Department of Obstetrics,
Gynecology and Reproductive Medicine

THIRTIETH ANNUAL RESIDENTS RESEARCH DAY

June 16, 2010

STATE UNIVERSITY OF NEW YORK
STONY BROOK
HEALTH SCIENCES CENTER

Stony Brook University Medical Center
Stony Brook, New York

PROGRAM OBJECTIVES

The purpose of this program is to provide a forum for discussion of original research findings and for the introduction, development, and review of new and most accepted approaches to the discipline of Obstetrics and Gynecology. Upon completion of the program, participants should be able to apply medical problem-solving skills, practice new approaches to manual and surgical skills, and utilize skills in evaluating new information.

CREDITS

The School of Medicine, State University of New York at Stony Brook, is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to sponsor continuing medical education for physicians.

The School of Medicine, State University of New York at Stony Brook, designates this activity for a maximum of 5.5 AMA PRA Category™. Physicians should only claim credit commensurate with the extent of their participation in the activity.

The American College of Obstetricians and Gynecologists has assigned 6 cognate credits to this program.

DISCLOSURE POLICY

All those in control of the content of its CME activities (planners, speakers, authors) sponsored by the School of Medicine are expected to disclose any real or apparent conflict of interest to the content of the educational program.

All commercial relationships that create a conflict with the planners, speakers, and authors control of content must be resolved before the educational activity occurs.

Department of Obstetrics, Gynecology and Reproductive Medicine
School of Medicine
Stony Brook University Medical Center
Thirtieth Annual Residents Research Day
June 16, 2010

Chairman: J. Gerald Quirk, M.D., Ph.D.

Residency Director: Todd Griffin, M.D.

Associate Residency Director: Michael Lydic, M.D.

RRD Program Director: Richard Bronson, M.D.

RRD Program Committee:
Deborah Duttge
Darlene Swords
Terry Leonbruno
Melanie Morgan

Departmental Faculty:

Kristen Alarcon, N.P.
Susan Altman, C.N.M., D.N.P.
Cecilia Avila, M.D.
David Baker, M.D.
Richard Bronson, M.D.
Lauri Budnick, M.D.
Christine Conway, M.D.
Michael Demishev, M.D.
Reinaldo Figueroa, M.D.
Heather Findletar, C.N.M., D.N.P.
Marie Frey, C.N.M.
Jennifer Griffin, N.P.
Todd Griffin, M.D.
Jessica Hilsenroth, C.N.M.
Jennifer Johnson, M.D.
Daniel Kiefer, M.D.
Pamela Koch, C.N.M.
Christina Kocis, C.N.M., D.N.P.
Laura Lesch, N.P.
Michael Lydic, M.D.
Goldie McBride, C.M.
Careen Mauro, C.N.M.
Alan Monheit, M.D.
Jolene Muscat, M.D.
Paul L. Ogburn, Jr., M.D.
Michael Pearl, M.D.
Lisa Rimpel, M.D.
Natalie Semenyuk, M.D.
Eva Swoboda, M.D.
Siamak Tabibzadeh, M.D.
Ann Visser, C.N.M.
Dympna Weil, M.D.

Martin L. Stone, M.D.
Professor Emeritus

LECTURER AND JUDGES

THIRTIETH ANNUAL RESIDENTS RESEARCH DAY

MARTIN L. STONE, M.D. LECTURER AND JUDGE

Vivian Von Gruenigan, M.D.	Chair of Ob/Gyn Summa Health System Akron, Ohio

JUDGES

Thomas Wilson, M.D.	Director of Pediatric Endocrinology Professor of Pediatrics Stony Brook University Medical Center
Douglas Lee, M.D.	Clinical Assistant Professor Department of Ob/Gyn Stony Brook University Medical Center

Departmental Residents

CHIEFS	Jerasimos Ballas, M.D. Shelly-Ann James, M.D. Lan Na Lee, M.D.
Administrative Chief	Randi Turkewitz, M.D.
PGY-3	Elizabeth Buescher, M.D. Joseph Chappelle, M.D. Elizabeth Garduno, M.D. Donald Phillibert, M.D. Chanda Reese, M.D.
PGY-2	Leia L. Card, M.D. Diana J. Garretto, M.D. James A. MacDonald, M.D. Cara S. Ninivaggio, M.D. Viveka R. Prakash, M.D.
PGY-1	Rosalie O. Alvarado, M.D. Jenny A. Blumberg, M.D. Alexis Gimovsky, M.D. Amanika Kumar, M.D. Michael J. Vizcarra, M.D.

ALUMNI RESIDENTS (CONTINUED)

2002-2003
Karen Chu, M.D., Private Practice, San Francisco, California
JoAnna Paolilli, M.D., Private Practice, Mineola, New York
Hera Sambaziotis, M.D., M.P.H., Albert Einstein Medical Center, Bronx, New York
Julie Welischar, M.D., Private Practice, Setauket, New York

2003-2004
Patricia Ardise, M.D., Private Practice, New Jersey
Anne Hunter, M.D.
Sara Petruska, M.D., Private Practice, Kentucky
Alejandra Turmero, M.D., Private Practice, Rhode Island

2004-2005
Heather McGehean, M.D.
Timothy Hale, M.D., Private Practice, Massachusetts
Joyce Rubin, M.D., Private Practice, Smithtown, New York
Vanessa Soviero, M.D., Private Practice, Smithtown, New York
Eva Swoboda, M.D., Stony Brook University Medical Center, Stony Brook, New York

2005-2006
Lynda Gioia, M.D., Private Practice, Tennessee
Olga Glushets, M.D., Urogynecology Fellowship, Brooklyn, New York
Meredith McDowell, M.D., Private Practice, Norwich, New York

2006-2007
Patricia Dramitinos, M.D., Urogynecology Fellowship, Cambridge, Massachusetts
Megan Lochner, M.D., Private Practice, Setauket
Christopher Paoloni, M.D., Private Practice, Virginia
Anita Patibandla, M.D., Private Practice, Ohio

2007-2008
Rupinder Bhangoo, M.D., Private Practice, Fishkill, New York
Kristen Patzkowsky, M.D., Minimally Invasive Fellowship, Ann Arbor, Michigan
Kelly van den Huevel, M.D., Private Practice, San Diego, California
Dympna Weil, M.D., Stony Brook University Hospital, Stony Brook, New York

2008-2009
Kirthi Katkuri, M.D., St. Elizabeth's Medical Center, MA
Nikole Ostrov, M.D. , Minimally Invasive Surgery Fellowship,
Stony Brook University Medical Center
Erin Stevens, M.D., Gynecologic Oncology Fellowship, SUNY Downstate,
Brooklyn NY

ALUMNI RESIDENTS (CONTINUED)

1993-1994
Ira Chan, M.D., Instructor, Beth Israel Hospital, Harvard Medical School, Boston, MA
Pui Chun Cheng, M.D., Gynecologic Oncology, New Orleans, Louisiana
Lawrence Weinstein, M.D., Private Practice, Kingston, New York

1994-1995
Ira Bachman, M.D., Private Practice, Cedarhurst, New York
Petra Belady, M.D., Private Practice, Bloomington, Indiana
Gloria Escamilla, M.D., Private Practice, Smithtown, New York
Lisa Farkouh, M.D., Private Practice, Denver, Colorado

1995-1996
Felicia Callan, M.D., Private Practice, Huntington, New York
Charles Mirabile, M.D., Private Practice, West Islip, New York
Karen Morris, M.D., Private Practice, Huntington, New York
James Stelling, M.D., Private Practice, Stony Brook, New York

1996-1997
Jacqueline Ammirata, M.D., Private Practice, West Islip, New York
Todd Griffin, M.D., Chief Medical Officer Stony Brook University Hospital, Stony Brook, New York
Hitesh Narain, M.D., Private Practice, Patchogue, New York
Florence Rolston, M.D., Private Practice, Southampton, New York

1997-1998
Salil Bakshi, M.D., Private Practice, Oakdale, New York
Wei Chu, M.D., Private Practice, East Islip, New York
David Reavis, M.D., Private Practice, Patchogue, New York
Marian Zinnante, M.D., Private Practice, Arlington, Texas

1998-1999
Robert Duck, M.D., Private Practice, Winchester, Virginia
Christopher Fabricant, M.D., Univ. of Texas, Southwestern Medical Center, Dallas, Texas
Anne Hardart, M.D., University of Southern California, Los Angeles, California
Lynne Macco, M.D., Private Practice, West Islip, New York

1999-2000
Vito Alamia, M.D., Private Practice, Southampton, New York
Terry Allen, M.D., Private Practice, Fairfax, Virginia
Mari Inagami, M.D., Private Practice, Westport, Connecticut
Jill Thompson, M.D., Private Practice, Northport, New York

2000-2001
Martina Frandina, M.D., New York Downtown Hospital, New York, New York
Dennis McGroary, M.D. Private Practice, Mt. Kisco, New York
Antonia Pinney, M.D., Private Practice, New Jersey

2001-2002
Siobhan Hayden, M.D., Mary Imogene Barrett Hospital, Cooperstown, New York
Antoun Khabbaz, M.D., Appalachian Regional Healthcare, Harlan, Kentucky
Dennis Strittmatter, M.D., Private Practice, Port Jefferson, New York

PROGRAM

8:30 - 8:35 *Welcome*
J. Gerald Quirk, M.D., Ph.D.
Chairman

8:35 - 8:45 *Introduction*
Richard Bronson, M.D.

8:45 - 9:00 **"***The Effects of Hyperoxia and Lipopoly Saccaride (LPS) on Inflammatory Mediator Expression in Sprague-Dawley Rat Pups*
Elizabeth Buescher, M.D.
Faculty Sponsors: Shetal Shah, M.D.
Craig Cohen, PhD.
Contributor: Erin Killeen, BS

9:00 - 9:15 Discussion and Questions
Discussant: Siamak Tabibzadeh, M.D.

9:15 - 9:25 *Preterm Premature Rupture of Membranes (PPROM): Neonatal Morbidity Between 32 and 36 6/7 Weeks of Gestation*
Viveka Prakash, M.D.
Faculty Sponsor: Reinaldo Figueroa, M.D.

9:25 - 9:30 Open Discussion

9:30 - 9:45 *Evaluating the Efficacy of Detailed Fetal Anatomic Ultrasonography to Detect Congenital Heart Disease*
Joseph Chappelle, M.D.
Faculty Sponsor: Reinaldo Figueroa, M.D.
Contributor: Lillian Meek, R.N.

9:45 - 10:00 Discussion and Questions
Discussant: Paul L. Ogburn, M.D.

10:00 - 10:10 *The Use of Body Mass Index in Pregnancy*
Diana Garretto, M.D.
Faculty Sponsor: Erin Stevens, M.D.

10:10 - 10:15 Open Discussion

10:15 - 10:25 *The Effect of Epidural Anesthesia in Preterm Labor*
James MacDonald, M.D
Faculty Sponsors: Rishimani Adsumelli, M.D.
Kathleen Dubrow, M.D.
Reinaldo Figueroa, M.D.

10:25 - 10:30 Open Discussion

10:30 - 11:00 Coffee Break

Program (continued)

11:00 - 12:00
Endometrial Cancer Survivorship: What does Obesity have to do with it?
Vivian Von Gruenigan, M.D.

12:00 - 12:15
The Impact of Obesity on Vaginal Birth after Cesarean Delivery (VBAC)
Donald Phillibert, M.D.
Faculty Sponsor: Reinaldo Figueroa, M.D.

12:15 - 12:30
Discussion and Questions:
Discussant: Jennifer Johnson, M.D.

12:30 - 12:40
A Retrospective Investigation of Endometrial Pathology in Relationship to the Bleeding Profiles of Premenopausal Females Undergoing Endometrial Biopsies in Order to Determine a Risk Categorization
Cara Ninivaggio, M.D.
Faculty Sponsor: Eva Swoboda, M.D.

12:40 - 12:45
Open Discussion

12:45 - 1:00
Is Ultrasound Finding of Single Umbilical Artery Associated with Fetal Cardiac Anomaly?
Chanda Reese, M.D.
Faculty Sponsor: Reinaldo Figueroa, M.D.

1:00 - 1:15
Discussion and Questions
Discussant: Alan G. Monheit, M.D.

1:15 - 2:15
Lunch

2:15 - 2:25
Chorioamnionitis: A Retrospective Study Analyzing the Accuracy of Clinical Diagnosis in Preterm Pregnancies
Leia Card, M.D.
Faculty Sponsor: Reinaldo Figueroa, M.D.

2:25 - 2:30
Open Discussion

2:30 - 2:45
Fasting Plasma Active Glucagon-like Peptide-1 in Pregnancies with and without Gestational Diabetes
Elizabeth Garduno, M.D.
Faculty Sponsors: Cecilia Avila, M.D.
Andrew Lane, M.D.

2:45 - 3:00
Discussion and Questions
Discussant: Michael Lydic, M.D.

3:00 - 3:15
Factors Predicting Gestational Age at Delivery Following Ultrasound-Indicated Cervical Cerclage
Jolene Muscat, M.D.
Faculty Sponsor: Paul L. Ogburn, M.D.

3:15 - 3:30
Discussion and Questions
Discussant: Reinaldo Figueroa, M.D.

ALUMNI RESIDENTS

1981-1982
Richard Scotti, M.D., Deceased
W. Robert Lockridge, M.D., New York

1982-1983
Deborah Davenport, M.D., Private Practice, East Setauket, New York
William Shuell, M.D., Private Practice, Southampton, New York

1983-1984
Robert O'Keefe, M.D., Private Practice, Setauket, New York
Alexandra Taylor, M.D.

1984-1985
Eva Chalas, M.D., Vice Chair of Ob/Gyn, Winthrop University Hospital, Mineola, New York
David Kreiner, M.D., Private Practice, Woodbury, New York

1985-1986
Jeffrey Porte, M.D., Private Practice, Setauket, New York
Gae Rodke, M.D., Private Practice, New York, New York

1986-1987
Lance Edwards, M.D., Private Practice, Port Jefferson, New York
Mindy Shaffran, M.D., Private Practice, Port Jefferson, New York
Christian Westermann, M.D., Private Practice, Stony Brook, New York

1987-1988
Timothy Bonney, M.D., Private Practice, West Islip, New York
Arlene Kaelber, M.D., Private Practice, East Setauket, New York

1988-1989
Michael Arato, M.D., Private Practice, Stony Brook, New York
Miriam Sivkin, M.D., Private Practice, Milford, Connecticut

1989-1990
Michael Klotz, M.D., Private Practice, Seattle, Washington
Paul Meyers, M.D., Riverside Hospital, Newport News, Virginia
Gustavo San Roman, M.D., Private Practice, Port Jefferson Station, New York

1990-1991
Cheri Coyle, M.D., Private Practice, Hampton, Virginia
Syau-fu Ma, M.D., Private Practice, Ridgewood, New Jersey
John Wagner, M.D., Private Practice, East Northport, New York

1991-1992
Brian McKenna, M.D., Private Practice, Smithtown, New York
Gerald Siegel, M.D., Private Practice, Commack, New York
Marie Welshinger, M.D., Women's Cancer Center, Morristown Memorial, Morristown, NJ

1992-1993
Theodore Goldman, M.D., Private Practice, East Northport, New York
Stephanie Mann, M.D., Private Practice, Los Angeles, California
Robert Scanlon, M.D., Private Practice, Kingston, New York

ALUMNI RESIDENTS

The Golden Scalpel Award

In Recognition of Demonstrating Excellence in Technical Skills

2001	Martina Frandina, M.D.
2002	Antoun Khabbaz, M.D
2003	Julie Welischar, M.D.
2004	Joyce Rubin, M.D.
2005	Eva Swoboda, M.D.
2006	Megan Lochner, M.D.
2007	Megan Lochner, M.D.
2008	Nikole Ostrov, M.D.
2009	Nikole Ostrov, M.D.

The Effects of Hyperoxia and Lipopolysaccharide (LPS) on Inflammatory Mediator Expression in Sprague-Dawley Rat Pups

Shetal Shah MD, Elizabeth Buescher MD, Craig Cohen PhD and Erin Killeen BS

Objective: The objective of this research is to determine if hyperoxia causes a pro-inflammatory response in Sprague-Daley Rat Pups, as compared to injection with lipopoly-saccharide.

Hypothesis: Hyperoxia-induced inflammation will synergistically increase levels of inflammatory mediators in the lungs of LPS-treated Sprague-Dawley Rat Pups.

Background: Supplemental oxygen is a mainstay of modern neonatal care. Hyperoxia is associated with retinopathy of prematurity and central nervous system depression via the "Bert Effect." In the lung, hyperoxia causes short-term inflammation, apoptosis, increased susceptibility of infection and decreased surfactant production; initiating a cycle of tissue injury, regeneration and fibrosis leading to the simplified alveolarization, and disrupted vascularization that characterizes chronic lung disease (CLD). This well-described mechanism, mediated by increased oxidative stress, overwhelms the decreased endogenous anti-oxidant defenses of the preterm infant, resulting in the formation of inflammatory reactive oxygen species. Data from our lab has shown hyperoxia alters lung levels of Heat Shock Protein 27, Interleukin 6, Interleukin 8, Interleukin 10, and Tumor Necrosis Factor-Alpha, as well as co-stimulatory molecules of Toll-like Receptors 2 and 4.
Neonates infected with gram-negative bacteria undergo a similar pro-inflammatory response in the lung with upregulation of tumor-necrosis factor alpha, toll-like receptors 2 and 4, interleukin 8, 12 and 1Beta. However the interactive effects of both these pro-inflammatory states, which together often occur clinically, has not been well characterized. The purpose of this experiment is to determine levels of known pro-inflammatory markers in the lung and serum of animals treated with both hyperoxia and LPS relative to controls.

Methods: Two litters of time-pregant Sprague- Dawley rat pups will be included in this study. Thee days after birth, half of each litter was treated with 5micrograms/kg of LPS via intra-peritoneal injection prior to exposure to >95% hyperoxia in a sealed chamber for 24 hours. The other half of each litter was injected with intraperitoneal normal saline, resuling in four cohorts: control, lipopolysaccharide alone, hyperoxia alone, and lipopolysaccharide plus hyperoxia. Immediately post-intervention pups were sacrificed. Lung tissue and serum from cardiac puncture were obtained. Frozen sections were made and immunohistochemistry was performed with antibodies for tumor-necrosis factor alpha, toll-like receptors 2 and 4, interleukin 8, 10 and IL-1Beta. Data was analyzed by pixelation of the digitzed images and compared using ANOVA testing. P value less than or equal to 0.05 were considered signficant.

Results: Results to date show that hyperoxia resulted in a statistically significant increase in TLR-2 $p<0.05$). Hyperoxia resulted in a statistically significant increase in TLR-4, IL-8, and IL-1beta with $p<0.05$. In TLR-2, the combination of hyperoxia and LPS resulted in statisticaly significant increased levels of expression. In TLR-4, IL-8, TNF-alpha, and IL-1beta, the combination of hyperoxia and LPS resulted in statistically significant lower levels than controls, For IL-10, LPS caused a decrease in the level of IL-10. Although hyperoxia alone did not significantly after the levels of IL-10, the combination of LPS and hyperoxia caused a significant decrease in IL-10 levels.

Conclusion: For TLR-2, the dual insult of hyperoxia and LPS resulted in a statistically significant increase in the level of expression. For TLR-4, IL-1beta, and TNF-alpha, there was a pattern of decreased levels of expression with the dual insult of hyperoxia plus LPS, possibly due to depletion of endogenous stores, however there is also data that shows that LPS can be protective in the hyperoxic state. Finally, it is possible that the dual insult of LPS and hyperoxia so overwhelmed the system, that, had we not sacrificed the pups immediately after the hyperoxic exposure, we would have seen a high death rate in that group. Further research in needed with ELISA, PCR, staining for lymphocytes, and delayed sacrifice of the pups to further elucidate the results found in this study.

Preterm Premature Rupture of Membranes (PPROM): Neonatal Morbidity Between 32 and 36 6/7 Weeks of Gestation

Viveka R. Prakash, MD and Reinaldo Figueroa MD

Introduction

Preterm delivery is defined as a delivery occurring before 37 weeks of gestation. Significant neonatal morbidities are associated with preterm deliveries. Although recent advances in neonatal care have decreased the incidence of significant morbidity and death in neonates between 32 and 37 weeks of gestation, there remain isolated cases of significant neonatal morbidity at these gestational ages. Controversy remains as to the management of patients with PPROM between 32 and 37 weeks of gestation.

Study Population

The medical records from patients admitted to the Obstetrics Service with a diagnosis of PPROM who delivered at Stony Brook Medical Center from July 2004 through December 2009 will be reviewed.

Inclusion Criteria

Singleton gestations with PPROM who delivered between 32 and 36 6/7 weeks will be included.

Exclusion Criteria

Patients with poor dating, major fetal anomalies, or fetal death on admission will be excluded.

Methods

PPROM will be diagnosed by presence of pooling of amniotic fluid on speculum examination, a positive ferning test, and/or the subjective complaints of loss of fluid from the vagina combined with decreased amniotic fluid on obstetric sonogram.

Gestational age will be determined by the last menstrual period, if it agrees with ultrasound estimation within 5 days (first trimester) or within 7 days (second trimester); otherwise, the pregnancy will be dated sonographically. Women in the third trimester who had not had a previous ultrasound examination to confirm dating will be excluded. In addition to the information stated above the medical charts of the mothers will be reviewed for the following information: age, ethnicity, parity, gestational age on admission (at diagnosis), history of previous preterm delivery, history of previous cesarean delivery, assisted reproduction, co-morbid conditions (i.e. diabetes, hypertension, asthma), cervical cerclage placement in this pregnancy, administration of antibiotics, administration of steroids, use of tocolytics, latency period (from PPROM to delivery), route of delivery, indication for delivery, diagnosis of clinical chorioamnionitis, white blood cell count on admission, diagnosis of placental abruption or cord prolapse, diagnosis of non-reassuring fetal heart tracing., and whether labor was spontaneous or induced.

Neonatal charts will be reviewed for the following information: gestational age at delivery, birth weight, sex, Apgar scores at 1 and 5 minutes, umbilical cord gases, neonatal sepsis (proven and suspected), respiratory distress syndrome (RDS), intraventricular hemorrhage (IVH), periventricular leukomalacia (PVL), bronchopulmonary dysplasia (BPD), necrotizing enterocolitis (NEC), patent ductus arteriosus (PDA), admission to the NICU, number of days admitted to the NICU, number of days on respirator, and perinatal death.

This study will attempt to determine the risk of significant neonatal morbidity in women with PPROM who delivered between 32 and 36 6/7 days of gestation. The neonatal outcomes will be stratified by gestational age at delivery.

AWARDS—PAST RECIPIENTS

The William J. Mann, M.D. Pathology Award

1982	Deborah Davenport, M.D.
1983	Deborah Davenport, M.D.
1984	Eva Chalas, M.D.
1985	Eva Chalas, M.D.
1986	Mindy Shaffran, M.D.
1987	Christian Westermann, M.D.
1988	Michael Arato, M.D.
1989	Paul Meyers, M.D.
1990	Syau-fu Ma, M.D.
1991	Cheri Coyle, M.D.
1992	Robert Scanlon, M.D.
1993	Robert Scanlon, M.D.
1994	Petra Belady, M.D.
1995	Charles Mirabile, M.D.
1996	James Stelling, M.D.
1997	Todd Griffin, M.D.
1998	Robert Duck, M.D.
1999	Jill Thompson, M.D.
2000	Jill Thompson, M.D. Terry Allen, M.D.
2001	Hera Sambaziotis, M.D., .M.P.H
2002	JoAnna Paolilli, M.D.
2003	Timothy Hale, M.D.
2004	Vanessa Soviero, M.D.
2005	Megan Lochner, M.D.
2006	Olga Glushets, M.D.
2007	Patricia Dramitinos, M.D.
2008	Kelly van den Heuvel, M.D.
2009	Erin Stevens, M.D.

The Robert L. Barbieri M. D. Research Award

(Formerly the Resident Research Award)

1981	Deborah Davenport, M.D.
1982	Alexandra Taylor, M.D.
1983	Deborah Davenport, M.D.
1984	Robert O'Keefe, M.D.
1985	Gae Rodke, M.D.
1986	Christian Westmann, M.D.
1987	Mindy Shaffran, M.D.
1988	Michael Arato, M.D.
1989	Syau-fu Ma, M.D.
1990	John Wagner, M.D.
1991	John Wagner, M.D.
1992	Robert Scanlon, M.D.
1993	Robert Scanlon, M.D.
1994	Ira Bachman, M.D.
1995	Felicia Callan, M.D.
1996	Todd Griffin, M.D. Marian Zinnante, M.D.
1997	Ann Hardart, M.D. Marian Zinnante, M.D.
1998	Ann Hardart, M.D. Jill Thompson, M.D.
1999	Vita Alamia, M.D.
2000	Mari Inagami, M.D.
2001	Dennis Strittmatter, M.D.
2002	JoAnna Paolilli, M.D.
2003	Sara Petruska, M.D.
2004	Anne Hunter, M.D.
2005	Lynda Gioia, M.D.
2006	Kristin Patkowsky, M.D.
2007	Kelly van den Heuvel, M.D.
2008	Nikole Ostrov, M.D
2009	Elizabeth Buescher, M.D.

AWARDS-PAST RECIPIENTS

The David Marzouk, M.D. Humanism in Medicine Award

In Recognition of Warmth, Compassion, and Devotion to the Profession of Medicine

1985	Eva Chalas, M.D.
1986	Timothy Bonney, M.D.
1987	Michael Arato, M.D.
1988	Michael Arato, M.D.
1989	Syau-fu Ma, M.D.
1990	Brian McKenna, M.D.
1991	Robert Scanlon, M.D.
1992	Stephanie Mann, M.D.
1993	Petra Belady, M.D.
1994	Felicia Callan, M.D.
1995	Elizabeth Folland, M.D.
1996	Florence Rolston, M.D.
1997	David Reavis, M.D.
1998	Vito Alamia, M.D.
1999	Lynne Macco, M.D.
2000	Siobhan Hayden, M.D.
2001	Anne Hunter, M.D.
2002	JoAnna Paolilli, M.D.
2003	Sara Petruska, M.D.
2004	Vanessa Soviero, M.D.
2005	Megan Lochner, M.D.
2006	Meredith McDowell, M.D.
2007	Dympna Weil, M.D.
2008	Rupinder Bhangoo, M.D.
2009	Nikole A. Ostrov, M.D.

Resident Teaching Award

In Recognition of Commitment, Dedication, and Enthusiasm in the Teaching and Nurturing of Medical Students

1990	Brian McKenna, M.D.
	John Wagner, M.D.
1991	Pui Chun Cheng, M.D.
1992	Pui Chun Cheng, M.D.
1993	Lawrence Weinstein, M.D.
1994	Todd Griffin, M.D.
1995	David Reavis, M.D.
1996	David Reavis, M.D.
1997	David Reavis, M.D.
1998	David Reavis, M.D.
1999	Vito Alamia, M.D.
2000	JoAnna Paolilli, M.D.
2001	JoAnna Paolilli, M.D.
	Hera Sambaziotis, M.D.
2002	Joyce Rubin, M.D.
2003	JoAnna Paolilli, M.D.
2004	Heather McGehean, M.D.
2005	Anita Patibandla, M.D.
2006	Anita Patibandla, M.D.
2007	Anita Patibandla, M.D.
2008	Jerasimos Ballas, M.D.
2009	Nikole A. Ostrov, M.D

Evaluating the Efficacy of a Detailed Fetal Anatomic Survey to Detect Congenital Heart Disease

Joseph Chappelle MD, Reinaldo Figueroa MD and Lillian Meek RN

Introduction: Congenital Heart Disease (CHD) is one of the most common congenital malformations. It occurs in approximately 8 out of every 1000 live births. Prenatal diagnosis of CHD allows for further prenatal evaluation and coordination of specialties available at tertiary care centers. A fetal echocardiogram is often performed when the fetus is at an increased risk for CHD. Additionally, many patients currently undergo a fetal anatomic survey prior to the fetal echocardiogram. The fetal anatomic survey includes evaluating the fetal heart and often identifies cardiac anomalies. The utility and cost effectiveness of a designated fetal echocardiogram after a normal fetal anatomic survey remains unclear.

Methods: The prenatal records of all patients who received both a fetal anatomic survey and a fetal echocardiogram at the University Associates in Obstetrics and Gynecology Ultrasound Unit were reviewed for detection of CHD. Charts were excluded from review if the initial fetal anatomic survey or the fetal echocardiogram were suboptimal, or if the studies were done on the same day.

Results: 921 charts were reviewed. Exclusions included: suboptimal fetal anatomic survey (n=223), suboptimal fetal echocardiogram (n=20), fetal anatomic survey and fetal echocardiogram done on same day (n=176). 502 charts were included in the final analysis and the total rate of CHD was 3.8% in the studied population. The positive predictive value of the fetal anatomic survey was 61.5% and the negative predictive value was 97.8%, with a sensitivity and specificity of 42.1% and 98.9% respectively.

Conclusion: The fetal anatomic survey detected the majority of the CHD in this study population. Of the 11 cases of CHD that were not detected on the fetal anatomic survey only 3 were likely to be of clinical significance. This would increase the negative predictive value of the test to >99%. The fetal anatomic survey is therefore an excellent method of screening for CHD and those patients that have a normal survey are unlikely to benefit from an echocardiogram.

Use of Body Mass Index in Pregnancy

Diana Garretto M.D. and Erin Stevens M.D.

Objective: In the United States, we are currently in the midst of an obesity epidemic. In 2007-2008, the prevalence of obesity was 35.5% among adult young women. This has led to many complications with pregnancy including macrosomia, increased blood loss, and increased risk of caesarian delivery. New initiatives are underway to help decrease this rapidly increasing number of obese pregnant women.

Recently, the Institute of Medicine has revised the amount of appropriate weight gain in pregnancy based on a person's pre-pregnancy BMI. What has not been published is what the appropriate BMI is at the time of delivery for these women. Also not yet investigated are if increases in BMI above the recommendations result in the same adverse outcomes.

We wish to examine the relationship of pre-pregnancy BMI to delivery BMI to see if the complications associated with obesity are also associated with those women who have inappropriate changes in their BMI during pregnancy.

The goal of our project, therefore, is twofold- define what the appropriate delivery BMI is, as well as the appropriate changes during pregnancy. Investigate the complications including macrosomia, blood loss, operative delivery, lacerations, and preeclampsia based on pre-pregnancy as well as delivery BMI to see if there is an association.

Materials and Methods: This is a retrospective chart review. Variables such as age, gravida, parity, gestational age at delivery, height, pre-pregnancy weight, pre-pregnancy BMI, delivery weight, delivery BMI, neonate weight, type of delivery, degree of laceration, presence/absence of diabetes, estimated blood loss at delivery, and presence of preeclampsia. The subjects will be patients who delivered at Stony Brook University Medical Center from January 2009 through February 2010. Data will be obtained from the medical record through a retrospective chart review. Subjects who do not have all data points available will be excluded.

AWARDS-PAST RECIPIENTS

The Martin L. Stone, M.D. Award

The Outstanding Resident in Recognition of Dedication, Commitment, and Service
(Formerly Resident of the Year Award)

1982	Robert O'Keefe, M.D.	**1995**	Ira Bachman, M.D.
1983	Eva Chalas, M.D.	**1996**	James Stelling, M.D.
1984	Jeffrey Porte, M.D	**1997**	Todd Griffin, M.D.
1985	Eva Chalas, M.D.	**1998**	David Reavis, M.D.
1986	Jeffrey Porte, M.D.	**1999**	Lynn Macco, M.D.
1987	Christian Westermann, M.D.	**2000**	Siobhan Hayden, M.D.
1988	Timothy Bonney, M.D.	**2001**	Martina Frandina, M.D.
1989	Michael Arato, M.D.	**2002**	Siobhan Hayden, M.D.
1990	Marie Welshinger, M.D.	**2003**	JoAnna Paolilli, M.D.
1991	John Wagner, M.D.	**2004**	Patricia Ardise, M.D.
1992	Pui Chun Cheng, M.D.	**2005**	Heather McGehean, M.D.
1993	Lawrence Weinstein, M.D.	**2006**	Lynda Gioia, M.D.
1994	Ira Bachman, M.D.	**2007**	Megan Lochner, M.D.
1995	Ira Bachman, M.D.	**2008**	Dympna Weil, M.D.
2008	Dympna Weil, M.D.	**2009**	Erin Stevens, M.D.

The Voluntary Clinical Faculty Award

In Recognition of and Appreciation for Outstanding Teaching and Service to the Residency Program

1995	Richard Halpert, M.D.	**2002**	Todd Griffin, M.D
1996	Christian Westermann, M.D.	**2003**	Philip Schoenfeld M.D.
1997	James Droesch, M.D.	**2004**	James Stelling, M.D.
1998	Deborah Davenport, M.D.	**2005**	James DroeschM.D.
1999	Christian Westermann, M.D.	**2006**	James Droesch, M.D.
2000	Abraham Halfen, M.D.	**2007**	Jeffrey Porte, M.D.
2001	Abraham Halfen, M.D.	**2008**	James Droesch, M.D.
		2009	James Stelling, M.D

APPENDIX

PAST AWARD WINNERS

AND

ALUMNI

The Effect of Epidural Anesthesia in Preterm Labor

James MacDonald MD, Kathleen Dubrow MD,
Rishimani Adsumelli MD and Reinaldo Figueroa MD

Objective: To determine the effects of epidural anesthesia on preterm labor patterns and see whether it can be another tool to slow preterm labor.

Background: Preterm delivery is the greatest source of neonatal morbidity and mortality and measures that prolong gestation up to 36 weeks can reduce poor outcomes. There are multiple drug regimens used to slow or stop the contraction pattern in preterm labor with varying levels of effectiveness. Epidural anesthesia is known to slow down term labor and may have the same effect in preterm labor.

Methods: A retrospective record review of mothers admitted with the diagnosis of preterm labor from January 1 to December 31st 2008 at Stony Brook Medical Center. The cohort will be divided into two groups: those that received epidurals for clinical indications and those who did not. Data will be collected from the Labor and Delivery Corometric Information System, the Perinatal Database, Eclipsys and from the Cerner Citrix Database. The primary outcome measure will be prolongation of pregnancy. Secondary outcomes will include neonatal morbidity, contraction frequency and length of labor. Investigated variables include maternal age, BMI, parity, multiple gestation, tocolytic agents, antibiotics, gestational age on admission, route of delivery, birth weight, APGARs and cord gases.

Results: Collection ongoing.

The Impact of Obesity on Vaginal Birth after Cesarean Delivery (VBAC)

Donald Phillibert Jr. MD, Heather Findletar CNM, DNP and Reinaldo Figueroa MD

Objective: To study the impact of Body Mass Index (BMI) on maternal and neonatal morbidity for women attempting a trial of labor after cesarean delivery (TOLAC).

Introduction: Obesity is a severe health risk to a pregnant woman and her fetus. Obesity complicates 18-38% of all pregnancies. VBAC is recognized as a safe and acceptable option after primary lower transverse uterine incision for a select group of patients. VBAC decreases the financial burden on the healthcare system. Repeat cesarean deliveries are associated with increased maternal and neonatal morbidity. There is varying data regarding obesity as a prognostic indicator for a successful VBAC.

Hypothesis: When controlling for confounding variables, a BMI that categorizes a women as obese or morbidly obese, significantly decreases the probability of a successful VBAC and increases maternal and neonatal co-morbidities.

Materials and Methods: Retrospective review of medical records of women with a primary low transverse cesarean delivery that then considered a trial of labor in their subsequent pregnancy. The data was taken from the Stony Brook University Hospital Nurse Midwifery practice database and included the deliveries between January 2004 and December 2008. The women were divided into underweight, normal, overweight, and obese as defined by The National Heart Lung and Blood Institute. The primary outcome of interest was VBAC success rate. Other outcomes of interest included maternal and neonatal complications. For purposes of comparison the women were grouped into the "not obese" versus "obese".

Results: Two hundred fifty nine women were included; 209 (80.7%) were not obese and 50 (19.3%) were obese. One hundred twenty two (58.4%) not obese women and 24 (48%) obese women underwent TOLAC (P = 0.21). There was no difference in VBAC success rate; 85 (69.7%) not obese and 18 (75%) obese women were successful in delivering vaginally (P = 0.81). Women in the not obese group were older (30.9 $\pm$ 5.8 y vs. 29.2 $\pm$ 5.2 y; P = 0.03) and obese women had more gestational diabetes mellitus (18% vs. 2.9%; P <0.001); otherwise, the two groups were similar in parity, gestational age, smoking, drug use, preeclampsia, asthma, post partum hemorrhage or need for blood transfusion. There were no differences in birth weight, incidence of LGA neonates, low 5 minute Apgar scores, and NICU admissions. Of those women who underwent TOLAC, there was no significant difference in reason for failed VBAC. In the not obese group, 46% (17/37) had non-reassuring fetal heart rate tracing (NRFHT) and 37.8% (14/37) had arrest of labor while in the obese group 16.7% (1/6) had NRFHT and 66.7% (4/6) had arrest of labor.

Conclusion: The data in this study did not confirm the hypothesis that obesity contributes to decreased success of VBAC.

Factors Predicting Gestational Age at Delivery Following Ultrasound-Indicated Cervical Cerclage

Jolene Muscat M.D. and Paul L. Ogburn, Jr., MD

Objective: To determine which, if any, maternal, obstetric and pregnancy characteristics predict gestational age (GA) at delivery following ultrasound-indicated cervical cerclage.

Study Design: Retrospective analysis of patients with singleton gestations who underwent ultrasound-indicated cerclage placement between January 2006 and December 2008. The following predictors were evaluated: maternal age, gravidity, parity, ethnicity, obstetrical history, prior cervical surgery, prior D&C, pre- and post-cerclage cervical length (CL), amniocentesis utilization, pre-cerclage exposure of the membranes to the vagina and GA at cerclage placement. The primary outcome evaluated was spontaneous preterm birth (SPTB) at <32 weeks. SPTB prior to 24 and 37 weeks were also evaluated as secondary outcomes. Statistical analysis was performed using the Wilcoxon Rank-sum or Fisher´s exact test when appropriate. Multivariable regression analysis was used to identify the independent effects of the predictors in determining GA at delivery.

Results: 80 patients met inclusion criteria. From the aforementioned predictors, only pre- and post-cerclage CL and pre-cerclage exposure of the membranes to the vagina were significantly associated with SPTB <32 weeks. Similar results were seen with SPTB prior to 24 and 37 weeks. The remaining predictors were not significant.

Table 1: Predictors of Delivery <32 weeks

Variable	Delivery < 32 weeks: No (n-64)	Yes (n=16)	P value
Pre-cerclage CL	1.5 (0-2.5)	0.5 (0-2.5)	0.0002
Post-cerclage CL	2.8 (o.5-5.0)	1.7 (0-4.8)	0.015
Membranes	21 (33%)	13 (81%)	0.0006

Data are median (range) or N(%)

Table 2: Odds Ratios for Delivery <32 weeks

Variable	Odds Ratio (95% CI)
Pre-cerclage CL ≤ 1.2 cm	14.6 (2.8076.0)
Post-cerclage CL ≤ 2.4 cm	6.184 (1.6-23.3)
Membranes exposed	8.6 2.1-35.4)

Conclusion: Pre- and post-cerclage cervical length and pre-cerclage exposure of the membranes to the vagina are the most predictive variables for spontaneous preterm delivery after ultrasound-indicated cervical cerclage.

Fasting Plasma Active Glucagon-like Peptide-1 in Pregnancies with and without Gestational Diabetes

Elizabeth Garduno MD MPH, Jarrett Santorelli BS, Alexander Kong, Jacob Mathai, Hyeong Jun Ahn PhD, Juana Gonzalez PhD, Andrew Lane MD and Cecilia Avila MD, MPH

Background: Glucagon-like peptide-1 (GLP-1) is an incretin hormone derived from proglucagon and is secreted from intestinal endocrine L-cells in response to nutrient ingestion. GLP-1 stimulates glucose-dependent insulin secretion, inhibits glucagon, reduces food intake and regulates pancreatic beta cell mass. Decreased GLP-1 has been noted in adults with type 2 diabetes mellitus. Alterations in GLP-1 levels may have important effects on the fetus such as development of the fetal pancreas. It was previously established by Ostrov et al. that GLP-1 could be quantified in umbilical cord blood. More recently, maternal GLP-1 levels have been shown to increase throughout gestation. We hypothesized that plasma GLP-1 levels would be lower in samples obtained from gestational diabetic mothers (GDM) as compared to those in non-diabetic mothers. We also hypothesized that fetal GLP-1 levels would not differ significantly between umbilical arterial and venous plasma.

Objectives: 1) to compare the levels of GLP-1 in patients with GDM with patients with normal GDM screening results and 2) to establish if the levels of GLP-1 in the umbilical artery were different from the levels in the umbilical vein at birth.

Methods: 17 GDM and 22 non-diabetic pregnant women who were scheduled for a term cesarean delivery were recruited. Maternal blood samples were obtained prior to delivery. After delivery of the infant, both venous and arterial umbilical cord blood were obtained. Dipeptidyl peptidase-4 (DPP-IV) inhibitor was added immediately to all samples from which GLP-1 was to be measured to inhibit proteolytic cleavage of GLP-1. The samples were centrifuged at 4°C for 5 minutes. Plasma was stored at -70°C. Electrochemiluminescent assay (Meso Scale Discovery®) was used to quantify GLP-1 in the samples.

Results: There was no significant difference in maternal, fetal arterial, or fetal venous GLP-1 in GDM versus non-diabetic mothers (t test, signed rank sum test). There was a positive correlation between maternal BMI and maternal GLP-1 ($r=0.33$, $p=0.05$), fetal arterial GLP-1($r=0.46$, $p=0.005$), and fetal venous GLP-1 ($r=0.45$, $p=0.005$). There were no significant differences between fetal arterial and venous levels of GLP-1 (paired t test).

Conclusions: No difference was found between GLP-1 levels in gestational diabetic and non-diabetic mothers. This unexpected finding may be due to small sample size, wide variation in levels of glycemic control amongst subjects with GDM, or elevated BMI in both groups. Our findings suggest GLP-1 is increased in association with elevated BMI but is not increased with GDM. There was no significant difference between GLP-1 levels in samples obtained from the umbilical arteries and vein. This might be explained by presence of the physiological shunts in the fetal circulation or the possibility of no fetal GLP-1 production in utero. Future studies involving fetal GLP-1 may be conducted on mixed umbilical cord blood.

A Retrospective Investigation of Endometrial Pathology in Relationship to the Bleeding Profiles of Premenopausal Females Undergoing Endometrial Biopsies in Order to Determine a Risk Categorization

Cara Ninivaggio MD and Erin E. Stevens MD

Objective: The purpose of the study is to characterize the bleeding patterns of premenopausal females that are most associated with abnormal pathology on an endometrial biopsy. This will be the first step of a multiphase project to create a risk assessment process to determine when to proceed with an endometrial biopsy in premenopausal females with irregular bleeding.

Null hypothesis: There will be no correlation between a diagnosis of endometrial pathology and different bleeding profiles.

Background: Endometrial cancer is the most common gynecologic malignancy in the United States. The mean age of diagnosis is 61, however, 5 to 30% of cases are found by endometrial sampling in those less than age 50. Additionally, a high incidence of premenopausal women with endometrial cancer have a synchronous ovarian cancer.

Few studies exist regarding the risk of development of endometrial pathology in this premenopausal cohort. This is likely due to a limited amount of data. However, of those that were published, proposed risk factors include increased body mass index (BMI), nulliparity, polycystic ovarian syndrome (PCOS), a history of infertility, and irregular menstrual cycles. Risk factors still under investigation include diabetes mellitus and hypertension. No study to date has categorized risk based on the patient's specific bleeding profile other than "irregular menses."

Method: A retrospective review of charts will be conducted of the cohort of all premenopausal patients between the ages of 18 and 50 who have had endometrial tissue sampling by Pipelle biopsy for an original complaint of any abnormal uterine bleeding. Those excluded will be patients who have preexisting malignancies. Records from Stony Brook University Hospital satellite clinic throughout the past 5 years will be assessed. Data to be collected include age, menstrual history and bleeding profile. The type of abnormal uterine bleeding to be evaluated are: amenorrhea, oligomenorrhea, menorrhagia, menometrorrhagia, and the length of time that the irregularities have existed. This will be compared to the outcomes of the endometrial biopsies, which will be hyperplasia or endometrial cancer. Based on a statistical analysis of our data and outcomes of tissue diagnosis, it will be investigated as to whether an association can be made between characterization of abnormal uterine bleeding and the existence of endometrial pathology. If there is an association, a risk categorization process will be attempted.

Planned Course: Obtain IRB approval, collect and analyze data.

Is Ultrasound Finding of Single Umbilical Artery Associated with Fetal Cardiac Anomaly?

Chanda Reese MD, Reinaldo Figueroa MD, Jolene Muscat MD and Lillian Meek RN

Objective: Our primary objective was to determine if the ultrasound finding of a single umbilical artery (SUA) is associated with fetal cardiac anomalies. The presence of other associated fetal anomalies and the ability to accurately diagnose these conditions in our antepartum testing unit were evaluated as a secondary objective.

Background: A fetus with a SUA complicates between 0.25% and 1% of pregnancies. Upon review of the literature, fetal cardiac anomaly has been associated with the finding of SUA in 0-15% of cases. For this reason, some have advocated for use of fetal echocardiogram evaluation in the antenatal period. Additionally, SUA has been associated with anomalies of the cardiovascular, renal, and gastrointestinal systems as well as chromosomal abnormalities and increased perinatal morbidity. Most studies do not indicate if cardiac anomalies associated with SUA occurred in isolation or if they occurred in association with other fetal anomalies. The purpose of this study was to determine if the ultrasound finding of SUA is associated with fetal cardiac anomaly, and if so, whether this association is limited to cardiac anomalies or indicative of other fetal abnormality.

Study Design: We performed a retrospective chart review of all patients who received a fetal anatomic survey between 1/2004 through 12/2009 and had the finding of SUA. Study subjects that could not be linked to neonatal outcomes or lacked placental pathology were excluded. Neonatal and placental pathology results were then reviewed to confirm presence of SUA after delivery and determine incidence of coexisting anomalies including congenital heart disease (CHD), renal, skeletal and gastrointestinal anomaly, chromosomal anomalies and intrauterine growth restriction.

Results: A total of 154 patients had sonographic evidence of single umbilical artery. 61 patients were excluded as neonatal and/or placental pathology records were not available, leaving 93 patients eligible for review.

72 of 93 patients (77%) had a confirmed two vessel cord (2VC) and 21 had a three vessel cord (23%). These 21 patients were excluded from further analysis. SUA occurred as an isolated ultrasound finding in 53 of 72 patients (74%). In the remaining 19 patients with 2VC, other fetal anomalies were appreciated on ultrasound.

In the group with isolated SUA, 24 of 53 patients had fetal echocardiography, all of which were normal. On subsequent neonatal evaluation, 1 cardiac anomaly (VSD with PDA) and 3 renal anomalies were identified. In the group with SUA and other anomalies, 2 of 19 patients had fetal echocardiography, both of which were normal. There were 2 cases of cardiac anomalies (1 TOF and 1VSD) both of which were diagnosed on fetal anatomical survey and did not have echocardiography. 15 other anomalies were also noted in this group and included cases of renal, gastro-intestinal, chromosomal, and skeletal anomalies. All were identified prior to delivery.

Conclusions: Ultrasound diagnosis of single umbilical artery occurs as an isolated finding in the majority of patients with confirmed 2VC upon delivery. Fetal echocardiography did not contribute significant clinical information above that obtained on fetal anatomic survey. Additionally, our antepartum testing unit was able to identify all cases of cardiac and associated fetal anomalies in this study cohort.

Chorioamnionitis: A Retrospective Study Analyzing the Accuracy of Clinical Diagnosis in Preterm Pregnancies

Leia Card MD and Reinaldo Figueroa MD

Objective: More than 12% of all deliveries in the United States are considered premature deliveries. 33% of preterm deliveries are diagnosed with histologic chorioamnionitis. Clinical chorioamnionitis is diagnosed in 1-10% of all pregnancies and its diagnosis is important because of the associated increased risk of neonatal and maternal morbidity and mortality. Prior studies have demonstrated a large discrepancy between the clinical and histological diagnosis of chorioamnionitis in preterm deliveries. The purpose of this study is to investigate the clinical diagnostic accuracy of chorioamnionitis in preterm deliveries and its effect on neonatal outcomes.

Study Design: We identified all singleton preterm infants delivered at Stony Brook Hospital from January 1, 2005 through December 31, 2008 from the Neonatal Intensive Care Unit database. A retrospective chart review will be conducted on both the maternal and neonatal records. Inclusion criteria are: any woman that delivered a live born singleton preterm infant (24 weeks gestation through 36 6/7 weeks gestation). Exclusion criteria are: multiple gestations, intrauterine fetal demise, and neonates with structural abnormalities or known chromosomal abnormalities. Maternal records will be reviewed for a clinical diagnosis of chorioamnionitis. We will define clinical chorioamnionitis as a maternal temperature of greater than 37.8 degrees Celsius and two or more of the following: uterine tenderness, malodorous vaginal discharge, elevated maternal white blood cell count (greater than 15,000 leukocytes/mm^3), maternal tachycardia (greater than 100 bpm) or fetal tachycardia (greater than 160 bpm). All placentas have been previously examined, and a diagnosis of histological chorioamnionitis will be noted if present on pathology report. Neonatal records will be reviewed for the presence of bronchopulmonary dysplasia, necrotizing enterocolitis, intraventricular hemorrhage, periventricular leukomalacia, respiratory distress syndrome and mortality. We will evaluate the percentage of preterm deliveries diagnosed with clinical chorioamnionitis that correlate with histological chorioamnionitis, and which combinations of diagnostic criteria most accurately diagnose preterm chorioamnionitis. Finally, we will analyze which specific clinical diagnostic criteria correlate with worse neonatal outcomes.

Results: In process of collection

Conclusions: Pending

Special thanks to Randi Turkewitz, MD