Prevention of Fecal and Urinary Incontinence in Adults

An NIH State-of-the-Science Conference

Program and Abstracts

December 10–12, 2007

William H. Natcher Conference Center
National Institutes of Health
Bethesda, Maryland

Sponsored by

National Institute of Diabetes and Digestive and Kidney Diseases, NIH
Office of Medical Applications of Research, NIH

Cosponsored by

National Cancer Institute, NIH
National Institute of Child Health and Human Development, NIH
National Institute on Aging, NIH
National Institute of Nursing Research, NIH
Office of Research on Women's Health, NIH

The Agency for Healthcare Research and Quality provided additional support to the conference development.

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
National Institutes of Health

About the Program

The National Institutes of Health (NIH) Consensus Development Program has been organizing major conferences since 1977. The Program generates evidence-based consensus statements addressing controversial issues important to healthcare providers, policymakers, patients, researchers, and the general public. The NIH Consensus Development Program holds an average of three conferences a year. The Program is administered by the Office of Medical Applications of Research within the NIH Office of the Director. Typically, the conferences have one major NIH Institute or Center sponsor, with multiple cosponsoring agencies.

Topic Selection

NIH Consensus Development and State-of-the-Science Conference topics must satisfy the following criteria:

- Broad public health importance. The severity of the problem and the feasibility of interventions are key considerations.
- Controversy or unresolved issues that can be clarified, or a gap between current knowledge and practice that can be narrowed.
- An adequately defined base of scientific information from which to answer conference questions such that the outcome does not depend primarily on subjective judgments of panelists.

Conference Type

Two types of conferences fall under the purview of the NIH Consensus Development Program: State-of-the-Science Conferences and Consensus Development Conferences. Both conference types utilize the same structure and methodology; they differ only in the strength of the evidence surrounding the topic under consideration. When it appears that there is very strong evidence about a particular medical topic, but that the information is not in widespread clinical practice, a Consensus Development Conference is typically chosen to consolidate, solidify, and broadly disseminate strong evidence-based recommendations for general practice. Conversely, when the available evidence is weak or contradictory, or when a common practice is not supported by high-quality evidence, the State-of-the-Science label is chosen. This highlights what evidence about a topic is available, the directions future research should take, and alerts physicians that certain practices are not supported by good data.

Conference Process

Before the conference, a systematic evidence review on the chosen topic is performed by one of the Agency for Healthcare Research and Quality's Evidence-Based Practice Centers. This report is provided to the panel members approximately 6 weeks prior to the conference, and posted to the Consensus Development Program Web site once the conference begins, to serve as a foundation of high-quality evidence upon which the conference will build.

The conferences are held over 2 1/2 days. The first day and a half of the conference consist of plenary sessions in which invited expert speakers present information, followed by "town hall forums," in which open discussion occurs among the speakers, panelists, and the general public in attendance. The panel then develops its draft statement on the afternoon and evening of the second day, and presents it on the morning of the third day for audience commentary. The panel considers these comments in executive session and may revise their draft accordingly. The conference ends with a press briefing, during which reporters are invited to question the panelists about their findings.

Panelists

Each conference panel comprises 12–16 members who can give balanced, objective, and informed attention to the topic. Panel members:

- Must not be employees of the Department of Health and Human Services.
- Must not hold financial or career (research) interests in the conference topic.
- May be knowledgeable in the general topic under consideration, but must not have published about or have a publicly stated opinion on the topic.
- Represent a variety of perspectives, to include:
  - Practicing and academic health professionals
  - Biostatisticians and epidemiologists
  - Clinical trialists and researchers
  - Public representatives (ethicists, economists, attorneys, etc.)

In addition, the panel as a whole should appropriately reflect racial and ethnic diversity. Panel members are not paid a fee or honorarium for their efforts. They are, however, reimbursed for travel expenses related to their participation in the conference.

Speakers

The conferences typically feature approximately 21 speakers; 3 present the information found in the Evidence-Based Practice Center's systematic review of the literature. The other 18 are experts in the topic at hand, have likely published on the topic, and may have strong opinions or beliefs. Where multiple viewpoints on a topic exist, every effort is made to include speakers who address all sides of the issue.

Conference Statements

The panel's draft report is released online late in the conference's third and final day. The final report is released approximately 6 weeks later. During the intervening period, the panel may edit their statement for clarity and correct any factual errors that might be discovered. No substantive changes to the panel's findings are made during this period.

Each Consensus Development or State-of-the-Science Conference Statement reflects an independent panel's assessment of the medical knowledge available at the time the statement was written; as such, it provides a "snapshot in time" of the state of knowledge on the conference topic. It is not a policy statement of the NIH or the Federal Government.

Dissemination

Consensus Development and State-of-the-Science Conference Statements have robust dissemination:

- Continuing Medical Education credits are available during and after the conference.
- A press conference is held the last day of the conference to assist journalists in preparing news stories on the conference findings.
- The statement is published online at http://consensus.nih.gov.
- Print copies are mailed to a wide variety of targeted audiences and are available at no charge through a clearinghouse.

The conference statement is published in a major peer-reviewed journal.

Contact Us

For conference schedules, past statements and evidence reports, please contact us:

NIH Consensus Development Program
Information Center
P.O. Box 2577
Kensington, MD 20891

1-888-NIH-CONSENSUS (888-644-2667)
http://consensus.nih.gov

General Information

CME

The National Institutes of Health/Foundation for Advanced Education in the Sciences (NIH/FAES) is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

The NIH/FAES designates this educational activity for a maximum of 13.25 *AMA PRA Category 1 Credits*.™ Physicians should claim only credit that is commensurate with the extent of their participation in the activity.

Your participant packet includes a CME evaluation form, which should be completed and returned either to the conference registration desk or by mail to claim credits.

Financial Disclosure

Each speaker presenting at this conference has been asked to disclose any financial interests or other relationships pertaining to this subject area. Please refer to the material in your participant packet for details.

Panel members signed a confirmation that they have no financial or other conflicts of interest pertaining to the topic under consideration.

Videocast

Live and archived videocasts may be accessed at http://videocast.nih.gov. Archived videocast will be available approximately 1 week after the conference.

Dining

The dining center in the Natcher Conference Center is located on the main level, one floor above the auditorium. It is open from 6:30 a.m. to 2:30 p.m., serving hot breakfast and lunch, sandwiches and salads, and snack items. An additional cafeteria is available from 7:00 a.m. to 3:30 p.m., in Building 38A, level B1, across the street from the main entrance to the Natcher Conference Center.

Message Service

The telephone number for the message center at the Natcher Conference Center is 301–594–7302.

Online Content

All materials emanating from the NIH Consensus Development Program are available at http://consensus.nih.gov.

Contents

What Are the Risk Factors for Fecal and Urinary Incontinence?

What Can Be Done To Prevent Fecal and Urinary Incontinence?

And

What Are the Strategies To Improve the Identification of Persons at Risk and Patients Who Have Fecal and Urinary Incontinence?

Background

Fecal incontinence (FI) and urinary incontinence (UI)—the inability to control bowel movements or urination, respectively—are conditions with ramifications that extend well beyond their physical manifestations. Many people find themselves withdrawing from their social lives and attempting to hide the problem from their families, friends, and even their doctors. The embarrassing nature of these conditions poses a significant barrier to seeking professional treatment, resulting in a large number of unreported, untreated individuals. Therefore, it is difficult to determine the accurate prevalence of these conditions, as well as any associated medical history trends. Incontinence is more likely to affect the aging population, although it is not considered a normal consequence of aging. As baby boomers approach their 60s, the incidence and public health burden of incontinence are likely to increase.

FI is a serious and embarrassing problem that affects up to 5% of the general population and up to 39% of nursing home residents. It affects people of all ages, but it is more common in women and the elderly. Bowel function is controlled by three factors: rectal sensation, rectal storage capacity, and anal sphincter pressure. If any of these are compromised, FI can occur. This condition can have many causes, including constipation, diarrhea, complicated childbirth, muscular or nerve damage, reduced storage capacity due to scarring or irritation, or pelvic dysfunction.

Although UI can affect people at all stages of life, it has been estimated that UI affects 38% of women and 17% of men 60 years of age and older. UI can occur if muscles in the wall of the bladder suddenly contract or if muscles surrounding the urethra suddenly relax. Women who have undergone childbirth are the most common at-risk population for UI. Pregnancy and delivery can weaken pelvic muscles, and reduced levels of the hormone estrogen following menopause can cause reduced muscle tone around the urethra, increasing the chance of leakage. Additionally, neurologic injury, birth defects, strokes, multiple sclerosis, and physical problems associated with aging have been reported to contribute.

Because incontinence is likely widely underdiagnosed and underreported, it has been difficult to identify both at-risk and affected populations. Also, because the biological mechanisms that cause both FI and UI are not well understood, it has been difficult to develop robust prevention and management strategies. Toward that end, the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) and the Office of Medical Applications of Research (OMAR) of the National Institutes of Health (NIH) will convene a State-of-the-Science Conference from December 10 to 12, 2007, to assess the available scientific evidence relevant to the following questions:

- What are the prevalence, incidence, and natural history of fecal and urinary incontinence in the community and long-term care settings?
- What is the burden of illness and impact of fecal and urinary incontinence on the individual and society?
- What are the risk factors for fecal and urinary incontinence?
- What can be done to prevent fecal and urinary incontinence?

- What are the strategies to improve the identification of persons at risk and patients who have fecal and urinary incontinence?

- What are the research priorities in reducing the burden of illness in these conditions?

Agenda

Monday, December 10, 2007

8:30 a.m. Opening Remarks
Stephen James, M.D.
Director, Division of Digestive Diseases and Nutrition
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health

8:40 a.m. Charge to Panel
Barnett S. Kramer, M.D., M.P.H.
Director
Office of Medical Applications of Research
Office of the Director
National Institutes of Health

8:50 a.m. Conference Overview and Panel Activities
C. Seth Landefeld, M.D.
Panel and Conference Chairperson
Chief, Division of Geriatrics
Director, Center on Aging
University of California, San Francisco
Associate Chief of Staff
San Francisco Veterans Affairs Medical Center
San Francisco, California

What Are the Prevalence, Incidence, and Natural History of Fecal and Urinary Incontinence in the Community and Long-Term Care Settings?

9:00 a.m. Definition and Epidemiology of Fecal and Urinary Incontinence
Kathryn L. Burgio, Ph.D.
Professor of Medicine
University of Alabama at Birmingham
Director
University of Alabama at Birmingham Continence Program
Associate Director for Research
Birmingham/Atlanta Geriatric Research, Education, and Clinical Center
Department of Veterans Affairs Medical Center
University of Alabama at Birmingham

9:15 a.m. Pathophysiology of Fecal Incontinence
Adil E. Bharucha, M.D., M.B.B.S.
Professor of Medicine
Division of Gastroenterology and Hepatology
Clinical Enteric Neuroscience Translational and Epidemiological Research Program
Mayo Clinic College of Medicine

What Are the Prevalence, Incidence, and Natural History of Fecal and Urinary Incontinence in the Community and Long-Term Care Settings? (continued)

9:35 a.m. Pathophysiology of Urinary Incontinence
Alan J. Wein, M.D., Ph.D. (Hon.)
Professor and Chair
Division of Urology
University of Pennsylvania School of Medicine and Health System
Chief of Urology, Hospital of the University of Pennsylvania
University of Pennsylvania Health System

9:50 a.m. Discussion

Participants with questions or comments for the speakers should proceed to the microphones and wait to be recognized by the panel chair. Please state your name and affiliation. Questions and comments not heard before the close of the discussion period may be submitted on the computers in the registration area. Please be aware that all statements made at the microphone or submitted later are in the public domain.

What Is the Burden of Illness and Impact of Fecal and Urinary Incontinence on the Individual and Society?

10:15 a.m. Economic Impact of Fecal and Urinary Incontinence on the Individual and Society—Direct and Indirect Costs
Philip B. Miner, Jr., M.D.
President and Medical Director
Oklahoma Foundation for Digestive Research
Clinical Professor of Medicine
University of Oklahoma

10:30 a.m. Impact of Fecal and Urinary Incontinence on the Health Consumer: Barriers on Diagnosis and Treatment—A Patient Perspective
Nancy J. Norton
President
International Foundation for Functional Gastrointestinal Disorders

10:45 a.m. Formal and Informal Caregiving Burden of Fecal and Urinary Incontinence
Sandra Engberg, Ph.D., R.N., C.R.N.P.
Chair
Health Promotion and Development
School of Nursing
University of Pittsburgh

11:05 a.m. Discussion

Monday, December 10, 2007 (continued)

What Is the Burden of Illness and Impact of Fecal and Urinary Incontinence on the Individual and Society? (continued)

11:30 a.m. Quality of Life for Patients With Fecal Incontinence
Liliana Bordeianou, M.D.
Instructor in Surgery
Department of Surgery
Harvard Medical School
Massachusetts General Hospital

11:50 a.m. Quality of Life for Patients With Urinary Incontinence
Jennifer T. Anger, M.D., M.P.H.
Assistant Professor of Urology
Department of Urology
University of California, Los Angeles

12:10 p.m. Discussion

12:30 p.m. Lunch
Panel Executive Session

What Are the Risk Factors for Fecal and Urinary Incontinence?

1:30 p.m. Evidence-Based Practice Center Presentation I: Prevalence, Incidence, and Risk Factors for Fecal Incontinence
Donna Z. Bliss, Ph.D., R.N., F.A.A.N., F.S.G.A.
Professor
School of Nursing
University of Minnesota

1:50 p.m. Evidence-Based Practice Center Presentation II: Prevalence, Incidence, and Risk Factors for Urinary Incontinence
Jean F. Wyman, Ph.D., R.N., F.A.A.N., F.S.G.A.
Professor
School of Nursing
University of Minnesota

2:10 p.m. Impact of Diabetes and Obesity on the Development of Fecal and Urinary Incontinence
Leslee L. Subak, M.D.
Attending Physician
Women's Health Clinical Research Center
University of California, San Francisco

2:30 p.m. Discussion

Monday, December 10, 2007 (continued)

What Are the Risk Factors for Fecal and Urinary Incontinence? (continued)

3:00 p.m. Do Pregnancy, Type of Delivery, and Postpartum State Increase the Risk for Development of Fecal and Urinary Incontinence?
Holly E. Richter, M.D., Ph.D.
Professor
Division Director, Women's Pelvic Medicine and Reconstructive Surgery
University of Alabama at Birmingham

3:20 p.m. Effect of Hormones on Fecal and Urinary Incontinence and Pelvic Organ Prolapse
Ingrid Nygaard, M.D., M.S.
Professor
Urogynecology and Reconstructive Surgery
Department of Obstetrics and Gynecology
University of Utah

3:40 p.m. Impact of Chronic Gastrointestinal Conditions, Such as Irritable Bowel Syndrome, Inflammatory Bowel Disease, and Constipation as Risk Factors for Fecal Incontinence
William E. Whitehead, Ph.D.
Professor of Medicine and Adjunct Professor of Obstetrics and Gynecology
Co-Director
Center for Functional Gastrointestinal and Motility Disorders
University of North Carolina at Chapel Hill

4:00 p.m. Risk Factors for the Development of Fecal and Urinary Incontinence—Age, Frailty, Dementia, Functional Impairment, and Institutionalization
John F. Schnelle, Ph.D.
Director
Vanderbilt Center for Quality Aging
Professor of Medicine
Vanderbilt University School of Medicine

4:20 p.m. Discussion

5:15 p.m. Adjournment

Tuesday, December 11, 2007

What Are the Risk Factors for Fecal and Urinary Incontinence? (continued)

8:30 a.m. Surgical Complications Including Prostatectomy and Other Urological Procedures
Patricia S. Goode, M.D., M.S.N.
Gwen McWhorter Professor of Geriatric Medicine
Medical Director
University of Alabama at Birmingham Continence Clinic
University of Alabama at Birmingham
Associate Director for Clinical Programs
Birmingham/Atlanta Geriatric Research, Education, and Clinical Center
Birmingham Veterans Affairs Medical Center
University of Alabama at Birmingham

8:50 a.m. Risk Factors for the Development of Fecal and Urinary Incontinence Following Anorectal Surgery, Colorectal Surgery, and Radiation Therapy for Colorectal Cancer
Ann C. Lowry, M.D., F.A.C.S., FASCRS
Adjunct Professor
Division of Colon and Rectal Surgery
Department of Surgery
University of Minnesota Medical School

9:10 a.m. Iatrogenic Disorders, Drug Side Effects, and the Development of Urinary and Fecal Incontinence
Alan J. Wein, M.D., Ph.D. (Hon.)
Professor and Chair
Division of Urology
University of Pennsylvania School of Medicine and Health System
Chief of Urology, Hospital of the University of Pennsylvania
University of Pennsylvania Health System

9:25 a.m. Discussion

9:55 a.m. Impact of Neurological Disorders, Such as Stroke, Spinal Cord Injuries, and Other Neurological Conditions on the Development of Fecal and Urinary Incontinence
Arnold Wald, M.D.
Professor of Medicine
Section of Gastroenterology and Hepatology
University of Wisconsin School of Medicine and Public Health

Tuesday, December 11, 2007 (continued)

What Are the Risk Factors for Fecal and Urinary Incontinence? (continued)

10:15 a.m. Impact of Depression and Other Psychiatric Conditions on the Development of Fecal and Urinary Incontinence
William D. Steers, M.D.
Hovey Dabney Professor of Urology
Chair
Department of Urology
University of Virginia

10:35 a.m. Discussion

What Can Be Done To Prevent Fecal and Urinary Incontinence?

And

What Are the Strategies To Improve the Identification of Persons at Risk and Patients Who Have Fecal and Urinary Incontinence?

10:55 a.m. Evidence-Based Practice Center Presentation III: Prevention, Screening, and Interventions for Urinary Incontinence and Fecal Incontinence
Robert L. Kane, M.D.
Professor
University of Minnesota

11:25 a.m. Impact of Exercise, Diet, Lifestyle, and Smoking in the Setting of Continence
Kathryn L. Burgio, Ph.D.
Professor of Medicine
University of Alabama at Birmingham
Director
University of Alabama at Birmingham Continence Program
Associate Director for Research
Birmingham/Atlanta Geriatric Research, Education, and Clinical Center
Department of Veterans Affairs Medical Center
University of Alabama at Birmingham

11:40 a.m. Prevention of Fecal and Urinary Incontinence and the Strategies To Improve the Identification of Persons at Risk
Diane K. Newman, R.N.C., M.S.N., C.R.N.P., F.A.A.N.
Co-Director
Penn Center for Continence and Pelvic Health
Division of Urology
University of Pennsylvania Medical Center

12:05 p.m. Discussion

12:40 p.m. Adjournment

Wednesday, December 12, 2007

9:00 a.m. Presentation of the draft State-of-the-Science Statement

9:30 a.m. Public Discussion

The panel chair will call for questions and comments from the audience on the draft statement, beginning with the introduction and continuing through each subsequent section in turn. Please confine your comments to the section under discussion. The chair will use discretion in proceeding to subsequent sections so that comments on the entire statement may be heard during the time allotted. Comments cannot be accepted after 11:30 a.m.

11:00 a.m. Panel Meets in the Executive Session

Panel meets in executive session to review public comments. Conference participants are welcome to return to the main auditorium to attend the press conference at 2:00 p.m.; however, only members of the media are permitted to ask questions during the press conference.

2:00 p.m. Press Conference

3:00 p.m. Adjournment

The panel's draft statement will be posted to http://consensus.nih.gov as soon as possible after the close of proceedings, and the final statement will be posted 4 to 6 weeks later.

Panel

Panel Chair: C. Seth Landefeld, M.D.
Panel and Conference Chairperson
Chief, Division of Geriatrics
Director, Center on Aging
University of California, San Francisco
Associate Chief of Staff
San Francisco Veterans Affairs Medical Center
San Francisco, California

Barbara J. Bowers, Ph.D., R.N., G.N.P., F.A.A.N.
Associate Dean for Research and Sponsored Programs
Helen Denne Schulte Professor
Director, Center for Excellence in Long-Term Care
University of Wisconsin–Madison School of Nursing
Madison, Wisconsin

Andrew D. Feld, M.D., J.D., F.A.C.G., A.G.A.F.
Chief, Central Division of Gastroenterology
Group Health Cooperative
Clinical Associate Professor of Medicine
University of Washington
Seattle, Washington
Rockwood Clinic
Spokane, Washington

Katherine E. Hartmann, M.D., Ph.D.
Deputy Director
Institute for Medicine and Public Health
Vice Chair, Obstetrics and Gynecology
Director, Women's Health Research at Vanderbilt
Vanderbilt Institute for Medicine and Public Health
Nashville, Tennessee

Eileen Hoffman, M.D., F.A.C.P.
Clinical Associate Professor of Medicine
New York University School of Medicine
New York, New York

Melvin J. Ingber, Ph.D.
Principal Scientist
Health Care Financing and Payment
RTI International
Washington, DC

Joseph T. King, Jr., M.D., M.S.C.E.
Chief of Neurosurgery
Veterans Affairs Connecticut Healthcare System
Associate Professor of Neurosurgery
Director of Neurosurgical Outcomes Research
Yale University School of Medicine
New Haven, Connecticut

William Scott McDougal, M.D.
Chief, Department of Urology
Massachusetts General Hospital
Walter S. Kerr, Jr. Professor of Urology
Harvard Medical School
Boston, Massachusetts

Heidi Nelson, M.D.
Fred C. Andersen Professor
Professor of Surgery
Mayo Medical School
Rochester, Minnesota

Endel John Orav, Ph.D.
Associate Professor
Department of Medicine (Biostatistics)
Division of General Internal Medicine
Brigham and Women's Hospital
Boston, Massachusetts

Michael Pignone, M.D., M.P.H.
Associate Professor of Medicine
Chief, Division of General Internal Medicine
University of North Carolina Department of Medicine
University of North Carolina Hospital
Chapel Hill, North Carolina

Lisa H. Richardson
Consumer Representative
National Chairperson of the Board Emeritus
Crohn's and Colitis Foundation of America, Inc.
Houston, Texas

Robert M. Rohrbaugh, M.D.
Director of Medical Studies and Associate Professor
Department of Psychiatry
Associate Chief of Psychiatric Service
Veterans Affairs Connecticut Healthcare System
West Haven Campus
West Haven, Connecticut

Hilary C. Siebens, M.D.
Professor of Clinical Physical Medicine and Rehabilitation
University of Virginia, Charlottesville
Seal Beach, California

Bruce J. Trock, Ph.D.
Director, Division of Epidemiology
Brady Urological Institute
Associate Professor of Urology, Epidemiology, Oncology, and Environmental Health Sciences
The Johns Hopkins University School of Medicine
Baltimore, Maryland

Speakers

Jennifer T. Anger, M.D., M.P.H.
Assistant Professor of Urology
Department of Urology
University of California, Los Angeles
Santa Monica, California

Adil E. Bharucha, M.D., M.B.B.S.
Professor of Medicine
Division of Gastroenterology and Hepatology
Clinical Enteric Neuroscience Translational and Epidemiological Research Program
Mayo Clinic College of Medicine
Rochester, Minnesota

Donna Z. Bliss, Ph.D., R.N., F.A.A.N., F.S.G.A.
Professor
School of Nursing
University of Minnesota
Minneapolis, Minnesota

Liliana Bordeianou, M.D.
Instructor in Surgery
Department of Surgery
Harvard Medical School
Massachusetts General Hospital
Boston, Massachusetts

Kathryn L. Burgio, Ph.D.
Professor of Medicine
University of Alabama at Birmingham
Director, University of Alabama at Birmingham Continence Program
Associate Director for Research
Birmingham/Atlanta Geriatric Research, Education, and Clinical Center
Department of Veterans Affairs Medical Center
University of Alabama at Birmingham
Birmingham, Alabama

Sandra Engberg, Ph.D., R.N., C.R.N.P.
Chair
Health Promotion and Development
School of Nursing
University of Pittsburgh
Pittsburgh, Pennsylvania

Patricia S. Goode, M.D., M.S.N.
Gwen McWhorter Professor of Geriatric Medicine
Medical Director
University of Alabama at Birmingham Continence Clinic
University of Alabama at Birmingham
Associate Director for Clinical Programs
Birmingham/Atlanta Geriatric Research, Education, and Clinical Center
Birmingham Veterans Affairs Medical Center
University of Alabama at Birmingham
Birmingham, Alabama

Robert L. Kane, M.D.
Professor
University of Minnesota
Minneapolis, Minnesota

Ann C. Lowry, M.D., F.A.C.S., FASCRS
Adjunct Professor
Division of Colon and Rectal Surgery
Department of Surgery
University of Minnesota Medical School
Minneapolis, Minnesota

Philip B. Miner, Jr., M.D.
President and Medical Director
Oklahoma Foundation for Digestive Research
Clinical Professor of Medicine
University of Oklahoma
Oklahoma City, Oklahoma

Diane K. Newman, R.N.C., M.S.N., C.R.N.P., F.A.A.N.
Co-Director
Penn Center for Continence and Pelvic Health
Division of Urology
University of Pennsylvania Medical Center
Philadelphia, Pennsylvania

Nancy J. Norton
President
International Foundation for Functional Gastrointestinal Disorders
Milwaukee, Wisconsin

Ingrid Nygaard, M.D., M.S.
Professor
Urogynecology and Reconstructive Surgery
Department of Obstetrics and Gynecology
University of Utah
Salt Lake City, Utah

Holly E. Richter, M.D., Ph.D.
Professor
Division Director
Women's Pelvic Medicine and Reconstructive Surgery
University of Alabama at Birmingham
Birmingham, Alabama

John F. Schnelle, Ph.D.
Director
Vanderbilt Center for Quality Aging
Professor of Medicine
Vanderbilt University School of Medicine
Nashville, Tennessee

William D. Steers, M.D.
Hovey Dabney Professor of Urology
Chair
Department of Urology
University of Virginia
Charlottesville, Virginia

Leslee L. Subak, M.D.
Attending Physician
Women's Health Clinical Research Center
University of California, San Francisco
San Francisco, California

Arnold Wald, M.D.
Professor of Medicine
Section of Gastroenterology and Hepatology
University of Wisconsin School of Medicine and Public Health
Madison, Wisconsin

Alan J. Wein, M.D., Ph.D. (Hon.)
Professor and Chair
Division of Urology
University of Pennsylvania School of Medicine and Health System
Chief of Urology, Hospital of the University of Pennsylvania
University of Pennsylvania Health System
Philadelphia, Pennsylvania

William E. Whitehead, Ph.D.
Professor of Medicine and Adjunct Professor of Obstetrics and Gynecology
Co-Director
Center for Functional Gastrointestinal and Motility Disorders
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina

Jean F. Wyman, Ph.D., R.N., F.A.A.N., F.S.G.A
Professor
School of Nursing
University of Minnesota
Minneapolis, Minnesota

Planning Committee

Planning Chair: Frank A. Hamilton, M.D., M.P.H.
Chief
Digestive Diseases Program
Division of Digestive Diseases and Nutrition
National Institute of Diabetes and Digestive and Kidney Diseases
National Institutes of Health
Bethesda, Maryland

David Atkins, M.D., M.P.H.
Chief Medical Officer
Center for Outcomes and Evidence
Agency for Healthcare Research and Quality
Rockville, Maryland

Alexis D. Bakos, Ph.D., M.P.H., R.N.C.
Program Director
Office of Extramural Programs
National Institute of Nursing Research
National Institutes of Health
Bethesda, Maryland

Lisa Begg, Dr.P.H., R.N.
Director of Research Programs
Office of Research on Women's Health
Office of the Director
National Institutes of Health
Bethesda, Maryland

Beth A. Collins Sharp, Ph.D., R.N.
Director
Evidence-Based Practice Centers Program
Center for Outcomes and Evidence
Agency for Healthcare Research and Quality
Rockville, Maryland

Catherine E. DuBeau, M.D.
Associate Professor of Medicine
Section of Geriatrics
University of Chicago
Chicago, Illinois

Patricia S. Goode, M.D., M.S.N.
Gwen McWhorter Professor of Geriatric Medicine
Medical Director
University of Alabama at Birmingham Continence Clinic
University of Alabama at Birmingham
Associate Director for Clinical Programs
Birmingham/Atlanta Geriatric Research, Education, and Clinical Center
Birmingham Veterans Affairs Medical Center
University of Alabama at Birmingham
Birmingham, Alabama

Joseph Kelaghan, M.D., M.P.H.
Program Director
Community Oncology and Prevention Trials Research Group
Division of Cancer Prevention
National Cancer Institute
National Institutes of Health
Bethesda, Maryland

Barnett S. Kramer, M.D., M.P.H.
Director
Office of Medical Applications of Research
Office of the Director
National Institutes of Health
Bethesda, Maryland

C. Seth Landefeld, M.D.
Panel and Conference Chairperson
Chief, Division of Geriatrics
Director, Center on Aging
University of California, San Francisco
Associate Chief of Staff
San Francisco Veterans Affairs Medical Center
San Francisco, California

Ann C. Lowry, M.D., F.A.C.S., FASCRS
Adjunct Professor
Division of Colon and Rectal Surgery
Department of Surgery
University of Minnesota Medical School
Minneapolis, Minnesota

Kelli K. Marciel, M.A.
Communications Director
Office of Medical Applications of Research
Office of the Director
National Institutes of Health
Bethesda, Maryland

Ernestine Murray, R.N., M.A.S.
Captain
U.S. Public Health Service
Senior Health Policy Analyst
Senior Advisor on Tobacco Use
Center for Outcomes and Evidence
Agency for Healthcare Research and Quality
Rockville, Maryland

Lata S. Nerurkar, Ph.D.
Senior Advisor for the Consensus Development Program
Office of Medical Applications of Research
Office of the Director
National Institutes of Health
Bethesda, Maryland

Diane K. Newman, R.N.C., M.S.N., C.R.N.P., F.A.A.N.
Co-Director
Penn Center for Continence and Pelvic Health
Division of Urology
University of Pennsylvania Medical Center
Philadelphia, Pennsylvania

Nancy J. Norton
President
International Foundation for Functional Gastrointestinal Disorders
Milwaukee, Wisconsin

Susan Rossi, Ph.D., M.P.H.
Deputy Director
Office of Medical Applications of Research
Office of the Director
National Institutes of Health
Bethesda, Maryland

Anne M. Weber, M.D., M.S.
Program Officer
Pelvic Floor Disorders Program
Contraception and Reproductive Health Branch
Center for Population Research
National Institute of Child Health and Human Development
National Institutes of Health
Bethesda, Maryland

William E. Whitehead, Ph.D.
Professor of Medicine and Adjunct Professor of Obstetrics and Gynecology
Co-Director
Center for Functional Gastrointestinal and Motility Disorders
University of North Carolina at Chapel Hill
Chapel Hill, North Carolina

A. Roger Wiederhorn, M.D., D.M.Sc.
Medical Officer
Division of Reproductive and Urologic Products
Office of Drug Evaluation III
Center for Drug Evaluation and Research
U.S. Food and Drug Administration
Silver Spring, Maryland

Rosemary Yancik, Ph.D.
Health Scientist Administrator
Geriatrics and Clinical Gerontology Program
National Institute on Aging
National Institutes of Health
Bethesda, Maryland

Abstracts

The abstracts are designed to inform the panel and conference participants, as well as to serve as a reference document for any other interested parties. We would like to thank sincerely the honored speakers for preparing and presenting their findings on this important topic.

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Please note that where multiple authors are listed on an abstract, the underline denotes the presenting author.

Definition and Epidemiology of Fecal and Urinary Incontinence

Kathryn L. Burgio, Ph.D.

Epidemiology is the study of the distribution and determinants of a disease or condition and includes its prevalence, incidence, and risk factors or correlates. Understanding the epidemiology of fecal incontinence (FI) and urinary incontinence (UI) is important to the search for potential causal factors, as well as protective factors, and the development of approaches to the primary and secondary prevention of these conditions.

Prevalence estimates vary widely for both FI and UI. This variation has been attributed to differences in definition of the conditions, data collection methods, populations selected, and sampling methods. Definitions used in epidemiological studies encompass a wide range of severity by incorporating various criteria based on frequency, type, quantity, duration, or symptom bother. Data collection has been conducted by in-person interview, telephone interview, or anonymous questionnaire. Each method carries the potential for under- or overreporting of incontinence and thus has the potential to influence prevalence estimates.

Fecal Incontinence

FI is usually defined as the involuntary loss of solid or liquid stool. Anal incontinence is a broader concept that includes loss of solid or liquid stool, flatus, or mucus. The two terms are sometimes used interchangeably, and some surveys have included the loss of flatus in the definition of FI.

Estimates of the prevalence of FI in community-based populations range from 0.4% to 18%.[1,2] Considering those studies that used the most unbiased methods, such as anonymous, self-administered questionnaires, estimates range from 11% to 15%.[3–5] Most surveys report increased prevalence of FI with age.[6,7] Population-based studies of older adults report prevalences of FI ranging from 3.0% to 32%.[7–9] Several studies have reported the FI is more common among women compared to men, but the literature is mixed on the issue of gender.

In nursing homes, prevalences range from 45% to 55%.[10,11] This range may be explained partly by FI being one of the most common reasons for nursing home admission.[4,8] FI in nursing homes has been associated with functional impairment, dementia, and sensory impairments—all factors that place a person at risk for institutionalization.

Studies of the incidence of FI in the general population are rare. One study reported a cumulative 5-year incidence of 8.5% among older adults.[7] Most other incidence studies have been conducted in special populations, such as patients undergoing medical or surgical procedures and postpartum women. Studies of FI in the postpartum period have identified anal sphincter injury during childbirth as a major cause of FI in young healthy women.[12,13]

Urinary Incontinence

UI is the involuntary loss of urine. A large literature exists on the prevalence of UI, primarily in women and in Caucasian populations. UI is uniformly more common among women compared to men by a ratio of 2:1. In women, prevalences based on liberal definitions range from 5% to

69%,[2] with most being in the 25% to 45% range.[14–16] In most studies that include a wide age range, the prevalence of UI increases progressively up to middle age, levels off until about age 70, and then increases steadily among older adults.[16,17]

Prevalence of UI is higher among women living in long-term-care settings. Prevalences range from 23% to 72%, with a median of about 55%.[18,19] Based on studies showing that presence of UI increases the risk of institutionalization, it has been assumed that this higher prevalence is due to selection. In addition, cognitive, functional, and sensory impairments that contribute to UI are also risk factors for placement in nursing homes.

Higher prevalences are also found in studies of pregnant women, in whom estimates range from 32% to 64% for any UI.[20,21] Prevalence tends to be low in the first trimester, greater in the second trimester, and even higher in the third trimester. Established risk factors for UI in women include age, parity, obesity, and cognitive and functional impairment. Other possible risk factors that have been investigated include race, fetal and obstetric factors, menopause, hormone therapy, hysterectomy, smoking, and family history.

Fewer studies have been published describing the prevalence of UI in men. In the general population of men, prevalence of UI ranges from 1% to 39%. UI increases steadily with age and ranges from 11% to 34% in older men.[22] As in women, prevalence of UI is higher among men in long-term-care settings.

Men undergoing prostatectomy are at particular risk for UI. Incontinence tends to be most severe immediately after surgery and to improve over time. Transurethral resection of the prostate (TURP) is associated with a fairly low incidence of UI (approximately 1%). However, radical prostatectomy carries a much higher risk of UI, with prevalences based on patient self-report ranging from 8% to 56% at 1 year following surgery.[23–25]

There are fewer studies of the incidence of UI. In community-dwelling women, 1-year incidence ranges from 1% to 11.1% for women under 60 years of age [26,27] and from 5% to 29% for those over 60 years.[7,28] Studies of the incidence of UI in men are rare. In older men, the 1-year incidence of UI ranges from 6.3% to 16.9% and is higher in the older age groups.[7,28]

Summary

UI and FI are prevalent conditions that affect men and women of all ages. Epidemiological data provide evidence for several risk factors, some of which are modifiable, and identify at-risk populations of men and women who could potentially benefit from prevention.

References

1. Macmillan AK, Merrie AE, Marshall RJ, Parry BR. The prevalence of fecal incontinence in community-dwelling adults: a systematic review of the literature. *Dis Colon Rectum.* 2004; 47:1341–1349.

2. Hunskaar S, Burgio KL, Clark A, et al. Epidemiology of urinary and faecal incontinence and pelvic organ prolapse. In Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence, 3rd International Consultation on Incontinence.* Health Publications Ltd., 2005:255–312.

3. Johanson JF, Lafferty J. Epidemiology of fecal incontinence: The silent affliction. *Am J Gastroenterol.* 1996; 91:33–36.

4. Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors: an underdiagnosed problem in the Australian community? *Med J Aust.* 2002;176:54–57.

5. Lam TCF, Kennedy ML, Chen FC, Lubowski DZ, Talley NJ. Prevalence of faecal incontinence: obstetric and constipation-related risk factors; a population-based study. *Colorectal Dis.* 1999;1:197–203.

6. Teunissen TAM, Lagro-Janssen ALM, van den Bosch WJHM, van den Hoogen HJM. Prevalence of urinary, fecal and double incontinence in the elderly living at home. *Int Urogynecol J.* 2004;15:10–13.

7. Ostbye T, Seim A, Krause KM, et al. A 10-year follow-up of urinary and fecal incontinence among the oldest old in the community: The Canadian Study of Health and Aging. *Can J Aging.* 2004;23:319–331.

8. Edwards NI, Jones D. The prevalence of faecal incontinence in older people living at home. *Age and Ageing.* 2001;30:503–507.

9. Goode PS, Burgio KL, Halli AD, et al. Prevalence and correlates of fecal incontinence in community-dwelling older adults. *J Am Geriatr Soc.* 2005;53:629–635.

10. Borrie MJ, Davidson HA. Incontinence in institutions: costs and contributing factors. *Can Med Assoc J.* 1992;147:322–328.

11. Nelson R, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes: prevalence and associations. *Dis Colon Rectum.* 1998;41:1226–1229.

12. Oberwalder M, Conner J, Wexner SD. Meta-analysis to determine the incidence of obstetric anal sphincter damage. *Br J Surg.* 2003;90:1333–1337.

13. Borello-France D, Burgio KL, Richter HE, et al. Fecal and urinary incontinence in primiparous women. *Obstet Gynecol.* 2006;108:863–872.

14. Thomas TM, Plymat KR, Blannin J, Meade TW. Prevalence of urinary incontinence. *Br Med J.* 1980;281:1243–1245.

15. Yarnell JW, Voyle GJ, Richards CJ, Stephenson TP. The prevalence and severity of urinary incontinence in women. *J Epidemiol Community Health.* 1981;35:71–74.

16. Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. A community-based epidemiological survey of female urinary incontinence: the Norwegian EPINCONT study. *J Clin Epidemiol.* 2000;53:1150–1157.

17. Hunskaar S, Lose G, Sykes D, Voss S. The prevalence of urinary incontinence in women in four European countries. *BJU Int.* 2004;93:324–330.

18. Palmer MH, German PS, Ouslander JG. Risk factors for urinary incontinence one year after nursing home admission. *Res Nurs Health. 1*991;14:405–412.

19. Sgadari A, Topinkovaa E, Bjornson J, Bernabei R. Urinary incontinence in nursing home residents: a cross-national comparison. *Age Aging*. 1997;26:49–54

20. Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol*. 1992;79:945–949.

21. Chiarelli P, Campbell E. Incontinence during pregnancy: prevalence and opportunities for continence promotion. *Aust N Z J Obstet Gynecol.* 1997;237:66–73.

22. Thom D. Variation in estimates of urinary incontinence prevalence in the community: effects of differences in definition, population characteristics and study type. *Am Geriatr Soc.* 1998; 46:473–480.

23. Fowler FJ, Barry MJ, Lu-Yao G, Roman MA, Wasson J, Wennberg JE. Patient-reported complications and follow-up treatment after radical prostatectomy. *Urology. 1993;*42:622–629.

24. Stanford JL, Feng Z, Hamilton AS, et al. Urinary and sexual function after radical prostatectomy for clinically localized prostate cancer: the Prostate Cancer Outcomes Study. *JAMA.* 2000;283:354–360.

25. Bishoff JT, Motley G, Optenberg SA, et al. Incidence of fecal and urinary incontinence following radical perineal and retropubic prostatectomy in a national population. *J Urol.* 1998;60:454–458.

26. Holtedahl K, Hunskaar S. Prevalence, 1-year incidence and factors associated with urinary incontinence: a population based study of women 50-74 years of age in primary care. *Maturitas.* 1998;28:205–211.

27. Waetjen LE, Liao S, Johnson WO, et al. Factors associated with prevalent and incident urinary incontinence in a cohort of midlife women: a longitudinal analysis of data. *Am J Epidemiol.* 2007;165:309–318.

28. Herzog AR, Diokno AC, Brown MB, Normolle DP, Brock BM. Two-year incidence, remission, and change patterns of urinary incontinence in noninstitutionalized older adults. *J Gerontol.* 1990;45:M67–M64.

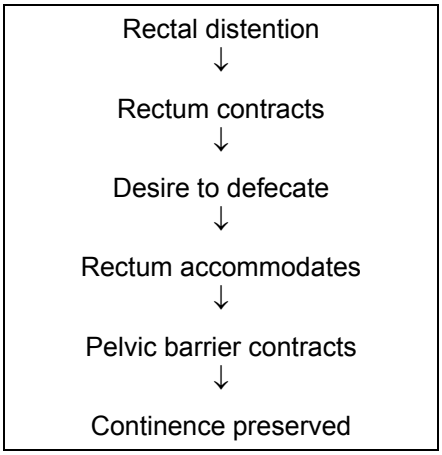
Pathophysiology of Fecal Incontinence

Adil E. Bharucha, M.D., M.B.B.S.

Mechanisms of Fecal Continence

Fecal continence is normally maintained by anatomical factors (i.e., pelvic barrier, rectal curvatures, and transverse rectal folds), recto-anal sensation, and rectal compliance.[1] The rectal segment above the middle fold is derived from the embryological hindgut, may contain feces, and is free to distend towards the peritoneal cavity. The lower rectum situated below the middle rectal fold is derived from the cloaca, surrounded by condensed extraperitoneal tissue, and is empty except during defecation. Rectal distention by stool induces rectal contraction, the sensation of urgency, and reflex relaxation of the internal anal sphincter, prompting defecation if socially convenient (Figure 1). If not, rectal contractions and the sensation of urgency generally subside as the rectum accommodates to continued distention. This, together with voluntary contraction of the external anal sphincter,[2] permits defecation to be postponed when necessary.[3] The factors that determine whether rectal distention is interpreted as a desire to defecate or to pass flatus are unclear.

Figure 1. Mechanisms of Fecal Continence



Etiology and Pathophysiology of Fecal Incontinence

Fecal incontinence (FI) is caused by anorectal dysfunctions and/or disordered bowel habits, resulting from a variety of conditions (Table 1). Some of these conditions (i.e., disordered

Table 1. Etiology of Fecal Incontinence

Condition	Possible Causes
Anal sphincter weakness	
Injury	Obstetric trauma, related to surgical procedures (e.g., hemorrhoidectomy, internal sphincterotomy, fistulotomy, anorectal infection)
Nontraumatic	Scleroderma, internal sphincter thinning of unknown etiology
Neuropathy	Stretch injury, obstetric trauma, diabetes mellitus
Anatomical disturbances of pelvic floor	Fistula, rectal prolapse, descending perineum syndrome
Inflammatory conditions	Crohn's disease, ulcerative colitis, radiation proctitis
Central nervous system disease	Dementia, stroke, brain tumors, spinal cord lesions, multiple system atrophy (Shy-Drager syndrome), multiple sclerosis
Diarrhea	Irritable bowel syndrome, post-cholecystectomy diarrhea

bowel habits, obstetric trauma, and neurological conditions) will be detailed in other presentations. FI is a multifactorial disorder. The pathophysiological mechanisms reflect the etiology of FI (Table 2), and more than one pathophysiological mechanism may contribute to FI in the same patient. Our understanding of the pathophysiology of FI, as detailed below, is primarily derived from clinic-based rather than community-based studies and predominantly in women.

Table 2. Anorectal Sensori-Motor Disturbances in Fecal Incontinence

Etiological Factor	Anal Sphincter Pressure	Threshold for Internal Sphincter Relaxation	Threshold for External Sphincter Contraction	Rectal Sensation*	Rectal Compliance	Pelvic Floor Function
"Idiopathic"	↓	↓	↓	↓ or ↑	↓	↓
Diabetes mellitus[4]	R ↓ S ↓	↔	↑	↓↓	↔	
Multiple sclerosis[4]	R ↔ S ↓↓	↓	↑	↓↓	↔	
Elderly patients with fecal impaction and incontinence[5]	R ↔ S ↔	↓	↑	↓		↓
Acute radiation proctitis[6]	R ↓ S ↓	NA	NA	↔	↓	NA
Chronic radiation injury[7]	NA	NA	NA	↑	↓	NA
Ulcerative colitis[8,9]	S ↓ incontinent patients	↓ (active colitis only)	NA	↑ (active colitis only)	↓ (active colitis only)	NA
Spinal cord injury, high spinal lesion, i.e., T12 or higher[10]	R ↔ S ↓	↓	↔	↓	↓	NA
Low spinal lesion, i.e., below T12	R ↓ S ↓	↔	↓	↓	↔	NA

Information pertains to patients with underlying disease and fecal incontinence.

↑ = Increased; ↓ = decreased; ↔ = no change; R = resting; S = squeeze sphincter pressure; NA = not available.

*Rectal sensation expressed as volume thresholds for perception; ↑ sensation indicates volume threshold for perception was lower compared to normal patients.

Pelvic Barrier

The anal sphincters and pelvic floor muscles (i.e., levator ani) comprise the pelvic barrier. The internal anal sphincter generates approximately 70% of anal resting tone; the external anal sphincter, which is a tonically active striated muscle, provides the balance. The puborectalis is a U-shaped component of the levator ani which blends with the upper aspect of the external

sphincter and maintains a relatively acute anorectal angle at rest. When continence is threatened, the external sphincter and pelvic floor can be contracted voluntarily to preserve continence. A majority of women with FI have reduced anal resting and/or squeeze pressures, reflecting weakness of the internal and/or external anal sphincters respectively (Table 2).[11,12] Anal sphincter damage due to obstetric or iatrogenic injury and pudendal neuropathy are common causes of anal sphincter weakness (Table 1). Endoanal ultrasound and magnetic resonance imaging (MRI) reveal anal sphincter injury, which manifests as defects, scarring, or thinning (i.e., atrophy).[12,13] Only MRI reveals atrophy of the external sphincter. External sphincter injury is associated with lower anal squeeze pressures, reflecting external sphincter dysfunction.[12,14]

Although these observations indicate a link between pelvic floor injury, weakness, and FI, several caveats apply. Most studies evaluating pelvic floor structure and function in FI are uncontrolled. Because anal pressures and pelvic floor motion are affected by age, gender, and techniques, these parameters should be compared with appropriate controls.[15–18] Because anal sphincter defects are observed even after uncomplicated vaginal delivery,[19,20] it can be challenging to ascertain the precise contribution of anal sphincter injury to anal weakness, particularly since the main known risk factor for anal injury (i.e., obstetric trauma), precedes FI by two to three decades.[21]

A pudendal neuropathy can also cause anal sphincter weakness. However, pudendal nerve function generally has been assessed by measuring nerve latencies, which we now recognize are flawed and should not be used to assess pudendal nerve function.[22] Needle electromyography (EMG) is necessary to identify neurogenic injury of the external anal sphincter. Neurogenic injury may result not only from a pudendal neuropathy but also from damage to nerves within the sphincter.[13] However, the expertise for conducting and interpreting needle EMG of the external sphincter is not widely available.

It is not widely appreciated that FI is also associated with atrophy, denervation, and impaired function of the puborectalis muscle, which is correlated with symptoms and improved after biofeedback therapy.[12,23,24] A subset of patients with FI have more generalized pelvic floor weakness (i.e., descending perineum syndrome), which is often associated with pelvic organ prolapse affecting the anterior and/or middle compartments.[25] Excessive perineal descent may stretch, and thereby damage the pudendal nerve. Excessive descent may also make the anorectal angle more obtuse. This impairs the flap valve normally responsible for maintaining fecal continence during increased intra-abdominal pressure. Sphincter pressures are lower in incontinent than in continent patients with the descending perineum syndrome.[25]

Rectal Compliance and Sensation

Patients with FI may have normal, reduced, or increased rectal sensation. Reduced rectal sensation allows stool to enter the anal canal, and perhaps leak before the external sphincter contracts.[11,26,27] Conversely, other patients with FI (47% in one large series)[11,12,28] have exaggerated rectal sensation (i.e., rectal hypersensitivity). Compared to patients with normal rectal sensation, patients with urge FI and rectal hypersensitivity have more frequent stools, use more pads, and report more lifestyle restrictions.[28] The mechanisms of rectal hypersensitivity are being studied. Because rectal perception is attributable not to rectal distention per se, but to the contractile response to distention,[29] rectal hypersensitivity in FI may be not primary, but perhaps partly secondary to an exaggerated contractile response to distention.[30] Indeed, rectal capacity is reduced in some women with FI. Reduced capacity is associated with rectal hypersensitivity and with the symptom of urgency.[12,30–32] Thus, it is conceivable that either

increased tone (i.e., reduced capacity or compliance), and/or an exaggerated contractile response to distention, and/or an alteration in passive properties (e.g., fibrosis) may amplify the increase in rectal tension during distention, thereby contributing to rectal hypersensitivity in FI.

Anal Sensation

Anal sphincter relaxation may occur during or independent of rectal distention, enabling the anal lining to periodically "sample" and ascertain the nature of rectal contents (i.e., gas, liquid, or stool).[33] In health, lidocaine anesthetizes the anal canal but does not affect continence for saline, suggesting that anal sensation is not critical for continence.[3] However, anal sampling occurred less frequently and anal sensation was reduced in incontinent patients, perhaps depriving them of sensory information.[33] The contributions of normal and disordered anal sampling to FI in patients with anorectal sensorimotor dysfunctions has not been studied.

Impaired Rectal Evacuation

Impaired rectal evacuation with retention of feces may contribute to FI in women, in elderly patients, and in men with a hypertonic sphincter (i.e., a long, high-pressured anal sphincter entraps small particles of feces during defecation and subsequently expels them, causing perianal soiling and discomfort).[12,34–36]

Other Factors

In addition to normal anorectal functions and stool consistency, mental faculties and mobility are necessary to preserve continence. Clinical observations and epidemiological studies suggest that irritable bowel syndrome (IBS) is a risk factor for FI.[37–38] IBS may be associated with accelerated small intestinal and/or colonic transit.[39] Conceivably, rapid delivery of colonic contents to the rectum may predispose to FI, particularly in patients with a dysfunctional rectal reservoir. In a preliminary report, however, only 3 of 20 patients with urge FI had accelerated colonic transit, and rectal sensorimotor dysfunctions, rather than accelerated transit, explained rectal urgency.[40] Similarly, rectal urgency was the most important risk factor for FI among women in the community, being associated on average with an eightfold increased risk for FI, even after controlling for a history of obstetric anal sphincter injury and other bowel symptoms (e.g., constipation and diarrhea).[38] Taken together, these data are consistent with the concept that in FI, rectal urgency is a distinct symptom which reflects rectal overactivity and/or rectal hypersensitivity rather than loose stools. Indeed, rectal urgency is associated not only with liquid but also with formed stools in healthy subjects.[41]

Pathophysiology of Fecal Incontinence in Men

Although the prevalence of FI is comparable among men and women in the community,[42,43] only a few studies have evaluated the pathophysiology of FI in men.[44] Clinical observations suggest that FI in men is frequently attributable to local causes (e.g., hemorrhoids, fistula, iatrogenic anal sphincter injury, or proctitis after radiotherapy for prostate cancer) or a rectal evacuation disorder.[35]

Summary and Gaps in Knowledge

FI is caused by an imbalance between stool consistency and anorectal functions in maintaining continence. Anal weakness is a recognized risk factor for FI, but the contributions of other risk factors, (e.g., disordered rectoanal sensation, and/or rectal compliance), which often coexist, to

FI are not. These mechanisms should be considered, particularly in incontinent patients with normal sphincter pressures.[45]

The critical questions about the pathophysiology of FI are:

1. What risk factors and pathophysiological mechanisms in women explain why FI generally presents several decades postpartum? Is the effect of age/menopause on anorectal functions modified by obstetric history?

2. What mechanisms are responsible for a smaller rectal reservoir in FI?

3. To what extent does neurogenic injury contribute to weakness of the pelvic barrier in FI?

4. Does disordered anal sensation contribute to FI?

5. What is the interaction between anorectal dysfunctions and disordered bowel habits in FI?

References

1. Bharucha AE. Pelvic floor: anatomy and function. *Neurogastroenterol Motility.* 2006;18:507–519.

2. Sun WM, Read NW, Miner PB. Relation between rectal sensation and anal function in normal subjects and patients with faecal incontinence. *Gut.* 1990;31:1056–1061.

3. Read MG, Read NW. Role of anorectal sensation in preserving continence. *Gut.* 1982;23:345–347.

4. Caruana BJ, Wald A, Hinds JP, Eidelman BH. Anorectal sensory and motor function in neurogenic fecal incontinence. Comparison between multiple sclerosis and diabetes mellitus. *Gastroenterology.* 1991;100:465–470.

5. Read NW, Abouzekry L. Why do patients with faecal impaction have faecal incontinence? *Gut.* 1986;27:283–287.

6. Yeoh EK, Russo A, Botten R, Fraser R, Roos D, Penniment M, Borg M, Sun WM. Acute effects of therapeutic irradiation for prostatic carcinoma on anorectal function. *Gut.* 1998;43:123–127.

7. Varma JS, Smith AN, Busuttil A. Correlation of clinical and manometric abnormalities of rectal function following chronic radiation injury. *Br J Surg.* 1985;72:875–878.

8. Rao SS, Read NW, Davison PA, Bannister JJ, Holdsworth CD. Anorectal sensitivity and responses to rectal distention in patients with ulcerative colitis. *Gastroenterology.* 1987;93:1270–1275.

9. Denis P, Colin R, Galmiche JP, Geffroy Y, Hecketsweiler P, Lefrancois R, Pasquis P. Elastic properties of the rectal wall in normal adults and in the patients with ulcerative colitis. *Gastroenterology.* 1979;77:45–48.

10. Sun WM, Read NW, Donnelly TC. Anorectal function in incontinent patients with cerebrospinal disease. *Gastroenterology.* 1990;99:1372–1379.

11. Sun WM, Donnelly TC, Read NW. Utility of a combined test of anorectal manometry, electromyography, and sensation in determining the mechanism of 'idiopathic' faecal incontinence. *Gut.* 1992;33:807–813.

12. Bharucha AE, Fletcher JG, Harper CM, Hough D, Daube JR, Stevens C, Seide B, Riederer SJ, Zinsmeister AR. Relationship between symptoms and disordered continence mechanisms in women with idiopathic fecal incontinence. *Gut.* 2005;54:546–555.

13. Stoker J, Halligan S, Bartram CI. Pelvic floor imaging. *Radiology.* 2001;218:621–641.

14. Terra MP, Deutekom M, Beets-Tan RG, Engel AF, Janssen LW, Boeckxstaens GE, Dobben AC, Baeten CG, de Priester JA, Bossuyt PM, Stoker J. Relationship between external anal sphincter atrophy at endoanal magnetic resonance imaging and clinical, functional, and anatomic characteristics in patients with fecal incontinence. *Dis Colon Rectum.* 2006;49:668–778.

15. McHugh SM, Diamant NE. Effect of age, gender, and parity on anal canal pressures. Contribution of impaired anal sphincter function to fecal incontinence. *Digest Dis Sci.* 1987;32:726–736.

16. Bannister JJ, Abouzekry L, Read NW. Effect of aging on anorectal function. *Gut.* 1987;28:353–357.

17. Jameson JS, Chia YW, Kamm MA, Speakman CT, Chye YH, Henry MM. Effect of age, sex and parity on anorectal function. *Br J Surg.* 1994;81:1689–1692.

18. Fox JC, Fletcher JG, Zinsmeister AR, Seide B, Riederer SJ, Bharucha AE. Effect of aging on anorectal and pelvic floor functions in females. *Dis Colon Rectum.* 2006;49:1726–1735.

19. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Eng J Med.* 1993;329:1905–1911.

20. Williams AB, Bartram CI, Halligan S, Spencer JA, Nicholls RJ, Kmiot WA. Anal sphincter damage after vaginal delivery using three-dimensional endosonography. *Obstet Gynecol.* 2001;97:770–775.

21. Bharucha AE, Zinsmeister AR, Locke GR, Seide B, McKeon K, Schleck CD, Melton LJI. Prevalence and burden of fecal incontinence: a population based study in women. *Gastroenterology.* 2005;129:42–49.

22. Diamant NE, Kamm MA, Wald A, Whitehead WE. American Gastroenterological Association medical position statement on anorectal testing techniques. *Gastroenterology.* 1999;116:732–760.

23. Bartolo DC, Jarratt JA, Read MG, Donnelly TC, Read NW. The role of partial denervation of the puborectalis in idiopathic faecal incontinence. *Br J Surg.* 1983;70:664–667.

24. Fernandez-Fraga X, Azpiroz F, Malagelada JR. Significance of pelvic floor muscles in anal incontinence. *Gastroenterology.* 2002;123:1441–1450.

25. Bartolo DC, Read NW, Jarratt JA, Read MG, Donnelly TC, Johnson AG. Differences in anal sphincter function and clinical presentation in patients with pelvic floor descent. *Gastroenterology.* 1983;85:68–75.

26. Allen ML, Orr WC, Robinson MG. Anorectal functioning in fecal incontinence. *Digest Dis Sci.* 1988;33:36–40.

27. Buser WD, Miner PB Jr. Delayed rectal sensation with fecal incontinence. Successful treatment using anorectal manometry. *Gastroenterology.* 1986;91:1186–1191.

28. Chan CL, Scott SM, Williams NS, Lunniss PJ. Rectal hypersensitivity worsens stool frequency, urgency, and lifestyle in patients with urge fecal incontinence. *Dis Colon Rectum.* 2005;48:134–140.

29. Corsetti M, Cesana B, Bhoori S, Basilisco G. Rectal hypersensitivity to distention in patients with irritable bowel syndrome: role of distention rate. *Clinical Gastroenterol Hepatol.* 2004;2:49–56.

30. Andrews CN, Bharucha AE, Seide B, Zinsmeister AR. Rectal sensorimotor dysfunction in women with fecal incontinence. *Am J Physiology; Gastrointest Liver Physiol.* 2007;292:G282–G289.

31. Siproudhis L, El Abkari M, El Alaoui M, Juguet F, Bretagne JF. Low rectal volumes in patients suffering from fecal incontinence: what does it mean? *Alimentary Pharmacol Thera.* 2005;22:989–996.

32. Deutekom M, Dobben AC, Terra MP, Engel AF, Stoker J, Bossuyt PM, Boeckxstaens GE. Clinical presentation of fecal incontinence and anorectal function: what is the relationship? *Am J Gastroenterol.* 2007;102:351–361.

33. Miller R, Bartolo DC, Cervero F, Mortensen NJ. Anorectal sampling: a comparison of normal and incontinent patients. *Br J Surg.* 1988;75:44–47.

34. Read NW, Abouzekry L, Read MG, Howell P, Ottewell D, Donnelly TC. Anorectal function in elderly patients with fecal impaction. *Gastroenterology.* 1985;89:959–966.

35. Parellada CM, Miller AS, Williamson ME, Johnston D. Paradoxical high anal resting pressures in men with idiopathic fecal seepage. *Dis Colon Rectum.* 1998;41:593–597.

36. Rao SS, Ozturk R, Stessman M. Investigation of the pathophysiology of fecal seepage. *Am J Gastroenterol.* 2004;99:2204–2209.

37. Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors: an underdiagnosed problem in the Australian community? *Med J Austr.* 2002;176:54–57.

38. Bharucha AE, Zinsmeister AR, Locke GR, Seide B, McKeon K, Schleck CD, Melton LJI. Risk factors for fecal incontinence: a population based study in women. *Am J Gastroenterol.* 2006;101:1305–1312.

39. Vassallo M, Camilleri M, Phillips SF, Brown ML, Chapman NJ, Thomforde GM. Transit through the proximal colon influences stool weight in the irritable bowel syndrome. *Gastroenterology.* 1992;102:102–108.

40. Murphy J, Chan CL, Pearce E, Newell M, Williams NS, Lunniss PJ, Garvie NW, Scott SM. The relationship between intestinal transit and rectal sensorimotor function in patients with urge fecal incontinence. *Neurogastroenterol Motil.* 2006;18:693. [Abstract]

41. Heaton KW, Ghosh S, Braddon FE. How bad are the symptoms and bowel dysfunction of patients with the irritable bowel syndrome? A prospective, controlled study with emphasis on stool form. *Gut.* 1991;32:73–79.

42. Perry S, Shaw C, McGrother C, Matthews RJ, Assassa RP, Dallosso H, Williams K, Brittain KR, Azam U, Clarke M, Jagger C, Mayne C, Castleden CM, Leicestershire M. Prevalence of faecal incontinence in adults aged 40 years or more living in the community. *Gut.* 2002;50:480–484.

43. Quander CR, Morris MC, Melson J, Bienias JL, Evans DA. Prevalence of and factors associated with fecal incontinence in a large community study of older individuals. *Am J Gastroenterol.* 2005;100:905–909.

44. Chen H, Humphreys MS, Kettlewell MG, Bulkley GB, Mortensen N, George BD. Anal ultrasound predicts the response to nonoperative treatment of fecal incontinence in men. *Ann Surg.* 1999;229:739–743; discussion 743–734.

45. Siproudhis L, Bellissant E, Pagenault M, Mendler MH, Allain H, Bretagne JF, Gosselin M. Fecal incontinence with normal anal canal pressures: where is the pitfall? *Am J Gastroenterol.* 1999;94:1556–1563.

Pathophysiology of Urinary Incontinence

Alan J. Wein, M.D., Ph.D. (Hon.)

The lower urinary tract (LUT) functions as a group of interrelated structures with a joint function in the adult to bring about efficient and low-pressure bladder filling, low-pressure urine storage with perfect continence, and periodic complete voluntary urinary expulsion, again at low pressure.

Urinary incontinence (UI) is defined as the involuntary loss of urine. The term is used in various ways. It may denote a symptom, a sign, or a condition. The pathophysiology of failure of the LUT to fill with or store urine adequately or to empty adequately must logically be secondary to reasons related to the bladder, the outlet, or both. A simple classification of the various subtypes of UI is seen in Table 1.

Table 1. Classification of Incontinence

- I. Extraurethral
  - A. Fistula (vesicovaginal, ureterovaginal, urethrovaginal)
  - B. Ectopic Ureter
- II. Urethral
  - A. Functional
    1. Due to physical disability
    2. Due to lack of awareness or concern
  - B. Post-void dribbling
    1. Urethral diverticulum
    2. Vaginal pooling of urine
  - C. Bladder overactivity
    1. Involuntary contractions
       - a. Neurologic disease or injury
       - b. Bladder outlet obstruction
       - c. Afferent activation (includes inflammation or infection)
       - d. Idiopathic
    2. Decreased compliance
       - a. Neurologic disease or injury
       - b. Fibrosis
       - c. Idiopathic
    3. Combination
  - D. Outlet underactivity
    1. “Genuine” stress urinary incontinence (GSI)
       - a. Lack of urethral support
       - b. Hypermobility, deficient “hammock”
    2. Intrinsic sphincter deficiency (ISD)
       - a. Neurologic disease or injury
       - b. Fibrosis
    3. Urethral instability
  - E. “Overflow” incontinence

There are situations in which urethral incontinence cannot be considered merely as an isolated abnormality of either bladder contractility or sphincter resistance. These situations, listed in Table 2 are more complicated to deal with, first, because they are more difficult to diagnose, and second, because one entity may adversely affect or compromise treatment of the other.

Table 2. Combined Problems Associated With Incontinence

Detrusor overactivity with outlet obstruction
Detrusor overactivity with impaired bladder contractility
Sphincteric incontinence with detrusor overactivity
Sphincteric incontinence with impaired bladder contractility

Absolute or relative failure to fill with and store urine adequately results from bladder overactivity (involuntary contraction or decreased compliance), decreased outlet resistance, heightened or altered sensation, or a combination.

Bladder Overactivity

Overactivity of the bladder during filling or storage can be expressed as phasic involuntary contractions (detrusor overactivity), as low compliance, or as a combination. Detrusor overactivity is most commonly seen in association with neurologic disease or injury; however, it may be associated with increased afferent input due to inflammation or irritation of the bladder or urethral wall, bladder outlet obstruction, stress urinary incontinence (perhaps due to sudden entry of urine into the proximal urethra), aging (probably related to neural degeneration), or may be idiopathic. Some hypothesize that decreased stimulation from the pelvic floor can contribute to phasic overactivity. Decreased compliance during filling or storage may be secondary to neurologic injury or disease, usually at a sacral or infrasacral level, but may result from any process that destroys the viscoelastic or elastic properties of the bladder wall.

Outlet Underactivity

Decreased outlet resistance may result from any process that damages the innervation or structural elements of the smooth and/or striated sphincter or support of the bladder outlet in the female. This may occur with neurologic disease or injury, surgical or other mechanical trauma, or aging. Classically, sphincteric incontinence in the female was categorized into relatively discrete entities: (1) so-called genuine stress incontinence (GSI) and (2) intrinsic sphincter deficiency (ISD), originally described as “type III stress incontinence.” GSI in the female was described as associated with hypermobility of the bladder outlet because of poor pelvic support and with an outlet that was competent at test but lost its competence only during increases in intra-abdominal pressure. ISD described a nonfunctional or very poorly functional bladder neck and proximal urethra at rest. The original implication of classical ISD was that a surgical procedure designed to correct only urethral hypermobility would have a relatively high failure rate, as opposed to one designed to improve urethral coaptation and compression. The contemporary view is that the majority of cases of effort-related incontinence in the female involve varying proportions of support-related factors and ISD. It is possible to have outlet-related incontinence due only to ISD but not due solely to hypermobility or poor support—some ISD must exist.

Stress- or effort-related UI is a symptom that arises primarily from damage to muscles and/or nerves and/or connective tissue within the pelvic floor. The urethra is normally supported by the action of the levator ani muscles through their connection to the endopelvic fascia of the anterior vaginal wall. Damage to this connection, or to the nerve supply, or direct muscle damage can, therefore, influence continence. Bladder neck function is likewise important, and loss of normal bladder neck closure can result in UI despite normal urethral support. In older writings, the urethra was sometimes ignored as a factor contributing to continence in the female, and the site of continence was thought to be exclusively the bladder neck. However, in approximately 50% of continent women, urine enters the urethra during increases in abdominal pressure. The continence point in these women (highest point of pressure transmission) is at the mid urethra.

Urethral hypermobility implies weakness of the pelvic floor-supporting structures. During increases in intra-abdominal pressure, there is descent of the bladder neck and proximal urethra. If the outlet opens concomitantly, stress UI ensues. In the classic form of urethral hypermobility, there is rotational descent of the bladder neck and urethra. However, the urethra may also descend without rotation (it shortens and widens), or the posterior wall of the urethra may be pulled (sheared) open while the anterior wall remains fixed. However, urethral hypermobility is often present in women who are not incontinent; thus, the mere presence of urethral hypermobility is not sufficient to make a diagnosis of a sphincter abnormality unless UI is also demonstrated. The “hammock hypothesis” of John DeLancey proposes that for stress incontinence to occur with hypermobility, there must be a lack of stability of the suburethral supportive layer: the effect of abdominal pressure increases on the normal bladder outlet, if the suburethral supportive layer is firm, is to compress the urethra rapidly and effectively. If the supportive suburethral layer is lax and/or movable, compression is not as effective. Intrinsic sphincter dysfunction denotes an intrinsic malfunction of the urethral sphincter mechanism itself. In its most overt form, it is characterized by a bladder neck and proximal urethra which are open at rest and is usually the result of prior surgery, trauma with scarring, or a neurologic lesion.

Urethral instability refers to the rare phenomenon of episodic decreases in outlet pressure unrelated to increases in bladder or abdominal pressure. The term urethral instability is probably a misnomer, because many believe that the drop in urethral pressure represents simply the urethral component of a normal voiding reflex in an individual whose bladder does not measurably contract, because of either myogenic or neurogenic reasons.

In theory at least, categories of outlet-related incontinence in the male are similar to those in the female. Sphincteric incontinence in the male is not, however, associated with intrinsic hypermobility of the bladder neck and proximal urethra but is similar to what is termed sphincter dysfunction in the female. It is generally due to prostatectomy, pelvic trauma, or neurologic disease or injury.

Overflow Incontinence

This descriptive term denotes leakage of urine associated with urinary retention. This is more common in the male than female. The primary pathophysiology is actually a failure of emptying, leading to urinary retention with “overflow” UI, resulting from either continuous or episodic elevation of intravesical pressure over urethral pressure. This generally results from outlet obstruction or detrusor inactivity, either neurologic or pharmacologic in origin, or may be secondary to inadvertent overdistention of the bladder.

General References

1. Wein AJ. Pathophysiology and classification of voiding dysfunction; In: Wein AJ, Kavoussi LR, Novick AC, Partin AW, Peters CA, eds. *Campbell-Walsh Urology. 9th ed.* Philadelphia, PA: Saunders/Elsevier; 2007:1973–1985.

2. Wein AJ, Moy ML. Voiding function and dysfunction: urinary incontinence. In: Hanno PM, Malkowicz SB, Wein AJ, eds. *Penn Clinical Manual of Urology.* Philadelphia, PA: Saunders/Elsevier; 2007:341–478.

Economic Impact of Fecal and Urinary Incontinence on the Individual and Society—Direct and Indirect Costs

Philip B. Miner, Jr., M.D.

Introduction

Failure to control the elimination of urine or stool causes psychological stress, complicates medical illnesses and management, and has major economic consequences. Urinary incontinence (UI) is a common problem with a great variation in the amount of incontinence and a complex differential diagnosis. Recent research related to fecal incontinence (FI) has unraveled much of the complexity of the pathophysiology. There are problems related to muscular failure of the pelvic floor due to surgical or accidental disruption of the muscular anal canal, neurologic dysfunction of the pelvic floor from suprasacral spinal cord injury, sacral nerve root injury, injury to the pelvic and rectal intramural nerves, and disorders of colonic and rectal function related to endocrine disorders, mucosal immune activation or inflammation. Complaints of incontinence cover a wide spectrum from involuntary, but recognized, passage of gas, liquid, or solid stool (urge incontinence) to unrecognized anal leakage of mucus, fluid, or stool (passive incontinence). The intensity of sensation related to defecation and the volume of stool involuntarily passed helps delineate the etiology of constipation, but the wide variety of etiologies and difficulty in defining the cause of FI with precision makes treatment solutions difficult to assess and interpretation of economic consequences complex. Personal impact is profound, as many individuals withdraw from all social contact while they remain tethered to their toilet in an attempt to minimize the episodes of incontinence. Minimal published data is available to help understand the personal and economic impact of incontinence.

Published studies on the social, personal, and economic impact of incontinence generally focus on a narrow part of the problem that cannot be extrapolated to the whole population—for example, FI in patients with diabetes which has a complex pathophysiology that includes sensory changes, primary anal canal abnormalities, and issues of small intestinal and colonic function that do not occur in other illnesses. In addition, the broad range of medical issues associated with diabetes is so complex that the incontinence issue becomes buried amongst the numerous other personal and economic costs of the disease. Although an economic cost analysis is feasible, the application of the information is limited to a narrow part of the population, and the patient's psychological issues are often dominated by underlying medical illness. When reviewing this topic, the patients for whom the economic cost is the greatest have other medical issues that overpower the cost of the incontinence (for example, spinal cord injury). Conversely, in the patients who pay the highest psychological price, the direct economic costs can be relatively small (for example, the patient with irritable bowel syndrome who is confined to home because of sudden explosive episodes of diarrhea that prohibit traveling as far as the shopping mall). The broad spectrum of incontinence and the limited information available makes this a difficult topic for generalized analysis.

Economic Impact

Diagnostic tests are not expensive. Evaluation is focused on obtaining assurance that the gross structure appearance of the anal canal, rectum, and colon is normal, and microscopic mucosal changes do not indicate inflammation that needs to be treated. Often, when abnormalities are found, successful intervention can be instituted. A variety of tests are available to assess

neurological or muscular function of the pelvic floor; the most common is anorectal manometry (ARM). The Medicare reimbursement for ARM is $299.[1] Unfortunately, although ARM is available in most cities with gastrointestinal motility equipment, true expertise in diagnosis and management is not readily available. ARM comprises a minuscule part of the curriculum for postgraduate education in gastroenterology and colorectal surgery. Correcting this deficit in training is pivotal if the treatment of incontinence is to progress.

Medical or surgical intervention costs are identifiable, but Malouf and colleagues grasped the problem accurately when they concluded that economic assessment is very difficult because of the lack of uniform study populations, variation in surgical techniques, and regional costs.[2] Often, the studied option for management is so unique to the institution or surgeon performing the study that application to other institutions or less skilled surgeons is impossible. Outcome measurements are not standard, follow-up periods are often brief, and procedure failures are difficult to integrate into the cost of surgery and the subsequent cost of care. Although continence scoring systems provide an index of clinical success, they do not measure a meaningful outcome (e.g., continence is often controlled by limiting activity in order to be close to a toilet).

An indirect measure of the extent and cost of incontinence is the marketing information on protective garments. Direct costs for the items that clearly represent protection for patients with incontinence (e.g., pads, diapers) was $255,000,000 for the institutional market and an additional $150,000,000 in the retail market. The total UI market for 1999 was estimated to be $2.607 billion. Other less obvious expenses in the incontinence market are more difficult to assess. Costs for ostomy supplies is an example, as it is difficult to know how much of the market is for ostomies created to manage incontinence and what portion of the market is for ostomies created for other purposes (for example, colon cancer).

The economic impact of incontinence, beyond the direct costs includes the payment of disability claims for patients with incontinence who are no longer able to work, is also in lost wages related to quitting work or retiring prematurely due to incontinence. The principal issue focuses on the patient who has incontinence and the disability or lost wages due to the inability to work. In the total economic assessment, families or friends who must leave their jobs to help the patient with incontinence must also be considered. Estimation of the cost in this sector is elusive, but it must be in the hundreds of millions of dollars a year.

In summary, the economic cost of incontinence is enormous and obvious, but the exact economic toll on the country's resources and on individual families is difficult to determine with precision. Economic models focusing on these issues should be developed to help garner resources into research on improving incontinence.

Personal Impact

The personal impact of incontinence is obvious to any reasonable person. Applying science has proven more difficult. Quality of life (QOL) instruments have been developed for a variety of illnesses to determine the impact of a variety of diseases.[3–8] No QOL instrument is necessary to communicate the suffering patients with FI and UI endure. Since all diseases, and often specific medical or surgical treatment, may decrease the QOL, the only reasonable QOL issues are related to a change in QOL with specific treatment programs. The development of QOL instruments for incontinence, with appropriate validation, is important for the assessment of different treatment protocols.

Social interaction is difficult to assess, as it is accompanied by isolation from depression and anxiety.[4,5,9–13] In our control trial of retraining techniques for FI, we were surprised by the common patient response that, after training, they were much better even though the diary of their incontinence episodes recorded the same number of incontinence episodes prior to the study and after retraining.[14] Their expression of improvement was related to an increase in activity level following FI managed by retraining.

Concluding Concepts

Our intuitive concepts are sufficient to understand the dimensions of the personal and economic impact of incontinence for the patient, but the ambiguity of data needs to be addressed to marshal the resources to help improve the lives of patients with incontinence. Specific suggestions for developing and channeling the resources necessary for comprehensive understanding of this important topic in incontinence are:

1. Proper economic analysis directed toward isolating the cost of incontinence, including medical and surgical options.

2. Economic analysis can justify financial support for medical research.

3. Medical management algorithms should be developed.

4. Developing new resources and maximizing the utilization of available resources to keep patients out of medical facilities.

5. Improved inpatient management algorithms for acute and chronic care.

References

1. Practice Management Information Corporation. *Medical Fees in the United States: Nationwide Charges for Medicine, Surgery, Laboratory, Radiology and Allied Health Services*. Los Angeles, CA: Author; 2003.

2. Malouf AJ, Chambers MG, and Kamm MA. Clinical and economic evaluation of surgical treatments for faecal incontinence. *Br J Surg.* 2001;88:1029–1036.

3. Edwards NI, Jones D. The prevalence of fecal incontinence in older people living at home. *Age Ageing.* 2001;30:503–507.

4. Stewart WF, Van Rooyen JB, Cundiff GW, Abrams P, Herzog AR, Corey R, Hunt TL, Wein AJ. Prevalence and burden of overactive bladder in the United States. *World J Urol.* 2003;20:327–336.

5. Melville JL, Walker E, Katon W, Lentz G, Miller J, Fenner D. Prevalence of comorbid psychiatric illness and its impact on symptom perception, quality of life and functional status in women with urinary incontinence. *Am J Obstet G.* 2002;187:80–87.

6. O'Keefe EA, Talley NJ, Zinsmeister AR, Jacobsen SJ. Bowel disorders impair functional status and quality of life in the elderly: a population-based study. *J Gerontol A Biol Sci Med Sci.* 1995;50:M184–M189.

7. Crowell MD, Schettler-Duncan A, Brookhart K, Barofsky I. Fecal incontinence: impact on psychosocial function and health-related quality of life (abstr). *Gastroenterology.* 1998;114:A729.

8. Fialkow MF, Melville JL, Lentz GM, Miller J, Fenner DE. The functional and psychosocial impact of fecal incontinence on women with urinary incontinence. *Am J Obstet Gynecol.* 2003;189:127–129.

9. Huppe D, Enck P, Kruskemper G, May B. Psychosocial aspects of fecal incontinence. *Leber Magen Darm.* 1992;22:138–142.

10. Nygaard I, Turvey C, Burns TL, Crischilles E, Wallace R. Urinary incontinence and depression in middle-aged United States women. *Obstet Gynecol.* 2003;101:149–156.

11. Mehta KM, Simonsick EM, Penninx BW, Schulz R, Rubin SM, Satterfield S, Yaffe K. Prevalence and correlates of anxiety symptoms in well-functioning older adults: findings from the health aging and body composition study. *J Am Geriatr Soc.* 2003;51:499–504.

12. Bogner HR, Gallo JJ, Swartz KL, Ford DE. Anxiety disorders and disability secondary to urinary incontinence among adults over 50. *Int J Psychiatry Med.* 2002;32:141–154.

13. Siracusano S, Pregazzi R, d'Aloia G, Sartore A, Di Benedetto P, Pecorari V, Gauschino S, Pappagallo G, Belgrano E. Prevalence of urinary incontinence in young and middle-aged women in an Italian urban area. *Eur J Obstet Gynecol Reprod Biol.* 2003;107:201–204.

14. Miner PB, Donnelly TC, Read NW. Investigation of mode of action of biofeedback in treatment of fecal incontinence. *Dig Dis Sci.* 1990;35:1291–1298.

Impact of Fecal and Urinary Incontinence on the Health Consumer: Barriers on Diagnosis and Treatment—A Patient Perspective

Nancy J. Norton

The impact of living as a person with urinary incontinence (UI) and/or fecal incontinence (FI) carries a significant burden. The nature of the condition presents unusual challenges. Incontinence is chronic, with often unpredictable symptom episodes that can be disabling. A social stigma is attached to incontinence and by attribution to the sufferer. Effective therapies are often elusive. Symptoms impair function and place demands on families as well as patients.[1]

Various factors influence whether or not a person will seek medical help and the individual's ability to adapt to their illness demands and benefit from treatment. Cultural, social, and psychological factors along with concepts of self-image and health expectations influence outcomes.[2]

UI or FI is a symptom of many different conditions. Establishing a diagnosis and assessing a therapeutic outcome of UI and FI has long been based on objective measurements such as quantity and frequency.[3] Objective measurements are not always practical, nor do they describe the true impact of incontinence on the patient.[2]

Illness Burden

The person with incontinence is faced with a persistent challenge of overcoming social and cultural taboos. Loss of control over elimination and public humiliation represent major threats to self-esteem.[4] Individuals will go to great lengths to keep their incontinence a secret. Revelation of this secret can have a profound effect on their well-being. The elderly are at risk for institutionalization.[5] Incontinent patients have been reported to be less likely to marry and to hold a normal job.[6]

Incontinence is accompanied by what is called a "second illness"—the reactions of the social environment and the stigma associated with the disorder. Stigmatization has been found to lead to social isolation, limited life chances, and delayed help seeking.[7] Numerous studies report that incontinence has a strong, if not devastating, impact on quality of life. Yet 50%–70% of incontinent persons do not seek help for their condition.[2–4,8,9]

Barriers to Care and Treatment

Barriers for seeking help include a lack of understanding of the condition, mistaken beliefs that symptoms are a normal part of aging or childbirth, and lack of knowledge about available treatments. Patients may not communicate their concerns to their physician or other healthcare providers because of embarrassment, fear of surgical interventions, or misconceptions of what constitutes a medical problem.[8,10]

In a study that found that primary care physicians ask few of their patients about incontinence, up to 70% of incontinent patients did not voluntarily report the problem, but more than 75% did report the condition when asked about it by their physician.[2] Physicians need to take the lead in

talking about incontinence. They need to ask their patients about bowel and bladder function, about the patient's ability to control it, and whether it is resulting in changes in daily routine. Patients and practitioners often refer to things differently, attaching their own interpretations to the reported symptoms. A simple question or two may be all that is needed to reveal the presence of incontinence.

The impact of symptoms on quality of life appears to be the main trigger for seeking help for UI. Few studies have been done to evaluate this in FI.[6,8]

Many people who are incontinent begin a gradual process of adaptation and accommodation of symptoms; this gives the individual the illusion of coping. Severity of symptoms may be a driving factor that brings people to their physician because they are no longer able to cope with the symptoms. Incontinent people have a need to perceive the benefits of treatment in order to overcome the emotional costs they will expend in revealing their incontinence to a physician.[8]

Changes Needed

The magnitude of the prevalence and burden of incontinence has been masked in this country by *silence* for far too long. There is a need to raise the comfort level in our society to discuss bowel and bladder problems in the context of more visible health concerns and to meet the challenge of removing the stigma.

Primary prevention needs to be a goal of all healthcare professionals, requiring a high level of community awareness and public education as well as health professionals' education.[11] How this will translate into behavioral change and what triggers health-seeking behavior are not yet fully understood. Behavioral change needs to occur within the provider community as well as on the public side.

Prevention may lie in developing new and different standards of care for patients, including awareness of how surgical interventions may cause or create injury resulting in FI.[12] Risk factors must be better understood. In nursing homes, continence could be improved with more toileting opportunities for residents.[13] More can be done on all fronts, not only to aid in preventing incontinence but to also improve the awareness around it, to make it easier for people to seek help, and to find solutions to managing the condition if not resolving it.

References

1. Royer A. *Life With Chronic Illness: Social and Psychological Dimensions*. Westport, Conn: Praeger Publishers; 1998.

2. Hajjar RR. Psychosocial impact of urinary incontinence in the elderly population. *Clin Geriatr Med.* 2004;20:553–564, viii.

3. Bharucha AE, Zinsmeister AR, Locke GR, Seide BM, McKeon K, Schleck CD, Melton LJ. Prevalence and burden of fecal incontinence: a population-based study in women. *Gastroenterology.* 2005;129:42–49.

4. Garcia JA, Crocker J, Wyman JF, Krissovich M. Breaking the cycle of stigmatization: managing the stigma of incontinence in social interactions. *J Wound Ostomy Continence Nurs.* 2005;32:38–52.

5. Mittness LS. The management of urinary incontinence by community-living elderly. *Gerontologist.* 1987;27:185–193.

6. Crowell MD, Schettler VA, Lacy BE, Lunsford TN, Harris LA, DiBaise JK, Jones MP. Impact of anal incontinence on psychosocial function and health-related quality of life. *Dig Dis Sci.* 2007;52:1627–1631. Epub 2007 Jan 9.

7. Schulze B, Angermeyer MC. Subjective experiences of stigma. A focus group study of schizophrenic patients, their relatives and mental health professionals. *Soc Sci Med.* 2003;56:299–312.

8. Shaw C, Tansey R, Jackson C, Hyde C, Allan R. Barriers to help seeking in people with urinary symptoms. *Fam Pract.* 2001;18:48–52.

9. Melville JL, Fan M-Y, Newton K, Fenner D. Fecal incontinence in US women: a population-based study. *Am J Obstet Gynecol.* 2005;193:2071–2076.

10. Horrocks S, Somerset M, Stoddart H, Peters TJ. What prevents older people from seeking treatment for urinary incontinence? A qualitative exploration of barriers to the use of community continence services. *Fam Pract.* 2004;21:689–696.

11. Newman DK, Denis L, Gruenwald I, Ee CH, Millard R, Roberts R, Sampselle C, Williams K, Muller N, Norton N. Continence promotion: prevention, education and organisation. In: Abram P, Cardozo L, Khoury S, Wein A, eds. *Incontinence Basics and Evaluation,* Vol. 1. Paris, France: Health Publications, 2005.

12. Collings S, Norton C. Women's experiences of faecal incontinence: a study. *Br J Community Nurs.* 2004;9:520–523.

13. Schnelle JF, Leung FW. Urinary and fecal incontinence in nursing homes. *Gastroenterology.* 2004;126:S41–S47.

Formal and Informal Caregiving Burden of Fecal and Urinary Incontinence

Sandra Engberg, Ph.D., R.N., C.N.R.P.

Caregiver burden is the strain or load borne by an individual caring for an older, chronically ill, or disabled family member or other person. It is a multidimensional response to the physical, psychological, emotional, social, and financial stressors associated with caring for another person.[1] For the purpose of this presentation, informal caregivers are persons who provide unpaid assistance to a family member or friend who needs help with one or more activities of daily living (ADLs). An estimated 44.4 million Americans are informal caregivers. The majority of those they care for are 50 years of age or older. Thirty percent of caregivers provide assistance with an average of eight or more ADLs and provide an average of 33 or more hours of care per week. Many of these caregivers report providing incontinence-related care (29%–53%) and/or toileting assistance (47%–68%).[2] Despite the high proportion of caregivers who report providing care related to managing or preventing incontinence, there is limited research examining the impact of incontinence on some aspect of caregiver burden (measuring burden, stress, fatigue, or hours of caregiving);[3–12] nearly half of the studies have been conducted outside the United States.[9–12] In most of these studies, all or the majority of the care recipients had dementia. There was considerable heterogeneity in the designs and methodologies used in these studies. Although findings varied, incontinence was generally associated with caregiver burden. Most of these studies examined the impact of urinary incontinence (UI)[3,5–8,10–12] on caregivers, with fewer studies examining the impact of fecal incontinence (FI) or combined FI and UI.[4,8] Incontinence-related caregiving for frail elders, particularly those with dementia, generally involves multiple care-related activities. Only one study examined what aspects of continence care were burdensome.[7] There is a dearth of research examining the impact of incontinence on caregivers of individuals without dementia.

The decision to place a care recipient in a nursing home can be seen as a proxy measure of caregiver burden. A number of investigators have examined UI and/or FI as risk factors for placing care recipients in a long-term-care facility,[3,9,13–21] with most studies conducted outside of the United States. The findings of these studies are mixed in relation to whether UI, FI, or combined incontinence increases the risk of institutionalizing an older care recipient.

This presentation also examines the limited research exploring the impact of incontinence on formal (paid) caregivers. These studies were conducted in nursing homes. In 2004, there were an estimated 1.5 million nursing home residents.[22] In 1997, nursing home residents needed assistance with an average of 4.4 physical ADLs, and almost 65% had UI and/or FI.[23] Their formal caregivers are predominantly nursing assistants. No studies were found that directly examined the impact of incontinence on formal caregiver burden. Formal caregiver burden may be a risk factor for absenteeism and turnover in long-term-care settings as well as having a negative impact on resident care, and bladder and bowel care may contribute to the burden. There is limited evidence examining outcomes that could be interpreted as indirect measures of incontinence-related burden, including (1) workload seen as a barrier to continence care,[24,25] (2) greater hours of care required when residents are incontinent,[26,27] (3) increased risk of caregiver injuries,[28,29] and (4) caregiver psychological stress.[30,31]

There is a lack of research on interventions to prevent UI and FI in caregiver-dependent individuals, including the impact of interventions on formal or informal caregiver burden. For the

majority of caregiver-dependent older individuals, interventions to prevent incontinence will include some type of toileting intervention. Evidence on the impact of toileting interventions on informal caregivers is very limited.[6,7,32] There is some research to suggest that toileting a caregiver-dependent older adult increases their workload,[6,7] but there is also evidence that a toileting program may reduce UI-related burden.[32] Jewart and colleagues examined the impact of anticholinergic medications on bladder function and caregiver burden in a small sample of 12 subjects. There were no significant differences in bladder function or caregiver burden prior to or after treating subjects with the anticholinergic medication (oxybutynin or tolterodine).[33] There is a need for research on measures to prevent FI and their impact on caregiver burden.

There is evidence that formal caregivers' adherence to toileting protocols is not adequate,[34–38] but the extent to which the noncompliance is related to the intervention's being a burden for staff is unknown. In addition, in all of these studies, the toileting program was implemented for residents with established UI. The use of regular toileting to prevent UI and FI has not been explored.

References

1. Kasuya RT, Polgar-Bailey P, Takeuchi R. Caregiver burden and burnout: a guide for primary care physicians. *Postgrad Med.* 2000;108:119–23.

2. National Alliance for Caregiving and AARP. Caregiving in the U.S. [National Alliance for Caregiving Web site]. Available at: http://www.caregiving.org/data/04finalreport.pdf. Accessed September 10, 2007.

3. Ouslander JG, Zarit SH, Orr NK, Muira SA. Incontinence among elderly community dwelling dementia patients: Characteristics, management, and impact on caregivers. *J Am Geriatr Soc.* 1990;38:440–445.

4. Noelker LS. Incontinence in elderly cared for by family. *Gerontologist.* 1987;27:194–200.

5. Langa KM, Fultz NH, Saint S, Kaberto MU, Herzog R. Informal caregiving time and costs of urinary incontinence in older individuals in the United States. *J Am Geriatr Soc.* 2002;50:733–737.

6. Flaherty JH, Miller DK, Coe RM. Impact of caregivers of supporting urinary function in noninstitutionalized chronically ill seniors. *Gerontologist.* 1992;32:541–545.

7. Engberg S, Sereika SM, McDowell BJ, Weber E, Brodak I. Effectiveness of prompted voiding in treating urinary incontinence in cognitively impaired homebound older adults. *J Wound Ostomy Continence Nurs.* 2002;29:252–265.

8. Grant J. Problems and associated feelings by family caregivers of stroke survivors during the second and third month of caregiving. *J Neurosci Nurs.* 2004;36:107–108.

9. Upton N, Reed V. The meaning of incontinence in dementia care. *Int J Psychiatr Nurs Res.* 2005;11:1200–1210.

10. Lim PPJ, Sahadevan S, Choo GK, Anthony P. Burden of caregiving in mild to moderate dementia: an Asian experience. *Int Psychogeriatr.* 1999;11:411–420.

11. Shinanoushi S, Kamei T, Hayashi M. Home care for the frail elderly based on urinary incontinence level. *Public Health Nurs.* 2000;17:468–473.

12. Onishi J, Suzuki Y, Umegaki H, Nakamura A, Endo H, Iguchi A. Influence of behavioral and psychological symptoms of dementia (BPSD) and environment of care on caregiver burden. *Arch Gerontol Geriatr.* 2005;41:159–168.

13. Tsuji I, Whalen S, Finucane TE. Predictors of nursing home placement in community-based long-term care. *J Am Geriatr Soc.* 1995;43:761–766.

14. Armstrong M. Factors affecting the decision to place a relative with dementia into residential care. *Nurs Stand.* 2000;14:33–37.

15. O'Donnell BF, Drachman DA, Barnes HJ, Peterson KE, Swearer JM, Lew RA. Incontinence and troublesome behaviors predict institutionalization in dementia. *J Geriatr Psychiatry Neurol.* 1992;5:45–51.

16. Tilvis RS, Hakala SM, Valvanne J, Erkinjuntt T. Urinary incontinence as a predictor of death and institutionalization in a general aged population. *Arch Gerontol Geriatr.* 1995;21:307–315.

17. Thom DH, Haan MN, VanDeen Eeden SK. Medically recognized urinary incontinence and risks of hospitalization, nursing home admission and mortality. *Age Ageing.* 1997;26:367–374.

18. Hope T, Keene J, Gelding K, Fairburn CG, Jacoby R. Predictors of institutionalization for people with dementia living at home with a carer. *Int J Geriatr Psychiatry.* 1998;13:682–690.

19. Thomas P, Ingrand P, Lalloue F, Hazif-Thomas C, Billon R, Vieban F, Clement JP. Reasons of informal caregivers for institutionalizing dementia patients previously living at home: The Pixel study. *Int J Geriatr Psychiatry.* 2004;19:127–135.

20. Bond MJ, Clark MS. Predictors of the decision to yield care of a person with dementia. *Australas J Ageing.* 2002;21:86–91.

21. Haupt M, Kurz A. Predictors of nursing home placement in patients with Alzheimer's disease. *Int J Geriatr Psychiatry.* 1993;8:741–746.

22. CDC/NCHS. Trends in nursing homes [National Center for Health Statistics Web site]. Available at: http://www.cdc.gov/nchs/data/nnhsd/nursinghomes1973-2004.pdf. Accessed October 5, 2007.

23. CDC. The changing profile of nursing home residents: 1985–1997 [Centers for Disease Control and Prevention Web site]. Available at: http://www.cdc.gov/nchs/data/ahcd/agingtrends/04nursin.pdf. Accessed October 5, 2007.

24. Lekan-Rutledge D, Palmer MH, Belyea M. In their own words: nursing assistants' perceptions of barriers to implementation of prompted voiding in long-term care. *Gerontologist.* 1998;38:370–378.

25. Mather KF, Bakas T. Nursing assistants' perceptions of their ability to provide continence care. *Geriatr Nurs.* 2002;23:76–81.

26. Cella M. The nursing costs of urinary incontinence in a nursing home population. *Nurs Clin North Am.* 1988;23:159–168.

27. Shih YCT, Hartzema AG, Tolleson-Rinehart S. Labor costs associated with incontinence in long-term care facilities. *Urology.* 2003;62:442–446.

28. Collins JW, Gwen BD. NIOSH research initiatives to prevent back injuries to nursing assistants, aides, and orderlies in nursing homes. *Am J Ind Med.* 1995;29:421–424.

29. Engst C, Chhokar R, Robinson D, Earthy A, Yassi A. Implementation of a scheduled toileting program in a long term care facility. *AAOHN J.* 2004;52:427–435.

30. Yu LC, Kaltreider DL. Stressed nurses dealing with incontinence patients. *J Gerontol Nurs.* 1987;13:27–30.

31. Jervis LL. The pollution of incontinence and the dirty work of caregiving in a U.S. nursing home. *Med Anthropol Q.* 2001;15:84–99.

32. Colling J, Owen TR, McCreedy M, Newman D. The effects of a continence program on frail community-dwelling elderly persons. *Urol Nurs.* 2003;23:117–131.

33. Jewart RD, Green J, Lu C, Cellar J, Tune LE. Cognitive, behavioral, and physiological changes in Alzheimer disease patients as a function of incontinence medications. *Am J Geriatr Psychiatry.* 2005;13:324–328.

34. Palmer MH, Bennett RG, Marks J, McCormick KA, Engel BT. Urinary incontinence: a program that works. *J Long Term Care Adm.* 1994;22:19–25.

35. Remsburg RE, Palmer MH, Langford AM, Mendelson GF. Staff compliance with and rating of effectiveness of a prompted voiding program in a long-term care setting. *J Wound Ostomy Continence Nurs.* 1999;26:261–269.

36. Campbell EB, Knight M, Benson M, Colling J. Effect of an incontinence training program on nursing home staff's knowledge, attitudes and behavior. *Gerontologist.* 1991;31:788–794.

37. Harke JM, Richgels K. Barriers to implementing a continence program in nursing homes. *Clin Nurs Res.* 1992;1:158–168.

38. Schnelle JF, Newman DR, Fogarty T. Management of patient continence in long-term care facilities. *Gerontologist.* 1990;30:373–376.

Quality of Life for Patients With Fecal Incontinence

Liliana Bordeianou, M.D.

Fecal incontinence (FI) is a common disease; its frequency is underreported by patients because of its associated social stigma.[1,2] One study found that the overall prevalence of this condition in U.S. outpatients is 18.4%.[3] Another study found even higher rates, approaching 21%, in the population of women presenting with urinary incontinence (UI) or pelvic prolapse.[4] In nursing home patients, the rates of this condition approach 47 % (a percentage that may reflect the fact that some of these patients are institutionalized for reasons relating to their incontinence in the first place).[5] Stratified by frequency of episodes, FI occurs daily in 2.7% of patients, weekly in 4.5%, and monthly or less in 7.1%.[4]

Although is often underreported and untreated, FI may have profound effects on patients. FI can present significant financial burdens (diapers, home care, nursing home care).[6] It is associated with various medical morbidities, including decubitus ulcers and urinary tract infections.[7] However, its most frequent impact is on quality of life.[8–10] Patients with FI often plan their life around easy and rapid access to toilet facilities. To accomplish this, they may curtail activities other members of society take for granted: shopping, going to the cinema, dining out, or having sexual intercourse.[11] They suffer from embarrassment, shame, and sometimes depression.[9] Not surprisingly, researchers using generic quality-of-life instruments, such as the Medical Outcomes Survey (SF-36), have found that patients with FI, on average, suffer a quality of life far below than that of the U.S. population as a whole, especially in the domains of vitality, social functioning, emotional role, and mental health.[10] Our review of published SF-36 studies in outpatients with various chronic diseases suggests that patients with incontinence have a qualify of life that is significantly lower than patients with, say, rheumatoid arthritis or diabetes, and equal to patients with inflammatory bowel disease.[10]

Fortunately, FI is not an “all or nothing” condition. Its severity may vary, from occasional (accidental) loss of gas control, to frank spillage of solid stool. Interestingly, while the severity of FI has an obvious impact on quality of life, it is not the only factor. In fact, in our study, the correlation between FI severity and quality of life, while present, was found to be rather weak.[10] Other factors that appear to affect an individual’s quality of life include the individual’s perception and experience of incontinence, as well as his or her age, medical comorbidities, financial status, and degree of adaptation to the condition.[12,13]

Because of this complex relationship between FI severity and quality of life, we suggest that at least two separate measurements be used to assess the burden of the disease and the impact of different treatment options targeting this condition. To assess severity of incontinence, one might consider utilizing, for example, the Fecal Incontinence Severity Index (FISI) developed by the American Society of Colon and Rectal Surgeons.[14] FISI addresses the leakage of gas, mucus, liquid or solid stool at varying frequencies. FISI assigns a cumulative subjective weighted score of 0–61 to each patient, where a value of 0 is no incontinence and 61 is incontinence to gas, liquid, mucus, and solid stool at least twice daily.

To assess quality of life, one might consider using, for example, the Fecal Incontinence Quality of Life Score (FIQL).[15] FIQL measures specific quality-of-life issues expected to affect patients with FI. The questions are grouped into four categories: lifestyle, coping/behavior,

depression/self perception, and embarrassment. Each category is scored 1–4 (1 = quality-of-life alteration present most of the time, 4 = none of the time).

FISI and FIQL are only two of a number of available scales, but they have been validated previously, and have become standard measurements in this area. In addition, their measurements have a relatively low degree of overlap.[16] However, whether these or other scales are used, the bottom line is the same: in assessing a particular treatment modality for the amelioration of FI, decisions should not be driven entirely by data suggesting incremental improvements in disease severity. Decisions also should be informed by an understanding of their potential impact on quality of life. Phrased another way: patients should not automatically be offered treatments that promise slight to moderate improvements in disease severity, unless these interventions also appear likely to produce some commensurate improvement in the patient's overall quality of life.

References

1. Whitehead WE, Wald A, Norton NJ. Priorities for treatment research from different professional perspectives. *Gastroenterology.* 2004;126:S180–S185.

2. Madoff RD, Parker SC, Varma MG, Lowry AC. Faecal incontinence in adults. *Lancet.* 2004;364(9434):621–632.

3. Johanson JF, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. *Am J Gastroenterol.* 1996;91:33–36.

4. Faltin DL, Sangalli MR, Curtin F, Morabia A, Weil A. Prevalence of anal incontinence and other anorectal symptoms in women. *Int Urogynecol J Pelvic Floor Dysfunct.* 2001;12:117–120; discussion 121.

5. Nelson R, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes: prevalence and associations. *Dis Colon Rectum.* 1998;41:1226–1229.

6. Mellgren A, Jensen LL, Zetterstrom JP, Wong WD, Hofmeister JH, Lowry AC. Long-term cost of fecal incontinence secondary to obstetric injuries. *Dis Colon Rectum.* 1999;42:857–865; discussion 865–867.

7. Parker SC, Thorsen A. Fecal incontinence. *Surg Clin North Am.* 2002;82:1273–1290.

8. Deutekom M, Terra MP, Dobben AC, Dijkgraaf MG, Baeten CG, Stoker J, Bossuyt PM. Impact of faecal incontinence severity on health domains. *Colorectal Dis.* 2005;7:263–269.

9. Rothbarth J, Bemelman WA, Meijerink WJ, Meijerink WJ, Stiggelbout AM, Zwinderman AH, Buyze-Westerweel ME, Delemarre JB. What is the impact of fecal incontinence on quality of life? *Dis Colon Rectum.* 2001;44:67–71.

10. Bordeianou L, Rockwood T, Baxter N, Lowry A, Mellgren A, Parker S. Does incontinence severity correlate with quality of life? Prospective analysis of 502 consecutive patients. *Colorectal Dis.* 2007; e-pub ahead of print.

11. Damon H, Dumas P, Mion F. Impact of anal incontinence and chronic constipation on quality of life. *Gastroenterol Clin Biol.* 2004;28:16–20.

12. Wilson M. The impact of faecal incontinence on the quality of life. *Br J Nurs.* 2007;16:204–207.

13. Varma MG, Brown JS, Creasman JM, et al. Fecal incontinence in females older than aged 40 years: who is at risk? *Dis Colon Rectum.* 2006;49:841–851.

14. Rockwood TH, Church JM, Fleshman JW, Kane RL, Mavrantonis C, Thorson AG, Wexner SD, Bliss D, Lowry AC. Patient and surgeon ranking of the severity of symptoms associated with fecal incontinence: the Fecal Incontinence Severity Index. *Dis Colon Rectum.* 1999;42:1525–1532.

15. Rockwood TH, Church JM, Fleshman JW, Kane RL, Mavrantonis C, Thorson AG, Wexner SD, Bliss D, Lowry AC. Fecal Incontinence Quality of Life Scale: quality of life instrument for patients with fecal incontinence. *Dis Colon Rectum.* 2000;43:9–16; discussion 16–17.

16. Baxter NN, Rothenberger DA, Lowry AC. Measuring fecal incontinence. *Dis Colon Rectum.* 2003;46:1591–1605.

Quality of Life for Patients With Urinary Incontinence

Jennifer T. Anger, M.D., M.P.H.

The goal of quality assurance in medicine is to preserve and improve patient care.[1,2] Quality-of-care research evaluates "the degree to which health services for individuals and populations increase the likelihood of desired health outcomes and are consistent with current professional knowledge."[3,4] Using the conceptual model established by Donabedian in 1966, medical quality comprises three components: structure, process, and outcome.[5]

Structure of care includes the equipment, resources, and provider experience necessary to provide care.[6] Structural measures for quality include characteristics such as physicians' board certification, volume of cases, and number of hospital beds available in a community. Process measures encompass the interactions between patients and practitioners and include interpersonal factors such as patient education, informed consent, and medical decisionmaking. Process of care also refers to the technical elements of care that transpire between doctor and patient, such as the extent of the history and physical examination, documentation of the work up, ordering of diagnostic and laboratory tests, as well as technical factors such as surgical technique. The recommendation and implementation of a treatment plan constitute an element of process of care and can serve as a measure of quality.

Outcome, in Donabedian's model, refers to a change in a patient's current or future health status that can be attributed to antecedent healthcare.[6] Outcomes consist of clinical variables such as survival rates, complications, health-related quality of life (HRQOL), satisfaction with care,[7] and health status.[3,4] Disease recurrence requiring a secondary treatment constitutes an element of outcomes and can also serve as a measure of quality. According to Donabedian, structural characteristics of the settings in which care takes place have a propensity to influence the process of care so that its quality is diminished or enhanced.[6] Similarly, changes in the process of care, including variations in its quality, will influence the effect of care on health status.[5]

Although a great deal of research on quality has been conducted in many areas of medicine and surgery, there is still a paucity of data on quality in the literature regarding the treatment of men and women with urinary incontinence (UI). Not only is there a need for the development of means to assess structure and process measures of quality of care for men and women with UI, but there is also a tremendous need to understand what specific outcomes a patient considers to be important. Pharmaceutical companies claim a high success rate with anticholinergic medications, the most commonly prescribed treatment for overactive bladder (OAB) symptoms and its associated urge UI. However, a large percentage of patients prescribed anticholinergic therapy actually stop taking their medication due to poor efficacy or problematic side effects including dry mouth, dry eyes, and constipation.[8,9]

Several variables are used to measure outcomes of care. These include mortality, complications, morbidity, objective clinical variables, and patient-centered outcomes, including HRQOL and patient satisfaction with care. Objective measurable clinical outcomes of UI treatment are multiple (Table 1). Outcomes measured are often a function of provider preference and practice patterns.

Table 1. Examples of Objective Clinical Outcomes of Incontinence Treatment

Outcomes of Medical or Surgical Treatment
Number of incontinence episodes per day or week
Number of voids per 24 hours
Volume voided
Subjective patient assessments: cured/improved/failed
Pad number/pad weight/pad size
Time to make it to toilet once urge starts
Percent urge versus percent stress incontinence episodes
Outcomes Specific to Surgical Treatment of Stress Incontinence
Surgical complications (bleeding, bladder injury)
Nonsurgical complications (pulmonary embolism, myocardial infarction)
New symptoms or findings of pelvic prolapse
Outlet obstruction/voiding difficulty/urinary retention
Urinary tract infection
“De novo” urge incontinence
Reoperation for incontinence
Reoperation for prolapse

In 1948, the World Health Organization defined health as “a state of complete physical, mental, and social well-being and not merely the absence of disease.”[10] HRQOL is a multidimensional construct that includes overall well-being, emotional well-being, somatic symptoms, functional ability, and social functioning.[11–13] HRQOL involves patients’ perceptions of their health and their ability to function in life.[14] Quality-of-life considerations are a critical factor in medical decisionmaking for UI.

Validated instruments are crucial in providing a comprehensive evaluation of how an illness and its treatment affect patients.[14] General HRQOL instruments include the RAND Medical Outcomes Study 36-Item Health Survey (SF-36),[15] the SF-12,[16] the Nottingham Health Profile,[17] and the Sickness Impact Profile,[18] to name a few. The many validated instruments used to assess general HRQOL encompass several domains, including physical, emotional, and social functioning. Unlike general HRQOL instruments, disease-specific quality-of-life instruments have been developed to measure the impact of a specific disease on HRQOL. Many such instruments for UI have been developed (Table 2) and are an important means of assessing the impact of UI on HRQOL.

Table 2. Validated Questionnaires Highly Recommended by the Third International Consultation on Incontinence[19,20]

I. Symptoms of UI	
A. Women	Urogenital Distress Inventory (UDI)[21]
	Urogenital Distress Inventory Short Form (UDI-6)[22]
	Incontinence Severity Index[23]
	The Bristol Female Lower Urinary Tract Symptoms (BFLUTS)[24]
B. Men	International Continence Society-male (ICSmale)[25]
	Danish Prostate Symptom Score (DAN-PSS)[26]
II. Quality-of-Life Impact of UI	
A. Men and women	Incontinence-Quality of Life (I-QOL)[27]
	Stress-Related Leak, **E**mptying Ability, **A**natomy, **P**rotection, **I**nhibition, **Q**uality of Life, **M**obility, and **M**ental Status (SEAPI-QMM)
B. Women	King's Health Questionnaire[28]
	Incontinence Impact Questionnaire (IIQ)[29]
	Incontinence Impact Questionnaire Short Form (IIQ-7)[22]
	Urinary Incontinence Severity Score (UISS)[30]
	CONTILIFE[31]
III. Combined Symptoms and Quality-of-Life Impact of UI	
A. Men and women	International Consultation on Incontinence Questionnaire (ICIQ)[32]
B. Women	Bristol Female Lower Urinary Tract Symptoms-Short Form (BFLUTS-SF)[33]
	Stress and Urge Incontinence and Quality-of-Life Questionnaire (SUIQQ)[34]
C. Men	ICSmale Short Form (ICSmaleSF)[25]
IV. Combined Symptoms and Quality-of-Life Impact of Overactive Bladder (OAB)	
A. Men and women	OAB-q[35]

Although some diseases affect both general and disease-specific quality of life, the effect of UI on general HRQOL is controversial. In a comparison of general HRQOL of 120 incontinent women and 313 age-matched controls, as measured by the Nottingham Health Profile, Grimby and colleagues found that women with stress or urge UI were much more likely to experience social isolation, emotional disturbances, and, in the case of women with OAB, sleep disturbances.[36] In contrast, Hunskaar and colleagues found that UI had a relatively low impact on general HRQOL among 70 self-referred incontinent women. However, they did find that women with urge UI demonstrated greater impairment in HRQOL than women with stress UI. Also, younger women were more affected in several domains, including mobility, sleep, emotional behavior, and social interactions.[37]

Controversy exists about the relationship between UI severity and patient-perceived HRQOL. In a study of HRQOL among incontinent community-dwelling adults, Wyman and colleagues found that neither incontinence severity nor duration correlated well with HRQOL impairment.[38] In a second study by Wyman and colleagues, the IIQ was used to measure the psychosocial impact of UI in 69 community-dwelling women aged 55 and older. They found significantly greater QOL impairment in women with urge UI (with or without stress UI) compared with women with stress UI alone. There were only modest correlations between psychosocial impact and both the number of incontinence episodes and leakage volume.[29] The unpredictability of leakage episodes, as occurs with OAB, may have a large impact on HRQOL for adults with UI.

A great deal of progress has been made in the area of UI-related health services research. The literature suggests that OAB/urge UI has a more profound impact on quality of life than stress UI. However, more research is needed to determine how much of the variation in overall HRQOL is explained by variation in UI-specific domains. In addition, outcomes of OAB treatment remain suboptimal. Despite the multitude of validated questionnaires available, one large problem still exists: It is not known what outcomes of treatment matter most to the patient.

References

1. Spencer BA, Steinberg M, McGlynn EA, Brumberger E, Litwin MS. Quality of care indicators for conventional and conformal external-beam radiation therapy in the treatment of early stage prostate cancer. *Int J Radiat Oncol Biol Phys.* 2003;57:S420–S421.

2. Miller DC, Litwin MS, Sanda MG, Montie JE, Dunn RL, Resh J, Sandler H, Wei JT. Use of quality indicators to evaluate the care of patients with localized prostate carcinoma. *Cancer.* 2003;97:1428–1435.

3. Spencer BA, Steinberg M, Malin J, Adams J, Litwin MS. Quality-of-care indicators for early-stage prostate cancer. *J Clin Oncol.* 2003;21:1928–1936.

4. Lohr K. *Medicare: A Strategy for Quality Assurance.* Washington, DC. National Academy Press, 1990.

5. Donabedian A. Evaluating the quality of medical care. *Milbank Mem Fund Q.* 1966;44(Suppl):166.

6. Donabedian A. Basic approach to assessment: structure, process, and outcome. In: *Explorations in Quality Assessment and Monitoring. Vol. 1.* Ann Arbor, MI: Health Administration Press; 1980;3:77–128.

7. Institute of Medicine Committee on Quality of Health Care in America. *Crossing the Quality Chasm: A New Health System for the 21st Century.* Washington, DC: National Academy Press; 2001:1–22.

8. Dmochowski, R. Improving the tolerability of anticholinergic agents in the treatment of overactive bladder. *Drug Saf.* 2005;28:583–600.

9. Dmochowski RR, Starkman JS, Davila GW. Transdermal drug delivery treatment for overactive bladder. *Int Braz J Urol.* 2006;32:513–520.

10. WHO. *Constitution of the World Health Organization, Basic Documents.* Geneva: WHO, 1948.

11. Guyatt GH, Naylor CD, Juniper E, Heyland DK, Jaeschke R, Cook DJ. Users' guides to the medical literature. XII. How to use articles about health-related quality of life. Evidence-Based Medicine Working Group. *JAMA.* 1997;277:1232–1237.

12. Guyatt GH, Bombardier C, Tugwell PX. Measuring disease-specific quality of life in clinical trials. *CMAJ.* 1986;134:889–895.

13. Cella DF, Bonomi AE. Measuring quality of life: 1995 update. *Oncology (Williston Park).* 1995;9:47–60.

14. Litwin MS. Outcomes research. In: Campbell MF, Wein AJ, Kavoussi LR, eds. *Campbell-Walsh Urology.* 9th ed. Philadelphia: W.B. Saunders; 2007:144–157.

15. Ware JE Jr, Sherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med Care.* 1992;30:473–483.

16. Ware J Jr, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care.* 1996;34:220–233.

17. Hunt SM, McEwen J, McKenna SP. Social inequalities and perceived health. *Eff Health Care.* 1985;2:151–160.

18. Bergner M, Bobbitt RA, Carter WB, Gilson BS. The Sickness Impact Profile: development and final revision of a health status measure. *Med Care.* 1981;19:787–805.

19. Nitti VW, Blaivas JG. Urinary incontinence: epidemiology, pathophysiology, evaluation, and management overview. In: Campbell MF, Wein AJ, Kavoussi LR, eds. *Campbell-Walsh Urology.* 9th ed. Philadelphia: W.B. Saunders; 2007:2046–2078.

20. Donovan J, Bosch R, Gotoh M, et al. Symptom and quality of life assessment. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence: Third International Consultation on Incontinence.* UK: Health Publications; 2005:519–584.

21. Shumaker SA, Wyman JF, Uebersax JS, McClish D, Fantl JA. Health-related quality of life measures for women with urinary incontinence: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Continence Program in Women Research Group. *Qual Life Res.* 1994;3:291–306.

22. Uebersax JS, Wyman JF, Shumaker SA, McClish DK, Fantl JA. Short forms to assess life quality and symptom distress for urinary incontinence in women: the Incontinence Impact Questionnaire and the Urogenital Distress Inventory. Continence Program for Women Research Group. *Neurourol Urodyn.* 1995;14:131–139.

23. Sandvik H, Hunskaar S, Seim A, Hermstad R, Vanvik A, Bratt H. Validation of a severity index in female urinary incontinence and its implementation in an epidemiological survey. *J Epidemiol Community Health.* 1993;47:497–499.

24. Jackson S, Donovan J, Brookes S, Eckford S, Swithinbank L, Abrams P. The Bristol Female Lower Urinary Tract Symptoms questionnaire: development and psychometric testing. *Br J Urol.* 1996;77:805–812.

25. Donovan JL, Abrams P, Peters TJ, Kay HE, Reynard J, Chapple C, De La Rosette JJ, Kondo A. The ICS-'BPH' Study: the psychometric validity and reliability of the ICSmale questionnaire. *Br J Urol.* 1996;77:554–562.

26. Hald T, Nordling J, Andersen JT, Bilde T, Meyhoff HH, Walter S. A patient weighted symptom score system in the evaluation of uncomplicated benign prostatic hyperplasia. *Scand J Urol Nephrol Suppl.* 1997;138:59–62.

27. Wagner TH, Patrick DL, Bavendam TG, Martin ML, Buesching DP. Quality of life of persons with urinary incontinence: development of a new measure. *Urology.* 1996;47:67–71; discussion 71–72.

28. Kelleher CJ, Cardozo LD, Khullar V, Salvatore S. A new questionnaire to assess the quality of life of urinary incontinent women. *Br J Obstet Gynaecol.* 1997;104:1374–1379.

29. Wyman JF, Harkins SW, Choi SC, Taylor JR, Fantl JA. Psychosocial impact of urinary incontinence in women. *Obstet Gynecol.* 1987;70:378–381.

30. Stach-Lempinen B, Kirkinen P, Laippala P, Metsänoja R, Kujansuu E. Do objective urodynamic or clinical findings determine impact of urinary incontinence or its treatment on quality of life? *Urology.* 2004;63:67–71; discussion 71–72.

31. Amarenco G, Arnould B, Carita P, Haab F, Labat JJ, Richard F. European psychometric validation of the CONTILIFE: a Quality of Life questionnaire for urinary incontinence. *Eur Urol.* 2003;43:391–404.

32. Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P. ICIQ: a brief and robust measure for evaluating the symptoms and impact of urinary incontinence. *Neurourol Urodyn.* 2004;23:322–330.

33. Brookes ST, Donovan JL, Wright M, Jackson S, Abrams P. A scored form of the Bristol Female Lower Urinary Tract Symptoms questionnaire: data from a randomized controlled trial of surgery for women with stress incontinence. *Am J Obstet Gynecol.* 2004;191:73–82.

34. Kulseng-Hanssen S, Borstad E. The development of a questionnaire to measure the severity of symptoms and the quality of life before and after surgery for stress incontinence. *BJOG.* 2003;110:983–988.

35. Coyne K, Revicki D, Hunt T, Corey R, Stewart W, Bentkover J., Kurth H, Abrams P. Psychometric validation of an overactive bladder symptom and health-related quality of life questionnaire: the OAB-q. *Qual Life Res.* 2002;11:563–574.

36. Grimby A, Milsom I, Molander U, Wiklund I, Ekelund P. The influence of urinary incontinence on the quality of life of elderly women. *Age Ageing.* 1993;22:82–89.

37. Hunskaar S, Vinsnes A. The quality of life in women with urinary incontinence as measured by the Sickness Impact Profile. *J Am Geriatr Soc.* 1991;39:378–382; erratum in *J Am Geriatr Soc.* 1992;40:976–977.

38. Wyman JF, Harkins SW, Fantl JA. Psychosocial impact of urinary incontinence in the community-dwelling population. *J Am Geriatr Soc.* 1990;38:282–288.

Evidence-Based Practice Center Presentation I: Prevalence, Incidence, and Risk Factors for Fecal Incontinence

Donna Z. Bliss, Ph.D., R.N., F.A.A.N., F.S.G.A., Tatyana Shamliyan, M.D.

Research on the epidemiology of fecal incontinence (FI) and dual (both fecal and urinary) incontinence has lagged behind that of urinary incontinence (UI). The absence of a standard and accepted definition of FI has hampered drawing conclusions about the epidemiology of both fecal and dual incontinence. Definitions vary by the inclusion of flatus, severity characteristics of FI, and subjective significance (e.g., requiring FI to be a social or hygienic problem). There is often a lack of data to determine whether dual incontinence is actually present. In this review, prevalence and incidence are reported for any FI (i.e., with or without UI), any anal incontinence (i.e., FI and flatus incontinence), dual incontinence, and FI only (without UI) when data are available. Associated factors and risks are reported for "any FI" (referred to hence as FI) when adjustment for other factors occurred. Prevalence is the proportion of individuals in a population having a certain condition or disease at a given point in time. Incidence is the number of new occurrences of a condition (or disease) in a population over a period of time.

Studies reported from 1989 through May 2007 were reviewed from a MEDLINE via PubMed, CINAHL, Cochrane databases, and manual searches of reference lists from systematic reviews and the proceedings of the International Continence Society. Eligible studies were large cross-sectional or cohort studies or case series of more than 100 subjects.

Prevalence in the Community

The prevalence of FI, anal, and dual incontinence could be pooled for men and women of different age groups living in the community (Table 1). The prevalence of FI and dual incontinence increased with age in both men and women. The prevalence of anal incontinence also increased with age in women, but this association could not be determined in men because of a lack of data. More women than men had FI at younger ages, but among the oldest, the prevalence between men and women was similar.

The prevalence of dual incontinence was similar between men and women under 65 years of age, higher in women 65+ years of age, and higher in men 80+ years of age. Although no study investigated FI vs. anal incontinence in the same sample, there was a consistent pattern of a higher prevalence (two- to four-fold) of anal incontinence than that of FI in women across age categories. This pattern suggests that combining incontinence of flatus and feces in the same definition may contribute to increased prevalence estimates. In a study in Japan, the age-adjusted prevalence of "FI only" was 2.1%.

Table 1. Community Prevalence of Fecal, Anal, and Dual Incontinence by Age and Sex

Type	Age in years	% Women (# of studies)		95% CI	% Men (# of studies)		95% CI
AI	19–44	22	(3)	16–28			
AI	45–64	17	(3)	10–24	2	(2)	0.03–3
AI	65+	33	(1)	27–39			
AI	80+	45	(1)	45–45			
FI	45–64	7	(5)	5–9	6	(8)	5–8
FI	65+	9	(3)	7–10	7	(12)	5–9
FI	80+	10	(3)	7–12	10	(3)	4–15
FI+UI	45–64	10	(3)	2–21	10	(2)	5–14
FI+UI	65+	10	(2)	8–12	8	(2)	6–10
FI+UI	80+	12	(2)	10–15	16	(2)	12–19
FI of liquid feces	45–64	7	(12)	5–8	3	(3)	2–5
FI of liquid feces	80+	1.5	(1)	1.5–1.5			
FI of solid feces	45–64	4	(14)	3–4	1	(3)	0.4–2
FI of solid feces	80+	2	(1)	3–4	2	(1)	2–2

AI=anal incontinence, FI+UI=dual incontinence, CI=confidence interval

Prevalence in Nursing Homes

In a large cross-sectional study of older nursing home (NH) residents in the United States, there was a 40% prevalence of dual incontinence. Almost one-half of the residents in Canadian long-term care hospitals had dual incontinence. The prevalence of dual incontinence was lower in NHs in the UK (4%) and in short-stay NHs (9%). The rate of FI ranged from 4% to 52%. Differences in FI prevalence were noted in subgroups of residents. Frail residents (16%), stroke survivors (25%), and those with dementia (45%) had the highest prevalence of frequent FI. In one study, the prevalence of "FI only" was 12.4%.

Severity of FI: The prevalence of incontinence of liquid feces as well as solid feces in middle-aged women was as least twice as high as in middle-aged men. The rate of incontinence of solid stool was similar in men and women aged 80+ years.

Incidence of FI

There were no studies of true incidence of FI or dual incontinence in adults living in the community or NHs. One study did report the cumulative incidence of FI in a cohort of old (65+ years of age) community-living adults. Overall, 7% of women developed FI after 5 years compared to 6% of men, but 27% of women had FI after 10 years compared to only 4% of men. The greatest difference between men and women occurred after 5 years in those 85+ years of age, in whom 34.4% of women compared to 13.3% of men had FI. More women than men in every age group had dual incontinence, with the greatest difference (2.4 times) occurring after 10 years in those 85+ years of age.

Factors Associated with FI

Inherent Factors (Age, Gender, and Race): In adults in the community, six of eight studies reported a significant association of age and FI; the two studies that did not show a significant association were of women after high-risk delivery and stroke survivors. The association of age and FI was no longer significant in NH residents, most of whom are elderly. The odds ratio (OR) of FI increased 1.3 times per decade in women in the community. The odds ratio for FI in postpartum women aged 35 years or more was 1.75. Men aged 85+ years were 3.41 times more likely to have FI than men aged 65–74 years. Two studies showed that men in the community were more likely than women to have FI, but two other studies showed no association between gender and FI. One study showed that being male in a NH increased the OR of having FI 1.2 times. One study showed that the odds of Asian women having birth-related FI were about 3 times higher than non-Asian women. In NH residents, non-White residents were twice as likely as Whites to have FI or dual incontinence.

Lifestyle Factors: There were no significant associations between body mass index (BMI) and FI in women in the community or in adults in NHs. Smoking, coffee drinking, or exercise were not significantly associated with prevalent or the cumulative incidence of FI in women in the community. Men in the community who drank coffee, compared to non-coffee drinkers, were twice as likely to have prevalent FI, but there was no association of coffee drinking and the cumulative incidence of FI. Men who smoked less than one pack per day were less likely, compared to nonsmokers, to have prevalent FI, but no association with smoking was found in the cumulative incidence of FI.

Bowel Pattern: Having loose/liquid stool consistency or diarrhea, but not constipation, was significantly associated with FI in community adults. In a NH study, diarrhea (OR=2.4, 95% CI=1.9–3), constipation (OR=1.3, 95% CI=1.2–1.4), and fecal impaction (OR=2.4, 95% CI=1.3–3.3) were associated with FI. FI was five times as likely to occur when people in the community experienced defecation urgency.

Functional and Cognitive Status: Mobility problems and living alone increased the likelihood of having FI 1.5 to 2.4 times, respectively, in people in the community. For stroke survivors in the community, impaired vision, functional limitations, or dysphagia increased the odds of FI 2 to 3.5 times. In NH residents, trunk restraints, tube feeding, limitations in activities of daily living (ADL), impaired vision, and decreased mobility were all factors associated with FI. Dementia was associated with FI in all studies of NH residents and in two of three studies in people living in communities. The odds of FI increased with more severe cognitive impairment; persons with an MMSE score <15 had a 250% increase in FI rates.

Obstetric Factors: Neither vaginal delivery nor Caesarean section (C-section) was significantly associated with FI; C-section was not associated with a significantly lower odds of FI. Single studies showed that birthweight, parity, or vaginal delivery after sphincter tear did not significantly increase odds of FI. Additional studies of parity and birthweight used the definition of anal incontinence, and the studies' findings were inconsistent. Use of forceps was associated with FI in one of two studies. Obstetrical damage to the anal sphincter increased the odds of FI by 200% to 300%.

Health and Comorbidities: A higher comorbidity index or self-perception of poor health by people in the community was associated with FI. Women in the community who had UI were approximately two times as likely to have FI as those without UI. NH residents aged 65+ years with UI were three times as likely to have FI, while those aged 85+ years with UI were

11–12 times as likely to have FI. In one study of stroke survivors in the community, UI was associated with six times higher odds of FI. Another study showed UI was significantly associated with FI (OR=18, 95% CI=9–37) only in the acute phase after stroke (days 7–10) but not at 3 months. Single studies showed that women with irritable bowel syndrome (IBS) or after menopause or hysterectomy with oophorectomy were approximately twice as likely to have FI. The odds for FI were highest after prostatectomy for prostate cancer (5.3), followed by prostate cancer (3.1), then by prostate disease (2.3).

Three studies of older adults and one of stroke survivors showed that diabetes increased the likelihood of FI by 1.7 to 3.1 times. Diabetes was not significantly related to FI in a community population with a mean age of 53 years or in NH residents; it was not a risk for the cumulative incidence of FI in older people in the community. Major depression was associated with a 273% increase in the rate of FI in women in the community in one of two studies and with a 283% higher increase in the rate in older men in the community; no association of depression and FI was found in NH residents.

Factors Associated With Fecal Incontinence Severity

Several factors related to bowel patterns were significantly associated with an increased prevalence of liquid feces: frequent diarrhea, loose/liquid stool consistency, and defecation urgency. Having UI, dementia, and surgery for hemorrhoids were other factors. Having defecation urgency always was the strongest factor associated with incontinence of solid stool (OR=20, 95% CI=3–11). Some factors associated with FI of solid feces were similar to those for liquid feces: having UI, dementia, and rectal surgery. Being male and having limited mobility were additional factors associated with FI of solid feces. Straining on defecation was protective for incontinence of both liquid and solid feces.

Greater frequency of FI was most strongly associated with defecation urgency all of the time (OR=11, 95% CI=5–26). However, any level of defecation urgency, loose/liquid feces, taking stool softeners for constipation, having hemorrhoids, a history of bowel resection, and UI were other factors. One study reported that a C-section lowered the odds of a greater frequency of FI, and use of forceps during delivery increased them.

The following bowel factors increased the odds of a greater amount of FI: defecation urgency some, most, or all of the time; loose/liquid feces; and history of a bowel resection. Other significant factors were having UI, mild or moderate memory problems, low back pain; and perceiving oneself as overweight.

Limitations

Variations in definitions of FI and its severity, few population-level studies with multivariate analyses, differences in samples, and inconsistency in factors adjusted in statistical modeling prevent firm conclusions. Pooled estimates and meta-analysis procedures could not be conducted in many instances. Data were inconsistently reported for FI severity characteristics (frequency, amount, consistency of leakage, and duration), and analyses of associated factors were few, so knowledge is limited. Use of a standard definition of FI that excludes flatus and determination of a minimum set of variables to be collected and used in multivariate analyses are recommended.

Conclusions

Age is strongly associated with FI in adults living in the community; other significantly associated factors include comorbidities, dementia, and stroke. Limited data suggest differences in factors associated with FI in men vs. women and in Asian women, but additional research is needed on the influence of gender and race on FI and its severity. Factors associated with FI that appear modifiable include functional and ADL limitations, restraint use, obstetrical anal sphincter damage, UI, diarrhea/loose stools, urgency and constipation/impaction in NH residents. Performing C-sections to prevent FI is not supported. Studies of FI incidence and risk factors are greatly needed.

General References

1. Bliss DZ, Fischer LR, Savik K, Avery M, Mark P. Severity of fecal incontinence in community-living elderly in a health maintenance organization. *Res Nurs Health.* 2004;27:162–173.

2. Harari D, Coshall C, Rudd AG, Wolfe CD. New-onset fecal incontinence after stroke: prevalence, natural history, risk factors, and impact. *Stroke.* 2003;34:144–150.

3. Macarthur C, Glazener C, Lancashire R, Herbison P, Wilson D, Grant A. Faecal incontinence and mode of first and subsequent delivery: a six-year longitudinal study. *BJOG.* 2005;112:1075–1082.

4. Nelson R, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes: prevalence and associations. *Dis Colon Rectum.* 1998;41:1226–1229.

Evidence-Based Practice Center Presentation II: Prevalence, Incidence, and Risk Factors for Urinary Incontinence

Jean F. Wyman, Ph.D., R.N., F.A.A.N., F.S.G.A., Tatyana Shamliyan, M.D.

Numerous epidemiological studies have led to advances in knowledge related to the prevalence and correlates of urinary incontinence (UI) in community-based and long-term-care adult populations. However, few studies have examined the development and natural history of UI, thus hampering understanding of how to best prevent its occurrence. Challenges in interpreting widely varying rates for UI, UI types, and UI severity, and in identifying potential risk factors can be attributed to differences in the definitions used for UI, study populations, sampling methods, measurement techniques, and the type of risk factors studied. Studies reported from 1990 through May 2007 were reviewed from MEDLINE via PubMed, CINAHL, Cochrane databases, and manual searches of reference lists from systematic reviews and the proceedings of the International Consultation on Incontinence. Eligible studies were large cross-sectional or longitudinal studies and case-control studies with more than 100 subjects.

Prevalence, Incidence, Progression, and Remission of Urinary Incontinence in Community-Based Populations

Prevalence. UI is a highly prevalent condition in the United States and worldwide. Prevalence rates for adult, community-based populations in the United States, Europe, and Asia range from 9% to 37% (combined men and women), with rates varying according to gender, age, and race/ethnicity. Women have higher rates than men, with estimates ranging from 8% to 44%, with daily UI reported by 7% to 32%, weekly UI by 11% to 36%, and monthly UI from 13% to 44%. UI in men is estimated at 3% to 39%, with 3% reporting daily UI, 3% to 8% weekly UI, and 15% monthly UI (one study only). In general, the prevalence of UI increases linearly with advancing age for both women and men, with adults aged 60 years and over or aged 80 and over having the highest rates as well as highest incontinence severity. The majority of population-based studies in the United States report a higher prevalence of UI in White women compared to Black, Hispanic, or Asian women. Similarly, non-Hispanic men were more likely than Hispanic men to have UI. There are inconsistent findings with respect to race in men. In one study, Black men had a higher rate of UI than White men, and in another study White men and American Indians had higher rates than Asians and Black men.

The prevalence of the different types of UI (stress, urge, mixed incontinence) also varies by age and gender. Stress and mixed UI are the most common types of incontinence in population-based samples of women. In incontinent women, rates for stress UI range from 14% to 63%, 6% to 66% for urge incontinence, and 20% to 62% for mixed incontinence. Most studies report higher rates of stress UI in young and middle-aged women, with higher rates of urge UI in older women. White women tend to have higher rates of UI and stress UI than Black, Hispanic, or Asian women. Black women are more likely to have higher rates of urge UI and mixed UI compared to Whites and Hispanics. In men, urge UI is the predominant type, followed by mixed UI and stress UI. In a large national survey of American men, rates for stress, urge, mixed, and other types of UI were 45%, 25%, 19%, and 12%, respectively, with older men more likely to report urge UI than younger men.

Incidence. Incidence rates also vary by age, gender, and race/ethnicity, with incidence rates being significantly higher in women and those in older age groups. One-year incidence rates in the adult population aged 40 years and over were reported at 6%. In American adults aged 65 and over, the 2-year incidence rate was 20%. One-year incidence rates in women varied from less than 1% in Norwegian women aged 50–74 years to 26% in American women aged 20–84 years, and 4% in UK men aged 40 to more than 20% in American men aged 60 years and over. There tends to be a consistent pattern of annual cumulative incidence rates across studies, with women having rates averaging between 1% to 4% per year. In the limited data available, incidence rates were similar between White and Black women. In a survey of American women 40–55 years of age, the 5-year cumulative incidence rates were highest for stress UI, followed by urge UI, and mixed UI. In this same survey, Whites and Japanese-American women had the highest incidence of stress UI compared to Chinese, Hispanic, and Black women. White women also had the highest incidence of urge UI; however, Black women had a higher incidence than Chinese, Japanese, and Hispanic women, respectively. Black women also had the highest incidence of mixed UI, followed by White, Chinese, Hispanic, and Japanese women. Data on the incidence rates of the different types of UI or comparisons by race/ethnicity are not available for men.

Progression and Remission. Data available on progression/remission rates indicate that UI is a dynamic condition, although remission rates tend to be relatively low. Evidence in adults aged 60 and over suggests that changes in UI severity over a 2-year period progress from continence to mild UI and from mild to moderate UI; few people advanced to severe UI. In a Canadian study of adults aged 65 years and over, 5- and 10-year incidence rates were 18% and 32% in women and 7% and 21% in men, respectively. Women and men may have different progression patterns. In one study involving adults 60 years of age and over, with a 2-year follow-up, women tended to develop stress UI and mixed UI as a primary condition, with urge UI as a secondary condition, whereas men developed urge UI, with stress UI as a potential secondary condition.

UI remission rates vary by gender, with women having more stable incontinence (e.g., lower remission rates) than men. In a large survey of UK adults 40 years of age and over, remission rates for both men and women across all age groups was 29%. Women had lower remission rates than men, with rates averaging 25%, whereas men had remission rates averaging 40%. Remission rates tended to decrease with age in women and men.

Prevalence, Incidence, Progression, and Remission of Urinary Incontinence in Long-Term-Care Populations

Prevalence. Prevalence estimates of UI in the overall long-term-care population are reported at 30%–65%; rates increase with advancing age in both women and men. The rates in women long-term-care residents are 74%–85% and in men 23%–72%. No evidence is reported on the prevalence of the different UI types, and limited data are available on racial/ethnic differences. In one large study involving long-term-care facilities in eight States, prevalence of UI was higher in Blacks than in Whites at admission, but after admission the rate became similar.

Incidence. Evidence is limited on the incidence of UI in nursing homes. One study reported UI incidence to be 27% at 2 months after admission and 19%–20% at 1 year.

Progression and Remission. Few studies on progression and remission rates are available in long-term-care settings. A small study reported a remission rate at 1 year after nursing home admission to be 10%.

Associated and Potential Risk Factors for Urinary Incontinence

Most of the evidence on risk factors comes from cross-sectional studies analyzing the association of selected variables with UI prevalence; limited data are available from longitudinal studies documenting which risk factors predict UI.

Genetic Factors. Family history of UI is associated with prevalent UI in women, with genetic factors most associated with the risk of prevalent stress UI and mixed UI.

Lifestyle Factors. Coffee was not associated with prevalent or incident UI in women, whereas tea drinking was associated. In contrast, drinking two cups of coffee was protective to having prevalent UI in men, whereas three cups or more was not protective. Carbonated beverages increased the odds of incident stress UI by 62% in women. Alcohol consumption was not associated with incident or prevalent UI in women, although one to six beverages/day compared to no alcoholic beverages was associated with prevalent UI in men. Smoking was associated with UI in men, and current smoking was associated with moderate to severe UI in women.

Occupational Status. Studies examining the effect of occupations with heavy lifting or labor on prevalent UI have not reported an association. However, women who do work in production facilities had higher rates of UI than those in academic positions.

Functional Status. Activity of daily living (ADL) impairments, decreased physical function, and cognitive impairment are strong predictors of UI and UI severity in women in the community and in long-term-care populations. ADL living impairments, physical dependency, and cognitive impairment are highly predictive of UI in long-term-care populations. Poor social support was associated with prevalent and incident UI in women aged 40–55 years.

Obesity. A higher body mass index (BMI) was associated with prevalent and incident UI in women and with prevalent UI in men.

Female Factors. Parity is a strong predictor of UI in women who are under 60 years of age, with women who have given birth or have had a greater number of childbirths being at increased odds of having UI. This effect tends to decrease with aging. Parity tends to increase the risk of stress UI but not urge UI. Vaginal delivery is associated with higher rates of UI, but this effect also tends to diminish with time. Spontaneous vaginal delivery, compared to C-section, is strongly associated with UI, with increased odds for developing each UI type. However, this effect tends to diminish for women aged 50 and older. There is some evidence that oxytocin increased the odds of having prevalent UI. Episiotomy and epidural analgesia were not related to UI. Studies are conflicting on the role of menopause. Some studies reported that menopause raised the risk for prevalent UI by 15% to 27%, but other studies did not report an association.

Urological Factors. Childhood enuresis and daytime incontinence were associated with an increased risk of urge UI in women. There is conflicting information on the role of lower urinary tract symptoms on UI in adulthood in women. Urinary tract infections tend to increase the odds of UI in women 20 years of age and over. Less is known about urological factors as a risk factor for UI in men.

Medical Comorbidities. Stroke is associated with prevalent UI and predicts incident UI. Some studies report that arthritis also is a predictor of UI and UI type. In most studies, cardiovascular disorders are not associated with or do not predict UI, although there is some conflicting

evidence, particularly with hypertension. Diabetes appears to be associated with prevalent UI in most studies in women and men (including stress, urge, and mixed UI in women), but diabetes was not a risk factor for incident UI in one study. The severity of diabetes may also be a risk factor. Other neurological disorders, such as Parkinson's disease, multiple sclerosis, and epilepsy, significantly increase the risk of UI (either/or prevalent and incident in most studies) in community and long-term-care settings. Constipation increases the odds of prevalent UI in women and predicts UI at 1 year postpartum.

Medications. Diuretics, hormone replacement therapy, topical estrogen treatment, and antidepressant use increased the risk of prevalent UI in women. Other medications were not associated.

Conclusions

Despite attempts by the International Consultation on Incontinence and the International Continence Society, there has been little consistency in the definitions and measurement of UI and other variables in epidemiological studies. Knowledge on risk factors that predict UI is limited by the lack of longitudinal designs and secondary data analyses of large population-based studies in which UI was not a primary focus. Standardization of terminology, potential risk factors, outcomes measured, and reporting methods is greatly needed to increase understanding of the development and natural history of UI. Future research is still needed on the incidence, severity, progression/remission, and risk factors of UI in community and long-term-care settings. Multivariate analyses are needed that can help determine the influence of age, gender, race/ethnicity, genetics, childhood voiding patterns, lifestyle factors, medications, medical conditions, and obstetric/gynecological factors. Potential modifiable risk factors in women are obesity, physical limitations, obstetrical anal sphincter rupture, diabetes, and constipation. Potential modifiable factors in men are caffeine intake, alcohol, smoking, physical activity, and obesity. Some evidence suggests that early prevention studies might be indicated in women with childhood voiding dysfunctions and familial risk factors.

General References

1. Boyington JE, Howard DL, Carter-Edwards L, Gooden KM, Erdem N, Jallah Y, Busby-Whitehead J. Differences in resident characteristics and prevalence of urinary incontinence in nursing homes in the Southeastern United States. *Nurs Res.* 2007;56:97–107.

2. Diokno AC, Estanol MV, Ibrahim IA, Balasubramaniam M. Prevalence of urinary incontinence in community dwelling men: a cross sectional nationwide epidemiological survey. *Int Urol Nephrol.* 2007;39:129–136.

3. Finkelstein MM. Medical conditions, medications, and urinary incontinence. Analysis of a population-based survey. *Can Fam Physician.* 2002;48:96–101.

4. Jackson RA, Vittinghoff E, Kanaya AM, Miles TP, Resnick HE, Kritchevsky SB, Simonsick EM, Brown JS; Health, Aging, and Body Composition Study. Urinary incontinence in elderly women: findings from the Health, Aging, and Body Composition Study. *Obstet Gynecol.* 2004;104:301–307.

5. Melville JL, Katon W, Delaney K, Newton K. Urinary incontinence in US women. A population-based study. *Arch Intern Med.* 2005;165:537–542.

6. Ouslander JG, Palmer MH, Rovner BW, German PS. Urinary incontinence in nursing homes: incidence, remission, and associated factors. *J Am Geriatr Soc.* 1993;41:1083–1089.

7. Waetjen LE, Liao S, Johnson WO, Sampselle CM, Sternfield B, Harlow SD, Gold EB. Factors associated with prevalent and incident urinary incontinence in a cohort of midlife women: a longitudinal analysis of data: study of women's health across the nation. *Am J Epidemiol.* 2007;165:309–318.

Impact of Diabetes and Obesity on the Development of Fecal and Urinary Incontinence

Leslee L. Subak, M.D.

Obesity, type 2 diabetes and urinary incontinence (UI) are common and costly disorders. Over 50% of American women are overweight (body mass index (BMI) 25–29.9 kg/m^2) or obese (BMI >30 kg/m^2), and type 2 diabetes is estimated to affect more than 12% of adults over age 40, including 19% of people over age 60.[1,2] UI affects nearly 50% of middle aged and older women.[3] Obesity and diabetes each account for expenditures of more than $100 billion per year,[4] and the direct cost of UI is more than $30 billion per year in the United States,[5] which is greater than the annual direct costs for all gynecological and breast cancers combined.[6]

Obesity and UI. In epidemiological studies, obesity is one of the strongest modifiable independent risk factors for UI.[7–14] Incontinence is reported to be 50%–100% more prevalent among overweight women and two- to fourfold more prevalent among obese women compared to women of normal weight. Several studies have observed an independent association of BMI with stress and mixed types of UI, with these types being two- to fourfold more prevalent in obese women.[7,8,14] Although data are limited, urge UI may also be associated with increasing BMI.[14] Waist circumference may be the specific aspect of obesity contributing to the prevalence and/or severity UI.[10,14]

Incident UI is also associated with increasing weight. Two recent population-based, prospective cohort studies demonstrated over 5–10 years of follow-up that the odds of incident weekly UI increased by 7%–12% for all types of UI (stress, urge, and mixed) for each 1 kg/m^2 increase in BMI.[14,15] Incident UI also increased with increasing adult weight gain.[15]

Weight reduction is an effective treatment for UI.[16–19] In two trials of overweight and obese women with UI randomized to either a low-calorie liquid-diet program or a lifestyle and behavior change program (Program to Reduce Incontinence by Diet and Exercise (PRIDE)) vs. a control condition, women in the intervention groups had significantly decreased weekly frequency of UI episodes.[18,19] Even modest weight reduction of 3%–5% is a clinically feasible treatment option for incontinence that has comparable efficacy to other nonsurgical treatments for UI as well as the additional benefits of weight reduction.

Although the mechanism of the obesity–UI association is unknown, it is theorized that excess body weight increases abdominal pressure, which in turn increases bladder pressure and urethral mobility, leading to stress UI, and exacerbates detrusor instability.[16,18] Higher waist-to-hip ratio has also been shown to increase risk for UI independent of BMI.[10,14]

Diabetes and UI. Recent evidence strongly suggests that that prediabetes (impaired fasting glucose) and diabetes are independent risk factors for UI.[7,10,11,13,14,20–22] In population-based observational studies, there is similar prevalence of UI, both overall and by type, among prediabetic and diabetic women[22] and UI is reported to be 50%–200% more prevalent in women with type 2 diabetes than among women with normal glucose levels.

Two observational studies showed that over 4–5 years of follow-up, women with type 2 diabetes were at a twofold increased risk of developing very severe UI.[14,21] Diabetes duration of 5 or

more years, insulin treatment, and microvascular complications such as peripheral neuropathy and retinopathy may be important risk factors for developing UI.[11,21–23]

Prevention or effective treatment of diabetes may also be an effective intervention for UI. Among women with prediabetes enrolled in the Diabetes Prevention Program (DPP), the prevalence of total weekly UI was significantly lower at the end of the trial among women randomized to the intensive lifestyle (weight loss and exercise) group than those randomized to metformin or placebo groups.[24] Ongoing investigation in the Action for Health in Diabetes (Look AHEAD) randomized controlled trial of a behavioral weight loss program will investigate UI outcomes among overweight and obese individuals with type 2 diabetes. Therapies for microvascular complications of diabetes may be beneficial in the prevention or treatment of incontinence,[22] and long-term follow-up in the DPP Outcomes Study and Look AHEAD will provide data on the effect of glycemic control and weight reduction on prevalent and incident UI among both prediabetic and diabetic populations.

Mechanisms by which type 2 diabetes may contribute to the development or severity of UI are not well understood.[25] Hyperglycemia in diabetics may cause an increased volume of urine, polyuria, or detrusor instability. Microvascular injury associated with diabetes, similar to the disease process involved in development of retinopathy, nephropathy, and peripheral neuropathy, might damage the innervation of the bladder or alter detrusor muscle function.

Fecal Incontinence. Limited studies have evaluated risk factors for fecal incontinence (FI), but evidence suggests that both obesity and diabetes are independent risk factors for FI. In population-based observational studies, FI is reported to be approximately 50% more prevalent in obese compared to normal weight women.[26–29]

FI is reported to be 40%–200% more prevalent in women with type 2 diabetes than in women with normal glucose levels.[27,29–32] One study observed a significant dose-response relationship between self-reported glycemic control and the prevalence of FI, with higher prevalence associated with poorer level of glycemic control.[32]

Increasing frequency of FI has been observed to be independently and positively associated with both obesity and diabetes.[27] In addition, women with both diabetes and obesity have a 3.5-fold higher likelihood of reporting FI compared to those without these prevalent conditions.[33]

FI is believed to result from an imbalance of the propulsive forces of stool with the resistive mechanisms of the pelvis. Conditions that cause increased abdominal pressure (obesity), increased intestinal motility or loose stool (diabetes), and sphincter or pelvic floor weakness from an anatomic defect or nerve damage (diabetes) may all contribute to FI.[27]

Summary. Obesity and diabetes are strong and independent risk factors for UI and FI. Since obesity and diabetes are preventable and modifiable conditions, the prospect of improved incontinence may help motivate people to undertake difficult lifestyle changes to reduce their more serious risks of obesity, diabetes, and their sequelae. For UI, future clinical trials of treatments among women with obesity and/or diabetes are critical. For FI, epidemiological studies are needed. Incontinence, obesity, and diabetes are common and costly: any treatment approach that can address these health problems simultaneously would be important for public health.

References

1. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. *JAMA.* 2001;286:1195–1200.

2. Harris MI, Flegal KM, Cowie CC, Eberhardt MS, Goldstein DE, Little RR, Wiedmeyer HM, Byrd-Holt DD. Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. The Third National Health and Nutrition Examination Survey, 1988–1994. *Diabetes Care.* 1998;21:518–524.

3. Hunskaar S, Burgio K, Diokno AC, Herzog AR, Hjalmas K, Lapitan M. Epidemiology and natural history of urinary incontinence. In: *Incontinence,* Abrams P, Cardozo L, Khoury S, Wein A, eds. Plymouth, UK: Health Publication Ltd; 2002.

4. Wolf AM, Colditz GA. Current estimates of the economic cost of obesity in the United States. *Obes Res.* 1998;6:97–106.

5. Wilson L, Brown JS, Park GE, Luc KO, Subak LL. Annual costs of urinary incontinence. *Obstet Gynecol.* 2001;98:398–406.

6. Varmus H. *Disease-Specific Estimates of Direct and Indirect Costs of Illness and NIH Support.* Bethesda, MD: Department of Health and Human Services, National Institutes of Health, Office of the Director; September 1997.

7. Danforth KN, Townsend MK, Lifford K, Curhan GC, Resnick NM, Grodstein F. Risk factors for urinary incontinence among middle-aged women. *Am J Obstet Gynecol.* 2006;194:339–345.

8. Hannestad YS, Rortveit G, Daltveit AK, Hunskaar S. Are smoking and other lifestyle factors associated with female urinary incontinence? The Norwegian EPINCONT Study. *BJOG.* 2003;110:247–254.

9. Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. A community-based epidemiological survey of female urinary incontinence: the Norwegian EPINCONT study. Epidemiology of Incontinence in the County of Nord-Trondelag. *J Clin Epidemiol.* 2000;53:1150–1157.

10. Brown J, Grady D, Ouslander J, Herzog A, Varner R, Posner S. Prevalence of urinary incontinence and associated risk factors in postmenopausal women. Heart & Estrogen/Progestin Replacement Study (HERS) Research Group. *Obstet Gynecol.* 1999;94:66–70.

11. Jackson RA, Vittinghoff E, Kanaya AM, Miles TP, Resnick HE, Kritchevsky SB, Simonsick EM, Brown JS; Health, Aging, and Body Composition Study. Urinary incontinence in elderly women: findings from the Health, Aging, and Body Composition Study. *Obstet Gynecol.* 2004;104:301–307.

12. Melville JL, Katon W, Delaney K, Newton K. Urinary incontinence in US women: a population-based study. *Arch Intern Med.* 2005;165:537–542.

13. Sampselle CM, Harlow SD, Skurnick J, Brubaker L, Bondarenko I. Urinary incontinence predictors and life impact in ethnically diverse perimenopausal women. *Obstet Gynecol.* 2002;100:1230–1238.

14. Waetjen LE, Liao S, Johnson WO, Sampselle CM, Sternfield B, Harlow SD, Gold EB. Factors associated with prevalent and incident urinary incontinence in a cohort of midlife women: a longitudinal analysis of data: study of women's health across the nation. *Am J Epidemiol.* 2007;165:309–318.

15. Townsend MK, Danforth KN, Rosner B, Curhan GC, Resnick NM, Grodstein F. Body mass index, weight gain, and incident urinary incontinence in middle-aged women. *Obstet Gynecol.* 2007;110:346–353.

16. Bump R, Sugerman H, Fantl J, McClish D. Obesity and lower urinary tract function in women: effect of surgically induced weight loss. *Am J Obstet Gynecol.* 1992;166:392–399.

17. Deitel M, Stone E, Kassam HA, Wilk EJ, Sutherland DJ. Gynecologic-obstetric changes after loss of massive excess weight following bariatric surgery. *J Am Coll Nutr.* 1988;7:147–153.

18. Subak LL, Whitcomb E, Shen H, Saxton J, Vittinghoff E, Brown JS. Weight loss: a novel and effective treatment for urinary incontinence. *J Urol.* 2005;174:190–195.

19. Subak LL, Smith WR, West D, Franklin F, Vittinghoff E, Creasman J, Richter HE, Burgio K, Gorin A, Macer J, Kusek JW, Grady D. A behavioral weight loss program significantly reduces urinary incontinence episodes in overweight and obese women. Paper presented at: Meeting of the American UroGynecological Society; September 2007; Hollywood, FL.

20. Ebbesen MH, Hannestad YS, Midthjell K, Hunskaar S. Diabetes and urinary incontinence—prevalence data from Norway. *Acta Obstet Gynecol Scand.* 2007:1–7. Epub ahead of print.

21. Lifford KL, Curhan GC, Hu FB, Barbieri RL, Grodstein F. Type 2 diabetes mellitus and risk of developing urinary incontinence. *J Am Geriatr Soc.* 2005;53:1851–1857.

22. Brown JS, Vittinghoff E, Lin F, Nyberg LM, Kusek JW, Kanaya AM. Prevalence and risk factors for urinary incontinence in women with type 2 diabetes and impaired fasting glucose: findings from the National Health and Nutrition Examination Survey (NHANES) 2001–2002. *Diabetes Care.* 2006;29:1307–1312.

23. Jackson SL, Scholes D, Boyko EJ, Abraham L, Fihn SD. Urinary incontinence and diabetes in postmenopausal women. *Diabetes Care.* 2005;28:1730–1738.

24. Brown JS, Wing R, Barrett-Connor E, Nyberg LM, Kusek JW, Orchard TJ, Ma Y, Vittinghoff E, Kanaya AM; Diabetes Prevention Program Research Group. Lifestyle intervention is associated with lower prevalence of urinary incontinence: the Diabetes Prevention Program. *Diabetes Care.* 2006;29:385–390.

25. Brown JS, Nyberg LM, Kusek JW, Burgio KL, Diokno AC, Foldspang A, Fultz NH, Herzog AR, Hunskaar S, Milsom I, Nygaard I, Subak LL, Thom DH; National Institute of Diabetes and Digestive Kidney Diseases International Research Working Group on Bladder Dysfunction. Proceedings of the National Institute of Diabetes and Digestive and Kidney Diseases International Symposium on Epidemiologic Issues in Urinary Incontinence in Women. *Am J Obstet Gynecol.* 2003;188:S77–S88.

26. Altman D, Falconer C, Rossner S, Melin I. The risk of anal incontinence in obese women. *Int Urogynecol J Pelvic Floor Dysfunct.* 2007;18:1283–1289.

27. Varma MG, Brown JS, Creasman JM, et al. Fecal incontinence in females older than aged 40 years: who is at risk? *Dis Colon Rectum.* 2006;49:841–851.

28. Melville JL, Fan M-Y, Newton K, Fenner D. Fecal Incontinence in US Women: a Population-Based Study. *Am J Obstet Gynecol.* 2005;193:2071–2076.

29. Uustal Fornell E, Wingren G, Kjolhede P. Factors associated with pelvic floor dysfunction with emphasis on urinary and fecal incontinence and genital prolapse: an epidemiological study. *Acta Obstet Gynecol Scand.* 2004;83:383–389.

30. Nakanishi N, Tatara K, Naramura H, Fujiwara H, Takashima Y, Fukuda H. Urinary and fecal incontinence in a community-residing older population in Japan. *J Am Geriatr Soc.* 1997;45:215–219.

31. Quander CR, Morris MC, Melson J, Bienias JL, Evans DA. Prevalence of and factors associated with fecal incontinence in a large community study of older individuals. *Am J Gastroenterol.* 2005;100:905–909.

32. Bytzer P, Talley NJ, Leemon M, Young LJ, Jones MP, Horowitz M. Prevalence of gastrointestinal symptoms associated with diabetes mellitus: a population-based survey of 15,000 adults. *Arch Intern Med.* 2001;161:1989–1996.

33. Lawrence JM, Lukacz ES, Liu IL, Nager CW, Luber KM. Pelvic floor disorders, diabetes, and obesity in women: findings from the Kaiser Permanente Continence Associated Risk Epidemiology Study. *Diabetes Care.* 2007;30:2536–2541.

Do Pregnancy, Type of Delivery, and Postpartum State Increase the Risk for Development of Fecal and Urinary Incontinence?

Holly E. Richter, M.D., Ph.D.

In March of 2006, the National Institutes of Child Health and Human Development and the Office of Medical Applications of Research of the National Institutes of Health convened a State-of-the-Science Conference to explore more fully the currently available data on cesarean delivery (CD) on maternal request.[1] A systematic review of the literature pertaining to both neonatal and maternal outcomes with CD on maternal request and vaginal delivery was presented both by an expert panel and by RTI-International–University of North Carolina Evidence-Based Practice Center through the Agency for Healthcare Research and Quality. Furthermore, presentations by clinical experts in the various areas of neonatal and maternal healthcare complemented the panel's presentations. For the maternal outcome of **urinary incontinence** (UI), level III (weak) data indicated that the rate of stress UI after elective CD was lower than after vaginal delivery; however, other covariates may influence these outcomes. For the outcome of anorectal dysfunction, including anal and **fecal incontinence** (FI), weak evidence suggested a reduced risk of anal incontinence in planned CD compared with unplanned CD or instrument-assisted vaginal deliveries. Existing evidence also demonstrated an association between sphincter disruption at the time of vaginal delivery and FI, especially when associated with midline episiotomy and instrument-assisted deliveries. This abstract provides an interim summary of an updated review of the literature on pregnancy and its effect on UI and FI.

UI has been attributed to pregnancy and childbirth, and its prevalence is common antenatally, with reported rates of 16%–65%.[2–6] UI during pregnancy may be a short-term condition for some women.[4,5] In a cohort of 523 women, Burgio and colleagues found a drop in the prevalence of UI from 60% to 11% by 6 weeks postpartum.[5] However, the presence of antenatal UI has also been found to be a predictor of longer term postpartum UI.[3,5,7] Other covariates should be considered when looking at the effect of pregnancy on the development of both short- and long-term UI, including primiparity versus multiparous patients (effect of prior deliveries),[8,9] vaginal delivery versus CD,[7,10] and use of episiotomy and forceps[5,10,11] among other factors. More recently, a prospective multicenter cohort study, the Childbirth and Pelvic Symptoms (CAPS) study, comprising three cohorts of primiparous women: 407 vaginally delivered women, with clinically recognized anal sphincter tears (n=407); 390 vaginally delivered controls, without a clinically evident sphincter tear; and 124 CD controls who delivered prior to labor. All three cohorts were prospectively followed for symptoms of UI and FI. UI symptoms were assessed by using the Medical, Epidemiological, and Social Aspects of Aging questionnaire.[12] UI symptom prevalence did not differ between the sphincter tear and vaginal delivery control groups (at 6 weeks, 34.8% vs. 35.4%, p=0.76; at 6 months, 33.7% vs. 31.3%, p=0.66) or between the vaginal delivery control and CD cohorts at 6 weeks (35.4% vs. 25.0%, p=0.32) and 6 months (31.3% vs. 22.9%, p=0.44).

Curiously, as women age, the association of childbirth and UI appears to consistently decrease. In a large Norwegian study,[9] the relationship of UI with parity was significant in younger women and absent in older women (>65 years).

To date, only one randomized trial has assessed the impact of planned vaginal delivery versus planned CD and its effect on UI.[13] In that study, 1,596 women from 110 centers worldwide completed questionnaires addressing UI symptoms at 3 months postpartum, and approximately one-half of the original subjects completed these questionnaires 2 years postpartum. Furthermore, the questions at the 3-month time addressed stress UI symptoms within the last 7 days, and at the 2-year time addressed symptoms within the last 3–6 months. At 3 months postpartum, women in the planned CD group had less UI than those in the planned vaginal delivery group (4.5% vs. 7.3%, RR=0.62, 95% CI=0.41–0.93). No difference was seen at 2 years, with UI noted in 17.8% in the planned CD group versus 21.8% for the planned vaginal delivery group.

FI, the involuntary loss of solid or liquid stool, can significantly affect quality of life.[14] Anal incontinence includes the involuntary loss of flatus. The prevalence of FI in community-dwelling women ranges from 0.4% to 18%, depending on the definition used and the population queried.[15] Among the many causes of FI, obstetric-related sphincter tears—one of the primary causes—have been the focus of research, treatment, and prevention. As the long-term results of primary sphincter repair have been reported to be as low as 44%,[16] research continues to investigate the relationship of obstetric delivery, sphincter tears, and the prevention of FI.

The Fecal Incontinence Postpartum Research Initiative undertook a population-based survey sent to women 3–6 months after delivery.[17] Fourteen percent (1,192/8,774) of respondents reported symptoms of FI. Body mass index (BMI) greater than 30, time pushing, forceps-assisted delivery, fourth-degree tear, and current smoking were associated with FI. The Kaiser Permanente Continence Associated Risks Epidemiologic Study was a cross-sectional sample of 12,200 women aged 25–84 years.[18] Using the validated Epidemiology of Prolapse and Incontinence Questionnaire, the overall prevalence of FI was 17%. Participants were then categorized into nulliparous, CD, or vaginally parous groups. The vaginally parous group had a higher prevalence of all pelvic floor disorder symptoms, including FI. Other recent studies have also continued to show an association between vaginal delivery and FI, especially with anal sphincter tear.[19,20]

The association of third- and fourth-degree sphincter tear and FI was first reported more than 10 years ago.[21] Sphincter tears may occur in up to 18% of deliveries[22] and may not be recognized at the time of delivery in 23%–35% of primiparous women.[22,23] As noted above, the prospective multicenter CAPS study consisted of three cohorts of primiparous women: 407 women delivered vaginally, with clinically recognized anal sphincter tears (*n*=407); 390 controls who delivered vaginally, without a clinically evident sphincter tear; and 124 CD controls who delivered prior to labor.[12] The presence of FI was measured with use of the Fecal Incontinence Severity Index, assessing symptoms at 6 weeks and 6 months postpartum. Women sustaining a sphincter tear compared to the controls who delivered vaginally reported more FI at 6 weeks (27% versus 11%, OR=2.8, CI=1.8–4.3, attributable risk 15%) and at 6 months (17% versus 8%, OR=1.9, 95% CI=1.2–3.2, attributable risk 9%). Severity of FI was significantly greater in those women with a sphincter tear. Risk factors for FI in the group with sphincter tears of the CAPS cohort included Caucasian race, antenatal UI, fourth- versus third-degree tear, older age at delivery, and higher BMI.[24]

A subset of CAPS patients underwent endoanal ultrasound studies, and they were queried about FI symptoms 6–12 months postpartum.[25] In the group with tears, the finding of internal sphincter tears was associated with greater FI symptoms. Women with combined tears (internal and external anal sphincter tears) had the highest degree of symptoms. The association of especially internal anal sphincter gaps by ultrasound postpartum, has been associated with

greater FI symptoms in other studies as well[26,27] and has broadened the focus of anal sphincter repairs from just the external sphincter to include the internal sphincter.

Evidence regarding the use of mediolateral episiotomy to prevent sphincter tears is inconsistent,[28] and evidence on other interventions such as delivery posture[29] and perineal massage[30] was inconclusive. The prevention of FI with use of CD has also not been conclusively proved.[12,31] The appropriate repair technique and association with FI has also been studied. A 2006 Cochrane review comparing overlapping and end-to-end repair did not find a difference in FI symptoms at 6 weeks and at 3, 6, and 12 months postpartum.[32] Recently, immediate postpartum ultrasonography has demonstrated missed sphincter tear rates as high as 35%;[33] this finding has sparked interest in immediate postpartum ultrasound.

References

1. NIH State-of-the-Science Conference Statement on cesarean delivery on maternal request. *NIH Consens Sci Statements.* 2006;23:1–29.

2. Nygaard I. Urinary incontinence: is cesarean delivery protective? *Sem Perinatology.* 2006;30:267–271.

3. Hvidman L, Foldspang A, Mommsen S, Nielsen JB. Postpartum urinary incontinence. *Acta Obstet Gynecol Scand.* 2003;82:556–563.

4. Viktrup L, Lose G, Rolff M, Barfoed K. The symptom of stress incontinence caused by pregnancy or delivery in primiparas. *Obstet Gynecol.* 1992;79:945–949.

5. Burgio KL, Zyczynski H, Locher JL, Richter HE, Redden DT, Wright KC. Urinary incontinence in the 12-month postpartum period. *Obstet Gynecol.* 2003;102:1291–1298.

6. Eason E, Labrecque M, Marcoux S, Mondor M. Effects of carrying a pregnancy and of method of delivery on urinary incontinence: a prospective cohort study. *BMC Pregnancy Childbirth.* 2004;4:4.

7. Foldspang A, Hvidman L, Mommsen S, Nielsen JB. Risk of postpartum urinary incontinence associated with pregnancy and mode of delivery. *Acta Obstet Gynecol Scand.* 2004;83:923–927.

8. Burgio KL, Locher JL, Zyczynski H, Hardin JM, Singh K. Urinary incontinence during a pregnancy in a racially mixed sample: characteristics and predisposing factors. *Int Urogynecol J Pelvic Floor Dysfunct.* 1996;7:69–73.

9. Rortveit G, Hannestad YS, Daltveit AK, Hunskaar S. Age- and type-dependent effects of parity on urinary incontinence: the Norwegian EPINCONT study. *Obstet Gynecol.* 2001;98:1004–1010.

10. Chaliha C, Digesu A, Hutchings A, Soligo M, Khullar V. Cesarean section is protective against stress urinary incontinence: an analysis of women with multiple deliveries. *Br J Obstet Gynecol.* 2004;111:754–755.

11. Foldspang A, Mommsen S, Djurhuus JC. Prevalent urinary incontinence as a correlate of pregnancy, vaginal childbirth, and obstetric techniques. *Am J Public Health.* 1999;89:209–212.

12. Borello-France D, Burgio KL, Richter HE, Zyczynski H, Fitzgerald MP, Whitehead W, Fine P, Nygaard I, Handa VL, Visco AG, Weber AM, Brown MB; Pelvic Floor Disorders Network. Fecal and urinary incontinence in primiparous women. *Obstet Gynecol.* 2006;108:863–872.

13. Hannah ME, Hannah WJ, Hodnett ED, Chalmers B, Kung R, Willan A, Amankwah K, Cheng M, Helewa M, Hewson S, Saigal S, Whyte H, Gafni A; Term Breech Trial 3-Month Follow-up Collaborative Group. Outcomes at 3 months after planned cesarean vs planned vaginal birth for breech presentation at term: The International Randomized Term Breech Trial. *JAMA.* 2002;287:1822–1831.

14. Hunskaar S, Burgio K, Clark A, et al. Epidemiology of urinary (UI) and faecal (FI) incontinence and pelvic organ prolapse (POP). In: Abrams P, Cardozo L., Khoury S, Wein A, eds. *Incontinence.* 2005 ed. UK: Health Publications Ltd., 2005.

15. Macmillan AK, Merrie AE, Marshall RJ, Parry BR. The prevalence of fecal incontinence in community-dwelling adults: a systematic review of the literature. *Dis Colon Rectum.* 2004;47:1341–1349.

16. Williams A. Third-degree perineal tears: risk factors and outcome after primary repair. *J Obstet Gynaecol.* 2003;23:611–614.

17. Guise JM, Morris C, Osterweil P, Li H, Rosenberg D, Greenlick M. Incidence of fecal incontinence after childbirth. *Obstet Gynecol.* 2007;109:281–288.

18. Lukacz ES, Lawrence JM, Contreras R, Nager CW, Luber KM. Parity, mode of delivery, and pelvic floor disorders. *Obstet Gynecol.* 2006;107:1253–1260.

19. McKinnie V, Swift SE, Wang W, Woodman P, O'Boyle A, Kahn M, Valley M, Bland D, Schaffer J. The effect of pregnancy and mode of delivery on the prevalence of urinary and fecal incontinence. *Am J Obstet Gynecol.* 2005;193:512–17; discussion 7–8.

20. van Brummen HJ, Bruinse HW, van de Pol G, Heintz AP, van der Vaart CH. Defecatory symptoms during and after the first pregnancy: prevalences and associated factors. *Int Urogynecol J.* 2006;17:224–230.

21. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Engl J Med.* 1993:329:1905–1911.

22. Fenner DE, Genberg B, Brahma P, Marek L, DeLancey JO. Fecal and urinary incontinence after vaginal delivery with anal sphincter disruption in an obstetrics unit in the United States. *Am J Obstet Gynecol.* 2003;189:1543–1549; discussion 1549–1550.

23. Pinta TM, Kylanpaa ML, Teramo KA, Luukkonen PS. Sphincter rupture and anal incontinence after first delivery. *Acta Obstet Gynecol Scand.* 2004;83:917–922.

24. Burgio KL, Borello-France D, Richter HE, Fitzgerald MP, Whitehead W, Handa VL, Nygaard I, Fine P, Zyczynski H, Visco AG, Brown MB, Weber AM; for The Pelvic Floor Disorders Network. Risk factors for fecal and urinary incontinence after childbirth: the Childbirth and Pelvic Symptoms Study. *Am J Gastroenterol.* 2007;102:1998–2004.

25. Richter HE, Fielding JR, Bradley CS, Handa VL, Fine P, FitzGerald MP, Visco A, Wald A, Hakim C, Wei JT, Weber AM; Pelvic Floor Disorders Network. Endoanal ultrasound findings and fecal incontinence symptoms in women with and without recognized anal sphincter tears. *Obstet Gynecol.* 2006;108:1394–1401.

26. Mahony R, Behan M, Daly L, Kirwan C, O'Herlihy C, O'Connell PR. Internal anal sphincter defect influences continence outcome following obstetric anal sphincter injury. *Am J Obstet Gynecol.* 2007;196:217e1–217e5.

27. Nichols CM, Nam M, Ramakrishnan V, Lamb EH, Currie N. Anal sphincter defects and bowel symptoms in women with and without recognized anal sphincter trauma. *Am J Obstet Gynecol.* 2006;194:1450–1454.

28. Carroli G, Belizan J. Episiotomy for vaginal birth. *Cochrane Database Syst Rev.* 2000;(2):CD000081.

29. Altman D, Ragnar I, Ekstrom A, Tyden T, Olsson SE. Anal sphincter lacerations and upright delivery postures—a risk analysis from a randomized controlled trial. *Int Urogynecol J.* 2007;18:141–146.

30. Beckmann MM, Garrett AJ. Antenatal perineal massage for reducing perineal trauma. *Cochrane Database Syst Rev.* 2006;(1):CD005123.

31. Nelson RL, Westercamp M, Furner SE. A systematic review of the efficacy of cesarean section in the preservation of anal continence. *Dis Colon Rectum.* 2006;49:1587–1595.

32. Fernando R, Sultan AH, Kettle C, Thakar R, Radley S. Methods of repair for obstetric anal sphincter injury. *Cochrane Database Syst Rev.* 2006;(3):CD002866.

33. Andrews V, Sultan AH, Thakar R, Jones PW. Occult anal sphincter injury—myth or reality? *BJOG.* 2006;113:195–200.

Effect of Hormones on Fecal and Urinary Incontinence and Pelvic Organ Prolapse

Ingrid Nygaard, M.D., M.S.

Hormones mediate numerous effects in women. Because many women first notice urinary incontinence (UI) in their late 40s and early 50s, the same time period in which major hormonal changes occur, estrogens were proposed as a logical treatment for UI. Indeed, there is ample biologic plausibility for a link between estrogen depletion and UI.[1] The urethra, bladder, and pelvic connective tissue and muscles have a rich supply of estrogen receptors. Estrogen increases vaginal maturation, periurethral blood supply, alpha-adrenergic receptor sensitivity, and sympathetic nerve density in the pelvis and inhibits bladder contractions in animals.

Clinical findings also suggested that hormones play a role in pelvic floor function. One-third of women in one study reported worsening bladder symptoms before menstruation. In a large population-based study, self-reported UI was increased in women who had midcycle bleeding or who recently noted decreased bleeding duration.[2]

In concordance with this basic science evidence and with clinical anecdotes, several uncontrolled studies with subjective outcomes showed that estrogen therapy of various types decreased UI, urgency, and frequency.

However, the early promise of estrogen's role as a therapeutic agent was not borne out by subsequent studies. Larger cohort studies found that women on estrogen had more UI than those who did not take this hormone.[3] In the 1980s and 1990s, many physicians prescribed estrogens to treat incontinence, however, such evidence cannot be used to conclude that estrogens caused the incontinence.

The most recent Cochrane review on this topic, last amended in 2003, concluded, based on 28 trials, with 2,926 women, available at the time, that about 50% of women treated with estrogen were cured or improved compared with about 25% of those on placebo.[4] Given the timing of this review, it necessarily did not include five large, randomized trials that collectively enrolled 30,914 women.[5–9] These carefully done randomized trials, that compared estrogen to placebo in a masked fashion, were strikingly consistent in their conclusions: at usual doses, estrogens, with or without progestins, increase UI severity and incidence, whereas low-dose transdermal estrogen has no effect.

Why might estrogen actually cause worsening of UI? Although estrogens are poorly studied to date, they appear to reduce collagen in the urethra and pelvic floor and increase collagen turnover.[10,11] In addition, women with stress urinary incontinence (SUI) may differ in important ways from continent women. For example, extracellular matrix protein expressions have been shown to be hormonally regulated in vaginal wall fibroblasts and also to differ between women with and without SUI.[12] In a study that found lower rates of estrogen-receptor staining in connective tissue, smooth muscle, and nerve fibers in women with SUI than in controls, the authors suggested that having fewer estrogen receptors in pelvic floor tissues might be related to SUI and also might explain why estrogen therapy is not effective in treating this condition.[13]

Selective Estrogen-Receptor Modulators (SERMs) impact pelvic floor function in different ways. Randomized trials comparing raloxifene to estrogen or placebo to date have shown no effect on

either UI or pelvic organ prolapse.[14] In contrast, an investigational study of levormeloxifene was halted after 10 months because of a marked increase in both UI and uterovaginal prolapse in women receiving this SERM compared to placebo.[15]

Androgens have anabolic effects on skeletal muscle and theoretically might improve pelvic floor muscles.[16] Castrating rats and thus removing their androgen source produced the same degree of myofiber atrophy in levator ani muscles as denervation alone.[17] Women also have androgen receptors in the levator ani muscles and cardinal ligaments. Whether androgens are useful for treating pelvic floor disorders has not been studied.

Other hormones, such as relaxin, may also play a role in maintaining continence, particularly during pregnancy.[18]

Very few studies have assessed the impact of hormones on either pelvic organ prolapse or fecal incontinence (FI). In an ancillary study of the Women's Health Initiative, women randomly assigned to estrogen plus progestin had similar prevalence and incidence rates of pelvic organ prolapse as those assigned to placebo.[19] In a small cohort study, after 6 months of estrogen therapy, anal resting and squeeze pressures improved in women with FI, but only one-fourth became asymptomatic.[20]

Future studies are needed to answer the following questions:

- Is the effect of hormones on UI mediated through the lower urinary tract or another route?
- What is the role of topical estrogens and androgens?
- Can SERMs or Selective Androgen-Receptor Modulators (SARMs) be developed to treat pelvic floor disorders?
- From an ultrastructural point of view, how do different hormones affect bladder, bowel, and pelvic function?
- Is there a critical window at which hormone therapy might have positive versus negative effects?
- Do xenoestrogens, present in many household and industrial products, affect bladder or bowel function?
- What role do other hormones, such as relaxin, play on pelvic floor function?
- Can we identify a subgroup of women who have UI who will respond to targeted hormonal therapy?

Understanding the answers to these questions through basic and clinical research has the potential to affect the lives of millions of women worldwide.

References

1. Griebling TL, Nygaard IE. The role of estrogen replacement therapy in the management of urinary incontinence and urinary tract infection in postmenopausal women. *Endocrinol Metab Clin North Am.* 1997;26:347–360.

2. Hvidman L, Foldspang A, Mommsen S, Bugge Nielsen J. Menstural cycle, female hormone use and urinary incontinence in premenopausal women. *Int Urogynecol J Pelvic Floor Dysfunct.* 2003;14:56–61.

3. Grodstein F, Lifford K, Resnick NM, Curhan GC. Postmenopausal hormone therapy and risk of developing urinary incontinence. *Obstet Gynecol.* 2004;103:254–260.

4. Moeher B, Hextall A, Jackson S. Oestrogens for urinary incontinence in women. *Cochrane Database Syst Rev.* 2003;2:CD001405.

5. Hendrix SL, Cochrane BB, Nygaard IE. Effects of estrogen with and without progestin on urinary incontinence. *JAMA.* 2005;293:935–948.

6. Waetjen LE, Brown JS, Vittinghoff E, et al. The effect of ultralow-dose transdermal estradiol on urinary incontinence in postmenopausal women. *Obstet Gynecol.* 2005;106:946–952.

7. Steinauer JE, Waetjen LE, Vittinghoff E. Postmenopausal hormone therapy: does it cause incontinence? *Obstet Gynecol.* 2005;106:940–945.

8. Vestergaard P, Hermann AP, Stilgren L, et al. Effects of 5 years of hormonal replacement therapy on menopausal symptoms and blood pressure—a randomised controlled study. *Maturitas.* 2003:46;123–132.

9. Goldstein SR, Johnson S, Watts NB, Ciaccia AV, Elmerick D, Murram D. Incidence of urinary incontinence in postmenopausal women treated with raloxifene or estrogen. *Menopause.* 2005;12:160–164.

10. Edwall L, Carlstrom K, Jonasson AF. Endocrine status and markers of collagen synthesis and degradation in serum and urogenital tissue from women with and without stress urinary incontinence. *Neurourol Urodyn.* 2007;26:410–415.

11. Jackson S, James M, Abrams P. The effect of oestradiol on vaginal collagen metabolism in postmenopausal women with genuine stress incontinence. *BJOG.* 2002;109:339–342.

12. Wen Y, Polan ML, Chen B. Do extracellular matrix protein expressions change with cyclic reproductive hormones in pelvic connective tissue from women with stress urinary incontinence? *Hum Reprod.* 2006;21:1266–1273.

13. Zhu L, Lang J, Feng R, Chen J, Wong F. Estrogen receptor in pelvic floor tissues in patients with stress urinary incontinence. *Int Urogynecol J Pelvic Floor Dysfunct.* 2004;15:340–343.

14. Waetjen LE, Brown JS, Modelska K. Effect of raloxifene on urinary incontinence: a randomized controlled trial. *Obstet Gynecol.* 2004;103:261–266.

15. Goldstein SR, Nanavati N. Adverse events that are associated with the selective estrogen receptor modulator levormeloxifene in an aborted phase III osteoporosis treatment study. *Am J Obstet Gynecol.* 2002;187:521–527.

16. Ho MH, Bhatia NN, Bhasin S. Anabolic effects of androgens on muscles of female pelvic floor and lower urinary tract. *Curr Opin Obstet Gynecol.* 2004;16:405–409.

17. Nnodim JO. Quantitative study of the effects of denervation and castration on the levator ani muscle of the rat. *Anat Rec.* 1999;255:324–333.

18. Kristiansson P, Samuelsson E, von Schoultz B, Svardsudd K. Reproductive hormones and stress urinary incontinence in pregnancy. *Acta Obstet Gynecol Scand.* 2001;80:1125–1130.

19. Nygaard I, Bradley C, Brandt D; Women's Health Initiative. Pelvic organ prolapse in older women: prevalence and risk factors. *Obstet Gynecol.* 2004;104:489–497.

20. Donnelly V, O'Connell PR, O'Herlihy C. The influence of oestrogen replacement on faecal incontinence in postmenopausal women. *Br J Obstet Gynaecol.* 1997;104:311–314.

Impact of Chronic Gastrointestinal Conditions, Such as Irritable Bowel Syndrome, Inflammatory Bowel Disease, and Constipation as Risk Factors for Fecal Incontinence

William E. Whitehead, Ph.D.

Chronic gastrointestinal symptoms and gastrointestinal disorders are among the strongest risk factors for fecal incontinence (FI) or anal incontinence (AI). (FI refers to involuntary loss of liquid or solid stool, whereas AI includes involuntary flatus as well. Soiling of underwear is sometimes distinguished from both.) The goals of this systematic review are to (1) estimate the relative risk for FI/AI associated with these symptoms and disorders, and (2) identify possible mechanisms.

Diarrhea. Population-based surveys in all age groups yielded odds ratios (ORs) of 2.5[1] to 4.89[2] for the relative risk of FI in subjects with diarrhea. A population-based study limited to men and women aged 75 or older[3] yielded a higher OR of 6.8 for FI alone and 7.7 for double incontinence (FI + urinary incontinence (UI)). Hypothesized mechanisms include (1) peristaltic motility that delivers high volumes of water or stool to the rectum, (2) reflex inhibition of the internal anal sphincter (IAS) secondary to peristaltic motility, and (3) decreased viscosity—liquid stool may require a stronger and more sustained contraction of the external anal sphincter and pelvic floor muscles to avoid leakage compared to formed stool.

Urgency. ORs of approximately 5 in population-based studies[1,2] suggest that urgency to defecate is a stronger predictor of FI than is diarrhea or other known risk factors. Although frequently associated with diarrhea, urgency is an independent risk factor.[1] The sensation of urgency is triggered by rectal distention, and physiological mechanisms for increased urgency include decreased compliance of the rectum and more rapid whole-gut transit.[4]

Constipation. Self-reported constipation (presumed to be mostly slow-transit type constipation) is not a risk factor for FI/AI. However, dyssynergic defecation and fecal impaction are significant risk factors.[5,6] Hypothesized mechanisms for FI in the presence of fecal impaction include (1) dilation of the IAS, (2) seepage of soft or liquid stool around the fecal mass, and (3) decreased ability to perceive rectal filling.[7]

Irritable Bowel Syndrome. ORs for FI in irritable bowel syndrome (IBS) range from 2 to 8, and up to 45% of patients with IBS report episodes of FI.[8] This association is consistently reported. The mechanisms mediating this association are presumed to be diarrhea and urgency—common symptoms of IBS. However, abdominal pain has been independently associated with FI.[5]

Inflammatory Bowel Disease (Ulcerative Colitis and Crohn's Disease). Estimates of the association between FI and inflammatory bowel disease (IBD) are variable because (1) only one population-based study is available, and (2) most patients described in the published literature have already undergone surgical and/or aggressive medical treatments which could affect the estimates. According to the only population-based study,[8] 41% of IBD patients have FI. Mechanisms for their FI include diarrhea and urgency, which are common symptoms of both ulcerative colitis and Crohn's disease. An additional mechanism specific to ulcerative colitis is reduced rectal compliance due to inflammation[9] or surgical creation of an ileal pouch. Mechanisms for FI that are specific to Crohn's disease include perianal fistulae, which may leak stool, and abscesses; both may damage the sphincter muscles.[10]

Hemorrhoids. From 48%[11] to 63%[12] of patients with prolapsed hemorrhoids report fecal soiling. However, solid and liquid stool incontinence are relatively rare. Hemorrhoids are more common in women than in men, and onset is frequently linked to vaginal childbirth.[13] Hemorrhoids are also related to chronic diarrhea.[11] The hypothesized mechanisms for fecal soiling associated with hemorrhoids are mechanical obstruction to closure of the anal sphincters and bleeding from hemorrhoids.

Rectal Prolapse. Fecal soiling occurs in an estimated 30%[14] to 50%[15] of patients with rectal prolapse. The likely mechanism for this is secretion of mucus from exposed rectal mucosa. Solid and liquid stool FI is less common.

Descent of the Perineum. There is a possible association between descent of the perineum and FI,[15] although this has not been seen in all studies. The likely mechanism for any association is weakness of pelvic floor muscles and damage to the pudendal nerve.

Rectocele. Rectocele shows no consistent association with FI.[15] It is a frequent finding on defecograph or pelvic magnetic resonance (MR) in healthy subjects, and this may lead to an erroneous clinical impression that rectocele is associated with FI.

References

1. Bharucha AE, Zinsmeister AR, Locke GR, et al. Risk factors for fecal incontinence: a population-based study in women. *Am J Gastroenterol.* 2006;101:1305–1312.

2. Kalantar JS, Howell S, Talley NJ. Prevalence of faecal incontinence and associated risk factors; an underdiagnosed problem in the Australian community? *Med J Aust.* 2002;176:54–57.

3. Stenzelius K, Mattiasson A, Hallberg IR, Westergren A. Symptoms of urinary and faecal incontinence among men and women 75+ in relation to health complaints and quality of life. *Neurourol Urodyn.* 2004;23:211–222.

4. Basilisco G, De Marco E, Tomba C, Cesana BM. Bowel urgency in patients with irritable bowel syndrome. *Gastroenterol.* 2007;132:38–44.

5. Siproudhis L, Pigot F, Godeberge P, Damon H, Soudan D, Bigard MA. Defecation disorders: a French population survey. *Dis Colon Rectum.* 2006;49:219–227.

6. Chassagne P, Landrin I, Neveu C, et al. Fecal incontinence in the institutionalized elderly: incidence, risk factors, and prognosis. *Am J Med.* 1999;106:185–190.

7. Barrett JA, Brocklehurst JC, Kiff ES, Ferguson G, Faragher EB. Anal function in geriatric patients with faecal incontinence. *Gut.* 1989;30:1244–1251.

8. Varma MG, Brown JS, Creasman JM, et al. Fecal incontinence in females older than aged 40 years: who is at risk? *Dis Colon Rectum.* 2006;49:841–851.

9. Farthing MJ, Lennard-Jones JE. Sensibility of the rectum to distension and the anorectal distension reflex in ulcerative colitis. *Gut.* 1978;19:64–69.

10. Michelassi F, Melis M, Rubin M, Hurst RD. Surgical treatment of anorectal complications in Crohn's disease. *Surgery.* 2000;128:597–603.

11. Johannsson HO, Graf W, Pahlman L. Bowel habits in hemorrhoid patients and normal subjects. *Am J Gastroenterol.* 2005;100:401–406.

12. Murie JA, Sim AJ, Mackenzie I. The importance of pain, pruritus and soiling as symptoms of haemorrhoids and their response to haemorrhoidectomy or rubber band ligation. *Br J Surg.* 1981;68:247–249.

13. Borders N. After the afterbirth: a critical review of postpartum health relative to method of delivery. *J Midwifery Womens Health.* 2006;51:242–248.

14. Ho YH, Muller R, Veitch C, Rane A, Durrheim D. Faecal incontinence: an unrecognised epidemic in rural North Queensland? Results of a hospital-based outpatient study. *Aust J Rural Health.* 2005;13:28–34.

15. Parmentier H, Damon H, Henry L, Barth X, Mellier G, Mion F. Frequency of anal incontinence and results of pelvic viscerography in 291 women with pelvic organ prolapse. *Gastroenterol Clin Biol.* 2004;28:226–230.

Risk Factors for the Development of Fecal and Urinary Incontinence—Age, Frailty, Dementia, Functional Impairment, and Institutionalization

John F. Schnelle, Ph.D.

Urinary incontinence (UI) is reported for 50%–65 % of nursing home (NH) residents, and fecal incontinence (FI) is found in most of the same people.[1] Immobility and dementia are the two primary risk factors for UI and FI in residents in the NH. Three separate studies showed that residents with UI and FI had significant mobility problems and an average Mini mental status score of 8–14, indicating severe cognitive impairment.[2–4] These data support other studies identifying immobility and dementia as the primary risk factors for FI.[5]

There is also ample evidence of lower urinary tract dysfunction among incontinent NH residents including reduced bladder capacity, high postvoid residuals and poor sphincter function.[6] However, these lower urinary tract disorders have not proven predictive of a resident's ability to regain continence through a behavioral program that addresses the risk factors of immobility and dementia.[6] There is less evidence describing the physiology of FI. One recent study reported that most NH residents with FI showed severe impairment of rectal sensation. More importantly, 75% showed a dyssnergic pattern characterized by high intrarectal pressure while straining, in combination with elevation of anal sphincter pressure.[7]

Treatment and Prevention

Most published treatments for UI and FI in NH residents have been directed to the risk factors of immobility and dementia. The logic is that immobility prevents people from getting to the bathroom and dementia reduces the motivation of both the resident to request assistance and of the caregiver to provide it. Furthermore, it is unlikely that any intervention directed to the medical risk factors for UI and FI (e.g., hyperactive bladder or poor rectal sensation) could be effective if residents are unaware of the need to toilet (dementia) or are unable to move to the toilet (mobility).

Treatments using prompted voiding have been evaluated most frequently in controlled trials and include three components relevant to immobility and dementia.[2–4] (1) Residents are approached every 2 hours and asked if they are wet or dry; (2) residents are prompted up to three times to request assistance; and (3) when residents ask for assistance, they are reinforced and provided assistance to the toilet. This simple intervention is labor intensive, does not involve treating abnormalities of the lower urinary tract, and is effective. In various clinical trials, 33%–60% of residents reduced the frequency of their incontinence to less than one episode per day or became continent after participating in a prompted-voiding program.[2–4] Supplementing prompted voiding with pharmacologic treatment for urge incontinence did not result in further significant clinical improvement.[4]

Two studies have estimated the effectiveness of prompted voiding programs in reducing the frequency of FI, thereby assessing the extent to which immobility and dementia contribute to this condition. In one study, residents significantly increased the number of appropriate defecations and significantly decreased UI in an intervention that offered toileting assistance every 2 hours. Although this intervention did not decrease significantly the frequency of FI, there

was a trend in this direction.[8] The second trial involved a comprehensive intervention that integrated toileting assistance (prompted voiding), a fluid-prompting protocol, and mobility exercise.[3] This program resulted in a significant decrease in FI episodes (from 0.6 to 0.3 episodes per day) and a significant increase in appropriate fecal voiding in the toilet. Residents also significantly increased fluid intake, decreased UI, and showed improvements in mobility endurance. However, constipation remained a significant problem, because 46% of the residents had no defecations (continent or incontinent) during a 2-day period when bowel movements were monitored. The lack of significant difference between the intervention and control groups in frequency of defecations during this period suggests that constipation was not alleviated by the intervention. Neither of these trials controlled for laxative use, medications with constipating side effects, or caloric intake, which was known to be very low. Anorectal function measures also were not available for the patients who participated in the above trials that measured FI.

In regard to prevention, it is important to note that residents who improved with prompted voiding did so within the first several days of the trial. This quick response suggests that prompted voiding was not effective because it taught residents new behaviors associated with continence. The more plausible explanation is that residents were primarily incontinent because of the failure of caregivers to provide care (prompting and assistance) that would have prevented incontinence or at least would have reduced its severity. If NH residents who could be responsive to toileting assistance were identified soon after admission and treated with prompted voiding, it is likely that most of the UI and FI in NHs could be prevented. Unfortunately, there are several reasons why such prevention programs are not implemented in NHs.

Preventing Incontinence in Nursing Home Residents

Ample data indicate that NH physicians and care providers do not provide good incontinence care. In regard to physicians, a review of charts in 10 NHs revealed that the most basic assessments recommended in practice guidelines to identify reversible causes of incontinence were seldom done.[9] Most importantly, physicians did not write orders to evaluate a resident's potential preferences for and responsiveness to toileting assistance.[9] This is a particularly egregious error, since there is good evidence that a resident's responsiveness to toileting assistance can be predicted accurately with a brief trial of toileting assistance.[6]

As for other NH care providers, residents with good memory have reported that toileting assistance occurs at an average rate of 0–2 episodes per day, with many residents reporting long waits for assistance and that they are reluctant to ask for toileting assistance because of staff reactions.[9] Direct care staff report that they do not change or toilet people consistently because of lack of time. Furthermore, there is evidence that medical record documentation of incontinence care provision by nursing aides is erroneous.[10,11] In short, the labor and staff management dynamics of preventing UI and FI do not offer providers an incentive to implement programs known to prevent or at least improve UI and FI.

Summary and Future Directions

It is clear that behavioral and caregiver management interventions must be a first line treatment for UI and FI. In the NH, both conditions are at least exacerbated and largely caused by the resident's inability to use the toilet. In FI, it is less clear if these behavioral interventions will be as effective as they are with UI due to the unknown role of constipation and dyssynergia.

Management and regulatory interventions at the organizational level will need to be implemented if UI and FI are to be prevented in the NH setting.

References

1. Chiang L, Ouslander J, Schnelle JF, Reuben DB. Dually incontinent nursing home residents: clinical characteristics and treatment differences. *J Am Geriatr Soc.* 2000;48:673–676.

2. Schnelle JF, MacRae PG, Ouslander JG, Simmons SF, Nitta M. Functional incidental training, mobility performance, and incontinence care with nursing home residents. *J Am Geriatr Soc.* 1995;43:1356–1362.

3. Schnelle JF, Alessi CA, Simmons SF, Al-Samarrai NR, Beck JC, Ouslander JG. Translating clinical research into practice: a randomized controlled trial of exercise and incontinence care with nursing home residents. *J Am Geriatr Soc.* 1995;43:1356–1362.

4. Ouslander JG., Schnelle JF, Uman G, Fingold S, Nigam JG, Tuico E. Does oxybutynin add to the effectiveness of prompted voiding for urinary incontinence among nursing home residents? *Am Geriatr Soc.* 1995;43:610–617.

5. Johanson JF, Irizarry F, Doughty A. Risk factors for fecal incontinence in a nursing home population. *J Clin Gastroenterol.* 1997;24:156–160.

6. Ouslander JG, Schnelle JF, Uman G, Fingold S, Nigam JG, Tucio C. Predictors of successful prompted voiding among incontinent nursing home residents. *JAMA.* 1995;273:1366–1370.

7. Leung WF, Beard MH, Grbic V, Habermann R, Rao SSC, Schnelle JF, Sepulveda ACC, VAGLAHS, UCLA; Center for Quality Aging, Vanderbilt VUMC, U Iowa. *Dyssynergia—key pathophysiologic mechanism for fecal incontinence (FI) in nursing home residents.* Paper presented at the American College of Gastroenterology Annual Scientific Meeting; October 13, 2007; Philadelphia, PA.

8. Ouslander JG, Simmons S, Schnelle JF, Uman G, Fingold S. Effects of prompted voiding on fecal continence among nursing home residents. *J Am Geriatr Soc.* 1996;44:424–428.

9. Schnelle JF, Cadogan MP, Yoshii J, Al Samarrai NR, Osterweil D, Bates-Jensen BM, Simmons SF. The Minimum Data Set urinary incontinence quality indicators: do they reflect differences in care processes related to incontinence? *Med Care.* 2003;41:909–922.

10. Lekan-Rutledge D, Palmer MH, Belyea M. In their own words: nursing assistants' perception of barriers to implement of prompted voiding in long-term care. *Gerontologist.* 1998;38:370–378.

11. Schnelle JF, Bates-Jensen BM, Chu L, Simmons SF. Accuracy of nursing home medical record information about care process delivery: implications for staff management and improvement. *J Am Geriatr Soc.* 2004;52:1378–1383.

Surgical Complications Including Prostatectomy and Other Urologic Procedures

Patricia S. Goode, M.D., M.S.N.

Prostatectomy for Cancer

Prevalence of urinary incontinence (UI). Determining rates of incontinence, the basis of determining risk, is particularly difficult due to inconsistent and nonstandardized measures. The Prostate Cancer Outcomes Study, a population-based, longitudinal cohort study of 1,288 African-American, White, and Hispanic men after radical prostatectomy, found incontinence rates of 13% preoperatively, with 3% using pads. At 1 and 5 years postoperatively, 65% had any incontinence at both time points, with 31% and 26% respectively using absorbent pads.[1] Far fewer men had frequent incontinence, 13% at 1 year and 11% at 5 years postoperatively.

Risk factors for postprostatectomy UI include older age, preoperative UI, previous transurethral resection of the prostrate (TURP), lack of preservation of both neurovascular bundles, total length of the posterior urethra, length of the urethral stump, presence of anastomotic stricture, and periurethral fibrosis on magnetic resonance imaging (MRI).[2–5]

Preventive interventions. Introduction of the anatomical surgical technique described by Walsh resulted in a decrease in the median time to continence from 5.6 to 1.5 months, and the rate of continence at 24 months increased from 82% to 95%.[2] A randomized study of placement of two anastomotic urethral suspension stitches increased Valsalva leak point pressure and postoperative continence rates.[6] A similar study from Japan demonstrated that the placement of an endopelvic anterior urethral stitch shortened time to continence.[7] Robotic-assisted laparoscopic prostatectomy is thought to have the potential for reducing postoperative complications, but more evidence is needed.

Three randomized, controlled clinical trials of perioperative pelvic floor muscle training programs have shown a significant reduction in duration and severity of incontinence after prostatectomy.[8–10] The studies varied in the intensity of the intervention, but produced continence rates of 88% vs. 56% at 3 months,[8] 83.4% vs. 47.5% at 12 months,[9] and median time to continence of 3.5 months vs. >6 months.[10] In the latter study, the number needed to treat to get one additional man out of pads by 6 months was 5, with a cost of $150 per man treated.[10] This is extremely cost-effective.

Prevalence of fecal incontinence (FI) has been shown to increase slightly after radical prostatectomy. A study of 665 men treated with radical prostatectomy reported, after a median of 2.6 and 6.2 years: fecal urgency in 3% and 5% vs. 4% and 1% for controls, and FI in 1% and 2% vs. 0% and 0% for controls.[11] Another study showed 2% of men with new or worsened FI 2 years after radical prostatectomy.[12]

Research priorities in prevention of postprostatectomy incontinence include: (1) use of standardized measures of incontinence and quality of life, so that results can be compared across trials; (2) continued innovation in surgical techniques; (3) determination of optimal timing, content, and intensity for pelvic floor muscle training; (4) addition of FI outcomes to intervention trials to prevent and treat postprostatectomy UI, (5) exploration of mechanisms that lead to UI, e.g., periurethral fibrosis, to develop preventive interventions.

Radiation Therapy for Prostate Cancer

External Beam Radiation

Prevalence of UI. The Late Effects Consensus Conference of 1995 established that the incidence of incontinence with pelvic external beam radiation therapy (RT) was 0%–10%, depending on the definition used.[13] In addition to UI, overactive bladder symptoms of urgency, frequency, and nocturia can be bothersome to patients and have considerable impact on quality of life. Dysuria, frequency, and urgency incidence was 23%–80% but usually subsided several weeks following RT.[13] The latency between RT and the onset of clinical symptoms can complicate the attribution of symptoms. The interval between RT and onset of bladder and urethral complications had a median time of 13–28 months.[13] In a series of 301 patients 2–3 years after RT, 35% reported incontinence, but only 6% required pads or other protective devices.[14]

Incontinence risk factors in a series of 1,192 prostate cancer patients treated with external beam RT 5 years previously included TURP before RT (10% vs. 6%, p=0.03), presence of Grade >2 acute genitourinary (GU) toxicity (11% vs. 5%, p=0.002), and TURP or dilatation after RT (8% vs. 1.5%, p=0.002).[15] There are no studies on interventions to prevent incontinence during or following RT.

Brachytherapy

Prevalence of incontinence. Brachytherapy (BT), the permanent implantation of interstitial radioactive seeds to treat prostate cancer, has been associated with incident overactive bladder symptoms and UI and FI. A French study of 308 men undergoing BT reported 63.9% of patients with urinary urgency, 66.0% diurnal frequency, and 62.8% nocturnal frequency at 2 months after seed implantation.[12] Although urinary symptoms reached a maximum at 2 months, they were still worse than baseline at 24 months including reports of urgency in 37.9%, diurnal frequency 36.8%, and nocturnal frequency in 30.8%. New or worsened UI was reported by 19.7% of men and FI in 8.9% at 24 months after seed implantation.[12]

Risk factors. Risk stratification for urinary tract morbidity, using the American Urological Association symptom score, flow rate, prostate volume, and postvoid residual volume, successfully separated a group at higher risk for GU morbidity (37% vs. 15%).[16]

Fecal Incontinence With External Beam Radiation and Brachytherapy

Both RT and BT for prostate cancer seem to be risk factors for incident fecal urgency and FI. In a study of 147 men who had undergone RT and 84 who had undergone BT (44 of whom had received supplemental external-beam RT as part of their primary therapy) at a median of 2.6 and 6.2 years after primary therapy, bowel symptoms measured by the Expanded Prostate Cancer Index Composite (EPIC) were as follows: percent with fecal urgency at 2.6 and 6.2 years: 14% and 14% respectively with RT, 19% and 10% for BT, and 4% and 1% for controls; percent with FI at 2.6 and 6.2 years: 4% and 9% with RT, 8% and 3% for BT, and 0% and 0% for controls.[11] As in UI, men with bowel symptoms prior to RT were at higher risk after RT, with an odds ratio of 7.4.[17]

Research priorities for external beam RT and BT include: (1) more clearly defining the risks for overactive bladder and UI and FI related to RT/BT, both acutely and 2 or more years following RT/BT, (2) determining if risk factors for UI and FI are different for external beam RT vs. BT,

(3) determining the optimal protocols for RT to achieve optimal cancer control and prevent overactive bladder and bowel symptoms as well as UI and FI, (4) determining if pelvic floor muscle training and multicomponent behavioral therapy programs can decrease the incidence of RT-associated overactive bladder, UI, and FI, and, if so, optimizing the protocols; (5) testing pharmacologic and dietary protocols to optimally decrease UI and FI, both alone and in combination with pelvic floor muscle training.

Transurethral Resection of the Prostate (TURP)

Prevalence. Procedures for benign prostatic hypertrophy (BPH) very infrequently result in new UI and more frequently resolve or reduce incontinence. In a study of 151 men, UI prevalence was 12% for daily leakage preoperatively and 2.8% postoperatively.[18]

Risk factors for incident incontinence following TURP are (1) less compliant bladder and detrusor overactivity on preoperative urodynamics,[19] (2) prior RT (external beam, BT, or particularly both),[20] (3) advanced local prostate cancer,[21] (4) neurologic diseases such as Parkinson's.[22] The prevalence of FI has not been shown to be affected by TURP. Only one study examined pelvic floor muscle exercises before TURP and showed increased strength but no clinically relevant changes in storage or voiding symptoms.[23]

Research priorities are: (1) establish the true predictive value of preoperative urodynamics for post-TURP incontinence, particularly in older patients and in those with neurologic diseases, (2) develop alternative treatments or modification in TURP surgical procedures for obstructive voiding symptoms and urinary retention in men who have had prior RT, (3) further identification of other high-risk groups for post-TURP incontinence.

Surgery for Bladder Cancer

Prevalence. Much less commonly performed than radical prostatectomy or TURP, radical cystectomy is indicated for muscle-invasive bladder carcinoma, recurrent T1 disease, or carcinoma in situ (CIS) unresponsive to intravesical chemotherapy or immunotherapy. Surgeons and patients may choose between ileal conduits and neobladders. One summary of the results of ileal neobladders reported total or good daytime continence rates in 88%–95% of patients, and some degree of nighttime leakage in most patients, but satisfactory nighttime continence rates of 66%–93%.[24] In 259 patients completing a Functional Assessment of Cancer Therapy instrument, quality of life did not differ between patients with bladder cancer having undergone radical cystectomy and those with an intact bladder or between continent and conduit urinary diversion groups.[25] Another study compared urinary and fecal symptoms in patients after RT for bladder cancer to symptoms in controls. UI occurring at least once a month was reported by 30% vs. 10% and FI at least monthly in 17% vs. 2%.[26]

Risk factors for urinary incontinence in orthotopic neobladder included maximum urethral closure pressure, maximum contraction amplitude, and baseline pressure at mid capacity.[27] In considering primary prevention, cigarette smoking is the most important risk factor for bladder cancer.

Research priorities include: Continuing to develop innovations in surgical, medical, and radiation therapy to optimally control bladder cancer and preserve continence.

References

1. Penson D, McLerran D, Feng Z, Li L, Albertsen P, Gilliland F, Hamilton A, Hoffman R, Stephenson R, Potosky A, Stanford J. 5-Year urinary and sexual outcomes after radical prostatectomy: results from the Prostate Cancer Outcomes Study. *J Urol.* 2005;173:1702–1705.

2. Eastham J, Kattan M, Rogers E, Goad J, Ohori M, Boone T, Scardino P. Risk factors for urinary incontinence after radical prostatectomy. *J Urol.* 1996;156:1707–1713.

3. Moore K, Truong V, Estey E, Voaklander D. Urinary incontinence after radical prostatectomy—can men at risk be identified preoperatively? *J Wound Ostomy Continence Nurs.* 2007;34:270–279.

4. Majoros A, Bach D, Keszthelyi A, Hamvas A, Mayer P, Riesz P, Seidl E, Romics I. Analysis of risk factors for urinary incontinence after radical prostatectomy. *Urol Int.* 2007;78:202–207.

5. Kordan Y, Alkibay T, Sozen S, Bozkurt Y, Acar C, Talu T, Bozkirli I. Is there an impact of postoperative urethral and periurethral anatomical features in post-radical retropubic prostatectomy incontinence. *Urol Int.* 2007;78:208–213.

6. Campenni M, Harmon J, Ginsberg P, Harkaway R. Improved continence after radical retro-pubic prostatectomy using two pubo-urethral suspension stitches. *Urol Int.* 2002;68:109–112.

7. Sugimura Y, Hioki T, Yamada Y, Fumino M, Inoue T. An anterior urethral stitch improves urinary incontinence following radical prostatectomy. *Int J Urol.* 2001;8:153–157.

8. Van Kampen M, De Weerdt W, Van Poppel H, De Ridder D, Feys H, Baert L. Effect of pelvic floor re-education on duration and degree of incontinence after radical prostatectomy: a randomized controlled trial. *Lancet.* 2000;355:98–102.

9. Manassero F, Traversi C, Ales V, Pistolesi D, Panicucci E, Valent F, Selli C. Contribution of early intensive prolonged pelvic floor exercises on urinary continence recovery after bladder neck-sparing radical prostatectomy: results of a prospective controlled randomized trial. *Neurourol Urodynam.* 2007 May 8; Epub ahead of print.

10. Burgio KL, Goode PS, Urban D, Umlauf M, Locher J, Bueschen A, Redden DT. Preoperative biofeedback assisted behavioral training to decrease post-prostatectomy incontinence: a randomized, controlled trial. *J Urol.* 2006;175:196–201.

11. Miller D, Sanda M, Dunn R, Montie J, Pimentel H, Sandler H, McLaughlin W, Wei J. Long-term outcomes among localized prostate cancer survivors: health-related quality-of-life changes after radical prostatectomy, external radiation, and brachytherapy. *J Clin Oncol.* 2005;23:2772–2780.

12. Buron C, Le Vu B, Cosset J, Pommier P, Peiffert D, Delannes M, Flam T, Guerif S, Salem N, Chauveinc L, Livartowski A. Brachytherapy versus prostatectomy in localized prostate cancer: results of a French multicenter prospective medico-economic study. *Int J Radiat Oncol Biol Phys.* 2007;67:812–822.

13. Marks L, Carroll P, Dugan T, Anscher M. The response of the urinary bladder, urethra, and ureter to radiation and chemotherapy. *Int J Radiat Oncol Biol Phys.* 1995;31:1257–1280.

14. Little D, Kuban D, Levy L, Zagars G, Pollack A. Quality-of-life questionnaire results 2 and 3 years after radiotherapy for prostate cancer in a randomized dose-escalation study. *Urology.* 2003;62:707–713.

15. Liu M, Pickles T, Berthelet E, Agranovich A, Kwan W, Tyldesley S, McKenzie M, Keys M, Morris J, Pai H. Urinary incontinence in prostate cancer patients treated with external beam radiotherapy. *Radiother Oncol.* 2005;74:197–201.

16. Wehle M, Lisson S, Buskirk S, Broderick G, Young P, Igel T. Prediction of genitourinary tract morbidity after brachytherapy for prostate adenocarcinoma. *Mayo Clin Proc.* 2004;79:314–317.

17. Peeters S, Heemsbergen W, Van Putten W, Slot A, Tabak H, Mens J, Lebesque J, Koper P. Acute and late complications after radiotherapy for prostate cancer: results of a multicenter randomized trial comparing 68 Gy to 78 Gy. *Int J Radiat Oncol.* 2005;61:1019–1034.

18. Deliveliotis C, Liakouras C, Delis A, Skolarikos A, Varkarakis J, Protogerou V. Prostate operations: long-term effects on sexual and urinary function and quality of life. Comparison with an age-matched control population. *Urol Res.* 2004;32:283–289.

19. Ameda K, Koyanagi T, Nantani M, Taniguchi K, Matusno T. The relevance of preoperative cystometrography in patients with benign prostatic hyperplasia: correlating the findings with clinical features and outcome after prostatectomy. *J Urol.* 1994:152:443–447.

20. Kollmeier M, Stock R, Cesaretti J, Stone N. Urinary morbidity and incontinence following transurethral resection of the prostate after brachytherapy. *J Urol.* 2005;173:808–812.

21. Gnanapragasam V, Kumar V, Langton D, Packard R, Leung H. Outcome of transurethral prostatectomy for the palliative management of lower urinary tract symptoms in men with prostate cancer. *Int J Urol.* 2006;13:711–715.

22. Chandiramani V, Palace J, Fowler C. How to recognize patients with Parkinsonism who should not have urological surgery. *Br J Urol.* 1997;80:100–104.

23. Tibaek S, Klarskov P, Hansen B, Thomsen H, Andresen H, Jensen C, Olsen M. Pelvic floor muscle training before TURP: a randomized, controlled, blinded study. *Scand J Urol Nephrol.* 2007;41:329–334.

24. Hautmann R, Volkmer B, Schumacher M, Gschwend J, Studer U. Long-term results of standard procedures in urology: the ileal neobladder. *World J Urol.* 2006;24:305–314.

25. Allareddy V, Kennedy J, West M, Konety B. Quality of life in long-term survivors of bladder cancer. *Cancer.* 2006;106:2355–2362.

26. Henningsohn L, Wijkstrom H, Dickman P, Bergmark K, Steineck G. Distressful symptoms after radical radiotherapy for urinary bladder cancer. *Radiotherapy Oncol.* 2002;62:215–225.

27. Koraitim M, Atta M, Foda M. Orthotopic bladder substitution in men revisited: identification of continence predictors. *J Urol.* 2006;176:2081–2084.

Risk Factors for the Development of Fecal and Urinary Incontinence Following Anorectal Surgery, Colorectal Surgery, and Radiation Therapy for Colorectal Cancer

Ann C. Lowry, M.D., F.A.C.S., FASCRS

Fecal incontinence impacts between 2% and12% of the general population. Although the etiology is often complex and poorly understood, fecal incontinence may be an unintended consequence of the treatment of common anorectal conditions and colorectal cancer in some patients.

Anorectal Conditions

In some anorectal diseases, incontinence is an element of the disease process (rectal prolapse, rectocele, sphincter injury) and may or may not improve after treatment of the condition. Incontinence is not generally associated with the common anorectal conditions of hemorrhoids, fissure, and fistulas but may occur after surgical treatment.

Hemorrhoids

Hemorrhoids affect approximately 10–15 million people in the United States; 10%–20% of whom are estimated to require surgery. Surgical options include Lord's dilatation, conventional hemorrhoidectomy, and stapled hemorrhoidopexy. One randomized trial compared dilatation to conventional hemorrhoidectomy; in long-term follow-up, 52% of patients reported some incontinence after dilatation.[1] Using endoanal ultrasound before and after dilatation, investigators found new internal sphincter defects in 76% of patients and external sphincter defects in 24%.[2]

In most case series of conventional hemorrhoidectomy, incontinence rates vary from 0.5%–20 %.[1,3,4] Stapled hemorrhoidopexy is a new technique in which a circumferential ring of excess rectal mucosa and submucosa is excised with a transanal stapler. Prospective data on postoperative incontinence comes primarily from randomized trials comparing stapled and conventional hemorrhoidectomy with short follow-up. In the studies reporting incontinence, the rate for both groups ranges between 0% and 3%.[5] It is unclear if the techniques differ in rates of incontinence. A Cochrane review found a nonsignificant trend toward increased soiling and incontinence with stapled hemorrhoidopexy.[5] Occult sphincter defects, use of retractors, and poor technique have been proposed as risk factors for incontinence.[6–8]

Anal Fissure

Most anal fissures respond to medical management; however, 20%–50% of chronic fissures persist. The standard therapy is a lateral internal sphincterotomy in which a portion of the internal sphincter is divided to reduce the anal resting tone. Fecal incontinence occurs postoperatively in 0%–38%; incontinence of flatus and soiling are more common than accidental bowel movements.[7,9,10] New medical therapies and Botox injection into the anal sphincter aim to heal fissures without the risk of incontinence. Although these methods are more effective than placebo, the healing rates are lower than those of lateral internal sphincterotomy.[11–13] The

technique of surgery (open versus closed), extent of sphincterotomy, and gender may influence rate of incontinence.[10,14,15]

Anal Fistulas

Fistulotomy cures fistulas but, depending upon the anatomy, may result in incontinence in 0%–44%; staged fistulotomy does not change the rates significantly.[16] Alternative modalities such as insertion of fibrin glue, a fistula plug, and endorectal advancement flaps are less successful in resolving the fistula but less likely to cause incontinence.

Technique	Success	Incontinence
Fibrin glue	14%–60%	0
Fistula plug	41%–85%	0
Advancement flaps	54%–93%	0%–35%[17–23]

The type of fistula, sphincter injury, occult defects, a short anal canal, age, and gender may affect the risk of incontinence.[16,24]

Rectal Cancer

Incontinence after treatment of rectal cancer may result from surgical treatment, either anterior resection or local excision, or adjuvant therapy. Incontinence of some degree is reported in 28%–49% of patients after anterior resection.[25,26] Loss of reservoir function and damage to the sphincter muscles are causative factors. Construction of a “neo-reservoir,” either a colonic J pouch or coloplasty, is designed to improve postoperative function. Randomized studies show better function at 6 months but no difference by 2 years.[27] A meta-analysis and multicenter randomized trial both report improved results with a J pouch.[28,29]

Local excision via either transanal excision or transanal endoscopic microsurgery (TEM) may be appropriate for early stage rectal cancer. Incontinence is reported in 20%–37% of patients and tends to improve with time.[30–32] At least for TEM, damage to the internal sphincter with a drop in resting pressure is noted.[32]

Local recurrence is lessened by adjuvant chemoradiation in regionally extensive rectal cancers. However, radiation increases the risk of incontinence posttreatment. In randomized studies of surgery with and without short-course radiation, the incidence of incontinence is approximately double in the group receiving radiation.[33,34] Functional results of long-course radiation utilized most commonly in the United States have not been studied in a randomized trial. Whether improved radiation techniques decrease the frequency of incontinence remains to be seen.

Conclusion

Treatments for common anorectal conditions and rectal cancer clearly have functional consequences. Prevention of the conditions is the most effective way of avoiding the problem; improved surgical or radiotherapy techniques and better identification of patients at risk may improve the results when treatment is required.

References

1. Konsten J, Baeten CG. Hemorrhoidectomy vs. Lord's method: 17-year follow-up of a prospective, randomized trial. *Dis Colon Rectum.* 2000;43:503–506.

2. Stamatiadis A, Konstantinou E, Theodosopoulou E, Mamoura K. Frequency of operative trauma to anal sphincters: evaluation with endoanal ultrasound. *Gastroenterol Nurs.* 2002;25:55–59.

3. Johannsson HO, Graf W, Pahlman L. Long-term results of haemorrhoidectomy. *Eur J Surg.* 2002;168:485–489.

4. McConnell JC, Khubchandani IT. Long-term follow-up of closed hemorrhoidectomy. *Dis Colon Rectum.* 1983;26:797–799.

5. Jayaraman S, Colquhoun PH, Malthaner RA. Stapled versus conventional surgery for hemorrhoids. *Cochrane Database Syst Rev.* 2006:CD005393.

6. Felt-Bersma RJ, van Baren R, Koorevaar M, Strijers RL, Cuesta MA. Unsuspected sphincter defects shown by anal endosonography after anorectal surgery. A prospective study. *Dis Colon Rectum.* 1995;38:249–253.

7. Tjandra JJ, Han WR, Ooi BS, Nagesh A, Thorne M. Faecal incontinence after lateral internal sphincterotomy is often associated with coexisting occult sphincter defects: a study using endoanal ultrasonography. *Austr N Z J Surg.* 2001;71:598–602.

8. van Tets WF, Kuijpers JH, Tran K, Mollen R, van Goor H. Influence of Parks' anal retractor on anal sphincter pressures. *Dis Colon Rectum.* 1997;40:1042–1045.

9. Casillas S, Hull TL, Zutshi M, Trzcinski R, Bast JF, Xu M. Incontinence after a lateral internal sphincterotomy: are we underestimating it? *Dis Colon Rectum.* 2005;48:1193–1199.

10. Garcia-Aguilar J, Belmonte C, Wong WD, Lowry AC, Madoff RD. Open vs. closed sphincterotomy for chronic anal fissure: long-term results. *Dis Colon Rectum.* 1996;39:440–443.

11. Richard CS, Gregoire R, Plewes EA, et al. Internal sphincterotomy is superior to topical nitroglycerin in the treatment of chronic anal fissure: results of a randomized, controlled trial by the Canadian Colorectal Surgical Trials Group. *Dis Colon Rectum.* 2000;43:1048–1057; discussion 1057–1058.

12. Nelson R. Non surgical therapy for anal fissure. *Cochrane Database Syst Rev.* 2006:CD003431.

13. Brown CJ, Dubreuil D, Santoro L, Liu M, O'Connor BI, McLeod RS. Lateral internal sphincterotomy is superior to topical nitroglycerin for healing chronic anal fissure and does not compromise long-term fecal continence: six-year follow-up of a multi-center, randomized, controlled trial. *Dis Colon Rectum.* 2007;50:442–448.

14. Elsebae MM. A study of fecal incontinence in patients with chronic anal fissure: prospective, randomized, controlled trial of the extent of internal anal sphincter division during lateral sphincterotomy. *World J Surg.* 2007;31:2052–2057.

15. Wiley M, Day P, Rieger N, Stephens J, Moore J. Open vs. closed lateral internal sphincterotomy for idiopathic fissure-in-ano: a prospective, randomized, controlled trial. *Dis Colon Rectum.* 2004;47:847–852.

16. Vasilevsky CA, Gordon PH. Benign anorectal: abscess and fistula. In: Wolff BG. Fleshman JW, Beck DE, Pemberton JH, Wexner SD, eds. *The ASCRS Textbook of Colon and Rectal Surgery.* New York: Springer, 2007;192–214.

17. Ortiz H, Marzo J. Endorectal flap advancement repair and fistulectomy for high trans-sphincteric and suprasphincteric fistulas. *Br J Surg.* 2000;87:1680–1683.

18. Champagne BJ, O'Connor LM, Ferguson M, Orangio GR, Schertzer ME, Armstrong DN. Efficacy of anal fistula plug in closure of cryptoglandular fistulas: long-term follow-up. *Dis Colon Rectum.* 2006;49:1817–1821.

19. Chew SS, Adams WJ. Anal sphincter advancement flap for low transsphincteric anal fistula. *Dis Colon Rectum.* 2007;50:1090–1093.

20. Golub RW, Wise WE, Jr., Kerner BA, Khanduja KS, Aguilar PS. Endorectal mucosal advancement flap: the preferred method for complex cryptoglandular fistula-in-ano. *J Gastrointest Surg.* 1997;1:487–491.

21. Johnson EK, Gaw JU, Armstrong DN. Efficacy of anal fistula plug vs. fibrin glue in closure of anorectal fistulas. *Dis Colon Rectum.* 2006;49:371–376.

22. van Koperen PJ, D'Hoore A, Wolthuis AM, Bemelman WA, Slors JF. Anal fistula plug for closure of difficult anorectal fistula: a prospective study. *Dis Colon Rectum.* 2007;August, E-pub ahead of print.

23. Zmora O, Mizrahi N, Rotholtz N, et al. Fibrin glue sealing in the treatment of perineal fistulas. *Dis Colon Rectum.* 2003;46:584–589.

24. Zbar AP, Beer-Gabel M, Chiappa AC, Aslam M. Fecal incontinence after minor anorectal surgery. *Dis Colon Rectum.* 2001;44:1610–1619; discussion 1619–1623.

25. Ho P, Law WL, Chan SC, Lam CK, Chu KW. Functional outcome following low anterior resection with total mesorectal excision in the elderly. *Int J Colorectal Dis.* 2003;18:230–233.

26. Rasmussen OO, Petersen IK, Christiansen J. Anorectal function following low anterior resection. *Colorectal Dis.* 2003;5:258–261.

27. Ho YH, Brown S, Heah SM, et al. Comparison of J-pouch and coloplasty pouch for low rectal cancers: a randomized, controlled trial investigating functional results and comparative anastomotic leak rates. *Ann Surg.* 2002;236:49–55.

28. Heriot AG, Tekkis PP, Constantinides V, et al. Meta-analysis of colonic reservoirs versus straight coloanal anastomosis after anterior resection. *Br J Surg. 2*006;93:19–32.

29. Fazio VW, Zutshi M, Remzi FH, et al. A randomized multi-center trial to compare long-term functional outcome, quality of life, and complications of surgical procedures for low rectal cancers. *Ann Surg.* 2007;246:481–488; discussion 488–490.

30. Dafnis G, Pahlman L, Raab Y, Gustafsson UM, Graf W. Transanal endoscopic microsurgery: clinical and functional results. *Colorectal Dis. 2*004;6:336–42.

31. Fenech DS, Takahashi T, Liu M, et al. Function and quality of life after transanal excision of rectal polyps and cancers. *Dis Colon Rectum.* 2007;50:598–603.

32. Herman RM, Richter P, Walega P, Popiela T. Anorectal sphincter function and rectal barostat study in patients following transanal endoscopic microsurgery. *Int J Colorectal Dis* 2001;16:370–376.

33. Peeters KC, van de Velde CJ, Leer JW, et al. Late side effects of short-course preoperative radiotherapy combined with total mesorectal excision for rectal cancer: increased bowel dysfunction in irradiated patients—a Dutch colorectal cancer group study. *J Clin Oncol.* 2005;23:6199–6206.

34. Pollack J, Holm T, Cedermark B, et al. Late adverse effects of short-course preoperative radiotherapy in rectal cancer. *Br J Surg. 2*006;93:1519–1525.

Iatrogenic Disorders, Drug Side Effects, and the Development of Urinary and Fecal Incontinence

Alan J. Wein, M.D., Ph.D. (Hon.)

More often than not, especially in the elderly or frail elderly patient, comorbidities, rather than a single discrete cause, contribute to the development or aggravation/worsening of both urinary incontinence (UI) and fecal incontinence (FI). These factors may be anatomic, physiologic, or pharmacologic. An iatrogenic contribution may result from the pharmacologic "side effects" of well-intentioned therapy. Pharmacokinetic (absorption, distribution, metabolism, clearance) issues, including drug–drug interactions, may significantly affect the unintended effects of drug therapy—again, especially in the elderly, who often retaking multiple medications. In considering the associations to be described, it should be noted that there is essentially no levels 1 or 2 evidence (Oxford guidelines) and opinions are based primarily on levels 4 and 3 evidence.

Urinary Incontinence

Drugs can contribute to stress (effort related) UI (SUI), urgency UI (UUI), overflow incontinence (OI), and functional incontinence (see Table 1 in abstract on "Pathophysiology of Urinary Incontinence," page 35). Any drug that depresses cognitive function and sensorium can contribute to **functional incontinence**. Agents which depress bladder contractility or increase outlet resistance may cause or worsen urinary retention and consequent **OI**. Agents which promote an increase in bladder tone or contractility can effectively decrease the volume threshold for detrusor overactivity, seemingly worsening **(UUI)**. The functional bladder capacity at which detrusor overactivity occurs can also be reduced by increasing the residual urine volume. Rapidly acting diuretics may aggravate UUI by increasing the rate of bladder filling and the frequency and intensity of afferent stimulation. Finally, any therapy which decreases outlet resistance can cause or aggravate **SUI**. A simple list is seen in Table 1.

Fecal Incontinence

At lease in the frail elderly, constipation is said to be the most important cause of FI and is cited as being prevalent, treatable, preventable, and frequently overlooked. The mechanism is hypothesized to be that a mass of hard stool decreases the ability to perceive the movement of new stool into the sigmoid or rectum, and reflexology dilates the internal anal sphincter, allowing liquid to escape. Agents which depress motility or increase stool consistency may act as offenders in this regard, causing so-called "overflow" FI. Loose stools (diarrhea) are hypothesized to be a risk factor for FI, especially in the elderly, by overwhelming an age-compromised sphincter mechanism. Table 2 lists pharmacologic possibilities for causing or contributing to FI.

Table 1. Agents Reported or Suspected To Cause or Contribute to New Onset or Worsening of Urinary Incontinence

Alpha-adrenergic antagonists	SUI*
Skeletal muscle relaxants	SUI
ACE inhibitors	SUI
Estrogen	SUI
Antipsychotic agents	SUI, UUI,† FI‡
Parasympathomimetic agents	UUI
Cholinesterase antagonists	UUI
Diuretics (short acting)	UUI
Agents causing constipation	UUI, OI§
Calcium channel blockers	OI, UUI
Antimuscarinic agents	OI
Alpha-adrenergic agonists	OI
Opioid analgesics	OI
Psychotropic drugs (sedatives, hypnotics)	OI, FI

*SUI, stress urinary incontinence

†UUI, urgency urinary incontinence

‡ OI, overflow incontinence

§FI, functional incontinence

Table 2. Agents Reported or Suspected To Cause or Contribute to New Onset or Worsening of Fecal Incontinence

Drugs causing diarrhea
Antibiotics
5HT4 agonists
Serotonin reuptake inhibitors (SRIs)
Protein pump inhibitors
Magnesium-containing antacids
Digoxin
Laxatives
Dietary ingredients or supplements causing diarrhea
Excess fiber
Lactose (in lactose intolerance)
Drugs causing constipation
Anticholinergic agents
Opiates
Iron supplements
Calcium channel antagonists

General References

1. Andersson K-E, Appel R, Cardozo L, Chapple C, Drutz H, Fourcroy J, Nishazawa O, Vela Navarette R, Wein AJ. Pharmacologic management of urinary incontinence. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence.* Paris, France: Health Publication Ltd.; 2005:809–854.

2. Fonda D, DeBeau CE, Harari D, Ouslandler JG, Palmer M, Roe B. Incontinence in the frail elderly. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence.* Paris, France: Health Publication Ltd.; 2005:1163–1240.

3. Norton C, Whitehead WE, Bliss DZ, Metsola P, Tries J. Conservative and pharmacological management of faecal incontinence in adults. In: Abrams P, Cardozo L, Khoury S, Wein A, eds. *Incontinence.* Paris, France: Health Publication Ltd.; 2005:1521–1564.

4. Thomas A, Woodard C, Rovner ES, Wein AJ. Urologic complications of nonurologic medications. In: Bewick C, Resnick MI, Seftel AD, eds. *Urologic Clinics North America.* Philadelphia, PA. Saunders/Elsevier; 2003:123–131.

Impact of Neurologic Disorders, Such as Stroke, Spinal Cord Injuries, and Other Neurological Conditions on the Development of Fecal and Urinary Incontinence

Arnold Wald, M.D.

Loss of voluntary control over bowel and bladder function frequently occurs in central nervous system (CNS) disorders. Fear of urinary incontinence (UI) and or fecal incontinence (FI) often results in social withdrawal and isolation. The issue of incontinence and its management has a significant impact on family members, caregivers, and community health services.

CNS disorders affect bowel function in a variety of ways, depending on the location and severity of damage, often resulting in variable loss of sensory and voluntary motor functions of the anorectum and urinary bladder. Whereas the enteric nervous system usually remains intact, lack of CNS modulation of the gastrointestinal tract may result in dysmotility, delayed colonic transit time, constipation, and anorectal dysfunction including FI.

The prevalence of bowel dysfunction in persons with CNS diseases is much higher than in the general population. It has been reported in as many as 70% of people with multiple sclerosis (MS), up to 75% of individuals with spinal cord injury (SCI), up to 68% of people with spina bifida, and up to 23% of persons after stroke.[1] These dysfunctions include both incontinence and constipation.

Despite the high prevalence and impact of FI and constipation in CNS disorders, evidence to support neurogenic bowel management is scanty, and programs for this patient group continue to rely on ritual, anecdote, and trial and error.

Bowel Dysfunction in Stroke

Prevalence estimates of FI in stroke survivors are somewhat lower than for UI but remain significant. Between 31% and 40% experience FI on hospital admission, 18% have FI upon discharge and between 7% and 9 % continue to have FI 6 months after their stroke.[2] Studies suggest that functional limitations are an important factor. FI is often more severe in stroke survivors than FI in age- and gender-matched populations without stroke. FI is often associated with increased morbidity and more social isolation for stroke survivors and their caregivers.

Urinary Dysfunction in Stroke

Prevalence estimates for UI in stroke survivors range from 32% to 79% upon hospital admission, decreasing to 25% to 28% at discharge and from 12% to 19% 6 months after the stroke.[2] Many stroke survivors have double incontinence, which is 4 times as high as in the population without stroke. UI falls into two categories: Urge UI and UI with reduced or no awareness of bladder filling before indication (IA-UI). IA-UI may be the strongest predictor of mortality and the need for institutional care at 3 months poststroke, in contrast to urge UI.[3] Poststroke UI is associated with decreased attentiveness, and those with IA-UI perform the poorest. In stroke survivors who recognize UI, one hypothesis is that primary treatment should aim to improve attentiveness, and improving processing speed as well as incorporating prompted voiding or perhaps pelvic floor muscle training.

Bowel Dysfunction in SCI

The number of persons with SCI in the United States has been estimated to be about 200,000; many are young and have an increasing life expectancy.[4] More than one-third of surveyed individuals with SCI rate bowel and bladder dysfunction as having the most important effect on their lives after injury, and many rank neurogenic bowel dysfunction as one of their major life-limiting problems.[5] Recent studies have defined three different neuropathophysiologic patterns associated with bowel dysfunction:[4] (1) present in >T7 injuries, is characterized by very high prevalence of constipation, substantial defecatory difficulty, and mild FI (Wexner score 4.5); (2) present in <T7, with preserved spinal reflexes, is characterized by some constipation, very substantial defecation difficulty, and mild FI (Wexner score 4.8); (3) present in <T7, with absent sacral reflexes, is characterized by modest constipation, less defecatory difficulty, and greater severity of FI (Wexner score 7.2). Identification of these patterns may be of help when designing therapeutic strategies. Many physicians who care for these patients, including gastroenterologists who are asked to consult for bowel dysfunctions, are poorly trained in this area and therefore lack the expertise to provide useful management advice to this population.

Urinary Dysfunction in SCI

Bladder dysfunction is common in patients with SCI as it is with suprapontine and subsacral disorders. Not only do these patients suffer from embarrassment, inconvenience, increased costs and burden of care, but persistently elevated detrusor pressure is associated with increased risk of upper urinary tract damage and complications.[6] Patients with SCI or spina bifida are at comparatively high risk for this, in contrast to its rarity in the MS population. Thus, a primary clinical objective of treatment of neurogenic lower urinary dysfunction is protection of the upper urinary tract by minimizing risk of pyelonephritis and stone disease, because renal failure continues to be a leading cause of mortality in patients with SCI who survive their initial injury.[7]

Because UI has little effect on mortality, it is often regarded as having a low priority in management programs, but this is shortsighted and overlooks the devastating effects that UI can have on patients in terms of confidence, physical and mental well-being, and overall quality of life. As with bowel dysfunction, nonurology specialists who care for these patients often lack interest or awareness of these problems associated with neurologic UI. This disinterest often results in suboptimal care of patients.

Bowel Dysfunction in Multiple Sclerosis

MS is a relatively common neurological disease affecting approximately 250,000 Americans. Onset of disease is often in the third or fourth decades, and 60% of patients are women. Both constipation and FI are common, and incontinence correlates strongly with the presence of genitourinary symptoms.

Anorectal sensory and motor abnormalities are common in patients with MS and FI. These include elevated thresholds of rectal sensory perception and impaired striated muscle contraction (external anal sphincter and puborectalis muscle), with preserved internal anal sphincter tone and function. Constipation may be protective of impaired continence mechanisms in this population and should not be treated aggressively.

Urinary Dysfunction in Multiple Sclerosis

Lower urinary tract dysfunction affects between 50% and 90% of people with MS sometime during the course of their disease.[8] Bladder storage problems often coexist with inadequate bladder emptying. The most common urodynamic finding is neurogenic detrusor overactivity, often with detrusor dyssynergia. Bladder dysfunction, however, does not necessarily correlate with urodynamic patterns or disease stages.

UI has important negative effects on quality of life. It is one of the main reasons that people stop working and may be a precursor to institutionalization. UI is associated with increased risk of falling in persons with urge incontinence who try to hurry to toilet facilities, and UI may be associated with comorbid conditions such as bed sores.

Although conservative treatment has been the mainstay in this population, neuromuscular electrical stimulation and intravesical injections of *Botulinum* toxin A have been shown to be beneficial, with improvement of quality of life.[9–11] *Botulinum* toxin A has been used to treat both detrusor overactivity and sphincter dyssynergia, but its use is limited because it is not reimbursed by Medicare and Medicaid.

References

1. Coggrave M, Wiesel PH, Norton C. Management of fecal incontinence and constipation in adults with central neurological diseases. *Cochrane Database Sys Rev.* 2007;CD002115.

2. Brittain K, Perry S, Shaw C, Matthews R, Jagger C, Potter J. Isolated urinary, fecal and double incontinence: prevalence and degree of soiling in stroke survivors. *J Am Geriatr Soc.* 2006;54:1915–1919.

3. Pettersen R, Saxby BK, Wyller TB. Poststroke urinary incontinence: one-year outcome and relationships with measures of attentiveness. *J Am Geriatr Soc.* 2007;55:1571–1577.

4. Valles M, Vidal J, Clave P, Mearin F. Bowel dysfunction in patients with motor complete spinal cord injury: clinical, neurological, and pathophysiological associations. *Am J Gastroenterol.* 2006;101:2290–2299.

5. Stiens SA, Bergman SB, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: clinical evaluation and rehabilitative management. *Arch Phys Med Rehabil.* 1997;78:S86–S102.

6. Denys P, Corcos J, Everaert K, et al. Improving the global management of the neurogenic bladder patient: Part I. The complexity of patients. *Curr Med Res Opin.* 2006;22:359–365.

7. Hinds JP, Wald A. Colonic and anorectal dysfunction associated with multiple sclerosis. *Am J Gastroenterol.* 1989;84:587–595.

8. Litwiler SE, Frohman EM, Zimmern PE. Multiple sclerosis and the urologist. *J Urol.* 1999;161:743–757.

9. McClurg D, Ashe RG, Lowe-Strong AS. Neuromuscular electrical stimulation and the treatment of lower urinary tract dysfunction in multiple sclerosis—a double blind, placebo controlled, randomized clinical trial. *Neurourol Urodynam.* 2007;e-pub ahead of print.

10. Schurch B, Denys P, Kozma CM, et al. Botulinum toxin A improves the quality of life of patients with neurogenic urinary incontinence. *Eur Urol.* 2007;52:850–859.

11. Denys P, Corcos J, Everaert K, et al. Improving the global management of the neurogenic bladder patient: Part II. Future treatment strategies. *Curr Med Res Opin.* 2006;22:851–860.

Impact of Depression and Other Psychiatric Conditions on the Development of Fecal and Urinary Incontinence

William D. Steers, M.D.

A large body of epidemiological evidence from clinic- or community-based populations suggests that psychiatric disorders such as depression and anxiety are several fold more common in men and women with lower urinary tract complaints, especially overactive bladder, urinary incontinence (Table 1), and bowel disorders (especially irritable bowel syndrome) compared to the general population. Clinicians treating individuals with these urologic and bowel complaints should be aware of the potential impact of these conditions on patient management. The bidirectional nature of these relationships implies that not only can urologic and bowel conditions cause emotional distress, but that patients with depression and anxiety are either at risk of developing lower urinary and bowel complaints or are more prone to seek medical attention. Basic research provides intriguing evidence for potential shared neurochemical pathways for stress, depression, and altered bladder/bowel function, relying on a central stress network. Key neurotransmitters include serotonin and corticotropin-releasing factor (CRF) (Figure 1). The ability of stress to alter promoter regions in genes regulating receptors for these transmitters provides exciting clues as to how severe stress due to sexual or physical abuse leads to psychiatric conditions and associated autonomic dysfunction. Moreover, bladder and bowel disorders often coexist in part due to common central control and local networks. Failure to adequately manage incontinence or to meet therapeutic goals in these patients may be due, in part, to an inability to target the underlying pathology. Conversely, failure to recognize and treat these emotional disorders may cause poorer outcomes in treating incontinence due to unrealistic expectations or failure in communication. Working in collaboration with mental health professionals in the management of concomitant emotional disorders and bladder and bowel disorders may improve patient care.

Table 1. Studies Reporting the Relationship Between Depression and Urinary Incontinence

Study	Dx Depression	N	Age (years)	Odds Ratio[a]	95% CI
Zorn et al., 1999[1]	BDI >12 and history	115 men and women	58 (mean)	2.3 5.2[b]	1.0–5.0 2.3–11.7
Black et al., 1998[2]	CES-D Scale >16	258 women	>65	1.94	1.46–2.59
Valvanne et al.,1996[3]	DSM-III	651 men and women	>75	4.5	2.3–8.8
Dugan et al., 2000[4]	Screener for depression	668 men and women	>60	1.45	1.01–2.09
Woo et al., 2006[5]	GDS ≥8	1,611 men and women	>70	0.58	0.16–2.12
Melville et al., 2002[6]	PRIME-MD PHQ	218 women	18–90	9.2[c] 13.5[d]	1.8–48.0 3.0–61.5

Table 1. Studies Reporting the Relationship Between Depression and Urinary Incontinence (continued)

Melville et al., 2005[7]	PRIME-MD PHQ	3,536 women	30–90	2.7[e] 3.8[f]	1.1–6.6 1.6–9.1
Nygaard et al., 2003[8]	Composite International Diagnostic Interview (CIDI)	5,701 women	50–69	1.82[g] 1.41[h]	1.26–2.63 1.06–1.87
Nygaard et al., 2003[8]	CES-D	5,701 women	50–69	1.33[g] 1.12[h]	0.91–1.94 0.83–1.51
Bogner et al., 2002[9]	GHQ (psychological distress)	781 men and women	50+	1.74[i] 1.56[j]	1.13–2.68 1.00–2.43
Vigod and Stewart, 2006[10]	CIDI-SF	69,003 women	18+	5.73	3.11–10.54

[a]Odds ratios were calculated for the likelihood of having depression, given the presence of incontinence. Odds ratios (95% confidence intervals (CI)) where the lower confidence limit is greater than 1.00 indicate a statistically significant increase in the likelihood of having depression.

[b]Cases of idiopathic urge incontinence.

[c]Cases of urge incontinence compared with stress incontinence (referent group).

[d]Cases of mixed incontinence compared with stress incontinence (referent group).

[e]Moderate incontinence compared with mild incontinence (as measured by Sandvik).

[f]Severe incontinence compared with mild incontinence (as measured by Sandvik).

[g]Severe incontinence compared with no incontinence, controlling for confounders.

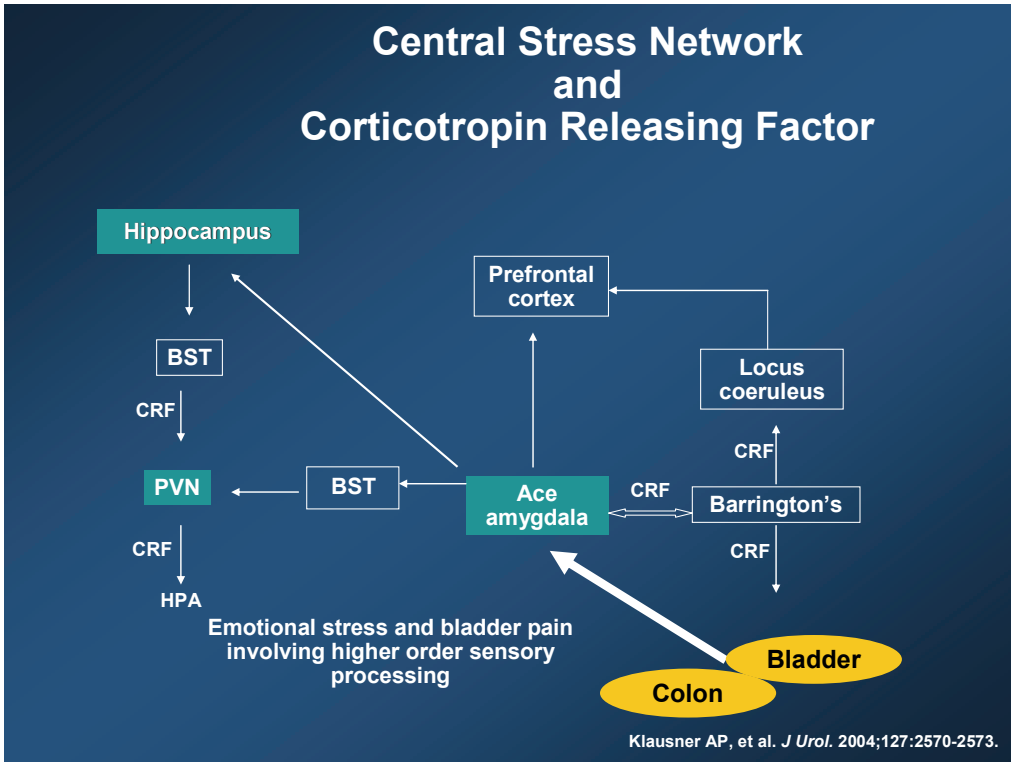
[h]Mild incontinence compared with no incontinence, controlling for confounders.

[i]Unadjusted.

[j]Adjusted for age, gender, ethnicity, and education.

BDI, Beck Depression Inventory; CES-D, Center for Epidemiological Studies-Depression; CIDI-SF, Composite International Diagnostic Interview-Short Form; DSM-III, Diagnostic and Statistical Manual III; GDS, Geriatric Depression Scale; GHQ, General Health Questionnaire; PRIME-MD PHQ, Primary Care Evaluation of Mental Disorders Patient Health Questionnaire.

Figure 1. Central Stress Network and Corticotropin-Releasing Factor*



*The central nucleus of the amygdala (Ace), bed nucleus of stria terminalis (BST), prefrontal cortex and paraventricular nucleus of the hypothalamus (PVN) form the central stress network. Corticotropin-releasing factor (CRF)-expressing projections from the Ace to Barrington's nucleus are activated during stress. Barrington's nucleus, which contains the pontine micturition center, sends CRF projections to locus coeruleus and the sacral spinal cord. This network may exert a profound influence on voiding in response to stressful stimuli. HPA, hypothalamic-pituitary-adrenal axis.

References

1. Zorn BH, Montgomery H, Pieper K, Gray M, Steers WD. Urinary incontinence and depression. *J Urol.* 1999;162:82–84.

2. Black SA, Goodwin JS, Markides KS. The association between chronic diseases and depressive symptomatology in older Mexican Americans. *J Gerontol A Biol Sci Med Sci.* 1998;53:M188–M194.

3. Valvanne J, Juva K, Erkinjuntti T, Tilvis R. Major depression in the elderly: a population study in Helsinki. *Int Psychogeriatr.* 1996;8:437–443.

4. Dugan E, Cohen SJ, Bland DR, Preisser JS, Davis CC, Suggs PK, McGann P. The association of depressive symptoms and urinary incontinence among older adults. *J Am Geriatr Soc.* 2000;48:413–416.

5. Woo J, Ho SC, Lau J, Yuen YK, Chiu H, Lee HC, Chi I. The prevalence of depressive symptoms and predisposing factors in an elderly Chinese population. *Acta Psychiatr Scand.* 1994;89:8–13.

6. Melville J, Walker E, Katon W, et al. Prevalence of comorbid psychiatric illness and its impact on symptom perception, quality of life, and functional status in women with urinary incontinence. *Am J Obstet Gynecol.* 2002;187:80–87.

7. Melville JL, Fan MY, Newton K, Fenner D. Fecal incontinence in US women: a population-based study. *Am J Obstet Gynecol.* 2005;193:2071–2076.

8. Nygaard I, Turvey C, Burns TL, Crischilles E, Wallace R. Urinary incontinence and depression in middle-aged United States women. *Obstet Gynecol.* 2003;101:149–156.

9. Bogner HR, Gallo JJ, Swartz KL. Anxiety disorders and disability secondary to urinary incontinence among adults over age 50. *Int J Psychiatry Med.* 2002;32:141–154.

10. Vigod SN, Stewart SD. Major depression in female urinary incontinence. *Psychosomatics.* 2006;47:147–151.

General References

11. Hyman PE, Cocjin J. Colon motility during a panic attack. *Psychosom Med.* 2005;67:616–617.

12. Klausner AP, Streng T, Na YG, Raju J, Batts TW, Tuttle JB, Andersson KE, Steers WD. The role of corticotropin releasing factor and its antagonist, astressin, on micturition in the rat. *Autonomic Neurosci.* 2005;123:26–35.

13. Lee KS, Dean McKinney T, Tuttle J, Steers WD. Alterations in voiding frequency and cystometry in the clomipramine-induced model of endogenous depression. *J Urol.* 2003;170:2067–2071.

14. Link C, Lutfey KE, Steers W, McKinlay JB. Is abuse causally related to urologic symptoms? Results from the Boston Area Community Health (BACH) survey. *Eur J Urol.* 2007;52:397–456.

15. Littlejohn JO Jr, Kaplan SA. An unexpected association between urinary incontinence, depression and sexual dysfunction. *Drugs Today.* 2002;38:777–782.

16. Melville JL, Delaney K, Newton K, Katon W. Incontinence severity and major depression in incontinent women. *Obstet Gynecol.* 2005;106:585–592.

17. Pavcovich LA, Valentino RJ. Central regulation of micturition in the rat the corticotrophin-releasing hormone from Barrington's nucleus. *Neurosci Lett.* 1995;196:185–188.

18. Penza KM, Heim C, Nemeroff CB. Neurobiological effects of childhood abuse: implications for the pathophysiology of depression and anxiety. *Arch Womens Ment Health.* 2003;6:15–22.

19. Steers WD, Herschorn, S, Kreder KJ, Moore K, Strohhehn K, Yalcin I, Bump RC. Duloxetine compared with placebo for treating women with symptoms of overactive bladder. *Brit J Urol Int.* 2007;100:337–345.

20. Watson AJ, Currie I, Curran S, Jarvis GJ. A prospective study examining the association between the symptoms of anxiety and depression and severity of urinary incontinence. *Eur J Obstet Gynecol Reprod Biol.* 2000;88:7–9.

Evidence-Based Practice Center Presentation III: Prevention, Screening, and Interventions for Urinary Incontinence and Fecal Incontinence

Robert L. Kane, M.D., Tatyana Shamliyan, M.D.

Clinical interventions to reduce urinary incontinence (UI) have been extensively reviewed by the Cochrane Incontinence Review Group, the International Consultation on Incontinence, and the Agency for Healthcare Research and Quality. Trials to reduce incidence and progression of UI targeted adults with risk factors of UI including obesity; pregnancy, parity, and hormone changes related to age in women; and prostate diseases in men. Most studies examined short-term curative effects of treatments in participants with incontinence. The basis for measuring successful treatment varied across the studies that examined different interventions.

Studies reported from 1989 through May 2007 were reviewed from searches of MEDLINE® via PubMed®, CINAHL, Cochrane databases, and manual searches of reference lists from systematic reviews and the proceedings of the International Continence Society. Study quality was analyzed using the following criteria: subject selection, length and loss of followup, intention-to-treat principle, masking the treatment status, randomization scheme, adequacy of randomization and allocation concealment, and justification of sample sizes.

Urinary Incontinence

We identified 248 randomized controlled trials (RCTs) that that examined the effects of clinical interventions on UI and included 192 RCTs that reported patient outcomes. Urinary continence after various interventions was defined as ability to control urination, self-reported in voiding diaries and standardized questionnaires (96 RCTs); negative pad test (23 RCTs); negative cotton swab test (2 RCTs); negative cough stress test (34 RCTs); or their combinations.

The effects of clinical interventions on UI in pregnant women (primary prevention). Continence rates after intensive exercise care and self-administered perineal massage were comparable to usual care. Pelvic floor muscle training (PFMT) with biofeedback and electrostimulation, started at 9 weeks after vaginal delivery, resulted in continence 10 times more often compared to usual care at 10 months of follow-up.

The effects of clinical interventions on UI in males with urological diseases (primary prevention). Two of eight trials reported significant increases in continence after active conservative treatments (pelvic floor rehabilitation that included verbal explanations, palpation, and Kegel exercises) compared to usual care. The majority of men with prostate diseases who were followed for 3–12 months after surgery were continent.

Effects of behavioral intervention on UI (primary prevention). The continence rates after a behavioral modification program implemented in 359 postmenopausal, continent women 55 years and older to prevent UI were the same as usual care at 12 months of follow-up.

Efficacy of PFMT compared to usual care (secondary prevention). PFMT resulted in continence in women more often than usual care did in 4 of 10 trials. The largest relative benefit on continence was observed after electromyography (EMG)-assisted biofeedback with PFMT compared to usual care in postmenopausal women with stress UI taking hormone replacement

therapy. Pooled relative benefits of PFMT (relative risk [RR]=7.1, 95% confidence interval [CI]=2.8-18) and PFMT combined with biofeedback (RR=11.2, 95% CI=2.2-56.4) were sensitive to one small RCT with 2-month follow-up. PFMT combined with bladder training increased continent rates by 175% compared to usual care (pooled RR=1.8, 95% CI=1.1-2.9)

Effectiveness of nonsurgical interventions for UI. In women, the rate of continence after treatments for stress UI varied from less than 10% in an RCT of 37 women at 4 months of follow-up to 60% in an RCT of 52 women at 15 years of follow-up after PFMT. Smaller trials tended to report greater continence rates. Women with urodynamically diagnosed stress UI were continent four times more often 15 years after intensive PFMT supervised by a physical therapist compared to home exercise groups (RR=4.02, 95% CI=1.54-10.53). Individualized PFMT and bladder training significantly increased continence rates by 158% compared to group exercises in 530 women after 12 months of followup.

Electrical stimulation for secondary prevention of UI. Urinary continence 1–6 months after electrical stimulation in women was improved in only one of six RCTs that reported patient outcomes.

Neuromodulation for secondary prevention of UI. In community-dwelling adults, this treatment showed significant relative benefit in one trial.

Injectable bulking agents for secondary prevention of UI. Bulking agents showed significant benefit in only one of four trials that reported patient outcomes.

The effects of hormone therapy on UI in women. Hormone therapy apparently has paradoxical effects. It improves continence when used topically, but hormone replacement therapy increases incontinence.

Urinary continence after surgical interventions. Among examined gynecological surgeries, intrafascial total abdominal hysterectomy significantly reduced the risk of urge UI compared to the extrafascial approach. Total or subtotal abdominal hysterectomy resulted in comparable continence rates 1 year after the surgery. The rate of continence was above 75% in the majority of RCTs of vaginal tension tapes and sling procedures. All RCTs compared the effectiveness of vaginal tapes and sling procedures to other treatments; cure rates were comparable. Among prolapse surgery to prevent UI, the continence rates varied substantially after the same procedures: from 3.1% to 85.1% after laparoscopic colposuspension and from 8.7% to 93.3% after laparoscopic Burch procedures.

Improvement in UI after clinical interventions. The majority of RCTs reported improvement in UI after vaginal tape and sling procedures, with rates from 2.2% after transobturator suburethral sling procedure to 100% after transvaginal antimicrobial synthetic mesh, depending on definitions of improvement. The rates were higher for stress UI after transvaginal antimicrobial synthetic mesh vesicourethral suspension by allogenic sling and suprapubic arc sling. The rates were lower (19.4%) after the same procedure on quantitative improvement in stress UI defined as a decrease of >50% in urine loss. A significant relative benefit was shown in one RCT that compared suburethral slingplasty with the suprapubic arc to intravaginal sling in 195 patients with urodynamic stress UI refractory to conservative management (RR 2.7, 95% CI 1.3-5.5). Improvement in self-reported UI and the pad weight test was reported in 12 RCTs of surgery for UI. One RCT reported improvement in more than 80% of women after laparoscopic colposuspension and Burch open colposuspension. No significant relative benefit was detected when different treatments were compared.

The effects of clinical intervention on UI. Some evidence suggested the preventive effects of clinical interventions on UI. Intensive lifestyle therapy to lose and maintain at least 7% of initial body weight and to engage in moderate-intensity physical activity reduced stress UI in women. PFMT reduced UI in pregnant women.

Fecal Incontinence

For fecal incontinence (FI), we identified 121 studies (118 RCTs and 3 multicenter trials) that examined FI in adults; 87 (84 RCTs) reported patient outcomes. We focused solely on FI and did not address anal incontinence or flatus per se. Of four RCTs testing pelvic floor muscle exercises to prevent FI, one showed a significant increase in continence. Of four RCTs testing pelvic floor muscle exercises to improve FI, one showed a significant benefit.

Among pregnant women, one RCT of PFMT with biofeedback showed no significant effect. Another RCT with self-administered perineal massage daily from the 34th or 35th week of pregnancy until delivery showed no significant effect. Assessment by nurses after delivery, with conservative advice on PFMT, reduced the risk of FI at 1 month but not at 6 years.

One RCT of diet supplemented with 25 g of psyllium/day showed no significant benefit after 1 month. An RCT of topical phenylephrine also showed no significant benefit after 1 month. An RCT of loperamide hydrochloride (Imodium®) for 7 days before surgery did not reduce FI after abdominal proctocolectomy. Three trials of botulinum toxin showed no effect, nor did a trial of isosorbide-5-mononitrate gel.

Early diagnosis of sphincter tear immediately after vaginal delivery, with endoanal ultrasonography followed by surgical repair, reduced FI. When end-to-end sphincter repair was compared with overlapping sphincter repair in seven RCTs, six reported patient outcomes with inconsistent benefit from end-to-end technique.

Artificial bowel sphincter reduced FI in a multicenter, prospective, nonrandomized clinical trial of 115 patients. However, 25% of patients experienced postsurgical infection that required surgical revision, and 37% needed reimplantations. None of nine RCTs that compared different surgical techniques for hemorrhoidectomy reported significant relative FI benefits. Of five RCTs comparing sphincterotomy with local administration of pharmacological agents, none reported significant relative benefit on continence or FI. Surgical procedures in anal sphincter in patients with high trans-sphincteric fistula, idiopathic fissure-in-ano, anorectal abscess, or neurogenic FI reduced progression of FI in one RCT of patients with idiopathic fissure-in-ano when closed sphincterotomy was compared to open.

Two trials of active anal stimulation yielded no significant benefits. One RCT of sacral nerve continuous stimulation showed improvement in FI.

Screening

If the basis of screening is response to a questionnaire and the definition of incontinence is a person's report, then testing the questionnaire's ability to detect can be tautological. Few studies have compared a questionnaire used in a general population with a diagnostic evaluation.

Urinary Incontinence Detection

Of the 15 studies that screened for UI in women by using scales and questionnaires, 13 studies involved clinic samples. The Positive Predictive Likelihood Ratio (PPLR) (how much the probability of disease increases if the test is positive) ranged from 1.3 to 15. The PPLR was higher for studies of older women 16.2 versus 2.7 for younger women.

Of the 19 studies screening for UI in women by using clinical history, 16 were conducted in clinic settings; 2 combined clinic and research samples; and 2 involve men.

The PPLR mean for younger women in clinics was 2.5 (12 studies), compared to 1.2 (2 studies) for older women in clinics.

Fecal Incontinence Detection

The Epidemiology of Prolapse and Incontinence Questionnaire demonstrated 87% sensitivity, 70% specificity, 61% positive predictive value, and a small PPLR (2.9) to diagnose FI in 294 enrolled women with pelvic floor prolapse compared to examiners' diagnosis.

The Bowel Symptom Questionnaire had 48.5% sensitivity, 79.2% specificity, 43.2% positive predictive value, and a small PPRL (2.3) to identify postpartum FI related to sonographic evidence of anoperineal trauma in 156 women.

Limitations

For incidence studies, baseline continence is assumed but not always explicit. Thus, it is not always clear when actual incidence is being tested. The inconsistencies in interventions used, populations, sampling strategies, and definitions make it difficult to achieve any level of useful summary.

Conclusions

Urinary Incontinence

PFMT and surgery have large effects; surgery is more likely to be curative. Sustained weight loss may play a useful role. Outcome measures varied widely: self-reported symptoms, signs, and improvement (versus actual continence); UI severity as assessed by voiding diaries; pad test weights; and condition-specific quality of life.

Fecal Incontinence

There is not yet strong evidence of strategies to prevent or ameliorate FI.

General

The criteria for deeming a treatment successful are not well established. Despite the ICS standardization document, the choice of outcomes should reflect the participant's perception of cure and quality of life rather than the provider's evaluation and testing through instrumentation. Studies are needed of subgroups by race, comorbidities, frailty, and concomitant treatments, as well as baseline pelvic floor dysfunctions.

General References

1. Albo ME, Richter HE, Brubaker L, Norton P, Kraus SR, Zimmern PE, Chai TC, Zyczynski H, Diokno AC, Tennstedt S, Nager C, Lloyd LK, FitzGerald M, Lemack GE, Johnson HW, Leng W, Mallett V, Stoddard AM, Menefee S, Varner RE, Kenton K, Moalli P, Sirls L, Dandreo KJ, Kusek JW, Nyberg LM, Steers W; Urinary Incontinence Treatment Network. Burch colposuspension versus fascial sling to reduce urinary stress incontinence. *N Engl J Med.* 2007;356:2143–2155.

2. Brown SR, Nelson RL. Surgery for faecal incontinence in adults. *Cochrane Database Syst Rev.* 2007;(2):CD001757.

3. Filocamo MT, Li Marzi V, Del Popolo G, Cecconi F, Marzocco M, Tosto A, Nicita G. Effectiveness of early pelvic floor rehabilitation treatment for post-prostatectomy incontinence. *Eur Urol.* 2005;48:734–738.

4. Glazener CM, Herbison GP, MacArthur C, Grant A, Wilson PD. Randomised controlled trial of conservative management of postnatal urinary and faecal incontinence: six year follow up. *BMJ.* 2005;330:337.

5. Janssen CC, Lagro-Janssen AL, Felling AJ. The effects of physiotherapy for female urinary incontinence: individual compared with group treatment. *BJU Int.* 2001;87:201–206.

6. Meyer S, Hohlfeld P, Achtari C, De Grandi P. Pelvic floor education after vaginal delivery. *Obstet Gynecol.* 2001;97:673–677.

Impact of Exercise, Diet, Lifestyle, and Smoking in the Setting of Continence

Kathryn L. Burgio, Ph.D.

Despite a large epidemiological literature that has identified risk factors for the development of incontinence, relatively little research has been conducted in the realm of prevention.

Pelvic Floor Muscle Exercise

Primary prevention of urinary incontinence (UI) has been investigated in three at-risk populations, using behavioral programs with pelvic floor muscle training and exercise: older women, childbearing women, and men undergoing radical prostatectomy.

Older Women. Diokno and colleagues designed a behavioral intervention for continent older women that involved group teaching sessions to educate the women about bladder function and teach pelvic floor muscle exercises. This was followed by individual sessions to evaluate and ensure proper exercise technique. A randomized trial of this intervention in continent older women demonstrated that fewer women in the intervention group subsequently developed UI.[1]

Childbearing Women. There is now a significant literature demonstrating that pelvic floor muscle training and exercise help prevent or attenuate incontinence in pregnant women and in the postpartum period.[2–7] In one randomized trial, women randomized to pelvic floor muscle exercise during pregnancy had lower rates of incontinence in late pregnancy and postpartum.[2] Other studies have documented the value of postpartum pelvic floor muscle exercises for reducing incontinence up to 12 months later.[4–7]

One of the strongest predictors of postpartum UI is UI during pregnancy. In fact, it has been reported as a risk factor for UI 5 years postdelivery.[8] This information opens the opportunity to better identify women who are experiencing UI during pregnancy, rather than merely waiting for the pregnancy to pass, and to counsel women about their increased risk and systematically offer conservative interventions to help prevent future UI.

Men Undergoing Radical Prostatectomy. Patient surveys have shown that 8%–56% of men report UI 1 year or more following radical prostatectomy.[9–11] Studies that have examined the effects of pelvic floor muscle training soon after surgery have yielded mixed results. In one trial, patients who were incontinent on day 15 after radical prostatectomy received weekly pelvic floor re-education for up to a year or a placebo therapy.[12] The treatment group showed advantages in duration and degree of incontinence. Other studies have not shown a benefit for training initiated 3 weeks,[13] 6 weeks,[14] or 8 weeks after surgery,[15] but were most likely underpowered.

A small body of literature exists demonstrating that preoperative behavioral training can reduce the duration and severity of postprostatectomy incontinence.[16,17] One randomized trial tested the effectiveness of a single preoperative session of biofeedback-assisted behavioral training to teach pelvic floor muscle control. Results showed that the intervention significantly reduced the duration, as well as the severity of UI in the 6-month postoperative period.[17] A similar trial that combined preoperative training and 3 months of postoperative treatment also found significant benefit compared to no intervention.[16] Future research might explore whether the impact of such

interventions would be greater if access to pre- and postoperative training were enhanced by integrating the program into urology practices.

Diet and Weight Loss

Obesity is now considered an established risk factor for UI and there is evidence that it places patients at increased risk for fecal incontinence (FI) as well. Studies of morbidly obese women report significant improvement in UI symptoms with large reductions in weight after bariatric surgery.[18,19] But significant improvements in UI have also been demonstrated with as little as 5% weight reduction in conventional weight-loss programs.[20] Considering the solid data on risk factors and the clinical weight-loss data, measures to prevent obesity reasonably could be expected to prevent UI in some women. A recent study has also demonstrated reductions in prevalence of FI from 19% to 9% with weight loss in patients undergoing bariatric surgery.[19]

Dietary Factors

It is widely believed that certain substances, such as caffeine, spicy foods, sugar substitutes, and carbonated beverages are bladder irritants and increase the occurrence or severity of urgency and UI. The best data exist for the role of caffeine. Studies have shown that caffeine increases detrusor pressure[21] and is a risk factor for detrusor instability.[22,23] In addition, elimination of caffeine from the diet has been shown to improve both stress and urge UI.[24–26]

Fecal impaction and constipation have been recognized as factors contributing to FI as well as to UI, particularly in nursing home populations.[27] Therefore, recommendations for adequate fluid and dietary fiber intake to maintain normal stool consistency and prompt attention to constipation may help to avoid FI as well as UI. Similarly, given the association between FI and diarrhea, dietary and pharmacological interventions to control diarrhea could be expected to help prevent FI. In general, data are lacking on the role of dietary factors in promoting or preventing incontinence.

Lifestyle: Healthy Bladder and Bowel Habits

It has long been thought that habitual frequent urination can contribute to reduced bladder capacity and lead to detrusor overactivity, urgency, and urge UI. Bladder training, which breaks the cycle of urgency and frequency by using consistent, incremental voiding schedules has been shown to improve UI in several clinical series and a randomized clinical trial.[28] Studies are lacking that track the natural history of people with various bladder habits. But, given the efficacy of bladder training, it is conceivable that identifying patients with frequent urination and teaching them about normal bladder habits, including normal frequency of urination, might help prevent the development of bladder control problems and warrants further study. It has also been suggested that prolonged voiding intervals, such as occur in employment situations with limited access to the toilet (nurses, teachers, production line workers), contribute to reduced bladder sensation and UI. Research is needed on the effects of prolonged voiding intervals, as well as the effects of establishing normal voiding habits.

Similarly, it is thought that certain bowel habits, like postponing bowel movements for long periods of time, can diminish sensation and contribute to development of FI. It would be reasonable to surmise that teaching normal bowel habits, such as establishing a routine for having bowel movements and responding to the urge to defecate, could have some value for prevention, but this approach requires further study.

Smoking

Although results have been mixed, the literature provides evidence for a link between smoking and UI as well as FI.[29–31] However, data are lacking on the effects of smoking cessation on incontinence.

References

1. Diokno AC, Sampselle CM, Herzog AG, et al. Prevention of urinary incontinence by behavioral modification program: a randomized controlled trial among older women in the community. *J Urol.* 2004;171:1161–1164.

2. Sampselle CM, Miller JM, Mims BL, Delancey JOL, Ashton-Miller JA, Antonakos CL. Effect of pelvic muscle exercise on transient incontinence during pregnancy and after birth. *Obstet Gynecol.* 1998;91:406–412.

3. Reilly ETC, Freeman RM, Waterfield MR, Waterfield AE, Steggles P, Pedlar F. Prevention of postpartum stress incontinence in primigravidas with increased bladder neck mobility: a randomized controlled trial of antenatal pelvic floor exercise. *BJOG.* 2002;109:68–76.

4. Wilson PD, Herbison GP, Glazener CMA, Lang G, Gee H, MacArthur C. Postnatal incontinence: a multicenter, randomized controlled trial of conservative treatment. *Neurourol Urodynam.* 1997;16:349–350.

5. Morkved S, Bo K. Effect of postpartum pelvic floor muscle training in prevention and treatment of urinary incontinence: a one-year follow-up. *BJOG.* 2000;107:1022–1028.

6. Glazener CM, Herbison GP, Wilson PD, et al. Conservative management of persistent postnatal urinary and faecal incontinence: a randomized controlled trial. *BMJ.* 2001;323:1–5.

7. Chiarelli P, Cockburn J. Promoting urinary continence in women after delivery: randomized controlled trial. *Br Med J.* 2002;324:1241–1243.

8. Viktrup L, Lose G. The risk of stress incontinence 5 years after first delivery. *Am J Obstet Gynecol.* 2001;185:52–87.

9. Fowler FJ, Barry MJ, Lu-Yao G, Roman MA, Wasson J, Wennberg JE. Patient-reported complications and follow-up treatment after radical prostatectomy. *Urology.* 1993;42:622–629.

10. Stanford JL, Feng Z, Hamilton AS, et al. Urinary and sexual function after radical prostatectomy for clinically localized prostate cancer: the Prostate Cancer Outcomes Study. *JAMA.* 2000;283:354–360.

11. Bishoff JT, Motley G, Optenberg SA, et al. Incidence of fecal and urinary incontinence following radical perineal and retropubic prostatectomy in a national population. *J Urol.* 1998;60:454–458.

12. Van Kampen M, De Weerdt W, Van Poppel H, DeTidder D, Feys H, Baert L. Effect of pelvic floor re-education on duration and degree of incontinence after radical prostatectomy: a randomized controlled trial. *Lancet.* 2000;355:98–102.

13. Mathewson-Chapman M. Pelvic muscle exercise/biofeedback for urinary incontinence after prostatectomy: an education program. *J Cancer Educ. 1*997;12:218–223.

14. Franke JJ, Gilbert WB, Grier J, Koch MO, Shyr Y, Smith JA Jr. Early post-prostatectomy pelvic floor biofeedback. *J Urol.* 2000;163:191–193.

15. Moore KN, Griffiths D, Hughton A. Urinary incontinence after radical prostatectomy: a randomized controlled trial comparing pelvic muscle exercises with or without electrical stimulation. *Br J Urol Int.* 1999;83:57–65.

16. Parekh AR, Feng MI, Kirages D, Bremner H, Kaswick J, Aboseif S. The role of pelvic floor exercises on post-prostatectomy incontinence. *J Urol.* 2003;170:130–133.

17. Burgio KL, Goode PS, Urban DA, et al. Preoperative biofeedback-assisted behavioral training to reduce post-prostatectomy incontinence: a randomized controlled trial. *J Urol. 2006*;175:196–201.

18. Bump R, Sugerman H, Fantl J, et al. Obesity and lower urinary tract function in women: effect of surgically induced weight loss. *Am J Obstet Gynecol.* 1992;166:392–397.

19. Burgio KL, Richter HE, Clements RH, Redden DT, Goode PS. Changes in urinary and fecal incontinence symptoms with weight loss surgery in morbidly obese women. *Obstet Gynecol.* in press.

20. Subak LL, Johnson CEW, Boban D, et al. Does weight loss improve incontinence in moderately obese women? *Intl J Urogyn.* 2002;13:40–43.

21. Creighton SM, Stanton SL. Caffeine: does it affect your bladder? *Br J Urol.* 1990;66:613–614.

22. Arya LA, Myers DL, Jackson ND. Dietary caffeine intake and the risk for detrusor instability: a case-control study. *Obstet Gynecol.* 2000;96:85–89.

23. Holroyd-Leduc JM, Straus SE. Management of urinary incontinence in women: scientific review. *JAMA.* 2004;291:986–995.

24. Tomlinson BU, Dougherty MC, Pendergast JF, Boyington AR, Coffman MA, Pickens SM. Dietary caffeine, fluid intake and urinary incontinence in older rural women. *Int Urogynecol J Pelvic Floor Dysfunct. 1*999;10:22–28.

25. Bryant CM, Dowell CJ, Fairbrother G. Caffeine reduction education to improve urinary symptoms. *Br J Nurs.* 2002;11:560–565.

26. Gray M. Caffeine and urinary continence. *J Wound Ostomy Continence Nurs.* 2001;28:66–69.

27. Ouslander JG, Schnelle JF. Incontinence in the nursing home. *Ann Intern Med.* 1995;122:438–439.

28. Fantl JA, Wyman JF, McClish DK, et al. Efficacy of bladder training in older women with urinary incontinence. *JAMA.* 1991;265:609–613.

29. Hannestad YS, Rortveit G, Daltveit AK, Hunskaar S. Are smoking and other lifestyle factors associated with female urinary incontinence? The Norwegian EPINCONT Study. *BJOG.* 2003;110:247–254.

30. Danforth KN, Townsend MK, Lifford K, Curhan GC, Resnick NM, Grodstein F. Risk factors for urinary incontinence among middle-aged women. *Am J Obstet Gynecol.* 2006;194:339–345.

31. Guise JM, Morris C, Osterweil P, Li H, Rosenberg D, Greenlick M. Incidence of fecal incontinence after childbirth. *Obstet Gynecol.* 2007;109:281–288.

Prevention of Fecal and Urinary Incontinence and the Strategies To Improve the Identification of Persons at Risk

Diane K. Newman, R.N.C., M.S.N., C.R.N.P., F.A.A.N.

Prevention of Fecal and Urinary Incontinence

Fecal incontinence (FI) and urinary incontinence (UI) are reasonably framed as public health problems, with an emphasis on primary prevention, because of their significant prevalence and chronicity and their preventable nature. Using this public health approach, key populations at risk of developing these conditions will be identified, risk factors demonstrated, and public awareness strategies developed to help individuals alter modifiable risk factors.[1] Although the evidence base for FI is more limited than that for UI, the conditions share many similarities with respect to risk and treatment, suggesting that similar benefits may derive from population-based strategies.[2] Primary prevention should be the goal of all healthcare professionals, as it means taking an active part in preventing the initial development of FI and UI.[3] Because the process of storing and expelling feces and urine is shaped by social rules for acceptable times and places for elimination, stigma is attached to incontinence.[4] Programs to increase health promotion and education about FI and UI may serve to deconstruct these barriers. Based on the literature, the 3rd International Consultation on Incontinence (ICI) recommended that (1) primary prevention studies should not be limited to individual interventions but also test the impact of population-based public health strategies, (2) pelvic floor muscle training (PFMT) should be a standard component of prenatal and postpartum care, (3) further randomized controlled trials should be conducted to test preventive effect of PFMT for men postprostatectomy, and (4) further investigation is warranted to assess the efficacy of PFMT and bladder training for primary prevention of FI and UI in older adults.[1]

Strategies To Improve the Identification of Persons at Risk

The majority of people who admit to FI and UI in prevalence surveys do not seek professional help. FI is a rarely talked about condition which can lead to severe embarrassment, isolation, anxiety, and depression.[5] One study reported that only 11% of affected women who were visiting a general gynecologist had sought care.[6] Healthcare professionals tend to underestimate the personal impact of FI. Therefore, it is imperative that they try to elicit symptoms of FI from their patients.

Help-seeking behavior for UI is not very different. Federal agencies have funded multiple research projects on the use of noninvasive behavioral treatments in the ambulatory and long-term-care setting. In a surge of increased Federal funding of UI research in the mid-1980s, significant development took place in the understanding of the prevalence, causation, assessment, and treatment of UI. This research demonstrated that UI is never normal, not even in elderly, institutionalized, demented persons; it outlines the causes of the problem and how to diagnose UI; and it concludes that UI is treatable, even curable, and can always be managed. However, the fact remains that individuals, primarily women, do not seek treatment.[7] Unless specifically addressed by a healthcare professional, most people are too embarrassed by their UI to seek medical help. UI usually comes to a healthcare provider's attention only when the patient complains of specific symptoms or when the patient complains of symptoms that are severe and unmanageable. However, this is not often. Also, many individuals believe that UI is an unavoidable component of aging or that treatment is ineffective or unavailable. To date,

studies estimate that at least 50% of women with UI do not ask for care.[8] A survey of women reported that only 38% initiated a conversation with a physician about incontinence.[9] Older adult women in a managed healthcare plan who had ready access to healthcare sought care, but women under age 55 did not.[10]

The lack of help-seeking behavior by individuals with FI and UI is a common phenomenon. Medical conditions that have greater stigma (and often are less life threatening) than FI and UI usually take a much longer time to be declared to a healthcare provider and even longer to family, friends, and others.[11]

One strategy for increasing FI and UI detection and screening is through education about persons at risk for developing FI and UI and guideline development. The ICI recommended compulsory inclusion of incontinence in the basic curriculum for physicians, nurses, physiotherapists, and allied health professionals.[1] FI and UI must be identified and preferably delivered as a separate topic, not fragmented between different modules of the educational curriculum. However, it is unclear if professional organizations, certification bodies, medical and nursing schools have accepted this challenge. In the past two decades, many U.S. Government agencies and professional organizations have issued practice guidelines on UI.[12–14] All of these guidelines were aimed at healthcare professionals and were intended to help standardize the assessment and management of UI in adults in both community and long-term-care settings, with an ultimate goal of improving the lives of persons with UI through early screening and treatment. None targeted screening for persons at risk. Although these readily available publications are widely quoted, they have failed to be included in the training of healthcare professionals or integrated into the practice of healthcare professionals. There is even some evidence to show that they have failed to increase screening or managing of UI by primary care providers[15] or to improve care in nursing homes.[16] Specialist education programs with relevant accreditation mechanisms (and planned periodic recredentialing) to safeguard patient interests need to be developed for urologists, gynecologists, gastroenterologists, specialist nurses, physical therapists, and others.

References

1. Newman DK, Denis L, Gruenwald I, Ee CH, Millard R, Roberts R, Sampselle C, Williams K. Continence promotion: prevention, education, and organisation for continence care. In Abrams PA, Cardozo L, Khoury S, Wein AJ, eds. *Incontinence: Proceedings from the Third International Consultation on Incontinence,* Plymouth, UK: Health Publication: 2005;35–72.

2. Miner PB. Economic and personal impact of fecal and urinary incontinence. *Gastroenterology.* 2004;126:S8–S13.

3. Sampselle CM, Palmer MH, Boyington AR, O'Dell KK, Wooldridge L. Prevention of urinary incontinence in adults: population-based strategies. *Nurs Res.* 2004;53:S61–S67.

4. Norton C. Nurses, bowel continence, stigma and taboos. *J Wound Ostomy Continence Nurs.* 2004;31:85–94.

5. Hawes SK, Ahmad A. Fecal incontinence: a woman's view. *Am J Gastroenterol.* 2006;101:S610–S617.

6. Boreham MK, Richter HE, Kenton KS, et al. Anal incontinence in women presenting for gynecologic care: prevalence, risk factors, and impact upon quality of life. *Am J Obstet Gynecol.* 2005;192:1637–1642.

7. Mardon RE, Halim S, Pawlson LG, Haffer SC. Management of urinary incontinence in Medicare managed care beneficiaries*. Arch Intern Med.* 2006;166:1128–1133.

8. Melville JL, Newton K, Fan M-Y, Katon W. Health care discussion and treatment for urinary incontinence in US women. *Am J Obstet Gynecol.* 2006;194:729–737.

9. Kinchen KS, Burgio K, Diokno AC, Fultz NH, Bump R, Obenchain R. Factors associated with women's decisions to seek treatment for urinary incontinence. *J Womens Health.* 2003;12:687–698.

10. Morrill M, Lukacz ES, Lawrence JM, Nager CW, Contreras R, Luber KM. Seeking healthcare for pelvic floor disorders: a population-based study. *Am J Obstet Gynecol.* 2007;197; e1–e6.

11. Fonda D, Newman DK. Tackling the stigma of incontinence—promoting continence worldwide. In Cardozo L, Staskin D, eds. *Textbook of Female Urology and Urogynecology.* 2nd ed. UK: Isis Medical Media, Ltd.;2006:75–80.

12. Center for Medicare & Medicaid Services. State operations manual, appendix PP—guidance to surveyors for long term care facilities. Tag F315, §483.25(d) Urinary Incontinence (Rev. 8, Issued: 06-28-05, Effective: 06-28-05, Implementation: 06-28-05): 208-223. Available at: http://www.cms.hhs.gov/manuals/Downloads/som107ap_pp_guidelines_ltcf.pdf. Accessed October 7, 2007.

13. Fantl JA, Newman DK, Colling J, et al. for the Urinary Incontinence in Adults Guideline Update Panel. *Urinary Incontinence in Adults: Acute and Chronic Management. Clinical Practice Guideline. No. 2. Update*. Rockville, Md: Agency for Healthcare Policy and Research; 1996. AHCPR Publication No. 96-0682.

14. Agency for Health Care Policy and Research. *Urinary Incontinence in Adults: Clinical Practice Guideline.* Rockville, Md: Public Health Service, U.S. Department of Health and Human Services; 1992. AHCPR Publication No. 92-0038.

15. Bland DR, Dugan E, Cohen SJ, Preisser J, Davis CC, McGann PE, Suggs PK, Pearce KF. The effects of implementation of the Agency for Healthcare Policy and Research urinary incontinence guidelines in primary care practices. *J Am Geriatr Soc.* 2003;51:979–984.

16. DuBeau CE, Ouslander, JG, Palmer, MH. Knowledge and attitudes of nursing home staff and surveyors about the revised federal guidance for incontinence care. *Gerontologist.* 2007;47:468–479.