

THE AMERICAN ASSOCIATION OF
**ENDOCRINE
SURGEONS**

Thirty-Second Annual Meeting



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American Association of Endocrine Surgeons

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American Association of Endocrine Surgeons

www.endocrinesurgery.org

AAES FUTURE MEETINGS

April 29 - May 1, 2012

Iowa City, Iowa

Ronald J. Weigel, MD, PhD

April 14 - 16, 2013

Chicago, Illinois

Peter Angelos, MD, PhD

April 27 - 29, 2014

Boston, Massachusetts

Richard A. Hodin, MD

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OFFICERS, COUNCIL & COMMITTEES

OFFICERS

Douglas B. Evans, President
Ashok R. Shaha, President-Elect
Gerard M. Doherty, Vice President
Peter Angelos, Secretary-Treasurer
Steven K. Libutti, Recorder

COUNCIL

Michael J. Demeure
Paul G. Gauger
William B. Inabnet, III
Electron Kebebew
Nancy D. Perrier
Sareh Parangi
Janice L. Pasieka
Julie Ann Sosa
Geoffrey B. Thompson

LOCAL ARRANGEMENTS CHAIR

Nancy D. Perrier

PUBLICATION AND PROGRAM COMMITTEE

Lawrence Kim, Chair
Douglas B. Evans, ex officio
Peter Angelos
Mark Cohen
Marybeth Hughes
Steven K. Libutti
Fiemu Nwariaku
John Olson
Jennifer Rosen
David Terris
Tina Yen

MEMBERSHIP COMMITTEE

Julie Ann Sosa, Chair
Electron Kebebew
Nancy D. Perrier

COMMITTEE ON EDUCATION AND RESEARCH

William B. Inabnet, III, Chair
Paul G. Gauger
Michael McLeod
Mira Milas
Barbra Miller
Sareh Parangi
Renu Sinha
Cord Sturgeon

FELLOWSHIP COMMITTEE

Allan B. Siperstein, Chair
Peter Angelos
William B. Inabnet, III
Greg Randolph
Melanie Richards
Tracy Wang

AACE REPRESENTATIVE

Martha A. Zeiger

ESC REPRESENTATIVE

Sonia L. Sugg

SOAC REPRESENTATIVE TO ABS

Richard A. Prinz

NSQIP REPRESENTATIVE

Julie Ann Sosa

PAUL LoGERFO COMMITTEE

Jack M. Monchik, Chair
John A. Chabot

NOMINATING COMMITTEE

Michael J. Demeure, Chair
Douglas B. Evans
Janice L. Pasieka

INFORMATION TECHNOLOGY COMMITTEE

Peter Angelos, Chair
Janice L. Pasieka
James Lee
Michael Yeh
Richard A. Hodin

AAES FOUNDATION

Geoffrey B. Thompson, Chair
John A. Chabot
Jack M. Monchik
Janice L. Pasieka
Robert Udelsman
Peter Angelos
William B. Inabnet, III

PAST OFFICERS

1980-1981

Norman W. ThompsonPresident
Orlo H. Clark Vice President
John M. Monchik.....Secretary-Treasurer

1981-1982

Norman W. ThompsonPresident
Orlo H. Clark Vice President
John M. Monchik.....Secretary-Treasurer

1982-1983

Edwin L. Kaplan.....President
Blake Cady Vice President
John M. Monchik.....Secretary-Treasurer

1983-1984

Stanley R. FriesenPresident
John A. Palmer Vice President
John M. Monchik.....Secretary-Treasurer

1984-1985

Leonard RosoffPresident
John M. Monchik..... Vice President
Stuart D. WilsonSecretary-Treasurer

1985-1986

Chiu-An WangPresident
Edward Paloyan..... Vice President
Stuart D. WilsonSecretary-Treasurer

1986-1987

Oliver BehrsPresident
Robert C. Hickey Vice President
Stuart D. WilsonSecretary-Treasurer

1987-1988

Edward Paloyan.....President
Caldwell B. Esselstyn..... Vice President
Stuart D. WilsonSecretary-Treasurer
Jon A. van Heerden.....Recorder

1988-1989

John R. BrooksPresident
Melvin A. Block Vice President
Richard A. Prinz.....Secretary-Treasurer
Jon A. van Heerden.....Recorder

PAST OFFICERS CONT.

1989-1990

Colin G. Thomas, Jr.....President
Carl R. Feind..... Vice President
Richard A. Prinz.....Secretary-Treasurer
Jon A. van Heerden.....Recorder

1990-1991

Caldwell B. Esselstyn.....President
Brown M. Dobyns..... Vice President
Richard A. Prinz.....Secretary-Treasurer
Robert D. Croom, III.....Recorder

1991-1992

Stuart D. Wilson.....President
Joseph N. Attie..... Vice President
Blake Cady.....Secretary-Treasurer
Robert D. Croom, III.....Recorder

1992-1993

Robert C. Hickey.....President
Patricia J. Numann..... Vice President
Blake Cady.....Secretary-Treasurer
Robert D. Croom, III.....Recorder

1993-1994

Orlo H. Clark.....President
Glen W. Geelhoed..... Vice President
Blake Cady.....Secretary-Treasurer
George L. Irvin, III.....Recorder

1994-1995

John M. Monchik.....President
Jon A. van Heerden..... Vice President
Jay K. Harness.....Secretary-Treasurer
George L. Irvin, III.....Recorder

1995-1996

Richard A. Prinz.....President
Jeffrey A. Norton..... Vice President
Jay K. Harness.....Secretary-Treasurer
George L. Irvin, III.....Recorder

PAST OFFICERS CONT.

1996-1997

Jon A. van Heerden.....President
George L. Irvin, III.....Vice President
Jay K. Harness.....Secretary-Treasurer
Quan-Yang Duh.....Recorder

1997-1998

Blake Cady.....President
E. Christopher Ellison.....Vice President
Paul LoGerfo.....Secretary-Treasurer
Quan-Yang Duh.....Recorder

1998-1999

George L. Irvin, III.....President
Barbara K. Kinder.....Vice President
Paul LoGerfo.....Secretary-Treasurer
Quan-Yang Duh.....Recorder

1999-2000

Jay K. Harness.....President
John S. Kukora.....Vice-President
Paul LoGerfo.....Secretary-Treasurer
Michael J. Demeure.....Recorder

2000-2001

Barbara K. Kinder.....President
Martha A. Zeiger.....Vice-President
Christopher R. McHenry.....Secretary-Treasurer
Michael J. Demeure.....Recorder

2001-2002

Clive S. Grant.....President
Miguel F. Herrera.....Vice-President
Christopher R. McHenry.....Secretary-Treasurer
Michael J. Demeure.....Recorder

2002-2003

Quan-Yang Duh.....President
Gary B. Talpos.....Vice-President
Christopher R. McHenry.....Secretary-Treasurer
Geoffrey B. Thompson.....Recorder

2003-2004

Paul LoGerfo.....President
Ashok R. Shaha.....Vice-President
Janice L. Pasioka.....Secretary-Treasurer
Geoffrey B. Thompson.....Recorder

PAST OFFICERS CONT.

2004-2005

John A. KukoraPresident
Andrew W. SaxeVice-President
Janice L. Pasieka.....Secretary-Treasurer
Geoffrey B. ThompsonRecorder

2005-2006

Robert Udelsman.....President
Collin J. WeberVice-President
Janice L. Pasieka.....Secretary-Treasurer
Douglas B. EvansRecorder

2006-2007

Christopher R. McHenry.....President
John B. HanksVice-President
Sally E. Carty.....Secretary-Treasurer
Douglas B. EvansRecorder

2007-2008

Geoffrey B. ThompsonPresident
Terry C. Lairmore.....Vice-President
Sally E. Carty.....Secretary-Treasurer
Douglas B. EvansRecorder

2008-2009

Michael J. DemeurePresident
Jeffrey F. MoleyVice-President
Sally E. Carty.....Secretary-Treasurer
Steven K. Libutti.....Recorder

2009-2010

Janice L. Pasieka.....President
Jeffrey E. LeeVice-President
Peter AngelosSecretary-Treasurer
Steven K. Libutti.....Recorder

THE OLIVER COPE MERITORIOUS ACHIEVEMENT AWARD

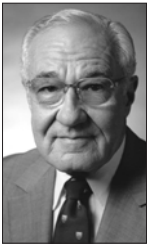
In April of 1984 at the American Association of Endocrine Surgeons Meeting in Kansas City, Drs. Edward Kaplan, Jack Monchik, Leonard Rosoff, Norm Thompson and Stuart Wilson proposed to the Council a new achievement award. The award honors a member of the AAES in recognition for contributions in the field of endocrine surgery as an investigator, teacher and clinical surgeon. It is not an annual award but is to be given to members of our Association who truly aspire to the spirit of this award.

On April 15th, 1985 at the annual meeting of the AAES in Toronto, our President Leonard Rosoff announced the first member to receive this award, Dr. Oliver Cope. In giving this award to Dr. Cope the decision of the Council was that from this day forward the award would be known as the Oliver Cope Meritorious Achievement Award for the American Association of Endocrine Surgeons.



Oliver Cope, MD

Professor of Surgery,
Harvard University and the
Massachusetts General Hospital
Awarded in Toronto in April 1985.



Stanley R. Friesen, MD, PhD

Professor of Surgery, University of Kansas
Awarded in Detroit, MI in April 1994
Dr. Friesen served as the President of our Association in 1983.



Norman W. Thompson, MD

Henry King Ransom Professor of Surgery, University of Michigan
Awarded in Atlanta, GA in April 2001
Dr. Thompson served as our inaugural President in 1980 and also in 1981.

THE OLIVER COPE MERITORIOUS ACHIEVEMENT AWARD CONT.



Jon A. van Heerden, MD

Professor of Surgery Mayo Clinic, Rochester, MN
Awarded in Charlottesville, NC in April 2004
Dr. van Heerden served as our Recorder from 1987-1989, as our Vice-President in 1994, and as President in 1996



Orlo H. Clark, MD

Professor of Surgery, UCSF Mount Zion Medical Center
Awarded in New York, NY in May 2006. Dr. Clark served as our inaugural Vice President in 1980 and also in 1981, and as President in 1993



Edwin L. Kaplan, MD

Professor of Surgery, University of Chicago
Awarded in Madison, WI in May 2009
Dr. Kaplan served as our President in 1982



George L. Irvin, III, MD

Professor Emeritus of Surgery, University of Miami
Awarded in Pittsburgh, PA in April 2010
Dr. Irvin served as our Recorder from 1993-1996, Vice President in 1996 and President in 1998.

HONORARY MEMBERS

Individuals who have made outstanding contributions to the discipline of Endocrine Surgical Disease

J. Aidan Carney, Pathologist

Stuart D. Flynn, Pathologist

Ian D. Hay, Endocrinologist

Virginia A. LiVolsi, Pathologist

A. G. E. "Ace" Pearse, Endocrinologist

Thomas S. Reeve, Endocrine Surgeon

F. John Service, Endocrinologist

Britt Skogseid, Endocrinologist

Robert Tuttle, Endocrinologist

William F. Young, Endocrinologist

RESIDENT/FELLOW RESEARCH AWARD WINNERS & POSTER COMPETITION WINNERS

The AAES Resident/Fellow Research Award was established in 1990 to encourage interest in endocrine surgery by those training as students and residents in general surgery. Presented work may be honored in either the Clinical or Basic Research categories.

The AAES Poster Competition was established in 2007.

1990

Michael J. Demeure - San Francisco, California

"Actin Architecture of Cultured Human Thyroid Cancer Cells: Predictor of Differentiation?"

Gerard M. Doherty - Bethesda, Maryland

"Time to Recovery of the Hypothalamic-Pituitary-Adrenal Axis After Curative Resection of Adrenal Tumors in Patients with Cushing's Syndrome"

1996

Jennifer Meko - St. Louis, Missouri

"Evaluation of Somatostatin Receptor Scintigraphy in Detecting Neuroendocrine Tumors"

Beth A. Ditkoff - New York, New York

"Detection of Circulating Thyroid Cells in Peripheral Blood"

1997

Herb Chen - Baltimore, Maryland

"Implanted Programmable Insulin Pumps: 153 Patient Years of Surgical Experience"

K. Michael Barry - Rochester, Minnesota

"Is Familial Hyperparathyroidism a Unique Disease?"

1998

Julie Ann Sosa - Baltimore, Maryland

"Cost Implications of the Different Management Strategies for Primary Hyperparathyroidism in the US"

David Litvak - Galveston, Texas

"A Novel Cytotoxic Agent for Human Carcinoid"

RESIDENT/FELLOW RESEARCH AWARD WINNERS & POSTER COMPETITION WINNERS CONT.

1999

Andrew Feldman - Bethesda, Maryland

"Results of Heterotrophic Parathyroid Autotransplantation: A 13 Year Experience"

Alan Dackiw - Houston, Texas

"Screening for MEN1 Mutations in Patients with Atypical Multiple Endocrine Neoplasia"

2000

Electron Kebebew - San Francisco, California

"ID1 Proteins Expressed in Medullary Thyroid Cancer"

2001

Nestor F. Esnaola - Houston, Texas

"Optimal Treatment Strategy in Patients with Papillary Thyroid Cancer: A Decision Analysis"

Katherine T. Morris - Portland, Oregon

"High Dehydroepiandrosterone-Sulfate Predicts Breast Cancer Progression During New Aromatase Inhibitor Therapy and Stimulates Breast Cancer Cell Growth in Tissue Culture: A Renewed Role for Adrenalectomy"

2002

Rasa Zarnegar - San Francisco, California

"Increasing the Effectiveness of Radioactive Iodine Therapy in the Treatment of Thyroid Cancer Using Trichostatin A (TSA), A Histone Deacetylase (HDAC)"

Denise M. Carneiro - Miami, Florida

"Rapid Insulin Assay for Intraoperative Confirmation of Complete Resection of Insulinomas"

2003

Petra Musholt - Hanover, Germany

"RET Rearrangements in Archival Oxyphilic Thyroid Tumors: New Insights in Tumorigenesis and Classification of Hürthle Cell Carcinoma"

Tina Yen - Houston, Texas

"Medullary Thyroid Carcinoma: Results of a Standardized Surgical Approach in a Contemporary Series of 79 Consecutive Patients from The University of Texas, M. D. Anderson Cancer Center in Houston"

RESIDENT/FELLOW RESEARCH AWARD WINNERS & POSTER COMPETITION WINNERS CONT.

2004

Rebecca S. Sippel – Madison, Wisconsin

“Does Propofol Anesthesia Affect Intra-Operative Parathyroid Hormone Levels During Parathyroidectomy?: A Randomized Prospective Trial”

David Finley – New York, New York

“Molecular Analysis of Hürthle Cell Neoplasms by Gene Profiling”

2005

Mark Cohen – St. Louis, Missouri

“Long-Term Functionality of Cryopreserved Parathyroid Autografts: A 13-Year Prospective Analysis”

Kepal N. Patel – New York, New York

“MUC1 Plays a Role in Tumor Maintenance in Aggressive Thyroid Carcinomas”

2006

Kyle Zano – Chicago, Illinois

“Cost-Effectiveness Analysis of Minimally Invasive Parathyroidectomy for Asymptomatic Primary Hyperparathyroidism”

Ashley Kappes Cayo – Madison, Wisconsin

“Lithium Ions: a Novel Agent for the Treatment of Pheochromocytomas and Paragangliomas”

2007

Tracy S. Wang – New Haven, Connecticut

“How Many Endocrine Surgeons Do We Need?”

David Yu Greenblatt – Madison, Wisconsin

“Valproic Acid Activates Notch1 Signaling and Inhibits Growth in Medullary Thyroid Cancer Cells”

RESIDENT/FELLOW RESEARCH AWARD WINNERS & POSTER COMPETITION WINNERS CONT.

2008

Elizabeth G. Grubbs - Houston, Texas

"Preoperative Vitamin D (VITD) Replacement Therapy in Primary Hyperparathyroidism (PHPT): Safe But Beneficial?"

Linwah Yip - Pittsburgh, Pennsylvania

"Loss of Heterozygosity of Selected Tumor Suppressor Genes in Parathyroid Carcinoma"

Poster: Pierre Leyre - Poitiers, France

"Does the Risk of Compressive Hematoma After Thyroidectomy Authorize One-Day Surgery?"

2009

Insoo Suh - San Francisco, California

"Candidate Germline Alterations Predisposing to Familial Nonmedullary Thyroid Cancer Map to Distinct Loci on Chromosomes 1 and 6"

Susan C. Pitt - Madison, Wisconsin

"Tertiary Hyperparathyroidism: Is Less Than a Subtotal Resection Ever Appropriate? A Study of Long-term Outcomes"

Poster: Matthew Nehs - Boston, Massachusetts

"Inhibition of B-RAFV600 Oncoprotein Prevents Cell Cycle Progression and Invasion In Vitro and Reduces Tumor Growth and Metastasis in an In Vivo Orthotopic Model of Thyroid Cancer"

Poster: Bian Wu - Los Angeles, California

"Utilization of Parathyroidectomy in the Elderly: A Population-Based Study"

2010

David T. Hughes - Ann Arbor, Michigan

"Routine Central Lymph Node Dissection For Papillary Thyroid Cancer"

Matthew A. Nehs - Boston, Massachusetts

"Thyroidectomy With Neoadjuvant Plx4720 Extends Survival And Decreases Tumor Burden In An Orthotopic Mouse Model Of Anaplastic Thyroid Cancer"

Poster: Aarti Mathur - Bethesda, Maryland

"Adrenal Venous Sampling in Primary Hyperaldosteronism: Standardizing A Gold Standard"

2010-2011 NEW MEMBERS

ACTIVE MEMBERS

Leaque Ahmed

New York, NY

Naris Nilubol

Bethesda, MD

Antonia E. Stephen

Boston, MA

Jessica E. Gosnell

San Francisco, CA

Steven E. Rodgers

Miami, FL

Stanley Z. Trooskin

New Brunswick, NJ

James Lee

New York, NY

Randall P. Scheri

Durham, NC

Tracy S. Wang

Milwaukee, WI

Christina L. Maser

Fresno, CA

Rebecca S. Sippel

Madison, WI

Kaare J. Weber

New York, NY

Peter J. Mazzaglia

Providence, RI

Michael R. Starks

Bangor, ME

ALLIED SPECIALIST MEMBERS

Gary L. Clayman

Houston, TX

David L. Steward

Cincinnati, OH

CORRESPONDING MEMBERS

Iihara Masatoshi

Tokyo, Japan

Martin K. Walz

Essen, Germany

2010-2011 NEW MEMBERS CONT.

CANDIDATE MEMBERS

Todd D. Beyer

Albany, NY

David Hughes

New York, NY

Christiana M. Shaw

Gainesville, FL

Jennifer Bocker

New York, NY

Monica E. Lopez

Houston, TX

Joyce Shin

Cleveland, OH

Glenda Callender

Louisville, KY

Adrienne Melck

Vancouver, BC

Gardner Smith

Birmingham, AL

Jennifer Cannon

Winston-Salem, NC

Tricia Moo-Young

Chicago, IL

Mark S. Sneider

St. Paul, MN

Tobias Carling

New Haven, CT

Shane Morita

Honolulu, HI

Sam Van Slycke

Aalst, Belgium

Emery Chen

Woodland, CA

Amy Quillo

Louisville, KY

Kimberly Vanderveen

Albuquerque, NM

Marlon Guerrero

Tucson, AZ

Daniel Ruan

Boston, MA

Avital Harari

San Francisco, CA

Alfredo Santillan

San Antonio, TX

RESIDENT / FELLOW MEMBERS

Cameron D. Adkisson

Jacksonville, FL

Carrie Jahraus

New York, NY

Daaron McField

Newark, DE

Amal Y. Alhefdhi

Madison, WI

Adam S. Kabaker

Pittsburgh, PA

Reese W. Randle

Salem, NC

Keith M. Baldwin

Providence, RI

Leon Kushnir

Delmar, NY

Rashmi Roy

Baltimore, MD

Donald M. Cheatem

Chicago, IL

Amanda M. Laird

Ann Arbor, MI

David F. Schneider

Maywood, IL

Jovenel Cherenfant

Evanston, IL

Christine Landry

Houston, TX

Robert Tasevski

Toronto, ON

Laura Eichhorn-Wharry

Detroit, MI

Cortney Lee

Temple, TX

N. Gopalakrishna Iyer

New York, NY

Jennifer L. Marti

New York, NY

2010-11 CONTRIBUTORS TO THE PAUL LOGERFO EDUCATIONAL RESEARCH FUND



Dr. Paul LoGerfo passed away September 16, 2003 during his tenure as President of the AAES. Dr. LoGerfo was very interested in education and clinical research, and in his honor the AAES established the Educational Research Fund to support educational and research activities of the Membership. As of press time, the following members and organizations contributed in 2010-11:

**Peter Angelos
Thomas A. Broadie
Samuel P. Bugis
Blake Cady
Herbert Chen
Gary C. Clark
Mark S. Cohen
Steven A. De Jong
Gerard M. Doherty
Mete Duren
Douglas B. Evans
Thomas J Fahey
Erin A. Felger
Douglas L. Fraker
Scott F. Gallagher
Clive Grant
John B. Hanks
Jay K. Harness
Keith S. Heller
Richard A. Hodin
William M. Hopkins
Marybeth Hughes
Emad Kandil
Electron Kebebew
Barbara K. Kinder
James Lee**

**John I. Lew
Steven K. Libutti
Chung-Yau Lo
LoGerfo Family
Carrie C. Lubitz
Dougald C. MacGillivray
Lloyd Mack
Michael R. Marohn
Christina L. Maser
Peter J. Mazzaglia
David McAneny
Kelly L. McCoy
Christopher McHenry
Adrienne L. Melck
Bradford K. Mitchell
Alberto S. Molinari
Jack M. Monchik
Patricia J. Numann
Takao Obara
Janice L. Pasieka
Subhash Patel
Walter E. Pofahl
Richard A. Prinz
Michael Roe
Anatoly F. Romanchishen
Irving B. Rosen**

**J.M. Sanchez-Blanco
Rick J. Schmidt
Frederic N. Sebag
Melwyn J. Sequeira
Ashok Shaha
Dietmar Simon
Renu Sinha
Rebecca S. Sippel
Carmen Solorzano
Sonia L. Sugg
Beth H. Sutton
Gary B. Talpos
Serdar T. Tezelman
Geoffrey B. Thompson
Norman W. Thompson
Doug R. Trostle
Jon van Heerden
James J. Vopal
Kristin E. Wagner
Martin K. Walz
Collin J. Weber
Ronald D. Wenger
Stuart D. Wilson
Michael W. Yeh
Martha A. Zeiger**

PAST MEETINGS

1980 - **Ann Arbor, Michigan**

Local Arrangements Chair: Norman Thompson

1981 - **Washington, DC**

Local Arrangements Chair: Glenn Geelhoed

1982 - **Houston, Texas**

Local Arrangements Chair: Robert C. Hickey

1983 - **San Francisco, California**

Local Arrangements Chair: Orlo Clark

1984 - **Kansas City, Kansas**

Local Arrangements Chair: Stanley Friesen

1985 - **Toronto, Ontario, Canada**

Local Arrangements Chair: Irving Rosen

1986 - **Rochester, Minnesota**

Local Arrangements Chair: Jon van Heerden

1987 - **Chicago, Illinois**

Local Arrangements Chair: Edwin Kaplan

1988 - **Boston, Massachusetts**

Local Arrangements Chair: Blake Cady

1989 - **Chapel Hill, North Carolina**

Local Arrangements Chair: Robert D. Croom

1990 - **Cleveland, Ohio**

Local Arrangements Chair: Caldwell B. Esselstyn

1991 - **San Jose, California**

Local Arrangements Chair: Maria Allo

1992 - **Miami, Florida**

Local Arrangements Chair: George L. Irvin

1993 - **Williamsburg, Virginia**

Local Arrangements Chair: H. Heber Newsome

1994 - **Detroit, Michigan**

Local Arrangements Chair: Gary B. Talpos

1995 - **Philadelphia, Pennsylvania**

Local Arrangements Chair: John Kukora

PAST MEETINGS CONT.

1996 - **Napa, California**

Local Arrangements Chair: Quan-Yang Duh

1997 - **Baltimore, Maryland**

Local Arrangements Chair: Robert Udelsman

1998 - **Orlando, Florida**

Local Arrangements Chair: Peter J. Fabri

1999 - **New Haven, Connecticut**

Local Arrangements Chair: Barbara Kinder

2000 - **Joint Meeting: London, United Kingdom/Lille, France**

Local Arrangements Chair: John Monchik

2001 - **Atlanta, Georgia**

Local Arrangements Chair: Collin Weber

2002 - **Banff, Alberta, Canada**

Local Arrangements Chair: Janice L. Pasieka

2003 - **San Diego, California**

Local Arrangements Chair: Jay K. Harness/John Kukora

2004 - **Charlottesville, Virginia**

Local Arrangements Chair: John B. Hanks

2005 - **Cancun, Mexico**

Local Arrangements Chair: Miguel F. Herrera

2006 - **New York, New York**

Local Arrangements Chair: Ashok R. Shaha

2007 - **Tucson, Arizona**

Local Arrangements Chair: Michael J. Demeure

2008 - **Monterey, California**

Local Arrangements Chair: Quan-Yang Duh

2009 - **Madison, Wisconsin**

Local Arrangements Chair: Herbert Chen

2010 - **Pittsburgh, Pennsylvania**

Local Arrangements Chair: Sally E. Carty

INVITED LECTURERS



Sunday, April 10, 2011
12:30pm- 1:00pm
Grand Ballroom ABDE

**Historical Lecture:
"Pheochromocytoma Resection:
Now and Then"**

Introduction: Nancy D. Perrier, MD

Speaker: **Jon van Heerden, MD**
Charleston, SC



Sunday, April 10, 2011
4:30pm - 5:30pm
Grand Ballroom ABDE

**Invited Lecturer:
"Major League Baseball - 2011
Economic and Health Related
Issues"**

Introduction: Douglas B. Evans, MD

Speaker: **Allan H. (Bud) Selig,**
9th Commissioner of Major League
Baseball

INVITED LECTURERS AT RECENT MEETINGS

- 1991 **Gregory B. Bulkley, MD**
Johns Hopkins University, Baltimore, Maryland
Endothelial Xanthine Oxidase: a Radical Transducer of Signals and Injury
- 1992 **Donald Coffey, PhD**
Bethesda, Maryland
New Concepts Concerning Cancer
- 1993 **John L. Doppman, MD**
National Institutes of Health, Bethesda, Maryland
Recent Advances in Endocrinologic Imaging
- 1994 **Gordon J. Strewler, MD**
San Francisco, California
The Parathyroid Hormone Related Protein: Clinical and Basic Studies of a Polyfunctional Protein
- 1995 **Ivor M.D. Jackson, MD**
Providence, Rhode Island
Regulation of TSH Secretion: Implications for Disorders of the Thyroid Function
- 1996 **Victor E. Gould, MD**
Rush-Presbyterian-Medical Centre, Chicago, Illinois
The Diffuse Neuroendocrine System: Evolution of the Concept and Impact on Surgery
- 1997 **Bertil Hamberger, MD, PhD**
Karolinska Institute, Stockholm
The Nobel Prize
- 1998 **Susan Leeman, PhD**
Boston University, Boston, Massachusetts
The NeuroPeptides: Substance P and Neurotensin
- 1999 **James Hurley, MD**
Cornell University, New York, New York
Post-Operative Management of Differentiated Thyroid Cancer
- 2000 **James Shapiro, MD**
University of Alberta, Edmonton, Alberta
Pancreatic Islet Cell Transplantation

INVITED LECTURERS AT RECENT MEETINGS

CONT.

- 2001 **Andrew F. Stewart, MD**
University of Pittsburgh, Pittsburgh, Pennsylvania
Parathyroid Hormone-Related Protein: From Hypercalcemia of Malignancy to Gene Therapy from Diabetes
- 2002 **William F. Young Jr., MD**
Mayo Clinic, Rochester, Minnesota
Adrenal-Dependent Hypertension: Diagnostic Testing Insights
- 2003 **Sissy M. Jhiang, MD**
Ohio State University, Columbus, Ohio
Lessons From Thyroid Cancer: Genetics and Gene Therapy
- 2004 **Edward R. Laws Jr, MD**
University of Virginia, Charlottesville, Virginia
The Diagnosis and Management of Cushing's Disease
- 2005 **David Duick, MD**
Phoenix, Arizona
Thyroid Nodules and Mild Primary Hyperparathyroidism: Examples of Clinical Perplexities or Unresolvable Conundrums
- 2006 **Michael Bliss, PhD**
University of Toronto, Toronto
Harvey Cushing and Endo-Criminology
- 2007 **Virginia A. Livolsi, MD**
University of Pennsylvania, Philadelphia, Pennsylvania
Thyroid Nodule FNA and Frozen Section: Partners or Adversaries
- 2008 **F. John Service, MD, PhD**
Mayo Clinic, Rochester, Minnesota
Hypoglycemia in Adults - 80th Anniversary of Hyperinsulinism
- 2009 **Jeffrey M. Trent, PhD**
Translation Genomics Research Institute, Phoenix, Arizona
Integrating Genetics, Genomics, and Biology Towards a More Personalized Medicine
- 2010 **Alexander J.B. McEwan, MB**
University of Alberta, Edmonton, Alberta, Canada
The State of the Art of Radionucleotide Imaging and Therapy in Patients with Neuroendocrine Tumors
-



CONFERENCE INFORMATION

OBJECTIVES

This program is designed for all surgeons seeking the latest developments in endocrine surgical technique and its related research. Through participation in discussions, attendees will be able to explain current developments in the science and clinical practice of endocrine surgery. Members and guests will be able to explain practical new approaches and solutions to relevant concepts and problems in endocrine surgical care.

CME CERTIFICATES AND EVALUATION FORMS

Please complete your evaluation form and return it to the AAES Registration Desk. You may pick up your CME Certificate at this time.

ACCREDITATION STATEMENT

This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of the American College of Surgeons and the American Association of Endocrine Surgeons. The American College of Surgeons is accredited by the ACCME to provide continuing medical education for physicians.

AMA PRA CATEGORY 1 CREDITS™

The American College of Surgeons designates this live activity for a maximum of **17.25 AMA PRA Category 1 Credits™**. Physicians should claim only the credit commensurate with the extent of their participation in the activity.



Division of Education
American College of Surgeons

DISCLOSURE INFORMATION

In compliance with ACCME Accreditation Criteria, the American College of Surgeons, as the accredited provider of this activity, must ensure that anyone in a position to control the content of the educational activity has disclosed all relevant financial relationships with any commercial interest. All reported conflicts are managed by a designated official to ensure a bias-free presentation. Please see the insert to this program for the complete disclosure list.

REGISTRATION

The 32nd Annual Meeting of the AAES will take place at the Hilton Americas-Houston in Houston, Texas. Registration fees are (postmarked before March 10, 2011) \$375 for AAES members, \$425 for non-members, \$225 for residents and fellows, and \$225 for spouses/guests. To register, visit the AAES Website at endocrinesurgery.org. You may register online, or download and fax the completed registration form to AAES, fax: 913-273-9940. The registration fee covers all scientific and social functions, except Saturday's Golf Outing, Tennis Tournament, NASA Tour, Baseball Game and Sunday's Fun Run, Yoga in the Park, and AAES Workshop: What Do I Do Now?.

HOTEL ACCOMMODATIONS

For the convenience of AAES members and guests we have reserved rooms at the Hilton Americas-Houston. However, it is important to make your hotel reservation early in order to ensure availability. The hotel is located downtown near the Minute Maid Baseball Park and the Toyota Center.

Hilton Americas-Houston

1600 Lamar Street
Houston, TX 77010
Telephone: 800-236-2905

Hotel reservations should be made at the Hilton Americas-Houston. The AAES group rate is \$200 per night plus tax.

AIR TRAVEL

The William P. Hobby Airport (HOU) is located approximately 30 minutes from The Hilton Americas-Houston while The George Bush Intercontinental Airport (IAH) is 35-45 minutes from the Hilton Americas-Houston. Houston is a major hub for Continental Airlines.

GROUND TRANSPORTATION

Taxis are readily available from both airports to the Hilton Americas-Houston. The cost for a taxi is approximately \$25 one way from the Houston Hobby Airport or \$45 one way from the George Bush Intercontinental Airport.

Airport Shuttle reservations may be made through Super Shuttle using the Discount Code 2T2A6. To make reservation call 877-300-4VAN (4826).

WEATHER

Springtime in Houston is lovely. Temperatures in mid-April range from high 70s to low 60s. For accurate weather closer to the date of the meeting, please check www.weather.com.

MEETING AGENDA

The 2011 meeting will use the “standard” AAES meeting format. The **Scientific Sessions** will commence Sunday afternoon and extend through Tuesday morning. The **Welcome Reception** will follow the Scientific Sessions on Sunday evening beginning at 7:00 pm. **The Gala Reception and Dinner Banquet** will be held on Monday evening. The Short Oral Presentations of the top 4 posters will be made on Tuesday morning, the meeting officially ends at 12:15pm on Tuesday, April 12th.

CONTACTS

American Association of Endocrine Surgeons

5810 W. 140th Terrace

Overland Park, KS 66223

Telephone: (913) 402-7102

Fax: (913) 273-9940

Email: meetings@endocrinesurgery.org

Web: www.endocrinesurgery.org

Nancy D. Perrier, MD

Local Arrangements Chair

University of Texas M. D. Anderson Cancer Center

Telephone: (713) 794-1345

Email: nperrier@mdanderson.org

PROGRAM OVERVIEW

Saturday, April 9, 2011

11:00am

Bus departs for Golf, Hotel Main Entrance

12Noon - 5:00pm

Annual Golf Outing, Hermann Park

12Noon - 5:30pm

NASA Tour, Bus Departs from Hotel Main Entrance

1:30pm

Bus Departs for Tennis, Hotel Main Entrance

2:00pm - 5:00pm

Annual Tennis Tournament, The Downtown Club at the Met

2:00pm - 5:00pm

AAES Council Meeting, Grand Ballroom A

6:05pm

Astros vs Marlins Baseball Game, Minute Maid Park

7:00pm - 9:00pm

Young Endocrine Surgeon's Social, House of Blues

Sunday, April 10, 2011

6:45am

Bus Departs for Fun Run, Hotel Main Entrance

7:30am - 8:30am

Fun Run to Benefit Paul LoGerfo Educational Research Fund, Memorial Park

8:00am - 9:00am

Yoga in the Park, Sarofim Picnic Lawn, on Discovery Green

PROGRAM OVERVIEW CONT.

8:00am - 5:30pm

Registration Open, Registration Desk 431

10:00am - 11:30am

What Do I Do Now? Management of Routine and Complex Thyroid Issues: The Surgical Perspective, Grand Ballroom I

Moderator: Mira Milas, MD

Speakers: Janice L. Pasieka, MD, Ian Hay, MD, Electron Kebebew, MD, Cord Sturgeon, MD, Steven Waguespack, MD, Robert Tuttle, MD

10:00am - 12Noon

Program Director's Meeting, Meeting Room 339A

12Noon - 12:30pm

Opening Session, Grand Ballroom ABDE

Opening Comments

Introduction of New Members

LoGerfo Educational Research Award

12:30pm - 1:00pm

Historical Lecture: "Pheochromocytoma Resection: Now and Then", Grand Ballroom ABDE

Introduction: Nancy D. Perrier, MD

Speaker: Jon van Heerden, MD

1:00pm - 3:00pm

Interesting Cases, Grand Ballroom ABDE

Moderator: Gerard Doherty, MD

2:00pm - 5:00pm

Industry Exhibits Open, Grand Ballroom CF

3:20pm - 4:30pm

Scientific Session I: Papers 1 - 4, Grand Ballroom ABDE

Moderator: Douglas B. Evans, MD

4:30pm - 5:30pm

Invited Lecturer, Major League Baseball - 2011 Economic and Health Related Issues, Grand Ballroom ABDE

Introduction: Douglas B. Evans, MD

Speaker: Allan H. (Bud) Selig, 9th Commissioner of Major League Baseball

7:00pm - 10:00pm

AAES Welcome Reception, The Grove Restaurant on Discovery Green

Monday, April 11, 2011

7:00am - 8:00am

Continental Breakfast, Grand Ballroom CF

7:00am - 3:00pm

Exhibits & Posters Open, Grand Ballroom CF

7:00am - 6:00pm

Registration Open, Registration Desk

7:45am - 9:30am

Scientific Session II: Papers 5 - 10, Grand Ballroom ABDE

Moderator: Gerard Doherty, MD

9:30am - 10:00am

Coffee Break & Poster Viewing, Grand Ballroom CF

10:00am - 11:45am

Scientific Session III: Papers 11 - 16, Grand Ballroom ABDE

Moderator: Lawrence Kim, MD

11:45am - 1:00pm

AAES Luncheon, Grand Ballroom GH

1:00pm - 2:30pm

Scientific Session IV: Papers 17 - 21, Grand Ballroom ABDE

Moderator: Peter Angelos, MD, PhD

2:30pm - 3:00pm

Coffee Break & Poster Viewing, Grand Ballroom CF

3:00pm - 4:30pm

Scientific Session V: Papers 22 - 26, Grand Ballroom ABDE

Moderator: Steven K. Libutti, MD

4:30pm - 5:15pm

Presidential Address: "Papillary Carcinoma of the Thyroid: Balancing Principles of Oncology with Emerging Technology", Grand Ballroom ABDE

Introduction: Gerard Doherty, MD

Speaker: Douglas B. Evans, MD

Medical College of Wisconsin, Milwaukee, WI

PROGRAM OVERVIEW CONT.

5:15pm – 6:00pm

AAES Business Meeting, Grand Ballroom ABDE

7:00pm – 7:30pm

New Members Reception, Meeting Room 340B

7:30pm – 10:00pm

Gala Reception & Banquet “Celebrate Discovery”, Grand Ballroom GH

10:00pm – Midnight

New President’s Reception, Skyline Ballroom

Tuesday, April 12, 2011

7:00am – 8:00am

Continental Breakfast, Grand Ballroom CF

7:00am – 10:00am

Exhibits & Posters Open, Grand Ballroom CF

8:15am – 9:30am

Scientific Session VI: Papers 27 – 30, Grand Ballroom ABDE

Moderator: Nancy D. Perrier, MD

9:30am – 9:55am

Coffee Break & Exhibits, Grand Ballroom CF

9:55am – 11:15am

Scientific Session VII: Papers 31 – 34, Grand Ballroom ABDE

Moderator: Julie Ann Sosa, MD

11:15am – 12Noon

Poster Snap Shot Presentations “Texas 4x4 Poster Competition”,

Grand Ballroom ABDE - 4 Judges, 4 Presenters, 4 Slides, 4 Minutes For Discussion

12Noon – 12:15pm

Meeting Wrap-up and Awards, Grand Ballroom ABDE

SCIENTIFIC PROGRAM

** Denotes Resident/Fellow Research Award Competition Paper*

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11:00am

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Annual Golf Outing, Hermann Park

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SCIENTIFIC PROGRAM CONT.

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What Do I Do Now? Management of Routine and Complex Thyroid Issues: The Surgical Perspective, Grand Ballroom I

Moderator: Mira Milas, MD

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10:00am - 12Noon

Program Director's Meeting, Meeting Room 339A

12Noon - 12:30pm

Opening Session, Grand Ballroom ABDE

12Noon – 12:15pm **Introduction of New Members**, Julie Ann Sosa, MD

12:15pm – 12:20pm **2011 Paul LoGerfo Educational Research Fund Award**,
William B. Inabnet, III, MD

12:20pm – 12:30pm **2010 Paul LoGerfo Award Recipient Presentation**,
Rebecca S. Sippel, MD

12:30pm – 1:00pm

Historical Lecture: "Pheochromocytoma Resection: Now and Then"

Grand Ballroom ABDE

Introduction: Nancy D. Perrier, MD

Speaker: Jon A. van Heerden, MD

1:00pm – 3:00pm

Interesting Cases, Grand Ballroom ABDE

Moderator: Gerard Doherty, MD

2:00pm – 5:00pm

Industry Exhibits Open, Grand Ballroom CF

3:20pm - 4:30pm

Scientific Session I: Papers 1 - 4, Grand Ballroom ABDE

Moderator: Douglas B. Evans, MD

3:20pm - 3:37pm

1. MOLECULAR PATHWAYS ASSOCIATED WITH MORTALITY IN PAPILLARY THYROID CANCER

Naris Nilubol, MD, Chotiya Sukchotrat, MS, Electron Kebebew, MD
National Cancer Institute

SCIENTIFIC PROGRAM CONT.

3:37pm - 3:54pm

***2. NECROPTOSIS IS A NOVEL MECHANISM OF RADIATION-INDUCED CELL DEATH IN ANAPLASTIC THYROID CANCER AND ADRENOCORTICAL CANCER**

Matthew A. Nehs, MD, Chi-Iou Lin, PhD, David E. Kozono, MD PhD, Edward Whang, MD, Nancy L. Cho, MD, Francis D. Moore Jr, MD, Daniel T. Ruan, MD
Brigham and Women's Hospital

3:54pm - 4:11pm

***3. NOVEL THERAPY FOR ANAPLASTIC THYROID CARCINOMA CELLS UTILIZING ONCOLYTIC VACCINIA VIRUS CARRYING THE HUMAN SODIUM IODINE SYMPORTER**

Sepideh Gholami, MD, Dana Haddad, MD, Chun-Hao Chen, MD, Nanhai Chen, PhD, Qian Zhang, MD, PhD, Pat B. Zanzonico, PhD, Aladar A. Szalay PhD, Yuman Fong, MD
Memorial Sloan-Kettering Cancer Center

4:11pm - 4:28pm

***4. A MULTICENTER COHORT STUDY OF TOTAL THYROIDECTOMY ALONE VERSUS TOTAL THYROIDECTOMY AND CENTRAL LYMPH NODE DISSECTION FOR CNO PAPILLARY THYROID CANCER**

Alexandra Popadich MD, Olga Levin BS, Lilah F. Morris MD, James E. Wiseman MD, James C. Lee, MBBS, M. Fazel MD, Stan B. Sidhu PhD, Leigh W. Delbridge MD, Fausto Palazzo MD, Mark S. Sywak MD, Michael Yeh MD
University of Sydney, Hammersmith and Charing Cross Hospitals, UCLA

4:30pm - 5:30pm

Invited Lecturer, Major League Baseball - 2011 Economic and Health Related Issues, Grand Ballroom ABDE

Introduction: Douglas B. Evans, MD

Speaker: Allan H. (Bud) Selig, 9th Commissioner of Major League Baseball

7:00pm - 10:00pm

AAES Welcome Reception, The Grove Restaurant on Discovery Green
Entertainment provided by Tweed Funk

Monday, April 11, 2011

7:00am - 8:00am

Continental Breakfast, Grand Ballroom CF

7:00am - 3:00pm

Exhibits & Posters Open, Grand Ballroom CF

SCIENTIFIC PROGRAM CONT.

7:00am - 6:00pm

Registration Open, Registration Desk

7:45am - 9:30am

Scientific Session II: Papers 5 - 10, Grand Ballroom ABDE

Moderator: Gerard Doherty, MD

7:45am - 8:02am

***5. THE EFFECT OF VITAMIN D LEVELS ON POSTOPERATIVE CALCIUM REQUIREMENTS AND SYMPTOMATIC HYPOCALCEMIA IN PATIENTS UNDERGOING PARATHYROIDECTOMY FOR PRIMARY HYPERPARATHYROIDISM**

Danielle Press, MD, Douglas Politz, MD, Jose Lopez, MD, and James Norman, MD
Norman Parathyroid Center

8:02am - 8:19am

***6. TRANSIENT ELEVATIONS IN INTRA-OPERATIVE PTH LEVELS RELATED TO ANESTHETIC TECHNIQUE**

Joe C. Hong, MD, Lilah F. Morris, MD, Edward Park, MD, Philip H. G. Ituarte, PhD, MPH, Dena Yassin, BS, Michael W. Yeh, MD
David Geffen School of Medicine at UCLA

8:19am - 8:36am

***7. NORMOCALCEMIC HYPERPARATHYROIDISM AFTER SUCCESSFUL PARATHYROIDECTOMY: A LONG-TERM ANALYSIS OF PTH VARIATIONS OVER 10 YEARS**

Melanie Goldfarb, MD, George L. Irvin III, MD, John I. Lew, MD
University of Miami Leonard Miller School of Medicine

8:36am - 8:53am

***8. RISK STRATIFICATION IN INDETERMINATE THYROID FNA BIOPSIES BASED ON MUTATION ANALYSIS**

Filippo Filicori, MD, Xavier M Keutgen, MD, Daniel Buitrago, MD, Hasan A AlDailami, MS, Thomas J Fahey III, MD, Rasa Zarnegar, MD
Weill Cornell Medical College

8:53am - 9:10am

***9. ELEVATED PARATHYROID HORMONE PREDICTS POST-OPERATIVE MORTALITY IN DIALYSIS PATIENTS UNDERGOING VALVE SURGERY**

Jyotirmay Sharma, MD, Huan Yan, BS, Collin J. Weber, MD, Robert A. Guyton, MD, Vinod H. Thourani, MD
Emory University School of Medicine

SCIENTIFIC PROGRAM CONT.

9:10am - 9:27am

***10.** THE PHENOTYPE OF PRIMARY HYPERPARATHYROIDISM WITH NORMAL PARATHYROID HORMONE LEVELS: HOW LOW CAN PTH GO?

Lucy B. Wallace, MD, Rikesh T. Parikh, MD, Louis V. Ross, BS, Peter J. Mazzaglia, MD, Christina Foley, MD, Joyce J. Shin, MD, Jamie C. Mitchell, MD, Eren Berber, MD, Allan E. Siperstein, MD, Mira Milas, MD
Cleveland Clinic

9:30am - 10:00am

Coffee Break & Poster Viewing, Grand Ballroom CF

10:00am - 11:45am

Scientific Session III: Papers 11 - 16, Grand Ballroom ABDE

Moderator: Lawrence Kim, MD

10:00am - 10:17am

***11.** POPULATION-LEVEL PREDICTORS OF PERSISTENT HYPERPARATHYROIDISM

Michael W. Yeh, MD, James E. Wiseman, MD, Stephanie D. Chu, BS, Philip H. G. Ituarte, PhD, In-Lu Amy Liu, BS, Kraig L. Young, BA, Avital Harari, MD, Philip I. Haigh, MD
David Geffen School of Medicine at UCLA

10:17am - 10:34am

***12.** COMPARISON OF 6-18F-FLUORO-L-DOPA, 18F-2-DEOXY-D-GLUCOSE, CT, AND MRI IN PATIENTS WITH PANCREATIC NEUROENDOCRINE TUMORS WITH VON HIPPEL-LINDAU DISEASE

Mio Kitano, MD, Corina Millo, MD, Peter Herscovitch, MD, Krisana Gesuwan, MSN, Aradhana M. Venkatesan, MD, Richard C. Webb, MD, Reza Rahbari, MD, Giao Q. Phan, MD, Marybeth S. Hughes, MD, Naris Nilubol, MD, W. Marston Linehan, MD, Steven K. Libutti, MD, Electron Kebebew, MD
National Institutes of Health

10:34am - 10:51am

13. Z-E SYNDROME ASSOCIATED WITH A HISTORY OF ALCOHOL ABUSE: COINCIDENCE OR CONSEQUENCE?

Stuart D. Wilson MD, Kara M. Doffek, Elizabeth A. Krzywda APNP, Edward J. Quebbeman MD, Kathleen K. Christians MD, Sam G. Pappas MD
Medical College of Wisconsin

10:51am - 11:08am

14. SSTR5 P335L-SPECIFIC MONOCLONAL ANTIBODY DIFFERENTIATES PANCREATIC NEUROENDOCRINE TUMOR (PNT) PATIENTS WITH DIFFERENT SSTR5 GENOTYPES

Guisheng Zhou, PhD, Marie-Claude Gingras, PhD, Shi-He Liu, MD, Robbi Catania, BS, Dean Edwards, David Dawson, MD, PhD, Kurt Christensen, BA, Giovanni Paganelli, Richard Gibbs, PhD, William Fisher, MD, F. Charles Brunicaardi, MD
Baylor College of Medicine

11:08am - 11:25am

***15. ACHIEVING EUGASTRINEMIA IN MEN1 PATIENTS: BOTH DUODENAL INSPECTION AND FORMAL LYMPH NODE DISSECTION ARE IMPORTANT**

Paxton V. Dickson, MD, Thereasa A. Rich, MS, Yan Xing MD, PhD, Nancy D. Perrier, MD, Douglas B. Evans, MD, Jeffrey E. Lee, MD, Elizabeth G. Grubbs, MD
University of Texas, M.D. Anderson Cancer Center

11:25am - 11:42am

***16. TARGETED PARATHYROIDECTOMY WITHOUT SESTAMIBI SCANS: CASE SERIES OF SURGEONS-PERFORMED ULTRASONOGRAPHY AS THE ONLY METHOD OF PREOPERATIVE LOCALIZATION**

Cindy M. Deutmeyer, MD, Denise Carneiro-Pla, MD
Medical University of South Carolina

11:45am - 1:00pm

AAES Luncheon, Grand Ballroom GH

1:00pm - 2:30pm

Scientific Session IV: Papers 17 - 21, Grand Ballroom ABDE

Moderator: Peter Angelos, MD, PhD

1:00pm - 1:17pm

17. THE INCIDENCE OF CENTRAL NECK MICROMETASTATIC DISEASE IN PATIENTS WITH PAPILLARY THYROID CANCER STAGED PREOPERATIVELY AND INTRAOPERATIVELY AS NO

Gilberto V. Teixeira, MD, PhD, Thiago Teixeira, STD, Fernando Gubert, MD, Horacio Chikota, MD, Ralph P. Tufano, MD
CEPON, Florianopolis, Brazil

1:17pm - 1:34pm

***18. PREOPERATIVE BASAL CALCITONIN LEVEL AND NOT EXTENT OF SURGERY CORRELATES WITH POSTOPERATIVE CALCITONIN LEVELS IN PATIENTS UNDERGOING INITIAL SURGICAL MANAGEMENT OF MEDULLARY THYROID CARCINOMA.**

Dana Yip, MD, Maria Hassan, Daniel Ruan, MD, Atul Gawande, MD, MPH, Randall D. Gaz, MD, Francis D. Moore, Jr., MD, Richard A. Hodin, MD, and Antonia E. Stephen, MD, Gilbert Daniels, MD, Gregory W. Randolph, MD, Sareh Parangi, MD, Carrie C. Lubitz, MD
Massachusetts General Hospital

1:34pm - 1:51pm

19. A QUANTITATIVE TOOL TO OBJECTIVELY ASSESS DEGREE OF SARCOPENIA IN PATIENTS WITH HYPERCORTISOLISM

Barbra S. Miller, MD, Kathy M. Ignatoski, PhD, Ceit Lindland, Stephanie Daignault, MS, Paul G. Gauger, MD, Gerard M. Doherty, MD, Stewart C. Wang, MD, PhD
University of Michigan

1:51pm - 2:08pm

***20.** COMPARISON OF CLINICAL AND IMAGING FEATURES IN SDHB+ VERSUS SPORADIC PARANGLIOMAS

Hari M. Trivedi, BS, Aradhana M. Venkatesan, MD, Karen T. Adams, CRNP, Electron Kebebew, MD, FACS, Karel Pacak, MD, PhD, DSc, Marybeth S. Hughes, MD, FACS
National Institutes of Health

2:08pm - 2:25pm

21. IS THE GENETIC SCREENING INDICATED IN APPARENTLY SPORADIC PHEOCHROMOCYTOMAS?

Maurizio Iacobone (1), Francesca Schiavi (2), Marzia Bottussi (1), Elisa Taschin (2), Giuseppe Opocher (2), Gennaro Favia
(1) Endocrine Surgery Dept, University of Padua, Italy
(2) Cancer Family Clinic, Veneto Institute of Oncology, Padua, Italy

2:30pm - 3:00pm

Coffee Break & Poster Viewing, Grand Ballroom CF

3:00pm - 4:30pm

Scientific Session V: Papers 22 - 26, Grand Ballroom ABDE

Moderator: Steven K. Libutti, MD

3:00pm - 3:17pm

22. CARDIAC DYSFUNCTION AND CATECHOLAMINE CARDIO-MYOPATHY IN PHEOCHROMOCYTOMA PATIENTS AND THEIR REVERSAL FOLLOWING SURGICAL CURE: RESULTS OF A PROSPECTIVE CASE-CONTROL STUDY

Dhalapathy Sadacharan, MS, Aditya Kapoor, MD, DM, Preeti Dabadghao, MD, DM, Anjali Mishra, MS, Amit Agarwal, MS, Ashok K. Verma, MS, FACS, Saroj K. Mishra, MS, FACS, Gaurav Agarwal, MD, FACS
Sanjay Gandhi Postgraduate Institute of Medical Sciences

3:17pm - 3:34pm

23. PROSPECTIVE APPLICATION OF OUR NOVEL PROGNOSTIC INDEX IN THE TREATMENT OF ANAPLASTIC THYROID CARCINOMA

Yorihisa Orita, MD, Iwao Sugitani, MD, Takeshi Amemiya, MD, Yoshihide Fujimoto, MD
Okayama University Graduate School of Medicine, Dentistry and Pharmaceutica

SCIENTIFIC PROGRAM CONT.

3:34pm - 3:51pm

***24. MOTOR AND SENSORY BRANCHING OF THE RECURRENT LARYNGEAL NERVE**

Samy A. Abdelghani, BSE, Ralph P. Tufano, MD, Eid Amer, MD, Saud Alrasheedi, MD, Kevin Kniery, BS, Emad Kandil, MD

Division of Endocrine and Oncologic Surgery, Department of Surgery, Tulane

3:51pm - 4:08pm

***25. PREDICTIVE FACTORS OF MALIGNANCY IN PEDIATRIC THYROID NODULES**

Rashmi Roy, MD, Guennadi Kouniavsky, MD, Eric Schneider, PhD, John A. Allendorf, MD, John A. Chabot, MD, Paul Logerfo, MD, Alan P.B. Dackiw, MD, Martha A. Zeiger MD, James A. Lee, MD

NYPH-Columbia University

4:08pm - 4:25pm

26. THE IMPACT OF FLUS ON THE RATE OF MALIGNANCY IN THYROID FNA: EVALUATION OF THE BETHESDA SYSTEM FOR REPORTING THYROID CYTOPATHOLOGY

James T. Broome, MD, Carmen C. Solorzano, MD

Vanderbilt University

4:30pm - 5:15pm

Presidential Address: "Papillary Carcinoma of the Thyroid: Balancing Principles of Oncology with Emerging Technology", Grand Ballroom ABDE

Introduction: Gerard Doherty, MD

Speaker: Douglas B. Evans, MD

Medical College of Wisconsin, Milwaukee, WI

5:15pm - 6:00pm

AAES Business Meeting, Grand Ballroom ABDE

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New Members Reception, Meeting Room 340B

7:30pm - 10:00pm

Gala Reception & Banquet "Celebrate Discovery", Grand Ballroom GH

10:00pm - Midnight

New President's Reception, Skyline Ballroom

Tuesday, April 12, 2011

7:00am - 8:00am

Continental Breakfast, Grand Ballroom CF

7:00am - 10:00am

Exhibits & Posters Open, Grand Ballroom CF

8:15am - 9:30am

Scientific Session VI: Papers 27-30, Grand Ballroom ABDE

Moderator: Nancy D. Perrier, MD

8:15am - 8:32am

27. THE IMPACT OF MICROSCOPIC EXTRA THYROID EXTENSION ON OUTCOME IN PATIENTS WITH CLINICAL T1 AND T2 WELL DIFFERENTIATED THYROID CANCER

Iain J. Nixon, MD, Ian Ganly, MD, PhD, Snehal Patel, MD, Frank L. Palmer, BA, Monica M. Whitcher, BA, Robert M. Tuttle, MD, Ashok R. Shaha, MD, Jatin P. Shah, MD
Department of Head and Neck Surgery, Memorial Sloan-Kettering Cancer Center

8:32am - 8:49am

28. EFFECT OF POSTOPERATIVE THYROTROPIN SUPPRESSION THERAPY ON BONE MINERAL DENSITY IN PATIENTS WITH PAPILLARY THYROID CARCINOMA: PROSPECTIVE CONTROL STUDY

Iwao Sugitani, MD, Yoshihide Fujimoto, MD
Cancer Institute Hospital

8:49am - 9:06am

29. ACTIVATION OF MTOR SIGNALING IN MEDULLARY AND AGGRESSIVE PAPILLARY THYROID CARCINOMAS

Maria A. Kouvaraki, MD, Chyssa Liakou, M.D., Adriani Paraschi, M.Sc., Kostantinos Dimas, PhD, Sofia Tseleni-Balafouta, MD, George Z. Rassidakis, M.D., Dimitrios Moraitis, MD
National and Kapodistrian University of Athens and Academy of Athens Biomed

9:06am - 9:23am

30. THE SURGICAL COMPLETENESS OF BILATERAL AXILLO-BREAST APPROACH ROBOTIC THYROIDECTOMY: COMPARISON WITH OPEN THYROIDECTOMY BY PROPENSITY SCORE MATCHING ANALYSIS

Kyu Eun Lee, MD, PhD, Do Hoon Koo, MD, Hyung Jun Im, MD, June Young Choi, MD, Kwan Yoon, MD, Hyuk Jae Choi, MD, Jin Chul Paeng, MD, PhD, June-Key Chung, MD, PhD, Seung Keun Oh, MD, PhD, Yeo-Kyu Youn, MD, PhD
Seoul National University Hospital

SCIENTIFIC PROGRAM CONT.

9:30am – 9:55am

Coffee Break & Exhibits, Grand Ballroom CF

9:55am – 11:15am

Scientific Session VII: Papers 31 – 34, Grand Ballroom ABDE

Moderator: Julie Ann Sosa, MD

9:55am – 10:12am

31. IS VENOUS THROMBOEMBOLISM A REAL RISK FOR THYROID CANCER PATIENTS UNDERGOING SURGERY?

Rachel A. Hadler, BA, Caroline E. Reinke, MD, Giorgos C. Karakousis, MD, Douglas L. Fraker, MD, and Rachel R. Kelz, MD

Hospital of the University of Pennsylvania

10:12am – 10:29am

32. WOULD SCAN, BUT WHICH SCAN? A COST-UTILITY ANALYSIS TO OPTIMIZE PREOPERATIVE IMAGING FOR PRIMARY HYPERPARATHYROIDISM

Tracy S. Wang, MD, Kevin Cheung, MD, Sanziana A. Roman, MD, Julie A. Sosa, MD
Medical College of Wisconsin, McMaster University, Yale University

10:29am – 10:46am

33. THYROID-SPECIFIC KNOCKOUT OF THE TUMOR SUPPRESSOR MITOGEN-INDUCIBLE GENE 6 ACTIVATES EGFR SIGNALING PATHWAYS AND SUPPRESSES NF- κ B ACTIVITY

Chi-lou Lin, PhD, Justine A. Barletta, MD, Matthew A. Nehs, MD, Zachary Morris, PhD, David B. Donner, PhD, Edward E. Whang, MD, Jaewook W. Jeong, PhD, Shioko Kimura, PhD, Francis D. Moore Jr., MD, Daniel T. Ruan, MD

Brigham and Women's Hospital

10:46am – 11:03am

34. SIMULATION TRAINING FOR THYROID LOBECTOMY IN CONJUNCTION WITH OPERATIVE TRAINING IMPROVES RESIDENT PERFORMANCE

Melissa A. Kath, MD, Nancy M. Carroll, MD, John A. Boswick, BA, Jack A. Sava, MD, Erin A. Felger, MD

Washington Hospital Center

11:03am – 11:15am

Discussion & Wrap-up

11:15am – 12Noon

Poster Snap Shot Presentations "Texas 4x4 Poster Competition"

Grand Ballroom ABDE - 4 Judges, 4 Presenters, 4 Slides, 4 Minutes For Discussion

Moderator: Janice L. Pasieka, MD

MEETING WRAP-UP AND AWARDS

12Noon - 12:15pm

Meeting Wrap-up and Awards, Grand Ballroom ABDE

Douglas B. Evans, MD, Ashok R. Shaha, MD, Nancy D. Perrier, MD



ABSTRACTS

** Denotes Resident/Fellow Research Award Competition Paper*

ABSTRACTS

1. MOLECULAR PATHWAYS ASSOCIATED WITH MORTALITY IN PAPILLARY THYROID CANCER

Naris Nilubol, MD, Chotiya Sukchotrat, MS, Electron Kebebew, MD
National Cancer Institute

Background: Aggressive tumor behavior is observed in a small subset of patients with papillary thyroid cancer (PTC). A better understanding of the molecular mechanisms involved in adverse outcomes is needed to effectively manage these patients. Our objectives were to identify pathways associated with unfavorable features and outcome in patients with PTC.

Methods: We performed genome-wide expression (GWE) analysis of 64 PTC human tissue samples. Clinical, pathologic and microarray data were analyzed to identify differentially expressed genes associated with an unfavorable outcome. Gene Set Enrichment Analysis (GSEA) was used to determine which molecular pathways are associated with mortality.

Results: GWE analysis identified 43, 115 and 40 genes that were significantly associated with gender, tumor differentiation and mortality, respectively, using a false discovery rate (FDR) of 5%. For mortality, GSEA revealed 9 enriched pathways involved in transfer RNA synthesis, porphyrin and chlorophyll metabolism, mitochondria and oxidative phosphorylation, and fatty acid synthesis (family wise error rate 25%). Leading edge gene analysis showed 40 genes significantly involved in the enriched pathways. Cluster analysis using 100 genes showed complete separation of patients by mortality.

Conclusions: To our knowledge, this is the first GWE analysis of PTC and adverse outcome. Nine molecular pathways were significantly associated with PTC mortality. A 100-gene signature completely separates patients with PTC with and without mortality.

ABSTRACTS CONT.

NOTES

*2. NECROPTOSIS IS A NOVEL MECHANISM OF RADIATION-INDUCED CELL DEATH IN ANAPLASTIC THYROID CANCER AND ADRENOCORTICAL CANCER

Matthew A. Nehs, MD, Chi-lou Lin, PhD, David E. Kozono, MD PhD, Edward Whang, MD, Nancy L. Cho, MD, Francis D. Moore Jr, MD, and Daniel T. Ruan, MD
Brigham and Women's Hospital

Background: Necroptosis is a recently described mechanism of programmed cellular death that is distinct from apoptosis and passive necrosis. Since radiotherapy is an important treatment for advanced thyroid and adrenal cancers, we sought to characterize the pattern of cellular death in response to external beam radiation. Here we hypothesize that necroptosis plays an important role in radiation-induced cell death in endocrine cancers.

Methods: Thyroid (8505c and TPC-1) and adrenocortical (SW13 and H295R) carcinoma cell lines were exposed to increasing doses of radiation (0,2,4,6 Gy) in the presence of the necroptosis inhibitor, Nec-1, the apoptosis inhibitor, zVAD, both compounds, or neither. H295R cells, which are deficient in RIP-1 kinase and therefore unable to undergo necroptosis, were used as a control. Survival curves were generated at increasing doses of radiation.

Results: Immunoblotting demonstrated robust expression of RIP-1 kinase in 8505c, TPC-1, and SW13 cells, but no expression in H295R cells. Nec-1 and zVAD increased cellular survival with increasing doses of radiotherapy for 8505c, TPC-1, and SW13. There was an additive effect on cellular survival when both inhibitors were used together. At 6 Gy, 8505c, TPC-1, and SW13 cell survival was significantly increased compared to controls by 40%, 33% and 31% with Nec-1 treatment, by 53%, 47% and 44% with zVAD treatment, and by 80%, 70% and 65% with both compounds, respectively ($p < 0.05$). H295R, which lacks the target of Nec-1, showed no change in survival with Nec-1 treatment. Additionally, the radiobiological parameter, D_q , which reflects sub-lethal radiation damage repair capacity, was significantly increased in 8505c, TPC-1, and SW13 cells when both Nec-1 and zVAD were used in combination to inhibit necroptosis and apoptosis together in the setting of a 6 Gy radiation treatment ($p < 0.05$).

Conclusions: Necroptosis contributes to radiation-induced cell death in anaplastic thyroid and adrenocortical carcinomas. This alternative programmed cell death pathway may explain the apoptosis-independent tumor regression observed with some targeted therapies for advanced endocrine cancers. Future studies should investigate necroptosis activation as an adjuvant to external beam radiation therapy.

NOTES

*3. NOVEL THERAPY FOR ANAPLASTIC THYROID CARCINOMA CELLS UTILIZING ONCOLYTIC VACCINIA VIRUS CARRYING THE HUMAN SODIUM IODINE SYMPORTER

Sepideh Gholami, MD, Dana Haddad, MD, Chun-Hao Chen, MD, Nanhai Chen, PhD, Qian Zhang, MD, PhD, Pat B. Zanzonico, PhD, Aladar A. Szalay PhD, Yuman Fong, MD
Memorial Sloan-Kettering Cancer Center

Background: Oncolytic viral therapy has shown promise in preclinical and clinical trials against a wide range of aggressive malignancies. Noninvasive deep tissue imaging tools are needed to assess therapeutic efficacy. Anaplastic thyroid carcinoma (ATC) is a fatal disease with resistance to radioiodine therapy due to loss of the intrinsic human sodium iodine symporter (hNIS). The objective of this study was to determine whether vaccinia virus carrying hNIS can be utilized to 1) kill ATC cells and 2) induce reexpression of hNIS in cells, thus facilitating deep tissue imaging via enhanced radioiodide uptake.

Methods: A replication-competent vaccinia virus (GLV-1h153) was derived from parental virus and modified to carry hNIS via homologous recombination. GLV-1h153 was tested against ATC cell lines 8505c, FRO, and 8305c for killing and replication via both cytotoxicity and viral plaque assays. Cellular uptake of radioiodine (131I) was determined using radioactivity uptake assays. Athymic nude mice with human ATC tumor xenografts (8505c) were treated with single intratumoral injection of GLV-1h153. Tumor deep tissue imaging was assessed via Tc-99m pertechnetate administration and gamma imaging.

Results: GLV-1h153 infected, replicated in, and killed all three ATC cell lines. GFP expression confirmed viral infection by 24 hours and was proportionate to both viral dosages and cytotoxicity results. At a multiplicity of infection (MOI) of 1.0, GLV-1h153 achieved a cell kill of nearly 100% in 8305c and FRO by day 5 and reached 70% cell kill even in the least sensitive cell line 8505c. GLV-1h153-infected ATC cells (8505c) demonstrated enhanced hNIS-specific radio uptake, with over 13-fold increase compared with uninfected control at 24 hours after infection at an MOI of 1.0. In vivo, GLV-1h153 facilitated imaging of hNIS expression in 8505c tumors via gamma scanning using Tc-99m pertechnetate.

Conclusions: GLV-1h153 is an effective oncolytic agent against ATC. Our results demonstrate hNIS-specific enhanced radiouptake by GLV-1h153-infected ATC cells, which facilitates deep tissue imaging. These results support GLV-1h153 as a promising new candidate for treating ATC. GLV-1h153 may also enhance ATC susceptibility to radioiodine therapy by inducing reexpression of hNIS and thus providing a novel treatment modality.

NOTES

*4. A MULTICENTER COHORT STUDY OF TOTAL THYROIDECTOMY ALONE VERSUS TOTAL THYROIDECTOMY AND CENTRAL LYMPH NODE DISSECTION FOR CNO PAPILLARY THYROID CANCER

Alexandra Popadich MD, Olga Levin BS, Lilah F. Morris MD, James E. Wiseman MD, James C. Lee, MBBS, M. Fazel MD, Stan B. Sidhu PhD, Leigh W. Delbridge MD, Fausto Palazzo MD, Mark S. Sywak MD, Michael Yeh MD
University of Sydney, Hammersmith and Charing Cross Hospitals, UCLA

Background: The role of central compartment lymph node dissection (CND) for papillary thyroid carcinoma (PTC) in patients without preoperative evidence of lymphadenopathy remains highly controversial. The aim of this study is to evaluate outcomes with and without central compartment node dissection (CND) with particular reference to stimulated thyroglobulin (sTG) levels and need for re-operative surgery.

Methods: A retrospective multicenter cohort study was undertaken. Data were pooled from three international Endocrine Surgery units. The study population included patients with PTC >1cm without preoperative evidence of lymph node disease (cN0). Outcomes for patients undergoing routine CND were compared with a historical control group treated with total thyroidectomy alone (TTx). Post-operative ablative radioiodine was administered in all cases. sTG was measured annually after initial surgery.

Results: 513 patients had surgery for cN0 PTC in the period 2000-2010. Group A comprised 294 cases having TTx alone. Group B had 219 cases undergoing TTx and CND. Mean tumor size was equivalent in the two groups (group A 24mm, group B 22mm, $p=0.2$). Mean follow up was 40 months. The cumulative dose of radioiodine was similar between groups (group A: 6.8GBq and group B: 6.9GBq, $p=0.8$). Overall sTG levels were significantly lower in Group B patients at final follow up (group A 9.0 versus group B 6.0ng/ml $p=0.01$). Sub group analysis showed no difference in sTG levels between groups for T1 tumors (group A 0.9 versus group B 0.7ng/ml, $p=0.5$). For T2, T3 and T4 tumors group B patients had lower sTG levels at final follow up (group A 17 versus group B 11ng/ml, $p=0.05$). The overall rate of local recurrence requiring re-operative surgery was equivalent between groups, 8% for Group A and 9% for Group B. The rate of local disease recurrence requiring re-operative surgery in the central compartment was greater for Group A (5.4%) compared to Group B (2.6%), $p=0.02$.

Conclusions: CND results in lower stimulated TG levels in patients with cN0 PTC, particularly those with T2-T4 tumors. Patients having routine CND for PTC have a lower rate of re-operation in the central compartment

NOTES

*5. THE EFFECT OF VITAMIN D LEVELS ON POSTOPERATIVE CALCIUM REQUIREMENTS AND SYMPTOMATIC HYPOCALCEMIA IN PATIENTS UNDERGOING PARATHYROIDECTOMY FOR PRIMARY HYPERPARATHYROIDISM

Danielle Press, MD, Douglas Politz, MD, Jose Lopez, MD, and James Norman, MD
Norman Parathyroid Center

Background: Low vitamin-D-25 is common in patients with primary hyperparathyroidism (PHPT). It has been suggested that low levels should be corrected prior to parathyroidectomy to avoid postoperative hypocalcemia.

Methods: A prospective, non-randomized study divided 1485 consecutive patients undergoing parathyroidectomy for PHPT into three groups according to preoperative vitamin-D-25 levels: Low (< 20 ng/ml, n=500), Normal (>30 ng/ml, n=500) and Supplemented (<20 supplemented to >40 ng/ml, n=485). Patients with levels between 20 and 30 ng/ml were not studied. Patients were placed on an identical postoperative oral calcium protocol and hypocalcemia symptoms were reported by phone or email; total calcium requirements for 2 weeks were calculated.

Results: The mean vitamin-D-25 level for all patients was 22.4+9 ng/ml. The mean for each group was: Low = 14.2+4 ng/ml; Normal = 36.3+9 ng/ml; and Supplemented = 14.9+5 ng/ml which was supplemented to 54.3+12 ng/ml. Average preoperative calcium, ionized calcium, PTH, and bone density was identical for all groups (11.4, 6.1, 119, -2.8, respectively). Two-week oral calcium requirements were identical for all three groups: 18.4g, 18.7g, and 18.6g, (p=0.93) as was the incidence of hypocalcemia symptoms: 8.1%, 7.9%, and 7.8% (p=0.80) (Low/Normal/Supplemented, respectively) No patient required IV calcium.

Conclusions: Although most patients with PHPT have low vitamin-D-25 levels, the incidence of hypocalcemic symptoms and the total postop calcium requirement is identical for patients with low, normal, or high vitamin-D. Supplementation of patients from very low to high levels has no clinical benefit.

NOTES

*6. TRANSIENT ELEVATIONS IN INTRA-OPERATIVE PTH LEVELS RELATED TO ANESTHETIC TECHNIQUE

Joe C. Hong, MD, Lilah F. Morris, MD, Edward Park, MD, Philip H. G. Ituarte, PhD, MPH, Dena Yassin, BS, Michael W. Yeh, MD
David Geffen School of Medicine at UCLA

Background: Parathyroid hormone secretion is stimulated by catecholamines and may thus be influenced by anesthetic technique. We examined the effect of different anesthetic techniques on post-induction intra-operative PTH (IOPTH) levels in patients undergoing initial surgery for sporadic primary hyperparathyroidism.

Methods: 102 patients were prospectively assigned to receive local anesthesia with monitored anesthetic care (MAC, n=36), general anesthesia with laryngeal mask airway (LMA, n=34), or general endotracheal anesthesia (GETA, n=32). IOPTH levels were drawn prior to induction and 3 minutes post-induction. Rapid sequential sampling was performed in a follow-up cohort to assess the duration of the anesthetic effect on IOPTH levels.

Results: All anesthetic techniques increased IOPTH levels from baseline at 3 minutes post-induction, though the average magnitude of the increase varied among techniques (MAC 30%, LMA 51%, GETA 66%, $p < 0.0001$). The proportion of patients exhibiting a >50% post-induction increase in IOPTH was 15%, 40%, and 59% for MAC, LMA, and GETA, respectively ($p < 0.005$). Sequential sampling revealed a peak anesthetic effect at 6 minutes post-induction, with the majority of patients recovering toward baseline pre-induction IOPTH levels by 9 minutes. Incorporation of the 3-minute post-induction IOPTH level into surgical decision-making according to the Miami criterion would have led to failed surgery in 3 of 13 patients (23%) with multiple gland disease receiving general anesthesia.

Conclusions: Significant, time-sensitive alterations in IOPTH levels occur after anesthetic induction. Pre-incision samples should be drawn more than 6 minutes after induction to avoid potentially inappropriate incorporation of anesthetic-related elevations in IOPTH into values used for surgical decision-making.

NOTES

*7. NORMOCALCEMIC HYPERPARATHYROIDISM AFTER SUCCESSFUL PARATHYROIDECTOMY: A LONG-TERM ANALYSIS OF PTH VARIATIONS OVER 10 YEARS

Melanie Goldfarb, MD, George L. Irvin III, MD, John I. Lew, MD
University of Miami Leonard Miller School of Medicine

Background: Although normocalcemic hyperparathyroidism (NCHPT) after successful parathyroidectomy (PTX) for sporadic primary hyperparathyroidism is well known, its long-term significance remains unclear. This study examines postoperative parathormone (PTH) variations and the recurrence rate of patients with NCHPT over a 10 year period.

Methods: Of 239 consecutive patients who underwent focused PTX with intraoperative parathormone monitoring from 1993 to 2000, 95 patients were followed for > 10 years. NCHPT was defined as normal serum calcium and elevated PTH levels (lab ranges varied by year & assay) > 6 months after PTX. Recurrence was defined as elevated serum calcium and PTH levels > 6 months after successful PTX. Risk factors for NCHPT, outcomes and patterns of postoperative PTH levels over 10 years were analyzed.

Results: Of 95 patients followed > 10 years, 42 had postoperative NCHPT whereas the remaining 53 patients had normal postoperative calcium and PTH levels for the entire period. When comparing both groups, univariate analysis showed male gender ($p=.041$) and higher preoperative PTH level ($p=.048$) as risk factors for NCHPT. Only male gender ($p=.008$) was significant by multivariate analysis. Overall recurrence rate was 6.3%; NCHPT did not predict recurrence in these patients. Four patterns of PTH variation were identified in patients with NCHPT. Group 1: 14 patients had 1 or 2 consecutive PTH elevations; none recurred. All but 3 had an explanation for NCHPT (low vitamin D levels, renal insufficiency ($Cr>1.5$) or physiologic variation). Group 2: 12 patients had multiple fluctuations in PTH levels; 2 recurred. All but one patient had physiologic variations. Group 3: 8 patients with mostly elevated PTH levels; 2 recurred. Of the rest, 3 showed physiologic variation, 1 had low vitamin D levels. Group 4: 8 patients with previously normal PTH levels with recent PTH elevation; 1 recurred. Of the rest, 2 had renal insufficiency, 2 had low vitamin D levels, and all showed physiologic variation.

Conclusions: NCHPT can occur after successful PTX for sporadic primary hyperparathyroidism, but does not itself predict recurrence. Postoperative PTH levels may vary over time, and factors such as physiologic variation, renal and vitamin D insufficiency may lead to secondary PTH elevation. No postoperative patients with 1 or 2 consecutive PTH elevations over 10 years developed recurrent disease. Nevertheless, patients with postoperative NCHPT should be monitored.

NOTES

*8. RISK STRATIFICATION IN INDETERMINATE THYROID FNA BIOPSIES BASED ON MUTATION ANALYSIS

Filippo Filicori, MD, Xavier M Keutgen, MD, Daniel Buitrago, MD, Hasan A AlDailami, MS, Thomas J Fahey III, MD, Rasa Zarnegar, MD
Weill Cornell Medical College

Background: Mutation analysis is a potential powerful adjunct tool to enhance the diagnostic accuracy of thyroid FNA biopsies. The risk of malignancy in indeterminate follicular neoplasms is 42% when atypia is identified and 17% when it is not. To refine recommendations based on FNA clinicians may rely on a negative mutation panel to exclude malignancy. We aimed to determine the effect of negative mutation analysis on the risk of malignancy in indeterminate lesions.

Methods: A literature review was performed to establish the mutation rate of the most commonly reported mutated genes in thyroid cancer: BRAF, RET, RAS, and PAX8/PPAR-gamma. Rates were applied to 466 patients with indeterminate FNAs at a tertiary referral center that underwent either a hemi-thyroidectomy or total thyroidectomy for definitive diagnosis. Statistical analysis was used to determine the risk of malignancy based on atypia status in patients with negative mutation analysis.

Results: Nine studies, which assessed the mutation rate in indeterminate FNAs were included. The mutation panel has a sensitivity of 40% and a specificity of 98% for cancer. There was a PPV of 93% and a NPV of 71%. We applied these rates to the 139/466 (30%) malignant lesions in our indeterminate group, 99 (71%) of which contained cytological atypia on FNA. Final pathology revealed 47% PTC, 33% FVPTC, 13% FTC and 7% other cancers. Overall, mutation analysis failed to identify 65% of thyroid cancers with indeterminate FNA. When atypia was present mutation analysis failed to identify 62% of thyroid cancers versus 72% of thyroid cancers in nodules without atypia. After excluding cancers identified by mutations, the overall risk of malignancy in the remaining indeterminate nodules decreased from 30% to 23%. In atypical lesions, the rate of malignancy declined from 42% to 31%, and in lesions without atypia it dropped from 17% to 13%.

Conclusions: Patients with a positive mutation in an indeterminate nodule should be considered for total thyroidectomy. Indeterminate follicular neoplasms with a negative mutation analysis continue to warrant a hemi-thyroidectomy for definitive diagnosis since the risk of malignancy remains significant.

NOTES

*9. ELEVATED PARATHYROID HORMONE PREDICTS POST-OPERATIVE MORTALITY IN DIALYSIS PATIENTS UNDERGOING VALVE SURGERY

Jyotirmay Sharma, MD, Huan Yan, BS, Collin J. Weber, MD, Robert A. Guyton, MD, Vinod H. Thourani, MD
Emory University School of Medicine

Purpose: Valve surgery in patients with preoperative dialysis is associated with a higher morbidity and mortality. Although serum parathyroid hormone (PTH) levels above 300 pg/mL are associated with increased mortality risk in dialysis patients; this correlation has not been investigated in patients undergoing cardiac valve surgery. The purpose of this study was to evaluate the use of perioperative PTH levels as a predictor of short- and mid-term mortality in dialysis patients undergoing valve surgery.

Methods: A retrospective analysis of 116 dialysis patients undergoing valve surgery with perioperative PTH levels between 1996 and 2007 at an US academic center was performed. Categorization of the patients was done 6 times using a single threshold PTH value starting at 100 pg/mL and proceeding by 100 pg/mL intervals until 600 pg/mL. Cox regression analysis was used to compare short-term mortality between patients with PTH < or >= 300 pg/mL. The analysis was adjusted for age, race, history of angina or myocardial infarction (MI), body mass index, antihypertensive use, and pre-operative calcium, phosphate, and hemoglobin. The patients were followed from the date of valve surgery until death or loss to follow-up.

Results: Patients with PTH >= 300 pg/mL (n=74) had a higher risk for post-operative mortality than those in the PTH < 300 pg/mL group (n=42) (hazard ratio (HR) 2.80; 95% CI 1.81-6.00). In the categorization modeling PTH of 300 pg/mL was a threshold for increased mortality. The regression model revealed that the median survival was improved in the PTH < 300 pg/mL group when compared to the PTH >= 300 pg/mL group (49.3 vs. 9.1 4 13.3 months, respectively). Other independent predictors that significantly decreased survival included serum phosphate (HR 1.46; 95% CI 1.03-2.05), history of angina (HR 2.04; 95% CI 1.15-3.62), history of MI (HR 1.89; 95% CI 1.10-3.25), and low hemoglobin (0.85; 95% CI 0.73-0.99).

Conclusions: Elevated perioperative PTH level >300 pg/mL is predictive of increased post-operative mortality after valve surgery in dialysis patients. Elevated PTH may serve as marker of poor outcome in this patient population. Hyperparathyroidism should be further investigated as a possible modifiable risk factor for post-operative mortality in this population.

NOTES

*10. THE PHENOTYPE OF PRIMARY HYPERPARATHYROIDISM WITH NORMAL PARATHYROID HORMONE LEVELS: HOW LOW CAN PTH GO?

Lucy B. Wallace, MD, Rikesh T. Parikh, MD, Louis V. Ross, BS, Peter J. Mazzaglia, MD, Christina Foley, MD, Joyce J. Shin, MD, Jamie C. Mitchell, MD, Eren Berber, MD, Allan E. Siperstein, MD and Mira Milas, MD
Cleveland Clinic

Background: While normocalcemic hyperparathyroidism is a well recognized phenotype of primary hyperparathyroidism (PHP), less is known about patients with high calcium but normal intact parathyroid hormone (iPTH). We aimed to describe this entity and designated it normohormonal primary hyperparathyroidism (NHPHP).

Methods: From a prospectively maintained database of patients undergoing bilateral parathyroid exploration for PHP, we identified and compared those with preoperative iPTH levels below (NHPHP) and above (typical PHP) normal reference peak (60 pg/ml).

Results: NHPHP occurred in 46 of 843 patients (5.5%) undergoing initial parathyroidectomy for PHP in the past 5 years. All NHPHP patients had hypercalcemia (11.1 mg/dl). Regarding preoperative iPTH, 7 patients (15%) had all values <40 pg/ml, 19 patients (41%) had all values <60 pg/ml; 20 patients (44%) had occasional values >60 pg/ml. Demographic and disease characteristics were similar in NHPHP and typical PHP. Unlike patients with elevated iPTH, nearly all NHPHP patients had additional routine laboratory testing and 9% had unconventional testing (selective venous sampling, SPEP, PTHrp, cancer screening) to confirm PHP diagnosis or exclude other etiologies, delaying surgery by an average of 6.5 months. No interfering antibodies were detected in the serum of 2 NHPHP patients with iPTH 5 and 15 pg/ml. Imaging correctly localized NHPHP parathyroid disease in 76%. At surgery, 74% of NHPHP patients had single adenomas. Among NHPHP patients, intraoperative iPTH was used in 96%. Post-mobilization, using the same assay used preoperatively, 82% showed an increase above normal (mean 279 pg/ml). In 4 patients, post-mobilization iPTH levels drawn simultaneously from multiple central and peripheral veins were similarly elevated. One patient had persistent and 1 patient developed recurrent PHP. Post-surgical iPTH levels were more suppressed among NHPHP patients: 21 pg/ml compared to 41 pg/ml for patients with preoperative iPTH 60-100 pg/ml, 56 pg/ml for patients with preoperative iPTH 100-200 pg/ml ($p < 0.0001$).

Conclusions: Lower PTH set points may exist in some patients with otherwise typical PHP features. Although high normal iPTH is inappropriate for hypercalcemia and should suggest PHP, this disorder may occur with iPTH levels as low as 5 pg/ml. Awareness of the unusual phenotype of NHPHP may facilitate earlier diagnosis and surgery.

NOTES

*11. POPULATION-LEVEL PREDICTORS OF PERSISTENT HYPERPARATHYROIDISM

Michael W. Yeh, MD, James E. Wiseman, MD, Stephanie D. Chu, BS, Philip H. G. Ituarte, PhD, In-Lu Amy Liu, BS, Kraig L. Young, BA, Avital Harari, MD, Philip I. Haigh, MD
David Geffen School of Medicine at UCLA

Background: Systematic study of outcomes associated with initial surgery for primary hyperparathyroidism (PHPT) has been limited by institutional, referral, and self-reporting biases. To avoid these biases, we evaluated parathyroidectomy outcomes within a vertically-integrated health care system encompassing 3.25 million annual enrollees.

Methods: All patients who underwent parathyroidectomy from 1995-2008 for biochemically-confirmed PHPT were studied. Patients with a serum calcium level exceeding 10.5 mg/dL before or after six months post-operatively were designated as having persistent or recurrent disease, respectively. Multivariate logistic regression was applied to assess the effect of demographic, biochemical, imaging, and hospital volume related variables on surgical outcome.

Results: 964 initial operations for PHPT were performed at 14 hospitals. Follow up calcium levels were available in 892 subjects (93%). The overall success rate of initial surgery was 92%, and 6% of patients developed recurrent disease. Age \geq 70 (OR 2.02, $p < 0.05$) and pre-operative calcium \geq 11.5 mg/dL (OR 1.66, $p < 0.05$) were predictive of persistent disease. High volume hospital (>100 cases) predicted against persistent disease (OR 0.35, $p < 0.05$) and was associated with an initial success rate of 96%. Factors that did not influence the likelihood of persistent disease were gender, comorbidity score, PTH level, nephrolithiasis, and sestamibi scan result. Of the patients with persistent ($n=76$) and recurrent ($n=52$) disease, 11 (9%) underwent re-operation.

Conclusions: Success rates of surgery for PHPT in an integrated healthcare setting are influenced by patient age, pre-operative calcium level, and hospital volume. Surgical outcomes may be optimized by designating high volume centers within such a system.

NOTES

*12. COMPARISON OF 6-18F-FLUORO-L-DOPA, 18F-2-DEOXY-D-GLUCOSE, CT, AND MRI IN PATIENTS WITH PANCREATIC NEUROENDOCRINE TUMORS WITH VON HIPPEL-LINDAU DISEASE

Mio Kitano, MD, Corina Millo, MD, Peter Herscovitch, MD, Krisana Gesuwan, MSN, Aradhana M. Venkatesan, MD, Richard C. Webb, MD, Reza Rahbari, MD, Giao Q. Phan, MD, Marybeth S. Hughes, MD, Naris Nilubol, MD, W. Marston Linehan, MD, Steven K. Libutti, MD, Electron Kebebew, MD
National Institutes of Health

Background: The utility of 18F-DOPA and 18F-FDG in the evaluation of pancreatic neuroendocrine tumors (PNETs) is not known. Our aim was to determine the clinical utility of 18F-DOPA and 18F-FDG in patients with PNETs in the setting of Von-Hippel-Lindau disease (VHL).

Methods: 61 patients with VHL and a pancreatic lesion(s) were prospectively evaluated with 18F-DOPA and 18F-FDG in conjunction with MRI and CT.

Results: 111, 52, 52, and 12 PNETs were identified by CT, MRI, 18F-FDG, and 18F-DOPA, respectively. MRI identified 3 PNETs that were not seen on CT and 2 patients had lesions in the liver not visualized on CT. 18F-FDG identified 1 PNET that was not seen on CT. Among the patients imaged with all four modalities, only 12 of 76 (15%) lesions were positive on 18F-DOPA and 46 of 76 (60%) lesions were positive on 18F-FDG. There were 11 18F-DOPA and 22 18F-FDG avid extra-pancreatic lesions (most in the adrenal glands). One patient underwent resection of an 18F-DOPA avid extra-pancreatic lesion in the lung, with pathology confirming a NET.

Conclusions: MRI and 18F-FDG may be helpful adjuncts to CT to confirm the presence of PNETs in patients with VHL. 18F-DOPA appears to have limited utility in identifying PNETs. Overall, pancreatic protocol CT is the most sensitive imaging modality for identifying PNETs in VHL.

NOTES

13. Z-E SYNDROME ASSOCIATED WITH A HISTORY OF ALCOHOL ABUSE: COINCIDENCE OR CONSEQUENCE?

Stuart D. Wilson MD, Kara M. Doffek, Elizabeth A. Krzywda APNP, Edward J. Quebbeman MD, Kathleen K. Christians MD, Sam G. Pappas MD
Medical College of Wisconsin

Background: This 47 year observational study suggests that sporadic Zollinger-Ellison (Z-E) syndrome, particularly duodenal wall gastrinomas (DWG), is associated with a history of alcohol abuse. Z-E patients with DWG, even with paraduodenal lymph node metastases, are long lived if gastric acid hypersecretion is controlled, whereas Z-E patients presenting with pancreatic gastrinomas are short lived. Recent studies (Fendrick 2009) indicate different genetic background between DWG and pancreatic gastrinomas. This study evaluates differences in drinking patterns and natural history of Z-E patients with duodenal wall vs pancreatic gastrinomas.

Methods: Thirty-nine consecutive Z-E patients were evaluated and followed from 1962 thru 2010. Gastric analysis, serum gastrin, secretin provocative test and imaging confirmed diagnosis. Drinking patterns of Z-E patients were compared to 3786 community patients studied in a cardiovascular data registry.

Results: There were 28 men and 11 women, mean age 52 years (range 33-73). All had gastric acid hypersecretion (BAO > 15 mEq) and hypergastrinemia. Overall 35 patients had extrapancreatic gastrinomas (34 DWG and/or paraduodenal lymph nodes, one antral gastrinoma). Four patients had a pancreatic gastrinoma. Total gastrectomy (TG) was done in 24 patients; there were 2 postop deaths. Four other deaths were alcohol related. Mean survival after TG was 16 years (2-35). Nine patients had operations to remove DWG and paraduodenal lymph nodes. Two patients have not been operated. Mean survival was 15 years (5-27). There were no deaths from tumor progression. Four patients presented with pancreatic gastrinomas and liver metastasis, all died from tumor progression at 2, 3, 7 and 11 years. Alcohol abuse (>50 gm/day) was documented in 25 of 34 (74%) patients with DWG and/or paraduodenal lymph nodes with tumor. Only 1 of the 4 patients who presented with a pancreatic gastrinoma and liver metastases had a history of alcohol abuse. The drinking patterns (drinks/day) of extrapancreatic DWG patients were dramatically different from the control group: abstainers: 3% vs 24%; 1-2 drinks: 16% vs 62%; 3-5 drinks: 29% vs 12%; 6+ drinks: 52% vs 2.5%. (P< 0.01)

Conclusions: Alcohol abuse appears to be an important risk factor for sporadic Z-E with extrapancreatic DWG. Liver metastases and tumor deaths were not observed in this subgroup, supporting the concept that DWG and pancreatic gastrinomas are different tumor entities.

NOTES

14. SSTR5 P335L-SPECIFIC MONOCLONAL ANTIBODY DIFFERENTIATES PANCREATIC NEUROENDOCRINE TUMOR (PNT) PATIENTS WITH DIFFERENT SSTR5 GENOTYPES

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Background: Somatostatin radionuclide therapy (SRT) is effective against pancreatic neuroendocrine tumors (PNT) in 20% of patients, however, 20% and 50% of patients have no or limited response, respectively. We hypothesize the reason must be due to lack of expression of SSTRs or expression of a hypofunctional SSTR in which the signal of the analogue is not transmitted into the cell. SSTR5 is one of major SSTRs responsible for SRT. We have shown that SSTR5 P335L is a hypofunctional single nucleotide polymorphisms (SNP). The purposes of this study are to: 1) analyze the distribution of SSTR5 P335L SNP in PNT and normal controls and 2) develop a SSTR5 P335L-specific monoclonal antibody (mAb) for diagnostics.

Methods: PAXgene Blood DNA kit was used to isolate genomic DNA from 12 PNT patients and 429 de-identified healthy adults from three ethnic groups (Caucasian, Hispanic and African American). SSTR5 genotype was determined with the TaqMan SNP Genotyping assay. Mouse anti-SSTR5 P335L mAb was generated by injecting a SSTR5 P335L-derived peptide-KLH conjugate in Freund's complete adjuvant into BALB/cJ mice. Transient lipofectamine transfections of SSTR5 P335L and WT SSTR5 were performed in HEK293.

Results: 1. In 28 PNT patients, 39% (n=11) had TT genotype for SSTR5 P335L, 32% (n=9) had CC genotype for WT SSTR5, and 29% (n=8) had CT genotype for both SSTR5 P335L and WT SSTR5. Control cohort showed genotypes of 75%, 27% and 6% for SSTR5 P335L in Caucasians, Hispanics and African-Americans, respectively. 2. SSTR5 P335L mAb recognized only SSTR5 P335L, but not WT SSTR5, in Western blotting and immunohistochemistry (IHC); 3) Western blotting using SSTR5 P335L mAb showed that PANC-1 and Mia PaCa-2 cells expressed SSTR5 P335L, with higher levels in PANC-1 cells, consistent with the TT genotype of SSTR5 in PANC-1 vs. the CT genotype of SSTR5 in Mia PaCa-2; and 4) IHC using SSTR5 P335L mAb detected immunostaining signals only from the PNT specimens with TT and CT genotypes, but not those with CC genotypes.

Conclusions: These data show the presence of the hypofunctional SSTR5 P335L SNP in 71% of a small group of PNT patients and its frequency is race-dependent in the normal cohort. Generation of a mouse SSTR5 P335L mAb that specifically recognizes SSTR5 P335L, but not WT SSTR5, provides a potential tool for clinical diagnosis of PNT. These data provide the justification to perform a study linking SSTR5 P335L genotype to response rates of SRT.

NOTES

*15. ACHIEVING EUGASTRINEMIA IN MEN1 PATIENTS: BOTH DUODENAL INSPECTION AND FORMAL LYMPH NODE DISSECTION ARE IMPORTANT

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Background: Appropriate surgical management of hypergastrinemia(HG) in patients(pts) with multiple endocrine neoplasia type 1(MEN1) is controversial, with debate regarding the role and extent of resection and ability to achieve eugastrinemia(EG).

Methods: An institutional MEN1 database was reviewed to identify pts with clinical and biochemical evidence of HG(defined as basal gastrin \geq 200pg/ml). The relationship of extent of surgery to achievement of EG was evaluated. Regional lymph node dissection(RLND) was defined as an anatomic operation that included peri-duodenal, peri-pancreatic head, portal and hepatic arterial nodes.

Results: 163 pts had MEN1;113(69%) had pancreaticoduodenal neuroendocrine tumors of whom 28(25%) had HG. Surgery was performed in 20 HG pts with a median follow-up(FU) of 51 months(mo). Operations performed were: duodenotomy+distal pancreatectomy with RLND(4), duodenotomy+RLND(3), pancreaticoduodenectomy+RLND(4), total pancreatectomy(2; 1 with RLND), distal pancreatectomy+/-enucleation without duodenotomy(8), and enucleation alone(1). Duodenal neuroendocrine tumors(gastrinomas) were identified in 5/6 pts who underwent duodenectomy and 6/7 pts who underwent duodenotomy. Pts who underwent RLND had a median of 16 nodes removed, vs. 1 in non-RLND pts. Nodal metastases were identified in 16/20(80%) pts; the median number of involved nodes was 3(range 1-15). Postoperative EG was achieved in 12 pts(60%), while 8(40%) had persistent HG. 1 pt rendered EG had a primary nodal gastrinoma, while 2 had preoperative imaging and operative confirmation of nodal disease outside the "gastrinoma triangle." Compared to pts with persistent HG, pts rendered EG more often underwent intra-operative duodenal inspection(11/12; 92% vs. 2/8; 25%, $p=.01$) and RLND(11/12; 92% vs. 3/8; 38%, $p=.03$); there was no relationship between pancreatic resection and achievement of EG($p=.32$). After a median FU of 44 mo, 10/12 pts rendered EG continued to have EG, including 3 pts with FU of >5 years.

Conclusions: Surgery can achieve EG in selected MEN1 patients with HG, and EG can be durable. Pts with HG undergoing surgery should have intraoperative evaluation of the duodenum. Given the high rate of regional nodal metastasis, RLND should also be performed. In contrast, the extent of pancreatic resection should be dictated by the extent and distribution of pancreatic neuroendocrine tumors, rather than by the presence of HG.

NOTES

*16. TARGETED PARATHYROIDECTOMY WITHOUT SESTAMIBI SCANS: CASE SERIES OF SURGEONS-PERFORMED ULTRASONOGRAPHY AS THE ONLY METHOD OF PREOPERATIVE LOCALIZATION

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Background: With the advent of targeted parathyroidectomy for treatment of sporadic primary hyperparathyroidism (SPHPT), preoperative localization studies have become increasingly important for operative planning. Previous studies have compared the accuracy of sestamibi scans (MIBI) vs. surgeon-performed ultrasound (SUS) in localizing hypersecreting glands. Although SUS has been proved to be just as accurate as MIBI scans, it is still common practice to obtain both studies in all patients before neck exploration increasing the cost of parathyroidectomy. The goal of this study is to demonstrate the feasibility of performing parathyroidectomy guided by intraoperative PTH monitoring (IPM) based on SUS localization alone without the added cost of MIBI scans.

Methods: 194 consecutive patients with SPHPT undergoing parathyroidectomy guided by IPM were studied. All patients had preoperative ultrasound performed by the same surgeon. 130/194 (67%) patients had only SUS (n=93) or were referred with a previously negative MIBI (n=41). Parathyroid localization information, intraoperative findings including intraoperative PTH levels, postoperative pathology and laboratory values were analyzed to determine the accuracy of the SUS in localizing abnormal parathyroid glands.

Results: 129/130 patients were eucalcemic postoperatively (82 followed >6 and 101 >2 months). The incidence of multiglandular disease was 10%. IPM predicted postoperative serum calcium levels in all patients. SUS correctly identified at least one abnormal parathyroid gland in 90% (117/130) of the patients. The sensitivity of SUS was 84%, specificity 88%, negative predictive value 93%, positive predictive value 73%, overall accuracy 87%. With the help of office-based differential jugular venous sampling in selected patients with equivocal or multiple suspicious lesions on SUS, unilateral neck exploration with IPM guidance was possible in the vast majority of the patients.

Conclusions: Preoperative SUS, without MIBI scans, is an accurate method of parathyroid gland localization before neck exploration in patients with SPHPT. However, IPM remains paramount in determining the extent of parathyroidectomy. This data confirms the feasibility of SUS as a single method for preoperative localization obviating the need for MIBI scans potentially decreasing the cost of parathyroidectomy and simplifying the preoperative work-up for the majority of the patients.

NOTES

17. THE INCIDENCE OF CENTRAL NECK MICROMETASTATIC DISEASE IN PATIENTS WITH PAPILLARY THYROID CANCER STAGED PREOPERATIVELY AND INTRAOPERATIVELY AS NO

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Background: In papillary thyroid cancer, the incidence of regional lymph node metastasis in the central compartment has been reported to be between 21% and 60%. The true incidence of micrometastases (nodes < 1cm harboring disease and not evident on pre-operative imaging or at the time of surgery) is less clear because of the lack of standardization to the central neck dissection.

Methods: This study sought to establish the rate of micrometastatic disease in the central neck for patients staged as NO by preoperative and intraoperative assessment. 72 consecutive patients with a diagnosis of papillary thyroid cancer without preoperative or intraoperative evidence of central neck metastases that underwent a total thyroidectomy were submitted to an elective central neck dissection ipsilateral to the lobe harboring the tumor or bilaterally when the primary tumor was located in the isthmus. The procedure was standardized and performed in accordance with the recommendation from the ATA consensus statement on the anatomy and terminology of central neck dissection. We studied the number of lymph nodes as a product of the dissection, the presence of nodal metastatic disease by histopathology and the complications related to the procedure.

Results: 30 patients underwent right central compartment dissections, 30 left and in 12 cases the dissection was bilateral. The range of dissected lymph nodes for the 60 unilateral central neck dissections was 3 to 28, with a mean of 7.45 (standard deviation 4.17). The number of lymph nodes dissected for the 12 bilateral central neck dissections ranged from 6 to 20 with a mean of 10.75 (standard deviation 4.75). The incidence of lymph node micrometastasis was 25.0% corresponding to 18 patients. 83.3% of patients did not experience any complications, 6.9% had temporary dysphonia and 8.3% temporary hypocalcemia. No cases of definitive vocal cord palsy or definitive hypocalcemia were observed after 3 months of the surgical treatment.

Conclusions: The incidence of central neck micrometastatic disease in patients with PTC deemed NO preoperatively and intraoperatively is 25%. This relatively low rate of micrometastatic disease emphasizes the need for a careful weighing of the risks and benefits of elective central neck dissection before being routinely employed.

NOTES

*18. PREOPERATIVE BASAL CALCITONIN LEVEL AND NOT EXTENT OF SURGERY CORRELATES WITH POSTOPERATIVE CALCITONIN LEVELS IN PATIENTS UNDERGOING INITIAL SURGICAL MANAGEMENT OF MEDULLARY THYROID CARCINOMA.

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Background: The optimal initial surgical management of medullary thyroid cancer (MTC) remains controversial and the use of biomarkers to guide the extent of surgery or postoperative surveillance has not been well studied in large series. Our hypothesis is that preoperative levels of calcitonin (CT) and carcinoembryonic antigen (CEA) correlate with extent of disease and that post-operative CT and CEA reflect the extent of surgery performed.

Methods: We retrospectively assessed clinicopathologic factors among patients with MTC undergoing at least total thyroidectomy at two tertiary-care hospitals from 1980-2009; these factors were correlated with pre- and post-operative biomarkers using regression analyses.

Results: Data were obtained from 102 patients (mean age 47 (41.9); 61% women), 74% with sporadic and 26% with hereditary MTC. Fine-needle aspirate correctly identified MTC in 69% of patients. Sixty-six patients had central compartment lymph node dissections, 38 underwent ipsilateral, and 12 had bilateral modified radical dissections. Median tumor size was 17 mm (IQR 1.5-30 mm). Tumor stage correlated with lymph node positivity (OR 2.2, CI [1.1, 4.4]) after adjusting for pre-operative CT. Tumor stage ($p=.003$), positive lymph nodes ($p=.02$), and c-cell hyperplasia (CCH, $p=.04$) were significantly associated with pre-operative CT levels on univariate analysis; however, only tumor stage ($p=.005$) was associated after multivariable adjustment. No patient with a pre-operative CT < 50 pg/mL ($n=17$) had lymph node metastases. On univariate analysis, postoperative CT and lymph node metastases ($p=.01$) were associated. Pre- and post-operative CT levels were correlated regardless of the extent of surgery, lymph node positivity, tumor stage, or hereditary MTC (adjusted $p=.05$). Post-operative CEA correlated with tumor stage (univariate $p=.02$) and positive margins (adjusted $p=.03$). Neither pre- nor post-operative CEA was significantly correlated with lymph node positivity or extent of surgery.

Conclusions: Preoperative serum CT level was associated with tumor stage and the only independent predictor of postoperative CT in our patients undergoing initial surgery for MTC. Future studies should prospectively evaluate preoperative CT as a determinate of the extent of initial surgical therapy.

NOTES

19. A QUANTITATIVE TOOL TO OBJECTIVELY ASSESS DEGREE OF SARCOPENIA IN PATIENTS WITH HYPERCORTISOLISM

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Background: Muscle weakness and wasting are known manifestations of hypercortisolism (HC). Central sarcopenia is a marker of frailty and predicts mortality. This study seeks to confirm that central sarcopenia can be used as a marker of disease severity and frailty in patients with HC, and can potentially be used to assess the need for more urgent intervention.

Methods: Using a novel technology developed at this institution, which breaks down a subject's CT scan into hundreds of thousands of granular data elements that are multi-dimensional in character, a measurement of photographic density can be transformed into a numerical density. Psoas muscle cross-sectional area was measured on CT scans of patients with HC using a defined protocol. Psoas muscle density (PMD) and bone mineral density (BMD) were recorded at specific areas on each CT scan. 24 hour urine cortisol (24HUC) levels were recorded and matched by date with each CT scan. A linear regression model was used to describe the relationship between HC and PMD, psoas muscle size, and BMD. A comparison of PMD in matched patients without HC was also performed.

Results: 44 patients (34 female) with HC and 24HUC levels matched to CT scans performed at similar times were identified. Median age was 47 years (14-80), median 24HUC 218 (9.5-39,500), median Mean Psoas Density 52.1 (27-72). Pearson Correlation Coefficient comparing 24HUC levels to PMD was -0.34 (p -value=0.025). In determining factors related to degree of HC and psoas density, significance was retained when gender, age and type of HC were included in the model (p =0.01). Type of HC (p =0.02) and gender (p =0.03) were significant. PMD was not affected by the side of adrenal abnormality if one existed. Seven patients had greater than one CT and cortisol measurements. Two patients had increasing cortisol levels between scans and showed a decrease in PMD as cortisol levels increased. Five patients had decreases in 24HUC with varying density values. 20 non-HC patients matched for age, sex and BMI showed higher (63 vs 52) PMD (p = 0.001) compared to those with HC.

Conclusions: Mean PMD is significantly related to 24HUC levels. It is not clear that when 24HUC decreases the density increases. Objective and comprehensive measures of the overall burden of HC, such as sarcopenia, may allow more precise assessment of overall frailty and the need for more urgent intervention before a threshold is reached that leads to increased morbidity and mortality.

NOTES

*20. COMPARISON OF CLINICAL AND IMAGING FEATURES IN SDHB+ VERSUS SPORADIC PARAGANGLIOMAS

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Background: Paragangliomas (PGLs) develop sporadically or in familial genetic conditions, most commonly due to germline B subunit mutations in the succinate dehydrogenase (SDHB) gene. There is limited data on the optimal screening and most accurate diagnostic imaging studies in patients with PGLs. Thus, we studied the clinical, genetic and imaging features of 125 patients with histologically confirmed PGLs.

Methods: The clinical, genetic (SDHB vs. no SDHx), imaging (CT, MRI, FDG-PET), and laboratory features of patients with PGL were obtained over a decade. Data were analyzed using either Fisher's exact test or Wilcoxon rank sum test.

Results: Mean age at diagnosis was younger in the SDHB+ group compared to the sporadic (no SDHx) group (28 y. vs 39 y., respectively, $p < .001$). More often the sporadic group had an acute presentation (18.8%) defined as hypertension, tachycardia, syncope, or acute pain compared to the SDHB+ group (7.1%) ($p = .14$). The rate of supradiaphragmatic neoplasms in the SDHB+ group was 16.7% compared to 4.7% in the sporadic group ($p = .11$). Metastatic disease rates were higher in the SDHB+ group (78.9%) vs. the sporadic group (48.3%) ($p < .001$). 38.5% of SDHB+ patients and 16.7% of sporadic patients had either metastases or multiple PGLs at presentation ($p < .05$). Overall recurrence rates and median tumor volume did not differ. However, 23% of SDHB+ patients had tumor volumes > 250 cc, whereas none in the sporadic group had tumors this large ($p < .05$). On CT, SDHB+ tumors enhanced more with a mean density of 108.7 Hounsfield units (HU) compared to 88.4 HU for the sporadic group ($p < .05$). All PGLs were FDG-avid, with a mean standard uptake value (SUV) of 12.3 for the SDHB+ group and 8.0 for the sporadic group ($p < .05$).

Conclusions: Clinically young age, large tumor size and higher rate of metastatic and multifocal PGLs were observed in SDHB+ patients. Higher SUV values at FDG PET and increased CT enhancement were observed in PGL due to an SDHB germline mutation. However, MRI T2-weighted signal intensity did not differ between SDHB+ and sporadic patients. Ongoing study is needed to elucidate the biological basis for these findings. Imaging characteristics including large tumor volume (> 250 cc), increased density on CT and elevated SUV values at FDG-PET imaging may warrant genetic screening. Because SDHB+ patients demonstrate more supradiaphragmatic lesions, whole body imaging may be of particular value in these patients.

NOTES

21. IS THE GENETIC SCREENING INDICATED IN APPARENTLY SPORADIC PHEOCHROMOCYTOMAS?

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Background: Pheochromocytoma (Pheo) is considered a sporadic disease in most cases. In the recent years, several germline mutations in genes involved in the pathogenesis of hereditary forms have been identified; thus, an increasing rate of genetically-based variants has been reported. However, the need for systematic screening of unsuspected germline mutations in patients with apparently sporadic forms is still debated. This study was aimed to assess the rate of germline mutations causing Pheo in a large, unselected, single-center series of patients suffering from Pheo in order to classify them as having either truly sporadic or hereditary disease.

Method: Demographics, clinical, and laboratory data at first presentation were assessed in 108 consecutive patients with Pheo. An extensive follow-up was performed; genetic testing for germline mutations of RET, MEN1, VHL, NF1, SDHD, SDHC, SDHB and TMEM127 was also performed. Apparently sporadic Pheo was defined as that without a suspicious personal or family history.

Results: Complete data were available for 71 patients. Twelve patients had evident hereditary disease at first presentation: NF1 and MEN2 in 4 cases; while no mutations were identified in 4 patients although the presence of familial bilateral Pheos. Among 59 patients with apparently sporadic Pheo, germline mutations were found in 9 patients: TMEM127 in 5 cases, SDHB in 2, VHL and SDHC in 1 case. Following the genetic screening, a recurrent Pheo was diagnosed in 2 affected patients; 4 relatives with subclinical Pheos were also identified. No differences were found between true sporadic and hereditary Pheo concerning age (mean 47 vs 41 yrs), sex ratio (F/M: 1.1 vs 1.3), tumor size and catecholamine levels, while bilateral and/or extrarenal tumors occurred most often in hereditary Pheos (50% vs 2%; $p < 0.0001$).

Conclusions: The rate of hereditary Pheo was higher than usually reported (29.6%), although the putative germline mutations remained unknown in 5.6% of cases. The heritability was evident at presentation only in 17% of cases; 15.2% of apparently sporadic forms were genetically inherited, and TMEM127 mutations represented the most frequent occurrence. Although the increased costs, the systematic genetic screening may lead to a stricter follow-up in index cases and to the presymptomatic detection of small tumors in relatives, with a reduced disease-related morbidity.

NOTES

22. CARDIAC DYSFUNCTION AND CATECHOLAMINE CARDIO-MYOPATHY IN PHEOCHROMOCYTOMA PATIENTS AND THEIR REVERSAL FOLLOWING SURGICAL CURE: RESULTS OF A PROSPECTIVE CASE-CONTROL STUDY

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Background: Recognition of Cardiac dysfunction(CVD) and catecholamine cardiomyopathy(CC) in Pheochromocytoma(PCC) patients is vital. Literature on these entities is in form of retrospective cases series, mostly small and without controls. We conducted, possibly first of its kind, prospective case-control study to document nature & extent of CVD & CC using objective parameters, and their reversal following surgical cure of PCC.

Methods: 35 PCC pts were evaluated with ECG, 2D-Echocardiography(ECHO), tissue Doppler imaging(TDI) and serum N-terminal pro Brain Natriuretic peptide(s-NT-proBNP) at diagnosis; after 2weeks of alpha blockade; and 7 days, 3 months & 6 months after curative PCC resection(lap/open). Same evaluation was done in controls- 10 normotensive non-PCC adrenal tumor pts before & after adrenalectomy; and 10 untreated essential hypertensives(HT).

Results: PCC pts had dilated left ventricle(LV) with larger mean LV diastolic dimension(LVIDd), LV end diastolic & systolic volumes(LVED,LVES) c.w. controls($p < 0.005$). Significant LV systolic dysfunction(LV ejection fraction-LVEF $< 45\%$) was present in 7(20%) PCC pts, 2 had critically low($< 30\%$)LVEF. Global indicator of myocardial performance(MPI) was significantly higher and indicator of diastolic dysfunction (transmitral Early/Late velocity ratio) significantly lower($p = 0.02$) in PCC. Mean TDI Ea, Aa & S values were not significantly different. Mean s-NT-proBNP levels were significantly higher($p = 0.0001$) in PCC pts(397 ± 579 pg/ml, range 15-3107, normal < 125) c.w. controls. s-NT-proBNP levels were > 125 pg/ml in 25(71%) and > 400 pg/ml in 7 PCC pts with severe CVD. LVEDD, LVED, LVES, LVEF improved significantly with alpha blockade and as early as 1-week post-surgery; with sustained improvement at 3 & 6mo. Diastolic function- Mitral E/A ratio and myocardial performance-MPI also improved after surgery with continued improvement at 3 & 6mo. s-NT-proBNP levels mirrored ECHO changes, with significant fall after alpha blockade, and sustained fall 1wk, 3mo & 6mo post-surgery. ECHO parameters & s-NT-proBNP normalized at 3-6 mo indicating reversibility of CVD after surgical cure of PCC.

Conclusions: Global LV diastolic & systolic dysfunction are common and specific to PCC. These improve rapidly with alpha blockade and as early as 1 week after PCC resection, and show subtle and continued improvement up to 3-6 months. s-NT-proBNP is sensitive indicator of myocardial dysfunction and precedes LV systolic derangement and improvement.

NOTES

23. PROSPECTIVE APPLICATION OF OUR NOVEL PROGNOSTIC INDEX IN THE TREATMENT OF ANAPLASTIC THYROID CARCINOMA

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Background: To determine appropriate therapeutic strategies for anaplastic thyroid carcinoma (ATC), we previously performed retrospective analyses of 44 patients with ATC treated between April 1976 and March 1999 (former cases). That study revealed that acute symptoms, large tumor >5 cm, distant metastasis, and leukocytosis \geq 10,000/mm³ were the most important independent factors for predicting prognosis of ATC. We devised a novel prognostic index (PI) by simply totaling the number of these 4 unfavorable prognostic factors present, from PI=0 to PI=4. We have since adopted the PI to decide treatment policy for ATC. In principle, when PI \leq 1, multimodal treatments comprising a combination of surgery, external beam radiotherapy and chemotherapy has been encouraged to prolong survival, while aggressive therapies have been avoided to maintain quality of life (QOL) for PI \geq 3. The validity of this therapeutic strategy was prospectively investigated.

Methods: Seventy-five patients with ATC were treated between April 1999 and December 2009 (PI \leq 1, n=25; PI \geq 3, n=29).

Results. Six-month survival rates (6MS) for PI \leq 1 and PI \geq 3 were 72% and 14%, respectively ($p < 0.0001$). Among PI \leq 1 patients, 11 (44%) underwent all three modes of treatment and showed significantly better survival than former cases (6MS: 73% vs. 56%; $p = 0.04$). Although locally curative surgery was performed on all 4 patients with UICC Stage IVA tumors and 11 of 15 patients with Stage IVB tumors, but only 1 of 5 patients with Stage IVC tumors, survival rates did not differ significantly between stages (6MS: IVA, 75%; IVB, 67%; IVC, 80%; $p = 0.68$). For patients with PI \geq 3, survival rates were equally dismal regardless of stage (6MS: IVA, 0%; IVB, 0%; IVC, 18%; $p = 0.26$). However, compared to former cases, the number of patients who underwent tracheostomy and who died directly from local disease (suffocation or bleeding) were significantly decreased (9/18 vs. 4/29, $p = 0.02$; 8/18 vs. 3/28, $p = 0.02$, respectively).

Conclusions: This prospective study confirmed that our PI is valid to anticipate the prognosis and promptly decide on treatment policy for patients with ATC. Adequate selection of patients for aggressive multimodal treatment or best supportive care based on PI may contribute to longer survival and better QOL.

NOTES

*24. MOTOR AND SENSORY BRANCHING OF THE RECURRENT LARYNGEAL NERVE

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Background: Recognition of extralaryngeal branching of the recurrent laryngeal nerve (RLN) is crucial, as inadvertent division of an unidentified branch may lead to significant voice, breathing or swallowing problems. The purpose of this study was to examine the rate of extralaryngeal bifurcation of the RLN and to demonstrate the distribution of the motor fibers within its branches.

Methods: This is a prospective study containing data collected from 211 patients over the course of 24 months. Operative data obtained includes the type of operation, location of the nerve, number of branches, the distance from the inferior border of the cricothyroid entry point to the point of bifurcation, the minimal stimulation level required (mA) to stimulate the nerve, and the location of the branches of the RLN that contain motor fibers that innervate the intrinsic muscles of the larynx.

Results: Data on 296 RLNs were collected (153 nerves were located on the right side and 143 nerves on the left). 128 of these RLNs (43.2%) were bifid, with a distribution of 65 (42.5%) bifurcations on the right, and 63 bifurcations (44.1%) on the left. Of the 82 patients dissected bilaterally, bilateral bifurcation occurred in 28 (34.1%) of them. The mean branching distance(SD) from the cricothyroid membrane on the right was 5.42 mm(2.18), while on the left it was 5.90 mm(2.20). The mean recorded minimal intensity for stimulation(SD) detected by the nerve monitor was 0.94 mA (0.39). In all 128 cases of bifid RLNs, the anterior branch contained only motor fibers to the vocal cords, while the posterior branch contained only sensory fibers based on the EMG reading.

Conclusions: Extralaryngeal bifurcation is present in 48.2% of the RLNs presented in this case series and the motor fibers of the RLN are exclusively located in the anterior branch of these bifurcations. These data indicate that the surgeon must have a high level of scrutiny when identifying the RLN to ensure that no branches are left vulnerable to inadvertent transection.

NOTES

*25. PREDICTIVE FACTORS OF MALIGNANCY IN PEDIATRIC THYROID NODULES

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Background: Many studies suggest that while most pediatric thyroid nodules are benign, there is a higher rate of malignancy than in adults. This belief has led clinicians to more aggressively pursue thyroidectomy in the pediatric population. In this study, we investigated whether certain clinical factors could predict malignancy in pediatric thyroid nodules.

Methods: Retrospective review of 78 consecutive thyroidectomies was conducted from 1994 to 2009 in patients between the ages of 7 and 18 years old at two tertiary medical centers. We performed analyses on the predictive value of 16 clinical factors for cancer including: demographics, clinical history, symptomatology, physical exam and USG characteristics, and fine needle aspiration (FNA) biopsy results. Student t-tests and Wilcoxon Rank Sum tests were used to examine continuous variables, and Chi-square tests were used to examine dichotomous variables. Regression models examined the relationship between predictors and malignancy.

Results: A total of 78 children aged 7 to 18 years were studied. Mean age was 15.4 (SD = 2.7) years, and 60 (76.9%) of patients were female. Pathology reports indicated the presence of malignancy in 43 (55.1%) subjects. After excluding individuals with missing data, malignancy was more likely among individuals with: 1) a family history of thyroid cancer (72.7% vs. 49.1%, $p = 0.059$), palpable lymphadenopathy (92.9% vs. 42.4%, $p = 0.001$) and hypoechoic nodules (72.2% vs. 36.0%, $p = 0.019$). None of the other predictors differed significantly in our sample. In a model controlling for age, gender and family history, palpable lymph nodes were associated with a greater than two fold increase in the risk of malignancy (RR 2.18, 95% CI 1.56 – 3.05). The positive predictive value (PPV) of FNA results were as follows: 0.94 for malignancy, 0.55 for indeterminate or suspicious lesions, and 0.79 for benign lesions.

Conclusions: In our cohort, thyroid cancer was found in 55% of pediatric patients with thyroid nodules. While family history of thyroid cancer and hypoechoic lesions are strongly associated with malignancy, patients with palpable lymph nodes appear to be at greatest risk. In addition, a benign FNA result in children is not as reliable as in adults. The presence of these three factors, even in the setting of a benign FNA, should heighten the suspicion of thyroid cancer in the pediatric population.

NOTES

26. THE IMPACT OF FLUS ON THE RATE OF MALIGNANCY IN THYROID FNA: EVALUATION OF THE BETHESDA SYSTEM FOR REPORTING THYROID CYTOPATHOLOGY

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Background: Indeterminate thyroid nodules continue to present a diagnostic dilemma for clinicians. The Bethesda System for Reporting Thyroid Cytopathology (BSRTC) was developed to refine cytology definitions, improve communication and clinical management. This study evaluates the impact of the BSRTC in a large cohort of patients undergoing thyroidectomy before and after its adoption at a single institution.

Methods: A total of 805 patients undergoing thyroidectomy for all indications in the pre-(Group-1: 7/07-1/09) and post-BSRTC (Group-2: 2/09-10/10) periods were reviewed. Cytologic diagnoses in group-1 included nondiagnostic, benign, follicular/Hürthle neoplasm, suspicious for cancer and cancer. Atypia/follicular lesion of undetermined significance (AUS/FLUS) was included in group-2. The percentage of each category, malignancy rate per diagnosis and rate of AUS/FLUS usage were calculated.

Results: Fifty-four percent (187/347) of group-1 patients had preoperative FNAs vs. 62% (282/458) in group-2. Group-1 FNA results included 3%(6) nondiagnostic, 48%(89) benign, 17%(32) follicular/Hürthle, 13%(25) suspicious for cancer and 19%(35) cancer. Group-2 results included 4%(11) nondiagnostic, 34%(96) benign, 12%(33) follicular/Hürthle, 10%(29) suspicious for cancer, 11%(31) cancer and 29%(82) AUS/FLUS. The malignancy rate for indeterminate FNAs in group-1 and-2 was 33% and 30%, respectively. The rate of cancer increased from 25% to 36% for follicular/Hürthle lesions and 44% to 52% in FNAs suspicious for cancer. The AUS/FLUS malignancy rate was 20%. Eighty-four percent of patients undergoing surgery for AUS/FLUS did so without a repeat FNA. AUS/FLUS was used in 154 of 1095(14%) FNAs reviewed since the adoption of BSRTC.

Conclusions: Implementation of the BSRTC led to an increased percentage of patients undergoing thyroidectomy for indeterminate results with the majority being AUS/FLUS. This new category was used more often than recommended (14% vs. 7%) with a higher than expected rate of malignancy (20% vs. 5-15%). It remains unclear whether this represents an over-utilization or an accurate number of AUS/FLUS based on the nonspecific criteria specified by BSRTC. Although the proportion of cytology specimens called follicular/Hürthle neoplasm decreased after BSRTC, the rate of malignancy increased. This experience suggests that adding the AUS/FLUS category has led to a more accurate separation of follicular lesions with differing risk of malignancy.

NOTES

27. THE IMPACT OF MICROSCOPIC EXTRA THYROID EXTENSION ON OUTCOME IN PATIENTS WITH CLINICAL T1 AND T2 WELL DIFFERENTIATED THYROID CANCER

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Background: The objective of our study was to report the impact of incidentally reported microscopic extrathyroid extension (ETE) on outcome in patients with clinical T1 or T2 (cT1/cT2) well differentiated thyroid cancer (WDTC). We also wanted to determine the effect of extent of surgery and adjuvant radioactive iodine (RAI) on outcome in patients with microscopic ETE.

Methods: From an institutional database of 1810 previously untreated patients with WDTC, we identified 984 patients (54%) who had thyroid surgery for cT1/T2 N0 disease. Of these, 869 patients were pathological T1/T2 tumors and 115 were upstaged to pathological stage T3 based on the finding of microscopic extrathyroid extension. Compared to the pT1/pT2 group, the pT3 group had more patients over the age of 45 years (66% versus 52%, $p=0.004$), were more likely to receive post operative RAI (57% versus 21%, $p<0.001$) and were more likely to have had total thyroidectomy (75% versus 56%, $p=0.002$).

Disease specific survival (DSS) and recurrence free survival (RFS) were analysed for each group using the Kaplan-Meier Method. In the pT3 group, factors predictive of outcome (age, gender, size of primary tumor, extent of thyroid surgery, use of RAI) were analysed by univariate and multivariate analyses.

Results: With a median follow up of 98 months (range 6-291), there was no significant difference in the 10 year DSS (99% versus 100%, $p=0.733$) or RFS (98% versus 95%, $p=0.188$) on comparison of the pT1/pT2 cohort and the pT3 cohort. Analysis of the pT3 group showed that male gender was predictive of poorer outcome but this was lost on multivariate analysis. Extent of thyroid surgery and administration of post operative RAI were not significant for recurrence on univariate or multivariate analysis in the pT3 cohort.

Conclusions: Outcomes in patients with well differentiated clinically intrathyroid cancers stage cT1T2N0 are excellent and not significantly affected by the discovery of microscopic ETE on histopathological analysis. The extent of surgical resection and administration of post operative RAI in patients with microscopic ETE does not have an impact on survival or recurrence.

NOTES

28. EFFECT OF POSTOPERATIVE THYROTROPIN SUPPRESSION THERAPY ON BONE MINERAL DENSITY IN PATIENTS WITH PAPILLARY THYROID CARCINOMA: PROSPECTIVE CONTROL STUDY

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Background: The influence of thyrotropin (TSH) suppression therapy (TST) on bone mineral density (BMD) remains contentious. Investigations into this issue have mostly been designed as cross-sectional studies and very few prospective control trials have been undertaken. We have carried out a randomized controlled trial (RCT) evaluating the effects of postoperative TST on disease-free survival for papillary thyroid carcinoma (PTC) since 1996. Accompanying the study, we prospectively verified the effects of TST on BMD.

Methods: For the RCT, participants were randomly assigned to receive TST (Group A, n=218, serum TSH levels kept below $0.01 \times \text{U/ml}$) or not (Group B, n=215, TSH adjusted to within normal ranges). Among these, female patients who agreed to annual examination of lumbar spine (levels L2-L4) BMD by dual-energy X-ray absorptiometry were enrolled. Patients receiving any medications for osteoporosis, hormone therapies for breast cancer, or treatment with steroid hormones, having Grave's disease, primary hyperparathyroidism, or postoperative hypoparathyroidism, showing bone metastasis or fracture, or who discontinued the RCT were excluded from analysis.

Results: A total of 144 patients in Group A (mean TSH, $0.07 \pm 0.10 \times \text{U/ml}$) and 127 patients in Group B (mean TSH, $3.14 \pm 1.69 \times \text{U/ml}$) were evaluated. Age and body mass index were identical between groups. Preoperative BMD expressed by T-score was -0.50 ± 1.42 in Group A and -0.60 ± 0.30 in Group B ($p=0.55$). Group B did not show a significant decrease in T-score until 5 years after surgery, whereas Group A showed a significant deterioration in T-score from 1 year after surgery. Among Group A patients, a significant decrease in T-score within 1 year was seen in patients 50-years-old or older, but not in those <50-years-old. After 5 years of continued TST (Group A), 20 patients showed T-score less than -2.0 and 100 patients did not. Compared to the latter patients, the former patients were significantly older (age 61.7 ± 8.6 years vs. 47.2 ± 12.9 years, $p<0.0001$) and had lower preoperative BMD (T-score -2.09 ± 0.59 vs. -0.01 ± 1.27 , $p<0.0001$).

Conclusions: This prospective study suggests that strict TSH suppression after surgery for PTC has adverse effects on BMD in women 50-years-old or older (postmenopausal women).

NOTES

29. ACTIVATION OF MTOR SIGNALING IN MEDULLARY AND AGGRESSIVE PAPILLARY THYROID CARCINOMAS

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Background: Mammalian target of rapamycin (mTOR), an important downstream kinase of the PI3K/AKT oncogenic pathway, may be involved in thyroid carcinogenesis. mTOR operates through two major downstream effectors: 4EBP1 that regulates the activity of the eukaryotic initiating factor 4E (eIF4E) controlling initiation of protein translation, and p70S6K, which activates the ribosomal protein S6 (rpS6) involved in ribosome biogenesis. We investigated the expression and activation patterns of mTOR signaling proteins in thyroid carcinoma cell lines and tumors and their association with histologic types and tumor aggressiveness.

Methods: Four cell lines were used including medullary (TT), anaplastic (ARO) and two novel cell lines of papillary thyroid carcinoma recently established in our laboratory. Total protein extracts from cells and paired (normal/tumor) patient samples were analyzed by Western blot for mTOR signaling proteins. In addition, specimens obtained from 45 patients with thyroid cancer including follicular (6), papillary (18), medullary (18) and poorly differentiated (3) carcinomas were analyzed using immunohistochemistry. Eight of the 18 papillary carcinomas were considered aggressive histologic variants. Antibodies used were specific for p-rpS6, p-4EBP1, 4EBP1, and eIF4E. Moreover, thyroid carcinoma cell lines were treated with rapamycin and the biologic effects on cell proliferation, viability and apoptosis were analyzed.

Results: Using Western blot analysis, p-rpS6, p-4EBP1, 4EBP1, and eIF4E were detected at a higher level in cultured and primary medullary and aggressive papillary thyroid carcinomas as compared to other tumor histologies and benign nodules. Similarly, mTOR signaling proteins were immunohistochemically expressed at a high level in medullary, poorly differentiated and aggressive variants of papillary thyroid carcinomas as compared with conventional papillary and follicular carcinomas ($p < 0.0001$). The level of eIF4E expression also correlated with tumor stage ($p = 0.03$). Treatment with increasing concentrations of rapamycin resulted in significant apoptotic cell death and decreased cell growth in medullary and aggressive papillary thyroid carcinoma cell lines.

Conclusions: mTOR signaling, which controls protein synthesis and recapitulates important biologic effects of PI3K/AKT pathway, is activated in clinically aggressive thyroid cancer types and represents a promising target for investigational therapies for these patients.

NOTES

30. THE SURGICAL COMPLETENESS OF BILATERAL AXILLO-BREAST APPROACH ROBOTIC THYROIDECTOMY: COMPARISON WITH OPEN THYROIDECTOMY BY PROPENSITY SCORE MATCHING ANALYSIS

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Background: The bilateral axillo-breast approach (BABA) robotic thyroidectomy (RT) has excellent cosmetic result. To assure the surgical completeness of BABA RT, we compared RT and conventional open thyroidectomy (OT) by means of the postoperative radioactive iodine (RAI) uptake of possible remnant thyroid tissue.

Methods: From February 2008 to January 2010, 425 patients who had received RAI ablation after BABA RT (184 cases) or OT (241 cases) for papillary thyroid carcinoma (PTC) were enrolled. From each group, 117 patients were respectively selected using propensity score matching of 8 separate factors. The remnant thyroid was measured by the neck-to-skull RAI uptake ratio on the first postoperative RAI ablation scan. Postoperative stimulated Tg levels, the total number of RAI ablation sessions, and the total RAI doses needed to achieve complete ablation were compared between the groups.

Results: There were no differences between the two groups in regards of the RAI uptake ratio (12.6 \pm 13.2 vs 13.4 \pm 13.2, $p = 0.487$), TSH level at ablation (107.1 \pm 68.7 vs 111.3 \pm 61.9 \times IU, $p = 0.474$), the mean stimulated Tg level (1.4 \pm 3.7 vs 1.4 \pm 3.9 ng/ml, $p = 0.541$), the proportion of the stimulated Tg level less than 1.0 ng/ml (64.6% vs 68.4%, $p = 0.336$), the total number of RAI ablation sessions (1.97 \pm 0.45 vs 1.99 \pm 0.50, $p = 0.780$), or the total RAI doses needed to achieve a complete ablation (65.6 \pm 34.8 vs 68.4 \pm 34.0 mCi, $p = 0.506$).

Conclusions: The BABA RT might achieve the same surgical completeness in regard of the RAI uptake after total thyroidectomy. The BABA RT might give a safe option for patients with thyroid cancer who are concerned with the scars in the neck area.

NOTES

31. IS VENOUS THROMBOEMBOLISM A REAL RISK FOR THYROID CANCER PATIENTS UNDERGOING SURGERY?

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Background: Venous Thromboembolism (VTE) is a leading cause of post-operative morbidity and mortality in cancer patients. Studies have reported that different malignancies may confer differential VTE risk, however, when deciding to anticoagulate patients undergoing surgical treatment for malignancy all cancers are weighted equally according to the current guidelines. Neck hematoma is a potentially lethal complication of thyroidectomy. We sought to confirm the incidence of VTE associated with thyroid surgery and determine the association between the thyroid cancer and VTE.

Methods: We performed a retrospective cohort study of patients who underwent thyroid surgery and were enrolled in the American College of Surgeons National Surgical Quality Improvement Program database (2005-2008). Patients were stratified by the presence (TCA) or absence (NTCA) of thyroid cancer. The incidence of 30 day post-surgery VTE was calculated and VTE risk factors were compared between groups using the student's t-test, Chi-Square and Fisher's exact tests as appropriate. Individual risk factor scores (RFS) based on current guidelines were calculated with and without TCA as a factor. Scores were compared with the student's t-test. Unplanned return to OR was compared using the Fisher's exact test.

Results: Sixteen VTE events were documented in 19,774 thyroid surgery patients studied. The incidence of VTE was 0.08% overall and in both the TCA (n=6048) and NTCA (n=13726) groups (0.08% vs 0.08%, p=1.00). TCA patients were younger, had lower BMI and were more likely to have had prior surgery than NTCA (<0.001, all). TCA operations were longer than NTCA procedures (130min v 114min, p<0.001). TCA patients were less likely to have CHF, be on bedrest and have lung disease (p=0.04, 0.04 and 0.02, respectively) than NTCA. TCA patients had slightly higher RFS prior to inclusion of TCA as a RF (mean 3.26 vs 3.21, p<0.001.) After adding an adjustment for TCA status to the RFS, 13% of TCA patients would have been re-classified as "high risk" for VTE. TCA patients were more likely to have an unplanned return to the OR (3.65% vs 1.84%, p<0.001)

Conclusions: The incidence of VTE following thyroid surgery is very low and is similar for TCA as NTCA thyroid surgery patients. VTE prophylaxis should be determined based on patient risk factors. Thyroid cancer does not appear to be associated with VTE; therefore, the inclusion of TCA as a risk factor for VTE may result in unnecessary anticoagulation.

NOTES

32. WOULD SCAN, BUT WHICH SCAN? A COST-UTILITY ANALYSIS TO OPTIMIZE PREOPERATIVE IMAGING FOR PRIMARY HYPERPARATHYROIDISM

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Background: Minimally invasive parathyroidectomy (MIP) for patients with primary hyperparathyroidism (pHPT) depends on accurate preoperative localization. This study examines the cost-utility of different imaging modalities, including sestamibi-SPECT (SS), ultrasound (US), and 4D-CT (CT).

Methods: A decision tree was constructed for patients with sporadic pHPT undergoing initial parathyroidectomy. Patients were randomized to 1 of 5 preoperative localization protocols: (1)US; (2)SS; (3)CT; (4)SS/US; and (5)SS/US and CT, if discordant. Patients with successful localization underwent outpatient MIP; patients with unsuccessful localization or multigland disease underwent bilateral exploration. The incremental cost-utility ratio (ICUR) of each protocol includes cost of imaging and all subsequent care, based on accuracy of localization. ICUR was determined from the societal perspective and reported in \$/quality-adjusted-life-year (QALY). Input data were obtained from the literature and Medicare. Sensitivity analyses were performed for relevant clinical inputs.

Results: In the base-case, US was the most cost-effective modality. US was least expensive, with a cost of \$6729, compared to \$7102 (CT), \$7530 (SS/US/CT), \$7573 (SS/US), and \$7916 (SS). The combined modalities were less expensive than SS alone because improved localization resulted in fewer bilateral explorations. QALYs were comparable across all modalities (range 29.77-29.83). The ICUR of US was comparable to CT (-\$36/QALY) and more favorable than SS (-\$20,824/QALY). US also was favorable compared to SS/US/CT, but with slightly lower quality of life, yielding an ICUR of (+)\$80,966/QALY. SS/US yielded equivalent QALYs, but US was less expensive. Sensitivity analyses demonstrated that the model was most sensitive to cost of surgery, sensitivities of US and CT, and probability of persistent disease. For example, decreasing US sensitivity from 90 to 60% would increase US costs by \$583; CT would then be favored with lower costs and higher quality of life.

Conclusions: In our model, US was the most cost-effective imaging modality for pHPT, followed by CT and SS/US/CT (when the initial two studies are discordant). However, cost-utilities were dependent on the reported sensitivities of high-quality US and CT. Our results suggest that surgeons should review data on the quality of available imaging modalities at their own institutions in order to optimize a strategy for preoperative localization.

NOTES

33. THYROID-SPECIFIC KNOCKOUT OF THE TUMOR SUPPRESSOR MITOGEN-INDUCIBLE GENE 6 ACTIVATES EGFR SIGNALING PATHWAYS AND SUPPRESSES NF- κ B ACTIVITY

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Background: Mitogen-inducible gene 6 (Mig-6) is a putative tumor suppressor gene and prognostic biomarker in papillary thyroid cancer. We hypothesized that targeted, thyroid-specific Mig-6 knockout in vivo would induce activation of pro-oncogenic signaling in thyrocytes.

Methods: We performed a thyroid-specific knockout using the Cre-loxP recombinase system; mice carrying the "floxed" Mig-6 allele were crossed with transgenic mice expressing Cre recombinase driven by the thyroid peroxidase (TPO) gene promoter. Mig-6 protein levels, as well as EGFR and ERK phosphorylation status, were determined by immunoblotting. Caveolae membrane, cell nuclear and cytosolic fractions were isolated and immunoblotting of EGFR and p65 NF- κ B determined EGFR internalization and NF- κ B activity.

Results: Four knockout and 4 control mouse thyroids were harvested at 2 months of age and the histology was reviewed by an endocrine pathologist for confirmation. Immunoblotting confirmed Mig-6 ablation in knockout mice. EGFR and ERK phosphorylation levels were increased in Mig-6 knockout compared to wild-type mice thyroids by immunoblotting. EGFR activation triggers EGFR internalization and cytoplasmic trafficking. In knockout mice, we observed that the total levels of EGFR were no different from wild-type. However, EGFR was absent in the caveolae-containing membrane fraction of knockout mice, indicating that Mig-6 depletion is associated with a change in the membrane distribution of EGFR. NF- κ B subunit nuclear translocation from the cytoplasm into the nucleus is a key mechanism for NF- κ B activation. We found that p65 was distributed in both the cytoplasm and nucleus in thyrocytes from knockout mice. In contrast, p65 was localized in the nucleus in the wild-type mice thyroids. These results indicate that Mig-6 loss decreases p65 activity.

Conclusions: Our results confirm the feasibility of targeted, thyroid-specific gene knockout as a strategy for studying the relevance of specific genes in thyroid oncogenesis in vivo. We are the first to suggest that Mig-6 works, in part, by altering the membrane distribution of EGFR, which would limit receptor degradation and favor signaling pathways.

NOTES

34. SIMULATION TRAINING FOR THYROID LOBECTOMY IN CONJUNCTION WITH OPERATIVE TRAINING IMPROVES RESIDENT PERFORMANCE

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Background: Reduced clinical exposure in general surgery training may make it more difficult for residents to achieve competence in performing thyroidectomy during training. Simulation (SIM) has been shown to improve performance of various procedures, but has not been evaluated in thyroidectomy. We hypothesized that standard operative training in conjunction with SIM training would improve resident performance of thyroid lobectomy.

Methods: We developed a novel, reproducible thyroidectomy bench model. After IRB approval, all PGY 3-5 residents at one institution were recruited for the study. A scoring rubric (12 domains) was created, modeled on validated rubrics for common medical procedures. It was based on specific, essential operative tasks for thyroidectomy. Using this tool, each resident's performance of thyroid lobectomy was scored by a thyroid surgeon prior to SIM training. Residents then completed a pre-SIM survey, performed SIM training, completed a post-SIM survey, and performed two total thyroidectomies with attending supervision to integrate the skills learned in SIM training. Each resident's performance was again scored when they performed their next thyroid lobectomy. Residents completed a post-study survey. The sign rank test was used to compare resident performance and attitudes before and after the training intervention.

Results: 14 of 16 PGY 3-5 residents completed the program (two residents were excluded for logistical reasons). 5 domains showed statistically significant improvement after training: dissection of the strap muscles ($p=.01$), superior pole vessels ($p=.002$), inferior pole vessels ($p=.03$), removal of lobe (.008) and overall performance ($p=.007$). Residents reported increased confidence in dissection of the RLN ($p=.03$) and in performing lobectomy overall ($p=.06$) after training; 86% thought SIM was helpful or very helpful. 93% thought the goals of the program were met.

Conclusions: SIM training in conjunction with operative training for thyroid lobectomy objectively improves resident skill, efficiency and confidence. Our inexpensive model is feasible for thyroidectomy SIM. We plan a larger study to assess the value of SIM alone using a control group. We will also study reproducibility and durability of SIM outcomes.

POSTER DISPLAYS

35. THE USE OF A BIPOLAR RADIOFREQUENCY ABLATION DEVICE UTILIZING NANOTECHNOLOGY FOR HEMOSTASIS DURING THYROID SURGERY.

Keith M. Baldwin DO, N. Joseph Espat MD, Ponnandai Somasundar MD
Roger Williams Medical Center

36. DOES THE FINAL INTRA-OPERATIVE PTH LEVEL REALLY HAVE TO FALL INTO THE NORMAL RANGE TO SIGNIFY CURE?

Alexandra E. Reiher, MD, Sarah Schaeffer, MS, ANP-BC, Herbert Chen, MD, and Rebecca Sippel, MD
University of Wisconsin

37. THYROID CANCER DETECTION WITH DUAL ISOTOPE PARATHYROID SCINTIGRAPHY IN PRIMARY HYPERPARATHYROIDISM.

Edwin O. Onkendi, MBChB, Melanie L. Richards, MD, Geoffrey B. Thompson, MD, David R. Farley, MD, Patrick J. Peller, MD, Clive S. Grant, MD
Mayo Clinic

38. ACTIVATION OF MEK1, A RAF-1 PATHWAY EFFECTOR, ALTERS MORPHOLOGY AND NEUROENDOCRINE PHENOTYPE IN MEDULLARY THYROID CANCER

Amal Alhefdhi, MD, Herbert Chen, MD, and Muthusamy Kunnimalaiyaan, PhD
Endocrine Surgery Research Laboratories, Department of Surgery, University of Wisconsin

40. SESTAMIBI IMAGING FOR PRIMARY HYPERPARATHYROIDISM: THE IMPACT OF SURGEON INTERPRETATION AND RADIOLOGIST VOLUME

Saqib Zia, MD, Rebecca Sippel, MD, Herbert Chen, MD
University Of Wisconsin Hospital

41. REOPERATIVE NODAL DISSECTION FOR RECURRENT PAPILLARY THYROID CANCER AFTER INITIAL TOTAL THYROIDECTOMY

David T. Hughes, MD, Amanda M. Laird, MD, Barbra S. Miller, MD, Paul G. Gauger, MD and Gerard M. Doherty, MD
Montefiore Medical Center/Albert Einstein College of Medicine and University of Michigan

42. GENETIC-POTENTIATED SYNERGETIC TARGETING OF MAPK AND PI3K PATHWAYS IN BRAF-MUTATED THYROID CANCER CELLS

Zakaria Y. Abd Elmageed, PhD, Micheal Xing, MD, Asim B. Abdel-Mageed, PhD, Emad Kandil, MD
Tulane University, School of Medicine

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43. CLINICAL SIGNIFICANCE OF NOVEL TUMOR-SUPPRESSOR ACTIVITY OF CELL ADHESION MOLECULE CD146 AND ITS DOWNSTREAM TARGET LATEXIN IN THYROID CANCER

Zakaria Y. Abd Elmageed, PhD, Krzysztof Moroz, MD, Asim B. Abdel-Mageed, PhD, Emad Kandil, MD

Tulane University, School of Medicine

45. RECEIVER OPERATING CHARACTERISTIC ANALYSIS OF INTRAOPERATIVE PARATHYROID HORMONE MONITORING TO DETERMINE OPTIMUM SENSITIVITY AND SPECIFICITY: ANALYSIS OF 896 CASES

Christina S. Foley, MD, Mira Milas, MD, Eren Berber, MD, Jamie Mitchell, MD, Joyce Shin, MD, Allan Siperstein, MD

The Cleveland Clinic

46. COMPARISON OF THERMAL PROFILES OF VESSEL SEALING TECHNOLOGY USING INFRARED THERMOGRAPHY

Carter T. Smith, MD, Barbara Zarebczan MD, Amal Alhefdhi MD, Herbert Chen MD, FACS

Department of Surgery, University of Wisconsin

47. ADRENALECTOMY IN OLDER AMERICANS HAS INCREASED MORBIDITY AND MORTALITY: AN ANALYSIS OF 5,144 PATIENTS

Hadiza S. Kazaure, B.Sc., Sanziana A. Roman M.D., Julie A. Sosa M.A., M.D*.

Yale University School of Medicine

48. PRIMARY HYPERPARATHYROIDISM: STILL A CONFUSING DIAGNOSIS?

Daniel X. Choi, MD, Daniel T. Ruan, MD, and Jacob Moalem, MD, FACS

University of Rochester

49. COMPARISON OF INTRA-OPERATIVE TIME UTILIZATION AND PERIOPERATIVE OUTCOMES FOR ROBOTIC VERSUS LAPAROSCOPIC ADRENALECTOMY

Koray Karabulut, MD, Allan Siperstein, MD, Eren Berber, MD

Cleveland Clinic Endocrinology and Metabolism Institute

50. ULTRASOUND USE BY ENDOCRINE SURGEONS

Roy Phitayakorn, MD, Sareh Parangi, MD, Richard A. Hodin, MD, Robert A. Sofferman, MD, Greg Randolph, MD, and Antonia E. Stephen, MD

The Massachusetts General Hospital

51. UTILITY OF THYROID PATHOLOGY SCREENING IN FIRST GRADE FAMILY MEMBERS WITH FAMILIAL PAPILLARY THYROID CARCINOMA

Jose Manuel Rodriguez, PhD, Diana Navas, MD, Antonio Ríos, PhD, Maria Balsalobre, MD, Nuria Torregrosa, MD, Manuel Reus, MD, Pascual Parrilla, PhD

Hospital Universitario Virgen de la Arrixaca

52. UTILITY OF THE ELASTOGRAPHY IN THE DIAGNOSIS OF MALIGNANCY OF THE THYROID NODULE PROSPECTIVE STUDY

Jose Manuel Rodriguez, PhD, Angela Cepedo, MD, Antonio Ríos, PhD, Maria Balsalobre, MD, Manuel Reus, MD, Pascual Parrilla, PhD
Hospital Universitario Virgen de la Arrixaca

53. CORRELATION BETWEEN BRAF MUTATION AND LYMPH NODE METASTASES IN PROPHYLACTIC AND THERAPEUTIC NECK DISSECTION. A PROSPECTIVE STUDY.

Simone E. Dutenhefner, MD, Suemi Marui, PHD, Claudio R. Cernea, PHD, Andre B. O. Santos, PHD, Milton Inoue, MD, Jose S. Brandão Neto, MD, Christina Chiang, MD, Erica Urbano, Julia Fukushima, Celso U. M. Friguglietti, PHD
Instituto do Cancer do Estado de São Paulo

54. CREATION OF A "PARATHYROID INDEX" NOMOGRAM TO PREDICT THE LIKELIHOOD OF ADDITIONAL HYPERFUNCTIONING PARATHYROID GLANDS DURING PARATHYROIDECTOMY

Haggi Mazeh, MD, Herbert Chen, MD, FACS, Rebecca S. Sippel, MD, FACS
University of Wisconsin

55. DISCRIMINATION OF BENIGN VERSUS MALIGNANT FOLLICULAR LESIONS OF THE THYROID GLAND USING GENE EXPRESSION OF FINE NEEDLE ASPIRATES

Sapna Nagar MD, Samreen Ahmed MPH, Nichole Urban MD, George Wilson PhD, Jan Akervall MD, PhD, and Peter Czako MD
William Beaumont Hospital

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Virginia Commonwealth University

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Sang-Wook Kang MD, Jae Hyun Park MD, Kyu Hyung Kim MD, So Hee Lee MD, Hang Rang Yu MD, Jong Ju Jeong MD, Woong Youn Chung MD, and Cheong Soo Park MD, FACS.

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Memorial Sloan-Kettering Cancer Center

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Mayo Clinic

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Departments of Surgery and Epidemiology, University of Iowa

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Center for Molecular Medicine and Surgery, Karolinska University Hospital Solna

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Department of Surgery; National Institute of Medical Sciences and Nutrition

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Xiao-Min Yu, MD, PhD, Muthusamy Kunnimalaiyaan, PhD, Herbert Chen, MD, FACS
Department of Surgery, and the UW Carbone Cancer Center, University of Wisconsin

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Thomas J. Wade, MD, Tina W.F. Yen, MD, MS, Douglas B. Evans, MD, Stuart D. Wilson, MD, Tracy S. Wang, MD, MPH
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University of Pittsburgh

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Endocrine Surgery Unit, Massachusetts General Hospital, Harvard Medical School

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M. D. Anderson Cancer Center

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University of Michigan

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University of Sydney Endocrine Surgical Unit

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University Hospitals-Case Western Reserve University School of Medicine

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Department of Endocrine Surgery, Hippocraton General Hospital of Athens

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Courtney Y. Lee, MD, Samuel K. Snyder, MD, Terry C. Lairmore, MD, Sean C. DuPont, BA, Daniel C. Jupiter, PhD

Scott & White Clinic/ Texas A&M HSC

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UCSF-East Bay Department of Surgery

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David F. Schneider, MD, MS, Gregory M. Day, BS, and Steven A. De Jong, MD, FACS
Loyola University Medical Center

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Michael J. Crowley, BS, Daniel Buitrago, MD, Filippo Filicori, MD, Xavier M. Keutgen, MD, Hasan Aldailami, BS, Theresa Scognamiglio, MD, Moonsoo Jin, D.Sc, Thomas J. Fahey, III, MD, Rasa Zarnegar, MD
Weill Cornell Medical College-New York Presbyterian Hospital

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Jovenel Cherenfant, MD, Tricia Moo-Young, MD, David J. Winchester, MD, Richard A. Prinz, MD
NorthShore University Healthsystem Hospital

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Chun-hao Chen, MD, Dmitriy Zamarin, MD, PhD, Sepideh Gholami, MD, Pingdong Li, MD, PhD, Richard Bakst, MD, Natalya Chernichenko, MD, Shuangba He, MD, Peter Palese, PhD, Jatin P. Shah, MD, Yuman Fong, MD, Richard J. Wong, MD
Memorial Sloan-Kettering Cancer Center

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Parth K. Shah, MD, Kinjal K. Shah, MD, Giorgos C. Karakousis, MD, Rachel R. Kelz, MD, Douglas L. Fraker, MD
Department of Surgery, University of Pennsylvania School of Medicine

93. EFFECT OF AN ORGANIZED, MULTIDISCIPLINARY APPROACH TO THYROID CANCER CARE ON PATIENT COMPLIANCE AND QUALITY OF SURVEILLANCE AT A LARGE, URBAN SAFETY-NET HOSPITAL

Sarah C. Oltmann, MD, Shelby A. Holt, MD, Stacey L. Woodruff, MD, Ildiko Lingvay, MD, Jeffrey H. Pruitt, MD, Fiemu E. Nwariaku, MD, Jennifer L. Rabaglia, MD
University of Texas Southwestern Medical Center

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Meena Said, MD, Philip I. Haigh MD, Trudy Poon MS
Kaiser Permanente Los Angeles Medical Center

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Robert Tasevski, MBBS, Erin Kennedy, PhD, Rinku Sutradhar, PhD, Nancy N. Baxter, PhD, David P. Goldstein, MSc, Jonathan Irish, MSc, Lorne E. Rotstein, MD, David R. Urbach, MSc
University of Toronto

BYLAWS

I. CORPORATION

- 1.1 NAME.** The name of the corporation is The American Association of Endocrine Surgeons.
- 1.2 PURPOSES.** The purposes for which the corporation is organized are as follows: The corporation is organized exclusively for the purposes set forth in Sections 501(c)(3) of the Internal Revenue Code of 1986 (or the corresponding provision of any future United States Internal Revenue law) (the “Code”), including, for such purposes, making of distributions to organizations that qualify as exempt organizations under Section 501(c)(3) of the Code. The objects of the corporation shall include: (1) advancement of the science and art of endocrine surgery and (2) maintenance of high standards in the practice and art of endocrine surgery; and doing anything reasonably in furtherance of, or incidental to, the foregoing purposes as the Council may determine to be appropriate and as are not forbidden by Section 501(c)(3) of the Code, with all the power conferred on nonprofit corporations under the laws of the State of Illinois.
- 1.3 NONPROFIT OPERATION.** The corporation shall be operated exclusively for scientific, literary and educational purposes within the meaning of Section 501(c)(3) of the Code as a nonprofit corporation. No Councilor or member of the corporation shall have any title to or interest in the corporate property or earnings in his or her individual or private capacity and no part of the net earnings of the corporation shall inure to the benefit of any Councilor, member, officer or any individual. No substantial part of the activities of the corporation shall consist of carrying on propaganda or otherwise attempting to influence legislation, nor shall the corporation participate in or intervene in any political campaign on behalf of (or in opposition to) any candidate for public office.

II. MEMBERSHIP

- 2.1 MEMBERSHIP.**
- A. Membership** in this Association shall be limited to surgeons of good professional standing, who have a major interest and devote significant portions of their practice or research to endocrine surgery, and who are certified by the American Board of Surgery or its equivalent in Canada, Central America, Mexico, and South America. In addition, membership shall be limited to fellows of the American College of Surgeons or its international equivalent.

B. Types of Members. There shall be seven types of members: Active, Senior, Allied Specialist, Honorary, Corresponding, Candidate, and Resident/Fellow.

1. Active members shall consist of original charter members and all members subsequently elected until they become eligible for senior membership. The number of active members shall not be limited.

1a. The candidates for Active membership would have attended at least one annual meeting (hereinafter “assembly”) of the American Association of Endocrine Surgeons prior to their application;

1b. The candidates for Active membership should be able to provide evidence of special interest in endocrine surgery;

1c. The candidates who are applying for Active membership, who have completed their Endocrine Surgical Fellowship, should be in practice at least for one year with special emphasis in endocrine operative surgery.

2. Senior members shall consist of Active members who have reached the age of 65 years or who have retired from active practice. Senior members shall have all the responsibilities and privileges of active members, excepting those regarding attendance at assemblies. Senior members are not required to pay dues.

3. Honorary members shall consist of individuals who have made outstanding contributions to the discipline of endocrine surgery. They shall have no voting privileges, are not eligible for election as officers, and are not subject to assessment for dues.

4. Corresponding members shall consist of individuals who meet all the same qualifications in their respective countries as active members. They shall have no voting privileges, are not eligible for election as officers, shall not have attendance requirements, but may be subject to dues at a reduced amount.

5. Allied Specialist members shall consist of specialists with American Board certification in their respective field or its equivalent in Canada, Central America, Mexico and South America. In addition, Allied Specialist membership shall be limited to Fellows of the American College of Surgeons or its international equivalent. Allied Specialist members shall have demonstrated a significant commitment to and documented excellence in clinical practice, education, and/or research in their area(s) of practice within endocrine surgery. Allied Specialist members shall have been in practice within their specialty for a minimum of five years beyond training. Non-physician scientists (PhD) with a demonstrated interest in, and who have made significant contributions to, the field of endocrine surgery, are also eligible for membership under the Allied Specialist category. Allied Specialist members shall pay dues as levied by the Council and approved by the

membership, shall have voting privileges, are subject to attendance requirements, shall have the right to attend the annual business meeting, can serve on committees, and are not eligible for election to office or Council.

6. Candidate members shall consist of individuals who have completed their surgical training and who are awaiting qualification as Active members. Candidate members are required to pay dues at a reduced rate, do not have voting rights, and may register for the annual meeting at a reduced rate. Candidate membership will be limited to a period of time no more than three years following completion of all continuous training to include residency and fellowship(s). A letter of sponsorship from an Active or Senior AAES member will be sufficient to be considered as a Candidate member. Candidate members are strongly urged to attend the annual meeting but need not have attended a prior meeting.

7. Resident/Fellow members shall consist of individuals who are currently training, either as surgical residents or fellows. Resident/Fellow members are required to pay dues at a reduced rate, do not have voting rights, and may register for the annual meeting at a reduced rate. Resident/Fellow membership is limited to the time that an individual is in a residency, research, or clinical fellowship training program. A letter of sponsorship from an Active or Senior AAES member will be sufficient to be considered as a Resident/Fellow member. Attendance at a prior meeting of the AAES is not required. Resident/Fellow members will become Candidate members upon completion of their training and upon request.

C. Election of New Members

1. Physicians fulfilling the requirements for Active membership stated in paragraphs 2.1A and 2.1B of these Bylaws who reside in the United States, Canada, Central America, Mexico or South America may be eligible for Active membership.

2. Application forms for Active or Corresponding membership shall be provided by the Secretary-Treasurer. Completed application forms signed by the proposed member, one sponsor, and two endorsees shall be delivered to the Secretary-Treasurer at least four months before the annual assembly. Completed applications shall be reviewed by Council, which has the right to accept or reject any application for membership in the Association. Names of prospective members recommended for election by the Council shall be submitted to the membership at the annual assembly. Election shall be made by secret ballot, by a three-fourths affirmative vote of the members present. A prospective member who fails to be elected at one assembly may be considered at the next two annual assemblies of the Association. If election

fails a third time, the prospective member's application may be resubmitted after a two year interval.

3. Prospective members for Honorary membership shall be proposed in writing to the Council through the Secretary-Treasurer. Prospective members approved by the Council will be elected by three-fourths affirmative vote of the Council and officers present.

4. Active members in good standing who subsequently take up practice in geographic areas outside of the United States, Canada, Central America, Mexico, or South America shall be changed to corresponding members of the Association.

D. Dues

Dues and assessments shall be levied by the Council and approved by the membership at the annual assembly.

E. Resignations / Expulsions

1. Resignations of members otherwise in good standing shall be accepted by majority vote of the Council.

2. Charges of unprofessional or unethical conduct against any member of the Association must be submitted in writing to Council. The Council's concurrence or disallowance of the charges shall be presented to the membership at the annual assembly executive session. A three-fourths affirmative vote of the members present shall be required for expulsion.

3. Any Active member who is absent from three consecutive annual assemblies without adequate explanation of this absence made in writing to the Secretary-Treasurer shall be dropped from membership in the Association by vote of the Council. Membership may be reinstated by vote of the Council.

4. Any member whose dues remain unpaid for a period of one (1) year shall be dropped from membership, provided that notification of such a lapse beginning at least three (3) months prior to its effective date. The member may be reinstated following payment of the dues in arrears on approval of the Council.

2.2 PLACE OF ASSEMBLIES. Annual and special assemblies of the members shall be held at such time and place as shall be determined by the Council.

2.3 ANNUAL ASSEMBLY. The annual assembly of the members of the corporation for election of Officers and Councilors and for such other business as may come before the assembly shall be held on such date and hour as shall have been determined by the members (or if the members have not acted, by the Council or the Chairperson), and stated in the notice of the assembly. If for any reason the annual assembly is not held on the determined date of any year, any business which could have been conducted at an annual assembly may be conducted at any subsequent special or annual assembly or by consent resolution.

A. During the annual assembly, there shall be an AAES Business Meeting of the membership. The business of the association shall be conducted at this time. The report of the nominating committee shall be presented to the membership during the AAES Business Meeting. Nominations may be made from the floor. Officers of the Association and Council members shall be elected by majority vote of the active and senior members during the AAES Business Meeting.

B. Any member of the Association may invite one or more guests to attend the annual assembly.

C. Abstracts for consideration for presentation must be authored or sponsored by a member of the following categories: Active, Corresponding, Senior, Honorary, or Allied Specialist.

2.4 SPECIAL ASSEMBLIES. Special assemblies of the members of the corporation may be called by the Council or the President and shall be called by the President or the Secretary-Treasurer at the written request of any 30 members of the corporation. No business may be transacted at a special assembly except the business specified in the notice of the assembly.

2.5 NOTICE OF ASSEMBLIES OF MEMBERS. Except as otherwise provided by statute, written notice of the place, day, and hour of the assembly and in the case of a special assembly, the purpose or purposes for which the assembly of the members of the corporation is called, shall be given not less than five (5) nor more than sixty (60) days before the date of the assembly to each member, either personally or by mailing such notice to each member at the address designated by the member for such purpose or, if none is designated, at the member's last known address.

2.6 WAIVER OF NOTICE. Whenever any notice whatever is required to be given under the provisions of the Illinois Not for Profit Corporation Act of 1986 ("the Act") or under the provisions of the articles of incorporation or bylaws of this corporation, a waiver thereof in writing signed by the person or persons

entitled to such notice, whether before or after the time stated therein, shall be deemed equivalent to the giving of such notice. Attendance at any meeting shall constitute waiver of notice thereof unless the person at the meeting objects to the holding of the meeting because proper notice was not given.

- 2.7 QUORUM OF MEMBERS ENTITLED TO VOTE.** A minimum of thirty (30) members eligible to vote shall constitute a quorum at the annual assembly to effect changes in the bylaws of the Association, to make assessments, to authorize appropriations or expenditures of money other than those required in the routine business of the Association, to elect officers, Council members and members, and to expel members. For the transaction of other business, the members entitled to vote present at any annual assembly shall constitute a quorum.

III. COUNCIL

- 3.1 COUNCIL.** The business and affairs of the corporation shall be managed by or under the direction of a Council which is the governing body of the corporation. The Council shall meet as often as necessary to conduct the business of the corporation.
- 3.2 NUMBER AND SELECTION OF COUNCIL.** The Council shall consist of the officers of the Association, the three immediate past Presidents, and six other Council members, as the membership shall from time to time determine. The Council shall be elected by majority vote of the Active and Senior membership during the AAES Business Meeting at its annual assembly and vacancies shall be filled in the manner specified in Section 3.4 below. Councilors (other than those elected to fill vacancies) shall serve for three (3) year terms, with two (2) Councilors being elected annually so as to provide overlapping terms.
- 3.3 REMOVAL.** Any Councilor may be removed from office with cause at any annual or special assembly of the members. No Councilor may be removed except as follows: (1) A Councilor may be removed by the affirmative vote of two-thirds of the votes present and voted, either in person or by proxy (2) No Councilor shall be removed at a meeting of members entitled to vote unless the written notice of such meeting is delivered to all members entitled to vote on removal of Councilors. Such notice shall state that a purpose or the meeting is to vote upon the removal of one or more Councilors named in the notice. Only the named Councilor or Councilors may be removed at such meeting. If the vote of Councilors is to take place at a special assembly of Councilors, written notice of the proposed removal shall be delivered to all Councilors no less than twenty (20) days prior to such assembly. Written notice for removal must include the purpose of the assembly (i.e., removal) and the particular Councilor to be removed.

- 3.4 VACANCIES.** Vacancies occurring in the Council by reason of death, resignation, removal or other inability to serve shall be filled by the affirmative vote of a majority of the remaining Councilors although less than a quorum of the Council. A Councilor elected by the Council to fill a vacancy shall serve until the next annual assembly of the membership. At such annual assembly, the members shall elect a person to the Council who shall serve for the remaining portion of the term.
- 3.5 ANNUAL ASSEMBLY.** The annual assembly of the Council shall be held at such place, date and hour as the Council may determine from time to time. At the annual assembly, the Council shall consider such business as may properly be brought before the assembly. If less than a quorum of the Councilors appear for such an annual assembly of the Council, the holding of such annual assembly shall not be required and matters which might have been taken up at the annual assembly may be taken up at any later regular, special or annual assembly or by consent resolution.
- 3.6 REGULAR AND SPECIAL ASSEMBLIES.** Regular assemblies of the Council may be held at such times and places as the Councilors may from time to time determine at a prior assembly or as shall be directed or approved by the vote or written consent of all the Councilors. Special assemblies of the Council may be called by the President or the Secretary-Treasurer, and shall be called by the President or the Secretary-Treasurer upon the written request of any two (2) Councilors.
- 3.7 NOTICE OF ASSEMBLIES OF THE COUNCIL.** Written notice of the time and place of all assemblies of the Council shall be given to each Councilor at least 10 days before the day of the assembly, either personally or by mailing such notice to each Councilor at the address designated by the Councilor for such purposes, or if none is designated, at the Councilor's last known address. Notices of special assemblies shall state the purpose or purposes of the assembly, and no business may be conducted at a special assembly except the business specified in the notice of the assembly. Notice of any assembly of the Council may be waived in writing before or after the assembly.
- 3.8 ACTION WITHOUT AN ASSEMBLY.** Any action required or permitted at any assembly of the Council or a committee thereof may be taken without an assembly, without prior notice and without a vote, if a consent in writing, setting forth the action so taken, shall be signed by all of the Councilors and all of any non-Councilor committee members entitled to vote with respect to the subject matter thereof, or by all the members of such committee, as the case may be. The consent shall be evidenced by one or more written approvals, each of which sets forth the action taken and bears the signature of one or more Councilors or committee members. All the approvals evidencing the consent shall be delivered to the Secretary-Treasurer to be filed in the

corporate records. The action taken shall be effective when all the Councilors or the committee members, as the case may be, have approved the consent unless the consent specifies a different effective date. Any such consent signed by all Councilors or all the committee members, as the case may be, shall have the same effect as a unanimous vote and may be stated as such in any document filed with the Secretary of State under the Illinois General Not for Profit Corporation Act.

- 3.9 QUORUM AND VOTING REQUIREMENTS.** A majority of the Councilors then in office and a majority of any committee appointed by the Council constitutes a quorum for the transaction of business. The vote of a majority of the Councilors or committee members present at any assembly at which there is a quorum shall be the acts of the Council or the committee, except as a larger vote may be required by the laws of the State of Illinois, these bylaws or the Articles of Incorporation. A member of the Council or of a committee may participate in an assembly by conference telephone or similar communications equipment by means of which all persons participating in the assembly can hear one another and communicate with each other. Participation in an assembly in this manner constitutes presence in person at the assembly. No Councilor may act by proxy on any matter.
- 3.10 POWERS OF THE COUNCILORS.** The Councilors shall have charge, control and management of the business, property, personnel, affairs and funds of the corporation and shall have the power and authority to do and perform all acts and functions permitted for an organization described in Section 501(c)(3) of the Code not inconsistent with these bylaws, the Articles of Incorporation or the laws of the State of Illinois. In addition to and not in limitation of all powers, express or implied, now or hereafter conferred upon Boards of Directors of nonprofit corporations, and in addition to the powers mentioned in and implied from Section 1.3, the Councilors shall have the power to borrow or raise money for corporate purposes, to issue bonds, notes or debentures, to secure such obligations by mortgage or other lien upon any and all of the property of the corporation, whether at the time owned or thereafter acquired, and to guarantee the debt of any affiliated or subsidiary corporation or other entity, whenever the same shall be in the best interests of the corporation and in furtherance of its purposes.
- 3.11 COMPENSATION.** Councilors shall receive no compensation for their services on the Council. The preceding shall not, however, prevent the corporation from purchasing insurance as provided in Section 5.1 nor shall it prevent the Council from providing reasonable compensation to a Councilor for services which are beyond the scope of his or her duties as Councilor or from reimbursing any Councilor for expenses actually and necessarily incurred in the performance of his or her duties as a Councilor.

IV. OFFICERS

- 4.1 OFFICERS.** The officers shall be a President, a President-Elect, a Vice President, a Secretary-Treasurer, and a Recorder.
- 4.2 ELECTION AND TERM OF OFFICE.** The President, President-Elect, and Vice President of the Association shall be elected for terms of one year each. The Secretary-Treasurer and Recorder shall be elected for three year terms. Officers of the Association shall be elected by majority vote of the active and senior members during the AAES Business Meeting.
- 4.3 REMOVAL.** Any officer or agent may be removed with or without cause by the Council or other persons authorized to elect or appoint such officer or agent but such removal shall be without prejudice to the contract rights, if any, of the person so removed. Election or appointment of an officer or agent shall not of itself create any contract rights.
- 4.4 PRESIDENT.** The President shall preside at Council assemblies and the annual members' assembly. The President shall appoint members to all standing and ad hoc committees and shall serve as an ex-officio member of each. Successors to vacated offices of the Association shall be appointed by the President until the position is filled at the next annual assembly. The President shall prepare an address to the annual assembly of the Association.
- 4.5 PRESIDENT-ELECT.** The President-Elect, in the absence or incapacity of the President, shall perform the duties of the President's office.
- 4.6 VICE PRESIDENT.** In the absence or incapacity of both the President and the President-Elect, the Chair shall be assumed by the Vice President
- 4.7 SECRETARY-TREASURER.** The Secretary-Treasurer shall keep minutes of the Association and the Council, receive and care for all records belonging to the Association, and conduct the correspondence of the Association. This office will issue to all members a written report of the preceding year's transactions to be read to the Council and membership at the annual assembly. The Secretary-Treasurer will prepare an annual report for audit. The Secretary-Treasurer shall have the authority to certify the bylaws, resolutions of the members and Council and committees thereof, and other documents of the corporation as true and correct copies thereof.
- 4.8 RECORDER.** The Recorder shall receive the manuscripts and edition of the discussions. The Recorder shall be custodian for the transactions of the Association.

V. INDEMNIFICATION

5.1 INDEMNIFICATION. Each person who is or was a Councilor, member, officer or member of a committee of the corporation and each person who serves or has served at the request of the corporation, as a Councilor, officer, partner, employee or agent of any other corporation, partnership, joint venture, trust or other enterprise may be indemnified by the corporation to the fullest extent permitted by the corporation laws of the State of Illinois as they may be in effect from time to time. The corporation may purchase and maintain insurance on behalf of any such person against any liability asserted against and incurred by such person in any such capacity or arising out of his status as such, whether or not the corporation would have power to indemnify such person against such liability under the preceding sentence. The corporation may, to the extent authorized from time to time by the Council, grant rights to indemnification to any employee or agent of the corporation to the fullest extent provided under the laws of the State of Illinois as they may be in effect from time to time.

VI. COMMITTEES

6.1 COMMITTEES. A majority of the Council may establish such committees from time to time as it shall deem appropriate and shall define the powers and responsibilities of such committees. The Council may establish one or more executive committees and determine the powers and duties of such executive committee or committees within the limits prescribed by law.

A. Standing committees of the Association shall consist of the Membership Committee (composed of the Council), Publication and Program Committee, Education and Research Committee, and Fellowship Committee.

B. The Nominating Committee shall consist of the President and two immediate past Presidents. The most senior past President is chairman of the committee.

C. All committees shall be chaired by members appointed by the President with the advice of the Council.

6.2 COMMITTEES OF COUNCILORS. Unless the appointment by the Council requires a greater number, a majority of any committee shall constitute a quorum, and a majority of committee members present and voting at a meeting at which a quorum is present is necessary for committee action. A committee may act by unanimous consent in writing without a meeting

and, subject to the provisions of the bylaws for action by the Council, the committee by majority vote of its members shall determine the time and place of meetings and the notice required thereof. To the extent specified by the Council or in the articles of incorporation or bylaws, each committee may exercise the authority of the Council under Section 108.05 of the Act; provided, however, a committee may not:

- A. Adopt a plan for the distribution of the assets of the corporation, or for dissolution;
- B. Approve or recommend to members any act the Act requires to be approved by members, except that committees appointed by the Council or otherwise authorized by the bylaws relating to the election, nomination, qualification, or credentials of Councilors or other committees involved in the process of electing Councilors may make recommendations to the members relating to electing Councilors;
- C. Fill vacancies on the Council or on any of its committees;
- D. Elect, appoint, or remove any officer or Councilor or member of any committee, or fix the compensation of any member of a committee;
- E. Adopt, amend, or repeal the bylaws or the articles of incorporation;
- F. Adopt a plan of merger or adopt a plan of consolidation with another corporation, or authorize the sale, lease, exchange or mortgage of all or substantially all of the property or assets of the corporation; or
- G. Amend, alter, repeal, or take action inconsistent with any resolution or action of the Council when the resolution or action of the Council provides by its terms that it shall not be amended, altered, or repealed by action of a committee.

VII. AMENDMENTS

- 7.1 AMENDMENTS.** These bylaws may be amended at the annual assembly of the membership provided a notice setting forth the amendment or a summary of the changes to be effected thereby is given to each member entitled to vote thereon in the manner and within the time provided in these bylaws for notice of the assembly. These bylaws may be amended at the annual assembly by a two-thirds affirmative vote of the members present. No amendment inconsistent with the Articles of Incorporation shall be effective prior to amendment of the Articles of Incorporation.

VIII. BOOKS AND RECORDS

- 8.1 BOOKS AND RECORDS.** The corporation shall keep correct and complete books and records of account and shall also keep minutes of the proceedings of its members, Council and committees having any of the authority of the Council, and shall keep at the registered or principal office a record giving the names and addresses of the Council and members entitled to vote. All books and records of the corporation may be inspected by any Councilor or member entitled to vote, or his or her agent or attorney for any proper purpose at any reasonable time.

IX. PARLIAMENTARY AUTHORITY

- 9.1 PARLIAMENTARY AUTHORITY.** The rules of parliamentary procedure in "Robert's Rules of Order, Revised", shall govern the proceedings of the assemblies of this corporation, subject to all other rules contained in the Articles of Incorporation and Bylaws and except that proxy voting shall be allowed in accordance with the Illinois General Not for Profit Corporation Act of 1986

X. SEVERABILITY

- 10.1 SEVERABILITY.** Each of the sections, subsections and provisions hereof shall be deemed and considered separate and severable so that if any section, subsection or provision is deemed or declared to be invalid or unenforceable, this shall have no effect on the validity or enforceability of any of the other sections, subsections or provisions.



GEOGRAPHICAL MEMBERSHIP DIRECTORY

2010 - 2011

GEOGRAPHICAL MEMBERSHIP DIRECTORY

Brazil

Porto Alegre

Molinari, Alberto S.

Sao Paulo

Aun, Frederico

Canada

ALBERTA

Calgary

Harvey, Adrian M.

Mack, Lloyd

Pasieka, Janice L.

BRITISH COLUMBIA

Vancouver

Bugis, Samuel P.

Melck, Adrienne L.

Schmidt, Nis

ONTARIO

Toronto

Rosen, Irving B.

Rotstein, Lorne E.

Tasevski, Robert

Urbach, David R.

QUEBEC

Montreal

Tabah, Roger J.

Chile

Santiago

Costa, Eduardo A.

Guatemala

Guatemala City

Penalzo, Marco A.

Mexico

Merida

Fajardo-Cevallos, Rafael E.

Mexico

Pantoja, Juan Pablo P.

Mexico City

Herrera, Miguel F.

Sierra-Salazar, Mauricio

USA

ALABAMA

Birmingham

Diethelm, Arnold G.

Smith, Gardner S.

Sperling, David

Mobile

Dyess, Donna Lynn

ARIZONA

Phoenix

Flynn, Stuart D.

Harding, Richard J.

Schlinkert, Richard T.

Scottsdale

Demeure, Michael J.

Van Lier Ribbink, Jeffrey A.

Tucson

Guerrero, Marlon A.

ARKANSAS

Little Rock

Kim, Lawrence T.

Mancino, Anne T.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

CALIFORNIA

Beverly Hills

Katz, Alfred D.

Duarte

Yim, John H.

El Macero

Wolfman, Earl

Fresno

Maser, Christina L.

Hillsborough

Lim, Robert C.

La Jolla

Bouvet, Michael

Sanford, Arthur

Los Altos

Allo, Maria D.

Los Angeles

Haigh, Philip I.

Hines, Oscar J.

Yeh, Michael W

Northstar-Truckee

Danto, Lawrence A.

Orange

Harness, Jay K.

San Diego

Block, Melvin A.

Clark, Gary C.

San Francisco

Clark, Orlo H.

Debas, Haile T.

Duh, Quan-Yang

Galante, Maurice

Gosnell, Jessica E.

Grogan, Raymon H

Harari, Avital

Hunt, Thomas K.

Mitmaker, Elliot J.

Shen, Wen Tsong

Yutan, Elaine U.

Santa Barbara

Latimer, Ronald G.

Santa Monica

Giuliano, Armando E.

Stanford

Greco, Ralph S.

Norton, Jeffrey A.

Sylmar

Zuckerbraun, Lionel

Woodland

Chen, Emery

COLORADO

Aurora

McIntyre, Robert C.

Raeburn, Christopher D.

Boulder

Brown, Dennistoun K.

Littleton

Liechty, R. Dale

CONNECTICUT

New Haven

Carling, Tobias

Foster, Jr., Roger S.

Milan, Stacey A.

Roman, Sanziana A.

Sosa, Julie Ann

Udelsman, Robert

DISTRICT OF COLUMBIA

Washington

Felger, Erin A.

Geelhoed, Glenn W.

DELAWARE

Newark

McField, Daaron

GEOGRAPHICAL MEMBERSHIP DIRECTORY

FLORIDA

Bay Pines

Goodgame, J. Thomas

Bonita Springs

Freier, Duane T.

Coral Gables

Irvin, George L.

Gainesville

Shaw, Christiana M.

Highland Beach

Hamburger, Stuart W.

Jacksonville

Adkisson, Cameron D.

Asbun, Horacio

Martin, J. Kirk

Smith, Stephen L.

Miami

Goldfarb, Melanie

Lew, John I.

Rodgers, Steven

Miami Beach

Dembrow, Victor D.

Safety Harbor

Schmidt, Rick J.

Stuart

Vopal, James J.

Tampa

Carter, Bradford

Fabri, Peter J.

Gallagher, Scott F

Norman, James G.

Politz, Douglas E.

GEORGIA

Atlanta

Sharma, Jyotirmay

Weber, Collin J.

Augusta

Mansberger, Arlie R.

Terris, David J.

Yeh, Karen A.

Lawrenceville

McGill, Julie F.

Marietta

Underwood, Robert A.

Savannah

Yeager, E. Stephen S.

HAWAII

Honolulu

Morita, Shane

Wong, Livingston

ILLINOIS

Aurora

Bloom, Allen D.

Chicago

Angelos, Peter

Cheatem, Donald M.

Fredland, Allan J.

Kaplan, Edwin L.

Moo-Young, Tricia A.

Patel, Subhash

Pickleman, Jack

Sturgeon, Cord

Evanston

Cherenfant, Jovenal

Prinz, Richard A.

Winchester, David J.

Hinsdale

Paloyan, Edward

Maywood

De Jong, Steven A.

Schneider, David F

North Chicago

Zdon, Michael J.

Oaklawn

Hopkins, William M.

Park Ridge

Hann, Sang E.

Zion

Staren, Edgar D.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

INDIANA

Indianapolis

Broadie, Thomas A.

Miskulin, Judiann

IOWA

Iowa City

Gurll, Nelson J.

Howe, James R.

Lal, Geeta

Sugg, Sonia L.

Weigel, Ronald J.

KANSAS

Kansas City

Cohen, Mark S.

Lake Quivera

Hermreck, Arlo S.

KENTUCKY

Louisville

Callender, Glenda G.

Goldstein, Richard E.

Quillo, Amy R.

LOUISIANA

Kenner

Woltering, Eugene A.

New Orleans

Jaffe, Bernard M.

Kandil, Emad

Shreveport

McDonald, John C.

MAINE

Bangor

Starks, Michael R.

Portland

Goldfarb, Walter B.

MacGillivray, Dougald C.

Radke, Frederick R.

Wu, Leslie S.

Vinalhaven

Kinder, Barbara K.

MARYLAND

Baltimore

Alexander, H. Richard

Dackiw, Alan P.B.

Gann, Donald S.

Marohn, Michael R.

Roy, Rashmi

Turner, Douglas J.

Zeiger, Martha A.

Bethesda

Hughes, Marybeth

Kebebew, Electron

Mathur, Aarti

Nilubol, Naris

Phan, Giao Q.

Chevy Chase

Stojadinovic, Alexander

MASSACHUSETTS

Auburndale

Silen, William

Boston

Beazley, Robert

Brooks, David C.

Gawande, Atul

Gaz, Randall D.

Hasselgren, Per-Olof J.

Hodin, Richard A.

Lubitz, Carrie C.

McAneny, David

Moore, Jr., Francis D.

Nehs, Matthew A.

Parangi, Sareh

Randolph, Gregory W.

Rosen, Jennifer E.

Ruan, Daniel T.

Stephen, Antonia E.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

MASSACHUSETTS CONT.

Brookline

Cady, Blake
Mowschenson, Peter

Burlington

Brams, David M.
Wei, John P.

Danvers

Narra, Vinod

Pittsfield

Curletti, Eugene L.

Shrewsbury

Patwardhan, Nilima

Springfield

Coe, Nicholas P.
Jabiev, Azad A.

Weston

Aliapoulos, Menelaos A.

MICHIGAN

Ann Arbor

Burney, Richard E.
Cerny, Joseph C.
Doherty, Gerard M.
Gauger, Paul G.
Laird, Amanda M.
Miller, Barbra S.
Thompson, Norman W.

Bloomfield Hills

Saxe, Andrew W.

Detroit

Eichhorn-Wharry, Laura I.
Talpos, Gary B.

Frankfort

Griffen, Ward O.

Lansing

McLeod, Michael K.

Midland

Sequeira, Melwyn John

MICHIGAN CONT.

Royal Oak

Czako, Peter F.
Reid, Darly A.

MINNESOTA

Minneapolis

Delaney, John P.
Najarian, John S.

Rochester

Carney, J. Aidan A.
Farley, David R.
Grant, Clive S.
Hay, Ian D.
McKenzie, Travis J.
Richards, Melanie L.
Service, F. John
Thompson, Geoffrey B.
Young, William F.

St. Paul

Sneider, Mark S.

MISSISSIPPI

Jackson

Parent, Andrew D.

Tupelo

Bowlin, John W.

MISSOURI

Columbia

Koivunen, Debra G.

Saint Louis

Ballinger, Walter F.
Brunt, L. Michael
Gillanders, William E.
Hall, Bruce L.
Moley, Jeffrey F.
O'Neal, Lawrence W.
Shieber, William

GEOGRAPHICAL MEMBERSHIP DIRECTORY

MONTANA

Kalispell

Sheldon, David G.

NEBRASKA

Papillion

Stanislav, Gregory

NEW JERSEY

Hackensack

Barbul, Adrian

Morristown

Whitman, Eric D.

Neptune

Shifrin, Alexander L.

New Brunswick

Trooskin, Stanley Z.

Paterson

Budd, Daniel C.

NEW MEXICO

Albuquerque

Quintana, Doris A.

Vanderveen, Kimberly

Rio Rancho

Miscall, Brian G.

NEW YORK

Albany

Beyer, Todd D.

Jahraus, Carrie B.

Bronx

Bocker, Jennifer M.

Hughes, David T.

Libutti, Steven K.

Buffalo

Cance, William G.

Cooperstown

Olson, John E.

Ryan, M. Bernadette B.

NEW YORK CONT.

Delmar

Kushnir, Leon

Ithaca

Foster, Cory L.

Lake Success

Sznyter, Laura A.

Mount Kisco

Spanknebel, Kathryn

New York City

Ahmed, Leaque

Allendorf, John D.

Brennan, Murray F.

Chabot, John A.

Fahey, Thomas J

Geha, Rula C.

Heller, Keith S.

Inabnet, III, William B.

Iyer, N. Gopalakrishna

Jahraus, Carrie B.

Lee, James

Marti, Jennifer L.

Ogilvie, Jennifer B.

Owen, Randall P.

Patel, Kepal N.

Shah, Jatin P.

Shaha, Ashok R.

Smith, Philip W.

Strong, Vivian E.

Tuttle, Robert M.

Weber, Kaare J.

Syracuse

Kort, Kara C.

Numann, Patricia J.

NORTH CAROLINA

Apex

Leight, George S.

Asheville

Humble, Ted H.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

NORTH CAROLINA CONT.

Chapel Hill

Croom, Robert D.
Thomas, Jr., Colin G.

Charlotte

Kercher, Kent
Pederson, Lee
Wagner, Kristin E.

Durham

Olson, Jr., John A.
Scheri, Randall
Tyler, Douglas S.
Wells, Jr., Samuel A.

Greenville

Pofahl, Walter E.
Pories, Walter J.

Raleigh

Faust, Kirk B.

Salem

Randle, Reese W.

Winston Salem

Albertson, David A.
Cannon, Jennifer

OHIO

Akron

Horattas, Mark C.

Cincinnati

Steward, David L.

Cleveland

Berber, Eren
Esselstyn, Caldwell B.
Mansour, Edward G.
McHenry, Christopher R.
Milas, Mira M.
Raaf, John H.
Shin, Joyce
Siperstein, Allan
Wilhelm, Scott M.

OHIO CONT.

Columbus

Arrese, David
Ellison, Christopher
Farrar, William B.
Phay, John E.

OREGON

Portland

Pommier, Rodney F.
Sheppard, Brett C.
Yu, Kelvin C.

PENNSYLVANIA

Abington

Borman, Karen R.
Kukora, John S.

Allentown

Hartzell, George W.

Center Valley

McDonald, Marian P.

Danville

Pellitteri, Phillip K.

Danville

Strodel, William E.

Harrisburgh

Yang, Harold C.

Hershey

Kauffman, Jr., Gordon L.

Palmyra

Shereef, Serene

Philadelphia

Cohn, Herbert E.
Fraker, Douglas L.
Kairys, John C.
LiVolsi, Virginia
Ridge, John A.
Yeo, Charles J.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

PENNSYLVANIA CONT.

Pittsburgh

Bartlett, David L.
Carty, Sally E.
Kabaker, Adam S.
McCoy, Kelly L.
Stang, Michael T.
Stremple, John
Yip, Linwah

Ridley Park

Amara, Shamly V.

Sayre

Trostle, Doug R.

RHODE ISLAND

Providence

Baldwin, Keith M.
Mazzaglia, Peter J.
Monchik, Jack M.

SOUTH CAROLINA

Charleston

Carneiro-Pla, Denise
Cole, David J.
van Heerden, Jon A.

Columbia

Brown, J. Jeffrey

Greenville

Lokey, Jonathan S.

Spartanburg

Orr, Richard K.

TENNESSEE

Chattanooga

Giles, Wesely H.
Roe, Michael

Knoxville

Nelson, Jr., H. Sperry
Zirkle, Kevin

TENNESSEE CONT.

Nashville

Abumrad, Naji N.
Broome, James T.
Solorzano, Carmen C.
Williams, Kathleen C.

TEXAS

Dallas

Holt, Shelby A.
Nwariaku, Fiemu E.
Steckler, Robert M.

Houston

Brandt, Mary L.
Brunicardi, F. Charles
Clayman, Gary
Jackson, Gilchrist L.
Landry, Christine S.
Lee, Jeffrey E.
Lopez, Monica E.
Perrier, Nancy D.

San Antonio

Santillan, Alfredo A.

Temple

Lairmore, Terry C.
Lee, Cortney Y.
Snyder, Samuel K.

Wichita Falls

Sutton, Beth H.

VIRGINIA

Charlottesville

Hanks, John B.

Falls Church

Broughan, Thomas A.
Lee, Louis C

Richmond

Grover, Amelia C.
Merrell, Ronald C.
Newsome, Jr., H. H.
Stevenson, Christina E.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

WASHINGTON

Spokane

Sinha, Renu

WEST VIRGINIA

Morgantown

Mitchell, Bradford K.

Ross, Arthur J.

WISCONSIN

La Crosse

Kisken, William A.

Madison

Alhefdhi, Amal Y.

Chen, Herbert

Kunnimalaiyaan, Muthusamy

Mack, Eberhard A.

Matze, Greg M.

Sippel, Rebecca S.

Starling, James R.

Wenger, Ronald D.

Milwaukee

Evans, Douglas B.

Wang, Tracy S.

Wilson, Stuart D.

Yen, Tina W.

CORRESPONDING COUNTRIES OF THE AAES

Australia

Beecroft

Reeve, Thomas S.

Frankston

Serpell, Jonathan William

St Leonards

Sywak, Mark

Sydney

Delbridge, Leigh W.

Sidhu, Stan

Warooona

Edis, Anthony J.

Austria

Vienna

Niederle, Bruno

Belgium

Aalst

Van Slycke, Sam

China

Hong Kong

Lo, Chung-Yau

Croatia

Zagreb

Bura, Miljenko

GEOGRAPHICAL MEMBERSHIP DIRECTORY

France

Francheville

Peix, Jean-Louis

Lille Cedex

Carnaille, Bruno M.

Marseilles

Henry, Jean-Francois

Sebag, Frederic N.

Poitiers

Kraimps, Jean Louis

Strasbourg

Mutter, Didier

Vandoeuvre les Nancy

Brunaud, Laurent

Germany

Duisburg

Simon, Dietmar

Dusseldorf

Roeher, Hans-Dietrich

Essen

Walz, Martin K.

Halle

Dralle, Henning

Mainz

Musholt, Thomas J.

Rostock

Klar, Ernst

Ulm

Weber, Theresia

Greece

Athens

Linos, Dimitrios A.

India

Lucknow

Agarwal, Gaurav

Mishra, Saroj K.

Israel

Hertzlia

Schachter, Pinhas P.

Italy

Padova

Iacobone, Maurizio

Padua

Favia, Gennaro

Pisa

Miccoli, Paolo

Rome

Bellantone, Rocco

Lombardi, Celestino P.

Raffaelli, Marco

Japan

Joto-ku, Osaka

Imamura, Masayuki

Kobe

Miyauchi, Akira

Nagoya

Imai, Tsuneo

Oita

Noguchi, Shiro

Osaka

Shiba, Elichi

Tokyo

Masatoshi, Iihara

Obara, Takao

Takami, Hiroshi E.

GEOGRAPHICAL MEMBERSHIP DIRECTORY

Netherlands

Utrecht

Vriens, Menno R.

Norway

Bergen

Brauckhoff, Michael
Varhaug, Jan E.

Russian Federation

Saint Petersburg

Romanchishen, Anatoly F.

Saudi Arabia

Riyadh

Al Sobhi, Saif S.

South Korea

Seoul

Youn, Yeo-Kyu

Spain

Barcelona

Moreno Llorente, Pablo

Sevilla

Sanchez-Blanco, J.M.

Sweden

Linkoping

Gimm, Oliver

Stockholm

Hamberger, Bertil

Uppsala

Akerstrom, Goran
Skogseid, Britt

Taiwan

Taipei

Lee, Chen-Hsen
Tu, Shih Hsin

Thailand

Bangkok

Defechereux, Thierry

Turkey

Istanbul

Duren, Mete
Terzioglu, Tarik
Tezelman, Serdar T.

Ukraine

Kiev

Kvachenyuk, Andrey

United Kingdom

Devon

Pearse, A.G.E.

London

Frilling, Andrea

Oxfordshire

Dudley, Nicholas E.

2011 MEMBER CONTACT INFORMATION

IMPORTANT!

Please indicate any changes and be sure to include your current email address

Name: _____

Mailing Address: _____

Institution: _____

Birthdate: _____

Spouse: _____

Phone: _____

Fax: _____

Email: _____

SUBMIT TO *REGISTRATION DESK

or to AAES Headquarters via

Email: information@endocrinesurgery.org

OR

Fax: **(913) 273-9940**

IN MEMORIAM

Brown Dobyns

Chardon, OH

Timothy Harrison

Rumford, RI

Jeffreys Macfie

Greenville, SC

William Remine

Ponte Vedra Beach, FL

William Snyder, III

Dallas, TX

