-septic 3.2±0.4kg, septic 3.2±0.6). Non-septic infants oxidised, on average, 7.7±0.9 of the dose received (Figure). Septic infants had a wide range of CPDR, but their overall CPDR, 9.6±1.4, was not significantly different from that of non-septic infants (Figure; p=0.3). There was no relationship between C-reactive protein (r2=0.04, p=0.48), alanine aminotransferase (r2=0.01, p=0.74) or alkaline phosphatase (r2=0.06, p=0.27) and CPDR.

Conclusions:

Sepsis does not appear to impair hepatic mitochondrial metabolism in infants. These findings have implications for nutrient provision and metabolism during sepsis.



Notes:

P7

SUBMUCOSAL GLAND DEVELOPMENT IN A FETAL TRACHEA HUMAN-XENOGRAFT MODEL: IMPLICATIONS FOR FETAL GENE THERAPY

Louis D. Le, MD, Sundeep G. Keswani, MD, Foong Y. Lim, MD, Mounira A. Habli, MD, Timothy M. Crombleholme, MD
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Purpose:

Previous work in our lab in a human-fetal trachea xenograft model suggests the potential for treating cystic fibrosis (CF) in utero. The target cells for postnatal gene therapy in CF are the tracheal submucosal glands (SMG), which have been inaccessible to postnatal gene transfer limiting the success of gene therapy. The aim of this study is to determine if SMG development in xenografted human fetal tracheas recapitulates normal fetal trachea development and its validity as a model for studying fetal gene therapy in the tracheobronchial tree.

Methods:

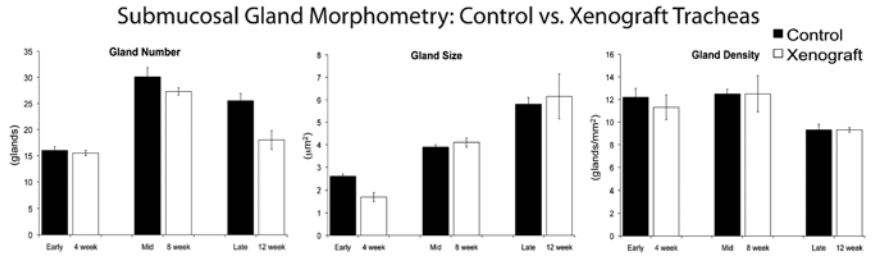
Human fetal tracheas were arbitrarily divided into three developmental phases, early (15-17wks gestational age [GA]), mid (18-20wks GA) and late (21-24wks GA). Fetal tracheas (15-24wks GA, n=12) were xenografted onto immunocompromised mice for 4, 8, and 12 weeks and compared to controls (GA matched fetal tracheas [n=9]). Tracheas were examined using morphometric analysis for SMG developmental staging using the Thurlbeck staging system and mucopolysaccarhide production by carbohydrate histochemistry.

Results:

In both the control and xenograft group, there is no significant difference(p=NS) in gland number, gland size and gland density from early through late phase(See Figure 1). Similarly, using the Thurlbeck staging system, xenografted tracheas demonstrate a similar progression through the 4 stages of SMG development as controls after an initial 4-week phase shift. Lastly, using carbohydrate histochemistry, control and xenografted tracheas demonstrate a characteristic pattern of acidic mucin production in the base of SMG signifying functional glands.

Conclusions:

Fetal trachea xenograft SMGs recapitulate normal fetal tracheal development and appears to be a valid model for studying human fetal gene transfer. The accessibility of SMG stem cells in early phase of tracheal development may afford a unique window of opportunity for gene transfer. This human-xenograft model has the benefit of providing access to human fetal trachea in vivo and permits the study of novel fetal gene therapy strategies.



Notes:

P8

AMNIOTIC CELLS AUGMENT FETAL WOUND HEALING

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Purpose:

Fetal wound healing holds valuable clues of translational relevance to wound management and remains to be fully understood. Amniotic mesenchymal stem cells have been previously shown to populate experimental fetal wounds. We sought to determine whether the presence of amniotic cells in fetal wounds is an epiphenomenon, or whether these cells play an active role in the healing process.

Methods:

After IACUC approval, fetal lambs (n=17) underwent creation of two symmetrical, size-matched skin wounds (n=34) at mid-gestation. Both wounds were encased by a titanium chamber hermetically anchored. One of the chambers was left open (control wound) and the other was closed with a semi-permeable, hydrophilic polyvinylidene fluoride membrane with 0.65μm pores, which allows for passage of water and all molecules, but not any cells (experimental wound). Survivors (n=10) had their wounds (n=20) submitted to multiple analyses at different time points, namely 5/6, 9, 14, 20 and 32 days post-operatively, before term. Statistical comparison were by repeated-measures ANOVA ($P<0.05$).

Results:

Experimental wounds showed a significantly slower healing rate compared to the control wounds ($P=0.005$). Elastin and collagen levels increased significantly over time in both wound types ($P<0.01$). However, paired comparisons indicated significantly lower elastin levels in experimental wounds at the 3 mid time points ($P<0.01$), with no significant differences in collagen levels. No significant changes over time, or between the wound types, were detected in hyaluronic acid levels. Immunohistochemistry for substance P, a known chemotactic fator for mesenchymal stem cells, was positive in both wound types.

Conclusions:

Fetal wound healing encompasses a cellular component derived from naturally occurring amniotic cells. While apparently not absolutely essential to the healing process, amniotic cells do expedite wound closure and enhance its extracellular matrix profile. Further scrutiny into the cellular component of fetal wound healing and its translational implications to wound management is warranted.

Notes:

P9

BILE DUCT EPITHELIAL CELLS; A NOVEL SOURCE OF NEUTROPHIL COLLAGENASE (MMP8)

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Background:

We have previously reported the importance of neutrophils and MMP8 during recovery from cholestatic injury and hypothesize that neutrophil MMP8 or neutrophil dependent activation of intrinsic hepatic MMP8 is critical to resolve collagen fibrosis. The purpose of this study was to further investigate the location and source of MMP8 and collagenase activity during cholestatic injury and repair.

Methods:

Cholestatic liver injury was created in rats by bile duct suspension for 7 days followed by decompression to recover from obstruction. Liver tissue obtained at the time of decompression or after 2 days of repair was processed for morphometric analysis, immunohistochemistry, Western blot and quantitative RT-PCR. Neutrophil whole cell lysates were prepared after harvest from injured and sham operated rats and examined for MMP8. Bile duct epithelial cells isolated from rats treated with choline-deficient diets treated with ethionine were cultured and examined for MMP8 protein. Collagen I in gel and in situ zymography were performed to detect collagenase activity, assess location and confirm MMP identification.

Results:

Gene expression of MMP8 increased in cholestatic injury compared to sham ($p<0.01$). Liver and neutrophil homogenates from injured and decompressed animals expressed both pro- and active- MMP8 protein. Isolated bile duct epithelial cell lysate was positive for active MMP8. Collagen I zymography demonstrated substantial substrate clearing in the neutrophil lane; a clear zone of substrate clearing was recognized in the injured liver and a limited zone in isolated bile duct lanes corresponding to MMP8 protein. In situ zymography revealed activity in the portal tracts.

Conclusion:

Our results reveal that hepatic MMP8 expression increases during injury and one source is likely bile duct epithelial cells. This novel finding of bile duct epithelial neutrophil collagenase (MMP 8) suggests the biliary tract responds to noxious stimuli during cholestatic injury and has the potential to participate in cellular recovery and fibrosis.

Notes:

P10

ENDOTHELIN CONVERTING ENZYME-1 PROMOTES RECYCLING OF THE NEUROKININ-1 RECEPTOR IN ENTERIC NEURONS

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Purpose:

The metalloendopeptidase endothelin converting enzyme-1 (ECE-1) is known to degrade neuropeptides in acidic endosomes. It is also known that ECE-1 knockout mice lack enteric neurons in the distal colon, a phenotype similar to Hirschsprung's disease. However, there is no known function for ECE-1 in enteric neurons. We hypothesize that ECE-1 promotes neurokinin 1 receptor (NK_1R) recycling in enteric neurons after exposure to substance P (SP).

Methods:

Rats were killed in accordance with IACUC guidelines. Small intestine was cut into 2cm segments, stretched and pinned onto a dish. Preparations were incubated with either ECE-1 specific inhibitor SM-19712 (10μM) or vehicle (ddH_2O) in physiologic salt solution ((PSS), 1hr, 37˚C). Segments were treated with either vehicle (ddH_2O) or SP 10nM (1hr, 4°C). Unbound SP was removed (PSS, 3x10min, 4°C) and allowed to recover in warm PSS (37°C, 5%CO_2) with or without SM-19712. Recovery times of 0, 10, 30, 60 and 120min were tested. Myenteric plexus whole mounts were prepared and processed for immunofluorescence using antibodies against NK_1R and ECE-1. Tissue was imaged by confocal microscopy and the subcellular distribution of NK_1R was analyzed using Image J software.

Results:

NK_1R and ECE-1 were coexpressed in myenteric neurons. In unstimulated neurons, NK_1R was at the plasma membrane, and ECE-1 was in endosomes. Image analysis of neurons is shown below.

Conclusion:

We have identified a new role for ECE-1 in enteric neurons, where ECE-1 promotes recycling of NK_1R to the plasma membrane following exposure to SP. Inhibition of ECE-1 with a specific inhibitor, SM-19712, caused retention of NK_1R in ECE-1 containing endosomes, effectively preventing recycling of NK_1R to the plasma membrane. Thus, ECE-1 promotes NK_1R trafficking in enteric neurons. Further investigation is needed to determine how ECE-1 deficiency result in loss of enteric neurons, and if prolonged retention of neuropeptide receptors in endosomes is a potential mechanism.

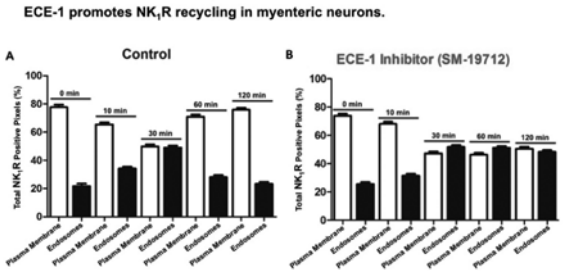

Figure 1. Image analysis of rat whole mounts showing the total percentage of NK_1R positive pixels at the plasma membrane (white bars) and in endosomes (black bar) with or without SM-91712 **A.** In Vehicle, SP treatment causes depletion of NK_1R from the plasma membrane into endosomes from 0 to 30 min, and NK_1R recycles to the plasma membrane by 120 min. **B.** SM-91712 incubation does not affect the internalization of NK_1R into endosomes, but significantly inhibits NK_1R recycling to the plasma membrane at 60 and 120 min (n = 6 rats; 4 images per group; > 60 neurons per group; *** P < 0.005 when compared to control)

Notes:

P11

MATRIX METALLOPROTEINASE-9 INDUCES VASCULAR HYPERPERMEABILITY FOLLOWING TRAUMATIC BURN INJURY

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Purpose:

Traumatic burn is known to be associated with marked increase in vascular permeability. Recent studies have demonstrated that Matrix Metalloproteinase-9 (MMP-9) is associated with vascular hyperpermeability. We hypothesized that MMP-9 is activated following acute burns and is associated with vascular hyperpermeability. The purpose of this study was to determine if MMP-9 inhibition attenuates vascular hyperpermeability.

Methods:

IACUC approval was obtained for this experiment protocol. Male Sprague-Dawley rats were subjected to a 40% total body surface area burn. MMP-9 levels were measured in sham serum and burn serum utilizing SDS-PAGE gelatin zymography. To determine if MMP-9 inhibition attenuates vascular hyperpermeability, rat lung microvascular endothelial cells were grown as monolayers on Transwell plates. Monolayers were treated with activated MMP-9(aMMP-9), and aMMP-9 + specific inhibitor of MMP-9 (MMP-9i). In a separate experiment, monolayers were treated with burn serum, sham serum, sham serum + MMP-9i, and burn serum + MMP-9i. Permeability was assessed by FITC albumin-flux across the monolayers as measured by change in fluorescence intensity. Vascular endothelial cell damage was assessed by immunohistochemistry for Beta-catenin, VE-cadherin, and F-actin. Extra-cellular matrix damage was assessed by laminin immunofluorescence.

Results:

Zymography for MMP-9 showed increased enzymatic activity in burn serum relative to sham serum, and this activity was significantly inhibited by MMP-9i (paired t-test, $n=4$, $p < .05$). Activated MMP-9 increased monolayer permeability relative to control (paired t-test, $n=5$, $p < .05$). MMP-9 inhibition significantly attenuated both burn and aMMP-9 induced monolayer permeability (paired t-test, $n=5$, $p < .05$). MMP-9 inhibition reduced the adherens junction complex damage induced by burn serum or aMMP-9, as well as extra-cellular matrix damage.

Conclusion:

Following traumatic burns MMP-9 is activated, and is a potent regulator of microvascular hyperpermeability. Inhibition of MMP-9 attenuates the microvascular hyperpermeability associated with burn injury.

Notes:

P12

EXPRESSION OF *IROQUOIS* GENES IS UPREGULATED DURING EARLY LUNG DEVELOPMENT IN THE NITROFEN INDUCED PULMONARY HYPOPLASIA

Takashi Doi, MD, Aušra Lukošiute, Jens Dingemann, MD, Elke Ruttenstock, MD, Prem Puri, MS, FRCS, FRCS (ED), FACS
The Children's Research Centre, Dublin, Ireland

Purpose:

The pathogenesis of lung hypoplasia associated with congenital diaphragmatic hernia (CDH) remains unclear. *Iroquois* homeobox (*Irx*) genes have been implicated in the early lung morphogenesis of *Drosophila* and vertebrates. In fetal rat, *Irx1-3* and *Irx5* gene expression is seen in the developing lung up to day 18.5 of gestation (D18.5), whereas *Irx4* is not expressed. Antisense knockdown of *Irx* genes displays loss of lung mesenchyme and dilated air spaces. In contrast, the hypoplastic lung in the nitrofen CDH model shows thickened mesenchyme and diminished air spaces. We designed this study to investigate the hypothesis that the *Irx1-3* and *Irx5* genes are upregulated during early lung morphogenesis in the nitrofen-induced hypoplastic lung.

Methods:

Pregnant rats were exposed to either olive oil or nitrofen on D9. Fetal lungs harvested on D15 were divided into two groups: Control and Nitrofen, and the lungs harvested on D18 were divided into three groups: Control, Nitrofen without CDH (CDH(-)) and Nitrofen with CDH (CDH(+)) (n=24 at each time point). Pulmonary mRNA expression levels of *Irx* genes were analyzed by real-time PCR. Immunohistochemistry was performed to evaluate protein expression of Irx family.

Results:

Pulmonary *Irx1-3* and *Irx5* mRNA expression levels were significantly upregulated in nitrofen group compared to controls at D15 (*$p<0.005$ vs Control, §$p<0.01$ vs Control)(Table). Strong Irx immunoreactivity was seen in the branching lung epithelium on D15 in nitrofen-induced hypoplastic lung compared to control lungs (Figure).

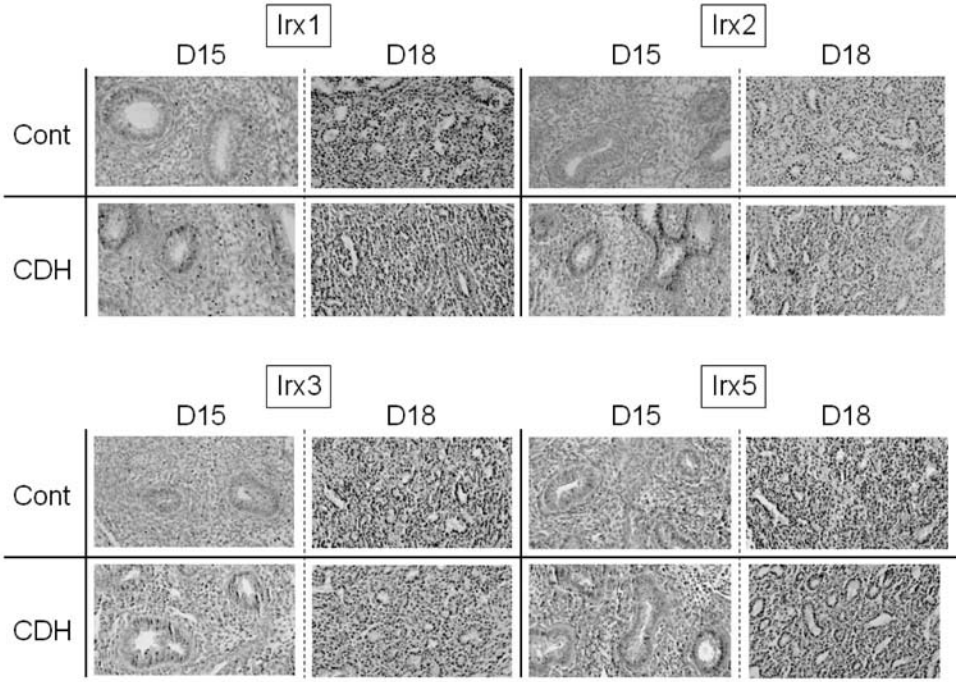
Conclusions:

Overexpression of pulmonary *Iroquois* gene family in the early lung development may cause pulmonary hypoplasia in the nitrofen-induced congenital diaphragmatic hernia model by inducing lung dysmorphogenesis with thickened mesenchyme and diminished air spaces.

Pulmonary mRNA Expression Levels of Irx Genes

		Control	Nitrofen	
D15	*Irx1*	0.75±0.67	3.18±0.90*	
	Irx2	4.03±1.58	11.53±5.39*	
	Irx3	0.85±0.28	1.77±0.53§	
	Irx5	1.27±0.72	3.28±0.68*	
		Control	CDH(-)	CDH(+)
D18	*Irx1*	7.34±1.53	6.91±1.74	5.32±1.53
	Irx2	10.95±3.77	10.53±3.93	11.51±8.01
	Irx3	2.01±0.54	2.17±0.63	2.39±1.12
	Irx5	4.13±0.62	4.15±0.89	3.39±1.39

Notes:


Irx1
D15
D18
Irx2
D15
D18
Cont
CDH
Irx3
D15
D18
Irx5
D15
D18
Cont
CDH

P13

TRACHEAL DEFECT REPAIR USING A PLGA-COLLAGEN HYBRID SCAFFOLD REINFORCED BY A COPOLYMER STENT WITH BASIC FIBROBLAST GROWTH FACTOR-IMPREGNATED GELATIN HYDROGEL

Yukihiro Tatekawa
Tsukuba University, Tsukuba, Japan

Purpose:

We investigated the rigid support, epithelial lining and regenerated cartilage in trachea defect repair using a biodegradable scaffold with basic fibroblast growth factor-impregnated gelatin hydrogel.

Material and Methods:

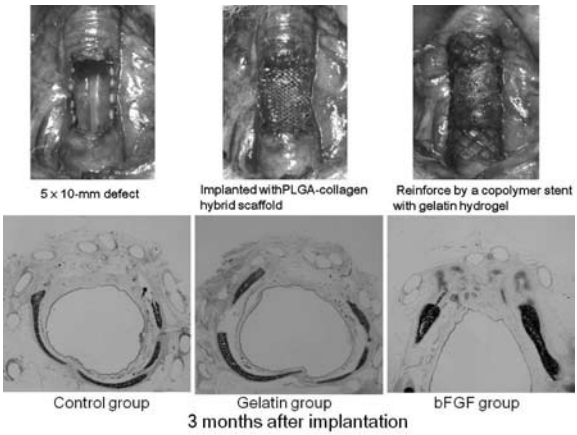
A 5 x 10-mm defect was created in the cervical trachea of rabbits. The defect was implanted with a poly-lactic-co-glycolic acid (PLGA)-knitted mesh-collagen sponge hybrid scaffold. The implanted trachea was reinforced by a copolymer stent of polycaprolactone and polylactic acid (P(CL/LA)) coarse fiber mesh. A gelatin hydrogel was used for providing a sustained release of basic fibroblast growth factor (bFGF). The reconstructed tracheas were divided into three groups with wrapped materials; none (control group: n=2), a gelatin hydrogel with saline (gelatin group: n=2), and a gelatin hydrogel with100 micro gram of bFGF (bFGF group: n=2), which were retrieved 1 or 3 months after implantation.

Results:

The reconstructed biodegradable scaffolds could maintain airway structure up to 3 months after implantation. Tracheal epithelialization was recognized from one month postoperatively in each group. Cartilage formation was strongly regenerated in the bFGF group and the defect portion was covered with the newly formed cartilage in an arched shape between the cartilage stumps.

Conclusion:

We confirmed the rigid support, epithelial lining and regenerated cartilage of artificial trachea using a biodegradable scaffold with basic fibroblast growth factor -impregnated gelatin hydrogel.



Notes:

P14

CELL LINEAGE TRACING OF TISSUE-ENGINEERED SMALL INTESTINE IN THE MOUSE MODEL DEMONSTRATES CONTRIBUTIONS TO THE STEM CELL NICHE AND THE ENTIRE EPITHELIUM

Frederic G. Sala, PhD, Jamil A. Matthews, MD, Allison L. Speer, MD, Dianne C. Skelton, Tracy C. Grikscheit, MD
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Purpose:

Lack of gastrointestinal tissue in children such as short bowel syndrome leads to severe morbidity and mortality. Current therapies do not offer adequate palliation. In previous work, we used tissue-engineered small intestine to rescue Lewis rats that underwent massive small bowel resection. Previously, we showed that the entire epithelium was generated from the implanted donor cells while the mesenchyme was of both donor and host origins. The purpose of this work is to identify the host and donor contribution of each cell type composing the tissue-engineered small intestine in the mouse model.

Methods:

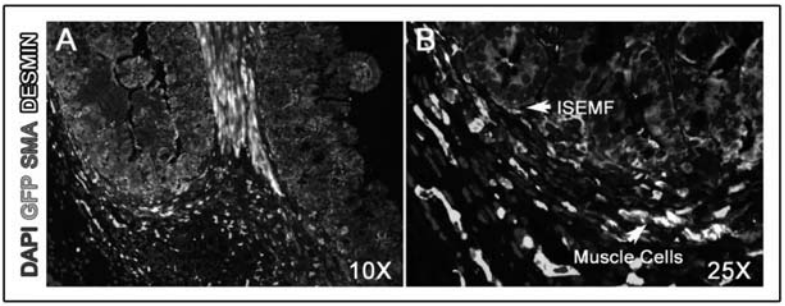
Organoid units (OUs), multicellular clusters of epithelial and mesenchymal cells, were derived from full-thickness small intestine of two-week-old mice that constitutively express Green Fluorescence Protein (GFP). OUs were isolated using our variation of the previous protocol reported in the Lewis rat. OUs were implanted on a biodegradable polyglycolic/polylactic acid scaffold into the omentum of irradiated NOD/SCID mice and harvested after 4 weeks. Immunohistochemistry was performed to identify the origin of each cell type.

Results:

Tissue-engineered small intestine was successfully generated. The entire epithelium was GFP positive as well as a substantial part of the mesenchyme. Specifically, a key component of the stem cell niche, the intestinal subepithelial myofibroblasts, was positive for both smooth muscle actin and GFP. Smooth muscle cells (smooth muscle actin and desmin positive) were also positive for GFP (Figure).

Conclusions:

Although organoid units were once believed to provide primarily epithelial cells, lineage tracing demonstrates a donor contribution to the entire engineered intestinal epithelium and an important contribution to the mesenchyme. These data suggest that the engineered tissue may grow from transplanted intact stem cell niches. Further evaluation of the donor and host contributions to tissue-engineered intestine will direct the most efficient population of donor cells and host conditions to establish future human therapies.



Notes:

P15

THE DEVELOPMENT OF NECROTIZING ENTEROCOLITIS RESULTS IN UPREGULATION OF THE WNT TARGET AND INTESTINAL STEM CELL SIGNATURE GENE, ASCL2: A NOVEL PATHWAY LEADING TO INTESTINAL STEM CELL IMPAIRMENT AFTER BARRIER INJURY.

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Division of Pediatric Surgery, Pittsburgh, PA, USA

Purpose:

Necrotizing enterocolitis (NEC) is the leading cause of death and disability from gastrointestinal disease in preterm infants, yet the underlying mechanisms remain largely unexplained. We have recently demonstrated a critical role for Toll-like receptor 4 (TLR4) in the pathogenesis of NEC, as TLR4 activation leads to reduced intestinal repair. We now focus our investigation on the intestinal stem cells (ISC) that are controlled by WNT gene signaling, specifically through transcription factor Ascl2. The role of the ISC in NEC is unknown, but given that ISCs control intestinal epithelial renewal, we now hypothesize that WNT signaling is increased in NEC in an attempt to restore gut barrier integrity.

Methods:

Experimental NEC was induced in newborn mice using combined enteric feeding and intermittent hypoxia and the terminal ileum was harvested for characterization of Ascl2 expression. In parallel, ISCs were harvested from newborn intestine using mechanical separation and differential centrifugation. To assess the role of TLR4 in Ascl2 expression, a dominant negative TLR4 (shTLR4) was created in an intestinal crypt cell line (IEC-6).

Results:

Experimental NEC was associated with a profound reduction in proliferation of the intestinal epithelium. Further, NEC was associated with a significant upregulation of Ascl2 (1.54 vs. 1.0, $p<0.05$). Significantly higher levels of Ascl2 (2.5 vs. 1.0, $p<0.05$) were found in harvested ISCs. In vitro, there was a significant increase in Ascl2 with TLR4 activation via LPS (1.59 vs. 1.0, $p<0.05$). Strikingly, however, in the shTLR 4 cells there was no change in Ascl2 expression after LPS (1.01 vs. 1.0, $p=0.9$).

Conclusions:

These data suggest WNT signaling is increased in NEC, possibly in a compensatory response to intestinal injury. When TLR4 signaling is blocked and cells are protected, there is no increase in Ascl2, suggesting that TLR4 signaling may play a role in the intestinal stem cell response to injury.

Notes:

P16

A NOVEL MODEL FOR INVESTIGATING THE EFFECTS OF VASCULAR ENDOTHELIAL GROWTH FACTOR (VEGF) IN THE MOUSE MESENCHYME.

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Saban Institute-Children's Hospital Los Angeles, Los Angeles, CA, USA

Purpose:

In intestinal maturation and repair the role of VEGF in mesenchyme-epithelium cross talk is not entirely defined. Intestinal epithelial stem cell proliferation depends on this signaling. Two major components of an intact intestinal stem cell niche are the intestinal subepithelial myofibroblasts (ISEMF) and the intestinal epithelial stem cells identified by DCAMKL-1. We sought to create a transgenic mouse with an underexpression of VEGF in the mesenchyme and to evaluate the effects on the intestinal epithelium.

Methods:

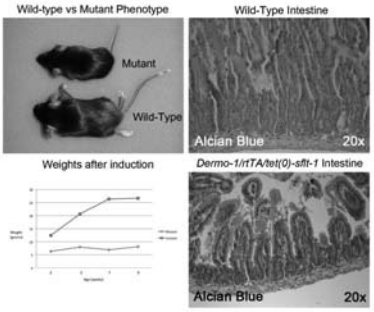
Adult mice carrying the transgene *Dermo-1-Cre*, a inducible mesenchyme-specific promoter and *rtTA*, a transactivator which is activated by *Cre* recombinase, were crossed with mice carrying the gene for *tet(0) sflt*, a soluble VEGF receptor. The resultant triple transgenic *dermo-1/rtTA/tet(0) sflt-1* genotype was confirmed by PCR. For transgene activation, the mice were fed doxycyline starting at postnatal day 0 (P0) via breast milk and then chow after weaning, and weights were followed. At P21, the small intestine and colon were stained with hematoxylin and eosin as well as immunohistochemically for DCAMKL-1, proliferative cell nuclear antigen (PCNA), desmin, and smooth muscle actin (SMA), a marker specific for ISEMF, which are further confirmed as negative for the marker desmin.

Results:

The *dermo-1/rtTA/tet(0) sflt-1* mice were smaller with slower weight gain compared their littermate controls. The intestinal villi of the *dermo-1/rtTA/tet(0) sflt-1* mice were smaller, with reduced villus height and number per high-powered field. DCAMKL-1 and PCNA positive cells were present in the crypts of both specimens. SMA positive ISEMF were noted in their usual location: the mesenchyme, surrounding each crypt and extending into the mesenchymal component of the villi.

Conclusion:

Here we demonstrate a novel inducible model for reduced VEGF expression in the mesenchyme, a useful tool to investigate the effect in the intestinal stem cell niche and other organ systems in which mesenchymal VEGF signaling is important.



Notes:

P17

COMPARISON OF SMALL MOLECULES AND ACTIVIN/WNT3A IN DERIVING DEFINITIVE ENDODERM FROM EMBRYONIC STEM CELLS.

Blair Roszell, MS[1], Ariel Seaton, BSc[1], Christine M. Finck, MD[2]
[1]University of Connecticut Health Center, Farmington, CT, USA, [2]Connecticut Children's Medical Center, Hartford, CT, USA

Purpose:

In prematurity, pulmonary hypoplasia accounts for 70% of neonatal mortality. Cell-based therapy is a novel and promising treatment option, but deriving definitive endoderm, the precursor of lung epithelial cells, from Embryonic Stem (ES) cells is still inefficient. Here, we compare growth factor-based and small molecule-based methods for deriving definitive endoderm, and assess these cells ability to further differentiate into alveolar epithelial type II cells.

Methods:

Mouse embryonic stem cell line E14 was differentiated using a low-serum growth factor containing medium (Activin20ng/ml/Wnt3a 1ng/ml) and compared to a low-serum small molecule contatining medium (IDE2, 1μM) for 6 days with a low density seeding method. These cells were characterized with immunofluorescent staining for markers Foxa2, Cxcr4 and E-Cadherin and yield was quantified using flow cytometric analysis. Next, the medium was switched to an optimized FGF2 (500ng/ml) supplemented medium for 5 days and assessed for markers of alveolar epithelial type II differentiation.

Results:

The use of small molecules improved the percentage yield of definitive endoderm from ESCs by 2.1-fold compared to an optimized activin/wnt3a containing medium, as quantified by flow cytometry. Day 6 endoderm derived from both methods was shown to engraft in E8.75 mouse whole embryo explants. Furthermore, both methods yielded cells that stain positive for endoderm markers Foxa2 and E-Cadherin. Finally, IDE2-derived endoderm was shown to have the ability to further differentiate into lung epithelial lineages by pro-surfactant C staining and RT-PCR detection of surfactant genes.

Conclusions:

Our results suggest that small molecules are an attractive and potent alternative to growth factor cocktails in differentiating ES cells to lung epithelial lineages. Though little is known about the mechanism of action of IDE2, we show that endoderm derived using this method is similar in phenotype and function from ES cells differentiated in activin/wnt3a containing medium.

Notes:

P18

DO PATIENTS WITH DOWN SYNDROME DEVELOP APPENDICITIS?

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[1]Children`s Hospital, Greenville Hospital System, Greenville, SC, USA, [2]Greenville Hospital System, Greenville, SC, USA

Purpose:

The increased incidence of specific GI pathologies in patients with Down syndrome (DS) is well known. There are over 400,000 individuals with DS in the United States. Given their number and the high incidence of acute appendicitis (AA) in the general population, one would expect a certain number of patients with DS to develop AA. However, experience at several institutions suggests that AA is rare in patients with DS. We sought to determine whether the annual incidence of AA is significantly lower in patients with DS than in the general population.

Methods:

A 13-year cross-sectional study (1996-2008) of our state hospital de-identified discharge database was performed to estimate the annual incidence of AA in patients with DS and in the general population; ICD-9 diagnosis codes were used to define AA and DS. Estimates were generated for both the pediatric (0-17 years) and adult (≥18 years) populations and were compared using Fisher's 95% confidence intervals [CI]. In addition, we queried our own hospital database for a 10-year frame (January 2000 - July 2009).

Results:

Analysis of the state data over the 13-year time frame revealed significantly lower AA incidence rates in the DS population (Table 1). Our own institution's database (~ 40,000 discharges annually) contained 0 patients with both diagnoses.

Conclusions:

The incidence of acute appendicitis is markedly lower in patients with Down syndrome than in the general population. Although the biologic basis for this remains unknown, this information is relevant in the evaluation of the acute abdomen in patients with Down syndrome. Additionally, prophylactic incidental appendectomy may not be warranted in Down syndrome patients.

Table 1. Incidence of acute appendicitis (AA) in Down syndrome (DS) vs. general population

Population	Age Group (years)	Total Discharges	Total with appendicitis (AA)	Incidence of appendicitis (AA) [CI]
General	0-17	1,164,073	10,341	8.9 [8.7, 9.1]
Down syndrome (DS)	0-17	3,561	9	2.5 [1.2, 4.8]
General	≥ 18	5,676,262	32,141	5.7 [5.6, 5.7]
Down syndrome (DS)	≥ 18	3,329	9	2.7 [1.2, 5.1]

Notes:

P19

AN EVIDENCE-BASED CLINICAL PROTOCOL FOR DIAGNOSIS OF ACUTE APPENDICITIS DECREASED THE USE OF COMPUTED TOMOGRAPHY IN CHILDREN: A PROSPECTIVE STUDY

Obinna O. Adibe, MD, Erik N. Hansen, MD, MPH, Albert J. Chong, MD, MPH, Lena Perger, MD, Richard Keijzer, MD, PhD, Oliver J. Muensterer, MD, PhD, Keith E. Georgeson, MD, Carroll M. Harmon, MD, PhD
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Purpose:

The increased use of computed tomography (CT) to diagnose appendicitis in children has led to a concern for the possibility of increased CT-related cancer morbidity. A long-term strategy by the National Cancer Institute aimed at reducing CT radiation exposure in children included use of selective strategies for pediatric imaging. We designed a clinical protocol for the diagnosis and treatment of appendicitis in children in an attempt to decrease the use of CT scans at our institution.

Methods:

After IRB approval, patients who had surgical consultation for suspected appendicitis were placed on the clinical protocol. Data concerning diagnosis and treatment were collected prospectively. Retrospective data from patients admitted to our institution with acute appendicitis prior to the clinical protocol were collected as historical controls. Statistical analysis was performed using the Mann-Whitney U test and Pearson's chi-square test.

Results:

One hundred twelve patients were diagnosed and treated by our protocol between June and September 2009. Of these, 73 patients underwent a laparoscopic appendectomy for acute appendicitis. This was compared to 146 patients from 2007. Total pre-operative CT use (in-house and outside institutions) decreased from 80.8% of appendectomy patients in 2007 to 60.2% presently ($p=0.004$). In-house CT use decreased from 71.2% to 24.7% ($p<0.001$). Pre-operative ultrasound use increased from 2.7% to 23.3% ($p<0.001$). The negative appendectomy rate remained the same (6.8%)

Conclusion:

Our preliminary findings suggest that the implementation of an evidence-based clinical protocol for the diagnosis and treatment of acute appendicitis in children may safely decrease the use of CT scans and increase the use of ultrasounds. This may be cost-effective while sparing the child of the potentially deleterious long-term effects of CT radiation.

Notes:

P20

EXPERIENCE PERFORMING 64 CONSECUTIVE STAPLED INTESTINAL ANASTOMOSES IN SMALL CHILDREN AND INFANTS.

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Univ Texas Southwestern Medical Center, Dallas, TX, USA

Abstract Body:

Purpose:

Intestinal anastomosis in small children has traditionally been performed using hand-sewn techniques. Some adult studies have suggested a reduction of anastomotic complications and improved patient outcomes using a stapled technique. Little data exist evaluating the efficacy of stapled intestinal anastomoses in the infant and pediatric populations.

Methods:

A retrospective review of the author's 5-year experience using a mechanical stapler to treat 64 consecutive children requiring intestinal anastomoses distal to the duodenum. In all cases, a side to side, functional end to end anastomosis was created. Postoperative complications, outcomes, and modifications made to the technique were identified.

Results:

Since 2004, 64 children (median age= 3 months, range= newborn to 24 months) have undergone procedures requiring intestinal anastomosis. Twenty-six of 64 children (41%) were one week or less in age. Twenty-seven children (42%) underwent colostomy, ileostomy or jejunostomy closure using a stapler following intestinal preparation. Thirty-seven children (58%) underwent a primary bowel resection and subsequent stapled anatomosis in treating small bowel/colon atresia (n=16), small bowel duplication (n=1), Meckel's diverticulum (n=3), NEC (n=2), intussusception (n=7), intestinal stricture (n=4), intestinal complications of a mesenteric band (n=2) and bowel perforation secondary to trauma (n=2). Complications included wound infection (n=2). One patient had a prolonged anastomotic ileus in the early postoperative period following closure of a colostomy and was found to have an anastomotic stricture (n=1). The stricture was revised using the intestinal stapler. No issues regarding anastomotic dilatation and subsequent stasis/overgrowth have been identified.

Conclusions:

These results suggest that stapled bowel anastomosis is a safe and effective approach applicable to a variety of surgical diseases in newborns and infants.

Notes:

P22

3-DIMENSIONAL COMPUTED TOMOGRAPHY (3-D CT) FOR EVALUATION AND MANAGEMENT OF CHILDREN WITH COMPLEX CHEST WALL ANOMALIES: USEFUL INFORMATION OR JUST PRETTY PICTURES?

Elizabeth H. Calloway, BA[1], Ali N. Chhotani, BS[1], Yueh Z. Lee, MD, PhD[2], J. Duncan Phillips, MD[3]
[1]UNC Chapel Hill School of Medicine, Chapel Hill, NC, USA, [2]UNC Chapel Hill Department of Radiology, Chapel Hill, NC, USA, [3]UNC Chapel Hill Department of Surgery, Chapel Hill, NC, USA

Purpose:

Shaded Surface Display (SSD) technology, with 3-D CT reconstruction, has been reported in a few small series of patients with congenital or acquired chest wall deformities. SSD images are visually attractive and educational, but many institutions are hesitant to utilize these secondary to cost and image data storage concerns. This study was designed to assess value of SSD in the evaluation and management of children with complex chest wall anomalies.

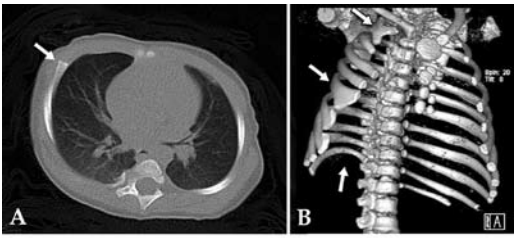
Methods:

Following IRB approval, we performed a retrospective review of records of 82 patients with chest wall deformities, evaluated with SSD, from 2002 to 2009. SSD usefulness, when compared with routine 2-D CT, was graded on a strict numerical scale from 0 (added no value besides education) to 3 (critical for surgical planning and patient management).

Results: There were 56 males and 26 females. Median age was 15.3 years (range: 0.6-41.1). Deformities included 56 pectus excavatum, 19 pectus carinatum, and 8 other/mixed deformities. 6 patients also had acquired asphyxiating thoracic dystrophy (AATD), and 11 (13%) had previous chest wall reconstructive surgery. In 25 (30%) patients, SSD was useful or critical. Findings underappreciated on 2-D images included: sternal abnormalities (29), rib abnormalities (28), and heterotopic calcifications (7). SSD changed or influenced operation choice (4), clarified bone versus soft tissue (3), helped clarify AATD (3), and aided in rib graft evaluation (3). Point biserial correlation coefficient analysis displayed significance for SSD usefulness in patients with previous chest repair surgery (Rpb=0.48, p<=0.001), AATD (Rpb=0.34, p=0.001), pectus carinatum (Rpb=0.27, p=0.008), and females (Rpb=0.19, p=0.044).

Conclusions:

Shaded Surface Display, when used to evaluate children and young adults with congenital or acquired chest wall deformities, provides useful or critical information for surgical planning and patient management in 1/3 of patients, especially those requiring a second operation, with acquired asphyxiating thoracic dystrophy, pectus carinatum, and females.



A: 2D-CT cross-sectional image, showing rib asymmetry and anterior protrusion of the right parasternal cartilages (variant of pectus carinatum).
B: SSD of same patient, showing bifid upper right rib, multiple lower rib anomalies, absent right12th rib; also helps to clarify asymmetric/unusual sternal segmentation.

Notes:

P23

PRE-CLOSURE FLUID RESUSCITATION INFLUENCES OUTCOME IN GASTROSCHISIS

Leigh A. Jansen, MD[1], Yi Lin, MSc[2], Ying MacNab, PhD[2], Erik D. Skarsgard, MD[1]
[1]BC Children's Hospital, Vancouver, BC, Canada, [2]University of BC School of Population and Public Health, Vancouver, BC, Canada

Purpose:

It might be reasonably assumed that in utero and postnatal fluid loss from gastroschisis (GS) bowel exposure predisposes to intravascular volume deficits which should be aggressively replaced immediately after birth. The purpose of our study was to evaluate effects of pre-closure intravenous fluid resuscitation on GS outcome.

Methods:

With IRB approval, cases were accrued from a national GS database. Risk variables analyzed included GA, BW, neonatal illness severity (SNAP-II) score, bowel injury severity and resuscitative intravenous fluid (crystalloid or colloid) administration within the first 6 hours of life, or prior to attempted closure, whichever came first. Outcomes analyzed included closure success (any method), urgent intended closure success, ventilation, TPN, and LOS days, and episodes of bacteremia. Linear and logistic regression analyses were performed.

Results:

407 live born GS cases were identified, of which 362 had complete resuscitative fluids data. Mean BW, GA and SNAP-II score were 2562 +/- 539 grams, 36.17 +/- 1.95 weeks and 9.97 +/- 12.65 respectively. 382 patients (96%) survived with mean post-closure ventilatory days, TPN days and LOS of 5.35 +/- 7.05, 41.03 +/- 44.60 and 50.68 +/- 55.32 days respectively. 162 patients received no supplemental fluid resuscitation, while 200 patients received a mean of 21.49 (0.81-134.81) cc/kg of intravenous fluid. The analyses demonstrated a significant, direct relationship between resuscitative volume and days of post-closure ventilation, TPN, LOS and episodes of bacteremia; specifically, every 17 cc/kg of fluid predicted one additional: ventilation day ($p=0.002$); TPN day ($p=0.01$); and hospital day ($p=0.01$); and 0.02 odds increase of an episode of bacteremia ($p=0.03$). These relationships persist after adjustment for previously validated predictive covariates (GA, SNAP-II score, bowel injury severity).

Conclusions:

Judicious, pre-closure fluid resuscitation is essential in early GS management. Excessive fluid offers no outcomes benefit and in fact imposes a deleterious effect on several survival outcomes.

Notes:

P24

A CRITICAL REVIEW OF PREMATURE INFANTS WITH INGUINAL HERNIAS: OPTIMAL TIMING OF REPAIR, INCARCERATION RISK, AND POSTOPERATIVE APNEA

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Purpose:

This study evaluates the optimal timing for repair, incarceration risk, and postoperative apnea rate in premature infants with inguinal hernias.

Methods:

A retrospective review of all premature infants undergoing inguinal hernia repair from 2006-08.

Results:

172 patients were identified; 81% were male, mean gestational age was 30.7±3.9 weeks, and mean birth weight was1428±713 grams. Overall comorbidities included: history of apnea (53%), history of mechanical ventilation (46%), history of GERD (28%), cardiac anomalies (26%), and history of IVH (10%). At the time of repair, mean postconceptional age was 46.6±6.5 weeks, mean weight was 3688±1334 grams, 18% required medication for chronic lung disease, 20% required antireflux medications, and 12% required home oxygen. 97% of patients underwent general anesthesia, 11% laparoscopic contralateral groin exploration, and 85% had bilateral inguinal hernia repair. 127 patients were evaluated in the clinic and scheduled for elective repair. 35 patients were discharged from the NICU with a known hernia and none developed incarceration prior to repair. There were no postoperative apnea episodes in any of these 127 patients. 45 patients had inguinal hernia repair prior to discharge from the NICU. 13% required prolonged (>48 hours) intubation after repair. The median time to discharge from the NICU after repair was 8 days (2-51 days). 8 patients (4.6%) developed an incarcerated hernia in this study. 5 patients incarcerated in the NICU prior to discharge and 3 patients had incarceration as their initial presentation.

Conclusions:

There is minimal risk of postoperative apnea for premature infants undergoing elective inguinal hernia repair, thus questioning whether routine hospital admission is required. Similarly, the risk of incarceration in premature infants discharged from the NICU with a known hernia is low. Repair of inguinal hernia prior to discharge from the NICU is associated with a prolonged hospital stay. Prospective studies are needed to validate these findings.

Notes:

P25

TEMPORAL ASSOCIATION BETWEEN BLOOD TRANSFUSION AND NECROTIZING ENTEROCOLITIS IN PREMATURE INFANTS

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Purpose:

Multiple clinical studies demonstrate increased mortality and morbidity associated with blood transfusions. Transfusions lead to tissue hypoxia, possibly due to a lack of nitric oxide (NO) in the stored blood, and may lead to an increased risk of necrotizing enterocolitis (NEC) in susceptible infants. Our hypothesis is that onset of NEC is associated temporally with blood transfusions in premature infants.

Methods:

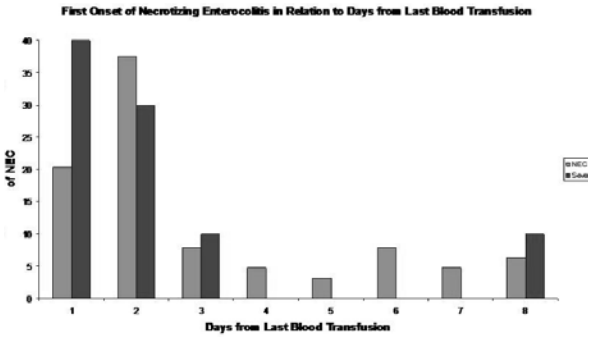
This is a retrospective review of premature infants admitted to a single NICU from July 2004 to July 2006. Clinical and demographic data were recorded including transfusions information. Infants were classified into three cohorts: no NEC, mild/moderate NEC, and severe NEC, based on clinical and radiological parameters. Only the first diagnosis of NEC was included. The data was analyzed using JMP statistics software using t-test, ANOVA and multivariate analysis with significance $P < 0.05$.

Results:

Of 767 infants, 143 had NEC (106 mild/moderate, 37 severe). Infants with either mild/moderateNEC or severe NEC had more total transfusions than those without NEC (control 1.3 transfusions, mild/moderate NEC 6.4, severe NEC 9, p<0. 05). Of the infants with NEC, 74 of 143 received a transfusion prior to the diagnosis of NEC (64 mild/moderate, 10 severe). Furthermore, a diagnosis of NEC was made within 48 hr after receiving a transfusion in 58% of the mild/moderate and 70% of the severe NEC infants (Figure 1). Infants with either mild/moderate NEC or severe NEC had equivalent gestational age, weight, gender, race, BUN, WBC, and HCT prior to transfusion.

Conclusion:

There is a temporal association between blood transfusion and onset of necrotizing enterocolitis in certain preterm infants. This onset of NEC following blood transfusion may be due storage lesions inherent in transfused blood. This association should factor into blood transfusion policies for premature infants.



Notes:

P26

OUTCOME OF PATCH REPAIR IN CONGENITAL DIAPHRAGMATIC HERNIA IN FETO ERA

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Purpose:
Prosthetic patches are being increasingly used as more "borderline" infants survive to undergo surgery for Congenital Diaphragmatic Hernia (CDH). Aim of this study was to evaluate the medium-term outcome following prosthetic patch repair in infants with CDH, with special reference to those, who have undergone Fetal Endoscopic Tracheal Occlusion (FETO) intervention.

Methods:
This was a ambispective review of all infants undergoing surgical repair of CDH (January 1994 - August 2009). FETO was used since May 2002. Hemi-diaphragm integrity was assessed by out-patient assessment/chest x-ray. Variables included recurrence, and presence or absence of gastro-oesophageal reflux (GOR) and chest wall deformity. Chi^2 analysis was used to compare categorical variables. Data are quoted as median (range). $P < 0.05$ was regarded as significant.

Results:
83 infants underwent surgical repair. Follow-up (median = 2 yrs (range 0.2 - 14 yrs) was available for 68 infants. Primary closure was achieved in 39/68 (57%), prosthetic patch closure in 29/68 (43%). The types included Permacol (n = 21, 72%), Goretex (n = 4, 14%), Surgisys (n=1, 3%) and Dacron (n=3, 10%). FETO intervention had been used in 41% (n=28) of these patients, of them 58% (n=19) required patch. Most important data have been summarised in table 1.

Conclusions:
FETO group of infants has higher incidence of prosthetic patch requirement and recurrence. Although the prosthetic patches group has a higher recurrence rate, encouragingly there is no evidence of an increase in other morbidities.

Table 1

Variables	Patch (n = 29)	Primary repair(n = 39)
Recurrence	7 (24%)	3 (8%) P value 0.08
Median time to recurrence (range)	1.3 (0.2-3.9) yrs	0.8 (0.7-1) yrs
Pectus excavatum;Assymmetry; Scoliosis	2 (7%);1 (3%) ; 0	5 (13%);2 (5%); 1 (3%)
GOR: Mild-moderate; Severe	6 (21%);3 (10%)	9 (23%);5 (13%)
Deafness	0	3 (8%)
Vision abnormal	0	1 (3%)
Follow-up	1 (0.2-8) yrs	4 (0.2-14) yrs

Notes:

P27

LAPAROSCOPIC TWO-STAGE FOWLER-STEPHENS ORCHIDOPEXY FOR ABDOMINAL TESTIS

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Purpose:

Laparoscopic first stage (vessel clipping) Fowler-Stephens orchidopexy is a well-established technique. Laparoscopic second stage is not widely performed by pediatric surgeons, however. We present our 15-year experience with fully laparoscopic two-stage procedures for cryptorchidism.

Methods:

All patients with non-palpable testis diagnosed between 1995 and 2008 were reviewed. Laparoscopic clipping of the testicular vessels was followed, at least 3 months later, by laparoscopic mobilization of the testis on a wide peritoneal pedicle and scrotal placement of the testis through a tract medial to the epigastric vessels. Testicular size was measured at completion of the second stage and at follow-up.

Results:

Forty-three children presented with a non-palpable testis despite careful examination, including examination under anesthesia. Three patients were excluded because of limited follow-up. Thus, 40 patients (42 testes) were reviewed. Median age at first operation was 11 months (range 7 months-14 years). At laparoscopy 6 testes were pelvic, 5 in a high location and 3 were at the internal ring ("peeping" testis). The mean interval between the two stages was 5 months (range 3-12 months). All 40 patients had at least two follow-up examinations, at 1 and 6-8 months. Overall success rate was 88% (37/42 testes). Two testes (5%) were proximal to the lower scrotum and underwent a subsequent inguinoscrotal orchidopexy. One testis was smaller at 6 months, compared with intraoperative measurements, and two (5%) were atrophied. There was no correlation between failure and initial location of the testis, but all five unsuccessful cases involved patients younger than 18 months.

Conclusions:

Full laparoscopic approach to the two-stage Fowler-Stephens orchidopexy yields results similar to or better than published series of open orchidopexy. Failure correlated with young age (<18 months) at the time of surgery.

Notes:

P28

TISSUE-ENGINEERED ESOPHAGUS IS A VERSATILE IN VIVO MOUSE MODEL WITH INTACT ARCHITECTURE

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Purpose:

Morbidity from esophageal atresia and esophagectomy is significant. Experimental substitution by onlay patch and interposition of tissue-engineered esophagus has been previously accomplished in a Lewis rat model. Lewis rats have been used for tissue engineering because of their larger size, resilience for successful survival surgeries, and historic precedent; however, investigation into the mechanism of engineered tissue formation has been limited as the molecular and genetic tools are not available. We hypothesized that tissue-engineered esophagus in the mouse could serve as a versatile *in vivo* model with therapeutic potential.

Methods:

Esophagus from 1-week-old non-obese diabetic, severe combined immunodeficiency, gamma chain deficient (NOD/SCID) mice was dissected. Organoid units, multicellular clusters containing both epithelium and mesenchyme, were isolated by a protocol previously established and validated in our laboratory and loaded onto biodegradable polyglycolic acid/poly-L-lactic acid scaffolds. The constructs were then implanted into the omentum of adult NOD/SCID mice. Four weeks later, all implants were harvested and analyzed with histology and immunohistochemistry. Adult native esophagus served as controls.

Results:

Tissue-engineered esophagus architecture replicated that of native esophagus and demonstrated appropriate cellular differentiation. Histology revealed tissue-engineered esophageal mucosa composed of a keratinized stratified squamous epithelium adjacent to a muscularis. Specific cytokeratins (CK) were used to distinguish between cell types. CK14-CK13+CK4+ cells confirmed the presence of differentiated suprabasal cells. CK14+CK13-CK4- cells were identified as proliferative basal cells and costained positive for proliferating cell nuclear antigen (PCNA). In addition, the muscularis stained positive for both alpha smooth muscle actin (SMA) and desmin.

Conclusions:

Tissue-engineered esophagus in the mouse resembles native esophagus and demonstrates appropriate cellular differentiation with an intact epithelium and mesenchyme. Further investigation into its cellular and molecular mechanism of formation is now possible in a versatile *in vivo* mouse model. Delineating the mechanism is an important precursor to future human esophageal replacement therapies.

Notes:

P29

ARGON BEAM COAGULATION NEGATES THE NEED FOR DRAINAGE IN THE EXCISION OF CYSTIC HYGROMAS

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Purpose:

Suction drainage is routinely used to prevent hematoma and seroma formation following the excision of cystic hygromas. However, these drains may be associated with longer hospital stays, patient discomfort, and disfiguring scars. We sought to identify if we could eliminate the need for drainage following the excision of these lymphatic malformations by utilizing intraoperative argon beam coagulation.

Methods:

Eleven patients underwent surgical excision of cystic hygromas in various locations including the axilla, neck, and lower extremity. The argon beam coagulator was used to ablate the entire operative field after the excision of the cystic hygroma in these patients. Wounds were closed without the placement of drains.

Results:

After a mean follow-up of thirty months (range 12-60 months), none of the patients has had wound complications, including seroma or hematoma formation. Additionally, there were no cystic hygroma recurrences. The length of stay in all patients was less than twenty four hours.

Conclusions:

We conclude that intraoperative use of the argon beam coagulator aids in the definitive excision of cystic hygromas possibly by sealing the lymphatics that contribute to seroma formation in addition to aiding in hemostasis thereby eliminating the need for drainage.

Notes:

P30

EXTRAHEPATIC PORTAL VEIN THROMBOSIS AFTER UMBILICAL CATHETERIZATION: IS IT A GOOD CHOICE FOR "REX SHUNT"?

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Purpose:

The management of portal hypertension in children has changed considerably in recent decades. Extrahepatic portal vein thrombosis (EHPVT) is an important cause of portal hypertension in children. Recently, the "Rex shunt" has been used successfully to treat these patients.

Methods:

Report of 19 children, mean age 5 years-old (5 mo to 14 years-old), with extrahepatic portal vein thrombosis (EHPVT), suffering from repeated gastrointestinal bleeding episodes, refractory to endoscopic treatment, that were eligible for a mesenteric-portal surgical shunt ("Rex shunt") with left internal jugular vein autograft. Eight children had idiopathic EHPVT, 9 had post umbilical catheterization EHPVT, one had portal vein agenesis and one had post transplant portal vein thrombosis.

Results:

It was possible to perform "Rex shunt" in all cases of idiopathic EHPVT (8), in 1 patient with post transplant EHPVT, in one case of portal vein agenesis, and in only one of the nine patients with post umbilical catheterization EHPVT (1). Warren surgery was performed in 4 of these last patients, and in one case we performed proximal splenorenal anastomosis because of intractable ascites. Thrombosis of the Rex shunt occurred in one patient with portal vein agenesis and associated cardiopaty, and one patient had stenosis of the left portal vein - jugular vein anastomosis that was successfully treated by percutaneous portography at 4 months after surgery. Current follow-up ranges from 3 to 26 months. Endoscopic status has significantly improved after surgery with resolution of the varices and hypertensive gastropathy. There was no recurrence of bleeding in all these patients until now.

Conclusions:

The mesenteric-portal shunt ("Rex shunt") should be considered in children with idiopathic EHPVT suffering from repeated gastrointestinal bleeding episodes, refractory to endoscopic treatment. Nevertheless, the indication of this kind of surgery in the post umbilical catheterization EHPVT group should be better evaluated.

Notes:

P31

SURGICAL INTERVENTION IN THE SETTING OF PARENTERAL NUTRITION ASSOCIATED CHOLESTASIS MAY EXACERBATE LIVER INJURY

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Purpose:

To compare post-operative direct bilirubin levels in patients with parenteral nutrition (PN)-associated cholestasis to those with resolved cholestasis at the time of a major abdominal operation.

Methods:

Medical records of all patients receiving intravenous fish oil as treatment for PN-associated cholestasis and who underwent a major abdominal operation (laparotomy) between 3/1/07 and 7/1/09 were reviewed from start of treatment to either resolution of cholestasis, PN cessation, or death. Patients who received treatment for < 1 month, had reversal of portal flow or portal vein thrombosis, or another major comorbidity were excluded. Twenty-three patients, who collectively underwent 27 major abdominal operations, were included and analyzed. Of these procedures, 12 occurred prior to resolution of cholestasis (defined as direct bilirubin < 2 mg/dL) and 15 occurred after resolution of cholestasis. Direct bilirubin levels were examined over time in relation to dates of major abdominal procedures.

Results:

Seventy-five percent (9/12) of procedures conducted prior to resolution of cholestasis were associated with an increase in direct bilirubin levels post-operatively. In contrast, only 13% (2/15) of procedures conducted following the resolution of cholestasis were associated with a post-operative direct bilirubin >2 mg/dL (P=0.001; odds ratio 4.47; 95% confidence interval 1.25-16.00).

Conclusions:

Patients who undergo major abdominal surgery while having cholestasis may experience an exacerbation of liver injury, as evidenced by increased direct bilirubin levels, which is less likely to occur in those who undergo such procedures upon resolution of cholestasis through treatment with intravenous fish oil. In the absence of clear clinical indication otherwise, it may be advisable to delay surgical intervention in the setting of PN-associated liver disease in order to avoid additional hepatic injury.

Notes:

P32

EFFECTS OF THE ADMINISTRATION OF PENTOXIFYLLINE AND PREDNISOLONE ON THE EVOLUTION OF PORTAL FIBROGENESIS SECONDARY TO BILIARY OBSTRUCTION - AN EXPERIMENTAL STUDY IN GROWING ANIMALS

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Purpose:

Many chronic liver diseases lead to progressive hepatic fibrosis and the need of liver transplantation. Studies have been demonstrated the possibility of drug modulation of hepatic fibrogenesis. Regarding biliary obstruction, it has been suggested that administration of corticosteroids could promote better late outcomes for biliary atresia children submitted to portoenterostomy. There is no published experimental study examining this issue, and models used to test potential antifibrogenic drugs, such as pentoxifylline, have not included growing animals.

Methods:

119 young rats (21st or 22nd days) were allocated into five groups: 1. CBDL + distilled water; 2. SHAM + distilled water; 3. CBDL + pentoxifylline (PTX); 4. CBDL + prednisolone (PRED); 5. CBDL + pentoxifylline + prednisolone (PTX+PRED). At the end of the study, animals were weighed and a hepatic fragment was collected for analysis. PTX animals exhibited increased weight gain compared to PRED or PTX+PRED groups.

Results:

Animals from the three therapeutic groups showed diminished collagen-filled area in portal spaces. Total portal space area was increased in PTX group.

Conclusions:
Hepatic fibrosis induced by bile duct ligation in young rats could be modulated by pharmacologic interventions. Administration of pentoxifylline and prednisolone, or the combination of both, resulted in diminished collagen-filled area in portal spaces.

Notes:

P33

LONG-TERM OUTCOME IN CHILDREN AFTER RESECTION OF CONGENITAL CYSTIC ADENOMATOID MALFORMATION (CCAM)

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Purpose:

CCAMs are lung lesions that pose significant risks without surgical excision, but the long-term outcome of patients who do undergo resection, particularly the incidence of infectious complications, chest wall and spinal deformities remains largely unknown. At our institution, we have established multidisciplinary Long-term Infant-to-adult Follow-up and Evaluation (LIFE) clinics to prospectively follow children with surgically correctable congenital anomalies such as CCAM. We present the data from the first three years of CCAM LIFE clinic.

Methods:

With IRB approval, we prospectively documented the long-term clinical characteristics of a cohort of children after postnatal CCAM resection between June 1998 and November 2007. At each visit, a complete evaluation, chest x-ray, and history are obtained. Parents complete a questionnaire documenting general, nutritional, and respiratory history.

Results:

Thirteen patients with CCAM have undergone comprehensive evaluation by the LIFE clinic. Twelve patients (92%) were diagnosed prenatally and born near term (38.7 ±2.1 wks). Seven (54%) thoracoscopic resections and six (46%) open thoracotomy resections were performed (mean age of 8.4 ±5.9 months). Pathology demonstrated seven macrocystic and six predominately microcystic lesions. Mean age at first LIFE clinic visit was 3.6 ±2.8 years and mean follow-up period was 3.84 (±2.9) years after resection. Mean O2 saturation was 98.3 (±1.4) % on room air at rest. Postoperative complication rates are summarized in Table 1.

Conclusions:

Initial results from our LIFE clinic indicate that although patients who undergo CCAM resection grow normally and have normal resting oxygenation; this patient population may have a high incidence of lower respiratory tract infections and asthma. Further investigation is ongoing.

Table 1. Long-term complication rates following resection of CCAM, n=13.

Postop follow-up (yrs)	Lower respiratory tract infections	Hospital/ED visits for respiratory symptoms	Asthma requiring medication	Pectus	Scoliosis	Auditory, speech or visual deficits
3.84 (+/- 2.9)	4 (31%)	3 (23%)	6 (46%)	1 (8%)	1 (8%)	4 (31%)

Notes:

PODIUM

1 – 3 minutes

A MULTI-INSTITUTIONAL COMPARISON OF PEDIATRIC APPENDICITIS OUTCOMES BETWEEN TEACHING AND NONTEACHING HOSPITALS

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Purpose:

In this era of heightened emphasis on patient outcomes, it is important to document the effect of residents acting as the surgeon for a surgical procedure. This study compares the outcomes of appendicitis between teaching and nonteaching institutions.

Methods:

A retrospective review from 1998-2007 was performed. The outcomes from two teaching institutions (each with its own General Surgery residency program) were compared to 11 nonteaching institutions. Study outcomes included postoperative morbidity and length of hospitalization (LOH). Data were analyzed using Wilcoxon rank-sum test and chi-squared analysis.

Results:

1472 patients were treated at the teaching institutions (mean age= 9.8 years, male= 63%) and 6431 at the nonteaching institutions (mean age= 10.8 years, male=62%). The perforated appendicitis rate was 37% at the teaching institutiosn and 30% at the nonteaching institutions (p <0.0001). Morbidity data are summarized in the table. LOH was similar for nonperforated (Teaching = 1.8±1.5 d, Nonteaching = 1.8±2.9, p=0.8) and perforated (Teaching = 5.8±3.3 d, Nonteaching = 5.5±3.4, p=0.07). Use of laparoscopy was higher in the nonteaching institutions for nonperforated appendicitis (Teaching=39%, Nonteaching=52%, p<0.0001). However, use of laparoscopy was similar for perforated appendicitis (Teaching=33%, Nonteaching=35%, p=0.5).

Conclusions:

Postoperative morbidity was similar in children with non-perforated appendicitis and lower in children with perforated appendicitis at teaching institutions. LOH was similar between teaching and nonteaching institutions. Overall, the presence of surgical trainees had no adverse impact on the quality of care for children with appendicitis.

Appendicitis Outcomes Between Teaching and Nonteaching Hospitals

	Teaching	Nonteaching	p
Non-perforated appendicitis (n)	935	4530	
Wound Infection	1.8%	2.0%	0.8
Abscess	0.1%	0.6%	0.09
Readmission	1.4%	2.3%	0.1
Perforated appendicitis (n)	537	1901	
Wound Infection	5.2%	8.2%	0.03
Abscess	9.3%	11.0%	0.3
Readmission	5.6%	9.7%	0.004

Notes:

2 – 3 minutes

PROSPECTIVE, RANDOMIZED TRIAL ASSESSING QUALITY OF LIFE IN PATIENTS WITH RUPTURED APPENDICITIS

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Purpose:

Ruptured appendicitis (RA) in children is commonly treated with either an early appendectomy (EA) or intravenous antibiotics and interval appendectomy (IA). Though either option treats the pathology of RA, the effect on the health and wellness of the child may differ; therefore, quality of life (QOL) was measured to determine if EA or IA was associated with improved physical or psychosocial wellness.

Methods:

A randomized, controlled trial was conducted comparing EA with IA with QOL as a secondary outcome. QOL was assessed utilizing a validated, parent-reported questionnaire (SF-10), at admission, discharge, one month after diagnosis and one month after completion of treatment. The SF-10 yields a physical (PHS) and psychosocial score (PSS). Normally distributed data were compared with a t-test; nonparametric data were analyzed with a Wilcoxon-rank sum test. The Institutional Review Board approved this study.

Results:

One hundred thirty patients with RA were randomized to either EA (n=64) or IA (n=66). One hundred eleven patients (89%) completed all four SF-10 forms. Median PHS on admission was 42.7 for both groups indicating that RA had a substantial impact on physical well-being (normal >50). Median PSS was slightly higher (EA-50.47, IA-53.9). Patients undergoing EA reported less physical impact compared with IA based on the change in PHS from admission to discharge (-6.0+/-2.5 vs -14.2+/-2.4, p=0.02); from admission to follow up (7.1+/-2.8 vs 0.38+/-2.7, p=0.09); and from admission to completion of therapy (16.7+/-2.3 vs 8.4+/-2.2, p=0.01). Changes in PSS from admission to each time point were also better in EA patients although of marginal statistical significance (p=0.05, 0.05, 0.03).

Conclusion:

When comparing change in QOL from admission scores, EA had less of a physical and psychosocial impact on patients at discharge, one month after diagnosis and at completion of therapy compared to IA.

Notes:

3 – 3 minutes

GLOWING IN THE DARK: TIME OF DAY AS A DETERMINANT OF RADIOGRAPHIC IMAGING IN THE EVALUATION OFABDOMINAL PAIN IN CHILDREN

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Purpose:

Radiology studies are commonly used to evaluate abdominal pain in children. Because ultrasound does not expose patients to ionizing radiation, it is often the preferred initial pediatric imaging study. Many institutions lack overnight ultrasound services and computerized tomography (CT) scanning becomes the only option. The purpose of this study is to quantify the difference in daytime and nighttime patterns of initial use of ultrasound or CT to evaluate pediatric abdominal pain and to measure consequent differences in radiation exposure and cost.

Methods:

We compared the daytime and nighttime use of ultrasound and CT for patients seen for abdominal pain by the Pediatric Surgery Service from January 2008 to July 2009 at a tertiary care center by performing a retrospective chart review of patients identified through the service's billing records (n=80). Day was defined as 7:30 AM to 5:30 PM. This study was exempt from institutional review board approval.

Results:

Patient age ranged from 8 months to 17 years (average, 9.8 years); 51% were female. The three most common final diagnoses were gastroenteritis, appendicitis, and constipation. During the day, the number of ultrasounds was almost 7 times greater than the number of CTs (47 vs. 7); at night, almost 3 times as many CTs were performed (ultrasound 7 vs. CT 19). The daytime average radiation dose per child was significantly lower than the average dose at night (day 0.52 mSv/patient, night 2.9 mSv/patient); the average cost per patient for radiological tests was lower for daytime patients ($2484 day vs. $4162 night) (all $p<0.05$).

Conclusions:

Dependence on computerized tomography scanning at night for evaluation of children with abdominal complaints results in higher average radiation exposure and cost per patient. Twenty-four hour ultrasound availability would decrease the average radiation dose and the cost for initial evaluation of children presenting with abdominal pain.

Notes:

4 – 3 minutes

PREOPERATIVE MECHANICAL BOWEL PREPARATIONS FOR PEDIATRIC GASTROINTESTINAL SURGERY: IS IT NECESSARY?

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Purpose:

In the adult surgery literature, there has been no data to date demonstrating benefits to mechanical bowel preparation (MBP) prior to gastrointestinal surgery (GI). However, there is a paucity of data relating to necessity of MBP prior to GI surgery in children. The present study aims to provide additional data.

Methods:

Following Institutional Review Board approval, charts from 1996 to 2009 were retrospectively analyzed from our institution. Inclusion criteria were patients who underwent GI surgery in ages 1-18. NICU and trauma patients were excluded. Primary outcomes were length of stay from OR date to discharge date, anastomotic leak, and infection. Comparisons were made between patients with MBP of all types, versus patients without MBP. Unadjusted comparison was made with t tests and chi square analysis. Multivariate regression analyses were performed adjusting for age.

Results:

A total of 90 patients were studied, with 86 with known MBP status. Among them, 47 (54.7%) had MBP. Mean (median) length of stay was 10.6 (6). One patient developed a leak (1.1%), and 7 patients developed infections (7.9%). On unadjusted analysis, the difference in LOS between MBP and non-MBP patients was not significant (13.2 vs 7.97, p=0.176), as well as rates of leaks (0.0% vs 2.6%, p=0.275) and infection (6.5% vs 7.7%, p=0.834). On multivariate analysis, MBP was also not significantly associated with LOS, leaks, or infections.

Conclusions:

The present study demonstrates no evidence that bowel preparations will reduce lengths of stay, anastomotic leaks, or infections following gastrointestinal surgery in children.

Outcomes with and without Mechanical Bowel Preparation in Pediatric Gastrointestinal Surgery

	MBP		Non-MBP		p value
n	46		39		
Age, mean (SD)	7.87	(7.52)	8.62	(7.28)	0.645
Lengths of stay, mean (SD)	13.20	(23.14)	7.97	(6.29)	0.176
Anastomotic leaks	0	0.0%	1	2.6%	0.275
Infections	3	6.5%	3	7.7%	0.834

Notes:

5 – 3 minutes

RESTORATIVE PROCTOCOLECTOMY WITH AND WITHOUT PROTECTIVE ILEOSTOMY IN A PEDIATRIC POPULATION

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Purpose:

A 19-year retrospective review of pediatric patients following restorative proctocolectomy (RP) with or without a protective ileostomy in the treatment of ulcerative colitis and polyposis syndromes.

Methods:

IRB approved data collection from hospital and office records. All patients had rectal mucosectomy with ileal pouch reservoir and hand-sewn ileal pouch anal anastomosis (IPAA). Group comparison by Fisher's exact test.

Results:

83 patients with ulcerative colitis (UC) and 7 patients with polyposis syndromes (age 2.0 to 21.8 years; mean 13.9 years) underwent restorative proctocolectomy over a 19-year period. 68 patients underwent IPAA without diverting ileostomy (56 patients underwent restorative proctocolectomy as single-stage procedures; 12 had abdominal colectomy and subsequent definitive IPAA without diverting ileostomy). 19 patients had IPAA with diverting ileostomy and subsequent closure of ileostomy. Three-stage procedures were performed in only 3 cases. Either S- or J-pouches were used at surgeon's discretion. There was no mortality in the series. An ileal pouch leak or pelvic abscess was identified in 2. Surgical pouch revision for retraction, efferent limb syndrome, prolapse, pouchitis or perirectal infections occurred in 18 (5/62 J-pouch, 13/28 S-pouch; p=0.0001). 14 patients (5/22 with diversion; 9/68 without diversion) developed small bowel obstruction (p=0.317). Overall, daytime and nighttime continence is excellent, and frequency ranges from 2 to 6 bowel movements per day with rare patients having nocturnal evacuations. Severe pouchitis, defined by chronicity of symptoms and/or recurrent perianal infections similar to Crohn's disease, has been identified in 8/19 UC patients after diverting ileostomy and 12/64 UC patients without protective ileostomy (p=0.063). 2 patients currently have an ileostomy because of chronic pouch-related problems. Pouchitis was not seen in the 7 polyposis patients.

Conclusions:

Restorative proctocolectomy without protective ileostomy is not associated with an increased morbidity, even in patients with active colitis, and may be appropriate for the majority of patients.

Notes:

6 – 3 minutes

OMEGA-3 FATTY ACID AND GROWTH FACTOR MANIPULATION OF INTESTINAL ANGIOGENESIS: A NOVEL APPROACH TO THE MANAGEMENT OF INFLAMMATORY BOWEL DISEASE

Keith A. Thatch, MD[1], Michael S. Katz, MD[1], Marian M. Haber, MD[2], Marshall Z. Schwartz, MD[1]
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Purpose:

Transgenic HLA-B27 Fischer rats demonstrate phenotypic changes similar to inflammatory bowel disease (IBD). Utilizing this animal model, we previously demonstrated the potential of HGF in ameliorating diarrhea, decreasing inflammation and stimulating non-pathologic angiogenesis. In this study we assess the effect of omega-3 fatty acid enriched (50% of total fat) feeds (Ω3 feed) with and without HGF supplementation on favorable neovascularization, cytokine production and inflammatory cell infiltration in this IBD model.

Methods:

Female transgenic HLA-B27 Fischer rats were divided into four groups. Saline or HGF was delivered via jugular vein by 14-day osmotic mini-pumps. **Group 1**: normal feed and saline (control, N=5); **Group 2**: Ω3 feed and saline (N=5); **Group 3**: normal feed and HGF (150 µg/kg/day, N=5); **Group 4**: Ω3 feed and HGF (N=5). Intestinal microvascular density (MVD), inflammatory cell infiltration, and inflammatory cytokine expression (TNF–α, IFN-γ, and IL-2) were assessed. Analysis of variance (ANOVA) was used to determine statistical significance.

Results:

Administration of Ω3 feed with HGF resulted in a significant reduction of ileal and colonic inflammatory cell infiltration ($p<0.05$), decreases in TNF-□, IFN-□, and IL-2 expression ($p<0.05$) and increase in MVD ($p<0.02$) in comparison to control animals. Ω3 feeds alone were not significant in reducing inflammatory cell infiltration, inflammatory cytokine expression and/or MVD. HGF alone significantly reduced inflammatory cell infiltration ($p<0.05$), was mixed in reducing inflammatory cytokine expression (TNF-□; $p>0.05$, IFN-□; $p<0.05$, IL-2; $p<0.05$) and had a significant increase in MVD ($p<0.05$).

Conclusion:

HGF provided the greatest response to the parameters measured in this IBD model where as Ω3 alone was not beneficial with any of the parameters measured. However, in combination there was a synergistic effect in modulating the intestinal inflammatory response. This is the first demonstration that Ω3 lipid dietary supplementation can synergistically enhance the growth factor (HGF) effects on non-pathologic angiogenesis and reduce inflammation parameters.

Notes:

7 – 3 minutes

RE-DILATION OF BOWEL AFTER INTESTINAL LENGTHENING PROCEDURES

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Purpose:
There are limited data on the long-term complications of patients undergoing a Bianchi or stepwise transverse enteroplasty (STEP) procedure. The aim of the study was to investigate such complications.

Methods:
This is a 15 year, single institution, retrospective chart review of patients who underwent an intestinal lengthening procedure (ILP). Re-dilation of bowel, length of bowel, ILP-related complications, their interval to development, and need for further procedures were analyzed. Comparisons were made using the unpaired t-test and Fisher's exact test.

Results:
One hundred and nineteen patients with SBS were identified, of which 13 had undergone an ILP. Eight patients had a STEP, and 6 patients had a Bianchi. One patient had a Bianchi followed by a STEP, two patients had two STEPs done. Mean follow-up length was 4 years (range 1-11.4 years) in the STEP group and 7.4 years (range 0.7-13 years) in the Bianchi group. Patients symptomatically re-dilated their small bowel in 9 out of 16 (56%) ILPs, (Table). Mean bowel diameter after ILP was 1.9 cm (range 1.5-2.75cm), and this re-dilated to a mean of 4.4cm (3.5-6.1cm) (p= 0.04). Other complications following re-dilation, included bacterial overgrowth (9/9), re-operation (5/9), gastrointestinal bleeding (4/9), adhesive small bowel obstruction (2/9), and stricturing (1/9). Although not significant, children who re-dilated tended to have shorter lengths of bowel.

Conclusions:
Re-dilation of lengthened bowel is common after ILP, and associated with worse outcomes, including a trend towards increased bacterial overgrowth, and a lower likelihood of weaning off PN. Future work will need to concentrate on the mechanisms which promote this re-dilation.

Outcomes after re-dilation

	Re-dilated (N=9)	No re-dilation (N=7)	p-value
STEP	67%	57%	0.75
Age at operation (years)	2.3±3.7	4.3±5.3	0.39
Time to re-dilation (years)	2.5±2.6	N/A	N/A
Bowel length (cm)	61±9	89±17	0.14
Number weaned off PN	11%	57%	0.11
Deaths	11%	14%	1.00
Bacterial Overgrowth	100%	43%	0.02
Gastrointestinal Bleed	44%	57%	1.00
Data shown as percentage or mean ± standard deviation. STEP - stepwise transverse enteroplasty; PN - parenteral nutrition			

Notes:

8 – 3 minutes

TRANSPLANTATION OF NEURAL CREST PROGENITOR CELLS INTO MURINE AGANGLIONIC RECTUM

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Purpose:

Prior studies demonstrated that neural crest progenitor (NCP) cells could be transplanted into normal rectum and survived for up to 35 days. Transplanted cells also differentiated and migrated in vivo. The objective of this study is to examine the fate of the NCP cells in the setting of aganglionic rectum.

Methods:

NCP cells were isolated from intestinal tissue of murine fetuses that expressed the green fluorescent protein (GFP). The cells were cultured in neuro-selective media for 3 days prior to transplantation. 250,000 cells, mixed with India ink for further identification, were injected transanally into the distal rectum of mice with aganglionosis of the rectum. Animals were euthanized at various post-injection time points, and the rectums were retrieved for analyses.

Results:

Following IRB approval, cells were successfully cultured prior to transplantation. By day 3, cultured cells were found to be forming clusters and had developed axonal-like projections in-vitro. All specimens demonstrated submucosal or subserosal deposition of cells in the distal rectum, and none of the animals died prior to the predetermined time points. Immunofluorescent staining demonstrated the presence of cells that expressed peripherin, a marker for neuronal cells, and S100, a marker for glial cells, in the area of injection up to day 35. Peripherin and S100 immunofluorescence in the region of injections decreased with time, although specimens from later time points (>21 days) demonstrated cells that expressed GFP and peripherin or S100 within the intestinal muscle layers and away from the sites of injection. Cells from earlier time points also stained positive for ki-67, a marker of proliferation.

Conclusions:

NCP cells can be successfully harvested, cultured, and transplanted into aganglionic rectum. Furthermore, transplanted cells staining for neural and glial markers persist to at least 35 days post injection. Whether these transplanted cells can form a functional enteric nervous system remains to be determined.

Notes:

9 – 3 minutes

INTRASPHINCTERIC BOTULINUM TOXIN DECREASES RATE OF HOSPITALIZATION FOR POST-OPERATIVE OBSTRUCTIVE SYMPTOMS IN CHILDREN WITH HIRSCHSPRUNG'S DISEASE

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Purpose:

Although most children with Hirschsprung's disease do well after pull-through surgery, some continue to have persistent obstructive symptoms that may lead to significant morbidity. Intractable symptoms traditionally have been treated with anal myectomy, which may be ineffective or complicated by long-term incontinence. The purpose of this study was to evaluate the effect of intrasphincteric botulinum toxin in the management of these children.

Methods:

Retrospective review of patients with Hirschsprung's disease treated at one medical center over a period of 10 years. Statistical analysis was done using the Wilcoxon's matched pairs test for continous variables.

Results:

Twenty-two patients received botulinum toxin: 17 males, mean age 8.4 years. Transition zone was rectosigmoid in 12 and long segment in 10. All patients had previously undergone pull-through surgery: Duhamel in 13 and Soave in 9. Patients received a median number of 2 botulinum toxin injections (range 1 to 23). The number of hospitalizations for obstructive symptoms significantly decreased from pre-injection (median 1.5, IQR 1-3) to post-injection (median 0, IQR 0-1), p=0.0003. The number of injections was lower in children with a rectosigmoid transition zone (median of 1 injection (IQR 1-3.5) than in those with long segment disease (median 3, IQR 1-15), p=0.04. 80% of patients had a good response to the first dose of botulinum toxin, 69% of them required additional injections. There were no short-term or long-term complications related to botulinum toxin.

Conclusions:

Intrasphincteric botulinum toxin significantly decreased the need for obstruction-related hospitalization in children who had undergone pull-through surgery for Hirschsprung's disease. Patients with long segment disease required more injections than those with rectosigmoid disease. Botulinum toxin is safe and effective, and should be strongly considered in the management algorithm for post-operative obstructive symptoms in children with Hirschsprung'a disease.

Notes:

10 – 3 minutes

HEPATICODUODENOSTOMY VERSUS HEPATICOJEJUNOSTOMY FOR RECONSTRUCTION AFTER RESECTION OF CHOLEDOCHAL CYST

Matthew T. Santore, MD, Brittany J. Behar, BA, Thane A. Blinman, MD, Edward J. Doolin, MD, Holly L. Hedrick, Peter Mattei, MD, Michael L. Nance, MD, N. Scott Adzick, MD, Alan W. Flake, MD
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Purpose:

Roux-en-Y hepaticojejunostomy (HJ) is currently the favored reconstructive procedure after resection of choledochal cysts. Hepaticoduodenostomy(HD) has been argued to be more physiologic and technically easier, but is feared to increase risks of cholangitis and bile gastritis. However, these risks have not been demonstrated in the literature. Here, we compare outcomes of the two procedures.

Methods:

A retrospective chart review identified 54 patients that underwent choledochal cyst resection within our institution from 1997 to 2009. Patients underwent HD or HJ according to surgeon preference. Demographic and outcome data were compared using two-tailed t-tests, and relative risk (RR).

Results:

Fifty four patients had repair of choledochal cyst. Biliary continuity was restored by HD in 33 (61%) and by HJ in 21 (39%). The two groups did not differ in terms of age at presentation, sex, or presenting symptoms. Excluding the five laparoscopic HD cases that were performed, HD required less total operative time than HJ (4.1 hours versus 5.2 hours, p=0.053). The HD group tolerated a diet faster (3.1 days compared to 5.6 days, p=0.04) but had similar hospital stay (8.1 days for HD vs.8.7 days for HJ, p=0.74). Complications were more common in HJ (HD = 15%, HJ = 23.8%, p =0.38, RR = 0.63 for HD). Three patients required reoperation (bile leak, stricture, ventral hernia) after HJ, but only one patient required reoperation after HD for a stricture (HD=3.0%, HJ=13.6%, p=0.11). Cholangitis occurred only once within this study, within the HJ group (HD 0% vs. HJ 3%, p=0.19).

Conclusion:

Hepaticoduodenostomy does not increase the risk of cholangitis and is less risky overall. In this series, Hepaticoduodenostomy required less operative time, allowed faster recovery of bowel function, and produced fewer complications leading to reoperation.

Notes:

11 – 3 minutes

LAPAROSCOPIC ADJUSTABLE GASTRIC BANDING IN ADOLESCENTS: SHORT-TERM RESULTS

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Purpose:

Childhood obesity has become recognized as an epidemic. Children and adolescents who fail to lose weight through diet and exercise programs have been offered weight loss surgery for several years. Our institution has performed laparoscopic adjustable gastric banding (LAGB) on 100 teenagers to date under an IRB-approved protocol and we report of our early results.

Methods:

Candidates for surgery were referred to our program. Enrollees underwent rigorous assessment for medical eligibility, compliance, and psychological well-being. Patients who met criteria and were approved by our team were offered LAGB. Post-operatively patients were followed monthly (minimum) until steady weight loss was achieved, then every 3 months thereafter. Comprehensive medical assessment is made every 6 months post-operatively.

Results:

100 patients ages 14-19 years have undergone LAGB. Pre-operative average weight is 305.0lbs and median body mass index (BMI) is 48.0. Five re-operations have taken place: port site bleeding, hiatal hernia repair, possible intestinal obstruction, and port slippage (2). 70 patients have been followed for at least 6 months. Average weight loss at 6 months was 27.7 lbs (range 58.0 to -4.5) and average change in BMI was 3.9 (range 11.8 t- -5.6). Co-morbid conditions such as hypertension, type II diabetes, and metabolic syndrome were uncommon.

Conclusion:

LAGB may be performed safely in adolescents and short term results suggest that LAGB may serve as an important tool to help them lose weight. Treatment of obesity in children and adolescents may ward off serious obesity-related co-morbidities.

Notes:

12 – 3 minutes

THE PREDICTIVE VALUE OF PREOPERATIVE PET-CT SCANS IN CHILDREN WITH CONGENITAL HYPERINSULINISM OF INFANCY

Augusto Zani, MD, Shireen Nah, MD, Ori Ron, MD, Virpi Smith, MD, Michael Ashworth, MD, Simon Eaton, PhD, Paolo De Coppi, PhD, Khalid Hussain, MD, Agostino Pierro, MD
Great Ormond Street Hospital for Children - University College London Institute of Child Health, London, United Kingdom

Abstract Body:

Purpose:

In Congenital Hyperinsulinism of Infancy (CHI), the use of preoperative ^{18}Fluoro-L-DOPA-PET-CT scan has recently been reported. The aim of this study was to evaluate the predictive value and accuracy of this technique in the discrimination between diffuse and focal pancreatic lesions and the anatomical localization of the focal lesions.

Methods:

Institutional ethical approval was obtained (09SG02). At our centre, between 2006 and 2009, PET-CT scan was done in 16 children with CHI (median age 2.5 months, range 2-11 months) who were not responding to medical therapy and underwent laparoscopic or open surgery. The results of the PET-CT scan were correlated with histological findings.

Results:

Diffuse disease: In 6 children the PET-CT scan indicated the presence of diffuse uptake in the pancreas supporting the genetic suspicion of diffuse disease. This was confirmed in all cases at frozen section and definitive histology.
Focal disease: In 10 children the PET-CT scan indicated focal uptake in the pancreas making a diagnosis of focal disease and localizing the lesion. These findings also corresponded to histology in all cases. During surgery frozen section was done, if the lesion was not present or not completely excised, dissection was continued. In 1 child the PET-CT scan was not accurate in defining the site of the focal lesion: the PET-CT scan indicated a focal lesion in the tail, but a subtotal laparoscopic pancreatectomy was required as the lesion was in the head.

Conclusions:

PET-CT scan discriminates between diffuse and focal forms of CHI with a sensitivity and specificity of 100%. In focal forms, the PET-CT scan is useful in the majority of patients in defining the site of the focal lesion. However, intraoperative histological confirmation of complete focal lesion resection is needed.

Notes:

13 – 3 minutes

SUCCESSFUL OUTCOME WITH ORGAN DONATION AND TRANSPLANTION FOLLOWING CARDIAC ARREST IN PEDIATRIC TRAUMA

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Purpose:

Previous studies indicate poor outcomes for pediatric trauma patients requiring cardiopulmonary resuscitation (CPR) following injury. Despite this knowledge, extensive resuscitation routinely continues in this population. The purpose of our study was to determine the percent survival of pediatric trauma patients requiring pre-hospital or trauma bay CPR, determine the proportion of organ donation among non-survivors, and to evaluate transplant outcomes from donors with a history of cardiac arrest.

Methods:

Children (age<18) who underwent pre-hospital or trauma bay CPR following blunt or penetrating trauma were identified from the trauma registry of a regional pediatric trauma center over a 10-year period (1998 to 2008). 53 children were identified who required CPR following both blunt and penetrating trauma. Mechanism of injury included passenger in an MVC (30.2%), MVC versus pedestrian or bicycle (30.2%), firearm injury (17.0%), non-accidental trauma or assault (14%), stabbing (5.7%), and falls (1.9%).

Results:

Overall survival was not impacted by mechanism of injury (p-value = 0.67). Death occurred in 45 children (84.5%), though eight children survived (15.1%). Of the children who died, organ, corneal or cardiac valve donation occurred in twelve (26.7%). All organ donors were victims of blunt trauma. Among donors, liver (41.7%), pancreas (33.3%), small bowel (25%), kidney (25%), lung (16.7%), and heart (33.3%) were transplanted. Graft survival at 1 month and 1 year was 93.3%.

Conclusions:

Pediatric trauma patients requiring CPR remain at high risk for death following blunt or penetrating trauma, though survival among our study population was higher than that previously reported in the literature. Improved patient survival, substantial rates of organ donation and excellent graft survival support continued aggressive resuscitation among pediatric trauma patients following cardiac arrest.

Notes:

14 – 3 minutes

SIGNIFICANCE OF FIRST RIB FRACTURES IN BLUNT PEDIATRIC THORACIC TRAUMA

Nicholas A. Hamilton, Brian T. Bucher, MD, Martin S. Keller, MD
Washington University, St Louis, MO, USA

Purpose:

To determine if first rib fractures are associated with an increased incidence of thoracic vascular injury in pediatric patients.

Methods:

Following IRB approval, the medical records of all children diagnosed with a first rib fracture or a central vascular injury treated at a state-designated level 1 pediatric trauma hospital from 2000-2008 were reviewed.

Results:

Thirty children (0.26% of patients; mean age: 10.6+/-5 years) were identified with 1st rib fractures or thoracic vascular injury due to blunt trauma. Twenty-nine children had 1st rib fractures. Ninety-seven percent had a fracture without cardiac or thoracic vascular injury. Two children were identified with thoracic vascular injuries; one (3%) had a first rib fracture with associated aortic dissection and the other had an aortic transection without rib fracture. Mediastinal abnormalities (indistinct aortic knob) were identified in 3 children, two with 1st rib fracture on initial chest radiograph. One child had a mediastinal hematoma without cardiovascular injury. Location and displacement of the 1st rib fracture was not predictive of associated injuries. Despite normal cardiovascular exams, 74% of children with a normal mediastinum on screening chest radiograph underwent additional imaging with computed tomography solely due to the presence of the 1st rib fracture. None of these patients were found to have associated intrathoracic injuries requiring further intervention. In children with first rib fractures and a normal mediastinum by screening chest x-ray, the negative predictive value of thoracic vascular injury was 100%.

Conclusions:

Children with first rib fractures without mediastinal abnormality on chest radiograph require no further workup for thoracic vascular injury.

Thoracic Vascular Injury in Patients with First Rib Fractures

	Aortic Injury	No Aortic Injury
Normal Mediastinum	0	27
Abnormal Mediastinum	1	1

Notes:

15 – 3 minutes

PARTIAL SPLENECTOMY FOR HEREDITARY SPHEROCYTOSIS: A MULTI-INSTITUTIONAL REVIEW

Keely L. Buesing, MD[1], Elisabeth T. Tracy, MD[2], Colleen Kiernan[3], Aimee Pastor, RN[4], Laura Cassidy, PhD, MS[1], J. Paul Scott, MD[1], Russell E. Ware, MD, PhD[5], Andrew M. Davidoff, MD[5], Frederick J. Rescorla, MD[3], Jacob C. Langer, MD[4], Henry E. Rice, MD[2], Keith T. Oldham, MD[1]
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Purpose:

Partial splenectomy has emerged as a surgical option for selected children with hereditary spherocytosis, with the goal of reducing anemia while preserving splenic function. However, a lack of large clinical studies has limited our understanding of this procedure. This multi-institutional study is the largest series to date to compile outcome data for partial splenectomy for hereditary spherocytosis.

Methods:

Clinical, laboratory and operative data were collected retrospectively after IRB approval from five North American pediatric hospitals with 62 children who underwent partial splenectomy for hereditary spherocytosis between 1990 and 2008. Statistical analysis was performed using paired t-tests, with significance defined as $p<0.05$.

Results:

Patient age ranged from 2 months to 17 years. Mean followup was 36 months (range 1-144 months). At one year following partial splenectomy, mean hemoglobin increased by 3.0 ± 1.4 g/dL ($p<0.0001$, n=27), reticulocytes decreased by 6.6 ± 6.6% ($p=0.0005$, n=18), and bilirubin levels decreased by 1.3 ± 0.9 mg/dL ($p=0.0007$, n=12). These effects were largely sustained throughout followup. Incidence of blood transfusions decreased from 30/54 patients (55.6%) preoperatively to 4/54 patients (7.4%) postoperatively. Patients with poor or transient hematologic response were found to have significantly increased splenic regeneration postoperatively compared to patients with a durable clinical response (maximal dimension 9.0 ± 3.4 cm vs. 6.3 ± 2.2 cm, $p<0.05$). Concurrent or deferred cholecystectomy was performed in 24/53 patients (45.3%). Clinically significant recurrence of anemia or abdominal pain led to a completion splenectomy rate of 4.84%. No patients developed systemic sepsis.

Conclusions:

Our multi-institutional review confirms earlier pilot studies, and suggests that partial splenectomy for hereditary spherocytosis leads to sustained reduction in hematologic signs and symptoms. Relatively few patients require later completion splenectomy. Our review of long-term followup data indicate that splenic regeneration may correlate with recurrent symptoms. Our data support partial splenectomy for select children with hereditary spherocytosis.

Notes:

16 – 3 minutes

PROTOCOLIZED MANAGEMENT OF INFANTS WITH CONGENITAL DIAPHRAGMATIC HERNIA: EFFECT ON SURVIVAL

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Purpose:

With the goals of standardizing care for our patients with congenital diaphragmatic hernia (CDH) and improving outcomes in all CDH patients, we introduced a new protocol for CDH management in 2006. Features of the new protocol include nitric oxide in the delivery room, gentle ventilation, lower criteria for extracorporeal membrane oxygenation (ECMO), and operative repair on ECMO after improvement in lung function and pulmonary hypertension. The purpose of this study was to assess outcomes after institution of this protocol and to compare results with historical controls.

Methods:

Following IRB approval, charts were reviewed of all newborns admitted to a large, metropolitan children's hospital from 2002-2009 with CDH diagnosis. Data recorded included delivery details, duration of ECMO, operative details, length of stay, co-morbidities/anomalies, complications, and survival. Outcomes of protocolized patients were compared to those from the pre-protocol era, as well as to data from the national CDH registry. Statistical analysis was performed using Student's t-test and Fischer's exact test, $P<0.05$ significant.

Results:

Comparison of the protocolized group (n=43) to the historical group (n=51) revealed no significant differences in gestational age, birth weight, APGAR scores, or comorbidities. 79% of protocolized patients were inborn (69% pre-protocol), and 93% underwent CDH repair (82% pre-protocol). Our new treatment strategies augmented use of ECMO (20% pre-protocol, 51% post-protocol) and substantially improved survival to discharge (67% pre-protocol, 88% post-protocol). Among patients treated with ECMO, survival increased to 82% (20% pre-protocol).

Conclusions:

Our new protocol significantly improved survival to discharge for newborns with CDH. We conclude that institution of such a protocol is valuable in improving outcomes for patients with CDH, and could be adopted nationally.

Comparison of CDH Patients in National Registry, Pre-Protocol, and Post-Protocol

	CDH Registry	Pre-Protocol	Post-Protocol	P-value for Pre- vs Post-Protocol
N	>4000	51	43	
% Treated with ECMO	35%	20%	51%	0.0020
% Survival to Discharge if ECMO	48%	20%	82%	0.0015
% Overall Survival to Discharge	68%	67%	88%	0.0154

Notes:

17 – 3 minutes

MID TERM FOLLOW-UP IN HIGH RISK CDH SURVIVORS: PATCHING THE DIAPHRAGM AFFECTS THE OUTCOME.

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Purpose:

The increased survival rate reached in congenital diaphragmatic hernia (CDH) infants has shown a concomitant increase in late morbidity. A recent report from CDH Study Group has showed that dimension of diaphragmatic defect is the only independent risk factor related to mortality. However it is not clear if the defect size may also influence late morbidity. Aim of the present study is to evaluate the influence of patch repair (proxy of diaphragmatic defects size) on mid term morbidity.

Methods:
All high risk (prenatal diagnosis and/or respiratory symptoms within 6 hours of life) CDH survivors, and treated at our Institution between 2004 to 2008, were followed up in a multidisciplinary outpatient clinic as part of a longitudinal prospective study. Auxological, gastro-esophageal reflux (GER), pulmonary (PFT) and orthopedic evaluations were determined at specific time point: 6, 12, and 24 months of age. Patients were divided on regard of patch repair. Unpaired t test and Fisher's exact test were used as appropriate. $P<0.05$ was consider significant.

Results:

61 out of 70 survivors (87%) were enrolled and prospectively evaluated in follow-up. Table summarizes main results.

Conclusions:

Our data suggest that patch repair correlates with higher morbidity at mid term follow-up. Poorer auxological outcome, increased rate of gastro-esophageal reflux, and altered PFT were observed during follow-up. Conversely patch repair doesn't increase chest wall deformities. Long-term evaluation is needed to better evaluate the entity of late sequelae in CDH survivors. Further studies are needed to define if patch repair is an indirect marker of more severe disease or patch repair itself is able to modify the prognosis of CDH survivors.

	Weight z score (median)	Lenght z score (median)	BMI z score (median)	GER %	Altered PFT %	Chest wall deformities %
6 mo patch-tot 46	-1,3 (-2,5;2,6)	-0,1 (-2,5;2,4)	-1,3 (-3,8;1,2)	19(41)	18 (39)	5 (11)
6 mo patch+ tot 15	-2,9 (-4,1;0,9)	-0,6 (-3,8;1,7)	-2,5 (-3,9;1,5)	13(87)	11 (73)	1 (7)
p	0.0001	0.02	0.02	0.002	0.04	1
12 mo patch- tot 35	-0,9 (-2,6;1,7)	-0,1 (-2,2;1,3)	-1,2 (-3,9;1,9)	13(37)	14 (40)	11 (31)
12 mo patch+ tot 14	-1,5 (-4,8;1,5)	-0,4 (-4,9;1,6)	-1,9 (-4,4;1,5)	11(79)	5 (36)	3 (21)
p	0.04	0.2	0.04	0.01	1	0.7
24 mo patch- tot 30	-0,7 (-2,7;2,1)	-0,4 (-1,7;1,9)	-0,9 (-3,3;2,9)	8 (27)	8 (27)	10 (33)
24 mo patch+ tot 13	-1,8 (-5,3;0,9)	-0,5 (-5;0,6)	-1,8 (-3,8;1)	9 (70)	3 (23)	6 (46)
p	0.01	0.1	0.03	0.02	1	0.5

Notes:

18 – 3 minutes

PREDICTING THE DEVELOPMENT OF CHRONIC PULMONARY HYPERTENSION IN PATIENTS WITH CDH ON ECMO: WHAT CAN WE DO AND WHEN

Jashodeep Datta, BS[1], Sharon E. Phillips[1], Edmund Y. Yang, MD, PhD[2]
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Purpose:

We retrospectively examined all patients with congenital diaphragmatic hernia (CDH) who required ECMO between 1992 and 2007 to determine if differences in ventilatory modes or pressures could be associated with chronic pulmonary hypertension (cPH).

Methods:

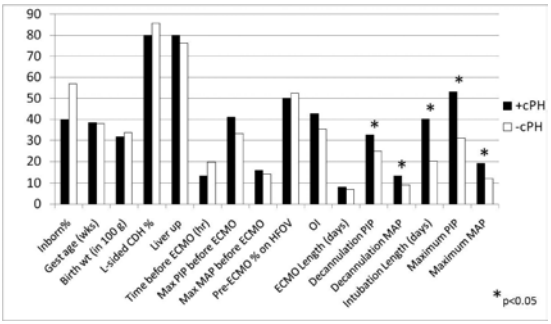
After IRB approval (#070648), we reviewed the records of CDH patients who required ECMO and survived to discharge. A diagnosis of cPH was made either by cardiac catheterization or echocardiogram at or after 3 months of age.

Results:

Thirty-two percent (10/31) of patients developed cPH. There were no differences in gestational age, birth weight, inborn status, side of CDH, or liver position between the groups. Before ECMO, patients with and without cPH were not significantly different. An equal proportion of patients received HFOV for similar duration. The maximum peak ventilatory pressure (PVP) and oxygenation index (OI) trended higher for patients with cPH (41.1 cm H_2O vs. 33.3 cm H_2O, p=0.25; 42.7 vs. 35.4 cm H_2O, p=0.25). The maximum mean airway pressure (MAP) and time to ECMO were not different between groups (16.1 vs. 14.4 cm H_2O, p=0.26; 13.2 vs. 19.8 hrs, p=0.46). The duration of ECMO was the same between the groups (8.3 vs. 7.1 days). But, the cPH patients required significantly higher PVP (32.7 vs. 25.0 cm H_2O, p=0.01) and MAP (13.5 vs. 9.2 cm H_2O, p=0.02) for ECMO decannulation. After ECMO, the maximum PVP and MAP increased dramatically for cPH patients (49.0 vs. 27.6 cm H2O, p=2.6e-5; 19.0 vs. 12.7 cm H2O, p=0.001). Thus, the overall time intubated was much longer for cPH patients (40.1 vs. 20.1 days, p=4.37e-06).

Conclusions:

Not until ECMO decannulation do we begin to see clinical differences separating patients who will ultimately develop cPH. Though pulmonary hypoplasia may ultimately dictate decannulation criteria, perhaps greater physiologic optimization before ECMO decannulation could improve the incidence or duration of cPH.



Notes:

19 – 3 minutes

NATIONAL TRENDS IN NITRIC OXIDE USE IN NEONATES WITH CONGENITAL DIAPHRAGMATIC HERNIA

Kimberly A. Ruscher, MD MPH[1], Kevin A. Modeste, MBBS[1], Stephen Neff[2], Brendan T. Campbell, MD MPH[2]
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Purpose:

Prospective, randomized trials have demonstrated that the use of nitric oxide (NO) to treat pulmonary hypertension in neonates with congenital diaphragmatic hernia (CDH) does not improve outcomes. The objective of this study is to describe national trends and costs associated with NO use in neonates with CDH.

Methods:

The Pediatric Health Information System (PHIS) database collects administrative data from 43 children's hospitals in the United States. We queried the PHIS database to identify all newborns born with CDH from January 1, 2008 through December 31, 2008. Patients with significant congenital heart disease were excluded from the analysis. Descriptive statistics were tabulated using Microsoft Excel.

Results:

In 2008, 562 CDH patients without congenital heart disease were identified. The overall mortality rate for CDH patients was 12.6% (n=71). The mortality rate for patients treated with NO was 45.7% (n= 58). Thirteen percent of patients were treated with ECMO (n=73), and 50.7% survived (n=37). Nearly a quarter of CDH patients (22.6%, n=127) were treated with NO for a mean NO charge of $120,508±$168,743 per patient. Looking at this another way, the sum of charges for NO therapy in CDH patients ($15,304,558) accounted for 10.6% of the total charges for the care provided to all 562 CDH patients ($144,978,746).

Conclusions:

In the United States nitric oxide is 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.

Notes:

20 – 3 minutes

FETAL CEREBRAL BLOOD FLOW IN CONGENITAL DIAPHRAGMATIC HERNIA.

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Purpose:

Congenital Diaphragmatic Hernia is increasingly diagnosed in the prenatal period, and parents expect individualized counseling based on severity. As part of the assessment fetal echocardiography is recommended. Left ventricular cardiac output is decreased in fetuses with CDH. This might have an impact on cerebral perfusion and/or cranial growth. This has so far not been studied.

Methods:

Retrospective review of 103 consecutive cases evaluated at a fetal medicine unit from a referral center offering fetal therapy for CDH. Fetal head circumference, biparietal diameter, transcerebral diameter, lung-to-head ratio, middle cerebral artery (MCA) peak systolic velocity (PSV), time averaged maximum velocity (TAMXV) and pulsatility index (PI) were retrieved from our database, transformed to gestational age independent scores and compared to the normal population. Subanalyses were made according to whether the CDH was left (n=86) or right sided (n=17) and to whether it was isolated (n= 86) or associated (n=17).

Results:

Cranial and cerebellar growth are within normal range in fetuses with CDH. MCA-PSV (z-score -1.47 ± 1.30) and MCA-TAMXV (z-score -1.16 ± 1.35) were significantly lower in CDH than in normal fetuses (z-test $p < 0.0001$ for both). MCA-PI was unchanged in CDH (z-score -0.04 ± 1.73, z-test $p = 0.65$). No differences in MCA blood flow were observed between the 4 subgroups. MCA-PSV z-score was correlated with lung size (left CDH: $R^2=0.08$, $p=0.009$; right CDH: $R^2=0.23$, $p=0.07$).

Conclusions:

Although cranial and cerebellar growth are within normal range, fetal cerebral perfusion is decreased in CDH. Prospective work has been initiated to assess prenatal neurologic development in this population.

Notes:

21 – 6 minutes

DECREASED CEREBRAL OXYGEN SATURATION DURING THORACOSCOPIC REPAIR OF CONGENITAL DIAPHRAGMATIC HERNIA AND OESOPHAGEAL ATRESIA IN INFANTS

Luca Giacomello, MD[1], Mark Bishay, MB ChB, MRCS[1], Merrill McHoney, MD, PhD[1], Paolo De Coppi, MD, PhD[1], Joe Brierley, MRCP, MRCPCH[1], Louise Harding, FRCA[2], Edward M. Kiely, FRCSI, FRCS[2], Joseph I. Curry, MBBS, FRCS[2], David P. Drake, FRCS[2], Kate Cross, FRCS[2], Simon Eaton, PhD[3], Agostino Pierro, MD, FRCS, FAAP[1]
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Purpose:

Congenital diaphragmatic hernia (CDH) and oesophageal atresia with tracheo-oesophageal fistula (OA/TOF) can be repaired thoracoscopically, but this may cause hypercapnia, acidosis, and reduced cerebral oxygenation. We aimed to evaluate the effect of thoracoscopy in infants on cerebral oxygen saturation, arterial blood gases and CO_2 absorption.

Methods:

Five infants weighing 2.9kg (2.4-3.9) underwent thoracoscopy between March and August 2009 (3 CDH and 2 OA/TOF). With ethical approval (09/H0714/2), serial arterial blood gases were measured in all patients, and regional cerebral oxygen saturation was measured in four neonates using near-infrared spectroscopy (INVOS®). Absorption of insufflated CO_2 was calculated from exhaled $^{13}CO_2/^{12}CO_2$ measured by isotope-ratio mass spectrometry. Data are presented as median (range).

Results:

Duration of operation was 75min (65-135) for CDH repair and 150min (120-180) for OA/TOF repair. 27% (14-47) of exhaled CO_2 originated from the pneumothorax. In one patient thoracoscopic CDH repair was converted to thoracotomy. This patient showed a drop in pH to 6.90, increasing after conversion to 7.44. $PaCO_2$ increased concomitantly to 15.5 then recovered to 5.4, while cerebral oxygen saturation fell to 42% before recovering to 68%. Overall, arterial pH decreased from 7.23 (7.00-7.39) at start of operation to 7.02 (6.90-7.19) intraoperatively and recovered to 7.22 (6.94-7.44) at end of operation. $PaCO_2$ increased from 9.1 (4.8-16.2) at start of operation to 13.4 (8.8-15.5) intraoperatively and then fell back to 9.4 (5.4-14.7) at end of operation. The intraoperative acidosis was associated with a decrease in cerebral haemoglobin oxygen saturation from 84% (70-95) at the start of operation to 69% (62-76) at end of operation. This had not recovered by 12hours [68% (66-76)] or 24 hours [69% (65-74)] post-operatively.

Conclusions:

This preliminary study suggests that thoracoscopic repair of CDH and OA/TOF may be associated with acidosis and decreased cerebral oxygen saturation. The effects of these phenomena on future brain development are unknown.

Notes:

22 – 3 minutes

SYMPTOMATIC VOCAL CORD PARALYSIS IN INFANTS OPERATED ON FOR ESOPHAGEAL ATRESIA AND/OR TRACHEO-ESOPHAGEAL FISTULA.

Francesco Morini, Barbara D. Iacobelli, MD, Alessandro Crocoli, MD, Sergio Bottero, MD, Marilena Trozzi, MD, Andrea Conforti, MD, Pietro Bagolan, MD
Bambino Gesù Children's Research Hospital, Rome, Italy

Purpose:

Dyspnea and stridor are common after esophageal atresia (EA) repair. Although a number of causes have been implicated in their pathogenesis, the role of vocal cord paralysis (VCP) has been rarely studied. Aim of our study was to describe the prevalence and pathogenesis of symptomatic VCP in a large cohort of patients (pts) operated on for EA and/or tracheo-esophageal fistula (TEF).

Methods:

Retrospective review of all consecutive pts treated for EA and/or TEF between August 1995 and June 2009. Symptomatic VCP suspected on the basis of persistent stridor, dyspnea, and/or dysphonia/aphonia, was confirmed by flexible laryngoscopy. Pts with and without VCP were compared for birth weight, gestational age, prevalence of associated anomalies, long gap (>3 cm or 3 vertebral bodies), cervical esophagostomy, anastomotic leak, azygos ligation, and major cardiac surgery by Mann-Whitney or Fisher's exact test as appropriate. Results are medians (interquartile ranges) or prevalence, $p<0.05$ was considered significant.

Results:

During the study period 174 consecutive pts with EA and/or TEF were treated; symptomatic VCP was detected in 7 (4%). Table shows main findings.

Main clinical finding in pts with and without VCP

	VCP+ (7 pts)	VCP- (167 pts)	p	RR (95% CI)
Gestational age (weeks)	37 (32-38)	38 (36-40)	0.3591	
Birth weight (gr)	2600 (2515-3365)	2540 (2120-2990)	0.2229	
Associated anomalies (N)	4	118	0.4279	0.2 (0.1-2.5)
Long gap (N)	5	41	0.0146	6.9 (1.4-34.6)
Cervical esophagostomy (N)	5	7	<0.0001	33.7 (7.3-156.1)
Anastomotic leak (N)	3	10	0.0097	9.3 (2.3-37.2)
Azygos ligation (N)	0	48	0.1921	0.0 (± ∞)
Major cardiac surgery (N)	0	4	1.000	0.0 (± ∞)

Conclusions:

VCP is a rare but important cause of respiratory morbidity in patients treated for EA and/or TEF, that should be ruled out in case of persisting respiratory morbidity. Surgical factors that may increase the risk of recurrent laryngeal nerve injury, such as cervical esophagostomy, should be limited as much as possible. Further systematic studies are needed to define the prevalence of acquired asymptomatic and congenital VCP in pts with EA and/or TEF.

Notes:

23 – 3 minutes

NECROTIZING ENTEROCOLITIS IS ASSOCIATED WITH NEONATAL INTESTINAL INJURY

Kristyn Mannoia, BA, Danilo Boskovic, PhD, Laurel Slater, BSN, RNC, Megan Plank, BS, Gerald Gollin, MD
Loma Linda University School of Medicine, Loma Linda, CA, USA

Purpose:

Some cases of necrotizing enterocolitis (NEC) may result from perinatal asphyxia and ischemia-reperfusion injury to the intestine in the neonatal period. This progression has been difficult to confirm as the epidemiological association of asphyxia and NEC is weak and there has been no documentation of intestinal injury in asphyxiated newborns. We hypothesized that a subset of premature newborns has subclinical, intestinal mucosal compromise that predisposes to the development of NEC days or weeks later.

Methods:

41 newborns of 24-33 weeks gestational age were identified and urine was collected in 6 hour aliquots over the first 3 days of life. The urinary concentration of intestinal fatty acid binding protein (iFABP), a sensitive and specific marker for intestinal mucosal injury, was determined by ELISA and a concentration of >1000 pg/ml was defined as elevated. NEC was determined on pathology or by radiological evidence.

Results:

iFABP was elevated in 25 subjects, including 7 of 7 who subsequently developed NEC, and 4 of 5 who died without developing NEC. Among those with a base deficit greater than 5 mEq/L at birth, 18 of 24 had elevated iFABP, although only 3 infants who developed NEC had metabolic acidosis at birth.

Conclusion:

In this population of premature newborns there was a substantial incidence of subclinical, intestinal mucosal compromise, particularly among those with a metabolic acidosis. All infants who subsequently developed NEC and all but one of those who died due to another cause had an elevated urinary iFABP during the first three days of life. This finding suggests a model for the pathogenesis of some cases of NEC whereby perinatal ischemia-reperfusion injury predisposes to intestinal compromise when feedings are initiated. In addition, neonatal iFABP assessment may represent a tool to identify infants at the highest risk for NEC and allow for the institution of focused, preventive measures.

Notes:

24 – 3 minutes

URINE BIOMARKERS OF PROGRESSIVE NECROTIZING ENTEROCOLITIS

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Purpose:

Progressive Necrotizing Enterocolitis (NEC) defined by the need for surgery, causes significant morbidity and mortality in premature infants. Previous epidemiological studies have failed to identify risk factors predictive of progressive NEC. Since we have previously reported the discovery of candidate biomarkers for progressive NEC in plasma, we hypothesized that peptide biomarkers of progressive NEC could be discovered in the urine of infants with early stage NEC.

Methods:

Patient clinical data and biologic sample collection was performed by the NEC Consortium. Thirty-four urine samples were collected and post-hoc identified as either progressive (n=17) or non-progressive NEC (n=17). Peptide spectra were generated using LC-MALDI. A nearest shrunken centroid (NSC) algorithm and ten-fold internal cross validation analyses were conducted on all samples to search for unique peptide features. Linear discriminate analysis (LDA) was used to select a panel of 14 biomarkers with minimal classification errors. Supervised and unsupervised cluster analyses were applied to interrogate the panel's predictive power. The panel's performance was further validated by ROC curve plotting of LDA-derived prediction scores that are calculated via bootstrapping methods on the re-sampled training data to derive a simulated test set.

Results:

The biomarker panel correctly classified 16 of 17 non-progressive (NP) and 15 of 17 progressive (P) NEC patients (p-value 2.1×10^{-6} (Fisher's) during supervised analysis. In unsupervised modeling, it accurately categorized 13 NP and 14 P NEC patients (p-value 1.5×10^{-3} (Fisher's). ROC curve analysis revealed the panel's robust discriminative performance with an AUC value of 0.997.

Conclusion:

Urine proteomic profiling and statistically rigorous feature selection were successfully combined to identify urine biomarkers with high sensitivity and specificity for progressive NEC at the time of initial diagnosis. Urine biomarkers may represent both potential diagnostic/prognostic tools for clinical development as well as an effective research vehicle for NEC risk stratification

Notes:

25 – 3 minutes

SURGERY FOR NECROTIZING ENTEROCOLITIS IN THE UNITED STATES: CURRENT PRACTICE AND MORTALITY

Melissa A. Hull, MD[1], Brian A. Jones, MD[1], Joseph H. Carpenter, MS[2], Michael Kenney, MS[2], Jennifer C. Michelle, MPH[2], David Zurakowski, PhD[1], Jeffrey D. Horbar, MD[2], Tom Jaksic, MD, PhD[1]
[1]Children's Hospital Boston, Boston, MA, USA, [2]Vermont Oxford Network, Burlington, VT, USA

Background:

Necrotizing enterocolitis (NEC) is a leading cause of surgical intervention and death in premature neonates. The goals of this study were to determine national birth weight dependent benchmarks for the mortality of surgical NEC and to describe the respective mortality of laparotomy and peritoneal drainage.

Methods:

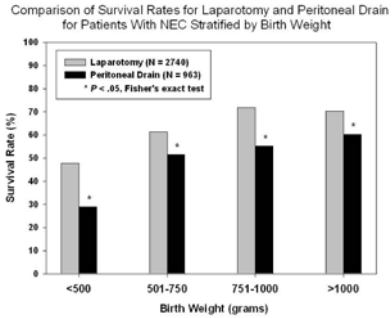
Following Institutional Review Board approval, data were prospectively collected on 79,445 very low birth weight neonates (<1500 g) treated in 545 U.S. hospitals between January 2006 and December 2007. 6852 infants had a diagnosis of NEC, including spontaneous intestinal perforation, based on stringent clinical, radiologic, and/or histopathologic findings. Patients were stratified by birth weight: 1000 g. Primary outcome was in-hospital mortality, and patients were followed until discharge or 1 year of age.

Results:

Of the 6852 neonates with a diagnosis of NEC, 3703 (54%) were managed with laparotomy or peritoneal drainage. Laparotomy was performed more frequently (n=2740, 74%) than peritoneal drainage (n=963, 26%), regardless of birth weight ($P < .0001$). Surgically managed NEC had an overall mortality of 37%. 44% of patients with peritoneal drainage had an additional laparotomy. Multivariate logistic regression identified birth weight and type of procedure (laparotomy or peritoneal drainage) as significant independent predictors of mortality (both $P < .0001$).

Conclusions:

The study cohort encompassed 2/3 of all very low birth weight neonates born in the U.S. during the data collection period. The overall mortality of surgically managed NEC was 37%, with lower birth weight being highly predictive of worse outcome. Laparotomy was performed significantly more frequently than peritoneal drainage. Although it is possible that lower risk patients were treated preferentially with laparotomy, this therapy was associated with higher survival than peritoneal drainage across all birth weight categories.



Notes:

26 – 3 minutes

MORTALITY OF NEONATAL SPONTANEOUS INTESTINAL PERFORATION: A MULTI-INSTITUTIONAL ASSESSMENT

Brian A. Jones, MD[1], Melissa A. Hull, MD[1], Joseph H. Carpenter, MS[2], Michael Kenny, MS[2], Jennifer C. Michelle, MPH[2], David Zurakowski, PhD[1], Jeffrey D. Horbar, MD[2], Tom Jaksic, MD, PhD[1]
[1]Children's Hospital Boston, Boston, MA, USA, [2]Vermont Oxford Network, Burlington, VT, USA

Purpose:

Spontaneous intestinal perforation is a disease of the neonate, often regarded as clinically distinct from necrotizing enterocolitis. The purpose of this study was to examine the mortality associated with operatively diagnosed spontaneous intestinal perforation in very low birth weight neonates in the United States.

Methods:

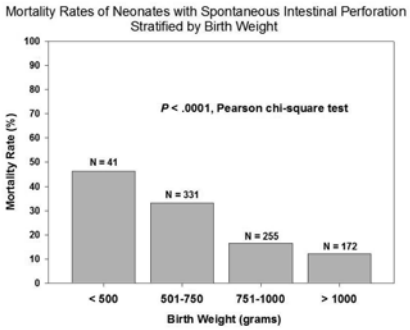
After Institutional Review Board approval, data was prospectively collected on 79,445 neonates with birth weight <1500g born in one of 545 U.S. hospitals between January 2006 and December 2007. Spontaneous intestinal perforation was defined as a focal perforation of the bowel without evidence of necrotizing enterocolitis or other bowel abnormality on visual inspection of the intestine at the time of surgery. The primary outcome was in-hospital mortality by one year of life. The Pearson chi-square test was used to analyze factors contributing to mortality with significance set at $P < .05$.

Results:

A total of 799 children were diagnosed with spontaneous intestinal perforation at laparotomy. The overall mortality rate was 24%. Birth weight was divided into four categories (<500g, 501-750g, 751-1000g, and >1000g). Spontaneous intestinal perforation was associated with patent ductus arteriosus (73%), indomethacin treatment (52%), and post-natal steroid administration (23%). Mortality was 46% in the <500g birth weight group; 33% in the 501-750g group; 16% in the 751-1000g group; and 12% in the >1000g group. Mortality decreased significantly as birth weight increased ($P < .0001$, Pearson chi-square test).

Conclusions:

The in hospital mortality associated with spontaneous intestinal perforation is high. Mortality of spontaneous intestinal perforation decreases significantly with increasing birth weight with a striking improvement in neonates >750g. These data offer the first reliable benchmarks for the mortality associated with spontaneous intestinal perforation.



Notes:

27 – 3 minutes

DOES MULTIDISCIPLINARY PRENATAL COUNSELING OR DELIVERY METHOD AFFECT OUTCOME IN GASTROSCHISIS?

Christopher W. Snyder, MD, MSPH, Joseph R. Biggio, MD, Donna T. Bartle, RN, BSN, Phillip R. Brinson, BS, Leandra A. Barnes, Keith E. Georgeson, MD, Oliver J. Muensterer, MD, PhD
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Purpose:

We investigated the effects of prenatal counseling and Cesarean section (C-section) versus vaginal delivery on intestinal functional outcome in gastroschisis.

Methods:

IRB approval was obtained. All gastroschisis patients treated at a single tertiary children's hospital between 1999 and 2009 were included. Mothers receiving multidisciplinary prenatal counseling were identified via fetal medicine clinic records. Delivery method (vaginal vs. C-section), patient characteristics, treatment details, and clinical outcomes were determined by chart review. Time to complete independence from parenteral nutrition (PN) was the primary outcome of interest. Effects of prenatal counseling and delivery method on time to PN independence were evaluated using a Cox proportional hazards model that included sex, birth weight, concomitant intestinal complications (e.g. atresia, perforation) , and early hypoalbuminemia.

Results:

Of 167 patients included, median birth weight was 2460 grams, median gestational age was 36 weeks, 45% were male, 46% were delivered vaginally, and 69% of mothers received multidisciplinary prenatal counseling. Bowel trauma during vaginal delivery occurred in one patient (1.3%). Prenatal counseling was associated with slightly higher median gestational ages (36.8 vs. 36.0 weeks, p=0.012) and birth weights (2549 vs. 2215 g, p=0.0002). Median time to PN independence was 38 days in the overall cohort. On multivariable modeling, only higher birth weight and absence of concomitant intestinal complications were significant predictors of PN independence. There was a strong trend toward more rapid PN independence among patients delivered vaginally (Hazard Ratio 1.48, 95% CI 0.99-2.23, p=0.058). Prenatal counseling had no effect on PN independence (HR 0.92, 95% CI 0.57-1.49, p=0.744).

Conclusions:

Birth weight and concomitant intestinal complications are the major determinants of PN duration in gastroschisis. Multidisciplinary prenatal counseling and planning does not hasten intestinal function. Vaginal delivery appears to be safe, and may provide benefits over Cesarean section with respect to intestinal functional outcome.

Notes:

28 – 3 minutes

VERTICAL EXPANDABLE PROSTHETIC TITANIUM RIB (VEPTR) DEVICE INSERTION: DOES IT IMPROVE PULMONARY FUNCTION?

Samir K. Gadepalli, Frances A. Farley, MD, Wan C. Tsai, MD, Peter J. Strouse, MD, Robert A. Drongowski, MS, Ronald B. Hirschl, MD
University of Michigan, Ann Arbor, MI, USA

Purpose:

We reviewed our experience with VEPTR implantation to determine if lung function and growth is augmented.

Methods:

From 2006-2009, 25 insertions and 45 expansions were performed in 22 patients. Demographic data were reviewed in conjunction with complications, pulmonary function tests (PFT), CT guided 3D-reconstructions to determine lung volumes, and satisfaction scores determined using the SRS-22 (scoliosis research society) questionnaire preoperative and at a mean of 1 year postoperative. The groups were also stratified by age (<or> 7 years because of lung growth potential), disease (scoliosis, Jeune, neuromuscular, other), or gender. Student t-test, ANOVA and Chi-squared analyses were performed with $p<0.05$ considered significant.

Results:

Each patient underwent 3.6 surgeries, spending 1.26±1.06 days in the ICU and 4.50±4.27 days in the hospital for each procedure. Mean age was 82.9±40.8 months. In all, 23 complications occurred with 15/23 treated nonoperatively: 2 transfusions for bleeding, 6 infections treated with antibiotics, 1 seroma, 4 neurologic (pain or numbness), and 2 pleural effusions. Reoperation was required in 4 for chest tube placement and 4 for hardware removal (for infection) and repositioning (for dislodgement). No statistically significant difference was seen in complications, PFT (FEV1, FVC, RV), lung volumes, and SRS-22 scores between gender, age, or disease categories. There was also no improvement in lung function, volume, or SRS-22 scores between the pre-and postoperative period.

		Preoperative	**Postoperative**	**P-value**
PFT	FEV1	52.2±30.4	50.0±16.3	0.81
	FVC	54.1±32.5	52.0±18.6	0.77
	RV	230.7±136.6	119.9±46.2	0.36
3DCTR	Total volume	670.0±221.6	674.6±219.7	0.92
SRS-22	Function	4.01±0.66	3.93±0.60	0.48
	Pain	3.99±1.04	3.82±0.89	0.45
	Self-Image	4.00±0.44	4.16±0.47	0.17
	Mental health	4.39±0.46	4.38±0.45	0.78
	Satisfaction	4.08±0.69	4.03±0.76	0.78

Conclusion:

Pulmonary function, lung volume, and patient subjective assessments did not increase following VEPTR placement. Our data suggest that VEPTR placement does not enhance pulmonary status.

Notes:

29 – 6 minutes

PEDIATRIC ACS NSQIP: FEASIBILITY OF A NOVEL, PROSPECTIVE ASSESSMENT OF SURGICAL OUTCOMES - A PHASE I REPORT

Mehul V. Raval, MD[1], Peter W. Dillon, MD[2], ACS NSQIP Pediatric Steering Committee[3]
[1]Division of Optimal Patient Care, American College of Surgeons; Division of Pediatric Surgery, Department of Surgery, Feinberg *School of Medicine, Northwestern University, Children's Memorial Hospital, Chicago, IL, USA, [2]Division of Pediatric Surgery, Department of Surgery, Penn State College of Medicine, Hershey, PA, USA, [3]American College of Surgeons and American Pediatric Surgical Association, Chicago, IL, USA*

Purpose:

The ACS National Surgical Quality Improvement Program (ACS NSQIP) provides validated, risk-adjusted assessment of surgical outcomes and facilitates the development of best practices. This study reports Phase 1 testing of an ACS NSQIP-Pediatric program at four pediatric hospitals.

Methods:

From October 2008 to May 2009, 114 data variables were prospectively collected for 3,317 patients, including rigorous 30-day outcomes, using a systematic sampling method; tailoring the ACS NSQIP methodology to all children's surgical specialties.

Results:

399 postoperative complications/occurrences were detected in 277 patients, representing 8.4% of the study population (Table). Of the patients with complications, 200 (72%) had 1, 42 (15%) had 2, and 35 (13%) had 3 or more complications. There were 12 deaths (0.36%) and 14 intra-operative occurrences (0.42%) detected, the most common postoperative complications were infection-88 (22%) (SSI-44, sepsis-26, pneumonia-12, UTI-6); post-operative airway/respiratory events-24 (6%); wound disruption-19 (4.8%); neurologic events-6 (1.5%) (nerve injury-3, stroke/CVA-2, coma-1); DVT-4 (1%); renal failure-4 (1%); and cardiac events-3 (<1%). Current sampling captures roughly 17.5% of cases across institutions. Unadjusted institutional complication rates ranged from 6.8% to 10.2%. Completeness of data collection for all variables exceeded 95% with 87% of patients having full 30-day follow-up across 4 sites, representing 183 surgeons and patients from 685 cities and 31 states.

Conclusions:

These data represent the first multi-institutional prospective assessment of specialty specific post-operative morbidity and mortality in children. Implementation of the ACS NSQIP-Pediatric has been successful and has facilitated refinement of sampling methodology, data collection practices, and data definitions. The program is now poised for institutional expansion that will permit the development of risk-adjusted models across surgical specialties.

Table

Specialty	Number of cases (%)	Number of cases with complications (%)	% of complications
General Surgery	1010 (30.4)	91 (9.0)	32.9
Otolaryngology	755 (22.8)	19 (2.5)	6.9
Orthopedics	598 (18.0)	50 (8.4)	18.0
Urology	391 (11.8)	16 (4.1)	5.8
Neurosurgery	368 (11.1)	77 (21.0)	27.8
Plastics	182 (5.5)	22 (12.1)	7.9
Others	13 (<1)	2 (15.4)	0.7
Total	3317 (100)	277 (8.4)	100

Notes:

30 – 3 minutes

ELEMENTS OF SUCCESSFUL INTESTINAL REHABILITATION

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Purpose:

The relative importance of current therapies for intestinal failure (IF) is unknown. We examined the effects of aggressive introduction of enteral feeds, rotating antibiotics, lipid sparing and fish oil therapies by comparing cohorts of patients cared for prior to (1998-06) and after (2006-09) the introduction of an algorithm with these elements.

Methods:

IF was defined as bowel length of < 40 cm, or Parenteral Nutrition (PN)> 60 days. Previously (1998-06) IF patients were cared for by a multi-disciplinary team but a protocol driven strategy to prevent PN associated liver disease (PNALD) was instituted in 2006. Data have been gathered prospectively with familial consent, and ethics board approval.

Results:

From 98-06, 33 patients were identified, with a 72% survival; 8/33 (27%) of patients received prophylactic antibiotics, 2/33 had lipid sparing PN; none received fish oil based lipids. The causes of IF were gastroschisis (30%) and atresia (21%). Average time to intestinal rehabilitation/death was 4.5 ± 3 months, 9/33 patients developed PNLD, all died. From 06-09, 30 patients have been followed, with 100% survival*, 23/30(77%)* received routine rotating enteral antibiotics, 15/30* had lipid sparing PN, 12/30 * received fish oil based lipid PN for an average of 121±160 days. Diagnosis were gastroschisis 15/30 (50%) and atresia 9/30 (30%). 28/30 are weaned from PN, at 3.4±7.2 months*, no patients developed PNLD* (* $p<0.05$ by Fischer's exact, student's t-test, or Kaplan-Meier analysis, data mean ± SD). Of the factors examined, lipid reduction and fish oil based lipid use were associated with the improved outcomes of survival and preserved liver function.

Conclusions:

The institution of an aggressive rehabilitation protocol with lipid reduction and the use of fish oil preparations appears to improve liver function, and intestinal rehabilitation in IF. Further studies to validate these findings in a larger cohort of patients are recommended.

Notes:

31 – 3 minutes

OUTCOMES FROM GASTRIC ELECTRICAL STIMULATION IN CHILDREN

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Purpose:

Gastric Electrical Stimulation (GES) is used in patients with intractable nausea and vomiting due to gastroparesis . The use of this modality has only been described in adults. We present our long term outcomes, an expanded experience, and use of temporary GES (tGES).

Methods:

Children and adolescents with refractory gastroparesis or other dysmotility, were selected for GES. Each case underwent tGES followed by permanent if successful. From 2004-2009, patient charts were reviewed and data regarding clinical presentation and course, pre and post symptom scores and long term outcomes were analyzed. Paired student's t test, and Mann Whitney's U test, were used for statistical analysis.

Results:

42 patients underwent 47 attempts at tGES. Almost 80% were female, and mean age at GES was 15 years. Endoscopic placement of tGES lead was done in 26 cases, while the remainder were placed through a gastrostomy tube. 50% responded to the temporary GES and were candidates for permanent stimulation. 21 patients underwent implantation of the permanent stimulator, and *all* experienced initial improvement of symptoms (Nausea ($p=0.002$), Vomiting ($p=0.04$), and total symptom score ($p<0.001$)). Four (19%) patients subsequently had the stimulator removed - two for recurrence of symptoms, one due to trauma and one for improved symptoms off therapy. The remaining 17 (81%) have had sustained improvement in symptoms. Follow up is from 1-5 years in this cohort. Medication use and hospital inpatient days were reduced ($p=0.002$). There were no adverse reactions from GES.

Conclusions:

This represents the largest experience of GES in children in the world. Our success rate is higher than adult reports. Our experience with tGES has allowed us to expand the role to other dysmotilities. Long term outcomes remain very good with sustained response to therapy and reduced use of resources. GES appears to be a safe therapy in children.

Notes:

32 – 3 minutes

RESULTS OF A LONGITUDINAL STUDY OF RIGOROUS MANUSCRIPT SUBMISSION GUIDELINES DESIGNED TO IMPROVE THE QUALITY OF CLINICAL RESEARCH REPORTING IN A PEER-REVIEWED SURGICAL JOURNAL

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Purpose:

After finding limitations in the quality of reporting of clinical research in the Journal of Pediatric Surgery, a voluntary checklist of reporting Guidelines was developed. Beginning in 2006, all clinical research manuscripts were requested to include the guidelines. The purpose of this study was to evaluate whether this requirement improved reporting quality.

Methods:

The Guidelines included 23 criteria in 3 subcategories Methods, Results, and Multiple treatment groups. Quality was evaluated by determining the percentage of applicable Guidelines criteria met to create a composite score. A randomized selection of 73 articles from the pre-Guidelines period (04/1997-06/2001) and 147 consecutive articles from the post-Guidelines period (03/2007-11/2008) were independently assessed by 2 reviewers. Global and subcategory composite scores were calculated for each article. Mean scores pre- and post-Guidelines were compared using a t-test, and the proportion of articles meeting different compliance levels were compared using chi square or Fisher's exact test as appropriate. A *p* value less than 0.05 was significant. This study was granted IRB exemption.

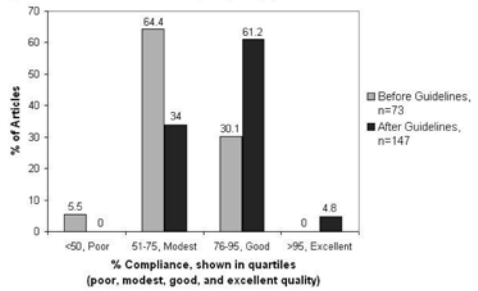
Results:

The use of the guidelines significantly increased the quality of research reporting in all sub-categories. Mean global composite scores increased from 72.2 pre-Guidelines to 80.1 post-Guidelines ($p<0.0001$). Methods increased from 71.9 to 78.6 ($p<0.0001$), Results increased from 77.2 to 83.0 ($p=0.002$), and Multiple treatment groups increased from 40.0 to 70.6 ($p=0.0003$). Figure 1 demonstrates an increased proportion of articles in the higher quality quartiles.

Conclusion:

We conclude that the introduction of the Guidelines has resulted in significant improvement in the quality of reporting in the *Journal* This is a notable achievement for guidelines which were voluntary and did not include enforcement by journal staff. We believe that other surgical journals should strongly consider the adoption of analogous guidelines in an effort to improve the quality of clinical research in Surgery.

Figure 1. Articles show shift to higher quality quartiles.



Notes:

33 – 3 minutes

MINIMAL VERSUS EXTENSIVE ESOPHAGEAL MOBILIZATION DURING LAPAROSCOPIC FUNDOPLICATION: A PROSPECTIVE RANDOMIZED TRIAL

Shawn D. St. Peter, MD[1], Douglas C. Barnhart, MD[2], Daniel J. Ostlie, MD[1], KuoJen Tsao, MD[3], Susan W. Sharp, PhD[1], Tracie Morgan, RN[4], Donna Bartle, RN[4], Carroll M. Harmon, MD[4], Keith E. Georgeson, MD[4], George W. Holcomb III, MD, MBA[1]
[1]Children's Mercy Hospital, Kansas City, MO, USA, [2]University of Utah/Primary Children's Medical Center, Salt Lake City, UT, USA, [3]University of Texas Medical Center, Houston, TX, USA, [4]University of Alabama Birmingham, Birmingham, AL, USA

Purpose:

Laparoscopic fundoplication is a common operation which has traditionally been performed with extensive esophageal mobilization. Retrospective data have shown that minimal esophageal mobilization may reduce the risk of postoperative transmigration of the fundoplication wrap into the lower mediastinum. Therefore, the role of esophageal mobilization during laparoscopic fundoplication was studied in a two-center, prospective, randomized trial.

Methods:

After IRB approval, patients were randomized to extensive esophageal mobilization after division of the phrenoesophageal membrane (MAX) or minimal esophageal mobilization leaving the anterolateral phrenoesophageal membrane intact (MIN). The primary outcome variable was postoperative wrap transmigration. Patients with a pre-existing hiatal hernia or requiring an open operation were excluded. Randomization was stratified for neurologic impairment. A posterior cruroplasty and four sophagocrural sutures were utilized in all patients. A contrast study was performed at 1 year and as indicated for symptoms.

Results:

From 2/2006 through 5/2008, 177 patients were enrolled in the study (n=90 MIN, n=87 MAX). There were no differences in demographics or operative time (Table 1). Mortality at 1 year was 7.8% in MIN group and 6.9% in the MAX group (P=0.99), none of which was related to the operation. Contrast studies were performed in 64 MIN patients and 71 MAX patients showing a higher transmigration rate in the MAX group (Table 2).

Conclusions:

Minimal esophageal mobilization during laparoscopic fundoplication decreases postoperative wrap transmigration and the need for a re-do operation.

Table 1 - Patient Characteristics at Operation

	MAX (N=87)	**MIN (N=90)**	***P*-Value**
Age (yrs)	1.9 +/- 3.3	2.5 +/- 3.5	0.30
Weight (kg)	10.7 +/- 11.9	12.6 +/- 18.2	0.44
Neurologically Impaired (%)	51.7	54.4	0.76
Operating Time (Minutes)	100 +/- 34	95 +/- 37	0.37

Table 2 - Outcome Data

	MAX (N=70)	**MIN(N=64)**	***P*-Value**
Postoperative Wrap Transmigration (%)	30.0 %	7.8 %	**0.002**
Need for Re-do Fundoplication (%)	18.4%	3.3 %	**0.006**

Notes:

34 – 6 minutes

IMPACT OF OMEGA 3 FATTY ACIDS ON LIVER FUNCTION TESTS AND LIVER HISTOLOGY IN CHILDREN WITH PARENTERAL NUTRITION DEPENDENT INTESTINAL FAILURE

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Purpose:

Omega-3 fatty acids (O3FA) are increasingly being utilized in treating pediatric intestinal failure associated liver disease (IFALD). Most reports note a dramatic decrease in direct bilirubin in the first few months of O3FA therapy but little information regarding long term IFALD outcomes. We report our experience with O3FA in 14 children with IFALD including the results of post-O3FA liver biopsies.

Methods:

After IRB approval, 14 IF patients receiving O3FA were prospectively evaluated. One patient died unrelated to IFALD. Three patients underwent visceral transplantation while on O3FA. Serum direct bilirubin and transaminase levels as well as liver biopsy results are reported. Parametric and non-parametric ANOVA were used with $p<0.05$ considered significant.

Results:

After starting O3FA the mean bilirubin concentration significantly decreased by 2 months and normalized by 6 months (Table). Serum AST significantly decreased after 6 months of O3FA. In contrast, the mean serum ALT and GGT levels did not significantly decrease by 12 months post O3FA. Liver biopsy results, ranging from 1 to 12 months following O3FA therapy, revealed extensive hepatic fibrosis. One patient had liver biopsies pre and post O3FA therapy with worsening bridging fibrosis but improved cholestasis and hepatocyte healing.

Conclusion:

Omega-3 fatty acids rapidly improve some serum parameters of cholestasis, including direct bilirubin. While our liver biopsy data suggests that this therapy does not prevent hepatic fibrosis/cirrhosis there is a suggestion that hepatocyte damage is reversed. These findings demonstrate the need for rigorous clinical trials to determine the optimum timing, dosing, and other variables regarding the utility of omega-3 fatty acids to ameliorate intestinal failure associated liver disease.

Mean concentration and standard deviation of liver tests pre and post O3FA therapy, *p<0.05

	Pre-O3FA	1 month	2 months	3 months	6 months	12 months
Direct Bilirubin (mg/dL)	6.91±3.42	6.31±3.77	3.75±3.56*	2.19±3.51*	0.17±0.40*	0.02±0.040*
AST (U/L)	388.86±377.04	323.77±213.50	216.00±128.96	162.45±136.33	74.70±28.34*	56.83±6.24*
ALT (U/L)	191.79±147.21	220.62±150.89	176.82±81.41	145.55±116.76	72.20±37.13	61.83±18.91
GGT (U/L)	149.79±96.75	182.50±119.85	241.10±251.60	159.45±114.17	137.57±118.97	110.00±99.12

Notes:

35 – 6 minutes

MORE OR LESS? - LESSONS LEARNED FROM LAPAROENDOSCOPIC SINGLE SITE SURGERY IN CHILDREN

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Purpose:

Laparoendoscopic single site surgery (LESS) is becoming increasingly popular for adult patients, but only a few studies have been reported so far in children. Compared to conventional laparoscopic surgery, the LESS approach entails some unique and novel challenges. The purpose of this study was to examine our first 114 pediatric LESS cases with particular attention given to technical difficulties.

Methods:

After IRB approval, data related to all LESS cases were prospectively collected. Pediatric surgeons involved in the cases were polled for technical difficulties encountered and their proposed operative solutions.

Results:

Over a period of 7 months, 114 pediatric LESS cases were performed in 113 pediatric patients (49 female) age 3 weeks to 19 years. Among these were 76 appendectomies (10 perforated, 10 interval, 56 acute appendicitis) 19 pyloromyotomies, 15 cholecystectomies, and 1 splenectomy. Seven appendectomies and 6 cholecystectomies employed a primary extra-umbilical, percutaneous 2mm grasping instrument along with the transumbilical trocars. None required laparotomy. Median operative times were 39±14min, 25±12min, and 81±21min for appendectomy, pyloromyotomy, and cholecystectomy, respectively. Intraoperative complications included bleeding, thermal injury to neighboring bowel, and intestinal perforation (n=1 each). The technical difficulties identified included: 1) instrument crowding, 2) fewer degrees of freedom for working instruments, 3) in-line endoscope view and 4) increased gas leak from multiple umbilical fascial puncture sites. Proposed coping strategies, respectively, are: 1) using instruments of different length, 2) working in the distal-proximal axis when possible, using roticulating instruments, 3) employing a 45° or swivel-head endoscope, and 4) using small fascial stab incisions, along with special trocars.

Conclusion:

Pediatric LESS procedures are feasible and result in no visible scarring. However there are a number of technical challenges which should be solved to lessen potential complications compared to traditional pediatric laparoscopic surgery.

Notes:

36 – 6 minutes

ON BOARD THE USNS COMFORT WITH CONTINUING PROMISE 2009 - A MODEL FOR HUMANITARIAN ASSISTANCE

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Purpose:

Surgical organizations have begun to focus their efforts on providing humanitarian assistance in international communities. Most surgeons do not have previous international experience and lack an understanding of what is expected and what care they can provide. The unknown factors include case types, patient volume, postoperative care and equipment. This abstract presents our model of humanitarian assistance and highlights the importance of preparation, host-nation involvement, and understanding the local politics of each country.

Methods:

In April-July 2009, the USNS Comfort deployed to provide humanitarian assistance to seven countries through Central and South America. We tracked cases, anesthesia, OR utilization and basic laboratory values.

Results:

These data represent the total mission of Continuing Promise 2009 including a total of 1141 surgical procedures of which 342 were pediatric (< 18 yo). The average number of pediatric cases for each country in seven days was 48.3+/-21.4 with a range of 24-84. The average age was 7.8 yrs (range 1 month - 18 yo). Our model included 2-3 days of preoperative screening for every 7 operative days, inclusion of host physicians, and a low threshold for rejection (rate of 43%, range 21-62%) and average ASA score of 1.3. Including all pediatric subspecialties, the most frequent procedures were: inguinal (23.6%) and umbilical hernias (13.0%). Whereas these were the most frequent procedure, the range and variety of cases varied widely. We had a very low complication rate (1.2%) including three wound infections and one early hernia recurrence. The OR utilization rate ranged between 72.9-91.4%.

Conclusions:

Our data represent the largest collection to date on the pediatric surgical care of children in a humanitarian effort. Our experience can be used to identify the most likely types of cases in South and Central America and as a model for the safe and efficient treatment of children in a developing country.

Notes:

37 – 3 minutes

THE CONTRIBUTION OF GASTROESOPHAGEAL REFLUX DISEASE TO WEIGHT LOSS IN NICU INFANTS

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Purpose:

To identify the contribution of GERD and other factors associated with weight loss in NICU infants.

Methods:

An IRB approved retrospective review was performed between January 2004 and December 2006 of 250 NICU infants under age 6 months, who underwent a 24-hour pH probe. Growth curves were reviewed at time of probe, 6 months later and study completion (2-5 years follow-up). Infants who did not maintain their growth curves were identified. Data were analyzed by Chi-square and logistic regression. P-values <0.05 were considered significant.

Results:

Follow-up was complete in 191. GERD was present and treated in 87(45.5%), (Nissen fundoplication-31, medications-56). Twenty-one(24.1%) lost weight (Nissen-7 and medications-14). Weight loss due to GERD occurred in none managed by Nissen and 2 managed with medications(9%). Nineteen lost weight unrelated to GERD (91%). GERD was not present in 104(54.5%) and 27/104(26%) lost weight. Weight loss due to GERD occurred in 7(26%) between 6 and 31 months after an initial negative pH probe (median-11 months). Weight loss unrelated to GERD occurred in 20(74%). Chi-square revealed: chromosomal or genetic syndromes, (p=0.001), GERD exacerbation, (p=0.005), ALTE, (p=0.024), evaluation for failure to thrive, (p=0.031), and inability to feed orally, (p=0.033) were associated with weight loss. Logistic regression revealed: chromosomal or genetic syndromes, OR 13.558 (95% CI 4.56-40.29) and inability feed orally, OR 2.68 (95% CI 1.24-5.85) were also associated. GERD exacerbation was significantly associated with weight loss in the initially GERD negative group (p=0.001).

Conclusion:

Infants with chromosomal or genetic syndromes and those who cannot attain oral feeds have poor growth, independent of GERD evaluation and therapy. Once GERD is identified and treated, its recurrence is not associated with weight loss. Those without GERD at the time of initial study who subsequently develop GERD symptoms, should be re-evaluated promptly before they show evidence of failure to thrive.

Notes:

38 – 6 minutes

PROSPECTIVE VALIDATION OF AN ABBREVIATED BEDREST PROTOCOL IN THE MANAGEMENT OF BLUNT SPLEEN AND LIVER INJURY IN CHILDREN

Shawn D. St Peter, MD[1], Susan W. Sharp, PhD[1], Saleem Islam, MD[2], Charles L. Snyder, MD[1], Ronald J. Sharp, MD[1], Walter S. Andrews, MD[1], J Patrick Murphy, MD[1], George W. Holcomb, III, MD[1], Daniel J. Ostlie, MD[1]
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Objective:

Current APSA recommendations for blunt spleen/liver injury (BSLI) entail bedrest equal to grade of injury plus one. Some institutions have questioned this protocol with retrospective data supporting shorter periods of bedrest. To validate the safety and quantify the impact of an abbreviated protocol, we instituted a prospective study with early ambulation.

Methods:

Following IRB approval, data were collected prospectively in all patients with BSLI from admission up to 8 weeks after discharge. There were no exclusion criteria and patient accrual was consecutive. Bedrest was restricted to one night for grade I & II injuries and two nights for grade ≥ III.

Results:

Between 11/2006 and 9/2009, 131 patients were admitted with BSLI. Mean age and weight were 10.4+/-4.5 years and 41.3+/-19.7 kg, respectively. Injuries included isolated spleen in 72(55%), liver only in 55(42%), and both in 4(3%). One splenectomy was required for a grade V injury. Transfusions were necessary in 24 patients(18%), with 18(13%) due to the injured solid organ. Bedrest for solid organ injury was applicable to 110 patients(84%), with a mean grade of injury of 2.6+/-1.0 and mean duration of bedrest of 1.6+/-0.6 days, resulting in 2.2+/-1.3 days of hospitalization. The need for bedrest was the limiting factor for length of stay in 86 patients (66%), for which mean grade of injury was 2.6+/-1.1 and mean duration of bedrest was 1.6+/-0.6 days, resulting in 1.8+/-0.9 days of hospitalization. There were 2 deaths, 1 from traumatic brain injury and 1 from a grade V liver injury. There were no patients readmitted for complications of solid organ injury.

Conclusions:

This prospective study has shown that an abbreviated protocol of one night of bedrest for grade I and II injuries and two nights for grade ≥ III can be safely employed, and result in dramatic decreases in hospitalization compared to the current APSA recommendations.

Notes:

39 – 3 minutes

HEPARIN-BINDING EGF-LIKE GROWTH FACTOR (HB-EGF) PROMOTES ENTERIC NEURAL CREST CELL MIGRATION

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Purpose:

Developmental defects of the enteric nervous system lead to a variety of disorders such as Hirschprung's disease and intestinal neuronal dysplasia. We have previously shown that HB-EGF exerts neuroprotective effects on injured neurons, and that deletion of the HB-EGF gene leads to enteric neuronal cell degeneration. However, the role of HB-EGF on enteric nervous system development remains unknown. This goal of this study was to assess the effect of HB-EGF on enteric neural crest cell (ENCC) migration in the developing gastrointestinal tract.

Methods:

Three methodologies were used: 1) whole mount PGP 9.5 immunohistochemistry was used to determine the extent of ENCC migration at different embryonic stages (E11.5-14.5) in HB-EGF knockout (KO) and wild type (WT) mice; 2) HB-EGF WT embryonic gut was excised en bloc at E11.5, cultured in the presence or absence of HB-EGF for 3 days, and then stained with the pan-neuronal marker PGP 9.5; 3) a chemoattractant assay was performed by culturing 2 mm segments of E11.5 WT midgut for 4 days in a collagen gel matrix containing HB-EGF soaked beads on one side and control beads at the opposite side. The outgrowth of ENCC from the midgut segment was determined by PGP 9.5 immunohistochemical staining.

Results:

ENCC migration was significantly delayed in HB-EGF KO mice compared to WT mice at E11.5-14.5 days. HB-EGF treatment of WT embryonic gut significantly promoted ENCC migration, as demonstrated by a significant increase in the ratio of ENCC migration distance towards the distal hindgut/total colon length ($82 \pm 3\%$ *vs*. $58 \pm 2\%$, $p<0.01$). Outgrowth of ENCC in WT E11.5 midgut was significantly stimulated by HB-EGF-soaked beads.

Conclusions:

HB-EGF is a potent neuronal chemoattractant factor that stimulates ENCC migration in the gut during development of the enteric nervous system. HB-EGF may represent a potential therapeutic strategy for the treatment of infants with neuro-intestinal disorders.

Notes:

40 – 3 minutes

THE MDM2 INHIBITOR NUTLIN-3A POTENTLY SUPPRESSES BOTH TUMOR ANGIOGENESIS AND TUMOR GROWTH IN NEUROBLASTOMA

Eugene S. Kim, MD, Danielle Patterson, MD, Dongbing Gao, Nikki Sikorski, Zaowen Chen, Jason S. Shohet, MD, PhD
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Purpose:

Anti-angiogenic therapy, a validated treatment in many human cancers, has not been effective in neuroblastoma in preclinical and clinical studies. Therefore, novel anti-angiogenic targets are being sought in neuroblastoma. The oncogene MDM2 is a major regulator of tumor suppressor p53 and also a regulator of hypoxia inducible factor-1α (HIF-1α). Inhibition of MDM2 has been shown *in vitro* to suppress HIF-1α and downstream vascular endothelial growth factor (VEGF), critical to tumor growth and angiogenesis. Therefore we hypothesized that MDM2 inhibition leads to decreased tumor angiogenesis and growth in an *in vivo* model of neuroblastoma by disrupting the MDM2-HIF-1-VEGF pathway.

Methods:

Human neuroblastoma cell line, SH-SY5Y, was implanted in the subrenal capsule of nude mice. Mice were treated with vehicle or Nutlin-3a (200 mg/kg/dose PO BID for 2 weeks). Mice were sacrificed, xenograft tumors preserved, and lectin perfusion angiography performed. Tumor tissue was immunostained for CD31, α-smooth muscle actin (αSMA), type-IV collagen, and TUNEL staining for apoptosis. Immunohistochemistry of xenograft tumors was quantified by digital image analysis. Statistical analysis of tumor weights was performed by Kruskal-Wallis technique and all other experiments were evaluated by Students T-test.

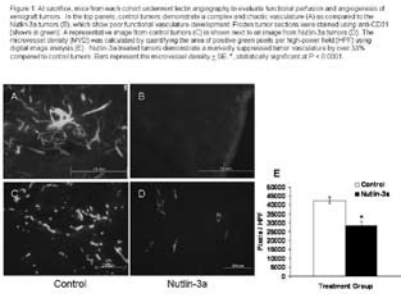
Results:

In the xenograft model of neuroblastoma, Nutlin-3a significantly suppressed tumor growth by 73% (p=0.02). Nutlin-3a treatment significantly decreased vascular perfusion (lectin angiography), microvessel density (CD31 staining, p<0.0001), vascular mural cell recruitment (αSMA staining, p<0.0001), and perivascular basement membrane development (type-IC collagen staining, p<0.0001) in xenograft tumors. Western blot analysis of Nutlin-3a-treated tumors demonstrated decreased expression of VEGF. Nutlin-3a treatment significantly increased tumor cell apoptosis and endothelial cell apoptosis.

Conclusions:

We conclude that Nutlin-3a potently suppresses tumor angiogenesis and growth in an *in vivo* model of neuroblastoma. As a therapy which may exert its effect through upregulation of p53-mediated apoptosis as well as downregulation of VEGF-mediated tumor angiogenesis, MDM2 inhibitors should be considered for use in patients with neuroblastoma.

Notes:



42 – 3 minutes

RESECTABILITY AND OPERATIVE MORBIDITY AFTER NEOADJUVANT CHEMOTHERAPY IN NEUROBLASTOMA PATIENTS WITH ENCASEMENT OF MAJOR VISCERAL ARTERIES

Barrie S. Rich, MD, Maureen P. McEvoy, MD, Natasha E. Kelly, MD, Edwin Oh, BS, Sara J. Abramson, FACR, Anita P. Price, FACR, Nai-Kong V. Cheung, MD, Brian H. Kushner, MD, Michael P. La Quaglia
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Purpose:

Image-defined major vessel encasement is a significant risk factor for surgical complications and incomplete resection for intermediate-risk tumors. However, no reports examine the impact of vessel encasement on complications or resectability in intermediate-risk or high-risk patients treated with neoadjuvant chemotherapy.

Methods:

With IRB waiver, we retrospectively reviewed medical records and the neuroblastoma database to identify 182 consecutive patients with circumferential vessel encasement who underwent resection between 1991 and 2008. Specifically, we evaluated resection rates and surgical morbidity, mortality, and outcome. All patients received neoadjuvant chemotherapy. We graded complications using the Clavien system.

Results:

Median age at diagnosis was 3.2 years. Stage distribution was 82%, 17%, and 1% for stages 4, 3, and 2B, respectively. 74% of primary tumors were adrenal, 26% retroperitoneal. MYCN amplification was observed in 20%. Vessel encasement included renal artery, SMA, or coeliac axis alone in 36, 7, and 5 patients, respectively. Encasement of both the renal artery and coeliac axis was found in 1 patient, renal artery and SMA in 5 patients, and coeliac axis and SMA in 19 patients. All 3 vessels were encased in 36 patients. Gross total resection rate was 95%. No operative or post-operative deaths occurred. In the 37 patients with complications (20%), only 1 was grade IVb, while 2 were grade IIIb, 1 grade IIIa, 25 grade II, and 8 grade I. Overall 5-year survival was 70.5% and was strongly correlated with gross total resection ($p<0.003$). No correlation existed, however, between gross total resection and encasement of the renal artery, coeliac axis, or superior mesenteric artery.

Conclusions:

Encasement of major visceral arteries in neuroblastoma patients after neoadjuvant chemotherapy does not preclude gross total resection. Although the overall complication rate was 20%, only 4 were grade III or IV. No mortality was observed.

Notes:

42 – 6 minutes

LOCAL CONTROL, SURVIVAL, AND OPERATIVE MORBIDITY AND MORTALITY AFTER RE-RESECTION AND INTRA-OPERATIVE RADIATION THERAPY FOR RECURRENT OR PERSISTENT PRIMARY HIGH-RISK NEUROBLASTOMA

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Purpose:

Patients with locally recurrent or persistent neuroblastoma are difficult to treat. Intraoperative radiation therapy (IORT) may improve local control in these patients. We describe our experience using IORT in patients with high-risk stage 3 or 4 neuroblastoma and locally recurrent or persistent primary tumors.

Methods:

With IRB waiver, we retrospectively determined local recurrence, survival, and morbidity in patients with recurrent/persistent high-risk stage 3 or 4 neuroblastoma in the primary site who received IORT using rapid dose-rate brachytherapy at our center between April 2000 and September 2009. We identified 42 consecutive patients, 41 for whom local recurrence data was available.

Results:

The median age at surgery was 5.6 years and follow-up after IORT 12 months. All patients had high-risk neuroblastoma, 12% stage 3 and 88% stage 4. All had received prior chemotherapy or surgery, while 88% had been treated with external beam radiation therapy (EBRT) with a median dose of 21.6 Gy. 37% had tumors with MYCN amplification. The rate of gross total resection of the recurrent/persistent primary tumor was 95%, and there were no operative or post-operative deaths. The median dose of IORT was 15 Gy (range, 8-20 Gy). Post-operative surgical complications occurred in 7 patients, including 5 cases of hydronephrosis, 1 bowel fistula, and 1 perforation. 60% of patients had no evidence of local recurrence 2 years after IORT (27% if MYCN amplified versus 68% for non-amplified tumors; p= N.S.). Overall survival was 35% at 2 years (10% if MYCN amplified versus 46% for non-amplified; $p<0.008$).

Conclusions:

Re-resection and IORT of locally persistent or recurrent primary tumors results in a high rate of local control with acceptable morbidity and mortality and overall survival. Though outcome is adversely affected by MYCN amplification, this approach should be strongly considered in this very high-risk group of patients.

Notes:

43 – 3 minutes

INHIBITION OF CYCLO-OXYGENASE 2 REDUCES EWING'S SARCOMA LUNG METASTASIS DURING VEGF BLOCKADE BY ALTERING EXPRESSION OF INFLAMMATORY PATHWAY GENE SETS

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Purpose:

Pulmonary metastasis is a leading cause of death in Ewing's sarcoma (EWS). We have reported that vascular endothelial growth factor (VEGF) blockade partially reduced lung metastasis in experimental EWS. COX-2 activity broadly influences both vascular remodeling and inflammatory states, each implicated in tumor metastasis. We hypothesized that SC236 would further limit metastasis in VEGF-blocked EWS xenografts, and that this reduction would be associated with altered expression of COX-2 or inflammation-related genes.

Methods:

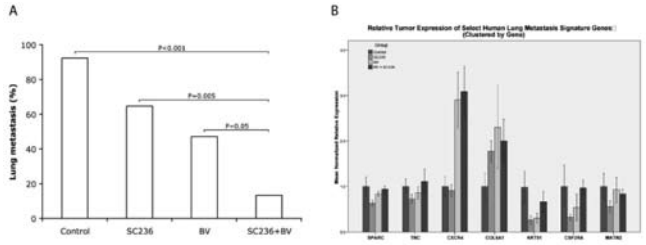
10^6 SKNEP1 EWS cells were implanted intrarenally in athymic mice (N=81). Mice were treated with SC236 (N=19), anti-VEGF antibody (bevacizumab, BV; N=20), SC236+BV (N=22), or vehicle (N=20). Mice were killed at 6 weeks, lungs were studied, and tumor RNA isolated. (1) Real-time PCR was performed for 7 genes in a validated COX-2-containing lung metastasis signature (LMS); and (2) HGU133A-Affymetrix microarrays analyzed, using a 67-geneset matrix constructed by querying the Molecular Signatures Database (5,542 total sets) for inflammation-related pathways, and Gene Set Expression Analysis (GSEA). Differential expression was assessed statistically using GSEA, GenePattern tools, and odds ratio calculation.

Results:

Metastatic incidence was reduced (47% to 13%, BV vs. BV+SC236-treated animals, p=0.0001). (Figure A) No significant differences between BV and BV+SC236-treatment were found for LMS genes. (Figure B) Twenty gene sets were significantly repressed in SC236+BV-treated vs. BV-treated tumors by GSEA. Leading-edge analysis indicated clustered overlapping subsets that predominantly included molecules involved in inflammation-pathway signaling.

Conclusion:

Expression of a lung metastasis gene set was not altered by the addition of SC236 to BV treatment, whereas GSEA analysis indicated broad significant repression of inflammation-related gene sets. Cytokines included in the repressed gene sets (eg IL4, IL12A and CCL24) have recently been implicated in prometastatic activity of tumor-associated monocytes and lung inflammation. Thus, SC236 may act in the context of VEGF inhibition by limiting the ability of tumors to induce an inflammatory state that facilitates metastasis.



(A) Reduction in lung metastasis incidence was seen when comparing BV and SC-236+BV treated mice (47% vs. 13%).
(B) No significant differences in BV (tan) and BV+SC-236 (purple) treatment were found for LMS genes.

44 – 3 minutes

MESENCHYMAL STEM CELLS (MSCs) DEPENDENT REGRESSION OF PULMONARY METASTASIS IS MEDIATED BY INHIBITION OF PLATELET DERIVED GROWTH FACTOR RECEPTOR-BETA (PDGFR-B)

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Purpose:

The overall survival for metastatic Ewing's sarcoma (ES) is ~15% and has not changed in over 15 years. The most common and fatal progression found in ES is pulmonary metastasis. Bone marrow derived mesenchymal stem cells (MSCs) have been found in preclinical models to induce regression of some human malignancies. There are no reports on the use of MSCs in the treatment of ES. We found, intravenous infusion of MSCs causes regression of pulmonary metastasis in ES and sought to understand the mechanism. MSCs preferentially 'home' to the lung compared to other organs. PDGFR-B is expressed in ES and promotes cell motility and invasion. We hypothesize that MSCs directly inhibit PDGFR-B and therefore the growth of pulmonary metastasis

Methods:

Increasing concentrations of human MSCs were co-cultured with human TC-71 ES cells. Protein levels of PDGFR-B and downstream regulators, pAkt, and mammalian target of rapamycin (mTOR) levels were measured after the addition of Rapamycin and compared to those cells cultured without Rapamycin. Normal lung epithelial cells were used as control. MCSs were then infused into an orthotopic xenograft model of ES after tumors were established.

Results:

By cell-cell contact, MSCs inhibited pAKT at a 5 to 1 ratio of MSCs to ES cells. Total Akt levels were not affected. Protein levels of pAKt were restored with the addition of Rapamycin. Further, MSCs inhibited PDGFR-B in dose dependent fashion. MSC infusion into our xenograft model induced regression of pulmonary metastasis.

Conclusions:

Human MSCs inhibit PDGFR-B by cell-cell contact. MSC inhibition of pAkt is restored by the addition of Rapamycin, indicating MSC inhibition of pAkT is mTOR dependent. This dual inhibition by MSCs describes a novel mechanism of growth inhibition of pulmonary metastasis in ES.

Notes:

45 – 3 minutes

SELECTIVE INHIBITION OF CYCLOOXYGENASE-2 (COX-2) SUPPRESSES METASTATIC DISEASE WITHOUT AFFECTING PRIMARY TUMOR GROWTH IN A MURINE MODEL OF EWING'S SARCOMA

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Purpose:

The mammalian target of rapamycin (mTOR) is a central signaling mechanism found in most human cancers and directly stimulates angiogenesis via a COX-2-mediated mechanism. mTOR suppression by rapamycin inhibits tumor growth and neovascularization but not lung metastases in a murine model of Ewing's sarcoma. We hypothesize that combining a selective COX-2 antagonist (celecoxib) with rapamycin would decrease lung metastases.

Methods :

Ewing's sarcoma cells (SK-NEP-1) were surgically implanted into the left kidney of athymic mice (n=40). The mice were divided into 4 treatment groups (control, rapamycin only, celecoxib only and combination). All mice were sacrificed at 6 weeks and the organs were harvested. Primary tumors were weighed and vasculature was examined using lectin angiography and immunohistochemistry for endothelial cells. Lung metastases were examined with a pediatric pathologist using H&E and immunohistochemistry for Ewing's markers. Tumor weights were analyzed with the unpaired t-test and lung metastases with factorial design.

Results:

Mean primary tumor weights were significantly reduced in the rapamycin treated groups (control 3.5 grams ± 0.58 SEM; rapamycin only, 1.02 grams ± 0.2, $p<0.001$; combination, 0.81 grams ± 0.26, $p<0.001$) but not in the celecoxib only group (3.6 grams ± 1.2). Lectin angiography and immunostaining for endothelial markers (platelet endothelial cell adhesion molecule-1, alpha smooth muscle actin, collagen-4) showed markedly decreased vascularity in the rapamycin treated groups but not in the celecoxib only group. Lung metastases (CD99 positive) in the celecoxib treated groups showed statistically significantly fewer metastases than non-celecoxib treated groups. (control 9/10 + for lung metastases; celecoxib only 3/9 +, $p<0.02$; combination 3/10, $p<0.02$; rapamycin only 7/10 +, NS).

Conclusion:

Celecoxib prevents lung metastasis in a murine model of Ewing's sarcoma with no effect on tumor size or neovascularization. COX-2 may represent a future potential target for metastatic disease prevention.

Notes:

46 – 6 minutes

TREATMENT AND OUTCOME OF PEDIATRIC PATIENTS SUFFERING FROM PERINEAL RHABDOMYOSARCOMA- RESULTS FROM THE COOPERATIVE SOFT TISSUE SARCOMA STUDIES CWS-86, -91, -96 AND -2002P

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Purpose:

Perineal rhabdomyosarcoma (PRMS) is uncommon. The aim of this study was to analyze the clinical course, treatment and outcome in patients suffering from PRMS treated within the Cooperative Soft Tissue Sarcoma Studies CWS-86, -91, -96 and -2002P.

Methods:

Patients with RMS were enrolled in the CWS-86, -91, -96, and -2002P trials, of which 34 had PRMS. All patients received three cycles of neoadjuvant chemotherapy. At week 9, patients were reassesed using CT- or MRI-scan. Depending on the tumor size, age, and response to chemotherapy, local therapy, consisting of radiotherapy and / or surgery, was initiated. After local therapy, adjuvant systemic therapy was continued.

Results:

Patient`s age ranged from 0 - 17 years (mean 10 y ± 6). 9 patients had embryonal RMS (ERMS), 25 had alveolar RMS (ARMS). Median follow up was 39.2 months ± 42.7. 59% of patients presented with advanced stage neoplasm (IRS-stage III). 9 patients (all ARMS) had metastatic disease. 18 patients had locoregional lymph node involvement (ERMS: 2, ARMS: 16). 5/9 patients with ERMS and 16/25 with ARMS received radiotherapy (32-64 Gy). Resection was performed in 14 patients (ERMS: 4, ARMS: 10). 10 patients underwent RT and surgery (ARMS: 7, ERMS: 3). Local relapse occurred in 2 patients with ERMS and 6 patients with ARMS. 7/9 patients with ERMS and 6/25 patients with ARMS are in first complete remission.

Conclusions:

Similar to RMS in other sites, histologic subtype seems to be to most important predictor of outcome in PRMS as well. Patients with ERMS seem to have a better outcome due to the higher failure rate in patients with ARMS.

Notes:

47 – 3 minutes

THYROID CARCINOMA IN CHILDREN IS BEST DIAGNOSED BY SURGICAL EXCISION

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Purpose:

Thyroid nodules in children are uncommon but often present an increased risk of malignancy in comparison to adults. Multiple diagnostic modalities are frequently employed to characterize thyroid nodules including ultrasound, radionuclide scans, fine needle aspiration (FNA), thyroid function tests, and evaluating patient demographics. We chose to evaluate if these diagnostic modalities influence treatment or signify a tendency for a nodule to represent a malignant lesion.

Methods:

A retrospective review of patients <21 years of age who underwent partial or total thyroidectomy from 2004-2009 was performed. Patient demographics, preoperative workup, type of procedure performed, and pathology results were recorded. Five patients with a history of MEN were excluded.

Results*:*

Forty-five patients underwent partial or complete thyroidectomy. Nine patients (20%) had malignant lesions, all of which were papillary carcinoma. The majority of patients were female (91%) with a similar average age for malignant and benign lesions (14.1 years and 14.2 years respectively). 91% of patients underwent pre-operative imaging with ultrasound, CT scan, or 1-123 scan. The average thyroid nodule size was 2.7 cm for malignant lesions and 2.9 cm for benign lesions. There were no hearld radiologic findings indicating a malignant lesion. FNA was performed in 49% of patients (n=22), 4 were interpreted as malignant and there was 1 false negative result, giving a sensitivity of 80 % and specificity of 100%.

Conclusions:

Thyroid disease is an uncommon occurrence predominantly in females. Other than an FNA indicating a malignancy, there does not appear to be any value to extensive preoperative workup, nor can patient risk be stratified based upon age, sex, or radiographic characteristics. We conclude that there is minimal utility in an extensive pre-operative work-up in a child with a thyroid nodule. Definitive diagnosis is best made by a surgical pathologist.

Notes:

48 – 3 minutes

IMPROVED SURVIVAL WITH LYMPH NODE SAMPLING IN WILMS TUMOR

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Purpose:

Adequate lymph node sampling is crucial to properly stage and treat Wilms tumor (WT) patients. We sought to determine the impact of number of lymph nodes examined on survival for children with WT.

Methods:

Data from the Surveillance, Epidemiology, and End Results (SEER) and Florida Cancer Data System (FCDS) were combined and queried for all patients < 20 years of age with WT (IRB approved).

Results:

Of 1,805 WT patients, 1,444 were identified who had lymph node (LN) data available following surgical resection. The majority of patients were 1 - 4 years old (63%) with a mean age of 3.3 ± 2.8 years for the entire cohort. Most patients were female (53%), white (78%), and non-Hispanic (78%). Only 40% (n=573) of patients received radiation therapy. A total of 380 patients (26%) had 0 LN sampled, while 714 (49%) had 1-5 LN, 211 (15%) had 6-10 LN, and 139 (10%) had >10 LN. The number of LN examined was not related to patient age, gender, race, or ethnicity. Overall 5-year survival for the cohort was 90.9%. By univariate analysis, 5-year survival was significantly lower for patients who had 0 LN sampled (87% vs 91% 1-5 LN, p=0.008; 93% 6-10 LN, p=0.01; 95% >10 LN, p=0.016). Multivariate analysis revealed no differences in outcome due to age, gender, race, or ethnicity, but patients not requiring radiation therapy had a lower mortality risk (HR 0.518, p=0.001). In addition, a survival advantage was noted for patients having 1-5 LN (HR 0.600, p=0.016), 6-10 LN (HR 0.521, p=0.048), and >10 LN (HR 0.403, p=0.039) compared to patients with 0 LN examined.

Conclusions:

Failure to biopsy lymph nodes during surgical resection of WT not only increases the risk of local recurrence due to understaging and inadequate adjuvant therapy, but is also and independent prognostic indicator of lower survival.

Notes:

49 – 3 minutes

OUTCOME OF RESECTION FOR PRETEXT III AND IV HEPATOBLASTOMA

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Children's Memorial Hospital, Chicago, IL, USA

Purpose:

It has been proposed that children with large hepatoblastomas may have better outcomes with transplantation rather than resection. This study evaluated the results of non-transplant hepatectomy in children with PRETEXT III and IV tumors who might otherwise have undergone transplantation.

Methods:

A review of all children with PRETEXT III or IV hepatoblastomas who underwent resection from 1998 to 2009 was performed. PRETEXT staging was determined by a radiologist blinded to the outcome of the patients.

Results:

Eighteen children (11 boys) were identified with a median age at diagnosis of 8 months. Four had metastasis at the time of diagnosis (3 pulmonary, 1 omental). Sixteen received neoadjuvant chemotherapy. Four patients had PRETEXT IV and 14 had PRETEXT III tumors at the time of surgery. Overall survival after resection was 89% with a median follow-up duration of 48 months. Twelve patients with PRETEXT IV (n=4) or centrally located PRETEXT III (n=8) tumors, and no metastatic disease at the time of definitive surgery, were potential transplant candidates. Resections in these twelve included: trisectionectomy in 8, mesohepatectomy in one, and non-anatomic extended hepatectomy in 3 patients, including 2 who required separate concurrent wedge resections. Ten of 11 (91%) potential transplant patients who were resected and have more than 1 year of follow up (median 51 months) are alive with no recurrence. The one patient who died had unfavorable rhabdoid histology and may have been a poor transplant candidate.

Conclusions:

We report 89% survival after aggressive non-transplant resection in all children with PRETEXT III and IV hepatoblastomas and 91% survival in patients who were potential transplant candidates. Deaths occurred in patients with unfavorable histology or metastatic disease. Surgeons experienced with advanced hepatobiliary techniques are justified in employing an aggressive non-transplant approach to the surgical management of selected patients with these large tumors.

Notes:

NEW TECHNOLOGY

50 – 5 minutes

PERCUTANEOUS FETAL ENDOSCOPIC TRACHEAL OCCLUSION FOR ISOLATED CONGENITAL DIAPHRAGMATIC HERNIA WITH UNFAVOURABLE PROGNOSIS.

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[1]University Hospitals Leuven, Leuven, Belgium, [2]King's College Hospital, London, United Kingdom, [3]Hospital Clinic, Barcelona, Spain

Purpose:

To describe the operative technique and summary of results of current experience with fetoscopic endoluminal occlusion of the trachea (FETO) in congenital diaphragmatic hernia (CDH). Cases are selected based upon measurement of an observed/expected lung-to-head ratio <27% and liver herniation.

Methods:

FETO is done under local or loco-regional maternal anesthesia and fetal administration of fentanyl (15 µg/kg), pancuronium (0.2 mg/kg) and atropine (20 µg/kg). External version helps position the baby. Instruments consist of a 3.3 mm cannula, 1.3 mm operative fetoscope within 3 mm sheath (Karl Storz). A detachable latex balloon (GVB 16, Nfocus Neuromedical Inc, Menlo Park, Ca) filled with 0.8 mL isotonic contrast is positioned under direct vision between the carina and vocal cords. Removal of the balloon is planned around 34 weeks either by ultrasound guided puncture, fetoscopy or in an emergency by EXIT following maternal bethametasone administration. A purpose designed balloon balloon extraction set has been developed for use during EXIT.

Results:

By 2008 210 cases (175 left, 34 right and 1bilateral) were done a median of 27.1 (range:23.0-33.3) weeks. The median duration was 10 (range:3-93) min. Preterm prelabor rupture of membranes (PPROM) within 3 weeks occurred in 16.7%. 48.0% were discharged alive from the hospital. Based on a prenatal prediction algorithm FETO increased survival from 24.1% to 49.1% for left, and from 0% to 35.3% ($p<0.001$) right sided cases (Jani et al, 2009).

Conclusions:

FETO in severe CDH is associated with a high incidence of PPROM and preterm delivery but an improvement in survival compared to earlier data from the antenatal CDH registry.

Notes:

51 – 5 minutes

PRE-CLINICAL REGULATORY VALIDATION OF AN ENGINEERED DIAPHRAGMATIC TENDON MADE WITH AMNIOTIC MESENCHYMAL STEM CELLS

Christopher G. Turner[1], Justin D. Klein[1], Shaun A. Steigman[1], Myriam Armant[2], Grace A. Nicksa[1], David Zurakowski[1], Jerome Ritz[2], Dario O. Fauza[1]
[1]Children's Hospital Boston, Boston, MA, USA, [2]CBR Institute for Biomedical Research, Boston, MA, USA

Purpose:

Engineered diaphragmatic repair has been proven viable experimentally. Under FDA oversight, we aimed at examining long term local and systemic safety, as well as efficacy, of an engineered diaphragmatic tendon graft built within clinically admissible guidelines, as a regulatory prerequisite for human trials of this methodology.

Methods:

After IACUC approval, newborn lambs with an experimental left diaphragmatic defect (n=27) were equally divided in 3 groups based on the type of implant used for repair, namely: a standard teflon patch; a clinically allowable, tri-layered composite acellular bioprosthesis; or the same bioprosthesis seeded with autologous amniotic mesenchymal stem cells expanded under Good Manufacturing Practice guidelines (not labeled, per the FDA). Comprehensive hematologic and serum chemistry tests were performed in all animals at various time points post-operatively. Animals from each group were euthanized at 7, 10 and 14 months (ovine adulthood) post-implantation, for multiple additional safety and efficacy analyses. Statistical comparisons were by exact logistic regression, two-way multivariate ANOVA and the F-test, as appropriate ($P<0.05$).

Results:

There were no deaths. None of the blood tests or full body autopsy specimens showed any abnormality. There was a significantly higher re-herniation rate ($P=0.014$) and failure (re-herniation or eventration) rate ($P=0.012$) in the acellular bioprosthetic group compared to the engineered group, with no significant differences between acellular and teflon implants. The modular tensile strength and total collagen levels were significantly higher in the engineered than in the acellular bioprosthetic implants ($P=0.016$ and 0.003, respectively). Lysozyme and myeloperoxidase immunohistochemistries were unremarkable in all grafts.

Conclusions:

In a large animal model, diaphragmatic repair with a clinically viable autologous tendon engineered with amniotic mesenchymal stem cells leads to improved outcomes when compared with an equivalent acellular bioprosthesis, with no local or systemic adverse effects. Clinical trials of engineered diaphragmatic repair appear practicable within regulatory guidelines.

Notes:

52 – 5 minutes

CARDIAC OUTPUT MEASUREMENTS IN NEONATES, ONE-HALF KILOGRAM TO FOUR KILOGRAMS

Andrew P. Bozeman, MD, Misael Rodriguez, MD, Joseph M. Van De Water, MD, Barbara Weaver, RN, Bao Ngoc T. Ho, BS, Robert L. Vogel, PhD, Don K. Nakayama, MD
Mercer University School of Medicine/ Medical Center of Central Georgia, Macon, GA, USA

Purpose:

To date there has been no practical means of measuring Cardiac Output (CO) in small children weighing less than ten kilograms. To fill this significant void in our hemodynamic assessment, especially in the growing numbers of premature neonates a new non-invasive device, requiring only the placement of four standard infant electrodes has been developed. It relies on the differences in red cell orientation between systole and diastole, to calculate Stroke Volume (SV).

Methods:

Obtaining one of these devices, Aesculon® (Cardiotronic, La Jolla, CA), we proceeded to do a thorough evaluation. With Institutional Review Board approval of this prospective study, as well as parental consent, measurements were made on infants admitted to our Neonatal Intensive Care Unit (NICU) with weights above one-half Kg and APGAR scores greater than three at five min.

Results:

Our analyses included 200 measurements on 102 premature and term infants. Ease of application with both patient acceptance and operator satisfaction was high. Using twenty infants and five operators, reproducibility was good with variance being < 1% and < 0.5% for inter- and intra-operator measurements. Utilizing measurements from 75 stable neonates with no structural cardiac defects by echocardiography CO and SV were compared to Weight (wt.), Body Surface Area (BSA) and Body Mass Index (BMI). The best correlations were with wt. ($r = 0.89$, $p < 0.001$ and $r = 0.93$, $p < 0.001$ for CO and SV).

Conclusion:

The easily obtained, reproducible, weight-related, CO values using this bedside device should prove very useful in our care of the NICU patient.

Notes:

53 – 5 minutes

DETERMINATION OF THE CELLULAR ORIGIN OF VASCULAR NEOTISSUE IN TISSUE ENGINEERED VASCULAR GRAFTS (TEVG)

Gustavo A. Villalona, MD[1], Rajendra Sawh-Martinez, BS[2], Tamar Mirensky, MD[3], Narutoshi Hibino, MD, PhD[2], Tai Yi, BS[2], Adam Shoffner, BS[2], Edward McGillicuddy, MD[3], Toshiharu Shinoka, MD, PhD[1], Christopher Breuer, MD[1]
[1]Yale New Haven Children's Hospital, New Haven, CT, USA, [2]Yale School of Medicine, New Haven, CT, USA, [3]Yale New Haven Hospital, New Haven, CT, USA

Purpose:

Tissue engineered vascular grafts (TEVG) hold great promise for use in children because of their growth potential. The purpose of this investigation is to determine the fate of seeded cells, and track the source and identity of cells that form the medial and intimal layers of the neovessel as it develops.

Methods:

(A)TEVG were seeded using green fluorescent protein (GFP+) bone marrow mononuclear cells (BM-MNC) on a biodegradable scaffold and implanted as an IVC interposition grafts in a C57BL/6 mice and tracked up to 14 days. (B) C57BL/6 chimeric mice were generated by rescuing lethally irradiated animals with bone marrow transplantation of GFP+ BM-MNC from male transgenic mice. Scaffolds were implanted as an Inferior Vena Cava (IVC) interposition grafts and harvested up to 6 months post surgery. (C) Composite vascular grafts composed of a GFP+ male mouse IVC anastomosed to a TEVG were implanted in a GFP- female host. Grafts were harvested up to 6 months after implantation and tracked using Fluorescence In-Situ Hybridization (FISH) for Y-chromosome (YChr) and GFP+ staining.

Results:

GFP+ seeded cells were replaced by host cells one-week after implantation. BM-derived macrophages (F4/80+ GFP+ YChr+) were abundant within the graft wall as determined by immunofluorescence, which decrease over time. Neomedia and neointima cells were all GFP- YChr, indicating that the cells expressing vascular cellular markers arose from the recipient. The composite graft further confirmed the vascular origins of cells in the neovessel group were many SMA+ and vWF+ were both GFP+ and YChr+ proximal to the GFP+ male IVC.

Conclusion:

Our results suggest that TEVG transform from a seeded scaffold into a neovessel that resembles a native vessel. The resulting neovessel is not derived from the seeded cells but instead through an immune mediated process of in-growth of endothelial and smooth muscle cells from the surrounding native vessel.

IACUCapprovalnumber: 2007-11160

Notes:

54 – 5 minutes

TOWARDS EFFECTIVE PEDIATRIC MINIMALLY-INVASIVE SURGICAL SIMULATION

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[1]Phoenix Integrated Surgical Residency, Phoenix, AZ, USA, [2]Banner Good Samaritan Simulation Education and Training Center, Phoenix, AZ, USA, [3]Arizona State University Department of Biomedical Informatics, Tempe, AZ, USA, [4]Phoenix Children's Hospital, Phoenix, AZ, USA

Purpose:

Simulation is increasingly being recognized as an important tool in the training and evaluation of surgeons. Currently there is no simulator that is specific to pediatric minimally-invasive surgery (MIS). An important technical difference between adult and pediatric MIS is the degree of motion scaling: smaller instruments and areas of dissection under greater optical magnification require finer, more precise hand movements. We have designed a simulator that alters motion scaling in an attempt to mimic operating conditions in pediatric patients and tested its validity by comparing pediatric and general surgeons.

Methods:

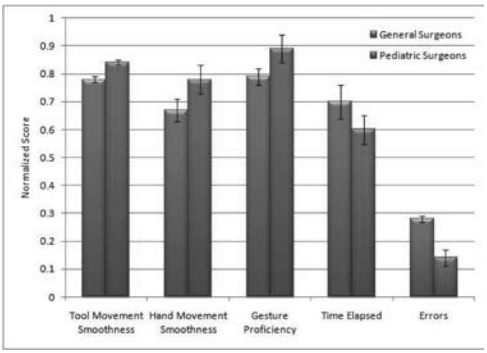
We programmed a virtual reality simulation of intracorporeal suturing with two modes in which the degree of motion scaling is changed to mimic the visiomotor conditions of either adult or pediatric MIS. The study participants, consisting of 9 pediatric and 6 general surgeons, interacted with the simulator via a joystick capable of life-like haptic feedback and completed multiple runs in both modes. The simulation program measured tool movement smoothness, time elapsed, and penetration errors as performance metrics. Additionally, motion-sensing gloves were worn to measure hand movement smoothness and gesture level proficiency, which is calculated using an algorithm of deconstructed hand and tool movements. These variables were then analyzed using ANOVA.

Results:

A graphical representation of the normalized metrics for the two groups is shown. For all measures excepting time elapsed, pediatric surgeons demonstrated statistically-significant superiority in psychomotor proficiency across the study ($p<0.05$).

Conclusion:

We demonstrate that pediatric surgeons possess unique MIS skills concordant with the visiomotor challenges they face in their practices. The design of our simulator is validated as a reproduction of pediatric MIS by its ability to evoke a difference between the two groups. This concept should be used in the development of specific simulators for creative new applications in the advancement of pediatric surgery.



Notes:

55 – 5 minutes

EXPERIENCE WITH DIAPHRAGM PACING IN REPLACING MECHANICAL VENTILATORS IN CHILDREN

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Purpose:

The diaphragm pacing system (DPS) has been shown to successfully replace mechanical ventilators for adult tetraplegics with chronic respiratory insufficiency. Spinal cord injury in young children is rare. However, younger children have a disproportionately high percentage of cervical involvement. Here we report our experience with DPS in children.

Methods:

Prospective, nonrandomized, controlled, interventional trials under FDA and/or IRB approval for use of DP. DP involves outpatient laparoscopic diaphragm motor point mapping to identify the optimum site where stimulation will cause maximum diaphragm contraction. Two percutaneous intra-muscular electrodes were implanted in each hemi-diaphragm and diaphragm conditioning ensues through with a programmed pacing unit to maximize diaphragm movement for respiration.

Results:

A total of 4 patients at one center were successfully implanted with the DPS system and weaned or being weaned from the ventilator from December 2008 to September 2009. There are two males and two females. The average age is 8.5 years with the range of 7 years to 10 years. The average weight was 26 Kg with the range of 15 to 36.3 Kg. The average tidal volume on ventilators was 375 with the range of 200c to 500cc. Length of time mechanical ventilation ranged from 1.5 years to 7 years. All patients tolerated the implantation procedure. Surgical times and anesthesia times were similar to the adult population. There were no operative or post operative complications. Three patients were discharged home a few hours after surgery. DPS was able to provide tidal volumes meeting basal requirements. One child is full time pacing with three in the conditioning process.

Conclusion:

The diaphragm pacing system can be successfully utilized in the pediatric population.

Notes:

PODIUM

56 – 3 minutes

MATERNAL SILDENAFIL DECREASES PULMONARY ARTERIAL WALL THICKNESS IN A FETAL OVINE DIAPHRAGMATIC HERNIA MODEL

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Background:

Lungs in infants with congenital diaphragmatic hernia (DH) are characterized by abnormal vasculature. Sildenafil citrate, a PDE5 inhibitor, has beneficial effects on pulmonary vascular remodeling and vasoreactivity and is used to treat pulmonary hypertension in pediatric and adult populations.

Purpose:

The purpose of this study was to assess the effects of prenatal administration of sildenafil in a fetal ovine DH model. We hypothesized that sildenafil would reverse the pulmonary vascular stigmata and physiologic severity of DH.

Methods:

After IACUC approval, timed pregnant ewes with twin gestations were divided into treatment (sildenafil 100mg mg daily) and non-treatment groups. In both groups, DH was created in 1 twin at 70 days gestations using established techniques. Maternal sildenafil administration was begun the day of DH. At 120 days, lambs were delivered by caesarean section and resuscitated for 2 hours. Physiologic parameters including arterial blood gases and oxygen requirements were recorded. After lamb sacrifice, lung vascular casting with barium was performed followed by morphometric analysis. Comparisons were made using Student's T-test and ANOVA.

Results:

Sildenafil was well-tolerated by pregnant ewes. There were no differences found in physiologic parameters between groups during the resuscitation. Whereas there was no difference in pulmonary vessel number between groups, lambs with DH who had received sildenafil had significantly decreased medial arterial wall thickness when compared to untreated lambs with DH (121 ± 70 vs. 170 ±63 micrometers, p<0.05, Figure 1)

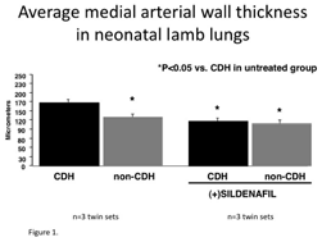

Figure 1.

Conclusions:

Based on this pilot study, prenatal administration of sildenafil may improve pulmonary vascular remodeling in the setting of congenital diaphragmatic hernia. Further study with a larger cohort of animals is underway.

Notes:

57 – 3 minutes

SERIAL AMNIOINFUSIONS PREVENT FETAL PULMONARY HYPOPLASIA IN A LARGE ANIMAL MODEL OF OLIGOHYDRAMNIOS

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Purpose:

Severe neonatal pulmonary hypoplasia incurs mortality rates approaching 71-95%. We sought to determine the utility of serial amnioinfusions through a subcutaneous implanted intra-amniotic catheter to prevent pulmonary hypoplasia in fetal obstructive uropathy.

Methods:

Fetal lambs (n=32) were divided into three groups at 94-100 days gestation (term=145 days). Group I (n=12) underwent a sham operation. Group II (n=15) underwent a complete urinary tract obstruction via ligation of the urachus and urethra in males, or bladder outlet in females with the placement of a subcutaneous tunneled intra-amniotic port-a-cath® and received no amnioinfusions. Group III (n=5) underwent creation of a complete urinary tract obstruction with a port-a-cath® placed as described in group II and received tri-weekly timed, serial amnioinfusions. All surgeries, treatments were done in accordance with IACUC approval. Survivors had serial ultrasounds and were sacrificed at comparable times. Lung tissue was analyzed by lung volume body/weight ratios and stereology. Statistical analysis was by ANOVA and Bonferroni comparisons, with significance set at $p<0.05$.

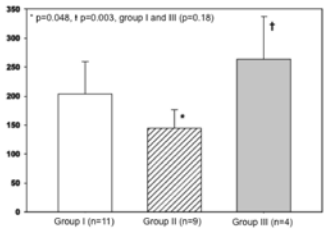
Results:

Overall fetal survival was 81.3% (26/32); 2 survivors were excluded due to infection. Ultrasound confirmed survival, presence of obstructive uropathy, and reconstitution of amniotic fluid in group III after infusion. At necropsy obstructed fetuses showed smaller lungs than treated and control animals on gross examination. Lung volume/body weight ratios were statistically significant between groups p=0.002, Bonferroni comparisons confirmed these findings as seen in Figure 1. Group I and group III showed comparable lung volume/body weight ratios, which were significantly different from group II. Airspace fractions were comparable between group I and group III (average=0.53 and 0.55 respectively) though both significantly greater than group II (average=0.48) ($p<0.001$).

Conclusions:

Serial amnioinfusions through an intra-amniotic port-a-cath® prevented pulmonary hypoplasia in an ovine model of complete obstructive uropathy. The use of an easily accessible device for amnioinfusions may be a viable option to treat oligohydramnios.

Figure 1. Lung Volume Body Weight Ratios



Notes:

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ENDOTHELIAL-DERIVED EPOXYEICOSATRIENOIC ACIDS PROMOTE COMPENSATORY LUNG GROWTH AFTER UNILATERAL PNEUMONECTOMY IN TRANSGENIC MICE

Hau D. Le, MD[1], Darryl C. Zeldin, MD[2], Mark W. Kieran, MD, PhD[1], Vincent E. de Meijer, MD, MSc[1], Matthew L. Edin, PhD[2], Craig R. Lee, PhD[3], Jo-Ann Vergilio, MD[1], Bruce D. Hammock, PhD[4], Mark Puder, MD, PhD[1], Dipak Panigrahy, MD[1]
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Background:

Epoxyeicosatrienoic acids (EETs) stimulate angiogenesis, in part via the VEGF signaling network. EETs are lipid mediators produced by cytochrome P450 epoxygenase CYP2C8. EETs in turn are metabolized by soluble epoxide hydrolase (sEH) to dihydroxyeicosatrienoic acids. Compensated lung growth is dependent on VEGF-induced angiogenesis. Thus, we hypothesize that endothelial cells (ECs) stimulate lung growth via production of EETs.

Methods:

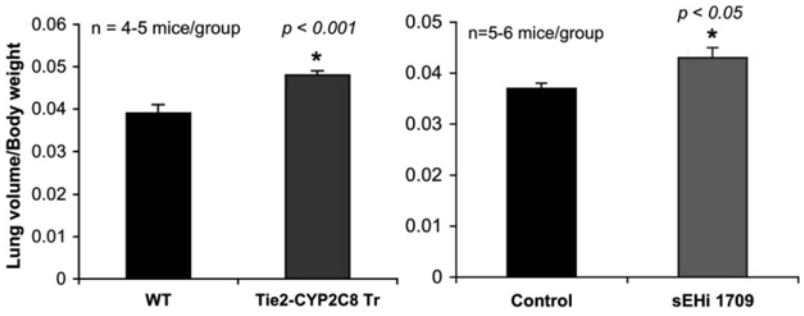
Transgenic (Tg) mice with EC-specific overexpression of CYP2C8 (Tie2 promoter-driven) were used to study the consequence of increased EETs on lung growth following left pneumonectomy. To confirm the effects of CYP2C8 overexpression, EET levels were increased by inhibition of soluble epoxide hydrolase (sEH). Morphometric analysis of the lungs was done on H&E-stained lungs. Cellular proliferation index was determined using BrdU immunostaining.

Results:

EC-specific overexpression of CYP2C8 promoted contralateral lung growth following unilateral pneumectomy. Lung volume/body weight and lung volume on day 4 post-pneumonectomy were increased by 23% ($p<0.001$) and 16% ($p<0.05$) in Tg mice compared to WT mice. In contrast, there was no significant change in baseline lung volume/body weight ratio after sham operation. The sEH inhibitor 1709 stimulated lung growth/body weight and lung volume by 14% ($p<0.05$) and 12% ($p<0.05$) on day 4 post-pneumonectomy compared to vehicle-treated mice.

Conclusions:

CYP2C8 overexpression in EC is sufficient to stimulate lung growth and may be mediated by an EET-mediated mechanism. Production of EETs in the endothelium may thus be a critical regulator of lung growth. Our results offer a mechanistic rationale for evaluating sEH inhibitors as novel therapeutics for pulmonary diseases related to lung immaturity, such as congenital diaphragmatic hernia and bronchopulmonary dysplasia.



59 – 3 minutes

SPHINGOSINE-1-PHOSPHATE ENHANCES INTESTINAL BARRIER FUNCTION BY MODULATING PARACELLULAR JUNCTIONAL PROTEINS

Jose Greenspon, MD[1], Jennifer A. Timmons, MD[1], Alexis D. Smith, MD[1], Riuyun Li, BS[2], Jian- Ying Wang, MD, PhD[2], Eric D. Strauch, MD[1], Douglas J. Turner, MD[1]
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Introduction:

Intestinal injury resulting in barrier disruption is implicated in many conditions including necrotizing enterocolitis and sepsis. Increasing data suggests that these conditions result in disruption of intercellular junction proteins. Recent work has implicated sphingosine-1-phophate (S1P) as a central mediator of endothelial barrier function. Although intestinal mucosa is abundant with S1P, its role in the regulation of intestinal barrier function is not known. The current study tests the hypothesis that S1P enhances intestinal barrier function via alterations of paracellular proteins even in an inflammatory milieu such as lipopolysaccharide (LPS) exposure.

Methods:

Differentiated intestinal epithelial cells (IEC-Cdx2L1 line) were exposed to S1P (0.5 to 10 μM). Small interfering RNA (siRNA) transfection, Western blot analysis, real-time PCR, and immunofluorescence studies were performed. Permeability was assessed by radiotracer measurement of 14C-labeled mannitol or transepithelial electrical resistance assays (TEER).

Results:

Real-time PCR and Western blot analysis revealed that levels of E-cadherin mRNA and protein were significantly increased after treatment with S1P. Exposure to S1P also improved epithelial barrier function as indicated by a decrease in paracellular permeability (~23%). Cortical staining of E-cadherin after S1P treatment resulted in redistribution to the periphery of the cells. Additionally, silencing of S1P receptor by siS1P1 resulted in an increase in permeability as compared to the baseline. Moreover, S1P caused an increase in phosphorylated occludin as compared to control. Additionally, treatment with LPS [50 μg/mL] decreased levels of E-cadherin and thus increased paracellular permeability, both of which were prevented by S1P pretreatment. Lastly, S1P pretreatment prevented the degradation of cortical occludin found to occur with LPS exposure.

Conclusions:

These results indicate that S1P promotes intestinal epithelial barrier function by regulating paracellular proteins, most likely through a mechanism involving the S1P1 receptor. These findings may represent a novel mechanism involved in epithelial integrity during inflammatory states such as NEC.

Notes:

60 – 3 minutes

THE INFLUENCE OF INTRA-LUMINAL CONTENTS ON INTESTINAL ADAPTATION UNDER THE SAME SYSTEMIC HORMONAL CONDITIONS

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Background:

Intestinal adaptation following massive intestinal resection requires
nutrient stimulation. However, the relative effects of direct mucosal stimulation by nutrients versus feed induced enteric hormonal stimulation are unknown. To investigate this, we created a model of resection leaving a bypassed segment of jejunum and ileum, comparing adaptation with and without exposure to luminal nutrients

Methods:

Male rats (250-300 g) underwent sham transaction or creation of a jejuno-ileal bypass, leaving 30 % of bowel in continuity (n=8/group). Animals were pair fed and followed for 7 days. Weight and postprandial systemic Glucagon-like peptide 2 (GLP-2) and (PYY) levels were quantified. Adaptation was quantified by intestinal gross and microscopic morphology and crypt proliferation in each intestinal limb of the bypass and the equivalent points in the sham intestine. Mucosal glucose active and passive carrier and growth factors were measured.

Results:

Bypass animals exhibited reduced weight gain, but significant adaptive changes with increased bowel diameter; Villus height, Crypt depth, Mid villus width, Villus density and crypt cell proliferation in both the jejunal and ileal segments of the alimentary limb in comparison to bypassed Jejunum and ileum and the sham control. Hormonal levels were increased in the bypassed animals (All comparisons $p<0.05$ by student's t-test)

Conclusions:

Despite consistent systemic trophic hormonal stimulation, small bowel adaptation occurred maximally in bowel segments stimulated by nutrients. Further studies to examine the local factors which mediate the nutrient induced adaptation response are indicated; such local factors could be useful therapy for patients with short bowel syndrome.

Notes:

61 – 3 minutes

EGFR INHIBITION IS ANTIANGIOGENIC IN EXPERIMENTAL NEUROBLASTOMA

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Purpose:

Survival for children with high risk neuroblastoma remains poor even after intensive multi-modal therapy. Therefore, new therapeutic approaches are necessary to help improve outcomes. The epidermal growth factor receptor (EGFR) is a transmembrane protein tyrosine kinase that plays a crucial role in regulating key cellular functions such as proliferation and survival. EGFR targeting agents have been shown to reduce tumor growth, but the mechanism remains unknown. EGFR has been implicated in the development of certain pediatric solid tumors, including neuroblastoma. We hypothesized that EGFR blockade would suppress neuroblastoma growth and angiogenesis.

Methods:

Approval was obtained by the Institutional Animal Care Committee. *In vitro* testing was performed using a colorimetric cell proliferation assay. *In vivo* evaluation used ten million cultured human, *MYCN* amplified, neuroblastoma cells which were implanted intrarenally in athymic mice (N=48). At 7 days, treatment with vehicle or the EGFR inhibitor, gefitinib, was begun (via daily gavage) and continued for 35 days. Control/treated cohorts (N=12 each) were euthanized at 35 and 49 days after treatment. Vasculature was assessed by perfusion and specific immunostaining for endothelium and vascular smooth muscle. Endothelial and smooth muscle cell apoptosis was determined by specific double-label immunostaining and TUNEL assay. Tumor weights were compared using Kruskal-Wallis analysis.

Results:

Tumor growth was suppressed by gefitinib at 35 days (75%, p=0.0004), but statistically significant rebound growth was noted after cessation of treatment. Perfusion and immunostaining analysis demonstrated diminished vasculature in the treated cohort. Both smooth muscle cells and tumor cells displayed increased apoptosis in treated tumors compared to controls.

Conclusions:

There is evidence that suggests EGFR functions to promote tumor proliferation and survival. Our data demonstrates both *in vitro* and *in vivo* tumor suppression with evidence of antiangiogenic properties affecting pericytes. These data advocate that targeting EGFR may provide a new pathway to provide antiangiogenic treatment in *MYCN* amplified neuroblastoma.

Notes:

62 – 3 minutes

CONSTRUCTING TRANSLATABLE TISSUE-ENGINEERED ARTERIAL GRAFTS: A NOVEL APPROACH

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Purpose:

Our group has already described the development of tissue-engineered arterial grafts composed of smooth muscle cells and endothelial cells seeded onto tubular scaffolds for use in repair of congenital heart defects. Although this approach holds great promise, it is a time consuming process requiring invasive extraction of host cells with prolonged periods of culture of the cells and grafts, thereby placing recipients at increased risks of infective complications and delaying treatment of the underlying processes. We sought to use host cells obtained on the day of surgery in the fabrication of arterial grafts for implantation later that same day, thereby making this technology easily translatable to the clinical setting.

Methods:

Sub-1mm internal diameter tubular scaffolds were constructed from biodegradable sheets of poly-L-lactic acid coated with a poly-□-caprolactone-lactide copolymer sealant solution. Scaffolds were statically seeded with fresh human bone marrow mononuclear cells and placed into autologous plasma prior to implantation as infrarenal aortic interposition grafts in SCID/bg mice (n=10) in accordance with IACUC guidelines. The mice were evaluated for up to 15 months following graft implantation with imaging, histologic characterization and immunohistochemical techniques to analyze the grafts at the various study time points.

Results:

The tissue-engineered vascular grafts constructed were successfully implanted into the arterial system. All grafts remained patent throughout the study despite not using antiplatelet agents or anticoagulants. By 6 months following implantation, grafts demonstrated a neo-intima containing endothelial cells, a neo-media containing smooth muscle cells and a neo-adventitia containing collagen as is similarly seen in the native artery. Developments of these cellular layers were even more pronounced as time from implantation increased.

Conclusions:

In conclusion, we have demonstrated a feasible method for the construction of tissue-engineered arterial grafts that can be fabricated and successfully implanted within a single day, thereby increasing the clinical applicability of this technology.

Notes:

63 – 3 minutes

CONDITIONAL MUTATION OF *FIBROBLAST GROWTH FACTOR RECEPTORS 1 AND 2* RESULTS IN AN OMPHALOCELE IN MICE ASSOCIATED WITH DISRUPTIONS ON VENTRAL BODY WALL MUSCLE FORMATION.

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Propose:

Fibroblast growth factor signaling is a lynchpin the morphogenesis of numerous organs and structures during development. We observed that *Fibroblast growth factor receptors 1* and *2* (*Fgfr1* and *Fgfr2*) are expressed during abdominal wall development in mice and hypothesized that conditional mutation of these genes would result in abdominal wall defects.

Methods:

IACUC approval for these studies was obtained. Section in situ hybridizations were performed for *Fgfr1* and *Fgfr2* on wild-type embryos at E11.5 and E13.5. Conditional mutation were generated by a single oral injection of tamoxifen (0.1 mg/g) at E8.5 in pregnant *Fgfr1c/c; Fgfr2 c/c* females that had been bred to *Fgfr2c/c; Rosa EsrCre/EsrCre* males. Embryos were harvested at E17.5, whole mount photographs were taken, embryos were fixed in Bouin's and paraffin sections were generated and stained with H&E. Comparisons were made with controls.

Results:

Fgfr1 is robustly expressed in ectoderm, lateral plate mesoderm and myoblasts whereas *Fgfr2* is expressed almost exclusively in ealy dermis and ectoderm of the abdominal wall. Conditional mutation of both *Fgfr2* alleles and one *Fgfr1* allele resulted in omphalocele in 32% of mutants. Histological examination demonstrated that the paniculus carnosis, a thin muscle layer between the abdominal wall musculature and the skin was discontinuous, thinner and closer to the dermis in mutants than in controls.

Conclusions:

We report a genetic model for omphalocele arising from mutation in *Fgfr1* and *Fgfr2* which exhibits disruptions in the development of the muscle layer closest to the skin. These findings are similar to those we've observed in giant human omphalocele and suggest that the primary abdominal wall and in particular the early ectoderm or dermis may function as a repository for positional information that regulates organization of adjacent myoblasts into specific muscle groups.

Notes:

64 – 3 minutes

FUNCTIONAL ANERGY OF NON-DELETED DONOR-REACTIVE HOST NK CELLS STABILIZES PRENATAL CHIMERISM

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Purpose:

Previous studies of allogeneic prenatal chimeras following in utero transplantation reveal the contradictory persistence of host NK cells expressing a cytotoxic activating receptor specific for the donor. We wondered if these potentially hostile NK cells were rendered anergic while developing in a stable chimeric environment. To address this possibility, we studied the response of these alloreactive cells toward donor-specific and non-specific stimuli.

Methods:

Balb/c ($H2^d$) fetal liver hematopoietic cells at 14 days postcoitum were transplanted into age-matched B6 ($H2^b$) fetal recipients. Chimeras were determined at 3 weeks after birth and the expression of alloreactive Ly49D+ host NK cells followed serially. The functional response of Ly49D+ splenic NK cells was measured at 9 weeks of age in a 4-hr IFN-γ assay in response to Ly49D (donor-specific), NK1.1 (non-specific) and PMA/ionomycin (non-specific). Results were compared to naive animals using a two-tailed Student *t* test assuming unequal variances.

Results:

The percentage of Ly49D receptor bearing NK cells was significantly lower in stable chimeras (n=5) when compared to controls (n=5). However, the intensity of Ly49D expression on the cell surface was comparable to naïve animals. In the functional assays, NK cells from stable chimeras were hypo-responsive compared to controls when stimulated through their Ly49D receptors (8.1% +/-3.0% vs. 15.1%+/-2.3%, $p<0.004$). However, Ly49D+ NK cells showed stimulation comparable to controls when stimulated through NK1.1 (28.5%+/-5.5% vs. 30.2%+/- 5.7%, $p=0.6$) or PMA/ionomycin (50.0+/-5.8% vs. 53.6+/-7.7%, $p=0.45$).

Conclusion:

Similar to alloreactive T cells, alloreactive NK cells persist in prenatal chimeras at a reduced frequency and exist in a state of donor-specific anergy. Future studies will examine the mechanisms regulating this anergy in the context of current models of NK cell immunobiology.

Notes:

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